CASE REPORT

Extracorporeal Membrane Oxygenation as a Bridge to Diagnosis of Pheochromocytoma Crisis

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Abstract

Aim and background: Pheochromocytoma (PCC), although rare, is one of the most dramatic and diagnostically challenging endocrine emergencies. Its unpredictable surges of catecholamines can lead to life-threatening conditions such as cardiogenic shock, malignant hypertension, and severe arrhythmia. Extracorporeal membrane oxygenation (ECMO) has become a crucial, life-saving intervention in these situations.

Case description: We described a case of a young male with chest pain and respiratory distress, and found that he had cardiogenic pulmonary edema. He went to venoarterial extracorporeal membrane oxygenation in view of hypoxia and pulmonary edema. After recovery of cardiac function and lung function, the patient was decannulated and subsequently extubated. Subsequently, on evaluation, he had elevated serum and urinary metanephrine, and on contrast-enhanced computed tomography of the abdomen, he had a right adrenal mass and a confirmed diagnosis of PCC.

Conclusion: Early consideration of ECMO can provide critical time for stabilization, advanced imaging, and biochemical evaluation in occult PCC.

Clinical significance: Extracorporeal membrane oxygenation can serve as a lifesaving bridge to diagnosis for patients experiencing unexplained pulmonary edema and cardiogenic shock due to an undiagnosed PCC.

Keywords: Case report, Pheochromocytoma crisis, Pulmonary edema, Stress cardiomyopathy, Venoarterial extracorporeal membrane oxygenation.

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Introduction

Pheochromocytoma (PCC), although rare, is one of the most dramatic and diagnostically challenging endocrine emergencies.[1] Its unpredictable surges of catecholamines can lead to life-threatening conditions such as cardiogenic shock, malignant hypertension, severe arrhythmia, acute respiratory distress syndrome (ARDS), and multiple organ dysfunction syndrome.[2]

Extracorporeal membrane oxygenation (ECMO) has become a crucial, life-saving intervention in these situations—not only as a rescue therapy but also as a bridge to diagnosis. By providing immediate circulatory and respiratory support, ECMO stabilizes hemodynamics, reverses end-organ hypoperfusion, and creates a period of physiological stability during which clinicians can conduct targeted investigations.[3] This support often uncovers underlying issues, such as an occult PCC that may remain hidden beneath refractory cardiogenic pulmonary edema or stress cardiomyopathy.

Case Description

A 25-year-old male presented to Utkal Hospital, Bhubaneswar, with chest pain, vomiting, and respiratory distress. Initially, he was given metoclopramide and pantoprazole, but his condition rapidly deteriorated, leading to a referral to our center. Upon evaluation, he was found to be suffering from acute lung injury, severe left ventricular dysfunction (on 2D ECHO-ejection fraction of 30%, global hypokinesia with apical ballooning), hepatopathy, and encephalopathy. A highly sensitive troponin I level of 152.5 ng/L was measured, indicating cardiac injury; the upper reference level for a healthy population is 19 ng/L. NTPROBNP was 3652.0 pg/mL (normal value < 125), suggesting pulmonary edema.

His chest X-ray, 2D ECHO image, and ECG are in Figures 1 to 3, respectively. His lab parameters are in Table 1.

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Patient consent statement: A written informed consent was obtained from the patient for the publication of details, which can include photographs and/or videos and/or case history to be published in any printed/online journals.

Due to hypoxia and pulmonary edema, he was intubated and put on mechanical ventilation. Later on, he was put on venoarterial extracorporeal membrane oxygenation (VA ECMO)

in view of cardiogenic pulmonary edema for four days with heparin anticoagulation. Over this period, the patient showed significant improvement in all organ functions. 2D ECHO showed normal cardiac contractility, and the chest X-ray improved. He was decannulated on day 5 and extubated on day 6. His ECMO settings are in Table 2. Given the swift recovery of his lungs and heart, he was evaluated for systemic causes of his rapid onset of multiple organ failures.

