Management of Post-Partum Pulmonary edema in an Intensive Care Unit (ICU) 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, who exhibited symptoms of shortness of breath, cough, abdominal pain, and orthopnea on the fifth day post-partum day. Following diagnosis of postpartum pulmonary edema, the patient received subsequent management in the Intensive Care Unit (ICU) of a Peripheral hospital. The patient's clinical condition improved, leading to discharge from the ICU on the eighth postpartum day. This case underscores the significance of coordinated care across various medical specialties and timely intervention in managing such conditions even in resource limited setting.

[**KEYWORDS**]

Pulmonary edema, postpartum, anesthesiologist, intensive care unit, vaginal delivery.

[INTRODUCTION]

Pulmonary edema is a potentially life-threatening condition characterized by the accumulation of fluid in the lungs, leading to impaired gas exchange and respiratory distress [1]. Pulmonary edema during pregnancy or the peripartum period, generally goes by various names such as Postpartum heart failure, postpartum heart disease, postpartum myocarditis, Meadows' syndrome, idiopathic myocardial degeneration associated with pregnancy, Zaria syndrome, and postpartum cardiomyopathy [2]. Peripartum cardiomyopathy 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]. Acute pulmonary edema during pregnancy and the postpartum period has an overall incidence of only 0.08% [4]. While it can occur in various clinical settings, including cardiac and renal disorders, it is rare in the postpartum period following normal vaginal delivery. However, when it does occur, it requires immediate attention and management to prevent serious complications.

[CASE PRESENTATION]

A 23-year-old, primigravida and with an uneventful antenatal period, presented at a peripheral hospital's emergency department on the fifth post-partum day. 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 saturation 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.

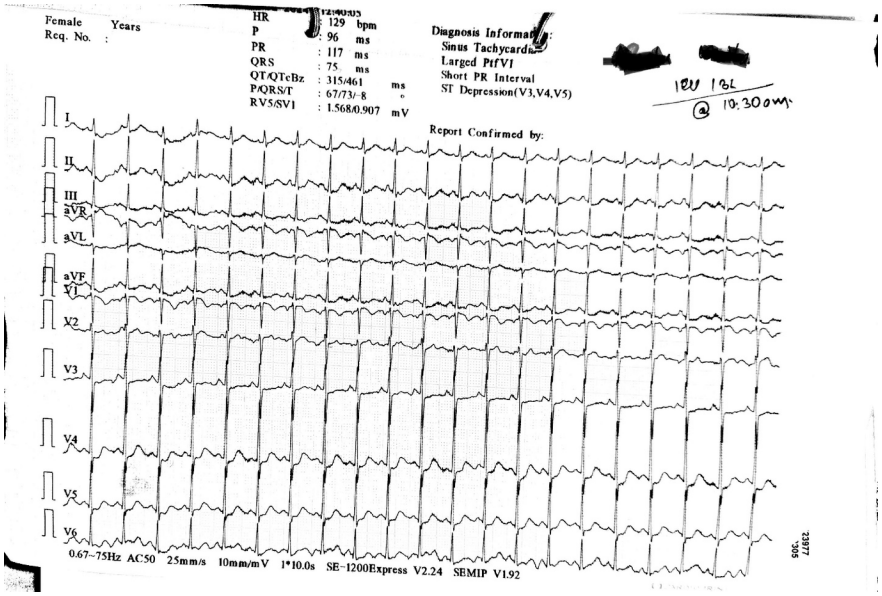

Figure 1: ECG of patient showing sinus tachycardia

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. A chest X-ray demonstrated diffuse bilateral infiltrates suggestive of pulmonary edema. Electrocardiography (ECG) showed only sinus tachycardia.

Bedside lung scanning revealed normal lung sliding with multiple kerley 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, including complete blood count, renal function tests, electrolytes, bleeding time, and clotting time, were all 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.

