International Journal of Research in Medical Sciences
Soni P et al. Int J Res Med Sci. 2026 Jun;14(6):2630-2635
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20261716

Case Report

Pheochromocytoma crisis presenting as refractory cardiogenic shock: anaesthetic and critical care challenges

Pankaj Soni, Sai Siva Ramakrishna Nandavarapu*, Rajeev Kumar

Department of Anaesthesiology and Critical Care, EMC Hospital, Amritsar, Punjab, India

Received: 03 March 2026
Revised: 13 April 2026
Accepted: 20 May 2026

***Correspondence:**
Dr. Sai Siva Ramakrishna Nandavarapu,
E-mail: ramakrishnanandavarapu@gmail.com

ABSTRACT

Pheochromocytoma is a rare catecholamine-secreting tumour of the adrenal medulla with heterogeneous clinical manifestations. Pheochromocytoma crisis is an uncommon but life-threatening presentation frequently complicated by cardiovascular collapse and multi-organ dysfunction. A 30-year-old male presented with undifferentiated shock, metabolic acidosis, acute heart failure, and renal failure. He was later diagnosed with pheochromocytoma-induced cardiogenic shock, likely worsened by inappropriate β-blocker therapy without prior α-blockade. Management required advanced critical care support, mechanical ventilation, renal replacement therapy, staged endocrine optimization, and definitive adrenalectomy. This case highlights the importance of maintaining a high index of suspicion for pheochromocytoma in patients with unexplained cardiogenic shock and labile hemodynamics, particularly when preceded by episodic headaches and inappropriate β-blocker exposure. Early imaging and multidisciplinary management are crucial for optimal outcomes.

Keywords: Pheochromocytoma crisis, Cardiogenic shock, Stress cardiomyopathy, β-blockers, Anesthesia, Critical care

INTRODUCTION

Pheochromocytoma is a rare neuroendocrine tumour arising from adrenal chromaffin cells and characterised by excessive catecholamine secretion.[1,15] Its prevalence is estimated at approximately 0.1–1% among hypertensive adults.[1,4] Clinical manifestations are protean, ranging from paroxysmal hypertension and headache to catastrophic cardiovascular collapse.[3,5]

Pheochromocytoma crisis represents an extreme state of catecholamine excess associated with catastrophic cardiovascular complications like cardiogenic shock, pulmonary oedema, arrhythmias, and multi-organ failure.[2,10,11]

Cardiovascular complications are reported in up to 40% of patients and may include stress-induced cardiomyopathy and acute heart failure.[3,6,14]

From an anaesthesiology and critical care perspective, management is uniquely challenging due to labile haemodynamic, paradoxical hypotension, impaired myocardial functional and contraindications to standard heart failure therapies.[12,16]

CASE REPORT

A 30-year-old male was brought to the emergency department in an unconscious state with cold, clammy extremities and markedly reduced urine output. There was no preceding history suggestive of infection, trauma, or toxin exposure.

Initial assessment

On arrival, the patient was hypotensive with a blood pressure of 60/40 mmHg and tachycardic. Capillary blood glucose was 142 mg/dl. Arterial blood gas analysis

revealed metabolic acidosis (pH 7.18) with elevated lactate (2.4 mmol/l), indicating global hypoperfusion.

Emergency management

Two large-bore peripheral intravenous cannulas were secured, and aggressive crystalloid resuscitation was initiated. A central venous catheter was placed for hemodynamic monitoring, and dual inotropic support with norepinephrine and dopamine was started due to persistent hypotension.

The patient's depressed consciousness with low GCS necessitated definitive airway protection, and he was intubated with 8.0 size endotracheal tube and mechanically ventilated. There was no clinical or laboratory evidence of sepsis.

Clinical history and diagnostic workup

Collateral history revealed recurrent episodes of severe headache associated with aggression and transient hypertension over the preceding weeks. He had been prescribed β-blockers and analgesics at a local hospital for hypertension without prior α-blockade.

Bedside transthoracic echocardiography demonstrated severe left ventricular systolic dysfunction with an ejection fraction of approximately 30%, consistent with catecholamine-induced cardiomyopathy.[6,18] Contrast-enhanced computed tomography of the abdomen revealed a large right adrenal mass adjacent to the upper pole of the kidney.

A diagnosis of pheochromocytoma-induced cardiogenic shock was made.[2,11]

ICU course

During the intensive care unit (ICU) stay, the patient developed acute kidney injury with anuria, necessitating one session of intermittent haemodialysis. Gradual improvement in renal function followed, with recovery of urine output. Hemodynamic stability improved allowing tapering of inotropes, and neurological status normalized. The patient was successfully extubated and transferred to the ward.

