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Fulminant antiphospholipid antibody syndrome complicated by *Aspergillus* tracheobronchitis

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ABSTRACT

Aspergillus fumigatus is a filamentous mold that causes infections in patients who are inmmunocompromised. We report a case of *Aspergillus* tracheobronchitis in fulminant systemic lupus erythematosus case. Diagnosis with more invasive diagnostic procedures & aggressive antifungal therapy is indicated at early stage.

1. Introduction

Pleuropulmonary infections are important cause for death in lupus patients [1]. Invasive aspergillosis most commonly presents as pulmonary disease; pneumonia/lung abscess. The risk factors for aspergillosis in SLE are high grade disease activity, neutropenia and prolonged use of immunosuppressives.

Case series by Katz et al. reveal that only 23 cases have been reported in literature, of which only one male patient survived [2]. In a review of 379 treated aspergillosis cases, by Denning et al., none had SLE [3]. The role of *Aspergillus* tracheobronchits in causing morbidity and mortality in systemic lupus erythematosus is rarely described [4]. We report a case of 31 year old man with fulminant systemic lupus erythematosus complicated by *Aspergillus* tracheobronchitis.

2. Case

31 year old man with occupation as an accountant was admitted with history of fever, rash and polyarthralgia for the past 3 months. The day of hospital admission being 0 day, and all other dates are given in reference to this fixed point. He had dry cough which was recent in onset. Evaluation at other centers showed pancytopenia and he had undergone antibiotic treatment and few days of antitubercular therapy (ATT). Patient examination revealed pallor, oral ulceration, candidiasis and subconjuctival hemorrhages along with maculopapular rash over trunk, legs and high grade fever. He presented with tachycardia and tachypnea. Central nervous system (CNS) examination was conscious, slightly irritable with no neuronal deficits and respiratory system had crackles at basal region (Fig. 1).

Laboratory investigations results showed hemoglobin level 9.1 gm%, total leukocyte count 2100 cell/mm^3 and erythrocyte sedimentation rate 80 mm/h. Peripheral smear showed pancytopenia. SGOT, SGPT, ALP, LDH as 299, 75, 220, 2312 U/L respectively and C reactive protein level 24 mg/L. Further HIV, Hepatitis Ag, Rheumatoid factor, Widal, VDRL, mantoux, brucella agglutination tests were negative. Blood and urine culture remained sterile. Urine protein was 3+, Echocardiography was normal and abdominal ultrasonography showed hepatosplenomegaly.

On the 2nd day he had two episodes of seizures & restlessness and brain Magnetic Resonance Imaging (MRI) was performed (Fig. 2).

Treatment was started with intravenous methyl prednisolone followed by cyclophosphamide with piperacillin tazobactum and phenytoin.

Anti-nuclear antibody test was positive with 1:2500 homogenous pattern and Anti dS DNA was positive. Cardiolipin antibody IgG was 18.4 GPL U/ml and IgM 8.9 MPL U/ml positive. Skin biopsy showed discontinuous weak granular band, strong IgM band and blood vessel deposit suggesting Systemic Lupus

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erythematosus. Bonemarrow was hypercellular with good cell trait and increase myeloid precursors and reactive marrow changes.

6–13 days later he developed breathlessness, cough and hypoxemia with chest infiltrates and noticeable decline in hemoglobin, white blood corpuscles and platelet count (Figs. 3 and 4).

He was treated with G-CSF, packed cell and platelet transfusion and antibiotics; meropenem, teicoplanin and oral steroids.

During 3rd week patient developed deep vein thrombosis of left leg which was treated with low molecular weight heparin. Steroid induced hyperglycemia was treated with insulin and oral candidiasis was treated with intravenous fluconozole. Improvement in hematological parameters was noticed with treatment. Fever, breathlessness, cough with moderate expectoration, tachypnea and restlessness persisted. Sputum for bacterial culture remained sterile and smears for acid fast bacilli were negative. Bronchoscopy showed whitish mucous patches occupying lateral wall of left main bronchus and apico-posterior orifice of upper lobe. Mucosal bleeding on touch was present.

Both sputum and bronchial lavage sent to mycology laboratory for culture investigation were inoculated on sabourauds dextrose agar (Hi-media Laboratories Ltd., Mumbai) and incubated at 28 °c which grew a moderately rapidly growing fungus with flat, powdery, blue green colonies consistent with *Aspergillus fumigatus* (Fig. 5).

Bronchial brush cytology showed numerous epithelial cells along with acute branching septate fungal hyphae suggestive of Aspergillosis. He was treated with Amphotericin B for 4 days; condition rapidly deteriorated and patient succumbed to death.

