

Electroconvulsive Therapy: Solution or a Cause of Tardive Dyskinesia?

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Abstract

Tardive dyskinesia is a motor disorder characterized by the appearance of involuntary and irregular movements in different body regions. It is usually caused by prolonged use of dopamine receptor blocking agents, especially by taking antipsychotics. Currently, there is no curative treatment for this disorder, although there are pharmacological strategies that reduce its impact. Electroconvulsive therapy has been successfully applied in several cases of tardive dyskinesia and has been proposed as a therapeutic alternative, although the available evidence is still insufficient. In the present article, we present a case of tardive dyskinesia which emerged following the application of electroconvulsive therapy. It is considered if there could be a relation between the application of this technique and the appearance of the motor disorder in specially predisposed individuals.

Keywords: Electroconvulsive therapy; Tardive dyskinesia; Major depression

Introduction

Tardive dyskinesia (TD) is a motor disorder resulting in involuntary, repetitive movements in areas such as mouth, tongue, face, trunk or limbs [1]. It is usually caused by prolonged use of dopamine receptor blocking agents, such as some antidepressants, antiepileptics or antiemetics, and is especially common due to the prolonged taking of antipsychotics [1-3]. Currently, there is no treatment that eliminates them, although there are diverse pharmacological strategies that reduce their impact, such as tetrabenazine or some antioxidant agents [2]. Electroconvulsive therapy (ECT) has been used several times to reduce TD with favourable results [1], although the level of evidence is still insufficient to propose it as a curative treatment [2]. Further on, we present a case of TD, which emerged after the application of ECT, and we propose whether there may be a relation between the appearance of the motor disorder and the application of this therapy. A comprehensive research has been conducted through evidence-based clinical resource such as Up-to-date, BMJ Clinical Evidence, Clinical Key, Medline and PubMed.

Case Report

An 85-year-old man, who had no relevant medical-surgical history, with a major depression refractory to various pharmacological treatments was admitted to the hospital. Since the onset of depression, the patient had received treatment with different groups of antidepressants, such as Selective Serotonin Reuptake Inhibitors (SSRIs), Selective Serotonin and Noradrenaline Reuptake Inhibitors (ISRN), Selective Dopamine Reuptake Inhibitors and Norepinephrine (IRDN), Mono-Amino Oxidase Inhibitors (MAOIs), Tricyclic Antidepressants and Tetracyclic Antidepressants. In addition, he had been on treatment with mood stabilizers and with 2.5 mg of olanzapine during 1 month, without ever achieving a remission of the depressive symptomatology. The first time he came to our clinic, about 2 years ago, he was being treated with desvenlafaxine at a dose of 100 mg per day. Due to the lack of improvement of the depressive clinic and to the appearance of delusional symptoms, he was treated with 9 sessions of ECT. Besides, the antidepressant was replaced by bupropion at doses of 150 mg daily and neuroleptic medication was introduced (risperidone at doses of 2 mg/day), achieving a partial remission of the symptomatology. In the next months, the depressive episode got worse, appearing autolytic ideation, so the bupropion dose was increased to 300 mg daily, with

the neuroleptic treatment being maintained at the previous doses. In addition, he received supportive therapy with cognitive-behavioural intervention with cognitive restructuring, coping strategies and stress reduction, but it was unsuccessful. A few weeks later it was decided to maximize the antidepressant regime and add mirtazapine 15 mg/day. Parkinsonism symptoms (bradykinesia and rigidity) were observed 3 months later, which was assessed by Neurology. A pharmacological origin of the motor disorder was suspected, so it was decided to suspend the treatment with 2 mg of risperidone, which the patient had taken for a year and a half. A partial improvement of the motor clinic was observed. The following weeks the affective symptomatology worsened. Further pharmacological readjustments (replacement of bupropion by desvenlafaxine 100 mg/day and subsequent dose increase to 150 mg daily, keeping mirtazapine at the previous doses) were equally ineffective. In the absence of positive results, it was decided to process another in-patient admission to reapply ECT. Seven sessions were administered, without observing a clear remission of the affective disorder. He also received supportive therapy with cognitive-behavioural intervention, without satisfactory results.

One week after the end of the ECT, involuntary movements at the or mandibular level, increasingly marked, were observed. These movements caused several mouth injuries from bites, pain and difficult food intake, and improved at night coinciding with sleep. The case was again evaluated by Neurology, which ruled out a parkinsonian disorder of neurological etiology and diagnosed tardive dyskinesia with a probable pharmacological cause. Benzodiazepine treatment was introduced in ascending pattern, with no response. Currently, the patient is being treated with 75 mg desvenlafaxine and 15 mg mirtazapine daily. An attempt was made to reduce the dose of desvenlafaxine in case it could be favouring the maintenance of the movements, but a clear deterioration of the depressive symptomatology was observed, with the appearance of irritability and decreased mood, so it was decided

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Received April 12, 2017; Accepted April 19, 2017; Published April 21, 2017

Citation: Varela Piñón M, Del Valle IO (2017) Electroconvulsive Therapy: Solution or a Cause of Tardive Dyskinesia? J Depress Anxiety S11:003. doi:10.4172/2167-1044.S11-003

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to maintain the dose at 75 mg/day. The dyskinesic movements are still present currently, they have increased their intensity and have spread to the trunk. They are becoming more and more disabling as they have caused a progressive loss of weight and, in addition, a frank emotional deterioration (pessimism, death thoughts) due to the limitations that they cause to him.

