

Pheochromocytoma-Induced Segmental Myocardial Dysfunction Mimicking an Acute Myocardial Infarction in a Patient with Normal Coronary Arteries

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We report a case of pheochromocytoma-induced segmental myocardial dysfunction and electrocardiographic abnormalities mimicking an acute anterior myocardial infarction, probably due to coronary spasm. Coronary angiography showed normal coronaries, and the electrocardiographic and echocardiographic changes resolved completely after therapy with an alpha-adrenergic blocker and tumor removal. Our case illustrates the importance of maintaining a high index of suspicion in patients presenting with an unexpected myocardial event and a hypertensive crisis.

Pheochromocytoma is a catecholamine-secreting tumor that arises from chromaffin tissue of the sympathetic nervous system. The usual manifestations of this tumor include palpitations, diaphoresis, headache, and paroxysmal hypertension. In addition to the classic symptoms, pheochromocytomas have been rarely associated with acute myocardial infarction and other cardiovascular complications¹. We report the case of a patient with an adrenal pheochromocytoma and normal coronary arteries who presented with electrocardiographic and echocardiographic findings consistent with an acute anterolateral myocardial infarction (MI), with complete reversal of these abnormalities after alpha-adrenergic treatment.

Case Report

A 46-year-old female with history of hypertension was hospitalized with nausea, vomiting, and diaphoresis for 2 days. On physical examination, her initial blood pres-

sure was 230/130 mmHg; heart rate, 132 bpm; and her skin was mottled. The rest of the examination was unremarkable. The electrocardiogram (fig. 1) showed sinus tachycardia with ST segment elevation and deep, symmetric T wave inversion in leads V2 thru V6. The T waves were also inverted in leads I, aVL, II, and aVF. The QT interval was markedly prolonged and Q waves were present in leads II, III, aVF.

She was taken to the cardiac catheterization laboratory where a coronary angiogram showed no significant coronary disease. The left ventriculogram (fig. 2) showed severe anterolateral and apical hypokinesis. During the procedure, the patient required mechanical ventilation, and the arterial blood pressure became very labile, going from as high as 320/240 mmHg to as low as 70/30 mmHg, warranting the use of vasodilators and, at times, vasopressors. The diagnosis of pheochromocytoma was suspected, and therapy with an alpha-adrenergic blocker was started, which slowly controlled the blood pressure.

Laboratory data included 24-hour urinary vanillylmandelic acid, metanephrine, epinephrine, and norepinephrine levels that were, respectively, 313 mg (normal, 2-10), 76 (normal, 0.3-0.9), 12339 ng (normal, 0-16), and 28316 ng (normal, 11-86). The blood urea nitrogen was 26 mg/dL and creatinine was 2.0 mg/dL. The peak CPK level was 951 IU/L with a normal MB fraction. A computed tomography revealed a rounded mass in the left adrenal gland.

Blood pressure returned to normal within 5 days of therapy with an intravenous alpha-adrenergic blocker. The patient was extubated, and renal function normalized. Serial echocardiograms were performed on days 1 and 10 of hospitalization. The first confirmed the findings on the left ventriculogram of severe hypokinesis of the anterior and apical walls, and the second demonstrated complete resolution of those abnormalities. After 10 days of therapy, the patient was sent to surgery where an 11.2 x 11.8 x 7.2 cm pheochromocytoma was removed uneventfully. A follow-up electrocardiogram obtained 3 months after the first one showed complete resolution of the ST segment and T wave changes (fig. 3).

