

A young woman with dysarthria, bradykinesia and ataxia

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PART A

A 23-year old female presented in the Emergency Department with dysarthria, bradykinesia and ataxia. The patient inhaled heroin one week before the onset of the symptoms. She was tachypnoeic but haemodynamically stable (blood pressure 145/90 mm Hg) with a normal sinus rhythm. Corneal reflexes were present bilaterally and the motor examination revealed axial myoclonus. Gener-

al examination revealed no cardiac murmurs whilst the chest and abdomen were unremarkable. No needle marks were observed on the skin. After a normal initial brain computed tomography (CT) scan, the patient was further evaluated with brain magnetic resonance imaging (MRI) (**Fig. 1**). A second MRI was performed after a 13 day hospitalisation (**Fig. 2**).



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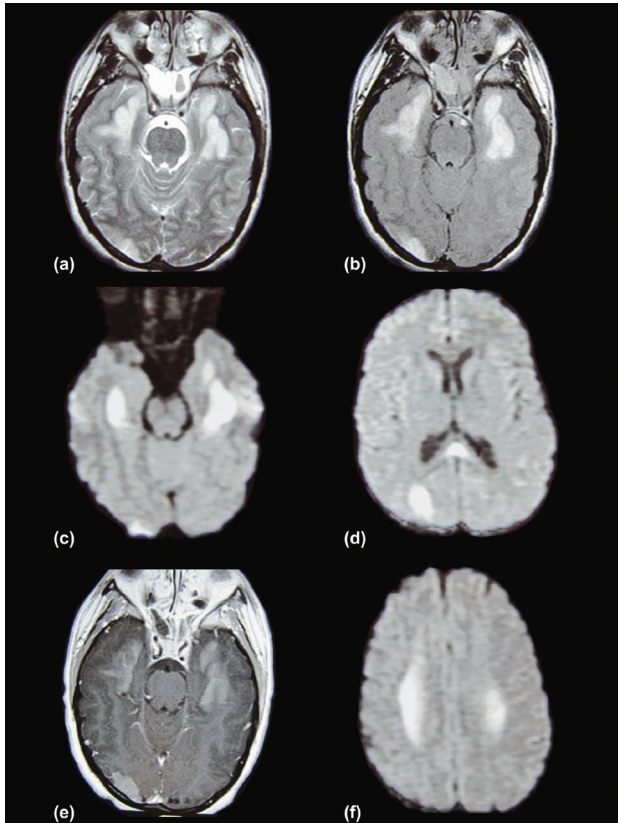


Fig. 1: Initial MRI: (a) Axial T2-weighted (T2W) image, (b) Axial fluid attenuation inversion recovery (FLAIR) image, (c) Axial diffusion weighted image (DWI), (d) Axial DWI image, (e) Contrast-enhanced axial T1W image, (f) Axial DWI image.

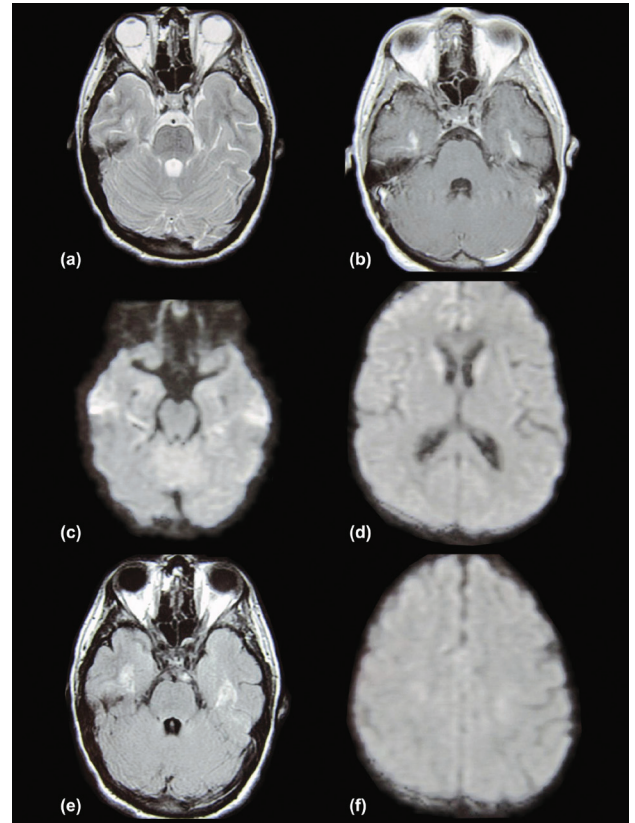


Fig. 2: MRI performed after 13 days of hospitalisation: (a) Axial T2W image, (b) Contrast-enhanced axial T1W image, (c) Axial DWI image, (d) Axial DWI image, (e) Axial FLAIR image, (f) Axial DWI image.