Test Time	2024 02 08 07:16:49		
Patient ID	131		
Cart.Lot	02305004CAFB		
Operator ID	admin		

131
10/25

Patient Information

Sample ID	20240208002		
Temperature	37.0		℃
FIO2	21		%

Blood Gas Values

pH	7.576	↑	
pO2	35	↓	mmHg
pCO2	28.0	↓	mmHg

Oximetry Values

Hct	34	↓	%
tHb(est).r	11.5		g/dL
sO2(est).r	77		%

Electrolyte Values

Na+	142		mmol/L
K+	2.7	↓	mmol/L
Ca++	0.98	↓	mmol/L
Cl-	106		mmol/L
Ca++(7.4).r	1.06		mmol/L

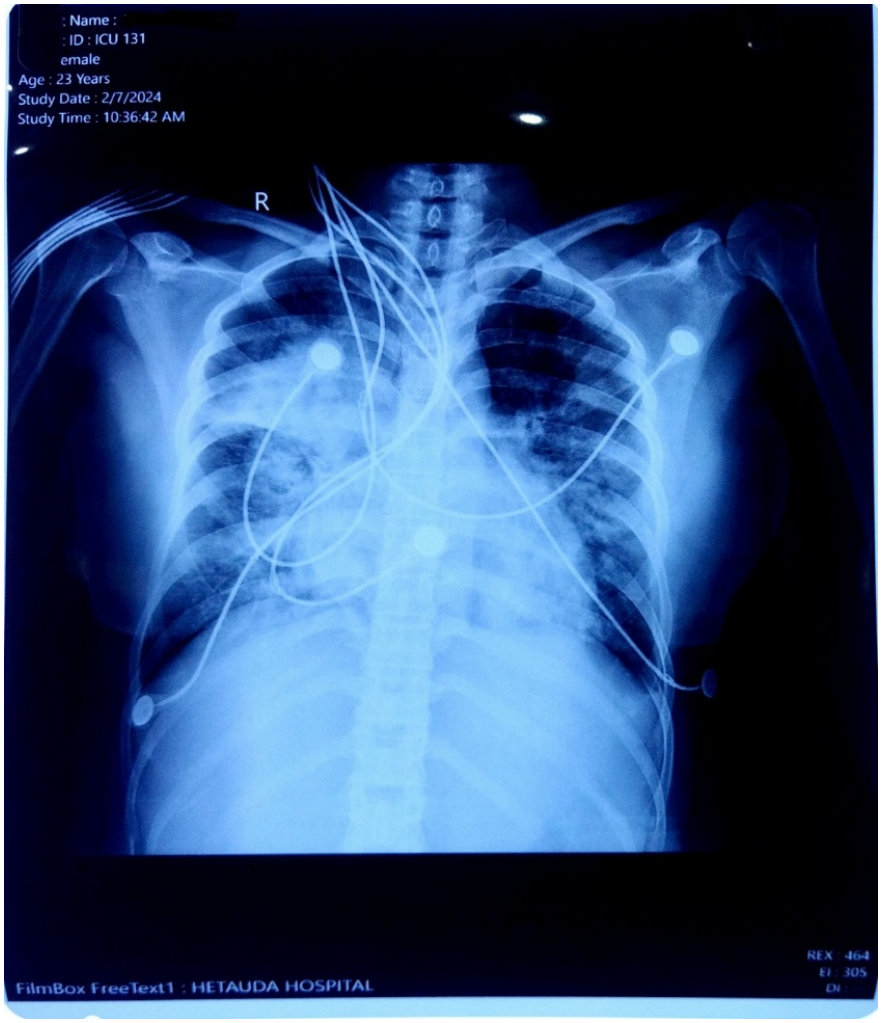

Figure 2: Chest x-ray showing bilateral diffuse infiltrates

Figure 3: ABG showing acute respiratory alkalosis

Investigations	Day of NVD	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

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

- NVD= Normal vaginal delivery
- 1st DOR= 1st day of readmission i.e. 5 days after normal vaginal delivery
- 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 intravenous GTN (glyceryl trinitrate), 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) was 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. IV Morphine (2 mg) was available as needed for pain relief. Close monitoring of vital signs, fluid intake and urinary output were 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 optimal 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 spirometry. Repeat chest X-ray showed resolution of pulmonary infiltrates.

