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Bernard-Von Burrow Flap and Unilateral Webster Modification for Reconstruction of Upper Lip

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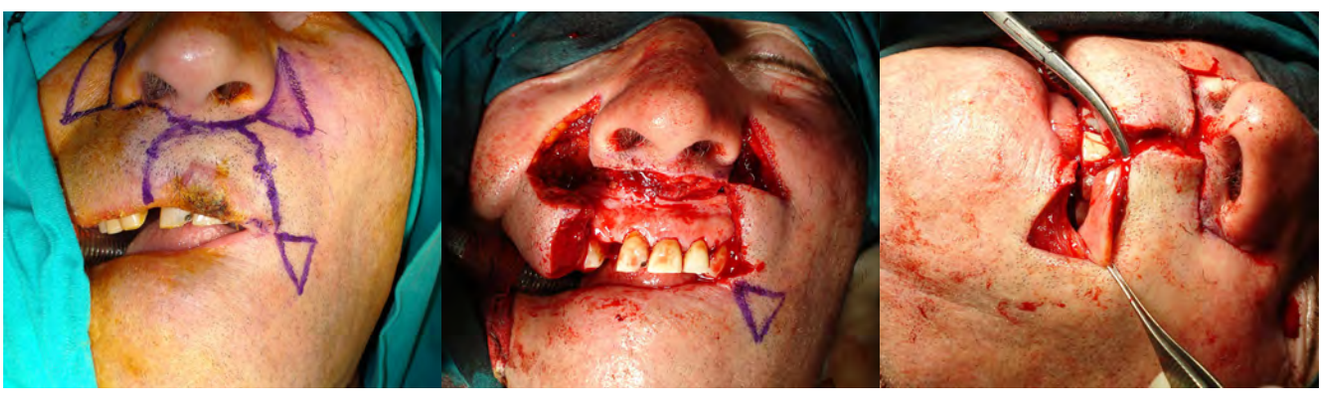

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JOURNAL OF CLINICAL AND ANALYTICAL MEDICINE

National Editorial Board

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Polytetrafluoroethylene-Covered Stent to Treat a Patient with Coronary Artery Aneurysm

Koroner Arter Anevrizması Olan Bir Olgunun Politetrafloroetilen Kaplı Stent ile Tedavisi

Treatment of Coronary Artery Aneurysm

Ekrem Aksu
Department of Cardiology, Kahramanmaras Necip Fazil City Hospital, Kahramanmaras, Turkey

Öz

Koroner arter anevrizması seyrek görülen, koroner anjiyografi sırasında tesadüfen tespit edilen bir anomalidir. En sık sebebi aterosklerozdur. Koroner arter anevrizması tedavisinde uzlaşı sağlanmış bir tedavi yöntemi yoktur. Tedavi planı yapılırken anevrizmanın büyüklüğü, eşlik eden rüptür, tromboz gibi komplikasyon durumu veya anevrizmanın bu olaylara yatkınlık durumu, eşlik eden koroner arter hastalığı göz önüne alınır. Koroner arter anevrizması tedavisinde konservatif yaklaşım, cerrahi girişim ve perkütan koroner girişim başlıca tedavi yöntemleridir. Makalemizde sirkumflex arterinde kritik darlığın eşlik ettiği koroner anevrizması olan, tedavisi politetrafloroetilen kaplı stentle yapılan bir vaka sunulmuş ve literatür gözden geçirilmiştir.

Anahtar Kelimeler

Koroner Arter Anevrizması; Koroner Anjiografi; Politetrafloroetilen Kaplı Stent

Abstract

Coronary artery aneurysms are uncommon anomalies, usually detected incidentally during coronary angiography. The most common cause of this anomaly is atherosclerosis. There is no consensus regarding the treatment of coronary aneurysms. The treatment plan should consider the magnitude of the aneurysm, accompanying rupture, complications such as thromboembolism or susceptibility to these events, and accompanying coronary artery disease. The main treatment methods are a conservative approach, surgical trial, and percutaneous coronary trial. In our article we present a patient with coronary artery aneurysm with a critical narrowing in the circumflex artery which was treated with a polytetrafluoroethylene coated stent, and review the literature.

Keywords

Coronary Artery Aneurysms; Coronary Angiography; Polytetrafluoroethylene Coated Stent

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Introduction

Coronary artery aneurysm is defined as dilatation of the coronary artery to a diameter more than 1.5 times that of the normal coronary artery segment. It is an uncommon anomaly. It is usually detected incidentally in patients undergoing coronary angiography [1]. The most common cause is atherosclerosis. Although rare, other reasons include inflammatory diseases, connective tissue diseases, infections, trauma, and congenital conditions [2].

Because it is a rare disease, there is no consensus on treatment of coronary artery aneurysm. Patients may be treated with a conservative approach that involves antiagregant and anticoagulant to prevent thrombus formation in the aneurysm and thromboembolism; a surgical approach involving aneurysm resection or ligation and coronary bypass surgery; or a percutaneous approach involving placement of graft-coated stent.

This study presents a case with circumflex coronary artery aneurysm accompanied by critical stenosis that was treated with polytetrafluoroethylene-covered stent placement, followed by review of the literature.

Case Report

A 66-year-old male patient who had no cardiovascular risk factors except for age was admitted to our clinic with class II exertional angina. On physical examination, blood pressure was 125/80 mmHg, pulse was 80/min, and cardiovascular and other system examinations were native. Routine blood tests, electrocardiogram, and transthoracic echocardiography did not detect any pathologic findings. Coronary angiography was chosen due to the patient's typical angina. Coronary angiography showed a saccular type aneurysm measuring 12*9mm in the proximal part of circumflex artery, noncritical stenosis in proximal, and 98% stenosis of critical lesion in distal (Figure 1). Percutaneous coronary intervention was planned. A 3.0*26mm bare metal stent (Liberte, Boston Scientific Corporation, USA) was placed at the distal lesion of the aneurysm. A 3.5*19mm polytetrafluoroethylene-covered graft stent (Direct-Stent, InSitu Technologies, USA) was placed at the aneurysm covering the proximal lesion and overlapping with distal stent placement. Post-dilatation was performed to overlap. Proximal optimization technique was applied using a 4.0*20mm noncompliant balloon (Mozec, Meril Life Sciences Pvt. Ltd., INDIA) inflated at 15 bars for 15 seconds. Full openness was achieved (Figure 2). The patient had stable clinical follow up and was discharged home with 100 mg acetylsalicylic acid (Coraspin, Bayer) per day lifelong and 75 mg clopidogrel (Baclan, Bayer) per day for six months. In the first, third, and 12 months of outpatient clinical controls the patient had no complaints. The patient had no pathological findings on physical examination and laboratory tests and was recommended for check-up every six months.

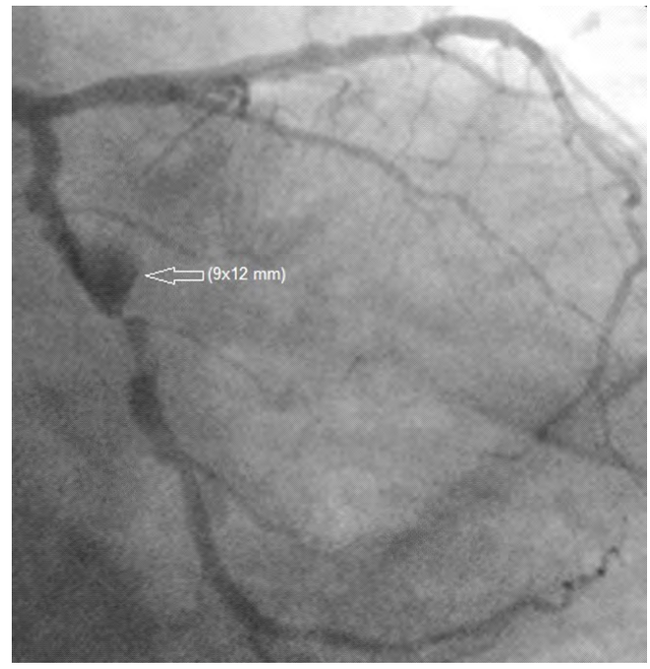

Figure 1. Saccular type circumflex artery aneurysm.

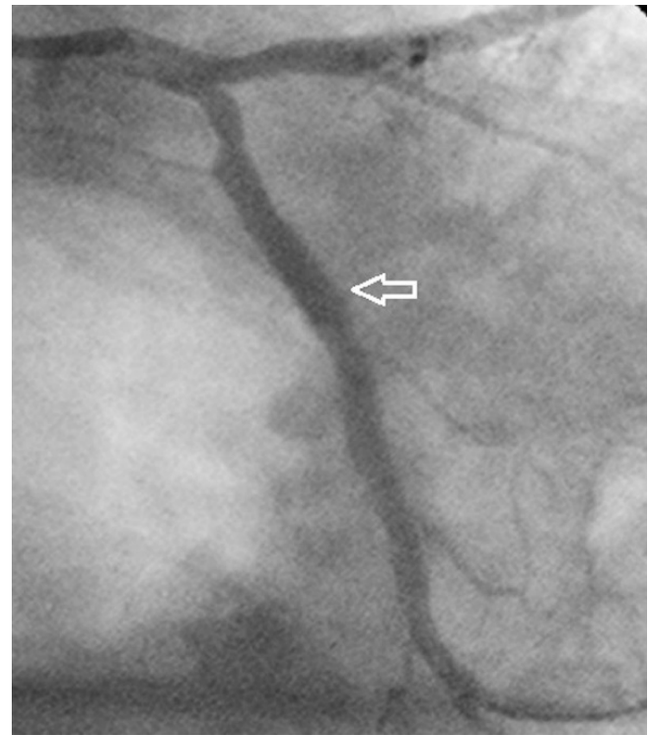

Figure 2. View of circumflex artery aneurysm after placement of polytetrafluoroethylene-covered graft stent.

Discussion

Coronary artery aneurysm is defined as dilatation of the coronary artery to a diameter more than 1.5 times that of the normal coronary artery segment diffusely or partly. It is an anomaly which is uncommon, asymptomatic, and most often incidentally detected during coronary angiography. The incidence ranges from 0.15% to 4.9% in different angiography series [1]. The most common cause is atherosclerosis. Rarely seen other reasons include inflammatory diseases such as Kawasaki disease, Takayasu disease, Systemic Lupus Erythematosus, connective tissue diseases such as Marfan syndrome, Ehler Danlos syndrome, infectious diseases such as Lyme disease, syphilis, and congenital conditions. In addition, trauma caused by interventional cardiology applications such as directional coronary atherectomy and coronary angioplasty has emerged as one of the rare causes [2]. This case was considered to be an aneurysm associated with atherosclerosis due to critical stenosis and accompanying vessel plaques.

There are two forms of coronary artery aneurysm: saccular and fusiform. Fusiform aneurysms are more common. Although sac-

cular aneurysms are seen less frequently, they are more susceptible to rupture, thrombosis, and fistulation [3]. The right coronary artery is the most commonly affected vessel. In order of frequency, the left anterior descending artery, the circumflex artery, and (rarely) the left main coronary artery are other affected vessels [4].

There is no consensus on treatment of coronary artery aneurysm. While planning the treatment, the size of the aneurysm, accompanying complications such as rupture, thrombosis, or susceptibility to these events, and accompanying coronary artery disease are taken into account. A conservative approach, surgery, and percutaneous coronary intervention are the main treatment methods. The treatment method for asymptomatic patients who have no accompanying complications such as compression, rupture, or thrombosis is a conservative approach including antiagregant therapy. The use of acetylsalicylic acid is recommended in patients with both coronary artery aneurysm accompanying coronary artery disease and isolated coronary artery aneurysm [5]. Surgical intervention is not recommended for aneurysms that are large, symptomatic, and accompanying cardiac pathology—especially coronary artery disease—due to risk of rupture or myocardial ischemia. Although there are many surgical methods, the basic principle is to maintain the repair or resection of the aneurysm along with providing continuity of coronary blood flow [6].

In saccular aneurysms that are not very wide or long, are symptomatic, and represent a high risk of thrombosis and embolism, placement of a polytetrafluoroethylene-covered stent is another method for patients suitable for percutaneous coronary intervention. Since 1990, clinical practices have started using polytetrafluoroethylene-covered stents because they are easy to implement and are a highly effective treatment method [7]. Use of these stents has taken a therapeutic place for most cases of iatrogenic coronary perforation and occasionally in the treatment of coronary artery aneurysm [8]. The most serious complication after placement of polytetrafluoroethylene-covered stent is the thrombosis of the stent due to reendothelization [7].

As in the case we're reporting, in which the aneurysm was not very wide, not accompanied with ischemic symptoms, and suitable for stent placement, percutaneous coronary intervention is an important alternative to surgical treatment. Because it shortens the length of hospitalization, is easily implemented, and is highly effective, percutaneous coronary intervention should be kept in mind in the treatment of coronary artery aneurysms.

Competing interests

The authors declare that they have no competing interests.

References

1. Espinola-Klein C, Rupprecht HJ, Erbel R, Nafe B, Brennecce R, Myer J. Ten-year outcome after coronary angioplasty in patients with single-vessel coronary artery disease and comparison with the results of the Coronary Artery Surgery Study (CASS). Am J Cardiol 2000;85(3):321-26.
2. Bruce F, Waller. Non-atherosclerotic coronary heart disease. In: Fuster V, Alexander RV, Rourke RA, editors. Hurst's the Heart. 10th ed. Philadelphia: McGraw-Hill Book Company, 2001;325-39.
3. Robinson FC. Aneurysms Of The Coronary Arteries. Am Heart j 1985;109:129-35.
4. Swaye PS, Fisher LD, Litwin P, Vignola PA, Judkins MP, Kemp HG, at al. Aneurysmal coronary artery disease. Circulation 1983;67(1):134-8.
5. al-Harthi SS, Nouh MS, Arafa M, al-Nozha M. Aneurysmal dilatation of the coronary arteries: diagnostic patterns and clinical significance. Int J Cardiol 1991;30(2):191-4.
6. Demirag MK, Keceligil HT, Yucel SM, Elmalı M. Aterosklerotik hastalığa eşlik eden sağ koroner arter anevrizması: Olgu sunumu. Türk Gögüs Kalp Damar Cerrahisi Dergisi 2009;17(3):214-7.
7. Briguori C, Sarais C, Sivieri G, Takagi T, Di Mario C, Colombo A. Polytetrafluoroethylene-covered stent and coronary artery aneurysms. Catheter Cardiovasc Interv 2002;55(3):326-30.
8. Lakova I, Colombo A. Treatment of a coronary aneurysm involving bifurcation stent system. Caheter Cardiovasc Interv 2005;64(2):169-72.

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Case Report
JCAM

Solitary Fibrous Tumor of the Pleura: A Report 5 Cases

Plevral Soliter Fibröz Tümörlü 5 Olgunun Sunumu

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Türk Toraks Derneği 19. Yıllık Kongresinde poster sunumu olarak sunulmuştur. 6-10 Nisan 2016 Antalya

Öz

Bu çalışmanın amacı, ender görülen plevral soliter fibroz tümörlerin tedavi ve takiplerinde nelere dikkat edilmesi gerektiğini saptamaktır. 2010 ile 2015 yılları arasında, kliniğimizde plevral soliter fibröz tümör tanısı alan 5 hastanın kayıtları geriye dönük olarak incelendi. Olguların yaş ortalaması 56,2 (40-66) idi. 2 olgu semptomatikti. Diğer 3 olgu tesadüfen çekilen radyolojik tetkikler sonucunda tanı almıştı. 2 olguda malign plevral soliter fibröz tümör tanısı konuldu ve bu 2 olguda erkekti. Ortalama tümör çapı 8,6 cm (5-13) idi. Bütün hastalara komplet rezeksiyon yapıldı. Hastaların hepsi düzenli takip edildi (9-75 ay). Sadece 1 hastada rekurrens izlendi. Plevranın soliter fibroz tümörleri nadir görülen neoplazmalardır ve büyük boyutlara ulaşabilir. Komplet cerrahi rezeksiyon, bu tümörlerin en uygun tedavisidir. Cerrahi sonrası hastalar rekurrens açısından yakın takip edilmelidir.

Anahtar Kelimeler

Plevra; Rezeksiyon; Soliter Fibröz Tümör

Abstract

The aim of this study is to determine treatment and management challenges associated with the rarely seen solitary fibrous tumor of the pleura. The patient files of five cases diagnosed with a solitary fibrous tumor of the pleura in our clinic between 2010 and 2015 were retrospectively analyzed. Mean age was 56.2 (range 40-66). Two cases presented with symptoms. The other three cases were diagnosed by incidental findings in imaging studies. Two cases, both male, were diagnosed as malignant solitary fibrous tumor of the pleura. Mean tumor size was 8.6 cm (range 5-13 cm). All patients had complete resection of their lesions. All patients were followed up regularly (9-75 months). Only one patient had recurrence of disease. Solitary fibrous tumor of the pleura is a rare neoplasm that can reach enormous sizes. Complete surgical resection is the most appropriate treatment option. Patients must be closely followed up after surgery to detect recurrence.

Keywords

Pleura; Resection; Solitary Fibrous Tumor

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Introduction

Primary tumors of the pleura can be classified as localized or diffuse neoplasms. The localized form, solitary fibrous tumor of the pleura, is classified as either malignant or benign. Solitary fibrous tumor of the pleura, which had been presumed to be a variation of malignant pleural mesothelioma, was subsequently shown to be originating from the submesothelial mesenchymal layer [1]. Solitary fibrous tumors are quite rare, making up only 5% of all pleural tumors. Of these, only 10-20% are malignant [2,3]. The incidence of the tumor is reported to be 2.8/100.000 [4]. More than a third of patients have no symptoms. For patients who do have symptoms, the most frequent complaints are chest pain, cough, and shortness of breath. Diagnosis is usually made by incidental findings in imaging studies. For localized forms, optimal treatment consists of total excision of the mass and long term follow-up. For malignant forms, patients receive chemoradiotherapy after surgery [5]. In this study, we obtained the records of five patients who were operated on for, and diagnosed with, solitary fibrous tumor of the pleura, and noted clinical and radiological features and postoperative follow-up information. We reviewed these cases with regard to the current literature.

Material and Method

For the six years through 2010 and 2015, there were five patients diagnosed with solitary fibrous tumor of the pleura in our clinic. The records for these cases were retrospectively reviewed. We evaluated all cases with a history of complaints, preoperative chest x-rays and other imaging of the chest, blood chemistry, and complete blood count and pulmonary function tests. All cases had thoracotomies. None of the cases had additional diagnostic interventions. Excised surgical materials were analyzed by the Pathology Department.

Results

Our cases have a mean age of 56.2 (range 40-66). Three of them were female and two of them were male. Only two of the patients were symptomatic. For the other patients, diagnoses were made incidentally during investigation of other diseases or routine follow-up of unrelated chronic conditions (IMAGE 1). All of the patients had thoracotomy; none had additional interventions for preoperative diagnosis. Tumors were totally excised in each patient (IMAGE 2). Mean tumor size was 8.6 cm (range 5-13 cm) (IMAGE 3). Only one patient required lobectomy for total excision of the tumor due to extensive adhesions to the left lower lobe. Three of the cases had their tumors in the right lung, two of them in the left. All cases of solitary fibrous tumor of the pleura originated from the visceral pleura.

Histopathologic examination showed that two of the tumors were malignant solitary fibrous tumors of the pleura. Neither of these cases had any evidence of tumor extending beyond the surgical margins, i.e. the surgical margins were clear for both these cases. It's noteworthy that both cases of malignant solitary fibrous tumor of the pleura were male patients. There were no postoperative complications and there was no operative mortality. The mean length of hospital stay was 4.8 days (range 3-6) (TABLE 1).

Cases were followed up on the postoperative first, third, sixth,

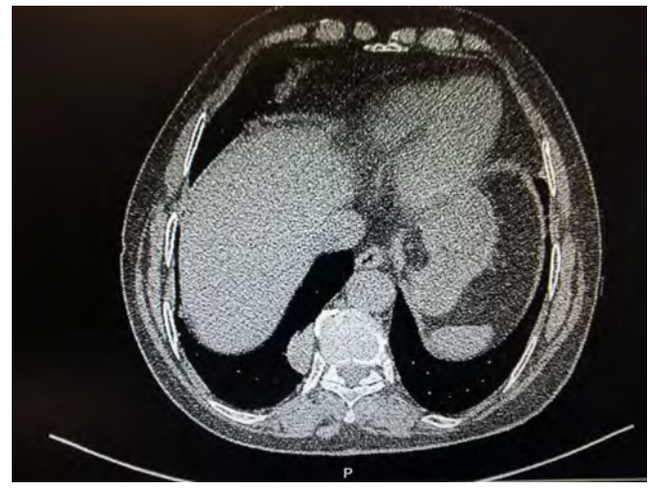

Image 1. Solitary fibrous tumor of the pleura in the paravertebral space next to the right lower lobe.

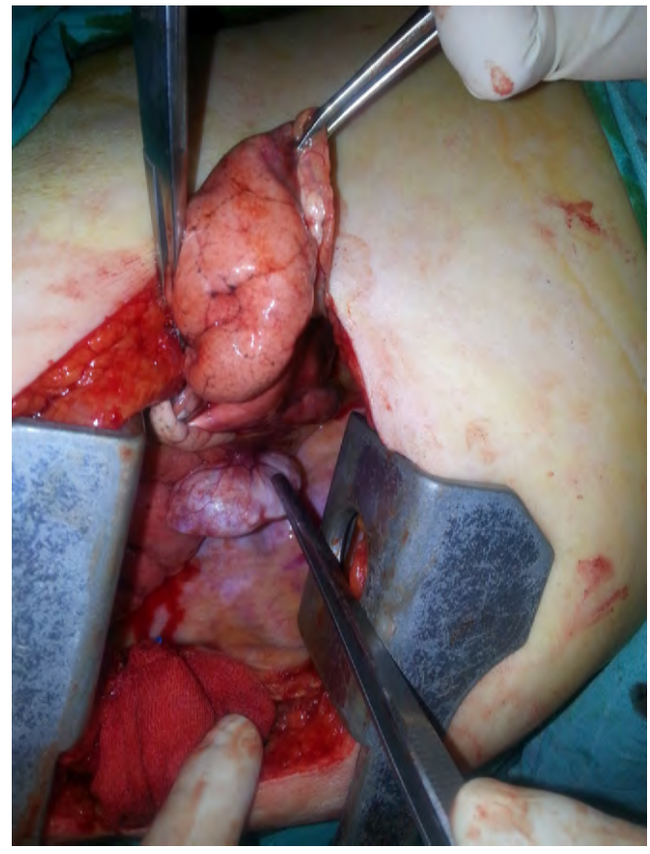

Image 2. Solitary fibrous tumor of the pleura attached to the right lower lobe with a pedicle.

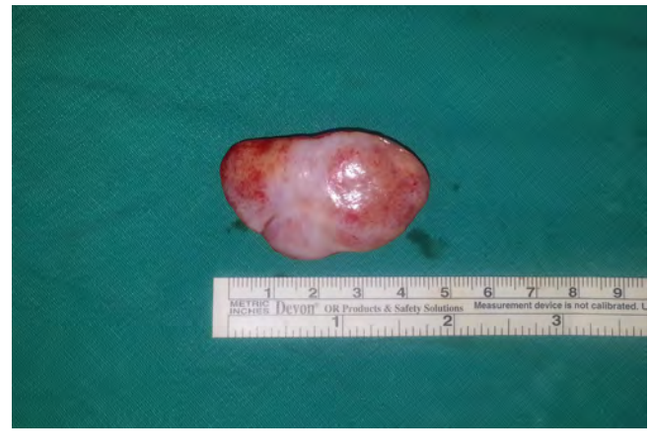

Image 3. Macroscopic image of the lesion excised from right lower lobe. A well-circumscribed mass lesion, which was attached to the right lower lobe with a pedicle, was reported to be a solitary fibrous tumor of the pleura based on its histopathology.

Table 1. Demographic and clinical features of patients.

	Age	Sex	Symptoms	Tumor size (cm)	Localization of tumor	Malign/Benign	Length of stay (days)	Length of follow-up (months)
1	58	Female	None	12	Right lower lobe	Benign	6	75
2	65	Female	None	5	Left upper lobe	Benign	3	60
3	52	Male	Chest pain and dyspnea	13	Left lower lobe	Malign	6	43
4	40	Male	Chest pain	8	Right lower lobe	Malign	3	29
5	66	Female	None	5	Right lower lobe	Benign	6	9

and twelfth months, and every six months thereafter. At follow-up, the patients were checked for any new complaints and abnormal findings in the physical examination. Complete blood count and blood chemistry was analyzed and radiologic tests were performed.

Only one patient, the one who had had a left lower lobectomy, went through a rethoracotomy for local recurrence. The tumor was excised totally in the reoperation.

Comments

Solitary fibrous tumor of the pleura is an extremely rare variety of neoplasm. In 1931, Klamperer and Rabin categorized pleural tumors as either diffuse or localized and stated that the diffuse type arises from multipotent mesothelial cells and are true mesotheliomas, whereas the localized type arises from subpleural areolar tissue [6][7]. There have been approximately 900 cases reported in the English literature [8].

Although the disease can be seen at any age, incidence peaks in the fifth and sixth decades [8]. In our series, 80% of the patients were in this range. Previous studies could not find a difference in the predisposition of this disease between sexes. In our study the female to male ratio was 3:2. Since the current literature states that malign solitary fibrous tumor of the pleura has similar incidence in both sexes [10], it is coincidental that both cases with the malignant form of the disease in our series were male patients.

More than a third of cases are asymptomatic. Symptomatic cases might have cough, shortness of breath, chest pain, hemoptysis, fever, chills, night sweats, fatigue, loss of appetite, sensitivity in the chest wall, pleural effusion, superior vena cava syndrome, and changes in electrocardiography [4] [12-14]. The rest of the patients were diagnosed incidentally during investigation of other diseases or during routine follow-up of unrelated chronic conditions.

Only 10-20% of solitary fibrous tumors of the pleura are malignant. In a multicenter retrospective case series of 50 patients, the recurrence rate of malignant solitary fibrous tumors of the pleura was 15% [5]. The aforementioned study recommended performing a chest wall resection, lobectomy, and even pneumonectomy if required for total excision of the tumor. For patients who have local recurrence, the recommended treatment is total excision of the recurrent tumor, if resectable. Patients with unresectable tumors should receive chemoradiotherapy.

Resection with video-assisted thoracoscopic surgery is possible in selected cases [9][15]. Whatever the choice of surgical technique, for optimal treatment total excision of the tumor is vital. It is the most important determinant of long term survival. To be specific, after total excision of benign solitary fibrous tumors of the pleura, the 5-year survival rate can be as high as 100%. The 5-year survival rate for patients with malignant solitary fibrous tumors is around 81% [5]. If total resection is not possible, another treatment modality is chemoradiotherapy. Survival rates for these patients are lower.

Recurrence of solitary fibrous tumors of the pleura is more common in the malignant form and is reported to be 15% [5]. Recurrence in the benign form is rare, but not unheard of. Local recurrence even after 15 years has been reported. Survival in cases with recurrence is most influenced by whether or not the recurrent tumor can be totally excised. For this reason, it's imperative that patients diagnosed with solitary fibrous tumor of the pleura are followed up at regular intervals. Radiotherapy is a treatment option for recurrence of malignant solitary fibrous tumors of the pleura. It has been reported that malignant solitary fibrous tumor of the pleura responds very well to radiotherapy [16].

In conclusion, the most important treatment element for both benign and malignant forms of solitary fibrous tumors of the pleura is total surgical excision. One should not refrain from aggressive surgical procedures for total excision of the tumor, particularly in the malignant form. Patients should be followed up at regular intervals postoperatively. If there is recurrence, eligible patients should have a repeat surgery. Outcomes for solitary fibrous tumors of the pleura are favorable with appropriate surgery and follow-up.

Competing interests

The authors declare that they have no competing interests.

References

1. Guerrini S, Ricci A, Osman GA, Mariotta S. Different clinical and radiological features of solitary fibrous tumor of the pleura: Report of two cases. Lung India 2016; 33(1):72-4.
2. Papadopoulos A, Porfyridis I, Christodoulides G, Georgiou A. A rare clinical case - Solitary fibrous tumor of the pleura. Respir Med Case Rep. 2015;16:117–9.
3. Zhao L, Liu J, Li Q, Wang HQ. Bone Scintigraphy in a Patient With Giant Malignant Solitary Fibrous Tumor of the Pleura. Clin Nucl Med 2016;41(6):474-5.
4. Mezzetti M, Panigalli T, Giudice F, Cappelli R, Giuliani L, Ravaglia F at al. Surgical experience of 15 solitary benign fibrous tumor of the pleura. Crit Rev Oncol Hematol 2003;47:29-33.
5. Lococo F, Cesario A, Cardillo G, Filosso P, Galetta D, Carbone L at al. Malignant Solitary Fibrous Tumors of the Pleura Retrospective Review of a Multicenter Series. J Thorac Oncol 2012;7(11):1698-706.
6. Akı H, Durak H, Kaynak K, Demirhan Ö, Öz B. Solitary fibrous tumor of the pleura. Cerrahpaşa J Med 2002;33:127-31.
7. Klemperer P, Rabin LB. Primary neoplasms of the pleura: a report of five cases. Arch Pathol 1931;11:385–412.
8. Supakul R, Sodhi A, Tamashiro CY, Azmi SS, Kadaria D. Solitary Fibrous Tumor of the Pleura: A Rare Cause of Pleural Mass. Am J Case Rep 2015;16: 854-7.
9. Sung SH, Chang JW, Kim J, Lee KS, Han J, Park SI. Solitary fibrous tumors of the pleura: surgical outcome and clinical course. Ann Thorac Surg 2005;79(1):303-7.
10. Boddaert G, Guiraudet P, Grand B, Venissac N, Le Pimpec-Barthes F, Mouroux J at al. Solitary fibrous tumors of the pleura: a poorly defined malignancy profile. Ann Thorac Surg 2015;99:1025–31.
11. England DM, Hochholzer L, McCarthy MJ. Localized benign and malignant fibrous tumors of the pleura. A clinicopathologic review of 223 cases. Am J Surg Pathol 1989;13(8):640-58.
12. Turkyılmaz A, Aydın Y, Dostbil A, Eroğlu A. Plevranın dev soliter fibröz tümörü

(İki olgu sunumu). EAJM 2007;39:145-7.
13. Briselli M, Mark EJ, Dickersin R. Solitary fibrous tumors of the pleura: Eight new cases and review of 360 cases in the literature. Cancer 1981;47:2678-9.
14. Perrot M, Fischer S, Bründler MA, Sekine Y, Keshavjee S. Solitary fibrous tumors of the pleura. Ann Thorac Surg 2002;74:285-93.
15. Takahama M, Kushibe K, Kawaguchi T, Kimura M, Taniguchi S. Video-assisted thoracoscopic surgery is a promising treatment for solitary fibrous tumor of the pleura. Chest 2004;125(3):1144-7.
16. Saynak M1, Bayir-Angin G, Kocak Z, Oz-Puyan F, Hayar M, Cosar-Alas R et al. Recurrent solitary fibrous tumor of the pleura: significant response to radiotherapy. Med Oncol 2010;27(1):45-8.

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Stress Ulcer Perforation in the Intensive Care Unit Patient Following Cardiac Surgery

Yoğun Bakım Hastasında Kardiak Cerrahiyi Takiben Gelişen Stres Ülser Perforasyonu

Stress Ulcer Perforation

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Öz

Gastrointestinal komplikasyonlar yoğun bakım hastalarında yaygın olarak tanımlanmıştır. Bunlardan stres-ilişkili mukozal hastalıkla bağlantılı ülser, kanama ve perforasyon hastanede kalışı ve mortaliteyi arttırabilmektedir. Bu yazıda akut myokard enfarktüsü sonrası yapılan koroner anjiografi esnasında oluşan, iatrojenik koroner damar perforasyonu neticesi gelişen perikardiyal tamponat nedeniyle ameliyat edilen yoğun bakım hastasında, stres ülseri perforasyonu ile ilişkili sıra dışı bir mortalite olgusunu sunuyoruz.

Anahtar Kelimeler

Ülser; Ülser Perforasyonu; Yoğun Bakım

Abstract

Gastrointestinal complications are common in intensive care unit patients. Some complications, including ulceration, bleeding, and perforation related to stress-related mucosal disease can prolong hospital stays and increase the risk of death. Herein we report on an unusual case of an intensive care unit patient who died following perforation of a stress ulcer. The patient had undergone surgery for pericardial tamponade, which occurred after iatrogenic coronary vessel perforation during coronary angiography for acute myocardial infarction.

Keywords

Ulcer; Ulcer Perforation; Intensive Care

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Introduction

Since Selye's description of the "alarm reaction," an immediate reaction to a stressor, it has been realized that the gastrointestinal (GI) tract is a major target of the stress response that leads to acute gastric and duodenal erosions. Stress ulcer syndrome refers to gastroduodenal erosions or ulcers that occur as acute responses to major physiological stressors. Clinically, these conditions may manifest as upper GI perforation [1].

Case Report

A 62-year-old man with no prior cardiac history presented at an emergency department one night because of chest pain at rest. The electrocardiogram showed an ascending ST segment elevation of 1 mm, with the J point on the isoelectric line, and a Q wave in leads II and III. His creatine kinase level was elevated (470 U/L; normal 0–171 U/L). The patient was diagnosed with acute inferior wall myocardial infarction and was transferred to our institution for primary percutaneous coronary intervention. Coronary angiography performed within one hour of admission to our hospital revealed thrombotic obstruction of the middle right coronary artery, which indicated angioplasty. However, a coronary rupture (depicted by significant leakage of the contrast agent into the pericardium) was observed as the guidewire was being advanced during the angiographic procedure. The echocardiogram revealed moderate to extensive circumferential pericardial effusion and evidence of cardiac tamponade. The patient's condition began to deteriorate and emergency surgical repair was performed. He was then transferred to a cardiac intensive care unit (ICU), where he was treated for approximately 30 days. Despite prophylactic administration of a proton pump inhibitor to prevent ulcer formation, as well as other therapeutic measures (energetic respiratory and circulatory support), the patient's condition deteriorated and the cardiac surgeon consulted with a general surgeon to discuss the patient's situation. During the initial consultation, he was found to be dehydrated and was experiencing cold sweats. His temperature was 38°C, his blood pressure was 97/54 mmHg, and his pulse rate was 101 beats/min. Abdominal examination revealed no abdominal movement and marked distension. His laboratory findings were as follows: white blood cell count, 15,700 /mm3 (neutrophils 91.5%); hematocrit, 31.1%; hemoglobin, 10.3 g/dL; platelet count, 346,000/mm3; alanine transaminase, 2 U/L; aspartate transaminase, 28 U/L; urea, 300 mg/dL; and creatinine, 3.97 mg/dL. A rectal examination revealed an empty rectum. Computed tomography was not performed because of dependence on the mechanical ventilator and severe acute renal impairment. Gastroscopy subsequently performed in the ICU revealed a perforation in the first part of the duodenum (Figure 1) and the patient underwent emergency surgery. Laparotomy revealed a perforated 1 cm ulcer on the anterior aspect of the proximal duodenal bulb and 200 mL of gastric contents mixed with biliary fluid in the peritoneal cavity. After peritoneal washing, the duodenal ulcer was repaired with three silk sutures and an omental patch. The remainder of the peritoneal cavity was explored and found to be normal. Postoperatively, the patient was transferred to our surgical ICU. Despite optimal management in the ICU, the patient's inflammatory markers, creatinine, and alanine transaminase continued to increase, and his urinary

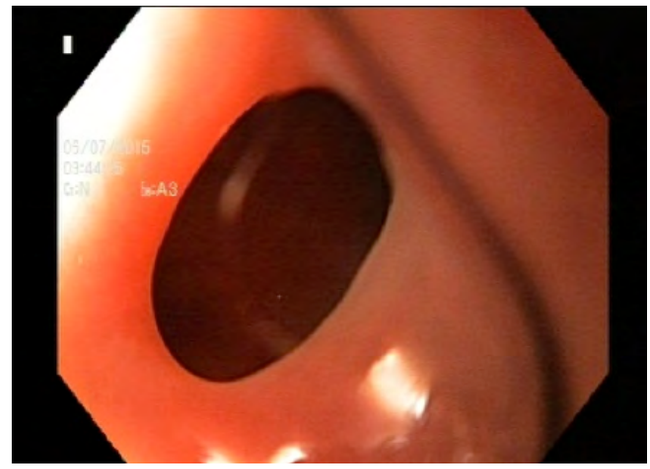

Figure 1. Endoscopic image of the perforation located in the proximal duodenal bulb.

output remained low. The patient had a difficult postoperative course that was complicated by wound infection and septicemia. The patient did not recover from septic shock. He was kept on a ventilator and inotropic drugs with antibiotics. The patient died from cardiopulmonary failure and shock four days following the second operation.

