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Case Report

Navigating the storm: a young woman's battle with refractory lupus nephritis complicated by pulmonary tuberculosis: a detailed case report

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ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune disease that is commonly treated with high dose of corticosteroids and other immunosuppressive medications. Patients with SLE are thus more likely to become infected with a variety of pathogens, including Mycobacterium tuberculosis. There are no established guidelines for treatment of tuberculosis in SLE patients with high disease activity due to a lack of relevant studies and management based on physician expertise. This article presents a case report of a 31 years old female with underlying SLE and refractory lupus nephritis previously treated with IV methylprednisolone pulse therapy and 6 doses of cyclophosphamide and Rituximab presented with Fever, cough with expectoration, pain abdomen and loose stools for a 15 days duration. Mini-BAL CBNAAT detected TB. She was treated with intravenous methylprednisolone and anti-tuberculous therapy, but the result was a fatal outcome.

Keywords: Lupus nephritis, Systemic lupus erythematosus, Tuberculosis

INTRODUCTION

SLE is marked by the presence of autoantibodies targeting self-antigens, leading to inflammation and damage multiple organs. Infections, cardiovascular disease and renal failure are the main contributors to mortality in these patients. The increased incidence of tuberculosis in those with SLE is linked to various immune system abnormalities and the use of immunosuppressive therapies.[1] The risk of tuberculosis in lupus patients is influenced by the local prevalence of the disease. SLE and tuberculosis can present similarly, complicating diagnosis, additionally, previous tuberculosis infections may trigger SLE in genetically susceptible individuals. Thus, it is crucial to differentiate between the two conditions in individual patients. This report underscores the need for routine tuberculosis screening in SLE patients residing in endemic areas.[2] Treatment decisions should be guided by local medical expertise, as there are no specific WHO guidelines for managing latent tuberculosis infection in these cases. This highlights the necessity for further research into the benefits and risks of testing and treating latent tuberculosis in patients undergoing steroid therapy or those with rheumatological disorders.

CASE REPORT

A 31 years old female with SLE for 5 years diagnosed based on the basis of malar rash, photosensitivity, oral ulcers and severe anaemia during her third pregnancy with ANA and ANTI RO +++ positivity and complement levels (C3-73, C4-16) she was started on oral prednisolone 10mg and Azathioprine 100 mg. She discontinued the medications due to GI side effects.

After a year she came for follow up and was started on 'Mycophenolate mofetil BD with oral Prednisolone and planned for renal biopsy. Renal biopsy reveals IF 11 GLOMERULI IgG 3+ C3 3+ C1Q3+ positive on capillary loops and Mesangium IGM IGA Negative. Patient treated with 6 doses of cyclophosphamide along with oral prednisolone 10 mg Bd. Despite completed the induction

phase of NIH protocol her symptoms of frothy urine, Bilateral leg swelling doesn't subsided and 24 hours urine protein reveals 9786 mg/day. Hence patient was planned for Rituximab injection and she lost her follow up for 2 months. After 2 months Rituximab 100 mg and methylprednisolone 125 mg was given. Later after a month duration she presented ER with Fever, pain abdomen, vomiting, loose stools for the past 15 days and cough with expectoration and shortness of breath for the past 5 days. At presentation, the young woman's vital signs were blood pressure 120/80 mmHg and heart rate 88 beats/min, providing essential clinical context for her condition of refractory lupus nephritis complicated by pulmonary tuberculosis in this detailed case report.

Clinical findings

On physical examination patient was conscious, oriented, afebrile. Blood pressure: 150/110 mmhg, pulse rate: 140/minute, temperature: 103 F, respiratory rate: 30/minute, Spo_2 was 78% on room air. Her chest examination reveals bilateral air entry with coarse crepitations. On same day a chest radiograph showed bilateral nodular opacities and HRCT chest report showed Multiple tiny centrilobular nodules seen in bilateral lung fields predominantly in right middle lobes. A well-defined soft tissue density nodule with peripheral rim of calcification in the superior basal segment of right lower lobe likely infective nodule.

