SHORT REPORTS

Metastatic tumour of right atrium mimicking constrictive pericarditis and tricuspid stenosis

Though secondary cardiac tumours are 20 times more common than primary lesions,¹ and cardiac metastases are found in up to 19% of necropsies undertaken on patients who have died of cancer,² correct diagnosis of secondary spread to the heart is rare. We describe a patient in whom an unrecognised right atrial metastasis was clinically mistaken for constrictive pericarditis while results of cardiac catheterisation suggested tricuspid stenosis.

Case report

A 54-year-old Kenyan man presented with tiredness and dyspnoea of two months' duration. He was wasted, afebrile, and had pronounced ascites and leg oedema. His pulse was 76 beats/min, regular, of low volume and not paradoxical, and blood pressure was 130/80 mm Hg. The jugular venous pressure was raised to the angle of the jaw and no abnormal pulsation was noted. The heart sounds were quiet, a third sound was present, and on two isolated occasions a soft, low-pitched, mid-diastolic murmur was heard at the left lower sternal border. The liver was enlarged to four finger breadths below the right costal margin. A chest x-ray film showed apical opacification and cavitation in both lungs, with a normal heart size. The electrocardiogram showed generalised low voltage. Although acid-fast bacilli were not detected, pulmonary tuberculosis and constrictive pericarditis were diagnosed, and anti-tuberculous and diuretic treatment was started. Cardiac catheterisation (table) showed a pressure gradient across the tricuspid valve suggesting tricuspid stenosis. While awaiting cardiac surgery the patient's condition deteriorated and he died.

Measurements of intracardiac pressure

	Phasic	Mean	Comment
Right atrium	a = 21 mm Hg; v = 21 mm Hg	16 mm Hg	Raised
Right ventricle	16/5 mm Hg		Normal
Pulmonary artery	22/10 mm Hg	15 mm Hg	Normal
Pulmonary wedge		10 mm Hg	Normal
Left ventricle	95/5 mm Hg		Low
Aorta	95/65 mm Hg	75 mm Hg	Low

Necropsy showed bilateral pulmonary tuberculosis and congestive cardiac failure. A firm, greyish, round tumour, 10 × 7 × 6 cm, was present in the right atrium lateral to the coronary sinus. The bulk of the tumour was above the tricuspid valve but it infiltrated through all the layers of the heart, including the valve's lateral cusp. A small right para-adrenal tumour nodule was found, but no other lesions were detected. Histological examination of the two tumours showed the features of infiltrating, undifferentiated carcinoma. Both were clearly metastatic lesions but their primary source could not be determined.

Comment

In general, the clinical effects of any cardiac tumour depend more on its site than on its pathological nature. The most common manifestations of cardiac metastases are pericarditis and pericardial effusions, arrhythmias, and heart failure.¹ Carcinoma of the breast, bronchus, and kidney and melanoma, leukaemia, and lymphoma are the most frequently reported primary lesions,² though in sub-Saharan Africa metastases from hepatocellular carcinoma, Kaposi's sarcoma, and Burkitt's lymphoma assume a greater importance.³

A secondary deposit confined to one of the heart chambers behaves essentially like a myxoma. When right-sided, such lesions may cause right heart failure, tricuspid murmurs, low-voltage electrocardiograms, and pressure gradients across the tricuspid valve suggestive of tricuspid stenosis.⁴ The clinical and haemodynamic findings of right atrial myxoma and constrictive pericarditis may also resemble each other closely.⁵ Other possible misdiagnoses of a right atrial tumour include cor pulmonale, thromboembolic pulmonary hypertension, and, in patients receiving treatment for known malignant disease, cardiac side effects from cytotoxic drugs.

The case reported here is of interest because of the perplexing physical and haemodynamic findings, but also as a reminder that meta-

static heart disease is more common than is generally realised. Patients with certain malignant diseases now live longer and often receive drugs that may themselves cause cardiac side effects. It is important to remember the possibility of the spread of tumour to the heart.

- McAllister HA, Fenoglio JF. *Tumors of the cardiovascular system*. Washington DC: Armed Forces Institute of Pathology, 1978.
- Young JM, Goldman IR. Tumor metastasis to the heart. *Circulation* 1954;9:220-9.
- Lothe F, Somers K. Secondary tumors of the heart and pericardium in Ugandan Africans. *Archives of Pathology* 1960;69:158-67.
- Soulié P, Acar J, Renault P, Lainée R, Azoulay. Les myxomes de l'oreillette droite. *Arch Mal Coeur* 1961;54:241-73.
- Emanuel RW, Lloyd WE. Right atrial myxoma mistaken for constrictive pericarditis. *Br Heart J* 1962;24:796-800.

