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Use of ECT in Autism Spectrum Disorder and/or Intellectual Disability: A Single Site Retrospective Analysis

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Abstract

Autism spectrum disorder (ASD) and intellectual disability (ID) are heterogenous and prevalent conditions which may occur in isolation or as a co-morbidity. Psychiatric co-morbidity is common with limited treatment options. Preliminary research into electroconvulsive therapy (ECT) for these conditions has been encouraging. Thus, further research in this patient population is warranted. We conducted a 10-year retrospective review of the electronic medical record and identified intellectually capable individuals with ASD (IC-ASD), and those with ASD+ID or ID who received at least three ECT treatments. 32 patients were identified of which 30 (94%) experienced positive clinical response, defined as a clinical global impression-improvement (CGI-I) score of 3 or less. The average retrospective CGI-I score across all groups was 1.97, and results of a t-test performed on CGI-I scores indicated improvement across all groups (t=−16.54, df=31,

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p<0.001, 95% CI = [1.72, 2.22]). No significant adverse events were identified based on clinical documentation. Our findings further support previous ECT research in this patient population.

Keywords

Autism; Intellectual Disability; Neurodevelopmental; Electroconvulsive Therapy; Neuromodulation

Autism spectrum disorder (ASD) is a highly heterogenous neurodevelopmental disorder (NDD) (Jannati et al., 2020) which presents with deficits in social interaction and communication along with restricted/repetitive pattern of behaviors and interests. (American Psychiatric Association, 2013) ASD is a condition of high prevalence (Lazoff et al., 2010; Shaw et al., 2021) and clinical need, (Becker et al., 2020; Howlin et al., 2004; Tillmann et al., 2019) which can present with co-morbid intellectual disability (ASD+ID). Historically referred to as mental retardation, intellectual disability (ID) is also a prevalent NDD (Maulik et al., 2011; Wittchen et al., 2011) and includes intellectual and adaptive deficits in conceptual, social, and practical domains. (American Psychiatric Association, 2013) The Centers for Disease Control estimates that 31-50% of individuals with ASD meet criteria for ASD+ID. (Christensen, 2019; Maenner, 2020) However, studies have found that only 6% of participants in ASD research have ASD+ID; (Russell et al., 2019) with rates of inclusion potentially worsening over time. (Stedman et al., 2019) Thus, a disparity of care has resulted due to limited treatment strategies for the most severely impacted autistic individuals. Yet, despite greater inclusion in clinical research, treatment options are often limited and side effect laden for intellectually capable individuals with ASD (IC+ASD) as well. (Alfageh et al., 2019; Hutton, 2008; Smith & Pierce, 2022; Zhou et al., 2021) For both IC+ASD and ASD+ID persons, pharmacologic treatment response rates across multiple co-morbidities are lower compared to the general population. Moreover, the only FDA approved pharmacologic treatments in ASD are aripiprazole and risperidone for irritability. At present, there are no FDA approved treatments for co-morbid psychiatric diagnoses in ASD or core feature of ASD. (Henneberry et al., 2021) Psychotherapeutically, preliminary studies incorporating cognitive behavioral therapy for treatment of psychiatric co-morbidity have been encouraging; though frequently exclude individuals with co-morbid ID. (Wood et al., 2020) Based on these trends, research investigating other potential treatment options for ASD+IC, ASD+ID, and ID individuals is urgently needed.

Since its introduction in 1938, electroconvulsive therapy (ECT) has demonstrated safety and efficacy in the treatment of catatonia, suicidal ideation, and affective disorders for neurotypical (NT) individuals across the life-cycle. (Bahji et al., 2019; Espinoza & Kellner, 2022; Hedley & Uljarević, 2018; Horowitz et al., 2018; Luccarelli et al., 2020a; Luccarelli, McCoy, Uchida, et al., 2021; Luccarelli, McCoy, Seiner, et al., 2021; Tørring et al., 2017) Moreover, encouraging reports and reviews have been published which point to ECT as a potentially efficacious treatment for symptoms common in ASD and ID. These include mood dysregulation, irritability, psychosis, self-injury, and catatonia. (Adıgüzel Akman et al., 2021; Consoli et al., 2013; DeJong et al., 2014; Desarkar et al., 2018; Dodd et al., 2016;

Eaton et al., 2021; Oakley et al., 2021; Park et al., 2020; Vaquerizo-Serrano et al., 2021; Wachtel, 2019; Withane & Dhossche, 2019)

Overall, ECT is a safe procedure. Common minor and self-limiting side effects include headache, muscle soreness, and post procedure nausea/vomiting. Cognitive side effects are common in ECT as well and include anterograde and/or retrograde amnesia. (Espinoza & Kellner, 2022) These cognitive difficulties often resolve in the weeks following treatment and do not appear to be of greater severity for adolescents and young adults. (Luccarelli, McCoy, Uchida, et al., 2021) Moreover, many patients experience improved cognitive functioning as psychiatric illness improves. The most concerning potential side effect of ECT is an acute cardiopulmonary event, which occurs in less than 1% of treatments. The risk can be further mitigated by consultation with cardiology for patients with a history of cardiac disease. Other serious adverse events associated with ECT are rare and include, but are not limited to, the following: cardiac arrhythmias, prolonged seizures, aspiration, and prolonged apnea. (Espinoza & Kellner, 2022)

However, access to ECT is limited due to provider availability, stigma, and restrictive legislation; especially for pediatric patients and individuals living in rural areas. (Espinoza & Kellner, 2022; Johnston, 2015; Luccarelli et al., 2020b; Miller et al., 2022; Sackeim, 2017) Additionally, the vast majority of ECT research in this patient population are case reports, case series, or smaller retrospective reviews. (Consoli et al., 2013; Park et al., 2020; van Waarde et al., 2001) To our knowledge there are no studies which compare the efficacy of ECT between IC+ASD, ASD+ID, and ID groups nor studies which investigate the use of ECT in the treatment of depression, bipolar disorder, schizophrenia, or other psychiatric co-morbidity in ASD or ID populations despite high prevalence. (Dunn et al., 2020; Mazza et al., 2020; Park et al., 2020) Thus, in this study we have conducted an exploratory single site retrospective analysis of IC+ASD, ASD+ID, and ID individuals who received ECT treatment over past ten years. We hypothesize that regardless of NDD or presenting psychiatric co-morbidity, clinical response rates to ECT will be high. Overall, we aim to expound upon the current literature in the field and provide insight into ECT treatment in this patient population.

