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Case Report

Heroin-induced leukoencephalopathy: Chasing the imaging findings ☆

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ABSTRACT

We present a case of a 29-year-old male who was brought into the hospital due to unresponsiveness and found to have heroin inhalational leukoencephalopathy (HLE). HLE is one component of a broad spectrum of opioid encephalopathies that is associated with heroin inhalation and other opioids. There is considerable overlap of HLE with other toxic and hypoxic-ischemic encephalopathies; however, the specific territories of brain involvement help distinguish it from other cerebral insults. The goal of this study is to help elucidate the findings of HLE and compare these findings to other toxic and hypoxic-ischemic encephalopathies.

Introduction

As of 2021, Opioid and opiates drug use affect 4.4% of the US population and 5.8% of the global population. Opioids and opiates (natural opioids such as heroin, morphine, and codeine) accounted for 75.4% of all drug related overdose deaths in the US in that year alone [1,2].

Opioids are widely known for its therapeutic properties due to its sedative and analgesic properties; however, there are a multitude of side effects related to opioids that range from mild symptoms such as nausea and vomiting to severe cases of hypoxia, pulmonary edema, and cyanosis. The opioid overdose triad is a term used to describe the typical presenting symptoms of opioid overdose: pinpoint pupils, respiratory depression, and decreased level of consciousness. Additionally, opioid tolerant patients fail to develop tolerance to the loss of hypoxic stimulus, which is thought to contribute to the higher risk of death from overdose [3,4]. Opioid overdoses can therefore produce both toxic and hypoxic encephalopathies and by extension, radiologic findings.

Opioid neurotoxicity comprises a broad pathophysiologic spectrum of findings, which include heroin vapor inhalation leukoencephalopathy (HLE, or “chasing the dragon” syndrome), Pediatric Opioid use-associated neurotoxicity with cerebellar edema (POUNCE) syndrome, Opioid-associated

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amnestic syndrome, and CHANTER (cerebellar, hippocampal, and basal nuclei transient edema with restricted diffusion) syndrome.

Case report

A 29-year-old male with a past medical history of asthma, hypertension, and type II diabetes mellitus, and CKD stage IIIa was found unresponsive. After administration of Narcan, seizure activity was noted. Pinpoint pupils were found on exam. Labs were notable for troponin of 837 ng/L (0 – 22 ng/L), respiratory acidosis with a pH of 7.15 (7.32 – 7.43), pCO2 elevated at 85 mmHg (41 – 54 mmHg), lactate elevated at 5.3 mmol/L (0.6 – 1.4 mmol/L), and a urine toxicology positive for opiates. Initial CT of the head showed ill-defined hypodensities in the subcortical white matter, bilateral basal ganglia and cerebellar hemispheres, suspicious for acute infarcts in the setting of drug toxicity (Fig. 1). Follow up CT head approximately 12 hours later showed diffuse cerebral edema with partial effacement of fourth ventricle and basilar cisterns as well as mild tonsillar and uncal herniation. MRI was then obtained which revealed restricted diffusion in the cerebral white matter, caudate nuclei, internal capsules, lentiform nuclei, hippocampi, vermis, cerebellar hemispheres with relative sparing of the U fibers (Fig. 2). Petechial hemorrhages were noted within the basal ganglia on GRI sequences (Fig. 3). Restricted diffusion was also demonstrated in the basal ganglia, hippocampi, and cerebellar hemispheres with petechial hemorrhage within the cerebellum, not shown. MRA revealed no hemodynamically significant stenosis in the intracranial or extracranial arterial systems. The combination of findings in addition to positive drug screening were compatible for heroin

