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Case Report

Acute postpartum pleural effusion causing distress for patient and obstetrician

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ABSTRACT

A 31-year-old Gravida 5 Para 2 Live 2 Abortion 2, without comorbidities, underwent spontaneous vaginal delivery at term. She was asymptomatic in postpartum and had puerperal sterilization under low risk on postnatal day 4. After fourteen hours of surgery, she experienced an acute onset of breathlessness, tachypnea, and orthopnoea. Workup revealed right-sided pleural effusion filling three-fourths of the cavity with consolidation on chest X- ray. Therapeutic thoracocentesis was performed draining 600 ml of straw-coloured fluid. She was started on the Piperacillin tazobactam combination. Due to the repeated collection and persistent symptoms, a continuous intercostal drain was placed after 4 days. Due to persistent fever spikes, antibiotics were stepped up to Linezolid and Meropenem. A negative result on the Mantoux test, CBNAAT, and IGRA test was obtained. ANA profiling revealed the presence of non-specific KU antibodies. Symptomatic improvement was noted, and the ICD was subsequently removed after 6 days of insertion. Pregnancy is an immunosuppressive state. Rapid reversal of this state in postpartum results in a flare-up of quiescent infection. Even auto-immune diseases flare up in postpartum. Understanding this phenomenon of immune reconstitution syndrome and its impact will help in the management planning of postpartum women without dilemmas.

Keywords: Pregnancy, Postpartum, Pleural effusion, Immune reconstitution syndrome

INTRODUCTION

Pregnancy is characterized by physiological immunosuppression with a decrease in proinflammatory host responses meant for tolerating growing allogenic fetuses.[1] Following delivery reversal of these changes takes place. This response may result in an acute exacerbation of latent infection or autoimmune disorder in the postpartum woman. Here we report a case of acute postpartum pleural effusion in a previously asymptomatic postnatal woman.

CASE REPORT

Mrs. X, 31-year-old, gravida 5 para 2 live 2 abortions 2, staff nurse by occupation, booked with us for pregnancy and got admitted with labor pain at term. She doesn't have any significant medical illness in the past. She had a spontaneous vaginal delivery and delivered an alive-term male baby. She was asymptomatic during the postpartum period and underwent puerperal sterilization on the 4th postnatal day under spinal anesthesia with low risk (ASA-I category). Her intraoperative and immediate post-operative periods were uneventful. After fourteen hours of surgery, she had a sudden onset of dry cough, breathlessness, orthopnea, right-side chest pain on inspiration, and coughing. There was no associated fever or hemoptysis. On examination, she was tachypneic, with tachycardia with a pulse rate of 140 beats per minute, blood pressure of 110/70 mmHg, and respiratory rate of 40/min. but her saturation remained stable at room air (SpO_2: 98%). There was no pedal edema and her jugular

venous pressure was not raised. On auscultation, there was decreased breath sound intensity in the right inter and infrascapular area. The abdomen was soft.

Complete blood count showed hemoglobin - 9.9 g/dl; total leucocyte count - 18400 cells/cu.mm (neutrophile 83%); platelet count - 317×1000/ul. Her liver function test and renal function tests were within normal limits. An electrocardiogram was done which showed sinus tachycardia. 2D echo showed a normal study with an ejection fraction of 60% (hence postpartum cardiomyopathy was ruled out). Chest X-ray revealed homogenous opacity in the right upper, middle, and lower zone, filling three-fourths of the pleural cavity (Figure 1).

Ultrasound-guided thoracocentesis was done for the right side moderate pleural effusion and drained 600 ml of straw-coloured fluid which was sent for fluid analysis. She was started on higher antibiotics Piperacillin tazobactam and Metronidazole for gram-negative, gram-positive, and anaerobic coverage. The patient showed a moderate improvement clinically following thoracocentesis. Pleural fluid analysis revealed predominantly neutrophils (70%), lymphocytes (20%), and macrophages (10%) with few reactive mesothelial cells with features suggesting acute inflammatory pathology. High protein levels (3.1 g/dl) pointed towards exudate. Though very low glucose (10 mg/dl) in pleural fluid was towards bacterial etiology, there were high adenosine deaminase (ADA) levels (47 U/l). To rule out tuberculosis, an interferon-gamma release assay (IGRA) and cartridge-based nucleic acid amplification test (CBNAAT) were sent.

