In addition, an 8-month interval between myocardial infarction and the subsegmental pulmonary embolism does not necessarily justify the association between these findings. In fact, patients with recent paradoxical embolism usually have simultaneous deep vein thrombosis or pulmonary embolism.³

With respect to coronary angiography, imaging findings are essential when making the differential diagnosis of myocardial infarction, because coronary artery embolisms present as small peripheral stops (embolisms are usually small and migrate distally). If coronary embolism is suspected, however, thrombus aspiration rather than angioplasty is preferable to avoid endothelial wall damage caused by the inflation of the balloon. In such cases, histopathologic evaluation of the thrombus aspirate may also be useful to substantiate further the origin of thrombotic material and to differentiate it from atheroembolism.⁴

Finally, we should bear in mind that a cor triatriatum dexter with an enlarged right atrium may in itself be a primary source of thrombus formation. Nonetheless, the persistence, to a greater or lesser extent, of the right sinus venosus valve may protect against pulmonary embolism acting as a filter at the cavoatrial junction.⁵ This could explain why this patient with a family history of pulmonary arterial embolism showed no Doppler data indicating deep vein thrombosis (all the vein thrombus could have migrated to the right cardiac chambers) and only had subsegmental pulmonary embolism.

Although we consider, as do Hussain and colleagues,¹ that the cor triatriatum dexter in this case should be resected because it produced moderate stenosis and that the atrial septal defect should be closed because of its large size, medical treatment aiming to prevent recurrence of venous thrombosis should also be mandatory, especially if the patient has a family history of pulmonary embolism.

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COR TRIATRIATUM DEXTER IN ADULTS: CLINICAL IMPLICATIONS

Reply to the Editor:

We thank Martínez-Quintana and Rodríguez-González¹ for their comments and appreciate the opportunity to respond.

We agree with Martínez-Quintana and Rodríguez-González¹ that pregnancy itself is a risk factor for myocardial infarction and that other diagnoses, such as atherosclerosis, spontaneous coronary dissection, and so on, need to be ruled out.² It is quite possible, even likely, that the thrombus on the membrane in the right atrium (RA) began to form during the pregnancy.³ The patient in our report did undergo a cardiac catheterization after she had inferior wall myocardial infarction, and it revealed occlusion of the posterolateral branch of the right coronary artery, with no evidence of coronary atherosclerosis, coronary dissection, or other abnormalities. She underwent an uncomplicated coronary angioplasty; whether balloon angioplasty is safe in the immediate postpartum period is beyond the scope of our article.

She came to us with pulmonary embolism 8 months later. Lower-extremity Doppler yielded negative results, and because she had family history of pulmonary embolism, coagulation studies, including factor V Leiden mutation, were performed. All were negative. During surgery, there was a large thrombotic mass on top of the thick, calcified membrane above the tricuspid valve in the RA, along with an atrial septal defect.³ The operative findings strongly suggested that the thrombotic mass had been developing for a long time, because the deeper portions were partly organized. The RA thrombotic mass is a plausible explanation of paradoxical embolism to the right coronary artery and resulting myocardial infarction in the absence of other findings on cardiac catheterization. Without other high-risk factors for deep venous thrombosis (DVT) in the patient and with normal coagulation studies, it also explains the pulmonary embolism. We disagree with Martínez-Quintana and Rodríguez-González¹ that an 8-month interval between the episodes rules out the association of both with the membrane.

We agree that cor triatriatum dexter with an enlarged RA may in itself be the source of thrombus formation, which was the case in our patient, with the calcified membrane acting as a nidus. We disagree that this membrane was a protection against a large pulmonary embolism.

We also disagree that the thrombus on the membrane was from a DVT, because it was very adherent to the membrane and had to be peeled off, reflecting that the thrombus was formed in situ. Because we strongly believe that the cause for the patient's pulmonary embolism (and myocardial infarction) was the thrombotic mass on the thickened membrane in RA (which was all surgically excised), especially with negative coagulation studies and absence of any documented DVT, we also do not agree with the recommendation that the patient should be placed on long-term medical prophylaxis for DVT despite her family history of pulmonary embolism. We used short-term anticoagulation and long-term aspirin therapy.

We greatly appreciate the opportunity to address the queries of Martínez-Quintana and Rodríguez-González.¹

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ASCENDING AORTIC ANEURYSMS AND VOLUMETRIC MEASURES

To the Editor:

We read with interest the article, “A prospective study of growth and rupture risk of small-to-moderate size ascending aortic aneurysms” by Geisbüsch and colleagues.¹ The volume of the ascending aorta can certainly be calculated when the diameter and the length of the vessel are known. A patient with a longer ascending aorta with normal transverse diameters can still have a high-volume ascending aorta. But a long ascending aorta is never 1 of the indications for surgery in the setting of a normal transverse diameter. However, when a patient has a very short and enlarged ascending aorta, he or she can still have a normal ascending aorta volume. It would be a mistake to ignore this patient based on volumetric parameters. We strongly believe that volume of the vessel should not be a criterion for a surgical approach.

The authors claim in their Table 1¹ that the presence of a bicuspid aortic valve was not a contributing factor for aortic dilation. However, bicuspid aortic valve is one of the major contributing factors to aortic dilation because the bicuspid valves disrupt blood flow creating an abnormal flow pattern within the vessel. We also request that the authors further explain the underlying mechanism/s so we can accurately apply anticoagulation therapy to the volume of the ascending aorta. Because there was a statistically significant finding (shown in Table 1¹), it deserves an explanation.

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GROWTH AND RUPTURE RISK OF SMALL-TO-MODERATE SIZE ASCENDING AORTIC ANEURYSMS: A PROSPECTIVE STUDY

Reply to the Editor:

Thank you for your interest in our article and your comment. The decision to operate on a patient with an ascending aortic aneurysm depends on a reliable and reproducible diagnostic method. To estimate the risk of rupture and death it is important to understand the natural history of thoracic aneurysms and take into account the morphology of the aneurysm and progression of the disease rather than looking at a single measurement (eg, diameter). We agree that a long aorta with a normal diameter is unlikely to rupture, whereas a shorter aorta with greater diameter is at higher risk for rupture. Both, however, could have the same volume measurement. Monitoring growth by taking diameters presents, we believe, a high risk of interobserver variability because measurements are often taken at different levels or angles. Also, the aorta is a 3-dimensional structure, which should not be described with a single linear measurement. Thus, calculating volumes is a more objective way of describing this structure and adds to the reliability of monitoring with computed tomography scans. To include the