Mosaic attenuation in bilateral lung fields. Multiple fibro atelectatic bands noted in bilateral lower and right middle lobes. Multiple small patchy fibrosis with volume loss in bilateral upper lobe. Bilateral mild pleural effusion. Possibly active infection. Patient was treated IV antibiotics, IV steroids, diuretics, beta blocker, anti-pyrectics, statins, anticoagulant, thrombopoietin receptor agonists.

Pulmonologist opinion was obtained advised for anti-fungal, anti-viral, broncho alveolar lavage. Later Mini BAL was sent for CBNAAT revealed MTB. Pulmonologist review was taken in view of CBNAAT advised for anti-tubercular drugs and ATT was started. On day 11 of post admission, she became hemodynamically unstable and went into bradycardia, hypotension and cardiac arrest CPR was started according to ACLS protocol. In-spite of resuscitate efforts patient succumbed to death.

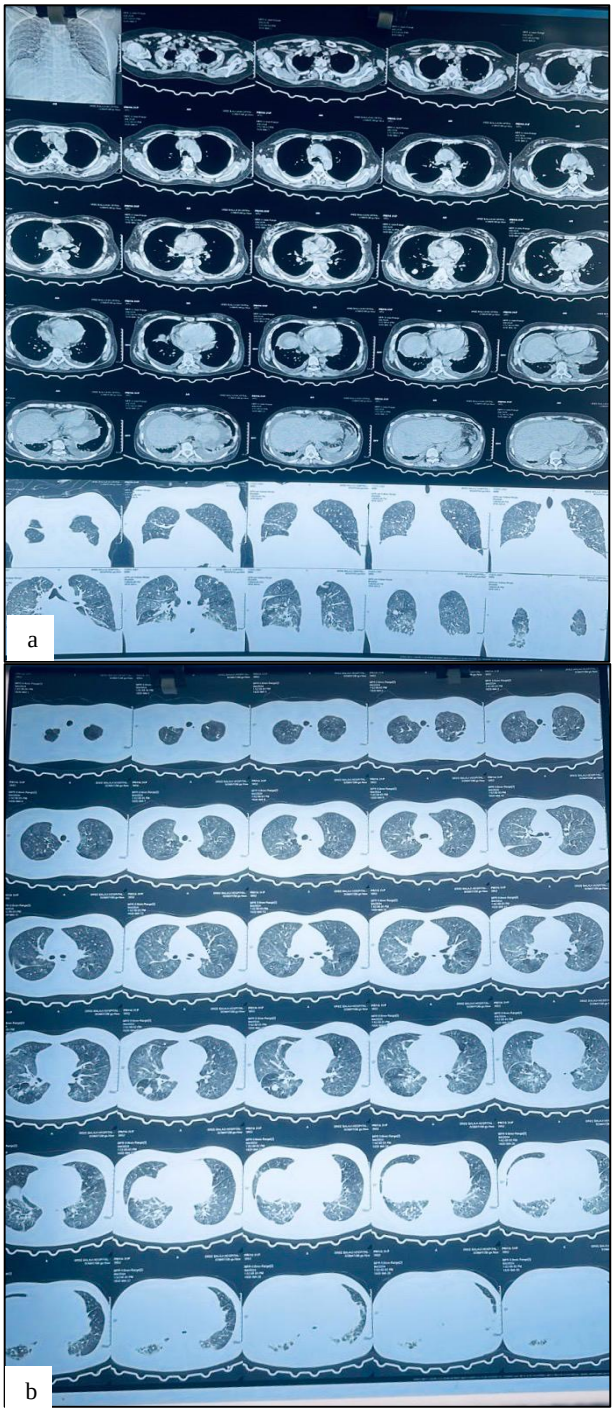

Figure 1 (a and b): HRCT chest showing multiple tiny centrilobular nodules in bilateral lung fields

Table 1: Laboratory test.

