Thallium-201 Imaging in a Patient with Mid-Ventricular Hypertrophic Obstructive Cardiomyopathy

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Findings specific to mid-ventricular hypertrophic obstructive cardiomyopathy were obtained in a patient by means of $^{201}$Tl planar myocardial scintigraphy. Namely, a myocardial band-like image dividing the left ventricle into two chambers was clearly shown. This was identified as hypertrophic muscle with sphincter-like muscular stenosis at the mid portion of the left ventricle.


In hypertrophic obstructive cardiomyopathy, a pressure gradient occurring between the left ventricular inflow and outflow tracts resulting from obstruction of the left ventricular outflow tract during systole has commonly been observed.

However, mid-ventricular hypertrophic obstructive cardiomyopathy (MVHOCM) has been noted as a variant type of hypertrophic obstructive cardiomyopathy, since Falicov et al., reported in 1976 on MVHOCM (1) where a pressure gradient was observed between the apex and the inflow and outflow tracts, resulting from an obstruction in the mid portion of the left ventricle.

We report here on a recent case of MVHOCM in which myocardial hypertrophy with muscular stenosis at the mid portion of the left ventricle was revealed by thallium-201 ($^{201}$Tl) myocardial scintigraphy.

CASE REPORT

The patient was a 39-yr-old housewife whose mother suffered from hypertrophic nonobstructive cardiomyopathy. Although she had no symptoms during childhood, she began to feel fatigued at age 33 yr, and cardiomegaly and an abnormal electrocardiogram were noted. From age 36 yr she complained of precordial pain and dizziness, and first visited our hospital in June 1984. Blood pressure was 100/70 mm Hg. There was a grade 3/6 systolic ejection murmur and mild cardiomegaly observed by chest x-ray. The electrocardiogram showed left ventricular hypertrophy and Q wave, ST elevation, and negative T wave change in lead III, and slight ST elevation also in aVF lead. M-mode echocardiography revealed normal thickness of the septum and the posterior wall, and systolic anterior movement (SAM) of the anterior mitral leaflet. Cross-sectional echocardiography demonstrated marked hypertrophy at the apex, along with hypertrophy of papillary muscle. Administration of a beta-blocker improved her symptoms, but since May 1986 ventricular extrasystoles were seen frequently, and precordial pain increased from March 1987. She was admitted on June 10. Chest x-ray on admission revealed slightly increased left ventricular enlargement as compared to the first examination. The electrocardiogram was almost unchanged, except that the duration of the QRS wave was slightly prolonged to 0.10 sec. Echocardiographic findings remained unchanged: marked hypertrophy near the apex, i.e., 20 mm each in the thickness of the septum and the posterior wall, respectively. Apical hypertrophic cardiomyopathy was suggested (Fig. 1). Gated blood-pool scintigraphy demonstrated an hourglass appearance of the left ventricle. A narrow neck connected the basal chamber with the apical chamber at end diastole, but the two chambers were entirely separated at the end systole. The basal chamber contraction was good with an ejection fraction of 60%, but the apical chamber exhibited dyskinesia during systole (Fig. 2).

The site demonstrated as the apex by echocardiography was deemed to correspond to the site of the narrow neck, which evidenced marked hypertrophy by echocardiogram. Accordingly MVHOCM was suspected, but it had to be differentiated from pseudoaneurysm which demonstrates a similar hour-glass shape of the left ventricle. Thallium-201 myo-

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cardiac image demonstrated posterior rotation of cardiac position (Fig. 3), and the 40° left anterior oblique (LAO) image corresponded to the usual anterior view image.

Imaging of the left ventricular cavity was difficult, because of marked muscular hypertrophy, in particular, hypertrophy of the septum and anterior wall, compared to the posterior wall, with increased uptake of 201Tl. In the area of the apex, the uptake of thallium-201 could not be observed on the images of LAO 40°, 60°, and 70°, but was slightly visible from the lateral view. The possibility of pseudoaneurysm was eliminated by the finding of 201Tl uptake in the myocardial wall, as compared with pseudoaneurysm, which is composed of organized hematoma lacking any elements of the myocardial wall.

