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Transient electromyographic findings in serotonergic toxicity due to combination of essitalopram and isoniazid

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ABSTRACT

Here, we report a case of serotonergic toxicity due to combination of essitalopram and isoniazid, which was rarely reported before. Moreover, we observed transient neurogenic denervation potentials in needle electromyography, which disappeared with the treatment of serotonergic toxicity. As to our best knowledge, this is the first case, reporting transient electromyographic changes probably due to serotonergic toxicity.

Key words: Denervation potentials, electromyography, essitalopram, isoniazid, serotonergic toxicity

Introduction

Serotonin toxicity (ST) is a rare clinical state, which may be serious and life-threatening. The most common reasons are usage of multiple serotonergic drugs together or combination of one serotonergic agent and one monoamine oxidase (MAO) inhibitor. Isoniazid (INH) is a weak MAO inhibitor^[1] and also may affect cytochrome enzyme system.^[2] So, it may interact with serotonergic agents like essitalopram.^[3] ST due to combination of isoniazid and essitalopram was rarely reported before.^[3] To suspect from the state and history of serotonergic drug usage are main factors for diagnosis.

Although clinical signs of ST on nervous system are well-known, no specific electrophysiological finding was demonstrated before. Here, we report a case of serotonin toxicity due to the combination of essitalopram and isoniazid. We also observed transient electromyographic findings, which may probably be due to serotonergic activity.

Case Report

A Forty-three-years-old female developed acute onset fever and confusion. She was receiving corticosteroids with the diagnosis of Takayasu arteritis for the recent three years. She was also taking isoniazid cause of a suspicious positive result in tuberculin skin test (PPD) for the last three months.

Neurological examination revealed fever, rigidity, clonus, bilateral positive Babinsky and Hoffman signs, and hyperactive deep tendon reflexes. Cranial and spinal magnetic resonance imaging (MRI) showed no abnormalities nor contrast enhancement. Nerve conduction studies were normal, but needle electromyography (EMG) demonstrated spontaneous neurogenic changes (fibrillation and positive waves) in her extensor digitorum brevis (EDB), tibialis anterior (TA), and gastrocnemius muscles. There were no chronic neurogenic signs.

Further investigations for toxic, nutritional, and infectious spinal cord diseases revealed nothing.

We learned that she had been taking essitalopram 20 mg/day for last month from her relatives.

With the help of this medical history, she was diagnosed as serotonergic toxicity, essitalopram was stopped, and the patient was hydrated. In her follow-up, she started to get better on the third day of drug-free period. After

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one week, she was completely improved. No rigidity, clonus, or pyramidal sign was detected. Repeated EMG was completely normal.

Discussion

Nowadays, the term serotonin toxicity (ST) is often referred to serotonin syndrome (SS).^[4] There is no specific laboratory or diagnostic test, so the diagnosis is mostly based on the clinical aspects.^[5] A history of serotonergic agent or increment in the dosage, four major or three major + two minor symptoms, exclusion of psychiatric, toxic, metabolic and endocrine diseases, and neuroleptic usage were needed for diagnosis.^[6] Our case fulfilled all criteria and revealed six major symptoms like myoclonus, tremor, rigidity, hyperreflexia, fever, and confusion. Although citalopram is widely used because of its favorable safety profile and lack of cytochrome P450 inhibition,^[7] it has been reported to cause serotonin toxicity, usually in the setting of an overdose.^[8,9] Citalopram is a substrate for CYP2C19 and 3A4, and isoniazide may inhibit cytochrome P450 (CYP450) isoforms, potent inhibition of CYP2C19 and CYP3A.^[2] INH is also a MAO inhibitor,^[1] so may affect the metabolism of anti-depressant drugs. INH plus escitalopram was rarely reported to be the cause of serotonergic toxicity.^[3] There are no prospective treatment studies for serotonin toxicity. General management measures include removal of the precipitating serotonergic agent and supportive strategies to control the state.^[10] In our case, general supportive treatment to stop escitalopram improved the clinical symptoms and signs of the patient. SS is a generalized toxic syndrome; therefore, both cognitive and neuromuscular findings may be observed. But, in most of the cases, clinical symptoms are more prominent in lower and distal part of extremities, which also may be a clue for a spinal process. As to our best knowledge, there is no specific electrophysiological finding of SS in the literature. The spontaneous denervation potentials that we observed during the etiological investigations of the patient may be due to a coincidental disease.

Nerve conduction studies were normal while there were acute denervation potentials, which lead to the idea of a pathology located more proximal to spinal ganglia. But, there were no abnormalities or contrast enhancement in her spinal MRI. Moreover, disappearance of spontaneous denervation potentials after treatment of serotonergic syndrome would be an evidence for toxic effects of serotonin on spinal cord cells. As a result, usage of INH inhibited the metabolism of serotonin and induced serotonergic toxicity in our case. Interaction of INH and escitalopram was rarely reported before; however, our case is the first to report probable electrophysiological findings due to serotonergic toxicity.

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