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Bidirectional Ventricular Tachycardia due to Pheochromocytoma: A Case Report

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ABSTRACT

Pheochromocytoma, a type of neuroendocrine tumor, can cause numerous symptoms and signs similar to those of other clinical conditions, with the classic triad being palpitations, headache, and diaphoresis. Patients with pheochromocytoma can present with various cardiac complications, including myocarditis, acute coronary syndromes, cardiomyopathy, heart failure, and arrhythmias. Here we report a case of pheochromocytoma that first presented with bidirectional ventricular tachycardia. The patient was initially diagnosed with acute viral myocarditis and was treated accordingly. A pheochromocytoma crisis with severe blood pressure fluctuation occurred after glucocorticoid administration, leading to further diagnostic work-up, which eventually revealed the adrenal pheochromocytoma.

1 | Introduction

Pheochromocytoma is a rare catecholamine-secreting neuroendocrine tumor that arises from the chromaffin cells of the adrenal medulla and sympathetic ganglia. The clinical presentation of pheochromocytoma includes paroxysmal hypertension, palpitations, headache, diaphoresis, and so on (Lenders et al. 2005). Catecholamine-induced cardiac injury is not uncommon. Cardiovascular complications of pheochromocytoma include myocarditis, acute coronary syndromes, cardiomyopathy, heart failure, arrhythmias, or even cardiac arrest (Afana et al. 2019). Here we report a case of pheochromocytoma that presented with severe heart failure accompanied by bidirectional ventricular tachycardia.

2 | Case Presentation

The patient, a 55-year-old male bus driver, was admitted to our hospital for the chief complaint of palpitations. He experienced dizziness and headache after taking some Viagra-like traditional Chinese medicine 8 h before the admission and developed palpitations later, without any chest pain, sweating, or episodes of syncope. In the emergency department, an immediate 12-Lead electrocardiogram (ECG) showed bidirectional ventricular tachycardia (see Figure 1) and soon the palpitations resolved spontaneously. A repeat 12-Lead ECG showed sinus tachycardia (see Figure 2). The patient had a history of hypertension for 7 years with chronic intermittent headache and was taking amlodipine routinely. Two weeks before the admission, he had a runny nose and took some cold medicine. Upon admission, his blood pressure rose to a level of 193/126 mmHg (left upper limb) and 192/116 mmHg (right upper limb), with a pulse of 139 bpm. General physical examination showed nothing of significance except for some moist rales at the base of both lungs, and the chest X-ray confirmed some infiltration in both lungs. Lab results are listed in Table 1. White blood cell and neutrocyte counts went up, indicating the presence of pneumonia. Echocardiography showed slight enlargement of the left ventricular chamber (LVD 56 mm), diffused left ventricular

Gui-yang Li and Man-Xin Lin contributed equally to this work.

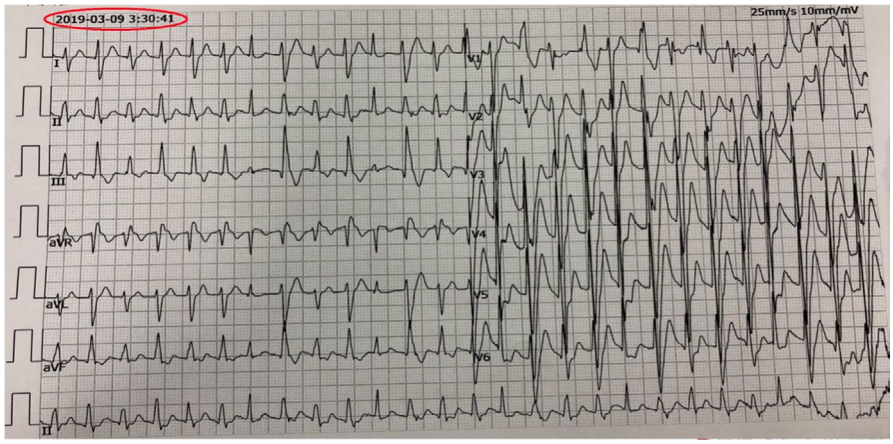

FIGURE 1 | First 12-Lead ECG showing bidirectional ventricular tachycardia.

