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CASE REPORT

A Rare Case of Heroin-induced Leukoencephalopathy after Intravenous Abuse in a Person Living with Human Immunodeficiency Virus

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Opioid intoxication can lead to varied neurologic complications including delirium, sedation, cognitive impairment, hallucinations, seizures, and myoclonus. One of the rare neurological impairments is toxin-induced leukoencephalopathy. Heroin-induced leukoencephalopathy (HLE) is a rare but critical neurological disorder seen in heroin users which carries high morbidity and mortality. It most commonly occurs with inhalational use of heroin also known as chasing the dragon syndrome. We report a 26-year-old person living with human immunodeficiency virus who presented with altered sensorium after intravenous heroin use. On examination, the patient was found to have dysautonomia with tachycardia and flushing of face and generalized rigidity of limbs with high-grade fever. Laboratory results were consistent with kidney and liver dysfunction with underlying rhabdomyolysis. An extensive Infectious workup turned out to be negative. Magnetic resonance imaging brain changes were consistent with toxin-induced leukoencephalopathy. The patient was managed conservatively with antioxidants and supportive care. This case highlights the clinical and radiological characteristics seen in HLE.

Key words: Case report, leukoencephalopathy, magnetic resonance imaging, opioid intoxication

INTRODUCTION

Opioid-induced leukoencephalopathy is a rare and serious complication that leads to long-lasting or even permanent neurologic impairments. It is characterized by typical magnetic resonance imaging (MRI) abnormalities. Among the various routes of abuse, the inhalation route is most commonly associated with heroin-induced leukoencephalopathy (HLE) seen in 60% of cases while the intravenous route constitutes about 30% of cases.[1] There is a difference in the presentation of patients with intravenous and inhalational abuse. Mental status changes, urine/fecal incontinence, and mutism are seen with intravenous use while cerebellar symptoms are commonly seen with inhalational use. These differences arise due to differences in the region of the brain involved.

CASE REPORT

A young male in his late 20s, presented to the medical emergency with gradually worsening sensorium, 1 day following intravenous heroin use and high-grade fever of around 104°F–105°F. He had a history of heroin abuse disorder for 4 months and was diagnosed case of human immunodeficiency virus for which he was on antivirals for the last 6 weeks. There was no history of vomiting, abnormal body movements, diarrhea, headache, jaundice, chest pain, or use of alcohol. On examination, the vitals of the patient were stable with fluctuating blood pressure between 110/70 and 150/90 with tachycardia (pulse rate – 130) and sweating and flushing of the face. On central nervous system examination-pupils were dilated and the plantar was downgoing. Neck rigidity was absent and the Glasgow Coma Scale was E1V1M1 for which he was intubated. There was symmetrical and generalized rigidity with hyperreflexia in both upper and lower limbs. Fundus examination of the patient was normal. The rest of the systemic examination was unremarkable.

Laboratory abnormalities included acute kidney injury with creatinine of 1.5 mg/dL, aspartate aminotransferase

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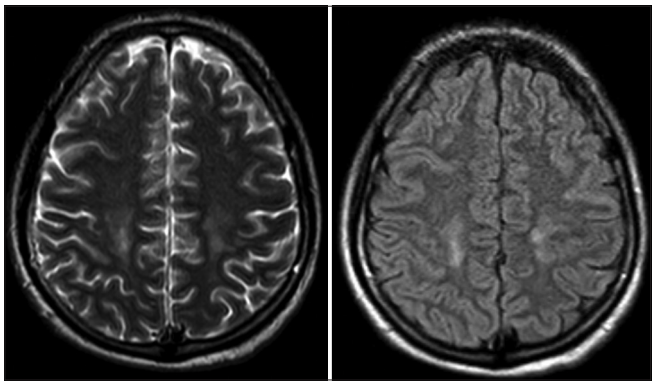

Figure 1: Axial section of T2/fluid-attenuated inversion recovery showing symmetrical bilateral hyperintensities involving the centrum semiovale

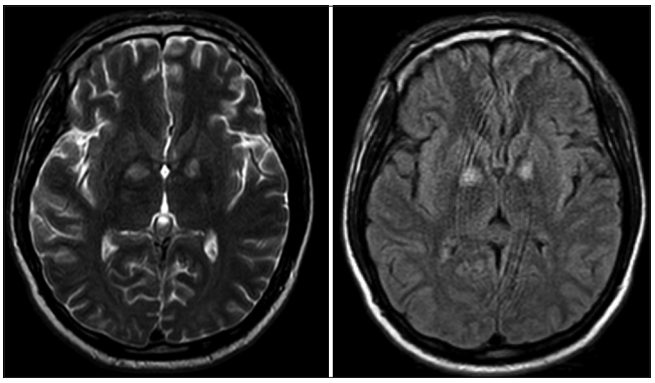

Figure 2: Axial section of T2/fluid-attenuated inversion recovery showing symmetrical bilateral hyperintensities involving the globus pallidum

