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Electroconvulsive therapy for anti-N-methyl-D-aspartate (NMDA) receptor encephalitis: A systematic review of cases

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ABSTRACT

Background: Anti-NMDA receptor encephalitis most commonly presents with psychiatric symptoms such as behavioural disturbance, catatonia and psychosis. Although the primary treatment is with immunomodulatory therapy, psychiatric symptoms often require adjuvant management. Side effects and treatment resistance limits the use of psychotropics, but the role of ECT has been minimally reviewed.
Objective: To review the safety and effectiveness of ECT for treatment of psychiatric symptoms in anti-NMDA receptor encephalitis.
Methods: A systematic literature review of PubMed, Embase and PsycInfo was performed from inception to June 2018.
Results: There were 30 cases of ECT used in anti-NMDA receptor encephalitis. Cases were typically young (mean age 27.7 years, SD 15.2) females (73.3%) with catatonia (86.7%). There was improvement of these symptoms in 65.2% of cases, interestingly without immunomodulatory therapy in 17.4%. ECT proceeded without complication in 86.7% of cases, with four cases prematurely ceasing ECT with further encephalitic deterioration. There were no anaesthetic complications.
Conclusions: ECT appears to be an effective and safe adjuvant treatment in anti-NMDA receptor encephalitis, particularly for catatonia.

Introduction

Anti-N-methyl-D-aspartate (NMDA) receptor encephalitis is an autoimmune disorder that most commonly presents to mental health services with psychiatric symptoms such as severe behavioural disturbance, mood disorder, catatonia and psychosis [1,2]. Anti-NMDA receptor encephalitis is estimated to account for up to 3% of all first episode psychosis presentations [3]. Progression of the disorder sees the development of neurological symptoms such as movement disorders, seizures, cognitive deficits and autonomic disturbance, often requiring intensive care admission and lengthy hospitalisation [1]. Anti-NMDA receptor encephalitis is more common in young females, with a reported ratio of approximately 3 females:1 male, and associated with ovarian teratoma or other malignancies in around 50% [1,2,4]. Tumour removal and immunomodulatory therapy are the standard treatments [1]. However, as the rate and the nature of the deterioration from psychiatric symptoms is variable and there is often a delay before CSF antibody confirmation of the disorder, an initial primary psychiatric diagnosis of mania or psychosis is common [5].

Treatment of psychiatric symptoms of anti-NMDA receptor encephalitis is complicated by resistance to traditional psychotropic agents, increased risk of extra-pyramidal side-effects and concern for lowering seizure threshold in patients already at risk of seizure [2,5]. Autonomic disturbance including cardiac arrhythmias and reduced level of consciousness also increase the risk of drug-related side-effects. Suspected neuroleptic malignant syndrome (NMS), seen more commonly in this disorder, often prompts anti-psychotic withdrawal [2]. For these reasons, electroconvulsive therapy (ECT) may be of use.

There is a limited body of literature describing use of ECT in single cases or small case series [6]. With increasing awareness and knowledge of this disorder among mental health professionals, we aimed to systematically review all published cases of ECT used in the treatment of symptoms in anti-NMDA receptor encephalitis,

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and ascertain whether it was safe and improved psychiatric outcomes.

Methods

PubMed, Embase and PsycInfo databases from inception to June 2018, were searched using terms: NMDA, NMDAR, NMDARE, N-methyl-D-aspartate encephalitis, ECT and electroconvulsive therapy. All articles types, including conference posters and abstracts, that had data for individual cases were included. Search and screening were independently conducted by two authors (NW, VG) at title, abstract and then full text level. References of selected articles were cross-checked to identify other potentially eligible studies. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement recommendations were followed and the study was pre-registered with PROSPERO, CRD42018102106 [7].

Cases were included if they received treatment of psychiatric symptoms with ECT. Those psychiatric symptoms occurred in the context of anti-NMDA receptor encephalitis diagnosed by positive serum or CSF IgG antibody. Cases with suspected but unconfirmed anti-NMDA receptor encephalitis, or those receiving ECT for other indications were excluded.

