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VITAMIN D DEFICIENCY IS ASSOCIATED WITH DISABILITY IN MULTIPLE SCLEROSIS PATIENTS INDEPENDENTLY OF OXIDATIVE AND NITROSATIVE STRESS

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Background and aims The aim of this study was to assess vitamin D status in patients with multiple sclerosis (MS) and to evaluate whether it was associated with oxidative and nitrosative stress (O and NS) markers and disability.

Methods This study included 137 patients with MS and 218 healthy controls. The markers evaluated were serum levels of 25-hydroxyvitamin D, lipid hydroperoxides, advanced oxidation protein products (AOPP), nitric oxide metabolites (NOx), and total radical-trapping antioxidant parameter TRAP/UA.

Results MS patients presented higher age ($p < 0.0001$), and lower 25(OH)D ($p = 0.002$), NOx, and TRAP/UA ($p < 0.0001$) than controls. When the age was used as an additional explanatory variable in binary logistic regression analyses, 25(OH)D, NOx, and TRAP/UA remained significant ($p = 0.016$, $p < 0.0001$, and $p = 0.002$, respectively). Patients with 25(OH)D < 20 ng/mL showed higher EDSS ($p = 0.016$) and lower AOPP ($p = 0.046$) than those with 25(OH)D ≥ 20 ng/mL. After the binary logistic regression analyses, EDSS remained significantly associated with vitamin D deficiency. We showed that lower levels of 25(OH) were associated with higher EDSS independently of variables such as O and NS (AOPP and NOx), age, sex, body mass index, ethnicity, disease duration, MS therapy, use of interferon beta, and clinical forms of MS (odds ratio: 1.380, 95% confidence interval 1.030–1.843, $p = 0.031$). Moreover, the study showed an association between serum levels of 25(OH)D and EDSS ($r^2 = 0.115$, $p = 0.002$), demonstrating that 25(OH)D may contributed with 11.5% of increase in EDSS.

Conclusions Our results suggest that vitamin D deficiency may be considered one of the predictors of the disability in MS patients, independently of their redox status.

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TRANSMEMBRANE ACTIVATOR AND CALCIUM-MODULATING CYCLOPHILIN LIGAND INTERACTOR AS A PROMISING NOVEL TARGETED THERAPY FOR SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and aims Nowadays, the definitive therapy to cure SLE has not been found yet. The newest drug available, Belimumab and Rituximab, cannot cure all kind of SLE manifestation. Moreover, both of those drugs also decrease the number of B cell in the body. *Transmembrane Activator and Calcium-Modulating Cyclophilin Ligand Interactor* (TACI) is an immunology based target therapy that expected to give a better result in induct remission without decreasing the number of B cell.

Aims Analyse the possibility of TACI inhibition as a targeted therapy for SLE, in order to accelerate the remission period with less side effects.

Methods We look up for scientif article comprehensively in Medline, Science Direct, PubMed, and Cochrane Database. We found 10 article based on bibliography and keywords from the database.

Results TACI expression is elevated in B cells from patients with SLE. Inhibition of TACI cause an inhibition to B cell differentiation, and also decrease the amount of plasma cell, which cause a decreasing quantity of autoantibody which circulate in the blood. Inhibition of TACI will fully protects the animals against autoantibody production, without having any impact on B cell survival. This inhibition also delays the onset of proteinuria, albuminuria, basement membrane thickening, tubulointerstitial fibrosis, and glomerulosclerotic disease. It also inhibits only the T-cell independent responses and T-cell dependent (IgA), but it will maintain T-cell dependent protective B-cell functions.

Conclusions Inhibition of TACI may give better outcome compared to current therapy. However, the study for now is only limited in mouse.

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EFFECTS OF AN ORAL SELECTIVE SPHINGOSINE 1-PHOSPHATE RECEPTOR MODULATOR IN A MOUSE MODEL OF SLE

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