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# Pheochromocytoma and Takotsubo Syndrome: An Ominous Duo

LETTER TO THE EDITOR

To the Editor,

In the clinical setting, pheochromocytoma has been suggested as a potential trigger of takotsubo syndrome (TTS) with a relatively worse prognosis.[1-3] In their recently published didactic case report, Vincenza Polito et al[1] have reported an atypical case of pheochromoyctoma-induced TTS with cardiogenic shock that was successfully managed with the use of levosimendan and intraaortic balloon pump (IABP). However, we would like to highlight particular implications of pheochromocytoma–TTS co-existence, and also we have information on a few points regarding their case.

First, the authors[1] should be commended for their timely diagnosis of an atypical TTS episode that universally presents with non-specific electrocardiographic (ECG) findings (as in the patient having ST-segment depressions in the leads V3-V6[1]) in the absence of classical apical ballooning on cardiac imaging.[4] Moreover, identification of pheochromocytoma as the trigger of TTS might also be considered quite challenging in the patient who demographically (elderly female) seemed to suffer just a classical emotionally triggered TTS at first glance.[1] Therefore, every effort should be made, regardless of demographic characteristics, to uncover any potential organic trigger (including pheochromocytoma) in such atypical TTS cases who are well known to harbor extreme adrenergic discharge (compared with classical TTS cases) potentially leading to arrhythmic episodes and acute coronary microvascular dysfunction.[2,4] Therefore, we wonder about the pattern of coronary slow flow (possibly emerging due to enhanced coronary microvascular resistance)[5] on coronary angiogram and documented malignant arrhythmias in their patient.[1]

Second, atypical TTS cases usually have a significant predisposition to persistent subclinical myocardial dysfunction due to the residual structural and metabolic alterations associated with substantial adrenergic discharge.[4] This mostly manifests as post-discharge exercise intolerance usually characterized by persistent disturbances in certain echocardiographic parameters including global longitudinal strain (GLS).[4,6] Accordingly, we wonder about serial GLS values of the patient, if any, on follow-up.

Third, a portion of patients with pheochromocytoma might also suffer varying degrees of chemical myocarditis usually presenting with diffuse myocardial edema, overt myocardial damage [usually detected with magnetic resonance imaging (MRI)], and substantial elevation in cardiac troponins.[2] In the setting of pheochromocytoma-induced TTS, co-existing chemical myocarditis might have important prognostic and therapeutic implications in the short and long terms.[2] Accordingly, was there any sign of accompanying chemical myocarditis (including excessive peak troponin value and specific echocardiographic findings) in the patient? Did they plan MRI with gadolinium enhancement after discharge in an effort to guide the long-term management?

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Fourth, sympathomimetic agents including dobutamine should be avoided in patients with TTS.[6] Moreover, these agents might be even more detrimental in those with a pheochromocytoma-induced TTS episode characterized by myocardial bombardment of circulating catecholamines in an excessive and prolonged manner. Importantly, levosimendan has been suggested as a promising alternative on top of mechanical support in TTS patients with hemodynamic compromise due to myocardial failure or acute mitral regurgitation.[4,6] Therefore, levosimendan might have been already preferred over dobutamine at the very onset of cardiovascular collapse in the patient.[1]

Finally, transient left ventricular outflow tract (LVOT) gradient has also been associated with extreme adrenergic discharge (as might be due to pheochromocytoma) in patients with TTS,[7] mostly in those with an apical ballooning pattern.[6] Importantly, inodilators (including levosimendan) and IABP should also be avoided in TTS patients with an emerging LVOT gradient, potentially suggesting a temporary left ventricular assist device as the sole management of hemodynamic compromise in these patients.[6] Interestingly, transition of wall motion abnormalities among different myocardial regions (from basal to apical, etc.) was previously suggested in the setting of TTS episodes associated with pheochromocytoma.[2,3] This form of specific TTS episode (characterized by rapidly alternating morphological patterns) was previously termed "fast wandering TTS."[7,8] Therefore, rapid evolution of significant LVOT gradient might be quite possible during the course of pheoc hromocytoma-induced TTS regardless of initial morphological patterns (basal, apical, etc.). This warrants frequent evaluation of wall motion abnormalities and LVOT velocity on echocardiogram for the timely diagnosis and management of LVOT gradient in these patients. Accordingly, did the authors detect any transition of wall motion abnormalities with or without temporary LVOT gradient in their patient?

In summary, the co-existence of pheochromocytoma and TTS might arise as life-threatening yet potentially overlooked clinical association.[1-3] Therefore, clinicians should keep this co-existence in mind for diagnosis, risk-stratification, and management of both conditions (and their potential complications) in a timely and proper manner.[1-3]

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