Clinical Reports

Phaeochromocytoma and mitral valve replacement

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A phaeochromocytoma was diagnosed following a mitral valve replacement and coronary artery bypass in a patient with progressive mitral regurgitation. Despite a previous adverse reaction to anaesthesia, this was not predicted perhaps due to his cardiac disease. Fever, leucocytosis and confusion were also prominent features. Haemodynamic control was achieved with the aid of labetalol.

Case report

A 73-year-old man of 80 kg was admitted for elective mitral valve replacement and single coronary artery bypass. A murmur of mitral regurgitation (MR) had been noted a year earlier when he suffered a small inferior myocardial infarction. Progressive dyspnoea on exertion (NYHA class 2) and recurrent pulmonary oedema led to the diagnosis of severe MR, 60 per cent right coronary artery occlusion and pulmonary hypertension. He denied chest pain and palpitations.

During induction of anaesthesia for retropubic prostatectomy seven years earlier, his blood pressure (BP) increased to 200/100 mmHg but settled with two per cent halothane. Near the end of the case, ventricular tachycardia developed requiring lidocaine boluses. Postoperative (PO) heart rate (HR) and BP were 110 min⁻¹ and 180/100 mmHg respectively but resolved over three days without treatment.

Admission medications were: digoxin 0.25 mg OD, propranolol 10 mg qid, isosorbide dinitrate 10 mg qid. The BP was 140/80 mmHg, HR 74 min⁻¹, and apart from the murmur of MR, physical examination was normal. The

Key words

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ECG showed ST and T wave changes inferiorly, and haematology and chemistry were normal except for a random blood sugar of 123 mg dl⁻¹ (N 60–115).

Induction of anaesthesia with lorazepam 3 mg, fentanyl infusion 60 µg·kg⁻¹, dimethyltubocurarine 10 mg, pancuronium 3 mg, and lidocaine 120 mg, preceded a difficult intubation. The BP increased to 200/80 mmHg, but settled at 140/60 mmHg after ten minutes. He received droperidol 4 mg for hypertension early during cardiopulmonary bypass, and morphine 45 mg near the end. After he was weaned from cardiopulmonary bypass (CPB), a BP of 220/100 mmHg was treated with nitroglycerine (TNG) 2 µg·kg⁻¹·min⁻¹ and sodium nitroprusside (SNP) 1.5 µg·kg⁻¹·min⁻¹. Heart rate was 70 min⁻¹ initially off pump, increasing to 100 min⁻¹ by the end of surgery.

In the first 24 hours PO, his BP fluctuated from 150/90 mmHg to 200/100 mmHg and HR gradually increased to 130 min⁻¹. His temperature persisted at 38.6°C and white count was 20×10^9 L⁻¹ or more for 72 hours. Haloperidol 2.5 mg IV was given several times for marked confusion. Haemodynamic control (BP 140/90 mmHg, HR 90 min⁻¹) was finally achieved with labetalol 400 mg bid PO, propranolol 80 mg qid PO, nifedipine 10 mg qid PO, alpramethyldopa 250 mg qid PO, and hydralazine 25 mg qid PO by the third PO day. Phentolamine 10 mg IV at this time produced no change in BP.

At this time, his 24-hour urinary vanillylmandelic acid (VMA) was 77 mg (N 2–7). A CT scan using ¹³¹I meta-iodobenzyl guanidine (MIBG) later showed a solitary functioning right adrenal tumour. Phenoxybenzamine therapy was commenced. A history of headaches, sweating, tremor or anxiety was denied on repeat questioning. Eight weeks later, a benign phaeochromocytoma was resected and the patient remains well two years later.

Discussion

The estimated incidence of phaeochromocytoma is 0.1 per cent of hypertensives in the general population. Thus, the anaesthetist needs a high index of suspicion for the condition which may occur as an incidental problem only once in his career. Fortunately, reports of undiagnosed

cases which manifest perioperatively^{1,2} suggest that almost all such patients have clinical findings documented preoperatively which are consistent with the diagnosis. Most often, these are hypertension, both persistent and paroxysmal, and paroxysmal headaches, sweating and palpitations.

This patient, like only a few others,³ had no suggestive history even in retrospect. The severe MR may have masked the hypertension so that an increase in systemic vascular resistance would manifest as increasing congestive heart failure or episodes of pulmonary oedema which, in this man, were attributed solely to a progressing valvular lesion. Other cases have been reported following CPB, but these have involved coronary artery bypass,^{4,5} aortic stenosis⁶ and aortic regurgitation.⁷

More significance could have been placed on the previous perioperative history, which was felt to evidence nonspecific cardiovascular disease at the time of occurrence. The gradual resolution of tachycardia and hypertension over three days is an uncommon pattern in our practice and has been noted to occur with phaeochromocytoma.⁵ Acute ventricular tachydysrhythmias⁸ and lapses of many years^{9,10} between the onset of symptoms and diagnosis have been documented.

This case shows striking similarity to another recently reported one,⁵ and confirms the behaviour of this endocrinopathy under high-dose fentanyl anaesthesia and CPB. Similar potential triggering factors were present, including intubation, morphine, hypothermia and especially droperidol.¹¹ Also, as his course progressed, he became refractory to high doses of multiple antihypertensive agents given parenterally in the acute phase. A phenolamine challenge was negative, but his BP did not come under control until after labetalol was added to his regimen. As this is a mixed adrenergic antagonist with alpha to beta blocking ratio of about 1:4,¹² this was a fortuitous choice in a patient whose symptoms suggest a tumour secreting both epinephrine (tachycardia, hyperglycaemia, fever, leucocytosis) and norepinephrine (hypertension).

Postoperatively, this patient behaved initially like a chronically hypertensive patient with good BP control using vasodilators. Urinary VMA may be elevated by the stress of cardiac surgery to double the normal range¹³ but his progressive clinical course and persistently high VMA concentration confirmed the diagnosis. Our index of suspicion was not raised until tachycardia developed. The fever and leucocytosis were suggestive findings¹⁴ which also were not initially appreciated.

In summary, phaeochromocytomas do not always present classically. Cardiac valve lesions and anaesthetic techniques and drugs that alter the catecholamine response can be modifying factors. Aggressive management reduces morbidity and mortality.

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Résumé

Un phéochromocytome a été diagnostiqué après un remplacement de valve mitrale et pontage aorto-coronarien chez un patient atteint de régurgitation mitrale progressive. Malgré des complications lors d'une anesthésie antérieure, le phéochromocytome ne pouvait être prédit possiblement à cause de sa maladie cardiaque. La fièvre, la leucocytose et la confusion furent des signes cliniques prédominants. Le contrôle hémodynamique fut acquis avec l'aide du labétolol.