Ribosomal P Antibodies Preceding the Onset of Sle-A Case Report


Dept of General Medicine, ESIC Medical College and PGIMSR, Rajajinagar, Bangalore
*Corresponding author: buddhatozen@yahoo.co.in

Received December 31, 2012; Revised February 03, 2013; Accepted April 02, 2013

Abstract Systemic lupus erythematosus (SLE) is an autoimmune disorder characterized by the production of auto antibodies directed against a variety of nuclear and cytoplasmic antigens. Auto antibodies to ribosomal Proteins are found in 15–30% of systemic lupus erythematosus (SLE) patients. Ribosomal P antibody is shown to predict SLE 1.7 years before clinical onset of SLE. This is a case of 28 year old female patient with a malar rash and nonspecific neurologic symptoms. The antibody profile showed that ANA ,anti- Ds dna and anti- Sm antibodies were negative but anti ribosomal P0 antibody was strongly positive(++) at presentation. A strong clinical suspicion and careful follow-up helped in making a diagnosis of SLE over a period of nine months.

Keywords: ribosomal P antibody, SLE, ANA

1. Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disorder characterized by the production of auto antibodies directed against a variety of nuclear and cytoplasmic antigens. Among the SLE-specific auto antibodies, one subset is directed against ribosomal P phosphoproteins P0, P1&P2. Ribosomal P auto antibodies (anti-P) occur almost exclusively in SLE. Auto antibodies to ribosomal P are found in 15–30% of systemic lupus erythematosus (SLE) [1], and have been associated with a number of clinical presentations including neuropsychiatric symptoms and are known to predict onset of SLE [2].

2. Clinical Summary

A 28 year old female patient presented with H/O tingling, numbness and pain in the right hand since 2 years followed by loss of touch and temperature senses since 6 months in the right little and ring fingers and palmar aspect of the right hand on medial side. On examination, the patient was found to have an erythematous papular rash on malar area of the face and multiple oral ulcers. The clinical examination of respiratory, cardiovascular and gastrointestinal systems was normal. She was suspected as SLE and her investigations revealed as follows. Complete blood count:- Hb-8.4gms, Platelets - 2.4Lakhs, total white blood cell count - 4600, ESR- 40mm at the end of one hour. The patient’s renal functions and liver function tests were normal. The imaging studies of the nervous system and cardia were normal. Nerve conduction studies revealed reduced action potentials in the ulnar nerve. The AUTO ANTIBODY profiling at presentation was as follows – ANA+, Ds DNA -negative, Anti Sm- negative, antibody to Rib.P0 (+++) strongly positive. The patient was followed up regularly and after 9 months her repeat ANA became positive (2+) with a full blown clinical picture of SLE with renal involvement.

3. Discussion

Anti-ribosomal P antibodies have been found to be associated with a more severe disease course [3] in SLE Anti-P prevalence in SLE varies widely by race, ranging from 10% to 40%. Antibodies to ribosomal P are directed against a family of phosphoproteins that are associated with the 60S ribosomal subunit. Ribosomal P proteins share a 22 amino acid carboxyl-terminal sequence that is thought to comprise the immunodominant epitope. Auto antibodies against ribosomal P are directed against three proteins P0 (38kD), P1 (19kD), and P2 (17kD) [4]. Several studies indicate the association between increased titres of Anti-rP antibodies in patient’s sera and active SLE disease. Anti-rP antibodies appear prior to diagnosis in 84% of anti-P positive patients and these antibodies develop on average 1.7 years prior to SLE diagnosis [5]. Auto antibodies in SLE can usually be detected in patient sera many years before SLE diagnosis and tend to accumulate in the years leading up to diagnosis while patients are still clinically asymptomatic. Furthermore, SLE-specific auto antibodies such as Anti-Sm and Anti-ds DNA appear closer to the time of diagnosis than non-specific auto antibodies such as Anti-Ro and Anti-La [6,7]. However, the accumulation and timing of anti-ribosomal P antibodies has not previously been analyzed.

4. Conclusion

SLE is nearly always diagnosed months to years after the onset of clinical symptoms and even longer after the
onset of auto antibodies. Antiribosomal antibodies can be used as early markers of the disease and careful follow-up of these patients can help in early diagnosis SLE.

References


