



Use of Gabapentin during ECT for Myalgia and Anxiety: Effects on Seizure Activity

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Authors' contributions

This work was carried out in collaboration between all authors. Author SLP performed the literature review, clinical care and writing. Author WHL performed the literature review, editing work and author KPD performed the literature review, writing, editing and clinical care. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aim: The goal was to determine if use of gabapentin could assist with management of myalgia and anxiety in a patient treated with Electroconvulsive therapy (ECT) for depression, without interfering with seizure activity during ECT.

Methods: A patient was scheduled for ECT but was reluctant to continue after completing the first session, due to myalgia and anxiety. Medical history and clinical data were noted. He had comorbid alcohol use disorder, so benzodiazepines were avoided. His response to ECT was followed while taking gabapentin for myalgia, anxiety and alcohol cravings.

Results: Gabapentin did not interfere with seizure intensity and did not reduce seizure duration. The patient experienced complete relief of myalgia within 24 hours and reduced anxiety and

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alcohol cravings while taking gabapentin. He experienced relief of depression through a course of seven ECT sessions.

Conclusion: Gabapentin appears to be a promising adjunct during ECT to manage anxiety and myalgia as it did not interfere with seizure duration or clinical effect on depression. Although randomized, controlled trials must be performed; this case supports its use during ECT for these symptoms.

Keywords: Anticonvulsants; ECT; gabapentin; alcohol use disorder.

1. INTRODUCTION

Electroconvulsive therapy (ECT) is an effective treatment for depression but has side effects, including myalgia and anxiety. Patients' concerns about these side effects often interfere with its implementation. Myalgia as a result of ECT occurs in 8.2% of patients and is one of the most distressing side effects of the treatment [1]. Anticonvulsants may be used to address myalgia, alcohol cravings or anxiety [2]. Most providers follow American Psychiatric Association's recommendations [2] to taper anticonvulsant medications during ECT; however, it may be continued at a low dose when used for anti-epileptic properties [2,3]. Myalgia and anxiety can be treated with benzodiazepines but benzodiazepines are partially or wholly contraindicated in patients with alcohol use disorder. In the case below, post-ECT treatment myalgia was treated successfully with gabapentin. Additionally, gabapentin reduced the patient's anxiety about ECT, increased subjective quality of sleep, and hence removed barriers to adherence. Gabapentin has been used for improvement of insomnia as well as neuropathy [4].

2. PRESENTATION OF CASE

A 56-year old Caucasian male was diagnosed using DSM-IV clinical criteria with treatment-resistant Major Depressive Disorder, recurrent without psychosis and Alcohol Use Disorder. His past psychiatric history was remarkable for depression with over twenty-five psychiatric hospitalizations. He was treated with multiple antidepressants at therapeutic dosages with only modest success, including: sertraline, fluoxetine, nortriptyline, amitriptyline, and had augmentation with quetiapine and aripiperazole (separately) while taking mirtazapine. He had made five suicide attempts-the most recent of which was by overdose on a tricyclic antidepressant combined with alcohol. He was successfully stabilized and

transferred to psychiatry. His most recent overdose occurred immediately after discharge from hospital and he expressed continued hopelessness and intent to commit suicide after medical stabilization, so the treating psychiatrist felt that a trial of ECT was indicated. The initial session of right unilateral D'elia multi-burst square wave ECT was delivered by Thymatron System IV with seizure duration of 87 seconds. Afterwards, the patient complained of myalgia, anxiety, insomnia and alcohol cravings. Gabapentin 300mg at meal times and 900mg at bedtime was started which relieved all of these symptoms rapidly. The duration of the second ECT session was 93 seconds. It was hypothesized that gabapentin might interfere with ECT so it was tapered. Seizure duration of the third session of ECT was 157 seconds at a gabapentin dose of 300 mg twice daily. As the patients' cravings, anxiety and myalgia decreased, gabapentin was discontinued. The patient completed five further ECT sessions without gabapentin with average seizure duration of 29 seconds and relief of depression.

