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REPLY: Adjustments of Electrocardiographic Criteria for the Diagnosis of Left Ventricular Hypertrophy



We appreciate Drs. Noubiap and Jingi for their interest in our recently published electrocardiographic (ECG) criteria for the diagnosis of left ventricular hypertrophy (1). They correctly pointed out that the diagnostic accuracy of the electrocardiogram varies within each patient depending on sex, age, and body mass index (BMI) (2). The Peguero-Lo Presti ECG criteria was adjusted for sex but not for age or BMI. In addition, other factors such as ethnicity, anthropometric differences, conduction abnormalities, cardiac fibrosis, type of hypertrophy, and lung pathology may attenuate the reproducibility and accuracy of the test (3). These inherent limitations of the electrocardiogram can be improved by adjusting the ECG voltage criteria in each individual patient.

In fact, several ECG left ventricular hypertrophy criteria have been published to accommodate such limitations, with improvement in accuracy (4). Nonetheless, we believe that adjusting for each individual characteristic is impractical and time consuming, particularly for a screening test. As an example, the sex-specific Cornell voltage criteria are more used than the BMI-adjusted version (3,4). Similarly, other combined criteria such as the Cornell product, the Romhilt-Estes score, and the Perugia score have been shown to improve accuracy but are far less used because of their complexity (5).

The Peguero-Lo Presti criteria was proposed to improve the diagnostic accuracy of the ECG while keeping its practicality as a rapid and easy tool for the everyday clinician. We believe, however, that our initial study needs to be expanded to answer more specific questions. We are currently in the process of assessing further the accuracy of these novel criteria after adjustment for the aforementioned variables.

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Rheumatoid Arthritis and Risk of Nonischemic Heart Failure



Closely Related Common Soil

After several literature reports on the association between heart failure (HF) and increased risk of cancer, a study on a large sample of patients with rheumatoid arthritis (RA) focused on risk of ischemic and nonischemic HF (1). The paper by Mantel et al. (1) from the Karolinska Institute in Stockholm sheds light on several aspects of the stated association between Swedish cohorts of nearly 46,000 RA patients (approximately 75% of patients were women) compared with matched general population. The authors found: 1) an increased risk of HF overall, regardless of the presence of ischemic heart disease; 2) that this could not be accounted for by cardiovascular and other comorbidities in RA; 3) the increased risk of HF did not precede RA onset, but rather emerged thereafter and was associated with high activity of RA; and 4) the increased risk was pronounced early for nonischemic HF. Mantel et al. (1) emphasized the clinical awareness and adequate assessment

of HF in patients with RA and called for exploration of the mechanisms by which inflammation and related factors contribute to cardiac decompensation.

Findings in this study with respect to HF, especially the link between high inflammatory RA activity and HF development, along with those on the association of HF with cancer, clearly suggest that inflammation may mediate via endothelial dysfunction and myocardial response pathways to HF. Added to the proinflammatory state, evidence exists for involvement of autoimmune activation in this process. Noteworthy is that a precipitous decline in serum levels of total and low-density lipoprotein cholesterol had been reported to precede the incidence of clinical RA in the experience of the Mayo Clinic (2), which, as shown in a meta-analysis, has a strong parallel to low lipoprotein(a) predicting type 2 diabetes (3). Cross-sectional case-control studies have shown an association between elevated levels of serum concentrations of β 2-glycoprotein I-lipoprotein(a) complexes and the presence of RA. Excess cardiovascular risk in RA also seems to be followed by immune processes typically illustrated by positivity of rheumatoid factor (RF), such that the risk of HF is known to be substantially higher among RF-positive patients with RA than among those who are RF-negative. Furthermore, renal “hyperfiltrators,” broadly congruent with having low serum creatinine levels, represent individuals with autoimmune activation who are actually at significantly higher risk of death and HF (4).

These considerations, collectively, support our hypothesis of enhanced low-grade inflammation and

autoimmune activation forming a unified underlying mechanism for diverse chronic diseases, including RA and HF (5). Further longitudinal studies are warranted to better identify the link between oxidative stress and incident RA and HF.

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