PP-102
Relation between Severity of the Disease and Arrhythmia Incidence in Patients with Psoriasis
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Background: Psoriasis is one of the most common chronic dermatologic diseases. The pathogenesis of psoriasis is not completely understood. It has been suggested that there is an association between psoriasis and cardiovascular disease. However, the exact mechanism is unknown. In this study we aimed to investigate the relation between psoriasis and arrhythmia incidence.

Methods: Patients with psoriasis pathologically confirmed by skin biopsy were included in the study. At the time of diagnosis, patients were evaluated with 24h Holter monitoring and results were compared.

Results: 100 patients with moderate (PASI≤10) and severe psoriasis (PASI>10) were included in the study. The moderate psoriasis group consisted of 70 patients, the severe psoriasis group had 30 patients. The mean age of the patients was 39±14 years old and 61% of the patients were male. There was no difference in age and sex distribution between the groups. Moderate psoriasis group had 24% (17 patients) and severe psoriasis group had 50% (15 patients) of arrhythmia incidence. The mean heart rate was 72±10 and 71±10 for moderate and severe psoriasis respectively. The heart rate variability was similar in both groups. Also, the mean QT interval and QTc interval were similar between the groups. The left atrium diameter measurements were similar between the groups as well. Only left ventricular wall thickness was significantly different between the groups (p<0.01). The left ventricular ejection fraction (LVEF) was higher in the moderate psoriasis group (p<0.01).

Conclusion: There was a positive correlation between the severity of psoriasis and the incidence of arrhythmia. Further prospective studies are needed in order to clarify this relation.

PP-103
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 1 (PAI-1) regulates fibrinolysis, and is involved in pathological conditions associated with cardiovascular diseases. Many studies have shown that PAI-1 polymorphisms may act as a risk factor for cardiovascular disease. However, the results have been inconsistent. The aim of our study was to investigate the role of (-675G/A) 4G/5G polymorphism on the risk of atrial fibrillation (AF) in patients with ischemic stroke.

Methods: One hundred and fifty-two patients with ischemic stroke and 150 healthy controls were included in the study. The PAI-1 4G/5G polymorphisms were analyzed in both groups by polymerase chain reaction (PCR) method. Distribution of the PAI-1 4G/5G polymorphism genotypes (Normal (5G/5G) genotype, heterozygous mutat genet (4G/5G) or homozygous (4G/4G) mutant genet) 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 the groups. There was no statistical difference in genotype distribution among the groups. The genotype distribution in nonvalvular AF who have had a stroke group was as follows: 5G/5G genotype frequency was 29 (41.4%), 4G/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 (32.9%), 4G/4G genotype frequency was 23 (27.3%) and 4G/4G genotype frequency was 10 (14.3%). There was no statistically significant difference between groups in genotype distributions.

Conclusions: 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.

PP-104
Necrotizing Cutaneous Vasculitis in a Patient with Infective Endocarditis and Severe Rheumatoid Arthritis
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A 68-year-old woman with fever, asthma, 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 EU/ml (N<10), and anti-cyclic citrullinated peptide (anti-CCP) was 56.5 U/ml (N<5) at admission. A month ago, a temporary pacemaker was applied to the patient due to drug-induced bradycardia, 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 leucocytoclasis (Figure 3a). In cases with IE and LCV, a trend to lower 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 longstanding, severe, seropositive, erosive, and nodular rheumatoid arthritis (RA). A 68-year-old woman with fever, asthma, 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 EU/ml (N<10), and anti-cyclic citrullinated peptide (anti-CCP) was 56.5 U/ml (N<5) at admission. A month ago, a temporary pacemaker was applied to the patient due to drug-induced bradycardia, 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 leucocytoclasis (LCV) (Figure 3a). In cases with IE and LCV, a trend to lower 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 arthritis (RA) is complicated by the inflammatory, 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 anti-CCP 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.