Methods

Study Population:

Using the SlicerDicer software found within the Epic Systems electronic medical record, (Epic SlicerDicer, 2021) we conducted a single site retrospective analysis of a private university hospital within the southern United States. Patient information was collected from individuals who received inpatient and/or outpatient ECT from the date 10/24/2011 to 10/18/2022. The primary analysis aim was to identify individuals under the inclusion criterion of the following: 1) received at least three ECT treatments as an inpatient and/or outpatient and 2) had a diagnosis of IC+ASD, ASD+ID, and ID based on free text and/or billing codes within the electronic medical record. Of note, in our analysis we identified patients who received a traditional acute series of ECT, defined as receiving ECT at least twice weekly. We also identified a smaller group of patients who did not undergo an acute series of ECT, but were treated on a weekly, biweekly, or monthly basis. We have

included both groups in this study. Patients were excluded if they had a co-morbid genetic syndrome or had not received a diagnosis of IC+ASD, ASD+ID, or ID. Our search was conducted using various combinations of the following terms: “autism”, “autism spectrum disorder”, “asperger’s syndrome”, “neurodevelopmental disorder”, “intellectual disability”, “intellectual developmental disorder”, “neuromodulation”, and “electroconvulsive therapy”. This study was approved and overseen by the institutional review board (#211979).

Case Selection:

As outlined in Figure 1, sixty-seven patients were initially identified in our search. All patients identified underwent ECT consultation by a psychiatrist within the health system. Two authors (JRS and CEH) reviewed each identified chart in the electronic medical record and reached an agreement on inclusion or exclusion. The following number of patients were excluded: twenty did not have a formal NDD diagnosis, nine had an NDD diagnosis but did not pursue ECT after consultation, two patients did not receive at least three ECT treatments, two had a diagnosis of DiGeorge syndrome, and two had a diagnosis of cerebral palsy without co-morbid ASD or ID.

Statistical analysis:

We used chi-squared tests to compare presenting symptom differences between groups using p-values and a robust effect size index that is equal to ½ Cohen’s *d*. (Vandekar et al., 2020) Clinical indication for ECT often included more than one presenting symptom. For each patient included in the study, we obtained a retrospective clinical global impressions-improvement (CGI-I) score (Busner & Targum, 2007) via author review of inpatient and/or outpatient progress notes, ECT procedural documentation, inpatient admission and/or outpatient intake notes, and consult documentation which explicitly discussed symptoms targeted by ECT. Using CGI-I data, we performed a t-test to investigate whether the mean CGI-I score was different from the null result of “no change” value of 4. Notably, seven patients underwent multiple acute series of ECT treatment. In all instances, CGI-I scores from only the most recent acute series were included in the statistical analysis.

Results

A total of thirty-two individuals met inclusion criteria. Sample demographics and statistics are provided for demographic, symptom, treatment, and clinical response variables in Table 1. Presenting symptoms, as determined by reason for ECT consultation, included catatonia (n=20), psychosis (n=12), depression (n=8), self-injury (n=6), and mania (n=1). Thirty patients received a traditional acute series of ECT, with one ASD+ID patient resuming ECT at a frequency of one treatment per month following the completion of three acute series. Additionally, two ASD+ID patients received ECT on a weekly or biweekly basis. Per information from progress and consultations notes in the medical record, the frequency of ECT for these patients was chosen based on the patients living in rural areas, challenges in transportation due to symptoms of negativism and agitation, as well as an inability to receive inpatient psychiatric care due to the patients’ need for support, and/or concern for decompensation when separated from primary caregivers. Co-morbid psychiatric diagnoses at the time of first ECT treatment are outlined in Table 2 for the ASD+ID acute series

group, Table 3 for the ASD+ID weekly, biweekly, or monthly ECT frequency group, Table 4 for the ID group, and Table 5 for the IC+ASD group. The most commonly used final ECT parameters were bitemporal electrode placement (27/32) with brief pulse (25/32), and charge set to 576 mC (25/32). Thirty of the thirty-two patients began treatment with an acute series of ECT. One ASD+ID patient received weekly ECT, and another received biweekly ECT from the onset of treatment. Additional information regarding both patients can be found in Table 3. For the purposes of our analysis, any ECT treatment which occurred at a frequency of less than two treatments in one week was defined as maintenance ECT (mECT). Thus, data from the two patients who did not undergo an acute series of treatment was termed mECT. Twenty-five of thirty-two patients pursued mECT. The median and average number of mECT treatments received per patient was 7 and 15, respectively. The median and average length of time between mECT treatments at 14 and 15.4 days, respectively. No significant adverse events were reported. The average rate of ECT treatments rose each year in this population, possibly due to provider level of comfort after treating similar cases. Moreover, three ECT providers at this site are adult as well as child and adolescent psychiatrists and may have recognized the need for ECT treatment in developmental delayed persons more readily based on their training and exposure to this patient population. Pharmacologically, concurrently administered antipsychotics, mood stabilizers, and benzodiazepines were reported given their potential impact on ECT seizure induction. (Zolezzi, 2016)

Regarding clinical response, 9/9 patients in the ASD+ID acute series group, 3/3 in the ASD+ID weekly, biweekly, or monthly ECT frequency group, 12/13 in the ID group, and 6/7 in the IC+ASD group had a score of 3 or less on the CGI-I, indicative of a positive clinical response. Overall, 30/32 (94%) patients experienced clinical improvement in their presenting symptomology. The average retrospective CGI-I score across all groups was 1.97, with a median of 2. Results of the t-test performed on the CGI-I indicated improvement across all groups ($t=-16.54$, $df=31$, $p<0.001$, 95% CI = [1.72, 2.22]). The two non-responders were in the ID and IC+ASD groups, they were also diagnosed with seronegative autoimmune encephalitis (SNAE). The patient with SNAE in the ID group received intravenous immunoglobin and mycophenolate which resulted in catatonia resolution. The patient in the IC+ASD group received treatment with lorazepam for catatonia due to SNAE over the course of one year; resulting in resolution of catatonia. Both non-responding patients presented with catatonia as their symptom targeted by ECT.