The patient was discharged from the ICU on the 8th postpartum day with stable vital signs and improved respiratory status. She was counseled regarding the importance of follow-up visits and advised on measures to prevent recurrence. On 5th day following her discharge, her general examination, systemic examination and investigations were all within normal range.

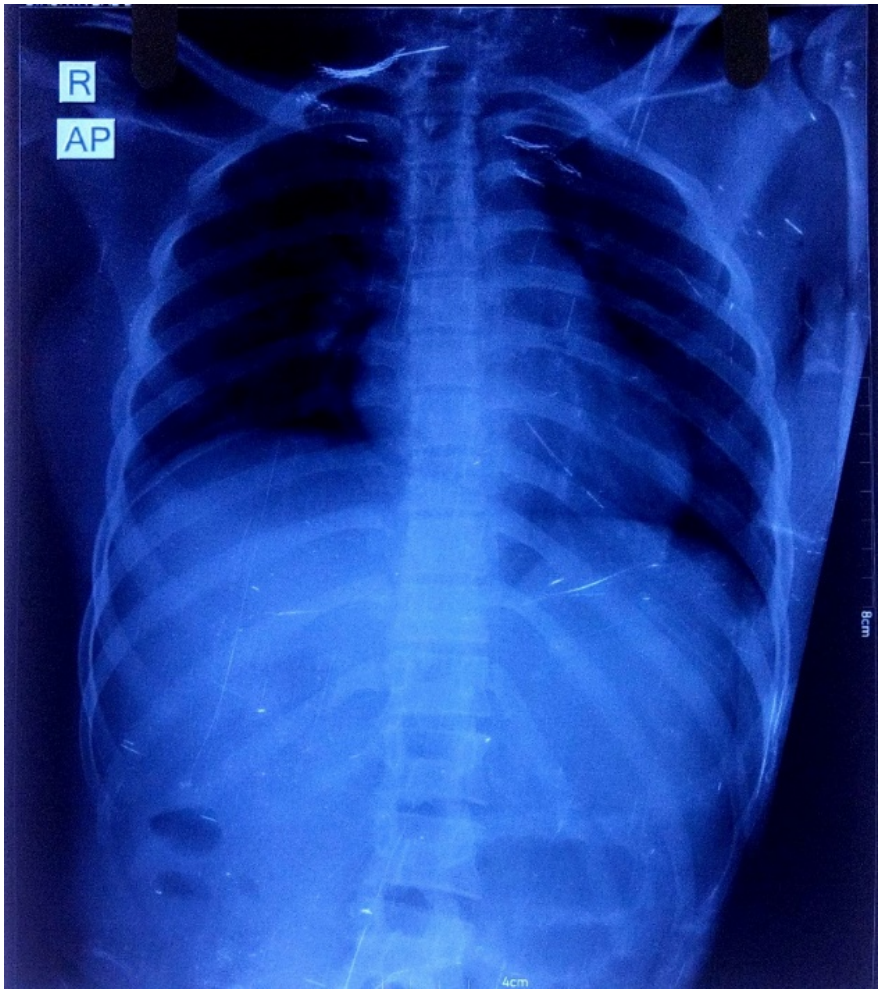

Figure 4: Normal chest xray of the patient at the time of discharge

Figure 5: ICU moniter showing normal 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 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 Post-Partum Cardiomyopathy(PPCM) 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 the patient was diastolic dysfunction during the third trimester of pregnancy. However, in our resource limited setting, echocardiography screening was done at the bedside which resulted in normal LVEF with no cardiac abnormality [8].

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.

[CONCLUSION]Pulmonary edema occurring after a normal 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. This instance highlights the significance of timely action, vigilant supervision, and teamwork in improving outcomes for patients experiencing postpartum pulmonary edema.

