<Response to the reviewers' comments>

Reviewer 1

It was a very valuable case. I have some topics that needed to be addressed:

When exactly did the patient's complaints begin?

(Reply) Thank you for your comment. Please refer to chief complaints section in case Presentation (Page 6, lines 8-9): "The symptom developed 3-4 months before the current visit."

Did the patient describe neurogenic claudication?

(Reply) Thank you for your helpful comment. Please refer to Chief complaints section in Case Presentation (Page 6, lines 9-10): "The patient denied neurogenic claudication or cramping pain of the lower extremities."

Was there a vascular risk factor in the patient's history? Did the patient have a history of diabetes mellitus and hypothyroidism?

(Reply) Thank you for your insightful comment. Please refer to the history of past illness section in case presentation (Page 6, lines 25-26): "The patient's hypertension and diabetes were controlled by medications and other vascular risk factors or thyroid diseases were absent.

"

Could you describe the patient's neurological examination in detail?

(Reply) Thank you for your critical question. Please refer to Physical examination section in Case Presentation (Page 7, lines 3-6): "Rigidity was absent and deep tendon reflexes, including both the knee jerk and ankle jerk reflexes, were normal. On straight leg raise test, the patient could achieve up to 80 and 90 degrees with the right and left leg, respectively. Mild tenderness was found at the lumbar

paraspinal muscle area; however, both legs had normal muscle strength.

"

How was muscle strength?

(Reply) Thank you for your valuable question. Please refer to physical examination section in case presentation (Page 7, lines 4-5): "Mild tenderness was found at the lumbar paraspinal muscle area, however, the muscle strength was normal in both legs."

How was serum creatine kinase level?

(Reply) Thank you for your helpful question. The patient had normal kidney function. Please refer to laboratory testing section in case presentation (Page 7, line 25): "Baseline blood urea nitrogen and creatinine levels were 15 and 1.08 mg/dL respectively."

When were imaging examinations performed?

(Reply) Thank you for your insightful question. Please refer to the imaging examination section in case presentation (Page 7, lines 9-10 and 12-14): "Results from the magnetic resonance imaging (MRI) examination five years ago in January 2014 during the first diagnosis of TN revealed no abnormalities around the trigeminal nerve… A lumbar spine computed tomography (CT) scan, performed two years ago in June 2017 as part of radiological examination for back pain, showed multilevel bulging discs…"

Did he have a sequence of MR diffusion?

(Reply) Thank you for your critical question. Please refer to the imaging examination section in case presentation (Page 7, lines 12-13): "Diffusion image was not obtained at MRI as the sequence was not included in our cranial nerve MRI protocol."

Why was MRI not performed for lumbar imaging?

(Reply) Thank you for your valuable question. Please refer to the discussion (Page 10, lines 9-12): "This case has a few limitations. First, a lumbar MRI would have provided a more accurate delineation of the back pain and leg weakness. However, the patient was a low-income worker who was covered under the Medical Aid program in Korea, and therefore, lumbar MRI was refused by the patient because of cost."

Why was the EMG not performed?

(Reply) Thank you for your helpful question. Please refer to the Discussion (Page 10, lines 12-15): "Second, an electromyography (EMG) was not performed when leg weakness was present. However, the patient's symptom did not correlate with physician's assessment of radiculopathy and the patient was reluctant to undergo a more aggressive work-up because of additional cost."

A rare side effect of milnacipran is hyponatremia. Could it have potentiated the effect of oxcarbazepine?

(Reply) Thank you for your insightful comment. Please refer to the Discussion (Page 10, lines 15-20): "Lastly, the concomitant medication, milnacipran, could have been the etiology, as hyponatremia is a rare adverse effect of the drug. Nevertheless, hyponatremia is more common among consumers of anticonvulsant drugs than in those on antidepressant medications including milnacipran [12]. As both hyponatremia and unsteady gait improved dramatically after discontinuation of oxcarbazepine, we suspected that the effect of milnacipran on hyponatremia was negligible in this case."

Reviewer 2

- Title: Should be modified.
- Abstract: satisfactory.
- Introduction: Well written.
- Case report: satisfactory.
- Discussion: Well described and the differentiation from spinal canal stenosis well documented.
- Conclusion: Useful. Interesting case report.

Reply:

Thank you for your comment. We have modified our title to "**Oxcarbazepine for trigeminal neuralgia may induce lower extremity weakness: a case report.**"