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A Case Of Lupus Pneumonitis

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Abstract

Systemic lupus erythematosus is a systemic autoimmune disease with multisystem involvement and is associated with significant morbidity and mortality.Approximately 90 % affected individuals are female and among young women SLE is one of the top 10 causes of death. Frequently diagnosed between ages 15 and 45 . Approximately 70% of patients follow a relapsing remitting course , the remaining divided equally between a prolonged remission and a persistently active disease.1 Here we report a young female who presented with complaints of high grade fever, cough with mucoid expectoration and breathlessness for 7days; rash over face, oral ulcers, polyarthralgia, myalgia and generalised swelling for 3 days. ANA was positive and complete profile showed anti Smith and anti RNP antibodies positive. Treatment was started with high dose pulse steroids - I.V. Methylprednisolone followed by oral Prednisone (1 mg/kg/day), Hydroxychloroquine (4 mg/kg/day) and parenteral antibiotics , patient was improved symptomatically.

Keywords: systemic lupus erythematosus ,Lupus pneumonitis, Anti smith ,Anti RNP

Introduction

36 year old female came with complaints of high grade fever, cough with mucoid expectoration and breathlessness for 7days; rash over face, oral ulcers, polyarthralgia, myalgia and generalised swelling for past 3days. O/E patient had pallor, anasarca, photosensitive malar rash, dyspnea, tachypnea and B/L basal coarse crepitations. Laboratory findings showed pancytopenia, hypoalbuminemia, normal Renal function test, elevated ESR and normal C-reactive protein. Peripheral smear shows – microcytic hypochromic anemia. Urine protein 3+ with 24 hours urinary protein 850 mg/day . CT chest showed Bilateral mild pleural effusion, multiple ground glass density nodules scattered in Bilateral lung parenchyma predominantly in upper lobes, area of consolidation in superior segment of left lower lobe, passive atelectasis of lung noted in basal segments in right lower lobe. Sputum for AFB & Culture and sensitivity negative. ANA was positive & further profile showed anti Smith & anti RNP antibodies positive. Serum C3 & C4 levels were low . According to the EULAR/ACR 2019 criteria, patient was diagnosed as systemic lupus erythematosus presenting with acute lupus pneumonitis & lupus nephritis with SLEDAI of 16. She was treated with high dose pulse steroids - I.V. Methylprednisolone for 3 days (10 mg/kg/day) followed by oral Prednisone (1 mg/kg/day), Hydroxychloroquine (4 mg/kg/day) and parenteral antibiotics. There was clinical & biochemical improvement evident by an improved SLEDAI of 12.