Catecholamine-induced cardiomyopathy is often reversible with appropriate treatment, as demonstrated in this case.[6,14,18]

Definitive management

Following stabilization, the patient was started on α-adrenergic blockade with an α blocker (prazosin) and advised a high-sodium diet for 14 days to optimize intravascular volume.[7]

Elective adrenalectomy was subsequently performed under general anaesthesia with invasive monitoring following multidisciplinary planning.[12,16] The intra-operative and post-operative periods were uneventful. The patient was discharged home one week later in stable condition.

Anaesthetic management

Preoperative considerations

Following hemodynamic stabilization and recovery from acute cardiogenic shock, the patient underwent preoperative optimization with α-adrenergic blockade using prazosin for 14 days along with dietary sodium supplementation. Adequate α-blockade is the cornerstone of management and reduces peri-operative mortality.[1,7,12] Particular attention was paid to volume status, given the antecedent episode of acute decompensated heart failure and renal dysfunction. Preoperative echocardiography demonstrated improvement in left ventricular systolic function compared to admission values, and renal parameters had normalized.

Given the history of catecholamine crisis, cardiomyopathy, and labile hemodynamic, the patient was categorized as high anaesthetic risk, and a multidisciplinary plan was formulated involving anaesthesiology, endocrinology, critical care, and surgery.

Monitoring and preparation

Standard ASA monitors were applied along with invasive arterial blood pressure monitoring prior to induction for beat-to-beat hemodynamic assessment.[12,17] A central venous catheter was maintained for vasoactive drug administration. Vasodilators (nitroglycerin), short-acting β-blockers (esmolol), vasopressors (phenylephrine, norepinephrine), and antiarrhythmics were prepared in advance to manage potential catecholamine surges or post-resection hypotension.

Induction of anaesthesia

Anaesthesia was induced using intravenous propofol, selected for its rapid onset, favourable titratability, and ability to attenuate sympathetic responses. To further blunt stress-induced catecholamine release and provide sympatholytic, a loading dose of intravenous dexmedetomidine at 1.5 µg/kg was administered prior to induction. Fentanyl 1.5 µg/kg was administered for analgesia and blunting stress response.

Dexmedetomidine was chosen for its highly selective α_2-adrenergic agonist properties, which confer central sympatholytic effects, reduce circulating catecholamine levels, and provide hemodynamic stability without significant respiratory depression.[16,19] This was particularly advantageous in a patient with a recent

pheochromocytoma crisis and compromised myocardial function.

Neuromuscular blockade was achieved using a non-depolarizing agent with minimal cardiovascular effects. Tracheal intubation was performed under deep anaesthesia, and care was taken to minimize sympathetic stimulation during laryngoscopy.

Maintenance of anaesthesia

Anaesthesia was maintained using a balanced technique with inhalational agents and controlled ventilation. Hemodynamic responses during surgical manipulation of the adrenal mass were closely monitored. Transient increases in blood pressure were effectively managed with deepening of anaesthesia and pharmacological modulation as required.

The dexmedetomidine infusion was continued intra-operatively at a titrated dose to maintain sympatholytic, facilitate anaesthetic sparing, and ensure smooth hemodynamic transitions during tumor handling. Tumour manipulation is known to provoke catecholamine surges and arrhythmias.[12,17] There were episodes of transient spikes in heart rate and blood pressure during tumour handling, which were taken care of by bolus doses of esmolol and nitroglycerin.

Post-tumor resection phase

Following adrenal vein ligation and tumor excision, the anticipated sudden withdrawal of circulating catecholamines was vigilantly anticipated. Hemodynamic parameters remained stable, and no significant hypotension was encountered, likely attributable to adequate preoperative α-blockade, volume optimization, and judicious intra-operative anaesthetic management.[7,16]

Emergence and postoperative care

Neuromuscular blockade was reversed, and the patient was extubated uneventfully in the operating room after confirming adequate respiratory effort and hemodynamic stability. He was transferred to a monitored postoperative care unit for continued observation. The postoperative course was uneventful, with no recurrence of hemodynamic instability or arrhythmias.

Anaesthetic perspective and learning points

Propofol, combined with dexmedetomidine and fentanyl, provided effective attenuation of sympathetic responses during induction and tumor manipulation. Dexmedetomidine played a key role in maintaining intra-operative hemodynamic stability through central sympatholytic and catecholamine suppression.

