Authors' reply

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Dear Sir,

We thank the letter writer for his response to our case report.[1] It led to further thought being put into this case among our authors and generated a robust discussion.

To re-illustrate the clinical picture, a 59-year-old Chinese woman had presented with throat tightness and worsening weakness. Pertinent points to note in her history include: (a) a main complaint of worsening dysphagia and throat tightness over the past two days; (b) inability to lift up her neck; and (c) orthopnoea. The listed symptoms all point to an impending myasthenic crisis in view of the rapid worsening of symptoms and the recent diagnosis of seropositive generalised myasthenia gravis (MG) one month prior. The orthopnoea could also be explained by the dependence on gravity to allow diaphragmatic expansion during an MG flare.

The decision at the emergency department, after consultation with the neurology consultant, was that there was no need for urgent intubation because the patient's airway was patent, there were no signs of respiratory distress, and the arterial blood gas did not show Type 2 respiratory failure. At that point, ST-elevation myocardial infarction necessitated activation of the catheterisation lab. We believe the myasthenic crisis occurred intra-catheterisation, when the patient acutely developed severe dyspnoea and appeared to be in respiratory distress just prior to the left ventriculogram.

Myasthenic crisis is a clinical diagnosis,[2] and the constellation of symptoms and signs up to the point of respiratory distress intra-catheterisation seem to suggest that it was the most likely cause. Admittedly, the patient's pupils were not examined for miosis or mydriasis. There was also no concomitant tachycardia during the episode. Even so, the clinical picture of acute progressively worsening weakness with bulbar symptoms over 48 hours, culminating in severe respiratory distress, suggests that myasthenic crisis was most likely.

Cholinergic crisis, which may be a side effect of anticholinergic medication, is a differential but was far less likely in this case. The patient had a dose of 60 mg three times a day of pyridostigmine, well below the recommended limit, a total daily dose of 960 mg. Furthermore, she had not presented with the other common features in a cholinergic crisis, lacrimation, defecation, emesis and urination.[3]

Additionally, acetylcholine receptor antibodies (AChR-Ab) titres were not assessed at the point of admission even with the suspicion of myasthenic crisis. The clinical utility would have been limited since the clinical picture already suggested a myasthenic flare in a patient with known seropositive MG. Trending such titres was also shown to be less useful for assessing treatment response.[4] Assessing AChR titres would perhaps have been more useful if the patient had not been diagnosed with myasthenia gravis.[5]

Lastly, we agree that identifying the precipitant of the myasthenic crisis is important, but this was left out of the case report. Revisiting the patient's history, following subsequent extubation, there was no recent tapering of her immunosuppressive medications prior to her admission. She had also been compliant to her pyridostigmine and prednisolone medications prescribed the month before. Thus, the identified precipitant for the myasthenic crisis was that of emotional stress surrounding the patient's work and her recent diagnosis of MG.

In summary, we defend the diagnosis of myasthenic crisis based on the constellation of our patient's symptoms, which were in keeping with a myasthenic crisis, even without performing AChR titres. The most likely precipitant for the myasthenic crisis was emotional stress.

Yours sincerely,

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