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Case Presentation

Jaw Subluxation as a Complication of Tardive Dyskinesia

Diana Paleacu Kertesz*

Abarbanel Mental Health Center, Tel Aviv University, Israel

Abstract

Tardive Dyskinesia (TD) is an iatrogenic complication caused by antipsychotic agents and rarely by other anti-depressive/antiepileptic or anti-nausea medication. It is mostly a benign condition with implications regarding esthetic issues but it can also impact social and emotional well-being. We are reporting a case in which severe TD ensued in an elderly lady with newly diagnosed dementia, who presented to the psychiatric ER with a Capgras syndrome and paranoia accompanied by behavioral disturbances. She was treated with 4 consecutive antipsychotic agents (haloperidol, brexiprazole, risperidone, and olanzapine) due to unresponsive psychosis in conjunction with biperiden and developed a severe case of TD, which was complicated by two successive episodes of jaw subluxation. In spite of the good outcome following the reduction of the subluxation, we emphasize the danger of this rare painful, and bothersome side-effect and recommend how to pharmacologically deal with the TD setting in which it occurred.

More Information

***Address for correspondence:** Diana Paleacu Kertesz, Abarbanel Mental Health Center, Tel Aviv University, Israel, Email: paleacu@me.com

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Introduction

Tardive Dyskinesia (TD) is an iatrogenic involuntary movement disorder following exposure to a Dopaminergic-related Blocking Agent (DRBA) causing D2 or possibly D3 receptor blockade, in most cases, but not all, an antipsychotic agent. The movement disorder characteristic of TD encompasses persistent, typically stereotyped movements in the face area and limbs. Over time, various other abnormal involuntary movements (chorea, tics, akathisia, myoclonus) have been described in people with TD and they were grouped under an umbrella term called "tardive syndrome." [1]. TD might appear within 3 months and in elderly patients within 1 month from treatment onset and the disorder persists for at least one month after stopping the offending drug [2]. It is one of the most bothersome side effects of antipsychotic agents but it has also been reported in patients treated with selective serotonin reuptake inhibitors (SSRI's), lithium, antiepileptic and anti-nausea drugs.TD appears mainly in schizophrenia patients, and sometimes in patients suffering from depression, bipolar disorder, anxiety, and insomnia for which these drugs are often used off-label [3]. The pathophysiology of TD is not very clear; it has been hypothesized to result from increased dopamine sensitivity causing receptor up-regulation due to chronic blockade of these receptors by DRBAs [4]. Interestingly, the underlying psychiatric disorder does not influence the risk of developing TD [5], long-term use of DRBAs and higher dosages, particularly in elderly women, confer the highest risk [6].

TD turns, in most cases, into a chronic condition leading to added motor disability on top of the psychiatric disease. In spite of the fact that it doesn't cause a life-endangering condition in the vast majority of cases, TD impacts social and emotional well-being, sometimes causing depression and social isolation. Severe cases can lead to complications leading to breathing difficulties when affecting the diaphragm or due to laryngospasm [7]. The best treatment for TD is prevention. Nevertheless, in most cases, the offending drug cannot be stopped, therefore the causative treatment should be replaced by clozapine and if the TD persists the VMAT2 inhibitors are recommended; either Xenazine [8] or one of the two new FDA-approved drugs: deutetrabenazine [9] or valbenazine [10].

We, hereby present a rare case of severe TD complicated by jaw subluxation. The case aims to enhance the fact that TD might be a seriously dangerous antipsychotic side effect with serious consequences, not only an esthetic nuisance, as usually perceived by the psychiatric community.

Case presentation

An 80-year-old lady of Polish Ashkenazy origin was brought to the psychiatric ER by the district psychiatry nurses. It was her first admission to a psychiatric facility and the hospitalization was done with the consent of the patient. The patient was previously known to be able to take care of her own affairs and had no guardian.

Her current illness

She was brought by the hospital brothers from district

lines, by virtue of an inspection order according to section 7 of the law, which was requested by an attorney, due to fear of a psychotic state with impaired judgment and control of reality throwing heavy objects from the window of the house. She was examined in the emergency department by an emergency psychiatrist. Relatively tidy, and quiet, wears glasses. According to her, her fake nephew told her that her building would be destroyed, and as she couldn't believe this would happen she had a big fight with him.

