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# Serotonin syndrome after SSRI and Mao-B inhibitor use: A case report

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## Abstract

An 83-year-old patient was hospitalized for an severe rigidity, excessive sweating and hallucinations. She was using both SSRI and MAOB inhibitor drugs. She had known parkinson's disease. We missed diagnosis at the first time. We increased the dose of levodopa and patient got worse. Then we thought serotonin syndrome and stopped her SSRI and MAO-B medications. After that her clinical condition got better. This case shows that the rare diagnosis of serotonin syndrome should be kept in mind in patients presenting with severe rigidity in Parkinson's disease patients.

## Keywords

Serotonin syndrome; Severe rigidity, MAO-B drugs; Parkinson's disease, SSRI drugs; Rasagiline.

## Abbreviations

SS: Serotonin Syndrome; MAOB: Monoamine oxidase inhibitor–B; SSRI: Selective serotonine reuptake inhibitors.

## Introduction

Serotonin Syndrome (SS) occur as a result of excessive activation of 5-HT (hydroxytryptamine) receptors in the central nervous system, vascular endothelial cells, and platelets. SS-related drugs among: antidepressants, antiemetics, antitussives, antibiotics, migraine medications, narcotic drugs, diet supplements and analgesics are listed [1]. When more than one of the serotonergic drugs is taken, the severity of the disease increases [2]. Major symptoms can start 6 hours after ingestion of the serotonergic agent, and a specific laboratory analysis method has not been defined yet [3]. Presence of serotonergic drug use in the patient's history and findings such as tremor, akathisia or clonus should make the clinician suspicious about this syndrome [3]. Among the treatment principles, discontinuation of the serotonergic

agent, intravenous hydration, and supportive treatment and control of existing agitations are recommended [1]. We present a case of serotonin syndrome, who has parkinson's disease and of major depressive disorder together.

## Case Report

A 83-year-old woman was brought to our neurology department by her son with complaints of nausea, vomiting, excessive sweating, halucinations and rigidity. Her medical history revealed the use of Sertraline 100 mg tablets due to a diagnosis of depressive disorder and rasagilin 1 mg tablets due to diagnosis of parkinson's disease. The patient was having 375 mg daily levodopa/benserazide combination due to diagnosis of parkinson's disease, antiagregans due to diagnosis of cerebrovascular and cardiovascular disease and ACEI drugs due to diagnosis of hypertension. The general condition of the patient was moderate, conscious, restricted oriented, limited cooperated to examination, and agitated. The Glasgow Coma Score was rated as 14. In the vital follow-ups, fever was 37.2°C, arterial blood pressure was 154/105 mmHg, and heart rate was 96/min. On physical examination, her pupils were isochoric and pupil reflex was bilaterally reactive. In the physical examination, patient was have severe rigidity, her left sided muscle strength was 3/5 due to diagnosis of cerebrovascular disease. This wasn't a new onset symptom. Other system examinations were evaluated as normal. The patient's self-care was inadequate. She was have altered mental status and confusion in the mental state evalution. She was having halucinations time to time. She could eat her meals with help. We initially missed the correct diagnosis. Because of her severe rigidity we started levodopa/carbidopa/entecapone combination therapy. We linked her other symptoms to diagnosis of delirium and we continued her ssri and mao-b inhibitor medications. In the follow-ups, the patient's consciousness deteriorated and tonic jaw contraction occurred. Because of this, she was unable to eat even with help. We started to feed her with nasogastric tube. Her orientation was gone. She wasn't able to talk because of jaw contractions. Her infection parameters was low although she was sweating a lot. We felt the need to review the diagnosis because of its response to levodopa dose escalation. We lowered the dose of levodopa, stopped the rasagilin and sertraline drugs. After we stopped her drugs, her condition improved. Her jaw contraction was gone and she could eat and talk again. When we evaluated the patient retrospectively after all these events, we found the diagnosis of serotonin syndrome appropriate.