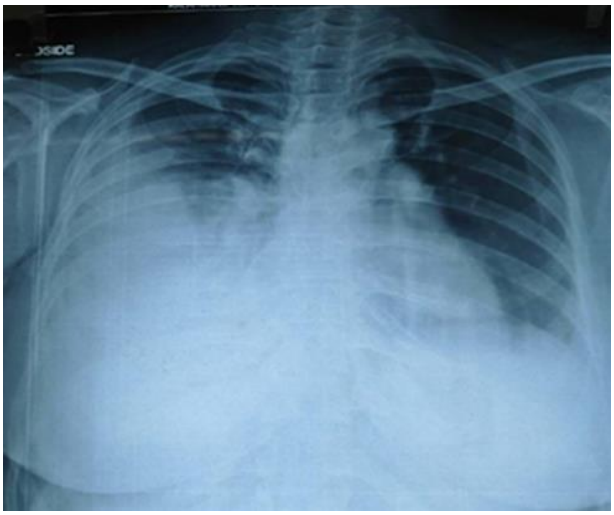

Figure 1: Chest X-ray AP view showing homogenous opacity in the right upper, middle, and lower zone.

On the next day, the patient started having fever episodes which were intermittent and not associated with chills and rigors, although there was a notable decrease in complaints of cough and breathlessness. Temperature spikes were reaching 101˚ with mild tachycardia PR: 112/minute and tachypnoea with stable BP of 100/70 mmHg and SpO_2 maintaining at 97% at room air. She continued to have decreased breath sounds in the infrascapular area. Fever investigations for dengue, malaria, scrub typhus, typhoid, and COVID antigens were negative. The Mantoux test did not reveal significant induration. D-dimer was <0.22 within the normal range. The patient remained on the same antibiotic regimen.

There was an ongoing fever and exacerbated breathlessness on the following day (the third day of symptom). A follow-up chest X-ray revealed a reappearance of pleural effusion (Figure 2).

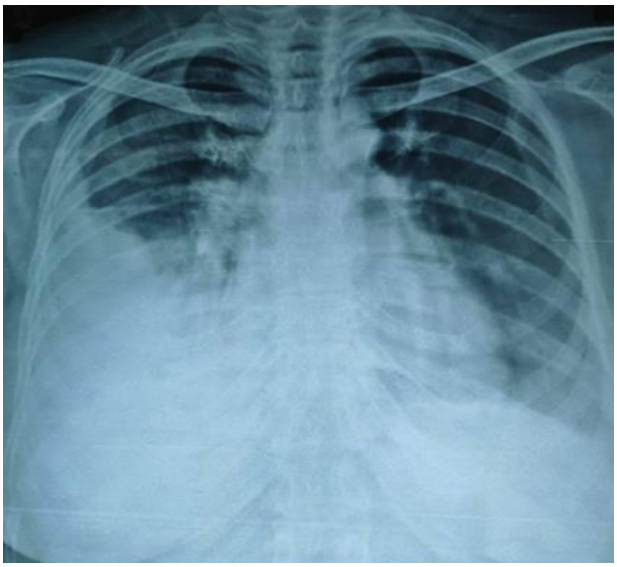

Figure 2: Chest X-ray AP view showing reappearance of pleural effusion.

Repeat pleural tapping was performed and 150 ml straw-colored fluid was drained. Pleural fluid culture, urine culture, blood culture, and high vaginal swab showed no growth. On the fifth day, the patient had persistent fever spikes, along with cough and breathlessness. As a repeat chest x-ray indicated persistent pleural effusion and ultrasonography (USG) thorax showed pleural effusion with strands, a decision was made to insert a continuous intercostal drain (ICD) and 200 ml of straw-coloured fluid. Antibiotics were escalated to Linezolid and Meropenem given non-response for 3 days and high fever spikes. Daily drain collection was 200-400 ml, with intermittent clamping. Fever began to subside.

Given the previous two abortions and the sudden onset of recurrent pleural effusion in a previously asymptomatic woman, anti-nuclear antibody (ANA) profiling was done to rule out autoimmune disorders. ANA was reported negative except for the presence of non-specific Ku antibodies which are considered insignificant. The rheumatoid factor also turned out to be negative. Tuberculosis workup of CBNAAT and IGRA were negative. After thorough consideration of all these results, we have effectively ruled out autoimmune disorders, tuberculosis, and malignancy. We came to a conclusion

that points toward a bacterial etiology, confirming the diagnosis as parapneumonic effusion.