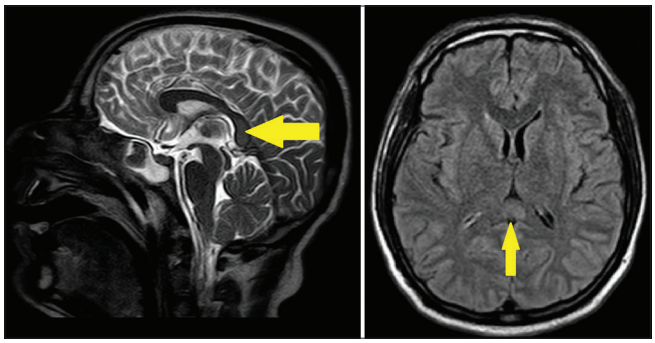

Figure 3: Sagittal section of T2 (horizontal arrow) and axial section of fluid-attenuated inversion recovery (vertical arrow) showing hyperintesity in the splenium of corpus callosum

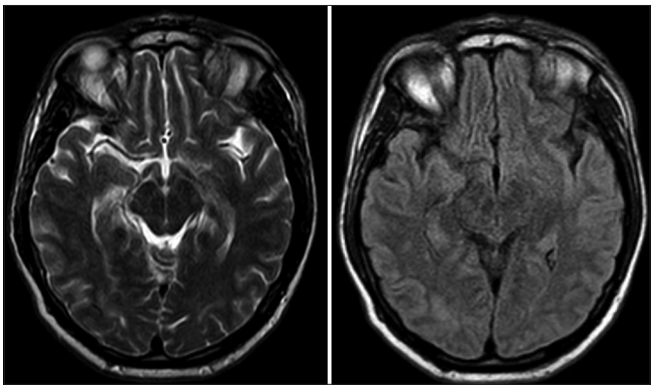

Figure 4: Axial section of T2/fluid-attenuated inversion recovery showing symmetrical bilateral hyperintensities involving the occipital white matter

of 872 U/L, and alanine aminotransferase of 1781 U/L. Hemoglobin was 12 g/dL and total leucocyte count was 13 × 10^9/L. Urine toxicology screen was positive for opioids. Noncontrast computed tomography of the head was done which was normal. Cerebrospinal fluid (CSF) examination showed 2 cells 100% lymphocytic with normal biochemistry (total protein – 26, glucose – 106, and adenosine deaminase – 1.3). Tuberculosis and herpes simplex virus testing in CSF yielded negative results. Transthoracic echocardiography was done to rule out infective endocarditis which was normal. Blood culture and urine culture were sterile with procalcitonin of 0.2. Tropical fever serologies were negative. Workup for hepatitis B and C, toxoplasmosis, and syphilis was negative. Serum levels of Vitamin B12 were adequate. There was also elevated total creatinine kinase and lactate dehydrogenase. Later, contrast-enhanced MRI brain was done after 5 days of admission which showed symmetrical bilateral hyperintensities involving centrum semiovale [Figure 1], globus pallidus [Figure 2], splenium of the corpus callosum [Figure 3] and parieto-occipital white matter [Figure 4] in T2-weighted images (T2WI) and also showing avid diffusion restriction [Figure 5] consistent with changes seen in opioid-induced leukoencephalopathy. Electroencephalogram showed generalized cerebral dysfunction likely encephalopathy. The patient was managed in the intensive care unit with intravenous antibiotics, antioxidants, and Vitamin E. The patient improved gradually and got extubated after 10 days of intubation with residual neurological deficits in the form of impaired memory and concentration and rigidity of limbs with dysarthria and urinary and fecal incontinence on follow-up. The patient was being maintained on regular physical rehabilitation. Ultimately, the patient succumbed to illness after 6 months of follow-up.