The pre-specified variables collected included: demographics, past medical and psychiatric history, initial and subsequent clinical presentation of encephalitis, investigations, psychotropic and other treatments, ECT timing and prescription, noted ECT side effects or other ECT considerations. Missing data was noted. Recognised anti-NMDA receptor encephalitis symptom clusters: behaviour disturbance, psychosis, cognitive, movement, seizures and autonomic, were used to classify initial presentation and course of illness [2,8]. Catatonia, considered for this study part of the behavioural disturbance cluster, was recorded if there was clear documentation of diagnosis using established criteria. The Bush Francis Catatonia Screening Instrument (BFCSI) was used to document the signs of catatonia and the Bush Francis Catatonia Rating Scale (BFCRS) noted when reported [9]. Treatment response and outcome was based on the information provided in the case reports and categorised as resolved, improved, no improvement or deterioration. Symptoms following treatment were recorded. Descriptive statistics were employed for analysis of the data.

Results

There were 781 citations identified in the databases after duplicates removed. Following screening at title, abstract and full text level, 29 studies comprising 30 cases were identified from 2007 to 2018 (Fig. 1). There were 22 females and 8 males with a mean age of 27.7 years (SD 15.2). The youngest case was 9 years and there were 10 cases under the age of 18 years. Encephalitis associated with ovarian teratoma accounted for four cases, with no other malignancy noted in the remaining cases.

The most common presenting feature of anti-NMDA receptor encephalitis in these cases was behavioural disturbance 21/30 (70%), all of these cases reporting agitation or severe irritability. There were 9 cases (30%) documenting catatonia on presentation and psychosis in 8 (36.7%). The presenting symptoms and those during the course of anti-NMDA receptor encephalitis can be seen in Fig. 2. During the illness course, catatonia developed in 26/30 cases (86.7%), and was noted to be fluctuating in 11 cases. An additional 3 cases which did not document catatonia had a BFCSI over 2, indicating a catatonic syndrome. The mean BFCSI was 4.3 and the most common catatonic symptoms were, in descending order, excitement, immobility/stupor, mutism, withdrawal, posturing, rigidity, stereotypy and perseveration. Combativeness was reported in 10 cases and impulsivity in 6 cases. Other psychiatric symptoms seen, included delusions and/or hallucinations (25 cases, 83.3%), mood disturbance (17 cases, 56.7%), pathological laughter/yelling (4 cases, 13.3%) and self-injury (2 cases, 6.7%).

Anti-psychotic medications were used in 25 cases (83.3%), with 17 cases requiring multiple anti-psychotics. Suspected neuroleptic malignant syndrome or severe extra-pyramidal symptoms resulted in the cessation of anti-psychotics in 14 cases. A trial of benzodiazepines (lorazepam) for catatonia was documented in 19 cases (63.3%).

The majority of cases (20/30, 66.7%) developed neurologic abnormalities, most commonly limb dystonia [8], orofacial dyskinesia [5], choreo-athetosis [5] and rigidity [5]. Autonomic disturbance (17/30, 56%) - including tachycardia [6], hyperthermia [6] and respiratory failure [5] - and seizures (16/30, 53%) were common. Cognitive deficits during the active phase of illness were reported in 13 cases (43.3%). Four cases (13.3%) had isolated psychiatric symptoms without other abnormalities. MRI brain was abnormal in 6/30 (20%) cases, reporting ischaemic foci [3], T2 FLAIR hyperintensity [2] and one case of an incidental temporal lobe cyst with no oedema or mass effect. There were no identified contraindications to ECT on neuroimaging. EEG on presentation was abnormal in 14 cases (46.7%), documented as diffuse slow waves [12], extreme delta brush [2] and one case demonstrating epileptiform features.

ECT was commenced prior to the diagnosis of anti-NMDA receptor encephalitis in 21 cases (70%). Only one case reported a relative contraindication for ECT; a history of pulmonary stenosis. In 5 cases antipsychotics were documented to continue during the course of ECT. The average number of sessions of ECT was 10 (range of 1–33 sessions), although this was not documented in 9 cases. Overall, the ECT dosage, placement and treatment course was poorly documented.

