

HYPERTROPHIC CARDIOMYOPATHY IN A YOUNG HYPERTENSIVE

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INTRODUCTION

In 1957, Brock¹ described 3 patients with hypertrophic cardiomyopathy of which one of these patients had previously been hypertensive. Brock postulated that obstruction of the left ventricle outflow tract was caused by concentric hypertrophy of the subvalvular region of the left ventricle, which in turn resulted in sustained systemic hypertension. Later in 1970, Hambly² reported 8 cases of hypertension coexisting with hypertrophic cardiomyopathy. We report a similar patient with hypertrophic cardiomyopathy presenting as a young hypertensive.

CASE REPORT

S.Y.C. is a 36 year old lady who was detected to have hypertension at the age of 34. She had no breathlessness, chest pain or syncopal attacks on exertion. There was no family history of hypertension, effort syncope or angina.

Physical examination revealed a 36 year old lady in no distress. Blood pressure was 160/110 mm Hg. and her pulse was 82 beats per minute, regular, with no femoral delay. Carotid pulses were strong and jerky. All peripheral pulses were palpable and equal. The apical impulse was strong at the fifth left intercostal space in the mid-clavicular line. No thrills were present. A grade 2/6 ejection systolic murmur was audible at the left sternal border, radiating to the base and a fourth heart sound was heard.

The electrocardiogram revealed Q waves in V₁ and V₂ with no left ventricular hypertrophy, as

seen in Fig. 1. The chest X-ray showed a normal size heart with no 'shouldering' of the left heart border seen. M-mode echocardiography demonstrated asymmetric septal hypertrophy and systolic anterior motion of the anterior leaflet of mitral valve and narrowed left ventricular cavity, consistent with hypertrophic cardiomyopathy, as illustrated in Fig. 2. There is also a mid systolic closure of the aortic valve as seen in Fig. 3. Carotid pulse recording confirms the mid systolic dip.

Vanillyl mandelic acid (VMA) and catecholamine determinations were normal. An intravenous pyelogram revealed no abnormality. Blood urea, serum electrolytes and creatinine clearance were also normal.

DISCUSSION

The association of hypertension and hypertrophic cardiomyopathy have been noted by several authors^{1,2,3,4,5,6,7,8}

In a report of 8 cases of hypertension with coexisting hypertrophic cardiomyopathy, Hambly² noted that 4 of their patients were young hypertensives. All the 8 patients had hypertension preceding clinical evidence or hypertrophic cardiomyopathy. The main presenting features in these patients were associated cardiac symptoms; changing electrocardiogram with development of Q waves; increasing heart size and evaluation of a new heart murmur.

Some of the investigators^{1,2,8} suggested that the hypertension was aetiologically related to subaortic muscular obstruction. Other causes of left ventricular hypertrophy such as aortic valvular stenosis and coarctation of aorta, may be associated with muscular subaortic stenosis, which clinically presents itself only after surgical correction of the valvular lesion or the coarctation.^{9,10,11}

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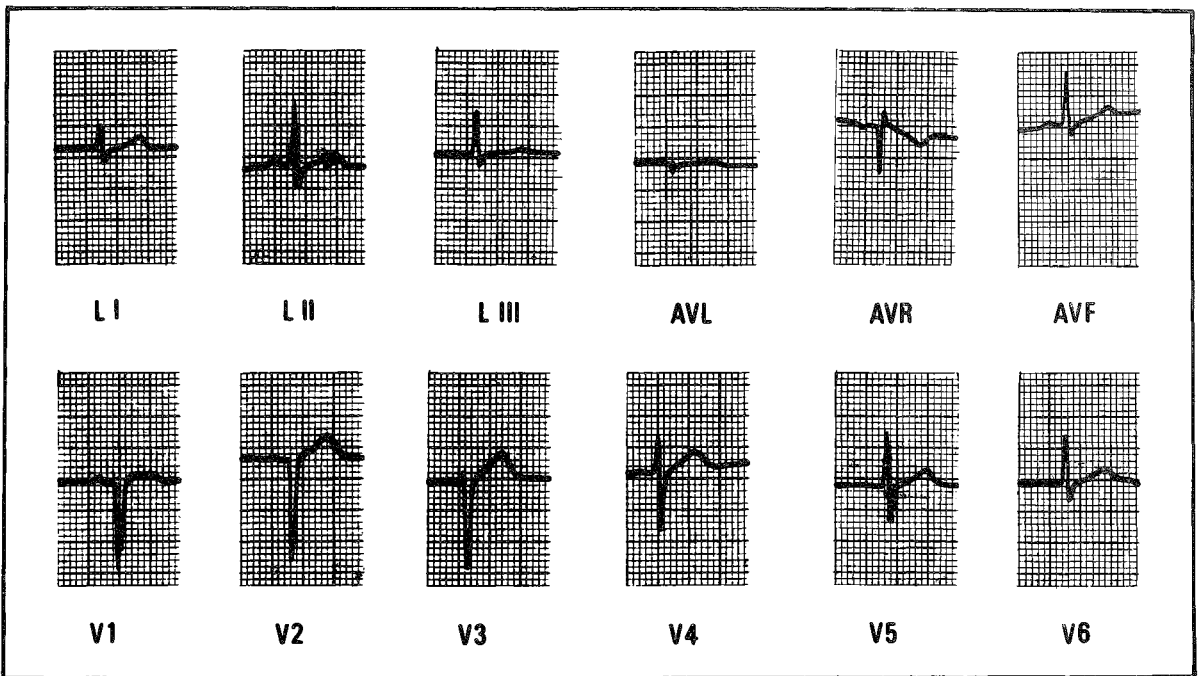