Discussion

The ECT uses a small electric current to produce a generalized cerebral seizure under general anesthesia [4]. It is mainly used in severe depression [5-8], although its use is also indicated in some cases of bipolar affective disorder, schizophrenia, schizoaffective disorder, catatonia, neuroleptic malignant syndrome or tardive dyskinesia [9,10].

Before starting ECT, it is recommendable to perform a complete medical evaluation and to explore possible antecedents that may contraindicate this therapy, such as cardiopulmonary diseases, central nervous system or major surgical interventions [11-14]. In our case, the patient had no relevant medical or surgical history that contraindicated its application, so the procedure was carried out in the usual way.

This technique may cause side effects of different severity [15]. Among the most common, and the mildest side effects, are headache [16-18], nausea or myalgias [19], as well as effects at a cognitive level that are often self-limited and require symptomatic management [20-22]. Other more severe but uncommon side effects include cardiac, pulmonary and cerebrovascular disorders. TD is not described as a possible direct side effect of the ECT application.

Many psychotropic medications, such as antidepressants [23], antipsychotics [24,25] or lithium [26], may be maintained during the course of ECT because of their synergistic effect, without compromising the safety or efficacy of the process [27,28]. Anticonvulsants [29] and benzodiazepines [30] interfere with ECT and it is recommended to be discontinued. In this case, antidepressant therapy was maintained during therapy (desvenlafaxine and mirtazapine, at a dose of 150 mg and 15 mg daily, respectively).

The ECT usually lasts from 6 to 12 sessions, and it's applied with a frequency of 2 or 3 times weekly to alternate days [31]. Our patient received 7 sessions, on a biweekly basis, without observing any significant change. Four days after the last session began to present dyskinesic movements at oromandibular level.

TD tends to appear in prolonged treatments with dopaminergic receptor blocking agents, being common due to the sustained intake of antipsychotics [32]. After withdrawal of neuroleptics, they usually remit in the course of months or years, although they sometimes persist in a chronic form [32]. However, it is important to note that not all patients receiving antipsychotics will develop TD. The reasons why only some of them present TD are still unknown. A number of factors have been proposed that could favour its emergence. Age is the risk factor that is most strongly associated with TD. It is suspected that at an older age there is an increased risk of developing this disorder and with a greater severity [33-35]. The existence of affective disorders, especially a major depressive disorder, neurological disease, or serious alcohol dependence, could be other factors predisposing to its development [36]. Cerebral structural abnormalities, cognitive deterioration, smoking or diabetes have been studied as possible factors favouring TD, without being able to be confirmed [37].

Neither there is a clear predisposition based on sex [38,39] although it could be somewhat higher in postmenopausal women [39].

Our patient was elderly, had major depressive disorder and prolonged exposure to antipsychotics (treatment with risperidone at a dose of 2 mg daily for approximately a year and a half) as significant risk factors, although neuroleptic medication had been withdrawn 6 months earlier of the appearance of TD. The possible role of desvenlafaxine in the maintenance or worsening of movements was studied, although TD was not described as a possible side effect of the use of this antidepressant.

For all of the above, we raised the possibility that the TD was triggered by the application of ECT, or alternatively, that it was a consequence of the neuroleptic treatment maintained where the ECT acted as a precipitant of the motor disorder, although the patient had already been without antipsychotics for 6 months and, until the application of ECT, did not present dyskinesic movements. Another hypothesis could be that ECT may have been a trigger precipitant of the symptomatology of an underlying primary disorder.

The current literature on the relationship between ECT and TD are scarce [1,40,41], which prevents us from knowing the actual influence of ECT on the appearance of these dysfunctional movements. Further knowledge about the mechanism of action of ECT is needed to ascertain its possible influence on the development of this neurological disorder.

After an exhaustive literature review, we have not found any other case that describes a possible relationship between the application of ECT and the occurrence of TD, although in the case discussed above it seems that this technique may have influenced somehow the development of the motor disorder.

Conclusion

Although ECT has been successfully employed in the management of TD, the case studied suggests that the application of this technique, in specially predisposed individuals, could influence somehow the appearance of dyskinesic disorders. Further studies are needed to help us understand the actual role of ECT in the development of TD and the mechanism by which this therapy could trigger its onset.

Conflicts of Interest and Source of Funding

The authors report no financial or ethical conflicts of interest regarding the content of the manuscript.

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Citation: Varela Piñón M, Del Valle IO (2017) Electroconvulsive Therapy: Solution or a Cause of Tardive Dyskinesia? J Depress Anxiety S11:003. doi:10.4172/2167-1044.S11-003

This article was originally published in a special issue, **Depression in Cancer Patients** handled by Editor(s). Dr. Domenico Chirchiglia, Chair of Neurosurgery, University of Catanzaro, Italy

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