Her nephew has a power of attorney. She agreed to sign for voluntary hospitalization.

In the Psychogeriatric Department, she was quiet, calm, and fully conscientious, showing psychomotor slowing. Orientation was fully preserved for time and place. Her delusions are without change. Denies suicidal thoughts or intentions to harm herself. There was no evidence of perception disorders. She was diagnosed with Capgras syndrome [11] and paranoid delusions for the first time in her life. A diagnosis of dementia was also suspected. Later on a Minimental state exam (MMS), she scored 20/30 showing a mild to moderate cognitive decline.

Treatment with haloperidol 2.5 mg was started, also biperiden 2 mgX1 for a few days, added due to the apparition of head tremor, very bothersome for the patient. Lorazepam would be given SOS as needed when she would manifest panic attacks. Two weeks later HPL was stopped due to an apparition of parkinsonism and Tardive Dyskinesia (TD) and brexpiprazol 1 mg was started.

Under this medication, she kept claiming that her nephew, her sister's son, was an impostor and there were many fake persons in her family surrounding her. She claims that the nephew told her that her building would be destroyed. But she can't believe this will happen, she is ready to return to the old house until she gets a "new house", she is convinced that she should get a new house. No suicidal thoughts.

Brexpiprazole was increased to a dose of 2 mg, but it didn't help with the delusional content, therefore it was stopped and risperidone was given for two days starting at 0.5 mg and increased to 2 mg per day, causing worsening of the motor symptoms without change in the delusional content. At this stage olanzapine was instituted at a dose of 5 mg IM and then increased to 10 mg IM for 3 days, followed by an oral dose of 10 mg per day.

At this stage (30 days from the start of hospitalization) the patient was suffering from very severe parkinsonism (motor UPDRS 48), with TD (very intense head and hand tremors) and very dysarthric, her slurred speech is impossible to understand, she cannot stand up on her own and had to be helped during ambulation. Her AIMS score was 32.

The next morning the patient presented with an asymmetric face and she was pointing to her right jaw indicating pain. A right central facial palsy was suspected, but no limb weakness was present and as local TMJ pain was severe a diagnosis of dislocated right TMJ was made. The patient was sent to the ENT service where she underwent a successful reduction for jaw subluxation.

The patient returned to our department. All neuroleptic treatment was stopped and amantadine 100 mg BID and tetrabenazine 12.5 mg X2 were given.

Three days later there was a significant improvement in the TD with an AIMS of 12, but still dysarthric and secondary parkinsonism quite significant. On the following day, the episode of subluxation reoccurred and it was fixed again by the ENT service. Further on, amantadine was increased to 300 mg/day, tetrabenazine to 50 mg/day and the psychiatric team was considering clozapine for her paranoid ideation. Also, we were negotiating with her health insurance provider a change of tetrabenazine to deutetrabenazine.

Discussion

The case of jaw dislocation due to severe TD is extremely rare but has been previously reported [12] and it has been treated in a number of ways including medical treatment and even DBS; this group suggested that pallidotomy is a treatment option for tardive jaw-opening dystonia and that dystonia of this type is driven by abnormal neural activities in the GPi [13,14]. In our case jaw subluxation was not associated with dystonia but very severe dyskinesia in the oral region, as a result of multiple neuroleptic drug use during a relatively short time in conjunction with an anticholinergic.

Conclusion

TD can rarely cause dangerous side effects; such as jaw dislocation. We want to emphasize that the use of anticholinergics together with antipsychotics as a preventive therapy for extrapyramidal symptoms (EPS) is an old bad habit in psychiatric practice and should be avoided. Also, we caution against the use of first-generation antipsychotics or an increased number of antipsychotics in a patient with a degenerative brain disorder, as it might be a predisposing factor for TD. In our case, it might have contributed to this very painful and dangerous side effect.

Ethical considerations

Before writing this case report we asked the patient for her permission and she consented. Nevertheless, she declined to be photographed or filmed for publication purposes.

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