DISCUSSION

Opioid intoxication leads to neurotoxic effects with possible long-lasting or even permanent neurologic impairments which include inattention, forgetfulness, and changes in personality to dementia, coma, and death. HLE is a rare neurological complication that is most often seen with inhalational use also known as chasing the dragon.[2] Its incidence with intravenous use is unknown. The pathogenesis of HLE is unknown but it is observed that unknown toxins produced after heating heroin

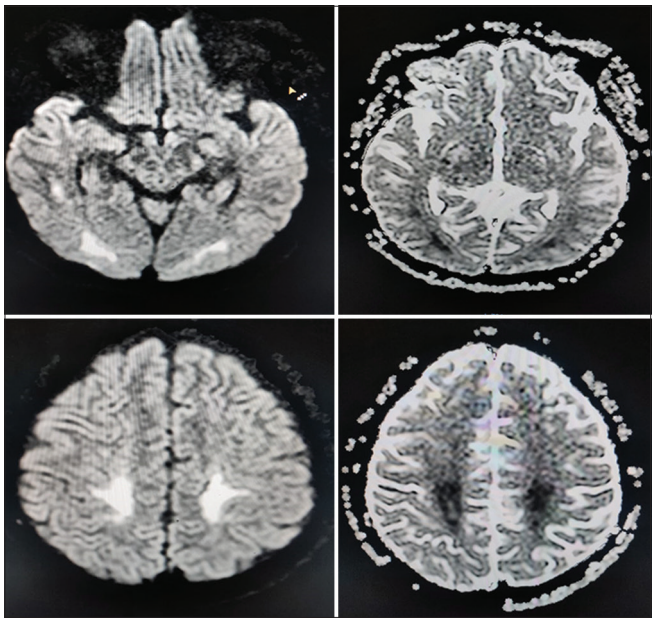

Figure 5: Axial section of diffusion-weighted imaging showing avid diffusion restriction in the occipital white matter and centrum semiovale with corresponding low signal on apparent diffusion coefficient

are implicated in the condition. Furthermore, mitochondrial dysfunction and inhibition of tetrahydrobiopterin metabolism play a role in the development of HLE.[3] The tetrahydrobiopterin is required for the conversion of tyrosine to levodopa. Thus, inhibition of tetrahydrobiopterin metabolism leads to parkinsonian symptoms seen in this disease there are three stages of clinical severity in HLE. Severe syndrome is characterized by impaired alertness with generalized motor impairment and language disorder as seen in our index case. There is a difference in the presentation of patients with intravenous and inhalational abuse. Mental status changes, urine/fecal incontinence, and mutism are seen with intravenous use while cerebellar symptoms are commonly seen with inhalational use.[1] These neurological deficits are accompanied by MRI abnormalities which differ with the method of administration of heroin. Typical MRI abnormalities seen with inhalational use include diffuse symmetrical white matter hyperintensities on T2WI and fluid-attenuated inversion recovery (FLAIR) images in the cerebellum, posterior limb of the internal capsule, and posterior cerebrum. While with intravenous use, MRI abnormalities include symmetrical white matter hyperintensities in T2WI and FLAIR in the periventricular region, splenium of corpus callosum, frontoparietal white matter, and centrum semiovale with sparing of cerebellum and brainstem.[4] Thus, cerebellar ataxia and dysarthria are the predominant symptoms seen with heroin inhalation as cerebellum is involved. While altered mental status, mutism and incontinence are seen with intravenous use due to frontoparietal white matter involvement. The treatment is mainly supportive with antioxidants with need of neurosurgical intervention in cerebellar herniation. Furthermore, the severe syndrome is usually seen subacutely within a period of 4–6 weeks.[5] Only three cases have been reported in the literature about the acute presentation with inhalational heroin abuse.[5] However, no case has been reported of acute presentation with intravenous use. This case is probably the first one. The prognosis of this disease is very poor with patients who survive often need prolonged physical and drug rehabilitation. Thus, a possibility of HLE should be kept in a patient presenting with altered sensorium after intravenous opioid use.

Declaration of patient consent

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its amendments. The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Data availability statement

The data that support the findings of this study are available from the corresponding author, Luthra V, upon reasonable request.

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Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Cheng MY, Chin SC, Chang YC, Wu T, Lim SN, Hsieh HY, *et al.* Different routes of heroin intake cause various heroin-induced leukoencephalopathies. J Neurol 2019;266:316-29.
2. Krinsky CS, Reichard RR. Chasing the dragon: A review of toxic leukoencephalopathy. Acad Forensic Pathol 2012;2:67-73.
3. Heales S, Crawley F, Rudge P. Reversible parkinsonism following heroin pyrolysate inhalation is associated with tetrahydrobiopterin deficiency. Mov Disord 2004;19:1248-51.
4. Özütemiz C, Roshan SK, Kroll NJ, Benson JC, Rykken JB, Oswood MC, *et al.* Acute toxic leukoencephalopathy: Etiologies, imaging findings, and outcomes in 101 patients. Am J Neuroradiol 2019;40:267-75.
5. Achamallah N, Wright RS, Fried J. Chasing the wrong dragon: A new presentation of heroin-induced toxic leukoencephalopathy mimicking anoxic brain injury. J Intensive Care Soc 2019;20:80-5.