Anticipation of post-resection hypotension and preparedness with vasoactive agents is critical in pheochromocytoma surgery. Careful titration of anaesthetic depth and avoidance of drugs causing sympathetic stimulation are paramount in patients with recent pheochromocytoma crisis.

Anaesthetic challenges in achieving hemodynamic stability and favourable outcome

Anaesthetic management of pheochromocytoma, particularly following a recent catecholamine crisis with cardiogenic shock, is uniquely challenging and requires meticulous planning, anticipation of extreme hemodynamic fluctuations, and precise pharmacological control. In this patient, several factors compounded the peri-operative risk and influenced anaesthetic decision-making.

Labile haemodynamic in the setting of recent catecholamine crisis

The principal challenge was the unpredictable and labile cardiovascular response due to persistent catecholamine effects and receptor hypersensitivity despite preoperative optimization. The patient had recently recovered from severe left ventricular systolic dysfunction, rendering him vulnerable to both hypertensive surges during tumor manipulation and profound hypotension following tumor resection.

Strategy adopted

Continuous invasive arterial blood pressure monitoring allowed real-time assessment and rapid intervention. Anaesthetic depth was carefully titrated to blunt sympathetic surges without precipitating myocardial depression.

Attenuation of sympathetic responses during induction and intubation

Laryngoscopy and tracheal intubation are potent triggers of catecholamine release and may precipitate life-threatening hypertension, arrhythmias, or myocardial ischemia in pheochromocytoma patients.

Strategy adopted

The combination of propofol induction with a high-dose dexmedetomidine loading (1.5 µg/kg) was deliberately chosen to achieve profound sympatholytic. Dexmedetomidine's central α_2-agonist action reduced sympathetic outflow, minimized tachycardia, and provided hemodynamic smoothness during airway manipulation.

IV fentanyl used as an analgesic also helps as an addon to attenuate the stress response to laryngoscopy and intubation.

Balancing myocardial protection with adequate perfusion

Recent pheochromocytoma-induced cardiomyopathy posed a dilemma between avoiding myocardial depression and ensuring adequate organ perfusion. Excessive anaesthetic depth risked hypotension, while inadequate depth risked catecholamine surges.

Strategy adopted

A balanced anaesthetic technique was employed, combining volatile anaesthetics with dexmedetomidine to achieve anaesthetic-sparing effects. This approach maintained stable systemic vascular resistance while avoiding excessive negative inotropy.

Hemodynamic instability during tumor manipulation

Direct manipulation of the adrenal tumor is known to cause sudden catecholamine release, leading to abrupt hypertension and arrhythmias.

Strategy adopted

Anticipatory deepening of anaesthesia during tumor handling, continued dexmedetomidine infusion, and readiness with short-acting vasoactive agents ensured effective control of transient hypertensive episodes. Importantly, no sustained hypertensive crisis occurred.

Risk of post-resection hypotension

Following adrenal vein ligation, abrupt cessation of catecholamine secretion can result in severe vasodilation and refractory hypotension, especially in patients with chronic vasoconstriction and intravascular volume depletion.

Strategy adopted

Adequate preoperative α-blockade, cautious volume optimization, and vigilant intra-operative monitoring allowed a smooth transition through the post-resection phase. Vasopressors were readily available but ultimately not required, underscoring the effectiveness of pre-emptive planning.

Emergence and extubating considerations

Emergence from anaesthesia can be associated with sympathetic stimulation, coughing, and hemodynamic instability.

Strategy adopted

Gradual emergence under continued sympatholytic ensured smooth extubation without tachycardia or blood pressure surges. Post-extubation monitoring was continued in a high-dependency setting.

Anaesthetic lessons and clinical implications

Patients with recent pheochromocytoma crisis and cardiogenic shock represent an extreme end of the anaesthetic risk spectrum.

Dexmedetomidine is a valuable adjunct in pheochromocytoma surgery for controlling sympathetic tone, reducing anesthetic requirements, and promoting hemodynamic stability. Successful outcomes depend on anticipation rather than reaction, with invasive monitoring, pharmacologic preparedness, and multidisciplinary coordination.

Anaesthetic management should be individualized, especially in the presence of myocardial dysfunction and prior inappropriate β-blocker exposure.

Table 1: Anaesthetic challenges in Pheochromocytoma surgery and strategies adopted.