Discussion

Stress-related mucosal damage (SRMD) of the GI tract was first described in 1971 by Lucas et al., who used the term stress-related erosive syndrome [2]. Since then, many terms have been used to describe SRMD in critically ill patients, including stress ulcer, stress erosion, stress gastritis, hemorrhagic gastritis, erosive gastritis, and stress-related mucosal disease. The incidence of SRMD has been reported to range from 6% to 100% in critically ill patients [3]. Stress ulcers develop within hours of major trauma or serious illness, most often in the proximal regions of the stomach. Endoscopy performed within 72 h of a major burn or cranial trauma revealed acute mucosal abnormalities in >75% of patients [4]. Stress ulcers are common complications in many clinical conditions, but they rarely perforate. They generally occur in the fundus or body of the stomach, but sometimes occur in the antrum, duodenum, or distal esophagus. Stress ulcers tend to be shallow and cause oozing of blood from superficial capillary beds. Deeper lesions may also occur, which can erode into the submucosa and cause massive hemorrhage or perforation [5]. Although not completely understood, the pathophysiology of stress ulcers is likely to be multifactorial. Inadequate systemic perfusion, mucosal blood flow, and cellular oxygenation appear to play important roles in the development of stress ulcers, as do decreased gastric pH, increased mucosal permeability, and alterations in normal protective mechanisms [6,7]. Stress ulcers are an important clinical disorder. Two major complications, upper GI perforation and bleeding, are significant concerns in critically ill patients and are associated with a high risk of death. GI complications are particularly common in critically ill patients and also carry a high risk of death. Many authors have reported the prevalence of GI complications and predictors of GI complications in specific patient populations. These data allow practitioners to evaluate not only the risk of complication but also the risk of death in individual patients. Chan et al. investigated the GI complications in neurosurgical

patients [8]. Their retrospective report of 526 patients revealed that 36 (6.8%) had endoscopically or surgically confirmed GI complications. All of these patients had GI bleeding, and two had evidence of ulcer perforation. Eleven patients died as a direct result of the GI complication. In a prospective study of 11,508 cardiac surgery patients, D'Ancona et al. identified 147 GI complications in 129 patients (1.2%) [9]. The complications included gastroesophagitis (12.2%), upper GI hemorrhage (28.6%), perforated peptic ulcer (4.7%), cholecystitis (6.8%), pancreatitis (8.8%), intestinal ischemia (11.5%), colitis (12.2%), diverticulitis (3.4%), intestinal occlusion (1.1%), and lower GI hemorrhage (0.7%). In a study by Tsiotos et al., perforation occurred after stress ulceration in <1% of patients in a cardiac surgery ICU [10]. The potential causes of stress ulcers are numerous and are related to patient characteristics such as age and underlying disease processes, as well as to interventions such as mechanical ventilation. The primary cause of stress in our patient was likely to be severe illness. In addition, the patient had had endotracheal intubation for an extended period of time. Most critically ill patients are unable to provide a history or description of their symptoms, which limits and/or delays the timely diagnosis of GI disorders. Therefore, it is essential that critical care specialists who suspect GI complications initiate preventive strategies and carefully monitor the patients for evidence of such complications. The optimal agent for acid suppression is unknown, and there is controversy and substantial variation in clinical practices regarding the choice of agent for acid suppression in critically ill patients. Based on the results of randomized trials and the recommendations of the American Society of Health System Pharmacists, all critically ill patients who are at high risk of GI complications such as bleeding and perforation should receive anti-ulcer prophylaxis [11]. Because of his poor clinical condition, our patient did not display the classic signs of peritonitis such as generalized tenderness and rigidity. Therefore, the diagnosis of bowel perforation was not initially apparent and could only be made after exploratory laparotomy in the ICU. Surgical exploration also revealed perforation of the first part of the duodenum, which was intractable despite intensive anti-ulcer therapy. There has been a significant decrease in the rates of elective surgery for treating uncomplicated peptic ulcers; however, complications such as perforation and obstruction persist and require urgent surgical management. The first report of a series of patients presenting with perforation of a duodenal ulcer was made in 1817 by Travers. Although the earliest description of surgical treatment was made by Mikulicz in 1884, the first successful operation for a perforated duodenal ulcer occurred in 1894 [12]. Mortality related to a perforated duodenal ulcer is dependent on the presence or absence of several risk factors. Individual risk can also be assessed using the Acute Physiology and Chronic Health Evaluation II system. Although prompt closure of the perforation offers the best chance for survival, it also creates additional stress exacerbating the patient's clinical state. Therefore, surgical repair must be done as quickly as possible. The overall mortality rate is approximately 10% in most studies. In the developing world the high morbidity and mortality rates of patients with a perforated duodenal ulcer are probably due to delayed presentation. In particular, patients in whom the diagnosis is overlooked almost always die. Risk factors affecting the prognosis of perforated ulcer included delayed treatment (> 24 h), preoperative shock (systolic blood pressure < 100 mmHg), and serious comorbidities. The mortality rate increases to 100% in patients with all three factors. This is consistent with our experience in the present case. Potential adverse effects of pharmacologic agents that suppress gastric acid secretion are particularly relevant to critically ill patients, and include nosocomial pneumonia and clostridium difficile infection. Other adverse effects, such as intolerance, drug interactions, and thrombocytopenia are rare. Despite these risks, prophylactic anti-ulcer therapy is essential to reduce the risk of stress ulcer-related complications. Perforated stress ulcers are potentially fatal complications in ICU patients. Such ulcers may be missed if practitioners are not alert to their likelihood in critically ill patients [13-15].

In conclusion, based on our limited experience with this patient, we wish to make the following recommendations: First, all ICU patients should receive prophylactic anti-ulcer therapy and possibly cimetidine; second, ICU specialists should be vigilant for complications of SRMD; and third, surgical closure of a perforation must be simple and performed quickly.

Competing interests

The authors declare that they have no competing interests.

References

1. Selye H. The general adaptation syndrome and the diseases of adaptation. J Clin Endocrinol Metab 1946;6:117–230.
2. Lucas CE, Sugawa C, Riddle J, Rector F, Rosenberg B, Walt AJ. Natural history and surgical dilemma of "stress" gastric bleeding. Arch Surg 1971;102(4):266–73.
3. Choung RS, Talley NJ. Epidemiology and clinical presentation of stress-related peptic damage and chronic peptic ulcer. Curr Mol Med 2008;8(4):253–7.
4. DePriest JL. Stress ulcer prophylaxis. Do critically ill patients need it? Postgrad Med 1995;98(4):159–8.
5. Cook DJ. Stress ulcer prophylaxis: gastrointestinal bleeding and nosocomial pneumonia. Best evidence synthesis. Scand J Gastroenterol Suppl 1995;210:48–52.
6. American Society of Health-System Pharmacists. ASHP Therapeutic Guidelines on Stress Ulcer Prophylaxis. ASHP Commission on Therapeutics and approved by the ASHP Board of Directors on November 14, 1998. Am J HealthSyst Pharm 1999;56(4):347–79.
7. Cook D, Heyland D, Griffith L, Cook R, Marshall J, Pagliarello J. Risk factors for clinically important upper gastrointestinal bleeding in patients requiring mechanical ventilation. Crit Care Med 1999;27(12):2821–7.
8. Chan KH, Mann KS, Lai EC, Ngan J, Tuen H, Yue CP. Factors influencing the development of gastrointestinal complications after neurosurgery: results of multivariate analysis. Neurosurgery 1989:25(3):378–82.
9. D'Ancona G, Baillot R, Poirier B, Dagenais F, de Ibarra JI, Bauset R, et al. Determinants of gastrointestinal complications in cardiac surgery. Texas Heart J 2003;30(4):280–5.
10. Tsiotos GG, Mullany CJ, Zietlow S, van Heerden JA. Abdominal complications following cardiac surgery. Am J Surg 1994;167(6):553–7.
11. Spirt MJ, Stanley S. Update on stress ulcer prophylaxis in critically ill patients. Crit Care Nurse 2006;26(1):18–9.
12. Andrew G Hill. Management of perforated duodenal ulcer. In:Holzheimer RG, Mannick JA, editors. Surgical Treatment: Evidence-Based and Problem-Oriented. Zuckschwerdt Publishers 2001.
13. Marik PE, Vasu T, Hirani A, Pachinburavan M. Stress ulcer prophylaxis in the new millennium: a systematic review and meta-analysis. Crit Care Med 2010;38(11):2222.
14. Houssa MA, Atmani N, Nya F, Abdou A, Moutakiallah Y, Bamous M, et al. Stress gastric ulcer after cardiac surgery: Pathogenesis risk factors and medical management. World Journal of Cardiovascular Diseases 2013;3(3):312-6.
15. Bateman BT, Bykov K, Choudhry NK, Schneeweiss S, Gagne JJ, Polinski JM, et al. Type of stress ulcer prophylaxis and risk of nosocomial pneumonia in cardiac surgical patients: cohort study. BMJ 2013;347:f5416.

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Superficial Dental Intervention Induced Myopericarditis: A Case Report

Yüzeyel Dental Girişimin Tetiklediği Miyoperikardit: Vaka Sunumu

Dental Intervention Induced Myopericarditis

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Öz

Dental girişim sonrasında gelişen miyoperikardit vakası literatürde şimdiye kadar raporlanmamıştır. Biz, yüzeyel dental girişim sonrasında plöritik tipte göğüs ağrısı ile hastaneye başvuran 26 yaşında bir erkek hasta sunduk. Hasta, labaratuar, elektrokardiyografik ve ekokardiyografik bulguları ile değerlendirildiğinde miyoperikardit tanısı aldı. Miyoperikardit etyolojisinde yer alan diğer nedenler dışlandıktan sonra, dental girişim öyküsü etyolojik neden olarak suçlandı. Bakteriyemiye sekonder gelişen kardiyak olaylar nadirdir ve sıklıkla infektif endokardit olarak prezente olur. Biz dental girişim sonrası miyoperikardit kliniği gelişen bir vakayı sunduk. Her dental girişim bakteriyemiye neden olabilir ancak her bakteriyemi kardiyak komplikasyonlar ile sonuçlanmaz. Miyoperikardit patogenezinde bakteri invazyonu, toksini yada eksternal uyarılara karşı gelişen immünolojik reaksiyonlar yer alır. Bu bağlamda, her bakteriyemi, immünolojik mekanizmalar aracılığı ile kardiyak komplikasyonlara yol açabilir.

Anahtar Kelimeler

Miyoperikardit; Dental Girişim; Bakteriyemi

Abstract

A myopericarditis case following dental intervention has not yet been reported in the literature yet. We present the case of a 26-year-old man admitted with pleurotic type chest pain following superficial dental intervention. He was diagnosed with myopericarditis based on laboratory parameters and electrocardiographic and echocardiographic findings. Other known causes of myopericarditis were excluded and dental intervention was concluded to be the probable cause of myopericarditis. Cardiac manifestations secondary to bacteremia are rare complications and most present as infective endocarditis. This case reports the first patient presented with myopericarditis following superficial dental intervention. All dental interventions can cause bacteremia; however, each case of bacteriemia does not result in cardiac complications. Bacterial invasion, toxins or immune responses to external stimulations act on pathogenesis of myopericarditis. In this regard, it is possible that bacteremia may lead to cardiac complications via immune mechanisms.

Keywords

Myopericarditis; Dental Intervention; Bacteriemia

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Introduction

Myopericarditis is an inflammatory process affecting the heart muscle and pericardium. Acute pericarditis is responsible for up to 5% of admissions to the emergency department and may result from drugs, infections, or autoimmune or idiopathic disorders [1]. There are four known mechanisms of myopericarditis etiopathogenesis: 1) direct damage to cardiac myocytes by the microorganism: 2) damage to infected myocyte via immunoresponse to bacteria: 3) cytotoxicity by viral antigen specific immunoreaction; and 4) cytotoxicity via a circulating toxin [2]. Cardiac manifestation secondary to bacteremia is very rare complication following superficial dental interventions. No cardiac complication has been reported in patients foloowing a dental thinning procedure before. In this paper, we report a case of myopericarditis probably having occured by an immunoresponse to bacteremia which develop following a dental thinning procedure. We evaluate the clinical and laboratory findings of myopericarditis and discuss them with respect to the available literature.

Case Report

We report the case of a 26-year-old man admitted with pleurotic type chest pain. He had no medical history of chronic diseases such as coronary artery disease, valvular heart disease, diabetes mellitus, or hypertension and had no familial history of cardiovascular disease. He didn't have a history of drug intake continuously or recently. He was not a smoker and did not have history of drug addiction. He had no viral or bacterial actual infection symptoms. He had been suffering from pleurotic type chest pain for five days and palpitation for three days before admission to our clinic. His physical examination did not show any relevant findings including fever, lymphadenopathies, rash, arthritis or heart murmurs. His abdominal examination was unremarkable. Laboratory finding were white blood cells count of 8.77 U/L (4.0-10.0 10^3 u/L), lymphocyte count of 1.08 U/L (0.6-3.4 10^3) and hemoglobin levels of 8.63 g/dl (13.2-17.3g/dL). There was an elevation in cardiac enzymes. Creatine-kinase (CK) was 1413 (< 190 U/L), CK-MB was 36 (0.6-6.3ng/dL), lactate dehydrogenase was 405 (< 190 U/L), and troponin I (TnI) was 1.65 (< 0.09 ng/mL). Erytrocyte sedimentation rate was 5 mm/h (<15 mmm/h) and C-reactive protein was 1.0 mgL (<10). Autoimmune (antinuclear antibody test with IFA was negative) and microbiological studies (EBV VCA IgM, CMV IgM, HSV type I-II IgM with ELISA were negative, influenza IgA PCR was negative, blood cultures were negative) failed to show an etiology for his disease. Electrocardiogram indicated sinusal tachycardia and nonspecific changes on inferior derivations (Figure1). Echocardiograpy showed echogenic focus on inferior segment of myocardium without any pericardial effusion (Figures 2-3). These findings indicated a diagnosis of myopericarditis and the patient was given nonsteroidal-anti-inflammatory-drugs. The patient was discharged after clinical symptoms and laboratory findings were resolved. We learned of a history of a dental thinning procedure two days before the initiation of chest pain according to the patient's detailed anamnesis. After excluding the other causes, we concluded that the etiology of myopericarditis was like related to the dental thinning procedure.

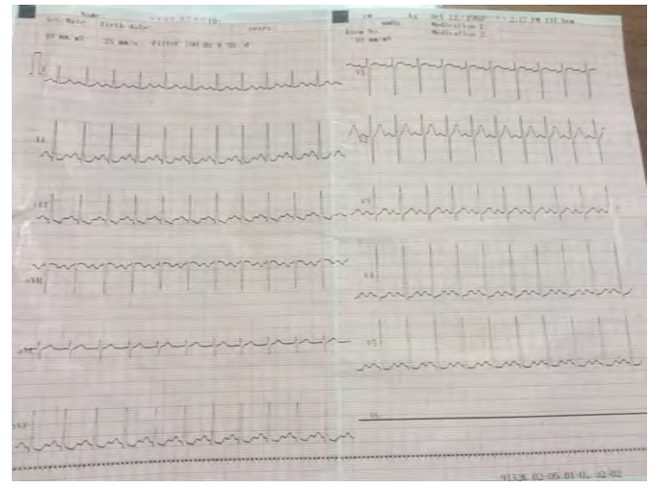

Figure 1. Electrocardiography. Electrocardiogram indicated the sinusal tachycardia and non specific changes on inferior derivations.

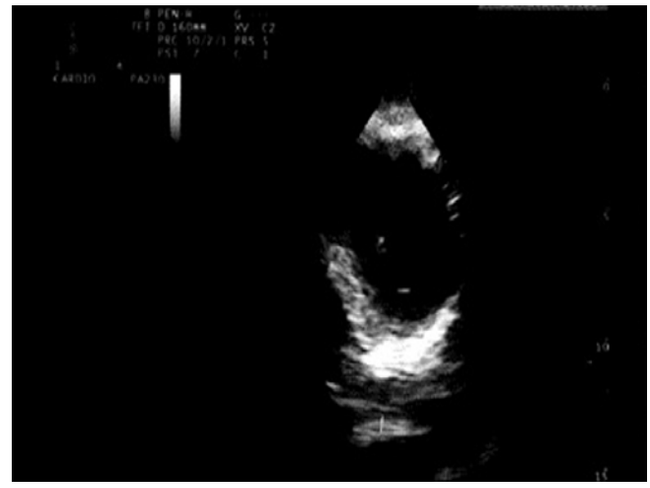

Figure 2. Echocardiography-Parasternal short axis view. Echocardiography showed the echogenic focus on inferior segment of myocardium.

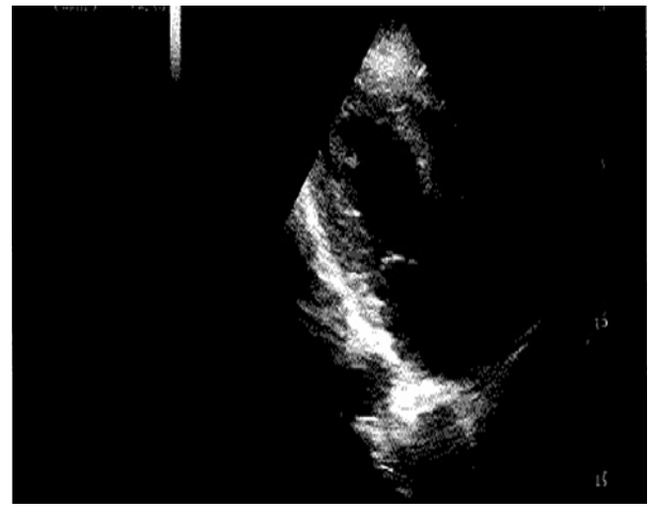

Figure 3. Echocardiography-Parasternal long axis view. Echocardiography showed the echogenic focus on inferior segment of myocardium.

Discussion

Myopericarditis is a myocardial injury added to the pericardial inflammation, on the basement. It can be clinically diagnosed by findings of acute pericarditis and elevated cardiac markers of injury (TnI or TnT, and CK-MB) without depressed left ventricular function by echocardiogram [3]. The pericardium may be affected by all categories of diseases, including infectious diseases such as viral, bacterial, fungal, parasitic, or

autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, scleroderma, Takayasu's arteritis, Behçet's syndrome, or rheumatoid arthritis. It can also be affected by neoplastic diseases such as pericardial mesothelioma, lung and breast carcinoma, lymphoma, or iatrogenic and traumatic conditions such as penetrating thoracic injury, esophageal perforation, radiation injury, or metabolic events such as uraemia and myxoedema. Classic myocarditis, an inflammation of the heart muscle, occurs as a result of the triggering of external antigens including viruses, bacteria, parasites or drugs (anthracyclines, cocaine, clozapine, sulfonamides, cephalosporins, penicillins and tricyclic antidepressants), or internal stimulants such as autoimmune activation against self-antigens [4]. In our case, we concluded that dental intervention was the probable cause of myopericarditis via immunostimulation against bacteria when other known causes had been eliminated.

Cardiac complication associated with dental intervention is mostly shown to be associated with infective endocarditis (IE) in the literature. Incidence of IE is approximately 10 per 100,000 of the population per year. The incidence of IE that is a result of dental treatment is as low as 4% [5]. The majority of patients who develop endocarditis were thought have a pre-existing cardiac defect. More recently, this perception has changed with the information that nearly half of the cases of endocarditis did not have known previous cardiac disease [6]. Guidelines had recommended that antibiotics be administered prior to invasive dental procedures to those patients who with a high risk of endocarditis. However, recent guidance by the National Institute for Health and Care Excellence (NICE) in England and Wales now recommends that antibiotics are not required. Many dental procedures cause bacteremia and this may lead to endocarditis in some people. Bacteria may enter the blood through a variety of portals but generally via mucosal surfaces. The gingiva and periodontal ligament which surrounds all teeth may cause a degree of inflammation and this lead to entry of bacterias to the bloodstream. Indeed, it has been demonstrated that daily activities such as toothbrushing can cause bacteremia [7,8]. In this regard, bacteremia have been not resulted with a cardiac complication mostly. However, underlying immunologic interactions could play a role in cardiac manifestations such as endocarditis or myopericarditis.

In our case we diagnosed myopericarditis by clinical presentation and elevated cardiac

enzymes. After excluding the other possible causes including autoimmune disorders, viral and bacterial infections, and drugs used, we suspected superficial dental intervention as an etiologic reason. As a result, we report the first case of myopericarditis that was secondary to superficial dental intervention. All dental interventions could can bacteriemia; however, each case of bacteremia does not result in cardiac complications. In this regard, it is possible that bacteremia may lead to cardiac complications via immune mechanisms.

Conclusion

Dental interventions should be considered in the differential diagnosis of myopericarditis etiology.

Competing interests

The authors declare that they have no competing interests.

References

1. Spodick DH. Acute cardiac tamponade. N Engl J Med 2003;349:684-90.
2. Knowlton KU, Savoia MC, Oxman MN. Myocarditis and pericarditis. In: Mandell GL. Bennett JE, Dolin R. Principles and practise of infectious diseases Churchill Livingstone, New York, 7th eds. 2010:1153-71.
3. Imazio M and Cooper LT: Management of myopericarditis. Expert Rev Cardiovasc Ther 2013;11:193 201.
4. Waite RA, Malinowski JM. Possible mesalamine-induced pericarditis: Case report and literature review. Pharmacotherapy 2002;391-4.
5. Gendron R, Grenier D, Maheu-Robert LF. The oral cavityas a reservoir of bacterial pathogens for focal infections. Microbes and Infection 2000;2:897–906.
6. Duval X, Delahaye F, Alla F, Tattevin P, Obadia JF, LeMoing V, et al. Temporal trends in infective endocarditis inthe context of prophylaxis guideline modifications: three
successive population-based surveys. Journal of the AmericanCollege of Cardiology 2012;59:1968–76.
7. Lucas V, Roberts GJ. Odontogenic bacteremia followingtooth cleaning procedures in children. Pediatric Dentistry 2000;22:96–100.
8. Roberts GJ. Dentists are innocent! 'Everyday' bacteremiais the real culprit: A review and assessment of the evidencethat dental surgical procedures are a principal cause of
bacterial endocarditis in children. Pediatric Cardiology1999;20:317–25.

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Bernard-Von Burrow Flap and Unilateral Webster Modification for Reconstruction of Upper Lip

Üst Dudak Rekonstrüksiyonunda Bernard-Von Burrow Flep ve Tek Taraflı Webster Modifikasyonu

Upper Lip Reconstruction

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Öz

Üst dudak karsinomları, ultraviyole ışınlarının daha az etkisinde kaldığı için, alt dudağa göre daha az görülmektedir. Buna bağlı olarak, üst dudak rekonstrüksiyon yöntemleri ve yöntemlerin uygulandığı hasta sayısı da daha azdır. Üst dudak defektlerinin rekonstrüksiyonunda şimdiye kadar birçok metod tanımlanmıştır. Üst dudak defektlerinin onarımında uygulanan rekonstrüksiyon metodları; transoral rotasyon flepleri (Abbe ve Estlander), sirkumoral ilerletme-rotasyon flepleri (Karapandzic ve Gillies), perialar kresentik eksizyon, ters Yús flep ve Bernard-Von Burrow flebi ve Bernard-Von Burrow flebi ile birlikte çift taraflı Webster modifikasyonu olarak sıralanabilir. Üst dudak karsinomu nedeniyle tümör eksizyonu yapılan ve rekonstrüksiyonunda Bernard-von Burrow flebin daha önce literatürde çok nadiren vurgulanan tek taraflı Webster modifikasyonu uygulanan olgu, literatür eşliğinde sunuldu.

Anahtar Kelimeler

Üst Dudak; Karsinom; Rekonstrüksiyon

Abstract

Since it is less influenced by ultraviolet rays, upper lip carcinomas are less common than those of the lower lip. Likewise, upper lip reconstruction methods and the number of patients that the methods applied are very limited. Several methods defined in the reconstruction of entire upper lip defects, so far. Reconstruction methods applied to the upper lip defects can be listed as follows: transoral rotation flap (Abbe and Estlander), circumoral advancement-rotation flap (Karapandzic and Gillies), perialar crescentic excision, reverse Yús flap, Bernard-von Burrow flap, and Bernard-von Burrow flap with double-sided Webster modification. In this article, a 58-year-old male patient who underwent tumor excision due to upper lip carcinoma by the Bernard-von Burrow flap and unilateral Webster modification method, previously very rarely emphasized in the literature is presented.

Keywords

Upper Lip; Carcinoma; Reconstruction

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Introduction

Besides the aesthetic properties of the upper and lower lips, important functional properties such as speech, facial expression, or oral competence are also important. In most series, lip carcinomas are the most common cancer type among the oral cavity cancers, while in some, the most common oral cancer is tongue carcinomas. The vast majority of lip cancers are squamous cell carcinoma. The second most common type of cancer is basal cell carcinoma that is more common in the upper lip [1,2]. The aim of lip reconstruction is to preserve both the aesthetic appearance and function as much as possible. Unlike the reconstruction of the lower lip, the preservation of the nasal base and philtrum in upper lip reconstruction is important [3]. If nasal base anatomy breaks down, then nasal airflow is also disrupted.

Reconstruction methods applied to the upper lip defects are: transoral rotation flap (Abbe and Estlander), circumoral advancement-rotation flap (Karapandzic and Gillies), perialar crescentic excision, reverse Yús flap, Bernard-von Burrow flap and Bernard-von Burrow flap with double-sided Webster modification [4,5]. We present the case of a patient who underwent tumor excision due to upper lip carcinoma with reconstruction with the Bernard-von Burrow flap and unilateral Webster modification method and a review of the current literature.

Case Report

A fifty-eight-year-old male patient was admitted with the complaint of a mass at the upper lip. Clinical examination revealed a crusty lesion at upper lip on the left side, 4 mm exceeding the vermillion border, a distance of 1 cm to the oral commissure, extending through the midline and 2x1.5 cm in diameter. He had an incision scar at lower lip and microstoma due to a previous intervention (Figure 1). There were no palpable lymphadenopathies at the neck. Incisional biopsy from the lesion was reported as squamous cell carcinoma. Under general anesthesia, tumor excision from the upper lip and reconstruction with the Bernard-von Burrow flap with unilateral Webster modification and left suprahyoid neck dissection operation was performed.

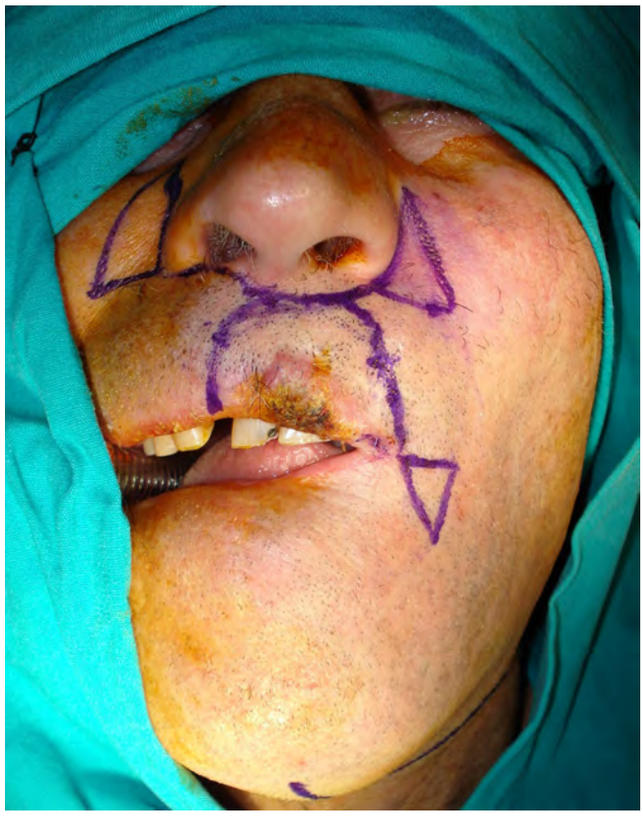

Figure 1. Preoperative view of the patient.

Vertical full-thickness incisions were made on each sides of the lesion leaving 1 cm to the lesion as a safety margin combined with horizontal full-thickness incision made 2 mm inferior to nasal base; skin, underlying tissues, and mucosa, together with the lesion, were excised (Figure 2). Burrow's triangles as

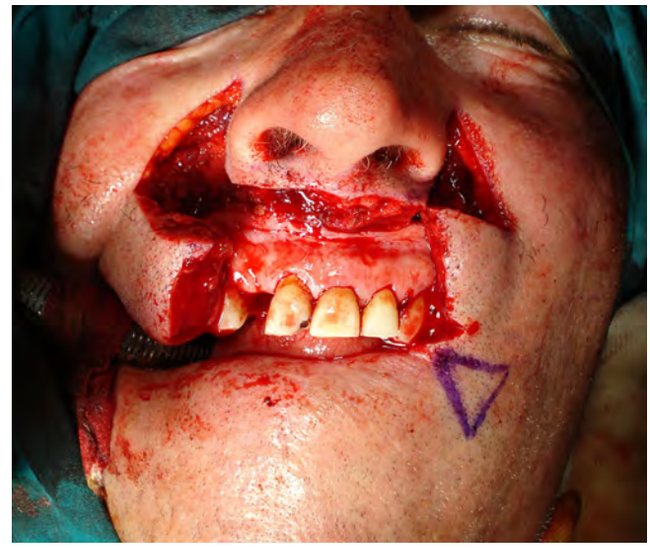

Figure 2. Perioperative view of the patient. Tumor at upper lip was excised and skin and subcutaneous tissue excisions were performed at Burrow's triangles.

the base running inferiorly and the apex superiorly were created along both nasolabial groove, triangles were excised, and Bernard flap was created. Another reverse triangle was drawn inferolaterally to the left oral commissure and one-sided Webster's triangle was created by excision of the skin and preserving the underlying mucosa. To form a line to the left upper lip vermillion, two inferiorly running edges of the mucosal triangle were cut and superiorly based mucosal flap was created (Figure 3). Opposing edges of each Bernard flaps were approximated

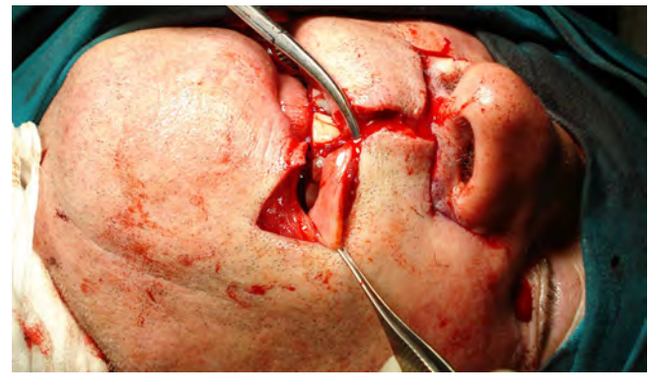

Figure 3. Superior-based mucosal flap created at the base of the Webster's triangle, and new vermillion.

finally and three layers closure was performed. Webster's mucosal flap was sutured as showing continuity with left upper lip and forming vermillion. The two other edges of Webster's triangle were also sutured in three layers. The operation was completed by performing left suprahyoid neck dissection (Figure 4). No metastatic lymphadenopathy was reported at pathological examination of the neck and the surgical margins were tumor free. Aesthetic and functional results were found to be satisfactory at postoperative sixth month control (Figure 5).

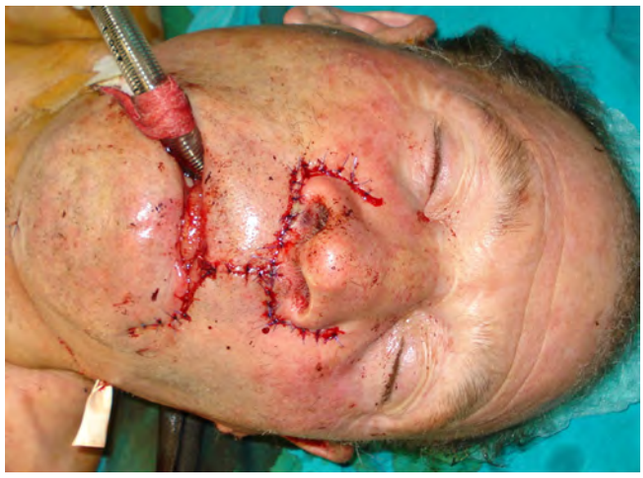

Figure 4. The patient's early postoperative appearance.

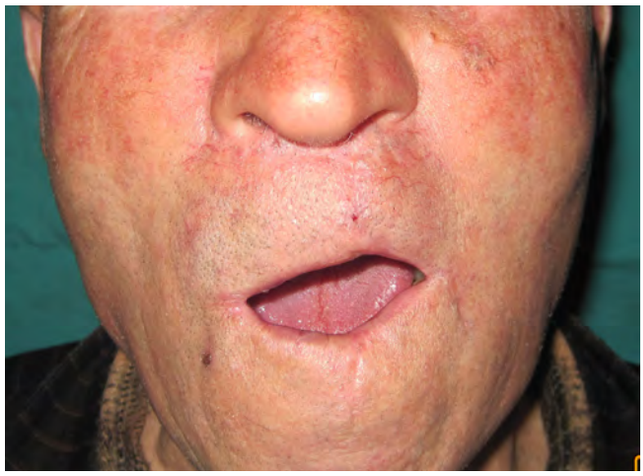

Figure 5. The patient's postoperative 6th month view.

Discussion

In the mid 19th century, Doctor Bernard and Doctor von Burrow simultaneously described cheek advancement flap for reconstruction of large upper lip defects (Bernard-von Burrow flap). The most important feature of this flap is formed by the triangles excised from the part of cheek proximal to the flap (Burrow's triangle). Excision of excess skin and muscle makes the advancement of flap easier. For upper lip reconstruction, an incision is made through the upper lip on both sides of the tumor with a horizontal connection at the base of the columella. Crescent or triangle-shaped areas are outlined in the nasolabial areas to permit straight horizontal advancement of the sides of the defect. The full-thickness crescent or triangle-shaped areas and the tumor are excised. Mobilization of the lateral flaps is achieved and three layer closure is performed [2,3,6].

Bernard- von Burrow's technique was later modified by Webster. Two lateral cutaneous triangles on each side of the chin at submental area are excised to allow for flap advancement. Mucosal triangles are preserved and tailored to form new vermillion for the reconstructed upper lip. These mucous membranes are then mobilized, preserving their base and new vermillion is formed [2,6]. This modification is applied two-sided and there are no cases in the literature concerning the implementation of this technique as one-sided. In our case, unlike the classic Webster modification, only one-sided reconstruction was performed for creating a new vermillion with an inferior triangle formed at the lesion side. We provided an acceptable mouth opening and function while avoiding microstomia.

Kriet et al. [7] applied the extended Abbe flap for repair of large upper lip defects, but cross lip flaps do not restore oral circumference.

Spinelli et al. [5] reported the utility of the alar crescent flap for partial and full-thickness reconstruction of upper lip defects of varying lengths. The technique provides good functional and aesthetic results, but the loss of philtral anatomic detail is reported as a disadvantage of this procedure.

Rotation flaps such as Gillies and Karapandzic are used for repair of large defects of the upper lip holding major part of the lip, but the disadvantage of these methods is reduction of oral competence in conjunction with microstomia [3].

Belmonte-Caro et al. [4] have modified the Yús technique and performed reverse Yús technique for upper lip reconstruction and they have reported very good aesthetic and functional outcomes for reconstruction of upper half lip defects.

Another technique that is applied to major defects of the upper lip is the Fujimori-Gate flap. A new upper lip is created by forming two inferior-based island flaps from the nasolabial area based on the angular artery and rotating them 90 degrees to the midline. This is reported as a reliable and a safe technique in the repair of large upper lip defects [8].

Chang et al. [9] have applied the technique of free temporal scalp flap for the reconstruction of large upper lip defects. It is a single-staged, relatively simple method of providing hair-bearing skin to the upper lip. They indicated that postoperative motor function and aesthetic results were almost normal at long-term follow-up.

In conclusion, the Bernard-von Burrow and unilateral Webster modification technique that we performed is easy to apply and is a reliable method in appropriate cases, with satisfactory postoperative mouth opening and aesthetic results. .

Competing interests

The authors declare that they have no competing interests.

References

1. Werning JW, Mendenhall WM. Cancer of the lip. In: Werning JW. ed. Oral Cancer; Diagnosis, management and rehabilitation. Thieme, New York; 2007:78-89.
2. Esclamado RM, Krause CK. Lip cancer. In: Bailey BJ, Healy Gb, Jackler RK, Pillsbury HC. III, Tardy E. JR, Johnson JT, eds. Head & neck surgery- otolaryngology. Lippincott Williams & Wilkins, Philadelphia; 2001:1299-311.
3. Renner G. Lip reconstruction. In: Day TA. and Girod DA. eds. Oral Cavity Reconstruction. Taylor & Francis, New York; 2006: 144-5.
4. Belmonte-Caro R, Cossio IP, Garcia GPA, Carranza TE. Reverse Yús flap for upper lip reconstruction. J Plast Reconstr Aesthet Surg 2010;63(2):148-50.
5. Spinelli HM, Tabatabai N, Muzaffar AR, Isenberg JS. Upper lip reconstruction with the alar crescent flap: A new approach. J Oral Maxillofac Surg 2006;64(10):1566-70.
6. Webster JP. Crescentic peri-alar cheek excision for upper lip flap advancement with a short history of upper lip repair. Plast Reconstr Surg 1955;16(6):434-64.
7. Kriet JD, Cupp CL, Sherris DA, Murakami CS. The extended Abbé flap. Laryngoscope 1995;105(9):988-92.
8. Aytekin A, Ay A, Aytekin O. Total upper lip reconstruction with bilateral Fujimori gate flaps. Plast Reconstr Surg 2003;111(2):797-800.
9. Chang KP, Lai CS, Tsai CC, Lin TM, Lin SD. Total upper lip reconstruction with a free temporal scalp flap: long-term follow-up. Head Neck 2003;25(7):602-5.

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A Case of Spinal Tuberculosis Which Caused Gibbosity and Accompanied by Epidural Abscess

Epidural Absenin Eşlik Ettiği ve Gibboziteye Neden olan Spinal Tüberküloz Olgusu

Spinal Tuberculosis Which Caused Gibbosity and Epidural Abscess

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This case report is presented as a poster on October 16, 2016 in TUSAD congress.

