EDITORIAL

Identifying a Previous Myocardial Infarction in Patients With Hypertensive Heart Disease: Lights and Shadows

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Electrocardiography and echocardiography are widely used in hypertensive patients with the aim of identifying left-ventricular hypertrophy and its serial changes, because such changes have important prognostic implications. Detection of left-ventricular hypertrophy is, however, only one of the many applications of these diagnostic techniques. In this issue of the Journal, Cicala et al address an important related issue (ie, the power of electrocardiography and echocardiography to detect a previous clinically symptomatic myocardial infarction in a cohort of hypertensive patients with left-ventricular hypertrophy). In this analysis of the echocardiographic study of the Losartan Intervention For Endpoint reduction (LIFE) study, the appearance of new Q waves on the surface electrocardiogram had a 29% sensitivity and 95% specificity, whereas the presence of new or worsened wall motion abnormalities on the echocardiogram had a higher sensitivity (68%) with a lower specificity (84%). In contrast, development of repolarization abnormalities did not correlate with the previous occurrence of myocardial infarction. Both methods provided valuable information in this regard, but the contribution of echocardiography appeared to be somewhat greater than that of electrocardiography.

The ability of electrocardiography and echocardiography to identify a previous myocardial infarction had been assessed before in a number of studies. However, data of the present study are both novel and clinically relevant under at least two viewpoints. This is the first attempt to quantify the accuracy of the electrocardiogram and echocardiogram in identifying a previous infarction in a cohort of subjects with left-ventricular hypertrophy, which can have a masking effect on both pathologic Q waves and wall motion abnormalities. The lack of a relationship between newly developed repolarization abnormalities and previous myocardial infarction in the specific setting of electrocardiographic left-ventricular hypertrophy is in part an expected finding, as repolarization changes in those patients are often due to ventricular strain rather than to ischemia, and the two patterns are not always easy to distinguish. Second, the LIFE study was carried out in the US, the United Kingdom, and the Nordic countries between 1995 and 2001, in an era of widespread use of thrombolysis and percutaneous revascularization for acute coronary syndromes. This is expected to reduce the sensitivity of a subsequent electrocardiographic or echocardiographic examination, given that prompt revascularization may attenuate the electric and functional consequences of a myocardial infarction. In this context, the findings of this study suggest that, even in the revascularization era, electrocardiography and echocardiography are useful in identifying previous clinical myocardial infarctions also among people with hypertensive left-ventricular hypertrophy.

The very low positive predictive values of electrocardiogram and echocardiogram in the study by Cicala et al are also worth commenting. Despite having a 95% specificity, the appearance of new Q waves did not correspond to a previous clinical diagnosis of acute myocardial infarction in 46 of 56 cases (82%, or 18% positive predictive value). Similarly, 88% of the new or worsened echocardiographic wall motion abnormalities (129 of 146 cases) were not preceded by a clinically recognized infarction. Individuals with left-ventricular hypertrophy are at increased risk of silent myocardial ischemia and unrecognized myocardial infarction. Taken together, these data suggest that silent myocardial infarction might be particularly common among patients with hypertensive left-ventricular hypertrophy.

What is the utility of performing repeated electrocardiographic and echocardiographic examinations in hypertensive patients? The low number of clinically recognized infarctions in the present study (35 for the electrocardiographic analysis and as low as 25 for the echocardiographic analysis) limits the precision of the estimates. Nevertheless, serial electrocardiographic examinations might be expected to pick up one unrecognized myocardial infarction per 100 patient-years in hypertensive patients with left-ventricular hypertrophy (46 events in 902
patients followed for 5 years). Even more notably, echocardiography is able to identify about 3 silent infarctions per 100 patient-years. Although some of these unrecognized myocardial infarctions may be false-positive findings, there is evidence that unrecognized infarctions are as likely as recognized ones to cause death or heart failure. The clinical implications of these findings are straightforward. Electrocardiography and echocardiography still remain simple and powerful, albeit defective, tools for identifying silent myocardial infarctions and improving risk stratification in patients with hypertensive heart disease.

References


