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Case Report: Occult Pheochromocytoma Masquerading as Takotsubo Syndrome: A Diagnostic Pitfall and Perioperative Challenge

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Case Report

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Abstract

Background

Pheochromocytoma classically presents with hypertension, headache, palpitations, and diaphoresis. However, occult variants may masquerade as stress-induced cardiomyopathy.

Case:

A 44-year-old woman presented with episodic palpitations, chest distress, and mildly elevated cardiac troponin I. Transthoracic echocardiography (TTE) showed focal basal inferior wall hypokinesia with mildly reduced left ventricular ejection fraction (51%). Coronary angiography revealed a mid-segment left anterior descending artery myocardial bridge without significant stenosis, whereas resting myocardial perfusion imaging showed reduced radiotracer uptake in the basal inferior wall. Abdominal computed tomography identified a 3.5-cm right adrenal mass, initially presumed to be an adenoma, with preoperative catecholamine and metanephrine levels near the upper limit of the normal range. During laparoscopic adrenalectomy, sudden severe hypertensive crisis (240/160 mmHg) occurred upon lower-limb repositioning. Postoperative pathology confirmed pheochromocytoma. On postoperative day 7, transthoracic echocardiography showed complete resolution of wall-motion abnormalities and normalization of left ventricular ejection fraction to 58%. The patient was discharged on postoperative day 11 and remained asymptomatic during one-year follow-up.

Conclusion

This case underscores that pheochromocytoma should not be prematurely excluded in patients with an adrenal incidentaloma and unexplained myocardial injury, even when catecholamine levels are near the upper limit of the reference range. Sudden intraoperative hypertensive crisis serves as a pivotal clue for occult disease, necessitating prompt multimodal vasoactive pharmacotherapy.

Introduction

Pheochromocytoma, a rare neuroendocrine neoplasm, classically presents with headache, palpitations, diaphoresis, and hypertension; however, only 25% exhibit this triad, while most present atypically and are incidentally discovered or misdiagnosed [1]. Massive catecholamine release can cause life-threatening complications, with 19.3% of patients developing significant cardio-cerebrovascular comorbidities [2]. Takotsubo syndrome (TTS), an acute reversible stress-induced cardiomyopathy with transient left ventricular dysfunction [3], is strongly associated with pheochromocytoma; catecholamine surge may induce myocardial injury mimicking acute coronary syndrome [4]. Screening for pheochromocytoma is recommended in unexplained stress cardiomyopathy, though diagnosis remains challenging when the tumor is occult, presenting with cardiac dysfunction without hypertension and near-normal biochemistry.

Herein, we report a rare case of a normotensive patient presenting with cardiac symptoms and myocardial injury, without significant coronary stenosis on angiography but with an adrenal mass on CT and coexisting left anterior descending (LAD) artery myocardial bridging. Near-normal catecholamines and the myocardial bridge initially led to misdiagnosis. However, focal basal inferior wall motion abnormality—outside the bridged LAD territory—coupled with rapid postoperative recovery suggested non-ischemic, catecholamine-mediated stress cardiomyopathy rather than bridge-related ischemia. Sudden severe hypertension during intraoperative repositioning prompted pathological confirmation of pheochromocytoma, highlighting the perioperative challenges of occult pheochromocytoma with atypical stress-induced cardiomyopathy.

Case report

A 44-year-old woman presented with a 10-year history of episodic palpitations (exertion- or emotion-triggered, self-limiting, 10 min each) and acute worsening over 4 days with chest tightness, retrosternal pain, and burning back pain. She denied hypertension, diabetes, or coronary artery disease, and was taking clopidogrel, pravastatin, and metoprolol. No relevant family history of neuroendocrine tumors or sudden cardiac death was reported. On admission, she was normotensive (113/69 mmHg; heart rate 64 bpm). Laboratory tests showed elevated cardiac troponin I (0.454 ng/ml; normal 0–0.03) and BNP (171 pg/ml; normal 0–100). ECG revealed diffuse ST-T changes; echocardiography showed a left ventricular end-diastolic diameter of 5.14 cm (Fig. 1A), impaired diastolic function, focal basal inferior wall hypokinesia, and mildly reduced LVEF (51%). Resting myocardial perfusion imaging confirmed reduced uptake in the basal inferior wall. Abdominal CT revealed a 3.5 cm right adrenal mass. Acute coronary syndrome was initially suspected.

Coronary angiography on day 2 revealed a mid-LAD myocardial bridge with 60–70% systolic compression and TIMI grade □ flow; other vessels were normal. Preoperative adrenal workup showed normal cortisol, ACTH, electrolytes, aldosterone, and renin. Twenty-four-hour urinary catecholamines and plasma metanephrines were within the normal range, though several values were at the upper limit (Table 1). Blood pressure during hospitalization fluctuated between 90–130/50–90 mmHg without hypertensive or orthostatic episodes. The mass was presumptively diagnosed as an adenoma, and laparoscopic adrenalectomy was planned.

