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Case Report

Opiate leukoencephalopathy in a pediatric patient

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ABSTRACT

A 17-year-old male presented from an outside facility in acute respiratory failure and profound hypotension. He was found to have classic MRI brain findings of opiate-induced leukoencephalopathy which was corroborated with urine drug screen and history. This entity has been described in the adults but is not as well recognized in the pediatric population. As the opiate epidemic continues to evolve, this case brings heightened awareness of this disorder to pediatric radiologists.

Introduction

Toxic leukoencephalopathy is a rare neurologic impairment that occurs as a result of opiate exposure. The process may present as acute or chronic. This entity is particularly relevant in today's age of opioid epidemic but is not commonly seen in the pediatric population. Presented here is a rare case of leukoencephalopathy in an adolescent due to opiate intoxication.

Case report

A 17-year-old male was found unresponsive in his bed on the morning of presentation by his parents. He had last been reported to be acting normally the night before he left the home, and his parents heard him come home at approximately 1:00 AM, 7 hours prior to being discovered in distress. The patient was found to have white foam around his mouth and was breathing in an agonal fashion. Emergency services were called. Upon arrival, the paramedics administered naloxone without a clinical response. He was hypoxic at 49% and hypotensive in the 60/40 mm Hg during transport. He was brought to the nearest area hospital where he was intubated in the emergency department. He was also started on norepinephrine and dobutamine. A CT performed at the outside institution was interpreted as normal. An initial urine toxicology screen showed the patient to have tramadol, opiates, tetrahydrocannabinol, and benzodiazepines in his system. At this point, the patient was transferred to a children's hospital for further workup and management, though the

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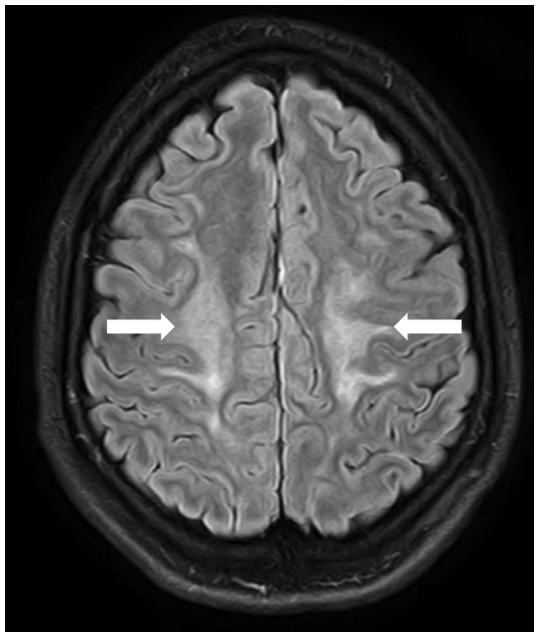

Fig. 1 – Axial FLAIR of the brain demonstrates confluent T2 hyperintensity throughout the superior centrum semiovale bilaterally (arrows).

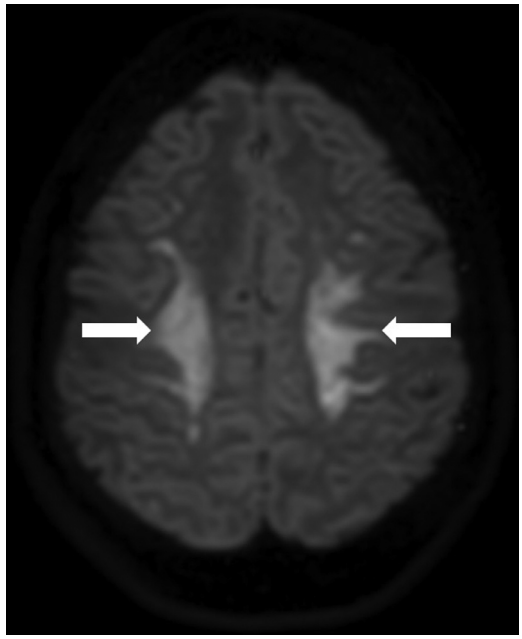

Fig. 2 – Axial diffusion weighted image of the brain exhibits hyperintensity in the region of white matter FLAIR signal abnormality (arrows).

