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Valvar aortic stenosis with unusual features

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Summary

This case report documents the co-existence of valvar aortic stenosis and hypertrophic obstructive cardiomyopathy with systemic hypertension and calcific mitral annulus, a combination which has not hitherto been reported. It is the purpose of this paper to help assess the true incidence of the co-existence of aortic stenosis and hypertrophic cardiomyopathy.

Introduction

The co-existence of aortic stenosis and hypertrophic cardiomyopathy (asymmetric septal hypertrophy) has been reported at operation by Ellis, Ongley and Kirklin (1962); after operation by Gordon (1962) and Braunwald *et al.* (1964); at post-mortem examination by Hurst and Logue (1966) who also estimated the prevalence to be 10% of patients with severe aortic valve stenosis. Parker, Kaplan and Connolly (1969), in an attempt to detect subaortic hypertrophy in patients with severe aortic valvar stenosis, discovered ten cases of co-existent aortic valvar stenosis and functional sub-aortic hypertrophy. Nanda *et al.* (1974) found six cases of aortic

valve disease with co-existing idiopathic hypertrophic subaortic stenosis. They regarded the combination to be rare and pointed to the value of echocardiography in the non-invasive diagnosis of this complex situation.

In the absence of aortic valve disease, functional sub-aortic stenosis occurring in the course of systemic hypertension was first documented by Brock (1957).

Case report

Systemic hypertension and aortic stenosis were first detected in a widow aged 67 years when she presented in 1964 with effort dyspnoea and mild angina of recent onset without any previous history of rheumatic fever. Her pregnancies and labour at the ages of 25, 28 and 35 years had been uneventful. Hypertension was controlled with methyldopa and a diuretic. Her symptoms improved and she herself discontinued the drug treatment after a few years. During the last 2 years she had had three syncopal and two near syncopal episodes with increasing effort dyspnoea, angina and relapse of hypertension.

Six months before her present admission, left ventricular failure supervened, since when she had been receiving digoxin and a diuretic. Her blood pressure fluctuated between 150/90 and 180/110 mmHg.

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FIG. 1. Echocardiogram showing dense echoes from the mitral annulus in keeping with calcification of the annulus.

FIG. 2. Echocardiogram showing ventricular septal (32 mm) and posterior left ventricular wall thickness in diastole (22 mm) with a small left ventricular cavity. Internal dimension in systole = 1.5 cm and in diastole 3.0 cm. Ratio of interventricular septal to posterior left ventricular wall thickness = 1.45. Estimated ejection fraction = 87%.

FIG. 3. Withdrawal pressure traces from the body of the left ventricle to aorta using a Cordis No. 7 N.I.H catheter. (a) L.V. apex to outflow tract: L.V. pressure = 260–280/+20 mmHg. Note L.V. alternans and notching halfway up the upstroke of the trace. It also shows post ectopic accentuation of L.V. pressure to 400/20 mmHg; (b) (continuous with trace a) shows a peak systolic gradient of 90–120 mmHg at aortic valve level.

No definite cause for hypertension was found. Propranolol was exhibited in small doses with considerable subjective improvement. Family history was not contributory. She smoked five cigarettes a day and consumed very little alcohol.

On examination, bilateral corneal arcus was evident; arterial pulse was unremarkable; blood pressure 150/90 mmHg; a systolic thrill was palpable over the left lower sternal edge and aortic area; the apical impulse was heaving in nature and the fourth heart sound was palpable. The first heart sound was normal and the rhythm was sinus. A late onset ejection murmur not preceded by a click was heard over the aortic area and left lower sternal edge. Retinoscopy showed no abnormality. Other systems were normal.

Investigations

(a) Chest X-ray showed moderate cardiomegaly with unfolded aorta but without any discernible post-stenotic dilatation of aorta; (b) cardiac fluoroscopy showed calcification of the mitral annulus with no aortic valve calcification; (c) ECG showed sinus rhythm with severe left ventricular hypertrophy and notched 'P' waves in aVL; (d) the phonocardiogram showed the fourth heart sound and late onset ejection murmur not preceded by a click; (e) blood lipid analysis revealed no abnormality; (f) echocardiograms: (1) Mitral echogram showed dense echoes from the region of the mitral annulus with normal mitral valve movements (Fig. 1). (2) Echogram to delineate interventricular septal thickness, left ventricular cavity and posterior left ventricular wall (Fig.

FIG. 4. (a) Left ventriculogram showing obliteration of mid-left ventricular cavity by systolic muscular contraction with division of the cavity into apical and outflow segments. (b) Left ventriculogram showing complete relaxation of muscular obstruction in diastole. The left ventricular wall is thick and the coronary arteries appear normal.

2); (g) left ventricular and aortic pressure traces (Fig. 3); left ventricular angiograms (Fig. 4).

