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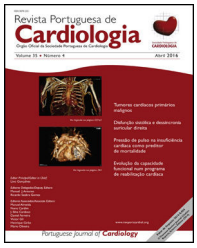

LETTER TO THE EDITOR

Basal takotsubo syndrome: A multifaceted entity with potential implications

Miocardiopatia de Takotsubo basal: uma entidade multifacetada com implicações potenciais

In clinical practice, takotsubo syndrome (TTS) occasionally manifests as atypical variants potentially affecting myocardial segments other than the left ventricular (LV) apex, including basal, midventricular, or other segments.[1,2] In their article recently published in the *Journal*, Miaris et al. reported an interesting case of basal TTS in a young woman following cesarean delivery.[1] Accordingly, we would like to make a few comments on this case and outline some further implications of basal TTS in this clinical setting.

First, diagnosis of atypical TTS variants is a significant challenge, largely due to the non-specific changes on the electrocardiogram (ECG) along with the absence of the typical apical ballooning pattern on imaging modalities.[2] Consistent with this, the patient was reported to be in normal sinus rhythm. However, we wonder about the authors' explanation regarding the potential mechanisms of documented relative bradycardia (50 bpm on the ECG), which seems quite unusual in such a TTS case under extreme stress. Accordingly, is it a drug-induced effect or a phenomenon associated with basal TTS? We hold the opinion that certain factors including the Bezold-Jarish reflex (potentially arising due to a sudden increase in circumferential wall stress of the LV base in association with dilatation and severe dysfunction of this segment) and episodes of sinus node ischemia – potentially associated with intermittent compression of coronary arteries including the circumflex artery (traveling along the left atrioventricular groove) by the dilated LV basal segments – might have served as the mechanisms of relative sinus bradycardia in this patient.

Second, atypical TTS variants (compared to the classical pattern) mostly arise as a consequence of more severe adrenergic discharge, which is potentially associated with serious clinical scenarios (neurological conditions, attempted suicide, etc.).[2] Therefore, it seems quite likely that the patient suffered extreme stress. On the other hand, severe adrenergic discharge in the setting of TTS was previously suggested to be associated with acute coronary microvascular dysfunction (presenting with coronary slow flow [CSF] in the major coronary arteries) and consequent adverse events including acute heart failure and arrhythmogenesis.[3] Accordingly, did the patient have a CSF pattern on invasive coronary angiography or cardiac arrhythmias during her hospital stay?

Third, basal TTS may also have important hemodynamic implications due to emerging acute mitral regurgitation (MR).[1,4] Characteristically, acute MR in patients with basal TTS may be considered a functional and reversible entity.[1,4] Recently, another case of basal TTS presenting with transient severe MR was also reported in a young woman following attempted suicide by drug overdose.[4] It thus seems likely that basal TTS has a particular predilection for young females under extreme stress.[1,4] Mechanistically, acute MR seems to arise in a subgroup of basal TTS patients presenting with excessive dilatation of the LV base and consequent mitral annular dilatation (as may have occurred in the present case[1]). However, dysfunction in the basal portions of the papillary muscles may also contribute to emerging acute MR in these patients. Nevertheless, the presence of a central jet on the echocardiogram suggests annular dilatation as the dominant mechanism of MR in the patient reported by Miaris et al.[1] Accordingly, we wonder about the mitral annulus diameter of the patient during her TTS episode. Importantly, the severity of MR should be thoroughly investigated in patients with basal TTS, particularly in those with mild LV systolic dysfunction, but with severe hemodynamic compromise. In this context, temporary LV unloading strategies such as intra-aortic balloon pumping could yield a significant therapeutic benefit in the acute setting.

Finally, the authors used global longitudinal strain (GLS) as an adjunctive diagnostic parameter in their patient.[1] However, we hold the opinion that assessment of GLS generally adds little to the conventional echocardiogram in diagnosing TTS. On the other hand, persistently abnormal GLS values following complete recovery from TTS could help identify cases with subclinical myocardial dysfunction in the long term, who usually present with symptoms such as exercise intolerance.[5,6] Moreover, survivors of atypical TTS variants may be more likely to suffer subclinical myocardial dysfunction, possibly due to the residual cardiomyotoxic effects of extreme adrenergic discharge on myocardial energetics and contractile reserve.[5,6] Accordingly, we wonder about the GLS values of the patient beyond one month (if any).

In conclusion, basal TTS could have important clinical implications in both the short and long term, mostly attributable to its specific cardiac location and associated factors (including extreme adrenergic discharge). However, further aspects of this phenomenon still need to be investigated.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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