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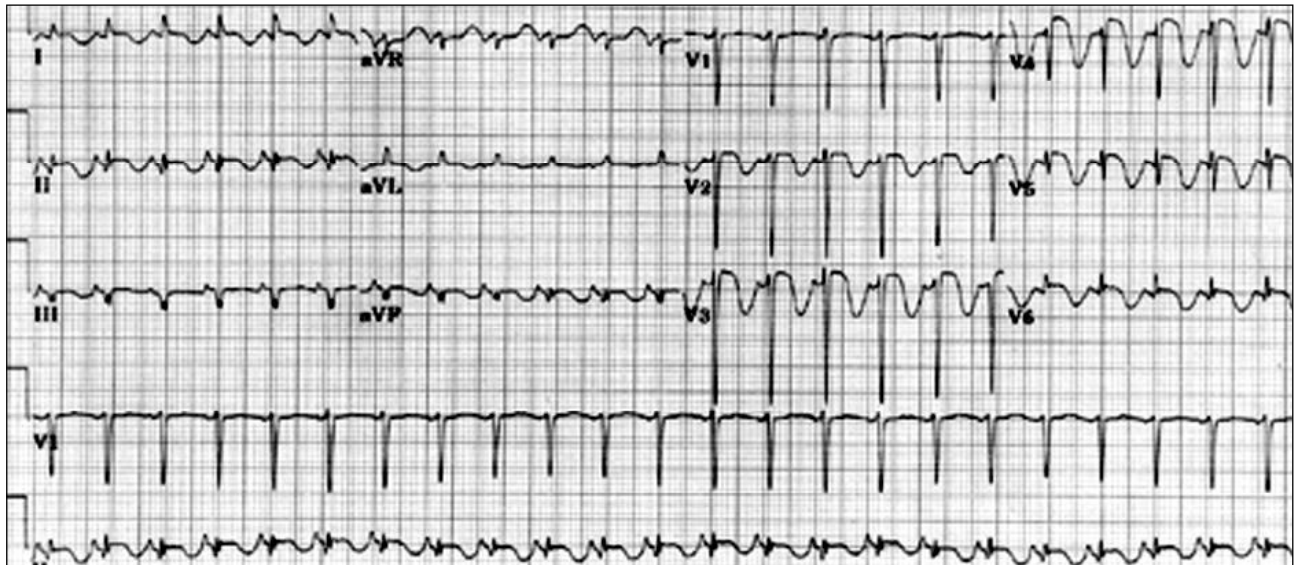


Fig. 1 - Electrocardiogram on admission showing ST segment elevation from V2-V6 and T wave inversion in V2-V6, I, II, aVL, and aVF. Note the marked prolongation in the QT interval.

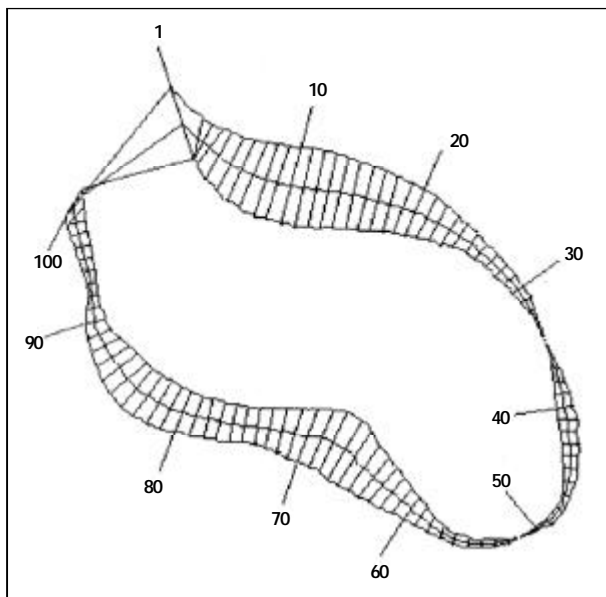


Fig. 2 - Diagram of the left ventriculogram in the right anterior oblique projection. The inner line represents the left ventricle at end-systole and the outer line at end-diastole. Severe anterolateral and apical hypokinesis is noted.

Before discharge, the patient admitted that she had been having frequent bouts of nausea, vomiting, and severe headaches since she was diagnosed with hypertension 5 years before. She continues to do well 1 year after discharge, with good blood pressure control and no recurrence of symptoms.

Discussion

Pheochromocytoma-induced myocardial disease may take the form of ventricular hypertrophy due to long-standing hypertension², dilated cardiomyopathy because of persistent and prolonged exposure to high levels of cate-

cholamines³, or, rarely, it may mimic an acute myocardial infarction⁴⁻⁹.

We report the case of a rare association between pheochromocytoma and reversible myocardial dysfunction in a patient with normal coronaries. Only a few cases of a pheochromocytoma crisis causing or mimicking an acute myocardial infarction have been reported in the literature. Their clinical presentations have varied significantly, from a silent myocardial infarction in one of the first cases reported in the literature⁴, to frank cardiogenic shock⁵. Striking electrocardiographic changes suggesting myocardial ischemia are often present in patients with pheochromocytoma and include marked prolongation of the QT interval, deep and symmetric T wave inversion, and ST segment changes¹⁰. Although these are very suggestive of myocardial ischemia, they have been described in other conditions such as subarachnoid hemorrhage and the use of certain drugs¹⁰. None of these situations is likely in the present case. Wall motion abnormalities have not been well characterized, but both segmental⁶ and global⁷ myocardial dysfunction have been reported. Although they are commonly associated, the ischemic ECG changes may not be accompanied by wall motion abnormalities⁸. Additionally, despite striking ECG changes and myocardial dysfunction, serum markers of myocyte necrosis may or may not be elevated⁷. In our case, the significant elevation in the CPK levels is probably of skeletal muscle origin and not myocardial injury because the MB fraction was normal.

