Electroconvulsive Therapy as a Treatment for Tardive Dyskinesia

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ABSTRACT

Objective: To review the published literature over the last 10 years for the use of electroconvulsive therapy (ECT) in tardive dyskinesia (TD), focusing on the efficacy of this treatment.

Data Sources: A comprehensive evidence search of the published literature in the last 10 years (2010–2020) was conducted using the search terms *electroconvulsive therapy*, *electroshock therapy*, *ECT*, *tardive dyskinesia*, and *tardive dystonia*. The review was limited to articles published in the English language. MEDLINE, Embase, PubMed, PsycInfo, Cochrane Library, Google Scholar, and the NICE (National Institute for Health and Care Excellence) guidelines were also searched.

Study Selection: Twenty-three case studies published within the last 10 years were retrieved. The search revealed 5 articles of potential relevance.

Data Extraction: The articles were analyzed by both authors to obtain clinical information relevant to meeting the objectives of the review.

Data Synthesis: The efficacy in using ECT for TD is derived only from case series and case reports. There were no controlled trials, and the evidence collated was limited and of low quality.

Conclusions: The review indicates that ECT could be considered as a treatment for TD. However, this treatment may only be considered when patients present with a coexistent refractory mood or affective disorder. Further clinical trials are needed to improve understanding regarding the efficacy, tolerability, and safety of using ECT in this patient group.

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Tardive dyskinesia (TD) is characterized by involuntary stereotypic movements in the oro-buccal-lingual region. This movement can include lip smacking or pursing, chewing, facial grimacing, tongue protrusion, and tongue movements inside the mouth. Some physicians reserve the term *tardive dyskinesia* exclusively for this presentation.[1] However, the term is used to define any tardive hyperkinetic movement disorder, which may include stereotypy, akathisia, dystonia, tremor, tics, chorea, and myoclonus. TD is a form of tardive syndrome. The term *tardive syndrome* refers to a group of iatrogenic hyperkinetic and hypokinetic movement disorders.[1]

All tardive syndromes are caused by exposure to an antipsychotic or dopamine receptor–blocking agent. They are characterized by their delayed onset days to months after initial exposure and continuation after the responsible agent has been removed. They can be precipitated by a dose reduction or withdrawal of neuroleptic agents.[1,2]

TD is estimated to affect 20%–50% of all patients treated with neuroleptics. However, this prevalence varies among age groups, and the risk increases with advanced age.[2] ECT may be a successful treatment in some cases. However, a greater risk of developing TD has also been associated with ECT.[3] In this review article, we accumulate all the recent evidence for the efficacy of ECT in treating TD and make recommendations following data analysis.

METHODS

We conducted a comprehensive evidence search of the published literature in the last 10 years (2010–2020). The search terms included *electroconvulsive therapy*, *electroshock therapy*, *ECT*, *tardive dyskinesia*, and *tardive dystonia*. The review was limited to articles published in the English language.

MEDLINE, Embase, PubMed, PsycInfo, Cochrane Library, Google Scholar, and the NICE (National Institute for Health and Care Excellence) guidelines were also searched. Twenty-three case studies published within the last 10 years were retrieved. The search revealed 5 articles of potential relevance. Our findings are summarized below and in Table 1.

RESULTS

Case Series: Yasui-Furukori et al, 2014

The authors[4] provide retrospective data on 18 patients with TD and tardive dystonia who received a course of ECT. There was no screening of psychopathology because the patients were in a stable mental state at the time of treatment. The medication history of the patients is also unknown. The severity of these symptoms and outcomes were measured using the Abnormal Involuntary Movement Scale (AIMS). Patients who demonstrated greater than 50% improvement in the AIMS score, in comparison to baseline, were described as showing treatment response. A partial response was described as a 25% improvement in the AIMS score relative to baseline.[4]

All of the patients derived some benefit from ECT for their TD. Some patients showed an effective response within 2 sessions of ECT. Patients

Clinical Points

- In most studies investigating the efficacy of electroconvulsive therapy (ECT) in tardive dyskinesia (TD), the severity and frequency of TD improved with ECT treatment.
- There is little evidence to confidently recommend ECT as an isolated treatment for TD.
- Controlled trials are required to assess the efficacy and safety of ECT in patients with TD.

Table 1. Summary of Current Evidence on the Use of ECT in Tardive Dyskinesia

- A case series by Yasui-Furukori et al (2014)[4] showed that all 18 patients with tardive dyskinesia and tardive dystonia had some symptomatic benefit from a course of ECT.
- Peng et al (2013)[5] present the case of a 41-year-old woman with paranoid schizophrenia and an 8-year history of tardive dyskinesia. Both her psychotic and motor symptoms improved with ECT.
- Varela and Del Valle (2017)[6] present the case of an 85-year-old man with a diagnosis of severe depression with psychotic symptoms who developed tardive dyskinesia after a course of ECT.
- Schneider and Fahn (2010)[7] describe 2 cases of tardive akathisia that improved with ECT.
- Takagai et al (2012)[8] report the case of a 34-year-old man with a diagnosis of bipolar affective disorder and refractory tardive dystonia that did not respond to ECT.