Effectiveness

There were 23 cases which provided information on effectiveness of ECT in anti-NMDA receptor encephalitis (Table 1). Of those, 15/23 (65.2%) noted improvement of psychiatric symptoms, and in nine of the cases the improvement was seen prior to immunotherapy. In four cases it was documented that there was complete resolution of psychiatric symptoms (catatonia [2], not stated [2]) with ECT, without any immunotherapy and after treatment resistance had been noted with psychotropics. In the six cases which improved with ECT and immunotherapy, it was documented that ECT was given due to insufficient response to immunotherapy alone. Interestingly, in two cases cognitive deficits were noted to resolve following ECT and in three cases there was also improvement of movement disorders, such as dyskinesia and rigidity. Catatonic symptoms were noted to improve in the 5 cases in which anti-psychotics were never used and in the 5 cases where anti-psychotics were continued throughout the ECT course.

ECT was felt to be ineffective in four cases and was ceased prematurely in four cases. In the four cases in which ECT was felt to be ineffective the indication was catatonia, there was more than one anti-psychotic trialled prior to ECT and the mean number of sessions was 9.5.

Safety

In 26/30 cases (86.7%) there were no safety concerns with ECT, however ECT was ceased prematurely in 4 cases (13.3%). In 2 cases (6.7%) ECT was ceased after one session due to development of seizures. In the other 2 cases (6.7%), ECT was ceased after 2 sessions due to concerns for neurological deterioration with limb dystonia

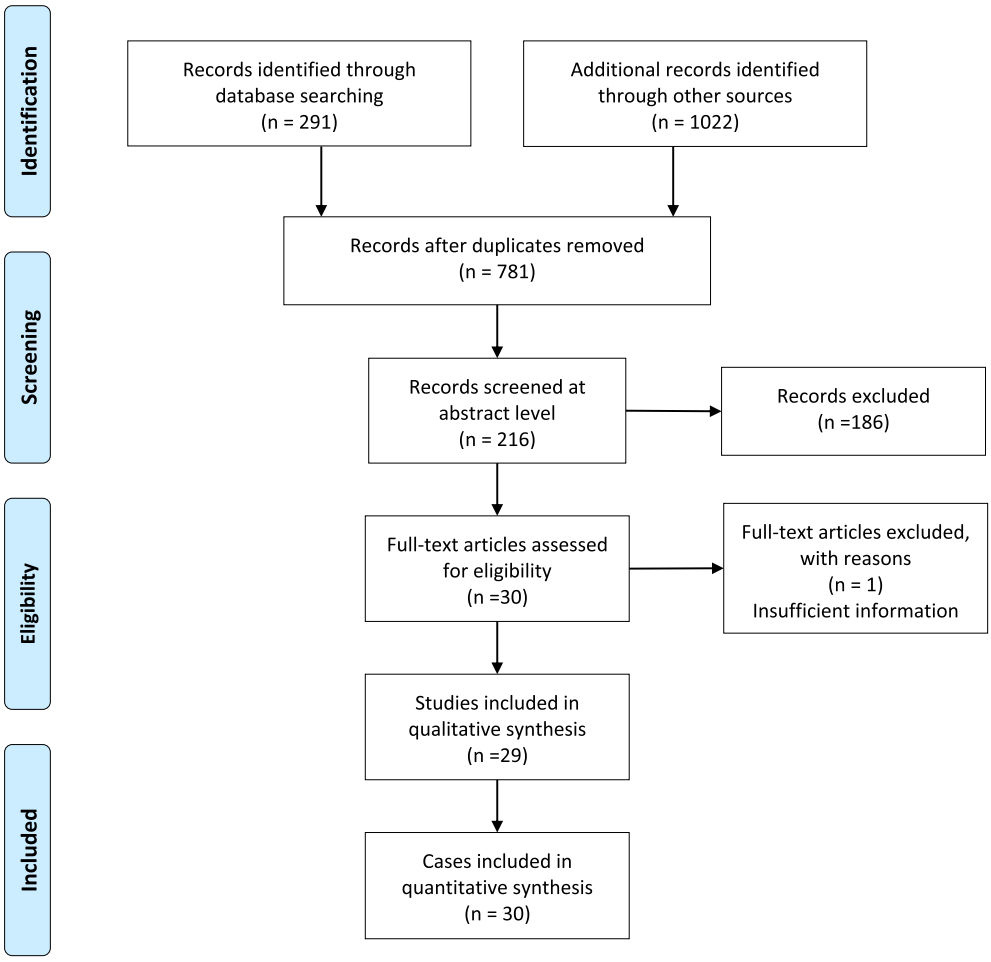

Fig. 1. PRISMA 2009 flow diagram.
ECT for Anti-NMDA receptor encephalitis.

