SILENT CEREBRAL MRI FINDINGS IN LUPUS NEPHRITIS AND NON NEPHRITIS PATIENTS

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Background: Lupus nephritis carries high morbidity and mortality and whenever added to neuropsychiatric manifestations lead to more unfavorable prognosis. The current work focused on LN patients comparing them to those without kidney affection, studying their cerebral MRI and its correlation with the histopathological classes of LN and disease activity.

Objectives: To study the effect of renal affection on the brain in SLE and their clinical significance with early detection and management of such lethal comorbidity.

Methods: Cerebral MRI and MRA were studied in 40 SLE patients without neuropsychiatric manifestation; 20 LN patients with different histopathological classes and 20 patients without kidney affection. Disease activity was assessed for all patients using SLE disease activity index.

Results: Abnormal MRI brain findings were more common in LN patients “though non significant” (P=0.9). The most common lesions were white matter hyperintense lesions. Number and size of such lesions were significantly higher in LN patients (1.8 fold that of non nephritis, P=0.003 and 0.03, respectively) and positively correlated with urea, creatinine, urinary albumin/creatinine ratio, SLEDAI, ESR, CRP, and grades of renal biopsy and negatively correlated with c3 and c4, cortical atrophy and prepotine space dilatation were also significantly higher in LN patients (P<0.01).

Conclusion: Asymptomatic MRI brain lesions in LN patients, they are usually clinically significant and correlate to laboratory parameters of LN, grades of renal biopsy, and disease activity independent to age, sex and hypertension.

REFERENCES

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LUPUS PODOCYTOPATHY-CASE REPORT

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Background: Lupus podocytopathy is a recently recognized new entity of lupus nephritis characterized by diffuse foot process effacement with or without mesangial expansion. Capillary wall immune deposits and glomerular proliferation are lacking in the histopathological analysis. (1) Clinically, it is manifested as nephrotic proteinuria in the majority of reported cases. (1) Nevertheless, there is insufficient knowledge about the epidemiology, precipitant etiology and choices of management. Here, we describe a case presented with significant proteinuria secondary to foot process effacement with the absence of immunocomplement deposition or glomerular inflammation.

Objectives: To describe unusual presentation of proteinuria in lupus patient secondary to podocyte effacement which is unusual underlying pathology.

Methods: 45 years old female diagnosed to have SLE 14 years back in the setting of malar rash, alopecia, arthritis and positive serology (ANA, Ds DNA). She was doing fine on HCQ till a years ago when she presented to the clinic with gradual onset lower limbs swelling.

Laboratory results showed a significant proteinuria 2781 mg/dl/day, urine analysis showed RBC 1-2 cells/microliter, WBC 2-3 cells/microliter, normal renal profile and normal C3-C4 counts. Kidney Kidney biopsy was taken and described a picture of minimal change glomerulonephritis with mild mesangial expansion. In addition, the study was negative for the presence of Immunocomplement deposition with the absence of capillary basement membrane and tubulointerstitial inflammation. Patient was treated with Mycophenolate Mofetil 2 grams twice per day and 1mg/kg/day oral steroid. 3 months later, protein was noticeably reduced in urine to 731 mg/dl/day but the patients had a side effect of MFM inform of significant weight loss, gastritis, and diarrhea. Azathioprine had been instituted alternatively with low dose prednisolone 10 mg. Proteinuria level had peaked again after 4 months of starting azathioprine to reach 5660 mg/dl/day. Kidney biopsy performed again which showed minimal light microscopic changes, the ultrastructural study showed extensive podocyte injury and foot process effacement up to 70% by the electron microscope. Mild mesangial proliferation and focal tubulointerstitial chronic changes. There was no evidence of endocapillary proliferation, subendothelial deposits or glomerular scarring. Immunofluorecence was negative for immunocompetent deposition.

Mycophenolate Mofetil has been instituted again but unfortunately, proteinuria inadequately responded and remained between 3-4 grams. Laterally, Cyclosporin 1.25 mg/kg/day has been commence few days ago. All laboratory results like renal profile and complements are with in normal values.

Results: Proteinuria was found to be attributed to diseased podocyte namely lupus podocytopathy. Unfortunately, she has inadequate response to azathioprine and mycophenolate mofetil.

Conclusion: Lupus podocytopathy is a recently recognized new entity of lupus nephritis characterized by diffuse foot process effacement without capillary wall immune deposits and glomerular proliferation. This case presentation illustrate an attention toward this new lupus entity. However, the lack of efficient knowledge about the frequency, clinical features and treatment necessitate further investigation.

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CLINICAL ASSOCIATIONS AND DIAGNOSTIC POTENTIAL OF REGULATORY-LIKE B-CELLS IN SJÖGREN’S SYNDROME

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Background: Some B-cell subsets contribute to the regulation of immune responses, mainly through the secretion of interleukin-10, which suppresses T-helper 1 (Th1) and Th17 cells and induces regulatory T-cells (Tregs). The role of Bregs and Tregs in Sjögren’s syndrome (pSS) pathogenesis is an active field of research.

Objectives: To evaluate the distribution of regulatory and effector T and B-lymphocytes, in patients with pSS and healthy controls (HC), and the relation between regulatory-like B-cell subsets and the pSS phenotype.

Methods: Fifty-seven pSS patients (2002 AECG criteria) and 24 HC were included. Circulating T and B-lymphocytes were characterized by flow cytometry and groups were compared. Significance was considered for p<0.05.

Results: Compared to HC, pSS patients had lower percentages (16.9 vs 32.0%, p=0.011) and absolute numbers (28 vs 81 cells/µL, p=0.001) of Breg-enriched CD24+CD27+ B-cells, and a decrease in CD24hiCD38hi B-cells percentages (6.2 vs 4.2%, p<0.001; 15.0 vs 19.6%, p=0.170), compared to anti-SSA-negative (n=19) patients (15.0 vs 19.6%, p=0.170), as well as lower absolute counts (26 vs 31 cells/µL, p=0.173). Anti-SSA-positive patients presented higher CD24+CD27+ B-cells percentages (6.2 vs 4.2%;