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Case Report

When beta-blocker unmasks the diagnosis: pheochromocytoma mimicking acute coronary syndrome. A case report

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ABSTRACT

Pheochromocytoma is a catecholamine-producing neuroendocrine neoplasm classically associated with secondary hypertension. We describe the case of a 63-year-old male who presented with intermittent precordial discomfort, paroxysmal hypertensive episodes, and a mild rise in cardiac troponin levels. He was referred to our institution with a presumptive diagnosis of acute coronary syndrome. Serial admission electrocardiograms showed no dynamic ischemic changes, and transthoracic echocardiography demonstrated preserved global systolic function without regional wall motion abnormalities. A noninvasive ischemic workup was therefore planned. Before performing coronary computed tomographic angiography (CCTA), the patient received propranolol, after which he developed a hypertensive crisis accompanied by recurrent precordial pain; an identical episode occurred following a second beta-blocker dose. CCTA ruled out flow-limiting coronary artery stenoses. In view of the clinical context, pheochromocytoma was suspected. Contrast-enhanced abdominal computed tomography identified a left adrenal mass, and urinary metanephrines were elevated. The patient underwent adrenalectomy, and histopathological examination confirmed a benign pheochromocytoma. Postoperative recovery was uneventful, and the patient remains asymptomatic at follow-up.

Keywords: Pheochromocytoma; Acute Coronary Syndrome; Adrenergic Beta-Antagonists (Source: MeSH-NLM).

RESUMEN

Cuando el betabloqueador revela el diagnóstico: feocromocitoma simulando síndrome coronario agudo: reporte de caso

El feocromocitoma es un tumor neuroendocrino productor de catecolaminas cuya presentación clínica puede simular un síndrome coronario agudo y representar un reto diagnóstico en la cardiología clínica. Presentamos el caso de un varón de 63 años con dolor precordial intermitente, hipertensión paroxística e incremento leve de troponinas. Fue referido a nuestra institución con diagnóstico de síndrome coronario agudo. Los electrocardiogramas de ingreso no evidenciaron signos de isquemia aguda; el ecocardiograma transtorácico no mostró trastornos segmentarios de motilidad. Se propuso una estratificación isquémica no invasiva. Previo a la angiografía coronaria por tomografía computarizada (CCTA), se administró propranolol, tras lo cual el paciente desarrolló crisis hipertensiva y dolor precordial. El mismo episodio se presentó luego de una segunda dosis de betabloqueador. La CCTA no identificó estenosis coronarias significativas. Ante la sospecha de feocromocitoma, la tomografía abdominal con contraste mostró un nódulo suprarrenal izquierdo y en la orina se identificaron metanefrinas elevadas. Se realizó una adrenalectomía y la histopatología confirmó un feocromocitoma benigno. El paciente evolucionó favorablemente, asintomático hasta la fecha.

Palabras clave: Feocromocitoma; Síndrome Coronario Agudo; Bloqueador de Receptores Beta-Adrenérgicos (Fuente: DeCS-BIREME).

Introduction

Phaeochromocytomas are catecholamine-secreting neuroendocrine tumours arising from chromaffin cells of the adrenal medulla; approximately 10% are extra-adrenal, known as paragangliomas, and up to 10% are malignant [1]. The mean age at diagnosis ranges between 40 and 50 years, and the annual incidence is estimated at 3-8 cases per million inhabitants. Their prevalence is 0.1-0.6% among hypertensive populations; however, these figures are likely underestimated, as autopsy studies have shown that up to 50% of diagnoses are post mortem [1,2].

Although most cases are sporadic, around 40% are associated with genetic disorders [2], most commonly Von Hippel-Lindau disease, Multiple endocrine neoplasia type 2, and Neurofibromatosis type 1 [3].

These tumours are characterised by adrenergic crises, including paroxysmal hypertension, headache, palpitations, and diaphoresis; however, clinical presentation may be atypical, making diagnosis challenging [2]. Prognosis is generally favourable, with a post-surgical survival rate of 98-100%, although the condition can be fatal if left undiagnosed [4].

We present the case of a patient referred to our institution with a diagnosis of acute coronary syndrome (ACS); however, this was ultimately confirmed to be a phaeochromocytoma with an atypical presentation.

Case report

A 63-year-old overweight man with no relevant family history, hypertension, or other comorbidities was referred with episodic oppressive chest pain associated with diaphoresis and a diagnosis of ACS. The episodes were self-limited, short in duration, and associated with hypertension. On admission, he was normotensive, haemodynamically stable, and asymptomatic.