Öz

Spinal Tüberküloz (STB), akciğer TB' ye göre çok daha nadir görülmektedir. Vertebra infiltrasyonu nedeniyle nörolojik defisit ve ortopedik problemlere neden olabilmektedir. Vakamız, 27 yaşında erkek hasta, öksürük ve bel ağrısıyla hastanemize başvurdu. PA grafisinde özellik yoktu. Akciğer bilgisayarlı tomografisinde, bilateral apikal infiltrasyon, torakal vertebrada multipl litik lezyon, gibbus deformitesi ve spinal kord basısı izlendi. Bronş lavaj sıvısında yayma ARB ve TB kültürü negatif geldi. Torako lumber MR' da epidural abse görünümü olması nedenli cerrahi abse drenajı yapıldı. Vertebrada litik lezyon nedenli kemik biopsisi yapıldı. Kemik biopsisi yayma ARB pozitif bulundu ve kültüründe M. Tuberculosis üredi. Biyopsi sitolojisinde nekrotizan granülomatöz inflamasyon görüldü. Tüberküloz tedavisi ve cerrahi stabilizasyon ile sırt ağrısı geriledi. Spinal TB, özellikle TB' nin endemik olduğu bölgelerde, bel ağrısıyla başvuran hastalarda ayırıcı tanıda düşünülmesi gereken önemli bir hastalıktır.

Anahtar Kelimeler

Spinal Tüberkülozis; Epidural Abse; Litik Lezyon

Abstract

Spinal Tuberculosis (STB) is much rarer than pulmonary tuberculosis. It can cause neurological and orthopedic problems due to infiltration of vertebrae. Here we report a 27-year-old male patient who applied with cough and low back pain. The chest Xray was unremarkable. In thorax computer tomography there were consolidations at the left and right upper lobes, multiple lytic lesions in the spine, spinal cord compression and gibbous deformity in the thoracic vertebrae. The acid resistant bacteria (ARB) smear and Mycobacterium tuberculosis (MT) culture of bronchial lavage fluid were found to be negative. Due to appearing to be an epidural abscess in thoraco-lumbar magnetic resonance imaging (MRI), surgical drainage was performed. For lytic lesions on the spine, bone biopsy was performed. ARB smear of bone biopsies was positive and MT isolated in the Lowenstein-Jensen culture. The cytology of the biopsy showed necrotizing granulomatous inflammation. Back pain decreased with anti tuberculosis treatment and surgical stabilization. We emphasize that, STB, in MT endemic and other developing areas, is an important consideration the differential diagnosis of patients presenting with low back pain.

Keywords

Spinal Tuberculosis; Epidural Abscess; Lytic Lesion

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Introduction

Spinal tuberculosis (STB) is a form of bone TB that is caused by infiltration of a vertebral arch [1]. In endemic and other developing countries, in case that present symptoms such as loss of appetite, weight loss and low back pain, STB should be considered in the differential diagnosis. The initial stages of STB are insidious; radiological findings develop slowly and there are no specific symptoms. Therefore diagnosis of STB may be delayed. Here, we present a case of STB that caused gibbosity and was accompanied by epidural abscess.

Case Report

A 27-year-old man was admitted to the hospital with a complaint of non-productive cough and low back pain occuring for the previous month. The low back pain was constant and had no relationship with physical effort. There were no other related features in the patient's medical history. The patient had tenderness on his back, but no other related features upon physical examination.

The erythrocyte sedimentation rate (ESR) was 38 / h and the sputum smear was negative for any bacteria. The chest X-ray of the patient was non-diagnostic (Figure 1). Non-specific antibiotherapy (amoxicillin-clavulanic acid) was initiated. The thorax computed tomography (CT) showed tree-in-bud infiltration at the right and left upper lobes of the lung and in the cavity in the right upper lobe (Figure 2).

Also, lytic lesions in the anterior and lateral spine, causing gibbous deformity and compression to the spinal cord were seen. The thoraco-lumbar magnetic resonance imaging (MRI) showed severe kyphotic deformation, epidural abscess and spinal cord compression on the T8-T10 levels. Also there was spondylodiscitis at the T3-T6 levels (Figure 3-4). The three acid resistant bacteria (ARB) smears were negative in induced sputum smear for Mycobacterium tuberculosis (MT). Due to bilateral apical infiltration of the cavity in the CT, fiberoptic bronchoscopy was performed. Bronchial lavage fluid that had been taken from bilateral upper lobes was positive for ARB.

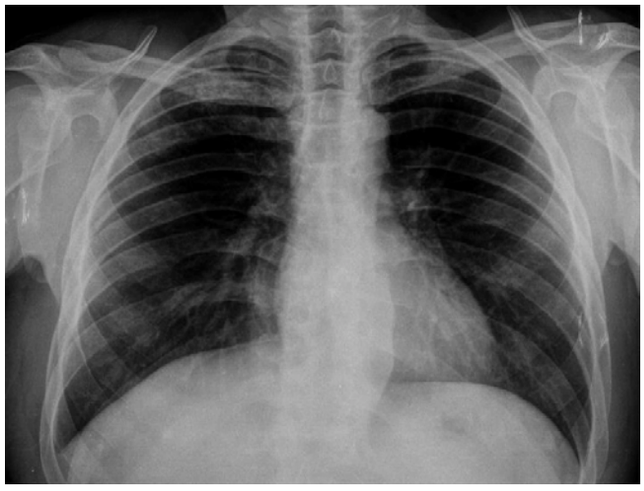

Figure 1. Initial PA Graphy

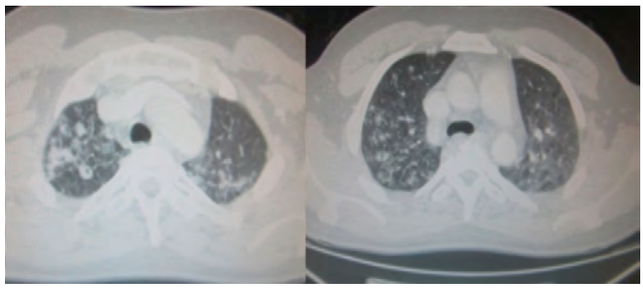

Figure 2. Thorax CT – Tree-in-bud infiltration at the right and left upper lobes of the lung and cavity in the right upper lobe.

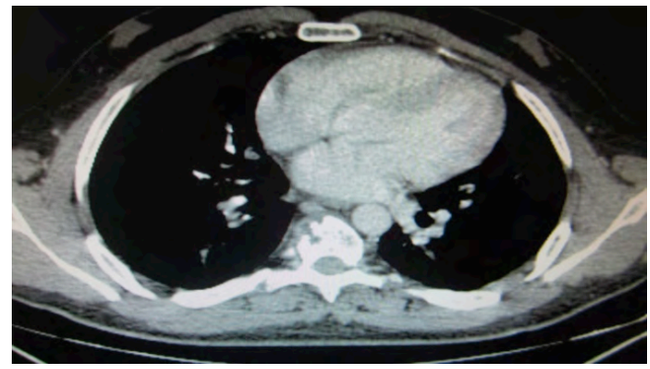

Figure 3. Thorax CT- Vertebrae lytic lesion

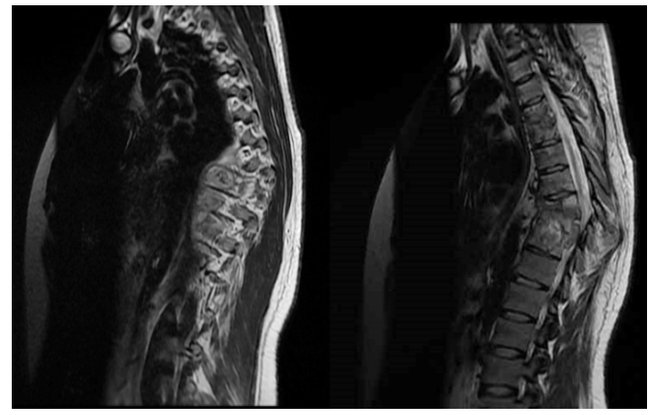

Figure 4. Thoraco-lumbar MRI - Severe kyphotic deformation, epidural abscess and spinal cord compression on the T8-T10 levels.

Spinal epidural abscess drainage and vertebral bone biopsy were performed from the thoraco-lumbar regions. Histopathology of the T3-T4 and T8-T10 vertebral bone biopsies detected necrotizing granulomatous infiltration. ARB smear of bone biopsies was positive and MT isolated in the Lowenstein-Jensen culture also. Anti-TB treatment was initiated as isoniazid 300 mg / day, rifampicin 600 mg / day, pyrazinamide 1.5 g / day, and ethambutol 1.5 g / day. Additionally, anterior surgical stabilization was implemented because of the infiltration of T8-T10. In the follow-up, the patient's symptoms had decreased.

Discussion

STB was first described in 1782 by Sir Percival Pott, so is also called Pott's disease [1]. STB is much rarer than pulmonary tuberculosis. It constitute 1-1.5% of all TB cases, and 15% of all extrapulmonary tuberculosis [2-3]. STB is especially seen in developing countries where it is endemic, such as Pakistan and India. But it is also seen in developed countries. Furthermore, STB cases have been increasing in recent years due to increased immundeficiency in cases of HIV infection, diabetes mellitus, cancer, etc. [4].

Patient complaints, radiological findings, microbiologic diagnosis of MT and necrotizing granulomatous inflammation in histopathology are crucial in the diagnosis of STB. Due to clinical and radiological similarities in differential diagnosis, it can often be confused with vertebral tumor and metastatic lesions. However, STB usually has anterior vertebrae involvement,

whereas tumors have more posterior vertebrae involvement [5]. In this case, anterior vertebrae involvement was observed.

STB may cause neurological deficits and spinal deformity. The most serious complications are paraplegia and tetraplegia. Although neurological deficits were not seen in this case, the possibility of neurological deficits was high because of spinal cord compression, epidural abscess and destruction of vertebrae. Therefore, early diagnosis and appropriate treatment of these cases is important.

Spinal epidural abscess (SEA) is quite rarely observed. It is mostly seen in the lumbar region with multiple vertebral articulation involvement. In this case, the involvement of three consecutive joints of the thoracal vertebrae was observed in the thoraco-lumbar MRI. Although SEA is predominantly due to predisposing factors such as HIV infection, diabetes and trauma, no other disease or predisposing factor was found in the case. SEA often appears to be secondary to pre-existing infection in another organ. In our case, epidural abscess was thought to have developed secondary to pulmonary tuberculosis.

The risk of kyphosis is quite high in STB. As the number of infiltrated vertebrae segments increases, the risk of kyphosis development increases accordingly. In another study, the kyphosis rate was found to be 38% in STB [6]. Due to consecutive three segment involvement, kyphosis was also observed in this case.

In the pharmacological treatment of spinal TB, antituberculous therapy (HRZE) consisting of isoniazid, rifampicin, pyrazinamide and ethambutol is recommended for at least nine months. In the intensive phase, two months HRZE treatment, and then 7-10 months HR treatment during the continuation phase is recommended [7]. Surgical treatment varies according to the degree of bone involvement. It has been shown that aggressive surgical treatment improves the efficacy of drug treatment and prevents neurological deficits [8]. Surgical anterior stabilization is required for long segment involvement of vertebrae. Posterior stabilization is applied to maintain an upper and a lower healthy segment. In addition to pharmacological treatment, surgical anterior stabilization was performed on our patient because of the long segment involvement of the vertebrae.

We emphasize that, in TB endemic countries and in developing countries, STB should be considered in the differential diagnosis in patients with back pain and destructive lesions of the vertebrae. Furthermore, surgery and/or anti-tuberculosis therapy should be given promptly after STB diagnosis.

Competing interests

The authors declare that they have no competing interests.

References

1. Gorse GJ, Pais MJ, Kusske JA, Cesario TC. Tuberculous spondylitis. A report of six cases and a review of the literature. Medicine (Baltimore) 1983 May;62(3):178-93.
2. Almeida A. Tuberculosis of the spine and spinal cord. Eur J Radiol 2005; 55:193-201.
3. Rajasekaran S. The problem of the deformity in spinal tuberculosis. Clin Orthop Relat Res 2002; 398:85-92.
4. Jain AK. Treatment of tuberculosis of the spine with neurologic complications. Clin Orthop Rel Res 2002; 398:75-84.
5. Aydın GB, Men S, Solak AŞ, Hücümenoğlu S. Metastazı taklit eden multisentrik spinal tüberküloz. Hacettepe Ortopedi Dergisi 2000; 10-4:118-21.
6. Necati T, Zeki O. Pott Hastalığının Tedavisinde Stabilizasyonun Yeri. J Kartal TR 2015;26(3):217–225. doi: 10.5505/jkartaltr.2015.94803
7. Recep A, editor. T.C. Sağlık Bakanlığı Tüberküloz Tanı ve Tedavi Uzlaşı Rehberi, Ankara; 2011;18-19,79.
8. Müslüman AM, Ziyal İM, Yılmaz A, Canpolat A, Duman H, ark. Pott hastalığında cerrahi tedavi sonuçlarını etkileyen faktörler. Türk Nöroşirürji Dergisi 2001;11:43-50.

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Unexpected Contralateral Femoral Artery Atheromatous Plaque Embolism After TAVI

TAVI Sonrası Beklenmedik Kontrlateral Femoral Arter Aterom Plak Embolisi

Femoral Artery Embolism After TAVI

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Öz

Transfemoral transkateter aort kapak implantasyonu ile ilişkilendirilen vasküler komplikasyonlar sıklıkla arteriyel kılıf yerleştirilmesinin istenmeyen sonuçlarıdır. Bu olguda Sağ femoral arter cerrahi kanülasyonu yapılmasını takiben gelişen sol ana femoral arter aterom plak embolisi sunulmaktadır. Transfemoral erişim yolu kullanılıyorsa, bilateral alt ekstremite nabızları yakından izlenmeli ve akut iskemi durumunda, acil operasyon gerekliliği unutulmamalıdır.

Anahtar Kelimeler

Aort Kapak Stenozu; Transkateter Aort Kapak Replasmanı; Ateroembolism; Embolektomi

Abstract

Vascular complications associated with transfemoral transcatheter aortic valve implantation are frequent and unfortunate consequences of arterial sheath insertion. The current report presents a case of atheromatous plaque embolization to the left common femoral artery when the right femoral artery is cannulated by surgical cutdown following transcatheter aortic valve implantation. If the transfemoral access route is used, bilateral lower extremity pulses should be monitored closely and, in the case of an acute ischemia, the necessity of emergency operation should be noted.

Keywords

Aortic Valve Stenosis; Transcatheter Aortic Valve Replacement; Ateroembolism; Embolectomy

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Introduction

Aortic stenosis (AS), the most common form of adult valvular heart disease, has become more prevalent in the aging population [1]. Transcatheter aortic valve implantation (TAVI) is an effective and reliable treatment method in patients with severe AS, who exhibit high surgical risk and are ineligible for surgical intervention. Despite being a less invasive method, this procedure can lead to various complications [2,3]. The current report presents a case of atheroma plaque embolization to the left common femoral artery, while the right femoral artery is cannulated by surgical cutdown.

Case Report

An 87-year-old woman with chest pain and NYHA (New York Heart Association) functional dyspnea class III-IV was referred to our institution with severe AS. Transthoracic echocardiography revealed a calcified and immobilized aortic valve. The mean aortic pressure gradient was 47 mmHg. The calculated aortic valve area was 0.70 cm^2 according to the continuity equation. The aortic annulus diameter was 23 mm, as measured by transesophageal echocardiography. There was no critical stenosis of the coronary arteries, and peripheral angiography revealed a femoral artery diameter of 6 mm (Figure 1A-B). She had a history of chronic obstructive lung disease and chronic renal failure (creatinine clearance: 32 ml/min). The patient's logistic Euroscore and Society of Thoracic Surgeons scores were calculated as 27% and 8.5%, respectively. The heart team (HT), which included cardiovascular surgeons, clinical and interventional cardiologists, as well as anesthesiologists, decided to proceed with TAVI using 26 mm CoreValve (Medtronic) by the transfemoral access route.

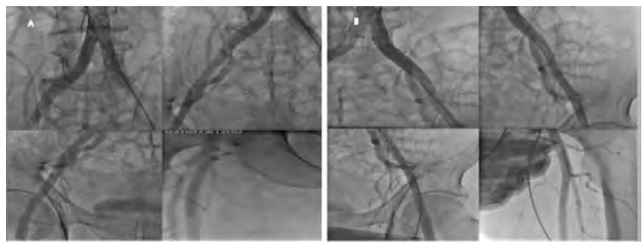

Figure 1. Pre-procedural angiographic images of aortoiliac bifurcation and the right iliofemoral arteries(A). Pre-procedural angiographic study of the left iliofemoral arteries(B).

Initially, unfractionated heparin (UFH) was administered perioperatively at a dose of 6000 IU (100 IU/kg) for anticoagulation. The antiplatelet regimen consisted of 100 mg acetylsalicylic acid and 75 mg clopidogrel administered before and after the procedure. After the induction of general anesthesia, the right common femoral artery (CFA) was cannulated by surgical cutdown. An 18 F introducer sheath was inserted through the right CFA. A 6F pigtail catheter was inserted for aortography through a 6F sheath to the left CFA and positioned at the aortic root during the procedure. A temporary pacemaker was placed in the right ventricle via the left femoral vein. A 0.035-inch Amplatz Super Stiff wire was inserted into the left ventricle through the 18 F sheath. Balloon dilatation of the stenotic aortic valve was performed with a balloon, under rapid pacing, using a temporary pacemaker. Then, a 26-mm CoreValve was deployed in the aortic annulus under angiographic guidance. Immediate post-procedural aortogram revealed the CoreValve to be in a good position. At the end of the process, the right CFA was surgically repaired.

After the procedure, the patient was taken to the intensive care unit, with continuous intravenous heparin therapy. During follow-up in the intensive care unit, the patient's left leg showed ischemic signs and became cold. The left femoral artery sheath was removed immediately. Distal extremity pulses were not palpable. Emergency Doppler ultrasonography revealed an occluded left common femoral artery.

The patient was immediately taken to the operating room. Under local anesthesia, the left common femoral artery was explored by transverse arteriotomy, first with a scalpel blade, then enlarged with Potts scissors. After performing embolectomy with 4F Fogarty catheter, arteriotomy was closed with 6-0 polypropylene suture. There was no thrombus in the femoral artery, but massive atherosclerotic plaque was removed from the femoral artery (Figure 2). After the operation, the distal pulses were palpable. The postoperative course was uneventful and the patient was discharged after four days with dual antiplatelet therapy.

Figure 2. The image of massive atherosclerotic plaque removed from the contralateral femoral artery.

Discussion

TAVI is a less invasive complex procedure in high-risk patients, but several complications can occur during or after the procedure. Consequently, transapical, trans-subclavian, transfemoral, and direct aortic access routes have been proposed for TAVI [4]. Among these, the transfemoral access route represents the most widely used and least invasive approach, but is also associated more frequently with vascular complications [5].

Vascular complications have been associated with significantly increased morbidity, mortality, and prolonged hospital stays [6]. The incidence of major vascular complications ranges between 5.6% and 17.3% [7]. The rate of contralateral femoral artery atheromatous plaque embolism is 1.1% (1 of 87 patients), in our experience.

The patients' pre-procedural evaluation must be performed cautiously to avoid major vascular complications. Pre-procedural angiography and/or CT can be useful to predict and avoid possible vascular complications during transfemoral TAVI. Careful patient screening and selection is essential, but complications can never be completely eliminated. It is important to be prepared for all mishaps that may occur, such as those related to large occlusion balloons, covered stents, surgical instruments, and bleeding. Most importantly, the surgeon should not hesitate to use non-iliofemoral access if the iliofemoral vessels are inadequate [4].

To determine the feasibility of an arterial approach, we used angiography to assess the presence and severity of iliofemoral disease with a femoral artery diameter of 6 mm. CoreValve's newer low-profile system with 18 F sheath (outer diameter: approximately 7 mm) was used in our approach and was deployed successfully with an uneventful procedure period.

Ideally, the minimal lumen diameter should exceed the diameter of the delivery system. However, in the absence of extensive calcification, bulky atheroma, or severe tortuosity, short segments of a relatively compliant artery 1 to 2 mm smaller in diameter than the intended sheath can often be safely cannulated [2].

In our case, the lumen diameter was 1 mm smaller than the sheath diameter and the intervention was successfully completed without any difficulty, but an atheromatous plaque embolization occurred despite the absence of difficulty during the sheath insertion. We believe that when the right common femoral artery was cannulated with a large-bore catheter, the atheromatous plaque was peeled off by the catheter and deposited at the iliac bifurcation. The embolization occurred during removal of the catheter from the iliac bifurcation to the left common femoral artery.

During the placement of a transcatheter aortic valve, iliofemoral artery intimal injury and peeling or rupture complications can occur [8].

Vascular access route complications during TAVI are a life-threatening problem that can only be treated successfully if immediately recognized and managed effectively. Because of the relatively large (18 F) sheath required, vascular complications may occur during and after CoreValve placement.

These complications are usually expected from the large arterial access routes used for sheath insertion.. Our case was unique, an atheromatous plaque embolization to the contralateral femoral artery, in which a sheath had not been inserted. To the best of our knowledge, this is the first case report of embolization of an atheromatous plaque of the contralateral CFA due to cannulation of the right CFA with an 18 F sheath during a TAVI procedure.

In cases of a relatively small-diameter femoral artery and difficult sheath insertion, other access routes should be considered. Also, pre-procedural angiography and/or CT can be useful to predict and avoid possible vascular complications during transfemoral TAVI. The surgeon must follow a meticulous and attentive technique in order to avoid vasospasm during the procedure. When the transfemoral access route is used, extremity pulses, ischemia signs, and hemodynamic monitoring should be followed closely, and the possibility of the development of bilateral acute lower extremity ischemia must always be kept in mind. In the case of an acute ischemia, the necessity for an emergency operation should be noted.

Competing interests

The authors declare that they have no competing interests.

References

1. Sawaya F, Stewart J, Babaliaros V. Aortic stenosis: Who should undergo surgery, transcatheter valve replacement? Cleve Clin J Med 2012;79(7):487-97.
2. Masson JB, Kovac J, Schuler G, Ye J, Cheung A, Kapadia S et al. Transcatheter aortic valve implantation: review of the nature, management, and avoidance of procedural complications. JACC Cardiovasc Interv 2009;2(9):811-20.
3. Burgazlı MK, Chasan R, Kavukçu E, Neuhof C, Bilgin M, Soydan N et al A. Transcatheter Aortic Valve Implantation: Our Experience and Review of the Literature. Balkan Med J 2012;29(2):118-23.
4. Ramlawi B, Anaya-Ayala JE, Reardon MJ. Transcatheter aortic valve replacement (TAVR): Access planning and strategies. Methodist Debakey Cardiovasc J 2012;8(2):22-5.
5. Hayashida K, Lefevre T, Chevalier B, Hovasse T, Romano M, Garot P et al. Transfemoral aortic valve implantation: new criteria to predict vascular complications. JACC Cardiovasc Interv 2011;4(8):851–8.
6. Rodés-Cabau J, Webb JG, Cheung A, Ye J, Dumont E, Feindel CM et al. Transcatheter aortic valve implantation for the treatment of severe symptomatic aortic stenosis in patients at very high or prohibitive surgical risk: acute and late outcomes of the multicenter Canadian experience. J Am Coll Cardiol 2010;55(11):1080–90.
7. Magri CJ, Chieffo A, Durante A, Latib A, Montorfano M, Maisano F et al. Impact of mean platelet volume on combined safety endpoint and vascular and bleeding complications following percutaneous transfemoral transcatheter aortic valve implantation. BioMed Research International 2013;2013:645265. doi: 10.1155/2013/645265.
8. Dagdelen S, Karabulut H, Senay S, Akyol A, Toraman F, Cagıl H et al Transcatheter aortic valve implantation in patients with high-risk aortic stenosis: a clinical follow-up. Turk Gogus Kalp Dama 2011;19(4):495-502.

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Idiopathic Pulmonary Hemosiderosis with Celiac Disease; Lane-Hamilton Syndrome

Çölyak Hastalığıyla Birlikte İdiyopatik Pulmoner Hemosiderosiz; Lane-Hamilton Sendromu

IPH with Celiac Disease; Lane Hamilton Syndrome

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Öz

Yirmi bir yaşında erkek hasta acil kliniğimize 2 gündür devam eden kanlı balgam, nefes darlığı, halsizlik ve yorgunluk şikayetiyle başvurdu. Tüm vital bulguları normal olmasına karşın parmak ucu oksijen saturasyonu %92 ölçüldü. Laboratuvar tetkiklerinde hemogramında hemoglobin:8.85g/dL, hematocrit:28.7% olarak tespit edildi. PT ve aPTT normaldi. Romatolojik laboratuvar tetkikleri negatifti. Hafif kanlı bronkoalveolar lavaj sıvısı patoloji preparatlarında "hemosiderin yüklü makrofajlar" görüldü ve idiyopatik pulmoner hemosiderosis (IPH) tanısı konuldu. Gastroduedonoskopi işleminde duedonumda nodülarite görüldü ve bu bölgeden biyopsiler alındı. Biyopsi sonucu çölyak hastalığı ile uyumlu duedonal mukoza villöz atrofisi" olarak rapor edildi. Lane-Hamilton sendromu tanısıyla olguya tedavide sadece glutensiz diyet önerildi. Altı ay takip sonrasında olguda tam remisyon gelişti. Sonuç olarak, çölyak hastalığı ve IPH birlikteliğinden dolayı, IPH'ye bağlı alveoler hemorajilerde, tanı aşamasında uyanık olmamız gerekmektedir. Lane-Hamilton sendromunda glutensiz diyet ile tam remisyon sağlanabilmektedir.

Anahtar Kelimeler

İdiyopatik pulmoner Hemosiderosiz; Çölyak Hastalığı; Lane-Hamilton Sendromu

Abstract

A 21-year-old male patient presented to our emergency department with the complaints of bloody sputum, respiratory difficulty, lethargy, and fatigue persisting for the previous two days. Fingertip oxygen saturation was 92%, while other vital signs were normal. Bilateral ground-glass opacities were present at thoracic computerized tomography. Laboratory findings were hemoglobin:8.85g/dL, hematocrit:28.7%. PT and aPTT values were normal. All rheumatologic laboratory tests were negative. Bronchoalveolar lavage was mildly hemorrhagic and "hemosiderin-laden macrophages" were observed in pathology specimens. The case diagnosed withidiopathic pulmonary hemosiderosis (IPH). Gastroduodenoscopy revealed nodularity in the duodenum, and mucosal biopsies taken from these duedonal regionswere reported as "villous atrophy in mucosal tissues in the duodenum compatible with celiac disease". The only recommended treatment was a gluten-free diet. At follow-up approximately 6 months later, complete remission was achieved. In conclusion, we should be aware, when seeing alveolar hemorrhage related to IPH, that celiac disease can accompany IPH. The concurrence of IPH and celiac disease is known as Lane-Hamilton syndrome. Complete remission in Lane-Hamilton syndrome can be achieved with a gluten-free diet.

Keywords

Idiopathic Pulmonary Hemosiderosis; Celiac Disease; Lane-Hamilton Syndrome

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Introduction

Idiopathic pulmonary hemosiderosis (IPH) is a disease of unknown cause that may lead to recurring alveolar hemorrhage. It isgenerally characterized by hemoptysis, iron deficiency anemia, and diffuse parenchymal infiltration in the lungs in childhood. Definitive diagnosis of IPH is made with histopathological observation of hemosiderin-laden macrophages in pulmonary tissue specimens, sputum, or bronchoalveolar lavage BAL [1]. If untreated, IPH may lead to pulmonary fibrosis [2]. Celiac disease (CD) is a malabsorption syndrome, also known as gluten enteropathy, proceeding with chronic diarrhea, weight loss, and findings of delayed growth and development. Inflammation and villous atrophy in the small intestine resulting from autoimmune enteropathy constitute the basic pathophysiology of the disease in genetically predisposed individuals with the digestion of foods containing gluten [3]. Lane-Hamilton syndrome, first described in 1971, is a rare condition representing a combination of IPH and CD [4]. While IPH responds well to corticosteroids, a gluten-free diet can reduce the need for corticosteroids in the treatment of Lane-Hamilton syndrome [5].

Case Report

A 21-year-old male patient presented to our emergency department with the complaints of bloody sputum, respiratory difficulty, lethargy, and fatigue persisting for the previous two days. Physical examination revealed pale sclera and fine rales in the basal areas of both lungs. Fingertip oxygen saturation was 92%, while other vital signs were normal. There was no specific disease or drug in his medical history. Bilateral ground-glass opacities were present at thoracic computerized tomography (CT) (Figure 1).

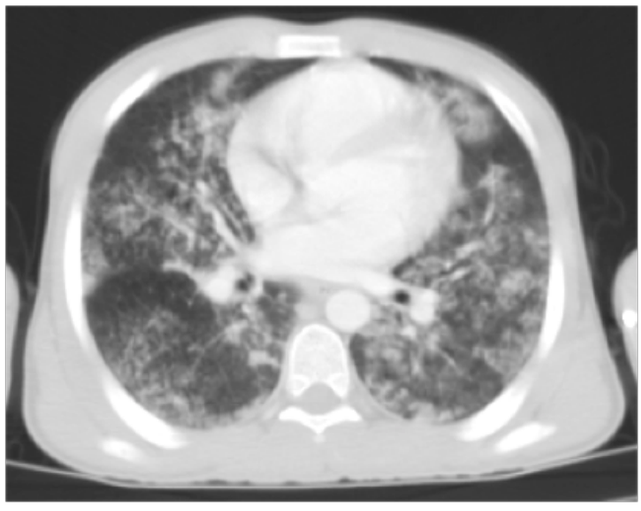

Figure 1. Parenchymal thoracic CT section; hemorrhage-related bilateral ground glass appearance

Laboratory findings were white blood cell:6.24K/uL, red blood cell:3.94 M/uL, hemoglobin: 8.85g/dL, hematocrit: 28.7%, mean corpuscular volume: 72.8 fL, mean corpuscular hemoglobin:22.5pg, mean corpuscular hemoglobin concentration:30.9 g/dL, platelet:355 K/uL, C-reactive protein:0.484 mg/dL, sedimentation:18/hour, glucose:118mg/dL, urea:25 mg/dL, creatinine:0.66 mg/dL, rheumatoid factor<20 IU/mL, iron:12 mg/dL, iron binding capacity:462 mg/dL, total iron binding capacity:464 mg/dL, ferritin: 20.06 ng/ml, vitamin B12:161 pg/mL, and folate:1.9 ng/ml.PT and aPTT were normal. The rheumatologic workup, including all of the antinuclear antibodies, anti-double stranded antibodies, antineutrophil cytoplasmic antibodies, and anti-glomerular basement membrane antibodies were negative.

Fiberoptic bronchoscopy revealed no endobronchial lesions. Bilateral BAL fluid was mildly hemorrhagic. BAL pathology was reported as “hemosiderin-laden macrophages. Therefore, IPH was diagnosed and we treated the patient with corticosteroid (methylprednisolone 1x120 mg) and tranexamic acid (250 mg, 3x1) for five days. The hemoptysis resolved entirely on the third day of treatment and the respiratory symptoms improved dramatically (fingertip oxygen saturation was 96%). The patient, who had hypochromic microcytic anemia, was consulted with the gastroenterology department on the fifth day of treatment. Tests to detect the presence of CD were positive: serum tissue transglutaminase IgA>200 RU/ml, anti-gliadin IgG 200.8 RU/ml, and anti-gliadin IgA >200 RU/ml. Gastroduodenoscopy revealed nodularity in the duodenum, and mucosal biopsies were taken from these duedonal regions (Figure 2A). The biopsies were reported as “villous atrophy in mucosal tissues in the duodenum compatible with celiac disease, focal intraepithelial lymphocyte increase, and crypt hyperplasia with accompanying inflammatory cell infiltration of plasma cells and eosinophils in the lamina propria” (Figures 2B,C,D). CD was diagnosed and we treated the patient with a gluten-free diet. The ground-glass opacities in the pulmonary regions at control thoracic CT resolved in the first week of the treatment. There were no longer any pulmonary symptoms. We discharged the patient on the eighth day of treatment. The only treatment recommendation was a gluten-free diet. At follow-up approximately 6 months later, complete remission was achieved. Hemoglobin levels had returned to normal, and there was no recurrence of bloody sputum or other pulmonary symptoms.

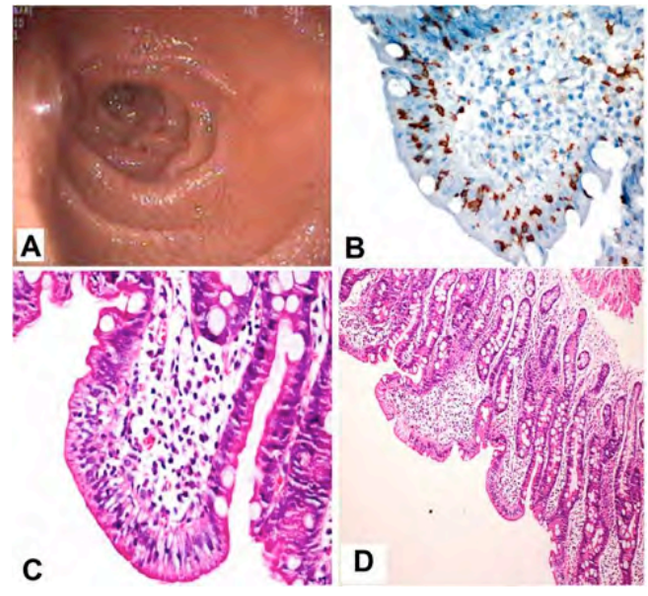

Figure 2. Endoscopic appearance of the duodenum. Appearance of the duodenal mucosa (scalloped feature)(A), Villous athrophy in the small intestine, increased intra-epithelial lymphocytes (hematoxylin-eosin staining) (B), Abundant inflammatory cell infiltration from plasma cells and lymphocytes in the lamina propria (hematoxylin-eosin staining) (C), Increased intra-epithelial lymphocytes staining positive with CD3 (D).

Discussion

Diffuse alveolar hemorrhage (DAH) is a clinical condition involving alveolar hemorrhage deriving from pulmonary microcirculation and alveolar injury. Several conditions involving immune and non-immune mechanisms can lead to DAH. IPH is rarely capable of causing DAH. IPH was first described, as 'brown induration,' by Virchow in 1864 [1]. The prevalence of IPH is estimated at 0.24-1.23/million and IPH is generally seen in adults under the age of 30 [6,7]. A combination of IPH and CD is known as Lane-Hamilton syndrome, the underlying pathogenic mechanism of which is unclear [4]. Parelman et al. implicated three mechanisms in the combination of IPH and CD. The first of these is the accumulation of immune complexes containing food allergens in the basal membranes of alveolar capillaries. The second is cross-relativity between alveolar basal membrane antigen and anti-reticulin antibodies, and the third is the potential involvement of adenovirus 12, a possible etiological agent in celiac disease, in IPH [8]. Studies have reported improvements in pulmonary symptoms associated with IPH following a gluten-free diet used in the treatment of CD [3,5]. In our case, 5-day corticosteroid therapy was administered for the treatment of IPH. After discharge the patient was prescribed a gluten-free diet as treatment. At 6-month follow-up there was no recurrence of either pulmonary or gastrointestinal system (GIS) symptoms with a gluten-free diet, and complete remission was achieved.

In conclusion, we should be aware, when there isalveolar hemorrhage related to IPH, that CD can accompany IPH with or without GIS symptoms. We need to further investigate whether patients with IPHhave GIS symptoms. It should be remembered that in Lane-Hamilton syndrome complete remission can be achieved with a gluten-free diet if the patient is pulmonary asymptomatic, and that corticosteroid therapy can thus be reduced. To the best of our knowledge, there are few case reports about Lane-Hamilton syndrome in the literature. The present case report may point the way to future research about Lane-Hamilton syndrome.

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Competing interests

The authors declare that they have no competing interests.

References

1. Ioachimescu OC, Sieber S, Kotch A. Idiopathic pulmonary hae¬mosiderosis revisited. Eur Respir J 2004;24(1):162–70.
2. Bakalli I, Kota L, Sala D, Celaj E, Kola E, Lluka R et al., Idiopathic pulmonary hemosiderosis a diagnostic challenge. Ital J Pediatr 2014;40:35.
3. Hammami S, GhediraBesbes L, Hadded S, Chouchane S, Ben Mer¬iem C, Gueddiche MN. Co-occurrence pulmonary haemosidero¬sis with coeliac disease in child. Respir Med 2008;102(6):935–6.
4. Lane DJ, Hamilton WS. Idiopathic steatorrhoea and idiopathic pulmonary haemosiderosis. Br Med J 1971;2(5753):89–90.
5. Sethi GR, Singhal KK, Puri AS, Mantan M. Benefit of gluten-free diet in idiopathic pulmonary hemosiderosis in association with celiac disease. Pediatr Pulmonol 2011;46(3):302-5.
6. Ohga S, Takahashi K, Miyazaki S, Kato H, Ueda K. Idiopathic pulmonary haemosiderosis in Japan: 39 possible cases from a survey questionnaire. Eur J Pediatr 1995;154:9945.
7. Kjellman B, Elinder G, Garwicz S, Svan H. Idiopathic pulmonary haemosiderosis in Swedish children. Acta Paediatr Scand 1984;73:5848.
8. Perelman S, Dupuy C, Bourrillon A.The association of pulmonary hemosiderosis and celiac disease. Apropos of a new case in a child. Ann Pediatr (Paris) 1992;39(3):185-8.

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Case Report
JCAM

Isolated Palmoplantar Lichen Planus

İzole Palmoplantar Liken Planus Olgusu

Palmoplantar Liken Planus

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Öz

Liken planus deri, deri ekleri ve mukozayı tutan benign, inflamatuar ve kaşıntılı bir dermatozdur. Liken planus'un aktinik, anuler, atrofik, büllöz, hipertrofik, foliküler, lineer ve ülseratif varyantları vardır. Tırnak ve oral mukoza tutulumu da görülebilmektedir. Palmoplantar liken planus ise liken planusun nadir görülen lokalize varyantıdır. Palmoplantar bölgede stratum korneum daha kalın olduğundan klasik morumsu papüller yerine sarımsı lezyonlar görülür. Palmoplantar tip, klasik liken planusun tipik klinik morfolojisinden farklılık gösterdiği için tanısal zorluk yaratmaktadır. Bu bölgedeki lezyonlar klasik klinik morfolojiye uymadığından tanısı zordur ve kolaylıkla başka hastalıklarla karışır. Burada izole palmoplantar tutulum gösteren bir liken planus olgusu sunmaktayız.