Table 1
Levels of catecholamines and their metabolites in blood and urine

Project	Result	unit	Indications	Reference Ranges
Catecholamines (24h Urine)				
Epinephrine	18.99	ug/day	-	0.00–20.00
Norepinephrine	55.10	ug/day	-	0.00–90.00
Dopamine	18.98	ug/day	-	0.00-600.00
Plasma Metanephrine Analogues				
Metanephrine	0.27	nmol/L	-	≤ 0.50
Normetanephrine	0.89	nmol/L	-	≤ 0.90
Catecholamines (Plasma)				
Epinephrine	91.84	pg/mL	-	0.00-100.00
Norepinephrine	258.99	pg/mL	-	0.00-600.00
Dopamine	38.28	pg/mL	-	0.00-100.00
Note: All biochemical indicators were within the normal reference range, with several parameters at the upper limit of normal.				

Upon arrival in the operating room, initial noninvasive BP was 121/89 mmHg, whereas invasive radial pressure (IBP) was 145/95 mmHg. After preoxygenation, anesthesia was induced with midazolam, propofol, sufentanil, and cisatracurium. During lower-limb repositioning for catheterization, IBP abruptly surged to 240/160 mmHg with a heart rate of 116 bpm. Urapidil 15 mg and propofol 50 mg were administered, followed by a second bolus of urapidil 15 mg and propofol 50 mg, reducing IBP to 106/71 mmHg before uneventful intubation. Immediately post-intubation, IBP acutely rose to 185/110 mmHg under 2–2.5% sevoflurane; five minutes later, systolic pressure dropped to 80 mmHg, requiring phenylephrine.

Pheochromocytoma was highly suspected, and although the anesthesiologist recommended aborting the procedure for preoperative optimization, the family insisted on proceeding. Aggressive fluid resuscitation (1000 mL lactated Ringer's and 500 mL hydroxyethyl starch) was initiated, with vasoactive agents titrated in real time. Intermittent intravenous phentolamine was administered before tumor mobilization. Approximately 30 minutes after incision, tumor manipulation caused IBP to rise to 210/128 mmHg; this was controlled with urapidil 20 mg, intermittent phentolamine, and sodium nitroprusside 0.4–0.8 μg/kg/min, achieving 150/80 mmHg. Esmolol 30 mg was given for tachycardia.

The interval from incision to adrenal vein ligation lasted 75 minutes. Immediately after tumor resection, IBP fell sharply to 85/40 mmHg; norepinephrine 0.2 μg/kg/min and dopamine 10 μg/kg/min were started, combined with a single bolus of epinephrine 50 μg, stabilizing IBP at approximately 120/60

mmHg. Total intraoperative fluid was 3700 mL, blood loss 15 mL, and urine output 800 mL. Total time from operating room entry to ICU transfer was 2 hours and 50 minutes.

She was transferred to the ICU intubated for hemodynamic support. Extubation was performed uneventfully on postoperative day 1, and she moved to the general ward on day 2. Pathology confirmed right adrenal pheochromocytoma (CK -, Vimentin -, CgA +, Syn +, S-100 -, Ki-67 1–2%). By postoperative day 7, echocardiography showed complete resolution of basal inferior hypokinesia, normalized LVEF (58%), and normal left ventricular end-diastolic diameter (4.90 cm) (Fig. 1B). The preoperative cardiac presentation was consistent with occult pheochromocytoma-induced TTS(basal variant), rather than ischemic heart disease due to the myocardial bridge. She was discharged on day 11 without recurrent symptoms and remained asymptomatic at 1-year follow-up.

Discussion

This case exemplifies occult pheochromocytoma predominantly manifested as atypical cardiac symptoms (episodic palpitations and acute chest distress) with normotension and near-normal catecholamine concentrations, an exceedingly rare clinical presentation that greatly hampered preoperative diagnosis. The attending cardiologist initially attributed the patient's chest pain and myocardial injury to the LAD myocardial bridge, given the 60–70% systolic compression observed on coronary angiography. This anatomical abnormality provided a plausible explanation for the elevated troponin and electrocardiographic changes, creating an anchoring bias that precluded further consideration of catecholamine-mediated disorders. However, several critical findings were incongruent with a myocardial bridge etiology. First, the focal wall motion abnormality was confined to the basal inferior segment, a territory supplied by the right coronary artery rather than the bridged LAD; thus, the segmental distribution of myocardial dysfunction was inconsistent with single-vessel coronary ischemia. Second, the absence of diffuse apical or anterior wall involvement argued against significant flow limitation from the LAD myocardial bridge. Third, the rapid and complete resolution of basal inferior hypokinesia with normalization of LVEF to 58% within 7 days postoperatively is characteristic of transient stress-induced cardiomyopathy rather than fixed structural coronary disease. These observations, combined with the intraoperative hypertensive crisis triggered by mere patient repositioning, ultimately redirected the diagnostic reasoning toward occult pheochromocytoma-induced Takotsubo syndrome.