The initial electrocardiogram showed a QS pattern in the inferior leads (II, III, aVF); however, there was no ST-segment elevation or reciprocal changes. High-sensitivity troponin T was 0.022 ng/mL (normal <0.005 ng/mL). Serial electrocardiograms showed no dynamic changes suggestive of acute ischaemia **(Figure 1)**. Transthoracic echocardiography demonstrated a preserved left ventricular ejection fraction (LVEF) of 76% with left ventricular hypertrophy, without evidence of apical ballooning or regional wall motion abnormalities.

Given the suspicion of non-ST elevation ACS and low cardiovascular risk, coronary computed tomography angiography (CCTA) was performed. As part of the protocol, propranolol 20 mg was administered orally to reduce heart rate below 65 beats per minute; however, minutes after administration, the patient developed a hypertensive crisis (210/110 mmHg) associated with diaphoresis and recurrence of chest pain. Intravenous nitroglycerin infusion

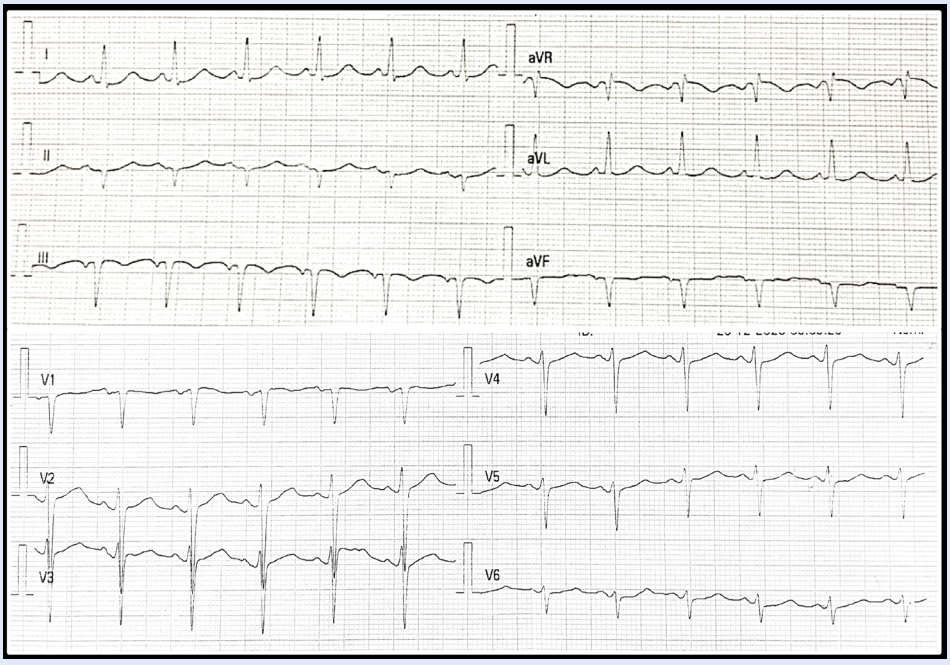

Figure 1. Twelve-lead electrocardiogram at admission. Sinus tachycardia, left axis deviation, and narrow QRS complexes are observed, with no evidence of bundle branch block or overt atrioventricular conduction abnormalities. Predominantly negative complexes are noted in the inferior leads, along with increased precordial voltages suggestive of left ventricular hypertrophy. No significant dynamic electrocardiographic changes were documented in serial tracings during hospitalisation.

was administered until symptom resolution. Six hours later, propranolol was re-administered, triggering the same clinical presentation. CCTA revealed left coronary dominance, a non-dominant right coronary artery arising anomalously from the left coronary sinus without an intra-aortic course, and mild atherosclerotic plaques in the left anterior descending and circumflex arteries **(Figure 2)**, thereby excluding type 1 myocardial infarction.

Due to the clinical triad of hypertension, diaphoresis, chest pain, and the triggering of symptoms after beta-blocker administration, a catecholamine-secreting tumour was suspected. Contrast-enhanced abdominopelvic computed tomography with washout protocol demonstrated a solid left adrenal nodule measuring 32 x 28 mm, suggestive of an atypical adenoma **(Figure 3)**.

