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Case Report
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Trifluperazine Induced Blepharospasm – A Missed Diagnosis

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Abstract

Tardive dystonia is a class of "tardive" movement disorder caused by antipsychotics and is specified by reflex muscle contraction, which may be tonic, spasmodic, patterned, or repetitive. This neurological disorder most commonly occurs as the repercussion of long-term or high-dose use of antipsychotic drugs. Tardive dyskinesia uncommonly inculpates the muscles of eye closure. Blepharospasm is a kind of focal tardive dystonia distinguished by persistent intermittent or persistent closure of the eyelids. Blepharospasm is an uncommon, persistently disabling medical condition rendering patient functionally blind and occupationally handicapped .We hereby tend to report a case of trifluoperazine induced tardive blepharospasm.

Case Presentation

A 46-year male patient reported to eye opd with complaint of progressive difficulty in opening his eyes for last two years .On examination he was unable to open his eyes voluntarily, sometimes thrusting his head backwards or rubbing his brow with his fingers during these episodes. Past and family history revealed no psychiatric or neurological illness. No past history of any other psychiatric/medical/surgical illness could be elicited.

On specifically asking about medication history, he gave history of trifluoperazine intake. Patient was diagnosed with schizophreniform disorder 3 years back and was on treatment since then. Initially, he was started with 5mg trifluoperazine in twice daily dosage which had to be increased to (15mg/day) within a month. He was kept on this maintenance dosage for 2 years. While being treated with trifluoperazine after 1 year patient developed frequent and forceful blinking of his eyelids. He would blink his eyes about 35-40 times in a minute when exacerbated by bright light.

During this course he consulted many doctors for blepharospasm but no cause could be established.

Vitals were found stable on examination. No other neurological deficit could be diagnosed other than the abnormal movements. Blood biochemistry, complete blood picture, ECG, EEG, and brain imaging were essentially normal. The patient had no prior personal or family history of blepharospasm or any other movement disorders.

Visual Acuity (with correction) was 20/25 Right eye (OD) and 20/20 Left eye (OS) and Extraocular Motility was Full in both eyes (OU). B/L/ Pupils were found reacting well to light OS, no RAPD OU. Intraocular Pressure was 15 mmHg OU. Confrontation Visual Fields were Full OU.

On External Examination presence of bilateral brow ptosis was noticed. Frequent spasms of the orbicularis oculi muscles, procerus, corrugators were present bilaterally causing forcible eyelid closure. Slit Lamp Examination revealed no abnormality .Dilated Funduscopic Examination revealed Normal disc, macula, vessels, and periphery OU.

Discussion

Blepharospasm is classified as a type of focal dystonia and has been reported to occur with atypical antipsychotics. Tardive syndrome (TS) is a group of hyperkinetic or hypokinetic movement disorders and sensory symptoms sharing the same pathophysiological basis. The etiological theories and treatment strategies have been elucidated recently [1,2]. This report presents a case of trifluoperazine-induced tardive dystonia.

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Trifluoperazine is a phenothiazine antipsychotic with high affinity for D2 receptors relative to D1 receptors [3]. It has lesser propensity to induce seizures than other antipsychotics and therefore it is considered a "good choice" in treatment of comorbid schizophrenia and epilepsy [4]. Trifluoperazine results in a variety of adverse reactions including sedation and weight gain, but to a lesser degree other antipsychotics [5,6]. Other adverse effects include postural hypotension, constipation, parkinsonism, priapism, and sexual dysfunction [7,8]. Trifluoperazine has been known for its high propensity to cause extrapyramidal side effects, such as tardive dyskinesia and akathisia [9,10].

By definition, tardive dyskinesia is a result of drug therapy. Tardive dyskinesia is a movement disorder resulting from taking certain drugs, distinctively a class of drugs called neuroleptics. These drugs effect the chemicals in the brain. One of the chemicals affected is dopamine, which has a pivotal character in control of movement. The extended use of neuroleptics leads to some aadaption in the motor system that steers production of involuntary movement. Since these movements are produced as late effect of taking these drugs, the dyskinesia is called tardive, which means late. Once these movements occur, they can be quite long-lasting and possibly permanent. Tardive dyskinesias can affect any muscle in the body, but they very commonly affect cranial nerve muscles. The muscles affected commonly are tongue, the jaw closing muscles, and the muscles around the mouth, but eyelid closing muscles can also be affected. When eyelid closing muscles are affected, there can be blinking or sustained closure of the eyelids, which could mimick blepharospasm. Movements of the tongue and lips are particularly emine Tardive Blepharospasm is worse in stress, fatigue, bright lights, watching television or driving, and social interactions while Sleep, relaxation, walking, talking and other "tricks" alleviate symptoms temporarily.

The early symptoms of blepharospasm include increased blink rate (77%), eyelid spasms (66%), eye irritation (55%), midfacial or lower facial spasm (59%), brow spasm (24%), and eyelid tic (22%). nt in tardive dyskinesia. These movements can also occur in rhythmic repetitive trains.

Because both blepharospasm and tardive dyskinesia can cause blinking or sustained closure of the eyelids, their appearance can be similar. However, tardive dyskinesia would only infrequently involve the muscles of eye closure. Therefore, unless the focal dystonia in the patient with blepharospasm has spread to involve the rest of the face, it ordinarily would not be difficult on clinical grounds to separate patients with blepharospasm and tardive dyskinesia.

Lastly, both blepharospasm and tardive dyskinesia are difficult to treat. There is no systemic medication that works well for either condition [11].

Conclusion

It is very important to consider possibility of tardive blepharospasm in a patient on trifluoperazine therapy. Patient must be referred to an ophthalmologist to look for possibility of tardive blepharospasm in case suspicion arises. Attention must be paid to specific cause and appropriate management because blepharospasm interferes with performance and enjoyment of day to day activities.

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