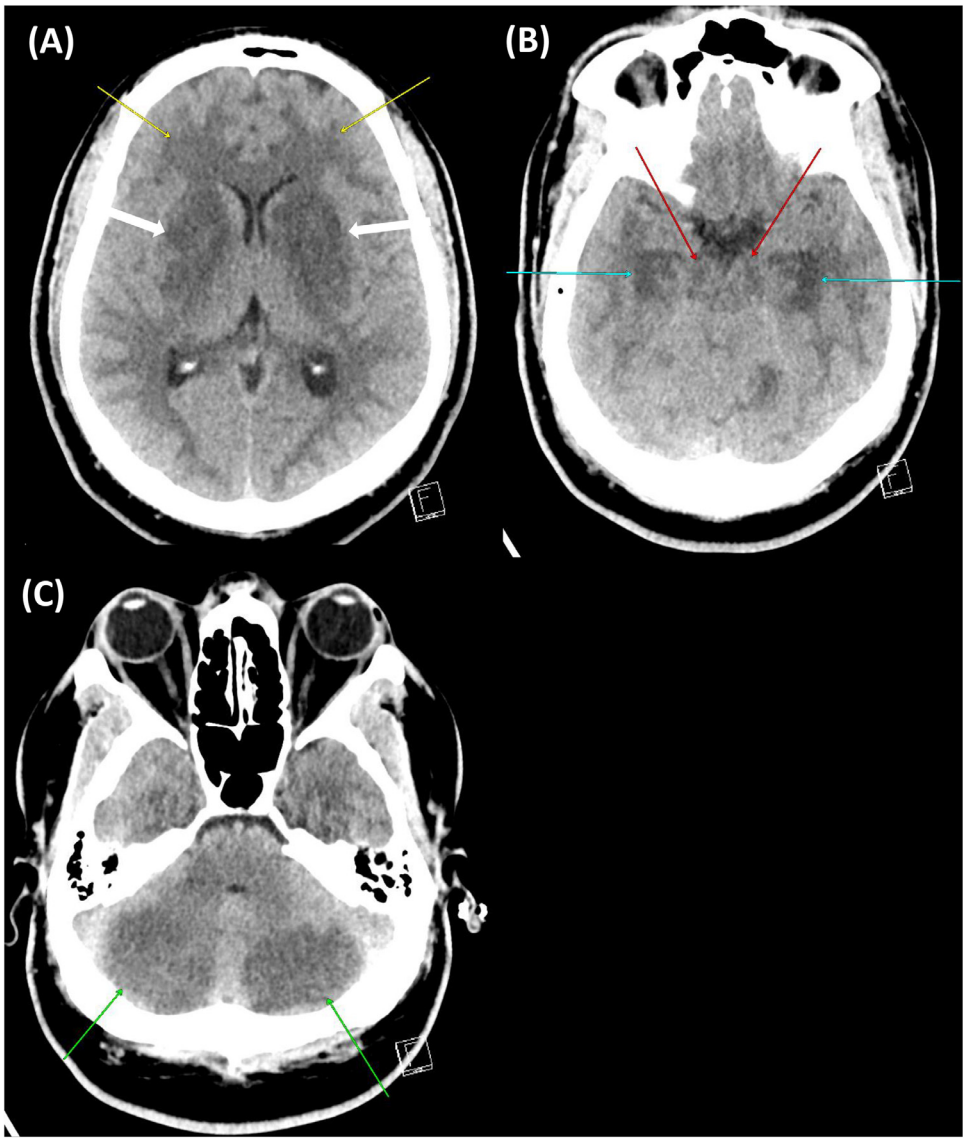

Fig. 1 – CT Head Axial images at the level of the lateral ventricles (A) and cerebral peduncles (B) and cerebellum (C) demonstrate hypodensity of the deep and subcortical white matter (yellow arrows), basal ganglia (white arrows), cerebral peduncles (red arrows), hippocampi (cyan arrows), and cerebellar hemispheres (green arrows).

