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Research Article

AN APPROACH TO PLEURAL EFFUSION IN SLE - A CASE REPORT

T V D Sasi Sekhar[1], M. Indra Keela Girish[2], K.S. Keerthi Vyas [3], Sravya V V K [4]

*[1]Professor of Medicine, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinoutpalli, Andhra Pradesh, India.
*[2,3,4] Post Graduates, Department of General Medicine, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation , Chinoutpalli, Andhra Pradesh, India.

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Corresponding Author:
M. Indrakeela Girish,
Department of General Medicine,
Chinoutpalli, Vijayawada,
Andhra Pradesh, India

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ABSTRACT

Systemic lupus erythematosis is a systemic autoimmune disease involving multi organ system.Patients with SLE have increased susceptibility to infections, most commonly tuberculosis with significant morbidity and mortality in endemic region.We report a case of young female presented with Breathlessness and fever.We diagnosed the patient was having S.L.E, and the cause of pleural effusion was due to T.B .It is challenging to diagnose TB in a case of SLE as they overlap with each other. Hence we publish this case as it will act as a guide in diagnosing T.B in S.L.E.

Key words: SLE, Tuberculosis , Pleural Effusion

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CASE REPORT:

A 19 yrs old female presented to ER with complaints of fever since 1 month, high grade, intermittent. She had gradually progressive exertional dyspnoea associated with dry cough. She had loss of appetite and loss of weight.

On examination, blood pressure was 110/60 mm Hg, pulse–130/min, febrile with 101F and 96% saturation on room air. Chest examination was suggestive of Left pleural effusion.

Investigation revealed microcytic hypochromic anemia with neutrophilia with adequate platelet counts, ESR 30mm/hr. Blood sugar, liver and renal function test were normal Chest X - ray showing consolidation of left mid and lower zone with bilateral pleural effusion.ECG - tachycardia with low voltage complexes and T wave inversions in v3 to v6.2D Echo revealed LVEF : 30% with normal LV size with moderate LV dysfunction, global hypokinesia,normal valves, mild TR, mild PAH(RVSP : 40).
Pleural fluid analysis: 42 cells with Neutrophils—20%. Lymphocytes 80%, Proteins- 3.0gm/dl,glucose - 9 mg%,ADA - 36.15(upto18), culture negative.

Tuberculosis was suggestive as the pleural was exudative and ADA positive andpatient was started on ATT with Tab Ethambutol 800mg + isoniazide 300 mg + pyrazinamide 750mg (2 tab)+rifampcin 450mg and Benadone 40 mg OD.

Patient didn't show any clinical improvement and during her hospital stay she developed ulcers in the mouth and buccal mucosa with Maculopapular rash present on the extensor surface of lower limbs, suspecting SLE ,serology for auto antibodies were sent.ANA -40.5 U/ml (positive > 25) and Anti ds DNA-891.7 IU/ml (< 20).Her thyroid profile and fundus was normal.

Patient was fitting into SLE criteriawithulcers in the mouth,buccal mucosa-1,anemia-1,polyserositis-1, ANA positive-1and anti ds DNA positive-1 total 5/11.she was started on tab prednisolone 40 mg od,tab HCQ 400mg bd,ATT was stopped.
On the 10th day of starting steroids she continued to have breathlessness and there is no resolution of pleural effusion in chest x-ray.

As our previous pleural fluid analysis showed ADA 36.15U/L .We reviewed the literature from Luis valdes ,David Alvarez told that if pleural fluid ADA < 47U/L ,pleural biopsy is specific to differentiate between Tuberculosis and other causes for raised ADA Levels in Pleural fluid.

We repeated pleural fluid analysis for ANA ,ADA. ADA-67.21 U/L (positive >23)ANA-2.74U/ ml(positive>25). Pleural biopsy showed granulomatous necrosis.We started the patient on Cat1 ATT and her general condition improved progressively

Discussion

Systemic lupus erythematosis is an autoimmune systemic disorder involving multi organ system.Infections are one of the leading cause of mortality and morbidity in SLE[2].They are prone to develop opportunistic infections

.Factors leading to the development of infections in SLE patients are :1)Genetic Factors :complement Deficiencies, Mannose –binding lectin, Fcgamma iii, GM-CSF,Osteopontin.2) use of immunosuppressive therapy[1].Infections play important role in the expression of SLE in genetically predisposed individuals[6].

