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CASE REPORT

Spongiform Leukoencephalopathy after Intravenous Heroin Use: Evaluation by Diffusion-weighted Imaging

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ABSTRACT

Spongiform leukoencephalopathy is a distinct pathological entity usually following inhalation of heroin, while spongiform leukoencephalopathy resulting from intravenous injection of heroin is very rare. The magnetic resonance imaging findings are non-specific with widespread symmetrical high-signal lesions in white matter. This report is of an intravenous heroin addict who presented with clinical features of encephalopathy with restricted diffusion on diffusion-weighted imaging. Diffusion-weighted imaging distinguished this drug-induced encephalopathy from other types of leukoencephalopathy. This is the third case report of spongiform leukocenpahalopathy showing restricted diffusion on diffusion-weighted imaging in the English literature.

Key Words*: Diffusion-weighted imaging, Heroin, Leukoencephalopathy, Magnetic resonance imaging, Spongiform*

INTRODUCTION

Spongiform leukoencephalopathy is a rare phenomenon that is usually caused by inhalation of heroin vapour and is pathologically characterised by spongiform degeneration of white matter. The clinical and imaging features are non-specific and resemble other more common demyelinating diseases and encephalopathies.[1,2] This report is of a patient with spongiform leukoencephalopathy with restricted diffusion in the cerebral white matter and internal capsule on diffusion-weighted imaging (DWI). DWI is a useful and non-invasive tool for distinguishing this syndrome from other pathologies.

CASE REPORT

A 41-year-old man was admitted to Tuen Mun Hospital with a 1-month history of increasing cognitive failure and double incontinence. He had been using heroin intravenously for more than 20 years. He had frequently been admitted to various hospitals with narcotic overdose and withdrawal symptoms. His wife noticed that he had developed abnormal behaviour, insomnia, and was unable to walk without aids 1 week prior to this admission.

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At physical examination, he was confused, unable to recall recent events, and talked nonsense during a conversation. Verbal response was poor with limited eye contact. He was afebrile and there was neck stiffness. Bilateral lower limb weakness, cerebellar ataxia, and akinetic mutism were observed. Fundal examination was normal and there was no evidence of papilloedema. All the blood tests were normal and included complete blood count, renal and liver function tests, erythrocyte sedimentation rate, and serology for syphilis. Lumbar puncture was performed with a normal opening pressure. Cerebrospinal fluid was clear with normal biochemistry and cell counts.

Urgent non-contrast computed tomography scan of the brain showed subtle symmetrical hypodensity of the periventricular white matter. Magnetic resonance imaging (MRI) of the brain showed symmetrical hyperintensity of the internal capsule, periventricular region, and centrum semiovale bilaterally on T2-weighted images and fluid-attenuated inversion recovery sequence (FLAIR) images (Figures 1a and 1b). DWI showed high-signal intensity of the periventricular region and centrum semiovale and internal capsule (Figures 2a and 2b). The apparent diffusion coefficient was decreased, which confirmed that the abnormality on DWI was caused by restricted diffusion rather than the 'T2 shine-through' effect. There was no abnormal