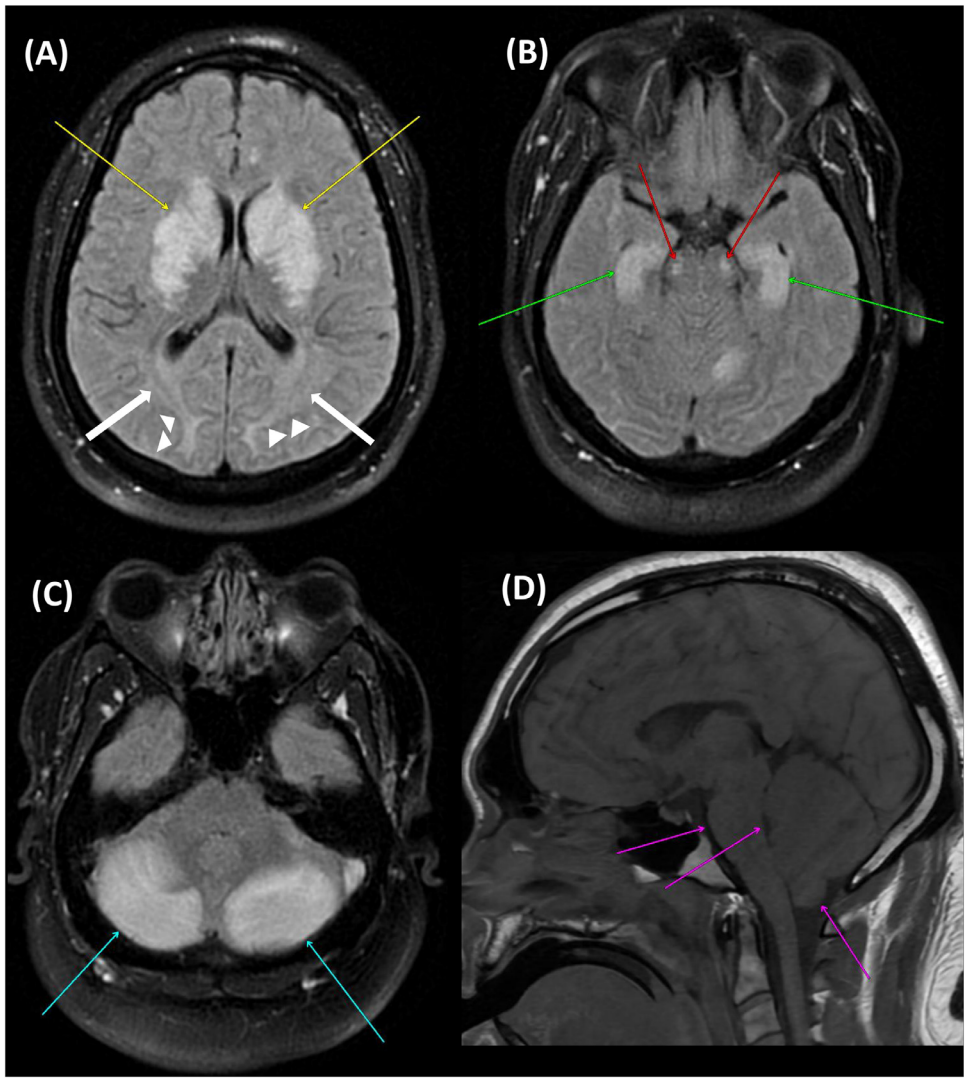

Fig. 2 – Axial T2 FLAIR Fat Saturated Images at the level of the lateral ventricles (A), cerebral peduncles (B), and cerebellum (C) as well as sagittal T1 (D) MR of the brain shows T2/FLAIR hyperintensity of the basal ganglia (yellow arrows), corticospinal tracts (red arrows), hippocampus (curved yellow arrows), and cerebellar hemispheres (cyan arrows) as well as the subcortical and deep white matter (white arrows) with sparing of the U-fibers (white arrowheads). Note tonsillar herniation and effacement of the fourth ventricle and prepontine cistern (magenta arrows).

inhalational leukoencephalopathy or "Chasing the Dragon" syndrome. The patient was admitted to the medical intensive care unit and is continuing to receive supportive care.

Discussion

Heroin inhalational leukoencephalopathy (HLE), or "chasing the dragon" syndrome is defined by the neurologic sequelae of heroin use, most notably by heating heroin powder over an aluminum foil which causes the heroin to melt into a reddish-brown liquid that emits a white smoke, resembling a dragon's tail. "Chasing" refers to inhaling the smoke through a tube. Tin/aluminum toxicity or mitochondrial dysfunction was initially suspected to produce the neurologic symptoms of HLE, but there has been no definitive supporting evidence. Histologically, spongiform degeneration of the white matter, sparing of the subcortical U-fibers, and vacuolar formation in the oligodendroglia and myelin sheaths are characteristic. Radiologically, HLE is defined as areas of diffuse symmetrical white matter T2/FLAIR hyperintensities in the cerebellum, posterior cerebrum with sparing of the U-fibers, corticospinal tract, and the hippocampus. Restricted diffusion may be seen in areas of T2/FLAIR hyperintensity. In more severe cases, involvement of the anterior cerebrum has been documented. Magnetic resonance spectroscopy if available will demonstrate reduced N-acetyl aspartate, reduced choline, and elevated lactate in the involved parenchyma [5].

The differential diagnosis of HLE includes hypoxic ischemic encephalopathy, CHANTER syndrome, Pediatric Opioid use-associated neurotoxicity with cerebellar edema (POUNCE) syndrome, and Opioid-associated amnestic syndrome. A mixed pattern hypoxic-ischemic encephalopathy