Anesthetic challenge	Clinical risk	Anesthetic strategy adopted	Impact on outcome
Recent pheochromocytoma crisis with cardiogenic shock	Severe hemodynamic lability, myocardial ischemia, circulatory collapse	Multidisciplinary planning; invasive arterial and central venous monitoring	Early detection and rapid correction of hemodynamic fluctuations
Labile blood pressure due to catecholamine excess	Hypertensive surges or refractory hypotension	Preoperative α-blockade and cautious volume optimization	Improved baseline hemodynamic stability
Sympathetic stimulation during induction and intubation	Tachyarrhythmias, hypertensive crisis, myocardial injury	Propofol induction combined with dexmedetomidine loading (1.5 µg/kg)	Blunted stress response and smooth airway instrumentation
Compromised left ventricular systolic function	Worsening heart failure, low cardiac output	Balanced anesthetic technique with anesthetic-sparing effect of dexmedetomidine	Preservation of myocardial function and adequate perfusion

Continued.

Anesthetic challenge	Clinical risk	Anesthetic strategy adopted	Impact on outcome
Catecholamine release during tumor manipulation	Sudden hypertension, arrhythmias	Anticipatory deepening of anesthesia, continued dexmedetomidine infusion, readiness with short-acting vasoactive agents	Controlled transient BP surges without major instability
Abrupt catecholamine withdrawal after tumor resection	Severe vasodilation and hypotension	Adequate α blockade, volume repletion, vigilant monitoring	Stable post-resection hemodynamics without vasopressor dependence
Emergence and extubation-related sympathetic response	Hypertension, tachycardia, myocardial stress	Gradual emergence under continued sympatholytic	Smooth extubation and stable immediate postoperative period

DISCUSSION

This case illustrates a rare but serious presentation of pheochromocytoma as refractory cardiogenic shock complicated by acute renal failure, posing unique diagnostic and therapeutic dilemmas in the critical care setting.

Pheochromocytoma crisis is a rare but life-threatening endocrine emergency.[2,10] Both adrenaline- and noradrenaline-secreting tumours have been implicated in pheochromocytoma crises. Evidence suggests that the clinical severity of crisis is not determined by the predominant catecholamine secreted, but rather by the degree of catecholamine excess and end-organ sensitivity.[11]

Excess adrenaline can induce myocardial injury through sustained β-adrenergic stimulation, leading to myocardial infarction, stress (Takotsubo-like) cardiomyopathy, or profound vasodilation with hypotension.[6,14,18] In this case, premature β-blockade likely caused unopposed α-adrenergic vasoconstriction, worsening myocardial dysfunction and precipitating failure — a well-described phenomenon.[1,7]

Diagnosis during pheochromocytoma crisis is inherently difficult. Measurement of plasma-free or urinary fractionated metanephrines remains diagnostic gold standard. However, in critically ill individuals, results may be confounded by stress, organ failure, and recent administration of catecholamine inotropes. Consequently, anatomical imaging (CT or MRI) assumes greater importance in the acute setting.[1,9]

The use of catecholamine-based inotropes in pheochromocytoma-induced shock remains controversial. Their mechanisms in a state of endogenous catecholamine excess are poorly understood, yet they may be unavoidable to maintain end-organ perfusion. Similarly, fluid resuscitation must be cautiously balanced against the risk of pulmonary oedema in patients with depressed ventricular function.

While α-blockade is the cornerstone of medical management in hypertensive pheochromocytoma crises, its initiation may be limited or contraindicated in profound hypotension. β-blockers, although essential in heart failure management, should only be introduced after adequate α-blockade, as premature use may precipitate catastrophic cardiovascular collapse.

A high-sodium diet is routinely recommended following α-blockade to restore catecholamine-induced volume contraction and prevent peri-operative hypotension. However, its role remains controversial in patients with concurrent acute heart failure and renal dysfunction, as illustrated in this case.

Peri-operative mortality has significantly declined with modern α-blockade, invasive monitoring, and multidisciplinary care.[12,17,20]

Recent literature emphasizes individualized haemodynamic strategies, particularly in patients with cardiogenic shock.[18,20]

CONCLUSION

This case underscores the importance of considering undiagnosed pheochromocytoma in patients presenting with unexplained cardiogenic shock and labile blood pressure, particularly in the presence of episodic headaches and prior inappropriate β-blocker therapy. Early recognition of this rare but reversible cause of shock requires vigilance, prompt imaging, and multidisciplinary collaboration. From an anaesthesiology and critical care perspective, individualized hemodynamic management is paramount to achieving favorable outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Lenders JWM, Duh QY, Eisenhofer G, Gimenez-Roqueplo A, Grebe SKG, Murad MH. Pheochromocytoma and paraganglioma: endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2014;99:1915-42.
2. Whitelaw BC, Prague JK, Mustafa OG, Schulte KM, Hopkins PA, Gilbert JA, et al. Phaeochromocytoma [corrected] crisis. Clin Endocrinol (Oxf). 2014;80(1):13-22.