Urinary metanephrines were requested. The initial result was negative; however, due to persistent clinical suspicion, a second test performed shortly after a hypertensive crisis was positive: 1.2 mg/24 h (normal: 0.05-1 mg/24 h) and a metanephrine/creatinine ratio of 0.76 (normal: <0.6), supporting the diagnosis. Perioperative management with alpha-blockade and optimisation of euvolaemia was initiated, followed by left adrenalectomy. Intraoperative findings included an adrenal gland measuring 5 x 4 cm with a 3 x 3 cm tumour. Histopathology revealed a brown-reddish nodule without atypical mitoses or capsular invasion, with a low Ki-67 proliferative index, confirming benign phaeochromocytoma **(Figure 4)**. The postoperative course was uneventful. At 2-year follow-up, the patient remains asymptomatic, without adrenergic crises, and urinary metanephrines are negative.

Discussion

Phaeochromocytoma is a catecholamine-secreting tumour characterised by adrenergic crises. The classic triad includes headache, diaphoresis, and tachycardia, although it is present in only 25% of cases. Atypical presentations are common and may complicate diagnosis [5]. Most patients develop hypertension, which is paroxysmal in approximately 30% of cases; however, 5-15% are normotensive and, paradoxically, up to 40% may present with orthostatic hypotension, attributed to hypovolaemia, beta-adrenergic vasodilation, and downregulation of adrenergic receptors [1,6,7].

Phaeochromocytomas arising from the right adrenal gland and those secreting higher levels of norepinephrine than epinephrine are more strongly associated with cardiovascular manifestations [8]. Myocardial involvement occurs in 32-65.4% of cases. The tumour is often referred to as a "great cardiovascular mimic", as its clinical presentation may resemble ACS, with oppressive chest pain, ST/T-segment abnormalities in up to 24% of cases, and elevated troponin levels [2,9]. Other cardiovascular manifestations include catecholamine-induced cardiomyopathy (hypertrophic, dilated, or Takotsubo-type), cardiogenic shock, aortic dissection, acute cerebrovascular events, pulmonary embolism, intracranial haemorrhage, and syncope. Palpitations are reported in 50-70% of patients, and documented arrhythmias include supraventricular arrhythmias, QT prolongation, ventricular tachycardia, and cardiac arrest [2,10].

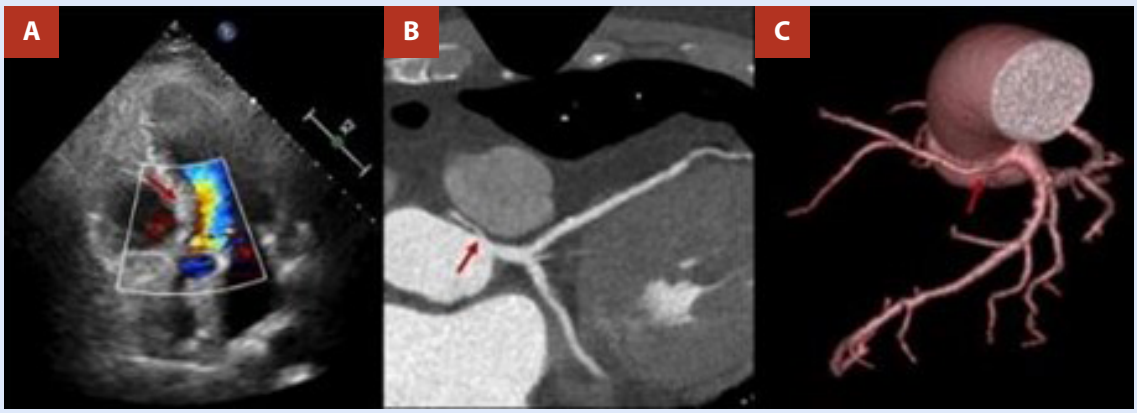

Figure 2. Multimodal cardiovascular imaging: transthoracic echocardiography and coronary computed tomography angiography. **A.** Transthoracic echocardiography showing left ventricular hypertrophy and dynamic left ventricular outflow tract obstruction, with a maximum estimated gradient of 45 mmHg (red arrow), without systolic anterior motion (SAM) of the mitral valve and no clinical impact. **B.** Coronary computed tomography angiography (CCTA), multiplanar reconstruction, demonstrating an anomalous origin of the non-dominant right coronary artery (RCA) from the left coronary sinus (red arrow), without an interarterial course or dynamic compression. Left coronary dominance is present; the left anterior descending and circumflex arteries show atherosclerotic plaques causing mild coronary stenosis. **C.** Three-dimensional CCTA reconstruction confirming the anomalous origin of the RCA (red arrow) with a non-malignant course, consistent with a prepulmonic trajectory.

