A Previously Healthy Adolescent With Acute Encephalopathy and Decorticate Posturing

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A 14-year-old previously healthy female was transferred from a local emergency department after being found unresponsive at home. Parental questioning revealed she had fever and pharyngitis 2 weeks before presentation. Past mental health history was negative, including concern for past or present suicidal ideation/attempts, suspected substance use, or toxic ingestion. In the emergency department, she was orotracheally intubated due to a Glasgow Coma Scale of 3. She was hemodynamically stable and euglycemic. Electrocardiogram showed sinus tachycardia. She underwent a noncontrast head computed tomography that was normal and subsequently underwent a lumbar puncture. She had a seizure and was given a loading dose of diazepam and fosphenytoin that led to cessation of extremity movements. She was subsequently transferred to the PICU for additional evaluation. Initial examination without sedation or analgesia demonstrated dilated and minimally responsive pupils, intermittent decorticate posturing, and bilateral lower extremity rigidity and clonus, consistent with a Glasgow Coma Scale of 5. Serum studies were unremarkable with the exception of mild leukocytosis. Chest radiograph only showed atelectasis. She was empirically started on antibiotics to cover for meningitis pending final cerebral spinal fluid test results. The pediatric neurology team was consulted for EEG monitoring, and the patient was eventually sent for computed tomography angiogram and magnetic resonance angiogram/venogram. We will review diagnostic evaluation and management of an adolescent patient with acute encephalopathy with decorticate posturing of unclear etiology.

CASE HISTORY WITH SUBSPECIALTY INPUT

Dr Yu Kawai (Senior Fellow, Pediatric Critical Care Medicine):

In April 2015, a previously healthy 14-year-old female was transferred from a local emergency department with altered mental status and witnessed generalized tonic–clonic seizure. The patient was found unresponsive in her bed the morning of her admission by her parents. Per their report, she was not responding to verbal or tactile stimuli, pupils were dilated, lips were noncyanotic, bilateral upper extremities were flexed, and fists were clenched toward her chest. She exhibited urinary incontinence. There were no signs of traumatic injury. Seven hours before this episode, she was in her usual state of health, conversant, asymptomatic, with baseline cognition, behavior, and emotion. Two weeks before presentation, the patient was noted to have had a fever of 38.8°C

abstract

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Dr Kawai led the initial writing of the manuscript, recruited all specialists involved for writing the manuscript, revised the manuscript, created the figures and tables, and was involved in the care of the patient; Ms DeMonbrun, Ms Chambers, and Drs Nolan, Malas, and Quasney contributed to the writing of the manuscript, revised the manuscript, and were involved in the care of the patient; Dr. Dolcourt contributed to the writing of the manuscript; and all authors approved the final manuscript as submitted.

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with odynophagia that resolved spontaneously within 24 hours. She lived at home in a rural town in Michigan with her biological parents. There were no known sick contacts, and her immunizations were up to date. She commonly played outside in a wooded area near a swamp by her home. The patient was in eighth grade with satisfactory academic performance and no history of learning difficulties. She socialized well with friends and was on the ice skating team. There were no recent reports of head injury or other sport-related traumatic injuries. The family did not have any pets and had not recently traveled outside the state. Per parental report, she had never smoked, used illicit drugs, or ingested alcohol. She had regular menses and was not reported to be sexually active. She had no reported past mental health history including no previous psychotropic use, psychotherapy, or psychiatric admissions. Her parents denied their daughter being exposed to recent major life stressors and denied witnessing signs of mood changes, anxiety, or suicidal ideation. She had no history of aggressive behavior or suicide attempts. There were no witnessed or reported toxic ingestions. Family history was negative for seizure disorder, autoimmune disease, bleeding disorder, arrhythmia, or malignancy.

