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Chasing The Dragon

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Chasing the dragon, a case of leukoencephalopathy associated with heroin inhalation

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Abstract

Although rare, toxic leukoencephalopathy (TLE) associated with heroin inhalation has been reported. “Chasing the dragon” may lead to progressive spongiform degeneration of the brain and presents with a large range of neuropsychological sequelae. This case is an example of TLE in a middle-aged white male with a history of polysubstance abuse. He presented with a three week history of progressive neuropsychological symptoms, including abulia, bradyphrenia, hyperreflexia, and visual hallucinations. He was initially suspected to have progressive multifocal leukoencephalopathy, however, JCV PCR was negative. MRI showed diffuse abnormal signal in the white matter, extending into the thalami and cerebral peduncles. Brain biopsy was performed, which revealed spongiform degeneration, and a diagnosis of TLE was made. The patient was then transferred to a skilled nursing facility. Clinical suspicion based on a thorough history and clinical exam findings is paramount in recognition of heroin-associated TLE. Although rare, heroin-inhalation TLE continues to be reported. As ‘chasing the dragon’ is gaining popularity among drug users, it is important for clinicians to be able to recognize this disease process.

Keywords

Opioid, Addiction, Heroin, Leukoencephalopathy

Introduction

Toxic leukoencephalopathy (TLE) associated with heroin abuse was first described in 1982.¹ “Chasing the dragon” is a method of heroin vapor inhalation in which a small amount of heroin powder is placed on aluminum foil, which is then heated by placing a match or lighter underneath. The white powder becomes a reddish-brown gelatinous substance that releases a thick white smoke resembling a dragon's tail. The fumes are “chased” or inhaled through a straw or small tube.² As inhalation is becoming more common amongst heroin users attempting to avoid complications of parenteral heroin use, clinical recognition of heroin-induced TLE is important in making the correct diagnosis and preventing unnecessary workup for other conditions.³

Case Presentation

A 37 year old white male accompanied by his wife presented to the emergency department (ED) with altered mental status. His wife reported that he had become progressively confused and disoriented and began having visual hallucinations over the previous three weeks. His past medical history was significant for polysubstance abuse, including heroin, which his wife reported that he had never used via injection and she was unsure if he was still using.

Two and a half weeks prior to his ED visit, he had reportedly overdosed on methadone and required intubation. He initially sought methadone because he had been feeling “exhausted” and obtained it from the street. He was intubated for three days. After extubation, he remained confused for approximately four days, at which time his mental status had improved to baseline and he was discharged. After discharge, he made an appointment with his psychiatrist and was

started on fluoxetine, clonidine, and alprazolam. According to his wife, it was after this appointment that he became confused, disoriented to time and place, and started having visual hallucinations of “shadows” near his child’s bedroom. The patient’s wife then took him back to the ED at the outlying facility, and the decision was made to stop his fluoxetine and decrease the doses of his alprazolam and clonidine. He was then sent home; his wife, however, reported that he showed no signs of improvement and that is when she made the decision to bring him to our facility.

According to the patient’s wife, he did not have any recent history of headaches, seizure-like activity, chemical or carbon monoxide exposure, travel, or sick contacts. His past medical history included polysubstance abuse, type 2 diabetes mellitus, bipolar I disorder, and attention-deficit-hyperactivity disorder. He has had no previous surgeries and his family history was noncontributory. His wife reported that he smoked one and a half packs of cigarettes per day and he drove a truck for work. Medications at the time of admission included lisdexamphetamine, clonidine, and alprazolam. Physical exam was significant for orientation only to himself, bradykinesia, bradyphrenia, and abulia, as well as hyperactive reflexes, inability to walk on his heels or toes, and incontinence. His speech was slow and monotone, he was easily distractible, and he scored 9/30 on mini-mental status exam.

