Case Report

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Management of Post- Partum Pulmonary Edema in an Intensive Care Unit of a Peripheral District Hospital: A Case Report

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Abstract

Pulmonary edema is an infrequent yet severe complication that may arise in the postpartum period following normal vaginal delivery. Prompt recognition and treatment are essential to prevent further deterioration. Here, we present a case involving a 23 year old primigravida, presented with shortness of breath and orthopnea on the fifth day after childbirth. Following diagnosis of postpartum pulmonary edema, the patient received subsequent management in the Intensive Care Unit. The patient's condition improved and patient was discharge. This case underscores the significance of coordinated care across various medical specialties and timely intervention in managing such conditions.

Keywords: Anesthesiologist; intensive care unit; post-partum vaginal delivery; pulmonary edema.

Introduction

Pulmonary edema is a potentially life-threatening condition characterized by accumulation of fluid in the lungs, leading to impaired gas exchange and respiratory distress.[1] This condition goes by various names such as postpartum heart failure, postpartum myocarditis, meadows' syndrome and postpartum cardiomyopathy.[2] It is an idiopathic cardiomyopathy occurring in the third trimester or up to 6 months post-partum and is seen most often in the first month postpartum.[3] Postpartum pulmonary edema has an overall incidence of only 0.08%.[4] It is rare in the postpartum period following normal vaginal delivery. However,

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when it does occur, it requires immediate management to prevent complications.

Case Report

A 23 year old, primigravida and with an uneventful antenatal period, presented at a peripheral hospital's emergency department on the fifth day after giving birth. She complained of worsening shortness of breath, coughing, abdominal pain, and difficulty breathing while lying flat. She had normal vaginal delivery with an episiotomy, resulting in a healthy baby girl weighing 3.5 kg, without any complications. The patient had no significant medical history or family history of chronic conditions such as hypertension, diabetes, or heart disease.

Upon examination, the patient had a Glasgow Coma Scale (GCS) score of 15 out of 15 and exhibited tachypnea (40 breaths per minute), tachycardia (119 beats per minute), and low blood oxygen levels (69%) while breathing on room air. Her blood pressure was 230/140 mmHg. Bilateral pitting edema was observed, alongside a normal body temperature. Lung auscultation revealed widespread crackling sounds on both sides.

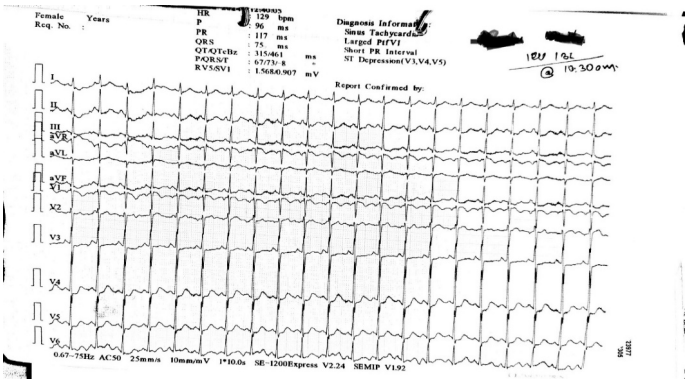

Figure 1: Sinus Tachycardia.

The patient was promptly transferred to the Intensive Care Unit (ICU), where oxygen supplementation was initiated at a rate of 10 liters per minute via a face mask. At this point, the patient's blood pressure was measured at 180/100 mmHg, pulse rate at 132 beats per minute, respiratory rate at 35 breaths per minute, oxygen saturation at 77%, temperature at 96.6 degrees Fahrenheit, and blood glucose level at 127 mg/dL.

Further investigations revealed arterial blood gas (ABG) findings consistent with respiratory alkalosis and hypoxemia. Electrocardiography (ECG) showed only sinus tachycardia (figure 1). A chest X-ray of fifth postpartum day demonstrated diffuse bilateral infiltrates suggestive of pulmonary edema (figure 2).

Bedside lung scanning revealed normal lung sliding with multiple B lines. Bedside echocardiography screening ruled out structural cardiac abnormalities or evidence of cardiogenic pulmonary edema. Additionally, bedside venous doppler of bilateral lower limbs was performed to exclude venous thromboembolism. Laboratory investigations (Table 1), including complete blood count, renal function tests, electrolytes, bleeding time, and clotting time, were within normal limits, except for a total white blood cell count of 16,900/cumm with 89% neutrophils. Slight elevations in serum glutamic pyruvic transaminase (SGPT) at 64 IU/L and serum glutamic oxaloacetic transaminase (SGOT) at 69 IU/L were noted, but they were deemed insignificant. Furthermore, the D-dimer test yielded negative results.

