Combined Apical Hypertrophic Cardiomyopathy and Left Atrial Myxoma*

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A patient had apical hypertrophic cardiomyopathy and left atrial myxoma. We believe that this is the first description of such a combination. (Chest 1992; 101:1149-50)

**HOCM** = hypertrophic obstructive cardiomyopathy; **IHSS** = idiopathic hypertrophic subaortic stenosis

Tumors of the heart are uncommon. With the advent of echocardiography, intracardiac tumors are being found more frequently and myxomas are the most frequent benign tumors. To our knowledge, however, association of a myxoma with apical hypertrophic cardiomyopathy has not been described.

**CASE REPORT**

A 67-year-old man was referred to this hospital for further evaluation of his cardiac status and therapy. He had once had a short period of treatment for hypertension many years before. Five years previously he had had a syncopal attack of about 10-min duration while standing at work and he experienced the same episode five times in the following year but none since. However, for two months prior to his hospital admission, he had had dyspnea on exertion and easy fatigability.

Medical history was noncontributory except for hypertension. Family history revealed no sudden death, cutaneous pigmentation, or endocrine disease.

Physical examination revealed only a soft, short rumbling diastolic murmur at the apex in the left lateral decubitus position. The loudness of the murmur fluctuated daily, and sometimes it was not audible. The blood pressure was 130/70 mm Hg and the heart rate was regular at a rate of 60 beats per minute. Results of the remainder of the physical examination were normal.

The electrocardiogram (ECG) revealed sinus rhythm and left ventricular hypertrophy (R wave in lead V1 = 6.4 mV), with giant negative T waves (depth in lead V4 = 1.6 mV) in the left precordial leads. Two-dimensional echocardiography showed a 35- by 45-mm space-occupying lesion in the left atrium originating from the atrial septum (Fig 1). The tumor did not protrude beyond the mitral orifice during diastole. The left ventricular wall mid and distal ventricular septum and apical myocardium were thickened. The left ventricular diastolic dimension was 50 mm and the walls were hyperkinetic. Systolic anterior movement of the anterior mitral leaflet was not noted.

Magnetic resonance imaging of the heart confirmed the findings shown by the echocardiograms (Fig 2). Cardiac catheterization revealed normal pulmonary artery and pulmonary artery capillary wedge pressures. There was no pressure gradient at rest between aorta and left ventricle. The left ventricular end-diastolic pressure was 22 mm Hg. Therefore, the diagnosis of apical hypertrophic cardiomyopathy associated with probable left atrial myxoma was made.

The patient underwent surgery to remove the left atrial tumor and a 45 x 35 x 30-mm pedunculated tumor of gelatinous consistency was found. Histologic examination confirmed the diagnosis of left atrial myxoma, and the patient had an uneventful recovery. A biopsy specimen of the left ventricular apex showed bizarre-shaped hypertrophied and bizarre arrangement (disarray) of myocardial muscle cells.

**DISCUSSION**

Hypertrophic cardiomyopathy, especially idiopathic hypertrophic subaortic stenosis (IHSS), or hypertrophic obstructive cardiomyopathy (HOCM), has been described in association with various congenital cardiac anomalies such as coronary arteriovenous fistula and a syndrome with somatic malformations. However, the only link between hypertrophic cardiomyopathy and left atrial myxoma is through lentigiosis. Familial lentigiosis has been reported to be associated with HOCM, and the term "progressive cardiomyopathic lentigiosis" has been proposed for the syndrome by Polani and Moyahan. Rees et al described a case of lentigiosis associated with left atrial myxoma but their case was not associated with a hypertrophic cardiomyopathy. We could find no literature describing an association of left atrial myxomas with other conditions. In this case, the patient stated that none of his family members or primary relatives were known to have either a myxoma, the appearance of spotty pigmentation, or any endocrine disease.

Our patient's history of hypertension merits discussion concerning the connection between hypertrophic cardiomyopathy and hypertension. Hypertensive patients often show echocardiographic features consistent with a hypertrophic cardiomyopathy that may be obstructive, nonobstructive, or apical. In our case, however, although the patient had a history of treated hypertension, he was normotensive at the time of hospital admission and had been without any antihypertensive therapy for many years. The high voltage

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in the left precordial leads (V1 and V6) with giant negative T waves strongly suggests that his apical hypertrophic cardiomyopathy is primary with hypertension playing only a small role toward the development of his left ventricular hypertrophy. Although this is the first description of a patient with apical hypertrophic cardiomyopathy associated with a left atrial myxoma, it is the second report of hypertrophic cardiomyopathy and left atrial myxoma.11 The coexistence may be more than a chance occurrence so that the future of left atrial myxoma should be ruled out when one sees a patient with a hypertrophic cardiomyopathy.

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Spontaneous Pulmonary Hemorrhage Following Coronary Thrombolysis

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Excessive bleeding is a major concern during the administration of thrombolytic therapy. Although the great majority of these events occur at sites of vascular interruption, major gastrointestinal, retroperitoneal, genitourinary, and central nervous system hemorrhage are known to occur. We present a patient who developed spontaneous pulmonary hemorrhage during thrombolytic therapy. Lack of recognition that the lungs too may be a site of spontaneous hemorrhage during thrombolytic therapy may lead to a considerable diagnostic and therapeutic delay. Pulmonary hemorrhage should be considered in the differential diagnosis of patients who receive thrombolytic therapy in whom new roentgenographic pulmonary infiltrates present accompanied by decreases in hematocrit value. (Chest 1992; 101: 1150-52)

Coronary thrombolysis, has become a standard mode of therapy for early treatment of acute myocardial infarction, and bleeding is a well described complication of this therapy. Although most of these events occur at vascular puncture sites, severe hemorrhage from gastrointestinal, retroperitoneal, central nervous system, and genitourinary sites are known to occur. The following report describes a patient who received thrombolytic therapy for myocardial infarction, and subsequently developed new bilateral pulmonary infiltrates, accompanied by a dropping hematocrit value and respiratory embarrassment. Clinical, laboratory, and roentgenographic data indicated pulmonary hemorrhage.

CASE REPORT

A 52-year-old man presented to St. Vincent’s Hospital with an acute anterior wall myocardial infarction. Chest pain had begun approximately one half hour prior to presentation. Physical examination demonstrated blood pressure of 130/80 mm Hg, pulse rate of 84 beats per minute, and respiratory rate of 16 per minute. Chest examination revealed clear heart sounds, an atrial gallop, and no murmurs or rubs. Rales were heard at both lung bases. The ECG was significant for an acute anterolateral wall myocardial infarction, sinus rhythm, and a right bundle branch block pattern. Approximately 1 h after presentation, nitroglycerine,

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