Conclusion: The CAREGIVERS questionnaire showed to be validated to assess the impact of pediatric rheumatic diseases.

References:

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AB0082

SERUM ALBUMIN LEVELS AND DEPRESSION IN JSLE
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Background: Albumin is a negative acute phase response protein synthesized in the liver, being an important marker of inflammation. Under inflammatory conditions, the transcapillary escape rate of albumin may increase, leading to hypoalbuminemia. Systemic lupus erythematosus (SLE) is a chronic condition involving multiple organ systems, inducing functional disability and psychological burden responsible for noteworthy depressive symptoms. Depression may be related with psychosocial, environmental and biological factors, disease activity and its severity. Several studies have shown that immune activation and increased concentrations of positive and decreased concentrations of negative acute phase proteins are involved in the pathogenesis of depression. As albumin has the capacity to bind homocysteine, lowered serum albumin levels leads to hyperhomocysteinemia, a well-known risk factor for depression. Moreover, hypoalbuminemia decrease the availability of tryptophan, an essential amino acid from which the neurotransmitter serotonin is derived, and induce oxidative stress, which further decreases antioxidant levels in people with depression.

Objectives: To assess the association between serum albumin levels and depressive symptoms in juvenile-onset SLE (JSLE) patients.

Methods: A cross-sectional sample of SLE patients, currently aged ≥16 years, completed a psychosocial assessment including quality of life (SF-36) anxiety and depressive symptoms (HADS) and cognitive assessment (MMSE), between October 2018- May 2019. Local Ethics Committee approved the study. All patients fulfilled both 2012 and 2019 EULAR/ACR classification criteria for SLE. Juvenile-onset was defined as age at diagnosis <18 years. Demographics and clinical characteristics were collected. Statistical analysis was performed with SPSS®.

Results: 35 patients were included, with current median (min-max) age of 22 (16-35) years, 29 (83%) females. Sex ratio (SD) was 1.1 (0.9-1.6). Median ESR was 19 (2-75) mm/h, CRP 1.65 (0.1-9.6) mg/L, albumin 41.6 (17.4-76.3) g/L, proteinuria 0.2 (0-3) g/dL, leukocyturia 0 (0-1362.7)/uL, erythrocytus 0 (0-501.9)/uL and anti-double stranded DNA 89.3 (10-800) U/mL. Mean C3 was 102.1 (21.6), C4 17.1 (7.4) mg/dL and creatinine 0.63 (0.1) mg/ dL. Median SLEDAI was 2 (0-12). All were ANA positive, 40 % positive for antinucleosssome antibodies, 25.7% anti-ribosanol P protein antibody, 11.4% anti-Sm antibodies and 31.4% anti-SSA antibody. 16.7% anti-SSB antibody. Antinucleosssome antibodies were positive in 48.6%, mucocutaneous in 77.1%, haemato logical in 45.7%, lupus nephritis in 49.2%, serositis in 8.6% and pulmonary interstitial disease in 2.9%. Mean (SD) total cholesterol values (TC) was 165.5 (44.7) mg/dL and LDL 94.5 (29.9) mg/dL. Median high-density lipoprotein was 52 (28-92) and triglycerides (TG) 81.5 (41-253) mg/dL. Median prednisolone dose was 5 (4-50) mg. 88.6% were treated with hydroxchloroquine, 31.4% with myophenolephate monophet and 14.3% with azathiohpine. TC was negatively correlated with serum albumin (p=0.043, rho=-0.378) and positively with SLEDAI (p=0.032; rho = 0.392), proteinuria (p=0.009; rho= 0.469) and leuko cytus (p=0.031; rho= 0.394). A positive correlation was found between LDL and proteinuria (p=0.043; rho= 0.385) and between TG and CRP (p=0.001; rho= 0.575). TG were also positively correlated with prednisolone daily dose (p=0.035; rho= 0.394). Mean LDL was higher in anti-Sm positive patients (p=0.022). No differences were found regarding anti-phospholipid antibodies. Nephritic lupus patients had worse lipid metabolism, but this did not reach statistical significance.

Conclusion: In our cohort, increased expression of TC, LDL and TGs is associated with disease activity in SLE. As expected, higher doses of prednisolone also correlated with lipid metabolism.

References:

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