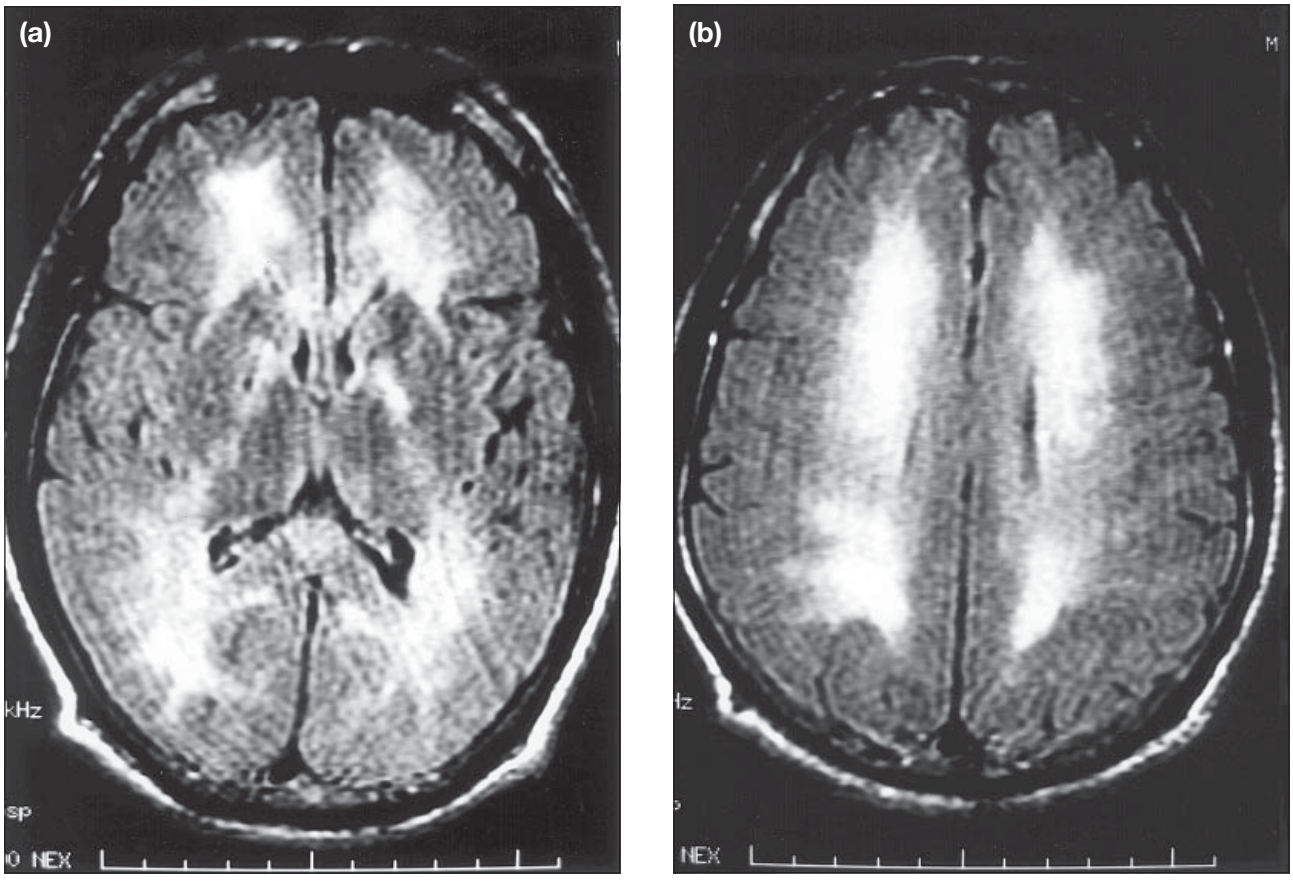

Figure 1. (a and b) Axial fluid-attenuated inversion recovery sequence images (TR 9002, TE 147) shows symmetrical high-signal intensity at the internal capsule, periventricular white matter, and centrum semiovale.

