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CASE REPORT

Life Threatening Pneumonia in Systemic Lupus Erythematosus

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ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune connective tissue disease characterized by multisystem involvement. Pulmonary manifestation of SLE varies from pleuritis to severe parenchymal involvement as pneumonitis mimiking as acute respiratory distress syndrome (ARDS). Other pulmonary manifestations of SLE include pulmonary artery hypertension, diffuse alveolar haemorrhage, pulmonary embolism, acute reversible hypoxaemia, and shrinking lung syndrome. We present the case report of a young woman who presented with severe lupus pneumonitis with central nervous system and renal involvement.

Case: A 20 yr female presented with chief complaints of mild grade fever and non productive cough since 1 month for which she was being treated by a local physician as a case of lower respiratory infection in form of oral antibiotics nature of which were not present. She developed acute onset of breathlessness with left sided chest pain since 4 days which was progressive in nature which brought her to Intensive care unit. Past history revealed that she was a known case of seizure disorder and on some anti-epileptics for 5 years nature of which was not known. She also gave history of NSAIDS intake occasionally for recurring non specific arthralgia since 5 yrs.

On examination patient was afebrile, conscious and oriented to time, place and person with stable vitals and dyspneic with respiratory rate of 30/min. General examination revealed absence of raised Jugular venous pressure, pallor, cyanosis, clubbing, icterus or palpable lymph nodes.

On systemic examination there were crepitation in left lower zone in left lung. Rest of systemic examination were normal. On investigating arterial blood gas (ABG) analysis revealed partial pressure of oxygen (PaO_2) of 61 mm Hg, partial pressure of carbon dioxide ($PaCO_2$) of 24 mm Hg, pH of 7.387, hyponatremia with sodium level of 107 mmol/l. Her Complete blood count (CBC) revealed total leucocyte count (TLC) of 26,400,with differential leucocyte count (DLC) of neutrophils (N) 82%, with hemoglobin (Hb) of 8.2 g/dl. Her liver and renal function were within normal limits.

Her chest radiograph revealed a left lower lobe consolidation. A provisional diagnosis of lower lobe pneumonia was made and she was started on broad spectrum antibiotic coverage

comprising of Injectable (Inj) Cefaperazone sulbactum, Inj. Vancomycin and Inj. Azithromycin. Her sputum and blood cultures were sent.

On day 2 of admission, she got more dyspneic and chest radiograph revealed involvement of her right lower lobe in addition to left lower zone. She also had mild fever. Her CBC next day showed TLC of 25900/mm^3, DLC with neutrophils of 79%. Her ABG revealed PaO_2-56 mm Hg, $PaCO_2$-19 mm Hg, pH-7.464.

Due to deteriorating ABG findings, she was intubated. She was induced with Thiopentone, fentanyl and long acting muscle relaxant vecuronium in appropriate doses and put on ventilator on assist control mode. Her blood gases improved subsequently. Her endotrachael tube aspirate cultures were taken aseptically and were sent for culture, sensitivity and Gram's staining. She was maintained on continuous relaxants and appropriate sedation. Apart from routine tests collagen profile of the patient was sent for investigations.

Next day her chest radiograph deteriorated further with bilateral involvement of upper, middle and lower lung fields .Her collagen profile revealed ANA-579.7 U/ml,(N <50), Anti –dsDNA-254.5 U/ml(N<25) positive in high titres. However her anti phosholipid and anti cardiolipin antibodies were found to be negative. Thus, a diagnosis of acute lupus pnemonitis was made.

She was instantly put on pulse methyl prednisolone 1g/day for 5 days. Her chest X-ray improved drastically next day and continued to do so for next 3 days (Fig. 1). Broad spectrum antibiotics was continued to prevent nosocomial infections. We continued to ventilate our patient on assist control mode.

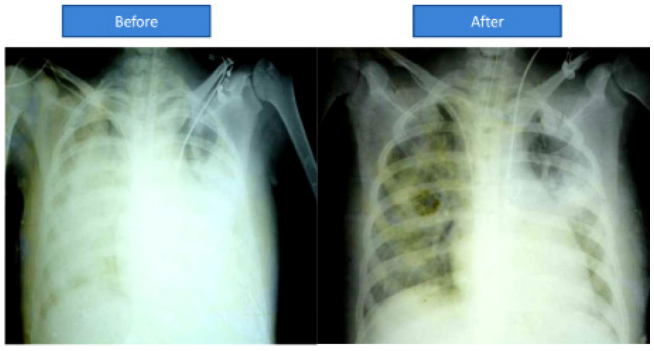

Fig. 1: Xray chest before and after use of methylprednisolone

After 5 days she was switched to oral tablet prednisolone 60 mg/day. Rest of the treatment continued.

But, after a week of treatment, she started to develop high grade fever with increase in TLC counts. She also developed hypotension and was put on appropriate vasopressors in judicious dose. Her endotracheal aspirates showed gram negative organisms which was later on found to be *Pseudomonas aeruginosa* which was sensitive to *Piperacillin tazobactum* and Gatifloxacin. She also developed ARDS with Pao_2/FIO_2 ratios well below 200 and compliance between 8 to 10 ml/cm water. We had to change her ventilator mode to Pressure controlled ventilation due to decreased lung compliance. Her urinary output also decreased gradually. On account of sepsis the patient unfortunately succumbed.

We collected her renal and lung biopsy specimens post mortem. Biopsy report confirmed lupus nephritis and lupus pnemonitis respectively.

DISCUSSION

Pulmonary manifestations of SLE includes pleuritis, pneumonia, pulmonary embolism, pneumothorax and pulmonary hemorrhage[1,2]. Among various pulmonary manifestation of SLE, lupus pneumonitis is life threatening. It may present at any stage in natural history of SLE. With a prevalence ranging from 1-10% in different series its mortality is high up to 50% [2,3]. Pathogenesis is not clear, but the pulmonary complications are thought to be the result of an immune complex mediated injury to pulmonary vasculature. Diagnosis remains a challenge because of varied presentation. Therefore high index of suspicion is needed in patients presenting with clinical manifestations of lupus. HRCT thorax can be done to know the extent of involvement. Blood and sputum cultures are necessary, and bronchoscopy or open-lung biopsy may be needed to exclude other conditions, such as pneumonia, alveolar hemorrhage, and other acute pulmonary processes that resemble lupus pneumonitis clinically and radiographically[4]. Lung biopsy reveals acute alveolar wall injury, alveolar hemorrhage, alveolar edema, hyaline membrane formation, and immunoglobulin and complement deposition. There have been no controlled trials addressing treatment of lupus pneumonitis.However, high dose corticosteroids. (I/V or Oral) is recommended. Role of other immunosuppressant not clear. Though they may be added if there is no response to corticosteroid therapy within 72 hours. In some case reports Intravenous immunoglobins (IVIg) was also given.

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