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## Myocardial stunning in a patient with occult pheochromocytoma

### Abstract

A young patient presented with electrocardiographic and echocardiographic signs of acute coronary syndrome, which turned out to be myocardial stunning as the consequence of an adrenergic crisis initiated by an ECG stress test. Heart failure improved rapidly and the patient was diagnosed with an occult catecholamine-secreting pheochromocytoma.

The physiopathology of myocardial stunning in catecholamine crisis is discussed.

*Key words: myocardial stunning; pheochromocytoma; exercise stress test; ventricular tachycardia*

### Introduction

Pheochromocytoma is known to produce various cardiovascular complications including hypertension, arrhythmias, heart failure, myocardial infarction and catecholamine-induced cardiomyopathy [1].

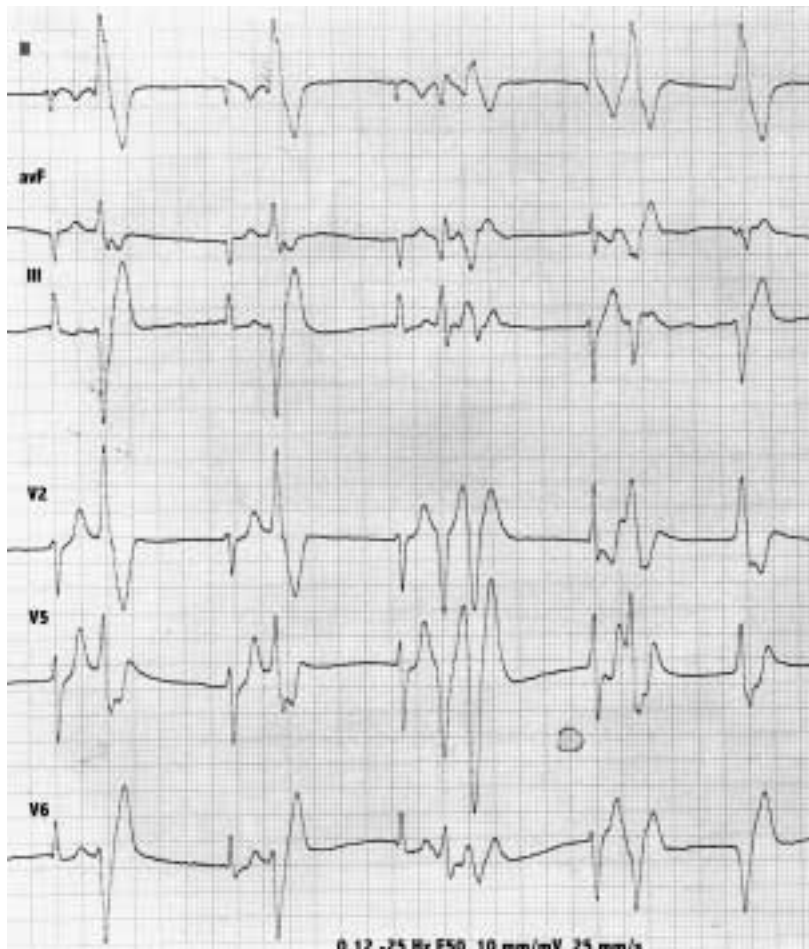
We present an unusual case of myocardial stunning secondary to adrenergic crisis initiated by ECG stress test in a patient with occult pheochromocytoma.

### Case report

A 48-year-old man was referred for a stress test to rule out a coronary heart disease. He gave a history of intermittent palpitations associated with chest pain, pallor, paresthesias and dizziness. The bouts were sudden in onset, not related to any obvious causative factors, nor related to exercise. Each episode lasted about fifteen minutes and occurred usually once or twice a week. Recently, the attacks became more frequent, occurring several times a day. The patient took no medications, no illicit drugs and, otherwise appeared to be in good health. He smoked two packs of cigarettes a day for twenty years but had no other risk factors for coronary artery disease.