Anahtar Kelimeler

Liken Planus; Palmoplantar

Abstract

Lichen planus is a benign, pruritic inflammatory dermatitis involving the skin, skin appendages and mucosa. Lichen planus have actinic, annular, atrophic, bullous, hypertrophic, follicular, linear and ulcerative variants. Nails and oral mucosa can also be involved. Palmoplantar lichen planus is a rare localized variant of lichen planus. Because stratum corneum is thicker in palmoplantar region, yellowish lesions are seen instead of classic purplish papules. Because it is different from the clinical morphology of the classic form of the lichen planus, palmoplantar type cause problems in diagnosis. Palmoplantar lichen planus is an extremely rare disease that can be easily confused with numerous other diseases, it can be easily missed. We present a case of lichen planus with isolated palmoplantar involvement.

Keywords

Lichen Planus; Palmoplantar

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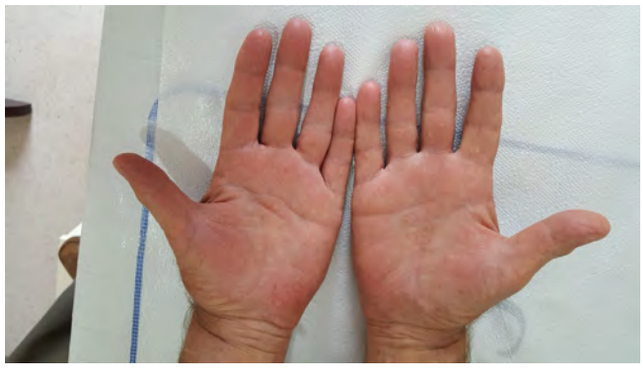
Introduction

Lichen planus is a benign, pruritic inflammatory dermatitis involving the skin, skin appendages and mucosa. Characteristic clinical finding is polygonal violaceous papules. Lichen planus has actinic, annular, atrophic, bullous, hypertrophic, follicular, linear and ulcerative variants. Nails and oral mucosa can be also involved [1]. Palmoplantar lichen planus (PPLP) is a rare localized variant of lichen planus [1-4]. Because it is different from the clinical morphology of the classic form of lichen planus, palmoplantar type cause problems in diagnosis [1,5]. This paper presents a clinically- and histopathologically-proven lichen planus patient with isolated palmoplantar involvement.

Case Report

A 60-year old male patient was admitted with itchy lesions in the palms and soles for about three months. He was previously treated with topical steroids and calcipotriol creams and moisturizers for palmoplantar psoriasis and palmoplantar keratoderma. Because the lesions in the soles but not those in the palms regressed, the patient was admitted to our clinic for further evaluation and treatment.

Past medical history and family history of the patient revealed no remarkable abnormalities. There was no previous use of medicines before the lesions appeared. Physical examination also revealed no abnormalities. On dermatologic examination, numerous yellowish keratotic papules of 2-3 mm in diameter were seen in bilateral palmoplantar regions [Figure 1 and 2].



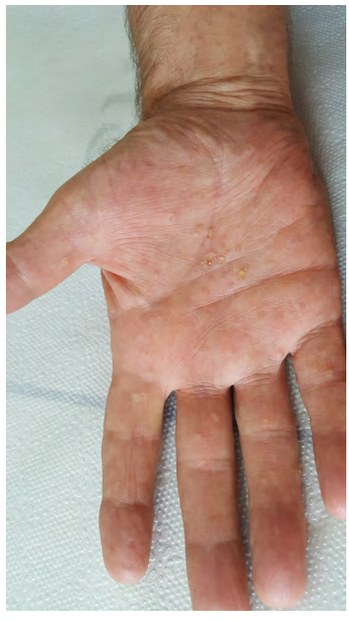


Figure 1. and Figure 2. Numerous yellowish keratotic papules of 2-3 mm in diameter were seen in bilateral palmar regions.

There was no lesions in other parts of the body. Hair, mucous membrane and nail examination was normal. Complete blood count, biochemistry and urine analysis were normal. Hepatitis markers were negative. Punch biopsy was taken from the palmar region of the left hand. On light microscopic examination, marked hyperkeratosis, wedge-shaped hypergranulosis and irregular acantosis were detected in the epidermis. An irregularity in the form of "sawtooth appearance" was found in the rete ridges. Band-like lymphocytic infiltration covering the dermoepidermal junction in the superficial dermis was observed. Histopathological findings were reported to be suggestive of "lichen planus" [Figure 3].

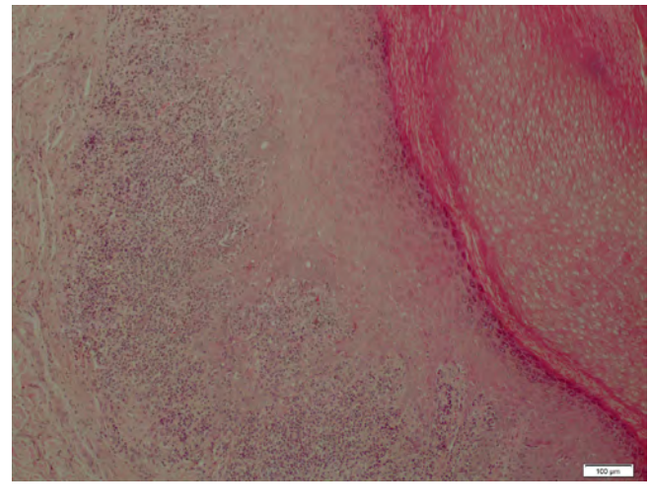


Figure 3. Histological examination of the lesion revealed hyperkeratosis, hypergranulosis, irregular acanthosis, irregularity in the form of "sawtooth appearance" was found in the ends of rete. Band-like lymphocytic infiltration in the superficial dermis (hematoxylin-eosin, original magnification×100).

The diagnosis of lichen planus was made in light of existing clinical and histopathological findings. Patient was treated with 32 mg/day oral methylprednisolone and topical clobetasol propionate. After achieving marked regression of the lesions at the second week of the treatment oral steroid treatment was stopped in 1 month by gradually decreasing the dose. No recurrence was observed during the follow-up period of 3 months.

Discussion

PPLP is a rare localized variant of lichen planus. Sanchez-Peres et al. have reported 26% palmoplantar involvement for lichen planus cases. It is more common in males than females with a peak age at third to fifth decades [5]. Because there are no other lesions in the body, diagnosis is difficult.

Because stratum corneum is thicker in palmoplantar region, yellowish lesions are seen instead of classic purplish papules [6]. PPLP can present with hypertrophic, diffuse squamous, punctate keratotic, diffuse keratoderma, erosive, pigmentated macular, vesicular, keratotic plaque and umbilicated papular morphological types [1,7].

The differential diagnosis of PPLP includes eczema, psoriasis, palmoplantar keratoderma, verruca vulgaris, secondary syphilis, lichen nitidus, arsenic keratosis and porokeratosis [5,7].

Histopathological features of PPLP are similar those of other types of lichen planus; skin biopsy is very helpful for the diagnosis [2]. In the present case, histopathological evaluation also supported the diagnosis of lichen planus.

The treatment options include topical and systemic steroids, acitretin, immunosuppressive agents (such as cyclosporine), PUVA, narrow band UVB, excimer laser and surgical methods [2,6]. Our case responded well to systemic and local steroid therapy.

Conclusion

In conclusion, because PPLP is an extremely rare disease that can be easily confused with numerous other diseases, it can be easily missed. Lichen planus should be kept in mind in the differential diagnosis of the diseases of palmoplantar region and histopathological examination should be performed in case of suspicion.

Competing interests

The authors declare that they have no competing interests.

References

1. Landis M, Bohyer C, Bahrami S, Bragan B. Palmoplantar lichen planus: A rare presentation of a common disease. J Dermatol Case Rep 2008;2:8-10.
2. Kim MJ, Choi M, Na SY, Lee JH, Cho S. Two cases of palmoplantar lichen planus with various clinical features. J Dermatol 2010;37:985-9.
3. Mehta V, Vasanth V, Balachandran C. Palmar involvement in lichen planus. Dermatol Online J 2009;15:12.
4. Rotunda AM, Craft N, Haley JC. Hyperkeratotic plaques on the palms and soles. Palmoplantar lichen planus, hyperkeratotic variant. Arch Dermatol 2004;140:1275-80.
5. Sanchez–Perez J, Rias Buceta L, Fraga J, Garcia-Diez A. Lichen planus with lesions on the palms and/or soles: prevalence and clinicopathological study of 36 patients. Br J Dermatol 2000;142:310-4.
6. Uçmak D, Azizoğlu R, Harman M. Palmoplantar lichen planus-a report of four cases. Journal of Clinical and Exerimental Investigations 2011;2:80-4.
7. Gutte R, Khopkar U. Predominant Palmoplantar Lichen Planus : A Diagnostic Challenge. Indian J Dermatol 2014;59:343-7.

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18-Year Nicotine Gum Addiction Treated with Bupropion: A Case Report

Bupropionla Tedavi Edilen 18 Yıllık Nikotin Sakız Bağımlılığı: Olgu Sunumu

Nicotine Gum Addiction

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Öz

Nikotin Replasman Tedavisi (NRT), uzun yıllardır sigara bırakma tedavisinde ilk seçenek olarak tercih edilmektedir. Nikotin replasman tedavisi, sigara içme isteğini ve yoksunluk belirtilerini azaltır. NRT tedavisi sırasında nadiren de olsa nikotin sakızına bağımlılık gelişebilir. Bu çalışmada 18 yıldır nikotin sakız bağımlısı olan ve tedavi için sigara bırakma polikliniğine başvuran ve bupropionla tedavi edilen bir olgu sunulmuştur. Nadir görülen nikotin sakız bağımlılığını tedavi etmede bupropionun da etkin bir seçenek olduğuna dikkat çekilmesi amaçlanmıştır.

Anahtar Kelimeler

Nikotin Sakız Bağımlılığı; Bupropion; NRT Komplikasyonu

Abstract

Nicotine Replacement Therapy (NRT) has been the preferred first option in smoking cessation treatment for many years. NRT reduces both the desire to smoke and the withdrawal symptoms. Nicotine gum addiction may rarely develop during NRT. In this study, we present a case who became addicted to nicotine gum for 18 years, was admitted to the smoking cessation polyclinic, and treated with bupropion. The aim of this case study is to highlight that bupropion is also an effective option to treat nicotine gum addiction, a rarely seen occurrence.

Keywords

Nicotine Gum Addiction; Bupropion; NRT Complication

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Introduction

According to data from the World Health Organization (WHO), the prevalence of cigarette smoking among persons aged 15 years and over was identified as 22% across the world. 6 million people die each year (1 person every 6 seconds) because of cigarette smoking or exposure to cigarette smoke [1].

Nicotine is the tobacco component that most contributes to the development of tobacco addiction. Nicotine acts by releasing various mediators in the body. These mediators include noradrenaline, acetylcholine, dopamine, 5-hydroxytryptamine, gamma aminobutyric acid, and endorphin [2].

Nicotine withdrawal syndrome is a group of symptoms that include irritability, insomnia, anxiety, decrease in concentration, confusion, feeling of hunger, impotence, and depression [3]. All smokers become addicted to nicotine in a short time and need medical support to quit smoking [4]. Medications used for smoking cessation are nicotine replacement therapy, bupropion, and varenicline [2].

In this study, we present a case study of a patient who became addicted to nicotine gum for 18 years, was admitted to the smoking cessation polyclinic, and was treated with bupropion.

Case Report

A 53-year-old male patient was admitted to the smoking cessation polyclinic in our hospital to treat nicotine gum addiction. The patient had started using nicotine gum to quit smoking 18 years previously and had become addicted to nicotine gum. He could not quit the gum use with his own efforts. He had been regularly using 6 pieces of 4 mg nicotine gum daily for 18 years. His physical examination and laboratory findings when admitted to the clinic were normal. A posteroanterior chest X-ray revealed bronchovascular markings. Bupropion was started at 150 mg once daily in the morning for the first three days and then was increased to 150 mg twice daily in the morning and evening. He was asked to stop using nicotine gum one week after the treatment was started. In a follow-up call 15 days later, he reported having stopped using nicotine gum at the end of the first week and also reported no longer having any desire for it. Bupropion therapy was terminated after three months of follow-up.

Discussion

Tobacco addiction, one of the leading causes of preventable deaths in the world, is difficult to treat. Addiction treatment is difficult because both physical and psychological addictions develop; intensive withdrawal symptoms appear if tobacco is discontinued. Various treatment programs are applied to people in smoking cessation centers. In addition to counseling and psychological support, pharmacological treatment is also used effectively. Nicotine replacement therapy, bupropion, and varenicline are the most effective and reliable agents among pharmacological medications.

Nicotine Replacement Therapy (NRT) has been preferred as the first option in smoking cessation treatment for many years. NRT increases smoking cessation at two times the rate of a control group [3]. It significantly reduces the desire to smoke and the withdrawal symptoms by providing a slow, steady amount of nicotine to the blood stream [4]. There are more than 188 studies and numerous meta-analytic studies on the subject in the literature. NRT preparations are transdermal patch, gum, nasal spray, sublingual tablet, and inhaler [2]. Nicotine gum has two forms, 2 mg and 4 mg. 2 mg is recommended for those who smoke <25 cigarettes/day and 4 mg is recommended for those who smoke ≥25 cigarettes/day. The total daily dose should not be over 24 pieces [2]. The most common side effects are irritation in the mouth, jaw pain, dyspepsia, hiccup, and nausea. Nicotine gum addiction may rarely develop during treatment [4].

Bupropion, which is an antidepressant acting through the dopaminergic pathway in the central nervous system, has been used in the United States and many other countries for 20 years. In two large randomized prospective clinical studies it was shown to significantly reduce the desire to smoke and was approved for smoking cessation treatment [2]. It is the most commonly used safe and effective treatment medication, behind NRT, in smoking cessation treatment [5]. The mechanism of action of bupropion for smoking cessation is not exactly known [2,5]. However, the smoking cessation effect is independent of its antidepressant effect [5]. Bupropion reduces the desire to smoke and the withdrawal symptoms. It has a high smoking quit rate at the end of the first year of treatment compared to placebo. Bupropion 150 mg tablets are a sustained release (SR) tablet [5]. 150 mg of bupropion is taken daily for the first three days and then 300 mg of bupropion is taken daily. The duration of treatment is usually 6-12 weeks, but bupropion can be safely used for a longer period of time.

In this paper we have described a rare case of nicotine gum addiction treated with bupropion. There are very few case reports in the literature on this subject.

In the United States in 1988, it was reported that a patient became addicted to nicotine gum used to treat tobacco addiction. No side effects were observed in the patient who had used nicotine gum for 4 years and who had asthma. It was concluded that nicotine gum has a high potency for tobacco addiction and no significant side effects [6]. In our patient, although he had been regularly using 24 mg nicotine gum daily for 18 years, no significant side effects were observed.

In Australia in June 2015, it was reported that a patient who had a 30-year history of high-dose nicotine gum use (up to 200 mg nicotine) and who experienced excessive sweating was treated with a nicotine patch. He stopped using nicotine gum within one week and did not feel the urge to use nicotine gum during the 6-month follow-up period. His excessive sweating improved immediately after stopping the use of nicotine gum. It has been concluded that nicotine patches can be an effective treatment for long-term nicotine gum addiction [7].

In this study, we report that bupropion can also be an effective option to treat nicotine gum addiction.

Competing interests

The authors declare that they have no competing interests.

References

1. Sayan İ, Tekbaş ÖF, Göçgeldi E, Paslı E, Babayiğit M. Bir eğitim hastanesinde görev yapan hemşirelerin sigara içme profilinin belirlenmesi. Genel Tıp Dergisi 2009;19(1):9-15.
2. Rennard SI, Daughton DM. Smoking cessation. Chest 2000;117(5):360-4.
3. Haustein KO. Pharmacotherapy of nicotine dependence. Int J Clin Pharmacol Ther 2000;38(6):273-90.

4. Campbell I. Nicotine replacement therapy in smoking cessation. Thorax 2003;58(6):464-5.
5. Roddy E. Bupropion and other non-nicotine pharmacotherapies. BMJ 2004:328(7438):509-11.
6. Mulry JT. Nicotine gum dependency: a positive addiction. Drug Intell Clin Pharm 1988;22(4):313-4.
7. Mendelsohn CP. Three Decades of High-Dose Nicotine Gum Dependence Treated With Nicotine Patches. Nicotine Tob Res 2016;18(5):1220-1.

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Hydatid Cyst Mimicking the Pancoast Tumor

Pankoast Tümörünü Taklit Eden Hidatik Kist

Hydatid Cyst Mimicking the Pancoast Tumor

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Öz

Elli sekiz yaşında kadın hasta, sol kol ve omuz ağrısı ile kliniğimize başvurdu. Yapılan tetkikleri sonucunda sol akciğer üst lob apikal segmentte vertebra destrüksiyonuna yol açan kitle tespit edildi. Pancoast tümörü olarak opere edilen ancak intraoperatif hidatik kist olduğu saptanan hasta, nadir gözükmesi ve endemik bölgelerde kist hidatik tanısının ön tanılar arasında akılda tutulması gerekliliğini vurgulamak amacıyla sunuldu.

Anahtar Kelimeler

Akciğer; Hidatik Kist; Pancoast

Abstract

A fifty-eight-year-old female patient was referred to our clinic with left-sided arm and shoulder pain. Upon radiological investigation, a mass in the apical segment of the left upper lobe causing vertebra destruction was detected. The patient was operated on with a working diagnosis of a Pancoast tumor. However, during the operation the lesion was identified as a hydatid cyst. This case report aims to emphasize that, despite its rarity, hydatidosis should be included among the differential diagnosis of Pancoast tumors in endemic regions.

Keywords

Lung; Hydatid Cyst; Pancoast

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Introduction

Hydatidosis is a zoonotic parasitic disease caused by Echinococcus granulosus and characterized by cystic lesions. Hydatid disease is seen worldwide and is especially prevalent in developing countries and in countries with widespread animal husbandry [1].

In adults, the liver (65-80%) and lungs (10-25%) are most often involved, whereas other organs are less often afflicted [2]. Cases with hydatid cysts located in the chest wall are rarely encountered. The focus in this area can be soft tissue, sternum, or ribs. Involvement of vertebrae is even more infrequent [3].

Although hydatidosis is generally considered easy to diagnose and treat, cases with atypically located cysts can prove difficult. Such cases are often diagnosed during surgery [2,4,5]. We present our case for two reasons: Because vertebral destruction is extremely rare and because hydatid cysts should always be kept in mind to prevent accidental spilling, since the case was diagnosed during surgery.

Case Report

A female patient age 58 was referred to the Thoracic Surgery outpatient clinic for arm and shoulder pain that had been gradually increasing for a year. Chest x-ray and computed tomography of the chest revealed a cystic mass lesion in the apical segment of the upper left lobe, with destruction of the second thoracic vertebra (Image 1). Since it was a nonhomogeneous lesion, differential diagnosis included malignancy, so a positron emission tomography (PET) scan was done and a maximum standardized uptake value of 5 was reported. The primary diagnosis was Pancoast tumor. However, findings on the PET scan did not warrant any invasive staging. There was no invasion of the first rib, nor was there any invasion of the brachial plexus or nearby vascular structures. Therefore, magnetic resonance imaging of the chest was not requested. Diagnostic bronchoscopy was performed. Endobronchial lesions were not observed. Bronchoalveolar lavage was performed. In the pathology report, atypical cells were not reported. Because the primary diagnosis was a Pancoast tumor, a left thoracotomy was performed after preoperative evaluation and preparation. During the operation, exploration showed a lesion of cystic nature. The cyst contents were aspirated, yielding a clear fluid. It was thought to be a hydatid cyst. Before cystotomy, hypertonic saline soaked sponges were placed around the cyst to protect the surgical field outside of the cyst from accidental spillage of cyst contents. With necessary precautions taken, the cystotomy was done, and we saw that there were numerous daughter vesicles, some of which had caused destruction of the second vertebral body (Image 2). Because destruction of the vertebral body was not to the extent of destabilization, there was no need for further intervention. After obtaining hemostasis, the operation ended uneventfully. There were no postoperative complications, and the patient was discharged on day six. Pathological investigation of surgical specimens confirmed Echinococcus granulosus infestation.

Discussion

Since the first renal hydatid cyst reported in 1702, hydatid disease has been known to involve various organs in the body [6]. Symptoms and signs vary according to the localization and size of cysts. Small and peripherally located cysts tend to be asymptomatic. Hydatid disease in the thoracic region is usually intraparenchymal. Extrapulmonary intrathoracic cysts may cause dysphagia or dyspnea due to mass effect on esophagus or trachea [2,4]. It may be difficult to diagnose atypically located complicated hydatid cysts. In these cases, the most frequent complaints are dyspnea and cough. Our patient was diagnosed as Pancoast tumor because of arm and shoulder pain.

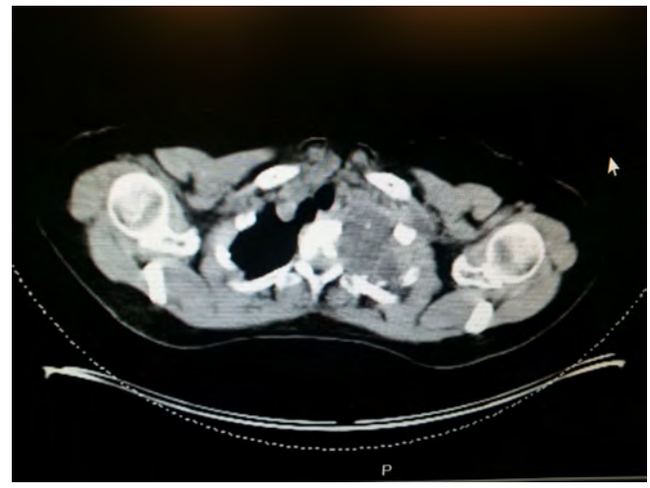

Image 1. Computed tomography of the chest, showing mass lesion in the apical segment of left upper lobe, with destruction of second vertebra.

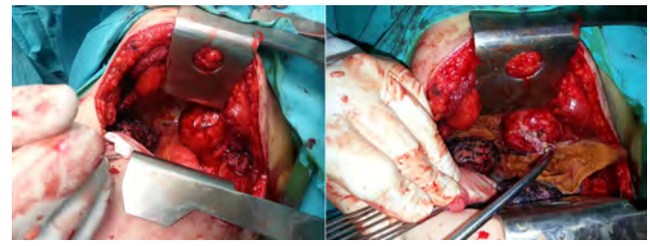

Image 2. Intraoperative view of the mass lesion causing vertebra destruction, and removal of the germinal membrane.

Review of the literature shows very few cases of hydatid disease mimicking Pancoast tumor and causing bone destruction. These patients' symptoms were similar to Pancoast tumor. Outcomes after surgical treatment are extremely favorable, as in our case.

While bone involvement in hydatidosis is reported to be between 0.9-2.0% intrathoracic extrapulmonary bone involvement is very rare [7]. In the presented case, there was invasion of the second vertebral body only, with no invasion of the first rib.

The most important diagnostic imaging techniques are radiography, computed tomography, and magnetic resonance imaging. Further imaging studies such as positron emission tomography should be performed if malignancy cannot be ruled out.

There are very few cases similar to ours that mimic Pancoast tumor [2, 8]. The case we have presented was thought to be a Pancoast tumor and operated on as such. During the operation, the mass was identified as extrapulmonary and of cystic nature. Therefore it was thought to be a hydatid cyst and a cystotomy was done.

In conclusion, it should be remembered that, although lung hydatidosis is a benign pathology, it can imitate lung malignancies both clinically and radiologically. It should be included among the differential diagnosis of malignancy, especially in endemic regions.

Competing interests

The authors declare that they have no competing interests.

References

1. İncekara F,Gülhan E, Üstün LN,Yazıcı Ü,Aydoğdu K, Fındık G ve ark. Torakal yaklaşım ile akciğer, karaciğer ve dalak kist hidatiklerinin cerrahi tedavisi. Turk Gogus Kalp Dama 2014;22(1):104-11.
2. Ceylan KC, Akpınar D, Polat H, Samancılar Ö, Usluer O, Alper H ve ark. Pulmoner arterin kist hidatiği. Turk Gogus Kalp Dama 2014;22(1):202-4.
3. Altuntaş B, Ceran S, Şener E. Unusual location of hydatid cyst: thoracic outlet. Turk Gogus Kalp Dama 2015;23(1):171-3.
4. Ulku R, Eren N, Cakir O, Balcı A, Onat S. Extrapulmonary intrathoracic hydatid cysts. Can J Surg 2004;47:95-8.
5. Pekmezci S, Kılıc İE. The diagnosis of the hydatid disease: imaging methods, serological and cytopathological examinations. Turkiye Klinikleri J Surg Med Sci 2006;2:13-6.
6. Durakbasa CU, Tireli GA, Sehiralti V, Sander S, Tosyali AN, Mutus M. An audit on pediatric hydatid disease of uncommon localization: incidence, diagnosis, surgical approach, and outcome. J Pediatr Surg 2006;41:1457-63.
7. Oguzkaya F, Akcali Y, Kahraman C, Emirogulları N, Bilgin M, Sahin A. Unusually located hydatid cysts: intrathoracacic but extrapulmonary. Ann Thorac Surg 1997;64:334-7.
8. Ozpolat B, Ozeren M, Soyal T, Yücel E. Unusually located intrathoracic extrapulmonary mediastinal hydatid cyst manifesting as Pancoast syndrome. J Thorac Cardiovasc Surg 2005;129:688-9.

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Acute Uterine Inversion Leading to Postpartum Bleeding: Case Report

Postpartum Kanamaya Neden Olan Akut Uterin İnversiyon: Olgu Sunumu

Acute Uterine Inversion

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Öz

Akut uterin inversiyon doğumun üçüncü evresinde nadir olarak görülebilen bir obstetrik acildir. Uterin inversiyon sıklığı 1200 -57000 'de bir doğum arasında değişmektedir. İnversiyon durumunda kan kaybı ve şok gelişebilmesi nedeni ile maternal mortalite gelişebileceğinden erken tanı ve erken müdahale önemlidir. Tanı klinik olarak koyulurken, uterin inversiyon geliştiğinde henüz en iyi yaklaşım netlik kazanmamış olsa da cerrahi olmayan manuel manevralar ile çeşitli cerrahi yaklaşımlar uygulanmaktadır. Biz bu vakada multipar bir hastada normal doğum sonrası 2. derece uterin inversiyon olup kanama ve atoni gelişmesi üzerine laparatomi ile uterusun normal hale getirilmesi olgusunu sunmayı amaçladık. 26 yaşında multipar (G3P2) hasta, 39. haftasında düzenli sancı şikayeti ile acil servise başvurmuş ve yapılan vajinal muayenesinde poşu mevcut ,4-5 cm %80 açıklık saptanması üzerine doğumhaneye kabul edilmiştir. Düzenli ağrıları olması sebebi ile spontan takip edilmiştir. Doğum 5 saat içinde gerçekleşmiş ve 3060 gr sağlıklı kız bebek dünyaya gelmiştir. Plasenta çıkmadan bir kez fundusa Crede manevrası uygulanmış ve 10 dakika içerisinde plasenta kendiliğinden ayrılmıştır. Plasenta ayrılmasını takiben aşırı uterin kanama izlenmiş , vajinal ekartör ile kanama kontrolü yapılırken miyoma uteri zannedilen , fakat uterin masajda fundusun hissedilmemesi üzerine uterin inversiyondan şüphenilerek manuel olarak inversiyon düzeltilmeye çalışılmış fakat başarılı olamayınca ve kanamanın fazla olması nedeni ile hasta ameliyathaneye alınmış ve laparatomi yapılarak redüksiyon sağlanmıştır. Histerektomi' ye gerek kalmamıştır. Hastanın genel durumunun iyi olması üzerine 4 gün sonra hasta şifa ile taburcu edilmiştir. Uterin inversiyon uterin fundusun ters dönerek serviksten dışarı çıkması durumudur. İnversiyonu hazırlayıcı faktörler kesin olarak bilinmediğinden öngörmek mümkün olmamaktadır. Tanı klinik olarak koyulurken, uterin inversiyon geliştiğinde henüz en iyi yaklaşım net olmasa da cerrahi olmayan manuel manevralar , medikal tedavi ve çeşitli cerrahi yaklaşımlar uygulanmaktadır. Özetle, postpartum kanama nedeni ve bir obstetrik acil olan uterin inversiyon nadir olarak görülse de ,oluştuğunda erken tanı konması ve kötü prognoz oluşmasını önlemede erken müdahale ve tedavi yaklaşımlarını bilmek çok önemlidir.

Anahtar Kelimeler

Obstetrik Acil; Uterin Kanama; Uterin İnversiyon

Abstract

Acute uterine inversion is an obstetric emergency that is a rare occurrence in the third stage of labour. The incidence of uterine inversion ranges from 1/1200 to 1/57000 deliveries. Early recognition and appropriate intervention are important as it may cause maternal mortality due to loss of blood and shock. While making a clinical diagnosis, various surgical approaches along with nonsurgical manual maneuvers are performed even if the best approach is not determined at the time of uterine inversion. We aimed to report the case of there positioning of the uterus of a multiparous patient by laparotomy following postpartum hemorrhage and atony caused by second degree uterine inversion following a vaginal delivery. A 26-year-old multiparous (G3P2) patient came to the emergency room with regular contractions in her 39 th week. She was hospitalized following examination performed as membrane intact and 4-5 cm 80% dilation were determined. She was followed up regularly due to her regular pains. She gave birth to a healthy baby girl weighing 3060 grams in five hours. The Crede maneuver had been executed once before the placenta came out, and placental separation took place within ten minutes. Following the placental separation, excessive uterine bleeding was observed. When performing bleeding control with a vaginal extractor, the patient was suspected of having myoma uteri. However, since the fundus could not be felt during the uterine massage and uterine inversion was thought to exist, manual repositioning of the uterus was performed without any success. The patient was taken to the operating room due to excessive bleeding that was reduced following laparotomy. Hysterectomy was not needed. The patient was discharged after four days with good general health. Uterine inversion is the state in which the uterine fundus reverses and extends beyond the cervix. It is not possible to foresee inversion as the factors that trigger inversion are not fully known.When uterine inversion occurs, while making a clinical diagnosis, medical treatment and various surgical approaches along with nonsurgical manual maneuvers are performed even when the best approach is not determined at the time of uterine inversion.To sum up, though uterine inversion is a rare obstetric emergency that causes postpartum bleeding, early recognition and appropriate intervention are essential to prevent poor prognosis.

Keywords

Obstetric Emergency Uterine Bleeding; Uterine Inversion

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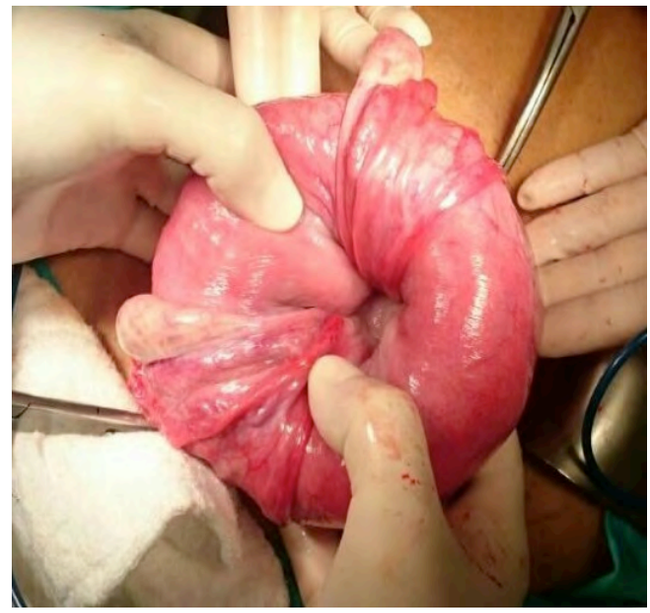
Introduction

Acute uterine inversion is an obstetric emergency that is a rare occurrence in the third stage of labour. Uterine inversion is described as the state in which the uterine fundus reverses and extends beyond the cervix. The incidence of uterine inversion ranges from1/1200 to 1/57000 deliveries [1-3]. Early recognition and appropriate intervention are important as it may cause maternal mortality due to loss of blood and shock [4]. Symptoms are similar to those of tumoral mass or myoma uteri incases of first and second degree inversion [5]. When uterine inversion occurs, uterine reduction can be achieved with the help of various surgical approaches along with nonsurgical manual maneuvers even if the best approach is not known at the time of uterine inversion.We aimed to present the case of repositioning the uterus by laparatomy due to the development of a second degree uterine inversion, bleeding and atony in a multiparous patient following vaginal delivery, including the approach to the atony that developed and the description, etiology, risk factors, diagnosis and treatment of inversion.



Picture 1.

Case Report

A 29-year-oldmultiparous (G3P2) patient came to the emergency room with regular contractions in her 39 th week and was hospitalized following her examination as membrane intact and 4-5 cm 80% dilation were determined. She had regular check-ups during pregnancy and did not have any illnesses. The stages of labour were followed as her fetal Non-Stress Test (NST) was reactive and she had regular contractions. Spontaneous rupture of the membrane occurred when the cervix was dilated to 7 cm. The delivery took five hours and she delivered a healthy baby girl weighing 3060 grams. Before the placenta came out, the Crede maneuver was executed once and placental separation took place within ten minutes. Following placental separation, excessive uterine bleeding was observed. When performing bleeding control with a vaginal extractor, no cervical os was monitored, yet there were masses resembling myoma uteri. However, since the fundus could not be felt during the uterine massage and uterine inversion was thought to exist, manual repositioning of the uterus was performed without any success. The patient was taken to the operating room due to excessive bleeding. The manual inversion was attempted again under general anesthesia, but it was an unsuccessful attempt. Laparotomy had to be performed. When the abdomen was opened with a Pfannenstiel incision, uterine inversion was determined (Picture 1). Haultain technique was performed due to the contraction ring, and the inversion was corrected. As atony and bleeding continued, the patient underwent an HB control (input Hb:11.2 hgb) with 6 hGb and was given a supportive treatment with 4 units of erythrocytes and 2 units of fresh frozen plasma replacement and uterotonic agents. As the bleeding continued after B- Lynch suture was applied to the uterus, bilateral hypogastric artery ligation was performed. Because the bleeding stopped without the need for a hysterectomy and atony was corrected, the abdomen was closed by placing a drain. The patient was discharged after four days with good general health.

Discussion

Uterine inversion is the state in which the uterine fundus reverses and extends beyond the cervix. Though it may occur during normal or cesarean delivery, approximately 5% of the inversions occur spontaneously and independently of puerperium [6]. Uterine inversion is classified according to the degree of inversion: 1st degree (the fundus is inside the endometrial cavity), 2nd degree (the fundus protudes through the cervical os), 3rd degree (the fundus protudes to or beyond the introitus), 4th degree (both the uterus and vagina are inverted). Based upon the timing of inversion it is classified as acute (occurs within 24 hours of birth), subacute (occurring after 24 hours but within 4 weeks of birth) and chronic (occurs after 4 weeks) [7].

The incidence of uterine inversion ranges from 1/1200 to1/57000 deliveries [1-3]. Its pathogenesis is unclear, but the most likely causes are strong traction of the umbilical cord, fetal macrosomia, short umbilical cord, congenital anomalies of the uterus, excessive fundal massage (Crede maneuver),and fundal placenta [8,9].

Diagnosis of uterine inversion is based on clinical signs and symptoms. Uterine inversion is suspected if the uterine fundus is observed in the vaginal introitus or when the fundus is palpated and there is excessive postpartum blood loss, or in the absence of uterine fundus during abdominal palpation. If the physical examination is not definitive and the patient is hemodynamically stable, radiographic imaging (ultrasonography and magnetic resonance) can be used in diagnosis [10].

If intervention does not occur soon after diagnosis , hypovolemic shock may develop. After uterine inversion is diagnosed, to minimize blood loss and to avoid contraction rings uterine reduction should be attempted with rapid pressure on the fundus from the vagina. This is known as the Johnson maneuver. If the Johnson maneuver fails, the patient should be taken under the care of the surgical and anesthesia team. Another experienced gynecologist and operating room staff should be called for help. Complete blood count and coagulation parameters should be studied while intravenous fluid is administered to the patient. Preparation of the blood products should be quickly performed. If uterine reduction fails even after uterine relaxants (nitroglycerine, terbutaline, magnesium sulphate and inhaler anesthetic

agents such as sevoflurane, desflurane, andisoflurane) are given, surgical approach is performed [11,12]. Methods such as laparoscopic assisted reduction, vaginal surgical approach, reduction using an obstetric vacuum, or hydrostatic reduction can be used, but asurgical approach with Huntington and Haultain techniques is applied most frequently [13,14,15]. The less invasive Huntington technique is recommended [7]. This method involves pulling bilateral ligaments by holding them with Allis clamps. Withdrawal is repeated when the other surgeon, if there is one, pushes through the vaginal route. Since this method generally fails when there is a contraction ring,the other surgical method, Haultain technique is used. In this method, after an anterior or posterior incision is made in the contraction ring, reduction is achieved manually. First the incision is sutured. The same method performed vaginally has beendescribed by Spinelli but no significant success has been observed in acute inversion cases. Hysterectomy is performed if reduction cannot be achieved by any of these methods [14].

