# A Case Report: Catecholamine-Secreting Adrenal Tumor-Inducing Cardiomyopathy

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**ABSTRACT:**

***Introduction:*** Catecholamine-secreting adrenal tumors (pheochromocytoma/paraganglioma) are rare neoplasms causing paroxysmal hypertension, metabolic disturbances, and cardiomyopathy. Diagnosis relies on biochemical tests (plasma-free metanephrines) and imaging (CT/MRI). Definitive treatment involves tumor resection with perioperative hemodynamic control.

***Case summary:*** A 47-year-old female with no prior hypertension presented with chest pain, hypertensive crisis (180/100 mmHg), hyperglycemia, and metabolic acidosis. Elevated norepinephrine (312.33 pg/mL) and abdominal MRI confirmed a right adrenal tumor (22×22×20 mm). A surgical resection was performed. Postoperatively, blood pressure normalized, and cardiac function improved (EF increased from 47% to 70% at the 2-month follow-up).

***Conclusion:*** Catecholamine-secreting tumors may induce severe cardiomyopathy even without a hypertension history. Early diagnosis and prompt surgery are critical for cardiac recovery and preventing complications.

*Keywords: Adrenal tumor, catecholamine, cardiomyopathy, pheochromocytoma.*

## I. Introduction

Catecholamine-secreting adrenal tumors (pheochromocytomas and paragangliomas) are rare neoplasms that arise from chromaffin cells of the adrenal medulla or extra-adrenal sympathetic ganglia. These tumors are capable of excessive catecholamine secretion including epinephrine, norepinephrine, and dopamine – leading to significant cardiovascular and metabolic disturbance [1, 4].

The prevalence of catecholamine-secreting adrenal tumors among hypertensive patients ranges from 0.2% to 0.6% [1]. Due to their often nonspecific clinical presentation, these tumors are frequently diagnosed late or overlooked. Common manifestations include paroxysmal or sustained hypertension, tachycardia, palpitations, headache, excessive sweating, anxiety, and glucose metabolism disorders [1, 5]. In certain cases, the tumor may lead to acute or chronic myocardial injury, including catecholamine-induced cardiomyopathy, heart failure, arrhythmias, and even sudden cardiac death.

Diagnosis of catecholamine-secreting tumors relies on both biochemical testing and imaging studies. Biochemical assessment

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involves the measurement of plasma free metanephrines in the supine position or 24-hour urinary metanephrines, both of which exhibit high sensitivity and specificity [1,4,6–9]. Imaging modalities such as computed tomography (CT) or magnetic resonance imaging (MRI) of the abdomen are commonly employed to localize the tumor. In cases where metastatic or extra-adrenal disease is suspected, positron emission tomography-computed tomography (PET-CT) using radiotracers such as 18F-FDOPA, 18F-FDA, 18F-FDG, or MIBG can provide additional diagnostic value [1,4,10–14].

The standard treatment for catecholamine-secreting adrenal tumors is surgical adrenalectomy, performed after appropriate preoperative management of blood pressure and heart rate using alpha-adrenergic blockers (e.g., phenoxybenzamine, doxazosin) and beta-blockers when indicated [1,4,15,16]. Timely intervention is essential in reducing the risk of cardiovascular complications such as heart failure, myocardial infarction, and stroke [4]. Additionally, long-term postoperative follow-up is necessary to detect tumor recurrence or metastasis, particularly in patients with germline mutations associated with pheochromocytoma or paraganglioma [3]. In this case report, we describe a patient with a catecholamine-secreting adrenal tumor presenting with atypical clinical features, highlighting the importance of considering this diagnosis in complex clinical scenarios. We also discuss diagnostic challenges, management strategies, and key lessons for clinical practice.

