

Mechanical bridge to recovery in pheochromocytoma myocarditis

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Background. A 27-year-old male presented with exercise-related symptoms of chest tightness, palpitations, breathlessness and severe headache, with occasional nausea, dizziness, and blurred vision. Apart from a family history of coronary artery disease there was no other medical history of note.

Investigations. Clinical examination, treadmill exercise test (Bruce protocol), electrocardiography, MRI of the abdomen, blood tests, chest radiography, coronary angiography, two-dimensional echocardiography, transesophageal echocardiography, microscopy of the tumor, ¹³¹I-iodine metaiodobenzylguanidine scan.

Diagnosis. Pheochromocytoma myocarditis.

Management. Intra-aortic balloon pump, levosimendan and dobutamine infusion, α -blockade with phentolamine, surgical removal of the pheochromocytoma, Levitronix® (Levitronix LLC, Waltham, MA) left ventricular assist device implantation.

Westaby, S. *et al.* *Nat. Rev. Cardiol.* 6, 482–487 (2009); doi:10.1038/nrcardio.2009.58

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Learning objectives

Upon completion of this activity, participants should be able to:

- 1 Describe the epidemiology of pheochromocytoma.
- 2 Identify elements of the diagnosis of pheochromocytoma.
- 3 Describe the cardiac effects of pheochromocytoma.
- 4 Specify the recommended treatment of pheochromocytoma myocarditis.

The case

A 27-year-old male soldier returned from Iraq with exercise-related symptoms of chest tightness, palpitations, and breathlessness followed by headache. On occasions he experienced nausea, dizziness, and blurred vision. With time, headache became the most distressing of the

symptoms and emerged as prodromal for the rest of the symptom complex. Apart from a family history of coronary artery disease there was no other medical history of note. The patient was referred by the Army for cardiological investigation at the local district general hospital. Clinical examination of the heart, lungs, and abdomen were unremarkable. His resting electrocardiogram was normal and his blood pressure (BP) was 117/68 mmHg. On a treadmill exercise test, the patient completed five stages of the Bruce protocol, achieving the maximum predicted heart rate without significant electrocardiographic changes. However, within 2 min of stopping exercise he developed a severe headache, paresthesia in the arms, and breathlessness at rest. His BP was then 240/110 mmHg and the electrocardiogram showed ST-segment depression. The cardiologist did not consider the symptoms to be typical of any specific disorder, but linked headache with the surge in BP. To further investigate the cause of hypertension, MRI of the abdomen was performed, together with blood tests for renin–aldosterone ratio and a 24 h urine collection for catecholamine screen. With these investigations pending the patient was allowed home, but advised not to recommence military duties.

One week later the patient presented at the emergency department of a different hospital with cardiogenic shock. His systolic BP was 50 mmHg and electrocardiography showed widespread, nonspecific ST-segment changes (Figure 1). Pulmonary edema was detected by auscultation of the lungs and confirmed by a chest radiograph. The cardiac silhouette was not considered to be enlarged. A friend of the patient related the history of hypertension. Without the benefit of unreported investigations from the previous hospital,

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Competing interests

The authors, the Journal Editor B. Mearns and the CME questions author C. P. Vega declare no competing interests.

myocardial infarction was suspected and the patient underwent insertion of an intra-aortic balloon pump (IABP). Coronary angiography showed normal vessels. Two-dimensional echocardiography showed globally impaired left ventricular (LV) contractility with a LV ejection fraction less than 15%. Hypotension proved refractory to epinephrine infusion. Realizing that the young man was deteriorating inexorably, the cardiology team sought help from a surgical center 150 miles away, where mechanical circulatory support could be provided. By this stage the differential diagnosis had shifted from myocardial infarction towards myocarditis or end-stage idiopathic dilated cardiomyopathy. During inter-hospital transfer, the patient continued to breathe spontaneously. An accompanying anesthetist used intravenous levosimendan and dobutamine infusions to raise the systolic BP to 65 mmHg.