Undiagnosed pheochromocytoma is associated with a high risk of catecholamine crisis during the perioperative period, with malignant hypertension as the most prominent clinical manifestation, which most commonly occurs during anesthesia induction and surgical tumor manipulation [5, 6]. In this case, the simple maneuver of lower limb repositioning for urethral catheterization triggered a massive release of catecholamines, leading to an episode of sudden severe hypertension. Urapidil, a short-acting, selective α1-adrenergic receptor blocker, was used for initial antihypertensive therapy; the suboptimal response to the first bolus was attributable to the potent vasopressor effects of the large amounts of released catecholamines, and the peak therapeutic effect of the second bolus effectively antagonized the vasopressor effects of circulating catecholamines [7]. The significant drop in blood pressure

following intubation was likely multifactorial, involving anesthetic-induced vasodilation and potential preoperative intravascular volume depletion, a typical pathophysiological alteration in patients with pheochromocytoma.

Following the clinical suspicion of pheochromocytoma, we promptly adjusted the anesthetic management strategy, which included the intensification of invasive hemodynamic monitoring, the implementation of emergency fluid resuscitation, and the combined use of vasoactive agents to control hemodynamic fluctuations. The combination of phentolamine (a non-selective α-adrenergic receptor blocker) and a continuous intravenous infusion of sodium nitroprusside effectively controlled the intraoperative hypertensive crisis, a finding that is consistent with the research results reported by Juszczak et al. [7]. In anticipation of preoperative intravascular volume depletion, a total of 1500 mL of intravenous fluid was administered prior to adrenal vein ligation, which effectively attenuated the degree and duration of post-resection hypotension. Continuous intravenous infusion of norepinephrine achieved a favorable blood pressure support effect following tumor resection, which is the first-line therapeutic option for hypotension in the immediate post-resection phase of pheochromocytoma surgery [5].

This case carries important clinical implications for clinicians involved in the management of patients with adrenal space-occupying lesions: (1) For patients presenting with unexplained chest pain or myocardial injury in conjunction with an adrenal space-occupying lesion, the possibility of occult pheochromocytoma should be considered, even in the presence of normotension and non-diagnostic catecholamine test results; (2) Sudden severe intraoperative hypertension is a pivotal diagnostic clue for occult pheochromocytoma; (3) For patients in whom pheochromocytoma is suspected intraoperatively, aggressive fluid resuscitation, rational administration of α/β-adrenergic receptor blockers and real-time invasive hemodynamic monitoring are the key measures to maintain perioperative hemodynamic stability; (4) Preoperative alpha-blockade and volume expansion remain the gold standard for known pheochromocytoma, and delaying surgery for medical optimization is mandatory whenever the diagnosis is established preoperatively [5]. However, when occult pheochromocytoma is unexpectedly recognized intraoperatively and discontinuation is not clinically viable—as in the present case, where the family insisted on proceeding after full risk disclosure—immediate aggressive hemodynamic optimization, including real-time invasive monitoring, aggressive fluid resuscitation, and titrated multimodal vasoactive therapy, is mandatory. A well-formulated emergency anesthetic protocol should therefore be instituted without delay to mitigate the risks of catecholamine crisis and post-resection cardiovascular collapse.

A limitation of this case report is that no further genetic testing was performed for the patient, and thus the potential genetic background of the pheochromocytoma remains unexplored. In future clinical practice, genetic testing can be considered for such patients to provide a more comprehensive diagnostic and therapeutic approach.

Pheochromocytoma can present with atypical clinical manifestations such as atypical cardiac symptoms, in the absence of hypertension and with non-diagnostic catecholamine test results, which

renders the condition prone to missed diagnosis and misdiagnosis. Sudden intraoperative hemodynamic fluctuations are an important diagnostic clue for this atypical form of pheochromocytoma. Thorough preoperative evaluation, vigilant intraoperative invasive hemodynamic monitoring and the timely and rational administration of vasoactive agents constitute the core principles of perioperative anesthetic management for such patients, which can effectively reduce the incidence of perioperative complications and improve surgical safety.

Declarations

Disclosures

Previous presentation in conferences: Not applicable

Conflicts of interest:

No potential conflict of interest relevant to this article was reported.

Ethics approval:

This study was approved by the Ethics Committee of the Affiliated Hospital of Jining Medical University (IRB number: 2025-05-C022).