A broad differential diagnosis was generated, including progressive multifocal leukoencephalopathy (PML), toxic brain injury, viral encephalitis, autoimmune encephalitis, spongiform encephalitis, and anoxic-ischemic brain injury. Initial lab workup, including CBC, CMP, LP, UA, ammonia, TSH, cortisol, folate, and B12 levels were within normal limits, and a urine drug screen was positive for benzodiazepines and amines, consistent with the patient’s prescribed medications. HIV testing, JC virus PCR, and herpes simplex PCR tests were negative.

Neurology and cardiology were consulted and performed a full stroke and seizure workup, including an EEG, TEE, EKG, and hypercoagulability studies, all of which were within normal limits. A non-contrast CT scan of the brain showed bilateral globus pallidus infarcts, which were interpreted as older lesions. An MRI of the brain (Image 1) revealed bilateral globus pallidus infarcts and diffuse abnormal signal noted on T2 weighted and FLAIR images throughout the white matter of the cerebral hemispheres with additional subtle signal abnormality extending into the thalami and cerebellar peduncles.

The MRI findings, in combination with the negative lab work, were strongly suggestive of a white matter toxic encephalopathy. It was noted, however, that JC virus PCR carries a sensitivity of 72-92% and the gold standard for diagnosing PML is with brain biopsy.⁴ Moreover, brain biopsy may aid in ruling out other pathologies. The patient underwent stereotaxic brain biopsy, which showed spongiform degeneration, suggestive of TLE. He was then transferred to a skilled nursing facility.

