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Impact of left ventricular noncompaction on brain

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To the Editors

We read with interest the article by Bistriceanu et al. [1] describing a 47-year-old male with ischaemic stroke in the territory of the right middle cerebral artery, which was classified as cardioembolic due to intracardiac thrombus formation in the context of left ventricular hyper-trabeculation (LVHT), also known as non-compaction. Their study is excellent, but has limitations that cause us concerns and which we would like to discuss.

We disagree with the statement that LVHT is congenital in each case [1]. Though rarely reported, several cases of LVHT have been reported in which LVHT was not present at birth, and therefore not congenital, but developed during post-partum life [2]. Acquired LVHT has been particularly reported in patients with neuromuscular disorders, in pregnant females, and in professional athletes [2]. There are also indications that acquired LVHT in pregnant females can disappear again after delivery or after cardiac surgery [3].

We also disagree with the statement that LVHT is a genetic disorder [1]. Though commonly associated with genetic disease, particularly chromosomal defects, LVHT and hereditary neuromuscular disorders, and although rare familial cases have been reported [4], a causal relation between any of these genetic defects and the intra- or extrauterine development of LVHT has not been proven.

The complications of LVHT not considered in the discussion of the index case are ventricular arrhythmias and sudden cardiac death (SCD). Ventricular arrhythmias can have neurological implications as they can be associated with heart failure or thrombus formation. Thrombus formation can occur either within the recessus between the trabeculations or within the left ventricular cavity. Because of the risk of ventricular arrhythmias, LVHT patients are recommended to undergo either repeated Holter monitoring or implantation of a loop recorder to detect malignant ventricular arrhythmias.

Detection of ventricular arrhythmias has therapeutic implications, because implantation of an implantable cardioverter defibrillator (ICD) should be considered in LVHT patients with recurrent syncopes or documented ventricular arrhythmias on long-term ECG recordings.

Neurological complications of LVHT not only include cardio-embolic stroke but also syncope, transitory ischaemic attack, or cerebral hypoxia in the case of heart failure, prolonged malignant ventricular arrhythmias, or insufficient resuscitation in the context of asymptomatic ventricular arrhythmias.

Because LVHT is most commonly associated with neuromuscular disorders [5], all patients with LVHT should be systematically investigated for myopathy. Accordingly, we would like to establish whether the index patient, who was seen by a neurologist because of the stroke, had any indications for peripheral nervous system disease.

Because LVHT is commonly associated with the phenomenon of late gadolinium enhancement (LGE) of the left ventricular myocardium [6], we would also like to ask whether the index patient underwent cardiac MRI, and if this phenomenon could be documented in his case as well. The pathophysiological consequences of this phenomenon are not fully understood, but it has been postulated that it represents intraventricular fibrosis. LGE is associated with the occurrence of major adverse cardiovascular events (MACE) [6].

To sum up, this interesting study has limitations that call the results and their interpretation into question. Addressing these issues would strengthen the conclusions and could improve the status of the study. LVHT has to be regarded as a risk

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factor of cardioembolic stroke, but before attributing stroke to LVHT, alternative aetiologies need to be thoroughly ruled out.

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