The pathophysiology of myocardial dysfunction associated with pheochromocytoma has been linked to either a direct toxic effect induced by catecholamines³, or myocardial stunning caused by coronary spasm⁹. These changes along with the ECG abnormalities are commonly reversible after treatment with alpha-adrenergic blockers and tumor removal^{5,7}. Although beta-blockers are certainly recommended in patients with an acute MI, they can worsen coronary spasm in cases of pheochromocytomas as a result of unop-

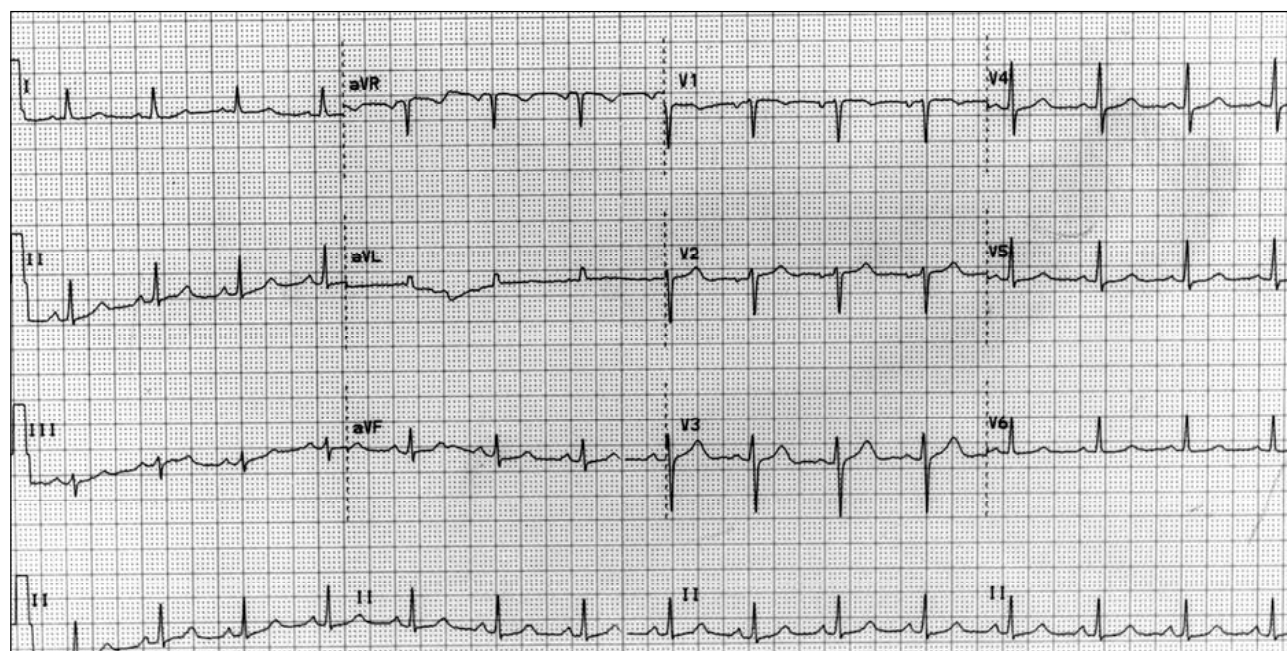


Fig. 3 - Electrocardiogram 3 months after presentation, showing complete reversal of the previous abnormalities.

posed alpha-adrenergic stimulation, and are, therefore, contraindicated in this circumstance.

Our case illustrates an uncommon presentation of a pheochromocytoma crisis with ECG and wall motion abnormalities mimicking myocardial infarction. The lack of elevation of myocardial necrosis markers and complete reversal of segmental myocardial dysfunction suggest stunning due to coronary spasm as the underlying mechanism. Our report also reinforces the importance of maintaining a high index of

suspicion in patients who present with an unexpected myocardial infarction associated with a hypertensive crisis. The relevance of a thorough history cannot be overemphasized, because in our case and in a number of cases reported in the literature, symptoms suggestive of pheochromocytoma are found to have been present for years prior to the acute event, and are frequently missed on presentation¹. These patients ought to be screened for pheochromocytoma, as early treatment may prevent serious morbidity and mortality.

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