Evidence by Shoenfeld and Schwartz available suggesting that monoclonal antibodies raised against TB can cross react with DNA[7], and detection of antibodies in patients with TB similar to that found in SLE[8].Evidence also available that prior TB infection may precipitate SLE in genetically susceptible patients and the incidence of TB in SLE patients is considered to be 15-fold higher. Ghosh et al[9]reported that prevalence of antecedent TB in patients with SLE to be21%, i.e., 40 times higher than the prevalence of TB in general population. Pleural biopsy histology and culture improves the diagnostic rate to about 90%.[10,11]

The ADA level in pleural fluid tends to be higher with tuberculosis than in other exudates.[12-14]With a concentration ofADA in pleural fluidof >40 U/I as a diagnostic criterion, the positive predictive value of pleural fluid concentrations of ADA for tuberculosis was 93.1%.[15]High levels of ADA present in lymphomas, malignancy, SLE , or pneumonia, rheumatoid pleurisy, whichhad pleural ADA concentrations below our diagnostic threshold[16]and makes the test less useful in countries with a low prevalence of tuberculosis.[21]

The prevalence of SLE with cardiacinvolvement is>50%with all three layers of the heart(pericardium, myocardium and endocardium).The clinical detection of myocarditis ranges from 3 to 15%[17]. Harvey *et al* found that myocarditis was the cause of heart failure in 8 of their 9 patients[18].Appenzeller studies on echocardiography show that it cannot definitively diagnose myocarditis, but global hypokinesia, in the absence of other known causes, is strongly suggestive.[19]

Studies showed that negative pleural ANA test makes a diagnosis of lupus serositis unlikely.[20]

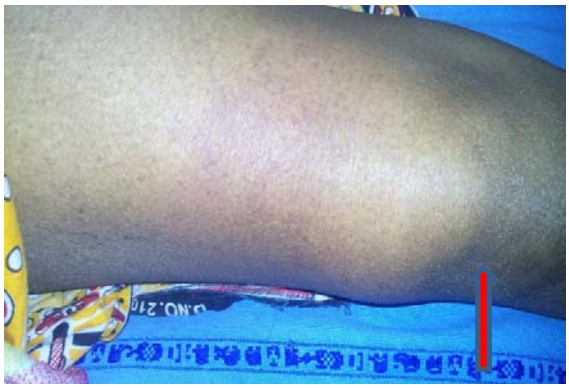

Fig 1: Maculopapular rash on the extensor surface of the lower limb

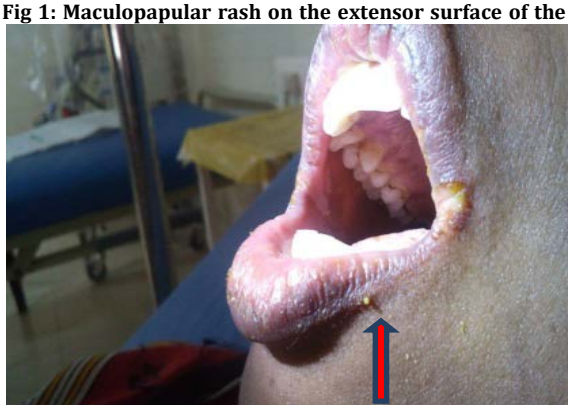

Fig 2: Ulcers in the angle of the mouth

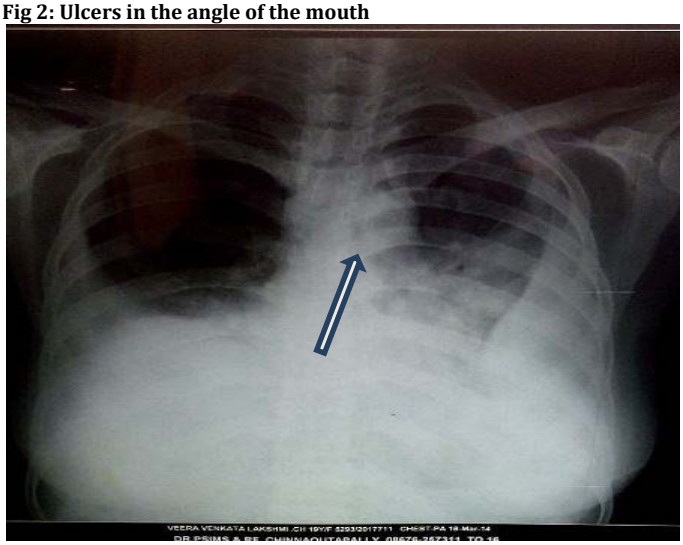

Fig 3: chest X – ray PA view showing consolidation of left mid and lower zone with pleural effusion

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