CASE REPORT

Chronic pericardial constriction linked to the antiparkinsonian dopamine agonist pergolide

K P Balachandran, D Stewart, G A Berg, K G Oldroyd

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Constrictive pericarditis is present when a fibrotic, thickened, and adherent pericardium restricts diastolic filling of the heart. Several drugs can cause pericarditis, which can lead to chronic pericardial constriction. A case of constrictive pericarditis in a patient receiving the antiparkinsonian drug pergolide is reported.

CASE REPORT

A 67 year old man with a previous history of Parkinson's disease was admitted in January 1999 with dyspnoea. His past medical history included ischaemic heart disease with single vessel coronary artery bypass surgery (saphenous vein graft to left anterior descending) in 1983. He had been relatively free of angina since. He was in atrial fibrillation and had signs of mild congestive heart failure. He was treated with digoxin and diuretics and anticoagulated. His extrapyramidal symptoms had developed five years before this presentation. He was well controlled initially with a levodopa-carbidopa combination but had deteriorated and required increasing doses. The ergoline dopamine agonist pergolide had been added as adjuvant therapy 11 months before presentation. This had resulted in improved control of symptoms. After discharge he required increasing doses of frusemide to control lower limb oedema. In addition, anginal symptoms appeared to have recurred and a cardiology opinion was sought. On review, he complained of breathlessness with normal daily activities. Ankle oedema was present and the jugular venous pressure was mildly raised. However he was back in sinus rhythm and the digoxin had been discontinued by his general practitioner. The electrocardiogram showed right atrial enlargement, right axis deviation, and left bundle branch block. Chest radiography revealed a normal cardiothoracic ratio and clear lung fields. Echocardiography demonstrated normal left ventricular function, severe right ventricular systolic dysfunction, and thickened pericardium posteriorly. A repeat study four months later showed that the pericardial thickening had increased and the right ventricular systolic function had deteriorated. It was decided to proceed with cardiac catheterisation.

Coronary angiography revealed an occluded left anterior descending artery with a patent saphenous vein graft and no other significant coronary disease. Right heart catheterisation revealed raised right sided pressures (mean right atrial pressure of 16 mm) despite his diuretic regimen. There was clear evidence of diastolic equalisation of right atrial, right ventricular diastolic, pulmonary capillary wedge, and left ventricular diastolic pressure wave forms in a pattern consistent with constrictive pericarditis. During fluoroscopy a significant area of curvilinear calcification was observed around the posterior aspect of the pericardium. The fluoroscopic findings were confirmed by computed tomography of the thorax (fig 1). The patient underwent pericardectomy in November 1999. Inspection of the pericardium revealed a large area of thickening and calcification inferiorly with several areas of soft lique-

Figure 1 Computed tomogram of mediastinum revealing calcification of the posteroinferior surface of the pericardium (arrow).

fied material, possibly old haematoma. There were widespread and dense pericardial adhesions with moderate right ventricular dilatation and normal myocardial contraction. The right ventricle decompressed immediately on opening the pericardium. The thickened pericardium was resected inferiorly and over the left ventricle. The postoperative period was complicated by atrial arrhythmias and left ventricular failure requiring reventilation. Four weeks after discharge the patient was readmitted with increasing dyspnoea. He had bilateral pleural effusions, larger on the right, and 1700 ml of straw coloured fluid was drained. Analysis revealed it to be a transudate and sterile. Echocardiography revealed persistent right ventricular dysfunction and pulmonary hypertension. Left ventricular function remained good. Pergolide was identified as a potential cause of pleural and pericardial effusions and fibrosis and was immediately withdrawn. An alternative non-ergot dopamine agonist pramipexole (Mirapexin) was started. Thereafter the patient's clinical status steadily improved. His diuretics have been withdrawn and there have been no further admissions.

DISCUSSION

Pergolide belongs to the group of ergoline dopamine agonist drugs. Others include bromocriptine, lisuride, and cabergoline. They are used in the later stages of Parkinson's disease and in the medical management of hyperprolactinaemia. Pergolide is substantially more potent than bromocriptine and is an agonist at both the D1 and D2 receptors. These drugs, in addition to the usual side effects associated with dopamine agonist therapy, share some properties with the

Learning points

- Always consider drug therapy as a potential aetiological factor.
- Ergolamine dopamine agonist drugs used in Parkinson's disease—that is, bromocriptine, pergolide, lisuride, cabergoline—are related to methysergide.
- The above drugs are associated with idiosyncratic fibrotic reactions that may lead to constrictive pericarditis.

parent family of ergot compounds, including the ability to induce pleuropulmonary and retroperitoneal fibrosis, erythromyalgia, and digital vasospasm. Retroperitoneal and mediastinal fibrosis is a well known side effect of another ergolamine derivative methysergide. This important side effect is believed to be an idiosyncratic reaction. Shaunak *et al* reported three patients with Parkinson's disease who developed pericardial, pleural, and retroperitoneal fibrosis after treatment with pergolide.¹ Symptoms had emerged on average two years after the institution of treatment and were sufficiently non-specific to cause significant delays in diagnosis. The erythrocyte sedimentation rate was raised in two patients in whom it was measured. Two patients treated with bromocriptine developed constrictive pericarditis 3–4 years after the start of treatment. Pericardectomy was required in both cases and in one of them, pleural effusion recurred seven months after pericardectomy leading to the withdrawal of bromocriptine. In the other patient an episode of mental confusion preoperatively prompted the cessation of bromocriptine.² Similar patterns of pleuropulmonary and pericardial disease have been linked to cabergoline.³ Lund *et al* successfully treated pergolide related retroperitoneal fibrosis in a parkinsonian patient with

replacement of pergolide with the non-ergolamine dopamine agonist ropinirole.⁴ In our patient the symptoms developed one year after the institution of pergolide therapy. A significant delay of six months occurred before the diagnosis was established, partly related to the assumption that the constriction was a late manifestation of previous cardiac surgery. Pergolide was suspected as the aetiological factor only after the readmission with bilateral pleural effusions after pericardectomy. This case emphasises the importance of considering concomitant medication as a cause of constrictive pericarditis.

Authors' affiliations

K P Balachandran, K G Oldroyd, Department of Cardiology, Hairmyres Hospital, East Kilbride
D Stewart, Victoria Infirmary, Glasgow
G A Berg, Western Infirmary, Glasgow

Correspondence to: Dr Keith G Oldroyd, Department of Cardiology, Hairmyres Hospital, Eaglesham Road, East Kilbride G75 8RG, UK; keith.oldroyd@laht.scot.nhs.uk

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