**2. Case report**

The patient was initially admitted to the hospital in January 2023, with a subsequent hospitalization in July 2023. We reported the case of a 47-year-old female patient, L.T.N, with no prior history of hypertension, presented to the emergency department with complaints of acute chest pain. On admission, her blood pressure was elevated at 180/100 mmHg. Prior to the first admission, the patient had no documented history of chronic illness. Six months before the second hospitalization, the patient experienced an acute episode requiring admission, during which elevated cardiac biomarkers were recorded: high-sensitivity troponin T (hs-TnT) level was 856.4 ng/L, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) level was 1201 pg/mL, indicating acute heart failure. Coronary angiography revealed normal coronary arteries. During hospitalization, the patient experienced an episode of supraventricular tachycardia. Transthoracic echocardiography showed a globally and relatively uniform hypokinesis of the left ventricular walls. Left ventricular systolic function, measured by the Simpson method, was 40%. The patient was treated for heart failure according to current clinical guidelines. After six days of treatment, the patient's symptoms stabilized; cardiac biomarkers and left ventricular systolic function normalized. At the time of discharge, despite low blood pressure, the patient was prescribed Spironolactone 25 mg and Bisoprolol 2.5 mg. The patient was followed up monthly at Hanoi Heart Hospital on an outpatient basis for five months.

In the sixth month following the initial hospitalization, the patient developed episodes of chest pain accompanied by blood pressure readings of 180/100 mmHg, shortness of breath, significant fatigue, nausea, and profuse

sweating. A 12-lead electrocardiogram (Figure 2.1) revealed sinus rhythm at 90 beats per minute, intermediate axis, ST-segment elevation of 1 mm in leads aVR and V1, and ST-segment depression greater than 1 mm in both inferior and precordial leads.

Transthoracic echocardiography revealed relatively uniform hypokinesis of the left ventricular walls, with no dilation of the left ventricular chamber. Left ventricular systolic function, assessed using the biplane Simpson method, was 47%. Severe mitral regurgitation (grade 3/4) of type I, moderate tricuspid regurgitation (grade 2/4), and significant pulmonary hypertension were observed, with an estimated systolic pulmonary artery pressure of 57 mmHg. Relevant laboratory tests were performed, as summarized in Table 2.1. Endocrine evaluations were performed, with results shown in table 2.2.

It is noteworthy that these tests were conducted while the patient was taking metoclopramide, spironolactone, and metoprolol. A repeat metanephrine test was performed after a two-week discontinuation of these medications, revealing a 24-hour urinary metanephrine level of 2,332.4 mcg. The patient was subsequently diagnosed with a catecholamine-secreting adrenal tumor (pheochromocytoma), which was identified as the cause of the myocardial injury. Surgical resection of the adrenal tumor was performed, followed by postoperative management of blood glucose and blood pressure (Figure 2.3).

Two months after surgery, the patient was re-evaluated at Hanoi Heart Hospital. At follow-up, the patient reported no chest pain, showed no signs of acidosis, and maintained normal blood pressure without the need for antihypertensive medication. Cardiac function had significantly improved, with an ejection fraction (EF) reaching 70%. At present, ten months post-discharge, the patient has not experienced any episodes of acute heart failure or hospitalizations due to myocardial injury.

