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Abruptio placentae with type II respiratory failure secondary to acute interstitial pneumonia responsive to steroids.

Muhammad Umer Salim
Patel Hospital

Syed Muhammad Mustahsan
Aga Khan University

Anum Fatima
Patel Hospital

CASE REPORT

Abruptio Placentae with Type II Respiratory Failure Secondary to Acute Interstitial Pneumonia Responsive to Steroids

Muhammad Umer Salim[1], Syed Muhammad Mustahsan[2] and Anum Fatima[1]

Abstract

Acute Interstitial Pneumonia (AIP) is categorized as Idiopathic Interstitial Pneumonia (IIP), in which the cause is unknown. A young female of 22 years presented in 34 weeks gestation with abruptio placentae (AP) and underwent Lower Segment Caesarian Section (LSCS) for AP. It progressed to type II respiratory failure secondary to AIP on 4th day post-surgery. It remained unresponsive when treated with noninvasive ventilation (NIV-BiPAP) along with antibiotics. Later, a trial treatment of pulse therapy of Methylprednisolone was executed on 7th day post-surgery which resulted in dramatic improvement in symptoms. It is uncommon to have type II respiratory failure secondary to AIP, and it is rarely steroid responsive.

Key Words: *Steroid responsive. Acute interstitial pneumonia. Abruption placentae. Methylprednisolone. Type II respiratory failure.*

INTRODUCTION

The term "acute interstitial pneumonia" (AIP) represents a group of lung disorders of unknown etiology, characterized by diffuse parenchymal lung involvement causing rapid onset of respiratory failure with potential to develop pulmonary fibrosis, occurring in patients without preexisting lung diseases.[1,2] This type of pneumonia differs from chronic forms by its sudden onset and rapid course of the disease.[3] AIP frequently starts with flu-like illness and progresses to hypoxemia and respiratory failure in most of the patients, requiring mechanical ventilation, with a few patients recovering completely with mortality rate of >70% in 3 months.[1,4] It is proved that an early intervention, such as an aggressive diagnostic approach, mechanical ventilation with lung-protective strategy, and the early institution of immunosuppressive drugs may improve clinical outcome in patients with AIP.[5]

The reason to report this case was the nature of AIP in this case that led to type II respiratory failure and its favorable response to steroid therapy.

Patients of AIP usually present with type 1 respiratory failure, unlike our patient; and almost all patients require mechanical ventilation, unlike our patient, which was maintained on NIV-BiPAP. Moreover, they are non-responsive to the systemic steroids unlike our patient which responded to the steroids.

[1] *Department of Critical Care, Patel Hospital, Karachi.*
[2] *Resident at Emergency Medicine, The Aga Khan University Hospital, Karachi.*

Correspondence: Dr. Syed Muhammad Mustahsan, Resident at Emergency Medicine, The Aga Khan University Hospital, Stadium Road, Karachi.
E-mail: mustu198@gmail.com

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CASE REPORT

A 22-year female was shifted to critical care unit from obstetrics ward, with sudden development of shortness of breath on 4th day post-LSCS. She was already transfused with 4 units of packed red blood cells (PRBC) within those 4 days.

Chest X-Ray, Echocardiography and C.T Scan Chest with Contrast were done. Echocardiography was reported normal, whereas chest X-ray showed bilateral lung infiltrates with alveolar shadowing, as shown in Figures 1a and 1b. CT scan showed bilateral minimal pleural effusion with multiple patchy areas of ground-glass opacities and consolidation with air bronchogram (Figure 2), suggestive of AIP.

