Introduction

A user who smokes heroin places its non-salt freebase form on a foil and heats it from below releasing pyrolysate vapors that resemble a tail of a dragon. The vapor is inhaled through a straw hence coined it “chasing the dragon.”

Toxic leukoencephalopathy (TLE) can be caused by a variety of substances including substances of abuse. Manifestations of TLE can range from inattention and forgetfulness to paresis, coma and death.

There are several mechanisms of cocaine-induced brain injury. It may be vascular, metabolic or immune-mediated. Levimazole, an adulterant in cocaine, has been implicated in causing leukoencephalopathy. Heroin is known to cause brain injury via demyelination and apoptosis of oligodendrocytes. Both are linked to mitochondrial dysfunction.

Case Report

HPI: 52/M found unresponsive in his car with reported ingestion of ½ bag of heroin. He was given 5 mg of naloxone and ventilated with a bag-valve mask. In the ED, he was stuporous but withdrew to noxious stimuli. Reflexes were normal on initial presentation. He had multiple electrolyte abnormalities including significant hyperkalemia, lactic acidosis, transaminitis, respiratory acidosis and AKI secondary to rhabdomyolysis. Toxicology was positive for cocaine but negative for amphetamines. Initial CT scan of the head was negative.

PMH and Social History: Bipolar disorder, Cocaine and Heroin abuse

Hospital course: He remained intubated for 6 days. Post-extubation, despite aggressive supportive treatment for metabolic derangements, he remained nonverbal and stuporous prompting neurology consult. MRI of the brain showed the images below. He was treated supportively with vitamins C and E and was medically stabilized, but was not able to fully recover neurologic function.

Discussion

Heroin induced TLE results when inhalation → mitochondrial dysfunction → demyelination of the white matter tracts. The process involves several stages:
1) motor restlessness and cerebellar dysfunction which occur in a course of days to weeks
2) hyperreflexia, spastic paresis, myoclonic jerks and choreo-athetoid movement
3) extensor posturing, central pyrexia and death

MRI findings include increased T2 and T2-FLAIR in the cerebellar and posterior cerebral white matter, with gray matter sparing.

Cocaine-related TLE manifests as motor and sensory impairment similar to stroke. Cocaine is neurotoxic by several mechanisms:
1) dopamine-mediated generation of free radicals → neuroinflammation and alteration in gene transcription
2) mitochondrial dysfunction → generalized demyelination → loss of axons and vacuolar degeneration

MRI findings include FLAIR and T2 signal abnormalities in periventricular areas of both hemispheres, with less involvement of the cerebellum and brainstem.

Antioxidants such as vitamins C, E and coenzyme Q10 have shown some therapeutic benefits. Neurotoxic-induced vasogenic edema → increased ICP, resulting in irreversible ischemia can be treated with steroids with some evidence of benefit.

Acknowledgement and References

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