(Accepted 18 August 1982)

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Photophobia in lithium intoxication

The neural signs and symptoms of lithium intoxication are well documented.¹ We report what we believe to be the first reported case of photophobia in this condition.

Case report

A 32-year-old woman with a 10-year history of manic-depressive psychosis was admitted to hospital after the rapid onset of a severe depressive episode. She also had polycystic ovary syndrome with amenorrhoea, obesity, hirsutism, acne, and infertility. She had been treated with lithium for four years and at the time of admission was taking 1600 mg at night. To maintain a stable lithium concentration it was decided to change the dose to 800 mg twice daily. After four days lithium concentration was 1.2 mmol/l (8.3 mg/l) and dosage was altered to 600 mg in the morning and 800 mg at night. Three days later she complained of polyuria, polydipsia, ataxia, nausea, abdominal discomfort, hot and cold "flushes," mild headaches, and photophobia. The photophobia was such that she would not open her eyes under normal ward illumination and experienced severe pain when light from an ophthalmoscope was shone in her eyes. Fine lateral nystagmus was also observed. Neurological examination showed no other abnormality and there were no signs of meningism. Temperature, pulse, blood pressure, and respiration remained normal.

Lithium intoxication was diagnosed, confirmed by a serum lithium concentration of 1.6 mmol/l (11 mg/l) and her rapid recovery over 24 hours on stopping lithium treatment. The only other drug being taken concurrently was thyroxine 0.1 mg daily for hypothyroidism (secondary to lithium treatment) and no other drug had been recently withdrawn. Lithium was restarted at a lower dose and the patient was discharged well, free from side effects.

Comment

A search of the published reports has revealed no similar case reports, though Lewy² has suggested that patients with manic depression may be hypersensitive to light. Some reviews have linked pineal function to both light sensitivity and mood regulation but this does not seem to explain the above observation. We would be interested to hear reports of any similar cases or to receive comments.

- ¹ Tyrer S, Shopsin B. Neural and neuromuscular side effects of lithium. In: Johnson FN, ed. *Handbook of lithium therapy*. Lancaster: MTP Press, 1980:289-309.
- ² Lewy AJ, Wehr TA, Goodwin FK, Newsome SP, Rosenthal NE. Manic depressive patients may be supersensitive to light. *Lancet* 1981;i:383-4.

(Accepted 18 August 1982)

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Bromocriptine-induced psychosis in acromegaly

Dopaminergic mechanisms have been implicated in the pathogenesis of schizophrenia.¹ Psychosis induced by the dopamine-receptor agonist bromocriptine has been described mainly with high-dose regimens in Parkinsonism and less frequently with the lower doses used in hormonal disorders.²⁻⁴ The following case is of interest because of the insidious onset of psychosis and the family history of paranoid schizophrenia.

Mean growth hormone and prolactin concentrations before, during, and after treatment

	Basal	After glucose	While taking bromocriptine			Weeks after having stopped bromocriptine	
			10 mg/day	20 mg/day	30 mg/day	1 week	6 weeks
Growth hormone (mU/l) (normal range <10)	587	400	128	147	114	696	556
Prolactin (µg/l) (normal range <20)	107	—	4.5	5.5	—	54	125

Case report

A 32-year-old married composer of popular music, previously a successful artist, presented in November 1981 with a breast lump, one month before the birth of his first child. For three years he had become increasingly immersed in his music and more irritable, and he and his wife had become socially isolated. He had no history of psychiatric disorder or drug abuse, but his mother had been successfully treated for a well-documented paranoid schizophrenic episode in 1973, when aged 60.

Acromegaly was diagnosed on the basis of a five-year history of enlargement of the face, hands, and feet and grossly raised growth hormone concentrations (table). Skull radiography showed an enlarged pituitary fossa with destruction of the posterior wall and floor. Computed tomography showed asymmetry of the sella floor and right-sided destruction but no appreciable suprasellar or extrasellar tumour extension. Visual fields were normal. An excision biopsy specimen of the breast lump showed duct ectasia with some "lactational" change.