She was transported by her parents to a local emergency department (ED) where she was unresponsive with absent gag reflex. Her mental status did not improve with a dose of naloxone. Her Glasgow Coma Scale (GCS) was 3, and she was orotracheally intubated for airway protection. Chest radiograph showed no acute cardiopulmonary processes. In the ED, she was hypothermic to 35.9°C and tachycardic to the 120s. She was otherwise normotensive, was on room air, euglycemic, and had normal electrolytes. A urine drug screen was negative for amphetamines, benzodiazepines, cocaine, barbiturates, cannabinoids, oxycodone, and other opiates. She had undetectable serum levels of acetaminophen, aspirin, and alcohol. Her pregnancy test was negative. She did have a leukocytosis to 13.9 K/μL, anemia with hemoglobin of 10.9 g/dL, and a normal platelet count. She had an electrocardiogram that showed sinus tachycardia with a ventricular rate of 125 beats per minute and a QTc of 473 milliseconds. A noncontrast head computed tomography (CT) showed no acute or chronic intracranial abnormalities. While in the radiology room, she had a seizure that was described as flexion of her upper extremities toward her chest with tremors of her lower extremities. She was given a loading dose of diazepam and fosphenytoin, and the seizure abated. Lumbar puncture was performed but without an opening pressure, and cerebral spinal fluid (CSF) studies were sent. She remained normotensive on minimal mechanical ventilator settings. Vancomycin, ceftriaxone, and acyclovir were initiated empirically for coverage against potential bacterial and viral meningitis. The decision was made to transfer the patient to the PICU. En route, she appeared to be "reaching toward her endotracheal tube" and was given a dose of fentanyl.

What did her initial exam demonstrate on arrival to the PICU?

Rebecca Chambers (Nurse Practitioner, Pediatric Critical Care Medicine):

On arrival, pupils were 8 mm bilaterally and minimally reactive to light. Ophthalmoscopic exam showed normal optic disc size and margin. She was noted to have intermittent, bilateral upper extremity flexion toward the chest with clinched fists and bilateral lower extremity extension with painful stimuli to the upper extremities, consistent with decorticate posturing and a GCS of 5. She also had bilateral lower extremity rigidity and inducible clonus, but neither was apparent in the upper extremities. Additional neurologic examination demonstrated intact corneal reflex bilaterally, present oculocephalic reflex, minimal facial grimacing with supraorbital pressure, and no cough reflex to tracheobronchial suctioning. Her degree of patellar and Achilles tendon reflexes were 3+ bilaterally. She had absent respiratory effort. On general examination, she was a well-nourished adolescent female with good hygiene. Head and neck exams were normal. Cardiovascular exam demonstrated tachycardia with normal rhythm without murmur, pericardial rub, or galloping beats. Respiratory exam revealed good bilateral air entry with clear vesiculobronchial breath sounds and normal work of breathing on the mechanical ventilator. Her abdomen was soft to palpation without organomegaly. Genitalia showed a sexual maturity rating of 5 without mucosal lesions or foreign body. Her extremities were well perfused with no peripheral edema. Her skin was warm, dry, and intact without rashes, lesions, or ecchymosis.

Dr Kawai:

What initial work-up did you perform?

Ms Chambers:

Sedatives and analgesics were held to monitor her neurologic status. Postpatient transport chest radiography confirmed appropriate endotracheal tube position and no acute cardiopulmonary processes were evident. The initial intake serum studies, including electrolytes, lactate, renal function, liver function, coagulation panel, and thyroid function, were unremarkable. A repeat urine drug screen, which would assess for amphetamines, benzodiazepines,

