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MYOCARDITIS IN SYSTEMIC LUPUS ERYTHEMATOSUS: CASE REPORT WITH A COMPLEX ETIOLOGICAL DIFFERENTIAL DIAGNOSIS

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BACKGROUND

Systemic lupus erythematosus (SLE) is an autoimmune, inflammatory, chronic, disease of indefinite etiology, which is allied to the presence of autoantibodies responsible for a large number of clinical spectra. Cardiac involvement in SLE occurs in about 50% of the cases, being pericarditis the most frequent. Myocarditis occurs in 8-10% of patients and may be due to multiple causes, being the association with disease activity the most frequent, followed by ischemia due to atherosclerosis and valvular dysfunction.

CASE REPORT

Female patient, 46 years old, diagnosed with SLE since 2011. On 03.10.2019 she started dyspnea on minimal exertion, lower limb edema and acute non-invasive diarrhea, motivating her hospitalization on May of the same year. At physical examination, the patient was in serious general conditions, dyspneic, febrile (38,5ºC), acyanotic, anicteric, tachycardic (98bpm), cardiac auscultation: regular rhythm in 3 times (gallop), respiratory auscultation: bibasilar crackles. In addition, C3 and C4 consumption, and active urinalysis (cylindruric, erythrocytic dismorphism, hematuria, proteinuria of 1600mg/24 hours), despite treatment with mycophenolate 3g/day and prednisone 40mg/day. The echocardiogram showed an abrupt drop in left ventricular ejection fraction (LV) from 55% to 26%, with diffuse hypokinesia. To exclude coronary ischemic disease, electrocardiogram (sinus tachycardia), markers of myocardial necrosis - negative, and cineangiocoronariography were performed: no obstructive lesions. Excluding myocardial ischemia, the investigation was directed to myocarditis. The myocardial magnetic resonance perfusion imaging showed late epicardial enhancement to the gadolinium corroborating with acute inflammatory myocarditis. Serologies for coxsakie, herpes simplex, toxoplasmosis, cytomegalovirus, hepatitis B and C, HIV1/2 negative; lupus anticoagulant and anticardiolipin IgG (10U-GPL/ml), cervical vessel doppler with acute venous thrombosis in left internal jugular vein. A hypothyroidism with TSH 0.00mUI/L, T4L 3.55ng/dl and TSH 40UI/L anti-receptor antibody was observed, and then started therapy with tapazole and anticoagulation, evolving with an important improvement of the condition.

CONCLUSION

This report describes the difficulty in defining the cause of myocarditis in a patient with SLE, since it was in a clear disease activity, besides being diagnosed with antiphospholipid syndrome and hyperthyroidism, also described as causes of myocarditis. Because of the progressive improvement with the antithyroid, it is believed that the major component of myocarditis is of this etiology. The authors draw attention to the occurrence of comorbidities whose manifestations may overlap with those of SLE, hindering the etiological diagnosis and compromising the therapeutic decision.