systemic inflammation and endothelial dysfunction, which promotes accelerated atherosclerosis.

Objectives: Evaluate the frequency of atheromatous plaques in patients with systemic lupus erythematosus.

Methods: Observational, prospective, cross-sectional study. Carotid Doppler was performed on patients with SLE from the external consultation of the rheumatology service from November 2019 to 2020. Inclusion criteria: > 18 years old, diagnosis SLE with classification criteria ACR 2007, 2002, 2012. Carotid Doppler, measurement of IMT.

Results: 116 patients met inclusion criteria, including 116 female controls. Mean age was 62.3 years. 14.65% (17) had atheromatous plaques, 99.4% (12) calcified plaques (5). 34.7% Dyslipidemia (63.1%) (73), obesity 34.7% (3), high blood pressure 23% (22), diabetes 3.4% (4), smokers 0% (0). The activity rate using SLEDAI showed 66.9% (80) without activity, 13.79% (16) low, 11.20% (13) moderate, 8.03% (7) high activity. About control group (116), 19.82% (23) showed atheromatous plaques, 39.13% (9) calcified plaques.

Conclusion: Our study shows that less than one-quarter of patients have atheromatous plaques in the carotid Doppler. In relation to LES activity, the vast majority are in low activity. We suggest the realization of Carotid Doppler in patients with low activity SLE for evaluation and monitoring of cardiovascular risk. Our study showed that there is no increased risk of atheroma plaque formation in SLE patients, compared to the general population.

REFERENCES:

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AB0327 DRUG-INDUCED LUPUS ERYTHEMATOSUS SECONDARY TO ANTI-TNF-α AGENTS IN PATIENTS WITH SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS

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Objective: Induction of autoantibodies is frequently observed in patients treated with TNF-α antagonist and the possible development of drug-induced lupus erythematosus (DILE) remains a matter of concern. The prevalence of DILE secondary to anti-TNF-α therapy is estimated around 0.5-1% and clinical features include arthritis/arthralgia, rash, serositis, fever, myalgias, cytophenias, among others. According to the literature, DILE secondary to anti-TNF-α agents differs in several ways from the clinical and laboratory findings typically associated with classic DILE.

Objectives: To estimate the incidence of induction of antinuclear antibodies (ANA) and DILE in a monocentric cohort of patients with spondyloarthropathies and psoriatic arthritis treated with anti-TNF-α agents. To describe the clinical and laboratory features and outcomes of patients with DILE.

Methods: We performed a retrospective analysis of patients with spondyloarthropathies and psoriatic arthritis treated with anti-TNF-α agents, from our University Hospital, who have been registered on the Portuguese Rheumatic Diseases Register (Reuma.pt) between July 2001 and December 2020. Patients with
positive ANA (titer > 1/100) before the anti-TNF-α therapy were excluded. Because specific criteria for the diagnosis of DILE have not been established, we considered the diagnosis in case of a temporal relationship between clinical manifestations and anti-TNF-α treatment and fulfillment of ACR/EULAR 2019 classification criteria for SLE. In patients with DILE, clinical features, laboratory findings, systemic therapies and outcome after discontinuation of medication were collected from reuma.pt and medical records. For the clinical and demographic predictors, continuous variables were analyzed using a two-sided t-test and categorical variables using a Fisher’s exact test. P-value <0.05 was considered statistically significant.

Results: In the spondyloarthritis group, 290 patients were included (44.8% females, mean age at diagnosis of 33.3 ± 11.5 years and mean disease duration of 15.1 ± 10.4 years) and in the psoriatic arthritis group, 116 patients were included (50.0% females, mean age at diagnosis of 40.1 ± 11.0 years and mean disease duration of 13.1 ± 6.8 years). In our study, we observed high serology conversion rates (positive ANA in 67.9% and 58.8% of patients with Spondyloarthritis and Psoriatic Arthritis, respectively), with similar conversion rates between different anti-TNF drugs. Three patients with spondyloarthritis (10%) and 1 patient with psoriatic arthritis (0.9%) developed DILE. Etanercept was the causative agent in 2 cases, infliximab and adalimumab in 1 case each, except for peripheral arthritis (new onset or abrupt worsening) occurred in 2 patients, serositis in 1 patient, constitutional symptoms in 2 patients, subnephrotic proteinuria in 1 patient, lymphopnia in 2 patients and hypocomplementemia in 1 patient. Specific treatment was prescribed to the 4 patients (oral corticosteroids) and they achieved complete recovery. After anti–TNF-α treatment interruption, no patient had recurrent disease. We observed that patients with DILE had a significantly longer disease duration (> 8.4 years; p=0.04) and a significantly longer duration of therapy with anti-TNF (> 4.0 years; p=0.04) when compared to patients without DILE.

Conclusion: Despite the frequent induction of autoantibodies, the development of DILE secondary to anti–TNF-α agents is rare. Our study demonstrates an incidence rate similar to other studies reported before. The clinical and laboratory characteristics of our patients with DILE attributable to anti–TNF-α agents differ significantly from DILE due to more traditional agents, as is described in literature. Overall, patients in this study had mild disease that improved after therapy discontinuation, without recurrence of the disease. It seems that a longer disease duration and a longer period under anti-TNF-α therapy may increase the risk of DILE development.

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