Though controversy exists about the time when the placenta should be removed if it fails to separate, it is commonly suggested that removal of the placenta after correction will result in decreased blood loss [16].

A single dose 1st generation cephalosporin antibiotic prophylaxis is recommended to prevent the development of endometritis after reduction has been achieved. From uterotonic agents oxytoxin (20-40 units), misoprostol 800 micrograms intravaginally, dinoprostone 20 mg rectally, or methylergonovine 200 micrograms should be administered intramuscularly in a maximum of 4 doses to prevent atony development [17].

In conclusion, although uterine inversion is a rare obstetric emergency that causes postpartum bleeding, early recognition and appropriate intervention are essential to prevent poor prognosis.

Competing interests

The authors declare that they have no competing interests.

References

1. Morini A, Angelini R, Giardini G. Acute puerperal uterine inversion: a report of 3 cases and an analysis of 358 cases in the literature. Minerva Ginecol 1994; 46:115-27.
2. Baskett TF. Acute uterine inversion: a review of 40 cases. J Obstet Gynaecol Can 2002; 24:953-6.
3. Witteveen T, Van Stralen G, Zwart J, et al. Puerperal uterine inversion in the Netherlands: a nationwide cohort study. Acta Obstet Gynecol Scand 2013; 92:334-7.
4. Watson P, Bejch N, Bowes V.A. Jr. Management of acute and subacute puerperal inversion of the uterus. Obstet. Gynecol 1980;55:12-6.
5. Occhionero M, Restaino G., Ciuffreda M, et al. Uterine inversion in association with uterine sarcoma: a case report with MRI findings and review of the literature. Gynecol Obstet Invest 2012;73:260-4.
6. Lupovitch A, England ER, Chen R. Non-puerperal uterine inversion in association with uterine sarcoma: case report in a 26-year-old and review of the literature. Gynecol Oncol 2005; 97:938-41.
7. Repke J, Ramin S, Barss V. Puerperal uterine inversion. Up to Date.com Nov-2016.
8. Lipitz S, Frenkel Y. Puerperal inversion of the uterus. Eur J Obstet Gynecol Reprod Biol 1988; 27:271-4.
9. Deneux-Tharaux C, Sentilhes L, Maillard F, et al. Effect of routine controlled cord traction as part of the active management of the third stage of labour on postpartum haemorrhage: multicentre randomised controlled trial (TRACOR). BMJ 2013; 28:346-f1541.
10. Hsieh TT, Lee JD. Sonographic findings in acute puerperal uterine inversion. J Clin Ultrasound 1991; 19:306-9.
11. Smith GN, Brien JF. Use of nitroglycerin for uterine relaxation. Obstet Gynecol Surv 1998; 53:559-65.
12. Yoo KY, Lee JC, Yoon MH, et al. The effects of volatile anesthetics on spontaneous contractility of isolated human pregnant uterine muscle: a comparison among sevoflurane, desflurane, isoflurane, and halothane. Anesth Analg 2006; 103:443-45.
13. Shepherd LJ, Shenassa H, Singh SS. Laparoscopic management of uterine inversion. J Minim Invasive Gynecol 2010; 17:255-7.
14. Antonelli E, Irion O, Tolck P,et al. Subacute uterine inversion: description of a novel replacement technique using the obstetric ventouse. BJOG 2006; 113:846-7.
15. Tan KH, Luddin NS. Hydrostatic reduction of acute uterine inversion. Int J Gynaecol Obstet 2005; 91:63-4.
16. You W., Zahn C. Postpartum hemorrhage: abnormally adherent placenta,uterine inversion and puerperal hematomas. Clin Obstet Gynecol 2006;49:184-97.
17. Adesiyun A. Septic postpartum uterine inversion. Singapore Med J 2007;48:943-5.

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Case Report
JCAM

Laparoscopic Fenestration of Posttransplant Lymphoceles: A Report of Three Cases

Posttransplant Lenfoselin Laparoskopik Fenestrasyonu: Üç Olgu Sunumu

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Öz

Lenfosel semptomatik olgularda etkili bir şekilde tedavi edilmelidir. Perkütan aspirasyon, perkütan drenaj ve skleroterapi tedavi alternatifleridir. Konvansiyonel veya laparoskopik olarak intraperitoneal kaviteye internal drenaj cerrahi yönetimde gerçekleştirilebilir. Biz lenfoselin laparoskopik tedavisini üç olgu ile sunuyoruz.

Anahtar Kelimeler

Posttranplant Lenfosel; Fenestration; Laparoskopi; Minimal İnvaziv; Böbrek Nakli

Abstract

Lymphoceles have to be treated efficiently when symptoms are present. Percutaneous aspiration, percutaneous drainage, and sclerotherapy are therapeutic alternatives. Conventional or laparoscopic internal drainage surgical management can be performed to the intraperitoneal cavity. We present three cases of the laparoscopic treatment of lymphoceles.

Keywords

Posttransplant Lymphoceles; Fenestrasyon; Laparoscopy; Minimally İnvasive; Renal Transplantation

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Introduction

Following renal transplantation, there are usually no clinical signs of perirenal fluid accumulation; however, in some cases, low patient well-being or inadequate graft kidney function could be indicative of such fluid accumulation. Along with urinomas, lymphoceles are among the recurring causes of perirenal fluid accumulation [1, 2, 3]. A post-transplant lymphocele represents a perigraft fluid aggregation occurring after the renal bed or the graft has been prepared; it is not the reason for urinary leakage or haemorrhage. Micro or macro graft kidney decapsulation leads to non-ligation between afferent lymphatic vessels and iliac vessels [4, 5]. Furthermore, lymphoceles may be triggered by the administration of powerful diuretics following a kidney transplant, increasinglymphatic flow [6, 7]. When considering causes of allograft lymph flow intensification, another aspect that should be taken into account is graft rejection. The majority of lymphoceles are symptomless and do not need to be treated. In these cases, given the parameters of renal function and its clinical course, appropriate administration is frequently a comprehensive follow-up. When symptoms do arise, an effective approach must be applied to treat the lymphoceles. Among the available approaches are percutaneous aspiration, percutaneous drainage, and sclerotherapy. A surgical procedure on the intraperitoneal cavity usually involves traditional or laparoscopic inner drainage. We have presented three cases of laparoscopic treatment of lymphoceles.

Case Report

Case 1

The first case is of a 25-year-old male patient with complaints of pressure within the right inguinal region. A successful open renal transplantation had been performed eight months earlier. The patient's urinary outflow decreased and his creatinine levels increased. After diagnostic procedures were performed it was deduced that a 120x64x31 mm lymphocele was present from the anterior of the right kidney to the bladder.(Picture 1).

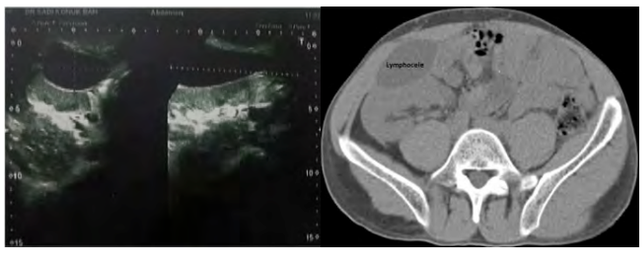

Figure 1. Preoperative ultrasound and computed tomography of lymphocele (Case 1).

Case 2

The second case is of a 31-year-old female upon whom a successful open renal transplantion had been performed five months earlier. After proper radiological diagnosis the presence of a 112x27x27mm lymphocele, inferior to the kidney and anterior to the bladder, was discovered (Picture 2).

Case 3

A 39-year-old female had undergone open renal transplantation one month earlier. In the patient's clinical and radiological examinations, a 220x140x42 cm lymphocele was detected.

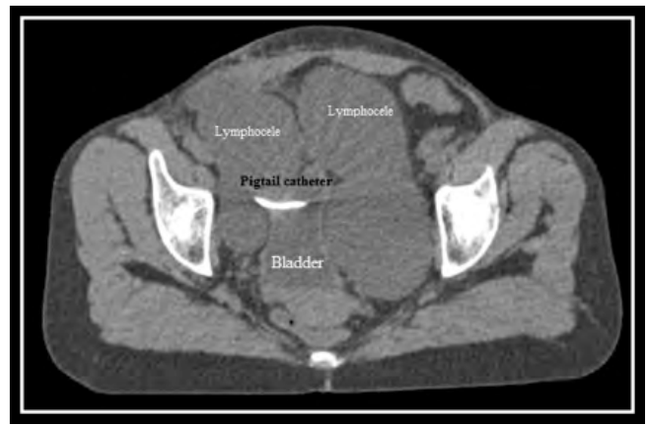

Figure 2. Lymphocele around the bladder and appearance of pigtail catheter (Case 2).

Surgical Treatment

Patients with a history of a previous failed percutaneous aspiration laparoscopic approach were managed with general anesthesia and the standard single dose of prophylactic antibiotics. The pneumoperitoneum was made by a Veress needle inserted in an infraumbilical location. The first trocar (10-mm) was inserted in the infraumbilical location followed by a second trocar (5-mm) along the anterior axillary line near the costal margin on the ipsilateral side and a third trocar (5-mm) was inserted in the midline, low in the hypogastrium. With the help of electrocautery and scissors, a peritoneal opening in the lymphocele wall was created, followed by removal of the lymphatic fluid. A pedicle of omentum in the lymphocele core was interfered with. The final step was a thorough examination of haemostasis at the lymphocele entry lines and the peritoneum (Picture 3).

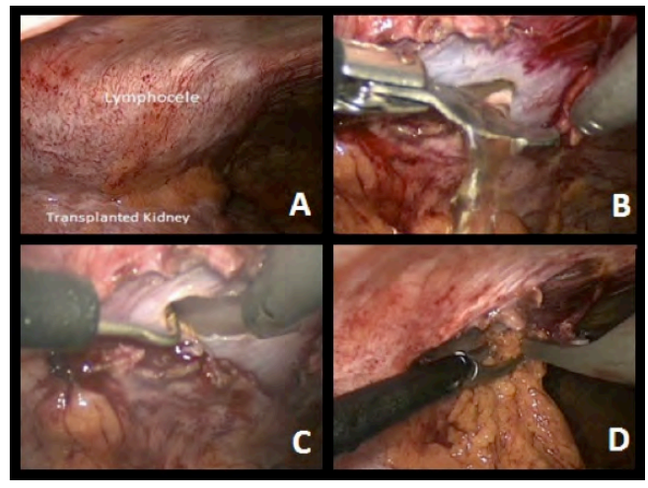

Figure 3. Surgical images. (A) Lymphocele and transplant kidney (B) Fenestration of lymphocele wall with scissors (C) Aspiration of the contents of lymphocele (D) Omental patch

Results

The entire procedures were each completed in three-quarters of an hour. No patient lost more than 100 ml of blood and no other problems were encountered. Likewise, no intraoperative complications occurred and patients recovered well after surgery. All patients' postoperative courses were uncomplicated. There were no disruptions to immunosuppression; oral administration commenced on the day of surgery. Post-operatively, the function of the graft remained constant and normalisation of serum

creatinine was seen.There was no graft rejection. The patients were discharged without any problems 2-5 days after their operations. Continuous follow-ups utilizing ultrasonography and computed tomography detected no recurrence of lymphoceles.

Discussion

Previous studies have reported different frequencies of posttransplant lymphoceles within the 0.6-20.0 % range [8, 9]. Symptoms that suggest lymphoceles are pain in the area of the pelvis or abdomen, caused by a pelvic mass that can be seen or palpated. Other side effects include, outcomes of venous or ureteral pressure, such as one-sided leg oedema and leg torment, hydronephrosis with resulting insufficiency of renal capacity, and profound vein thrombosis [10]. Fever and leucocytosis are indicative of infectious complications of lymphatic fluid accumulation. Ultrasound and occasionally computed tomography are used in the preoperative diagnosis of lymphoceles [11]. To prevent urinoma and superinfection it is advisable to undertake percutaneous aspiration or drainage and microbiological sample analysis prior to internal drainage. Patients without any symptoms are not usually given any treatment. Types of treatments for symptomatic lymphoceles include recurrent percutaneous aspiration of lymphatic fluid [4, 12], percutaneous drainage [13], sclerosing substance administration [12, 14], and surgical internal drainage into the peritoneal cavity.

Infected lymphoceles should never be drained into the abdominal cavity because of potentially mortal consequences for the immunosuppressed patient. Percutaneous aspiration or drainage, and sclerotherapy do have acceptable success rates of 80–90%, and are easy to perform. Nevertheless, after this treatment, incomplete resolution and high recurrence rates of up to 80% are reported. Furthermore, there is risk of haemorrhage and infection, principally after recurrent punctures or insertion of a drainage tube. An especially long duration of drainage therapy over several weeks is an exceptional disadvantage. Parra et al. [13] noted that a success rate of about 73% still came with a 26% risk of infection.

Scleropathy can create complications for sequential surgical procedures, and, as a result, the graft kidney or nearby structures may be damaged [2–4, 11, 12, 15]. High repetition rates, infection rate, and treatment duration are all factors that should be taken into consideration. The best treatment in the case of recurring lymphatic fluid drainage into the peritoneal cavity is open surgical internal drainage. The operation was first outlined in 1966 by Byron et al. [16], but the operation has limitations in that it must be performed under general anaesthetic, causes surgical trauma, and patients need a long time to recover. In 1991, McCullough et al.[17] pioneered the laparoscopic surgical approach for treatment of lymphoceles after the introduction of laparoscopic surgery. Four years later, in 1995, the significance of this type of surgery was advocated by Gill et al. [18]. Its advantages include reduced haemorrhage, a reduced need for analgesics, earlier food ingestion post-operatively , and a shorter duration of recovery. In 1997, Fornara et al. [19] reported that when eight cases were compared, open surgery, lasted 115 minutes on average, while laparoscopic surgery was completed in an average of 42 minutes. Based on extensive experience, successful outcome rates and the short operating time of laparoscopic surgery are critical advantages in favor of the laparoscopic procedure over open surgery. In 2009, Iwan-Zietek et al. [20] ,embraced an outline which distinctly showed data that advocated for the preference of surgical, especially laparoscopic, management of posttransplant lymphoceles. The authors reported that percutaneous drainage should be attempted first to cure a lymphocele, but reported that; laparoscopic fenestration is a safe and practicable technique that should be used after unsuccessful and inefficient percutaneous drainage [20]. The three cases in this report show the definite benefit of surgical treatment because there were no recurrences. Based on the findings of earlier studies, laparoscopic surgery was decided to be the best approach for treating lymphoceles.

Conclusion

Based on studies in the literature and the cases in our report, laparoscopic fenestration is a method with short operating times, low infection rates, less blood loss, and decreased postoperative pain.It has especially been found to be a successful, uncomplicated, established, and effective solution to complications of percutaneous drainage and needle aspirated lymphoceles.

Competing interests

The authors declare that they have no competing interests.

References

1. Braun WE, Banowsky LH, Straffon RA, Nakamoto S, Kiser WS, Popowniak KL et al. Lymphocytes associated with renal transplantation. Report of 15 cases and review of the literature. Am J Med 1974; 57: 714–29.
2. Burgos FJ, Teruel JL, Mayayo T, Lovaco F, Berenguer A, Orte L et al. Diagnosis and management of lymphoceles after renal transplantation. Br Journal of Urol 1988; 61: 289-93.
3. Howard RJ, Simmons RL, Najarian JS. Prevention of lmymphoceles following renal transplantation. Ann Surg 1976; 184: 166-8.
4. Schweizer RT, Cho SI, Kountz SL, Belzer FO. Lymphoceles following renal transplantation. Arch Surg 1972; 104: 42-5.
5. Koene RA, Skotnicki SH, Debruyne FM. Spontaneous renal decapsulation with excessive fluid leakage after transplantation. N Engl J Med 1979; 300: 1030-1.
6. Szwed JJ, Maxwell D, Kleit SA, Hamburger RJ. Angiotensin 1 diuretics and thoracic duct lymph flow in the dog. Am J Physiol 1973; 224: 705-8.
7. Pedersen DC, Morris B. The role of the lymphatic system in the rejection of homografts: a study of lymph from renal transplants. J Exp Med 1970; 131: 936-69.
8. Hamza A, Fischer K, Koch E, Wicht A, Zacharias M, Loertzer H et al. Diagnostics and therapy of lymphoceles after kidney transplantation. Transplant Proc 2006; 38: 701-6.
9. Zietek Z, Sulikowski T, Tejchman K, Sieńko J, Janeczek M, Iwan-Zietek I, et al. Lymphocele after kidney transplantation. Transplant Proc 2007; 39: 2744-7.
10. Ulrich F, Niedzwiecki S, Fikatas P, Nebrig M, Schmidt SC, Kohler S et al. Symptomatic lymphoceles after kidney transplantation multivariate analysis of risk factors and outcome after laparoscopic fenestration. Clin Transplant 2010; 24: 273-80.
11. Greenberg BM, Perloff LJ, Grossman RA, Naji A, Barker CF. Treatment of lymphoceles in renal allograft recipients. Arch Surg 1985; 120: 501-4.
12. Gilliland JD, Spies JB, Brown SB, Yrizarry JM, Greenwood LH. Lymphoceles: percutaneous treatment with povidone – iodine sclerosis. Radiology 1989; 171: 227-9.
13. Parra RO, Jones JP, Hagood PG. Laparoscopic intraperitoneal marsupialization: report on a new treatment for lymphoceles. Surg Laparosc Endosc 1992; 2: 306-11.
14. Pope AJ, Ormiston MC, Bogod DG. Sclerotherapy in the treatment of a recurrent lymhocele. Postgrad Med 1982; 58: 573-4.
15. Braun WE, Banowsky LH, Straffon RA, Nakamoto S, Kiser WS, Popowniak KL et al. Lymphoceles associated with renal transplantation. report of 15 cases and review of the literatures. Am J Med 1974; 57: 714-29.
16. Byron RL Jr, Yonemoto RH, Davajan V, Townsend D, Bashore R, Morton DG. Lymphocysts: surgical correction and prevention. Am J Obstet Gynecol 1966; 94: 203-7.
17. McCullough CS, Soper NJ, Clayman RV, So SS, Jendrisak MD, Hanto DW. Laparoscopic drainage of a post-transplant lymphocele. Transplantation 1991; 51: 725-7.
18. Gill IS, Hodge EE, Munch LC, Goldfarb DA, Novick AC, Lucas BA. Transperi-

toneal marsupialization of lymphoceles: a comparison of laparoscopicand open techniques. J Urol 1995; 153: 706-11.
19. Fornara P, Doehn C, Fricke L, Hoyer J, Jocham D. Laparoscopy in renal transplant patients. Urology 1997; 49: 521-7.
20- Iwan-Zietek I, Zietek Z, Sulikowski T, Nowacki M, Zair L, Romanowski M et al. Minimally invasive methods for the treatment of lymphocele after kidney transplantation. Transplant Proc 2009; 41: 3073–3076.

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Anomalous Origin And Inter-Arterial Course of the Right Coronary Artery: A Case Series

Anormal Orjinli ve İnterarteryal Seyir Gösteren Sağ Koroner Arter: Olgu Serisi

Anomalous Right Coronary Artery Series

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Öz

Radyoloji kliniğimizde, Kasım 2014 ve mayıs 2016 tarihleri arasında göğüs ağrısını, koroner revaskülarizasyon prosedüründen sonra koroner arteri ve olası koroner arter anomalilerini değerlendirmek için amacıyla refere edilen 1200 hastaya ÇKBT yapıldı. BT anjiyografi yapılan hastalar arasından koroner anomalisi olan hastalar değerlendirildi. Toplam x sayıda hastada koroner anomali olduğu gözlendi. Bu hastaların altısında (erkek:3, kadın:3) anormal orjinli RCA saptandı. Olguların özgeçmişinde anormal çıkışlı RCA kaynaklı MI veya iskemi nedenli hastaneye başvuru yoktu. Tüm hastaların ekokardiyografileri doğaldı. ÇKBT' de sol koroner arter orjinine yakın fakat ayrı olarak, sol sinüs valsalvadan orjin alan sağ koroner arter izlendi (resim 1). Tüm hastalarda RCA' nın proksimal kesimi, aorta ve pulmoner arter arasında interarteryal seyir göstermekteydi (resim 2). ÇKBT' de anormal orjinli RCA' da perkütan koroner girişim gerektirecek anlamlı stenoz saptanmadı. Olguların tamamı medikal olarak tedavi edildi ve yaklaşık yirmi dört aylık izlem sonrasında, kötü kardiyak bir olay görülmedi.

Anahtar Kelimeler

Koroner Arter Anomalisi; İnterarteryal Seyir; Koroner BT; Taşikardi

Abstract

In our radiology clinic, between November 2014 and May 2016, we obtained MSCT from a total of 1,200 patients who had presented with chest pain, and who had undergone coronary revascularization procedures, in order to evaluate coronary arteries and possible coronary artery anomalies. Among the patients who had computed tomography (CT) angiography, the patients with coronary anomalies were evaluated. In total, 10 patients with coronary anomalies were included. An anomalous origin of the RCA was detected in six of these patients (3 males and 3 females). None of the patients was admitted to hospital for myocardial infarction (MI) or ischemia caused by an anomalous origin of the RCA. The ECG findings of all patients were normal. Close to the origin of the left coronary artery, but separately, a right coronary artery originating from the left sinus of Valsalva was observed in the MSCT (Figure 1). The proximal part of the RCA had an interarterial course between the aorta and pulmonary artery in all patients (Figure 2). No significant stenosis in an anomalous origin of the RCA requiring percutaneous coronary intervention was detected in the MSCT. All patients were treated medically and no unfavorable cardiac event was observed during approximately two years of follow-up.

Keywords

Coronary Artery Anomaly; Interarterial Course; Coronary CT; Tachycardia

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Introduction

A right coronary artery (RCA) originating from the left sinus of Valsalva is a rare congenital anomaly that accounts for 0.1% of patients undergoing angiography. There is an abnormal inter-arterial course in 99% of the patients with an anomalous origin of the RCA. An anomalous origin of the RCA can be benign and asymptomatic, but it can also be highly risky due to its anatomical characteristics [1]. Its clinical manifestations are quite variable and can be associated with sudden cardiac death, myocardial ischemia, and syncope [2]. The development of electrocardiography (ECG) gated multi-slice computed tomography (MSCT) enables detection of coronary anomalies definitively and in a non-invasive manner [3]. Herein, we present six RCA cases with left sinus of Valsalva origin and inter-arterial course in the light of the literature.

Case Report 1

A 72-year-old female patient presented with complaints of atypical chest pain and hypertension. Her medical history revealed hypertension and stent placement in the proximal part of the left circumflex (LCx) artery 20 years previously. Hypercholesterolemia was detected in the laboratory examination. The ECG findings were normal. Echocardiography (ECHO) showed segmental wall disturbance in the left ventricle and dilatations of the left atrium and left ventricle. In the MSCT, the right coronary artery originated from the left sinus of Valsalva and its proximal 2 cm segment with a thin calibration had a course between the pulmonary artery and the aorta. In the middle part of the left anterior descending (LAD) artery, there were mixed plaques causing moderate stenosis in the lumen. The patient was prescribed hypertensive and anti-hyperlipidemic drugs. She was followed at six-month intervals for 24 months, and no ischemic symptom was observed.

Case Report 2

A 26-year-old male patient was admitted to our cardiology outpatient clinic with a complaint of palpitation. On admission his heart rate was high and irregular, ranging between 90 and 120 bpm. There was no history of smoking, alcohol use, or comorbidity in his medical history. His laboratory test results were normal. Tachycardia was seen on the ECG and ectopic beats were noted. In the MSCT, the right coronary artery with a normal calibration originated with an acute angle from the left sinus of Valsalva and had a course between the pulmonary artery and aorta. No atherosclerotic plaques causing stenosis in the vessels were detected. Medical treatment was initiated, and he is still under follow-up.

Case Report 3

A 50-year-old female patient presented with complaints of dyspnea and chest pain. Her medical history revealed diabetes. Her laboratory analysis showed that the blood glucose was regulated and she had dyslipidemia. There was sinus rhythm on the ECG. The ECHO findings were normal. In the MSCT, the right coronary artery originated from the left sinus of Valsalva and its proximal 3 cm segment with a thin calibration had a course between the pulmonary artery and aorta. There were soft plaques that did not cause significant stenosis in the lumen of the LAD and LCx arteries. Medical treatment was initiated and she is still under follow-up.

Case Report 4

A 75-year-old female patient presented with complaints of dyspnea, fatigue, and atypical chest pain. Her medical history revealed hypertension and her coronary angiography showed non-critical stenosis. There was sinus rhythm on the ECG. The ECHO showed left ventricle hypertrophy and degenerative aorta stenosis. In the MSCT, the right coronary artery originated from the left sinus of Valsalva and its proximal 1.5 cm segment with a thin calibration had a course between the pulmonary artery and aorta. Also, there were multi-segmental mixed plaques causing mild-to-moderate stenoses along the lumen of the RCA. Medical treatment was initiated and she was followed at six-month

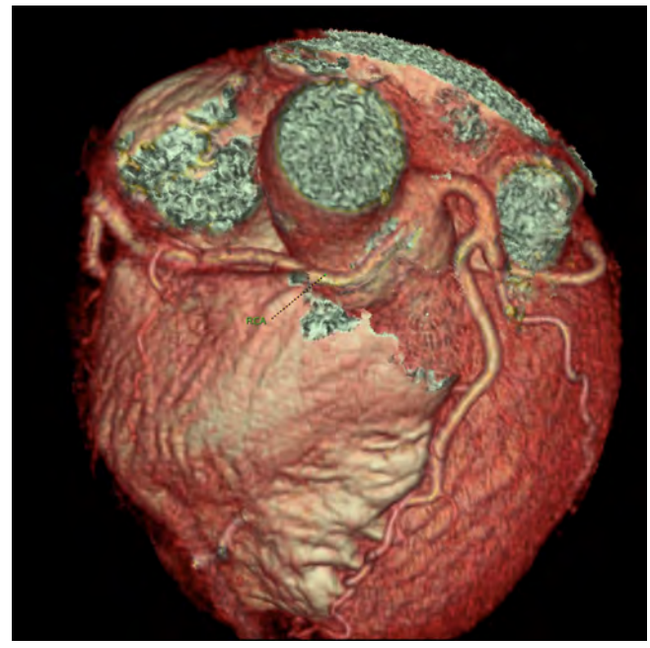

Figure 1. The right coronary artery origination from the left sinus of Valsalva was observed.

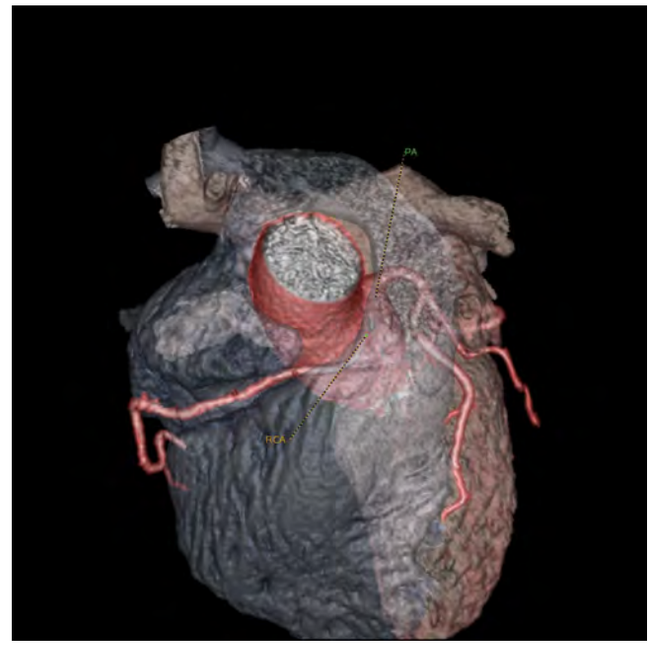

Figure 2. The proximal part of the RCA has an inter-arterial course between the aorta and pulmonary artery.

intervals. During 16-months of follow-up, no ischemic symptom was observed.

Case Report 5

A 65-year-old male patient presented with complaints of palpitation and dyspnea while climbing stairs. There were coronary heart disease and dyslipidemia in his medical history. The ECG and ECHO findings were normal. In the MSCT, the right coronary artery originated from the left sinus of Valsalva and its proximal 2 cm segment with a thin calibration had a course between the pulmonary artery and aorta. A stent was seen extending from the proximal origin of the RCA to 5 cm distally with a patent lumen. Also, there were stents in the distal parts of the LCx and LAD arteries with patent lumens. Medical treatment was initiated and he was followed at six-month intervals. During the 21-month follow-up, no ischemic symptom was observed.

Case Report 6

A 51-year-old male patient presented with complaint of palpitation and dyspnea during long distance walking. His medical history revealed diabetes. There was sinus rhythm on the ECG. The ECHO showed Grade 1 diastolic dysfunction. In the MSCT, an aberrant right coronary artery originated from the left sinus of Valsalva with an acute angle and its proximal 2.5 cm segment with a thin calibration had a course between the pulmonary artery and aorta. No atherosclerotic plaques causing significant stenosis within the lumens of coronary arteries were detected. Medical treatment was initiated and he was followed at six-month intervals. During 21 months of -month follow-up, no ischemic symptom was observed.

Discussion

Abnormal coronary arteries are rare and their clinical results may range from asymptomatic to sudden cardiac death [1]. The incidence of abnormal right coronary arteries in coronary angiography studies ranges from 0.06 to 0.5% [4]. In addition to this, the incidence of an anomalous origin of the RCA has been reported to be 0.92% in the overall population and is higher than the incidence of left main coronary artery origin [5].

The coronary artery with an anomalous origin may have an inter-arterial, retro-aortic, pre-pulmonic, or septal (sub-pulmonic) course. However, the most frequent course is inter-arterial [3]. The incidence of sudden death in patients with an inter-arterial course is 25-40%; half of the reported cases are associated with exercise [3]. In the literature, various anomalous origins of the RCA have been reported including the descending thoracic aorta, left main artery (LMA), LCx, from left sinus of Valsalva or above, from the pulmonary artery, or below the aortic valve [4]. Following its abnormal origin, the RCA lies in the anterior, posterior, or at the base of the heart between the great arteries. The course at the base of the heart between the great arteries is known as malignant type anomaly due to the risk of extrinsic compression and sudden death [4].

If coronary anomaly causes a complaint, the symptoms commonly occur before the age of 30 and its occurrence is strongly correlated with exercise [6]. The clinical manifestations can vary from angina, acute myocardial infarction, syncope, and heart failure to sudden death. Inter-arterial course of aberrant coronary arteries is a risk factor for sudden death [2]. In the anomalies without an inter-arterial course, sudden cardiac death results from the narrowed orifice or emergence of aberrant coronary artery with an acute angle [7].

The cardiac symptoms may be caused by pinching between the aorta and pulmonary artery or increased cardiac output during exercise [6]. A dilated aortic root or the compression of coronary artery lumen between the aorta and pulmonary artery restricts blood flow. Sudden arrhythmias such as paroxysmal supraventricular tachycardia are caused by these hemodynamic changes [6]. The intussusception of the origin part of the RCA into the aortic wall is described as the intramural part and it may be compressed by the dilated aorta during increased cardiac output [2]. The presence of abnormal slit-like ostium in the aortic wall and emergence of the RCA with an acute angle may restrict coronary blood flow [2]. The emergence of aberrant coronary artery with an acute angle from the aorta and a tortuous course of its proximal part accelerate atherosclerosis [2].

Most of the anomalous origins of the RCA are detected incidentally. Clinical presentation and disease course may substantially vary among asymptomatic patients. Clinical outcomes are usually benign, and the incidence of sudden death associated with coronary anomalies among these patients is very low. Conservative treatment is ideal particularly for those who do not have an inter-arterial course [4]. In young patients (<35 years) and for patients who have angina symptoms and ischemia during an exercise stress test, percutaneous coronary intervention (PCI) or surgical treatment is usually considered. Surgery is not needed in elderly patients without angina symptoms or ischemic findings [5]. Excellent results have been obtained with medical treatment in these patients in the long-term, and no cardiac ischemia was reported [8].

Treatment options include medical treatment in conjunction with restriction of high-force physical exercise, placement of a stent into the compressed vessel, or surgical treatment. Although medical treatment is effective and less invasive, high-force physical exercise should be avoided, since there is a potential risk for sudden cardiac death [8]. Suggested surgical treatment options are unroofing the coronary artery, re-implantation of the coronary ostium, and coronary artery bypass grafting [7]. In the presence of an intramural segment in the abnormal coronary artery, the intramural segment of the coronary artery is dissected and re-implanted into the correct sinus of Valsalva. Unroofing is a technically established and safe approach to correct the anomaly [8]. It can be also applied to abnormal arteries with slit-like ostium and a supracommissural intramural course. Vessel translocation or re-implantation is recommended as a treatment option in patients without an intramural segment [8]. The literature review showed that coronary artery bypass grafting would be the most appropriate choice of treatment in patients older than 60 years old in whom concomitant atherosclerotic stenosis is detected in the RCA, as assessed by angiography [8].

Conclusion

Coronary CT angiography is complementary to coronary angiography. The signs of various coronary arterial anomalies on CT and an under¬standing of the clinical importance of these

anomalies are es¬sential for an accurate diagnosis and for treatment planning for the patient.

Competing interests

The authors declare that they have no competing interests

References

1. Fuglsang S, Heiberg J, Byg J, Hjortdal VE. Anomalous origin of the right coro¬nary artery with an interarterial course and intramural part. Int J Surg Case Rep 2015;14:92-4.
2. Angelini P. Coronary artery anomalies: an entity in search of an identity. Circula¬tion 2007;115(10):1296–305.
3. -Satija B, Sanyal K, Katyayni K. Malignant anomalous right coronary artery detected by multidetector row computed tomography coronary angiography. J Cardiovasc Dis Res 2012;3(1):40-2.
4. Suryanarayana P, Lee JZ, Abidov A, Lotun K. Anomalous right coronary artery: case series and review of literature. Cardiovasc Revasc Med 2015;16(6):362-6.
5 Lee BY. Anomalous right coronary artery from the left coronary sinus with an interarterial course: is it really dangerous? Korean Circ J 2009;39(5):175-9.
6- Angelini P, Velasco JA, Flamm S. Coronary anomalies: incidence, pathophysiol-ogy, and clinical relevance. Circulation 2002;105(20):2449-54.
7. Fedoruk LM, Kern JA, Peeler BB, Kron IL. Anomalous origin of the right coronary artery: right internal thoracic artery to right coronary artery bypass is not the answer. J Thorac Cardiovasc Surg. 2007;133(2):456-60.
8. Matsumura K, Matsumoto H, Hata Y, Ueyama T, Kinoshita T, Kuwauchi S, et al. Anomalous Right Coronary Artery Arising from the Left Sinus of Valsalva in a Young Athlete. Intern Med 2016;55(1):55-8.

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Rare Complication of Intracavernosal Injection Therapy: Breakage of Needle Inside a Penis

İntrakavernözal Enjeksiyon Tedavisinin Nadir Bir Komplikasyonu: Penis İçerisinde İğne Kırılması

Breakage of Needle Inside Penis

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Öz

İntrakavernözal enjeksiyon sonrası ağrı, ekimoz, priapism dışında, nadir olarak penis içerisinde iğne kırılması gibi yan etkiler de görülebilmektedir. 63 yaşında intrakavernözal enjeksiyon sırasında enjeksiyon iğnesi penis içinde kırılmış olan hastaya görüntüleme yöntemleri sonrası cerrahi müdahele yapılarak iğne penisden dışarı çıkarılmıştır. İntrakavernözal enjeksiyon tedavisi erektil disfonksiyon tedavisinde etkili ve başarılı bir şekilde uygulanabilir bir yöntemdir. Bu tedaviye başlanmadan önce hastaya tedavinin uygulama şekli ve penis içinde iğne kırılması dahil olabilecek tüm yan etkiler ayrıntılı olarak anlatılmalıdır.

Anahtar Kelimeler

İntrakavernozal Enjeksiyon; İğne Kırılması; Komplikasyon; Erektil Disfonksiyon

Abstract

After intracavernosal injection, side effects such as pain, ecchymosis, priapism, and a very rare complication like needle breakage inside the penis can be observed. We present a case with surgical removal of needle breakage inside the penis. Our case was a 63-year-old patient, who suffered breakage of the needle inside his penis during intracavernosal injection and after screening methods, received surgical intervention to remove the needle. Intracavernosal injection therapy is a method that can be used effectively and successfully in the treatment of erectile dysfunction. Before performing this therapy, the patient should be informed in detail about the implementation of the therapy and all possible side effects including breakage of the needle inside the penis.

Keywords

Intracavernosal Injection; Needle Breakage; Complication; Erectile Dysfunction

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Introduction

The intracavernosal injection method that is used in the treatment of erectile dysfunction was defined in 1982. It is a second line treatment method that is used in patients who do not benefit from oral phosphodiesterase 5 enzyme inhibitors (PDE5i) and it provides satisfactory results in sexual functions [1, 2]. However, there are some local and systemic complications associated with intracavernosal injection. Early complications are pain, ecchymosis, priapism, and syncope and the most frequent late complications are cavernosal fibrosis with a rate of 1.9-16% [3, 4]. In this article, we aimed to present a surgical removal of needle breakage inside the penis that rarely occurs during intracavernosal injection.

