

CASE REPORT



## Uncontrolled recurrent myasthenia gravis exacerbations secondary to chronic gabapentin use

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### ABSTRACT

Gabapentin is an anticonvulsant medication that reduces synaptic transmission by decreasing presynaptic voltage-gated Ca<sup>2+</sup> and Na<sup>+</sup> channels. It is approved to treat focal seizures but also used to treat post-herpetic and neuropathic pain. Although uncommon, there have been three reported cases of myasthenia gravis exacerbation associated with gabapentin in the literature. We present a patient with uncontrolled recurrent myasthenia gravis exacerbations secondary to chronic gabapentin use and provide a review for the three published cases.

### ARTICLE HISTORY

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### KEYWORDS

Gabapentin; myasthenia gravis

### 1. To the editor

Gabapentin is an anticonvulsant medication that reduces synaptic transmission by decreasing presynaptic voltage-gated Ca<sup>2+</sup> and Na<sup>+</sup> channels. It is approved to treat focal seizures but also used to treat post-herpetic and neuropathic pain[1]. Although uncommon, there have been three reported cases of MG exacerbation associated with gabapentin in the literature [2–4]. We present a patient with uncontrolled recurrent MG exacerbations secondary to chronic gabapentin use.

### 2. Case report

A 77-year-old man with medical history of MG diagnosed 1 year ago with multiple MG exacerbations for the past year returned for neck weakness, dysphagia, intermittent diplopia, and arm weakness consistent with his usual symptoms of MG exacerbations. He has had frequent MG exacerbations while treated with pyridostigmine, with the most recent exacerbation earlier the same month, another episode 2 months prior, and multiple exacerbations almost monthly during the past year. Prior hospitalizations for his MG exacerbations resolved with combinations of intravenous methylprednisolone, plasmapheresis, and intravenous immunoglobulin.

On admission, he was found to have decreased strength in all extremities, diplopia, and dysphagia. AChR binding antibody was elevated to 3.9 nmol/L (positive>0.5 nmol/L). Computed tomography of the chest was negative for thymoma. During the hospital course, he developed worsened respiratory distress consistent with MG crisis requiring intubation. He was treated with intravenous methylprednisolone, mycophenolic acid, an increased dose of pyridostigmine, and five

rounds of plasmapheresis, which resolved his acute MG crisis. A review of home medications revealed patient had been taking gabapentin for the past year for neuropathic pain. He did not have risk factors known to trigger MG crisis such as infection, stress, or recent surgery. The recurrent MG exacerbations were speculated to be secondary to his gabapentin use. His gabapentin was discontinued given reports [2–4] of MG exacerbations associated with gabapentin. At discharge, no dose changes to his original home medication pyridostigmine were made. Symptoms of MG exacerbations were monitored. At the six months follow-up, no further exacerbations were reported.

### 3. Discussion

We report a case of recurrent MG exacerbation in a patient with chronic gabapentin use. The patient's uncontrolled recurrent MG exacerbations while taking gabapentin coupled with the resolution of MG exacerbations after discontinuing gabapentin led us to believe that gabapentin contributed to this patient's uncontrolled MG exacerbations. Other medications with the potential to exacerbate weakness include certain local anesthetics, beta-blockers, calcium channel blockers, antiepileptics (phenytoin and gabapentin), phenothiazines, diuretics, procainamide, magnesium, and opioids. In normal patients, these effects are usually of no consequence, but in patients with MG, they may exacerbate muscle weakness presenting as respiratory or bulbar weakness. This can be especially deleterious in the presence of residual anesthetic agents[5].

To our knowledge, this case represents the 4th case in the literature reporting gabapentin's deleterious effects on MG. In 2000, gabapentin unmasking of MG was first

**Table 1.** Cases of myasthenia gravis exacerbation associated with gabapentin.

Case Number; Authors; Published year	Age	Gender	Indication for Gabapentin	Onset of MG exacerbation after starting Gabapentin	Clinical Symptoms	Treatment
1; Boneva et al; 2000	67	Female	Severe calf cramps	2 month	Slurring of speech, dry mouth, ptosis, chewing and fatigue in speech	Discontinuing gabapentin and received pyridostigmine
2; Scheschonka et al; 2002	64	Female	Post-herpetic neuralgia	1 week	Increased weakness	Discontinuing gabapentin
3; Sheen et al; 2010	65	Male	Numbness in hands and thighs	1 week	Dysphagia and vomiting, trouble talking, impaired tongue movement, chest tightness on swallowing	Discontinuing gabapentin and received pyridostigmine and methylprednisolone
4; Chien et al; 2019	77	Male	Neuropathic pain	Unknown as patient had recurrent MG exacerbation for 1 year while on gabapentin	Neck weakness, dysphagia, intermittent diplopia, arm weakness, difficulty breathing	Discontinuing gabapentin and received intravenous methylprednisolone, mycophenolic acid, increased dose of pyridostigmine, and 5 rounds of plasmapheresis

MG: Myasthenia Gravis.

reported in a patient without previous neuromuscular symptoms (Table 1)[2]. The same findings were reproduced in a rat model suffering from experimental autoimmune MG[2]. Subsequently, a report described a patient with antibody-positive MG complicated by herpetic neuralgia treated with gabapentin with worsened weakness that resolved with discontinuing gabapentin (Table)[3]. Recently, Sheen et al. reported a patient who was diagnosed with MG after treated with gabapentin for dysarthria, dysphagia, paresthesia, and muscle weakness in arms and legs (Table)[4]. In summary, while gabapentin exacerbation of MG is exceedingly rare, providers should consider it as a possible side effect of gabapentin.

### Authorship

All authors had full access to data during design and drafting of this manuscript. All authors were responsible for conception and design. JLC drafted the manuscript, VB revised the article, and HRM gave final approval of the manuscript.

### Disclosure statement

No potential conflict of interest was reported by the authors.

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