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CASE REPORT

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Unexpected pheochromocytoma leading to cardiac arrest during the perioperative period: a case report and literature review

Jian Guo[1], Yuting Qiu[1], Xiaojin Zhang[2], Yitao Qian[1] and Jianhong Xu[1*]

Abstract

Background Pheochromocytomas (PCCs) are rare neuroendocrine catecholamine (CA)-secreting tumours that originate from chromaffin tissue and can produce and store CAs. Unexpected PCCs pose a serious threat to the perioperative safety of patients and a considerable challenge to anaesthesiologists because of the risks of fatal hypertensive crises and other stresses.

Case presentation A 37-year-old woman who was scheduled for tonsillectomy and palatopharyngoplasty under general anaesthesia experienced a malignant cardiovascular event after induction, which was characterized mainly by a sharp increase in heart rate and blood pressure, ultimately leading to cardiac arrest and the occurrence of secondary long QT syndrome. Based on the perioperative clinical manifestations, measurements of plasma and urinary CAs, postoperative bilateral adrenal computed tomography results and surgical pathological results, the patient was diagnosed with an undiagnosed PCC.

Conclusions Anaesthesiologists should pay attention to patients with recurrent chest tightness, as these patients may have an undiagnosed PCC. Extreme hypertension and tachycardia during the perioperative period may indicate a PCC. We should not automatically use beta-adrenergic receptor blockade while overlooking the importance of alpha-adrenergic receptor blockade. If a serious malignant cardiovascular event occurs in patients with an undiagnosed PCC during the perioperative period, multidisciplinary comprehensive treatment is crucial.

Keywords Pheochromocytoma, Cardiac arrest, Long QT syndrome

Background

Pheochromocytomas (PCCs) are rare neuroendocrine tumours with a prevalence ranging from 0.05 to 0.1% [1]. PCCs are characterized by excessive release of catecholamines (CAs) and are often neglected because they present symptoms similar to those of other prevailing clinical conditions, such as the classic triad of headaches, palpitations, and profuse sweating, as well as a variety of other signs and symptoms. Furthermore, a small proportion of patients with PCCs are asymptomatic [1–4]. PCCs are associated with increased risks of perioperative haemodynamic instability and life-threatening events during

*Correspondence:
Jianhong Xu
1197058@zju.edu.cn
[1]Department of Anaesthesiology, The Fourth Affiliated Hospital, School of Medicine, Zhejiang University, Yiwu, Zhejiang 322000, China
[2]Department of Obstetrics, The Fourth Affiliated Hospital, School of Medicine, Zhejiang University, Yiwu, Zhejiang 322000, China

anaesthesia; therefore, the anaesthetic management of patients with PCCs is far from satisfactory, even though various techniques are available [5, 6]. Unexpected PCCs, especially in patients who have undergone other types of surgeries, pose significant intraoperative challenges to anaesthesiologists [7].

In October 2023, a patient who was scheduled for tonsillectomy and palatopharyngoplasty was treated in our hospital. The patient experienced significant haemodynamic fluctuations after anaesthesia induction, ultimately leading to cardiac arrest. After comprehensive multidisciplinary treatment, the patient recovered from crisis to safety. Eventually, the patient was diagnosed with an undiagnosed PCC. The patient underwent extremely dangerous surgical and anaesthesia procedures. This rare case highlights that anaesthesiologists should devote special attention to patients with an undiagnosed PCC who are undergoing other types of surgeries.

Case presentation

A 37-year-old woman was admitted to the hospital after complaining of snoring for 3 years. Twenty-four-hour respiratory sleep monitoring revealed an apnoea hypopnea index (AHI) of 28.9, with a minimum oxygen saturation of 90%. She was diagnosed with tonsillitis and obstructive sleep apnoea hypopnea syndrome and was scheduled for tonsillectomy and palatopharyngoplasty. The patient had previously reported good physical activity, with 8 metabolic equivalents and a New York Heart Association class I. The patient reported a history of seeking medical attention due to chest tightness, but no significantly abnormal results, including heart rate, dynamic electrocardiogram, myocardial enzyme spectrum, or cardiac troponin levels, were found. The electrocardiogram was normal, with a QT interval of 380 ms and a QTc interval of 467 ms (the normal QTc interval for females should be less than 450 ms). The results of lung computed tomography (CT) and related blood tests were normal.

