

Case Report of Toxic Leukoencephalopathy Induced by Heroin Abuse

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(Submitted: 29 March 2022 – Revised version received: 18 April 2022 – Accepted: 10 May 2022 – Published online: 26 September 2022)

Abstract

Toxic leukoencephalopathy is a rare disease that may present with different signs and symptoms, it requires combination of clinical signs and symptoms along with radiographic evidence to reach to a diagnosis. We report the case of a young man who presented with decreased level of consciousness and found that he took an overdose of heroin prior to admission, after investigations the patient was found to have toxic leukoencephalopathy. Its uncommon disease, however, it should be in the differential diagnosis of any patient coming with some neurological and behavioral changes along with substance abuse, mainstay treatment is supportive.

Introduction

Toxic leukoencephalopathy is a rare condition that needs both clinical symptoms and signs along with radiological evidence of changes in white matter and it can result from a variety of conditions.^{1,2} The cause of these changes could be infectious, or side effect of chemotherapy or other immunosuppressive therapy or drug overdose.³ Major drugs that are associated with toxic leukoencephalopathy are ethanol, cocaine, toluene, methylenedioxymethamphetamine or ecstasy and heroine.⁴ The patients could present as simple as inattention to severe symptoms such as coma or even death.⁵ The triad of exposure, neurological and behavioral changes along with classic Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) findings should raise the suspicion of toxic leukoencephalopathy, in MRI there will be hyperintensity of the cerebral white matter and on CT we will find diffuse hypodensity of the white matter.⁶ The clinical outcome and prognosis depends on many factors such as the cause, the type of drug ingested and the amount, in addition to that the duration of exposure and underlying diseases, the outcome could vary from complete reversibility to death.⁷

We are reporting a case of young man was admitted initially as a case of encephalitis and was treated accordingly, however, this patient was found to have toxic leukoencephalopathy caused by heroin overdose.

Case Report

A 24-year-old gentleman known to have heroin use disorder based on Diagnostic and Statistical Manual of Mental Disorders (DSM-V) criteria. He is using heroin through intravenous route since the age of 13 years. He was admitted several times in addiction unit for detoxification and rehabilitation during the past years. He was able to abstinence from heroin abuse for 7 months. He was not on methadone and he just relapsed before this admission. He was admitted with heroin overdose after he took high dose of 250 ml of heroin which was mixed with other substances intravenously, which is considered as a high dose, hence, required intensive care unit (ICU) admission and intubation. The patient presented unconscious, he had pinpoint pupils and he was given several doses of naloxone. The patient was treated in ICU for quite some time, as he developed aspiration pneumonia, sepsis, and rhabdomyolysis, therefore, he was treated with antibiotics and other supportive measures.

The patient underwent CT of the brain the next day of admission which showed: "Plain CT head showed symmetrical bilateral cerebral white matter extensive and confluent hypodense areas with involvement of corpus callosum". Since the patient was hemodynamically unstable to go for MRI brain, he was treated for the possibility of meningoencephalitis, he was started on Ceftriaxone and acyclovir after obtaining cerebrospinal fluid (CSF) sample, which turned out to be negative. However, the patient was not improving initially, hence, the antibiotics were upgraded to Meropenem.

After that patient is extubated, he was hemodynamically stable and was shifted from intensive care unit (ICU) to a regular ward. During his stay, he was delirious and agitated for few days initially, then he responded well to haloperidol injections. Later, he expressed guilty feelings and dysphoria regarding his relapse. His examination showed that he does have impaired short-term memory and he has generalized reduction in power and strength which improved with assistance and physiotherapy.

The patient underwent MRI of the brain later on which showed: **Figure 1A** and **1B** shows Axial FLAIR MR Images show confluent symmetric hyperintensity in the periventricular and supraventricular cerebral white matter with involvement of the corpus callosum. However, there is sparing of the subcortical white matter. **Figure 2A** and **2B** shows Axial DWI b1000 show high signal intensity in the symmetric areas of FLAIR hyperintensity involving the periventricular and supraventricular white matter as well as the corpus callosum. Those areas correspond with high signal intensity on the ADC map (not shown). After excluding all other possibilities and with the mentioned MRI findings, the patient was diagnosed to have Toxic Leukoencephalopathy.