Abbreviation: ECT = electroconvulsive therapy.

also demonstrated significant improvement in their global assessment of functioning (GAF) scores. Seven patients (39%) responded to ECT using their criteria. Eleven patients were identified as partial responders. Of the 18 subjects, there were 10 women and 8 men with ages ranging between 19 and 65 years. Thirteen patients had a diagnosis of schizophrenia, and the other 5 had unipolar depression.

Some patients were not prescribed any medications. Others received antidepressants or antipsychotics during the ECT course. The prescribed antipsychotics included aripiprazole (1.5–12 mg daily), risperidone (1–3 mg daily), and olanzapine (2.5–10 mg daily). The doses and medications remained consistent during the course of ECT.[4] All patients were treated with bilateral ECT, which was administered 3 times weekly. Propofol and succinylcholine were used as induction agents. The length of ECT courses ranged from 6 to 15 treatments, and the average course length was 10.5 ± 2.4 treatments. All patients tolerated ECT with no significant adverse effects noted.

The authors[4] refer to a prospective study from 1990 wherein one patient's TD improved with ECT but 8 of the other participants experienced no change. The authors[4] propose mechanisms for the action of ECT, including the prevention of super sensitization of postsynaptic dopamine receptors that contribute to the development of tardive states. ECT may enhance dopaminergic transmission and also target the γ-aminobutyric acid (GABA)ergic system. ECT may also affect the blood-brain barrier, which allows antipsychotic and antidepressant medication to enter the brain and alleviate motor symptoms at a constant oral dosage.

In this study,[4] there was no measurement of cognition after the course of ECT. The study[4] was of low quality, and the results need to be interpreted with some caution. However, overall, the data were promising in demonstrating the use of ECT as a potential treatment for TD.

Case Study: Peng et al, 2013

The authors[5] present the case of a 41-year-old woman with paranoid schizophrenia (diagnosed when she was 22 years of age) and an 8-year history of TD. Both her psychotic and motor symptoms improved with ECT. At the onset of illness, she presented with auditory hallucinations and delusional beliefs of being possessed.

During the earlier stages of her psychotic illness, drug compliance was poor, and this impacted her personal functioning. She was sensitive to adverse effects from antipsychotic medications and developed a tremor and muscle rigidity after taking sulpiride (600 mg daily) for 2 weeks. Her compliance improved, and in 2002 she developed an orofacial dyskinesia after taking risperidone (3 mg daily) for 1 year. This medication was later changed to quetiapine (200 mg daily) and then olanzapine (15 mg daily). However, both medications caused oversedation and weight gain. The patient preferred to restart risperidone (3 mg daily), and she remained on this treatment for the next 8 years. There were fluctuating psychotic symptoms and signs of persistent dyskinesia.[5]

The patient was admitted to the hospital in May 2010 due to worsening auditory hallucinations, delusions of control, and risk of violence. TD was clearly visible with pouting and puckering of her lips, mouth chewing, twisting and spreading of upper limbs, and foot tapping. She was treated with ECT to target the severe psychotic symptoms and also due to medication noncompliance. She had bilateral ECT every other day (total 11 sessions) with seizure duration ranging from 20 to 35 seconds. Thiamylal sodium (150 mg intravenously) was used as the anesthetic agent. She was prescribed risperidone (3 mg daily through the ECT treatment course).[5]

There was significant improvement in both the psychotic symptoms and the TD. The improvement in TD was confirmed with a reduction in her AIMS score from 13 before ECT to 5 after ECT. There was now "only minimal" pouting and puckering of her lips and athetoid movements of the upper limbs. There was a reduction in her Positive and Negative Syndrome Scale score from 77 to 42, with residual auditory hallucinations and prominent negative symptoms. She was discharged after 1 month of hospitalization. The improvement in TD and psychotic symptoms was maintained during her 2-year follow-up period. She remained on the risperidone prescription (3 mg daily) during this timeframe.[5]

Case Study: Varela and Del Valle, 2017

This case study[6] describes the emergence of TD following ECT in an 85-year-old man. The patient had no relevant medical history. He had a treatment-refractory

severe depressive disorder with psychotic symptoms and was admitted to the hospital. He had been treated with psychotherapy and various classes of antidepressants in the past including selective serotonin reuptake inhibitors, selective serotonin-norepinephrine reuptake inhibitors, a norepinephrine-dopamine reuptake inhibitor, monoamine oxidase inhibitors, and tricyclic and tetracyclic antidepressants. He also had trials of mood stabilizers and antipsychotics, which included low-dose olanzapine (2.5 mg daily) and risperidone (2 mg daily).[6]

