Takotsubo cardiomyopathy: a potential complication in the setting of acute prosthetic valve dysfunction

To the editor Takotsubo cardiomyopathy (TTC) is a specific transient type of acute myocardial disease which usually arises in the setting of stressful conditions associated with severe adrenergic discharge.[1] Clinically, this phenomenon strongly mimics acute coronary syndromes (ACSs), and may present with typical (apical ballooning) or atypical (focal, diffuse, etc, variants) myocardial wall motion abnormalities on imaging.[1] In their recently published well-written article, Perzanowska-Brzeszkiewicz et al[2] reported a case of mitral prosthetic valve dysfunction and thrombosis (leading to severe stenosis) presumably complicated by non–ST-segment elevation myocardial infarction due to coronary embolism. However, a coexisting TTC episode (with an atypical presentation) might also be quite likely in the present case.

In clinical practice, atypical variants of TTC might potentially be misdiagnosed as nonobstructive ACSs (those attributed to coronary vasospasm, coronary slow flow, and secondary triggers, etc), and hence; requires a high index of suspicion for their diagnosis.[1] On the other hand, TTC evolution in association with prosthetic valves was previously reported particularly in the postsurgical or infective endocarditis settings.[3] It seems likely that the patient presented in the case[2] might have suffered an atypical TTC episode largely attributable to severe physical stress associated with acute heart failure (hypoxia, etc) and possible emotional stress (sense of impending doom). In this context, demographic features of the patient (an elderly woman), absence of a visible thrombus on coronary angiogram, substantial levels of N-terminal pro-B-type natriuretic peptide (31 769 pg/ml) along with a slight elevation of cardiac troponin (0.134–0.113 ng/ml)[2] might also substantiate a coexisting atypical TTC episode.[4] Accordingly, we wonder whether segmental wall motion abnormalities (on echocardiogram) fully recovered or persisted during follow-up. Of note, atypical TTC variants might also have a worse prognosis due to higher levels of adrenergic discharge.[1] Did the patient have a coronary slow flow pattern on coronary angiogram (indicative of severe adrenergic discharge)[1]?

More rarely, subtle or overt cerebrovascular embolic events might also lead to TTC evolution (mostly atypical variants)[1] due to acute disturbances in specific areas of central autonomic regulation.[5] Therefore, a cerebrovascular embolic fragment (emanating from the surface of bioprosthetic valve thrombus) might also be associated with TTC evolution in this case. Accordingly, we wonder whether the patient had any coexisting neurological findings on admission.

In summary, TTC evolution (besides ACSs), might be also regarded as a potential complication in patients with acute prosthetic valve dysfunction with or without coexisting thrombus formation potentially suggesting important diagnostic and prognostic implications in these patients.

ARTICLE INFORMATION

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Authors' reply We would like to thank for interest in our article and for valuable comments.

Indeed, a diagnostic workup in our patient was very challenging. She was referred to our clinic with non–ST-segment elevation myocardial infarction (NSTEMI) as the initial diagnosis. However, urgent echocardiography showed massive thrombosis of mitral prosthesis. In this case, systemic embolization, including thrombotic infarction, seemed very possible.

Coronary embolism is a heterogenous cause of myocardial infarction with nonobstructive coronary arteries.[1] It can be diagnosed clinically and should be suspected when acute myocardial infarction occurs in a person with an underlying condition which predisposes to systemic embolism including valvular pathologies.[2] The clinical diagnosis of coronary embolism can be complicated by its potential for recanalization, such that coronary emboli may cause infarcts in territories supplied by angiographically-normal coronary arteries.[3]

In fact, we found no evidence of a coronary thrombus on coronarography. However, this examination was performed 3 days after the onset of chest pain. The distal embolization may have occurred at this time. Thromboemboli tend to lodge distally in normal coronary arteries that are becoming intramyocardial, causing small but transmural myocardial infarcts.[4]

In their comment, Yalta et al[5] suggest takotsubo cardiomyopathy (TTC) with an atypical presentation as a potential complication of acute prosthetic valve dysfunction. Takotsubo cardiomyopathy can mimic acute coronary syndrome and sometimes it is very difficult to differentiate it from acute coronary syndrome, especially myocardial infarction with nonobstructive coronary arteries. The diagnosis of TTC requires the presence of all 4 of the following: transient wall motion abnormalities with or without apical involvement (the regional wall motion abnormalities extend beyond a single epicardial vascular distribution), absence of obstructive coronary disease or acute plaque rupture at the angiography, new ECG abnormalities or elevation in the cardiac troponin level (with the absence of pheochromocytoma or myocarditis).

In our patient, transthoracic echocardiography revealed regional wall motion abnormalities within the inferolateral wall and intraventricular septum corresponding to the right coronary artery region. Unfortunately, we do not know the result of the next echocardiography after discharge from our hospital. Coronarography showed no coronary artery stenosis and no coronary slow flow pattern.

Standard electrocardiography showed 0.5 to 1 mm ST-segment elevation in the III and aVF leads and ST-segment depression in the I, aVL, and V_6 leads and the level of cardiac troponin was elevated with cardiac troponin T maximum value of 0.134 ng/ml, and N-terminal pro-B-type natriuretic peptide greater than 31 000 pg/ml.

Upon looking for an explanation for the clinical symptoms and abnormal results of additional tests in our patient, we concluded that the thrombotic infarction was the most likely cause.

Of course, focal TTC associated with the area of vascularization of one coronary artery is also possible; however, it is rather rare. Cardiac magnetic resonance imaging showing late gadolinium enhancement in the infarct area would have confirmed NSTEMI diagnosis, while rapid normalization of left ventricular contraction abnormalities would suggest TTC, However, none of them was performed.

In conclusion, TTC could be a good explanation for observed abnormalities in our patient, but in our opinion, NSTEMI seems to be more likely.

ARTICLE INFORMATION

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