Anaesthesia was induced using 0.3 mg/kg remimazolam tosilate, 0.5 μg/kg sufentanil, and 0.6 mg/kg rocuronium. The patient developed a sudden paleness of the lips and facial features, accompanied by a gradual increase in heart rate and blood pressure (BP). The maximum heart rate (HR) was 180 bpm, and the highest BP was 223/170 mmHg. A single dose of esmolol (30 mg) was administered via intravenous (IV) injection, but no significant improvement occurred. After multiple intermittent doses of esmolol 30 mg IV (cumulative dose of esmolol, 120 mg), the HR decreased to 116 bpm, and the BP decreased to 80/53 mmHg and epinephrine 80 μg was administered IV immediately. After 5 min, HR decreased to 78 bpm and BP decreased to 44/23 mmHg. No significant changes in HR and BP were detected after 20 μg of adrenaline was administered intravenously. When the auscultation of the heart was unclear, an additional dose of adrenaline (1 mg) was administered, and immediate cardiopulmonary resuscitation was performed. Reassessment revealed a HR of 160 bpm and a BP of 167/111 mm Hg after 3 min. Successful left radial artery puncture and right internal jugular vein puncture catheterization were performed after the arrival of the multidisciplinary rescue team led by anaesthesiologists. Real-time blood gas analysis revealed a pH value of 7.235, blood glucose level of 22.6 mmol/L, blood potassium level of 2.7 mmol/L, blood lactate level of 11.4 mmol/L, and a base excess (BE) value of -7.0. Two hundred millilitres of sodium bicarbonate, 6 U of insulin, 20 ml of 10% potassium chloride and 40 mg of methylprednisolone were subsequently administered for symptomatic supportive treatment. After treatment with 25 mg of propafenone and 50 mg of amiodarone via IV injection, there were no significant changes in heart rate. The HR decreased from 160 bpm to 110 bpm after implementing synchronized electroconversions at 50 J. The circulatory system stabilized after maintenance with low doses of norepinephrine and adrenaline. The patient was transferred to the intensive care unit (ICU) for further treatment after immediate surgery.

Further tests performed in the ICU revealed that the levels of troponin, creatine kinase MB, and lactate dehydrogenase; activated partial thromboplastin time; and thrombin time were elevated. The patient developed persistent hypokalaemia, with a minimum blood potassium level of 2.1 mmol/L. Despite active potassium supplementation treatment, the patient still experienced ventricular fibrillation 15 h after being transferred to the ICU. An electrocardiogram revealed frequent recurrent premature ventricular contractions, short paroxysmal twisted ventricular tachycardia attacks and ST segment elevation. Emergency coronary angiography was performed by a cardiologist and revealed that there was no significant stenosis in any coronary segment. Based on the patient's medical history and electrocardiogram results, the possibility of long QT syndrome (LQT) was considered. To avoid the use of drugs that prolong the QT interval, supplementation with potassium and magnesium to maintain K+at 4.0 mmol/L or above was performed to treat LQT. The tracheal catheter was removed two days after the patient was transferred to the ICU. After 5 days of comprehensive treatment in the ICU, the patient was transferred to the general ward. The detailed clinical process is shown in Table 1.