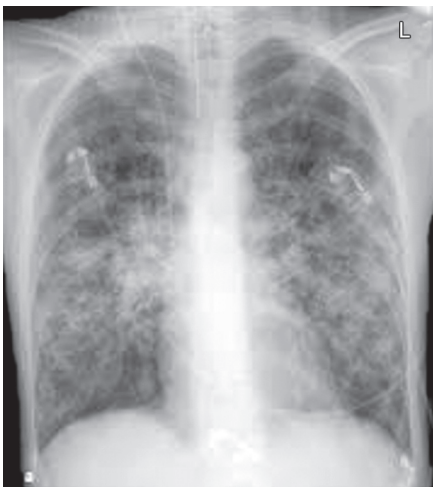


Fig. 1: Bilateral infiltrates suggestive of pulmonary edema

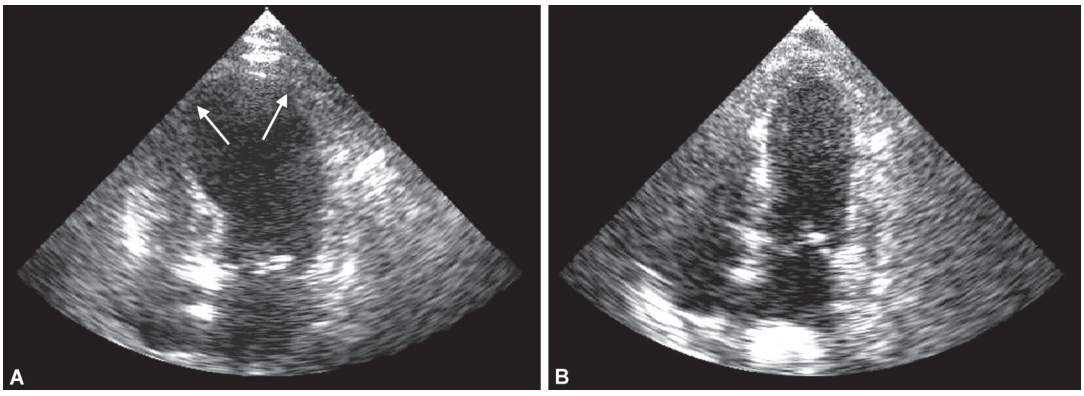
During the evaluation, the following information was gathered:

- History: The patient reported a history of anxiety and palpitations for the past 2 years and had been on multiple anti-anxiety medications.
- Family history: There was a family history of pituitary adenoma in his aunt.

Biochemical tests revealed elevated levels of metanephrines and normetanephrines in a 24-hour urine and plasma analysis, as in Table 3. A contrast-enhanced computed tomography (CECT) scan of the abdomen suggested a heterogeneous enhancing mass in the right adrenal gland, as in Figure 4. The patient was discharged on day 9 and scheduled for right adrenalectomy one month later.

Discussion

Pheochromocytoma can release large amounts of catecholamines, either continuously or intermittently. This can lead to persistent or paroxysmal hypertension, multiple organ dysfunction, and metabolic disorders. The primary clinical manifestations include a triad of symptoms: headache, palpitations, and sweating. In severe cases, it can result in a hypertensive crisis, heart failure, cardiomyopathy, myocardial infarction, and ARDS.[4]