PART B

Diagnosis: Heroin inhalation toxic leukoencephalopathy or “Chasing the dragon” syndrome

The use of heroin, a diacetyl derivate of morphine, is common among drug addicts. Heroin may be used in different ways: intravenously, subcutaneously, smoked, sniffed or inhaled.

The term “chasing the dragon”, or “chinesing” or “chinese blowing” originates in the 1920s’ China and became rather popular in the 1950s in Hong Kong, since that impure drug form was cheap and thus widely consumed. It involves heating a small amount of powder placed on an aluminum foil and sucking the produced vapour with a straw or pipe. Heroin intended for smoking is usually 30% to 40% pure, since the purest forms degrade too quickly for effective smoking. Additionally, according to reports, heroin is mainly available in a non-intravenous base form [1].

That form is linked to toxic leukoencephalopathy, although the exact pathogenesis is still unclear, with characteristic and highly specific signal abnormalities on MRI, which can greatly aid in proper diagnosis. Typical imaging findings involve bilateral symmetric lesions to the cerebellar white matter, posterior cerebral white matter, especially in the occipital lobe, the posterior limb of internal capsule and the cerebellar peduncles, with sparing of the anterior limb of the internal capsule, the dentate nuclei and the subcortical U-fibers [2, 3]. The frontal region may be relatively spared as well, while affected areas may or may not show diffusion restriction. However, less common presentations with predominate involvement of the frontoparietal lobes, as well as hippocampal involvement have also been described [4, 5]. The term leukoencephalopathy generally refers to a disease of the white matter of the brain and therefore this entity involves motor, sensory and visual systems. Leukoencephalopathy can also disrupt cognitive and emotional functions. The clinical manifestations of the disease reflect the areas of the brain damaged as the disease progresses. Clinical features range from inattention, forgetfulness and personality changes, to dysarthria, ataxia, dementia, coma and death.

In our case, evaluation with MRI imaging (Fig. 1) showed symmetric hyperintensity concerning both the temporal lobes, the splenium of the corpus callosum, the centrum

semiovale and an area in the right occipital lobe on T2W images and FLAIR sequence.

DWI showed abnormal restricted diffusion in these areas, while contrast-enhanced axial T1W image demonstrated enhancement bilaterally in the temporal lobes and right occipital lobe.

The MRI findings improved drastically in 13 days (Fig. 2) and the patient gradually recovered. The new MRI showed the absence of hyperintensity on T2W images, with the exception of the lesions located in the temporal lobes; however these were found markedly reduced in size whereas a mild residual enhancement remained in post contrast T1W images. No restriction of diffusion was noted on the DWI images.

The first and largest outbreak of this type of toxic leukoencephalopathy linked to “chasing the dragon” was reported in 1982 in the Netherlands and included 47 cases, 11 (23%) of whom died [6]. Since then, similar cases have been reported sporadically in both Europe and the United States.

The primary hypothesis, although a specific aetiology has not been identified, is that a contaminant in the heroin or a combustion by-product [7] causes the lesions mentioned above. Additionally Buxton et al [8] support that there is also a dose-dependent response effect rather than a genetic predisposition to the manifestation of the disease.

It has been shown that severe changes occur in the white matter, termed vacuolating myelinopathy, which are characterised by the formation of vacuoles in the oligodendroglia and result in spongiform degeneration [9]. Mitochondrial dysfunction may be critical as well in the development of heroin leukoencephalopathy, as suggested by mitochondrial changes on specimens from brain biopsies. MR spectroscopy may show abnormally elevated intracerebral lactate in the affected white matter, as well as decreased levels of N-Acetylaspartate in the white matter, gray matter and cerebellum, postulating that these findings reflect mitochondrial toxicity [10]. Thus, MR spectroscopy is not essential in the diagnosis but may help in elucidating the nature of the condition [3].

Clinical improvement after administering antioxidant therapy, such as coenzyme Q and vitamin supplements,