Two days after being transferred to the general ward, the patient's bilateral adrenal CT scan revealed a tumour on the left adrenal gland measuring 4.14 cm×3.80 cm (Fig. 1). Blood CA measurements revealed that her free methoxyepinephrine level was >1000 pg/ml and her free methoxyepinephrine level was >500 pg/ml. Moreover,

Table 1 Clinical course of the patient

Time	Event	Disposal and response
Day 1		
16:20	Entered the operating room	
16:25	Anaesthesia induction	0.3 mg/kg remimazolam tosilate, 0.5 μg/kg sufentanil, and 0.6 mg/kg rocuronium IV
16:28	Sudden paleness of lips and facial features, accompanied by a gradual increase in HR and BP. The maximum HR was 180 bpm, and the highest BP was 223/170 mmHg.	Esmolol 30 mg IV, no significant improvement. Multiple intermittent doses of esmolol 30 mg IV. Cumulative dose of esmolol 120 mg.
16:33	Tracheal intubation. HR 110 bpm, BP 135/113 mmHg	
16:43	Start of surgery	
16:45	HR 116 bpm, BP 80/53 mmHg	Epinephrine 80 μg IV
16:46	Right tonsillectomy	
16:50	HR 78 bpm, BP 44/23 mmHg, surgery suspended	Adrenaline 20 μg IV, no significant changes
16:51	HR 82 bpm, BP 37/34 mmHg, unclear cardiac auscultation	Immediate chest compressions and calling for help
16:51 – 16:54	Cardiopulmonary resuscitation	Adrenaline 1 mg IV, sodium bicarbonate 250 ml intravenous drip
16:54	Re-evaluation revealed pulse and HR	
16:55 – 17:12	A multidisciplinary rescue team led by anaesthesiologists arrived	Ultrasound-guided left brachial artery and right internal jugular vein puncture catheterization. Adrenaline and norepinephrine pump injection based on circulation parameters. According to HR and BP, methylprednisolone 40 mg, propafenone 25 mg, amiodarone 25 mg, and calcium chloride 0.3 g were administered successively.
17:12	HR 160 bpm, BP 169/103 mmHg	50 J synchronous electric cardioversion. HR reduced from 160 bpm to 110 bpm.
17:15		Transesophageal echocardiography showed no significant abnormalities.
17:20	Auscultation of wet rales in the lungs, bloody discharge seen in the tracheal tube.	Positive end-expiratory pressure 5 cmH_2O
17:23	Blood gas analysis revealed a pH value of 7.235, blood glucose level of 22.6 mmol/L, blood potassium level of 2.7 mmol/L, blood lactate level of 11.4 mmol/L, and BE value of -7.0.	200 ml of sodium bicarbonate, 6 U of insulin, 20 ml of 10% potassium chloride, 40 mg of methylprednisolone and 10 mg furosemide were subsequently administered.
18:00–18:15	Surgery continued, left tonsillectomy, palatopharyngeal reconstruction.	
18:21	Blood gas analysis revealed a pH value of 7.308, blood glucose level of 16.6 mmol/L, blood potassium level of 3.9 mmol/L, blood lactate level of 8.9 mmol/L, and BE value of 1.2.	Adrenaline and norepinephrine pump injection based on circulation parameters.
18:25	Transferred to ICU	
Day 2		
06:02	Ventricular fibrillation	Electric defibrillation, potassium supplementation, amiodarone, adrenaline and norepinephrine pump injection.
07:49	Persistent hypokalaemia with a minimum blood potassium level of 2.1 mmol/L	Potassium supplementation, amiodarone
18:22	Electrocardiogram revealed recurrent frequent premature ventricular contractions, short paroxysmal twisted ventricular tachycardia attacks and ST segment elevation.	Multidisciplinary consultation and emergency coronary angiography were conducted.
Day 3		
14:46	Tracheal tube removal	
Day 5		
10:00	Transferred to the general ward	

24-hour urine CA examination revealed that her free norepinephrine level was 348.3 μg/24 h, and her free epinephrine level was >144.0 μg/24 h. The possibility of PCC was considered given the patient's relevant clinical symptoms, laboratory tests and CT results.

Two months later, the patient underwent left adrenal tumour resection under general anaesthesia. Postoperative pathology revealed left adrenal PCC (Fig. 2).

Discussion and conclusions

In this case report, a young female patient experienced a malignant cardiovascular event during tonsillectomy and palatopharyngoplasty after anaesthesia induction.