and oro-facial dyskinesia reported. There were no documented complications from anaesthesia, with no requirement for reintubation or additional bloods pressure/cardiac rhythm support.

Overall, complete recovery from anti-NMDA receptor encephalitis was documented in 18 cases, partial recovery in 10 cases, and not documented or lost to follow up in two cases. Of note, the four cases in which ECT was prematurely ceased made full recoveries following immunotherapy. In the four cases in which ECT was felt to be ineffective, two had complete recovery with immunotherapy and in two there was no documentation. When recovery was partial this was due to ongoing cognitive deficits, as seen in Fig. 2, and in five of these cases the cognitive deficits were only documented after treatment. Recurrence of anti-NMDA receptor encephalitis was seen in one case, treated successfully with further immunotherapy.

Discussion

The cases described here, which have used ECT for the treatment of psychiatric symptoms, have similar demographic and initial clinical presentation characteristics as the broader group of anti-NMDA receptor encephalitis cases [1,2,8]. Interestingly, most cases received ECT prior to a definitive diagnosis of anti-NMDA receptor encephalitis, a not uncommon occurrence given the diagnostic delay from a complex clinical presentation, potential reluctance to perform lumbar punctures on psychiatric patients and long turnaround times for antibody testing. Almost all of these cases had catatonia, and ECT was used for refractory psychiatric symptoms, or when side effects required cessation of psychotropic medications. Catatonia is common in anti-NMDA receptor encephalitis, present in around a third of cases overall, and carries significant risks of long-term immobility, deconditioning and hospital acquired infections as well as the severe consequences of malignant catatonia [2,10].

ECT is highly effective in treating all types of catatonia with reported catatonic remission rates between 82% and 96% [10–12]. Both excited and stuporous catatonic symptoms were effectively treated in these cases at a comparable efficacy. ECT is thought to be the primary treatment of choice for catatonia with autonomic disturbance, malignant catatonia or where a rapid treatment response is required [12]. This may be particularly important for catatonia associated with anti-NMDA receptor encephalitis, where dysautonomia is common and where prolonged catatonia may have more severe sequalae in the setting of additional neurological concerns [1]. It should be noted that potentially the use of antipsychotics may have contributed to the catatonic motor symptoms in these cases, and anti-psychotic withdrawal rather than commencement of ECT, may have resulted in symptom improvement. However, catatonia was seen at presentation in nine cases, and developed in five cases untreated with antipsychotics. Additionally, improvement of catatonic symptoms occurred in five cases where antipsychotics had been continued

