Letter to the Editor: Value of Cardiac Magnetic Resonance Imaging in the Diagnosis of Left Ventricular Hypertrabeculation/Noncompaction

We read with interest the article of Korcyk et al. (J Cardiovascular Magnetic Resonance 2004;6:569–576) who present a 26-year-old man with left ventricular hypertrabeculation/noncompaction (LVHT) (Korcyk et al., 2004). The report raises several issues, and we would like to bring these to the attention of the readers of the Journal.

That LVHT due to intrauterine arrest of myocardial compaction has not been proven so far. On the contrary, it has been reported that LVHT develops during adulthood (Finsterer and Stöllberger, 2001). Furthermore, many other pathomechanisms explaining the development of LVHT have been proposed (Stöllberger and Finsterer, 2004a). LVHT is not necessarily characterized by hypokinesia; it has also been found in well contracting left ventricles (Ichida et al., 1999; Stöllberger et al., 2002). There are no follow-up studies that confirm the statement of the authors according to which all LVHT hearts decline to heart failure. There is no definite evidence that LVHT is associated with an abnormally high rate of thromboembolism (Ichida et al., 1999; Stöllberger and Finsterer, 2004b).

There are no grounds that the echocardiographic criteria for LVHT should be simply adopted by cardiac magnetic resonance imaging (CMRI). The statement that CMRI is a valid method to diagnose LVHT is not in accordance with a recent study in 8 patients with echocardiographically diagnosed LVHT, which was confirmed by CMRI in only 6 patients (Weiss et al., 2003).

When measuring myocardial mass by CMRI, it is unclear how the authors differentiated flow artefacts and papillary muscles from myocardial trabeculations. It is not clear how many and which short axis imaging planes were choosen, based on which criteria, to measure left ventricular mass.

No information is provided about follow-up and cardiac therapy of the patient, although the authors request such information. Did the patient have elevated levels of creatine-kinase? It is not reported whether the patient or his relatives complained of any neuromuscular symptoms and whether they underwent a neurologic evaluation. LVHT is associated with neuromuscular disorders in up to 80% of the cases (Stöllberger et al., 2002).

No results of the ultrastructural investigation of the myocardial biopsy are given. How can the authors be certain that earlier performance of CMRI in the presented patient would have obviated the need for myocardial biopsy?

Was LVHT excluded echocardiographically in all 10 patients with dilated cardiomyopathy in whom they measured trabecular mass and compared it with the results of the reported patient? Was the perfusion of the trabeculations reduced in these 10 patients?

Myocardial scarring is not always found in LVHT when histologically investigated. It is still not proven if cardiac arrhythmias in LVHT are due to scar and fibrosis. Another potential pathomechanism might be abnormalities in the cardiac conduction system.
Pathohistological investigations of false tendons, which can be associated with LVHT, have shown that they contain Purkinje fibers, elements of the conduction system (Lotkowski et al., 1997).

Overall, final assessment of the value of CMRI in the diagnosis of LVHT needs to be deferred until controlled studies on larger cohorts of patients are available.

Claudia Stöllberger, M.D.
2nd Medical Department,
Krankenanstalt Rudolfstiftung,
Wien, Österreich

Josef Finsterer, M.D.
Neurological Department,
Krankenanstalt Rudolfstiftung,
Wien, Österreich

REFERENCES