cocaine, barbiturates, cannabinoids, oxycodone, and other opiates, was sent. Because our urine drug screen does not detect other drugs of abuse, such as phencyclidine, γ-hydroxybutyrate, lysergic acid diethylamide, designer amphetamines, and tryptamines, we also sent a comprehensive urine gas chromatography and mass spectrometry (GCMS) test to broaden the evaluation for toxic ingestion despite parental denial of the possibility of toxic ingestion. Complete blood count showed leukocytosis to 16.2 K/μL with neutrophil predominance of 88.9%, anemia of 10.4 g/dL with low mean corpuscular volume to 71.9 fL, and normal platelet count of 260 K/μL. Infectious workup included a normal urinalysis and a negative respiratory infection polymerase chain reaction (PCR) panel via nasal swab detecting adenovirus, coronavirus 229E/HKU1/NL63/OC43, human metapneumovirus, human rhinovirus, enterovirus, influenza A/B, parainfluenza 1–4, respiratory syncytial virus, bordetella pertussis, chlamydophila pneumoniae, and mycoplasma pneumoniae. Furthermore, blood, urine, and mini-bronchoalveolar lavage cultures were sent. Herpes simplex virus PCR was added to the CSF sample from the local ED, which were all pending at this time. Given that the patient met systemic inflammatory response syndrome criteria and her exposure to wooded areas, we examined for tick or other insect bites, including her oral and vaginal mucosa. Because she also may have been exposed to the swamp area, we changed ceftriaxone to piperacillin/tazobactam to cover for *Pseudomonas* species, while continuing vancomycin and acyclovir. Our initial differential diagnoses included toxic ingestion either in abuse of a substance or with suicidal intent, traumatic injury, intracranial anatomic or vascular processes, or an acute infection. Given the evolution of the patient's presentation, lack of reported mental health history, lack of collateral information to suggest an ingestion, and current evaluation, infectious etiology became higher on the differential. The pediatric neurology team was consulted for additional evaluation.

Dr Kawai:

At this point, what other neurologic workup is necessary?

Dr Danielle Nolan (Senior Resident, Pediatric Neurology):

Our initial neurologic examination revealed dilated and unreactive pupils with no response to painful stimulation in all extremities. Her lower extremity exam was worse than the upper extremity exam, showing increased rigidity and clonus comparatively. Decorticate posturing had resolved at this time. Her seizure in the ED was described as quick flexion of her arms with possible tremors of her legs. Our initial differential included toxic ingestion and status epilepticus due to infectious, metabolic, autoimmune, or anatomic cause, with seizure activity higher on the list of acute concerns while waiting for the urine GCMS test; therefore, we recommended long-term video EEG monitoring. The study was negative for epileptiform activity, but did demonstrate diffuse slowing consistent with moderate encephalopathy.

Dr Kawai:

We still do not have an etiology for the patient's presentation. What is our next step?

Dr Nolan:

A repeat neurologic examination about an hour after our initial evaluation revealed decorticate posturing to painful stimulation. Given no other changes in the clinical evaluation and no additional history, this evolution of presentation made brainstem ischemia, specifically basilar artery thrombosis, our most urgent concern. Basilar artery thrombosis is a potentially fatal neurologic condition that can present with subtle CT findings that requires urgent evaluation and management if suspected. Clinically, patients can exhibit a wide variety of neurologic symptoms, including altered level of consciousness, paresis, and convulsions. At this time, the head CT images from the local ED became available and demonstrated a possible hyperdense basilar artery concerning for basilar artery thrombosis (Fig 1). Subsequently, an emergent head CT angiogram of the head and neck was obtained. This was normal, revealing no evidence of thrombus. However, it is possible for a cerebral vascular accident to be CT-occult. Several studies have shown that only one-third to two-thirds of lesions are detected on CT due to the timing of onset and associated diffusion changes.[1] Therefore, she then had a brain and cervical spine magnetic resonance angiogram/venogram (MRA/MRV) to evaluate for structural changes, such as ischemic changes not visualized on the CT, arterial and venous thrombosis, and arterial dissection.

Dr Kawai:

Are any of the pending laboratories and images starting to return with results?

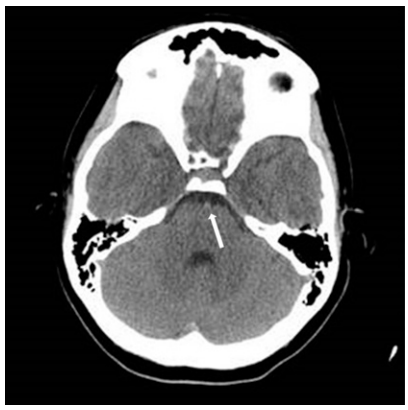

FIGURE 1
Noncontrast head CT showing hyperdensity (arrow) of the basilar artery, suggestive of thrombosis.

