Uniform criteria for diagnosing noncompaction by cMRI and echocardiography are warranted

Josef Finsterer, Sinda Zarrouk-Mahjoub

Uniform criteria for diagnosing noncompaction by cMRI and echocardiography are warranted

To the Editor,

We read with great interest the article by Akhbour et al. (1) published in Anatol J Cardiol 2015; 15: 550-5 entitled "Electrocardiographic findings in correlation to magnetic resonance imaging patterns in African patients with isolated ventricular noncompaction" on cardiac magnetic resonance imaging (cMRI) and electrocardiographic (ECG) findings in 24 patients with left ventricular hypertrabeculation (LVHT)/noncompaction. Systolic function and arrhythmia were not correlated with the number of non-compacted segments or the number of segments showing late gadolinium enhancement (LGE) (1). We have the following comments and concerns.

Though LVHT is presumably congenital in majority of the cases, it can be also acquired, such as in neuromuscular disorders (NMDs), (2) pregnant females (3), and athletes (4). Acquired LVHT suggests that LVHT is not only due to the failure of the embryonic compaction process but also may result from the adaptation of the myocardium to hemodynamic dysfunction.

We do not agree with the definition of LVHT for not allowing the presence of any other cardiac abnormality except LVHT (isolated LVHT). Non-isolated LVHT is frequent and is also LVHT.

How do the authors explain the missing correlation between the number of LGE segments and ventricular tachycardia? Was the group size too small? Was the correlation different when subendocardial, transmural, and mid-myocardial LGE were separately evaluated? Was the LGE pattern patchy or diffuse? Possibly, cMRI fails to display all degrees of fibrosis, particularly fibrosis of the endocardium or early evolving fibrosis? Possibly, ventricular arrhythmias are not correlated with the number of LG -segments but with the volume or area of the LGE lesions? It is also conceivable that fibrosis in LVHT is ethnically different; for instance, Caucasians show a positive correlation between fibrosis and arrhythmias, whereas Africans do not, similar to the results in the present study. How did the authors quantify arrhythmias to correlate them with the number of LVHT fibrotic segments?

Arrhythmias may not only result from myocardial fibrosis but also result from ischemia. There are some indications that perfusion of the non-compacted layer is worse than that of the compacted layer (5). Possibly, the amount of arrhythmias correlates with myocardial scintigraphy. The frequent occurrence of LBBB may not only result from myocardial fibrosis but also from trabeculations, which predispose for prolonged propagation of the excitation.

We do not agree with the statement that cMRI is the method of choice to diagnose LVHT (1). The method of choice is echocardiography, but in case the echocardiographic diagnosis is uncertain, cMRI should be performed. Both techniques supplement each other, but they produce false positive and false negative results. As long as there are no common generally accepted LVHT diagnostic criteria either for cMRI or for echocardiography and as long as there is no gold standard for diagnosing LVHT, the reliability of both methods remains limited.

Atrial fibrillation was found in 17% of patients (1). Did these patients also present with thrombi within the intertrabecular spaces?

Was intraventricular thrombus formation associated with cardiac function?

Overall, this interesting study could profit from including patients other than Africans, from increasing the group size, and from evaluating the LGE extension. The negative correlation found could be explained by the absence of a uniform definition of LVHT, thus including patients who do not have LVHT or excluding patients who definitely have LVHT.

Josef Finsterer, Sinda Zarrouk-Mahjoub[1]
Krankenanstalt Rudolfstiftung; Vienna-*Austria*
[1]Genomics Platform, Pasteur Institute of Tunis; Tunis-*Tunisia*

References

1. Akhbour S, Fellat I, Fennich N, Abdelali S, Doghmi N, Ellouali F, et al. Electrocardiographic findings in correlation to magnetic resonance imaging patterns in African patients with isolated ventricular noncompaction. Anatol J Cardiol 2015; 15: 550-5. **[CrossRef]**
2. Finsterer J, Stöllberger C, Schubert B. Acquired left ventricular noncompaction as a cardiac manifestation of neuromuscular disorders. Scand Cardiovasc J 2008; 42: 25-30. **[CrossRef]**
3. Gati S, Papadakis M, Papamichael ND, Zaidi A, Sheikh N, Reed M, et al. Reversible de novo left ventricular trabeculations in pregnant women: implications for the diagnosis of left ventricular noncompaction in low-risk populations. Circulation 2014; 130: 475-83. **[CrossRef]**
4. D'Ascenzi F, Pelliccia A, Natali BM, Bonifazi M, Mondillo S. Exercise-induced left-ventricular hypertrabeculation in athlete's heart. Int J Cardiol 2015; 181: 320-2. **[CrossRef]**
5. Sato Y, Matsumoto N, Matsuo S, Imai S, Yoda S, Tani S, et al. Subendomyocardial perfusion abnormality and necrosis detected by magnetic resonance imaging in a patient with isolated noncompaction of the ventricular myocardium associated with ventricular tachycardia. Cardiovasc Revasc Med 2009; 10: 66-8. **[CrossRef]**

Address for Correspondence: Univ. Prof. Dr. Josef Finsterer
Postfach 20, 1180 Vienna-*Austria*
Phone: +43-1-71165-92085
Fax :+43-1-4781711
E-mail: fifigs1@yahoo.de

DOI:10.5152/AnatolJCardiol.2015.6659

Author`s Reply

The authors of this mentioned article did not send any reply to this Letter to the Editor in spite of our insistent requests.