Figs 2A and B: Arrow depicting apical ballooning; there is global hypokinesia

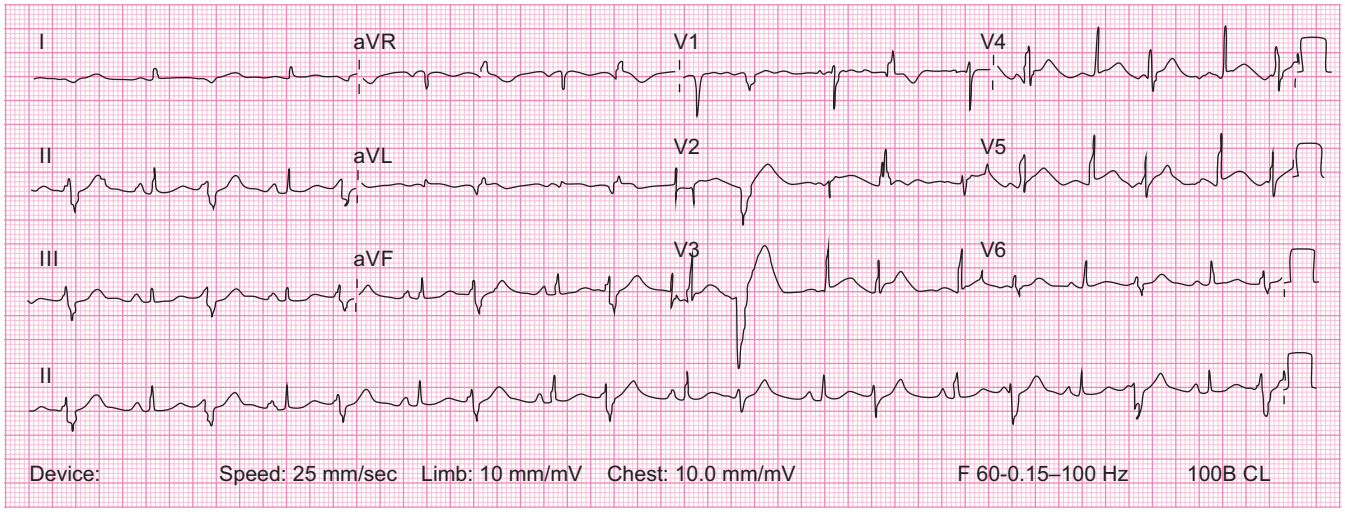


Fig. 3: ECG showing ST-T changes

Table 1: Lab investigations of the patient

Lab investigations	*Reference*	*17/9*	*19/9*	*20/9*	*22/9*	*24/9*	*22/8*	*24/8*	*25/8*
Hb	12–16 gm/dL	11.6	11	10.8	11	10.9	10.4	10.1	10.3
TLC	4–11*10^3/μL	11.4	10.5	10.1	9.6	10.2	10.9	11.5	10.5
TPC	1.5–4.5 L/μL	360k	300k	296k	250k	210k	180k	200k	240k
Urea	7–20 mg/dL	24	40	36	46	58	35	30	28
Creatinine	0.6–1.2 mg/dL	0.8	0.7	0.4	0.5	0.7	0.6	0.8	0.6
INR		1.2			1.3				
SGOT/SGPT	(5–40 μ/L)/(7–56 μ/L)	1,435/1,785			600/587			66/55	
Bilirubin	0.1–1.1 mg/dL	1.5			2.4			1.1	
ET culture				Sterile					
2D ECHO		Apical ballooning, global hypokinesia, severe LV dysfunction		Normal LV function					

Table 2: Extracorporeal membrane oxygenation settings

ECMO setting	*17/9*	*18/9*	*19/9*	*20/9*	*21/9*
Flow	3.5 L	3.6 L	3.5 L	3.5 L	2 L
RPM	3,500	3,600	3,500	3,400	3,400
FiO_2	100	60	40	40	35
Sweep gas flow	3.5 L	4 L	3.8 L	3.7 L	2 L
ACT	167	165	158	160	166

Table 3: Plasma metanephrine and normetanephrine, and urine metanephrine

	Value	*Reference*
Plasma metanephrine free	385.0 ng/L	7.90–88.70 ng/L
Plasma normetanephrine free	1390.0 ng/L	20.10–135.40 ng/L
Urine metanephrine over 24 hours	509.4 μg/ 24 hours	44–261 μg/ 24 hours

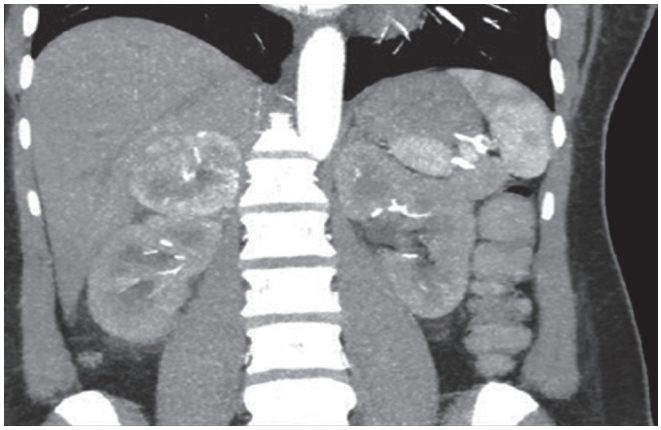


Fig. 4: Showing right adrenal mass

The patient in our case presented with an insidious onset of symptoms and had no prior history of hypertension, which complicated the diagnosis. She had a medical history of anxiety disorder and was taking antianxiety medications. Diagnosing PCC is quite challenging, especially in patients without hypertension and those experiencing acute multiple-organ dysfunction. In this case, the patient reported chest pain and respiratory distress. Upon evaluation, she was found to have pulmonary edema and left ventricular dysfunction characterized by apical ballooning. Extracorporeal membrane oxygenation was used to stabilize her organ function. After decannulation, further assessment and imaging revealed an adrenal mass that was intermittently releasing catecholamines, which explained the patient's symptoms.