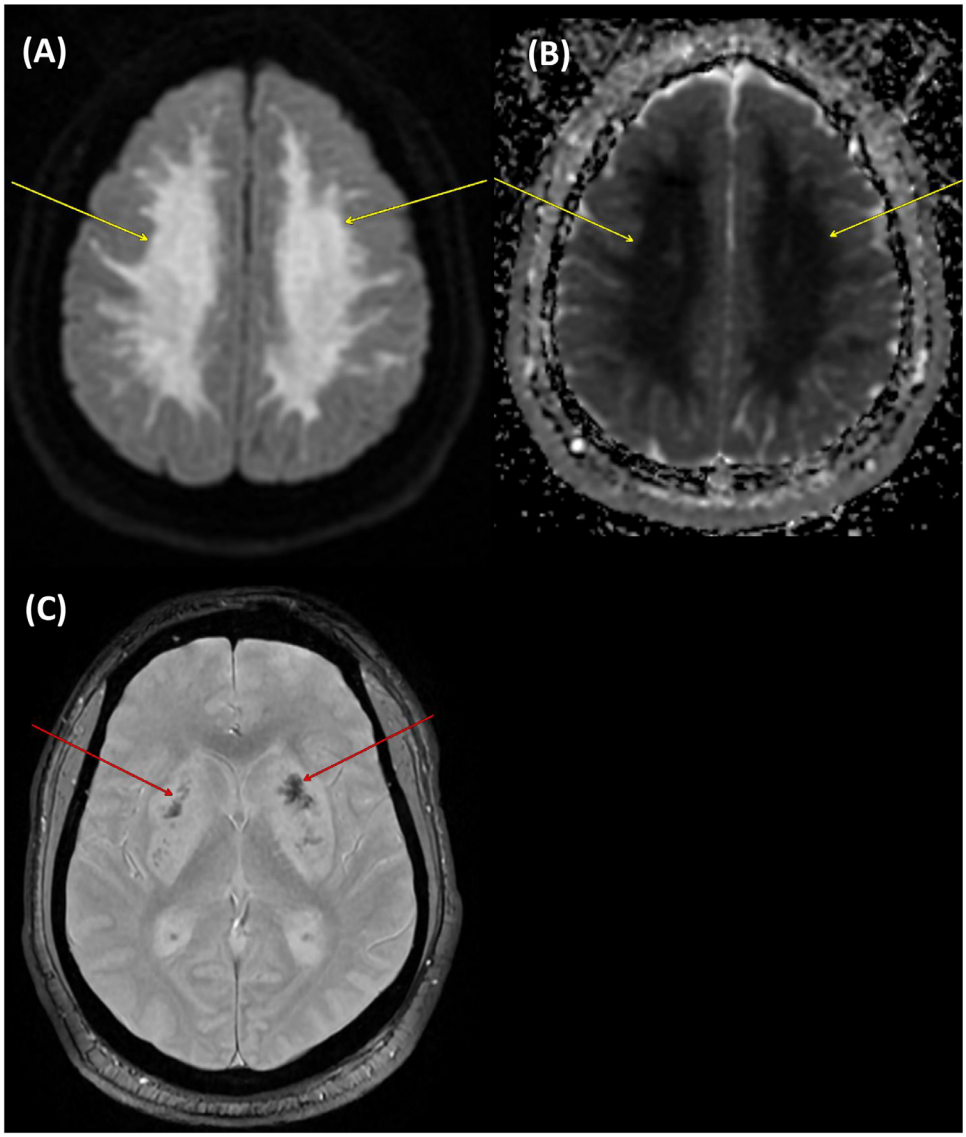

Fig. 3 – Axial DWI (A), ADC (B), and GRE (C) MR of the brain shows diffusion restriction within cerebral white matter (yellow arrows) and areas of petechial hemorrhage within the basal ganglia (red arrows).

can present with similar imaging findings as HLE, however subcortical sparing and lack of thalamic findings, as seen in this patient is not typical [6]. Like HLE, CHANTER syndrome affects the hippocampi, cerebellum, and basal ganglia, however diffuse cortical involvement is not seen. Opioid associated amnestic syndrome affects the hippocampus, with many findings overlapping with CHANTER syndrome, but subcortical hyperintensity is not usually seen. POUNCE syndrome preferentially affects the cerebellum with or without supratentorial white matter involvement, but the age of the patient and basal ganglia excludes this diagnosis.

Interestingly, diffuse involvement of basal ganglia was noted, and has only been documented in a few cases of opioid overdose [6]. We suspect that some component of hypoxic encephalopathy or CHANTER syndrome is involved, as the hippocampus, deep gray matter nuclei, and cerebellum are preferentially affected in the early stages. Considering the profound respiratory depression caused by opioid overdose, a combination of toxic and hypoxic encephalopathy may be possible. Treatment of HLE as with most opioid related toxicities is largely supportive, with possible neurosurgical intervention for cerebral or cerebellar herniation. Long term rehabilitation is often required for these patients, as permanent impaired functionality is common even in mild cases.

Patient consent

All relevant patient information was anonymized and the manuscript only includes non-identifiable images. The informed consent for publication has been acquired.

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