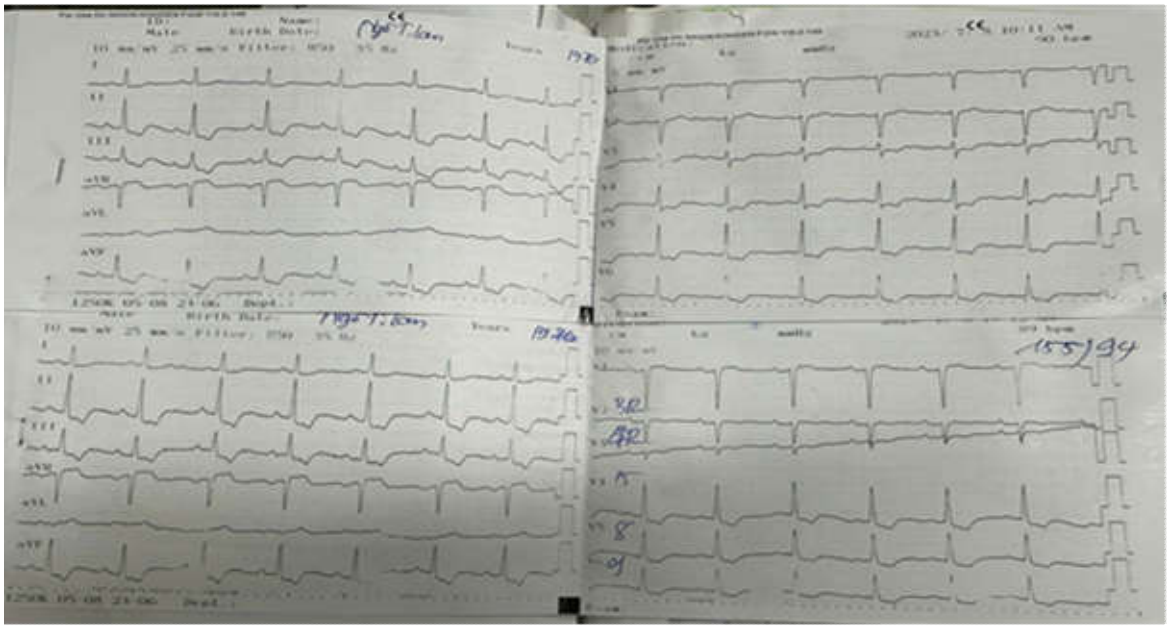


**Figure 2.1. Electrocardiogram at hospital admission**

**Table 2.1: Laboratory Result at hospital admission**

| | | Value | Reference range |
|---|---|---|---|
| Random plasma glucose (mmol/L) | | **24** | 4.4 – 7.2 |
| HbA1C (%) | | 5.1 | < 6.4 |
| Sodium (mmol/L) | | 135 | 135 – 145 |
| Potassium (mmol/L) | | 4.2 | 3.5 – 5.1 |
| pH | | **7.30** | 7.35 – 7.45 |
| HCO3 (mmol/L) | | **10** | 23 – 29 |
| PaCO2 (mmHg) | | **21** | 35 – 45 |
| Lactate (mmol/L) | | **14** | 0.4 – 2.2 |
| Anion gap | | **28** | |
| Leukocytes (G/L) | | **19** | 4.0 – 10.0 |
| % Neutrophils | | **88** | 50 – 70 |
| Pro-Calcitonin (ng/mL) | | **3.4** | < 0.5 |
| Troponin Ths (ng/L) | 1st sample | **198** | < 14 |
| | 2nd sample (1h later) | **423** | < 14 |

**Table 2.2: Catecholamine testing**

| | Value | Reference range |
|---|---|---|
| **Renin (mcUI/mL)** | **79.9** | |
| **Aldosterone (ng/dl)** | **36.2** | |
| Adrenalin mcg/24h (mcg/day) | 16.66 | 0 -20 |
| Dopamine (mcg/day) | 211.82 | 0 -600 |
| Noradrenaline (mcg/day) | 35.35 | 0 -90 |
| Metanephrin (mcg/day) | **2332.4** | 52-341 |
| Normetanephrin (mcg/day) | **641.3** | 88 - 444 |

