

CASE REPORT

Pre-operative tachycardia is not always a sign of sepsis or pain: a case report of pheochromocytoma

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ABSTRACT

Pre-operative tachycardia is a frequent clinical manifestation commonly attributed to pain, anxiety, or systemic infection. However, uncommon endocrine emergencies such as pheochromocytoma may present with nonspecific cardiovascular instability and remain clinically unrecognized until catastrophic perioperative deterioration occurs. This case report describes a 26-year-old female who presented with a painful hyperemic right breast mass accompanied by persistent tachycardia and respiratory distress. In accordance with CARE case report principles, clinical data were retrospectively collected from medical records, including serial hemodynamic monitoring, laboratory investigations, electrocardiography, thoracic imaging, perioperative management, and intensive care interventions. Initial evaluation demonstrated persistent tachycardia (153 bpm), tachypnea, and hypertensive episodes that were disproportionate to the localized breast pathology. Despite surgical intervention and initial postoperative stabilization, the patient developed progressive hemodynamic instability characterized by persistent tachyarrhythmia, a Burch-Wartofsky Point Scale score of 20, severe hyperglycemia (858 mg/dL), ketonuria, and electrolyte imbalance, which eventually culminated in asystolic cardiac arrest. This constellation of cardiovascular and metabolic abnormalities strongly suggested a pheochromocytoma crisis precipitated by surgical stress and excessive catecholamine release. Catecholamine-induced insulin resistance and impaired peripheral glucose utilization likely contributed to the profound metabolic derangement observed in this patient. This case underscores the importance of recognizing unexplained perioperative tachycardia and hyperglycemia as potential indicators of occult pheochromocytoma. Early endocrine evaluation and appropriate perioperative preparation are critical to prevent fatal cardiovascular complications and improving surgical outcomes.

Keyword: Case report, catecholamines, hypertension, pheochromocytoma, tachycardia.

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INTRODUCTION

Pre-operative tachycardia is a common finding usually attributed to sepsis, pain, or anxiety. However, it may also represent a manifestation of a more serious endocrine emergency such as pheochromocytoma. Pheochromocytoma is a catecholamine-producing neuroendocrine tumor arising from the chromaffin cells of the adrenal medulla. Although uncommon, it carries significant perioperative mortality when the disease remains unrecognized. Patients may present with episodic hypertension, palpitations, diaphoresis, headache, hyperglycemia, or cardiovascular collapse triggered by stress, anesthesia, or surgery. This case report aims to highlight the importance of comprehensive pre-operative evaluation and the early recognition of unexplained tachycardia as a potential sign of a pheochromocytoma crisis.

METHODS AND SUBJECT

This case report describes a 26-year-old female patient who was admitted for surgical management of a right breast mass. Informed consent was obtained from the patient's family, and patient's identity was anonymized throughout the manuscript. Institutional ethical review was waived because this report involved a retrospective analysis of a single clinical case without intervention beyond standard medical care. Clinical data were retrospectively collected from medical records during the patient's hospitalization in August 2025. Data extraction was independently reviewed by the authors and included the clinical presentation timeline, perioperative monitoring, laboratory investigations, imaging findings, electrocardiography (ECG), glucose measurements, urinalysis, medications

administered, anesthetic management, intensive care interventions, and patient outcomes. Diagnostic evaluations included serial blood pressure measurement, thoracic imaging, ECG monitoring, blood glucose testing, electrolyte analysis, and urinary ketone analysis. The Burch-Wartofsky Point Scale (BWPS) was applied to assess the possibility of a thyroid storm, where a score below 25 suggests that a thyroid storm is unlikely. Perioperative management included the surgical intervention for the breast mass, administration of propranolol, postoperative intensive monitoring, mechanical ventilation, and advanced cardiovascular resuscitation measures. The patient was followed until her death on 9th August 2025.

