A 37-year-old Chinese professional soccer player with an abnormal electrocardiogram (FIGURE 1) required ankle surgery. He had had an isolated syncopal episode while intensely training a year ago, but his medical history was otherwise unremarkable.

On examination, he appeared fit. His vital signs were normal. The apical pulse was sustained on palpation and was not displaced. Auscultation revealed an S4 heart sound.

Transthoracic echocardiography revealed apical hypertrophic cardiomyopathy (FIGURES 2 AND 3) and absence of regional wall-motion abnormalities. He underwent uneventful ankle surgery.

FIGURE 1. In this patient with apical hypertrophic cardiomyopathy, electrocardiography shows a loss of septal Q waves, high QRS voltage, and repolarization abnormalities with deep T-wave inversion, especially in lead V6 (arrow). This is particularly reflective of an apical electrical cardiac event. Such abnormalities are also evident in the neighboring leads V2 and V4.

THE CLINICAL PICTURE

Hypertrophic cardiomyopathy apical variant

The prevalence of hypertrophic cardiomyopathy is 0.2% to 0.5%; the apical variant constitutes about 15% to 25% of cases in Chinese and Japanese cohorts and about 3% of cases in American cohorts.1–3

In typical hypertrophic cardiomyopathy, the left ventricle, especially the interventricular septum, is thickened, but the left ventricular chamber size is normal or small. In severe cases, the left ventricular outflow tract can become very narrowed, resulting in accelerated blood flow, which may further increase in the presence of hypovolemia, peripheral vasodilation, and increased cardiac contractility. The Venturi effect thus created may entrain a typically malformed anterior mitral valve leaf-
HYPERTROPHIC CARDIOMYOPATHY

Parasternal short-axis views

Mitral valve commissure  Papillary muscles  Apex

![Parasternal short-axis views](image)

FIGURE 2. On transthoracic echocardiography, parasternal short-axis views show gross hypertrophy of the apex with relatively normal thickness of the left ventricle (LV) at the level of the mitral valve. The apex is so hypertrophied that it is obliterated at end-systole. This does not occur at the mitral level in the apical variant of hypertrophic cardiomyopathy.

Recent data challenge the conventional wisdom that the apical variant has a benign prognosis. In patients without a history of recurrent syncope, chest pain, or heart failure, perioperative risk is probably not significantly increased. The differential diagnosis includes myocardial ischemia or infarction, electrolyte disturbances, effects of drugs (eg, digoxin), and subarachnoid hemorrhage. Plain or contrast-enhanced echocardiography or cardiac magnetic resonance imaging, or both, can help confirm the diagnosis. Long-term management should be guided by the patient’s symptoms.
FIGURE 3. On transthoracic echocardiography, the apical four-chamber view (left) shows apical hypertrophy. The subcostal four-chamber view (right) shows apical hypertrophy only at the left ventricular apex, with the classic “spade” sign visible in the left ventricle (LV).

REFERENCES


ADDRESS: Anthony M.-H. Ho, MD, FRCP, FCCP, Department of Anesthesiology and Perioperative Medicine, Victory 2, Kingston General Hospital, 76 Stuart Street, Kingston, ON, Canada, K7L 2V7; e-mail: hoamh@hotmail.com