



Clinical Letter

Serotonin Syndrome and Hippocampal Infarction

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Patient description

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

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

Memory testing two weeks after the ingestion showed poor short-term memory that improved at the three-month re-evaluation (Table). Because of his memory deficits, he required new provisions at school that allowed him access to notes during examinations. He also developed focal epilepsy.

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, but 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 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 both selective serotonin reuptake inhibitors and cocaine inhibit the serotonin transporter, 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 patient demonstrates a novel association between serotonin syndrome and bilateral hippocampal infarctions and provides a potential explanation. Memory deficits in this setting may improve. Our report illustrates potential complications of serotonin syndrome that should be treated.

Disclosure: The authors have no conflicts of interest to declare.

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Statement of Ethics

The authors have no ethical conflicts to disclose

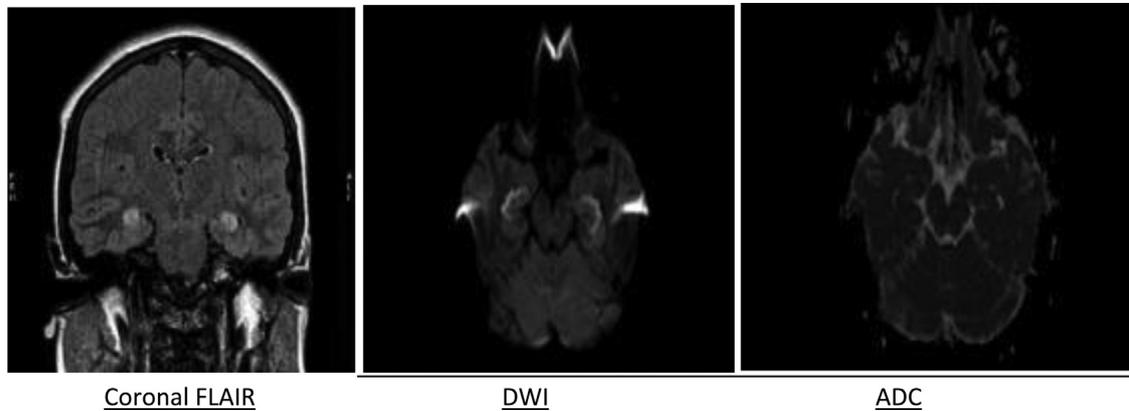


FIGURE. Magnetic resonance imaging brain without contrast on hospital day 15 showing acute diffusion restriction within the hippocampus bilaterally with corresponding T2 hyperintensity and increased FLAIR signal, consistent with cytotoxic injury, likely post-ischemic.

TABLE. Wide Range Assessment of Memory and Learning, Second Edition (WRAML-II) at Two Weeks and Three Months Postingestion Showing Improving Memory Deficits

WRAML-II	Classification at Two Weeks	Classification at Three 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

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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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