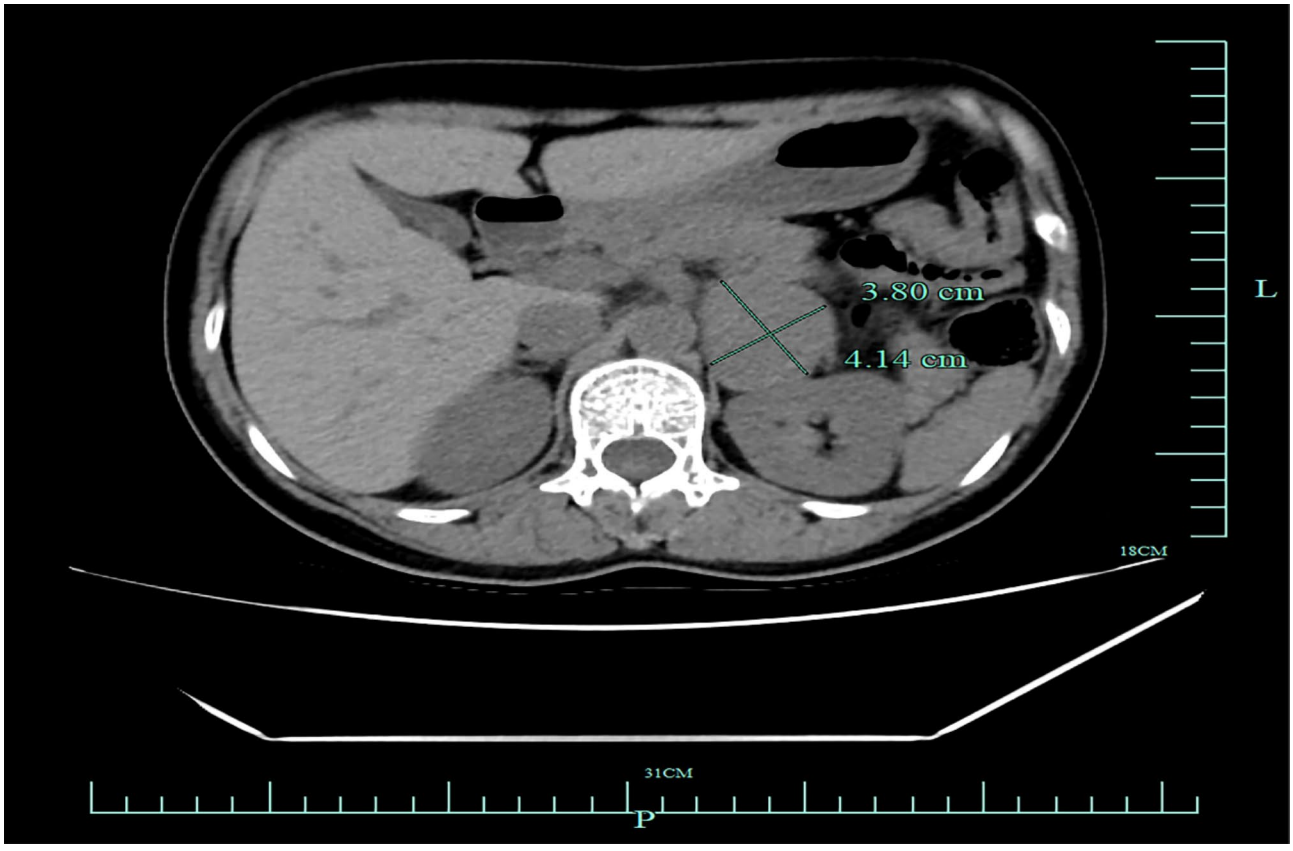

Fig. 1 Bilateral adrenal CT. The left adrenal gland tumour was 4.14 cm×3.80 cm in size