RESULTS AND DISCUSSION

Results

The patient was initially admitted to the hospital with tachycardia, tachypnea, and a hyperemic mammary mass. Although, she was originally scheduled to undergo a surgical procedure, her symptoms worsened, prompting her to present to the Emergency Department (ED). In the ED, the patient immediately received Cefixime (200 mg) and Dexketoprofen (25 mg) orally. Due to the patient's deteriorating condition, the surgeon decided to proceed with the operation intervention. The surgery proceeded smoothly, the patient's vital signs were stable, and the patient was subsequently transferred to the recovery room. During observation, the patient exhibited hypertension (125/86 mmHg) and a Burch-Wartofsky Point Scale score of 20. The condition deteriorated to severe hyperglycemia (858 mg/dL), ketonuria, and electrolyte imbalance, eventually leading to asystole.

Table 1. Clinical Hemodynamic and Metabolic Progression

Time / Location	Clinical Findings & Vital Signs	Metabolic & Laboratory Status
Surgical Clinic (06 Aug 2025)	The patient presented with a painful mass in the right breast, fever and shortness of breath for the past three days. Tenderness was noted in the right mammary region. The patient was scheduled for surgical intervention.	-
Emergency Department (07 Aug 2025)	The patients presented with tachycardia (153 bpm), tachypnea, a febrile state, and hyperemic mammary mass.	Initial laboratory results and thoracic imaging findings were within normal limits. The patients underwent stabilization and preparation.
Recovery Room (07 Aug 2025)	The patient exhibited hypertension (125/86 mmHg), persistent tachycardia (135 bpm), and a BWPS score of 20.	An ECG was performed, and propranolol 10 mg was administered (resulting in a heart rate of 122 bpm).
Post-Operative Room (07 Aug 2025)	The patient developed declining consciousness, Stage 1 hypertension (139/93 mmHg), and tachycardia (123 bpm).	A GDS level of 858 mg/dL, ketonuria, and electrolyte imbalance were detected.
ICU (07 Aug 2025) (08 Aug 2025) (09 Aug 2025)	The patient was intubated and placed on mechanical ventilation. The patient developed a decrease in blood pressure (112/67 mmHg), tachycardia (173 bpm), cold acral, and a CRT >3s. There was an absence of pulse and spontaneous respiration, absent pupillary light reflexes bilaterally, and electrocardiography (ECG) showed asystole. The family requested a do-not-resuscitate (DNR) order. The patient was pronounced deceased at 14:22.	Blood sugar decreased to 456 mg/dL; ECG progressed to asystole despite resuscitation.

Table 2. Patient Progress Based on Operative Processes

Operative Process	Vital Sign	Physical Examination	Laboratory Findings
Pre-Operative	• Temperature 38,2 C • Respiratory Rate: 25x/minutes • Blood Pressure: 100/60 mmHg • Heart Rate: 153 bpm • Peripheral Oxygen Saturation: 97%	Local Status ad regio mammae dextra: swelling, hyperemia, and tenderness.	Initial laboratory results and thoracic imaging findings were within normal limits. The patient underwent stabilization and preparation.
Intra-Operative	• Temperature 37,4 C • Respiratory Rate: 16x/minutes • Blood Pressure: 120/60 mmHg • Heart Rate: 80 bpm • Peripheral Oxygen Saturation: 99 % on O₂ Ventilation through LMA.	Not conducted.	Not conducted.
Post-Operative	• Temperature 36,2 C • Respiratory Rate: 22x/minutes • Blood Pressure: 119/91 mmHg • Heart Rate: 153 bpm • Peripheral Oxygen Saturation: 99% on O₂ 3 lpm through Nasal Canule	Local Status ad regio mammae dextra surgical wound covered with gauze	A GDS level of 858 mg/dL, ketonuria, and electrolyte imbalance were detected.

