Psychology & Psychotherapy

Case Report Open Access

Generalised Non-Convulsive Status Epilepticus (NCSE) following Electro-Convulsive Therapy

Lloyd Reeve-Johnson[1]*, Alston Unwin[2] and Harry McConnell[3]
[1]*Faculty of Health, Queensland University of Technology, Brisbane, Australia*
[2]*Director of Medical Services, Belmont Private Hospital, Brisbane, Australia*
[3]*Clinical Sub-Dean, Griffith University, Gold Coast, Australia*

Abstract

Electroconvulsive therapy is an effective treatment for severe depression. Non-convulsive status epilepticus is a serious, uncommon yet probably under-reported side-effect. This paper considers risk factors that have occurred in other cases and proposes alcohol withdrawal as an additional risk factor for consideration when selecting suitable patients.

Keywords: Non-convulsive status epilepticus; NCSE; Risk factors; ECT; Electroconvulsive therapy

Introduction

Electro-Convulsive Therapy (ECT) is an effective treatment used for severe depression. Electric current applied via electrodes on the head induces a seizure which is typically self-limiting and usually lasts less than a minute [1]. Complications are usually short lived and include headache, memory loss and confusion. More serious is the potential for prolonged seizure although extensive literature review reveals only fourteen detailed reports of convulsive *status epilepticus* over a 33 year period following ECT. Even less frequent is Non Convulsive Status Epilepticus (NCSE) with twelve cases located in the literature of which only five have been described in detail over the same period. A literature search of the Cochrane Library database and PubMed using advanced terminology was performed searching the following terms and synonyms: status epilepticus +non-convulsive +/- electro-convulsive therapy +/- ECT +/- EMG. This is the first Australian case report that we can find in the literature reflecting local under-diagnosis. NCSE can lead to neuronal injury [2] and without EEG cannot be detected and even in the conscious patient may be difficult to differentiate from acute confusion. We have taken the opportunity to consider some common factors from these cases for patients undergoing ECT which may help identify patients at increased risk.

Case

A 41 year old, 62 kg (BMI 19.8 kg/m^2) childless professional lady with no history of epilepsy was admitted for treatment to Belmont Private Hospital with suicidal ideation and ongoing major depression for over 20 years. She had an ongoing history of alcohol abuse (12-14 units per night) and apart from an incidentally discovered

Factor V Lieden had no other remarkable medical history. There were no neurological deficits. She failed to respond to desvenlofaxin at increasing doses up to 150 mg mane with concurrent quetiapine to a maximum dose of 200mg. Mirtazapine 30 mg nocte had been discontinued 14 days before the seizure and in the hospital setting alcohol intake was restricted. Other medications were zolpide tartrate 25 mg nocte, chloral hydrate 20 mls nocte and dothiepin hydrochoride 50 mg bedtime-tid for insomnia. She was also receiving Sodium Valproate (VPA) 400 mg BD aimed at maintaining blood levels of approx 600 µmol/L, lithium Carbonate 250 mg TID, 40 mg diazepam for alcohol withdrawal and PRN paracetamol 1 g approximately every other night for headache. Her only reported drug interaction or allergy previous to this was with penicillin. She had no history of seizures.

She had received eight previous ECT treatments over the past four weeks with no major adverse events, experiencing a minor degree of confusion and no memory loss. Small improvements in symptoms were noted from the seventh ECT treatment, amounting to a marginal improvement in her General Assessment of Function (GAF) score of no more than 10 (improving from a rating of 40-50). This amounted to a lessening of serious social impairment and inability to interact socially to symptoms of flat effect and elective avoidance behaviors. She consented to two further ECT treatments.

On the ninth treatment occasion no epileptiform EEG or EMG activity was recorded with initial charges of 401.3 mC for 7.5 seconds or 602.2 mC 7.6 seconds. The third attempt involved 1013.8 mC for 8 seconds. After 15 seconds an electrographic seizure was captured, with generalized epileptiform activity without any clinical EMG change. The EEG registered bilateral seizure activity. She was considered to be tolerant to benzodiazepine and her current dosage of VPA was 800 mg daily. Phenytoin sodium (15 mg/kg) was administered over a 15 second period by intravenous injection at 28 seconds, but EEG seizure activity persisted for a total of 1130 seconds. It was then terminated with intravenous propofol (2 mg/kg). The patient recovered fully with no adverse effects to memory or headache and the period of recovery and confusion post-anesthetic was not significantly different to other general anesthetic recoveries.