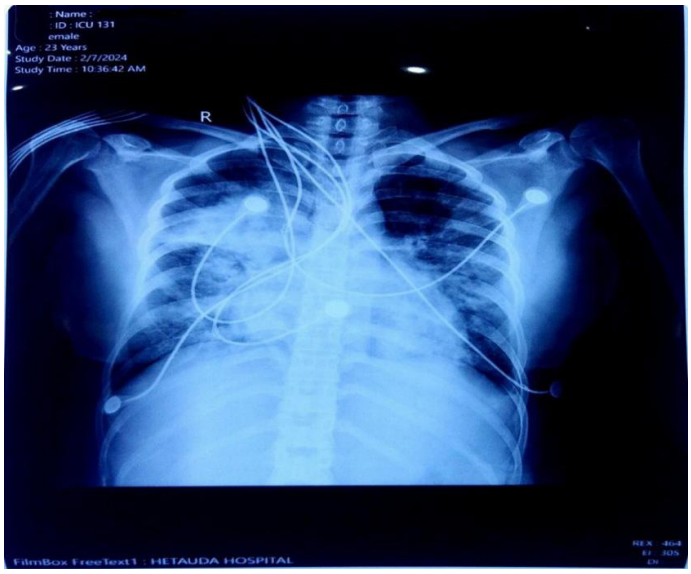

Figure 2: Bilateral Heterogeneous Opacity in both Middle and Lower Lobe.

Table 1: Investigations of the patient during the course of hospital stay.

Investigations	1st Day of admission	1st DOR	2nd DOR	3rd DOR
Hb (gm%)	12.9	10.4	11.2	11
Platelet count (/cumm)	3,53,000	3,31,000	3,01,000	3,00,000
Tlc (/cumm)	15,300	16,000	16,000	14,000
Neutrophil (%)	90	89	83	80
Lymphocyte (%)	8	8	15	10
Rbs (md/dl)	95	92	95	90
B. Urea (md/dl)	18	28	38	46
S. Creatinine (md/dl)	0.4	0.54	0.94	0.16
Sodium (mmol/l)	137	136	138	140
Potassium (md/dl)	3.8	3.9	4.19	5.0
ABG				
Ph	7.57	7.45		
po2	35	98		
pco2	20	32		
hco3	25.4	23		
Lactate	1.1	0.8		

***1st DOR (Day of Readmission) = 1st day of readmission i.e. 5 days after admission*2nd DOR= 2nd day of readmission**

*3rd DOR= 3rd **day of readmission**

Management

The patient was swiftly transferred to the Intensive Care Unit (ICU) under the supervision of an anesthesiologist for further management. She was given supplemental oxygen through a face mask at a high flow rate of 10 liters per minute to ensure her blood oxygen levels stayed above 92%. To alleviate pulmonary congestion and breathing difficulties, she received intravenous diuretics: a 40 mg dose of furosemide initially, followed by another 40 mg dose after 10 minutes, and then 20 mg twice daily. Additionally, she was administered GTN (glyceryl trinitrate) through an IV, starting with a 100 mcg bolus dose followed by a continuous infusion of 5 mcg per minute to manage her blood pressure, decrease heart load, and enhance oxygen levels.

Broad spectrum antibiotics (Piperacillin 4 gm + Tazobactam 0.5 gm) were also started, along with a prophylactic dose of Enoxaparin (40 units) injected subcutaneously. Hydrocortisone (100 mg three times daily) was administered intravenously, and nebulization with a mixture of salbutamol, ipratropium, and normal saline (in a ratio of 1:1:2) was done three times daily. Intravenous morphine (2 mg) injection was available for pain. lose monitoring of vital signs, fluid intake and output, and urinary output was initiated, and fluid intake was restricted to prevent further fluid overload. The patient's response to treatment was carefully tracked through repeated arterial blood gas analyses and chest X-rays. Over the following 48 hours, the patient's respiratory symptoms and oxygen levels gradually improved.

Outcome

The patient's clinical condition continued to improve with conservative management in the ICU. She was weaned off after 48 hours and transitioned of supplemental oxygen via face mask to nasal cannula. Diuretic therapy was gradually tapered, and the patient's fluid balance normalized. Patient was initiated on chest physiotherapy and incentive spirometry. Repeat chest X-ray showed resolution of pulmonary infiltrates (figure 3).