Autism spectrum disorder with intellectual disability who received ECT in acute series:

Nine patients were included in this group (Tables 1 & 2). The mean age at the time of initial presentation was 21 years, with eight biologically male and one biologically female patient reported. Catatonia was the most frequent presenting symptom (n=8), followed by self-injury (n=5), and psychosis (n=1). The mean number of ECT treatments in an acute series was 8.9. Bitemporal electrode placement and brief pulse was utilized in all cases. Notably, ultra-brief pulse was used for patient 5 on Table 2 during the first acute series; this was transitioned to brief pulse for a second acute course at a later time. Regarding psychiatric co-morbidity, three cases of schizophrenia and two cases of type 1 bipolar disorder were reported, along with single cases of attention/deficit hyperactivity disorder, intermittent

explosive disorder, post-traumatic stress disorder and unspecified anxiety disorder. Patients number 5, 7 and 8 received multiple courses of acute ECT treatment as outlined in Table 2. Over the course of treatment, 7/9 patients were prescribed a second-generation antipsychotic (SGA) and a mood stabilizer or antiepileptic, 6/9 a benzodiazepine, and 2/9 an NMDA receptor antagonist. Regarding clinical response, for all acute series of ECT received in this group; the average CGI-I score was 1.9.

Autism spectrum disorder with intellectual disability who received ECT weekly, biweekly, or monthly:

Three patients were included in this group (Tables 1 and 3). All patients were biologically male, diagnosed with ASD+ID, and had an average age of 24.7 years at the time of initial presentation. Patients 1 and 2 did not undergo an acute series of ECT due to challenges in transportation brought about by ongoing symptoms of negativism and agitation, as well as an inability to receive inpatient psychiatric care due to the patients' need for support and/or concern for decompensation when separated from primary caregivers. Patient 1 also lived in a rural area, further compounding the difficulties in transportation. Patients 1 and 2 had a positive clinical response, both scoring a CGI-I of 2. However, attempts to reduce the frequency of ECT treatment to lower than every 14 days for patient 1 and every 6 days for patient 2 have resulted in rapid recurrence of catatonic symptomology. For patient 1, five attempts have been made to transition the patient to mECT. However, catatonia has returned with each attempt. As per Table 3, patient 3 underwent three acute series of ECT with minimal improvement. However, the patient and his family returned for ECT and patient 3 has received 6 mECT treatments with a median of 21 days between each treatment. After this change, patient 3 has shown some mild clinical improvement; with a change in CGI-I score from 4 to 3. Specifically, patient 3 has resumed non-verbal communication. Notably, concerns regarding temporal lobe epilepsy have been expressed for this patient, though the diagnosis has yet to be confirmed. At present, patient 3 continues to receive ongoing mECT.

Intellectual disability:

Thirteen patients were included in the ID group (Tables 1 & 4). The mean age at initial presentation was 31.5 years, with two biologically male and eleven biologically female patients reported. Presenting symptoms included catatonia (n=6), self-injury (n=5), and psychosis (n=2). Regarding ECT parameters, the average number of treatments in an acute series of ECT was 10.2. Nine patients received bitemporal electrode placement, three right unilateral, and one bifrontal. Brief pulse was used in nine cases, and ultra-brief in four. Co-morbid psychopathology included the following: major depressive disorder (n=5), type 1 bipolar disorder (n=3), unspecified psychosis (n=2), schizoaffective disorder, bipolar type (n=1), generalized anxiety disorder (n=1), post-traumatic stress disorder (n=1), and schizophrenia (n=1). Over the course of treatment, 8/13 patients were prescribed a SGA, 7/13 a mood stabilizer or antiepileptic, and 7/12 a benzodiazepine. Regarding clinical response, for all acute series of ECT received in this group; the average CGI-I score was 1.9.

Intellectually capable individuals with autism spectrum disorder:

Seven patients were included in the ASD+IC group (Tables 1 & 5). The mean age on initial presentation was 20.6 years, with four biologically male and three biologically

female patients reported. Presenting symptoms included psychosis (n=5), depression (n=4), catatonia (n=2), and mania (n=1). The average number of ECT treatments in an acute series was 10.4. Bitemporal ECT electrode placement occurred in six cases and right unilateral placement in one. Brief pulse was used in five cases and ultra-brief in two. Co-morbid psychopathology included major depressive disorder (n=5), generalized anxiety disorder (n=2), attention/deficit hyperactivity disorder (n=2), obsessive compulsive disorder (n=1), type 1 bipolar disorder (n=1), and schizophrenia (n=1). Over the course of treatment, 5/7 patients were prescribed a SGA, 3/7 a mood stabilizer or antiepileptic, 2/7 a benzodiazepine, and 2/7 an NMDA receptor antagonist. Regarding clinical response, for all acute series of ECT received in this group; the average CGI-I score was 2.1.

Discussion

The aim of our study was to investigate the use of ECT for persons with diagnoses of IC+ASD, ASD+ID, and ID. Overall, regardless of specific symptoms targeted by ECT, the clinical response rate was 94% (30/32) across all three groups, with an average and median CGI-I of 2. No significant adverse events reported. The two cases which did not respond to treatment initially presented with catatonia. Both cases were thought to be due to SNAE and responded to long term lorazepam treatment or intravenous immunoglobin and mycophenolate. Similarly, temporal lobe epilepsy has long been considered for patient 3 on Table 3. Patient 3 required three acute series of ECT and then ongoing mECT to experience a mild clinical response.