Discussion

A number of potential risk factors for NCSE have been suggested. These have included being over 70 years of age and applying right unilateral stimulation [3], medications that lower seizure threshold such as lithium [1,4], paroxitine [5,6], thioridazine [7] and haloperidol [6,8]. Grogan et al. [1] suggested these to be necessary cofactors in the precipitation of NCSE. However the literature is not conclusive and all

***Corresponding author:** Lloyd Reeve-Johnson, Queensland University of Technology, Brisbane, Queensland Q4070, Australia, Tel: +07 3202 5701; E-mail: lloyd@goydpark.com

Received February 06, 2014; **Accepted** March 24, 2014; **Published** April 01, 2014

Citation: Reeve-Johnson L, Unwin A, Connell HM (2014) Generalised Non-Convulsive Status Epilepticus (NCSE) following Electro-Convulsive Therapy. J Psychol Psychother 4: 138. doi: 10.4172/2161-0487.1000138

of the above have been used safely in many patients [9]. No other cases recorded patient's alcohol intake history or withdrawal and we consider this to be a relevant factor in this case. Benzodiazepine reduction was also feature of this case and is noted to have occurred in three other cases of NCSE after ECT [6,10,11]. In six of nine reported cases of NCSE patients had received multiple ECT treatments (mean 6.5).

The causes of NCSE remain unknown, however of the twelve cases identified over a 33 year period, nine were female and three male. The median age was 41 years old. There was one case of an 18 year old male receiving thoridazine (100 mg/day) and trihexyphenidyl [7], the mean age of the other cases was 45 years old (range 26-87). Of six cases which described the seizure length the median length was 3.5 days (range 350 seconds to 5 days) with three described as bilateral, two as unilateral and the rest uncharacterized. In one case NCSE was described after the first treatment [3], however in all other cases NCSE occurred after multiple treatments. The mean number of treatments before NCSE was recorded was 6-7 treatments (range 1-9). Both in our case and in others where VPA was used [8] refractory seizures were readily controlled. Similar to this case others also reported incomplete seizure control with phenytoin and diazepam [1,3,6,8,10].

In a number of cases including our own, neuroleptic drugs had been used thioridazine, haloperidol [1,7], haloperidol [6], riperidone and in this case quetiapine [10].

Without ongoing EEG monitoring, detection of NCSE can be difficult to differentiate from acute confusional states post ECT or possibly other neuroleptic drug interactions. It is likely that NCSE is under-diagnosed particularly as it can manifest after a delay of many hours or days after ECT. NCSE should therefore be included in the differential diagnosis of any patient displaying unusual behaviour after ECT.

Routinely repeated EEG monitoring is appropriate during the first week post-ECT in any patient where there is any doubt about their behaviour or response. Patients on neuroleptic medication, those suspected of having a lower seizure threshold (e.g on Lithium) or those who have recently had alcohol or benzodiazepine withdrawal should be viewed as having elevated risk of NCSE when undergoing ECT.

References

1. Grogan R, Wagner DR, Sullivan T, Labar D (1995) Generalized nonconvulsive status epilepticus after electroconvulsive therapy. Convuls Ther 11: 51-56.
2. Fountain NB (2000) Status epilepticus: risk factors and complications. Epilepsia 41 Suppl 2: S23-30.
3. Smith K, Keepers G (2000) Nonconvulsive status epilepticus after ECT. Am J Psychiatry 157: 1524.
4. Weiner RD (1981) ECT-induced status epilepticus and further ECT: a case report. Am J Psychiatry 138: 1237-1238.
5. Curran S (1995) Effect of paroxetine on seizure length during electroconvulsive therapy. Acta Psychiatr Scand 92: 239-240.
6. Srzich A, Turbott J (2000) Nonconvulsive generalised status epilepticus following electroconvulsive therapy. Aust N Z J Psychiatry 34: 334-336.
7. Rao KM, Gangadhar BN, Janakiramaiah N (1993) Nonconvulsive Status Epilepticus after the Ninth Electroconvulsive Therapy. Convuls Ther 9: 128-129.
8. Varma NK, Lee SI (1992) Nonconvulsive status epilepticus following electroconvulsive therapy. Neurology 42: 263-264.
9. Kellner CH, Nixon DW, Bernstein HJ (1991) ECT--drug interactions: a review. Psychopharmacol Bull 27: 595-609.
10. Parker V, Nobler MS, Pedley TA, Sackeim HA (2001) A unilateral, prolonged, nonconvulsive seizure in a patient treated with bilateral ECT. J ECT 17: 141-145.
11. Conway CR, Nelson LA (2001) The combined use of bupropion, lithium, and venlafaxine during ECT: a case of prolonged seizure activity. J ECT 17: 216-218.

Citation: Reeve-Johnson L, Unwin A, Connell HM (2014) Generalised Non-Convulsive Status Epilepticus (NCSE) following Electro-Convulsive Therapy. J Psychol Psychother 4: 138. doi: 10.4172/2161-0487.1000138