Discussion

The clinical manifestations were driven by excessive catecholamine production, which normally maintains homeostasis but can also cause paroxysmal hypertension and tachyarrhythmia.^{1,8} Catecholamines also increase insulin resistance and impair glucose uptake, explaining the patient's hyperglycemia and ketosis.^{5,6} A critical weakness in this case was that the initial clinical assessment focused primarily on localized infection as the cause of the patient's symptoms, while the possibility of an underlying neuroendocrine disorder such as pheochromocytoma was not considered early in the diagnostic process. This narrow diagnostic approach may have delayed recognition of catecholamine excess, especially in patients presenting with persistent pre-operative tachycardia, labile blood pressure, diaphoresis, and unexplained cardiovascular instability.⁷

Statement of Main Findings

This case highlights a fatal perioperative progression of a suspected pheochromocytoma, initially masked by a localized inflammatory presentation of a breast mass.^{3,4} The patient demonstrated a classic hemodynamic instability triad; persistent tachycardia, paroxysmal hypertension, and sudden cardiovascular collapse, triggered by the physiological stress of surgery.^{1,10,11} These findings may represent more than surgical stress alone and are highly suggestive of a pheochromocytoma crisis, a hypermetabolic state characterized by life-threatening multi-organ failure.^{3,4}

1) Relation to Previous Studies and Pathophysiology

The patient's initial heart rate of 153 bpm was disproportionate to the clinical presentation of a mammary tumor. Previous studies emphasize that pheochromocytoma acts as "the great masquerader," often being mistaken for sepsis or anxiety.^{8,10} While the Burch-

Wartofsky Point Scale (BWPS) (which has a several parameters such as thermoregulatory dysfunction, central nervous system effects, gastrointestinal or hepatic dysfunction, cardiovascular dysfunction, congestive heart failure severity, and precipitating event) score of 20 correctly suggested that a thyroid storm was unlikely, it served as a critical clinical indicator that the tachycardia was driven by a hypermetabolic surge.^{12,13}

The excessive production of catecholamines (norepinephrine and epinephrine) plays a central role in maintaining this state of instability.^{1,8} Norepinephrine (NE) and epinephrine (EPI) produce different cardiovascular effects because they act on different adrenergic receptors in the heart and blood vessels. NE predominantly stimulates α 1-adrenergic receptors on vascular smooth muscle, causing intense peripheral vasoconstriction that markedly increases systemic vascular resistance and blood pressure, which may progress to a hypertensive crisis. In contrast, EPI has a stronger affinity for β 1-adrenergic receptors in the heart, leading to increased heart rate, myocardial excitability, and conduction velocity, thereby predisposing patients to paroxysmal tachyarrhythmias such as ventricular tachycardia and atrial fibrillation.

Previous studies have further shown that excessive β 1 stimulation increases cyclic AMP and calcium influx in cardiac cells, enhancing automaticity and triggering malignant arrhythmias. In severe cases, recurrent catecholamine surges can lead to profound electrical instability, myocardial exhaustion, circulatory collapse, and eventually asystole, in the intensive care unit (ICU), in which cardiac electrical activity ceases completely. These life-threatening events are particularly common in pheochromocytoma patients with uncontrolled catecholamine secretion and require rapid hemodynamic stabilization and antiarrhythmic management.^{1,11}

2) Metabolic Masquerade and Pseudodiabetes

A striking feature of this case was the extreme hyperglycemia (GDS 858 mg/dL) and ketonuria. This phenomenon is described in the literature as “pseudodiabetes”.^{3,6} High levels of circulating catecholamines, particularly epinephrine and norepinephrine released by pheochromocytoma, profoundly disrupt normal glucose metabolism by impairing insulin activity and increasing glucose production. Catecholamines activate α_2 -adrenergic receptors located on pancreatic β -cells, which suppresses insulin secretion and reduces the body’s ability to lower blood glucose levels. At the same time, these hormones stimulate glycogenolysis, the process by which glycogen stored in the liver and muscles is rapidly broken down into glucose, leading to an acute rise in blood sugar concentration.

In addition, catecholamines enhance gluconeogenesis, a metabolic pathway that generates new glucose from amino acids and other substrates in the liver. Simultaneously, peripheral tissues such as skeletal muscle and adipose tissue become less responsive to insulin, resulting in decreased cellular glucose uptake and worsening hyperglycemia. This combination of reduced insulin secretion, excessive hepatic glucose output, and peripheral insulin resistance creates a severe catecholamine-induced diabetic state.