Catatonia was the most common presenting symptom for patients diagnosed with ASD+ID or ID and was present in two individuals with an ASD+IC diagnosis. For patients with ASD+ID, self-injury was the second most common presenting symptom. This is of clinical importance as recurrent self-injury is a common and debilitating symptom in this patient population. Moreover, recent literature has suggested that recurrent self-injury is a symptom along the catatonia spectrum for ASD+ID individuals. (Wachtel et al., 2018) Catatonia itself is an affective and psychomotor condition with distinct physical examination findings. While most often associated with schizophrenia, affective disorders, and medical illnesses; (Wachtel et al., 2011) interest in catatonia presenting in NDDs has risen in recent years. (Vaquerizo-Serrano et al., 2021) A recent meta-analysis by Vaquerizo-Serrano and colleagues found that 20.2% of individuals with ASD had features of catatonia; presenting most often with new onset speech impairment, negativism, and aggression. (Vaquerizo-Serrano et al., 2021)

Thus, while traditionally described symptoms of catatonia do occur for individuals with ASD or other NDDs; consideration of recurrent-self injury, aggression, loss of verbal abilities, and worsening negativism should be considered when considering the diagnosis of catatonia and ECT as a treatment option. Critically, a missed diagnosis and/or delay to treatment may result in progression to malignant catatonia, a condition associated autonomic instability and rates of mortality as high as 10%-20% if left untreated. (Walther et al., 2019) Diagnostically, recent literature suggests that most cases of catatonia in individuals with ASD occur in the absence of an underlying medical or psychiatric condition; (Consoli et al., 2012; Withane & Dhossche, 2019) though medical work up to rule out organic causes

should always be pursued. (Park et al., 2020) From the treatment perspective, catatonia is most often managed with a combination of IV lorazepam and ECT. Encouragingly, the results of our study and others report high clinical response rates of catatonia in ASD when treated with ECT. (Park et al., 2020; Vaquerizo-Serrano et al., 2021) However, the efficacy and tolerability of lorazepam in the treatment of catatonia in ASD has recently been called into question (Vaquerizo-Serrano et al., 2021) and recent research has reported low ECT utilization in pediatric catatonia; (Luccarelli et al., 2022) further highlighting the need for ECT availability given the potential life threatening complications associated with treatment delay; often driven by restrictive legislation, stigma, and limited ECT provider availability. (Espinoza & Kellner, 2022; Miller et al., 2022)

Depression, mania, and psychosis were reported in the IC+ASD group and were common in the patients diagnosed with ASD+ID or ID. ECT is a well-established treatment of suicidal ideation, mood disorders, and psychosis in NT individuals. (Bahji et al., 2019; Espinoza & Kellner, 2022; Hedley & Uljarević, 2018; Horowitz et al., 2018; Luccarelli et al., 2020a; Luccarelli, McCoy, Seiner, et al., 2021; Luccarelli, McCoy, Uchida, et al., 2021; Tørring et al., 2017) This is a point of consideration as individuals with ASD and ID are at an elevated risk for mood disorders, psychosis, self-injury, and suicidal behaviors; (Dodd et al., 2016; Hedley & Uljarević, 2018; Hepburn et al., 2014; Horowitz et al., 2018; Ludi et al., 2012; Oakley et al., 2021; O'Halloran et al., 2022; Schwartzman et al., 2021; Segers & Rawana, 2014) yet no clear treatment options are well researched, especially in cases of ASD+ID. We also found that bitemporal electrode placement and brief pulse duration was the most commonly used ECT parameter across all groups. While right unilateral electrode placement is often preferred due to the lower risk of cognitive side effects, (Kellner et al., 2010) bitemporal electrode placement in ECT has long been considered the definitive treatment of catatonia and psychosis, (Park et al., 2020; Paus et al., 2001) common presenting symptoms in our study.

The high response rate seen in our study and in other reports, (Adıgüzel Akman et al., 2021; Consoli et al., 2013; DeJong et al., 2014; Desarkar et al., 2018; Dodd et al., 2016; Eaton et al., 2021; Oakley et al., 2021; Park et al., 2020; Vaquerizo-Serrano et al., 2021; Wachtel, 2019; Withane & Dhossche, 2019) is tempered by small samples sizes and the inherently open label nature of ECT. Moreover, a neurobiologic explanation of treatment response to ECT is difficult to characterize given the poorly understood mechanism of ECT and neurobiologic underpinnings of ASD and/or ID; all of which are likely influenced by multiple biological systems including neuroendocrinologic, neuroplastic, and others. (Casanova et al., 2020; Iwase et al., 2017; Singh & Kar, 2017) However, one of the leading hypotheses behind the ASD is the parvalbumin deficiency hypothesis. (Hashemi et al., 2017; Lee et al., 2017; Steullet et al., 2017) Reduced numbers of parvalbumin-expressing cells have been reported in human postmortem brain samples (Hashemi et al., 2017) and animal models of ASD. (Lee et al., 2017) Additionally, reduced levels of parvalbumin expression are associated with ASD-like behavioral deficits and sensory-motor symptoms associated with ASD. In animal models, long term reversal of parvalbumin deficits by pharmacologic or cell type specific gene rescue normalize or diminish these symptoms. (Lee et al., 2017; Mukherjee et al., 2019; Selimbeyoglu et al., 2017) Thus, researchers have identified an excitatory:inhibitory imbalance as a potential etiology and treatment target in ASD. (Rojas

& Wilson, 2014; Smith et al., 2022; Sokhadze et al., 2009; Steullet et al., 2017) The excitatory:inhibitory imbalance may represent glutaminergic cortical excitotoxicity, (Rojas, 2014) hyperplasticity due to dysfunction of *N*-methyl-D-aspartate receptor mediated long-term depression and potentiation-like plasticity mechanisms, and/or inhibitory GABAnergic dysfunction. (Buzsáki & Wang, 2012; Casanova et al., 2020; Jeste & Nelson, 2009; Smith et al., 2022) One explanation is that ECT may induce neurogenesis of inhibitory interneurons to offset possible cortical excitotoxicity, or that ECT reduces cortisol mediated reduction of inhibitory interneuron neuroplasticity as has been reported in animal models. (Hellsten et al., 2002; Inta et al., 2013; Singh & Kar, 2017; Wennström et al., 2006)