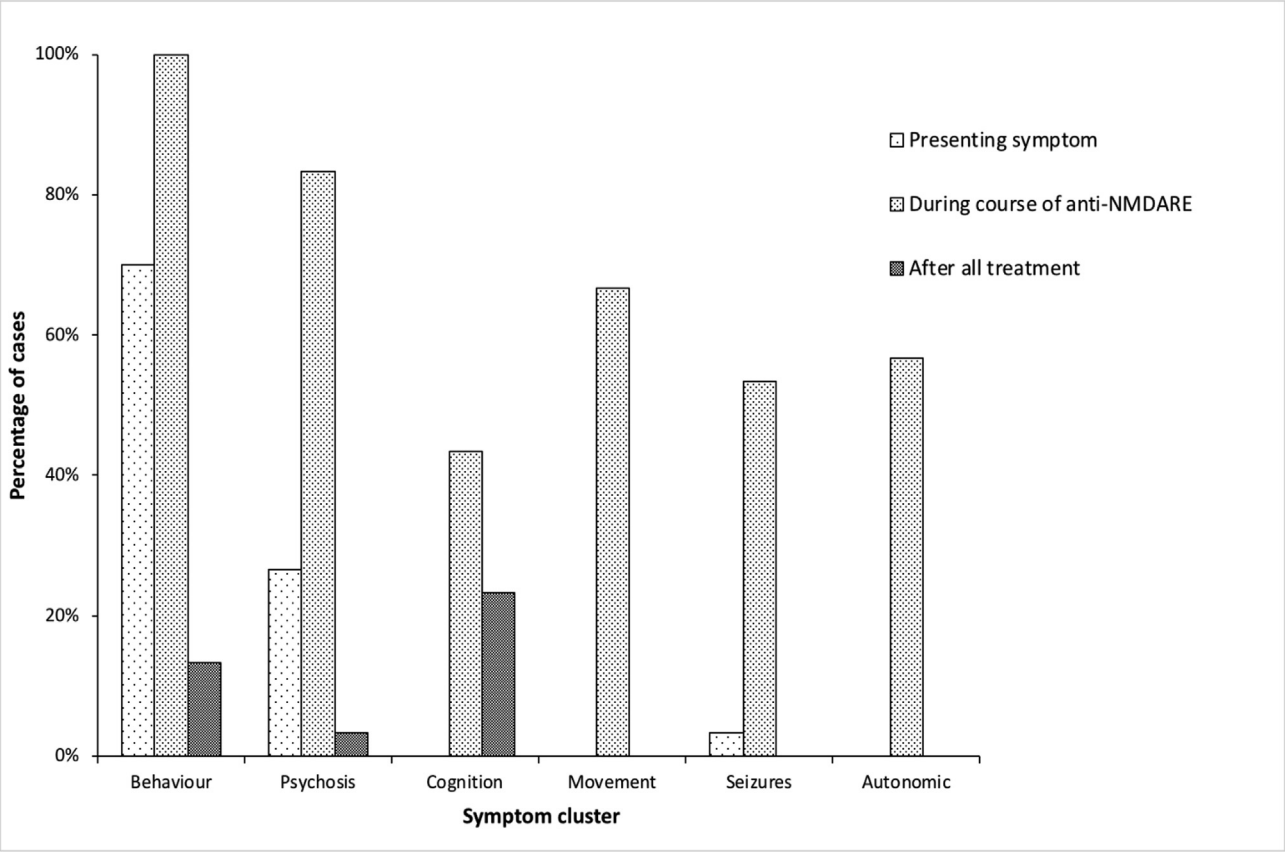

Fig. 2. Anti-NMDARE symptom clusters.

during ECT. Thus, catatonic improvement was felt to be primarily related to the use of ECT.

In addition to the impact on psychiatric symptoms, ECT may have other beneficial effects for cases with anti-NMDA receptor encephalitis. ECT has been shown to be efficacious in NMS, status epilepticus, and for the dyskinesias and other motor symptoms in disorders such as Parkinson's disease; potentially accounting for some of the beneficial motor effects seen in these cases [13–15]. ECT has a known anti-convulsant effect and has been demonstrated in animal models to prevent kindling, which may be beneficial to cases of anti-NMDA receptor encephalitis, known to progress to seizures in around 70% [8,16]. Case series of epileptic psychiatric patients undergoing ECT, have shown it to be a safe and effective treatment, with no anti-epileptic medication dose adjustments required in the majority [17]. It is possible that in the cases here in which ECT was ceased due to seizure development, or other neurological symptoms, this was more reflective of the natural progression of anti-NMDA receptor encephalitis rather than a true side effect of ECT.