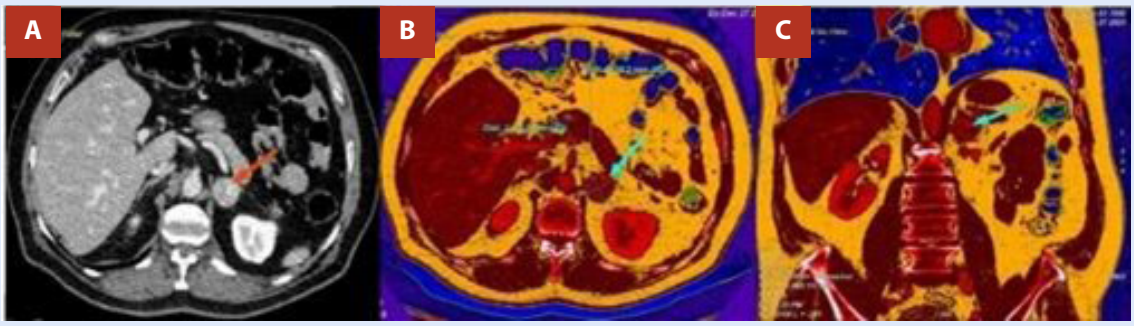

Figure 3. Adrenal characterisation using computed tomography washout protocol. **A.** Contrast-enhanced abdominopelvic CT showing a well-defined solid nodular lesion measuring 32 x 28 mm in the left adrenal gland (red arrow). **B, C.** Washout protocol demonstrating attenuation values of 35 Hounsfield units (HU) in the non-contrast phase, 99 HU in the venous phase, and 44 HU in the delayed phase, with absolute washout of 85% and relative washout of 55%, findings consistent with an atypical adrenal adenoma (light blue arrow).

The pathophysiology of cardiac involvement involves coronary vasospasm at both epicardial and microvascular levels mediated by alpha-adrenergic stimulation, associated with smooth muscle medial thickening, functional no-reflow, and an imbalance between oxygen supply and demand. These processes lead to endothelial dysfunction and increased platelet aggregation. In addition, catecholamine toxicity increases myocardial cell membrane permeability, elevates intracellular calcium and reactive oxygen species, and results in myocardial stunning and apoptosis [11,12]. Inflammatory infiltration, myocardial fibrosis, and beta-adrenergic receptor downregulation have also been described [10,13].

In our patient, the occurrence of angina following propranolol administration can be explained by beta-adrenergic receptor blockade in the vascular bed. In a hypercatecholaminergic state, the autonomic nervous system is deprived of its compensatory vasodilatory pathway, leading to severe peripheral and coronary vasoconstriction, abrupt increases in afterload and blood pressure, and heightened myocardial oxygen demand, ultimately resulting in hypertensive crisis and anginal chest pain. This explains the fundamental therapeutic principle in phaeochromocytoma: alpha-adrenergic blockade should be initiated before considering beta-adrenergic blockade [1,6].