The patient was directly discharged from the ICU in the 8th postpartum day 2024-2-11 with stable vital signs and improved respiratory status (figure 4). She was counseled regarding the importance of follow-up visits and advised on measures to prevent recurrence. On further follow up after fifth day, her general examination, systemic examination and investigations were within normal range.

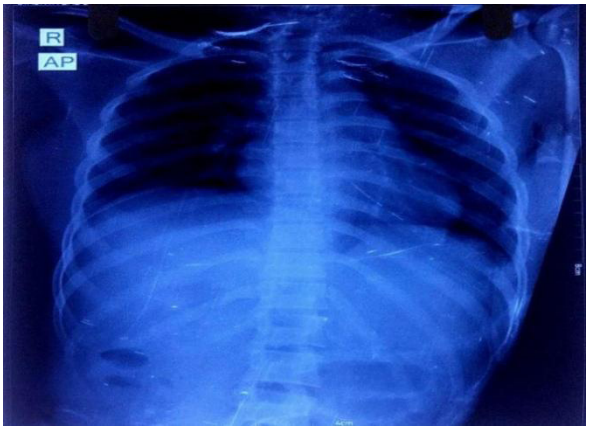

Figure 3: Normal chest x-ray of the patient at the time of discharge.

Figure 4: ICU monitor showing improve vitals of the patient at the time of discharge.

Discussion

Pulmonary edema following normal vaginal delivery is a rare but potentially serious complication that requires prompt recognition and management.[5] While the exact pathophysiology is not fully understood, it is believed to be multifactorial, involving fluid shifts, hemodynamic changes, and altered vascular permeability.[6] Postpartum pulmonary edema can be either cardiogenic (peripartum cardiomyopathy, pre-existing valvulopathies, myocardial ischemia, and pre-eclampsia causing heart failure) or noncardiogenic (iatrogenic fluid overload, excessive tocolytic use, thyroid disease, sepsis, and ARDS) in origin.[7] Studies have shown that there is an increased risk of pulmonary edema associated with cesarean and spontaneous preterm delivery. Our patient did not meet the criteria for any of the above causes. In study done by Kakogawa et al on Department of Obstetrics and Gynecology, St Marianna University School of Medicine, Kanagawa, Japan, patient was Managed with oxygen, diuretics, morphine and beta-blockers contrasting from our study where we used oxygen, diuretics, hydrocortisone and GTN infusion for management of the case.[8] A new position statement from a European Society of Cardiology working group on PPCM (Postpartum cardiomyopathy) has expanded the definition of the condition. It now describes PPCM as an idiopathic heart condition marked by heart failure caused by

decreased left ventricular function occurring towards the end of pregnancy or in the months post-delivery, with no other identifiable cause of heart failure.[9]

Kakogawa et al study reported that cause of heart failure in this patient was diastolic dysfunction during the third trimester of pregnancy however, in our study only echocardiography screening was done at the bedside.[8] As it is difficult to differentiate heart failure due to systolic dysfunction from that caused by diastolic dysfunction the finding of our echo screening was normal LVEF with no any cardiac abnormality due to lack of proper echocardiographic machine and probe as well as unavailability of cardiologist in our setting.

In Kakogawa et al study an elevated level of serum prolactin was found, as the 16-kDa cleavage product of prolactin as well as C1 inhibitor deficiency is a major contributor to PPCM.[8, 10] The deficiency of C1 inhibitor led to the onset of acute heart failure, marked by a combined dysfunction in both systolic and diastolic phases, attributed to the leakage of capillaries throughout the body.[11] However, we are unable to test serum prolactin and C1 inhibitor due to limited resources and unavailability of test in peripheral setting. A retrospective investigation studying BNP levels in pregnancy found that women who encountered adverse maternal cardiac events during this period had BNP levels exceeding 100 pg/mL.[12]

Although measuring serial BNP levels was helpful there are limited data available on the value of BNP levels when evaluating volume status during pregnancy, so in our study, we have not done serial BNP monitoring. In our case, multidisciplinary management involving anesthesiologists and obstetricians played a crucial role in the successful outcome. Prompt initiation of supportive measures, including supplemental oxygen, diuretic therapy, and GTN, helped alleviate symptoms and improve respiratory function. Close monitoring and serial assessments were essential in guiding therapeutic interventions and ensuring optimal patient care.

Summary

Pulmonary edema occurring after a regular vaginal delivery is uncommon yet can be a serious complication necessitating quick identification and comprehensive treatment. Anesthesiologists, given their proficiency in critical care and airway handling, hold a central position in managing such scenarios, especially in peripheral hospitals with constrained resources.

Conflict of Interest: None

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