Tackling Ataxia After "Chasing the Dragon": The Use of Buspirone in the Rehabilitation of a Patient with Heroin Induced Toxic Leukoencephalopathy

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ABSTRACT

The topic of heroin use is often associated with images of needles and injections, overlooking the fact that heroin is also snorted or smoked in our community. Because of the risk of infection, many users resort to smoking heroin to achieve a high. A potentially fatal consequence of this practice is Heroin Induced Leukoencephalopathy (HIL). This process may be the result of a toxin activated in the heating process and causes damage to the white matter of the brain with associated neurological symptoms. No treatments have yet been proven to alter the disease course. In this case study, we describe a 30 year old male who presented with profound ataxia and dysarthria because of HIL. We discuss the history, physical and radiological findings leading to a diagnosis of HIL and relate these to the pathological findings and pathophysiology in this disease. Finally, we present Buspirone as a potential treatment for the ataxic component of this condition.

KEYWORDS: *heroin, leukoencephalopathy, buspirone*

CASE REPORT

A 30-year old male presented to an Outpatient Neuro-Rehab Department one year after developing increasing falls, weakness, dysarthria and ataxia. He was unable to stand unaided and his mobility was restricted to foot- propelled wheelchair. He had profound dysdiadochokinesia and dysmetria such that he could no longer use the dominant (right) arm for purposeful tasks. His cognition was adequate. Based on his presentation, the patient was questioned and endorsed a history of heroin inhalation.

MRI (figures 1-4) non-contrast studies of the brain were obtained and showed symmetric white matter changes in the parietal lobes, internal capsule, corpus callosum, brainstem and large bilateral lesions in the cerebellum.

HEROIN INDUCED LEUKOENCEPHALOPATHY

"Chasing the Dragon" refers to inhalation of the heated vapour of the free-base form of heroin. The heroin powder is placed on tinfoil above a flame until it liquefies, vaporizes and is then inhaled. The smoking of heroin is reported to have arisen in Shanghai, China in the 1920's using porcelain bowls and bamboo straws.[2] The use of tinfoil in this practice is now known as "Chasing the Dragon" because heroin vapour rising into the air appears like the tail of a dragon.[2]

Table 2. Glossary of terms used to describe the patient's physical exam.[1]

Term	Definition
Ataxia	An inability to coordinate voluntary muscular movements.
Dysarthria	Difficulty in articulating words due to disease of the central nervous system.
Dysmetria	Impaired ability to estimate distance in muscular action.
Dysdiadochokinesia	Impaired ability to make movements exhibiting a rapid change of motion that is caused by cerebellar dysfunction.

A complication of "Chasing the Dragon" is Heroin Induced Leukoencephalopathy (HIL). HIL was first described in the Netherlands in 1982 when forty-seven patients with encephalopathy were found to have a common history of inhaling heroin bought in the same neighbourhood.[3] Since this series, HIL remains a rare condition described in under one-hundred cases. This number may reflect under-recognition. The incidence of HIL has been reported to be increasing, possibly due to increased use of 'chasing the dragon' to avoid the risk of HIV and HCV infection from intravenous use.[4]

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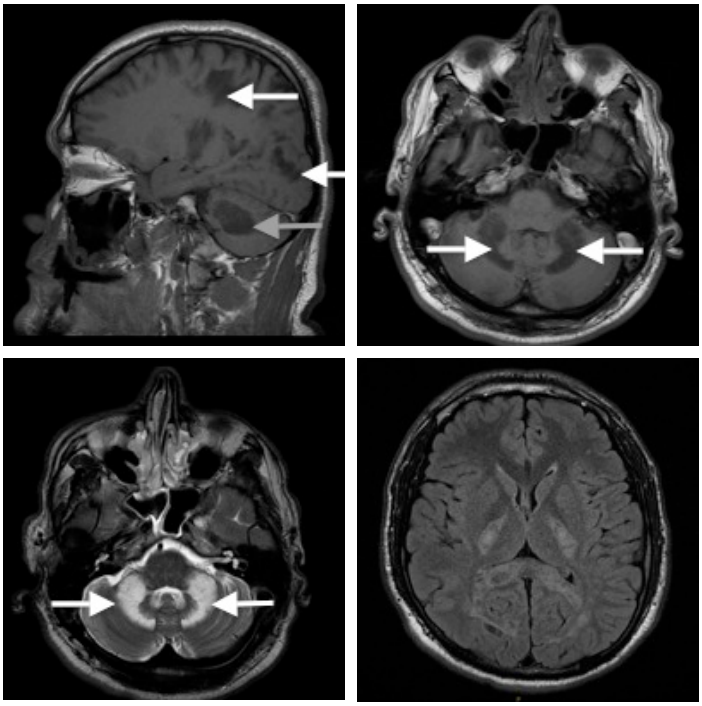

Top Left: Figure 1. T1 saggital image shows prominent white matter changes in the parietal and occipital lobes (white arrows), extending through the internal capsule and into the midbrain, pons and medulla and cerebellum (gray arrow).
Top Right: Figure 2. T1 transverse image shows large bilateral lesions in the cerebellum (white arrows).
Bottom Left: Figure 3. T2 image shows symmetrical white matter hyperintensity in the cerebellum (white arrows).
Bottom Right: Figure 4. T2 flair shows the bubbly cystic appearance of spongiform leukoencephalopathy.