On arrival at the surgical center, the patient was cerebrally obtunded, anuric, and acidotic, but still breathing spontaneously and able to respond to command. CT revealed an 80 mm adrenal tumor above the left kidney (Figure 2). He was then taken directly to the operating room and placed on cardiopulmonary bypass via median sternotomy. A hemofilter was incorporated into the bypass circuit and phentolamine was used to provide α -blockade. Transesophageal echocardiography showed persistently poor LV contractility. To avoid exacerbation of cardiac failure, β -blockade was not used.¹ The median sternotomy was extended to the left subcostal region to provide access to the tumor, which was removed uneventfully without a rise in systemic perfusion pressure. The tumor weighed 101 g and measured 70 × 65 mm. Microscopy confirmed pheochromocytoma (Figure 3).

A trial separation from cardiopulmonary bypass was abandoned because of poor LV contractility. A Levitronix® (Levitronix LLC, Waltham, MA) LV assist device (LVAD), which can be kept *in situ* safely for at least 30 days,² was then implanted by cannulation of the left atrium and ascending aorta. Circulatory support was then transferred to the LVAD. The right ventricle did not require inotropic support. The device dramatically reduced LV end diastolic and pulmonary artery pressures, providing resolution of the ST-segment elevation. With a measured LVAD output of 4.5 l/min, the median sternotomy was closed over the cannulas and the patient was transferred to the intensive care unit with a mean systolic BP of 85 mmHg. The IABP was left *in situ* to provide pulsatility. Urine flow resumed soon afterwards. During the course of the operation the urine catecholamine results were obtained from the first hospital. These showed very high levels of norepinephrine, epinephrine, metenephrine, and normeteneprhine diagnostic of pheochromocytoma (Table 1).

Over the next few days, pulmonary edema cleared and there was progressive improvement in LV contractility. Renal function returned to normal. After 6 days of LVAD support, the patient was slowly weaned from the

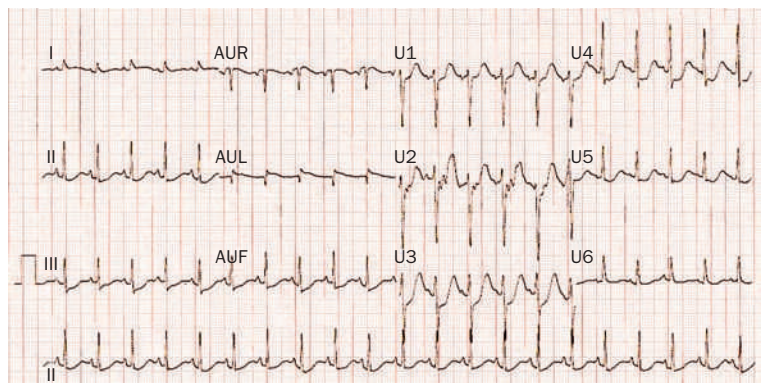


Figure 1 | The patient's electrocardiogram after he presented with cardiogenic shock, and showing widespread nonspecific ST-segment changes.

device and returned to the operating room for decannulation. The IABP was kept *in situ* for a further 48 h. Unexpectedly, repeat urinary catecholamine assay on the fifth postoperative day showed persistently elevated normeteneprhine, but no second tumor was found when the CT scan was reviewed. A radioisotope, ¹³¹iodine metaiodobenzylguanidine scan was performed to visualize any other catecholamine-containing ectopic or metastatic pheochromocytoma. When none were found it was concluded that the response to surgical stress was responsible for the sustained normeteneprhine elevation. The patient had an uneventful recovery and was discharged from hospital 3 weeks after admission. At 3 month follow-up, the patient had a normal LV ejection fraction and no symptoms