For younger patients with ASD, ID, catatonia, psychosis, and/or other psychiatric conditions which would necessitate use of ECT; even less is known regarding what influence ECT may have on developing neurobiology. However, our results and those from other studies investigating the clinical efficacy and side effect profiles of ECT in adolescents and young adults, are encouraging. (Castaneda-Ramirez et al., 2022; Luccarelli, McCoy, Seiner, et al., 2021; Luccarelli, McCoy, Uchida, et al., 2021) In addition, case reports beginning as early as the 1940s have noted safe administration of ECT in pediatric patients (Sirgiovanni, 2021) and little is known regarding possible long-term implications of untreated psychosis or catatonia if ECT treatment is indicated but unavailable. While much more research is needed, this is an area worthy of consideration for future investigations. Specifically, we would recommend ECT providers consistently utilize systematic clinical assessment tools to allow for more precise data to be obtained. Furthermore, future ECT research and clinical care may also consider administering brief and developmentally appropriate cognitive tests for patients before, during, and after receiving ECT. Thus, allowing for greater investigation into cognitive outcomes of ECT, severe psychiatric illness, and the interface of the two; especially in cases of NDDs.

Limitations:

Overall, our study had several limitations. First, our sample was heterogenous and statistically significant only when incorporating the retrospective CGI-I regardless of presenting symptoms. Additionally, the data does not contain information regarding socioeconomic status and our study was a single site retrospective analysis. Therefore, the quality of our findings are limited due to reduced generalizability, as well as a lack of blinding and randomization. Regarding the interplay of socioeconomic status and access to ECT, we would encourage future research to address this area given challenges associated with ECT access for individuals in rural areas, (Johnston, 2015) as was reported for patient 1 on Table 3. Another limitation of our study is that not all patients underwent an acute series of ECT as seen on Table 3. However, it is notable that these individuals were unable to present for an acute series due to the severe quality of their symptomology and a lack of inpatient psychiatric options; thus reflecting the well described paucity of clinical care and research for the most severely impacted of autistic individuals. (Lord et al., 2022)

Another limitation includes the retrospective manner in which the CGI-I was collected, as well as a lack of other systematic measurement-based tools in determining improvement in presenting symptomology; though there are very few measurement-based tools designed for

individuals with NDD or ASD, especially for pediatric persons. (McFayden et al., 2021) One notable exception includes catatonia. The Bush-Francis Catatonia Rating Scale (BFCRS) is often used in research and clinical work. (Bush et al., 1996) However, the BFRS is designed for NT adults and may not well characterize catatonia for pediatric individuals or those with NDDs. Specifically, symptoms of urinary incontinence, nudism, acrocyanosis, and others are included in the Pediatric Catatonia Rating Scale and Kanner catatonia rating scale for individuals with NDDs. (Benarous et al., 2016; Bush et al., 1996; Carroll et al., 2008) Future work should consider inclusion of such scales for screening and symptom monitoring, as well as address the need for developmentally informed measures. In addition, specific IQ scores and reports of adaptive functioning were not included in charts reviewed. This information would be useful in determining if clinical response to ECT is correlated with the degree of IQ impairment or adaptive functioning and should be investigated in future work. Overall, the lack of quantifiable and systematic measurement-based assessments in our study may have resulted in less accurate and/or consistent reporting of symptoms, adding an additional limitation to our results.

Conclusions:

Overall, there was a significantly high rate of clinical response to ECT in the IC+ASD, ASD+ID, and ID presenting with co-morbid catatonia, self-injury, psychosis, mania, and depression. We are also hopeful that continued reports of lifesaving and life sustaining ECT treatment in this high need patient population, will lead to less restrictive legislation, destigmatization, and greater ease of access for patients and their families.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability:

The data that support the findings of this study are available from the corresponding author, JRS, upon request.

Abbreviations:

ASD	Autism spectrum disorder
NDD	neurodevelopmental disorder
ASD+ID	autism spectrum disorder with co-morbid intellectual disability
ID	intellectual disability
IC+ASD	intellectually capable autism spectrum disorder

ECT	electroconvulsive therapy
NT	neurotypical
CGI-I	clinical global impressions improvement scale
mECT	maintenance ECT
SNAE	seronegative autoimmune encephalitis
SGA	Second-generation antipsychotic
BFCRS	Bush Francis Catatonia Rating Scale

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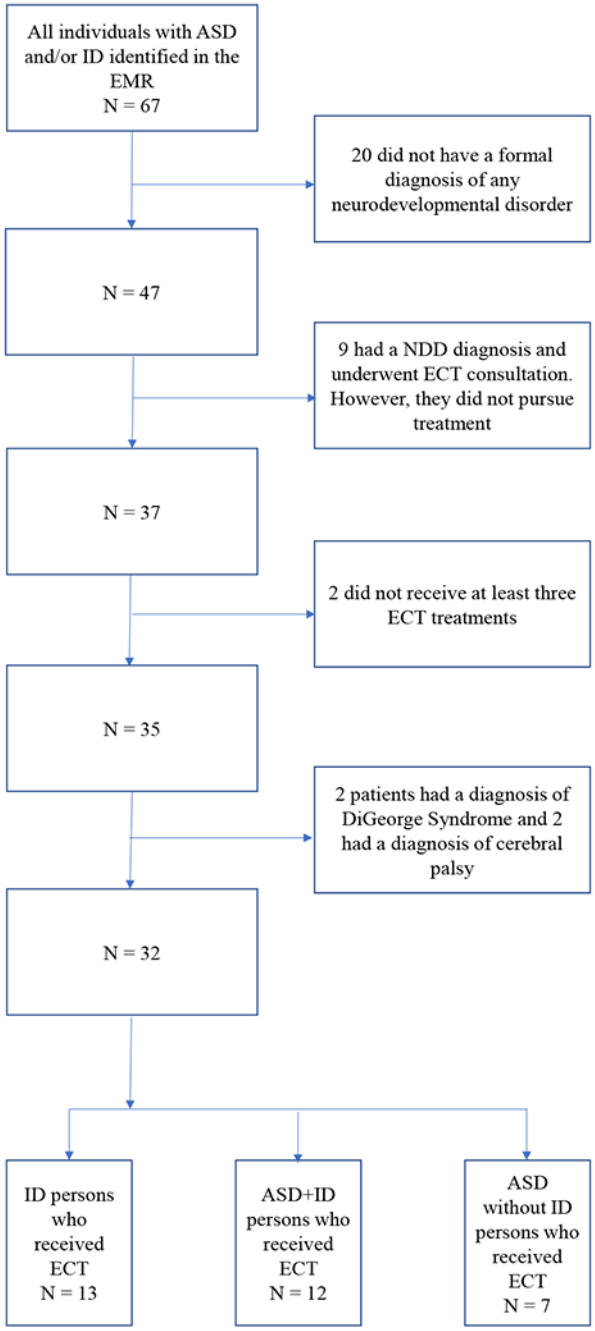