Andrea DeMonbrun (Nurse Practitioner, Pediatric Critical Care Medicine):

CSF white blood cell count was undetectable with 35 per mm^3 of red blood cells present. CSF glucose was 78 mg/dL and protein count was 18 mg/d. Blood, urine, and CSF cultures were still noted to be negative, but it had only been 12 hours since the cultures had been sent. Herpes simplex virus PCR on CSF was negative, and acyclovir was discontinued. Repeat urine drug screen with an analytic time of 1 hour returned positive for opioid, but this was due to the fentanyl she received en route to our PICU. Urine GCMS test was still pending because analytic time can take up to 24 hours. MRA/MRV of the brain and cervical spine were both normal with no acute changes (Figs 2 and 3).

Dr Kawai, MD:

Given a lack of an etiology, are we sure that she did not ingest any substances not identified on the urine drug screen?

Ms DeMonbrun:

Ingestion became a greater consideration in our differential. Because we were actively evaluating the patient, we were repeatedly attempting to glean clues to the etiology from additional collateral history. We thought it prudent to reevaluate the presenting history about 8 hours after the patient had arrived to our PICU because of the lack of clear etiology.

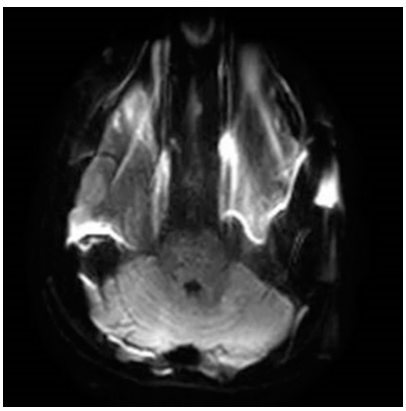

FIGURE 2
MRI of the brainstem showing no diffusion changes to suggest ischemia (motion and equipment artifact present).

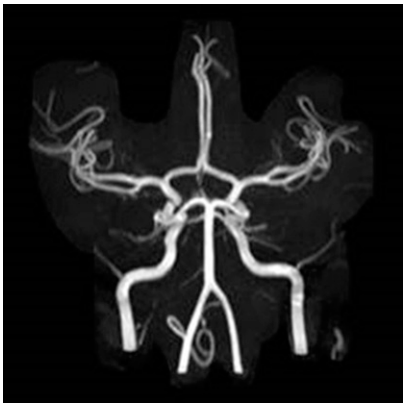

FIGURE 3
MRA of the brain showing no evidence of arterial thrombosis/dissection.

The patient's father was recently diagnosed with acute back pain and was prescribed cyclobenzaprine immediate-release 10 mg daily. The patient's mother had been diagnosed remotely with major depressive disorder and had been taking bupropion sustained-release 150 mg twice daily and venlafaxine extended-release 150 mg daily. On further questioning, there was some maternal family history of unipolar depression. Her parents continued to emphasize not witnessing any emotional, behavioral, or cognitive disturbance in their daughter. She was described as pleasant, well adjusted, and without significant irritability or social isolation. She had been doing well academically and competing actively in ice skating. Her parents did not report a history of bullying at school, romantic relationships, significant conflict with peers, recent losses in the family, or other traumatic events. The parents denied any endorsed dysphoric mood, anhedonia, sleep or appetite disturbance, self-injurious behavior, or suicidal ideation. The parents stated they were with the patient throughout the day before admission and reported no missing over the counter or prescription medication bottles at home. Despite their low suspicion, we encouraged the parents to return home to assess if any tablets were missing from their medication bottles. The father immediately called us from home to let us know that 10 tablets of cyclobenzaprine, 40 tablets of bupropion, and 10 tablets of venlafaxine were missing. The urine GCMS test 6 hours later confirmed that she had ingested cyclobenzaprine, bupropion, and venlafaxine.