Discussion of diagnosis

Pheochromocytoma

Pheochromocytoma is a catecholamine-producing tumor that arises from the adrenal medulla (80–85%) or from extra-adrenal abdominal or extra-abdominal paraganglion tissue. The prevalence of pheochromocytoma in patients with hypertension is 0.1–0.6%. A relatively high incidence (0.05%) of pheochromocytoma in autopsy studies indicates that many undetected tumors cause premature mortality.³ Hereditary tumors are associated with germline mutations that cause multiple endocrine neoplasia type 2 (*RET* gene), von Hippel–Lindau syndrome (*VHL* gene), neurofibromatosis type I (*NFI* gene), and the familial paragangliomas (*SDHB* and *SDHD* genes). Bilateral adrenal tumors occur in 10–20% of familial pheochromocytoma. Although metastases are rare (up to 5%) from isolated adrenal pheochromocytomas, the prevalence of malignant disease is around 33% for extra-adrenal pheochromocytomas and even greater where associated with specific mutations in the *SDHB* and *SDHD* genes, which encode the B and D subunits of mitochondrial succinate dehydrogenase.³ The sporadic forms of pheochromocytoma usually occur in patients aged 40–50 years, while the hereditary forms present earlier.

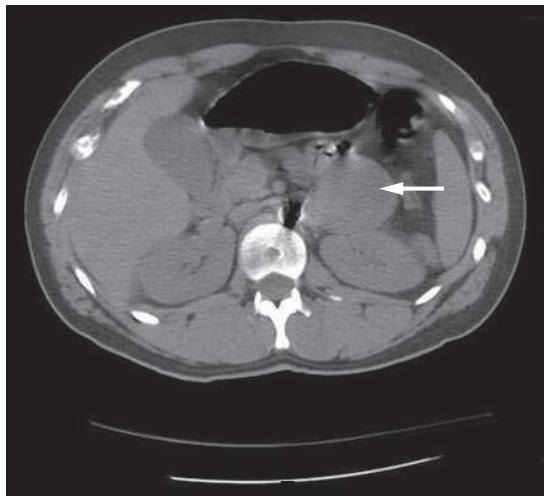


Figure 2 | CT scan after the patient had been transferred to a surgical center. The image shows an 80 mm round lesion (arrow) adjacent to the upper pole of the left kidney.

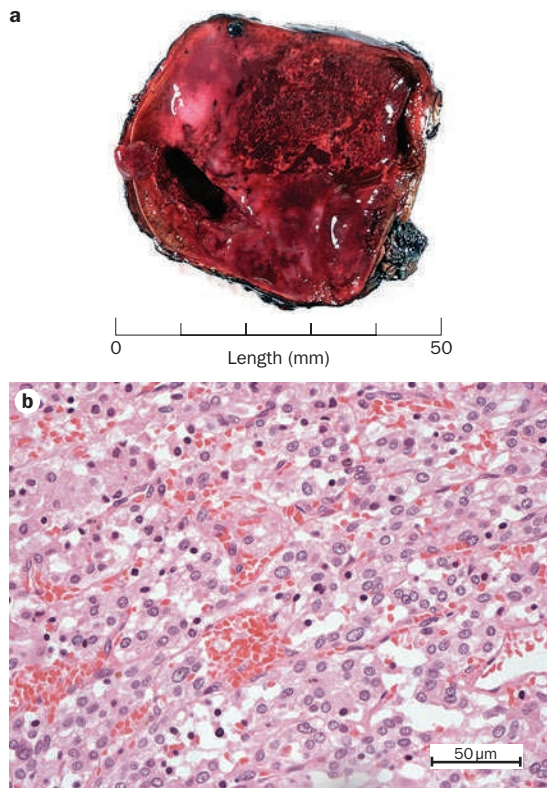


Figure 3 | Tumor histology. **a** | Histological examination showed the capsule of the pheochromocytoma to be intact and there was no obvious parenchymal necrosis. A thin rim of adrenal cortex was found in some areas together with a relatively normal adrenal gland measuring 40 × 20 mm at one edge of the main lesion. The parenchyma was hemorrhagic with prominent vasculature. **b** | Tumor microscopy showed multiple mitotic figures in some areas together with vascular invasion that could indicate aggressive pathology. In the absence of metastases or breach of the capsule, however, there are no universally accepted histological criteria for distinguishing benign from malignant pheochromocytomas.

The patient in this case will undergo genetic testing because hereditary tumors can be associated with other neoplasms or further pheochromocytomas.