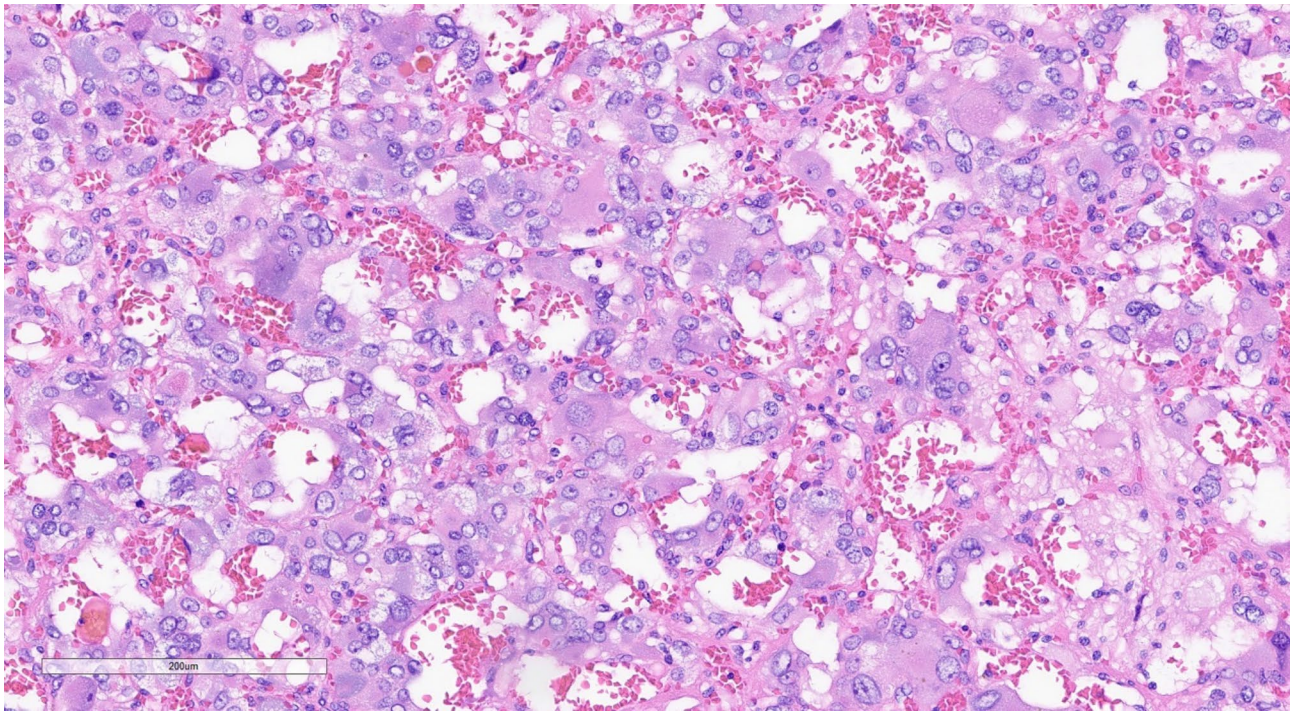

Fig. 2 Pathological diagnosis results. The postoperative pathological diagnosis was PCC. Tumour cells have an alkaline cytoplasm, mild nuclear atypia, a patchy distribution, and abundant interstitial blood vessels

This event was characterized mainly by a sharp increase in heart rate and blood pressure, ultimately leading to cardiac arrest. Based on the perioperative clinical manifestations, postoperative bilateral adrenal CT results, and measurements of plasma and urinary CAs, the patient was suspected to have an undiagnosed PCC. Two months later, the surgical pathological results of the patient's adrenal tumour resection confirmed this suspicion. As previously reported, unexpected PCCs pose a serious threat to the perioperative safety of patients and substantial challenges to anaesthesiologists [7–10].