Test/ date	4-Sep	6-Sep	8-Sep	10-Sep	11-Sep	12-Sep	13-Sep	14-Sep
Haemoglobin (gm/dl)	9.4	7.1	7	7.3	7.5	6.7	7.9	6.5
PCV	29.6	21.8	21.4	22.9	23.3	20.8	24.9	20.7
Total WBC	4380	4840	47400	3280	2230	3390	3900	170
Platelet	1.2 lakhs	71000	77000	55000	44000	1.14 lakhs	81000	56000
Renal profile								
Urea	48.43	84.2	95.19	78	77.4	108	120.73	104.8

Continued.

Test/ date	4-Sep	6-Sep	8-Sep	10-Sep	11-Sep	12-Sep	13-Sep	14-Sep
Creatinine	1.33	2.92	2.87	2.3	2.23	2.7	2.42	1.72
Uric acid	9.46	11.83	10.51	8.2	7.31	7.9	7.88	6.92
Sodium	141	136	135.9	134.3	133.5	140.3	141.6	145.1
Potassium	3.3	3.42	3.86	3.75	3.45	4.13	4.32	5.5
Chloride	111	113.1	114.9	113.9	114.2	113.2	116.5	119.9
Viral serology	Negative							
Serum procitonin	8							
Urine protein	127							
Urine spot creatinine	0.066							
Urine spot pcr	1914.6							
C3 complement		0.97						
C4 complement		0.37						
PT				11.3	11.5			
APTT				37.4	33.8			
INR				0.98	1			
Serum fibrinogen				227.3				
Serum ferritin								
Blood pressure (mmhg)	120/80							
Heart rate (bpm)	88							
Respiratory rate (breaths/min)	18							
AFB smear				Negative				
Fungal KOH				Negative				

DISCUSSION

Pulmonary tuberculosis in SLE patients may manifest differently than in immunocompetent patients, with a higher incidence of miliary disease due to delayed diagnosis and non-specific clinical symptoms that mimic lupus.[3] Therefore, early diagnosis of pulmonary tuberculosis is essential for proper treatment. High-dose steroid therapy is a significant risk factor. Several studies have found that SLE patients received a higher cumulative dose and mean daily dose of prednisolone prior to tuberculosis diagnosis.[4]

According to one study, each gram dose of prednisolone increases the risk of developing tuberculosis by 23%. To more convincingly demonstrate the role of steroids in increasing the risk of tuberculosis, the effect of cortisol on the immune response to *Mycobacterium tuberculous* antigens was investigated in vitro. Corticosteroid-induced immunosuppression in SLE patients directly increases susceptibility to pulmonary tuberculosis. This highlights the critical need for careful monitoring of patients receiving high-dose steroids.

Early recognition of tuberculosis symptoms and timely intervention are essential to reduce complications and improve clinical outcomes in this vulnerable population. At presentation, the patient's vital signs were: blood pressure 120/80 mmHg, heart rate 88 beats/min, respiratory rate 18 breaths/min and temperature 37.2 °C. It demonstrated that cortisol, within physiological range, can inhibit the mycobacterial antigen-driven proliferation of cells as well as the production of interferon-gamma, implying that endogenous levels of cortisol may contribute to the decreased lymphoid cell response to mycobacterium antigen.[5]

CONCLUSION

In conclusion, SLE patients with tuberculosis have a high mortality. In this case report emphasizes the importance of formal guidelines for co-management of active tuberculosis in SLE patients who are on immunosuppressive therapy. Regular monitoring for active tuberculosis in endemic areas is crucial to treat infection in the early stages before it spreads due to increased immunosuppressant dosage, as tuberculosis symptoms and causes are similar to SLE. Furthermore, this case report will help in the future to establish guidelines as we treat both diseases concurrently with a risk of poor outcome.

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