The symptoms of pheochromocytoma depend on whether catecholamine secretion is persistent or intermittent and whether the predominant hormone is norepinephrine or epinephrine—in this patient it was normetanephrine. A diagnosis of pheochromocytoma should be considered in patients with hypertension and unexplained symptoms. Orthostatic hypotension can also be a presenting feature of pheochromocytoma and is thought to be caused by impairment of the sympathetic postural reflexes. The patient in this case displayed the classical episodic symptoms of severe headache, palpitations, chest pain, paresthesia, and a sense of impending doom. Sweating, tremor, and abdominal pain with nausea, vomiting, or diarrhea are other frequent manifestations of pheochromocytoma. Between paroxysms, patients can be normotensive and symptom free. This patient experienced symptom attacks after exercise when BP and cardiac afterload remained elevated, but heart rate and cardiac output fell. Although the symptom complex might seem characteristic, fewer than 0.1% of hypertensive patients have a pheochromocytoma.³

Most patients with pheochromocytoma secrete large amounts of norepinephrine, although epinephrine-secreting and dopamine-secreting tumors also occur.⁴ Patients with these tumors typically present with hypotension, cardiomyopathy, or cardiogenic shock when β -adrenergic stimulation overrides α -adrenergic stimulation. The most reliable screening method is 24 h urinary excretion of metanephrines. In a single voided urine specimen, more than 1 μg of metanephrines per mg of creatinine is indicative of pheochromocytoma. Levels of plasma catecholamines also provide a sensitive indicator and the tests are more reliable if blood and urine samples are collected during a hypertensive attack. When symptoms and biochemical tests suggest pheochromocytoma, CT or MRI are the most accurate means of locating the tumor, provided that it is larger than 5 mm in diameter. A metaiodobenzylguanidine radioisotope scan may also be required to detect ectopic or metastatic pheochromocytoma of less than 5 mm in diameter.

Cardiac dysfunction and pheochromocytoma

Electrocardiographic abnormalities—including T-wave inversion and LV hypertrophy in response to hypertension—occur in the majority of patients with pheochromocytoma.⁵ Sinus tachycardia, paroxysmal supraventricular tachycardia, and supraventricular ectopic activity resemble dysrhythmias in thyrotoxicosis and are caused by the direct chronotropic effects of catecholamines or from sudden increases in BP. Reflex bradycardia is mediated by baroreceptor stimulation. A sudden dramatic rise in BP can cause electrocardiographic changes, such as transient ST-segment elevation, T-wave inversion, and ST-segment depression,

which can lead to myocardial injury.⁵ These changes are, however, transient and reversible. By contrast, high catecholamine levels can cause structural and biochemical injury termed 'catecholamine myocarditis', which often proves fatal.⁶ Autopsy studies of patients who have died from pheochromocytoma show that half have active myocarditis with LV failure and pulmonary edema.⁷ Structural changes include focal degeneration and contraction band necrosis of the myocytes, inflammatory cell infiltration, medial thickening of small and medium size coronary arteries, and interstitial fibrosis. There may also be defective storage of endogenous amines and increased concentration of free fatty acids in the myocyte. The macroscopic findings in the patient in this case were consistent with acute myocarditis, but a myocardial biopsy sample was not obtained.

Sardesai *et al.* described six patients with pheochromocytoma presenting with pulmonary edema.⁸ Autopsies showed that all five of the patients who died from cardiogenic shock had normal sized hearts with catecholamine-induced focal myocardial necrosis and inflammatory cell infiltration. Pheochromocytoma tissue has been transplanted into rats that, within 6 weeks, reproducibly sustain cardiac injury—comprising multifocal lesions of replacement fibrosis, myocyte contraction band necrosis, and inflammatory infiltrates.⁹ Animal models simulating major stress also manifest focal myocardial necrosis or myofibrillar degeneration.¹⁰ The same pathological process has been identified in humans who have died from the stress of physical assault, but without internal injuries.¹¹ In addition Pavin *et al.* reported two patients with cardiogenic shock occurring in the aftermath of severe emotional stress.¹² Chest X-ray in both individuals showed pulmonary edema. Blood catecholamines measured 12 h after the stressful event showed highly elevated epinephrine and norepinephrine concentrations, which reverted to normal within 3 days. There was improvement in cardiac function 1 week later, and coronary angiography was normal. Pheochromocytoma, coronary artery disease, and viral myocarditis were ruled out in each case. Pulmonary edema was probably caused by LV failure, but also by the direct effects of catecholamines on the pulmonary vasculature. Sympathetic stimulation increases pore size in pulmonary capillaries, causing pulmonary alveolar transudation. Despite functional impairment, the left ventricle was not dilated and contractility of the basal segments was unchanged.