In patients with pheochromocytoma, these metabolic disturbances may become especially prominent during periods of stress, tumor manipulation, or intraoperative catecholamine surges. Persistent hyperglycemia may further aggravate cardiovascular stress, impair wound healing, and increase postoperative morbidity. Therefore, careful perioperative monitoring of glucose levels and hemodynamic status is essential in patients with catecholamine-secreting tumors.⁶ Simultaneously, catecholamines stimulate glycogenolysis and gluconeogenesis while decreasing glucose uptake in peripheral

tissues.^{5,6} This metabolic environment promotes the ketosis observed in our patient, mimicking diabetic ketoacidosis but driven entirely by the adrenal tumor.^{4,6}

3) Weaknesses and Strengths in Study

The most important part of this discussion is acknowledging the diagnostic limitations. The clinical relevance of this case is high for surgical practitioners, as it illustrates how source control for a localized infection (the breast mass) may be insufficient if a systemic neuroendocrine condition is overlooked.^{7,9} A major limitation of this study was the absence of pre-operative biochemical screening (Metanephrines testing), as the clinical focus was diverted by the hyperemic breast mass.^{2,9} However, the strength of this study lies in its honest and succinct documentation of the transition from stable surgical status to multi-organ failure, providing a vital lesson in vigilance for persistent tachycardia.^{3,11}

4) Additions to Knowledge and Future Directions

This case adds to the knowledge that tachycardia should not be treated as a symptom in isolation.^{1,9} We highlight the implications that the combination of tachycardia and severe hyperglycemia without a history of diabetes serve as a red flag for pheochromocytoma.^{5,6,8} Future studies should establish standardized screening protocols that incorporate bedside electrocardiography (ECG) and glucose monitoring as early diagnostic tools for patients presenting with unexplained hemodynamic instability, particularly those with fluctuating hypertension, tachycardia, arrhythmias, or episodic hypotension suggestive of pheochromocytoma. The literature emphasizes that excessive catecholamine secretion can produce severe cardiovascular manifestations, including hypertensive crisis, myocardial ischemia, cardiomyopathy, and malignant arrhythmias, which may initially mimic other acute cardiac conditions.

Continuous bedside ECG monitoring could therefore facilitate early detection of transient tachyarrhythmias, ST-segment abnormalities, QT prolongation, or catecholamine-induced electrical instability before catastrophic cardiovascular collapse occurs. Simultaneously, bedside glucose monitoring is important because catecholamine excess suppresses insulin secretion through α 2-adrenergic receptor activation and promotes glycogenolysis and gluconeogenesis, leading to stress-induced hyperglycemia and metabolic instability. The integration of these simple and noninvasive tools into emergency and intensive care settings may improve the early recognition of occult pheochromocytoma in patients with unexplained hemodynamic swings.^{7,9,14}

These findings further highlight that perioperative hemodynamic fluctuations remain a major challenge in pheochromocytoma management and require meticulous cardiovascular surveillance and individualized monitoring strategies. Early ECG and glucose abnormalities may therefore serve as important warning signs that prompt further biochemical testing for catecholamine excess. Screening protocols could potentially reduce delays in diagnosis, prevent severe cardiovascular complications, and improve perioperative outcomes through earlier hemodynamic stabilization and targeted treatment.^{7,9,14} Furthermore, this case reinforces that any elective surgery should be postponed until the patient is cooled down with alpha-blockade for at least 7–14 days to prevent the intraoperative crisis seen here.^{2,9,15}

CONCLUSION

This case emphasizes that persistent pre-operative tachycardia accompanied by unexplained hypertension and hyperglycemia should prompt consideration of pheochromocytoma as a differential diagnosis. Early recognition and appropriate perioperative endocrine

evaluation are essential to reduce the morbidity and mortality associated with a pheochromocytoma crisis.

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DECLARATION OF INTERESTS

The authors declare that they have no conflict of interest.

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