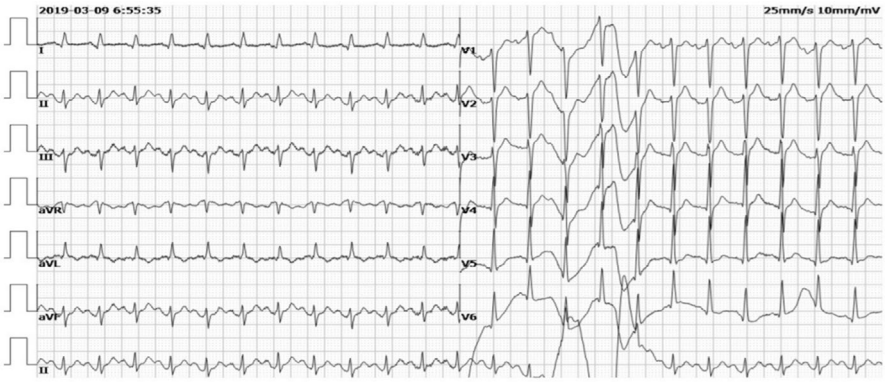

FIGURE 2 | Repeat 12-Lead ECG showing sinus tachycardia after the ventricular tachycardia had resolved.

TABLE 1 | Laboratory test results.

Biochemical tests	Complete blood count	hsTnT	NT-proBNP
CK 560.2 U/L	WBC 26.43×10^9/L	209.3 pg/mL	430.4 pg/mL
CK-MB 24.2 U/L	NEUT 23.85×10^9/L		
GLU 11.11 mmol/L	HGB 169 g/L		
Cr 190.5umol/L	PLT 340×10^9/L		
BUN 11.14 mmol/L			
K 3.68 mmol/L			
Na 123.01 mmol/L			
AST 78.2 U/L			
ALT 25.6 U/L			

hypokinesis, mild mitral regurgitation, and severe reduction of contractile function (LVEF 22%).

Based on initial clinical manifestations, myocarditis and pneumonia were suspected, and Methylprednisolone (200 mg q.d) and Moxifloxacin (400 mg i.v. q.d) were given along with other symptom-oriented medications such as furosemide, pantoprazole, and high-dose vitamin C injection. The treatments alleviated his symptoms, yet his blood pressure fluctuated dramatically on the third day after admission. The systolic blood pressure ranged from 90 to 240 mmHg, with the diastolic pressure reading between 50 and 140 mmHg, which gave us a hint that secondary causes of hypertension should be investigated. Biochemical tests of catecholamine, ultrasound, and CT scans of the adrenal gland were performed, along with other further examinations (see Table 2; Figures 3 and 4) that revealed a possible pheochromocytoma originating from the left adrenal gland. After recovering from the pheochromocytoma crisis, the patient underwent laparoscopic

adrenalectomy, and pathological examinations (see Figure 5) confirmed the diagnosis of pheochromocytoma. Upon the 4-month follow-up, the patient appeared in a good condition. His blood pressure was stable, the ECG showed no abnormalities (see Figure 6) and the echocardiography showed an intact cardiac structure and significantly improved cardiac function.

TABLE 2 | Plasma catecholamine and urine vanillylmandelic acid levels.

Subtype	Value	Reference value
Plasma adrenaline	1620 pg/mL	0–62 pg/mL
Plasma norepinephrine	2700 pg/mL	0–145 pg/mL
Plasma dopamine	4320 pg/mL	0–207 pg/mL
Urine vanillylmandelic acid	20.8 mg/24 h	0–13.6 mg/24 h

3 | Discussion

Pheochromocytoma is easily misdiagnosed for its variable and nonspecific clinical presentation, and was therefore called "the Great Mimic" or "the Great Masquerader". Only 40% of the patients may present with typical symptoms such as the classic triad of headache, palpitations and generalized sweating. The key to diagnosing pheochromocytoma is to first think of it, and an accurate diagnosis can only be made after careful exploration (Bednarek-Tupikowska et al. 2009). Over the years

