



## A Case of Hypothyroidism with Anti-dsDNA Negative Lupus Nephritis Presenting as Pericardial Effusion: A Diagnostic Dilemma

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### Abstract

*Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease that may involve many different organs and display a variable clinical course. From a pathogenetic point of view, the production of several autoantibodies characterizes the disease. The diagnosis of SLE is based on characteristic clinical findings of the skin, joints, kidneys, and the central nervous system, as well as on serological parameters such as antinuclear antibodies (ANA), in particular, antibodies to dsDNA.<sup>[1]</sup> Here we present the case of a 30-year-old female patient, known case of hypothyroidism, who presented with polyserositis and renal involvement and upon investigations was diagnosed to be a case of Anti-dsDNA negative lupus nephritis.*

### Introduction

SLE is an autoimmune disease, potentially involving any organ/system, with a remitting-relapsing course<sup>[2,3]</sup>. It is characterized by the production of a wide range of autoantibodies, resulting from polyclonal B cells activation, impaired apoptotic pathways, or idiotypic network dysregulation. Among these antibodies, anti-dsDNA represent the hallmark of SLE.<sup>[4,5]</sup> Due to the high frequency (ranging from 70% to 98%), sensitivity, and specificity (57.3% and 97.4%, resp.), the presence of these autoantibodies could be virtually diagnostic for SLE.<sup>[4]</sup> Despite the central role of these antibodies in the disease pathogenesis, a percentage of SLE patients, ranging from 2 to 30%, result negative for anti-dsDNA.<sup>[5]</sup>

### Case Presentation

A 30-year-old female patient, known case of hypothyroidism since 3-4 months, on irregular treatment for the same, presented with complaints of generalised body swelling (on and off) since 2-3 months, loose stools since 4 days and decreased urine output for 2 days. She also gave history of h/o multiple joint pain involving knees and small joints of hands and foot, shortness of breath on exertion and excessive hair fall. She denied any h/o rash or recurrent oral ulcers, pain abdomen or reddish discoloration of urine. On examination, pallor and bilateral pitting pedal oedema was present. Bilateral crepitations were present at lung bases. Heart sounds were muffled and abdominal distension with free fluid was present. Routine investigations revealed pancytopenia and deranged Renal function tests during the course of stay in the hospital. ECG of the patient revealed

low voltage QRS complexes. Urine C/E revealed minimal proteinuria. Chest x ray showed unilateral pleural effusion and pericardial effusion. Also, ultrasound of the abdomen revealed ascites. 2D-Echo was suggestive of moderate pericardial effusion with a maintained ejection fraction. ESR of the patient was highly raised at 175mm/1<sup>st</sup> hour.

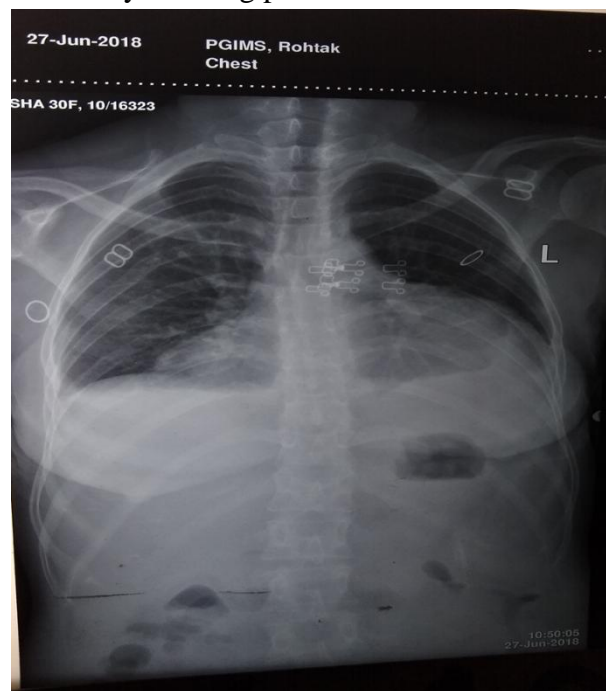
Therapeutic pericardiocentesis was done in view of moderate pericardial effusion. Ascitic, pleural and pericardial examination of the patient showed exudative, lymphocytic predominant picture. In view of these findings and low-grade fever, patient was empirically started on Antitubercular therapy while awaiting the report of TB-PCR which came out to be negative. The patient showed no improvement of symptoms after institution of ATT. Meanwhile repeat urine complete examination revealed nephritic range proteinuria and dysmorphic RBCs. Considering arthralgia, pancytopenia, polyserositis, significantly raised ESR and renal involvement, patient was worked up for Systemic lupus erythematosus. ANA was found to be positive at a titre of 1:160. Anti dsDNA was found to be negative and C3, C4 complement levels were reduced at 22.5 mg/dl and < 8 mg/dl respectively.

In view of renal involvement and persistent proteinuria, a renal biopsy was done which revealed a Stage 2 Lupus nephritis.

#### Investigations on Admission

|                     |                             |
|---------------------|-----------------------------|
| Hb                  | 6.7g/dl                     |
| TLC                 | 2000 cells/cumm             |
| APC                 | 1.0 lakh/cumm               |
| PBF                 | Microcytic, hypochromic     |
| B.urea/S.creatinine | 144/3.9 mg/dl               |
| S. uric acid        | >15 mg/dl                   |
| S. albumin          | 1.8 g/dl                    |
| ESR                 | 175 mm/1 <sup>st</sup> hour |
| TSH                 | 7.4 microIU/L               |
| ANA by IFA          | Positive; 1:160             |
| C3 and C4           | 22.5 mg/dl and <8 mg/dl     |
| Anti dsDNA          | Negative                    |

Chest X ray showing pericardial effusion



#### Discussion

Anti-dsDNA antibodies are listed as an immunologic criterion for the classification of SLE by the American College of Rheumatology and SLICC. A percentage of SLE patients, ranging from 2 to 30%, result negative for anti-dsDNA. Previous studies have demonstrated that serositis resulted significantly more frequently in anti-dsDNA -ve SLE compared to anti-dsDNA +ve SLE and significantly higher frequency of renal involvement in persistently positive patients, thus confirming the pathogenetic role of anti-dsDNA in the kidney injury.<sup>[6]</sup> Historically it had been postulated that anti-dsDNA antibodies play a role in the initiation of lupus nephritis. Now, many studies suggest that anti-dsDNA antibodies may not be required for the pathogenesis of lupus nephritis and a growing interest is devoted to other antibodies detected in the serum of SLE patients, as evident by the classification criteria recently proposed by the Systemic Lupus International Collaborating Clinics.

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