Figure 1:
Flow diagram of study inclusion

Table 1.

Patient Demographics and Statistical Analysis

	Autism with co-morbid intellectual disability (N=12)	Autism without co-morbid intellectual disability (N=7)	Intellectual Disability (N=13)	All patients (N=32)	Test Statistic	Effect Size
Demographics						
Biologically Male	11/12 (91.67)	4/7 (57.14)	2/13 (15.38)	17/32 (53.12)	$X^2(2)=14.64$, P<0.001	0.63
Age	21.92(6.067)	20.57(4.504)	31.54(15.37)	25.53(11.58)	F(2,29)=3.44, P=0.045	0.37
Present ing Symptom for ECT						
Catatonia	10/12 (83.33)	2/7 (28.57)	8/13 (61.54)	20/32 (62.50)	$X^2(2)=5.67$, P=0.06	0.34
Self Injury	6/12 (50.00)	0/7 (0.00)	0/13 (0.00)	6/32 (18.75)	$X^2(2)=12.31$, P<0.001	0.57
Psychosis	2/12 (16.67)	5/7 (71.43)	5/13 (38.46)	12/32 (37.50)	$X^2(2)=5.67$, P=0.06	0.34
Depression	0/12 (0.00)	4/7 (57.14)	4/13 (30.77)	8/32 (25.00)	$X^2(2)=8.09$, P=0.02	0.44
E CT Pulse Width						
Ultra-brief pulse	1/12 (8.33)	2/7 (28.57)	4/13 (30.77)	7/32 (21.88)	$X^2(2)=2.07$, P=0.35	0.05
Brief Pulse	11/12 (91.67)	5/7 (71.43	9/13 (69.23)	25/32 (78.12)	$X^2(2)=2.07$, P=0.35	0.05
ECT E lectrode Placement						
Right Unilateral	0/12 (0.00)	1/7 (14.29)	3/13 (23.08)	4/32 (12.50)	$X^2(2)=3.06$, P=0.22	0.18
Bitemporal	12/12 (100.00)	6/7 (85.71)	9/13 (69.23)	27/32 (84.38)	$X^2(2)=4.49$, P=0.11	0.28
Bifrontal	0/12 (0.00)	0/7 (0.00)	1/13 (7.69)	1/32 (3.12)	$X^2(2)=1.51$, P=0.47	0.00
CI inical Response						
Resolution of Presenting Symptom	10/12 (83.33)	6/7 (85.71)	12/13 (92.31)	28/32 (87.50)	$X^2(2)=0.49$, P=0.78	0.00
CGI	2.00 (0.426)	2.14 (0.900)	1.85 (0.800)	1.97 (0.695)	F(2,29)=0.418, P=0.662	0.00

Table 2.

Demographics, clinical data, electroconvulsive therapy parameters, and treatment response for patients with autism spectrum disorder and co-morbid intellectual disability receiving acute series

Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT	Psychiatric co-morbidity at the time of ECT	Antipsychotics, Mood stabilizers and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No. and median days	Notes on clinical response
1	14	Male	Catatonia Self-injury	Attention-deficit/ hyperactivity disorder	Olanzapine 20 mg Divalproex Sodium 1,500 mg Lorazepam 4 mg	14 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia and self-injury
2	20	Male	Catatonia Self-injury	Bipolar I disorder	Clozapine 200 mg Divalproex Sodium 2,000 mg	16 treatments CGI-I: 3	Bitemporal	Brief 576 mC	NR	First acute series ended without symptom resolution due to the patient developing pneumonia Patient was admitted medically; a second acute series was initiated 3 weeks later
	20	-	-	-	No changes	5 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia and self-injury
3	33	Female	Catatonia Psychosis	Schizophrenia	Olanzapine 10 mg Divalproex Sodium 1,000 mg Lorazepam 4 mg	8 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia and psychosis Lost to follow up for 1 year
	35	-	-	-		7 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia and psychosis
4	20	Male	Self-injury	Unspecified anxiety disorder	Lamotrigine 750 mg Lithium 1,200 mg Clonazepam 1 mg	10 treatments CGI-I: 2	Bitemporal	Brief 576 mC	22 mECT 28 days	Resolution of self-injury. *No longer receiving mECT*
5	26	Male	Catatonia Psychosis	Schizophrenia	Olanzapine 10 mg Lorazepam 9 mg	8 treatments CGI-I: 2	Bitemporal	Ultrabrief 230 mC	NR	Resolution of catatonia and psychosis, able to engage in treatment planning Lost to follow up for 1 year
	27	-	-	-	Paliperidone 3 mg Lorazepam 12 mg	7 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Similar therapeutic response to previous acute series Lost to follow up for 5 months
	27				Paliperidone 9 mg Lorazepam 6 mg	6 treatments CGI-I: 2	Bitemporal	Brief 576 mC	5 mECT 12 days	Similar therapeutic response to previous acute series Lost to follow up for 1 month

