A 29-year-old man with a history of drug and alcohol abuse was admitted to the intensive care unit in coma. Schizophrenia was diagnosed three years before, but he voluntarily rejected treatment. His parents revealed suspicion of heroin inhalation 24 hours ago. CT examination was performed within 24 hours of the onset of symptoms.

Imaging Findings:

Unenhanced CT (Fig. 1) showed small hypodense foci in corona radiata, semiovale centres and posterior limb of the left internal capsule. No abnormalities were identified in the brainstem or cerebellum.

48 hours after the CT acquisition a brain MRI was performed. T2-weighted images showed diffuse symmetric area of abnormal high signal intensity in the periventricular and subcortical white matter, with respect of the U fibres, and almost symmetrical hypointense foci affecting semiovale centres, globus pallidi and splenium of corpus callosum (Fig. 2).

Axial diffusion-weighted (DWI) images showed areas of high signal intensity distributed diffusely in the white matter of the frontal, temporal, and occipital lobes and splenium of corpus callosum, with corresponding hypointensity on the apparent diffusion coefficient (ACD) maps, indicating restricted diffusivity (Fig. 3).

Axial T1-weighted image after administration of intravenous gadolinium (Fig. 4) showed diffuse white matter enhancement indicating increased permeability of the blood-brain barrier.

These findings suggested drug abuse leukoencephalopathy.
Discussion:

Heroin-induced spongiform leukoencephalopathy is a rare toxic leukoencephalopathy produced after inhalation of heroin pyrolysate vapors, which was first reported by Wolters et al. in 1982 [1]. The chemical that causes toxic adverse effects in white matter is thought to be an occasional impurity that crosses the blood-brain barrier when heroin is heated. Users heat the powder on aluminium foil and inhale the smoke. This practice is known as “chasing the dragon” [3].

The clinical presentation varies greatly, there are three distinct stages and patients may remain in one stage or pass through two or all three. During the initial stage, cerebellar symptoms are observed. In the intermediate stage worsening of the cerebellar and extrapyramidal symptoms are observed and finally progressive stretching spasms, akinetic or hypotonic mutism or death indicate the terminal stage. In most such cases, there has been a latency of a day or more between inhalation of the drug and the first symptoms. Heroin leukoencephalopathy is associated with an estimated mortality rate of 23% [4]. Histopathologic findings reveal formation of vacuoles within the myelin sheath of the deep white matter, damaging the oligodendrocytes and resulting in spongiform degeneration [5].

The final diagnosis is based on the patient’s history and the neurological and imaging findings. The clinical symptoms and imaging findings on CT and MRI are highly specific, presenting as symmetric lesions of high signal intensity on T2-weighted images in the cerebellar and occipital white matter, the posterior limb of the internal capsule, the splenium of the corpus callosum and the medial lemniscus in the brainstem [2, 4]. The reason for this striking pattern of involvement is unknown. Typical findings are also the sparing of the anterior limb of the internal capsule and of the dentate nuclei of the cerebellum [3]. DWI/ADC changes in the acute stages are usually consistent with restricted diffusion secondary to cytotoxic oedema, while in subacute/chronic stages there is an increased diffusivity due to myelin breakdown and neuronal loss.

In conclusion, the clinical and imaging findings of toxic spongiform leukoencephalopathy after heroin inhalation are highly specific. In this case the cerebellum was normal but white matter and corpus callosum were affected and heroine inhalation was confirmed the day before, which confirmed the diagnosis. The neurological deficit is typically irreversible, with progression to death in some patients. There is no effective therapy, but coenzyme Q treatments may limit the extent of the disease.

Differential Diagnosis List: Toxic leukoencephalopathy after heroin inhalation, Hypoxic-ischaemic disease, Neurotoxic drugs, Environmental and endogenous toxins, Posterior reversible encephalopathy syndrome

Final Diagnosis: Toxic leukoencephalopathy after heroin inhalation

References:


Yin R, Lu C, Chen Q, Fan J, Lu J (2013) Microvascular damage is involved in the pathogenesis of heroin induced...
Description: T2-weighted images: symmetric area of abnormal high signal intensity in periventricular and subcortical white matter, and symmetrical hyperintense focal lesions in semiovale centres, globus pallidi and splenium of corpus callosum. Origin: Servicio de Radiología. Hospital de la Ribera. Alzira, Valencia.
**Description:** Restricted diffusion distributed diffusely in the white matter of the frontal, temporal, and occipital lobes and splenium of corpus callosum. The findings were compatible with cytotoxic oedema. **Origin:** Servicio de Radiología. Hospital de la Ribera. Alzira, Valencia.
Description: T1-weighted images after administration of intravenous gadolinium showed diffuse white matter enhancement indicating increased permeability of the blood-brain barrier.
Description: Small hypodense foci in corona radiata, semiovale centres and posterior limb of the left internal capsule. Origin: Servicio de Radiología. Hospital de la Ribera. Alzira, Valencia.