[NOTES]

Conflict of interest All authors declare that they have no potential conflicts of interest.

Patient consent

The patient involved in this study have granted consent for the publication of this case report.

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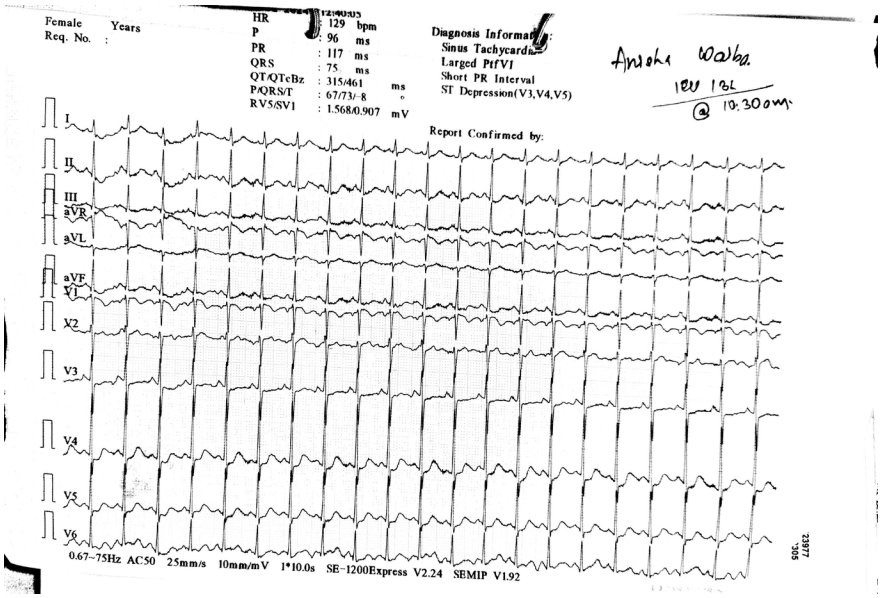

