

LETTER

Management of noncompaction requires optimisation

Dear Editor,

In a recent article, which appeared in the September 2018 issue of *Echo Research and Practice* (volume 5, pages K59–K62, <https://doi.org/10.1530/ERP-18-0015>), Gurunathan *et al.* reported a case in which a 32-year-old male with left-ventricular hypertrabeculation/noncompaction (LVHT), who experienced an ischemic stroke 1 year after detection of LVHT, which was attributed to this rare type of cardiomyopathy (1). We wish to stimulate discussion by raising the following points.

We do not agree with the statement that echocardiography is sufficient for diagnosing LVHT (1). Even experienced echocardiographers may overlook LVHT, particularly if the apex cannot be visualised ideally. In case of additional hypertrophic cardiomyopathy, LVHT may be hidden behind the thickened myocardium (2). In these cases, cardiac MRI (cMRI) is required to confirm or exclude LVHT, if suspected. Furthermore, there is no general consensus about the definition and diagnostic criteria of LVHT, why the inter-observer variability is high (3) or why LVHT is frequently over- or under-diagnosed, depending on the experience of treating cardiologists, diagnostic facilities and the definition of LVHT applied. Disagreement on the diagnosis of LVHT often makes it difficult to compare cases and studies about LVHT from different centres worldwide.

We also do not agree with the statement that the classical presentation of LVHT is heart failure (1). The classical presentation of LVHT is the asymptomatic presentation; this is why LVHT is most frequently only detected accidentally. Heart failure, cardiac embolism and ventricular arrhythmias including sudden cardiac death are regarded as rare complications of LVHT, although profound studies on this matter have been carried out infrequently.

Since atrial fibrillation (AF) may also be a complication of LVHT (4), and since AF is a frequent cause of embolic stroke, we should be informed about the individual history, if the patient ever experienced palpitations or arrhythmias prior to ischemic stroke and if the ECG

on admission or during hospitalisation ever showed permanent or paroxysmal AF. If standard ECG does not document any arrhythmias, long-term ECG recordings of LVHT patients should be carried out to detect their propensity to AF or to ventricular arrhythmias.

Concerning Fig. 3, the stroke volume appears to have been large, suggesting occlusion of a large intracerebral artery. Thus, we should be informed about the results of computed tomography-angiography (CTA), particularly if any of the large arteries was occluded, and if the patient required acute thrombolysis on admission or mechanical thrombectomy. Since follow-up cerebral computed tomography showed a marked shift of the left hemisphere to the right, we should be informed if decompression by craniotomy was ever considered or carried out.

The patient was reported to have been anticoagulated after the stroke (1). What was the length of the interval after which oral anticoagulation was started? Which type of oral anticoagulant was applied: a vitamin-K antagonist or a direct oral anticoagulant (DOAC)? If the authors regarded prevention of embolization as an important aspect of therapy, why did they not begin anticoagulation therapy at detection of LVHT at the age of 32 years?

Overall, this interesting case report could be made more meaningful by discussing the variable definitions and diagnostic criteria of LVHT, by discussing the inter-observer variability when diagnosing LVHT, by discussing the acute management of stroke and by discussing the indication for oral anticoagulation and thus prevention of cardiac embolism.

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