

Left ventricular non-compaction and hypertrophic cardiomyopathy: two overlapping diseases or two manifestations of the same cardiomyopathy? Response to the letter concerning the article: “Left ventricular non-compaction associated with hypertrophic cardiomyopathy in the same patient”

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We thank Dr. Finsterer and Dr. Stollberger for their valuable comments on our paper [1].

First, we agree that both left ventricular non compaction (LVNC) and hypertrophic cardiomyopathy (HCM) can be associated with neuromuscular disorders [2]. Our patient did not report any symptoms suggestive of myopathy. He was also referred to a neurologist, who did not find any abnormality. Of note, creatine kinase serum level was normal.

Second, LVNC and HCM have been accepted as distinct cardiomyopathies and classified as genetic cardiomyopathies by the American Heart Association [3]. We concur that since LVNC was reported to disappear and was even inducible in animal models, its genetic origin could be conflictual and debatable. Recently, Lorca et al. [4] studied three families with both LVNC and HCM diseases, with different common mutations in each family and autosomal dominant inheritance. Within one family, the investigators found the coexistence of both diseases in two patients, with a possible pathogenic candidate described: mutation of sarcomere cardiac b-myosin heavy chain gene (MYH7 L620P). In the two other families, the same mutation was described (MYBPC G263NX) with phenotype variability, and only one case associating both LVNC and HCM in each family [4]. Further genetic studies should be performed to try to answer to the following question: are HCM and LVNC two overlapping diseases or two different manifestations of the same cardiomyopathy spectrum? Unfortunately, we did not perform genetic analysis because the patient's family refused it.

Finally, regarding complications, our patient showed heart failure symptoms and signs with mildly impaired left ventricular ejection fraction, in addition to episodes of non-sustained

ventricular tachycardia on 24-h Holter electrocardiography recording. According to sudden cardiac death score related to HCM, implantable cardiac defibrillator was indicated and thus implanted. Although the left atrium was dilated (area 27 cm²), no atrial fibrillation was found on 24-h Holter electrocardiogram recordings or on different telemetries; therefore, anti-coagulation was not indicated.

To conclude, LVNC can be either primary (congenital) or secondary. When it is primary, LVNC genetic determination is close to that of HCM. Further genetic studies are needed to better elucidate this relationship and to better identify the pathogenic mutations.

Conflict of interest: none declared

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