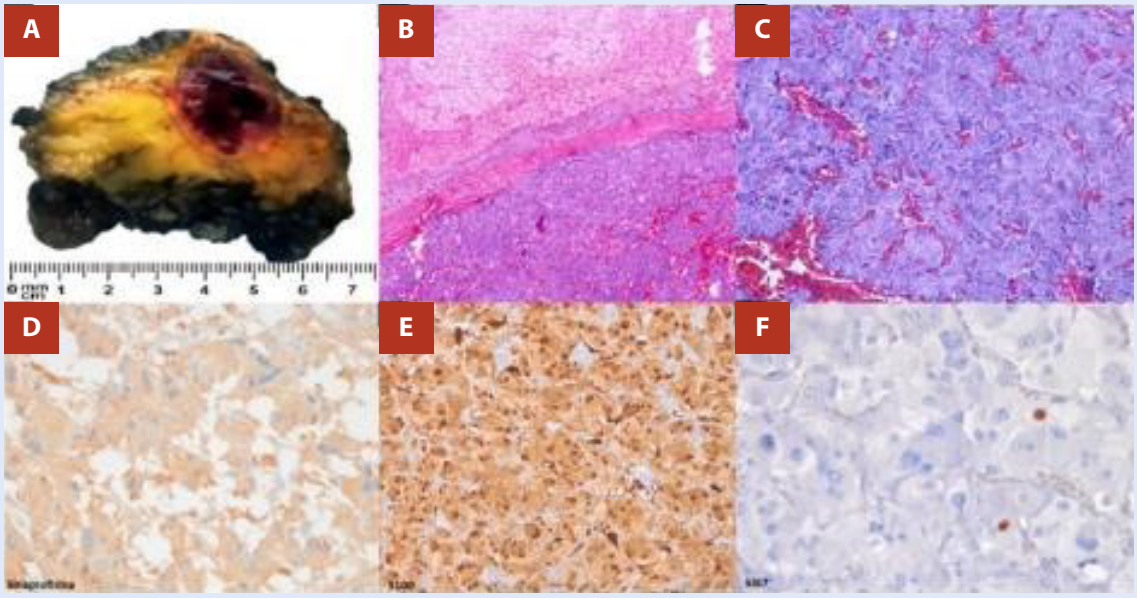

Figure 4. Histopathological and immunohistochemical confirmation of adrenal phaeochromocytoma. **A.** Left adrenalectomy specimen showing a well-circumscribed 25 x 23 mm brownish nodule with haemorrhagic areas, without macroscopic involvement of the cortex or capsule. **B.** Light microscopy showing neoplastic proliferation arranged in nests separated by a delicate capillary network. **C.** Tumour cells with granular basophilic cytoplasm, vesicular nuclei, and conspicuous nucleoli. **D-F.** Immunohistochemistry demonstrating diffuse synaptophysin expression **(D)**, S100 protein positivity in sustentacular cells **(E)**, and a Ki-67 proliferative index <1% **(F)**, without necrosis or significant atypia, consistent with a low-risk (benign) phaeochromocytoma.

CCTA is recommended as a non-invasive ischaemic stratification tool in patients with suspected ACS and low risk (Class IIa, level of evidence A), as in our case [13]. CCTA demonstrated an anomalous origin of the right coronary artery without high-risk features and non-obstructive coronary artery disease [14], thereby excluding type 1 myocardial infarction. Regarding invasive coronary angiography, the use of contrast media in these patients may precipitate hypertensive crises or heart failure due to increased catecholamine release. Additionally, there is a risk of tumour haemorrhage related to anticoagulation during the procedure [15]. Therefore, non-invasive coronary imaging and accurate aetiological diagnosis were essential in this case. Scintigraphy is a functional imaging technique based on the uptake of metaiodobenzylguanidine (MIBG), a norepinephrine analogue, by adrenal tissue. It has higher specificity than computed tomography or magnetic resonance imaging and is indicated when these modalities are negative but clinical suspicion of phaeochromocytoma remains high [16,17].

For diagnosis, plasma and urinary metanephrines should be requested; however, false-negative results may occur when sampling is not performed close to adrenergic crises, in tumours smaller than 1 cm or 50 g, in the presence of tumour necrosis, due to pre-analytical errors, or in atypical secretory phenotypes [5,18].

The definitive treatment of functioning phaeochromocytoma is surgical resection, which can reverse cardiovascular complications [19]. Perioperative management is critically important. First, relative hypovolaemia should be corrected, and alpha-blockers should be administered for 7-14 days prior to surgery [2,9]. Subsequently, and never beforehand, beta-adrenergic blockade may be introduced for the control of tachyarrhythmias [6].

The main postoperative complications are hypotension, due to the abrupt fall in catecholamine levels, and hypoglycaemia secondary to rebound hyperinsulinaemia. Cure is confirmed through metabolite quantification from the tenth postoperative day onwards, as secretion may persist during the first postoperative week. If metanephrine levels remain elevated, adrenal scintigraphy should be performed. Thereafter, annual follow-up is recommended to exclude recurrence [20].

As limitations of this case, follow-up duration was relatively short (2 years), and genetic testing was not performed.

In conclusion, phaeochromocytoma is a well-recognised mimic of ACS and should be considered in the differential diagnosis of patients presenting with angina in the absence of significant coronary lesions, particularly when chest pain occurs concomitantly with hypertensive crises. In such cases, beta-blockers may precipitate a catecholaminergic crisis due to unopposed alpha-adrenergic activity, leading to intense vasoconstriction, a sudden increase in afterload, and exacerbation of myocardial ischaemia, providing an important clinical clue to the underlying aetiology.

Ethical aspects

This case report was approved by the institutional ethics committee, and written informed consent for publication was obtained from the patient.

Author contributions

AVB, LEG, IGG, MAM, DGC: Conceptualisation, investigation, original draft writing, and manuscript review and editing.

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