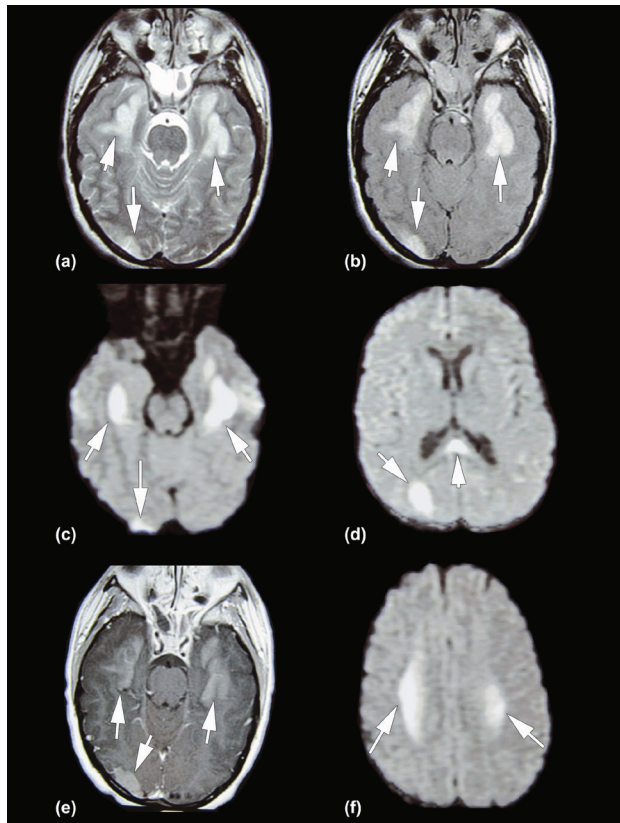


Fig. 1: Initial MRI: (a) Axial T2-weighted image, (b) Axial FLAIR image: hyperintensity in the temporal lobes and an area of hyperintensity in the right occipital lobe. (c) Axial DWI reveals restricted diffusion bilaterally in the temporal lobes and right occipital lobe, in the right occipital lobe and splenium (d) and in the centrum semiovale (f). (e) Contrast-enhanced axial T1W image demonstrates enhancement bilaterally in the temporal lobes and right occipital lobe (corresponding areas with arrows).

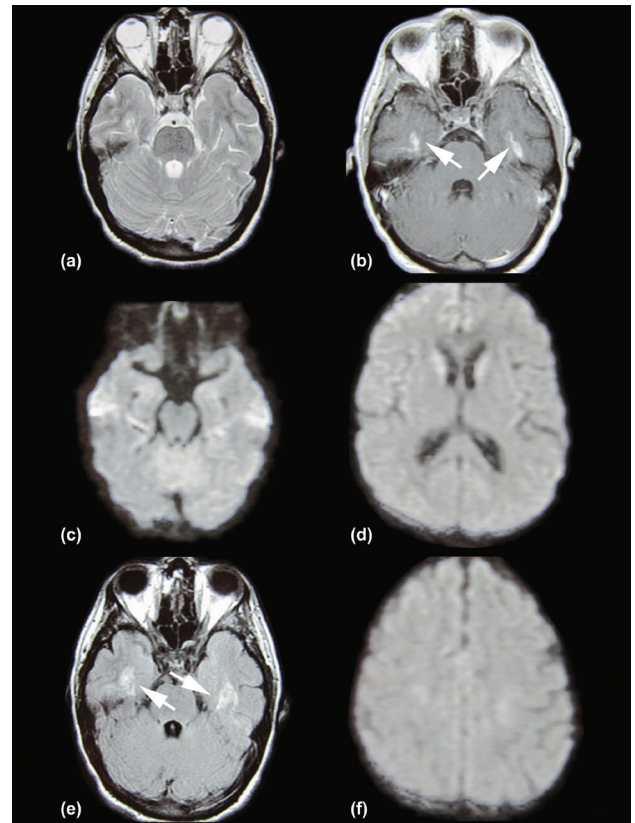


Fig. 2: MRI performed after 13 days of hospitalisation: (a) Axial T2W image showing no hyperintensity. (b) Contrast-enhanced axial T1W image demonstrates mild residual enhancement in the temporal lobes (c) Axial DWI shows no restriction of diffusion in the temporal lobes, in the right occipital lobe and splenium (d) and in the centrum semiovale (f), (e) Axial FLAIR image shows mild residual hyperintensities in both temporal lobes (corresponding areas indicated with arrows).

may be beneficial as seen in our patient and reported in a few other cases [10].

The syndrome presented herein has similar findings to other leukoencephalopathies with widespread white matter hyperintensity on T2W images involving both supra and infratentorial compartments. A reasonable differential should include global hypoxic/ischaemic insult, cerebral vasculopathy, posterior reversible encephalopathy syndrome, hypoglycaemic encephalopathy, progressive multifocal leukoencephalopathy, acute disseminated encephalomyelitis, as well as toxic leukoencephalopathies related to drug administration such as methotrexate, cisplatin, cyclosporine, metronidazole,

ethanol, methanol, cocaine and arsenic. In our case, relevant patient history played a key part in establishing the diagnosis. Since most of the reports have been published in the form of clinical cases, it is difficult to determine possible risk factors for this condition in biochemical and molecular level [8].

MRI is the imaging modality which allows the diagnosis of toxic leukoencephalopathy, determines the extent of the pathology and assesses the effect of treatment during follow up exams. **R**

Conflict of interest

The authors declared no conflicts of interest.



KEY WORDS

Chasing the dragon; Heroin toxicity; Toxic leukoencephalopathy; MRI

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