PCCs are rare neuroendocrine CA-secreting tumours that originate from chromaffin tissue and can produce and store CAs as well as other substances, such as VIP, PTH- and calcitonin-related peptides, opioids, CRH, ACTH, histamine, chromogranin, and interleukin-6 [11–13]. The most common symptoms and signs of PCCs are palpitation, headache and hypertension due to excessive release of CAs, and PCCs need to be differentiated from panic syndrome, thyrotoxicosis, anxiety, hypoglycaemia, etc [2, 3, 14, 15]. PCCs are considered a rare cause of hypertension, occurring in approximately 1 in 500 adults [4]. Only half of patients with PCCs have paroxysmal hypertension or normotension [1]. Among 200 cases of PCC-related crisis identified in 187 articles, the most common symptom was headache (39.5%), the heart was the most common organ damaged by a PCC-related crisis (99.0%), followed by the lungs (44.0%) and the kidney (21.5%) [16]. In this case, the patient was ultimately confirmed to have a PCC. However, the patient's clinical symptoms were atypical, with no hypertension and only chest tightness, leading to a missed diagnosis of a PCC. However, it was also possible that the patient had hypertension that was not detected. A 24-hour BP pattern might be of some diagnostic value for the patient; this procedure requires the attention of anaesthesiologists [17].

PCCs may lead to fatal hypertensive crises under anaesthesia or other stressors; therefore, the diagnosis of PCCs is important [18]. In addition to clinical manifestations, Fang F et al. [2] reported that the diagnosis of PCCs requires both evidence of excessive release of CAs and anatomical localization of CA-secreting tumours. Compared with plasma or urinary CAs, plasma-free metanephrine or urinary fractionated metanephrine seem to have greater diagnostic value for the biochemical diagnosis of PCCs [18, 19]. Witteles RM et al. [20] reported that the most sensitive laboratory diagnostic tests were plasma total CAs (95%) and urine total metanephrine (100%) and that testing for urine vanillylmandelic acid, which is less expensive and easier to perform than many other tests, had a slightly lower sensitivity (89%). CT is more commonly used for detecting PCCs, but its sensitivity is only 88%, whereas magnetic resonance imaging and iodine I-131 metaiodobenzylguanidine scintigraphy have a sensitivity of 100% [21]. The incidence of PCCs has been increasing over time due to the increasing demand for imaging exams and improved methods for measuring CA metabolites [22]. To date, germline mutations in five genes have been identified to be responsible for familial PCCs [18]. Early diagnosis of PCCs is highly important, and physicians need to be familiar with the relevant clinical manifestations and diagnostic steps to increase the degree of clinical suspicion of PCC and establish a timely diagnosis [15]. Although the patient in our case was young, no family history related to PCC was found. Owing to the atypical clinical symptoms of our patient, we did not conduct further examination for a PCC, which ultimately led to an undiagnosed PCC. For patients with unexplained chest tightness, CT of the adrenal gland and levels of plasma total CAs and urine total metanephrine may help diagnose a PCC.

The perioperative period of patients with PCCs requires comprehensive management. Without adequate preparation of PCCs, the release of excessive amounts of CAs, especially during anaesthetic induction or surgical removal, can produce life-threatening cardiovascular complications and other systemic disturbances [23]. Preoperative sequential use of alpha-adrenergic receptor blockade and volume expansion followed by beta blockade is helpful for achieving cardiovascular stability and decreasing uncontrolled intraoperative surges in blood pressure [19, 24]. The use of β-blockers should be maintained when tachycardia and tachyarrhythmias are present, but they should always be administered after effective control of hypertension with α-blockade; on average, β-blockers may be used 3 days after the introduction of α-blockade [3]. Compared with anaesthetic drugs and procedures, surgery for PCC removal is associated with hypertensive emergencies, and tumour size and location affect the number of crises and the dose of antihypertensives used [25]. Therefore, for anaesthesiologists, sufficient preoperative preparation should include determining the location and size of the tumour. Safe anaesthesia still depends on adequate preoperative preparation and assessment together with careful monitoring during the perioperative period [26, 27]. In terms of anaesthesia methods for patients with PCC, combined epidural-general anaesthesia was not effective in attenuating hypertensive responses but could have exacerbated perioperative hypotension [28, 29]. Perioperative haemodynamic management in patients with PCC requires a thorough understanding of the pharmacology of various drugs [30]. Additional antihypertensive (mainly alpha-blocking) agents are also essential to prevent and overcome hypertensive crises during anaesthesia. Preoperative and intraoperative beta-blockade can only be used as adjuvant therapy, mainly to control tachycardia and

