

## Pathophysiology of Non-compaction Remains Enigmatic

To the Editor,

With interest, we read the article by Kiss et al<sup>1</sup> about cardiac magnetic resonance imaging parameters in 81 patients with left ventricular non-compaction, also known as left ventricular hypertrabeculation (LVHT), but with normal left ventricular (LV) indices.<sup>1</sup> Left ventricular hypertrabeculation patients nonetheless had increased LV volume, LV mass, reduced LV ejection fraction, and reduced global circumferential strain.<sup>1</sup> It was concluded that LV functional and strain characteristics of the LVHT cohort differ<sup>2</sup> significantly from healthy controls.<sup>1</sup> Since the LV muscle mass was larger in men as compared to females, gender-specific diagnostic criteria are warranted.<sup>1</sup> We have the following comments and concerns.

Left ventricular hypertrabeculation can be complicated by heart failure, intraventricular thrombus formation, and ventricular arrhythmias.<sup>2</sup> We should be told how many of the included patients had elevated pro-brain natriuretic peptide values, a history of thromboembolism, and a history of syncope. It should be also mentioned in how many patients intraventricular or inter-trabecular thrombi were detected.

Missing is the data of the current medication that LVHT patients were regularly taking. Cardiac medication may influence global circumferential strain. We should also know how many patients were anticoagulated because of atrial fibrillation, how many required antiarrhythmics, and how many had heart failure treatment.

Since LVHT can be complicated by ventricular arrhythmias, some patients may require device treatment. Thus, we should be informed how many carried an implanted cardioverter defibrillator (ICD), how many had a cardiac re-synchronization therapy (CRT) system, how many had an ICD with a CRT system, and how many had an ICD with a pacemaker.

Since LVHT is frequently associated with genetic disorders,<sup>3</sup> we should be informed about the genetic background of the included patients. Left ventricular hypertrabeculation is also frequently associated with neuromuscular disorders (NMDs),<sup>4</sup> why we should be told if the 81 patients were systematically investigated for NMDs and how many were diagnosed with NMD.

Left ventricular hypertrabeculation can be associated with late gadolinium enhancement (LGE).<sup>5</sup> Thus, we should be informed if the included patients were investigated for LGE and if there were any differences between those with and without LGE.

Left ventricular hypertrabeculation may undergo dynamic changes with regard to morphology and function over time. This has been particularly observed in pregnant females, athletes, and NMDs. Thus, we should know if the 81 included patients underwent follow-up investigations to assess if morphology and extent of LVHT and cardiac functions changed over time.

Though rare, LVHT may be present in several family members, suggesting a genetic pathophysiological background. We should know how many of the

### LETTER TO THE EDITOR

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included patients were related to each other and in how many of the included patients systematic investigations for LVHT had been carried out.

In conclusion, the study lacks substantial information about individual patients. Current medication, devices, family relations, follow-up investigations, previous history for syncope, cardio-embolism, or heart failure, LGE, and genetic background should be provided. To unravel the enigma LVHT, comprehensive data in individual patients with LVHT should be collected and reported.

## REFERENCES

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