The patient had a course of 9 ECT treatments in the past and achieved partial remission with this and combination pharmacologic therapies. He later developed parkinsonism including bradykinesia and rigidity with the combination of antipsychotic and antidepressant treatment. Following a neurology review, risperidone, which was prescribed for the last year and a half, was discontinued. This led to some improvement in motor symptoms, although there was subsequent deterioration of his affective symptoms. His pharmacologic therapy was adjusted, and he was prescribed desvenlafaxine (150 mg daily) and mirtazapine (15 mg daily). There was no improvement, and he had 7 ECT treatments with supportive psychotherapy. There was no remission of his affective symptoms with this intervention.[6]

A week after the end of his ECT course, the patient developed TD. He had already been without antipsychotic medication for 6 months during this time. The TD manifested with involuntary mandibular movements. The TD caused a number of oral injuries from biting. There was associated pain, and he experienced difficulty with oral intake. The symptoms improved during the night. He had another neurology review, and benzodiazepines were started. The benzodiazepines were ineffective, and the clinicians attempted to reduce the dose of desvenlafaxine. However, there was deterioration in his depressive symptoms, and he was maintained on a dosage of desvenlafaxine (75 mg daily) and mirtazapine (15 mg daily).

The TD remained and also spread to his trunk. The symptoms were progressively becoming worse and causing more impairment. There was associated weight loss, and the subsequent disability affected his emotional well-being. The authors[6] were unable to identify any further cases of TD precipitated by ECT in their literature review.

Case Series: Schneider and Fahn, 2010

Schneider and Fahn[7] present an abstract that describes 2 cases of tardive akathisia successfully treated with ECT. Both patients failed treatments with reserpine and tetrabenazine but were later successfully treated with ECT. The first patient was a 49-year-old woman with TD and tardive akathisia. The onset of these symptoms followed treatment with haloperidol. There was remission of the akathisia with ECT, and the improvement was maintained over the next 5 years.[7]

The second patient was a 54-year-old woman who developed tardive akathisia after treatment with thioridazine. She was treated with ECT and remained symptom free at 3-week follow-up. Both patients had no longer-term adverse effects from ECT. Schneider and Fahn[7] noted that only 1 previously published case study reports on the efficacy of ECT in tardive akathisia.

Case Study: Takagai et al, 2012

The authors[8] discuss the case of a 34-year-old Japanese man with a diagnosis of bipolar disorder. He had multiple manic relapses during the course of this illness. He had a refractory tardive dystonia, which did not respond to ECT. Prior to the onset of dystonia, he had been treated with various antipsychotic medications including haloperidol and risperidone and the mood stabilizer lithium. He developed tardive syndrome after exposure to haloperidol (9 mg daily) and lithium. There were involuntary muscular contractions in both his upper and lower limbs. Haloperidol was subsequently switched to risperidone (6 mg daily).[8]

The patient later developed left tilting of his trunk, right-sided torticollis, opisthotonic trunk extension, and spasms of both upper and lower limbs. These abnormal movements disappeared during sleep. The patient did not smoke or use excessive alcohol or illicit substances. There was no family history of neurologic disorder. He had a neurology review and extensive investigations. Risperidone was discontinued, and he had pharmacologic trials of various agents to treat the movement disorder. He also had 12 sessions of ECT in July 2010 with no change in the dystonia. The patient later showed a positive response when he was treated with aripiprazole following another manic relapse.[8]

CONCLUSION

There is a paucity of recent literature on the efficacy of ECT as a treatment for TD. We found 1 case series of 18 patients and 5 other case studies. Most of the patients with TD demonstrated a positive response to ECT, with the retrospective study by Yasui-Furukori et al[4] providing the most promising data. The improvement was sustained without the need for maintenance ECT. However, there is 1 case study[8] that reports no treatment response with ECT and another[6] that suggests the onset of TD following ECT treatment.

In most of the studies, ECT was well tolerated and there were no significant adverse effects. There is very little information provided regarding cognition before and after ECT treatment. We advise that ECT may be considered in cases when there is a treatment-resistant psychotic or affective illness and coexistent TD. There is little robust evidence to advise on using ECT as an isolated treatment for TD. It is well known that TD can have negative outcomes and cause significant distress.

Overall, the data in the last 10 years are of low quality and comprise only case reports and series. The current evidence has not contributed to providing a greater depth of knowledge in the use of ECT in TD. Further trials that provide high-quality data are required to develop adequate therapies for this potentially disabling iatrogenic disorder.

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