Dr Kawai:

Now knowing that she had polypharmacological ingestion, does her neurologic manifestation fit with a clear diagnosis?

Dr Nasuh Malas (Assistant Professor, Pediatric Consultation–Liaison Psychiatry):

Severe serotonin syndrome (SS) does fit and is caused by elevated levels of serotonin in the central nervous system related to the ingestion of agents that enhance serotonergic activity.[2] Cyclobenzaprine is a skeletal muscle relaxant that has a tricyclic structure similar to tricyclic antidepressants, which has selective serotonin and norepinephrine reuptake inhibitor activities.[3] Bupropion is an antidepressant that inhibits dopamine and norepinephrine reuptake with little impact on serotonergic activity. However, bupropion has been reported to be associated with SS.[4,5] Venlafaxine is another antidepressant that functions primarily as a serotonin and norepinephrine reuptake inhibitor.[6] Venlafaxine has been associated with SS in isolation, even at low doses, and the potential for SS increases significantly with coadministration with other agents that can potentiate serotonergic activity.[7] Numerous other agents, including antimicrobial agents (linezolid), antiemetics (ondansetron), and

analgesics (fentanyl) have also been implicated given their potential serotonergic properties.[8,9] Ingesting cyclobenzaprine, bupropion, and venlafaxine simultaneously, as well as a possible contribution with administration of fentanyl during transport, certainly would place the patient at increased risk of developing SS.

Dr Kawai, MD:

What is the clinical presentation of SS, and what are the differential diagnoses?

Dr Bram Dolcourt (Assistant Professor, Emergency Medicine and Medical Toxicology):

SS manifests with the classic triad of neuromuscular excitation (tremor, hyperreflexia, rigidity, and clonus), autonomic instability (mydriasis, diaphoresis, hyperthermia, tachypnea, tachycardia, hypertension), and altered mental status (agitation, confusion, and comatose state).[2] Interestingly, neuromuscular findings, including tremor, hyperreflexia, and clonus, are more pronounced in the lower extremities compared with upper extremities, which was the case for this patient. These signs and symptoms can start within hours of ingestion. The diagnosis of SS is made clinically with the aid of the Hunter Serotonin Toxicity Criteria Decision Rules, which exhibit 84% sensitivity and 97% specificity for the diagnosis of SS (Fig 4).[10]

The differential diagnoses for SS include neuroleptic malignant syndrome (NMS), anticholinergic toxicity, malignant hyperthermia, sympathomimetic toxicity, thyrotoxicosis, idiopathic malignant catatonia, hyperactive delirium, and meningitis/encephalitis. SS and NMS have overlapping features.[11] In the case of polypharmacy of unknown drugs, such as in this case, differentiating SS, NMS,

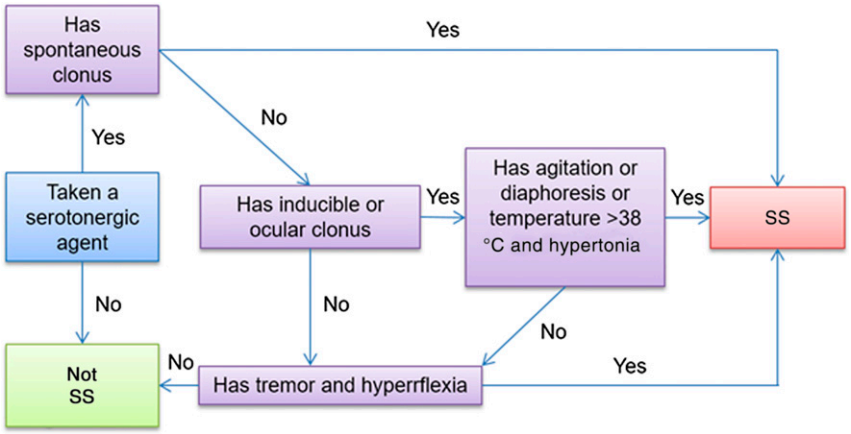

FIGURE 4
Hunter Serotonin Toxicity Criteria decision rules for SS.

and neuromuscular movements from other drug toxicity can be challenging. Similarities and differences between SS and NMS are shown below in Tables 1 and 2.[8,9,12] Also, it's important to keep in mind that bupropion toxicity can present with clinical features similar to SS, such as agitation, diaphoresis, and tachycardia, but severe cases usually present with seizures, hypertension, and arrhythmias, which were lacking in our patient.[3,13] Myoclonus can also occur in bupropion toxicity, but is extremely rare.