3. Prejbisz A, Lenders JWM, Eisenhofer G, Januszewicz A. Cardiovascular manifestations of pheochromocytoma. J Hypertens. 2011;29:2049-60.
4. Agarwal G, Sadacharan D, Kapoor A, Batra A, Dabadghao P, Chand G, et al. Cardiovascular dysfunction and catecholamine cardiomyopathy in pheochromocytoma patients and their reversal following surgical cure: results of a prospective case-control study. Surgery. 2011;150(6):1202-11.
5. Zelinka T, Petrák O, Turková H, Holaj R, Strauch B, Kršek M, et al. High incidence of cardiovascular complications in pheochromocytoma. Horm Metab Res. 2012;44(5):379-84.
6. Stolk RF, Bakx C, Mulder J, Timmers HJ, Lenders JW. Is the excess catecholamine secretion in pheochromocytoma the cause of cardiomyopathy? Circ Heart Fail. 2013;6:752-8.
7. Pacak K. Preoperative management of pheochromocytoma. J Clin Endocrinol Metab. 2007;92:4069-79.
8. Manger WM, Gifford RW. Pheochromocytoma crisis. Endocrinol Metab Clin North Am. 1993;22:303-14.
9. Wang K, Liang J, Shen S. Pheochromocytoma with acute heart failure as a complication—emphasis on the etiology of acute heart failure for diagnosis and treatment: a case report. BMC Cardiovascular Disorders. 2025;25(1):605.
10. Scholten A, Cisco RM, Vriens MR, Cohen JK, Mitmaker EJ, Liu C, et al. Pheochromocytoma crisis is not a surgical emergency. J Clin Endocrinol Metab. 2013;98:581-91.
11. Riester A, Weismann D, Quinkler M, Lichtenauer UD, Sommerey S, Halbritter R, Penning R, Spitzweg C, Schopohl J, Beuschlein F, Reincke M. Life-threatening events in patients with pheochromocytoma. European J Endocrinol. 2015;173(6):757-64.
12. Kinney MA, Warner ME, vanHeerden JA, Horlocker TT, Young WF Jr, Schroeder DR, et al. Perianesthetic risks and outcomes of pheochromocytoma resection. Anesth Analg. 2002;95:1467-73.
13. James MF. Use of vasopressors in pheochromocytoma. Curr Opin Anaesthesiol. 2010;23:45-50.
14. Agarwal V, Kant G, Hans N, Messerli FH. Takotsubo-like cardiomyopathy in pheochromocytoma. Int J Cardiol. 2011;153(3):241-8.
15. Neumann HPH, Young WF Jr, Eng C. Pheochromocytoma and paraganglioma. N Engl J Med. 2019;381:552-65.
16. Lentschener C, Gaujoux S, Tesniere A, Dousset B. Anaesthetic management of pheochromocytoma. Br J Anaesth. 2011;107:658-70.
17. Senne M, Wichmann D, Pindur P, Grasshoff C, Mueller S. Hemodynamic instability during surgery for pheochromocytoma: a retrospective cohort analysis. J Clin Med. 2022;11(24):7471.
18. Kiernan CM, Du L, Chen X, Broome JT, Shi C, Peters MF, et al. Predictors of hemodynamic instability during surgery for pheochromocytoma. Ann Surg Oncol. 2014;21(12):3865-71.
19. Talke P, Chen R, Thomas B, Aggarwall A, Gottlieb A, Thorborg P, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion. Anesth Analg. 2000;90:834-9.
20. Brunaud L, Boutami M, Nguyen-Thi PL, Finnerty B, Germain A, Weryha G, et al. Both preoperative alpha and calcium channel blockade impact intraoperative hemodynamic stability similarly in the management of pheochromocytoma. Surgery. 2014;156(6):1410-7.

Cite this article as: Soni P, Nandavarapu SSR, Kumar R. Pheochromocytoma crisis presenting as refractory cardiogenic shock: anaesthetic and critical care challenges. Int J Res Med Sci 2026;14:2630-5.