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Lacosamide-Induced Visual and Auditory Hallucinations in an Elderly Patient Treated for Refractory Trigeminal Neuralgia: A Case Report

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Background: Trigeminal neuralgia (TN) is a severe neuropathic facial pain syndrome that mainly affects older adults. Carbamazepine and oxcarbazepine are standard therapies, but their tolerability is often limited in the elderly. Lacosamide is a potential alternative through its modulation of slow sodium channel inactivation, yet its neuropsychiatric adverse effects remain poorly documented.

Case Report: A 71-year-old man with right-sided TN refractory to carbamazepine and intolerant to oxcarbazepine was started on lacosamide. Pain decreased 30% to 40% at 150 mg twice daily, but daytime visual and auditory hallucinations emerged. Clinical, imaging, and laboratory evaluations were normal. Reducing the dose to 100 mg twice daily resolved the hallucinations while maintaining partial pain control.

Conclusions: Our case highlights a rare dose-dependent neuropsychiatric adverse effect of lacosamide in an elderly patient with TN. Clinicians should monitor perceptual symptoms during titration and consider dose adjustment rather than discontinuation when such effects occur.

Key words: Trigeminal neuralgia, lacosamide, hallucinations, elderly, neuropsychiatric adverse effects

BACKGROUND

Trigeminal neuralgia (TN) is a debilitating neuropathic syndrome marked by brief, paroxysmal, electric shock-like facial pain along one or more branches of the trigeminal nerve. The incidence increases significantly with age, especially after 50, leading to major functional and psychological burden. First-line treatments, including carbamazepine and oxcarbazepine, act through inhibition of voltage-gated sodium channels, but their side effect profile—such as sedation, hyponatremia, and interactions—often complicates their use in older individuals. Lacosamide, an antiepileptic agent that enhances slow inactivation of sodium channels, offers a mechanistically distinct approach that may provide analgesia with a different safety profile (1). However, its use in TN is not yet well established, and observations of neuropsychiatric adverse effects remain rare.

We describe here a case of lacosamide-induced hallucinations in a 71-year-old patient treated for refractory TN.

CASE PRESENTATION

A 71-year-old retired male civil engineer presented with a 2-year history of right-sided facial pain consistent with classical TN. His attacks were paroxysmal, lasting 30 seconds to 2 minutes, and localized to the maxillary (V2) and mandibular (V3) divisions. The pain was reliably triggered by chewing, speaking, and light palpation of

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the right cheek. He reported significant anticipatory fear of triggering pain and social withdrawal, as he avoided talking and eating in company.

His past medical history included well-controlled hypertension, type 2 diabetes, and a longstanding anxiety disorder. Neurological examination revealed heightened sensitivity (hyperalgesia) and abnormal unpleasant sensations (dysesthesia) in the right V2-V3 area. Reflexes of the trigeminal nerve (corneal, masseter) and facial motor function were normal. No other neurological deficits were found, and cognitive screening was intact.

Magnetic resonance imaging of the brain, including focused sequences on the trigeminal root and brainstem, showed no mass lesion, vascular loop, or demyelinating lesion. Laboratory tests, including electrolytes, renal and liver function, glucose, and inflammatory markers, were all within normal range.

Therapeutically, the patient first received carbamazepine titrated up to therapeutic doses according to clinical guidelines (1,600 mg/d), without meaningful reduction in attack frequency or intensity. A subsequent trial of oxcarbazepine (1,200 mg/d) produced a modest ~20% pain reduction, but was associated with disabling sedation that significantly impaired activities of daily living.

Given the limited response and poor tolerability of standard agents, lacosamide was initiated at 50 mg twice daily and escalated to 100 mg twice daily in one week, with a further increase to 150 mg twice daily planned. At this higher dose, the patient reported a 30% to 40% decrease in pain intensity, improved function, and resumed more normal social behavior. However, within days, he experienced new symptoms: predominantly daytime visual hallucinations (fleeting silhouettes, shadowy shapes) and auditory hallucinations (muffled whispers or indistinct voices), which disrupted his concentration, reading, and interpersonal interactions. Despite these perceptual disturbances, he retained insight without delusions, disorientation, or psychomotor agitation.