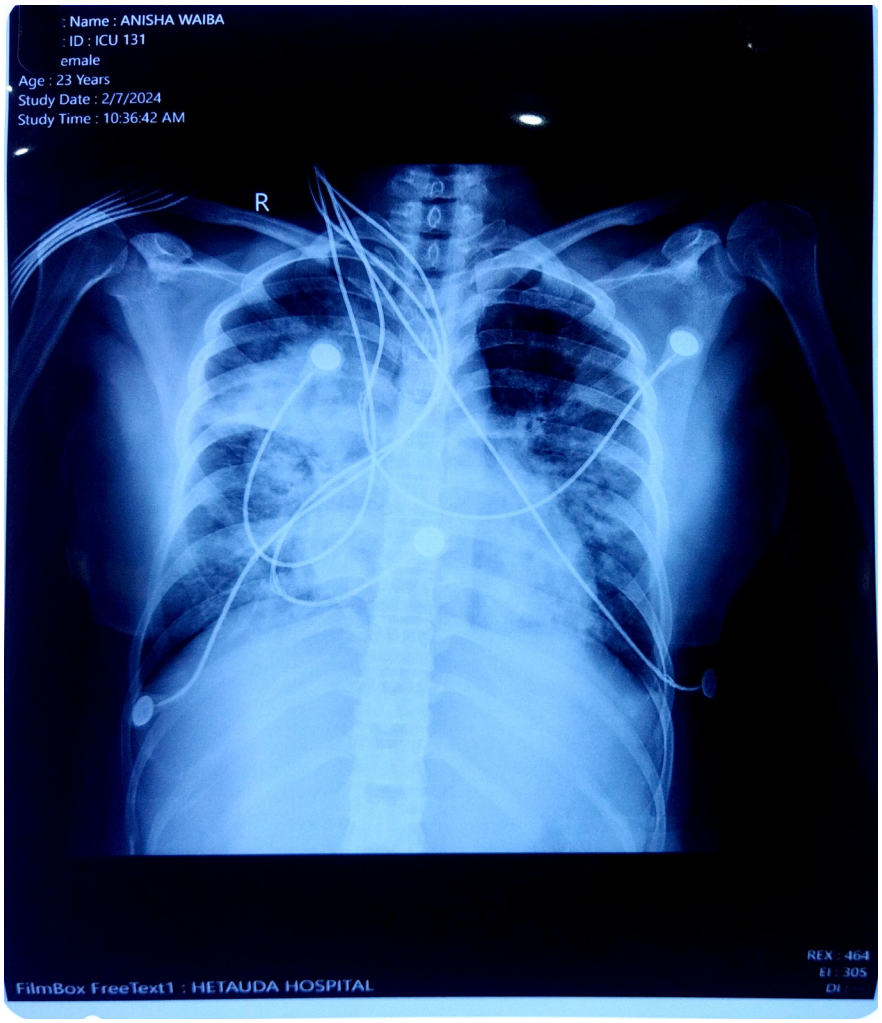

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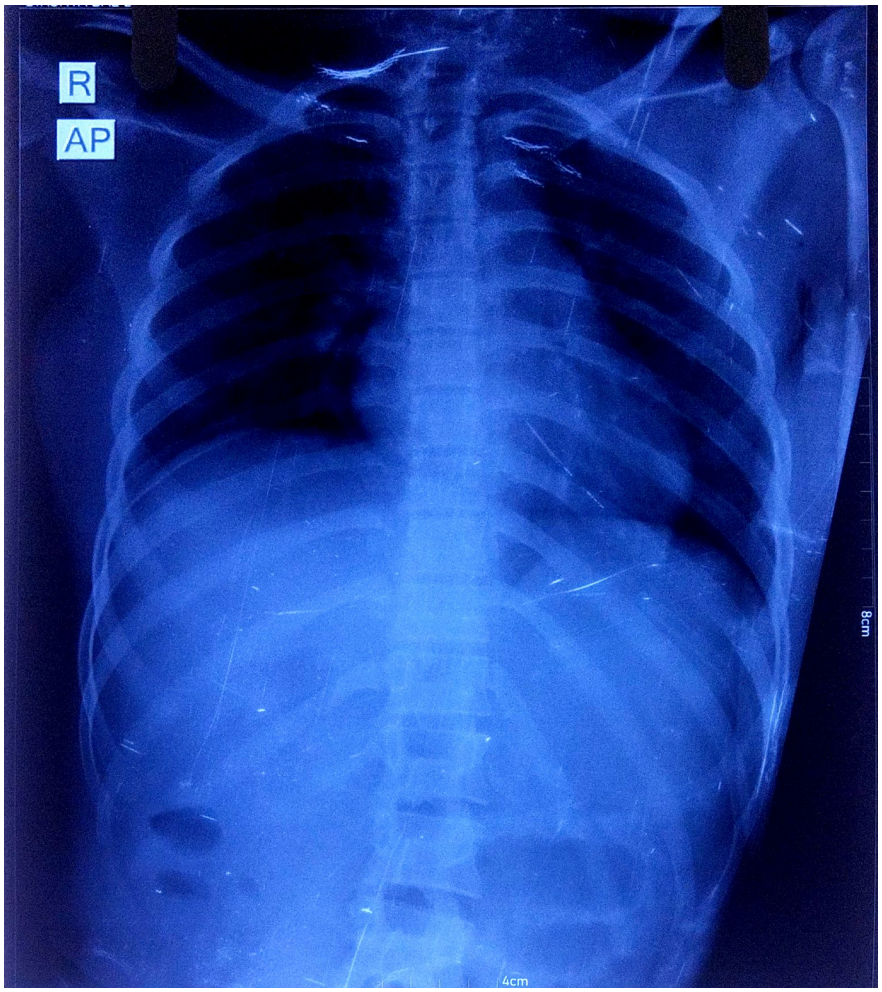
Metabolite Values

Glu	7.3	↑	mmol/L



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