

video which may further clarify the presence of non-compaction of the myocardium.

Regarding the improvement seen in SaO₂, we think that mild improvement in SaO₂ was associated with the bed-rest of the patient and oxygenotherapy; however, it did not reach the optimal level.

There was a misinterpretation of the systolic pulmonary artery pressure. We measured the tricuspid transvalvular pressure gradient as 94 mmHg, as shown in Fig. 2. We added the supposed pressure of the right atrium of 10 mmHg, and this yielded a value >100 mmHg. We

agree that systolic pressure should be the same in the aorta and pulmonary artery in the absence of aortic valve disease and/or pulmonary valve disease.

Sincerely,

On behalf of the authors,

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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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Figure. A short-axis view distal to the papillary muscles showing a thrombus (arrow) with noncompaction of the ventricle.

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Author's reply

Dear Editor,

Dr. Ali discusses thromboembolic events in myocardial noncompaction patients with complementary information and references which supplement our article. In the letter, there is no specific question to be answered regarding our case.

We appreciate the author for sharing this comprehensive information. Although routine use of anti-

coagulation in myocardial noncompaction patients is not well-documented, we use warfarin for six months due to apparent risk for cerebral thromboembolism. In our case, left ventricular ejection fraction was slightly decreased (45%) and remained unchanged during the follow-up. The patient has been asymptomatic for a year after cessation of warfarin.

Sincerely,

On behalf of the authors,

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Tissue Doppler evaluation of the effects of major lung resection on cardiac functions

Dear Editor,

We read with interest the article by Çölkesen et al.^[1], which is the first study in the literature evaluating cardiac functions by tissue Doppler echocardiography in the early postoperative period of major lung resection (up to 3 months).

We want to comment on some weak points about the inclusion and exclusion criteria of the patients.

1. In the Methods section, it is clearly stated that patients with diastolic dysfunction were excluded. However, data in Table 2 for preoperative echocardiographic findings include the following:

- Mitral diastolic velocities (E velocity 90 ± 23 cm/sec, A velocity 92 ± 23 cm/sec)
- Tricuspid diastolic velocities (E velocity 67 ± 13 cm/sec, A velocity 65 ± 19 cm/sec).

Tissue Doppler diastolic parameters:

- Mitral annulus (E' 9 ± 2 cm/sec, A' 10 ± 2 cm/sec)
- Tricuspid annulus (E' 9 ± 2 cm/sec, A' 15 ± 3 cm/sec)

When we analyze these data, we see that the mean values of E and A are close to each other for both mitral and tricuspid diastolic velocities, and the mean value of E' is smaller than A'.

Based on these findings, we consider that the patients meet the criteria for stage II diastolic dysfunction (pseudonormal pattern).^[2,3] Therefore, we want the authors to define the method they used to exclude diastolic dysfunction, other than they mentioned in the article.

2. The authors state that patients with an FEV₁/FVC ratio less than 0.60 were excluded to avoid right heart modifications related to "severe" chronic obstructive pulmonary disease (COPD). However, it is known that the presence of airflow limitation is defined by a postbronchodilator FEV₁/FVC <0.70.^[4] According to the criteria for staging the severity of COPD, the FEV₁ value must be used. The spirometric classification based on FEV₁ for the severity of COPD includes four stages:^[4-6]

Stage I: Mild (FEV₁/FVC <0.70 and FEV₁ $\geq 80\%$ predicted)

Stage II: Moderate (FEV₁/FVC <0.70 and $50\% \leq$ FEV₁ <80% predicted)

Stage III: Severe (FEV₁/FVC <0.70 and $30\% \leq$ FEV₁ <50% predicted)

Stage IV: Very severe (FEV₁/FVC <0.70 and FEV₁ <30% predicted or FEV₁ <50% predicted plus chronic respiratory failure).

For this reason, to avoid right heart modifications related to severe COPD, we suggest that the authors use FEV₁ values for exclusion of severe COPD (stage III and IV).