Myocardial band-like images were intensively depicted, dividing the left ventricle into two chambers on the images of LAO 40°, 60°, 70°, and from the lateral view, suggesting sphincter-like muscular stenosis at the mid portion of the left ventricle.

Left ventricular catheterization showed systolic intraventricular pressure of 177 mmHg in the apex, 95 mmHg in the outflow tract, and 97 mmHg in the aorta, indicating a pressure gradient of ~ 80 mmHg between the apex and the outflow tract and aorta (Fig. 4).

Left ventricular cineangiography revealed systolic cavity obliteration of the ventricle in its mid portion due to muscular hypertrophy (Fig. 5). The basal chamber showed hyperdynamic contraction, while the apical chamber showed dyskinesia. Coronary angiography did not reveal any vascular stenosis lesions.

The patient's symptoms were improved by an increased dose of beta-blocker, and she is still attending our outpatient clinic.

DISCUSSION

Echocardiography is the dominant technique in evaluation of hypertrophic cardiomyopathy. Observation
FIGURE 3
Thallium-201 resting myocardial scintigraphy: Posterior rotation of cardiac position is shown. The 40° left anterior oblique (LAO) image corresponds to the usual anterior view image. Imaging of the left ventricular cavity is difficult, because of marked hypertrophy particularly in the septum and anterior wall. There is perfusion defect in the apex, where no uptake of $^{201}$TI is seen on the images of LAO 40°, 60°, and 70°. Severely reduced uptake in the apex is observed, however, from the lateral view. Myocardial band-like images in the mid ventricular portion are shown dividing the left ventricle into two chambers on the images of LAO 40°, 60°, 70°, and lateral view, suggesting sphincter-like muscular stenosis at the mid portion of the left ventricle.

of all cross-sectional images of the left ventricle, however, is not possible due to direction restrictions of the ultrasonic beam. Therefore, information about the apical region, and the middle and lower septum can not be easily obtained. It is also difficult with echocardiography to evaluate myocardial tissue characteristics such as fibrosis coexisting with myocardial hypertrophy.

On the other hand, thallium imaging is useful in evaluating hypertrophy from the increased myocardial mass suggested by increased thallium uptake. The clearly demarcated thallium perfusion defect could be judged to be myocardial fibrosis, and tissue characterization of the myocardium might also be possible to some extent. A further advantage is that the entire myocardial image of the left ventricle can be observed.

In the present case, the limitations of echocardiography were demonstrated. More valuable information was obtained by $^{201}$TI myocardial imaging and cardiac blood-pool imaging. In particular, $^{201}$TI myocardial imaging revealed the hypertrophic muscle mass with severe sphincter-like narrowing in the mid portion of the left ventricle, a finding specific to MVHOCM. Thus, $^{201}$TI myocardial imaging could be extremely useful in the noninvasive diagnosis of MVHOCM.

No other cases of imaging hypertrophic muscle mass exhibiting muscular stenosis in MVHOCM have been reported.

The hourglass appearance of the left ventricle demonstrated by gated blood-pool scintigraphy had to be distinguished from pseudoaneurysm (2–5). Thallium-201 myocardial imaging revealed myocardial thallium activity in the left ventricular apex and suggested fibro-

FIGURE 4
LV pressure recordings: Left ventricular catheterisation shows a pressure gradient of −80 mmHg between the apex and the outflow tract and the aorta: systolic pressure of 177 mmHg in the apex, and 95 mmHg in the outflow tract and 97 mmHg in the aorta.
sis in the apex with severely reduced uptake. Such scintigraphic findings were conclusive in establishing that it was not a pseudoaneurysm.

Wigle et al., asserted the possibility that mid-ventricular obstruction might result from nonobstructive hypertrophic cardiomyopathy combined with apical infarction, as is frequently observed when apical aneurysm accompanies MVHOCM (6). In the present case, however, $^{201}$TI myocardial imaging demonstrated hypertrophic muscle mass in the mid-ventricular portion dividing the left ventricle into two chambers. It is suggested that sphincter-like muscular stenosis caused mid-ventricular obstruction and markedly increased apical intraventricular pressure leading to fibrosis and aneurysm formation in the apex.

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