Patients with AIP present with acute onset of breathlessness, fever and cough followed by type 1 (hypoxic) respiratory failure, unlike our case, who presented with type II (hypercapnic) respiratory failure. Her blood cultures and H1N1 for swine flu were negative. She neither filled in the history of transfusion-related acute lung injury (TRALI), nor did she have any antigen exposure to label her as a case of Hypersensitivity Pneumonitis

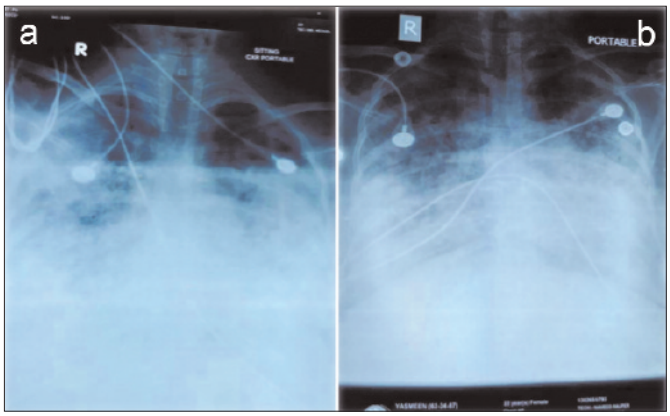

Figure 1: Chest X-ray showing bilateral lung infiltrates.

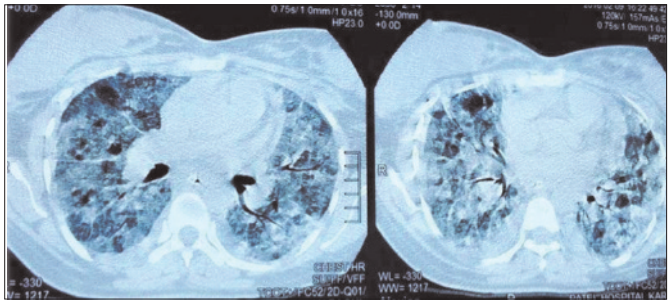

Figure 2: CT scan showing multiple, pately areas of ground-glass opacities and consolidation with air bronchogram.

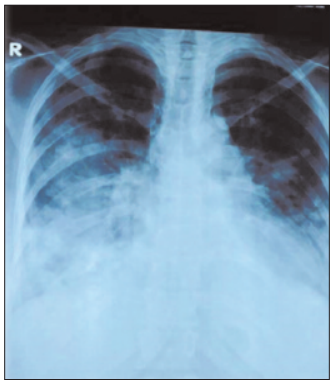

Figure 3: Chest X-ray showing marked improvement of bilateral lung infiltrates.

(HP), although she had pulmonary squeaks on auscultation, like patients with HP. Bronchoalveolar lavage could not be done as she was breathless and hypoxic.

She was given intravenous pulse therapy of Methyl-prednisolone 1g for 5 days, and responded impressively after the first dose of steroids, as shown in (Figure 3) with vanishing of lung infiltrates. She visited in the outpatient department twice for obstetric follow-up after getting discharged from the hospital. Her breathlessness had been decreased since the day of discharge from the hospital.

DISCUSSION

AIP resembles acute respiratory distress syndrome (ARDS) radiologically and physiologically; and is considered to represent the small subset of patients with idiopathic ARDS.[1] It is unique as it has a high initial case fatality ratio but, potentially, a more favorable long-term prognosis for survivors of the initial insult.[2] The exact pathogenesis of AIP is still obscure. The histological hallmark of AIP is interstitial fibrosis and oedema with type II pneumocyte hyperplasia.[3] Diagnostic assessment involves chest radiography, high-resolution CT (HRCT), lung function tests, lung biopsy analysis and serological tests. Exact diagnosis of AIP is essential due to its high mortality ratio.[4] CT assessment is potentially helpful in predicting patient prognosis in AIP, regardless of the degree of physiologic abnormality.[6] However, AIP has a relatively worse prognosis, regardless of CT findings.[7] Unfortunately, there is less data available on the role of steroid therapy in AIP; and if the steroid trial fails then cyclosporine (CsA) with corticosteroids has shown some efficacy in steroid-resistant cases.[8]

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