

General

Pharmacologic Management of Neuropathic Pain After Lumbar Spine Surgery: A Case-Based Review

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Chronic neuropathic pain following lumbar spine surgery remains a common and challenging clinical problem in pain medicine, often persisting despite surgical intervention and injections. This case-based educational review describes a 62-year-old man with multiple prior lumbar surgeries who developed persistent bilateral lower-extremity neuropathic pain consistent with persistent pain after spinal surgery. Initial treatment with gabapentin, titrated to therapeutic doses, resulted in minimal clinical improvement, prompting transition to pregabalin with moderate symptom relief. The addition of tramadol for breakthrough pain further improved functional status and sleep quality. Through this clinical scenario, key pharmacologic principles are reviewed, including the mechanisms of action, pharmacokinetics, safety considerations, and practical differences between gabapentin and pregabalin, as well as the role of tramadol as a dual-mechanism analgesic for mixed neuropathic and nociceptive pain. This teaching case is of particular educational value for trainees in orthopedics, pain management, anesthesiology, and primary care, where stepwise pharmacologic decision-making and longitudinal management of complex post-surgical pain syndromes are frequently encountered.

TEACHING CASE

A 62-year-old man presents to a chronic pain clinic with persistent low back pain and bilateral lower-extremity neuropathic symptoms. His history is notable for multiple prior lumbar spine surgeries, including L4–L5 and L5–S1 decompression and fusion procedures performed over the past decade. Despite surgical intervention, he continues to report burning, tingling, and electric-like pain radiating down both legs.

The patient describes his pain as constant, rated 7/10 on average, with worsening symptoms at night that interfere with sleep and daily functioning. Physical examination demonstrates decreased sensation in a bilateral L5 distribution without new motor deficits. Imaging shows postsurgical changes without clear evidence of recurrent compressive pathology.

He is initially started on gabapentin 300 mg three times daily, which is gradually titrated to 600 mg three times daily over several weeks. Despite appropriate titration, he reports minimal improvement in pain severity or function. Given inadequate response, gabapentin is discontinued and he is transitioned to pregabalin 100 mg three times daily, resulting in moderate improvement in neuropathic symptoms.

For breakthrough pain, tramadol 50 mg every 6 hours as needed is added, leading to further functional improvement

and improved sleep. He tolerates the regimen without significant adverse effects.

TEACHING QUESTIONS

QUESTION 1

What is the primary mechanism of action of gabapentin in the treatment of neuropathic pain?

- A. Direct antagonism of NMDA receptors
- B. Inhibition of serotonin reuptake
- C. Binding to the $\alpha 2\delta$ subunit of voltage-gated calcium channels
- D. Interaction with GABA-A receptors

QUESTION 2

Which statement best describes the mechanism of action of pregabalin?

- A. Identical to gabapentin but with higher potency and more predictable absorption
- B. Selective μ -opioid receptor agonism
- C. Inhibition of sodium channel inactivation
- D. Direct activation of spinal GABA receptors

QUESTION 3

Which patient factor is most important to consider when prescribing gabapentin or pregabalin?

- A. Hepatic function
- B. Renal function
- C. Body mass index
- D. Serum albumin level

QUESTION 4

Which adverse effect is most commonly associated with gabapentinoids and should be routinely discussed with patients?

- A. QT prolongation
- B. Sedation and dizziness
- C. Severe hepatotoxicity
- D. Agranulocytosis

QUESTION 5

Which mechanism best explains tramadol's analgesic effect in this patient?

- A. Pure μ -opioid receptor agonism
- B. Cyclooxygenase inhibition
- C. Combined weak μ -opioid agonism and inhibition of serotonin and norepinephrine reuptake
- D. NMDA receptor antagonism

ANSWERS AND DISCUSSION

QUESTION 1

Correct Answer: C

Gabapentin binds to the $\alpha 2\delta$ subunit of voltage-gated calcium channels, reducing calcium influx at presynaptic terminals and decreasing release of excitatory neurotransmitters such as glutamate and substance P.¹⁻³ This mechanism reduces neuronal hyperexcitability associated with neuropathic pain.

QUESTION 2

Correct Answer: A

Pregabalin shares the same primary mechanism as gabapentin by binding to the $\alpha 2\delta$ subunit of voltage-gated calcium channels. However, pregabalin has greater potency, faster onset, and more predictable pharmacokinetics, which can translate into improved clinical response.³⁻⁵

QUESTION 3

Correct Answer: B

Both gabapentin and pregabalin are renally excreted with minimal hepatic metabolism. Dose adjustment is essential in patients with chronic kidney disease to reduce the risk of accumulation, sedation, and neurotoxicity.^{6,7}

QUESTION 4

Correct Answer: B

The most common adverse effects of gabapentinoids include sedation, dizziness, and peripheral edema.⁸ These effects are dose-dependent and more pronounced in older adults and those with renal impairment.

QUESTION 5

Correct Answer: C

Tramadol provides analgesia through a dual mechanism: weak μ -opioid receptor agonism combined with inhibition of serotonin and norepinephrine reuptake, enhancing descending inhibitory pain pathways.^{9,10} This multimodal mechanism makes it particularly useful for mixed nociceptive-neuropathic pain states.⁹⁻¹²

EDUCATIONAL DISCLAIMER

This case is a fictional teaching example created solely for educational purposes and does not represent a real patient. It was designed as a teaching case to illustrate general principles of pharmacologic management of chronic neuropathic pain and should not be construed as medical advice. Clinical decisions must be individualized and guided by current evidence and professional judgment.

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