The physical examination was unremarkable except for an S3 with a protomesosystolic murmur extending to the left axilla. The blood pressure measured in the left and right arms

**Figure 1**  
ECG seven minutes after  
completion of exercise  
stress test.

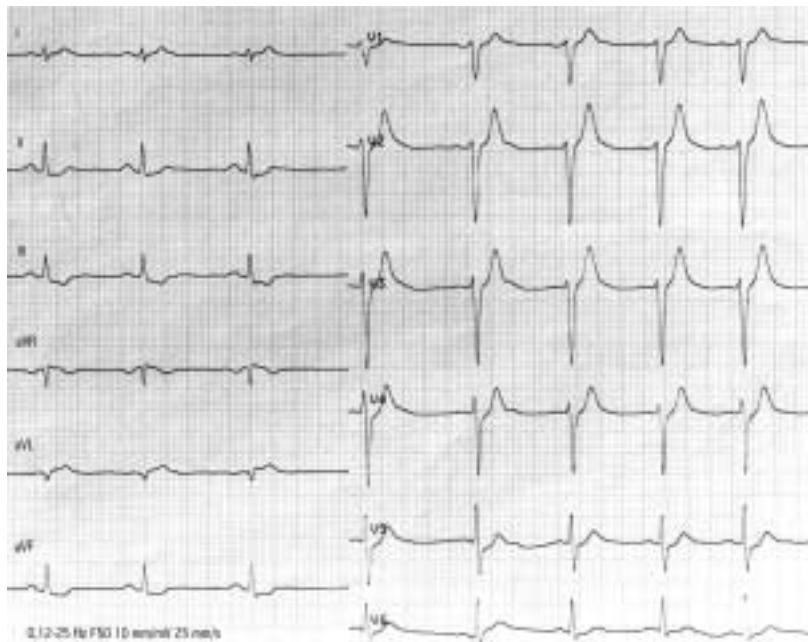


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**Figure 2**  
ECG twenty minutes after completion of exercise stress test (ventricular tachycardia).



**Figure 3**  
ECG 18 hours after completion of exercise stress test.



was 134/94 and 156/100 mm Hg respectively. The chest x-ray, the ECG and the echocardiogram were normal. On the exercise stress test, the patient produced an effort of 175 watts without symptoms or ECG changes. However, seven minutes into recovery, he began to complain of dizziness, abdominal pain and palpitations. His systolic blood pressure rose to 230 mm Hg, with couplets and triplets of polymorphic ventricular extrasystoles on the ECG (fig. 1). Twenty minutes after completion of the stress test, the patient went into ventricular

**Table 1**  
Blood levels of cardiac enzymes.

	day 1	day 2	day 3	day 4
CK (U/L) (norm <300)	181	366	460	450
CK-MB (%)		25 (7%)	33 (7%)	33 (7%)
Troponin I (ng/ml) (norm <0.4)	0.077	0.914	1.519	0.743

**Table 2**  
24-hour urinary levels of catecholamines.

	day 1	day 4
Epinephrine (<110 nmol/24 h)	6415	298
Norepinephrine (<472 nmol/24 h)	1355	129
Dopamine (<5000 nmol/24 h)	3505	1900
Normetanephrine (<3.3 µmol/24 h)	7.8	3.1
Metanephrine (<1–2 µmol/24 h)	37.4	11.2
Vanilmandelic acid (<35 µmol/24 h)	–	35

tachycardia (VT) (fig. 2). During the VT, which resolved spontaneously after three minutes, the patient maintained a blood pressure over 200 mm Hg. A subsequent ECG revealed infero-lateral ST depression (fig. 3). The patient was transferred to the CCU with a diagnosis of unstable angina. The echocardiogram obtained soon after arrival in the CCU showed diffuse hypokinesis in the infero-postero-lateral walls and basal septal segment. The ventricular ejection fraction was markedly decreased (40%) with signs of left ventricular dilation.