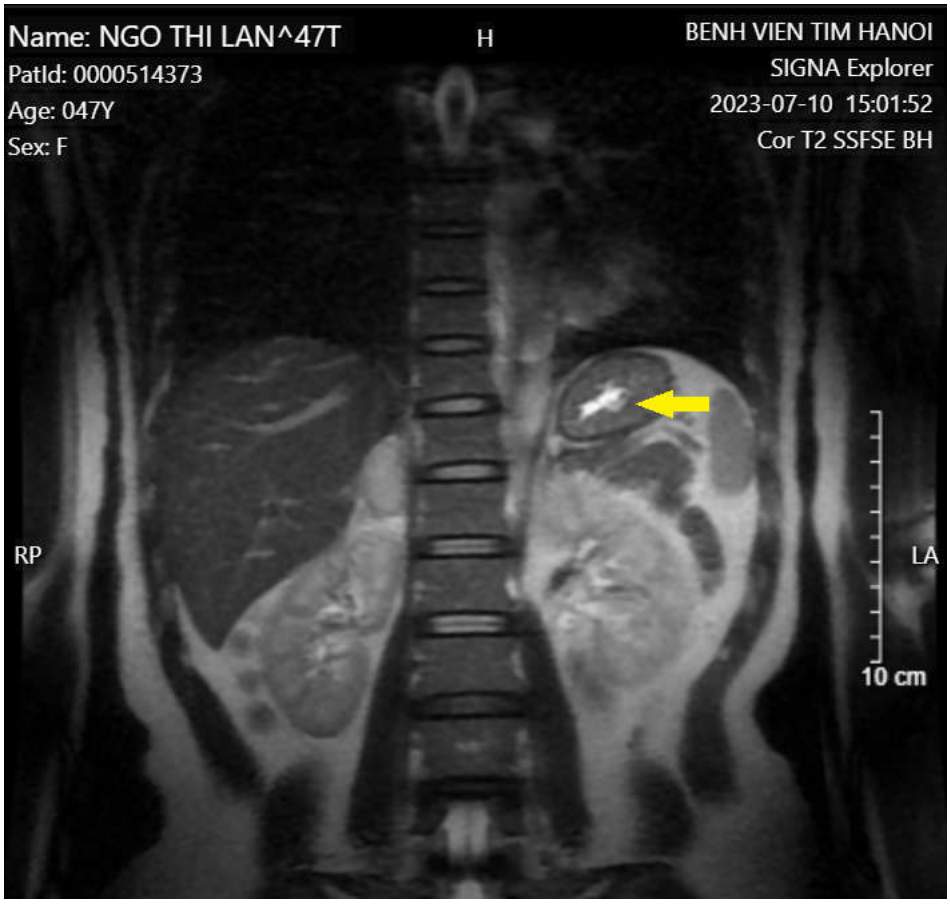
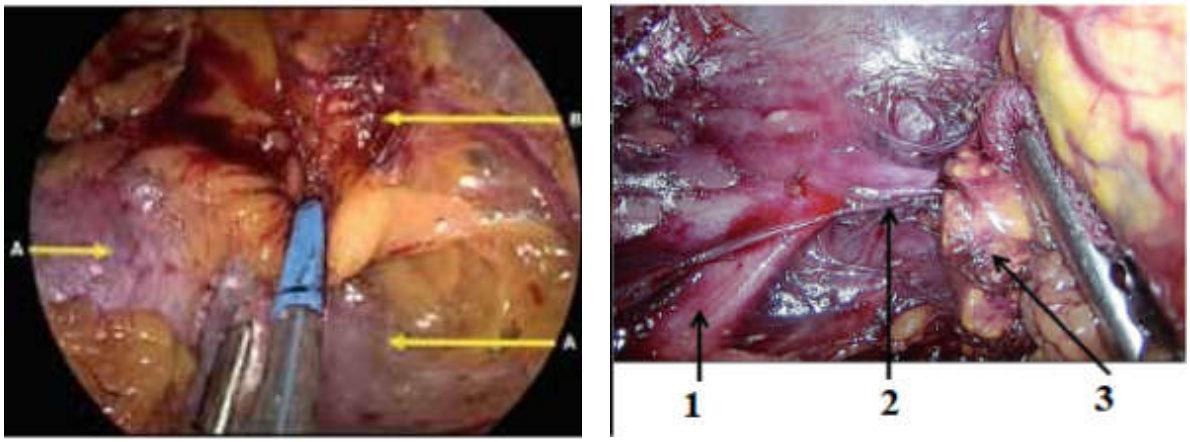



