Diaphoresis: A Presentation of Serotonin Syndrome From Vortioxetine

To the Editor: The clinical presentation of serotonin syndrome spans a wide spectrum—from benign to lethal—due to increased serotonergic activity in the central nervous system.[1] The classical features of serotonin syndrome are indistinguishable in the early stages, posing a diagnostic challenge.[1] We present the case of a patient who developed serotonin syndrome while taking vortioxetine 10 mg/d (therapeutic dose). To our knowledge, this is the first report of serotonin syndrome with vortioxetine.

Case report. A 69-year-old woman, hospitalized for the past 2 weeks for psychosis and urinary tract infection, developed diaphoresis. She had a history of dyslipidemia, late-onset psychosis, and generalized anxiety disorder. Amisulpiride 800 mg/d had been discontinued 10 days ago due to akathisia. She had been taking propranolol 30 mg/d and vortioxetine 10 mg/d for the last year.

The patient was alert and orientated. Her mattress was drenched with sweat. Her vital signs included a temperature of 98.1°F (36.7°C), blood pressure of 160/100 mm Hg, heart rate of 88 beats/min, and respiratory rate of 22 breaths/min. Findings of heart and lung examinations were unremarkable. Upper limbs were rigid with occasional tremors. Reflexes were brisk in all limbs, plantars were upgoing with no limb clonus, and no ocular clonus was present. Her electrocardiogram result was within normal limits. She had a white blood cell count of 13.3×10^9/L, a renal panel result that was within normal limits, and an elevated creatine kinase level (CK) (3,086 U/L).

The most likely diagnosis was serotonin syndrome (per the Hunter Serotonin Toxicity criteria[2]). Neuroleptic malignant syndrome was also considered in the differential diagnosis, as it mimics serotonin syndrome. However, reflexes are hypoactive in neuroleptic malignant syndrome. Given the patient's recent urinary tract infection, sepsis was also considered. Sepsis is cited as the reason for hospital admission in 12% of patients with serotonin syndrome—second only to altered consciousness.[3]

Vortioxetine was discontinued, and the patient was given intravenous saline 3 L/d and regular doses of clonazepam. She became less restless, her CK level decreased from 1,056 U/L to 536 U/L, and a urine culture result was negative. However, 2 days later, she was noted to be more agitated and was started on cyproheptadine. Her condition resolved 5 days after diaphoresis onset.

Diaphoresis is a common, yet nonspecific, symptom in medicine. Acute onset of diaphoresis should raise concern of a serious underlying medical condition. Only 21% of patients with serotonin syndrome have diaphoresis at diagnosis, compared to more notable signs like agitation, tachypnea, tachycardia, and fever.[3]

In our case, hyperreflexic responses provided the most insight into the diagnosis of serotonin syndrome. A bedside neurologic examination with tendon hammer and torchlight is an indispensable tool when making this diagnosis, especially in the current era that is heavily dependent on laboratory and radiologic tests. The absence of fever and masking of sympathetic features by propranolol use compounded the mild presentation of symptoms in our patient. The absence of clonus could have been caused by increased muscle tone.

Use of selective serotonin reuptake inhibitors in clinical practice has increased.[4] Early presentation of serotonin syndrome can be vague and easily dismissed. Therefore, physicians should have a heightened awareness for this condition.

References

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