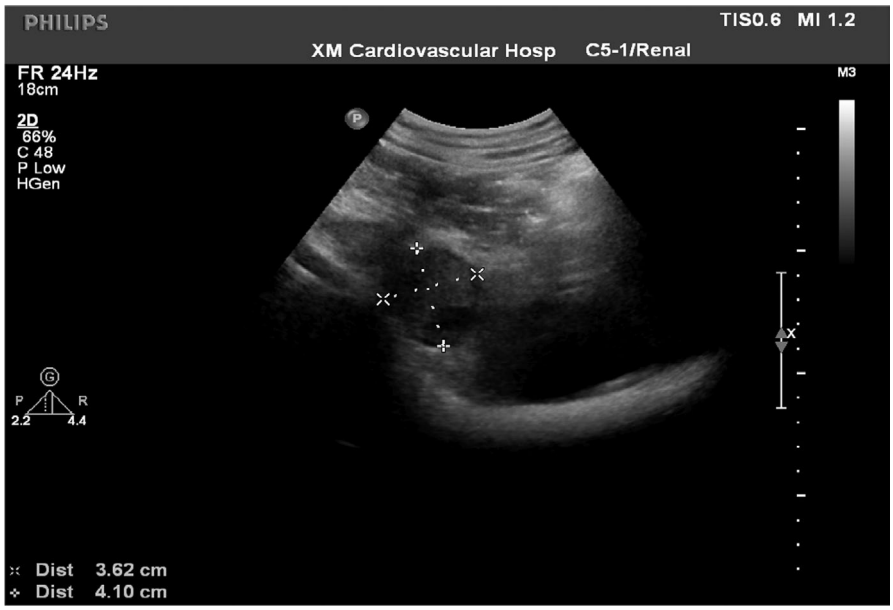

FIGURE 3 | Ultrasonography of the left adrenal gland showing a substantial hypoechoic mass with clear boundary. Size: 3.62 × 4.10 cm.

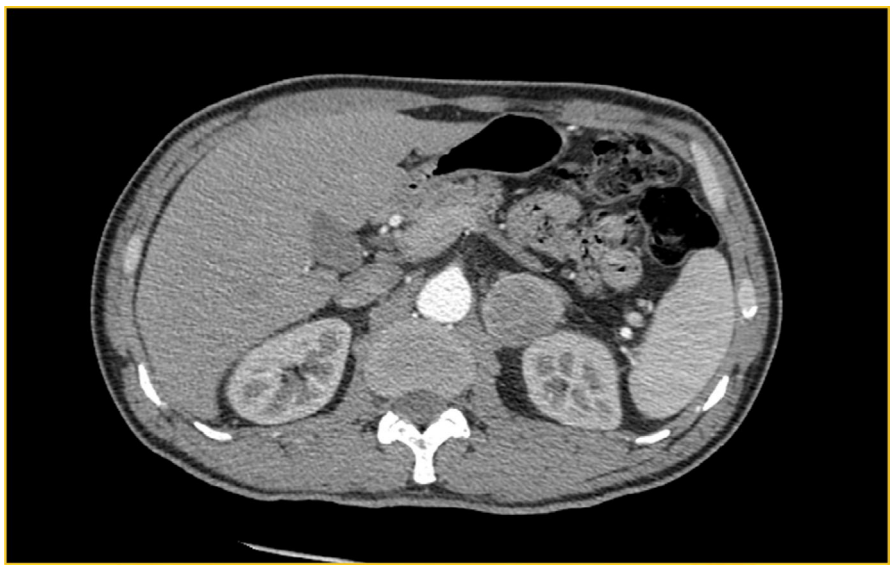

FIGURE 4 | Contrast-enhanced CT scan showing that the mass on the left adrenal gland can be partially enhanced.

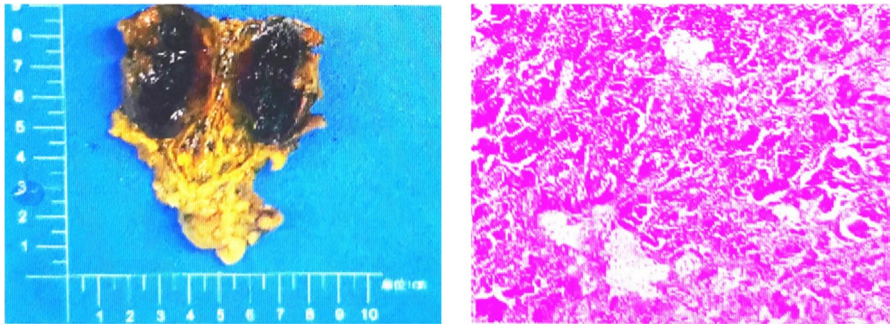

FIGURE 5 | Pathology confirmed pheochromocytoma without intravascular tumor thrombus and nerve invasion. The tumor cells showed acinar or organ-like arrangement with basophilic or dichromophilic cytoplasm and abundant blood sinuses can be seen between cell nests. Immunohistochemical results: CK-P (−), CgA (+), CD56 (+), S-100 (+), Ki-67 (+, positive rate about 1%).

