Recurrent Atrial Flutter in Apical Hypertrophic Cardiomyopathy

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SUMMARY

Palpitations are a symptom often reported by patients with apical hypertrophic cardiomyopathy (HCM), yet the arrhythmias associated with this type of HCM have not been studied adequately. Herein, a case of persistently recurrent atrial flutter in a 63-year-old Greek man with apical HCM is presented. Synchronized direct-current shocks were used twice during his hospitalization in order to convert atrial flutter to sinus rhythm. No definite precipitating factor for the induction of atrial flutter was identified.

Key Words:

Atrial flutter Hypertrophic cardiomyopathy Arrhythmias

SINCE Yamaguchi et al¹⁾ first described apical hypertrophic cardiomyopathy (HCM) in Japanese patients, reports of this unique form of non-obstructive HCM in patients outside of Japan have appeared in the literature.^{2),3)} However, in contrast to other forms of HCM, there have been very few reports of the arrhythmias associated with this particular variant of HCM.⁴⁾ We describe the first reported case of recurrent atrial flutter in a patient with apical HCM.

CASE REPORT

A 63-year-old Greek man was admitted to the hospital because of palpitations which he had been feeling for 15 days. The electrocardiogram (ECG) showed atrial flutter with 2:1, 3:1, and 4:1 atrioventricular (AV) conduction (Fig. 1A). Episodes of atrial flutter, of 2 hours to 6 days duration, had

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been reported during the previous 6 years. Their mean rate was 1 episode per month in spite of the antiarrhythmic drugs, of all the known classes, that the patient had received. His last medications were digoxin, verapamil, and

Fig. 1. A: ECG showing type I atrial flutter (atrial rate is about 300 beats/min). B: ECG with sinus rhythm and inverted T waves predominantly in leads V₄₋₆. Maximum depth of the T waves in V₄ is 6 mm. R wave in lead V₅ is 23 mm and the SV₁+RV₅ value is 33 mm.

propafenone. Symptoms other than palpitations were not reported. There was no history of arterial hypertension. On examination, blood pressure was 115/80 mmHg. Cardiac auscultation revealed variable intensity of the first heart sound and a 2/6 systolic ejection murmur. Chest x rays, and blood chemistry, except for cholesterol (280 mg%), as well as thyroid function tests were within normal limits. The 24-hour ECG recording showed atrial flutter with 1:1 to 5:1 AV conduction. Atrial flutter was converted to sinus rhythm using a synchronized direct-current (DC) shock of 50 joules. The ECG showed moderately negative T waves and marginally high QRS voltage in the precordial leads (Fig. 1B). Afterwards, the patient was given amiodarone as an intravenous loading dose for 4 days and then continued orally. Transthoracic and transesophageal two-dimensional echocardiography demonstrated apical hypertrophy of the left ventricle (Fig. 2). Left atrial dimension was 4.17 cm. Other echocardiographic and Doppler findings were normal. Cardiac catheterization and angiography revealed normal coronary arteries, normal intracardiac pressures (left ventricular end-diastolic pressure was 14 mmHg) and a left ventriculogram with a typical "spade-like" configuration at end-diastole and vigorous systolic contraction (Fig. 3). Ejection fraction was calculated to be 60%. Ten days after cardioversion, atrial flutter recurred. Rapid atrial pacing was unsuccessful and a second 50-joule DC shock, under a temporary pacemaker stand-by, restored sinus rhythm. The patient was discharged, receiving amiodarone and oral anticoagulants. Oral anticoagulation was administered throughout the patient's hospitalization.

Fig. 2. Diastolic (left) and systolic (right) transesophageal two-dimensional echocardiographic views of the left ventricle (LV) and left atrium (LA). Apical hypertrophy (H) is depicted.

Fig. 3. End-diastolic (top) and end-systolic (bottom) left ventriculogram in right anterior oblique projection. A "spade-like" end-diastolic appearance and a vigorous systolic contraction are apparent.

DISCUSSION

Although palpitations are a symptom often reported by patients with apical HCM,¹⁾⁻³⁾ the arrhythmias associated with this type of HCM have not been studied adequately. Hayano et al⁴⁾ described a case of Lown-Ganong-Levine syndrome and supraventricular tachycardia in a patient with apical HCM. To our knowledge, no report of atrial flutter in association with apical HCM has been previously published. Supraventricular arrhythmias have been reported in conjunction with HCM, yet atrial flutter is not a common finding even in these patients.^{5),6)} Atrial flutter itself is relatively uncommon. Furthermore, no definite causal relationship between arrhythmias and HCM has been identified.⁷⁾ Similarly, no particular precipitating factor for the induction of atrial flutter was detected in our case, except for the potential contribution of the mild left atrial enlargement.

Persistently recurring atrial flutter, as in our case, is usually associated with underlying heart disease such as cardiomyopathy and coincidental occurrence (and reoccurrence?) of the arrhythmia is not very likely, although it is difficult to exclude it.⁸⁾ Mild left atrial enlargement could be considered a potential contributor to the induction of the arrhythmia but not a substrate for it since the atrial flutter reentrant circuit is located predominantly in the right atrium.^{9),10)} However, the common form of atrial flutter, i.e. with negative atrial waves in the inferior leads and positive waves in aV_R and aV_L, and with continuous undulation of the baseline, as was the type I atrial flutter in this case, supposedly originates in the low part of one of the atria.^{11),12)} Besides, the right atrium was not enlarged in this case. Finally, as for the particular type of apical HCM in our Greek patient, "non-Japanese type" ECG features coexisted with "Japanese type" angiographic ones, implying that such subclassification of apical HCM is not strictly applicable.³⁾

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