Dr Kawai:

Have there been any reports of neurologic posturing associated with severe SS?

Dr Dolcourt:

Not in the pediatric population, but there is 1 reported case series of 2 adults with decerebrate posturing thought to be due to reversible cerebral vasoconstriction syndrome associated with SS. Both of these patients had severe, life-altering neurologic insults.[14] There is also a pediatric case report of neurologic posturing secondary to bupropion-induced status epilepticus leading to cardiac arrest, but it is difficult to attribute the posturing directly to the bupropion.[15] Our patient is unique in that she had posturing without MRI findings suggestive of cerebral vasoconstriction nor seizures detected on the EEG.

TABLE 1 Serotonin Syndrome versus Neuroleptic Malignant Syndrome: Similarities

Altered mental state
Agitation, confusion, and comatose state
Autonomic disturbances
Hyperthermia, tachycardia, tachypnea, hypertension, and diaphoresis
Mydriasis (common in serotonin syndrome, atypical in neuroleptic malignant syndrome)

Dr Kawai:

How is a patient with SS managed?

Dr Malas:

Treatment of SS includes the discontinuation of serotonergic agents and supportive care. The use of benzodiazepines has strong support for alleviation of agitation, neuromuscular excitability, and vital-sign abnormalities.[16] In severe SS, cardiac, airway, and respiratory functions need to be closely monitored. Any patient suspected of SS with significant autonomic instability, particularly severe hyperthermia (temperature >41.1°C), should be transferred to an intensive care setting with consideration of airway protection by endotracheal intubation.[8] Fever is caused by muscle rigidity and is not due to central hypothalamic temperature

TABLE 2 Serotonin Syndrome versus Neuroleptic Malignant Syndrome: Differences

	Serotonin Syndrome	Neuroleptic Malignant Syndrome
Pathophysiology	Increased serotonin activity	Decreased dopamine activity
Agents	Antidepressants	Antipsychotics
	Antiemetics	Antiemetics
	Antiepileptics	
	Antibiotics	
	Analgesics	
	Stimulants	
	Illicit drugs	
Neuromuscular excitability	Hyperreflexia	Hyporeflexia
	Extremity and ocular clonus	Rigidity > tremors
	Tremors > rigidity	
Symptom onset	Within 24 h of ingestion	Days after ingestion
Laboratory findings	None	Elevated creatinine phosphokinase, lactate dehydrogenase, aspartate aminotransferase, and white blood cell count
		Low serum iron level
Treatment[a]	Cyproheptadine	Dantrolene
		Bromocriptine
		Amantadine
		Electroconvulsive therapy
Symptom resolution after treatment	Within a few days	Usually takes days to weeks

[a] In addition to discontinuing causative agents, maintaining cardiopulmonary stability, supportive care, and benzodiazepine use for neuro-agitation.

dysregulation, so antipyretics have a limited role.[8]

Although our patient with SS did not have hyperthermia, her initial GCS and presentation necessitated airway protection with endotracheal intubation and mechanical ventilation. The patient was also placed on continuous cardiac monitoring. Serial electrocardiograms were obtained every 6 hours to monitor for QTc prolongation or evidence of dysrhythmia. She did receive intermittent doses of lorazepam with improvement in clinical status. We considered administering cyproheptadine, a histamine and serotonin antagonist, yet given the rapid clinical response to benzodiazepines, this treatment was not warranted. In severe SS, vital signs can fluctuate widely and rapidly, so the patient should be closely monitored for at least 48 hours or until the patient shows gradual and sustained symptomatic improvement.