A PCC crisis can occur spontaneously or be triggered by various factors, including tumor manipulation, trauma, surgical pressure in areas outside the adrenal glands, and certain medications such as glucocorticoids, β-blockers, metoclopramide, and anesthetics.[5] Studies have indicated that metoclopramide stimulates the release of catecholamines by acting as a dopamine receptor antagonist and a 5-hydroxytryptamine receptor 4 antagonist.[6]

Pheochromocytoma can present with Takotsubo cardiomyopathy, which is also known as stress cardiomyopathy, broken heart syndrome, or apical ballooning syndrome.[7] The 2018 European consensus established new international diagnostic criteria for diagnosing Takotsubo syndrome (TTS). Patients with TTS experience transient left ventricular dysfunction, which can manifest as midventricular, basal, or focal wall motion abnormalities, as well as apical ballooning. Pheochromocytoma may contribute to this syndrome.[8] An excess of catecholamines can lead to cardiomyopathy. These catecholamines can cause direct damage to the myocardium through mechanisms including calcium overload, oxidative stress, apoptosis, fibrosis, and activation of the renin-angiotensin-aldosterone system.

Pheochromocytoma, which can present with pulmonary edema as its first symptom, is a rare condition that often leads to rapid death. This complication can occur both during the course of the disease and after the surgical removal of the tumor. The causes of pulmonary edema associated with PCC can be classified into cardiac and non-cardiac origins.

In most cases, pulmonary edema is cardiogenic, resulting from conditions such as cardiomyopathy, myocardial infarction, and severe arrhythmias. On the other hand, non-cardiogenic pulmonary edema is believed to be caused by a transient increase in pulmonary capillary pressure induced by catecholamines, leading to pulmonary venoconstriction and changes in pulmonary capillary permeability.[9,10] For instance, Sukoh et al.[11] reported a case of a patient with PCC who developed acute non-cardiogenic pulmonary edema. Analysis of bronchoalveolar lavage fluid revealed an accumulation of pulmonary neutrophils, suggesting that the catecholamines released by PCC may promote the gathering of

neutrophils in the lungs, thereby contributing to the development of ARDS.

The patient in our case presented with atypical symptoms of respiratory distress and chest pain, leading to the decision to place him on VA-ECMO. It was suspected that metoclopramide contributed to his crisis by causing a surge in catecholamines. The ECMO support not only allowed for organ recovery but also provided crucial time for further evaluation.

Conclusion

This case demonstrates that ECMO can be a lifesaving bridge to diagnosis for a patient experiencing unexplained cardiogenic pulmonary edema due to an occult PCC. When traditional resuscitation efforts fail and the cause of the patient's deterioration, including hypoxia and cardiovascular instability, is unclear, early consideration of ECMO can provide critical time for stabilization, advanced imaging, and biochemical evaluation. This approach prevents the patient from suffering the potentially fatal consequences of ongoing catecholamine-driven shock. The successful diagnosis and definitive treatment of pheochromocytoma in this situation highlight the importance of ECMO, not only as a rescue therapy but also as a valuable diagnostic tool in cases of refractory shock.

Clinical Significance

- The mortality rate for PCC, especially when it presents with left ventricular dysfunction and pulmonary edema, is high, making timely diagnosis essential.
- Pheochromocytoma can occasionally present with chest pain and respiratory distress, which are considered unusual symptoms.
- A PCC crisis can occur spontaneously or be triggered by various factors, including tumor manipulation, trauma, surgical pressure in areas outside the adrenal glands, and certain medications such as glucocorticoids, β-blockers, metoclopramide, and anesthetics.
- Extracorporeal membrane oxygenation can serve as a lifesaving bridge to diagnosis for patients experiencing unexplained pulmonary edema and cardiogenic shock due to an undiagnosed PCC.

Declarations

Ethics Waiver/Approval

Ethical approval was waived as this is a single case report in accordance with institutional guidelines.

Artificial Intelligence (AI) Disclosure

No AI tools were used in the preparation of this manuscript.

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None.

Authors' Contributions (CRediT Format)

Patient management: Rashmi R Pattanayak and K Smita Reddy.
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