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Letter to the Editor

Neuroimaging findings and follow-up in two cases of severe ethylene glycol intoxication with full recovery

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1. Introduction

We report the early neuroimaging findings and follow-up in two cases of severe ethylene glycol (EG) intoxication showing initial widespread lesions in the basal ganglia (BG), thalami, temporal lobe, brainstem and cerebellum, with total recovery on follow-up.

2. Case 1

A 37-year old male presented with consciousness impairment attributed to the ingestion of an anti-freeze solution. At admission, the patient's Glasgow coma score (GCS) was 7, Kussmaul breathing, miosis, absent tendon reflexes with a body temperature of 35 °C. Brainstem reflexes were unremarkable.

The patient was mechanically ventilated and sedated. Chest radiograph showed right basal aspiration pneumonia.

Blood work-up revealed renal failure and high anion gap metabolic acidosis (Table 1). Plasma EG concentration measured using gas chromatography was 19.1 mg/dL.

The patient was immediately administered fomepizole with intravenous loading dose of 15 mg/kg followed by a continuous 1 mg/kg/h infusion during the first 24 h along with hemodialysis. Patient management included additional hemodialysis sessions at days 4, 7, and 10.

CT scan at H24 showed brain edema and marked hypodense lesions in the BG, thalami and brainstem. (Fig. 1A–B). On day 3, MRI revealed bilateral and symmetrical FLAIR hyperintense lesions in the BG, thalami, internal and external capsule, medial temporal lobe, brainstem, and cerebellum, and less extensive lesions on diffusion-weighed image (DWI) without diffusion restriction (Fig. 1C–K). MRI follow-up on day 12 showed total recovery.

The patient regained normal level of consciousness and physical examination became normal on day 13. The patient had further nephrological follow-up. He fully recovered few months later.

3. Case 2

A 77-year old female became increasingly agitated. On the scene, the patient presented GCS of 15 but was mechanically ventilated and sedated to allow her transport. At admission, less than 4 h after ingesting an unknown amount of an anti-freeze product, the vital signs and clinical examination were unremarkable except for a temperature of 35 °C and pupils in miosis. Laboratory studies (Table 1) revealed renal failure and plasma EG concentration of 61.7 mg/dL. She received an intravenous 15 mg/kg loading dose of fomepizole, and an infusion of 1 mg/kg/h during the 8-h hemodialysis session. MRI at H36 revealed brain edema and hyperintense lesions in the BG, thalami, hippocampi, dorsal pons, on FLAIR (Fig. 1L–M) and in the dorsal pons on DWI without diffusion restriction. She rapidly improved and was successfully extubated on day 2. MRI follow-up on day 8 showed total recovery.

4. Discussion

EG poisoning produces usually successive stages including an initial latency phase followed by the onset of metabolic acidosis, renal insufficiency and multi-organ failure [1].

Except the initial EG-related transient inebriation, central nervous system (CNS) impairment is usually delayed, evidencing that EG toxicity results from its conversion to toxic metabolites, thus explaining the lack of correlation between symptoms and EG concentrations. Severe encephalopathy appearing a few hours after poisoning, with coma and absence of brainstem reflexes was reported in cases of massive ingestion usually followed by the patient's death [2]. EG intoxication has been reported to mimic an acute basilar artery occlusion [3].

CT shows diffuse cerebral edema 24 to 48 h post-ingestion. During the second and third days hypodensities involving BG, thalami, midbrain and upper pons are observed, disappearing after 5–35 days [2, 4–6].

In our first case, early CT findings (24 h), presented the same pattern with an additionally medulla involvement.

Hemorrhagic lesions in lenticular nuclei (LN) after EG poisoning have been also reported on initial CT in 2 patients: in the pallidi in a patient who completely recovered [7] and in the putamina in another patient with fatal outcome [8].

On the other hand MRI abnormalities, reported in few cases were consistent in one case with putaminal necrosis [6], revealing hemorrhagic necrosis in 2 other cases: in the thalami, pallidi and putamen at day 8 in a patient presenting acute Parkinson's syndrome [9] and of the LN at day 9 in a fatal case [8].

MRI showed in Case 1, at the early stage (day 3), bilateral and strikingly symmetrical hyperintense lesions in FLAIR and DWI of the BG, internal and external capsule, thalami, hypothalamus, medial temporal lobes (amygdala, hippocampe, parahippocampic gyrus), the midbrain (sparing the red nuclei and the corticospinal tracts), posterior pons, the medulla and the cerebellum. There was no diffusion restriction on ADC map. In Case 2, the distribution of lesions was similar but less extensive. This pattern of lesions seems to correlate with the potential areas of CNS dysfunction in EG poisoning [6].

The window of opportunity to find these lesions seems to be the first 3 days.

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Table 1
Biological data on admission.

	Normal range	Case 1	Case 2
pH	7.34–7.45	6.77	7.13
$PaCO_2$, mm Hg	32–45	26.5	23.6
HCO_3, mmol/L	20–26	3.8	7
Creatinine, μmol/L	53–105	334	650
Urea, mmol/L	3.0–7.0	9.5	5.3
Lactates, mmol/L	0.70–2.10	6.54	1.3 L
Anion gap, mmol/L	3.0–11	34.2	28.3
EG level, mg/L	0	191	616.6

Interestingly the most extended MRI abnormalities were observed in Case 1 presenting the most severe neurological impairment with the lowest EG level.

As CNS impairment is related to conversion of EG into toxic metabolites, this observation is probably related to the longer interval between ingestion and admission in Case 1, compared to Case 2. The patients completely recovered.

