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Relation between Severity of the Disease and Arrhythmia Incidence in Patients with Psoriasis

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Aim: The aim of this study is to investigate relation between severity of the disease and heart rate variability, diastolic dysfunction and arrhythmia in patients with psoriasis vulgaris.
Patients and Method: Fifty- three psoriasis patients (28 F, 25 M), 30 healthy volunteer were enrolled in the study. Severity of the psoriasis disease was evaluated by Psoriasis Area and Severity Index (PASI) score. The patients who have PASI score $\leq$10 were groupped as mild psoriasis and the patients who have PASI score >10 were grouped as severe psoriasis. Patients with hypertension, diabetes mellitus, thyroid disease, chronic liver disease, chronic renal failure, valvular heart disease, congestive heart failure, coronary artery disease, cerebrovascular event history and patients who have history of medical treatment which can lead to arrhythmia were excluded. BUN, creatinine, AST, ALT, fT3, fT4, TSH, fasting blood glucose, total cholesterol, trygliseride, HDL cholesterol, LDL cholesterol, TNF-α and Hs-CRP were tested in all patients. All patients were evaluated with transthorasic echocardiography. Twenty-four-hour ambulatory electrocardiogram holter monitoring was performed.
Results: Diastolic blood pressure measurements of patients with psoriasis were significantly higher than control group (p:0.001). HDL cholesterol values in both mild and severe psoriasis groups were significantly lower than the control group (p<0.05). Hs-CRP level in severe psoriasis group was significantly higher than the other groups (p:0.001). Left ventricular end diastolic diameter, end systolic diameter, LVEF, septum and posterior wall thickness', left atrium diameter measurements were similar among the groups.Mean standard deviation of mean of normal-to-normalin intervals (SDANN) which one of the parameters of the heart rate variability was in mild psoriasis group 130.1$\pm$39.2 msec, in severe psoriasis group 118.4$\pm$39.9 msec and in control group 126.7$\pm$39.3 msec (p:0.012). Frequency of supraventricular ectopy and ventricular ectopy are similar among the groups. Serious conduction defects, supraventricular tachycardia and ventricular tachycardia were not determined in 24-h holter monitoring.
Conclusıon: HDL cholesterol level in patients with psoriasis was lower than control group.Mean SDANN mean, mean and night percentage of R-R intervals with more than 50 msec variation (PNN50), mean and night root mean square of differences between adjacent normal-normal intervals (RMSSD) which one of the parameters of the heart rate variability were lower in severe psoriasis group. As previous studies showed, reduced heart rate variability is a risk factor for cardiovascular morbidity and total mortality. Increased cardiovascular risk may be related with those findings in patient with psoriasis.In this study, our data suggests possible explanation for high incidence of cardiovascular disease in psoriasis patients and need to further prospective clinical trials.

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The Role of (-675G/A) 4G/5G Polymorphism of the Plasminogen Activator Inhibitor-1 Gene on Atrial Fibrillation with Ischemic Stroke in Turkish Population