Case Report

The 63- year- old male patient, who was known to have Type 2 diabetes mellitus for about 10 years, was given a PDE5i therapy with a diagnosis of erectile dysfunction. However, it failed, and intracavernosal injection therapy was recommended to the patient as a second line therapy. A 28 -gauge insulin injector was broken while the patient was applying a papaverine injection to the right corpus cavernosum and he was immediately admitted to the emergency room. Deep palpation of the penis revealed stiffness in the right lateral side of the penis. In the right half of the penis, hyperemia on a small area consistent with the needle insertion was observed; no open wound or needle tip in the skin of the penis was observed (Picture- 1a). In the laboratory examinations, no pathology except high blood glucose levels was detected, and no hematuria was observed in the urine. Opacity associated with the needle inside the penis was observed in the X-ray imaging (Picture- 1b), while a linear hyperechogenicity of 8.5 mm in diameter, extending the right tunical tissue under the skin, was observed in the penile Doppler ultrasound. Penile exploration was planned for the patient. After degloving the penis, the needle located in the region of the right tunica albuginea was removed with the help of needle forceps (Pictures- 1c, 1d). No complication developed during intraoperative and postoperative period follow-ups. After receiving detailed information and practice regarding intracavernosal injection, he has continued his erectile dysfunction treatment.

Discussion

While PDE5i therapies remain popular today as a first line therapy in the treatment erectile dysfunction, intracavernosal injection therapy is currently used as a second line therapy in a reliable and effective manner [5]. Furthermore, intracavernosal injection therapy is preferred by patients in our country because it is less costly than PDE5i therapy. It is necessary to resolve complications that may develop during this therapy in a way that minimizes harm to the patient and does not cause them to switch to oral therapy or to experience doubts about receiving intracavernosal injection therapy. In the resolution of these complications, there is a wide range of options from symptomatic treatment to surgical intervention. When we examine the literature, there are a few case presentations of needle breakage inside a penis during intracavernosal injection and its treatment.

Akyol et al. has explained the technical reasons for breakage of needle inside the penis. The administration of the needle obliquely to the corpus cavernosum longitudinal line results in further forward movement in the corpus cavernosum and the needle may break when retracted as a result of improper bending force. It was also reported that using the correct injection method, practice with a physician, and manual skill, can prevent these complications; socio-economic status is also a factor [6]. There are few publications in the literature about intracorporeal needle breakage. Nevertheless, it has been reported in the literature that penile fibrosis and lateral deviation develop after deferred treatment [7].

Iacono and Barra have recommended that, if possible, the needle should be manually retracted; if it is not possible, it should be removed surgically [8]. Wayland Hsiao et al. presented in their case that location of the needle can be determined with the guidance of a portable ultrasound and that it can be retrieved without circumcision and without completely degloving the penis. The foreign object can be removed with a direct incision in the penis skin [9]. In this case, we preferred to remove the broken needle surgically, according to the general literature. In conclusion, intracavernosal injection therapy should be practically described to the patient, taking into consideration his socio-cultural background. The patient should be informed of all possible early and late complications of this therapy including breakage of needle inside the penis.

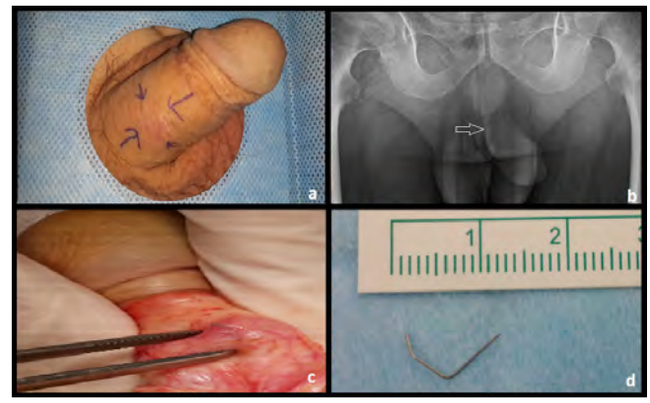

Figure 1. In the right half of the penis, the hyperemic lesion area indicated by the arrows (a); The needle opacity indicated by the arrow in the X-ray imaging (b); Operation image (c); Macroscopic view of the needle (d).

Competing Interests

The authors declare that they have no competing interests.

References

1. Virag R. Intracavernous injection of papaverine for erectile failure. Lancet 1982;8304:938.
2. Hsiao W , Bennett N, Guhring P, Narus J, Mulhall JP. Satisfaction profiles in men using intracavernosal injection therapy. J Sex Med 2011;8:512–7.
3. Porst H. Ten years of experience with various vasoactive drugs. Comparative studies in over 4000 patients. Int. J. Impotence Res 1994;6:149.
4. Chen R, Lakin M, Montague DK, Ausmundson S. Penile scarring with intracavernosal injection therapy using prostaglandinE1: a risk factor analysis. J Urol 1996;155:138–40.
5. Beer SJ, See WA. Intracorporeal needle breakage: An unusual complication of papaverine injection therapy. J Urol 1992;147:148-50.
6. Akyol I, Adayener C, Senkul T, Karademir K, Baykal K, Iseri C. Broken needle in the penis as a complication of intracavernous injection therapy. Gulhane Med J 2007; 49:40-1.
7. Shamloul R, Kamel I. A broken intra cavernous needle: successful ultrasound-guided removal. J Sex Med 2005; 2(1):147-8.
8. Iacono F, Barra S. Intracorporeal needle breakage as an unusual complication of intracavernous self-injection. Tech Urol 1998; 4:54-5.

9. Hsiao W, Lian F, Goodgame B, Ritenour CW, Angell J, Master VA. Breakage of Needle during Intracavernosal Injection and Use of Portable Ultrasound Guidance for Removal. Case Rep Urol 2013; 2013:215492.

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Case Report
JCAM

Left Side Located Cholelithiasis Case

Sol Taraf Yerleşimli Kolelitiazis Olgusu

Left-Sided Gallbladder

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Öz

Sol taraf yerleşimli safra kesesi round ve falsiform ligamentin solunda bulunan safra kesesidir. Sık görülmeyen bir anatomik anormalliktir. Laparoskopik kolesistektomi en sık uygulanan cerrahi prosedürlerden bir tanesidir ve sol taraf yerleşimli safra kesesi durumunda güvenlik kurallarına uyulduğu ve özel stratejiler dikkate alındığı sürece laparoskopik cerrahi güvenli gözükmektedir. Laparoskopik kolesistektomi esnasında tanımlanan sol taraf yerleşimli safra kesesi olgusunu bildirirken sol taraf yerleşimli değişik safra kesesi formlarını ve bu farklılığın cerrahi ile ilişkisini tartışmaktayız.

Anahtar Kelimeler

Sol Taraf Yerleşimli Safra Kesesi; Laparoskopik Kolesistektomi; Aberan Safra Kesesi

Abstract

A left-sided gallbladder is a gallbladder located on the left side of the round and falciform ligament. It constitutes an uncommon anatomic abnormality. Laparoscopic cholecystectomy is one of the most commonly performed surgical procedures and in case of left-sided gallbladder, it seems to be safe as long as safety rules are followed and specific strategies are considered. We report a case of a left-sided gallbladder identified during laparoscopic cholecystectomy and discuss the different forms of left-sided gallbladder and the surgical relevance of this anomaly.

Keywords

Left-Sided Gallbladder; Laparoscopic Cholecystectomy; Aberrant Gallbladder

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Introduction

The gallbladder develops from an endodermal outpouching of the hepatic diverticulum and sits just beneath the right lobe of the liver [1]. The position of the gallbladder in relation to the liver may vary, with documented cases such as transverse, intrahepatic, retrodisplaced, and left-sided [2]. Such variants are very rare: reported cases of a left-sided gallbladder have been no more than 200; the incidence is 0.4% [3]. Left-sided gallbladder is often accompanied by biliary, portal venous, and other anomalies that might lead to intra-operative injuries. Hence, identification of this anomaly is important when performing laparoscopic cholecystectomy.

Case Report

A 44-year-old woman presented with right upper quadrant pain. Physical examination was unremarkable. Laboratory data were within normal limits. Ultrasound demonstrated cholecystolithiasis with no evidence of cholecystitis or choledocholithiasis. The patient was informed of the diagnosis, and a laparoscopic cholecystectomy was performed using the standard 4-port technique. Intra-operative view of the gallbladder indicated that it was located to the left of the falciform ligament (Figure 1,2). Meticulous dissection of the Calot triangle revealed that the cystic duct joined the common hepatic duct on the right side. The cystic artery was arising normally from the right hepatic artery. The gallbladder was detached from the liver bed by a antegrade dissection. The procedure was completed successfully. The patient was discharged home on postoperative day 1 and had an uneventful postoperative period. Pathological examination confirmed choleystolithiasis.

Discussion

Despite common variations in hepatobiliary anatomy, congenital anomalies of gallbladder position are rare. There are four types of aberrant gallbladder: (I) left-sided; (II) intrahepatic;(III) transverse; and (IV) retrodisplaced [2]. Of these four, the left-sided gallbladder without situs inversus viscerum is the rarest. It was first described by Hochstetter in 1886 [4]. Although the exact etiology of left-sided gallbladder has not been established definitely, there are many theories. If the cystic duct from a left-sided gallbladder joins the common hepatic duct from its right side, the variation has been regarded as a defect migration; if the cystic duct merges with the common hepatic duct on its left side or drains into the left hepatic duct, left-sided budding of the gallbladder/cystic duct anlage in the embryo has been proposed [5,6]. Moreover, some authors have regarded the left-sided gallbladder as the result of a poorly developed quadrate lobe while others have seen the right-sided round ligament as the true cause of the problem [3,5]. Associated anomalies with left-sided gallbladder include complete or partial situs inversus, duplicated gallbladder, hypoplastic bile duct, anomalous pancreato-biliary ductal junction, absence of the quadrate lobe, accessory liver, and anomalous intrahepatic branching of the portal vein [7]. It has also been associated with complex congenital abnormalities such as the main bile duct lying in front of the first part of the duodenum and malrotation of the intestine, an anteduodenal portal vein, an anular pancreas, agenesis of the dorsal pancreas, polysplenia and highly mobile right colon, liver cyst, and with intrapelvic ectopic testis with ectopic vesica and an umbilical hernia [3,7]. Recent data indicate that routine ultrasonography in patients with gallstone disease may fail to identify left-sided gallbladder disease. A left-sided gallbladder increases the risk of bile duct injury during laparoscopic cholecystectomy [3]. As associated anomalies are to be expected with left-sided gallbladder, the surgeon has to be cautious while performing laparoscopic cholecystectomy. Using specific operative strategies, such as mirror image port setup, fundus first dissection, placement of additional ports, use of existing port sites with manipulation of falciforme ligament, different patient positioning, and intra-operative cholangiography, can be the key to the successful and safe management of these patients [8].

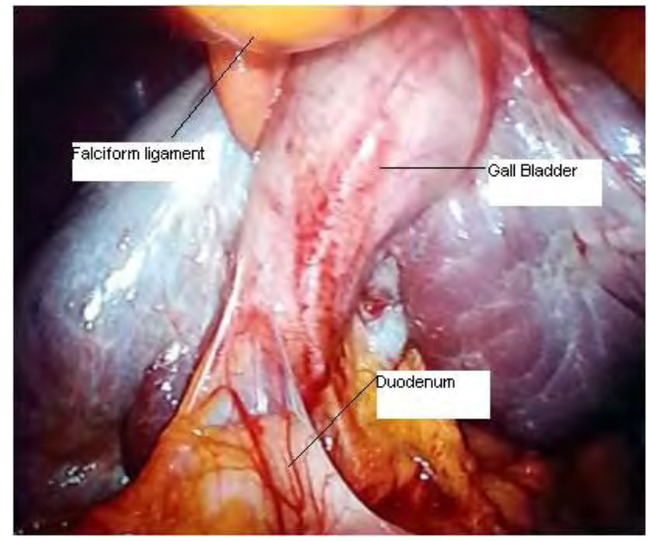

Figure 1. Gallbladder's position in surgery

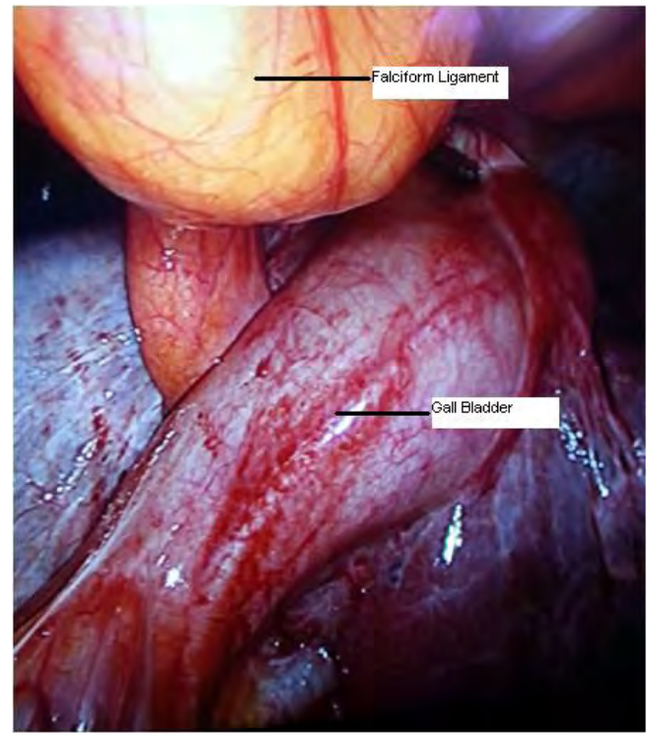

Figure 2. Gallbladder' position in surgery

Conclusion

Left-sided gallblader is often discovered during surgery. If the surgeon is experienced and aware of possible associated anomalies of left-sided gallbladder, laparoscopic cholecystectomy can be performed successfully.

Competing interests

The authors declare that they have no competing interests.

References

1. Gary C. Schoenwolf. Larsen's human embryology (Thoroughly rev. and updated 5th ed.). Philadelphia: Development of the Gastrointestinal Tract Churchill Livingstone/Elsevier 2015.p.341-75.
2. Gross RE. Congenital anomalies of the gallbladder. Arcg Surg 1936;32:131-62.
3. Strong RW, Fawcett J, Hatzifotis M, Hodgkinson P, Lynch S, O'Rourke T, et al. Surgical implications of a left-sided gallbladder. Am J Surg 2013;206(1):59-63.
4. Dhulkotia A, Kumar S, Kabra V, Shukla HS. Aberrant gallbladder situated beneath the left lobe of liver. HPB (Oxford) 2002;4(1):39–42.
5. Stringer MD. Gallbladder and Extrahepatic Bile Ducts. Bergman's Comprehensive Encyclopedia of Human Anatomic Variation 2016.p.1261-71.
6. Iskandar ME, Radzio A, Krikhely M, Leitman IM. Laparoscopic cholecystectomy for a left-sided gallbladder. World J Gastroenterol 2013;19:5925-8.
7. Colovi R, Colovic N, Barisic G, Atkinson HD, Krivokapic Z. Left-sided gallbladder associated with congenital liver cyst. HPB 2006;8(2):157-8.
8. Nastos C, Vezakis A, Papaconstantinou I, Theodosopoulos T, Koutoulidis V, Polymeneas G. Methods of safe laparoscopic cholecystectomy for left-sided (sinistroposition) gallbladder: A report of two cases and a review of safe techniques.Int J Surg Case Rep 2014;5(10):769-73.

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The Benign Childhood Acute Myositis Due to Rotavirus Infections: Case Report

Rotavirüs Enfeksiyonuna Bağlı Çocukluk Çağının Selim Akut Miyoziti: Olgu Sunumu

Rotavirus Infection Myositis

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Öz

Çocukluk çağının akut selim miyoziti özellikle okul çağındaki genellikle erkek çocukları etkileyen, alt ekstremitenin distal kaslarını simetrik olarak tutarak yürüme güçlüğüne neden olan ve kendiliğinden düzelen bir klinik durumdur. Akut dönemde, ilerleyerek çocuğun yürümesini engellemesi ve ağrıya yol açmasıyla hekim ve ana babaları telaşlandırabilir. En sık influenza viruslarına bağlı olarak ortaya çıkmaktadır. Ancak diğer virüsler ile de nadiren görülmektedir. Tipik laboratuvar bulgusu kreatinfosfokinaz düzeyindeki yükselmedir. Ayırıcı tanıda Guillain-Barré sendromu, müsküler distrofiler, dermatomiyozit, piyomiyozit ve büyüme ağrıları vardır. Bu yazıda 5,5 yaşındaki bir kız hastada rotavirüs enfeksiyonuna ikincil olarak gelişen akut myozit tablosu sunulmuştur.

Anahtar Kelimeler

Rotavirüs Enfeksiyonları; Çocuk; Myozit; Gastroenterit; Komplikasyonlar

Abstract

Benign acute childhood myositis usually affects males, especially school children, causes difficulty in walking by keeping the distal muscles of lower extremity symmetrically, and is a clinical condition that resolves spontaneously. In the acute stage, it can alarm the physician and the parents by blocking the child's walking and leading to pain. It arises most frequently due to influenza viruses, while it is rarely seen with other viruses. The typical laboratory finding is an increase in the level of keratin phosphokinase. The differential diagnosis includes Guillain-Barré syndrome, muscular dystrophy, dermatomyositis, pyomyositis, and growing pain. This paper presents an acute myositis case developing secondarily in a rotavirus infection in a 5.5-year-old female patient.

Keywords

Rotavirus Infection; Child; Myositis; Gastroenteritis; Complications.

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Introduction

Rotavirus infection in childhood is the most common cause of acute gastroenteritis. Although it mainly locates in the intestinal tract, it may rarely manifests itself outside of the intestinal tract. Also, neurologic complications, encephalitis, febrile convulsion and aseptic meningitis have been detected.

Benign Acute Childhood Myositis (BACM) is a clinical condition of bilateral calf pain that develops suddenly immediately after viral infections, usually affects school-age children, is characterized by walking difficulties, and resolves spontaneously [1]. Usually developings due to post-infectious pathogens, myositis associated with rotaviruses is rarely reported [2]. A typical laboratory finding is a high level of serum keratin phosphokinase (CPK) [3]. Knowing detailed characteristics of benign acute childhood myositis will prevent unnecessary tests and treatments.

In the literature, only four cases of myositis associated with rotavirus infection have been reported to date [4]. In this paper, we present a case of benign acute childhood myositis developing secondarily associated with rotavirus gastroenteritis infection in a 5.5-year-old.

Case Report

A 5.5-year-old girl presented with fever, fatigue, weakness, and vomiting to the emergency clinic in our hospital. Upon determining the CPK of the patient to be 9806 U/L, the patient was brought to the neurology clinic. Three days later, as her current complaints increased, she was brought to our emergency department. In addition to the patient's previous complaints, diarrhea, inability to walk, and pain in both thighs had already begun. Due to malnutrition and general weakness of the patient, she was admitted to our Child Health and Diseases Department in the hospital. In her physical examination, the general physical state of the patient was moderate. The patient's vital findings were stable. In her neurological examination, vulnerability to palpation on both sides of the quadriceps muscle was observed. However, no swelling, redness, or laceration was detected. In muscle strength examination, all extremities were detected as normal. There was no loss of sensation. Deep tendon reflexes were normoactive. Laboratory examinations of the patient's blood count, erythrocyte sedimentation rate, and C-reactive protein were normal. Biochemical tests performed on the patient are shown in Table 1. Rotavirus antigen was found positive in the stool test. The patient was diagnosed with BACM associated with rotavirus. The patient was discharged from the hospital with a marked improvement in her general condition. A week later, her physical examination and lab findings were found to be normal.

Table 1. The course of the laboratory findings of our patient.

	AST N:0-31U/L	ALT N:0-34 U/L	LDH N:0-247 U/L	GGT 0-32 U/L	CPK 0-145 U/L
1st day of disease	290	88	652	16	9806
3st day of disease	174	80	466	16	2728
6st day of disease	56	49	286	15	175
discharge after 1 week	28	24	242	16	102

Discussion

BACM was defined for the first time in 1957 by Lundberg [1]. as a sudden calf pain and inability to walk described in 74 children following a viral upper respiratory tract infection, often occurring after the 1-5 day prodromal period and resolving spontaneously [2]. The pain is particularly pronounced when the patient first gets up in the morning. In the etiology, there are often Influenza type A and B viruses, RSV, adenoviruses, HSV, EBV, or CMV [4,5]. In our case, we did not test these when forming the differential diagnosis because there were no symptoms suggestive of viral upper respiratory tract infection.

Since patients complain about an inability to walk, stand up, and step onto their feet, all families become extremely worried about their children and refer to the emergency departments of hospitals. Rajajee et al. reported that cases of BACM are most frequently directed to the clinic with a preliminary diagnosis of GBS [5]. However, in GBS, a two-sided muscle weakness and pain moving upward is observed, decreased deep tendon reflexes are detected in physical examination, and serum CPK level is within normal limits.

The majority of patients with myositis associated with rotavirus are over 2 years of age [5]. On the other hand, most children with rotavirus gastroenteritis are under 2 years of age. In a case series examining 40 patients diagnosed with BACM, 55% of them were male, an average onset time of symptoms was 5.3 days, and the duration of the prodromal phase symptoms was 3.97 days [5].

There are many culprits likely to lead to muscle pain in children. During the course of viral infections, a widespread myalgia can be seen. However, BACM typically presents with bilateral gastrocnemius muscle involvement. Important clues in differentiating BACM from muscular dystrophy is the apparent lack of pain in muscular dystrophy due to increased serum CPK and no improvement in clinical findings during the course of the disease. There are several typical skin conditions that can cause similar clinical findings in children, such as dermatomyositis, which presents with a progressive bilateral proximal muscle weakness, an increased CPK level, EMG changes, unusual muscle biopsy findings, edema, and purplish color change in the eyelids [6].

For the diagnosis of BACM, the most typical diagnostic finding is an increased CPK level in the serum [3]. Moderate degree CPK has been reported to range from 558 to 6800. In our case, the CPK level was 9806. It is recommended that, due to the possibility of increased muscle cell damage, diagnosticians should be alert to rhabdomyolysis and kidney failure and should monitor the pain and urine color [7]. In our case, renal function tests and urine analyses that were performed because of these possibilities were normal.

The mechanism responsible for BACM associated with rotavirus has not yet been determined. However, it can be characterized as damage resulting from direct invasion of immunological reactions triggered by virus particles inside the muscle tissue [8]. Since the symptoms are short-term and improve spontaneously, further examinations such as electromyography and muscle biopsy have been rarely consulted [5].

To summarize: BACM is a clinical condition with viral infection that is self-limiting in the healing process, with a prognosis that

is very good and does not require clinical treatment. The observation of rapid improvement in clinical findings is significant for differentiating BACM, which doesn't require further tests and resolves spontaneously, from other diseases that may have more severe courses and will require different treatment options.

Competing interests

The authors declare that they have no competing interests.

References

1. Lundberg A. Myalgia cruris epidemica. Acta Paediatr Scand 1957;46:18-31.
2. Hu JJ, Kao CL, Lee PI, Chen CM, Lee CY, Lu CY, et al. Clinical features of influenza A and B in children and association with myositis. J Microbiol Immunol Infect 2004;37:95-8.
3. Zafeiriou DI, Kataos G, Gombakis N, Kontopoulos EE, Tsantali C, et al. Clinical features, laboratory findings and differential diagnosis of benign acute childhood myositis. Acta Paediatr 2000;89:1493-4.
4.Yamamoto K, Fukuda S, Minami N, Kanai R, Tsukamoto K, et al. Acute myositis associated with concurrent infection of rotavirus and norovirus in a 2-year-old girl. Pediatric Reports 2015;7:5873:58:51-3.
5. Rajajee S, Ezhilarasi S, Rajarajan K. Benign acute childhood myositis. Indian J Pediatr 2005;72:399-400.
6. Compeyrot-Lacassagne S, Feldman BM. Inflammatory myopathies in children. Pediatr Clin North Am 2005;52:493-520.
7. Tabbutt S, Leonard M, Godinez RI, Sebert M, Cullen J, Spray TL, et al. Severe influenza B myocarditis and myositis. Pediatr Crit Care Med 2004;5:403-6.
8. Warren JD, Blumbergs PC, Thompson PD. Rhabdomyolysis: a review. Muscle Nerve 2002;25:332-47.

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Esophagial Perforation Due to Chicken Bone Ingestion: Multidetector CT Findings

Esophageal Perforation Due to Chicken Bone Ingestion: Multidetector CT Findings

Mediastinitis Due to Esophageal Perforation

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Öz

Yabancı cisim impaksiyonuna bağlı gelişen özefagus perforasyonunda hayatı tehdit edici komplikasyonların önlenebilmesi için erken tanı ve tedavi önemlidir. Erişkinlerde genelde perforasyon sebebi et, balık ve tavuk kemikleridir. Boğaz ağrısı, disfaji ve kusma sık görülen semptomlardır, ancak perforasyon eşlik ederse medastinit gibi ciddi komplikasyonlar gelişebilir. 52 yaşında erkek hasta, 6 gün önce tavuk eti yedikten sonra ortaya çıkan boğaz ağrısı ve disfaji şikayetleriyle acil servise başvurdu. Toraks bilgisayarlı tomografisinde (BT)tavuk lades kemiğine ait Y şeklinde yabancı cisim gözlendi. Çok kesitli BT ile özefagus perforasyona bağlı mediastinal hava ve sıvı koleksiyonu gibi mediastinit bulguları da saptandı. Özefagustaki yabancı cisimler ve bunlara bağlı komplikasyonları göstermede çok kesitli BT önemli bir rol oynar.

Anahtar Kelimeler

Yabancı Cisimler; Özofagus Perforasyonu; Mediastinit; Çok Kesitli Bilgisayarlı Tomografi

Abstract

Esophageal perforation (EP) due to foreign body impaction requires early diagnosis and treatment in order to prevent life-threatening complications. EP in adults is most frequently caused by the impaction of meat, fish, and chicken bones. The common symptoms are sore throat, dysphagia, and vomiting, but in the presence of perforation, mediastinitis and other complications can occur. In our case, a 52-year-old man was admitted to the emergency room with a six-day history of sore throat and dysphagia after eating chicken. Computed tomographyof the thorax revealed a Y-shaped foreign body resembling the wishbone of a chicken. Mediastinal air secondary to esophageal perforation and further evidence of mediastinitis were also discovered with multidetector computed tomography (MDCT). This case shows that MDCT plays an important role in detecting esophageal foreign bodies and their complications.

Keywords

Foreign Bodies; Esophageal Perforation; Mediastinitis; Multidetector Computed Tomography

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Introduction

Esophageal perforation (EP) due to foreign body (FB) impaction is rare and requires prompt treatment [1, 2, 3]. The most common esophageal FB diseases in adults are caused by impacted meat or other food. Fish bones in particular have been known to cause EP in many countries [1, 2, 4, 5]. Cases of spontaneous and iatrogenic ruptures have also been observed. About 80–90% of ingested FBs pass spontaneously without complications, 10–20% of them require endoscopic removal, and approximately 1% require surgical procedures [1, 6]. A sharp object in the esophagus must be removed within 24 hours to minimize the risk of perforation, mediastinitis, or abscess [3, 4].

Case Report

A 52-year-old man was admitted to the emergency room with a six-day history of sore throat and dysphagia after eating chicken. He was afebrile on arrivaland presented with an elevated WBC count (18.3x109/L). Computed tomography (CT) of the thorax revealed a Y-shaped FB resembling the wishbone of a chicken in his proximal esophagus (Figure 1). Pneumomediastinum secondary to esophageal perforation and evidence of mediastinitis were also observed using multidetector CT (MDCT) (Figure 2). Upper gastrointestinal endoscopy confirmed the diagnosis and the chicken bone was removed from the patient's esophagus with a snare. An over-the-scope clip was applied to treat the esophageal perforation. A right thoracotomy was performed, which detected necrotizing mediastinitis. The mediastinal cavity was cleaned with betadine and saline. Empyema developed in the right hemithorax after the operation (Figure 3). The patient was discharged after two months of medical therapy.

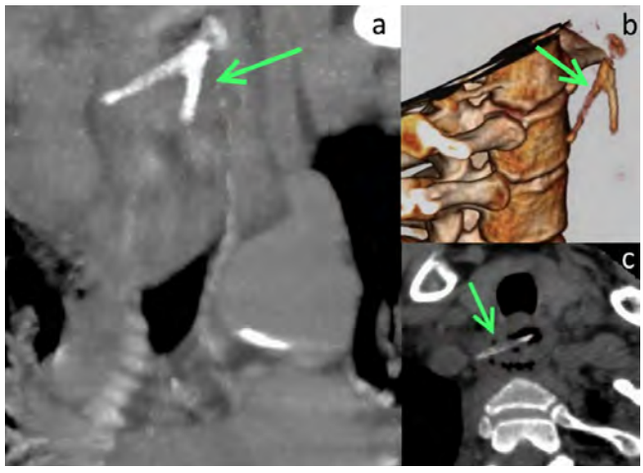

Figure 1. CT images of wishbone of the chicken in the esophagus. MIP and 3D images (a, b). Note that the chicken bone is extending outside the esophagus, representing esophageal rupture (c).

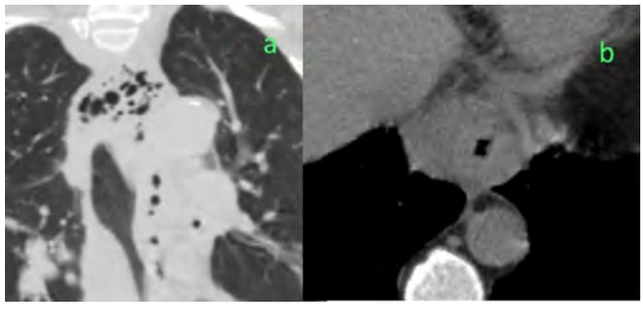

Figure 2. Pneumomediastinum (a). Paraesophageal fluid collections representing mediastinitis (b).

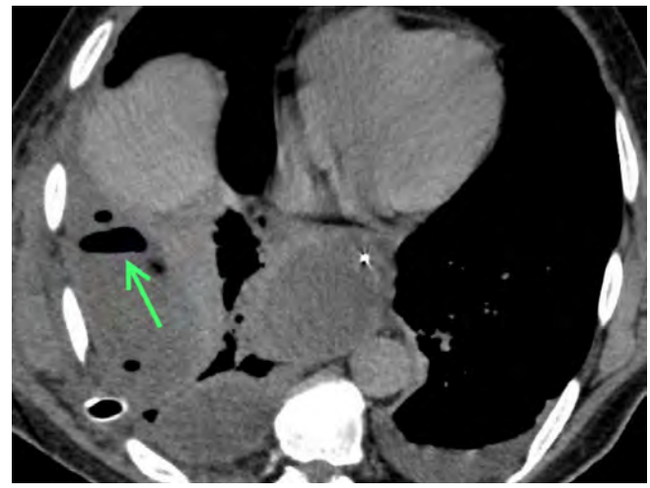

Figure 3. Right sided empyema and suspended air bubbles.

Discussion

Ingestion of FBs is a common occurence, and the majority of such cases involve spontaneous passage of the FB through the esophagus [1, 6, 7]. Some patients may remain asymptomatic for many years, thoughEP is a potentially life-threatening condition with high rates of morbidity and mortality [1, 4]. EP occurs in only 1–2% of FB ingestion cases [6]. More attention should be paid to sharp FBs (e.g., metallic objects, chicken and fish bones, and toothpicks) due to a higher risk of perforation and mediastinitis [1, 2, 3, 4, 7]. EP due to FB ingestion usually occurs in the cervical esophagus, whereas spontaneous or iatrogenic injuries occur distally [1, 6]. The time that has passed between esophageal injury and initiation of treatment is the most important factoraffecting mortality in cases of EP [4]. In this case, the patient was admitted to the emergency room six days after ingesting a chicken bone that had caused the perforation of his esophagus and mediastinitis. As this case demonstrates, early removal of FBs is necessary to prevent the development of mediastinitis [3, 4, 5].

Any degree of sore throat, dysphagia, odynophagia, difficulty breathing, and vomiting are common symptoms in esophageal FB diseases. FB sensation and localized pain are some patients' main complaints in the early periods of such diseases. As the disease progresses or complications develop, localized inflammatory or systemic symptoms, such as swelling of the neck, hematemesis, dysphagia, dyspnea, fever, and chest pain can occur [5, 6].

FBs in the alimentary tract should be evaluated carefully to identify their exact anatomic location [2]. Fiberoptic laryngoscopy or chest and neck X-rays can be performed to this end, but they are not always successfully diagnostic. Cervical or mediastinal emphysema, pleural effusion, or radiopaque objects may be seen on X-rays [1, 6]. FBs need to be radiopaque in order to detect them with X-rays, but radiolucent FBs are common and may not be noticed on radiographs [1, 3]. The most common radiolucent objects that can be ingested are fish and chicken bones, wood, plastic, and thin metal [1]. As such, radiography may not be a sufficient examination method for the detection of esophageal FBs [2]. Removal of these FBs must be performed within 24 hours of ingestion to prevent perforation and fistula formation [1, 4, 8]. The exact location of a foreign object can be

determined byMDCTquickly and with high diagnostic accuracy [2, 3]. MDCT should therefore be the first choice of imaging method to detect sharp esophageal FBs that do not show up byclinical inspection or radiographs. Slightly calcified FBs can appear on CT, which has higher contrast and spatial resolution [2]. CT has previously detected esophageal bone impactions with 90–100% sensitivity, with positive predictive rates as high as 99% (6). Wall thickening, surrounding soft tissue changes, and free air, and complications such as wall perforation, fistulae, and mediastinitis can also be detected with CT [1, 2, 3]. If complications are suspected, an intravenous contrast agent would be a great asset for the diagnosis process (1).

Proper treatment of esophageal FB diseases depends on the individual clinical situation. Conservative or surgical treatment options can be utilized [6]. Sharp objects, such as chicken or fish bones, should be removed carefully by endoscopy to prevent EP [6, 8]. In cases where perforations have been caught early, primary repair is preferred, but esophageal exclusion/diversions and thorough drainage may be needed. If there is no evidence of mediastinal contamination, endoscopic clips may be used in the treatment of acute esophageal perforations [8].

In conclusion, esophageally impacted FBs require quick diagnosis and treatment because of the risk of serious complications such as perforation and mediastinitis. Therefore, the first choice of diagnostic method should be MDCT, which can not only localize the FB, but will also display wall and surrounding soft tissue changes.

Competing interests

The authors declare that they have no competing interests.

References

1. Guelfguat M, Kaplinskiy V, Reddy SH, DiPoce J. Clinical guidelines for imaging and reporting ingested foreign bodies. AJR Am J Roentgenol. 2014 Jul;203(1):37-53. doi: 10.2214/AJR.13.12185. Review. Erratum in: AJR Am J Roentgenol. 2014;203(3):694.

2. Ma J, Kang DK, Bae JI, Park KJ, Sun JS. Value of MDCT in diagnosis and management of esophageal sharp or pointed foreign bodies according to level of esophagus. AJR Am J Roentgenol 2013;201(5):W707-11. doi: 10.2214/AJR.12.8517.

3. Liguori C, Gagliardi N, Saturnino PP, Pinto A, Romano L. Multidetector Computed Tomography of Pharyngo-Esophageal erforations. Semin Ultrasound CT MR 2016;37(1):10-5.

4. Park IH, Lim HK, Song SW, Lee KH. Perforation of esophagus and subsequent mediastinitis following mussel shell ingestion. J Thorac Dis 2016;8(8):E693-7.

5. Kim EY, Min YG, Bista AB, Park KJ, Kang DK, Sun JS. Usefulness of Ultralow-Dose (Submillisievert) Chest CT Using Iterative Reconstruction for Initial Evaluation of Sharp Fish Bone Esophageal Foreign Body. AJR Am J Roentgenol 2015;205(5):985-90.

6. Aronberg RM, Punekar SR, Adam SI, Judson BL, Mehra S, Yarbrough WG. Esophageal perforation caused by edible foreign bodies: a systematic review of the literature. Laryngoscope 2015;125(2):371-8.

7. Mondin RM, Fandiño M, Carpes LF, Tang J, Ogilvie LN, Kozak FK. Esophageal wishbone extraction: not for the chicken-hearted. Ann Otol Rhinol Laryngol 2015;124(1):79-82.

8. Triadafilopoulos G, Roorda A, Akiyama J. Update on foreign bodies in the esophagus: diagnosis and management. Curr Gastroenterol Rep 2013;15(4):317. doi: 10.1007/s11894-013-0317-5.

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From the Symptoms of an Undiagnosed Mother to the Infant with Congenital Myotonic Dystrophy

Tanısız Annenin Semptomlarından Konjenital Myotonik Distrofili Bebeğe

Congenital Myotonic Dystrophy

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Neonataloji BD, Necmettin Erbakan Üniversitesi, Konya, Türkiye

Öz

Konjenital myotonik distrofi (KMD) neonatal dönemde, hipotoni, respiratuar yetmezlik ve beslenme problemleri ile bulgu veren klinik spektrumu geniş bir hastalıktır. Aile öyküsü tanıda önemli olmakla birlikte neonatal dönemde bebeğin tanı alması annenin ya da nadiren babanın tanı almasını sağlayabilir. Burada neonatal dönemde hipotonisite ve solunum yetmezliği ile prezente olan erkek hastada, annenin halsizlik, kas ağrısı ve çok uyuma şikayetlerinden yola çıkılarak genetik analizle KMD tanısına varıldı. Vakamızda olduğu gibi, hipotonik bebeklerde yalnızca aile öyküsünün derinleştirilerek KMD tanısının daha erken konabileceği akılda tutulmalıdır.

Anahtar Kelimeler

Konjenital Myotonik Distrofi; Hipotonik İnfant; Polihidroamniyoz; Dismorfik Yüz; Solunum Yetmezliği

Abstract

Congenital myotonic dystrophia (CMD) is a disorder with a wide clinical spectrum, characterized by hypotonia, respiratory failure, and nutritional challenges in the neonatal period. Although familial history is important in the diagnostic process, diagnosing the infant in the neonatal period may, conversely, lead the mother, or rarely the father, to be diagnosed. Here, a male infant presenting with hypotonicity and respiratory failure in the neonatal period was diagnosed with CMD through genetic testing by looking at the complaints of fatigue, muscle pain, and hypersomnia in the mother. As in our case, it should be kept in mind that CMD can be diagnosed at an early stage only by focusing on the familial history in hypotonic infants.