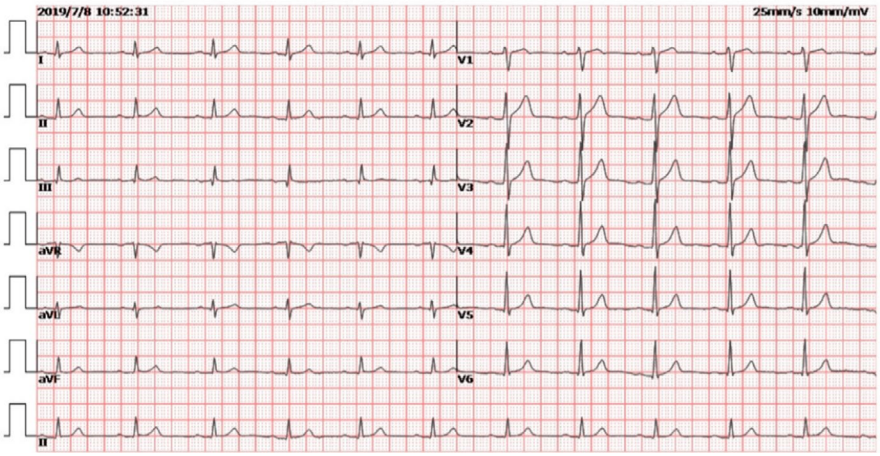

FIGURE 6 | ECG upon the 4-month follow-up showing no abnormalities.

pheochromocytoma-associated cardiovascular complications have been proposed and reported, which include "myocarditis," "myocardial infarction," "reversible cardiomyopathies," and "transient electrocardiographic repolarization changes" (S and Falhammar 2019). Monomorphic, bidirectional, or polymorphic VT have all been reported as a result of excessive catecholamine secretion (Nazari et al. 2020).

Our case illustrated a rare pheochromocytoma presentation with bidirectional ventricular tachycardia (see Figure 1). On the first ECG, a beat-to-beat alternation of the frontal wide QRS axis could be seen, especially conspicuous in lead V1. Meanwhile, a narrow QRS complex, which implies sinus capture, was also present. Bidirectional ventricular tachycardia is quite rare. The most common causes of bidirectional ventricular tachycardia are digitalis toxicity and catecholamines overproduction. Catecholaminergic polymorphic ventricular tachycardia is a rare inheritable disease which has a heterogeneous genetic basis (Velcea et al. 2017). Pheochromocytoma, which secretes catecholamine, may precipitate end organ damage in the heart. Catarina Quina-Rodrigues speculated that catecholamines might have impinged on calcium channels in the cardiomyocyte, causing calcium-overload in these cells, leading to after-depolarization that triggered the arrhythmia (Quina-Rodrigues et al. 2019). Increased myocardial oxygen consumption due to tachycardia, augmented inotropy, and heightened wall stress from elevated BP may also contribute to pro-arrhythmic ischemia (Nazari et al. 2020). However, the pathophysiologic process remains unclear. A "ping-pong" model was proposed to explain the mechanism behind bidirectional ventricular tachycardia. It is speculated that there exist two different heart rate thresholds for delayed after-depolarization in specific cardiac regions. These two myocardial areas provoke each other mutually and maintain the BVT in a "ping-pong" fashion (Jebberi et al. 2019). If the heart rate ceased to be in accordance with the threshold for the delayed after-depolarization in these specific regions, BDVT will not sustain, confirmed by the fact that BDVT terminated spontaneously in this case.

Also, there are several other lessons we should learn from this case. When this patient first presented with ventricular tachycardia, heart failure and elevated cardiac troponins after having undergone an acute respiratory tract infection 2 weeks ago, myocarditis was highly suspected, and he was given steroids. A pheochromocytoma crisis with severe blood pressure fluctuation occurred after systemic glucocorticoid administration. Several similar cases have been reported (Barrett et al. 2015; Eisenhofer et al. 2007). Glucocorticoids can increase the activity of phenylethanolamine-N-methyltransferase, which catalyzes the conversion of norepinephrine (noradrenaline) to epinephrine (adrenaline). In addition, steroids can enhance the "permissive effect" of catecholamines on vascular tone and blood pressure, causing the severe blood pressure fluctuation that may cause unexpected damage. Also, when dealing with

unexplained cardiac damage, heart failure or arrhythmia, we need to be alert in excluding pheochromocytoma.

4 | Conclusion

Pheochromocytoma can manifest itself in various symptoms and signs that mimic numerous other clinical conditions. BDVT may be an atypical clinical presentation of pheochromocytoma. Glucocorticoid administration could induce catecholaminergic crisis in patients with pheochromocytoma and should be carefully evaluated before being put into use if necessary. Resection of the chromaffin cell tumor may relieve the symptoms and reverse the impaired cardiac functions.

Author Contributions

Gui-yang Li and Man-Xin Lin recruited the subject, collected and analyzed the clinical data, and wrote the manuscript. Fa-Guang Zhou and Qiang Li helped collect clinical data, analyze the data, and reviewed the manuscript. All authors read and approved the final manuscript.

Ethics Statement

The study was approved by the Ethics Committee of Xiamen Cardiovascular Hospital, Xiamen University, and performed in line with the principles of the Declaration of Helsinki.

Consent

Informed consent has been obtained from the patient regarding the use of his clinical data for publication.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author, Li Qiang, upon reasonable request.

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