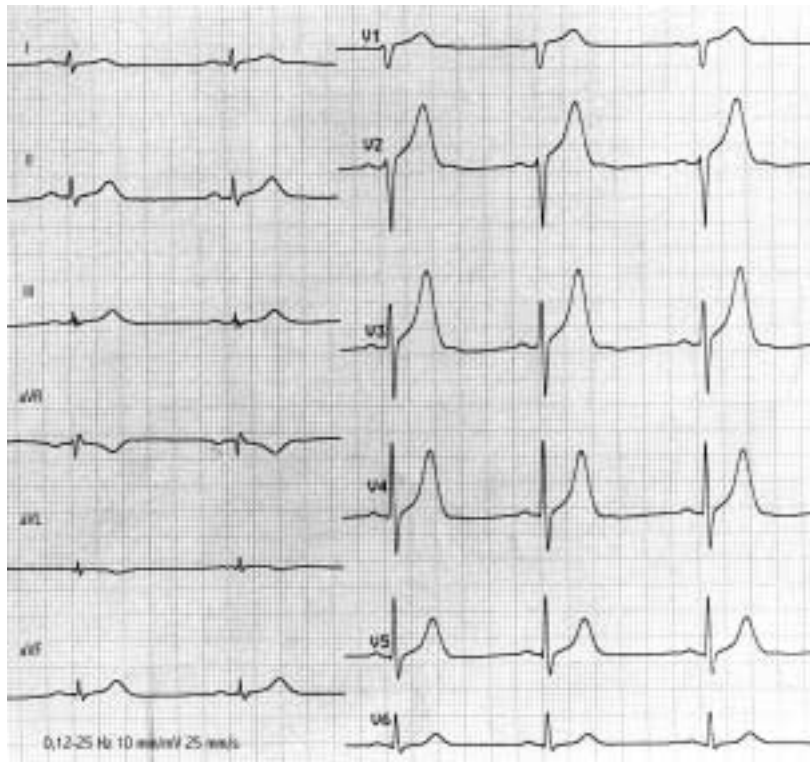
The patient was treated with oxygen, heparin, aspirin and beta-blockers. Cardiac enzymes rose slightly (table 1) but no lesions were detected in the coronary angiogram.

A second echocardiographic exam performed and read by the same physician 72 hours after admission to the CCU revealed a vigorous contractile performance with normal ejection fraction. The ECG abnormalities had also completely resolved (fig. 4). Late potentials were detected in a signal averaging-ECG. An intracardiac electrophysiologic study, however, found no abnormalities of either sinus node function or atrioventricular conduction. Moreover, there were no inducible ventricular arrhythmias.

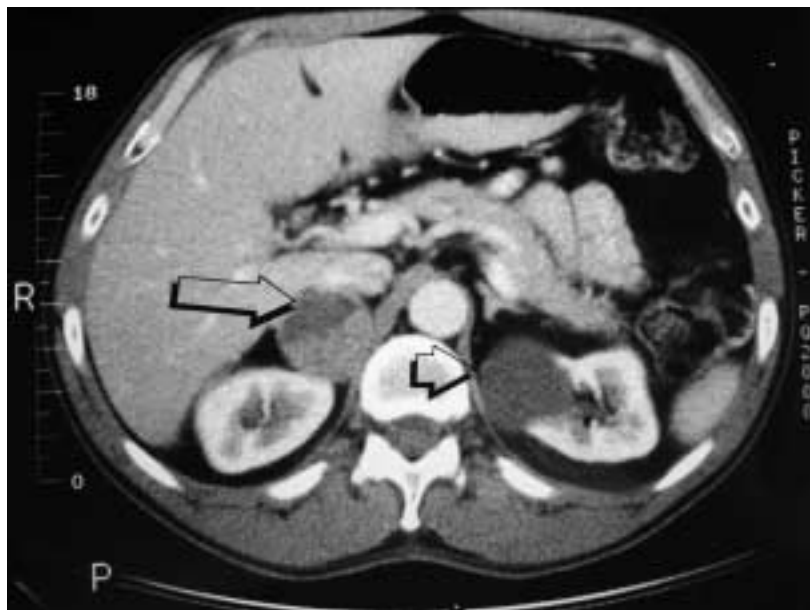
A 24 hour dosage of urinary catecholamines yielded markedly elevated adrenaline and vanillylmandelic acid levels whereas noradrenalin was slightly elevated (table 2). The abdominal echography was silent but a 43 mm nodular tumor above the right kidney in contact with both the inferior vena cava and

**Figure 4**

ECG three days after completion of the exercise stress test.

**Figure 5**

CT-scan of the abdomen (long arrow: renal cyst; short arrow: pheochromocytoma).



the liver was visualised on the CT scan. The tumor had a heterogeneous parenchyma, no calcifications and showed marked peripheral enhancement after contrast injection (fig. 5). The diagnosis of left adrenal pheochromocytoma was confirmed with I 123 MIBG planar and SPECT scintigraphy. No other ectopic or secondary localizations were found. Associated MEN syndrome, von Recklinghausen

and von Hippel-Lindau diseases were ruled out by family history, physical examination and laboratory tests. The patient was given labetalol for 3 weeks and scheduled for elective laparoscopic left adrenalectomy. General anesthesia was induced with etomidate, fentanyl and rocuronium, and maintained with desflurane, fentanyl and rocuronium. The intraoperative course was stormy with hypertensive episodes that were difficult to control in spite of high inspirational fractions of volatile agent and use of sodium nitroprusside, phentolamine and nitroglycerine. After the left adrenal vein was clamped, the patient became haemodynamically stable. In particular, there was no need for a vasopressor infusion. The postoperative course was uneventful and the patient was discharged home three days after surgery.