3. DISCUSSION

Gabapentin was chosen to treat several symptoms. The first indication was for myalgia. Its mechanism of analgesic action is suspected to be a result of binding to alpha-2-delta calcium channel subunits resulting in decreased release of glutamate and substance P, suppressing neuronal excitability following nerve or tissue injury [5]. It is shown to reduce spontaneous pain and allodynia, and is productive in neuropathic pain syndromes like post-herpetic neuralgia, diabetic neuropathy and multiple sclerosis [5,6]. Gabapentin does not change nociceptive thresholds and therefore is not a conventional analgesic like opiates [6]. It was also chosen due to effectiveness in patients with alcohol dependence for withdrawal-related symptoms of insomnia, dysphoria, cravings and anxiety [7-9]. It was used for anxiety due to its partial GABA receptor agonist properties rather than its

anticonvulsant properties and because it is not habit-forming. ECT related myalgia typically occurs after the first treatment of ECT, peaking at 24 hours and usually disappearing after the second to the fourth treatment [10-12]. The mechanism of ECT-induced myalgia is unknown. Some investigators suggest that pain and myalgia are induced from convulsive movements or fasciculations caused by anesthetics [12]. Succinylcholine, which is well known to cause myalgia, fasciculations, and increased myoglobin [11], was used in ECT treatment of the patient in this study. However, one group showed that the dose of succinylcholine does not correlate with the severity of post-treatment myalgia which contradicts the hypothesis that myalgia is a side effect from the anesthetic [13]. Other studies that compared myalgias after ECT found that convulsive movements and strength of fasciculations were not related to subjective complaints of pain [10,11]. This suggests that myalgia is neither caused by muscle damage from contraction nor iatrogenic effects of succinylcholine. We hypothesized that gabapentin assisted with relief of neuropathic pain rather than muscle damage. However, it is also possible that in this patient myalgia simply abated with time rather than due to treatment with gabapentin. Usually, myalgias abate after several ECT treatments, whereas this patient's myalgia resolved within 24 hours so his improvement was more rapid than in other reports, suggesting but not proving that gabapentin facilitated resolution of myalgia. Controlled trials of multiple subjects would be necessary to determine this. The American Psychiatric Association's recommendations are to taper anticonvulsant medications during ECT; however, they may be continued at a low dose when used for anti-epileptic properties [2]. Some evidence supports that concomitant use of anticonvulsant medications during ECT may not interfere with seizure threshold or duration with no severe adverse effects or complications [2,14]. While literature exists that supports the safety of combining anticonvulsants with ECT, no evidence currently suggests that the combination increases therapeutic efficacy [14]. A report of two case studies describes using gabapentin and valproate concomitantly with ECT successfully for intractable seizures [15]. In our case, seizure length actually increased with the use of gabapentin, and seizure length decreased after cessation of gabapentin, so further controlled scientific examination of the effect of gabapentin

on seizure duration is indicated. It is not possible to determine whether gabapentin contributed to relief of the patient's depression and there are few randomized controlled trials of use of gabapentin for augmentation of treatment for depression in the literature. One randomized, controlled study in adjunctive treatment of depression in cocaine users did not show significant anti-depressant effects [16], but another randomized, controlled trial in depressed hemodialysis patients with neuropathy did show improvement of depression [4]. An observational study on patients with diabetic neuropathy also showed improvement in both pain and depression [17]. It is possible that since both anticonvulsants and electroconvulsive therapy have anticonvulsant and mood stabilization properties they may act synergistically [18,19]. Therefore, it is possible that gabapentin also contributed to antidepressant effects as well as anxiety and relief of myopathy, but our case study cannot establish this-further controlled trials would be necessary. Standardized rating scales were not used to quantitate the degree of depression or anxiety of this patient, which is a limitation of this study and would have improved understanding of its effects.

4. CONCLUSION

ECT's antidepressant effects occurred despite either addition or tapering of gabapentin in this case and concurrent use of gabapentin did not reduce seizure duration. Gabapentin seemed to have a variable effect on seizure duration. In fact, its use seemed to lengthen seizure duration. The patient also experienced relief of anxiety and reduced alcohol cravings. A significant limitation of this study is that his depression, anxiety and physical symptoms were not followed with structured rating scales, only with subjective report of relief from these symptoms. Therefore, although more systematic studies must be undertaken, this case study suggests that continuation of gabapentin may be considered during ECT when treating co-morbid mood and substance use disorders for both anxiety symptoms and myalgia without interfering with ECT.

CONSENT

Informed consent was obtained from the patient.

ETHICAL APPROVAL

The local Institutional Review Board and Research & Development Committee approved this article for publication. During our research the procedures were in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 1983.

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COMPETING INTERESTS

None of the authors have any competing interests to declare.

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