## Discussion

Serotonin (5-HT) is a neurotransmitter synthesized from the amino acid L-tryptophan in the presynaptic area and localized in the Raphe nucleus in the central nervous system. Serotonin concentration is mainly regulated at postsynaptic receptors by «feedback loop», «re-uptake mechanism and metabolism». Seven types of serotonin receptors have been described in the literature, and it has been reported that serotonin receptors located in the central nervous system are responsible for the regulation of behavioral state, thermoregulation, sleep-wake cycle, muscle tone and pain perception. It has been reported that serotonin receptors located in cells outside the central nervous system are localized in thrombocyte and intestinal enterochromaffin cells, and regulate vascular tone and gastrointestinal mobility [1,3]. In the literature, specific postsynaptic hydroxytryptamine1A (HT1A) receptors are responsible for the

antidepressant and anxiolytic effects; Stimulation of postsynaptic 5-HT1A and 5-HT2A receptors has been reported to cause serotonin syndrome. It has been reported that the incidence of serotonin syndrome development is 14-16% in case of overdose of serotonergic drugs; While high-dose use due to a single drug has been reported in many cases, combinations of two or more serotonergic drugs have also been frequently mentioned. Even with the use of drugs at therapeutic doses, cases of serotonin syndrome have been reported [1,4,5]. In the literature, selective serotonin reuptake inhibitors (SSRI), serotonin-norepinephrine reuptake inhibitors (SNRI), tricyclic antidepressants, monoamine oxidase inhibitors, fentanyl, methadone, tramadol, ondansetron, metoclopramide, cocaine, ecstasy and some other illegal drugs are included [3]. SS is a picture characterized by mental, autonomic and neuromuscular dysfunctions; Clinical findings may occur immediately after taking the drug or in less than 24 hours. There is no laboratory method to confirm the diagnosis, and the diagnosis can be made mostly with the presence of serotonergic drug use in the patient's history and clinical findings that develop after other diseases are excluded [6]. The complaints of our case developed after the concomitant use of MAO-B inhibitor drug and SSRI drug.

In the differential diagnosis of this syndrome, especially in terms of neurological symptoms, neuroleptic malignant syndrome, dystonic reaction, carcinoid syndrome and encephalitis are in the first place, and the combination of clonus, hyperreflexia and "flushing" are shown as the most specific findings for serotonin syndrome [6]. A defined consensus and/or guideline for the treatment of SS has not yet been found in the literature. . Among the supportive treatments, external cooling, intravenous hydration, use of antihypertensive drugs, administration of anxiolytic and sedative agents for agitation, and in severe cases muscle paralysis and switching to mechanical ventilation are recommended [2,6]. It is argued that benzodiazepine-type agents cause nonspecific inhibitory effects on serotonergic transmission and, thanks to this feature, they can take an important place in treatment [7]. In this case, the use of a toxic level (2800 mg) of serotonergic agent (sertraline), tachycardia, tachypnea, and tonic-clonic contractions in the extremities occurred 8 hours after taking the drug, agitation, hallucination, hyperthermia and generalized convulsions accompanied the picture at the end of 18 hours, and this The persistence of the findings for 24 hours suggested that the patient developed serotonergic syndrome. As a matter of fact, it was possible to remove the patient from this syndrome by stopping the absorption of the suicidal agent into the body by giving activated charcoal after gastric lavage, as well as trying to accelerate its elimination from the body with intravenous hydration and starting benzodiazepine infusion for concurrent convulsions. On the other hand, by giving cyproheptadine, a 5-HT1A/2A receptor antagonist agent, to the patient, it was aimed to quickly reverse the effects of the serotonergic agent that passed into the body. As a matter of fact, it was observed that this agent made the treatment applied to the patient more effective thanks to its rapid solubility in body fluids [5,8]. On the other hand, since it has been shown that the hyperthermia in SS results from increased muscle activity, no antipyretic agent was given in this case, and external cooling was preferred instead [6]. Thanks to all these treatments, hemodynamic stability was achieved at the end of 24 hours. In conclusion, in this case, the patient and the syndrome were treated effectively and successfully, thanks to the detection of a history of serotonergic drug use, the early recognition of the mental, autonomic and neurological finding triad, and the early application of pharmacological antagonism and other supportive medical treatments. Because serotonin syndrome is a rare disease, it can be overlooked. However, when

maob inhibitors and ssri drugs are used together, serotonin syndrome should definitely come to mind. Since muscle rigidity is a well-known symptom of the Parkinson's disease patients, it can be confused with the muscle rigidity of serotinin syndrome. In our patient, the outcome was not fatal, since the clinical course was not serious. However, it can have fatal consequences in a patient with a poor clinical course. Therefore, detailed questioning of the drugs used is necessary for early diagnosis and intervention. As a result, the determination of the history of serotonergic drug and mao-b inhibitor use, early recognition of the mental, autonomic, and neurological finding triad, and the early application of supportive medical treatments can be effective and successful management of the case.

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