The mechanisms of action of ECT remain unclear, although animal studies have shown complex effects on NMDA receptor number and activity, as well as effects on glutamate, GABA and dopaminergic neurotransmitters, all of which are proposed to be dysregulated in catatonia [18–20]. It has therefore been hypothesised that ECT may be particularly beneficial in anti-NMDA receptor encephalitis, targeting the neurocircuitry effects of the antibodies on the brain, although this has not been further confirmed [21]. Conversely, it could be considered that ECT may just be temporarily attenuating the catatonic and motor symptoms, whilst effect of the immunomodulatory treatments or the natural progression of the disorder occurs. First line immunotherapy or tumour removal achieves symptom improvement for around 50% of anti-NMDA receptor encephalitis patients over 4 weeks [5,8]. However, for some, improvement may come up to 24 months later, even with second line immunotherapy, during which time the person is exposed to the risks of ongoing encephalitis such as sustained catatonia [5,8].

There are several important considerations that should be borne in mind prior to ECT commencement in cases of anti-NMDA receptor encephalitis including appropriate consent and exclusion of relative contraindications. In this series, one-third of cases were under 18 years of age and although(22) [22] ECT is rarely used to treat adolescents and children, it is indicated for debilitating or life-threatening conditions such as malignant catatonia [13]. Consent and other legal obligations require thorough discussion with family and other decision makers, not only for adolescents but for all without capacity, expected to be the majority of cases with anti-NMDA receptor encephalitis. Relative contraindications to ECT such as raised intracranial pressure and space occupying lesions are uncommon in anti-NMDA receptor encephalitis. Although, neither occurred in these cases, ECT proceeded safely in the context of neurological symptoms, seizures, autonomic disturbance, EEG and MRI abnormalities. Further reassurance may be provided by the literature examining ECT use in post stroke and subcortical vasculopathies, having similar neuropathological insults as encephalitis. This suggests efficacy and tolerability but potentially increased rates of post-ECT delirium [22].

The anaesthetic considerations highlighted in the anti-NMDA receptor encephalitis literature were not raised in any of these cases, perhaps reflecting the brief anaesthetic required for ECT

Table 1
ECT for Anti-NMDA receptor encephalitis.

Author	Year	Gender	Age	Indication for ECT	BFCSI	ECT details	ECT Outcomes
Florence	2009	F	16	N.S.	3	N.S.	Improved symptoms
Khadem	2009	F	57	Catatonia	4	12 sessions	N.S
Braakman	2010	M	47	Catatonia	4	7 sessions Bi-lateral	Resolved catatonia
González-Valcárcel	2010	F	15	Catatonia	I.I.	12 sessions	Resolved catatonia, relapsed after 1 year
Caplan	2011	F	27	N.S.	1	6 sessions	Partial improvement with some ongoing behavioural disturbance
Creten	2011	M	9	Catatonia	2	N.S.	Resolved catatonia
Kamran Mirza	2011	F	14	Catatonia	4	7 sessions	No improvement
Torgovnick	2011	M	52	Catatonia	3	12 sessions	No improvement. Lost to follow up
Matsumoto	2012	M	18	Catatonia	4	13 sessions	Resolved catatonia
Mann	2012	F	14	Catatonia	8	7 sessions	Improved catatonia, cognition, mobility and autonomic features
Wilson	2013	F	14	Catatonia	BFCRS21	14 sessions	Improved catatonia and movement
Ramanathan	2013	F	17	Catatonia	5	13 sessions	Improved catatonia
Yuksel	2014	F	23	Catatonia	I.I.	N.S.	N.S.
Hermans	2015	F	20	Catatonia	2	N.S.	Improved catatonia
Hermans	2015	F	23	Catatonia	6	N.S.	No improvement
Huang	2015	F	25	N.S.	2	1 session	Ceased due to neurological deterioration
Jones	2015	M	17	Catatonia	7	2 sessions Bi-temporal	Improved catatonia
Koksal	2015	F	25	Catatonia	2	2 sessions	Ceased due to seizures
Kramina	2015	F	15	Catatonia	6	2 sessions	Ceased due to neurological deterioration
Peng	2015	F	30	N.S	2	31 sessions	Not stated
Rajahram	2015	M	35	Behaviour refractory to immunotherapy	5	Multiple sessions	N.S.
Miller	2016	F	30	Catatonia	2	33 sessions	N.S.
Murdie	2016	F	17	Catatonia	I.I.	N.S.	Improved catatonia and paranoia
Sunwoo	2016	F	27	Catatonia and refractory dyskinesia	5	13 sessions, brief pulse	Improved catatonia and dyskinesia
Gough	2017	F	71	Catatonia	I.I	Multiple sessions	N.S.
Hermans	2017	F	25	Catatonia	6	1 session	Ceased due to seizures
Kar	2017	M	21	Catatonia	7	12 sessions	Improved catatonia
Medina	2017	F		Catatonia	BFCRS24	6 sessions bi-temporal	Resolved catatonia
Rong	2017	F	52	Psychotic depression	5	2 sessions	N.S.
Palakkuzhiyil	2018	M	47	Catatonia	7	N.S.	No improvement