**Figure 2.2. Abdominal MRI scan with T2-weighted sequence. (the yellow arrow indicates an adrenal tumor)**




(a) A: Kidney, B: Adrenal Tumor

(b) 1,2,3: Inferior vena cava, Adrenal vein and Adrenal tumor

**Figure 2.3. Intraoperative image during adrenal tumor resection**.

## III. Discussion

Catecholamine-secreting adrenal tumors (pheochromocytomas and paragangliomas) are rare causes of secondary hypertension but can lead to severe cardiovascular complications if not diagnosed and treated promptly [2]. Most reports suggest that cardiomyopathy, severe coronary vasospasm, myocarditis, and tachyarrhythmias result from excessive catecholamine release, leading to elevated cardiac troponin levels and reduced left ventricular systolic function [17,18]. In this case, the patient had no prior history of hypertension or diabetes mellitus but was admitted with paroxysmal tachycardia and hypertensive crisis, accompanied by significant myocardial injury, as evidenced by markedly elevated troponin T levels and left ventricular dysfunction. This underscores the importance of early recognition of adrenal tumors in patients Adrenal tumors can result in various forms of cardiomyopathy, including Takotsubo cardiomyopathy and catecholamine-induced cardiomyopathy [19,20]. In this case, coronary angiography revealed no obstruction, and echocardiography demonstrated relatively uniform hypokinesis of the left ventricular walls without apical ballooning, consistent with catecholamine-induced cardiomyopathy. These two types of cardiomyopathy may share a common pathophysiological mechanism involving excessive catecholamine secretion, which leads to microvascular dysfunction, epicardial vasospasm, direct myocardial toxicity, severe vasoconstriction, ischemia, and oxidative stress-induced myocardial injury [9]. Acute pulmonary edema is more frequently observed in catecholamine-induced cardiomyopathy. Furthermore, patients with catecholamine cardiomyopathy typically present with higher left ventricular mass index, increased relative wall thickness, and more pronounced hypertension compared to those with Takotsubo cardiomyopathy [21].

Diagnosis of adrenal tumors in this case was particularly challenging due to the nonspecific initial clinical presentation. Suspicious features included paroxysmal hypertension, glucose metabolism disorders, metabolic acidosis, and elevated blood lactate levels. The diagnosis was confirmed by 24-hour urinary metanephrine measurement after drug withdrawal and abdominal MRI, which identified an adrenal mass, guiding appropriate therapeutic intervention.

Various treatment strategies are available to manage the effects of catecholamine excess, including pharmacologic agents such as alpha-blockers, calcium channel blockers, beta-blockers, and tyrosine hydroxylase inhibitors, as well as surgical resection, radiofrequency ablation, and radiotherapy [22]. In patients with tachyarrhythmia, heart failure, or angina, beta-blockers such as propranolol, atenolol, or metoprolol are indicated. However, beta-blockers should never be administered without prior effective alpha-blockade, as unopposed beta-adrenergic receptor inhibition may precipitate hypertensive crises and even cardiac arrest [23,24]. This mechanism likely contributed to the acute myocardial injury in this patient, who was receiving bisoprolol without alpha-blockade prior to the second hospitalization.

Surgical resection of the tumor is the definitive treatment. In this case, the patient

recovered well following surgery, with normalization of blood pressure without the need for antihypertensive medication and a significant improvement in cardiac function – ejection fraction increased from 47% to 70% within two months. This demonstrates that catecholamine-induced myocardial injury can be reversible if the underlying cause is promptly and effectively treated.

## IV. Conclusion

This case highlights the importance of considering catecholamine-secreting adrenal tumors, such as pheochromocytoma or paraganglioma, in the differential diagnosis of patients presenting with hypertensive crises, metabolic disturbances, or unexplained cardiac symptoms—even in the absence of a prior history of hypertension. Atypical presentations can lead to delays in diagnosis and increase the risk of life-threatening complications. Early recognition, appropriate biochemical and imaging evaluation, and timely surgical intervention are critical to improving clinical outcomes. Clinicians should maintain a high index of suspicion for this rare but potentially fatal condition, especially in complex or unexplained clinical scenarios.

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