
REPLY

Although we appreciate the interest of Dr. Abinader concerning our study on apical hypertrophic cardiomyopathy (A-HCM), (1) we would like to address the issues he has raised.

First, it is to be recognized that the initial reference to A-HCM was that of Sakamoto et al. (2) in 1976, not Yamaguchi et al. in 1979 (3), as stated by Dr. Abinader. According to Sakamoto, who recently reviewed his experience with 126 patients, A-HCM is quite often associated with giant T-wave negativity on the left precordial leads (4). However, giant T-wave is not necessary for the diagnosis, which is based on “thickening of the myocardium of unknown cause localized and confined to the ventricular apex” as detected by any one of available imaging techniques (4). This definition was used in our study. All of our patients had the thickest myocardium in the apical region of the left ventricle, with some degree of septal hypertrophy in 16 patients, as demonstrated by echocardiography or magnetic resonance imaging (MRI). In that respect, our patients do not resemble the Maron et al. cases (5), but are identical to those described by Japanese investigators (2, 4).

It is well recognized that A-HCM occurs in non-Japanese patients (6–8) and represents about 7% of the HCM population in our center (1). Dr. Abinader refers to his experience of 11 cases followed for 5 to 20 years, which is similar to ours, dealing with 105 cases followed for 13.6 ± 8.3 years. We are well aware that patients with A-HCM infarct their apex and may develop mid-ventricular obstruction and apical aneurysm formation. In our series, 4 of the 11 patients with myocardial infarction (MI) and normal coronary arteries developed apical aneurysm; in one patient a large aneurysm was surgically resected for management of recurrent ventricular tachycardia. We did not observe a significant mid-ventricular obstruction in any of these patients. Patients with apical aneurysms all developed Q-waves, a significant decrease in R-wave amplitudes and had less T-wave negativity after infarction. None of the patients who did not have MI or who did not develop an apical aneurysm had Q-waves. We did not analyze changes in R-wave amplitudes in our study.

We do not agree with Dr. Abinader’s statement that long-term studies are lacking due to the rarity of true A-HCM outside Japan. Our long-term results based on 105 patients followed for up to 32 years indicate that cardiovascular mortality of 0.1% per year in A-HCM is much lower than in other forms of HCM. Therefore, the prognosis with regard to mortality is benign. However, the prognosis is not so benign with regard to cardiovascular morbidity, as 30% of patients develop serious morbidity events such as MI, arrhythmias, and strokes. We have established risk stratification to identify the patients most likely to develop these complications. Finally, we agree with Dr. Abinader that, because of significant morbidity, A-HCM patients should continue to be followed closely by cardiologists with special interest in HCM, and that further genetic studies of this form of HCM are needed.

Interaction of Herbal Drugs With Digoxin

I enjoyed reading the recent study by Valli and Giardina (1) on drug interactions of herbal therapies with cardiovascular effects. It was quite comprehensive. However, I would like to make some additional comments on the interaction of herbal drugs with digoxin.

Numerous herbs containing cardiac glycosides have been identified as containing digoxin-like substances, including milkweed, lily of the valley, Siberian ginseng, and hawthorne berries (2). Ginseng may falsely elevate digoxin levels (2). A Chinese herb called kushen (川芎), has digoxin-like properties (3). Another Chinese medicine, which is very popular in Japan (4) and is called kyoishin (歸脾 "to save the heart"), has been documented to crossreact with digoxin assays (5,6). It was determined that one