CT images were not sent with the patient for review or additional opinion. He was found to be in cardiogenic shock with severe end-organ dysfunction and multisystem organ failure. Further history from his friends revealed that he had taken a combination of fentanyl, heroin, and alprazolam referred to as "white China" approximately 8 hours before he was found down. The patient did not remember how he took the drugs, but he thought it was only heroin without the additional substances. The patient's parents stated that he had used marijuana and benzodiazepines to manage stress but were not aware of any recent use. There had been no opiate use previously.

An MRI of the brain performed on day 3 of admission showed diffuse FLAIR hyperintensity and restricted diffusion in the white matter of the bilateral posterior centrum semiovale as well as the occipital lobes (Figs. 1–3). The occipital lobes also demonstrated associated T2 and FLAIR signal hyperintensity. The subcortical U fibers were relatively spared. The patient was diagnosed with opiate-induced leukoencephalopathy based on the characteristic imaging findings and positive urine drug screen.

The treatment for this disease is supportive while allowing the drug to clear, and the patient received this supportive care while in the hospital. He was then transferred to an inpatient rehabilitation facility and then later to a substance abuse program. Physical exam following discharge revealed residual right hemiparesis and neuropathic pain in the distal right lower extremity which was managed on gabapentin. As is the case with stroke and trauma, this patient's long-term outcome depends on the extent of the brain injury that the patient endured.

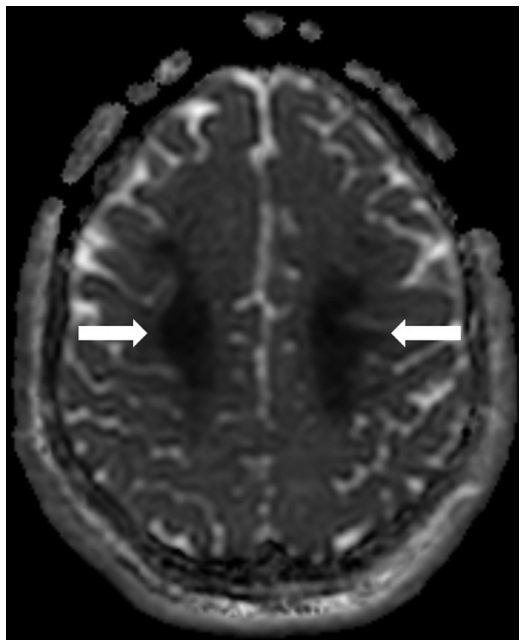

Fig. 3 – Apparent diffusion coefficient correlation shows hypointense signal (arrows) confirming restriction of diffusion throughout the affected white matter.

Discussion

Given the current opioid epidemic in this country, it is important that pediatric radiologists recognize the findings of

toxic leukoencephalopathy, specifically opioid-induced toxic leukoencephalopathy. Differential considerations include stroke, metabolic disorder, infection, demyelinating process, and hypoxic ischemia. The confluence of signal abnormality and diffusion restriction in the central white matter seen in this patient are much more concordant with opiate leukoencephalopathy. Subsequent encephalomalacia is common and was observed in this patient as well.

While most deaths due to opiate toxicity are in the adult population, there are a growing number of pediatric patients who are overdosing from opiates and ultimately dying. The mechanism of opioid-induced leukoencephalopathy has not been fully elucidated, but these findings are more common in heroin inhalation ("chasing the dragon") [1–2]. Pathologically, this produces spongiform degeneration of the involved white matter with vacuolization of the oligodendrocytes [3]. To date, a valid incidence of this disorder in the pediatric population has yet to be obtained, as pediatric opiate-induced leukoencephalopathy is described sparingly in case reports without specific epidemiologic investigation.

Characteristic MRI findings may prompt the diagnosis in an unresponsive child and provide information useful to guide therapy.

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