The final etiological diagnosis was Systemic Lupus Erythematous (SLE) of multiple systems; with hematological involvement like pancytopenia, neurological involvement indicating seizure, central nervous system vasculitis, renal involvement with lupus nephritis, Antiphopholipid antibody syndrome with venous thrombosis and positive cardiolipin antibody followed by *Aspergillus* tracheobronchitis due to *A. fumigatus*.

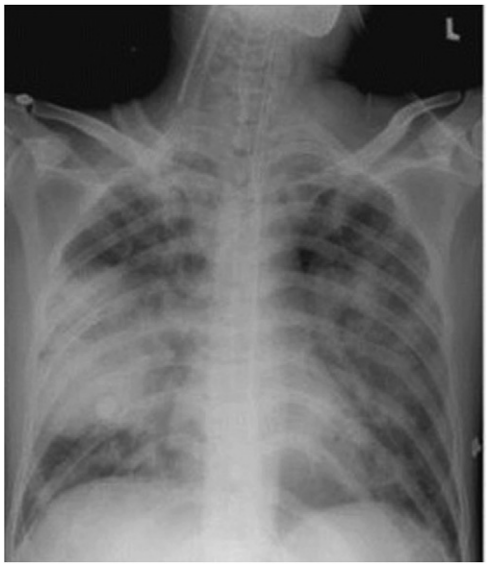

Fig. 3. X-ray showing rapid worsening of chest infiltrates over 4–5 days after admission.

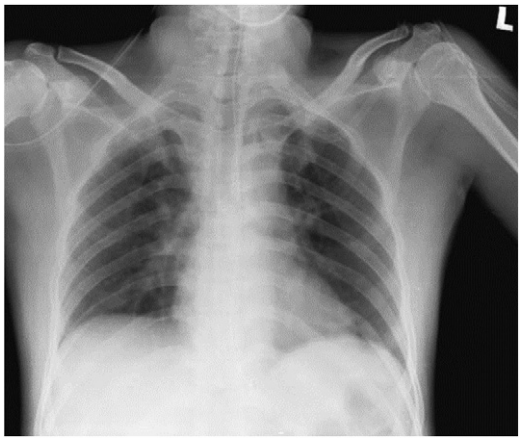

Fig. 1. Chest X-ray image on day of admission.

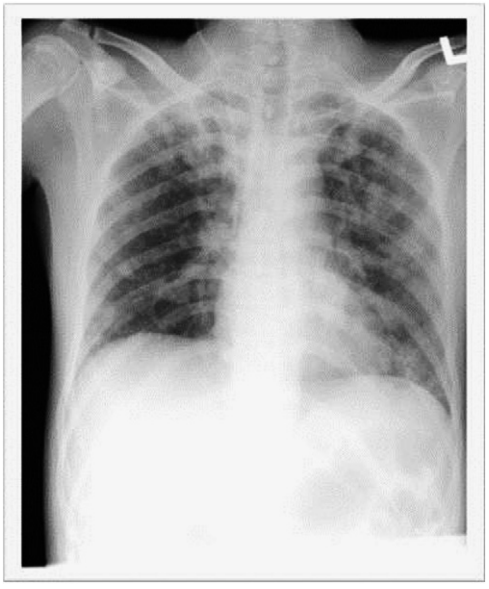

Fig. 4. Chest X-ray on 10th day of admission.

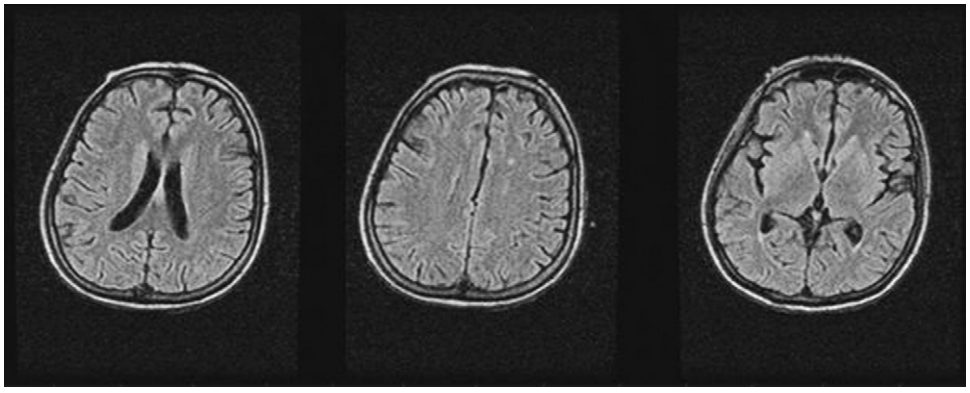

Fig. 2. MRI showing features of vasculitis of left fronto temporal region.