

Noncompaction may not only be non-isolated but also myopathic. Commentary to the article: “Left ventricular non-compaction associated with hypertrophic cardiomyopathy in the same patient”

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With interest we read the article by Laaroussi et al. [1] about a 19-year-old male with left ventricular hypertrabeculation/noncompaction (LVHT). We have the following comments and concerns.

LVHT may not only be associated with mutations in sarcomeric genes causing cardiomyopathy, but also in sarcomeric and non-sarcomeric genes causing skeletal muscle myopathy. Did the authors consider myopathy in the presented patient? Up to 80% of patients with LVHT suffer from a neuromuscular disorder [2]. Was the patient referred to a neurologist? Patients with myopathy may manifest with fatigue, exercise intolerance, muscle cramps, myalgias, weakness, wasting, fasciculations, or complications such as respiratory insufficiency, orthopaedic abnormalities, hyper-CKaemia, or rhabdomyolysis, and we should be informed if any of these manifestations was present.

We do not agree that LVHT is a genetic disease [1]. Although LVHT is frequently associated with mutations in various genes or chromosomal defects, a causal relation has never been proven. Because LVHT may be non-congenital and may even disappear [3], it is conceivable that LVHT is a secondary phenomenon in primary cardiac disease, such as cardiomyopathy or heart failure. In transgenic mice it has been shown that LVHT is even inducible [4].

LVHT is complicated by heart failure, ventricular arrhythmias, or stroke/embolism [5]. Did the patient develop any of these complications? Was systolic dysfunction in the presented patient due to LVHT or due to hypertrophic cardiomyopathy? The patient seems to be particularly at risk for stroke/embolism since he presented with an enlarged left atrium, which is frequently associated with atrial fibrillation. Was atrial fibrillation ever recorded on Holter electrocardiogram? Did the patient undergo electrophysiological studies? Did the authors ever consider oral anticoagulation?

LVHT may be a hereditary condition [6]. Although it is mentioned that other family members were screened, it is

not reported for which abnormalities these relatives were screened and how many had LVHT.

Lastly, we need to remind the reader that the association of LVHT with hypertrophic cardiomyopathy is not unique and has been repeatedly reported. LVHT has been additionally described in association with dilated cardiomyopathy.

Overall, this study would be more meaningful if supplementary data had been provided, particularly if the patient was screened for myopathy, chromosomal defects, or complications of LVHT. It would also be helpful to revise previous echocardiographic investigations to determine if LVHT was congenital or acquired.

Conflict of interest: none declared

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