ORIGINAL RESEARCH PAPER

Psychiatry

STATUS EPILEPTICUS POST ECT: NEED FOR MANDATORY EEG SEIZURE MONITORING

KEY WORDS: ECT, Anaesthesia, Status epilepticus, seizure

Dr. Anand Lingeswaran

ABSTRACT

Status epilepticus has been reported during ECT treatment. Reliance on observable motor seizure as sole therapeutic indicator of ECT may allow the unexpected occurrence of status epilepticus in rare cases that can be life threatening. We report a psychiatric patient who was given ECT for major depression without EEG monitoring who developed status epilepticus. After four successful therapeutic ECT induced motor seizures, status epilepticus developed after recovery from general anaesthesia for the last ECT. EEG monitoring if available could have detected prolonged seizure and emergence of status epilepticus. Hence we propose EEG monitoring is always mandatory for ECT delivery in all settings.

INTRODUCTION

ECT treatment has been delivered safely and in a useful manner in many life threatening psychiatric conditions. Despite its amazing therapeutic effect, life threatening unexpected complications also occurs. One such rare adverse effect is status epilepticus, a neurological emergency and it has been linked to prolonged ECT seizures (120 seconds). Status epilepticus (SE) which is defined as 30 minutes of sustained seizure activity or 2 or more seizures during this time period without the full recovery of the patient causes significant hypoxemic damage to the hippocampal neurons has a high mortality rate of 37%. (1,2,3) In the context of ECT, seizure that continues beyond 120 seconds, repetitive stimulation to achieve therapeutic seizure without EEG monitoring of seizure, and hyperoxygenation have been reported as rick factors that may precipitate SE with ECT. Although, clinicians delivering ECT treatment widely concur on the higher accuracy of EEG monitoring of ECT seizure, still only relying on the occurrence of a generalized tonic-clonic motor seizure of 15 to 25 seconds is ECT is delivered with only motor seizure. (2) In this context, we present a case of a patient who developed status epilepticus post ECT without EEG recording, to strongly highlight the merits, safety and benefits of routine EEG seizure monitoring for ECT. (3, 4)

Case Report

We report the case of 35 years old patient, with recurrent depressive disorder, current episode of severe depression without psychotic symptoms diagnosed based on ICD-10 criteria. She was responding gradually to Mirtazapine 45mg and since her oral intake was poor, Electroconvulsive Therapy (ECT) was chosen as treatment option to augment the response. She had never received ECT before and had two previous episodes of depression. ECT treatment was discussed with her and a written informed consent was obtained from the patient. Physical examination revealed a constitutionally small physical built and she weighed 34 kg during admission. All her pre-ECT investigations like CBP, ESR, blood sugar, blood urea, serum creatinine, ECG, and chest –Xray were within normal limits. Fundus examination was normal with no signs of raised intracranial tension. A preanaesthetic clearance was obtained and patient was administered injection thiopentone 100 mg i.v, and succinylcholine 25mg based on her body weight. To reduce oral secretions 0.2mg of injection glycopyrrolate was given intravenously. A rubber mouth gag was placed safely in her mouth to prevent tongue-bite and buccal mucosal injury. After adequate gel application, a bitemporal handheld ECT electrode dose of 111 millicoulombs (mC) was given using a brief pulse ECT machine without EEG recording. A therapeutic motor seizure of tonic-clonic type was observed for 41 seconds with spontaneous cessation. No post-ECT or anaesthesia recovery related concerns were noted. Four similar ECT treatments with same dosing parameters were given and motor seizures ranged from 33 to 46 seconds. Clinically, the patient showed gradual recovery of oral intake, psychomotor activity, and depression following the third ECT. To maintain the improvement, a fifth ECT was given with the same anaesthetic dosage and treatment parameters. But on this instance, after complete cessation of motor seizure after 43 seconds, there was no evidence of any observable motor seizure. Pulse rate was gradually dropping to normal levels and oxygenation was resumed to assist recovery. Unexpectedly, within a minute, the patient was noted to develop clonic movements of her masseter muscles bilaterally and within seconds this seizure spread to the upper limbs only. Manual jaw thrust was applied to stop the seizure but did not work and the seizure continued beyond 5 minutes and oxygen saturation (SpO2) dropped to 87%. Injection Midazolam 2mg i.v bolus was given to abort the seizure. Seizure stopped for a few seconds and restarted. For the next 30 minutes, the patient was repeatedly having multiple GTCS, each episode lasting about 6 minutes. Immediately she was shifted into intensive care unit and was managed by a team of anaesthetists, physicians and treating psychiatrist. Status epilepticus was confirmed and the appropriate management protocol was initiated. SpO2 monitoring, head-end elevation was done and a plastic airway was tightly fixed to maintain Spo2 and to avoid tongue bite. After 3 minutes following midazolam, Injection Lorazepam 2mg i.v bolus three times (6mg) stopped the seizures briefly only. After 15 minutes post lorazepam, two doses of Injection Leviteracetam 500mg i.v bolus was given, with a total of 1000mg. By then, the interval between seizures increased indicating positive response to antiepileptic. After 30 minutes, while planning to administer i.v metered infusion of injection Thiopentone 100mg, the seizure stopped and the whole body was relaxed. SpO2 reached 99% and lungs had bilateral clear air entry. Random blood sugar was 112mg/dL.

