Recurrent ventricular tachycardia during the electroconvulsive therapy procedure: A case report

Sir,

Electroconvulsive therapy (ECT) is one of the lifesaving treatments for patients with various severe mental disorders. The initial cardiovascular response to the electrical stimulus includes initial parasympathetic upsurge, lasting for 10–15 s, during which the patient is observed to have bradycardia, hypotension, and transient asystole. This is followed by a sympathetic upsurge, which peaks at 3–5 min after the electrical stimulation, during which patients may have increased blood pressure (BP), tachycardia, and cardiac arrhythmias. Occasional patients also develop sinus arrhythmias, atrial and ventricular premature beats, and minor ST-T changes during the postimmediate postelectrical stimulation. Sustained ventricular tachycardia (VT) is rarely reported during the ECT procedure, and the literature is limited to few case reports.

Here, we report a 24-year-old female patient who had VT after three consecutive ECTs. A 24-year-old female suffering from paranoid schizophrenia since the age of 20 years presented to inpatient unit following a suicidal attempt. Her mental state examination revealed the presence of delusion of reference and delusion of persecution, auditory and somatic hallucinations, made act and thought broadcast. The patient also reported that she made suicide attempt under the influence of commanding auditory hallucinations. In the inpatient unit, she was initially treated with risperidone up to 5 mg/day for 6 weeks, but she showed minimal improvement in psychotic symptoms. While on risperidone, she showed emergence of depressive symptoms and worsening in the intensity of commanding hallucination, asking her to harm herself. Following this, ECT was considered. Her pre-ECT evaluation did not reveal any abnormality in the hemogram, serum electrolytes, liver function test, renal function test, electrocardiogram (ECG), and X-ray chest posterior–anterior view. Her ECG showed a heart rate of 70 beats/min (BPM), which was regular, and there was no evidence of any arrhythmia. Routine monitoring of BP during the week before ECT showed readings of systolic BP to vary from 110 to 124 mmHg and diastolic BP to vary from 76 to 84 mmHg. During the first ECT, she was getting the same dose of risperidone, and no new medication was added. She was given ECT, with atropine as a premedication and thiopentone and succinylcholine as the inducing agents. Immediately, after the first ECT (receiving the electrical stimulus), her heart rate decreased to 48 BPM, and she required treatment with injection atropine 0.6 mg iv. With this, her heart rate improved, but the patient developed monomorphic VT with hypotension (BP = 60/40 mmHg), which resolved spontaneously in 30 s, without any intervention. At this point, the possibility of atropine-induced VT was considered. Her heart rate and BP were monitored for the next 3 days, which did not reveal any abnormality. Following this, she was considered for second ECT during which atropine was avoided as
Persistent VT has been rarely reported with the ECT procedure. In fact, in view of this rarity, initially, possibility of ECT-associated VT was not considered in the index case. A review of available literature revealed existence of only four case reports, which have reported similar association. Of these, in three cases, there was no past or current medical history of cardiac illness, as seen in the index case. Detailed cardiac evaluation in these cases also did not reveal any abnormality, as in the index case. One of the reported cases had a history of ischemia, and the patient was on quinidine and digoxin at the time of receiving ECT. Of the four reported cases, two cases required the use of chest compression, one patient had spontaneous resolution of VT, and one patient was managed with cardiac massage and midazolam.

Index case developed VT thrice, every time temporally related to the electrical stimulus, with no evidence of VT before, after, and during the intervening periods between the ECTs. Further, efforts were made to remove the probable offending medications during the ECT, and patients did not have any VT during the intervening period between the three ECTs, with the continuation of the same medication. All these provide further credence to this association in the index case. In the index case, VT always required interventions.

To conclude, the present case report suggests that sustained VT is a rare side effect of ECT. Accordingly, whenever a patient receiving ECT develops VT, a possibility of ECT-associated VT must be considered.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

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Sir,

We read the article by Narasimha et al. [1] with interest. The authors have presented the data on the use of two drugs (N-acetylcysteine and baclofen) in a condition with very little pharmacotherapy options, cannabis use disorders (CUDs), with the caveat that it can only provide preliminary evidence as this was not a randomized controlled trial. These drugs are already in use for different indications.

The study gives data on 72 patients treated for the primary diagnosis of CUDs. But, 47 (65.2%) of their sample had a comorbid psychiatric illness and would have received some psychotropic medications. But curiously, they have not considered that this could also be a contributing factor to the benefits they attribute to the study drugs (N-acetylcysteine and baclofen) and/or psychosocial intervention. If they had considered this as a covariate in their analysis, the findings would have been different. There is evidence that combined treatment with psychotropics and other drugs is more effective in cases of substance use disorders with comorbidity.

[2]

Further, they have not separately mentioned the outcomes for the 25 (34.8%) patients who did not have other psychiatric comorbidities and hence did not receive any other psychotropics, except the study drugs. Although a small number, this would be noteworthy. They have mentioned all other limitations. We agree with them that this study provides only very preliminary evidence that has to be evaluated by further studies.

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