Lentiform Nuclear Degeneration Following Methanol Ingestion

Fig. 1. MRI brain, T2WI in sagittal section (A) showing hyperintense lesion (arrow) in lentiform nucleus. T1WI in sagittal section (B) showing hypointense lesion (arrow) in the same area. T2WI in axial (C) and coronal section (D) showing hyperintense lesion (arrow) in lentiform nucleus on both side.

A 30 year male, regular alcohol consumer for 10 years presented with sudden visual loss in both eyes 30 hours after ingestion of illicit liquor adulterated with methanol. He did not have headache, periorbital or retrobulbar pain.

He was conscious, oriented and his visual acuity was reduced to finger counting and perception of light in both the eyes. Pupils were dilated (4.5 mm) and non-reactive to light. Optic discs were pale with attenuated blood vessels. Neurological and systemic examinations were normal.

Arterial blood gas analysis, at admission revealed high anion-gap (AG) metabolic acidosis (AG 27 mEq/L, pH 7.3, bicarbonate 10 mEq/L). Cerebrospinal fluid analysis was normal. Brain-stem auditory evoked potentials (BAEP) were normal but the P100 response in the visual evoked potentials (VEP) was prolonged (125 msec-1) suggestive of severe optic neuritis. Magnetic resonance imaging (MRI) of brain showed bilateral basal ganglia involvement with hyperintense lesion measuring 1 cm near the peripheral lentiform nuclei on T2-weighted images (T2WI) with flair sequence and hypointense lesion on T1-weighted images (T1WI), characteristic of toxic demyelination (Fig. 1). He was treated with haemodialysis, vitamin B1, B6, B12, folinic acid and methylprednisolone with initial improvement in his visual acuity (6/9 right eye, 6/12 left eye) but subsequently lost his vision. This type of biphasic course is known with methanol intoxication.

Lenticular degeneration with methanol ingestion is uncommon and it was first reported on imaging in 1980.1 Similar types of lesion may be found in manganese and carbon monoxide intoxication, chronic porto-systemic encephalopathy, Hallervorden Spatz disease and hypoxic ischaemic injury. In methanol poisoning the characteristic lesion is putaminal necrosis and on long term follow-up these patients may present with extrapyramidal syndrome.

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