Keywords

Congenital Myotonic Dystrophia; Hypotonic Infant; Polyhydramnios; Dysmorphic Face; Respiratory Failure

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Introduction

Congenital myotonic dysthropia (CMD) is a neuromuscular disorder with an autosomal dominant hereditary transmission. In CMD, a disorder with a prevalence of 1/100,000 across the world, facial dysmorphic signs, such as fishmouthing, long face, ptosis, and temporal muscle atrophy are remarkable findings seen at the time of delivery. Patients with CMD present with severe hypotonia, talipes equinovarus, artrogryposis, weakened weeping and sucking, and respiratory failure. Congenital cataracts, thyroid dysfunction, and cardiac involvement are also seen among cases with CMD, but more rarely [1,2]. CMD develops as a result of the repetition of the myotonic dystrophy protein kinase (DMPK) gene in chromosome 19q13 due to the increased cytosine-thymine-guanine (CTG) trinucleoid in the 3' untranslated region. In such patients, the repetition of CTG is generally found to be more than 1,000 [2], and the mortality is between 30-40% in the neonatal period [3]. Here, we aimed at presenting how an infant with the complaints of hypotonicity and respiratory failure during the neonatal period was diagnosed with CMD by focusing on the infant's symptomatic, but previously undiagnosed, mother.

Case Report

Our case was born at term to a 23-year-old mother as the second living infant after the second pregnancy through cesarean section, and the infant's Apgar scores were 5 and 7 at minutes 1 and 5, respectively. Resuscitated in the delivery room, the patient was taken to the neonatal department after the intubation. The patient's parents with non-kinship also had a 2-year-old healthy daughter. The prenatal history of the patient revealed polyhydramnios. While the patient's weight, head circumference, and height were 2750 gr (25-50 p), 35 cm (90 p), and 48 cm (50-75 p) respectively, the physical examination also revealed prominent root of nose, antraverted nostrils, higher palate, retrognathia and micrognathia, and inverted-V-shaped upper lip (Figure 1). The cardiological and gastrointestinal systems examination of the patient conducted following intubation and mechanical ventilation were within normal limits. On the neurological examination, hypotonicity and hyperlacticity in all extremities were present, and no deep tendon reflexes were recorded in the lower extremities. Bilateral talipes echinovarus deformity was observed, and metabolic tests of the patient with syndromic presentation were detected to be normal. Investigated in terms of hypotonicity, the level of creatine kinases (CK) (85 U/L) was at normal level. Cranial magnetic resonance imaging (MRI) demonstrated subdural hemorrhage on the right parietal region. The speech defects in the patient's mother drew attention. Although previously evaluated many times in the neurology and psychiatry departments due to the complaints of hypersomnia and fatigue, the mother had not been diagnosed with anything. On physical examination, the mother displayed myotonic findings such as masklike face and difficulty in relaxation after gripping fingers (Figure 2). After performing electromyography (EMG), myotonic discharges were determined in the mother, and so the patient was genetically evaluated in terms of CMD. As a result of the genetic testing, at least 50 CTG repeat size was detected in an allele of the myotonic dystrophia protein kinase (DMPK) gene, and the patient was diagnosed with CMD. The patient was supported with mechanical ventilation for 20 days during the hospital stay. On day 58, the patient was discharged with oral feeding and without supplemental oxygen.

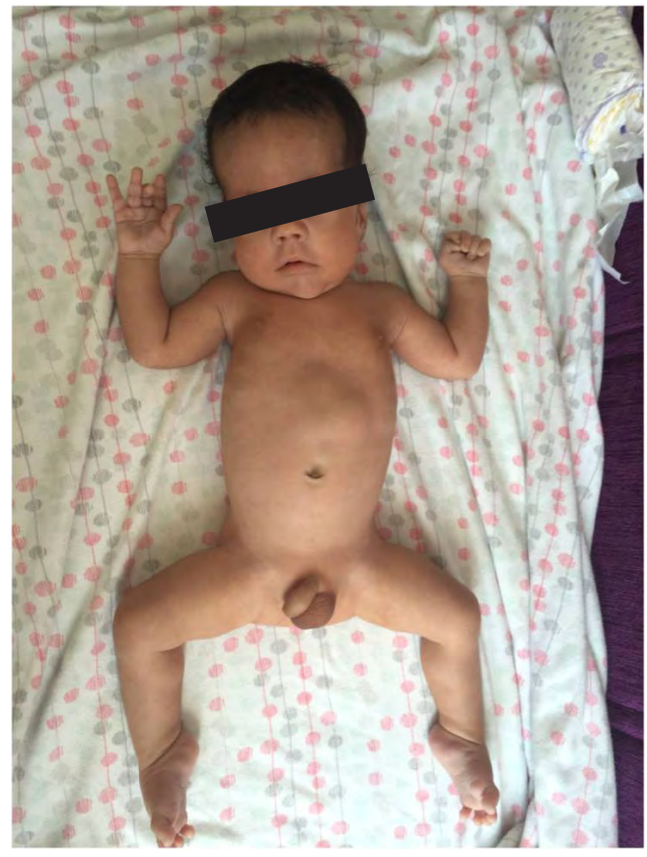

Figure 1. Floppy infant with bilateral talipes echinovarus deformity

Figure 2. Patient's mother showing masklike face and infant with antraverted nostrils, retrognathia and micrognathia, and inverted-V-shaped upper lip

Discussion

The clinical features of CMD were first described by Hans Steinert et al. at the beginning of the 1900s [4]. There are two different forms of the disease, both characterized by muscle weaknesses, myotonia, and cataracts. Various genetic differences are observed between type 1 myotonic dystrophy (MD), the most severe form seen at birth or in childhood, and type 2 MD seen during adulthood. While the repetition of CTG is observed in the DMPK gene in type 1 MD, the repetition of cytosine-cytosine-

thymine-guanine (CCTG) repeat expansion occurs in zinc finger protein 9 (ZNF9) gene in type 2 MD. Our case was a type 1 MD patient with a 50 repeat expansion of CTG in the DMPK gene. In patients affected by the condition in the neonatal period, respiratory failure stemming from the weakness of respiratory muscles is one of the most significant challenges. The existence and severity of respiratory failure define the prognosis. While the mortality rate due to respiratory failure is almost 25% in patients needing mechanical ventilation for more than 30 days, the rate increases up to 40% in infants seriously affected by CMD [5]. In our patient, the need for ventilation ceased at the 26th day after birth.

Whilest the congenital form is generally seen in the infants of symptomatic mothers with multisystemic involvement, serious complaints can also be observed in the neonatal period in the infants of mothers with mild symptoms who are not yet diagnosed. Consistent with the literature, the mother of our patient diagnosed with type 1 MD and needing long-term ventilation had yet to be diagnosed. The most commonly encountered cardiac defects in type 1 MD patients are conducting abnormalities, with the rate of 40%. Minor conducting defects can be detected via electrocardiography (ECG) alone. Because CMD is a disorder with a progressive course, the use of an implantable cardioverter defibrillator or pacemaker is proposed in order to prevent sudden cardiac death often seen in such patients [6]. Even though the ECG performed on our patient did not reveal any conducting defect, the patient was followed up by the pediatric cardiology department. In addition, studies have illuminated the association between the width of CTG repetition size in type 1 MD patients and the prevalence and progression of cardiomyopathy. For this reason, the detection of CMD cases at an early stage is important to prevent disease progression by properly following up these patients through ECG.

Even if it is not observed in the neonatal period, myotonia is detected in all cases prior to 11 years of age [5]. The earlier the symptoms of CMD began in the mothers, the more CMD risk increases in the infants. In our patient's mother, myotonia symptoms had been present since her infancy. In a study, Esplin et al. assumed MD as an important cause in idiopathic polyhydramnios. In cases with idiopathic polyhydramnios and fetal swallowing dysfunction, myopathy due to X linked myopathy, congenital myotonic dystrophy, and congenital myopathy are three neuromuscular disorders to be ruled out [7]. In our patient, the history of polyhydramnios also drew attention to such an association, and such signs as the dysarthria detected in the mother, masklike face, marked muscle weakness increasing with pregnancy, and fatigue were the guiding lights for the diagnostic approach to the infant. As with our patient, the fact that while a mild form of the disease is present in the mother, a severe form of CMD is observed in the infant and this demonstrates that tissue mosaicism and other genetic factors are also effective in determining the severity of disease phenotype. In the study performed by Cobo et al., it was shown that the risk of developing CMD is higher during the neonatal period in cases where the number of maternal alleles is more than 300 repetitions [8]. While the congenital form of CMD is observed in only 10% of the infants with less than 300 CTG repetitions, 60% of the infants of mothers with more than 300 repetitions were congenitally affected by the condition [8]. However, more than half of the affected mothers are not diagnosed with myotonic dystrophy or have no other complaints. As in our patient, diagnosing the infant with CMD is generally a factor leading to the diagnosis of the mother.

In infants whose mothers present with the clinical signs of a muscle disorder, the diagnosis can be achieved in a shorter period through genetic testing with no need for detailed tests. Genetic tests lead to certain diagnosis and are more cost-effective. The techniques such as electromyography (EMG) and muscle biopsy used to diagnose muscle disorders are insufficient in the diagnosis of CMD during the neonatal period. Although effective to demonstrate myopathic potentials, EMG is not useful because myotonic discharges are not observed during the neonatal period [2]. The assessment of muscle biopsy with different stainings is important in the diagnosis of CMD; however, these unconventional procedures are uncommon in most laboratories [9]. If muscle biopsy is evaluated properly, then the increase in the number of central nucleus, atrophy of type 1 bindings, clusters of pyknotic cells, and angular fibers can be observed.

Among the problems anticipated in the follow-up period of the patients with CMD, retardation in psychomotor development, growth retardation, nutritional deficiencies, and constipation are frequently observed [10]. Therefore, patients with CMD should be followed up by other training departments.

In conclusion, CMD can be diagnosed at an early stage in infants with the help of detailed familial histories. Investigating the mother with reference to the diagnosis of the infant can give clues as to the prognosis of the condition. With this report, we aimed to emphasize the importance of observation and history as a cost-effective diagnostic procedure; more-recently developed techniques or technologies are not necessarily required or effective.

Competing interests

The authors declare that they have no competing interests.

References

1. Campbell C. Congenital myotonic dystrophy. J Neurol Neurophysiol 2012; S7:1.
2. Bass N, LotzeT.E, Miller G. Hypotonia and Neuromuscular Disease in the Neonate in Fanaroff and Martin's Neonatal-Perinatal Medicine. 10th ed. Saunders Elsevier; Philadephia 2015; 950-63.
3. Ho G, Cardamone M, Farrar M. Congenital and childhood myotonic dystrophy: Current aspects of disease and future directions. World J Clin Pediatr 2015;4: 66–80.
4. Thornton C.A. Myotonic Dystrophy. Neurol Clin 2014; 32:705-19.
5. Volpe JJ. Neuromuscular disorders: Muscle involvement and restricted disorders. In: Neurology of the Newborn. 5th ed. Saunders Elsevier; Philadephia 2008. p. 801.
6. Nigro G, Papa AA, Politano L. The heart and cardiac pacing in Steinert disease. Acta Myol 2012;31(2):110–6.
7. Esplin MS, Hallam S, Farrington PF, Nelson L, Byrne J, Ward K. Myotonicdystrophy is a significantcause of idiopathicpoly- hydramnios. Am J Obs Gynecol 1998; 179: 974-7.
8. Cobo AM, Poza JJ, Martorell L, Lopez de Munain A, Emparanza JI, Baiget M. Contribution of molecular analyses to the estimation of the risk of congenital myotonic dystrophy. J Med Genet 1995;32:105–8.
9. Campbell C, Levin S, Siu VM, et al. Congenital myotonic dystrophy: Canadian population-based surveillance study. J Pediatr 2013; 163:120-5.
10. Domingues S, Alves Pereira C, Machado A, Pereira S, Machado L, Fraga C, Oliveira A, Vale I, Quelhas I. Congenital myotonic dystrophy in a Neonatal Intensive Care Unit: caseseries. Arch Argent Pediatr 2014;112:18-22.

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Case Report
JCAM

A Rare Case in Pediatric Neurology: Complex Regional Pain Syndrome

Pediyatrik Nörolojide Nadir Bir Olgu: Kompleks Bölgesel Ağrı Sendromu

Complex Regional Pain Syndrome in Child

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Öz

Kompleks bölgesel ağrı sendromu (KBAS) duyusal, trofik ve motor anormallikler ile nitelenen çok etmenli bir ağrı sendromudur. Tip 1 KBAS'ta bir sinir hasarı saptanmazken, tip 2 KBAS'a belirlenebilen bir sinir hasarı eşlik eder. Çocukluk ve adölesan çağında nadir olduğu düşünülmesine karşın, son 20 yılda daha iyi tanımlanmaya başlamıştır. Ancak, erişkin ve çocukluk çağı KBAS klinik bulgularının farklılığı, özel laboratuar testlerin ve görüntüleme tekniklerinin bulunmayışı, tanıda bir yıla kadar gecikmeye neden olmaktadır. KBAS'ta erken tanı ve tedavi iyi prognoz ölçütü olduğundan, hastalığın klinisyenler tarafından farkındalığı önemlidir. Bu yazıda, sağ bacakta ağrı, renk değişikliği ve yürüme güçlüğü ile başvurup, KBAS tip 1 tanısı alan, rehabilitasyon ve pregabalin tedavisine çok iyi yanıt veren 11 yaşında bir erkek hasta sunulmuştur.

Anahtar Kelimeler

Tip 1 Kompleks Bölgesel Ağrı Sendromu; Çocuk; Livedo Retikülaris; Ekstremite Ağrısı

Abstract

Complex regional pain syndrome (CRPS) is characterized by multifactorial pain disorder in combination with sensory, autonomic, trophic and motor abnormalities. CRPS type 1 refers to cases in which no specific nerve injury is identified, while type 2 are cases accompanied by identifiable nerve damage. Once considered to be a rare disorder among children and adolescents, CRPS has become better recognized over the past two decades. The clinical differences between adult and pediatric CRPS and the lack of specific laboratory tests and imaging techniques cause a delay in diagnosis of up to one year. Awareness of the syndrome is very important for the early diagnosis and treatment and is a positive prognostic factor. Here we present an 11-year-old male patient with pain, color changes of the skin and motor deficit in the right leg diagnosed with CRPS type 1. The patient responded well to rehabilitation and pregabalin treatment.

Keywords

Complex Regional Pain Syndrome Type 1; Child; Livedo Reticularis; Extremity Pain

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Introduction

Complex regional pain syndrome (CRPS), formerly known as reflex sympathetic dystrophy, is a chronic neuropathic pain syndrome that is believed to be a result of a dysfunction in the central or peripheral nervous system. It is characterized by persistent burning pain and vasomotor changes. Generally only one extremity is involved; however, any part of the body may be affected [1]. CRPS type 1 occurs without a definable nerve lesion, whereas type 2 is associated with a definable nerve lesion usually secondary to a trauma [2]. Patients might describe allodynia (non-painful stimuli evoking pain), hyperalgesia (painful stimuli evoking more intense pain than usual) and hyperpathia (repeated painful stimuli causing exaggerated response) [2]. Here, we present an 11-year-old male patient diagnosed with CRPS type 1 who was referred to our pediatric neurology clinic with difficulty in walking, pain and color changes of the skin of his right leg.

Case Report

An 11-year-old male patient was admitted with pain, color changes and weakness in the right leg. The color change had first appeared 5-6 months previously. It had grown in size and was accompanied by an intense pain. Weakness and difficulty in walking were his more recent complaints. He stated that during a bath, the intensity of pain increased in contact with hot and cold water and the blue discoloration of the skin became darker. Sometimes he felt cold in the region of the color change. His mother stated that he is generally shy and he never wants to go to the school, and that this intense pain recently affected his school attendance negatively.

Results of a neurological examination were normal. Livedo reticularis was noticed in the region of the pain (Figure 1). Laboratory investigations including complete blood count, renal and hepatic function tests, electrolytes, C-reactive protein, and creatine kinase levels were all normal. Viral serology was negative. Screening for vasculitis revealed negative results. Doppler ultrasonography for deep venous thrombosis results were normal. Electroneuromyography did not show any pathological changes. All cranial, spinal and right femur magnetic resonance imaging (MRI) were reported to be normal.

The diagnosis of CRPS type 1 was established according to the modified Budapest International Association for the Study of Pain (IASP) criteria [1]. The patient was consulted with the Physical Medicine and Rehabilitation (PM&R) Department. Treatment with paracetamol and codeine (addition of peripheral and central analgesic effect), vitamin C, and 75 mg pregabaline twice daily were started. He was given stretching and muscle-strengthening exercises because of weakness, atrophy and shortness in the right quadriceps muscle. Muscle-strengthening exercises for the other muscle groups of the lower extremity were also started because of weakness related to immobilization. His complaints decreased markedly after two weeks of treatment. The intense pain of his right leg reoccurred just once, when his grandfather, who had come from his village for a visit, returned home. Fluoxetine was started and his complaints subsided gradually within a few weeks of treatment. Monthly control visits for 6 months revealed that school attendance had improved.

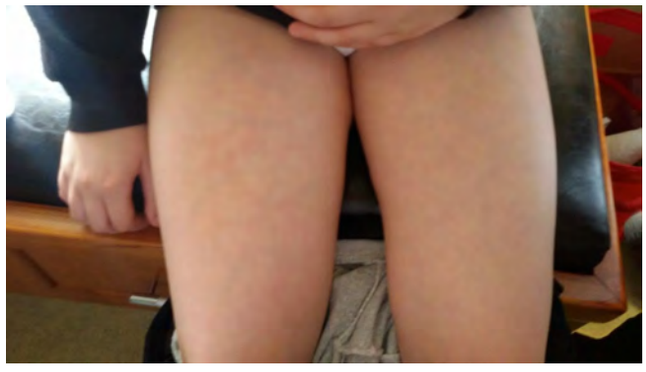

Figure 1. Livedo reticularis on the right thigh of the patient.

Discussion

In childhood, CRPS type 1 is a rare condition causing diagnostic challenges [3,4]. It is more common in school-aged children and mainly adolescent females, with a mean age at onset of 11-12 years [5]. Compared with adults, pediatric patients with CRPS type 1 have a higher incidence of lower extremity involvement and almost half of the patients have no history of trauma preceding the onset of symptoms [5]. Tan et al. [6], in a study of 78 children under age 16 with CRPS type 1 seen between 1980-2014, found that in children CRPS type 1 is less likely to be associated with edema and skin temperature is mostly cold, whereas edema and warm skin temperature are more commonly observed in adults with CRPS type 1. Psychogenic factors and anxiety are thought to play a major role in the development of CRPS, especially in childhood [7]. In contrast to the female preponderance described in the literature, our case was an 11-year-old male. Lower extremity involvement, lack of an initiating trauma, cooler feeling in the region of the color change, and lack of edema were features consistent with the literature.

The pathophysiology is not completely understood; however, current opinion is that activation of cutaneous nociceptors as a result of tissue damage stimulates unmyelinated C fibers and A delta afferents, leading to a neurogenic inflammation. Sympathetic pain has a major role in the early periods of the disorder. Peripheral sensitization occurs with the release of cytokines as a result of neurogenic inflammation and the release of algesic neuropeptides such as substance P. Central sensitization follows as a result of activation of N-methyl-D-aspartate (NMDA) receptors and altered functions at the level of the dorsal root ganglion [2].

The diagnosis of CRPS type 1 is clinical and a high level of vigilance is needed to make the diagnosis. Average delays in diagnosis of up to a year have been reported in the 1990's [8]. More recent studies have shown marked improvement with an average delay of about 3 months, which still is not ideal [5]. The differential diagnoses include inflammatory arthritis, cellulitis, osteomyelitis, deep venous thrombosis, malignancies and chronic vascular abnormalities [2].

The treatment of CRPS type 1 is multidisciplinary. Rehabilitation is the primary mode of treatment. The basic principles involve posture, range of motion, muscle stretching and strengthening exercises. The aim of the therapy is the prevention of immobilization caused by the persistent pain. Psychotherapy and psychiatric evaluation is a part of the treatment in child-

hood [5]. Nonsteroidal anti-inflammatory drugs, central analgesics, amitriptyline, pregabalin, gabapentin, corticosteroids and vitamin C can be used for pain management. In childhood, the response rate to conservative treatment is good, ranging from 70 to 90 %; however, recurrences have been reported in 30-50 % of cases [5,6]. Low et al. [5] reported that in children who were diagnosed early in the disease course (< 3 months), symptom resolution occurred much more rapidly than in those diagnosed later.

In conclusion, although rare, CRPS type 1 should be considered in the differential diagnosis of extremity pain. Being familiar with the symptoms and signs of the disorder makes early diagnosis and intervention possible and are positive prognostic factors.

Competing interests

The authors declare that they have no competing interests.

References

1. Marinus J, Moseley GL, Birklein F, Maihöfner C, Kingery WS, van Hilten JJ. Clinical features and pathophysiology of complex regional pain syndrome. Lancet 2011;10:637-48.
2. Ofluoğlu D, Akyüz G. Kompleks bölgesel ağrı sendromu tip 1: genel klinik yaklaşım. Türk Fiz Tıp Rehab Derg 2007;54:112-5.
3. Ayvaz A, İçağasıoğlu FD. Reflex sympathetic Dystrophy in children. J Clin Anal Med 2011;5(6):521-3.
4. Saltık S, Sözen HG, Basgul S, Karatoprak EY, Içağasıoğlu A. Pregabalin Treatment of a Patient With Complex Regional Pain Syndrome. Pediatr Neurol 2016;54:88-90.
5. Low KA, Ward K, Wines AP. Pediatric complex regional pain syndrome. J Pediatr Ortop 2007;27(5):567-72.
6. Tan EC, Zijlstra B, Essink ML, Goris RJ, Severijnen RS. Complex regional pain syndrome type 1 in children. Acta Paediatr 2008; 97: 875-9.
7. Borchers AT, Gershwin ME. Complex regional pain syndrome: A comprehensive and critical review. Autoimmun Rev 2014;13:242-65.
8. Wilder R, Berde C, Wolohan M, Vieyra M, Masek B, Micheli L. Reflex sympathetic dystrophy in children. Clinical characteristics and follow-up of seventy patients. J Bone Joint Surg 1992;74:910-9.

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A Case of Behcet Disease Characterized by the Appearance of a Mass in the Lungs

Akciğerde Kitle Görünümlü Behçet Hastalığı Vakası

Behcet Disease Mass in the Lung

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Öz

42 yaşında erkek hasta on gündür devam eden sağ omuz ve göğüs ağrısı, öksürük hemoptizi şikayetiyle hastaneye başvurdu. Hastanı titremekle yükselen 38°C ateşi vardı. Laboratuvar incelemesinde; lökosit:21.1 K/uL(91.4 % nötrofil), trombosit:564 K/uL, hemoglobin:13.8 g/dl, C-reactive protein: 8.61 ve eritrosit sedimentation hızı :104 mm/s olarak tespit edldi.Hastanın fizik muayenesi doğaldı. Öz geçmişinde 30 paket/yıl sigara kullanımı ve yedi yıldır Behçet Hastalığı mevcuttu. Pa akciğer grafisinde sağ üst paratrakeal alanda dansite artışı mevcuttu. Çekilen toraks BT'de sağ akciğer üst lobda 55x34 mm ebadında lobule konturlu kitle lezyon görüldü. Hastaya bronkoskopi yapıldı fakat endobronşial lezyon ya da kanama odağı görülmedi. Transtorasik ince iğne aspirasyon biyopsisiyle kitle lezyondan biyopsi alındı. Patoloji sonucu nekrotizan vasculit olarak rapor edildi. Bu sonuç Behçet hastalığının akciğer tutulumu ile uyumluydu. Tedavi olarak immunsupresif tedavi başlandı. 2 ay sonra çekilen kontrol toraks bilgisayar tomografide lezyonun kaybolduğu görüldü.

Anahtar Kelimeler

Behçet Hastalığı; Nekrotizan Vaskülit; Hemoptizi

Abstract

A 42-year-old male patient was admitted to the hospital with a cough, hemoptysis, and complaints of pain in his right shoulder and chest for 10 days. The patient had intermittent fever 38°C with chills. Laboratory results were: leucocytes:21.1 K/uL, platelets:564 K/uL, hemoglobin:13.8 g/dl, C-reactive protein: 8.61, and erythrocyte sedimentation rate: 104 mm/h. Physical examination was normal. The patient had a history of smoking 30 packets/year and Behcet disease (BD) for seven years. Chest radiography showed an increased density on the right paratracheal area. Thorax computed tomography (TCT) revealed a lobulated mass lesion 55×34 mm on the upper lobe of the right lung. Bronchoscopy was performed but neither a mass nor a bleeding focus was detected. A CT-guided transthoracic fine-needle aspiration biopsy was performed. The pathological examination was reported as "necrotizing vasculitis." These results were compatible with pulmonary involvement of the BD. Immunosuppressive therapy was initiated; after 2 months, a control TCT was within normal limits.

Keywords

Behçet Disease; Necrotizing Vasculitis; Hemoptysis

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Introduction

Bechet's disease is a chronic inflammatory disorder of unknown etiology characterized by recurrent attacks. It was first described by Hulusi Behçet in 1937. Except for mucocutanous lesions, BD is a multisystem disorder affecting the musculoskeletal system, the vascular system, the gastrointestinal tract, the lungs, and the eyes [1,2]. Pulmonary parenchymal involvement of BD is not common. In the present case, we discuss the challenges in the stage of diagnosis of BD.

Case Report

A 42-year-old male patient was admitted to the hospital with a cough and hemoptysis and complaints of pain in his right shoulder and chest; the patient's symptoms had persisted for nearly 10 days. The patient reported intermittent fever with chills; his fever measured in the clinic was 38°C. Laboratory results were as follows: leucocytes: 21.1 K/uL (91.4% neutrophils), platelets:564 K/uL, hemoglobin:13.8 g/dl, C-reactive protein: 8.61, and erythrocyte sedimentation rate: 104 mm/h. Biochemical tests were normal.

Upon physical examination, bilateral hemithorax was equally active in breathing; rale and rhonchus were not heard. The patient's other system examinations were normal. The patient had a history of smoking 30 packets of cigarettes per year, and he was still an active smoker. The patient had been diagnosed with Behcet disease (BD) for nearly seven years. On the basis of the history that we obtained, we learned that the patient did not receive checkups on a regular basis because of his BD, and he did not receive regular treatments for his disease. We consulted the patient with the dermatology clinic. The dermatology consultation report stated that the patient had a history of recurrent oral and genital ulcers and that the previous pathergy test was positive. The upper zone of the right paratracheal area revealed an increase in density in a chest radiograph (Figure 1). Empirical antibiotic treatment was initiated. The patient underwent a CT scan of his thorax because of complaints of hemoptysis and his history of smoking. We observed a lobulated mass lesion 55×34 mm in the posterior segment of the upper lobe of the right lung in the pleural neighborhood (Figure 2a-b). We accordingly performed a bronchoscopy; neither a mass nor a bleeding focus was detected. A CT-guided transthoracic fine-needle aspiration biopsy was performed. Necrotizing vasculitis signs were revealed in a pathological examination of the obtained material. Because of the patient's BD diagnosis, these findings were believed to be consistent with pulmonary involvement of the disease. Immunosuppressive therapy was initiated; after 2 months of treatment, a control thorax CT was within normal limits (Figure 3).

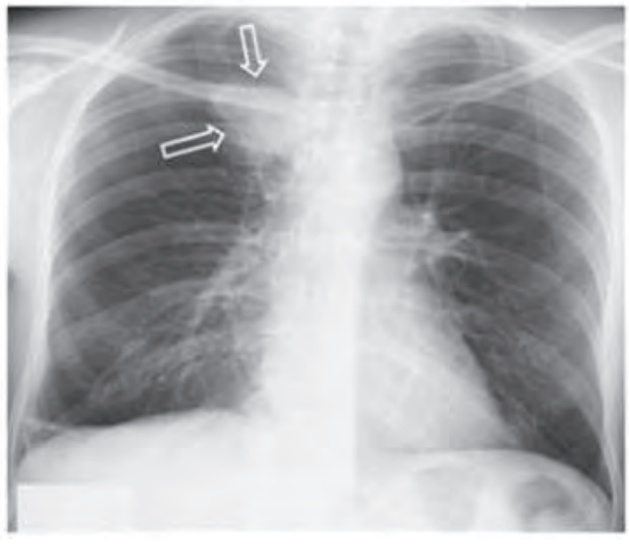

Figure 1. Posterior anterior chest graphy showed right paratracheal mass lesion (white arrows).

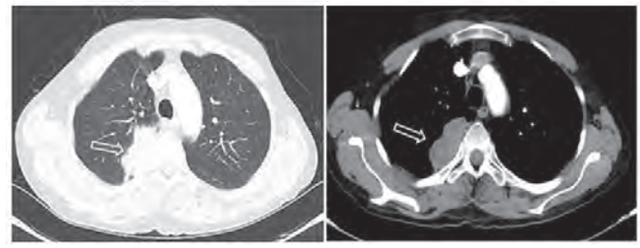

Figure 2. Thorax computed tomography images showed a lobulated mass lesion 55×34 mm in diameter at the posterior segment of the upper lobe of the right lung (white arrows).

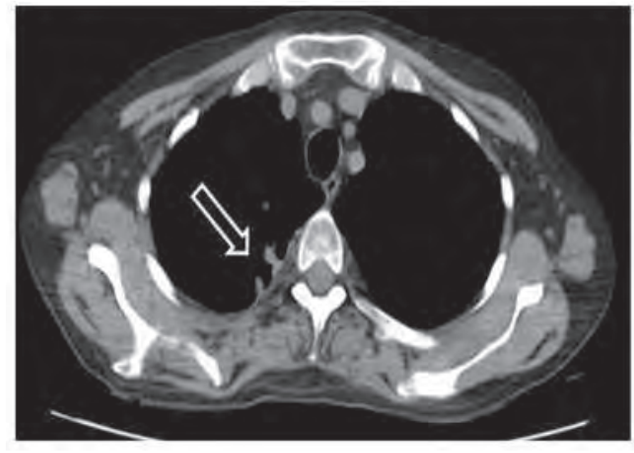

Figure 3. Control thorax computed tomography image was normal after medical treatment (white arrow).

Discussion

Pulmonary involvement has been reported in 1.0-7.7% of all patients with BD [2]. Pulmonary artery aneurysms, arterial and venous thrombosis, pulmonary infarction, recurrent pneumonia, bronchiolitis obliterans organized pneumonia, pleural effusion, and mediastinal lymphadenopathy are the primary features of pulmonary involvement in BD [2,3]. Patients exhibit symptoms such as episodes of dyspnea, cough, chest pain, fever, phlegm, and hemoptysis [3,4]. Hemoptysis, the most frequent symptom in BD with pulmonary involvement, is life-threatening. In this case, the patient's most important reason for admission was hemoptysis. A conventional chest radiography is the most common diagnostic method used to evaluate pulmonary involvement and in follow-up treatment [3,5]. The apparent pulmonary artery, the oligemic area resulting from perfusion defects (which occur due to the occlusion of the pulmonary artery branches), and the appearance of hilar and perihilar masses depending on aneurysms can be detected in chest radiography [6]. We did not observe chest X-ray findings indicative of pulmonary artery pathology. High-resolution CT data are useful for distinguishing parenchymal lesions resulting from pulmonary artery aneurysms, infarct, and pulmonary artery thrombus from other le-

sions. Spiral CT angiography is the best radiological method for evaluating pulmonary problems. In a contrast-enhanced thorax CT of the patient, we observed a 55 mm ×34 mm lobulated mass lesion in the posterior segment of the right upper lobe in the pleural neighborhood. Because of the characteristics of the lesion and the patient's history of smoking, we performed a bronchoscopy to look for malignancy. Pathology was not detected in the bronchoscopy, and we performed a transthoracic CT-guided biopsy. Histopathological examination of biopsies or surgical specimens is another useful method for diagnosing BD [3]. In the biopsy specimen taken from the patient, we found necrotizing vasculitis.

In the literature, pulmonary involvement in BD is typically in the form of pulmonary artery aneurysms and pulmonary vascular lesions; parenchymal involvement has rarely been reported [5]. Pulmonary parenchymal involvement, on the other hand, shows the activation of the disease [6]. In this case, pulmonary parenchyma was normal, and there was no evidence of pulmonary vascular involvement in the patient's contrast-enhanced thorax CT. Considering that the patient is a current smoker, the upper lobe lesion in the posterior segment was interpreted as an indicator of malignancy.

Anti-inflammatory and/or immunosuppressive drugs are used for the treatment of BD based on the severity of disease [2]. Medical treatments consisting of corticosteroids and cyclophosphamide have been found to be successful [2,4]. Our patient had been diagnosed with BD but had not received regular treatment for his condition. Immunosuppressive therapy was initiated. Upon follow-up, we found that the patient's clinical and radiological findings had improved.

Conclusion

Bechet's disease is common in Turkey, and pulmonary involvement is a rare form of pulmonary vascular involvement. Pulmonary parenchymal involvement is even rarer. Since hemoptysis may be the first symptom, BD should be kept in mind, and relevant research should be conducted when a patient with hemoptysis arrives at the hospital without exhibiting additional symptoms. In this case, the patient's history of smoking and the detection of a lesion in the thorax CT had been interpreted as an indicator of possible malignancy, but the biopsy results revealed vasculitis based on the histopathological examination, which was an unexpected finding.

Competing interests

The authors declare that they have no competing interests.

References

1. Hiller N, Lieberman S, Chajek-Shaul T, Bar-Ziv J, Shaham D. Thoracic manifestations of Behçet disease at CT. Radiographics 2004;24(3):801-8.
2. Erkan F, Gül A, Tasali E. Pulmonary manifestations of Behçet's disease. Thorax 2001;56(7):572-8.
3. Malekmohammad M, Emamifar A. Pulmonary Nodules as an Initial Manifestation of Behçet's Disease. Case Rep Rheumatol 2014;2014:869817.
4. Serir Aktoğ , Onur Fevzi Erer, Gülcan Ürpek, Ömer Soy, Gültekin Tibet. Behçet Hastalğında Multipl Pulmoner Arter Anevrizmaları: Siklofosfamid ve Kortikosteroid Tedavisinden Sonra Klinik ve Radyolojik Remisyon. Toraks Dergisi 2001;2(2):35-8.
5. Vydyula R, Allred C, Huartado M, Mina B. Surgical lung biopsy to diagnose Behcet's vasculitis with adult respiratory distress syndrome. Lung India 2014;31(4):387-9.
6. Gülbay BE, Kaya A, Acıcan T, Gülbay M. Radyooji Dersleri 3: Behçet Hastalığında Akciğer Tutulumu. Tüberküloz ve Toraks Dergisi 2001; 49(3): 412-6.

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Bronchiectasis Due to a Hidden Underlying Cause; Look to See

Altta Yatan Gizli Bir Nedene Bağlı Bronşektazi; Görmek İçin Bakın

Bronchiectasis Due to a Rare Cause

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Bu çalışma, TÜSAD 36. Ulusal Kongresi – Solunum 2014, 15-19 Ekim 2014, İzmir'de elektronik poster olarak sunulmuştur.

Öz

Bronşektazi, bronşların kalıcı dilatasyonu ve akciğerin fibrozisi ile karekterize kronik bir akciğer hastalığıdır. Değişik etiyolojik faktörlere ikincil olarak geliştiği için, cerrahi kararı vermeden önce dikkatli bir değerlendirme çok önemlidir. Bu sunuda bronşektazinin nadir bir nedeni sunulmuş olup, bu hastalarda bronkoskopinin öneminin altı çizilmiştir.

Anahtar Kelimeler

Bronşiyal Hastalık; Bronkoskopi; Fistül (Trakeoözofajial)

Abstract

Bronchiectasis is a chronic lung disease that is characterized by permanent dilatation of the bronchi and fibrosis of the lung. Because it is secondary to different etiological factors a careful evaluation is crucial before surgical decision making. Herein a rare cause of bronchiectasis is reported, underlining the importance of bronchoscopy for these patients.

Keywords

Bronchial Disease; Bronchoscopy; Fistula (Tracheoesophageal)

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Introduction

Bronchiectasis is a chronic lung disease that is characterized by permanent dilatation of the bronchi and fibrosis of the lung [1]. Although many patients seem to have no associated disease leading to the development of bronchiectasis, there are many conditions, such as infections, alfa 1 antitrypsin deficiency, cystic fibrosis, immotile cilia syndrome, etc., that have been recognized as causing bronchiectasis. Other than these, a rare cause of bronchiectasis, tracheoesophageal fistula (TEF), discussed in this case report should be kept in mind.

Case Report

A 24 year-old-male, who was followed due to chronic and recurrent infections with the diagnosis of bronchiectasis, was referred to our department for the surgical management of bronchiectasis. He complained of large amounts of purulent sputum. Thorax computed tomography (CT) demonstrated cystic bronchiectasis at the middle lobe and basal segments of the right lower lobe (Figure 1a). Fiberoptic bronchoscopy, performed for a preoperative evaluation, pointed out an orifice at the posterior wall of the trachea leading us to think of a tractus such as a TEF (Figure 1b). Esophagoscopy and esophagography did not identify a fistula. When thorax CT was reevaluated retrospectively, the radiological finding was much more visible (Figure 1c). Therefore a surgical intervention was planned based on this underlying primary pathology (TEF). An oblique incision was performed from the left side of the neck. The fistula was demonstrated revealed with the guidance of intraoperative bronchoscopy (Figure 2a,b) and repair was done followed by a sternohyoid muscle flap transposition (Figure 2c). The postoperative period was uneventful. After the treatment of TEF, the primary pathological process, the patient remains under follow-up for a secondary residual disease.

