

Background

- Neuropathic pain (NP) is common after spinal cord injury (SCI). Hyperexcitable nociceptors are hypothesized to mediate SCI-induced NP [1].
- Calcitonin gene-related peptide (CGRP) is a neuropeptide expressed in C-fibers and A δ afferents transmitting pain to the dorsal root ganglion [2,3].
- Animal and human studies [2,4] demonstrate new aberrant immunoreactive CGRP-fiber sprouting within the dorsal horn after SCI, facilitating nociceptive input [4,5].
- In animals with SCI, this sprouting is associated with allodynia and hyperalgesia [2].
- CGRP inhibitors (CGRPi) have recently been FDA-approved for migraine prevention.

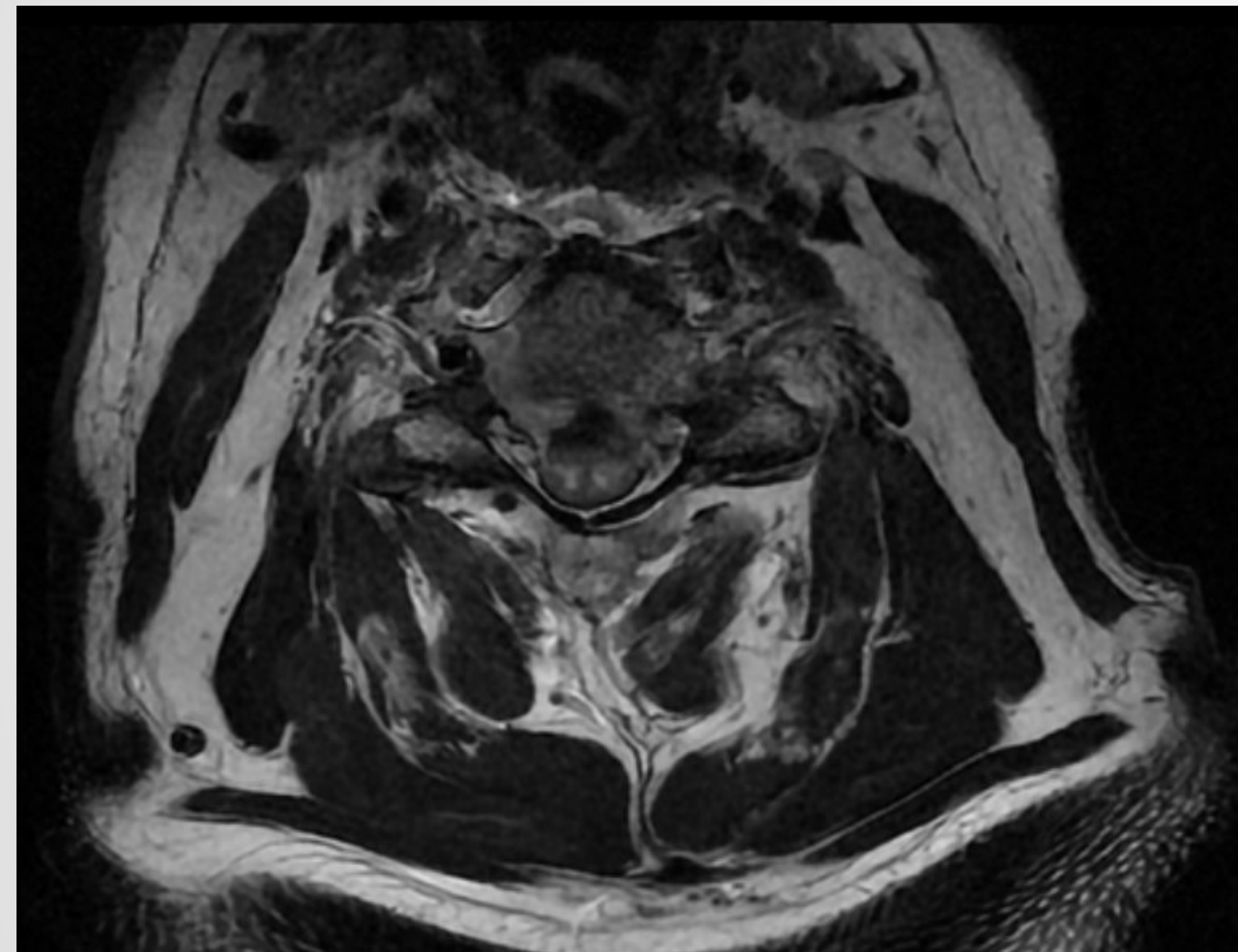


FIGURE 1. T2-weighted sagittal FRFSE (A) and axial FSE MRI sequences, revealing severe spinal canal stenosis at the C3-C4, C4-C5, and C5-C6 levels, and increased cord signal from the C3-C5 level, suggestive of cord contusion.

Results

- Patient-reported pain scores via DVPRS revealed reductions from 6.2/10 pre-CGRPi to 5.4/10 post-CGRPi initiation.
- The patient tolerated opioid weaning, and opioid requirements declined from 55.3 to 35.3 MME.
- The patient reported reduced migraine frequency and gradual NP improvement.
- Allodynia improved and the patient was able to tolerate cares and E-Stim.
- Six weeks post-CGRPi initiation the patient reported the “best day” since his injury, in terms of pain.

