LETTER TO THE EDITOR

Can cardiac magnetic resonance imaging resolve cardiac and neurological enigmas of left ventricular hypertrabeculation/non-compaction?

Est-ce que l’IRM peut résoudre les énigmes cardioneurologiques d’un VG non compacté?

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With interest, we read the article by Thuny et al. about the side-by-side comparison of cardiac magnetic resonance imaging (CMRI) and echocardiography in 16 patients with echocardiographically suspected left ventricular hypertrabeculation/non-compaction (LVHT) [1]. We have the following questions.

Echocardiographic data indicate that LVHT may be overdiagnosed in black Africans [2]. Were there any black Africans among the included patients, and did the morphology and distribution of LVHT differ between Africans and Caucasians?

The authors stated that none of the patients had signs or symptoms of a neuromuscular disease. Were they investigated neurologically, and if so, by which means? Since LVHT is frequently associated with neuromuscular disease, when systematically screened, it would be interesting to compare CMRI findings in patients with and without neuromuscular disease [3].

According to Table 1, five patients had a family history of LVHT. Were any of these relatives included among the five study patients? If so, were the pattern and distribution of LVHT different or similar in the relatives? LVHT is frequently associated with genetic disorders. Were the patients screened for mutations in genes reported to be associated with LVHT [4]?

LVHT frequently is associated with ventricular arrhythmias, systolic dysfunction and thromboembolic events. Did any of the 16 patients experience a stroke or peripheral embolism? How many of the patients received oral anticoagulation, and how many received pharmacotherapy for heart failure or rhythm abnormalities?

Can you provide an explanation for the discrepancy between left ventricular ejection fraction assessed by echocardiography and CMRI, especially in patients 1, 3, 5 and 15, in whom the discrepancy exceeded 10%? How did the observers differentiate LVHT from papillary muscles and aberrant bands, which might pretend to be LVHT, especially in the short-axis views and in the basal myocardial segments? It is indicated that only the maximal ratio between non-compacted and compacted myocardial layer was used for the analysis. Did the echocardiographic and CMRI observers agree on the segment where the maximal ratio was measured?

In three patients, pathological analysis of the hearts was performed, but the findings of this analysis and the comparison with echocardiography and CMRI were not presented. Since there is a lack of data correlating patho-anatomy with imaging findings in LVHT, it would be interesting to see these results.

LVHT may develop during the course of an individual’s life, as well as disappear, and an undulating pattern has been described in children [5,6]. Was it possible to analyse previous echocardiographic or CMRI recordings in order to assess whether LVHT developed or changed over time? Did the authors perform any follow-up investigations of the included patients and did they observe changes in LVHT? Since CMRI delineates the extent of LVHT better than echocardiography, it might also diagnose LVHT more accurately in those cases, in which echocardiography is uncertain or the diagnostic criteria are not completely fulfilled.

As reported previously, the study confirms that the septal segments are only rarely affected by LVHT [7]. Can you provide any explanation for these findings?

In conclusion, from this well-documented series of patients with LVHT, still more information and data should be obtained, which could contribute to the clarification of some of the enigmas of LVHT.

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Conflicts of interest statement

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References


Claudia Stöllberger∗, Josef Finsterer
Krankenanstalt Rudolfstiftung Wien, Vienna, Austria

∗ Corresponding author. Steingasse 31/18, 1030 Vienna, Österreich, Austria. Fax: +43 1 945 42 91.
E-mail address: claudia.stoellberger@chello.at (C. Stöllberger).

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