Dear Editor,

We have read the recent case report by Emini et al. with great interest. The authors reported an interesting case with noncompaction of ventricular myocardium (NVM) in a 27-year-old man followed-up with a “large ventricular septal defect” (“single ventricle”). Emini et al. discussed the pathophysiology of NVM and highlighted the importance of careful echocardiography in diagnosing NVM. In our opinion, some points of this work are not sufficiently clear.

The authors noted left ventricular trabeculations and deep intertrabecular recesses as the diagnostic criteria of NVM. However, in the presented case, they only showed the hypertrabeculations and deep recesses in the right ventricular portion of the single ventricle (especially in Figure 1b). Isolated left ventricular involvement in NVM is satisfactory for the diagnosis, although the sole right ventricular involvement requires further investigation as the right ventricle is structurally a trabecular ventricle. As in the reported case, accompanying elevated pressure (due to large interventricular septal defect) in the right ventricle leads to hypertrophy and elongation of trabeculas. Accordingly, definition of NVM requires the presence of hypertrabeculations and intertrabecular recesses in the left ventricle by clearly presenting the borders of the noncompacted and compacted layers.\(^\text{[2]}\)

The authors reported pulmonary artery systolic pressure as 95 mmHg in the text, whereas they reported pulmonary artery systolic pressure as >100 mmHg in the figure legend of Figure 2. I am confused about the pulmonary artery systolic pressure of this case. Actually, pulmonary artery systolic pressure should be the same as the systolic systemic arterial pressure (100 mmHg) in the absence of aortic or pulmonary stenosis (which were not noted) as both chambers share common ventricular pressures.

Beyond the above-mentioned points, authors reported admission \(\text{SaO}_2\) as 72% and they noted improved \(\text{SaO}_2\) of 86% after the therapeutic interventions. Unresponsiveness of cyanosis (and \(\text{SaO}_2\)) is the clinical hallmark of cyanotic congenital heart diseases differentiating them from pulmonary diseases. Therefore, I wonder about the mechanism of \(\text{SaO}_2\) improvement and the treatment modalities improving \(\text{SaO}_2\).

We hope that the above-mentioned issues might add to the value of the case report by Emini et al.

Sincerely,

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REFERENCES

Author’s reply

Dear Editor,

We thank Mr. Yıldız, whose comments gave us the opportunity to discuss our case report published previously in your journal.

We agree with the author that the presence of intertrabecular recesses in the left ventricle is not very clear. It could be due to poor-quality images and also to difficulties in obtaining two-dimensional echo scans, because the patient was not clinically stable during the echocardiographic examination. However, we were convinced that the left ventricle was also non-compacted. We have sent the editor an echo
Myocardial noncompaction recognized following a transient ischemic attack

Thromboembolism in young patients with noncompaction cardiomyopathy: more than what we thought

Dear Editor,

I read with interest the case report by Karabulut et al.[1] about a transient ischemic event in a young man with noncompaction of the ventricular myocardium (NCVM). Although this complication was well documented by Oechslin et al.[2] its occurrence in children and young adults is still probably underestimated. The presence of multiple trabeculations with deep intertrabecular recesses leads to blood stagnation and clot formation as was previously reported in three pediatric patients (Figure).[3] It is well documented that the left ventricle function may improve within days of presentation,[3,4] but this event may then provoke embolization, which was reported in children as young as 2 years of age. Serious or even fatal outcome may ensue when the left ventricle thrombus is large, and immediate anticoagulation or thrombolysis should be instituted.[5] Routine use of anticoagulation drugs in NCVM is controversial;[6] however, if there is poor ejection fraction, anti-platelet therapy is mandatory. Awareness about this important life-threatening complication of NCVM in young patients can help prevent/reduce mortality and morbidity.

Sincerely,
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