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Background: Plasminogen activator inhibitor type 1 (PAI-1) regulates fibrinolysis, and the common promoter region variants -675G/A (4G/5G) is associated with increased thrombotic risk. High levels of PAI-1 have been implicated as a risk factor for cardiovascular disease. The 4G allele is associated with higher levels of PAI-1, and might increase the risk for intravascular thrombosis. Atrial fibrillation (AF) renders individual patients at risk for development of an atrial thrombus. AF increases the risk of stroke by 4-5-fold, particularly in the elderly. Several pathophysiological mechanisms of the left atrial thrombogenesis have been suggested. Therefore, genetic variations affecting expression levels and activity of haemostatic factors could presumably modulate the risk of thrombogenesis in AF. We investigated the PAI-1 4G/5G (-675G/A) polymorphisms in nonvalvular AF with Ischemic Stroke.
Methods: The PAI-1 4G/5G polymorphisms were analysed in 70 patients with nonvalvuler AF who have had a stroke and 70 healthy individuals with no documented episode of AF matched for age, race and sex. The PAI-1 4G/5G polymorphisms were identified by polymerase chain reaction (PCR) method. Distribution of the PAI-1 4G/5G polymorphism genotypes (Normal (5G/5G) genotype, heterozygous mutant genotype (4G/5G) or homozygous (4G/4G) mutant genotype) were determined in study population. Demographic characteristics and risk factors for AF and stroke were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. There was no statistical difference in genotype distribution among the groups. The genotype distribution in nonvalvuler AF who have had a stroke group was as follows: 5G/5G genotype frequency was 29 (41.4%), 5G/4G genotype frequency was 27 (38.6%) and 4G/4G genotype was 14 (20%). The genotype distribution in control group was as follows: 5G/5G genotype frequency was 37 (52.9%), 4G/5G genotype frequency was 23 (32.9%) and 4G/4G genotype frequency was 10 (14.3%). There was no statistically significant difference between groups in genotype distributions.
Conclusıons: Our results suggest that the 4G/5G polymorphism of the PAI-1 gene appears not to be associated with nonvalvular AF with ischemic stroke in Turkish population.

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Necrotizing Cutaneous Vasculitis in a Patient with Infective Endocarditis and Severe Rheumatoid Arthritis

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Acute or subacute bacterial endocarditis should be suspected in any patient with a pacemaker manifesting a persistent febrile illness. Embolic complications of IE can occur as vasculitis, and vasculitis is also a serious complication that develops in a minority of patients with longstanding, severe, seropositive, erosive, and nodular rheumatoid arthritis (RA).

A 68-year-old woman with fever, asthenia, night sweats, arthralgia, weight loss, and necrotizing skin ulcers on the body and lower extremities (Figure 1a) was admitted to our clinic. She had been followed for RA for over 20 years. The laboratory findings revealed granulocytosis, anaemia of chronic infection, high levels of CRP, and sedimentation rate, and low levels of C4. The rheumatoid factor (RF) was 929 IU/ml (N<10), and anti-cyclic citrullinated peptide (antiCCP) was 56.5 U/ ml (N< 5) at admission. A month ago, a temporary pacemaker was applied to the patient due to drug-induced bradyarrhytmia, but the pacemaker's lead fractured and remained inside the right ventricle while it was removed. The two major criteria for the diagnosis of IE was confirmed by revealing vegetations on the fractured lead and the tricuspid valve by echocardiography (Figure 2a-b), and methicillin- resistant coagulase-negative Staphylococcus aureus in two of the four blood cultures drawn >12 h apart. Vancomycin and imipenem were commenced for the treatment of IE. The biopsy specimen from the skin ulcers revealed leucocytoclastic vasculitis (LCV) (Figure 3a). In cases with IE and LCV, a trend to low serum complement levels was evident.This was associated to an immune complex-mediated process initiated by antigen products of the infectious agent responsible for the IE, or to the result of abnormal immunoregulation related to the infectious disease.

Vasculitis is a serious complication of RA that develops in a minority of patients with this disease, and it is not uncommon in patients with IE. The diagnosis of rheumatoid vasculitis (RV) was confirmed by the history, clinical and laboratory findings of our patient. Methylprednisolon 500 mg intravenous bolus followed by 40mg peroral daily was administered. In spite of all taken measures the patient died of severe sepsis on the 50th day of her admission.

Rheumatoid vasculitis usually develops in people with at least 10 years of severe disease when the inflammatory arthritis is 'burned out. High titers of RF are reported to be the strongest predictor of the development of RV and antiCCP antibodies may be helpful in distinguishing RV from other forms of small vessel vasculitis. Numerous medications used to treat RA have also been proposed as triggers of RV, partly because of some similarities between drug-induced hypersensitivity vasculitis and RV. In particular, the treatment of RA with oral glucocorticoids has been implicated in the development of RV. Our patient had been RA for over 20 years and on corticosteroid therapy of different dose regimens since then.