



What triggers Takotsubo in cases without an obvious trigger?

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We read with interest the article by Mori *et al.* about a 64-year-old female with a history of rheumatoid arthritis starting 8 years earlier, who developed Takotsubo syndrome (TTS) 3 days prior to admission for chest pain and dyspnoea (1). No definite trigger for TTS was detected but cardiac magnetic resonance imaging (cMRI) suggested the presence of pericarditis, which was assumed not to have been causative (1). We have the following comments and concerns.

The main disadvantage of the report is that the trigger of TTS could not be specified. In the vast majority of the cases, TTS is triggered by external or internal stress associated with a catecholamine storm, overactivity of sympathetic nerves, or microvascular dysfunction in the setting of systemic inflammation (2). Since pericardial effusion in the presented patient resolved without application of steroids, the authors regarded pericardial effusion as a complication of TTS rather than the cause of TTS. Thus, it remains unclear what triggered TTS in this particular patient. Identification of the trigger, however, is important as the trigger should be eliminated. This is of paramount importance as TTS can recur. To detect the triggering stress, it can be helpful to take the individual history carefully and to look for events patients sometimes may not be able to talk about easily. This is particularly the case for psychological stress due to fearful, painful, or humiliating experiences. In such cases, patients need to be enabled to re-establish confidence in their habitual environment and in their caregivers. In case a patient still cannot open himself to give access to his inner world, relatives or close friends need to be consulted.

A second point requiring discussion is the diagnosis of

pericarditis (1). Pericarditis was diagnosed by cMRI but no puncture of the pericardial effusion was carried out. Thus, the diagnosis remains speculative and is not based upon strong indicators for the disease. Since the patient was under an effective immunosuppressive therapy with methotrexate at the time TTS developed, it is rather unlikely that rheumatoid arthritis caused pericarditis. Since TTS is characterised by systolic dysfunction and since systolic dysfunction is frequently associated with pericardial effusion, it is more likely that pericardial effusion developed as a complication of heart failure rather than due to inflammation. In this respect, we miss the values of the parameters fractional shortening and ejection fraction, key indicators for normal or abnormal systolic function (3). The ejection fraction could have been determined not only on echocardiography but also on cMRI (4).

Another shortcoming of the report is that reference limits for the parameters listed in Table 1 were not provided. As a consequence, it is difficult to figure out if there was disease activity of rheumatoid arthritis at the time of TTS or not and if TTS was associated with elevated proBNP. According to our reference limits gamma-glutamyl peptidase was elevated and it would be interesting to know if this reflects right heart failure with liver congestion. It should be also indicated at which timepoint after the onset of TTS these parameters were determined. Particularly parameters indicating heart failure may fluctuate considerably with the stage of the disease.

It would be also useful to mention if the patient received cardiac therapy for TTS or not. In case she received heart failure therapy, it is conceivable that heart failure therapy contributed to the resolution of the pericardial effusion.

Overall, this interesting case could be more meaningful if more effort had been undertaken to detect the trigger of TTS, if the nature of pericardial effusion would have been realised, if reference limits of serum parameters would have been provided, and if it would have been mentioned if the patient received cardiac therapy for TTS or not.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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