Case Description

A 79-year-old male with history including migraines and C3 AIS C quadriplegia secondary to a fall with C4 vertebral fracture experienced insidious onset, progressive upper extremity NP following his injury. The quality of pain was electrical and burning, described as “grabbing an electric wire fence.” Severe associated allodynia and hyperalgesia limited the patient’s ability to tolerate nursing cares (i.e. repositioning, turns, bathing), and therapy modalities (i.e. range of motion, electrical stimulation). MRI of the cervical spine ruled out syringomyelia. The patient was treated with scheduled acetaminophen, pregabalin, and duloxetine. Numerous opioid analgesic regimens were trialed (including oxycodone, tramadol, hydromorphone, and topical buprenorphine), either without significant analgesia or with significant sedation. Given the patient’s history of migraines, erenumab, a once monthly injectable CGRPi, was started.

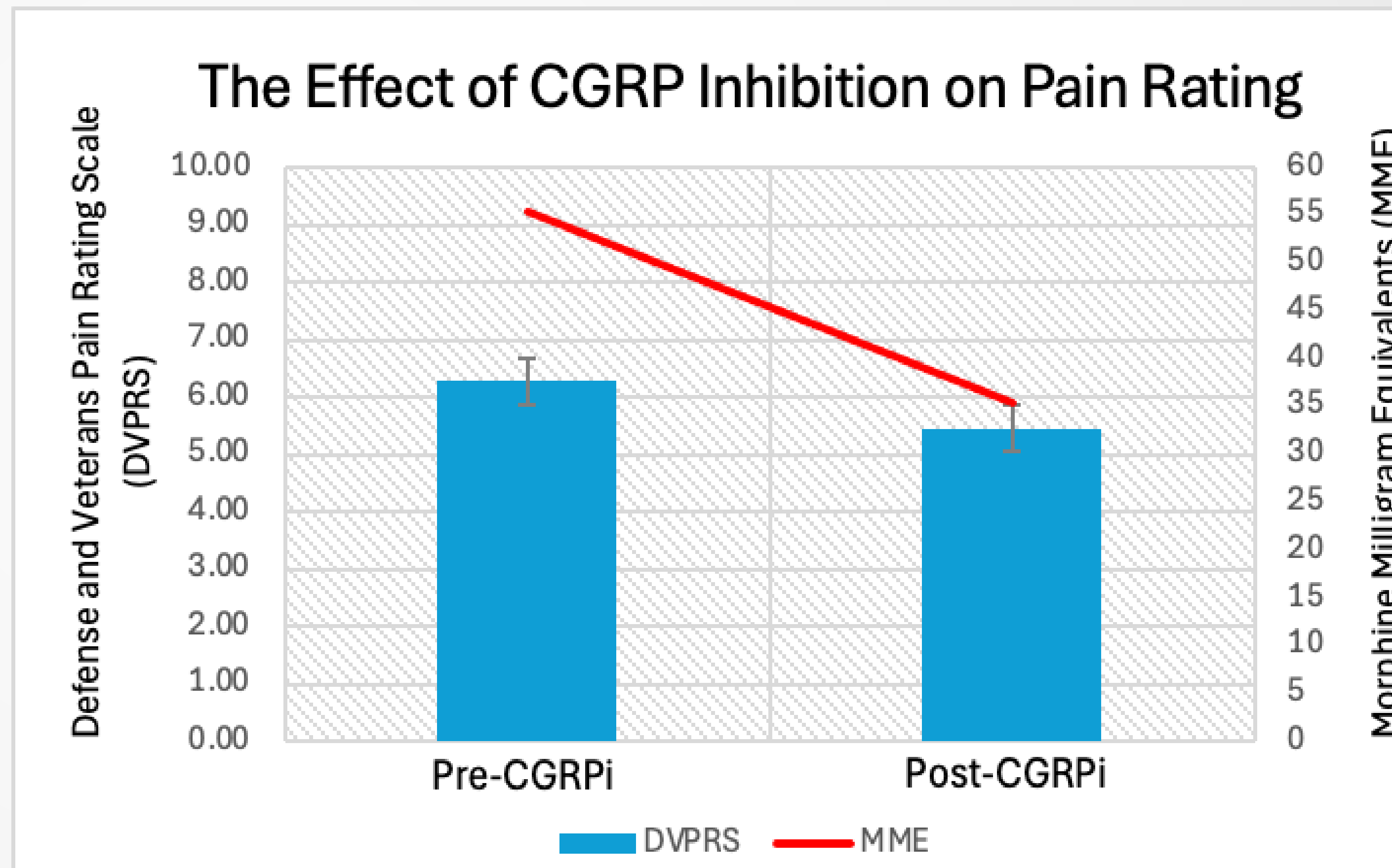


FIGURE 2. Primary Y-axis: graphical representation of mean patient reported pain scores during admission before (n=316) and after (n=400) CGRP initiation, including standard errors of the mean. P-value = 1. Secondary Y-axis: Daily MME pre- and post-CGRPi initiation during admission.



Conclusions

- Given the extensive expression of CGRP, antagonism of CGRP may aid in managing pain states other than migraine.
- These findings support further investigation of CGRP inhibition as a therapeutic strategy for SCI-associated NP.

References

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