Neurologia i Neurochirurgia Polska
Polish Journal of Neurology and Neurosurgery
2023, DOI: 10.5603/PJNNS.a2023.0030

ISSN: 0028-3843, e-ISSN: 1897-4260

Response to Letter to the Editors regarding article entitled 'Left ventricular non-compaction cardiomyopathy and ischaemic stroke'

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Key words: left ventricular non-compaction cardiomyopathy (LVNC), ischaemic stroke

To the Editors

We thank Finsterer and Stöllberger for their valuable feedback [1], and we would like to respond to it.

A study from 2018 that involved a molecular analysis of 107 genes in 95 adult patients with LVNC in order to characterise the genetic spectrum of this disorder found that TTN, HCN4, MYH7 and RYR2 were the most common mutations among these patients.

The results of this study give substance to the idea that LVNC is basically a genetic disease. However, according to the European Society of Cardiology, LVNC is listed among unclassified cardiomyopathies, and according to the American Heart Association it is among the genetic cardiomyopathies [2].

Another interesting research field in this area is the association and overlapping with other conditions.

Several decades ago, the association of the phenotype with congenital heart diseases was first acknowledged and attributed to the additional burden imposed by the congenital defects on the LV [3].

Other associations between non-compaction cardiomyopathy and neuromuscular disorders can be found, i.e. Duchene muscular dystrophy, Becker muscular dystrophy, myotonic dystrophy, metabolic myopathy, and Leber's hereditary optic neuropathy [4].

No such neuromuscular pathology was detected in our patient, nor any other peripheral nervous system disorder.

We discussed in our article some complications such as cardiac arrhythmias, cardiac failure and sudden death. Cardiac monitoring is important because the treatment is related to cardiac failure management or an implantable defibrillator in ventricular arythmias [5]. In our patient, ECG monitoring did not reveal atrial fibrillation or any other cardiac arrhythmia at that moment. Further cardiac investigations did not reveal any thrombus between the trabeculations or within the left ventricular cavity. The phenomenon of late gadolinium enhancement (LGE) was not described on cardiac MRI.

However, we analysed the brain DWI-MRI imaging, and this revealed acute stroke in the right superficial MCA territory and left PCA territory. This is known as MACI (multiple acute cerebral infarcts in multiple arterial territories) and is radiologically defined as more than one acute ischaemic lesion in at least two cerebral territories. Several studies have emphasised cardiogenic embolism to be the primary aetiology for this group.

Further investigations in our case did not reveal a cervical or intracranial atherosclerosis that could have explained successive embolisation and affected clinical assessment or MRI timing. A cardio-embolic mechanism was hypothesised and the treatment with DOACs was started (the EF was

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Received: 11.04.2023 Accepted: 13.04.2023 Early publication date: 05.05.2023

< 20% and this represents another criterion according to other studies) [5, 6].

Conflicts of interest: *None.*
Funding: *None.*

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