

Letter to the Editor

Myasthenic Crisis Must Be Confirmed Before Accusing It as the Cause of Takotsubo Syndrome

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In a recent article, Ho *et al.*, reported about a 59yo Chinese female with myasthenia gravis (MG), diagnosed one month prior to presentation and being treated with prednisolone and pyridostigmine (Ho, R.Y.J. *et al.*, 2019). She developed Takotsubo cardiomyopathy (TTC) during a presumed myasthenic crisis (Ho, R.Y.J. *et al.*, 2019). The study raises the following comments and concerns.

We do not agree that a myasthenic crisis triggered TTS in the presented patient. It is strongly conceivable that there was no myasthenic crisis at all. The patient developed respiratory distress during coronary angiography (Ho, R.Y.J. *et al.*, 2019). It cannot be excluded that respiratory insufficiency was a clinical manifestation of TTS. Furthermore, a myasthenic crisis is characterised by mydriasis and tachycardia but the presented ECG does not show tachycardia. We should know if there was miosis or mydriasis during coronary angiography. We should also be informed about the pyridostigmine dosage the patient was taking at the time of TTS, to exclude a cholinergic crisis, characterized by miosis and bradycardia (Adeyinka, A., & Kondamudi, N.P. 2019). Since a myasthenic crisis may be associated with high titers of post-synaptic acetylcholine-receptor (AchR-ab) (Kanzato, N. *et al.*, 1999), we also should be informed about the patient's AchR-ab titer. Knowing the AchR-ab titer is crucial, even if the clinical presentation outside a crisis does not correlate with the AchR-ab titers (Eymard, B. 2009).

Assuming that the patient had a myasthenic crisis, we should know the precipitant of the crisis. Frequent precipitants include physical stress, aspiration pneumonia, infections, perimenstrual state, pregnancy, sleep deprivation, surgery, environmental stress, emotional stress, pain, temperature extremes, or tapering of immune-modulating medication (Wendell, L.C., & Levine, J.M. 2011). Additionally, MG is associated with anxiety, depression and insomnia (Jordan, H., & Ortiz, N. 2019). Were any of these precipitants present before the occurrence of TTS?

Overall, this interesting case report could be more meaningful if arguments for the presence of a myasthenic crisis were provided, if AchR-ab titers were provided, and if the precipitator of the presumed myasthenic crisis was identified.

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