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Letter to the editor

Takotsubo syndrome in Parkinson's disease requires extensive diagnostic workup

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Letter to the Editor,

We read with interest the article by Lazaros et al about a 79-year-old male with Parkinson's disease who was admitted for chest pain and dyspnea during exercise, dizziness, and near-syncope.[1] After workup with a clinical examination, ECG, echocardiography, and coronary angiography, Takotsubo syndrome (TTS) was diagnosed.[1] TTS was described as atypical, as the ECG was almost normal.[1] We have the following comments and concerns.

The main shortcoming of the paper is that no clear trigger for the occurrence of TTS could be identified. However, the patient presented with dizziness and near-syncope on admission and clinical manifestations occurred after physical stress.[1] As Parkinson's disease may be associated with other CNS disorders such as epilepsy, we should know whether the patient experienced seizures before admission or had a history of epilepsy. Seizures are the second most frequent cerebral trigger of TTS and thus need to be excluded in each TTS case in which the trigger remains unknown.[2] Identifying the trigger in TTS is crucial, as TTS may recur and may be accessible to treatment.

We should also be informed about the stage of Parkinson's disease in the index case and whether there was already cognitive impairment or dementia as well as frequent complications of Parkinson's disease. Assuming that the patient had dementia already, it is conceivable that TTS was triggered by an episode of severe fear due to excitation, hallucination, or confusion.

Parkinson's disease may be also associated with migraine,[3] and migraine has been reported to trigger TTS in single cases.[2]

As the patient presented with exceptional dyspnea and dizziness, we should be informed if pulmonary embolism was excluded.[4]

The fact missing in this report is the current medication. Which type of medication did the patient receive for Parkinson's disease and which type of drugs were administered for arterial hypertension? Knowing the current medication is crucial, as it may be responsible for arterial hypotension in admission and as it may have the potential to trigger TTS[5] For example, TTS has been reported after entacapone intake[6] supporting the idea that COMT inhibition increases the bioavailability of catecholamines triggering TTS. With regard to this, we should also know whether the anti-Parkinson medication was effective or ineffective and associated with any side effects.

Additionally, it would be interesting to know the proBNP serum values, as the ejection fraction (EF) was 25%. As TTS mimics myocardial infarction also with regard to laboratory findings, we should also know whether creatine kinase was elevated or not.

Finally, nothing is reported about the outcome of TTS in the index patient. ECG recovers maximally after 10 weeks and echocardiography becomes normal within maximally 6 weeks. We should know when systolic function and the wall motion abnormalities recovered completely in this patient.

Overall, this interesting case could be more meaningful if epilepsy, migraine, pulmonary embolism, and drugs were excluded as triggers of TTS and if the outcome was described. However, the report underscores that patients with TTS should undergo extensive investigations to detect the trigger of TTS.

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