Informed consent:

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Clinical trial registration number

Not applicable

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Author Contribution

Conceptualization and Data curation Jing-Jing Cui,, Feng Yang. Investigation and Methodology: Jing-Jing Cui, Feng Yang, Supervision, Validation,Visualization: Ai-Mei Li.Writing – original draft: Jing-Jing Cui,Writing – review and editing: Jing-Jing Cui; Ai-Mei Li

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Figures

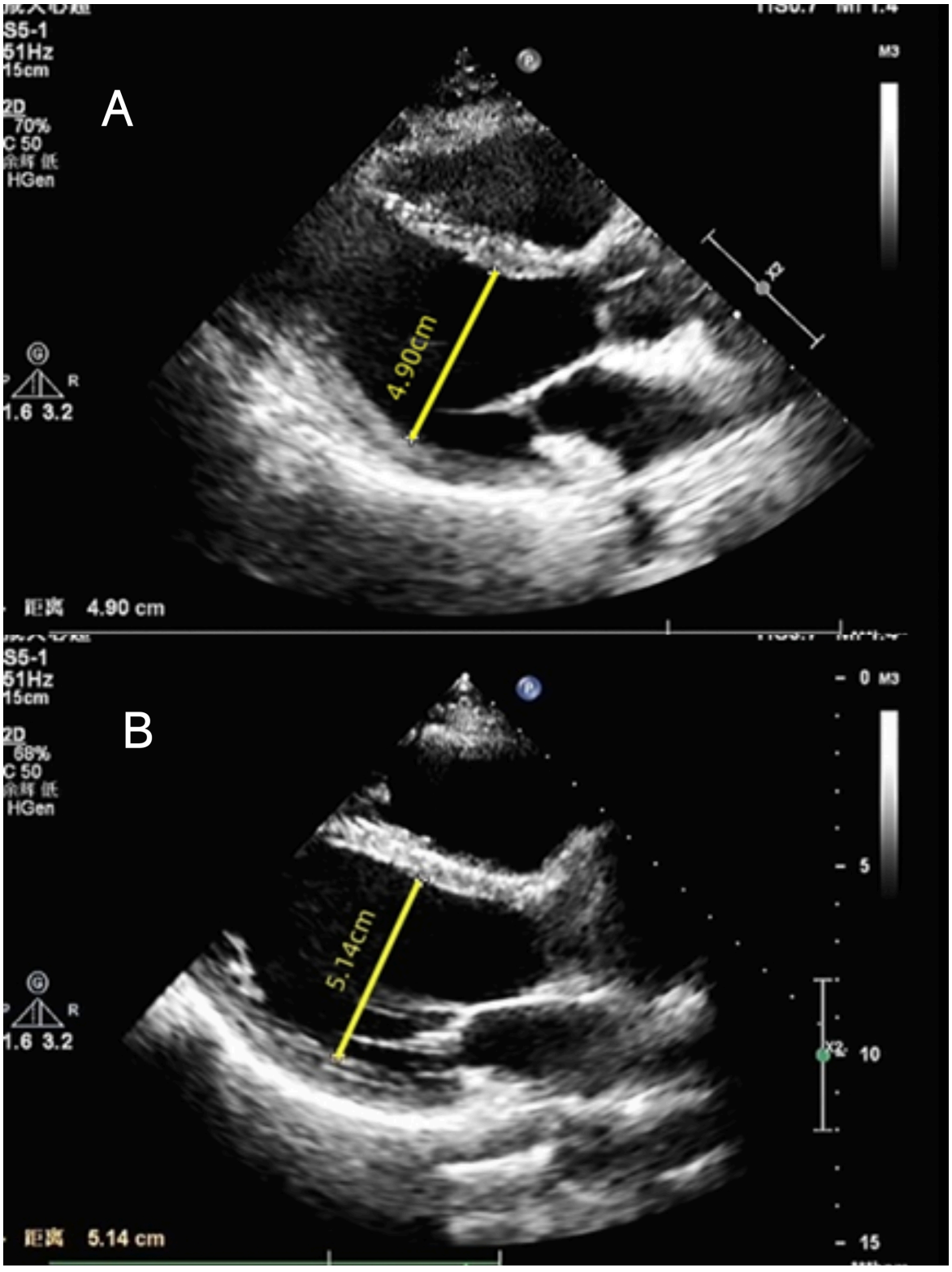

Figure 1

Cardiac ultrasound before and after surgery.

A) Preoperative echocardiography showed a left ventricular transverse diameter of 5.14cm.

B) Postoperative echocardiography showed a left ventricular transverse diameter of 4.90cm.

Image not available with this version

Figure 2

Abdominal CT imaging of pheochromocytoma patients.

The right-sided mass indicated by the arrow is an adrenal space-occupying lesion (later confirmed as pheochromocytoma by histopathology).

Supplementary Files

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