ESC HEART FAILURE
ESC Heart Failure 2020; **7**: 1997–1998
Published online 21 May 2020 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/ehf2.12753

LETTER TO THE EDITOR

Unravel the genetic background of noncompaction before relating it with myocardial hypoperfusion

With interest, we read the article by Cerar *et al*. about myocardial perfusion in left ventricular hypertrabeculation/noncompaction (LVHT).[1] They measured left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) by echocardiography and looked for myocardial ischemia by single-photon emission computed tomography (SPECT) at rest and on stress. Myocardial ischemia, defined as summed difference score ≥2 between rest and stress, was found in 11/41 patients. These 11 patients had lower LVEF, lower GLS and higher N-terminal pro-B-type natriuretic peptide levels than the remaining 30.[1]

We have the following questions and concerns:

In contrast to the statement that 'cardiomyocytes in non-compacted areas are therefore nourished only by diffusion from the ventricular cavity, leading to intramural perfusion' histological investigations show that trabeculations in LVHT contain normal coronary vessels.[2,3]

We scrutinize the sentence 'no studies have shown significant improvement by guideline-based heart failure therapy'. Although there are no prospective randomized trials investigating pharmacotherapy or device therapy, it has been shown that beta-adrenoceptor blockade in LVHT may induce reverse remodelling.[4] Cardiac resynchronization therapy (CRT) may provide beneficial effects in heart failure.[5]

LVHT may be associated with coronary artery disease (CAD). How was CAD excluded in the investigated patients? Did they undergo coronary angiography?

According to which echocardiographic criteria were LVHT diagnosed? The patients underwent cardiac magnetic resonance imaging (CMRI), but we miss CMRI findings in the results, especially if there was any congruence in areas of late gadolinium enhancement by CMRI and ischemia by SPECT. Was there any congruence between the segments with perfusion abnormalities and location of the trabeculations?

Among the ECG findings, only the prevalence of sinus rhythm is reported. The prevalence of Q-waves, repolarization abnormalities, and bundle branch block and their association with perfusion abnormalities would be of interest. In Table 1, it is indicated that 30/41 patients had an implantable cardioverter/defibrillator; however, the rate of patients with CRT devices is not given.

LVHT is frequently associated with neuromuscular disorders (NMD). NMD affect prognosis of LVHT patients.[6] Thus, it would be interesting to know how many patients of Cerar *et al*. were investigated by a neurologist and how many suffered from NMD and if there were differences in the perfusion abnormalities. Furthermore, LVHT is associated with >100 genetic defects.[7,8] We should know which of the known LVHT genes were mutated in the included patients.

Interpretation and relevance of the interesting results of Cerar *et al*. would be more yielding if more clinical and genetic data would be provided.

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DOI: 10.1002/ehf2.12753