Bromocriptine 2.5 mg daily was started on 28 January 1982, increasing to 10 mg over the next two weeks and to 30 mg over the following month; this was continued until his admission in a psychotic state on 30 April. In retrospect, two weeks after starting bromocriptine he had become totally absorbed in his music, believing that it had a unique, inexpressible "three-dimensional" quality. His musical ideas became more bizarre and complex over the next 10 weeks and he spent long periods in bed engrossed in their interrelated meanings. Ten days before admission he had a vivid dream of a mother with a child with Down's syndrome, which he took to be a direct experience of God. He became preoccupied with ideas of good and evil and increasingly frightened. This led to a delusional misinterpretation that the devil was outside their home, and he and his wife fled to relatives. They began to suspect that his disturbance was due to the bromocriptine, which he discontinued, and they came to the casualty department. Up to this point his wife had shared his ideas, but this changed when he stated that she was evil and absconded with their child from casualty. He returned within hours, was admitted to a medical ward, and was later transferred to the psychiatric unit.

On admission he was in a fearful, paranoid, agitated state, showing partial insight in the presence of clear consciousness and absence of overt mood disorder, hallucinations, passivity phenomena, or formal thought disorder. On treatment with thioridazine 250 mg daily his paranoid ideas disappeared within a week. He was discharged taking trifluoperazine, which he failed to continue, and was readmitted two weeks later, for 10 days, with a mild recurrence of paranoid ideation that responded to trifluoperazine 10 mg daily. This was tailed off over a month with no recurrence of psychotic symptoms. Subsequent pituitary surgery was planned.

Comment

This psychotic episode appears to have been precipitated by bromocriptine. There is evidence of a liability to levodopa-induced affective disorder in those with a family history of affective illness.⁵ This case suggests an analogous genetic vulnerability to bromocriptine-induced psychosis. A family history of schizophrenia in addition to a personal history may be relevant in considering the use of bromocriptine.

- ¹ Crow TJ. The biochemistry of schizophrenia. *Br J Hosp Med* 1978;25:532-44.
- ² Parkes D. Bromocriptine. *N Engl J Med* 1979;301:873-8.
- ³ Pearson KC. Mental disorders from low dose bromocriptine. *N Engl J Med* 1981;305:173.
- ⁴ Steinbeck K, Turtle JR. Treatment of acromegaly with bromocriptine. *Aust NZ J Med* 1979;9:217-24.
- ⁵ Mendlewicz J. Application of genetic techniques to psychiatric research. In: Frazer A, Winokur A, eds. *Biological bases of psychiatric disorders*. New York: Spectrum Publications, 1977:87.

(Accepted 18 August 1982)

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Treatment of digoxin overdose with antigen-binding fragments of digoxin-specific antibodies

Severe digoxin toxicity due to accidental or deliberate overdose is rare, but is often life-threatening, especially if there is pre-existing cardiac disease. Treatment with gastric lavage, adjustment of electrolyte balance, antiarrhythmic drugs, pacing, and, when necessary, low-voltage DC shock is not always effective and the ultimate aim must be to lower circulating free digoxin concentrations. Dialysis and haemoperfusion clear digoxin but blood concentrations are rapidly restored from the large extravascular pool.¹ We treated a patient with severe digoxin toxicity using digoxin-specific antibodies and we report the efficacy of this treatment in a potentially lethal condition.

Case report

A 60-year-old man with a history of left ventricular failure, mitral regurgitation, and intermittent atrial fibrillation treated with digoxin, diuretics, and warfarin, was admitted after a suicide attempt with 12.5 mg digoxin, 150 mg warfarin, and 40 mg diazepam. On examination he was alert, in sinus rhythm at 60 beats/min, and blood pressure was 110/60 mm Hg. He was not in overt heart failure and cardiac signs were unchanged. Electrolyte balance and cardiac enzyme activities were normal. Chest x-ray film showed an enlarged heart. There was complete left bundle-branch block and left axis deviation on the electrocardiogram as previously, but the P-R interval was now prolonged to 0.28 seconds and there were frequent ventricular ectopics, some of which were multiple and multifocal. The initial serum digoxin concentration was 13 g/l (therapeutic range up to 2.2 g/l) and prothrombin index 7.9.

Gastric lavage was carried out one and a quarter hours after the overdose. A lignocaine infusion was started and a prophylactic temporary transvenous pacemaker inserted. Potassium concentrations were closely controlled. Despite these measures bradycardia worsened and the electrocardiographic changes persisted. We decided to lower the digoxin concentrations with antigen-binding fragments of sheep digoxin antibody. After preliminary test