## Discussion

Pheochromocytoma is a rare but well-known cause of hypertension, which occurs in about 90% of the cases. Cardiac complications are common and protean, presenting with arrhythmias, myocardial ischemia, infarcts, as well as dilated or hypertrophic cardiomyopathies.

Our case suggests that the exercise stress test may have stimulated the liberation of catecholamines from the adrenal medulla thereby increasing myocardial irritability, as evidenced by the ventricular extrasystoles and ventricular tachycardia. This viewpoint is in accord with Telenius-Berg who demonstrated that exercise stress test raises plasma catecholamines, especially epinephrine, in patients with pheochromocytoma associated with multiple endocrine neoplasias Type II [2]. In Telenius-Berg's study, however, the patients did not experience any disruption of cardiac electrical stability. This observation is in contrast with the high number of reported cases of arrhythmias occurring in the context of pheochromocytoma [3–6].

In pheochromocytoma, supraventricular tachycardia occurs more often than ventricular arrhythmia [3]. Nevertheless, ventricular tachycardia [4], including "torsades de pointes" [5], or more rarely ventricular fibrillation [6] has also been described. In our case, we observed silent ischemic electrocardiographic changes together with a marked and diffuse left ventricular hypokinesia. Both ECG changes and the contractile dysfunction completely resolved within 72 hours. ST segment depression, low amplitude or inverted T waves

are suggestive of ischemic insults and are often seen in pheochromocytoma [7]. Prolonged QT intervals are also frequently observed in these patients [8, 9]. Usually these electrocardiographic abnormalities resolve steadily within five to ten days.

Very few cases of cardiac hypokinesia in pheochromocytoma have been reported in the literature [3, 8, 10]. As a rule, the hypokinetic ventricular segments were rather small in size [10–13] and tended to show a slow recovery over weeks or even months [11, 14]. Rarely, as in the present case, did the echocardiogram reveal large area of myocardial contractile dysfunction that fully recovered within just a few days [3, 8, 15].

In our patient, the short duration of a diffuse hypokinesia suggests myocardial stunning as a result of extensive coronary artery vasoconstriction. The hypothesis of myocardial stunning in patients with pheochromocytoma has been considered previously [11–13].

The underlying mechanism is poorly understood. The conjunction of several factors such as the alpha- and beta-mediated stimulating effects of catecholamines could induce marked cardiac ischemia and subsequent stunning. Severe but brief ischemic insults followed by restoration of normal or near normal coronary flow are essential prerequisites for myocardial stunning. It appears that our patient underwent each of these typical changes.

The early infero-lateral ischemic abnormalities on the ECG, the near global diffuse hypokinesia and the finding of normal coronary arteries are consistent with a transient alpha adrenoceptor stimulation – mediated diffuse coronary vasospasm. This interpretation is supported by other clinical reports that have implicated both coronary [1, 3, 16, 17] and peripheral vasospasms [1] caused by pheochromocytoma.

Moreover, in experimental studies, there is evidence that pheochromocytoma cardiomyopathy can be caused by an alpha-mediated vasoconstriction since it can be prevented by alpha adrenoceptor blocking drugs [18]. Marked coronary flow reduction and large increases in afterload during hypertensive crises, together with increased inotropy and heart rate due to concomitant beta stimulation will greatly augment myocardial oxygen demand and consumption and promote ischemic changes. Thus, in pheochromocytoma, episodes of myocardial stunning may well constitute a series of initial injuries which, over time, could steadily evolve into myocarditis [19] and a cardiomyopathy with possible

dilation, especially if myocardial infarctions complicate its course [9].

A special comment is warranted with regard to the preparation of this patient for surgery and anesthesia. The patient received labetalol alone, a beta-blocker with little alpha blocking properties. As indicated by the anesthesiologist, the alpha blockade proved to be insufficient during surgery. Clearly, labetalol alone should not be used to prepare a patient for pheochromocytoma surgery. Another issue is related to the choice of the volatile agent for the maintenance of anesthesia. We used desflurane because of its great manageability despite its known sympathomimetic propensity. This effect appears to be minor especially when large doses of opioids are used and, like Lippman et al. [20] we agree that this factor plays little or no role in the maintenance of appropriate intraoperative haemodynamic conditions.

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