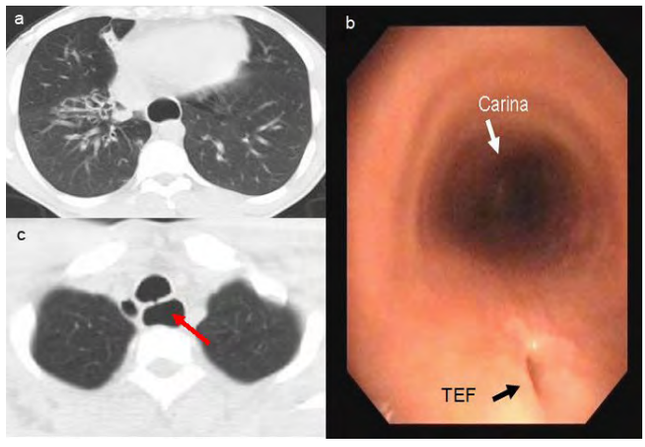

Figure 1. a. Thorax computed tomography section showing bronchiectasis at the middle and lower lobes of the lung. b. Bronchoscopic view of the orifice at the posterior wall of trachea, considered a TEF. c. Computed tomography section supporting our consideration of TEF.

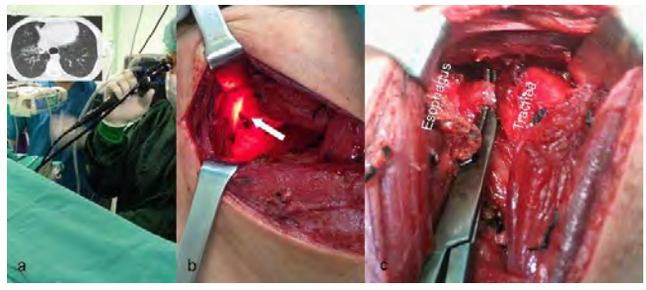

Figure 2. a. Intraoperative bronchoscopy was used as a guide to determine the orifice of the fistula. b. White arrow showing the illuminated trachea. c. Intraoperative view of the abnormal tractus between the esophagus and trachea.

Discussion

Bronchiectasis is a chronic, destructive lung disease that is frequently secondary to infective diseases. The indications for surgical resection are localized disease, failure of medical treatment, sufficient respiratory and cardiac reserve, frequent infections, presence of a chronic cough and hemoptysis, deterioration of the quality of life, a destroyed lobe, and sequelae [1,2]. But as in our case, if there is an underlying chronic primary pathology a surgical intervention may result in disaster. Therefore it is important to make a careful examination to fully investigate etiological factors before deciding on surgical management.

Bronchoscopic evaluation of bronchiectasis patients is necessary for the aspiration of secretion, to determine probable infective agents, and to inspect endobronchial masses or foreign bodies [3]. In this case, determining the orifice of occult TEF in adulthood saved the patient from a parenchymal loss and possible inoperability in the future.

The diagnosis of TEF in adults can be difficult and often delayed. A high level of suspicion is necessary for the diagnosis. The various endoscopic and radiological techniques are not entirely reliable in identifying a fistula [4]. Diagnosis is mostly done by esophagography-esophagoscopy. However, in small orifices, clinical evaluation and bronchoscopic findings should also be prominently considered, in the light of the contribution of this reported case.

The current study indicated that, for bronchiectasis patients, precise preoperative investigation and planning are important to identify even very rare causes before rendering an irreversible decision such as resective surgery. This will protect patients as well as pulmonologists and thoracic surgeons from potential disaster.

Competing interests

The authors declare that they have no competing interests.

References

1. Miller JI. Bacterial infections of the lungs and bronchial compressive disorders. In: Shields T, Lo Cicero J, Ponn RB, editors. General Thoracic Surgery. 5th ed. Philadephia, USA: Lippincott Williams & Wilkins; 2000. p.1039-52.
2. Çobanoğlu U , Sayır F, Sertoğullarından B. Surgical Treatment of Hemoptysis: Analysis of 17 Cases. J Clin Anal Med 2012;3(2):198-202.
3. Gursoy S, Ozturk AA, Ucvet A, Erbaycu AE. Surgical management of bronchiectasis: the indications and outcomes. Surg Today 2010;40(1):26-30.
4. Ng J, Antao B, Bartram J, Raghavan A, Shawis R. Diagnostic difficulties in the management of h-type tracheoesophageal fistula. Acta Radiol 2006;47:801-5.

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Case Report
JCAM

Surgical Treatment of Acute Mediastinitis Due to Fish Bone

Balık Kılçığına Bağlı Gelişen Akut Mediastinitin Cerrahi Tedavisi

Acute Mediastinitis

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Öz

Akut mediastinit ciddi bir durumdur ve çabuk tedavi edilmelidir. 66 yaşında kadın hasta ateş ve konfüzyonla acil servise başvurdu. Öyküsünde 11 gün önce balık yedikten sonra öksürdüğü ve hastaneye başvurmadan bir gün önce bir balık kılçığının ağzına geri geldiği öğrenildi. Toraks bilgisayarlı tomografi(BT) görüntülemesinde mediastinal alanlarda hava ve yüksek dansiteli sıvı koleksiyonları mevcuttu. Mediastinit tanısı ile hastaneye yatırıldı. Operasyon öncesinde özofagusu incelemek için endoskopi yapıldı ve özofagusta küçük bir nekrotize alan görüldü. Preoperatif hazırlık yapıldıktan sonra hasta ameliyata alındı ve abse drene edildi. Postoperatif 35.günde yoğun bir tedaviden sonra taburcu edildi.

Anahtar Kelimeler

Mediastinit; Kılçık; Cerrahi Tedavi

Abstract

Acute mediastinitis is a serious condition that needs to be treated quickly. A 66-year-old female patient was admitted to the emergency department with fever and confusion. In her medical history she said that she coughed after eating fish 11 days earlier and a fish bone returned to her mouth one day before the admission to hospital. Her thorax CT scan showed air and high-density liquid collections in the mediastinal compartments. She was hospitalized with a diagnosis of mediastinitis. An endoscopy performed before the operation to examine the esophagus revealed a small necrotized area. The patient underwent surgery and the abscess was debrided after preoperative preparation. She was discharged on the postoperative 35th day after an intensive course of treatment.

Keywords

Mediastinitis; Fish Bone; Surgical Treatment

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Introduction

Ingestion of a foreign body into the esophagus is common in children but rarely seen in adults. Acute mediastinitis due to esophageal foreign bodies is a life-threatening condition with a mortality of 17-25% [1,2]. Early diagnosis and treatment is life-saving for patients. Medical and surgical treatment may be applied. Patients usually refer to hospital with fever, tachycardia, and chest pain, and blood tests show infection associated with an abscess. Computerized tomography (CT) should be used when mediastinitis is clinically suspected.

Case Report

A 66-year-old female patient was admitted to the emergency department with fever and confusion. In her history she said she coughed after eating fish 11 days earlier. She had been admitted to hospital two times before. A laryngoscopy was performed but no foreign body was seen. A fish bone returned to her mouth one day before this admission to hospital. Physical examination noted decreased breath sounds on the right side. Laboratory results were as follows: white blood cell count 23,700/mm3 (reference: 4,000-10,000), neutrophil dominance 85% (reference: 37-73), hemoglobin 9.6g/dL (reference:12.1-17.2), C-reactive protein (CRP) >160mg/dL (reference: 0-10), erythrocyte sedimentation rate (ESR) 28mm/h. X-ray revealed air-fluid levels at the mediastinum (Figure 1) and a thorax CT scan showed air and high density liquid collections in mediastinal compartments (Figure 2). After the evaluation of these findings the patient was hospitalized with a diagnosis of mediastinitis, oral feeding was discontinued, and sulbactam/cefoperazone sodium was begun before surgery. Endoscopy was performed to examine the esophagus and a 2 mm necrotized area was seen at 15 cm from incisor; no foreign body was seen in the esophagus. Posterolateral thoracotomy was performed through the 4th intercostal space and the lung was adherent to the chest wall on examination. An abscess located in the posterior mediastinum was drained between superior of vena azygos and inferior pulmonary artery. Isotonic saline with antibiotics was used for the thoracic lavage. A Foley catheter was placed in the space remaining after removal of the posterior mediastinum abscess (Figure 3). We used effective antibiotics and antifungal drugs in the thoracic lavage for the microbiological culture. Surgery wasn't planned for the esophagus because the perforated area in the esophagus was too small for surgery and healed spontaneously after the fish bone was ejected. Atrial fibrilation after the surgery, probably unrelated to the operation, was treated with metoprolol. Total parenteral nutrition began on the second day and the necrotized area was fully healed on the re-evaluating endoscopy at the 25th day. Oral feeding started after the endoscopy. The patient was discharged on the 35th day and she was healthy at the 6 month follow-up.

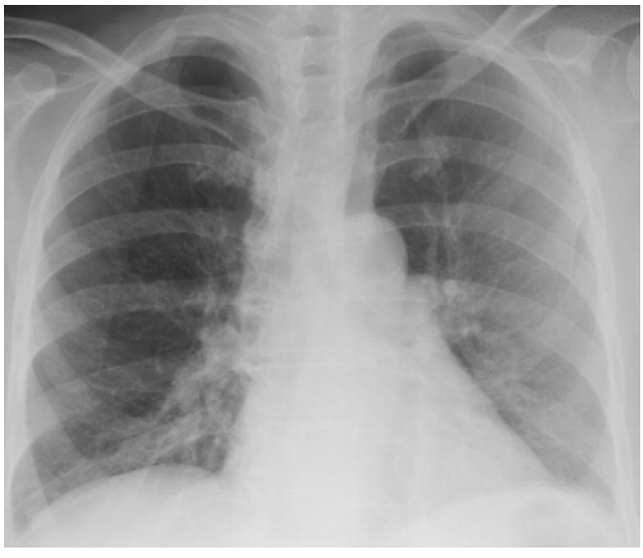

Figure 1. X-ray revealed air-fluid levels at mediastinum

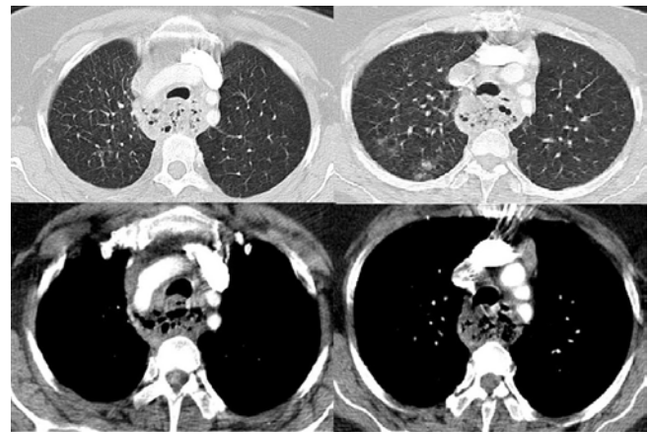

Figure 2. Thorax CT scan showed air and high-density liquid collections in mediastinal compartments.

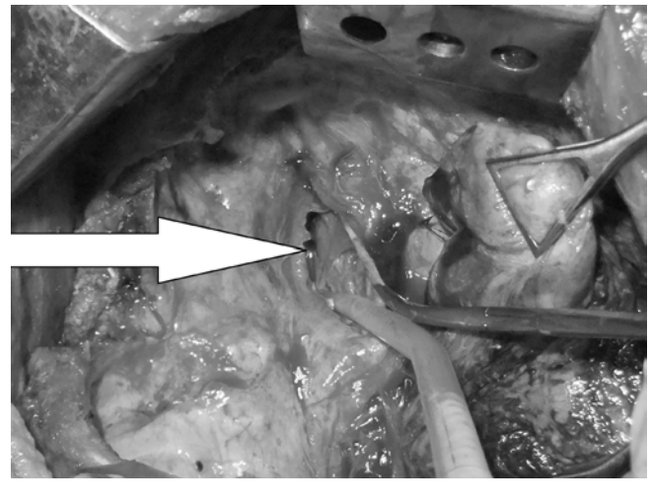

Figure 3. Foley catheter placed into pouch.

Discussion

Swallowing of foreign bodies is common throughout people's lives, but most of the foreign bodies are removed through the stool without causing any problems. An endoscopic or surgical removal is required in only 10–20% of the cases [5]. Pointed materials such as chicken bones, fish bones, and toothpicks most often cause gastrointestinal system perforation [3]. Swallowed fish bones are the most common cause of intestinal perforation associated with foreign bodies in Hong Kong [4] because of their pointed sharp tips and long bodies [4]. Because most of the fish bones are invisible on x-ray due to their size, clinical history should be investigated in detail. Only 32% of ingested fishbones can be identified radiographically [8] Many different imaging techniques can be used for foreign bodies. When there is clinical suspicion of a foreign body, bi-directional x-ray images should first be taken along with a thorax CT scan

if necessary. Metallic foreign bodies can be seen easily on x-ray. Air and high-density liquid collections in mediastinal compartments may be indicative of mediastinitis. There is a long list of serious complications as a consequence of esophageal foreign bodies, including perforation, retropharyngeal abscess, mediastinitis, and fistulas [6]. The mortality rates are as high as 50% as a result of subsequent intrathoracic infection [7]. Conservative or surgery are the treatment modalities for mediastinitis. If there is clinical evidence of infection and collections of liquid in the mediastinal compartment upon scanning, surgery is a good choice for treatment. We recommend thoracic lavage with antibiotics after an effective surgery. Necrotic tissue, bacteria, and their toxins must be drained from the mediastinum. On the other hand, total parenteral nutrition and wide-spectrum antibiotics may be used for the medical treatment of mediasitinitis.

Conclusion

Our case emphasizes the danger of esophageal perforations and delayed diagnosis of mediastinitis caused by foreign body ingestion. A thorough history obtained from the patient and timely imaging techniques will lead physicians to the correct diagnosis early. Posterolateral thoracotomy can be a good approach to drain the abscess.

Competing interests

The authors declare that they have no competing interests.

References

1.Vidarsdottir H,Blondal S, Alfredsson H, Geirsson A, Gudbjartsson T. Oesophageal perforations in Iceland: A whole population study on incidence, aetiology and surgical outcome. Thorac Cardiovasc Surg 2010;58:476–80.
2. Markar SR, Mackenzie H, Wiggins T, Askari A, Faiz O, Zaninotto G, et al.Management and outcomes of esophageal perforation: A national study of 2,564 patients in England. Am J Gastroenterol 2015;110:1559–66.
3. Chu KM, Choi HK, Tuen HH, Law SY, Branicki Fj, Wong J. A prospective randomized trial comparing the use of the flexible gastroscope versus the bronchoscope in the management of foreign body ingestion. Gastrointest Endosc 1998; 47: 23-7.
4. Goh BK, Tan YM, Lin SE, Chow PK, Cheah FK, Ooi LL, et al. CT in the preoperative diagnosis of fish bone perforation of the gastrointestinal tract. AJR Am J Roentgenol 2006; 187: 710-4.
5. Ginsberg G.G. Management of ingested foreign objects and food bolus impactions. Gastrointest Endosc 1995;41(1):33–8.
6. Loh KS. Complications of foreign bodies in the esophagus. Otolaryngol. Head. Neck Surg 2000;123(5):613–6.
7. Balasubramaniam SK. A review of the current management of impacted foreign bodies in the oesophagus in adults. Eur Arch Otorhinolaryngol 2008;265(8):951–6.
8. Ngan JH, Fok PJ, Lai EC, Branicki FJ, Wong J. A prospective study on fish bone ingestion. Experience of 358 patients. Ann Surg 1990;211:459-62.

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Thoracic Aortic Injury Due to Thoracoscopic Sympathectomy: A Case Report

Torakoskopik Sempatektomiye Bağlı Torasik Aort Yaralanması

Aortic Injury Due to Sympathectomy

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Öz

Torakoskopik sempatektomi palmar ve aksiller hiperhidrozun tedavisinde kullanılan bir yöntemdir. Bu teknik daha az ağrıya ve erken işe dönmeye olanak sağlar.39 yaşında erkek hasta palmar ve aksiller hiperhidroz ile hastaneye başvurdu. Hastamıza çift taraflı torakoskopik sempatektomi uyguladık. Aort yaralanmasına bağlı masif hemotoraks gelişti ve intraoperatif tedavi edildi.

Anahtar Kelimeler

Aort Yaralanması; Torakoskopik Sempatektomi; Hemotoraks

Abstract

Thoracoscopic sympathectomy is a procedure for the treatment of palmar and axillary hyperhidrosis. This technique offers less pain and an earlier return to work than other surgical treatments. A 39-year-old male was admitted to hospital with palmar and axillary hyperhidrosis. We performed thoracoscopic sympathectomy bilaterally. Massive haemothorax due to aortic injury occured and was treated intraoperatively.

Keywords

Aortic Injury; Thoracoscopic Sympathectomy; Haemothorax

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Introduction

Hyperhidrosis is a common disorder characterized by abnormally increased sweating that affects many people, with a prevalence of about 0.5%. This condition is associated with psychological, emotional, and social status. Video-assisted thoracoscopic sympathectomy is a new procedure for the treatment of palmar and axillary hyperhidrosis. This technique has become popular because of its low morbidity and mortality rates, and the benefits that minimally invasive surgery offers to patients [1]. The most common side effect is compensatory hyperhidrosis and major complications are rare. We present a male patient with palmar and axillary hyperhidrosis who experienced an aortic injury during the thoracoscopic sympathectomy operation.

Case Report

A 39-year-old male patient was admitted to our hospital with palmar and axillary hyperhidrosis His medical history included a coronary angiography performed due to chest pain 4 years earlier and showed no abnormalities. A thoracoscopic right T3-T4 sympathectomy was performed with two 5mm incisions over the axillary hairline and a left T3-T4 sympathectomy was performed subsequently in Semi-Fowler's position under general anesthesia. The patient woke up often during the surgery. Approximately 2 mm of injury due to cauterization with the hook occured in the descending aorta at the T4 level and bleeding occurred during the left sympathectomy. Anterolateral thoracotomy was immediately performed. A total of 2000cc of hematoma and blood drained. The aorta was explored and repaired with 4/0 prolene suture. Three suspensions of erythrocytes were transfused during the surgery and the patient was extubated in the operation room. He stayed in the intensive care unit for two days. A thorax CT performed during his hospitalization did not show any other aortic injury. He was discharged after a 5-day hospital stay.

Discussion

Most people do not consider hyperhidrosis a disease, but emotional palmar-axillary wetness affects lives both socially and professionally. Although the pathophysiology is unclear, its believed to be caused by hyper-stimulation or over-activity of the sympathetic nervous system that passes through the upper thoracic ganglia [2]. There are several forms of treatment for hyperhidrosis. Medical treatment includes anti-perspirants, anti-cholinergic drugs, iontophoresis, and botulinum toxin injections. Local excision of the sweat glands and thoracoscopic sympathectomy are the surgical treatment options. Endoscopic transthoracic sympathectomy is a minimally invasive procedure with several advantages over open surgery in the treatment of palmar and axillary hyperhidrosis [3]. Different surgical techniques can be used for the endoscopic transthoracic sympathectomy such as ablation, resection, and interrupting the thoracic sympathetic chain by clips. In a study by Garcia and Espana, intervention on the T3-4 ganglia is identified as the most successful method for combined palmar and plantar hyperhidrosis [4]. We also prefer this approach for palmar and plantar hyperhidrosis because of the high rate of patient satisfaction. In our experience, orotracheal intubation with a double-lumen endotracheal tube is the best choice for intra-operative ventilation. The surgeon needs adequate and continuous relaxation to prevent injuries during the surgery. The most common side effect of this method is compensatory sweating due to thermoregulatory mechanisms [5]. Pneumothorax, haemothorax, chylothorax, pleural effusion, lung injury, Horner's Syndrome, nasal obstruction, rhinitis, wound infection, and intercostal neuralgia may be seen intra- and peri-operatively. Cameron has reported a case of subclavian artery injury that required 34 units of blood transfusion [6]. In a prospective study by Dominique, one case with a tear of the subclavian artery and two cases of chylothoraces resolved without sequelae [7]. Patients should clearly be warned of the side-effects and complications of thoracoscopic sympathectomy before surgery.

Conclusion

Our case is the first massive injury case during thoracoscopic sympathectomy in the literature. Appropriate patient selection and adequate anesthesia management are important to avoid complications of hyperhidrosis.

Competing interests

The authors declare that they have no competing interests.

References

1.Kauffman P, Cinelli M, Wolosker M, Leao LE. Treatment of palmar hyperhidrosis by cervico-thoracic sympathectomy. AMB Rev Assoc Med Bras 1978; 249(1): 29-30.

2.Drott C, Gothberg G, Claes G. Endoscopic transthoracic sympathectomy: an efficient and safe method for the treatment of hyperhidrosis. J Am Acad Dermatol 1995; 33: 78-81.

3.Byrne J, Walsh TN, Hederman WP. Endoscopic transthoracic electrocautery of the sympathetic chain for palmar and axillaryy hyperhidrosis. Br J Surg 1990; 77:1046-9.

4.Garcia F, Espania A. Usefulness of bilateral sympathectomy using video-assisted thorascopic surgery in the treatment of essential hyperhidrosis. ActasDermosifiliogr 2008; 99: 523-711.

5.Chou SH, Kao EL, Lin CC, Chang YT, Huang MF. The importance of classification in sympathetic surgery and proposed mechanism for compensatory hyperhidrosis: experience with 464 cases. Surg Endosc 2006; 20(11): 1749-53.

6.Cameron AE. Complications of endoscopic sympathectomy. Eur J Surg 1998; 164: 33-5.

7.Gossot D, Kabiri H, Caliandro R. Early complications of thoracic endoscopic sympathectomy: A prospective study of 940 procedures. Ann Thorac Surg 2001;71:1116-9.

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Is this Massive Lumbar Disc Herniation? What is the Treatment?

Bu Görünüm Masif Lomber Disk Hernisi Midir? Tedavisi Nedir?

Massive Lumbar Disc Herniation

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To the editor:

A 24-year-old woman was admitted with low back pain radiating to right leg. She had been complaining of mild low back pain for 3 years. On physical examination there were no neurological deficits except for slight hypoesthesia of the right L5 dermatome and mildly painful lumbar spine movements. The severity of pain was at level 5 on the Visual Analogue Scale (VAS). We treated conservatively using nonsteroidal anti-inflammatory drugs, hot applications and organized activities of daily living lasting for a week. We performed a lumbar MRI (magnetic resonance imaging) scan because there were no changes in the clinical findings of the patient. The pathological evaluation was proposed by the radiologist for differential diagnosis because of the appearance of a pathological mass or a giant mass of extruded disc herniation at the L5-S1 intervertebral disc level and a compressed right S1 nerve root (Fig.1). Two surgeries, a right-L5 laminectomy and a L5-S1 microdiscectomy, were performed after consultation with the neurosurgery clinic. Pathological examination showed that the mass was part of the herniated disc fragment. Postoperative pain (VAS:3) decreased and the patient was followed under conservative treatment with monthly controls. During this period, the clinical findings of the patient did not change. After postoperative 4 months, lumbar MRI showed that the right paramedian extruded disc herniations at the L5-S1 disc level were smaller than in the previous image (Fig 2).

There are four stages in the development of a herniated disc, from disc bulging to sequestration. There is no correlation between the clinical findings and the size of the hernia [1]. Moreover, there is not yet a consensus on which method of treatment should be chosen in which stage of lumbar disc herniation (LDH) [2,3]. In recent years, because MRI scanners have become easily accessible, it may sometimes be decided to proceed with surgery according to the size of the disc herniation, ignoring the patient's clinical status [2,3]. However, better regression resultimg from conservative management of large disc herniation has been reported [3]. Therefore, it is argued that the patient should not be rushed to surgery based on the size of the hernia material on a MRI [2]. A massive LDH might resemble pathologic masses such as schwannoma or meningiomas [4]. In this case, consulation with radiologist for differential diagnosis is essential. In this way, confirmation of a pathologic diagnosis in cases where a massive LDH can not be distinguished from a tumor, as in our case, will be accurate.

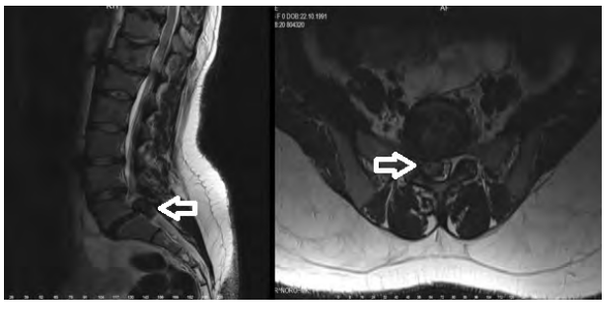

Figure 1. Sagittal and transvers T2-weighted MRI of the lumbar spine shows giant mass of extruded disc herniation that was at the L5-S1 intervertebral disc level and compressed right S1 nerve root or pathological mass

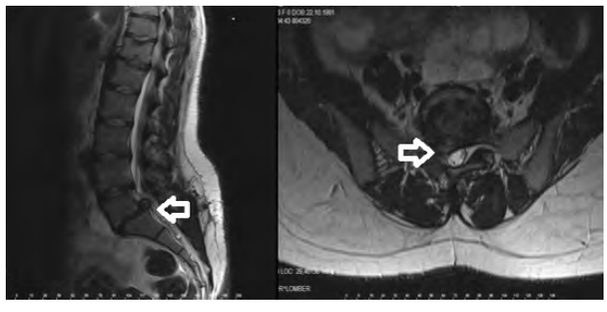

Figure 2. Sagittal and transvers T2-weighted lumbar MRI (postoperative) showed that right paramedian extruded disc herniations are smaller than in the previous image in the L5-S1 disc level

References

1. Jeon CH, Chung NS, Son KH, Lee HS. Massive lumbar disc herniation with complete dural sac stenosis. Indian J Orthop 2013;47:244-9.
2. Ocak FDM, Karaaslan M, Tutar I, Konuralp N. Evaluation of the efficacy of conservative therapies with clinical parameters and magnetic resonance imaging in lumbar disc herniations. Turk J Phys Med Rehab 2007; 53:108-12.
3. Benson RT, Tavares SP, Robertson SC, Sharp R, Marshall RW. Conservatively treated massive prolapsed discs: a 7-year follow-up. Ann R Coll Surg Engl 2010;92:147-53.
4. Ellenberg M, Ilechukwu I. Gadolinium-enhanced disc lesion. Am J Phys Med Rehabil 2004; 83:550.

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Ultrasound-Guided Selective Ulnar Nerve Block at the Level of Mid-Forearm for Outpatient Procedure

Günübirlik Cerrahide Orta Önkol Seviyesinden Yapılan Ultrasound Eşliğinde Selektif Ulnar Sinir Bloğu

Ultrasound Guided Ulnar Nerve Block

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To the editor:

Post-operative pain and delayed discharge are common problems for patients undergoing outpatient procedures [1]. Insufficient pain management, prolonged motor block, and opioid-related nausea and vomiting are the main causes of delayed discharge, resulting in not being able to accept new patients [2]. Ultrasound-guided regional anesthesia may be an ideal technique for providing optimal analgesia and reducing opioid use [3]. The use of ultrasound enables visualization of nerves and the observation of the needle tip and spread of the injected local anesthetic.

In this report, discuss ultrasound-guided selective ulnar nerve block at mid-arm level in a patient who did not agree to receive general anesthesia and was not satisfied with the previous regional anesthesia experience due to development of complete motor block.

A 21-year-old female patient was admitted to the orthopedics outpatient clinic due to swelling and pain in the 5th finger of the left hand. It was decided that she would undergo surgical excision with the pre-diagnosis of exocytosis (Figure 1a-b). On her pre-anesthetic visit one day before the operation, she did not have a known disease. Explanations regarding general anesthesia and regional anesthesia options were provided. It was planned that she would undergo the operation with USG-guided ulnar block because she wished to receive regional anesthesia and did not agree to a motor block in her arm.

The left arm was marked for ulnar nerve block under ultrasound guidance and covered; 2 mg of midazolam was administered intravenously. The ulnar nerve was visualized neighboring the ulnar artery with the Esaote My Lab 30 USG device (Figure 2). Nerve tracing was followed and the mid-level of the forearm was reached. 1% lidocaine was applied subcutaneously for skin infiltration. 4 ml of 0.5% bupivacaine and 4 ml of 2% lidocaine mixture were administered to the inferior and posterior parts of the nerve under USG guidance using 50 mm of USG-visible needle with in-plane technique. Nerve stimulator was not used because the ulnar nerve was visualized easily. The sensory block was checked with the pinprick test 20

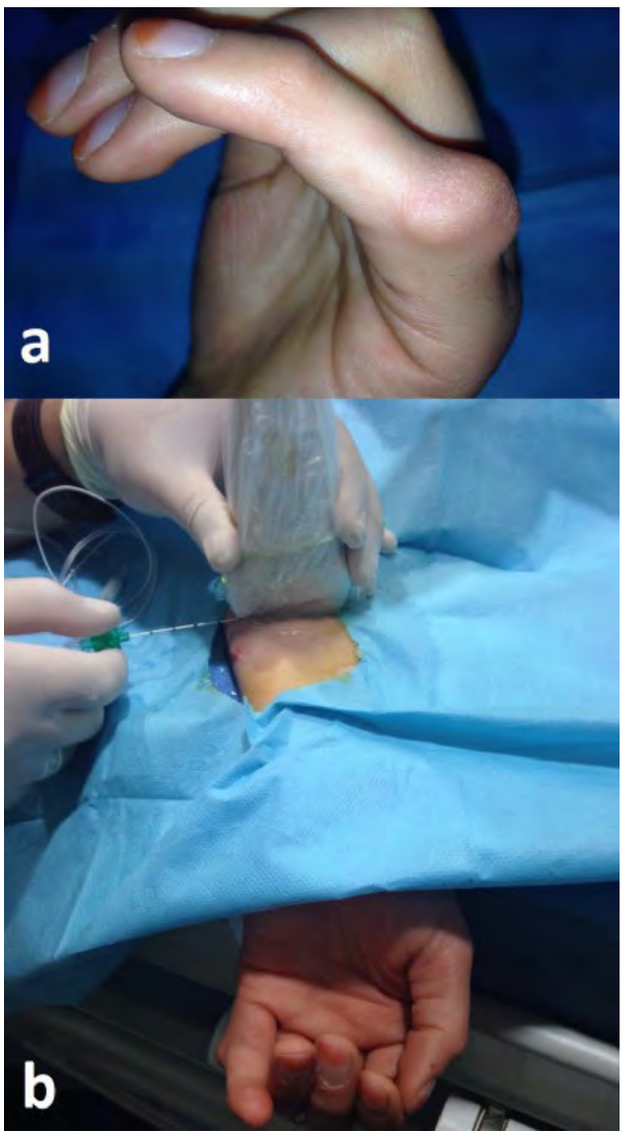

Figure 1. Left hand 5th finger exocytosis (a) Ultrasound probe and needle position(b)

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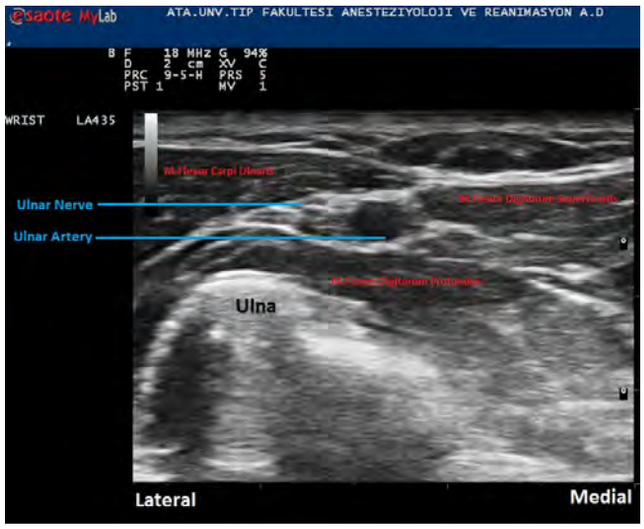

Figure 2. Ultrasound image of ulnar nerve

minutes later and the operation commenced. The patient did not need sedation or additional analgesia during the procedure. The patient comfortably tolerated the operation, which took 45 minutes, under isolated ulnar nerve block. She was discharged on the same day without loss of post-operative motor function. The post-operative analgesia time was 185 minutes.

Brachial plexus blocks may be an appropriate option for patients for whom the use of a tourniquet for forearm surgery is planned, because these blocks include whole nerves. Use of short-acting local anesthetic drugs cannot sufficiently relieve post-operative pain, particularly following bone surgery [4]. In the study of McCartney et al., the time of the first analgesic need was determined as 97 minutes following 1.5% lidocaine administration to the brachial plexus [5]. Alternatively, use of long-acting local anesthetic leads to prolonged motor block and an unpleasant feeling such as a dead arm after discharge [6]. Anesthetists may be reluctant to discharge after prolonged peripheral nerve blocks. We preferred ulnar nerve block at mid-arm level as the most appropriate option as it does not cause motor block and provides sufficient anesthesia. Isolated nerve block becomes an appropriate option for hand surgery in patients for whom a tourniquet would not be used.

These blocks should be applied from distal as possible for preventing motor block and providing anesthesia only in the surgical field [7]. We followed the ulnar nerve under USG guidance at the wrist level and performed the nerve block procedure at mid-arm level. While the patient had a long post-operative analgesia period through long-acting local anesthetic, no loss of motor function occurred.

In conclusion, USG-guided selective nerve blocks may be an appropriate alternative option in outpatient hand surgery procedures in which a tourniquet would not be used, as it provides anesthesia only in the operative field, does not cause motor block, enables early discharge, provides sufficient post-operative analgesia, and enables avoidance of general anesthesia.

Competing interests

The authors declare that they have no competing interests

References

1. Rawal N. Postdischarge complications and rehabilitation after ambulatory surgery. Curr Opin Anaesthesiol 2008;21(6):736-42.
2. Rodgers J, Cunningham K, Fitzgerald K, et al. Opioid consumption following outpatient upper extremity surgery. J Hand Surg Am 2012;37(4):645-50.
3. Fingerman M. Regional anesthesia for outpatient hand surgery: ultrasound-guided peripheral nerve block. J Hand Surg Am 2011;36(3):532-4.
4. Kuntz F, Bouaziz H, Bur MD, et al. Comparison between 1.5% lidocaine with adrenaline and 1.5 % plain mepivacaine in axillary brachial plexus block. Ann Fr Anesth Reanim 2001;20(8):693-8.
5. McCartney CJ, Brull R, Katz J, et al. Early but no long-term benefit of regional compared with general anesthesia for ambulatory hand surgery. Anesthesiology 2004;101(2):461-7.
6. Klein SM, Buckenmaier CC III. Ambulatory surgery with long acting regional anesthesia. Minerva Anestesiol 2002;68(11):833-41.
7. Dufeu, Nicolas et al. Efficacy and Safety of Ultrasound-Guided Distal Blocks for Analgesia Without Motor Blockade After Ambulatory Hand Surgery. Journal of Hand Surgery 2014;39(4):737-43.

Original Image
JCAM

Bilateral Accessory Breast Induced By Pregnancy

Gebelikle İndüklenen Biletarel Aksesuar Meme Dokusu

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Accessory breast tissue is a normal embryonic residue which can be found on the milk line between the axillary and the inguinal region. In women accessory breast tissue is usually observed during lactating and pregnancy. Based on the tissue region and size they may not require treatment. They may vanish on their own but also cause serious pain and cosmetic problems [1, 2].

On her 6th day after c/s on her first pregnancy, 28 year old female patient presented with pain in her chest and axillary region. Physical examination revealed soft, hairy and mobile masses with oedema. Masses did not contain nipples or areolas (Figure 1). History did not reveal any diseases or drug use. After the ultrasonograhphic assesment masses were diagnosed as accessory breasts. Two weeks of symptomatic treatment relieved patients pain and the ectopic breast image dissappeared.

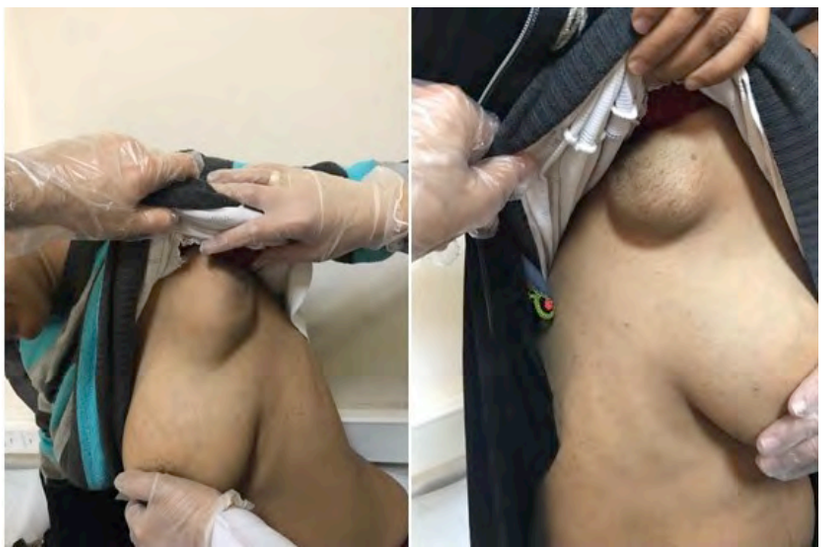

Figure 1. Left (A) and right (B) axillary region

References

1. Özdamar MY, Atabey F, Soyuer I. Adolesan bir kızda aksesuar meme: nadir bir aksiller kitle olgusu. Bozok Tıp Derg 2014;4(2):71-4
2. Grama F, Voiculescu Ş, Vîrga E, Burcoş T, Cristian D. Bilateral axillary accessory breast tissue revealed by pregnancy. Chirurgia (Bucur) 2016; 111(6):527-31.

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