Dr Kawai:

What was the rest of her PICU course after the diagnosis was made?

Ms DeMonbrun:

All cultures were negative at 48 hours, and antibiotics were discontinued. After extubation, the patient quickly became lucid and reported feeling lonely, hopeless, and sad for 6 months in the context of social isolation at school and increased parental conflict at home. She admitted to ingesting her parents' medications as a suicide attempt and had not revealed her suicidal ideations to anyone. The parents did locate her diary later during her hospital course, which expressed her intent in detail.

Dr Kawai:

How is she doing now?

Ms Chambers:

The patient and family participated in psychoeducation on suicide. The child psychiatry consultation team reviewed safety planning, including lethal means restriction in the home. The family did not have any firearms in the house. After medical stabilization, she was eventually transferred to the inpatient psychiatry unit. She underwent cognitive behavioral therapy, attended group therapy, and was initiated on fluoxetine 10 mg daily for her major depressive disorder. She was eventually discharged from the hospital with a plan for close primary care physician and psychiatric follow-up.

Dr Kawai:

In a patient with a previous diagnosis of severe SS, restarting a selective serotonin reuptake inhibitor (SSRI) can be an uncomfortable decision for many providers. Can you comment on this?

Dr Malas:

Previous SS is not a contraindication to use serotonergic agents in the future. Often times a patient's depression or anxiety may be so impairing that reintroduction of an SSRI is necessary. Use of any serotonergic agents should only be done after complete resolution of SS. A washout period to ensure no residual effects from the offending agents can take several days to a week depending on the half-life of the initial serotonergic agents used. In the outpatient setting, it is common practice to wait 2 weeks before restarting a serotonergic agent. In an inpatient setting with close clinical supervision, this period is often shorter and dictated by the agents ingested, their half-life, and other clinical circumstances.

Serotonergic agents can be prescribed for a variety of conditions, and therefore, prevention of future episodes of SS is predicated by education, close communication with all involved in the care of the patient, as well as limiting polypharmacy as much as possible.[7] In our case, fluoxetine was started at 10 mg 7 days after her ingestion and monitored for an additional 4 days before discharge. The half-life of fluoxetine can range from 1 to 4 days.[17] Furthermore, fluoxetine

has a psychoactive byproduct, norfluoxetine, that can have a half-life of 7 to 15 days.[17] Use of an SSRI with a longer half-life is not precluded by a previous history of SS, yet should be considered in the monitoring of the patient.

Dr Kawai:

Are there any long-term outcomes associated with severe SS?

Dr Nolan:

I am not aware of any long-term neurologic deficits associated specifically with SS. I also could not find any literature to support outpatient follow-up with neurology once clinical features of SS resolve, and this decision should be made by clinical judgement on a case-by-case basis.

Dr Michael Quasney (Professor, Pediatric Critical Care Medicine):

We found this case to be intriguing diagnostically because our differential diagnoses fluidly moved with the patient's presentation and evaluation. This case highlights the importance of keeping a broad differential and continuing to gather collateral history for diagnostic clarification. Parents often can have a poor understanding of their child's internalizing mental health symptoms. Therefore, reliance on parental report alone should not rule out toxic ingestion in an adolescent with altered mental status.

In summary, this is a case of a 14-year-old previously healthy female who was found unresponsive at home and eventually developed decorticate posturing secondary to severe SS. This is the first case report describing neurologic posturing with severe SS in a pediatric patient. The patient eventually admitted to toxic polypharmacy ingestion with suicidal intent and received inpatient and outpatient psychiatric care. She is currently doing well at home and in school with no neurologic deficits with established outpatient mental health care.

ABBREVIATIONS

CSF: cerebral spinal fluid
CT: computed tomography
ED: emergency department
GCMS: gas chromatography and mass spectrometry
GCS: Glasgow Coma Scale
MRA/MRV: magnetic resonance angiogram/venogram
NMS: neuroleptic malignant syndrome
PCR: polymerase chain reaction
SS: serotonin syndrome
SSRI: selective serotonin reuptake inhibitor

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