After 45 minutes, patient regained consciousness and the thiopentone infusion was allowed to complete. The patient was kept under ICU observation for the next 6 hours, all vital parameters became normal and no further seizure was seen. Airway was removed and she maintained spontaneous breathing with 99% SpO2. Gradually oral feeding was started and after 4 hours post recovery, she was shifted to the general ward where she remained stable. After 24 hours, a bedside neuropsychological evaluation was specifically conducted to assess her memory and it showed intact immediate, recent and remote memory.

DISCUSSION

Non-therapeutic prolonged (>120 seconds) seizures post ECT have been related to hyperoxygenation during the anaesthesia, cerebral excitability from seizure, Multiple monitored ECT. A seizure under general anaesthesia causes unexpected adverse events such as aspiration, apnoea,

bronchospasm, laryngeal spasm, cardiac asystole, retrograde amnesia, post-ictal confusion, orthostatic hypotension, status asthmaticus, and status epilepticus. Newer operational definitions have suggested that seizures lasting longer than 5 minutes are unlikely to discontinue spontaneously and should be treated.4 This is a more useful definition since treatment for SE is not delayed. In addition, individual unprovoked seizures can on occasion be observed and may not warrant aggressive treatment.

Through this case, we need to review retrospective research that will improve our understanding of mandatory safety methods to be utilized in ECT delivery in any setting. EEG monitoring certainly provides serious merits of uncomplicated ECT treatment. One study reported that mean ECT motor seizure duration was at least 25 seconds shorter than of EEG seizures. Moreover, the mean ratio of motor seizure and EEG seizure duration was 0.68 with a range of 0.46 - 0.90, and this ratio was lowest in prolonged seizures lasting beyond 120 seconds [3], highlighting the fact that clinicians tend to underestimate actual ECT seizure duration by looking only at motor seizure. The preventable neuronal cell damage and hypoxemia due to prolonged seizure (120seconds) is grossly missed. [3]. Another study proved the poor sensitivity of only motor seizure monitoring by reporting that just 21.4% of ECT seizure was missed through motor seizure monitoring alone.

There is no doubt that status epilepticus may not be easy to identify as in our case where clonic motor activity started only in the masseter area, and EEG might have definitely revealed continuation of actual seizure. In resource poor healthcare settings, the lack of EEG support strict need for EEG monitoring of seizure during ECT is not always available and followed and high reliance is placed on motor seizure duration only. Given the complication of status epilepticus seen in our case that might have been detected on EEG, revisiting of relevant research from the past that supports the need for EEG during ECT provides better knowledge. Also, where ECT is given without EEG monitor, the clinician might end up restimulating with a higher electrical current which increases the risk of prolonged seizures.

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