Fig. 1 Electrocardiogram of patient showing Q waves in V_1 and V_2 .

The diagnosis of hypertrophic cardiomyopathy in a patient with hypertension can be difficult unless the blood pressure is lowered.² In a patient treated for hypertension in whom a systolic murmur subsequently develops, hypertrophic cardiomyopathy should be suspected.

Recent studies using M-mode echocardiography have shown a characteristic systolic movement of anterior mitral leaflet in hypertrophic cardiomyopathy.^{12,13,14} However "systolic anterior motion of mitral valve" may be absent in those patients who have no subaortic obstruction at the time of study.¹⁵

In several autopsy series of patients with hypertrophic cardiomyopathy, the ventricular septum has been described as asymmetrically hypertrophied when compared with the left ventricular free wall.^{16,17,18,19} Recently, however, advances in echocardiographic techniques have made it possible to measure the thickness of the septum and left ventricular posterior free wall reliably and noninvasively.^{20,21,22}

Goodwin,²³ noted the difficulty with M-mode echocardiography in differentiating hypertensive heart disease from "non-obstructive hypertrophic cardiomyopathy". However, when obstruction was present, mid systolic closure of the aortic valve and

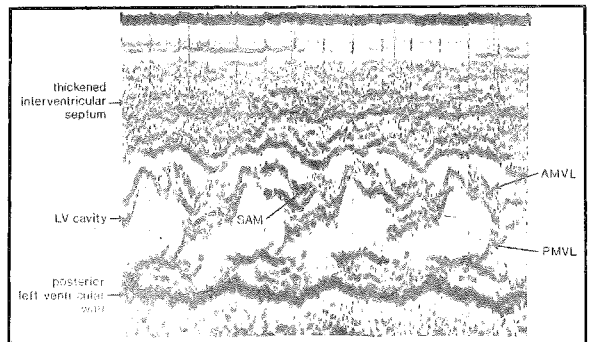


Fig. 2 Echocardiogram demonstrating asymmetric septal hypertrophy, systolic anterior motion of mitral valve (SAM) and narrowed left ventricular cavity.

systolic anterior motion of the mitral valve served to differentiate the two diseases.

The characteristic asymmetric septal hypertrophy, systolic anterior motion of the mitral valve and mid systolic closure of aortic valve are well illustrated in this patient's echocardiograms.

The management of hypertension in patients with hypertrophic cardiomyopathy had been a problem in some cases.² Hambly² noted atypical response of the hypertension to treatment. Despite good control of the hypertension, radiological examination showed the heart had enlarged and

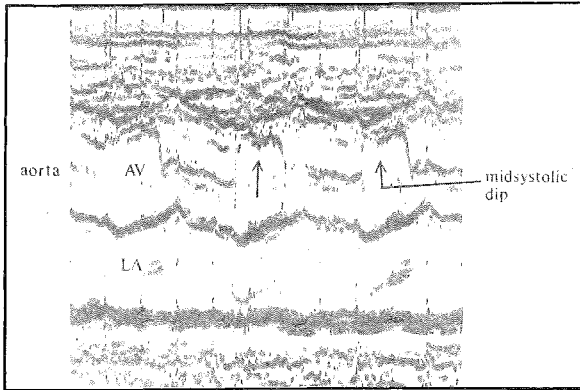


Fig. 3 Echocardiogram illustrating midsystolic dip of aortic valve.

the electrocardiogram revealed left ventricular hypertrophy. Two of the 8 patients were resistant to treatment. Our patient, however, responded fairly satisfactorily to propranolol.

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