CARDIOVASCULAR RISK IN RHEUMATOID ARTHRITIS. CASE REPORT

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CARDIOVASCULAR RISK IN RHEUMATOID ARTHRITIS. CASE REPORT (Abstract):
We present the case of a 57-year-old rural patient diagnosed with stage 4 seropositive rheumatoid polyarthritis and Steinbrocker class III functional capacity at the age of 45; the patient was initially treated with DMARDs (methotrexate and leflunomide) and corticosteroids, discontinued due to the diagnosis of pulmonary fibrosis; he then received biological therapy with infliximab, discontinued following the detection of some axillary lymphadenopathies (complex workup confirming they were of inflammatory type in the context of the collagen disease), later switched

The diseases of the connective tissue are characterized by a complex multifactorial etiology and pathogenesis. Mortality and cardiovascular morbidity are higher in individuals with rheumatoid polyarthritis (RP) compared to the general population. Rheumatoid polyarthritis is an independent risk factor for cardiovascular disease. The degree of disease activity in RP is directly correlated with the cardiovascular risk (1). The early and aggressive treatment of RP reduces the rate of cardiovascular events. The association of therapy with statins and angiotensin converting enzyme inhibitors reduces the cardiovascular risk in patients with RP (2).

CASE REPORT
We present the case of a 57-year-old rural patient diagnosed with stage 4 seropositive rheumatoid polyarthritis and Steinbrocker class III functional capacity at the age of 45; the patient was initially treated with DMARDs (methotrexate and leflunomide) and corticosteroids, discontinued due to the diagnosis of pulmonary fibrosis; he then received biological therapy with infliximab, discontinued following the detection of some axillary lymphadenopathies (complex workup confirming they were of inflammatory type in the context of the collagen disease), later switched
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to rituximab, with therapeutic response and a decrease in disease activity scores. The patient presented to the hospital for dyspnea on light-moderate exertion and palpitations with onset approximately 24 hours earlier, with irregular, fast heartbeats following prolonged high-intensity physical activities. He associated the traditional risk factors for coronary disease – diabetes mellitus, dyslipidemia, obesity, arterial hypertension.

Physical examination showed: good general health, absence of fever, conscious, third-degree obesity (BMI=41.20 kg/m²), gynecomastia (fig. 1), lipodermatosclerosis, SpO₂=97% on room air, BP=160/100 mmHg, ventricular rate = 65 beats/minute, rhythmical heart noises, aortic atheromatosis, abdominal panniculus adiposus mobile with breathing, sensitive to palpation in the right-side hypochondrium, without signs of peritoneal irritation, hepatomegaly located 2 cm below the costal margin. Examination of the musculoskeletal system revealed: peripheral algofunctional syndrome (irreducible flexum of the left elbow at 10 degrees), „rheumatoid hand“ appearance with bilateral tumefaction of radiocubitocarpal joint and metacarpophalangeal joints II-III, bilateral swan neck deformity of the middle finger, bilateral Z-deformity of the thumb, radial deviation of the carpal bones and ulnar deviation of fingers, atrophy of the dorsal interosseous muscles, bilateral tumefaction of the knee with liparthritsis, without local warmth, cracking upon movement, discrete bilateral tumefaction of the tibiotalar joint, bilateral deforming arthrosis of the metatarsophalangeal joint of the big toe, „rheumatoid foot“ appearance.

Electrocardiogram findings: pauci symptomatic episodes of bradycardia – polymorphic extrasystolic ventricular arrhythmia expressed through doublets, ventricular tachycardia phases/beats (fig. 2). Bio humoral tests revealed hyperglycemia, hypercholesterolemia, hepatic cytolysis syndrome, absence of the inflammatory syndrome and myocardial cytolysis. Immunological assessment revealed the presence of the rheumatoid factor and elevated level of anti-cyclic citrullinated peptide antibody (250 IU). Urine test revealed proteinuria and chest X-ray the absence of progressive pleuropulmonary lesions, rectilinear left mid aortic arch, aortic knob calcification, right-hand side basal accentuated pulmonary sketch (fig. 3). The abdominal and pelvic ultrasound imaging confirmed the hepatic steatosis and pancreatic lipomatosis and did not describe other changes. The echocardiogram revealed ischemic heart disease with delayed relaxation type diastolic dysfunction and incipient degenerative aortic valve changes, ejection fraction = 52%, progressing on the background of a chronic immune-inflammatory status, to which a complicated diabetes mellitus is added.

Fig. 1. Third degree obesity
Amiodarone i.v. (I C Indication, European Heart Journal Guidelines 2015)

**Fig. 2.** Electrocardiogram evolution
According to the SCORE (Systematic COronary Risk Evaluation) charts which are used to assess the total death risk due to cardiovascular disease (coronary and non-coronary) in the last 10 years, the patient was at intermediate risk (3%) (3,4). Following the analysis of Framingham score parameters, which estimate the coronary cardiovascular accident risk in the following 10 years, the patient also showed an intermediate risk (14.5%) (3, 4).

The disease activity scores in collagen disease [DAS28 (Disease Activity Score28) = 3.68; SDAI (Simplified Disease Activity Index) = 14.9; CDAI (Clinical Disease Activity Index) = 14] indicated a moderately active disease with an average functional impact (HAQ (Health Assessment Questionnaire) = 1.75) and EULAR (European League Against Rheumatism) response = 0.51 (5).

The electrocardiographic evolution was slowly favorable under specific treatment, arrhythmia being interpreted within the context of silent myocardial ischemia of rheumatoid polyarthritis and diabetic neuropathy.

The prognosis is poor, not only due to the immediate complications of the associated pathology, but also to the distant complications which progress to locomotor disability, development of opportunistic infections or neoplasia because of the immunocompromised state.

**DISCUSSION**

We reported the case of an obese patient with diabetes, hypertension and dyslipidemia who associated an autoimmune disease – rheumatoid polyarthritis – (with higher incidence in women!), showed no response to DMARDs and anti-TNF (Tumor Necrosis Factor) antibodies, with cardiovascular risk score multiplied (x1.5) by the presence of the systemic disease, and who developed symptomatic episodes of tachycardia with important prognostic consequences.

The cardiovascular and rheumatic diseases can coexist (6, 7). The risk of cardiovascular events in rheumatoid polyarthritis is independent, yet amplified by the presence of traditional risk factors (8, 9). Endothelial dysfunction represents the initial link in the development of vascular complications and can be induced by the oxidative stress generated by hyperglycemia and/or insulin resistance (10, 11). The correct and early risk stratification is essential for guiding the treatment. The therapeutic scheme is guided by patient’s comorbidities (12).

In these patients, a careful post-discharge monitoring is necessary. Long-term monitoring must include lifestyle change, correction of risk factors and secondary prevention (13). The early recognition and aggressive therapeutic approach of rheumatic disease will reduce the risk of
cardiovascular disease and improve survival and quality of life (14, 15).

CONCLUSIONS
This case describes a patient with rheumatoid inflammatory disease and high cardiovascular risk. Rheumatoid arthritis and cardiovascular diseases share common pathophysiological mechanisms, which have as consequence the acceleration of process of the atero-forming process. Cardiovascular risk assessment and adoption of cardiovascular disease prevention strategy are integral part of the management of patients with rheumatoid arthritis. Patients with chronic immunoinflammatory RA require regular cardiac screening (clinical examination, electrocardiogram and, in certain conditions, echocardiography). The cardiovascular affection in connective tissue disease requires a dual treatment: of the underlying disease and of the cardiac affection itself according to the current specific guidelines.

REFERENCES