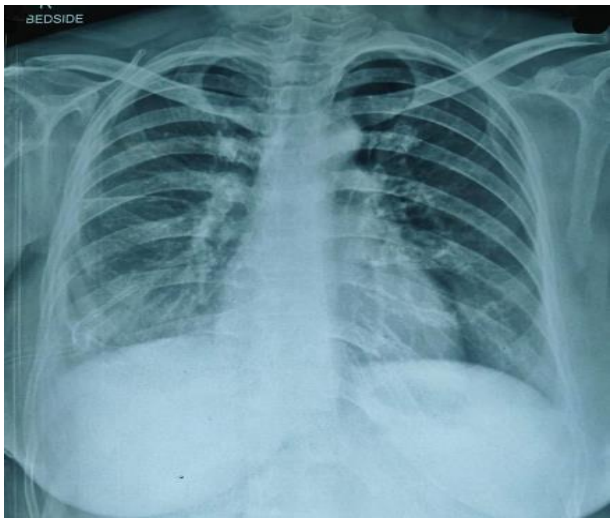

Figure 3: Chest X-ray AP view showing complete resolution of effusion.

Patient assessment for the following three days revealed significant symptomatic improvement. Subsequent chest X-rays indicated complete resolution of effusion (Figure 3).

ICD was removed on the fifth day of placement and she was discharged. She was on follow-up for 3 months at regular intervals, when she did not develop any recurrence of symptoms.

DISCUSSION

Explosive pleuritis/acute pleural effusion is a profound pleural space infection in the setting of pneumonia with the dramatic progression of pleural disease within a matter of hours, generally in less than 24 hours.[2,3] Since her chest X-ray was normal before puerperal sterilisation and she developed a large pleural effusion within fourteen hours of surgery, we were initially clueless about her diagnosis. As pneumonia, congestive cardiac failure, malignancy, and pulmonary embolism are the common causes of pleural effusion in adults, we did our workup toward them. We thought of autoimmune connective tissue disorders (systemic lupus erythematosus, and rheumatoid arthritis), and drug-induced effusions as an etiology in her case and ruled them out based on history and investigations. Severe pre-eclampsia is also reported to cause massive pleural effusion which was not in her case. Benign pleural effusion can happen in postpartum as a result of physiological changes in pregnancy (increased blood volume, decreased colloid osmotic pressure, deceased pleural fluid drainage, and diaphragm movement due to a gravid uterus) and repeated Valsalva manoeuvres during delivery.[4-6] But women are generally asymptomatic in such benign pleural effusion and it is typically detected through chest radiography or USG. They usually resolve spontaneously within one week after delivery.

The concept of immune reconstitution syndrome and exacerbation of infections after pregnancy was brought to the limelight by Singh and Perfect in 2007.[7] Usually, the concept of immune reconstitution syndrome is in the context of HIV infection and solid-organ transplantation. However, any situation involving a rapid change in immune status can result in immune reconstitution syndrome. An increase in progesterone, cortisol, norepinephrine, and 1,25-dihydroxyvitamin in pregnancy plays a major role in modulating immune responses. Due to the shift in the immunological system from T2 helper response in pregnancy to T1 helper response in the postpartum period, there is an enhanced proinflammatory response.[7] These changes predispose to bacterial, fungal, and viral infections in postpartum.[9] Autoimmune disorders that are exacerbated by a T1 helper response are suppressed in pregnancy and flare up during the postpartum period.[8]

In our case, the patient is a staff nurse by occupation working in the Emergency department of a government hospital, which involves constant exposure to a variety of pathogens. Though she was asymptomatic in pregnancy, during the phase of postpartum reversal of physiological changes, the patient developed parapneumonic effusion. Timely diagnosis and intervention with insertion of ICD coupled with escalation of antibiotics played a synergistic role in the comprehensive restoration of her overall health. Empirical broad-spectrum coverage is needed, as isolation of causative agents is often difficult from pleural fluid cultures. Knowledge of these changes in the immune system, immediate workup of the patient, and timely management helped in preventing catastrophe in our case.

CONCLUSION

Pregnancy is an immunosuppressive state. Rapid reversal of this state in postpartum results in a flare-up of quiescent infection. Even auto-immune diseases flare up in postpartum. Understanding this phenomenon of immune reconstitution syndrome and its impact will help in the management planning of postpartum women without dilemmas.

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