other rhythm disturbances [6]. Nonselective β-adrenergic blockade should not be used as the treatment of choice for PCC patients before α-blockade is administered because cardiovascular collapse may occur [9]. Compared with esmolol, landiolol is a better choice because of its high cardioselectivity, high β1 selectivity and receptor affinity, resulting in a more potent chronotropic effect and a less potent hypotensive effect [31]. As reported in our case, if a PCC is misdiagnosed, preoperative preparation is incomplete, and serious cardiovascular events may occur during anaesthesia induction. When severe hypertension and tachycardia occur, the importance of alpha-blocking agents should be prioritized, since excessive and frequent use of esmolol might be associated with severe secondary hypotension and cardiac arrest. Fortunately, we identified cardiac arrest early and implemented cardiopulmonary resuscitation as early as possible, which provided valuable time for the recovery of the patient's heart function. The patient was saved because multidisciplinary comprehensive treatment was performed during the perioperative period.

The patient was also diagnosed with LQT in the ICU. LQT represents a heterogeneous family of cardiac electrophysiologic disorders characterized by QT prolongation and T-wave abnormalities on the electrocardiogram, which are commonly associated with syncope; however, sudden cardiac death can occur due to torsades de pointes [32]. Advanced age, hypokalaemia, a history of heart failure, and structural heart disease could increase the risk of QT prolongation [33]. During the perioperative period, patients are exposed to numerous pharmacologic agents and physiologic perturbations that are known to prolong the QTc interval, agents that are torsadogenic, or both [34]. In this case, the preoperative QTc interval of the patient was slightly prolonged, but it did not meet the diagnostic criteria for LQT according to the consultation opinion of cardiovascular physicians. However, the patient was diagnosed with acquired LQT in the ICU, which may be related to perioperative medication and cardiopulmonary resuscitation after cardiac arrest. Maintaining electrolyte stability and avoiding hypoxia, hypocapnia, hypercapnia, and hypothermia were important for the treatment of acquired LQT in this patient [34]. Fortunately, the patient experienced significant improvement after comprehensive multidisciplinary treatment.

Conclusions

In this case, we reported a malignant cardiovascular event related to an undiagnosed PCC after anaesthesia induction. The clinical manifestations of the patient were atypical and included mainly chest tightness. Our experience emphasizes that anaesthesiologists should pay attention to patients with recurrent chest tightness, as these patients may have an undiagnosed PCC. Extreme hypertension and tachycardia during the perioperative period may also indicate the presence of a PCC. In response to this situation, anaesthesiologists should not automatically use beta-adrenergic receptor blockade while overlooking the importance of alpha-adrenergic receptor blockade. Excessive and improper use of beta blockade may lead to cardiac arrest and the occurrence of secondary LQT. If a serious malignant cardiovascular event occurs in patients with undiagnosed PCC during the perioperative period, multidisciplinary comprehensive treatment is crucial.

Abbreviations

PCCs	Pheochromocytomas
CAs	Catecholamines
AHI	Apnea hypopnea index
LQT	Long QT syndrome
CT	Computed tomography
IV	Intravenous
ICU	Intensive care unit
BP	Blood pressure

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Author contributions

Jian Guo contributed to performing of anesthesia and drafting of manuscript. Yuting Qiu, Xiaojin Zhang and Yitao Qian contributed to data collection. Jianhong Xu contributed to performing of anesthesia and revising the manuscript. All authors have read and approval the final manuscript.

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Data availability

All data related to this case report are contained within the manuscript.

Declarations

Ethics approval and consent to participate

All methods were performed in accordance with the relevant guidelines and regulations. Written informed consent for publication of the clinical details and clinical images was obtained from the patient.

Consent for publication

Written informed consent for publication of the clinical details and clinical images was obtained from the patient.

Competing interests

The authors declare no competing interests.

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