HISTORY AND CLINICAL FINDINGS

HIL is suspected in any patient presenting with neurological impairment and a history of inhaling heroin. The most common signs and symptoms are ataxia, motor restlessness, apathy, behavioral change, aggressive behavior and confusion.[2]

Radiology

HIL affects the white matter.[5] This pattern is reflected best on MRI, where T2 and FLAIR sequencing shows symmetric white matter hyper-intensities in the posterior cerebral and cerebellar white matter, cerebellar peduncles, splenium of the corpus callosum and posterior limb of the internal capsule.[6] Affected white matter is most often found in the posterior regions of the brain, but rarely can affect the frontal regions of the brain as well.

Diagnosis

HIL is a clinical and radiological diagnosis. The history of heroin inhalation associated with neurological impairment is classic. However, other diagnoses like traumatic, ischemic or other toxic brain injury must be considered. MRI images showing characteristic white matter changes are virtually pathognomonic.[6]

Pathology

Gross neuropathological findings include slight softening of white matter and edema. Light microscopy shows spongiform degeneration of white matter with vacuolation of myelin sheaths and scattered axonal bodies in the absence of necrosis, inflammation or vascular abnormality.[5]

Pathophysiology

The history of clusters of patients afflicted with HIL and often the use of heroin obtained from a common source suggest a toxic etiology for the lesions.[7] Also, the pattern of spongiform degeneration of white matter is found in other conditions resulting from exposure to toxins such as tin.[8] Despite this evidence, the pathophysiology of HIL remains poorly understood. A latent period between the toxin exposure and the presentation of symptoms often occurs- a toxicological phenomenon referred to as 'coasting', which has been described for other neurotoxins.[9] The toxic substance may be deposited in the lipid-rich myelin where it remains and is slowly released causing ongoing tissue destruction and progression of symptoms. This may account for negative toxicology results in a number of cases. Also, the toxin may induce a metabolic change that persists following the withdrawal of the offending substance. By the time the patient becomes symptomatic, the toxin is no longer detectable in the blood stream.

Natural History and Prognosis

The natural history of HIL is variable. A latent period of days to months exists between toxic exposure and clinical presentation.[10] Once symptoms develop, they typically progress for 2-3 weeks in the absence of continued exposure, though they have been reported to continue progressing for up to 6 months.[6,11] In the original description of this disease, three clinical stages were defined(Table 1); however, this scale helps prognosticate in only the most severe cases.[2,6]

Table 1. Clinical Stages of HIL

Stage of illness	Feature
Initial	Soft speech Cerebellar ataxia Motor restlessness Apathy
Intermediate	Pseudobulbar lesions Spastic paraparesis Tremor/myoclonic jerks Choreoathetoid movements
Terminal	Stretching Spasms Hypotonic Paresis Akinetic Mutism Cantral Pyrexia Death

Patients may stay in the initial stage, progress into the intermediate stage, or progress through all three stages with one quarter entering the final stage leading to death. Survivors have been described to have slight to dramatic degrees of spontaneous improvement. Therapy with antioxidant therapy including Co-Enzyme Q10 has been associated with dramatic improvement in two cases.[7]

TREATMENT

Recovery from HIL varies and does not appear to correlate with the severity of presentation.[2] Antioxidant treatment with Co-Enzyme Q10 has not been rigorously studied to declare if it is disease altering.

Buspirone hydrochloride, is primarily used as an anxiolytic agent in the treatment of general anxiety disorder, and is thought to act as a serotonergic 5-hydroxy tryptamine1A agonist (5HT1A) as well as a D2-Dopamine agonist/antagonist.[12] Case-reports, open label, and double blind trials have shown that Buspirone also has a modest effect to improve symptoms related to hereditary cerebellar ataxias, but this drug has never before been used to treat ataxia secondary to HIL.

CASE RESOLUTION

The patient was started on a regimen of Co-Enzyme Q10, vitamin C and D and was admitted to the In-Patient Rehab Unit ten weeks later. One week after admission he was started on a trial of Buspirone along with dextroamphetimine for his profound slowness.

No changes were noted in the two-month period on the antioxidant therapy prior to his in-patient rehabilitation. Within ten days of administration of the Buspirone, there was a significant improvement in dysdiadochokinesis, ataxia, and dysmetria. While no objective measures were used, his handwriting became legible, and his fine motor tasks improved.

Over the course of admission his mobility and speech also improved. He was able to walk supported with a two-wheeled walker. However, fatigue still limited his mobility.

Over the course of the following year, the patient continued to make significant gains taking Buspirone and participating in outpatient physical, occupational and recreational therapy. He began to walk unaided, and his speech became more fluent, and his handwriting improved drastically.

CONCLUSION

"Chasing the Dragon" is a common practice in our community and its use may be increasing.[4] HIL is a rare, but potentially devastating consequence of this method of heroin consumption that can be fatal. HIL often leaves patients with persistent neurological deficits such as ataxia. Anti-oxidant therapy, which needs to be studied further, is one of the few options in treatment of HIL. Buspirone is a serotonergic drug that has been studied in the treatment of hereditary ataxia and was observed to have a dramatic effect on the ataxia in this patient. This case underscores that clinical and MRI diagnosis of HIL is essential, as even in a fulminant presentation of HIL, measures should be taken to provide essential life support, acute care and long term rehabilitation as dramatic improvements may be seen.

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