Later, the patient was treated with supportive measures, hence, his signs and symptoms improved, therefore, he was discharged home with a referral letter to rehabilitation center. Verbal consent was given by the patient to write this case report and publish it.

Discussion

Toxic leukoencephalopathy is an uncommon condition, there should be high index of suspicion if a patient presented as acute encephalopathy with substance ingestion. However, it should be differentiated from other conditions with similar

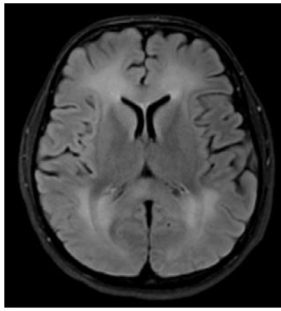


Figure 1A

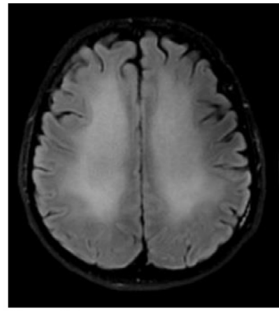


Figure 1B

Fig. 1 Figure 1A and 1B shows Axial FLAIR MR Images show confluent symmetric hyperintensity in the periventricular and supraventricular cerebral white matter with involvement of the corpus callosum. However, there is sparing of the subcortical white matter.

presentation, because its highly reversible by removal of the cause and supportive measures.¹ When there is suspicion of leukoencephalopathy caused by toxins, detailed history should be taken from the patient and urine toxicology screen should be done as well. A careful psychiatric history also need to be taken especially if there is a history of suicidal attempt as one of the causes of Toxic leukoencephalopathy is carbon monoxide poisoning as it's a very potent leukotoxic agent and one of the most important history information is to document change in mental status of the patient, which is very important in diagnosing toxic leukoencephalopathy.⁵

Heroin leukoencephalopathy has been described as "Chasing the dragon syndrome" either by inhalation or intravenous use of heroin. In the literature, its noticed that heroine leukoencephalopathy is associated with 23% mortality, however, it is under recognized.⁸ The major cause of heroine being neurotoxic is that it has high affinity to bind to mu receptors specialty in the cerebellum and limbic system. There are other hypothetical pathophysiological mechanisms of the cause of toxic leukoencephalopathy, one of them is that hypoxia may have synergetic effect with the toxins and vice

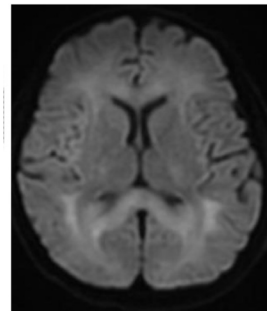


Figure 2A

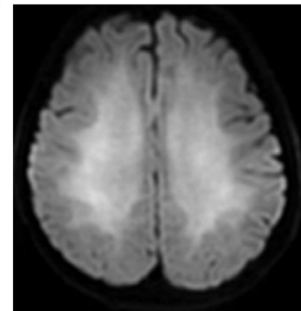


Figure 2B

Fig. 2 Figure 2A and 2B shows Axial DWI b1000 show high signal intensity in the symmetric areas of FLAIR hyperintensity involving the periventricular and supraventricular white matter as well as the corpus callosum. Those areas correspond with high signal intensity on the ADC map (not shown).

versa. The other mechanism is vascular injury.² Our patient has increased risk for heroin overdose and its associated neurotoxicity which is attributed to loss of tolerance after long period of abstinence and then injecting high dose of heroin while relapsed.

The MRI findings of toxic leukoencephalopathy due to heroin use shows symmetrical cerebellar white matter, posterior cerebral white matter in the occipital lobe along with posterior limb of internal capsule.⁴

The treatment of toxic leukoencephalopathy is prevention of using the leukotoxins as the treatment is limited for supportive measures and rehabilitation. Usage of antioxidants such as high dose vitamin C and E has been reported in some studies along with coenzyme Q10.^{2,9}

Conclusion

In conclusion, toxic leukoencephalopathy is a rare disease that is reversible, it should have a high index of suspicion when a patient presented and have a unique feature in the MRI. The treatment is supportive. ■

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