Laboratory parameters remained stable, with no metabolic derangement, no hyponatremia, and normal renal and liver function. Electrocardiogram showed no conduction abnormalities. Given the temporal association, the hallucinations were attributed to lacosamide. The dose was reduced to 100 mg twice daily, after which the hallucinations resolved within a matter of days while maintaining partial analgesic benefit. Over 6 months of follow-up, he continued on this maintenance dose with stable partial pain control and no recurrence of perceptual symptoms.

Our case illustrates a rare, but important, neuropsychiatric adverse effect of lacosamide in an elderly patient with TN: daytime visual and auditory hallucinations. While lacosamide's tolerability profile is generally favorable, case reports in epilepsy populations have documented similar phenomena. For example, Gomez-Ibanez et al (2) described reversible psychosis associated with lacosamide, and Pinkhasov et al (3) reported hallucinations and mood disturbances during dose escalation.

The pathophysiological basis for these effects remains speculative. From a pharmacological standpoint, lacosamide selectively enhances slow inactivation of voltage-gated sodium channels, distinguishing it from classical sodium channel blockers. While this mechanism may confer improved tolerability, it may also alter cortical and limbic excitability in susceptible individuals. Elderly patients, particularly those with psychiatric comorbidities or polypharmacy, may represent a population at increased risk for neuropsychiatric adverse effects. In addition, age-related changes, such as increased blood-brain barrier permeability and altered pharmacodynamics, may further increase central nervous system sensitivity to this modulation.

In our patient, 2 features are particularly instructive. First, the adverse effect was dose related: symptoms emerged at 150 mg twice daily, but remitted after reduction. Second, insight was preserved, and symptoms could be reversed without complete discontinuation, allowing retention of analgesia. These observations suggest that neuropsychiatric toxicity does not necessarily mandate drug cessation, but rather careful dose adjustment and monitoring.

From a therapeutic standpoint, lacosamide's partial analgesic effect (30% to 40% reduction) aligns with emerging evidence. Cohort studies (4) and small clinical trials (5) have demonstrated meaningful improvement in pain as well as acceptable tolerability. Despite this progress, lacosamide should not be viewed as a panacea: patient selection is critical, particularly in older individuals with comorbid psychiatric conditions or polypharmacy.

In clinical practice, our case supports several recommendations. Clinicians initiating lacosamide in elderly patients with TN should adopt a cautious titration schedule, routinely inquire about perceptual symptoms during follow-up, and consider dose reduction rather

than systematic drug discontinuation if hallucinations occur. Incorporation of neuropsychiatric monitoring, possibly including baseline and follow-up cognitive or perceptual assessments, may be warranted. Informed consent should include discussion of rare, but potentially distressing, neuropsychiatric adverse effects.

From a research perspective, our report highlights an important knowledge gap. Future studies should aim to identify clinical or pharmacological predictors of lacosamide-induced hallucinations, particularly in nonepileptic populations. Prospective registries and controlled trials focusing on elderly patients with TN are needed to better characterize incidence, risk factors, and reversibility of these adverse effects, and to refine patient selection and dosing strategies.

Importantly, the clinical presentation in our case differs markedly from lacosamide overdose, in which neurological depression, seizures, coma, and cardiovascular abnormalities are predominant. In contrast, our patient developed isolated visual and auditory hallucinations at therapeutic dosing, with preserved insight and complete reversibility after dose reduction, supporting a dose-related neuropsychiatric adverse effect rather than toxic encephalopathy (6). Serum lacosamide concentrations were not measured, as therapeutic drug monitoring is neither routinely recommended nor readily available in clinical practice; this omission represents a limitation of the present report and precludes correlation between plasma levels and the occurrence of neuropsychiatric adverse effects.

CONCLUSIONS

In summary, our case documents lacosamide-induced visual and auditory hallucinations in a 71-year-old patient treated for refractory TN. The symptoms were dose dependent and reversible with dose reduction, allowing continued analgesic benefit. While lacosamide remains a promising alternative for TN in patients intolerant to first-line therapies, clinicians should be vigilant for neuropsychiatric side effects, especially in elderly individuals. Future research should aim to define predictors of such toxicity and optimize therapeutic strategies.

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