Comment on: Takotsubo cardiomyopathy, or 'broken-heart syndrome', with concomitant myasthenic crisis

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Dear Sir,

I read with interest the article by Ho et al about a 59-year-old Chinese woman with myasthenia gravis (MG) who was diagnosed one month prior to presentation and treated with prednisolone and pyridostigmine.[1] She developed Takotsubo cardiomyopathy (TC) during a presumed myasthenic crisis.[1] The study raises the following comments and concerns.

I do not believe that a myasthenic crisis triggered TC in the presenting patient. It is conceivable that there was no myasthenic crisis at all, and that the patient developed respiratory distress during coronary angiography.[1] It cannot be concluded that respiratory insufficiency was a clinical manifestation of TC. Furthermore, a myasthenic crisis is characterised by mydriasis and tachycardia, but the patient's electrocardiogram did not show tachycardia. The attending physician should determine 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 TC, to exclude a cholinergic crisis characterised by miosis and bradycardia.[2] Since a myasthenic crisis may be associated with high titres of postsynaptic acetylcholine receptor antibody (AChR-Ab),[3] we should also be informed about the patient's AChR-Ab titre. Knowing this is crucial, even if the clinical presentation outside a crisis does not correlate with the AChR-Ab titres.[4]

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 medications.[5] Additionally, MG is associated with anxiety, depression and insomnia.[6] Were any of these precipitants present in this patient before the occurrence of TC?

Overall, this interesting case report could be more meaningful if arguments for the presence of a myasthenic crisis and AChR-Ab titres had been provided, and if the precipitator of the presumed myasthenic crisis had been identified.

Yours sincerely,

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