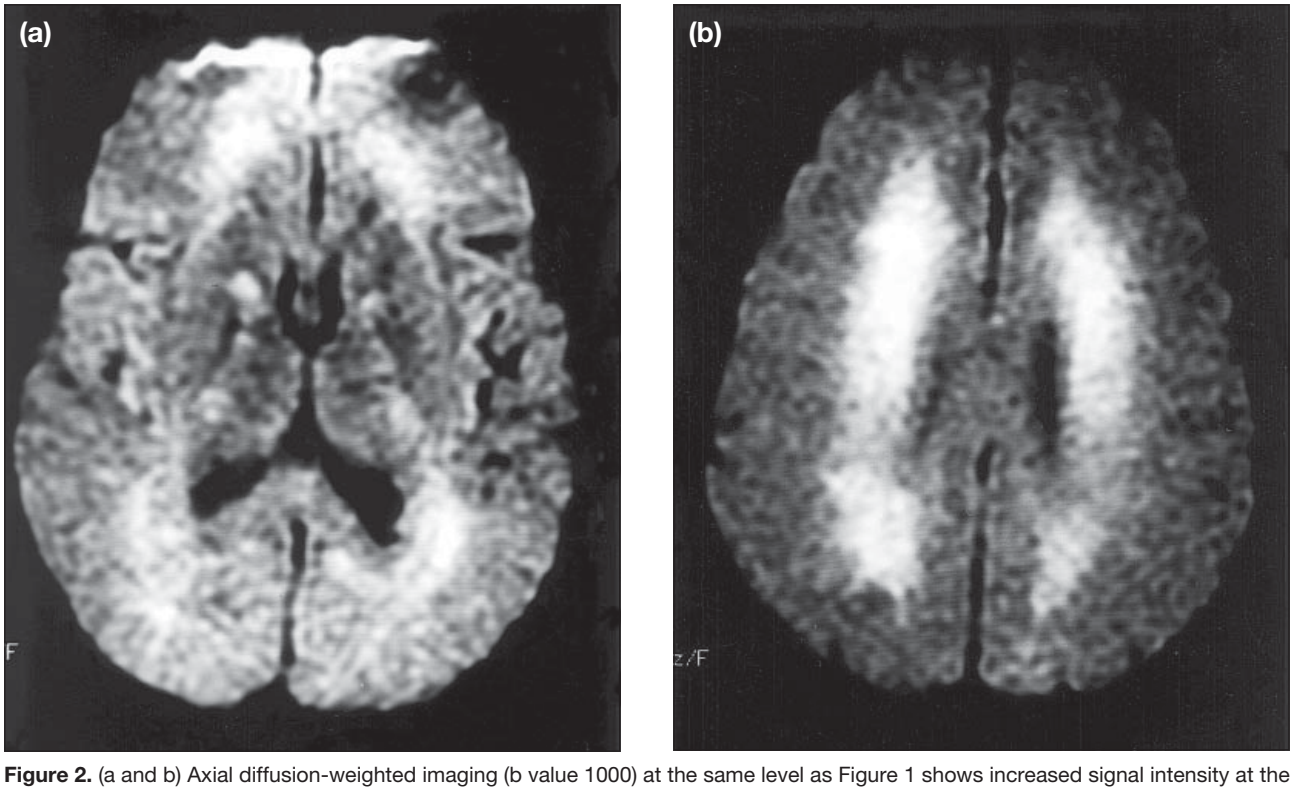

Figure 2. (a and b) Axial diffusion-weighted imaging (b value 1000) at the same level as Figure 1 shows increased signal intensity at the internal capsule, periventricular white matter, and centrum semiovale.

contrast enhancement in the gadolinium-enhanced images.

The diagnosis of spongiform leukoencephalopathy was made based on the clinical features and imaging findings. The patient was treated conservatively with haloperidol and benzodiazepam. His lower limb power improved and double incontinence subsided 4 days after admission. However, impairment in cognitive function persisted. He was discharged and a follow-up appointment in the psychiatric outpatient clinic was arranged. Unfortunately, he was admitted to another hospital following a narcotic overdose a few days after discharge.

DISCUSSION

Heroin intoxication may induce a variety of neuropathologic changes. The pathophysiology may be secondary to hypoxic-ischaemic insult, hypotension, cerebral oedema, or infections.[3] Heroin-induced spongiform leukoencephalopathy is a distinct entity that was first reported by Wolters et al.[1] In 1982, 47 drug addicts developed encephalopathy after inhaling heated heroin vapour in Holland. Several small series were subsequently recorded in Europe, the USA, and Taiwan.[4] Recently, spongiform leukoencephalopathy after intravenous injection of heroin has also been reported. However, it is extremely rare.[5,6]

The clinical features are characterised by cerebellar ataxia, dysarthria, akinetic mutism, and spastic quadriparesis. The majority of patients have an acute onset, although some may have a delayed onset after heroin overdose complicated by a period of prolonged unconsciousness.[1-4,6] Significant clinical improvement has been observed in some patients, either spontaneously or after treatment with antioxidants, including oral coenzyme Q. However, the majority of patients have persistent or progressive cognitive dysfunction and the mortality rate is estimated to be approximately 25%.[1,4]

Spongiform leukoencephalopathy is distinguished from other encephalopathies by the histopathological hallmark of vacuolar degeneration of cerebral white matter. Electron microscopy shows fluid entrapment between the myelin lamellae and absence of demyelination in these patients.[1] It has been postulated that there is a lipophilic toxin-induced process due to contaminants in the vapour, which is enhanced by hypoxia, although a definite toxin has not been identified.[1,7]

The imaging findings of heroin-induced spongioform leukoencephalopathy on MRI have been documented. The most common findings are bilateral symmetrical high-signal lesions in the white matter of the cerebrum and cerebellum on T2-weighted images. High-signal intensity in the internal capsule, corpus callosum brainstem, and lemniscus medialis has also been reported.[1,2,4,7-9] Myelopathy is rare and it is believed to be caused by ischaemia.[3] Although MRI is highly sensitive in detecting the white matter lesions, it is not specific for spongiform leukoencephalopathy. The imaging findings can mimic other types of leukoencephalopathies including demyelinating or metabolic diseases.

Diffusion MRI in spongiform leukoencephalopathy was first used by Chen et al, who demonstrated restricted diffusion in the cerebral white matter and claimed that diffusion imaging may be useful in distinguishing spongiform leukoencephalopathy from other demyelinating diseases.[5] These authors postulated that accumulation of fluid between the myelin lamellae by splitting at the intraperiod lines increased the anisotropy of water diffusion in the white matter and accounts for the increased signal intensity on DWI.[5] On the other hand, demyelinating diseases increase the extracellular space, which may result in an increase in water diffusion. Similarly, vasogenic oedema also increases water diffusion due to accumulation of fluid outside the myelin sheath, which decreases signal intensity on DWI.[5] These 2 common entities of hyperintense white matter changes on T2-weighted imaging can therefore be distinguished from spongiform leukoencephalopathy with the use of diffusion MRI.

Chronic toluene abuse can also result in development of symmetric T2 hyperintense white matter lesions in the cerebrum and cerebellum. Demyelination and gliosis are the histopathologic changes underlying these white matter lesions, which should demonstrate decreased signal intensity on DWI. T2-weighted hypointensity in the thalami and basal ganglia is another MRI feature seen in chronic toluene abusers, which helps to distinguish this disease entity from heroin-induced spongiform leukoencephalopathy.[10]

CONCLUSIONS

Spongiform leukoencephalopathy after intravenous use of heroin is rare and its clinical and imaging findings are non-specific and may mimic other more common encephalopathies, demyelinating, or metabolic diseases. DWI is a valuable tool in establishing the diagnosis.

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