LV dysfunction in response to stress (also known as Takotsubo cardiomyopathy, or apical ballooning syndrome) is an increasingly well-recognized entity thought to be related to myocardial stunning, but without coronary stenoses.¹³ High levels of epinephrine trigger a switch in intracellular signal trafficking from Gs protein to Gi protein signaling via the β -2-adrenoceptor. Although this switch protects against the proapoptotic effects of intense activation of β -adrenoceptors, it is negatively inotropic, with

Table 1 | 24 h urine catecholamine and catecholamine metabolite levels

Catecholamine/metabolite (unit)	Preoperative	Fifth postoperative day	Normal range
Normetephrine (μ mol daily)	70.1	11.7	0–3.60
Metephrine (μ mol daily)	17.5	1.6	0–1.90
Norepinephrine (μ mol daily)	6.0	NA	0.07–0.48
Epinephrine (μ mol daily)	1.1	NA	0–0.10
Dopamine (μ mol daily)	2.4	NA	0.49–2.85
Urine volume (l daily)	2.95	1.88	0.8–2.0

Abbreviation: NA, not available.

greatest effect in the apical myocardium. These events mimic apical myocardial infarction with electrocardiographic changes similar to those in acute coronary syndromes. Massive catecholamine secretion may cause coronary vasoconstriction and explain ischemia at the LV apex. Catecholamines also increase sarcoplasmic calcium concentration, which causes myocyte necrosis. Hypocalcemia can also have a role in the etiology of cardiogenic shock, particularly in patients with β -receptor downregulation. Olson *et al.* suggested that epinephrine-secreting pheochromocytomas cause hypocalcemia through increased calcium use by the tumor or by bone calcium deposition resulting from tumor hypersecretion of adrenomedullin.⁴ Reversibility of the lesion might be related to shortness of exposure to excessive catecholamines, resulting in stunning and metabolic abnormalities rather than irreversible necrosis.

Treatment and management

The patient's survival depended upon rapid inter-hospital transfer and provision of effective and reliable circulatory support until the left ventricle recovered. Successful excision of the pheochromocytoma during intractable cardiogenic shock could only have been performed safely using cardiopulmonary bypass.

Surgery is the only definitive treatment for pheochromocytoma, but preoperative pharmacological management is necessary to prevent systemic vasoconstriction. Medical treatment should include α -blockade (phenoxybenzamine, or phentolamine in an unstable patient) to control hypertension, followed by β -blockade (propranolol) to prevent sinus tachycardia or catecholamine-induced arrhythmias. Although unopposed α -blockade can result in severe reflex tachycardia, prior β -blockade and loss of β -adrenoceptor mediated vasodilatation may precipitate hypotensive crisis in patients with pheochromocytoma.¹ Sloan and Thompson¹ reported propranolol-induced pulmonary edema and cardiogenic shock, while Wood *et al.*¹⁴ described collapse with unrecordable BP following an oral dose of captopril. Other authors have suggested that steroids could trigger acute heart failure by augmenting the sensitivity of myocardial cells to catecholamines.¹⁵ Alternative drugs include labetalol (a combined

Table 2 | Reversibility of acute and chronic heart failure by cause

Heart failure	Potentially reversible	Not reversible
Acute	Post cardiomyotomy stunning Acute viral myocarditis Acute myocardial infarction Postpartum cardiomyopathy Catecholamine myocarditis	Giant cell myocarditis
Chronic	Idiopathic dilated cardiomyopathy	Ischemic cardiomyopathy Amyloid or sarcoid cardiomyopathy Toxic or alcoholic cardiomyopathy Restrictive cardiomyopathy

α-adrenoceptor and β-adrenoceptor blocker) or calcium-channel blockers either alone or in combination with an α-adrenergic receptor blocker.