N.S.: Not stated.
I.I.: Insufficient information.
BFCSI: Bush Francis Catatonia Screening Instrument.
BFCRS: Bush Francis Catatonia Rating Scale.

compared with that for surgical removal of teratomas and other malignancies. Anaesthetics that act at the NMDA receptor, such as nitrous oxide and ketamine, may behave unpredictably in anti-NMDA receptor encephalitis [23]. Ketamine was not used in any of these cases, but has been the subject of increasing interest for potential anti-depressant and neuroprotective cognitive effects, with less anti-convulsant activity than proprofol [24,25]. Propofol, the most common ECT anaesthetic, has been used without concern in anti-NMDA receptor encephalitis [23]. A non-depolarising muscle relaxant may be a safer alternative to succinylcholine, in the setting of potential hyperkalaemia with catatonic rigidity or longstanding immobility [26].

Autonomic disturbance with alterations of temperature, heart rate and blood pressure, as well as respiratory depression, arrhythmias and asystole have been described in anti-NMDA receptor encephalitis [1]. Tachycardia and fever occurred in over half of these cases without disruption to the ECT course, however, more severe autonomic disturbances such as central hypoventilation may pose greater concern, especially as prolonged airway protection with intubation does not routinely occur with ECT administration [27]. ECT can result in a brief parasympathetic mediated bradycardia, hypotension and asystole, followed by a more prominent sympathetic response with hypertension and tachycardia [4]. Awareness, assessment and availability of support is recommended.

The cognitive effects of ECT in anti-NMDA receptor encephalitis are unclear, with both the resolution of cognitive deficits and onset of such deficits reported in these cases. Cognitive deficits, as initial symptoms as well as in both the active and recovery phases of anti-NMDA receptor encephalitis are common, thought to be due to impact on mesial-temporal structures [1,19,28]. Early effective treatment may result in improved cognitive outcomes, of which ECT may assist [8]. However, ECT has known cognitive side-effects, generally limited to the first few days after treatment, with resolution beyond baseline seen after that in domains such as working memory, processing speeds, anterograde memory and executive functioning [29]. Conflicting results may also be related to the inconsistent or non-specific documentation of cognition in these cases.

The cases identified represent a small fraction of published anti-NMDA receptor encephalitis cases and cannot be generalised to those with non-psychiatric symptoms [1,2]. Since data was collected from case reports, the results are subject to selection, reporting and publication bias. Poor documentation potentially has led to an underestimation of catatonic symptoms and reduced BFCSI scores and results in lack of clarity to which symptoms have improved following ECT. There was limited documentation of ECT frequency, dose, electrode positioning, seizure quality and duration, response and adverse events, preventing further analysis for ECT optimisation.

The primary treatment for anti-NMDA receptor encephalitis is immunomodulatory therapy, but ECT appears to be a safe, effective adjuvant treatment, especially for psychiatric symptoms such as catatonia. In less than 15%, ìt was prematurely ceased, with no lasting adverse consequences for the patient. Availability of appropriate monitoring, support and anaesthetic expertise may dictate the environment in which ECT is performed. Informed patient consent may be difficult and legal aspects, especially in adolescents and children, need careful consideration. The risks of deferring appropriate ECT treatment in anti-NMDA receptor encephalitis or delaying ECT until this disorder is excluded must be evaluated. Further research to define the ECT parameters and confirm safety in a prospectively recruited cohort would be beneficial. However, acknowledgement of the existing literature for safe ECT use in catatonia and medically complex cases is reassuring.

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Declaration of interest

None.

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