Acute bilateral BG lesions on MRI may be produced by other toxics, especially methanol, CO, cyanides, and by acute metabolic condition, i.e. hypoxia.

Methanol intoxication causes bilateral putamina necrosis and variable degrees of hemorrhage, associated with hemorrhagic necrosis of the subcortical white matter and cerebellar necrosis. Our cases show a pattern of brain lesions not previously reported in EG intoxication and quite different than the pattern of other lesions with bilateral acute BG involvement.

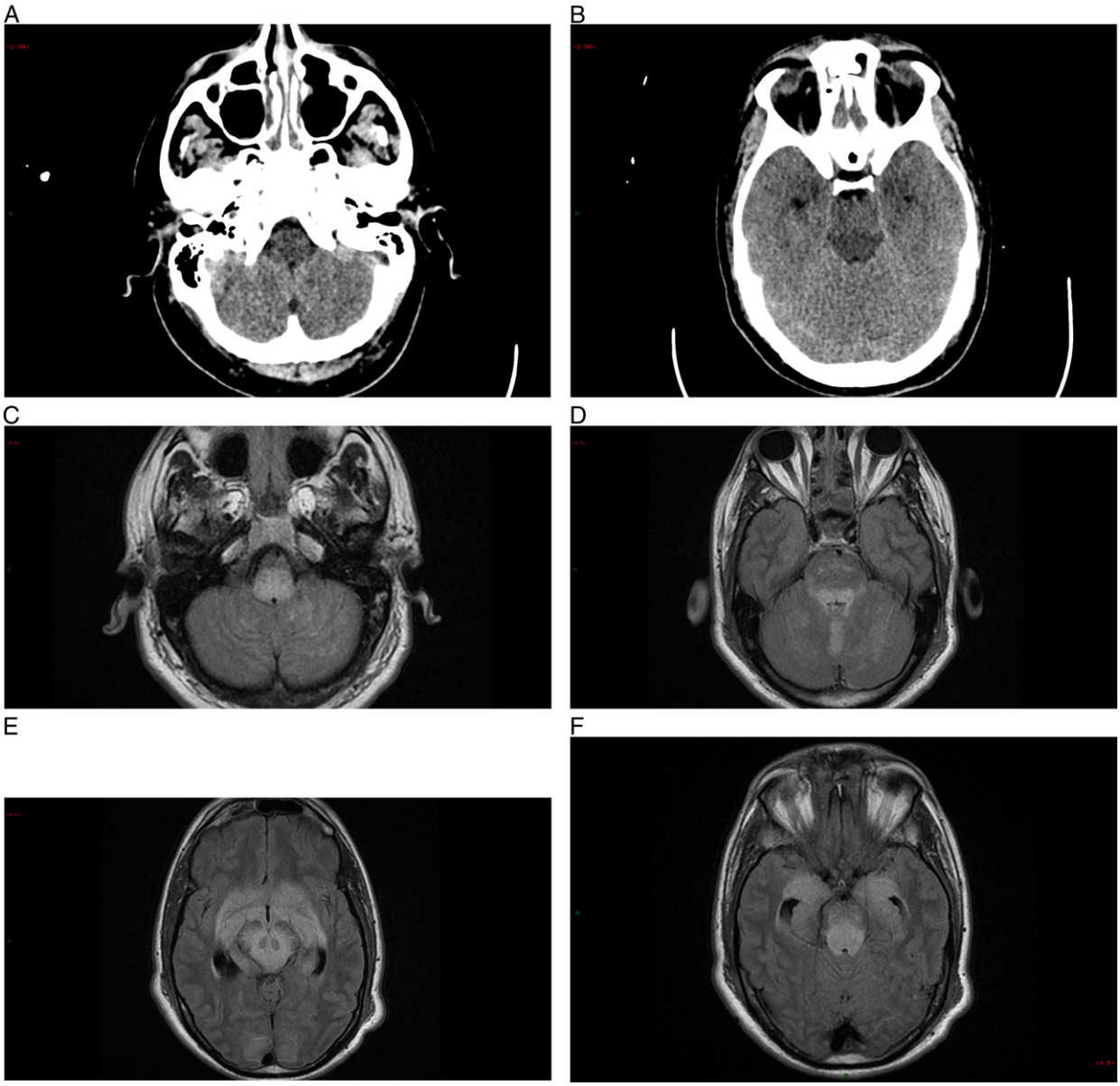

Fig. 1. A–J — Brain imaging in Case 1. Non-enhanced CT-scan obtained 24 h after EG ingestion, shows hypodensity of the medulla (A) and the posterior pons (B). MRI at day 3: FLAIR shows hyperintensity of the medulla (C), the posterior pons and cerebellum (D), midbrain, sparing the red nuclei and the cortico-spinal tracts (E) medial temporal lobe (Amygdala, hippocampi, parahippocampi gyrus) (F), the thalami, the caudate and lenticular nuclei, the posterior limb of the internal capsule and the external capsule (G). Note the enlarged temporal and occipital horns of the lateral ventricles secondary to the third ventricule compression. Diffusion-weighted images show hyperintensity of the tectum of the medulla (H), pons and amygdala (I), thalami and lenticular nuclei (J). The apparent diffusion coefficient map shows high values at the same areas (K). L–M: MRI at 36 h in Case 2. FLAIR shows hyperintensity of the tectum of the pons (K) and the lenticular and caudate nuclei, thalami and hippocampi (L). Diffusion shows slight hyperintensity at the dorsal pons (M).