CORRESPONDENCE

Isolated Right Ventricular Myocardial Infarction in Patients with Coronary Artery Bypass Grafts

To the Editor:

I read with interest the article in the *Texas Heart Institute Journal* by Franco and colleagues[1] about isolated right ventricular myocardial infarction (RVMI). First, I want to thank the authors for reminding us that isolated RVMI can easily be misdiagnosed as acute anterior myocardial infarction (MI) and should be considered in the differential diagnosis.

In clinical practice, isolated RVMI is rare in comparison with anterior MI.[2] There are important differences in the approach to treating these types of MI, particularly in regard to vasodilator medication. Therefore, the differential diagnosis is important, especially in centers without the capability of performing primary percutaneous coronary intervention. ST-segment elevation in the precordial leads is characteristic of anterior MI caused by left anterior descending coronary artery occlusion. However, ST-segment elevation in leads V_1 through V_3 or V_4 has also been reported during isolated RVMI. The decrease in magnitude of the ST-segment elevation in the precordial leads V_1 through V_3 or V_4 and progressive regression of ST segments without Q-wave formation on electrocardiography suggests isolated RVMI rather than anterior MI.[3]

Franco and colleagues mentioned some causes of isolated RVMI. Another cause is proximal acute occlusion of a dominant right coronary artery (RCA) in a saphenous vein graft to the RCA.[4] In this circumstance, the inferior part of the left ventricle is preserved by the saphenous vein graft and does not become ischemic. If the right ventricular branch of the RCA does not receive enough retrograde blood supply from the graft, occlusion of the proximal dominant RCA can induce isolated RVMI. Therefore, isolated RVMI should be considered in patients who have histories of coronary artery bypass grafting and ST-segment elevation in leads V_1 through V_3 or V_4 on electrocardiography.

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Focusing on Cor Triatriatum Dexter and Atrial Septal Defects

To the Editor:

We read with interest the article in the *Texas Heart Institute Journal* by Vukovic and colleagues,[1] and we have a few comments that we think are of note.

Cor triatriatum dexter (CTD) and prominent eustachian valve are thought to result from the incomplete and abnormal regression of the embryonic right valve of the sinus venosus, caused by abnormal fetal circulation. Whereas the left leaflet of the sinus venosus is incorporated into the interatrial septum and forms part of the septum secundum, the right leaflet is reabsorbed, forming the eustachian and thebesian valves.

A prominent Chiari network and eustachian or thebesian valve might simulate CTD and produce insignificant flow accelerations. However, in CTD (as in the case report[1]), the valve is attached to the atrial septum. In the case of a prominent or giant eustachian valve, there are no attachments, irrespective of obstruction.[2] In addition, CTD is frequently associated with right-sided abnormalities such as atrial septal defect (ASD). For this reason, although successful percutaneous ASD closure has been reported in patients who have prominent eustachian valves,[3] extra care should be exercised in patients with CTD: septal attachments on the atrial septum might lead to entanglement, misplacement, or leftward bowing because of a high right atrial pressure and embolization of the device. In addition, proper deployment and device stability might be prevented by inadequate definition of the ASD margins, overlap of the valve remnant, and under- or oversizing of the device.[4]

As stated by the authors,[1] the patient's symptoms depend primarily on the degree of right atrial septation and the size of the sinoatrial orifice. However, a large, prominent eustachian valve can sometimes obstruct the inferior vena cava and lead to trapped catheters, supraventricular arrhythmias, bacterial endocarditis, and thrombosis with subsequent pulmonary or systemic embolism. For this reason, if the patient will be undergoing heart surgery for other reasons, the membrane or fibrous band should be removed, even if it is not obstructive.

Finally, left-to-right shunting secondary to restrictive CTD and a patent foramen ovale or ASD might lead to central cyanosis and symptoms consistent with increased portal venous pressure, such as ascites, coagulopathy, hepatic dysfunction, and protein-losing enteropathy.[5] In such cases, ASD closure should be undertaken after surgical or percutaneous balloon correction of CTD.[6]

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Myocardial Apical Hypertrophy and Takotsubo Cardiomyopathy

To the Editor:

I appreciated the article by Roy and colleagues[1] in the *Texas Heart Institute Journal*. To the authors' knowledge, theirs were the first reported cases in which apical-variant hypertrophic cardiomyopathy was masked by apical ballooning from stress-induced cardiomyopathy. The authors added information about 5 reported cases of patients with hypertrophic cardiomyopathy (HCM) who had experienced an episode of takotsubo cardiomyopathy, all of whom had the obstructive HCM "with asymmetric septal hypertrophy, not apical-variant HCM." What was actually observed is a recently detected phenomenon of apparent left ventricular (LV) apical hypertrophy,[2,3] which occasionally is seen in the subacute and chronic phase of convalescence from takotsubo cardiomyopathy. It is caused by transient myocardial edema.[4] Kato and colleagues[3] observed apical hypertrophy of the LV at approximately 3 weeks after onset, when the wall motion had improved; the ventricular wall gradually became thinner, and the transient apical hypertrophy was attributed to hypertrophic signaling in the myocardium, which was stimulated by catecholamines.[3] Myocardial edema with a hypertrophic LV apex has been reproducibly detected on cardiac magnetic resonance images[4] and echocardiograms. Whether the LV hypertrophy[1] represents apical HCM or takotsubo-induced myocardial edema can be resolved by observing subsequent electrocardiograms of these patients for chronically persisting giant negative T waves[5] and R waves[6] in the mid-precordial leads, and by comparing old and follow-up echocardiograms.

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