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Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT	Psychiatric co-morbidity at the time of ECT	Antipsychotics, Mood stabilizers and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No. and median days	Notes on clinical response
	28				Lorazepam 8 mg	5 treatments CGI-I: 2	Bitemporal	Brief 576 mC	10 mECT 14 days	Similar therapeutic response to previous acute series *No longer receiving mECT*
6	17	Male	Catatonia Self-injury	Schizophrenia	Lamotrigine 500 mg Lithium 1,200 mg Clonazepam 1 mg Alprazolam 1 mg	5 treatments CGI-I: 2	Bitemporal	Brief 576 mC	42 mECT 28 days	Resolution of catatonia and self-injury. *Currently receiving ongoing mECT*
7	21	Male	Catatonia Self-injury	Bipolar 1 disorder	Risperidone 3.5 mg Divalproex Sodium 1,500 mg Memantine 10 mg	6 treatments CGI-I: 1	Bitemporal	Brief 576 mC	NR	Resolution of catatonia and self-injury. mECT not pursed, symptoms returned within one month
	21					10 treatments CGI-I: 1	Bitemporal	Brief 576 mC	7 mECT 5 days	Resolution of catatonia and self-injury *Currently receiving ongoing mECT*
8	16	Male	Catatonia	Post-traumatic stress disorder	Quetiapine 50 mg Memantine 20 mg Clonazepam 9 mg	14 treatments CGI-I: 2	Bitemporal	Brief 576 mC	2 mECT 7.5 days	*Resolution of catatonia* Lost to follow up for 1 month
					Divalproex Sodium 500 mg Memantine 20 mg Clonazepam 13.5 mg	7 treatments CGI-I: 2	Bitemporal	Brief 576 mC	10 mECT 9 days	Resolution of catatonia Currently receiving mECT
9	22	Male	Catatonia	Intermittent Explosive Disorder	Lurasidone 120 mg Divalproex Sodium 2,500 mg	14 treatments CGI-I: 2	Bitemporal	Brief 576 mC	12 mECT 7.5 days	*Resolution of catatonia* Currently transitioning to mECT

NR: Did not receive mECT

Table 3.

Demographics, clinical data, electroconvulsive therapy parameters, and treatment response for patients with autism spectrum disorder and co-morbid intellectual disability receiving weekly, biweekly, or monthly

Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT	Psychiatric co-morbidity at the time of ECT	Antipsychotics, Mood stabilizers and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No., median days	Notes on clinical response
1	29	Male	Catatonia Self-injury	Bipolar I disorders	Olanzapine 20 mg Divalproex Sodium 2,000 mg Lithium 600 mg Lorazepam 4 mg	*No acute series, has treatments every 2 weeks for 6 years* CGI-I: 2	Bitemporal	Brief 576 mC	130 mECT 13 days	Resolution of self-injury and catatonia with a return to baseline when receiving ECT at a higher frequency than q2 weeks. 5 attempts have been made to transition the patient to mECT. When frequency is greater than q2 weeks, catatonia has returned. *Patient initially presented for treatment every 2 weeks due to transportation difficulties from a rural area, as well as intermittent symptoms of negativism and aggression.*
2	16	Male	Catatonia	Bipolar 1 disorder	Quetiapine 200 mg Memantine 20 mg Clonazepam 21 mg	*No acute series, received weekly treatment* CGI-I: 2	Bitemporal	Brief 576 mC	28 mECT 6 days	Resolution of catatonia, specifically robust return of communicative abilities and reduction in aggression. *When treatment frequency is greater than weekly, catatonia has returned. Patient initially presented for weekly ECT treatment due to difficulty in transport due to intermittent symptoms of negativism and aggression*
3	29	Male	Catatonia Psychosis	Bipolar I disorder	Aripiprazole 5 mg Lorazepam 6 mg	8 treatments CGI-I: 4	Bitemporal	Brief 576 mC	NR	Did not respond to ECT, concerns were raised regarding temporal lobe epilepsy
	29	-	-	-	Memantine 20 mg Lorazepam 6 mg	11 treatments CGI-I: 4	Bitemporal	Brief 576 mC	2 mECT 22.5 days	Returned for additional trial of ECT, did not respond.
	30	-	-	-	Risperidone 2 mg Oxcarbazepine 600 mg Lorazepam 24 mg	30 treatments CGI-I: 4	Bitemporal	Brief 576 mC	NR	Returned for additional trial of ECT, did not respond.
	30	-	-	-	Risperidone 2 mg Oxcarbazepine 600 mg Lorazepam 32 mg	*No acute series, received treatments monthly or every other month* CGI-I: 3	Bitemporal	Brief 576 mC	6 mECT 21 days	Returned for additional trial of ECT given failure of other treatments, began to respond non-verbally and saying yes or no. Previously patient was non-speaking. *Currently receiving ongoing mECT*

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NR: Did not receive mECT

Table 4.

Demographics, clinical data, electroconvulsive therapy parameters, and treatment response for patients with intellectual disability receiving acute series

Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT time of ECT	Psychiatric co-morbidity at the and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	Antipsychotics, Mood stabilizers	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No. and median days	Notes on clinical response
1	42	Female	Catatonia	Major depressive disorder	Lorazepam 20 mg	12 treatments CGI-I: 2	Bitemporal	Brief 576 mC	1 17 days	Resolution of catatonia *No longer receiving mECT*
2	13	Female	Psychosis	Schizoaffective disorder, bipolar type	Clozapine 150 mg Iloperidone 5 mg Divalproex Sodium 500 mg	18 treatments CGI-I: 2	Bitemporal	Ultrabrief 568 mC	17 14 days	Resolution of psychosis, then lost to follow up *No longer receiving mECT*
3	18	Male	Catatonia	None	Lorazepam 14 mg	6 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia
4	55	Female	Catatonia Depression	Major depressive disorder with psychotic features	None	5 treatments CGI-I: 2	Right Unilateral	Ultrabrief 230 mC	NR	Resolution of catatonia and depression, then lost to follow up
	58	-	-	-	Olanzapine 5 mg	13 treatments CGI-I: 2	Right Unilateral	Ultrabrief 384 mC	NR	Similar therapeutic response to previous acute series, then lost to follow up
	60	-	-	-	Olanzapine 5 mg	7 treatments CGI-I: 2	Right Unilateral	Ultrabrief 384 mC	NR	Similar therapeutic response to previous acute series
5	37	Female	Catatonia	Major depressive disorder and generalized anxiety disorder	Divalproex Sodium 1,000 mg Lorazepam 12 mg	14 treatments CGI-I: 4	Bitemporal	Brief 576 mC	NR	Did not respond to ECT. Diagnosed with seronegative autoimmune encephalitis. Patient received intravenous immunoglobin and mycophenolate; catatonia resolved. 10 days between treatment #10 and #11 due to transfer to medical facility from inpatient psychiatry.
6	16	Female	Psychosis	Major depressive disorder and post-traumatic stress disorder	Aripiprazole 25 mg	10 treatments CGI-I: 1	Bifrontal	Ultrabrief 455 mC	9 mECT 9 days	Resolution of psychosis *No longer receiving mECT*

Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT time of ECT	Psychiatric co-morbidity at the and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	Antipsychotics, Mood stabilizers	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No. and median days	Notes on clinical response
7	36	Male	Catatonia Psychosis	Schizophrenia	Clozapine 250 mg	15 treatments CGI-I: 1	Bitemporal	Brief 576 mC	1 mECT 30 days	Resolution of catatonia and psychosis *No longer receiving mECT*
8	30	Female	Catatonia	Unspecified psychosis	Lorazepam 3 mg	8 treatments CGI-I: 2	Bitemporal	Brief 576 mC	7 mECT 28 days	Resolution of catatonia *No longer receiving mECT*
9	26	Female	Psychosis Depression	Major depressive disorder with psychotic features	Asenapine 10 mg Lamotrigine 100 mg Lorazepam 3 mg	10 treatments CGI-I: 1	Bitemporal	Brief 576 mC	17 mECT 15 days	Resolution of psychosis and depression *Currently receiving ongoing mECT*
10	17	Female	Psychosis Depression	Unspecified psychosis	Clozapine 400 mg Lamotrigine 50 mg	9 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of psychosis and depression
11	64	Female	Depression	Bipolar I disorder	Lurasidone 50 mg Lamotrigine 200 mg	5 treatments CGI-I: 2	Right Unilateral	Ultrabrief 460 mC	23 mECT 21 days	Resolution of depression
12	32	Female	Catatonia	Bipolar I disorder	Divalproex Sodium 1,000 mg Lorazepam 1.5 mg	6 treatments CGI-I: 1	Right Unilateral	Brief 576 mC	NR	Resolution of catatonia
13	24	Female	Catatonia	Bipolar I disorder	Quetiapine 50 mg Divalproex Sodium 1,250 mg Lorazepam 1 mg	15 treatments CGI-I: 2	Bitemporal	Brief 576 mC	NR	Resolution of catatonia

NR: Did not receive mECT

Table 5.

Demographics, clinical data, electroconvulsive therapy parameters, and treatment response for patients with autism spectrum disorder without intellectual disability receiving acute series

Patient no.	Age at first ECT	Biologic sex	Symptom(s) targeted by ECT	Psychiatric co-morbidity at the time of ECT	Antipsychotics, Mood stabilizers and/or Antiepileptics, NMDA antagonists, and Benzodiazepines (total daily mg)	No. of acute treatments and retrospective CGI-I	ECT electrode placement	ECT pulse and charge	mECT No. and median days	Notes on clinical response
1	17	Male	Catatonia	Major depressive disorder, obsessive compulsive disorder, and attention deficit hyperactivity disorder	Memantine 10 mg Lorazepam 9 mg	12 treatments CGI-I: 4	Bitemporal	Brief 576 mC	2 mECT 15.5 days	Did not respond to ECT. Diagnosed with seronegative autoimmune encephalitis The patient responded to treatment lorazepam over the course of 1 year
2	24	Female	Psychosis Depression	Major depression disorder with psychotic features and generalized anxiety disorder	Aripiprazole 15 mg	13 treatments CGI-I: 2	Right Unilateral	Brief 576 mC	4 mECT 7 days	Resolution of depression and psychosis *No longer receiving mECT*
3	28	Male	Depression	Major depressive disorder	None	10 treatments CGI-I: 2	Bitemporal	Ultrabrief 192 mC	13 mECT 28 days	Resolution of depression *No longer receiving mECT*
4	23	Male	Psychosis Depression	Major depressive disorder generalized anxiety disorder, and attention deficit hyperactivity disorder	Olanzapine 15 mg Lithium 1,500 mg	17 treatments CGI-I: 2	Bitemporal	Brief 576 mC	2 mECT 6.5 days	Resolution of depression and psychosis *No longer receiving mECT*
5	19	Male	Psychosis Mania	Bipolar I disorder	Olanzapine 20 mg Divalproex Sodium 2,500 mg	7 treatments CGI-I: 2	Bitemporal	Brief 576 mC	1 mECT 14 days	Resolution of mania and psychosis *No longer receiving mECT*
6	16	Female	Catatonia Psychosis	Schizophrenia	Risperidone 4 mg Oxcarbazepine 1050 mg Lorazepam 1 mg	6 treatments CGI-I: 2	Bitemporal	Ultrabrief 236 mC	NR	Resolution of catatonia and improvement in psychosis
7	17	Female	Psychosis Depression	Major depressive disorder with psychotic features	Lurasidone 40 mg Amantadine 200 mg	13 treatments CGI-I: 1	Bitemporal	Brief 320 mC	2 mECT 6.5 days	Resolution of depression and psychosis *Did not return for ongoing mECT due to needle phobia*

NR: Did not receive mECT