Discussion

The diagnosis of aortic stenosis was based on the history of syncope, angina and effort dyspnoea with signs of a heaving apical impulse, systolic thrill in the aortic area, ejection murmur and electrocardiographic evidence of left ventricular hypertrophy. Severe aortic stenosis at valve level was confirmed by the withdrawal pressure trace from the body of the left ventricle to aorta (Fig. 3). However, the absence of an ejection click, post-stenotic dilatation of the ascending aorta, aortic valve calcification and the presence of systolic thrill over the left lower sternal edge raised the possibility of sub-valvar aortic stenosis. The withdrawal pressure trace excludes a discrete sub-valvar chamber as seen with a sub-aortic diaphragm or muscular ring or bar.

The aortic trace form in patients with co-existing aortic stenosis and hypertrophic cardiomyopathy has not been clearly described so far and needs further clarification. This forms a part of a future study. Despite a significant peak systolic gradient at aortic valve level, the aortic trace is not typical of tight aortic stenosis in this case.

The echocardiogram (Fig. 2) showed inter-ventricular septal to posterior left ventricular wall ratio of 1.45 (normal, 1.3), with a small left ventricular cavity and an ejection fraction of 87%, these features are in keeping with hypertrophic cardiomyopathy. The typical abnormal systolic anterior movement of the anterior mitral leaflet in hypertrophic obstructive cardiomyopathy was not ob-

served in this case presumably because of calcified mitral annulus with possible extension to the base of the leaflets.

The left ventriculogram (Fig. 4a and b) delineated the typical features of hypertrophic obstructive cardiomyopathy in patients with the injection of contrast material under pressure probably acting as a stimulant producing obstruction.

It is important to detect the presence of hypertrophic cardiomyopathy in patients with severe aortic valve disease for cardiac surgery may involve valve replacement with myotomy or myectomy of the muscle-bound left ventricular outflow tract. This may be followed by β -blockade therapy. In this case β -blockade appears to have controlled her symptoms to date and cardiac surgery was not undertaken because of the patient's reluctance.

The occurrence of hypertrophic cardiomyopathy in the course of systemic hypertension has been documented in the past; however, its co-existence with mitral annulus calcification and severe aortic stenosis form the unusual features of this case. Such concomitance raises the possibility of multifactorial pathogenesis of hypertrophic cardiomyopathy.

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Hypocalcaemia and convulsions

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Summary

Hypocalcaemia may manifest with tetany, convulsions and even status epilepticus. Recognition of underlying hypocalcaemia in convulsions is mandatory because the fits may not be adequately controlled by anti-convulsant drugs which may also aggravate hypocalcaemia. Vitamin D, by relieving hypocalcaemia, reduces the frequency of convulsions and may even eliminate them.

Introduction

A sustained fall in the physiologically active ionized calcium of the plasma is due to a failure of the homeostatic mechanisms regulating the plasma calcium within a narrow normal range. Such homeostatic mechanisms involve the integrated actions of parathyroid hormone, vitamin D and possibly calcitonin. Besides parathyroid insufficiency and dietary deficiency of vitamin D, magnesium deficiency, anticonvulsant drugs, chronic renal failure, and steatorrhoea are the other common causes of hypocalcaemia. Hypocalcaemia is a well recognized cause of tetany and convulsions. Anti-convulsant drugs may be advised for such patients which do not control the seizures and may aggravate hypocalcaemia, setting up a vicious cycle necessitating increasing doses of anticonvulsant drugs.

These disturbances of the calcium metabolism are illustrated in three cases now described.

Case reports

Case 1

A 40-year-old male presented with what was diagnosed as a multinodular goitre. This was re-

moved and histopathology revealed follicular carcinoma. Total thyroidectomy was undertaken 3 months later and replacement therapy thyroxine 0.3 mg/day was given. The patient developed paraesthesiae in all four limbs within a week of surgery, for which he was given calcium gluconate tablets orally. Fifteen months after thyroidectomy he complained of failing vision which was attributed to presbyopia but later proved to be a cataract which was successfully removed. The patient had a fit resembling grand mal seizure 2 months later. Since then he has had several convulsive fits which have been treated with phenobarbital. Bilateral papilloedema was noticed 2 months after the first seizure. There was generalized constriction of fields of vision. Electroencephalogram revealed slow waves from the frontal areas. X-ray of the skull, ventriculography and carotid angiography were normal, as was the cerebrospinal fluid. Plasma calcium on two occasions was recorded as 9 mg/100 ml (2.25 mmol/l) and 7 mg/100 ml (1.75 mmol/l) respectively. Possibility of space occupying lesion in brain due to secondaries from thyroid carcinoma was excluded and the patient was treated with phenobarbital (150 mg/day). The patient had more fits while on phenobarbital; these were partly controlled with intravenous calcium gluconate. The patient was referred to the authors 23 months after thyroidectomy. He was depressed and worried because he was to lose his job in view of uncontrolled epilepsy. Chvostek's and Trousseau's signs were positive. There was bilateral cataract. The plasma calcium was 6.6 and 7.0 mg/100 ml (1.65 and 1.75 mmol/l) on two occasions (sp. gr. 1.027); plasma inorganic phosphorus was 4.5 mg/100