To the Editor: An 89-year-old woman was admitted with a 3-day history of dysphagia and lower jaw twitching. She had a history of hypertension, diabetes mellitus, surgically corrected left proximal humeral fracture, and right C5 dermatome postherpetic neuralgia. The jaw twitching had caused dysphagia with an inability to drink liquids. Physical examination confirmed the persistent lower jaw myoclonus (Supplementary Video; http://links.lww.com/CM9/A48). The patient was fully conscious and had no twitching or nystagmus in any of her other extremities. The results of neurological examination and biochemical analysis (including complete blood profile, electrolyte level, random glucose concentration, renal function test, and liver function test) were all within the normal range. Computed tomography of the brain was unremarkable and an electroencephalogram showed no evidence of seizure. A careful review of her prescribed medications revealed the following: the patient complained of allodynia over the right C5 dermatome around 3 months prior and was prescribed gabapentin. The dosage of gabapentin was increased to 300 mg total dissolved solids (TDS) around 2 months prior. Creatinine levels were normal but her glomerular filtration rate, as estimated by the Cockcroft-Gault equation, was 36 mL/min. Since the patient’s relatives supervised her medication intake, overdosage was not possible. Gabapentin was discontinued and replaced with pregabalin 50 mg BD and the patient was prescribed valium 2 mg TDS transiently. The jaw myoclonus subsided on the second day. After a consultation with a speech therapist, the patient resumed oral intake and remained tolerant of a normal diet. Upon review after 1 month, the patient remained free from jaw myoclonus.

Common causes of acute dysphagia in elderly patients include acute stroke and delirium. Reversible causes, such as jaw myoclonus, are rarely reported. Myoclonus refers to a sudden, brief, shock-like involuntary movement. It can be caused by a hypoxic brain injury, metabolic imbalance, focal brain lesion, medication, or viral infection. Our patient scored a six on the Naranjo Adverse Drug Reaction Probability Scale, indicating that gabapentin was the probable culprit of an adverse drug reaction. From the literature, 0.1% to 12.5% of patients using gabapentin can suffer from myoclonus. Gabapentin is cleared by the kidneys and, hence, its half-life can be prolonged to over 20 h in patients with chronic kidney disease (CKD), compared to 5 to 8 h in patients with normal renal function. Our patient suffered from stage three CKD, which is likely to have contributed to the development of the gabapentin-induced toxicity. The patient had been prescribed a dose of gabapentin appropriately adjusted according to her CKD. This is our second experience of an appropriate dose of gabapentin leading to myoclonus in a patient with CKD. Previous reports on gabapentin-induced myoclonus are usually multifocal (ie, occurs asynchronously in at least two limbs). Other medications that may induce jaw myoclonus include cefepime. From the literature, gabapentin-induced myoclonus is self-limiting. Low-dose benzodiazepine or renal replacement therapy should be considered in cases of severe myoclonus.

In conclusion, clinicians should be aware of reversible causes of acute dysphagia including jaw myoclonus. Gabapentin could be a cause of drug-induced jaw myoclonus.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Reversible dysphagia due to gabapentin-induced jaw myoclonus

Chun-Him Hui1, Jackson Ka-Chun Leung2, Richard Shek-Kwan Chang3, Yat-Fung Shea4

1Respiratory Division, Department of Medicine, Queen Mary Hospital, The University of Hong Kong, Hong Kong, China; 2Medical Oncology Division, Department of Medicine, Queen Mary Hospital, The University of Hong Kong, Hong Kong, China; 3Neurology Division, Department of Medicine, Queen Mary Hospital, The University of Hong Kong, Hong Kong, China; 4Geriatrics Division, Department of Medicine, Queen Mary Hospital, The University of Hong Kong, Hong Kong, China.
Conflicts of interest
None.

References