

Myopathy, athleticism, pregnancy, race, and chromosomal defects need to be considered in noncompaction



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To the Editor,

With interest we read the article by Asfalou et al. [1] about the clinical characteristics of 23 Moroccan patients with left ventricular hypertrabeculation/noncompaction (LVHT). We have the following comments and concerns.

The proportion of men was higher than previously reported. Was this due to the fact that the study was carried out in a military hospital where usually more men than women are treated? Was it due to a selection bias?

The percentage of patients presenting with left bundle branch block was high [1]. What is the explanation for this? Is it attributable to endocardial fibrosis, frequently found in LVHT? How many had heart failure and how many had systolic dysfunction? Was there a history of sudden

cardiac death in any of the patients or their relatives?

The proportion of patients with pulmonary hypertension is unexpectedly high (48%) [1]. This is in contrast to what has been reported thus far. How do the authors explain this high rate of pulmonary hypertension? Was it acute or chronic pulmonary hypertension? Was the prevalence of pulmonary disease increased in the cohort?

What was the rationale to establish oral anticoagulation with vitamin K antagonists in 28% of the patients? Did those with a history of thromboembolism present with intertrabecular thrombi, atrial fibrillation, or severe heart failure?

We do not agree that the study was the first to analyse prognostic factors of LVHT [1]. Several studies about prognosis, prognostic factors, and outcome have been published [2]. One of these studies identified advanced age, presence of a

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neuromuscular disorder (NMD), heart failure New York Heart Association III, atrial fibrillation, and sinus tachycardia as predictors of mortality [2].

What do the authors expect from a genetic study to support the embryonic hypothesis? Why should a certain mutation suggest the notion that LVHT is congenital? The relation between mutations so far detected and LVHT is vague and not all carriers of the same mutation develop LVHT [3]. Thus, a certain mutation does not predict LVHT and other factors than genetic ones seem to be involved in the development of LVHT.

LVHT is frequently associated with NMDs, pregnancy, chromosomal defects, athleticism, and Black African ethnicity [4]. Were any of these conditions present in any of the 23 patients?

Is the high prevalence of LVHT in the present study attributable to the fact that military members are usually athletic? In athletes, the prevalence of LVHT has been shown to be increased [5].

There are several indications that LVHT is more prevalent in Black Africans than Caucasians. How many of the included patients were Black Africans and how many Caucasians?

Although the authors mention that no NMD was detected in any of the 23 patients, we should be informed if the 23 patients were prospectively

investigated for NMDs. This is necessary since many NMDs are subclinical at presentation or present with only mild or nonspecific phenotypic features.

Overall, this interesting study should be supplemented by more clinical data, and by thorough discussion of the indication for oral anticoagulation and the genetic work-up, and counselling of LVHT patients.

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