FIG -1a

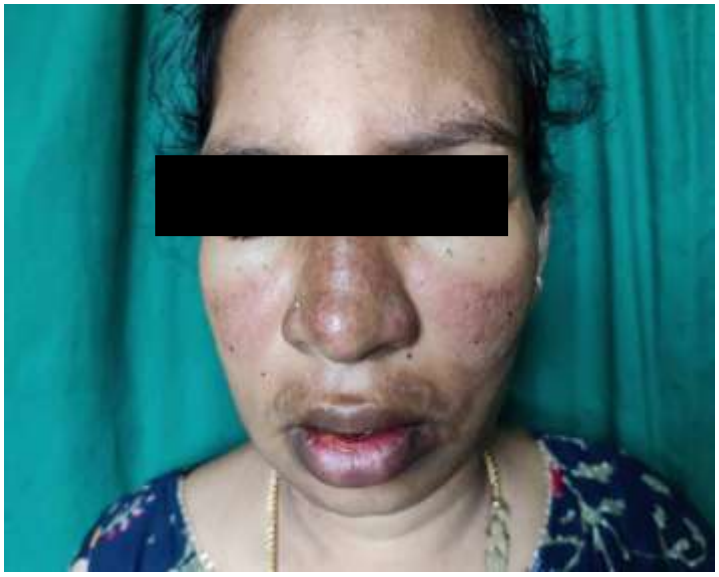

FIG 1b

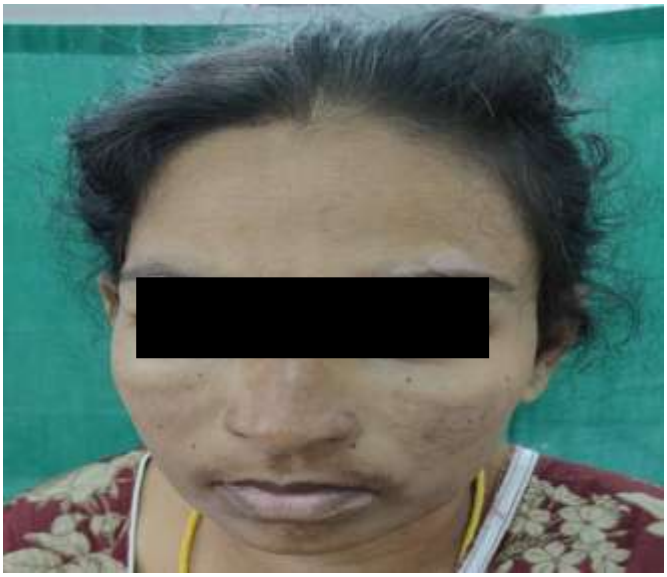

FIG 2a

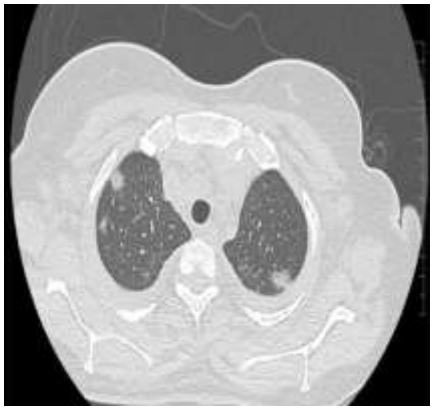

FIG 2b

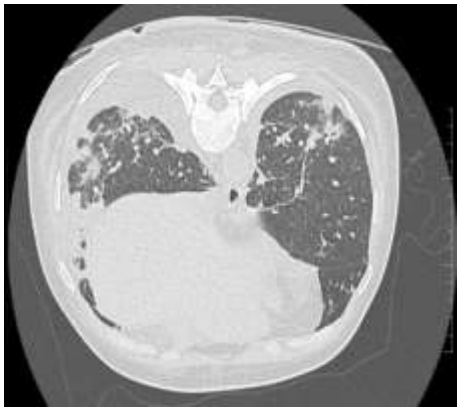

FIG 2c

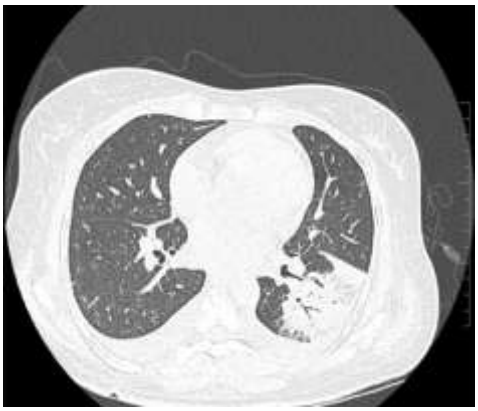

FIG 2 a – Bilateral mild pleural effussion

FIG 2b – Multiple groundglass density nodules scattered in bilateral lung parenchyma predominantly in upperlobes

FIG 2 c – Area of consolidation in superior segment of left lowerlobe.

Discussion:

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with multisystemic involvement. The condition has several phenotypes, with varying clinical presentations from mild mucocutaneous manifestations to multiorgan and severe central nervous system involvement .Several immunopathogenic pathways play a role in the development of SLE several genetic, immunological, endocrine, and environmental factors play a role in the etiopathogenesis of SLE.(2) Some gene mutations that are rare but are considered very high risk for the development of SLE include deficiencies of early complement components C1q, C1r, C1s (>90% risk), C4 (50%), C2 (20%), and TREX1. Some of the other genes associated include HLA-DRB1, HLA-DR2, HLA-DR3, HLA-DRX, TNFAIP3, STAT-4, STAT-1, TLR-7, IRAK1/MECP2, IRF5-TNPO3, ITGAM , Female sex and hormonal influence are significant

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risk factors for SLE. Several drugs have been implicated in causing a lupus-like phenomenon by causing demethylation of DNA and alteration of self-antigens. While procainamide and hydralazine have the highest incidence of causing drug-induced lupus, Ultraviolet rays and sun exposure lead to increased cell apoptosis and are well-known triggers for SLE.(2) Several viral infections have been implicated, and the underlying mechanism is thought to be molecular mimicry. Antibodies against Epstein-Barr virus (EBV) are more prevalent in children and adults with SLE

SLE predominantly affects women of childbearing age, with a female to male ratio of 9 to 1. The risk, however, decreases after menopause in women, Age also plays a vital role in SLE, and although the disease is more common in childbearing age in women, it has been well reported in the pediatric and elderly population(3). SLE is more severe in children SLE in children tends to be more severe than in adults, with a high incidence of malar rashes, nephritis, pericarditis, hematologic abnormalities, and hepatosplenomegaly.

Pulmonary manifestations of SLE are wide-ranging and can include disorders of the lung parenchyma (such as interstitial lung disease and acute pneumonitis), pleura (resulting in pleurisy and pleural effusion) and pulmonary vasculature [including pulmonary arterial hypertension (PAH), pulmonary embolic disease, and pulmonary vasculitis] & shrinking lung syndrome. Acute lupus pneumonitis is a rare manifestation of SLE that has been reported to occur in 1– 4% of patients . Clinically, acute lupus pneumonitis presents in the context of a systemic flare of SLE in addition to dyspnea, cough (including hemoptysis) and pleuritic chest pain(4). Fever is commonly associated with the acute presentation, thus making it a clinical challenge to differentiate from infection. in lung histology in acute lupus pneumonitis , lymphocytic infiltrates and alveolar damage with associated interstitial edema have been reported in both lung biopsy samples and at post-mortem assessment(4).

Acute lupus pneumonitis is a rare manifestation of systemic lupus erythematosus with clinical presentation similar to pneumonia, explaining its misdiagnosis leading to diagnostic delay.(4)Acute lupus pneumonitis must be suspected in patients who are non responsive to conventional treatment for pneumonia and when extrapulmonary characteristics of systemic lupus erythematosus are noted.

Treatment:

Systemic corticosteroids (either high dose oral or pulsed iv) plus either cyclophosphamide, rituximab ,mycophenolate mofetil ,azathioprine , possibly IV immunoglobulin.

Treatment Given

She was treated with high dose pulse steroids - I.V. Methylprednisolone for 3 days (10 mg/kg/day) followed by oral Prednisone (1 mg/kg/day), Hydroxychloroquine (4 mg/kg/day) and parenteral antibiotics. There was clinical & biochemical improvement evident by an improved SLEDAI of 12

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