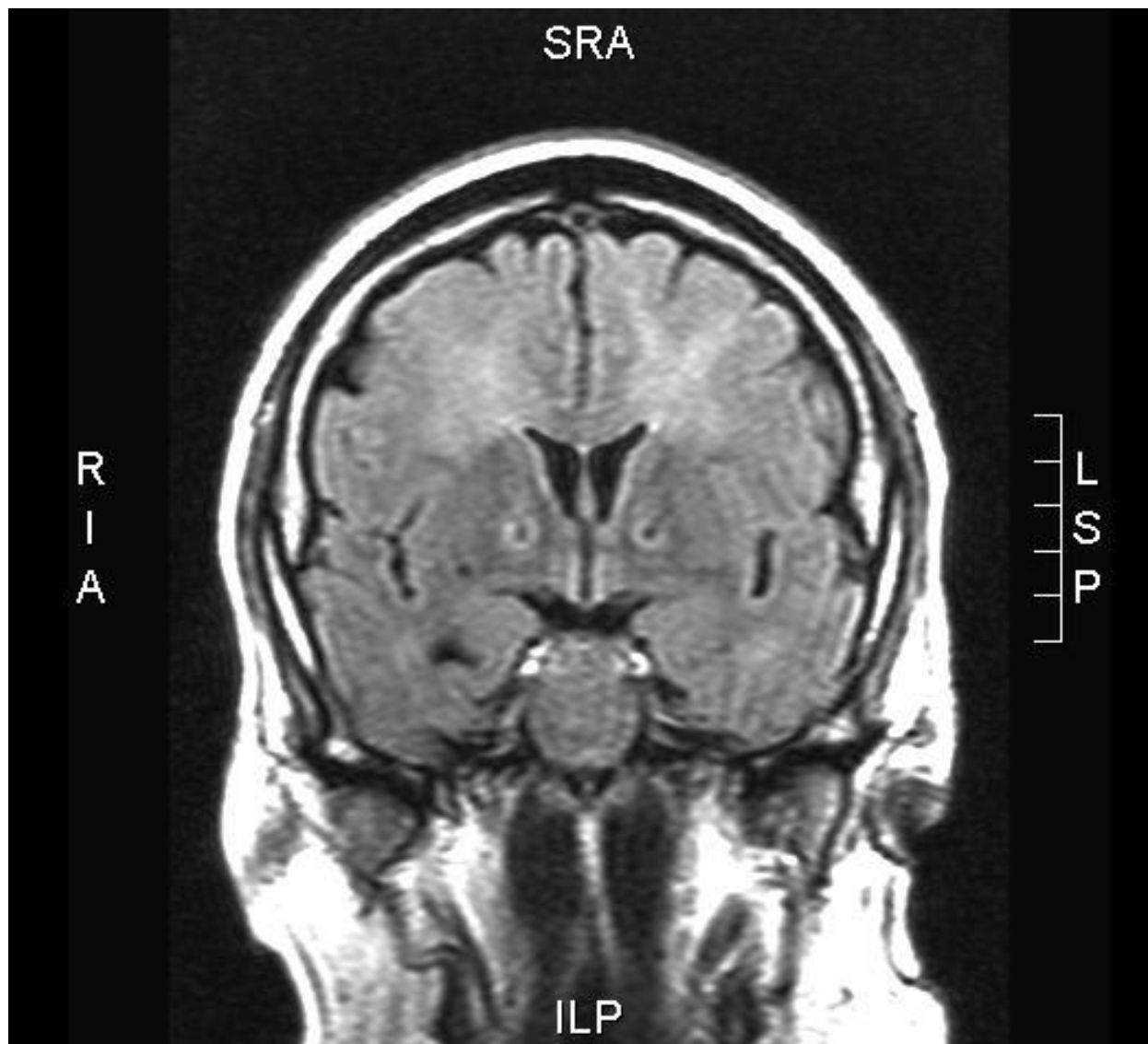


Image 1: Brain MRI FLAIR showing diffuse leukoencephalopathy and bilateral globus pallidus infarcts.

Discussion

Since its first description, there have been several case reports of heroin-inhalation TLE. This continues to be a problem, not only locally but abroad.⁸ Although the mechanism of neurologic injury is unknown, it has been hypothesized that unidentified substances in the heroin pyrolysate (the product of chemical change caused by heating) may be responsible.¹ Some suggest the mechanism is related to inhibition of tetrahydrobiopterin which is required for conversion of tyrosine to levopa and tryptophan to serotonin. In the initial stage of the disease process, patients typically present with pseudobulbar speech, cerebellar ataxia, restlessness, apathy, abulia, and bradyphrenia, with pseudobulbar speech and cerebellar ataxia being the two most frequent presentations. In approximately half of patients with initial symptoms, the disease progresses to an intermediate stage over a period of two to four weeks. Most frequently noted symptoms

during the intermediate stage include pyramidal tract signs, hyperactive reflexes, tremors, myoclonus, spastic hemiplegia or quadriplegia, chorea, and athetosis. Approximately one-fourth of patients will progress to a terminal stage before death. These patients most frequently exhibit stretching spasms, hypotonic/areflexic paresis, akinetic mutism, and central pyrexia.¹ Though anoxic encephalopathy is a possibility secondary to overdose and intubation, it is unlikely as most of the symptoms of anoxic injury are not reversible and our patient returned to baseline function.

The diagnosis of heroin-inhalation TLE is clinical, making a thorough social history and recognition of characteristic features of the utmost importance. While other diagnostic tests (i.e. brain MRI, brain biopsy) are usually performed, they are more useful in ruling out other pathologies and are not necessary for making the diagnosis, although they may be supportive.^{1,3} In heroin-inhalation TLE, brain MRI typically shows diffuse, symmetrical white matter hyperintensities on T2 and FLAIR images in the cerebellum, posterior cerebrum, and posterior limbs of the internal capsule. Brain biopsy, when performed, usually shows spongiform degeneration with relative sparing of subcortical U-fibers.³ There is no treatment for heroin-inhalation TLE, and mortality ranges between 23 and 48% of patients affected, with the most frequent cause of death being respiratory failure.^{1,5} In rare cases, mild clinical improvement in symptoms has been noted with antioxidant therapy including coenzyme Q10, vitamin E, and vitamin C.^{3,6} Surviving patients should be referred to physical and drug rehabilitation programs. In our patient, we believe that the further deterioration was related to the progression of the disease TLE. The fact that heroin (along with carbon monoxide, methamphetamine, inhalants, etc.) can lead to TLE⁹ should embolden the provider to maintain a high index of suspicion and broad differential in patients with similar presentations.

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