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S. French , C. Wray

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French S, Wray C

Oregon Health and Science University, Pediatric Neurology, Portland (OR) USA

*Corresponding author. Oregon Health and Science University, Pediatric Fellowship Program, Mail Code: CDRCP Rm 3214, 707 SW Gaines St, Portland, OR 97239-2998. Tel.: (505)412-5716; Fax: 503-494-6868

CASE REPORT

The patient is a 15-year-old boy who presented with serotonin syndrome after an intentional overdose by ingestion of 60 tablets of bupropion (300mg) and 30 tablets of vilazodone (20mg). Prior to the ingestion he was taking several advanced classes and received good grades in school.

At presentation, the patient had hallucinations, fever, and intermittent tremor without seizures. He became hypotensive and hypoxic with SpO₂ in the 80s for roughly 1 minute, prompting immediate intubation. He was treated with vasopressors and cyproheptidine. He also received lorazepam and fosphenytoin because of his tremors, but video electroencephalogram (EEG) demonstrated that these were not epileptic. EEG showed a single right central 17 second focal subclinical seizure. Head magnetic resonance imaging (MRI) demonstrated acute isolated bilateral hippocampal anoxic injury (Fig 1).

Memory testing at two weeks post-ingestion showed poor short term memory that improved at the 3-month reevaluation (Tab 1). Because of his memory deficits, the patient required new provisions at school that allowed him access to notes during exams. He also developed focal epilepsy.

Fig. 1. MRI brain without contrast on hospital day 15 showing acute diffusion restriction within the hippocampus bilaterally with corresponding T2 hyperintensity and increased FLAIR signal, consistent with cytotoxic injury, likely postschismic.

Table 1. Wide Range Assessment of Memory and Learning, second edition (WRAML-II) at 2 weeks and 3 months post ingestion showing improving memory deficits.

WRAML-II	Classification at 2 weeks	Classification at 3 months
Story Memory		
Immediate Recall	Borderline Low	Average/Age-Typical
Delayed Recall	Low	Average/Age-Typical
Delayed Recognition	Borderline Low	Average/Age-Typical
Verbal Learning		
Immediate Recall	Low Average	Average/Age-Typical
Delayed Recognition	Low	Average/Age-Typical
Delayed Recall	Low	Average/Age-Typical
Picture Memory		
Immediate Recall	--	Low Average
Delayed Recognition	--	Average/Age-Typical

DISCUSSION

The etiology of infarction in this patient is likely multifactorial with serotonin syndrome being the strongest contributor. He was hypotensive and hypoxic with possible transient hypoperfusion, though the brevity of this state makes it unlikely to fully explain his infarcts. His subclinical seizure would have increased cerebral metabolic demand and may have made his brain more susceptible to ischemic injury. While status epilepticus alone in the setting of hypoxia can cause bilateral hippocampal infarcts,¹ this patient's seizure was too short and focal to explain his injury.

Serotonin syndrome can cause stroke,² and patients on selective serotonin reuptake inhibitors (SSRIs) have been reported to develop reversible segmental vasoconstriction of cerebral arteries.³ Parallels in mechanism of injury may be drawn between cocaine abuse and serotonin syndrome because SSRIs and cocaine inhibit the serotonin receptor SERT, and, similar to bupropion, cocaine inhibits reuptake of norepinephrine and dopamine. Isolated bilateral hippocampal infarcts have been reported in the setting of cocaine abuse.⁴ The mechanism of injury is thought to be cerebral vasospasm and vascular thrombosis.⁵

This case identifies a novel association between serotonin syndrome and bilateral hippocampal infarctions with a potential causative explanation and provides prognostic guidance that resulting memory deficits may improve. It adds to our knowledge of potential complications of serotonin syndrome that should be evaluated for and treated.

STATEMENT OF ETHICS

The authors have no ethical conflicts to disclose

DISCLOSURE STATEMENT

The authors have no conflicts of interest to declare

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AUTHOR CONTRIBUTIONS

Sophia French, MD: Wrote, revised, and approved work

Carter Wray, MD: Revised and approved work

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