For patients who present with cardiogenic shock, the use of inotropic support is unlikely to improve the clinical condition. Continued exposure to high levels of catecholamines leads to chronic β-adrenergic desensitization,¹⁶ which results in decreased effectiveness of the β-adrenergic-receptor-stimulating inotropic agents in the failing heart. Patients with poor LV function already have fewer functional β-adrenergic receptors and less responsiveness to inotropic agents.¹⁷ β-adrenergic agents improve cardiac output only at the expense of increased myocardial oxygen consumption. Diastolic dysfunction persists and might deteriorate further. Patients with dilated cardiomyopathy also manifest a decline in β-adrenergic receptor function and receptor density. By contrast, myocardial unloading with an LVAD is known to reverse receptor downregulation in failing hearts.¹⁶ This process results in an improved myocardial response to sympathetic stimulation in mechanically unloaded ventricles.

Mechanical ‘bridge to myocardial recovery’ is an established treatment for conditions that cause acute myocardial failure (Table 2).² The rationale for this strategy is clear; a dilated failing heart with elevated wall tension, impaired subendocardial blood flow, and increased oxygen consumption is unlikely to recover spontaneously or with inotropic drive. The LVAD unloads the failing ventricle, boosts coronary and systemic blood flow, and promotes myocardial recovery at the cellular and metabolic level. Improvement occurs over days, weeks, or months depending upon

pathology and duration of the disease process. Patients with myocarditis or postischemic stunning can recover in 5–10 days.² Those with idiopathic dilated cardiomyopathy might require months of unloading. A range of pulsatile and continuous-flow LVADs are suitable for this purpose. The Levitronix® centrifugal pump was selected for this patient because it is safe and effective for at least 30 days.

Catecholamine-induced myocarditis can be added to the list of indications for LVADs. Grasselli *et al.* described successful mechanical support in a 47-year-old female who presented with multisystem failure.¹⁷ Takagi *et al.* reported cardiogenic shock after a patient with occult pheochromocytoma received dexamethasone.¹⁵ Steroids could have triggered shock by augmenting the sensitivity of the myocyte to catecholamines. When an IABP failed to improve hemodynamics, a percutaneously established extracorporeal circuit resolved the situation. Similarly, Grinda *et al.* reported short-term extracorporeal circulatory support superseded by the use of an implanted DeBakey® (MicroMed Cardiovascular, Inc., Houston, TX) LVAD in a patient with pheochromocytoma.¹⁸ After recovery, patients require continued surveillance, but usually have normal LV function.¹³ The risk of tumor recurrence in the remnant adrenal gland is 10%, but metachronous tumor development in the contralateral adrenal gland may occur in 30% of hereditary cases.³ Equally, there is no infallible method to predict metastatic disease from histological findings in the resected tumor.

Conclusions

Pheochromocytoma can cause rapid onset of cardiogenic shock in response to intense exercise, β-blockade, or steroid administration. Catecholamine-induced myocarditis is characterized by contraction band necrosis and may rarely occur in patients without pheochromocytoma who are subject to intense stress. Inotropic support is rarely of benefit and might worsen cardiac failure in patients with pheochromocytoma. Cardiopulmonary bypass allows safe excision of the tumor during cardiogenic shock and mechanical left ventricular unloading then provides an opportunity for the heart to recover. Catecholamine-induced myocarditis can be included in the list of indications for LVAD therapy.

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Acknowledgments

Written consent for publication was obtained from the patient. Charles P. Vega, University of California, Irvine, CA, is the author of and is solely responsible for the content of the learning objectives, questions and answers of the MedscapeCME-accredited continuing medical education activity associated with this article.