Is Antiribosomal P Protein Antibody Associated With Neuropsychiatric Systemic Lupus Erythematosus?

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Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease involving multiple antibodies and complex pathogenesis. Neuropsychiatric SLE (NPSLE) is a devastating manifestation of SLE that involves the central nervous system (CNS), producing neurologic and psychiatric symptoms. Seizure manifestation is a common presentation of NPSLE and predictive of poor prognosis.[1] The pathogenesis of NPSLE is multifactorial, involving inflammatory mediators and autoantibodies.

Multiple studies support an association with antiribosomal P antibody and NPSLE, while other studies do not.[2-5] Thus, antiribosomal P antibody remains speculative as a diagnostic tool.

We present a case of SLE with development of subsequent neuropsychiatric symptoms and positive antiribosomal P antibody. The utility of antiribosomal P antibody should be further investigated in NPSLE.

Case Presentation

Patient: A 36-year-old female with past medical history of SLE, mixed connective tissue disease, hypothyroidism, Reynaud's, and hypertension presented to the hospital with a chief complaint of witnessed seizure, fevers, and altered mental status. She had one witnessed seizure at home by her husband and denied history of prior seizure disorder. Patient was initially diagnosed with SLE and prescribed hydroxychloroquine 200 mg BID.

Clinical Course: Vital signs were stable. MRI brain showed mild cerebral volume loss. Lumbar puncture was unremarkable. CT angiography chest was remarkable for bilateral pulmonary infiltrates. Urinalysis was remarkable for 2+ proteinuria. Findings were consistent with severe flare-up of SLE with pulmonary involvement, CNS involvement, pancytopenia, and new-onset proteinuria. She was given high-dose steroids with improvement in symptoms. She did not have any further episodes of seizures and was discharged on 60 mg PO prednisone.

Follow-up: She was started on additional immunosuppression with mycophenolate mofetil in outpatient rheumatology, in addition to hydroxychloroquine therapy. AVISE was positive for antiribosomal P antibody, previously noted to be negative on initial diagnosis of SLE (Figure 1). AVISE checked on follow-up appointment revealed decreased antiribosomal P antibody, with most recent titer 51.0 U/ml. She was referred to outpatient neurology for further evaluation.

Figures

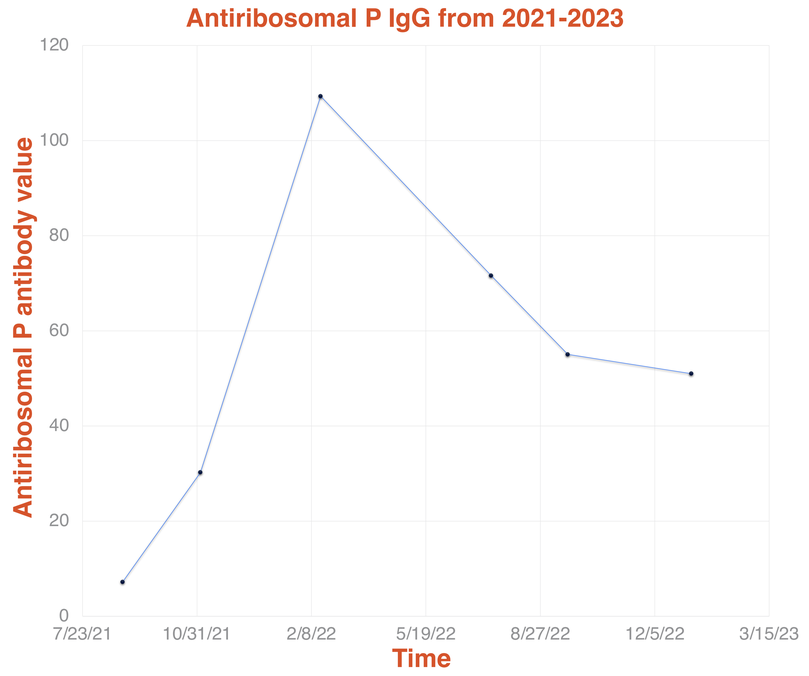
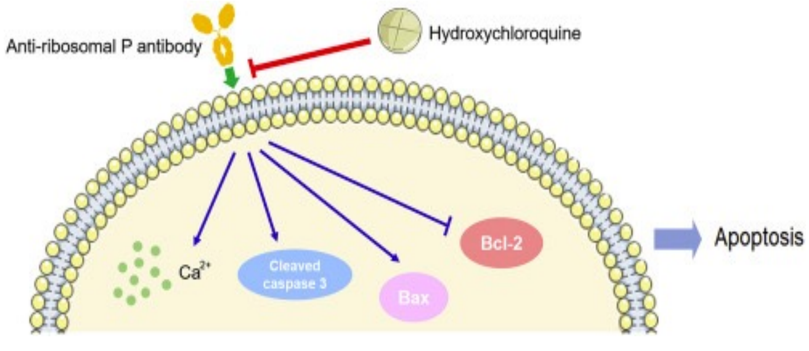


Figure 1: Antiribosomal P protein antibody trends of the patient from serial AVISE panels. Reference range < 20 negative, ≥ 20 positive.



Hydroxychloroquine Alleviates the Neurotoxicity Induced by Anti-ribosomal P Antibodies by Zhao, Xinnan; Yang, Pingting. From: The Journal of Neuroimmunology. Published September 14, 2021. Volume 358. Pages 577648.

Figure 2: Pathogenesis of antiribosomal P antibody inducing damage on neural cells. Antiribosomal P antibody binds to neural cells, leading to rapid influx of calcium into neurons and subsequent apoptosis. Hydroxychloroquine eases damage on neural cells and may play neuroprotective role in NPSLE.[6]

Discussion

Antiribosomal P antibody has been associated with diffuse neuropsychiatric symptoms of SLE, including headache, cognitive impairment, stroke, acute encephalopathy, seizures, depression, and psychosis. Antiribosomal P antibody is one of the most specific antibodies for NPSLE with estimated specificity of 80%.[4] Antiribosomal P antibodies appear in up to 46% of SLE patients.[2]

This autoantibody binds to neuronal cell surface protein distributed in memory, cognition, and emotion leading to rapid influx of calcium into the cell, causing apoptosis (Figure 2).[6,7]

Previous metanalysis concluded that antiribosomal P antibody provides limited diagnostic value and is poor at differentiating disease phenotypes of NPSLE.[4,5] Other recent prospective studies have shown that antiribosomal P antibody in serum greatly increases mortality in diffuse NPSLE and remains a major factor, indicating poor prognosis.[8,9] However, antiribosomal antibody P as a specific prognostic factor for NPSLE remains unknown.

This case highlights a patient who initially presented with SLE without neuropsychiatric symptoms that later developed diffuse CNS involvement from SLE flare, correlating with a rise of antiribosomal P protein antibody. Further studies on the prognostic value of antiribosomal P is warranted in NPSLE.

Conclusion

Positive antiribosomal P antibody and high disease activity may be risk factors for NPSLE. Presence of antiribosomal P may indicate poor prognosis, and neuropsychiatric symptoms decreases survival rates among SLE patients. Routine screening of antiribosomal P antibody may serve as a diagnostic tool for NPSLE. While the mechanism of antiribosomal P antibody is not fully understood, this may represent a future target for molecular exploration of NPSLE.

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