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Case Report
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Parkinsonism, a Sequel of Severe Self-Induced Methanol Poisoning

Herishanu YO[1], Merkin E[1] and Shelef Y[2]*

[1]Department of Neurology, Soroka University Medical Center, Ben-Gurion University of the Negev, Israel

[2]Department of Radiology, Soroka University Medical Center, Ben-Gurion University of the Negev, Israel

Abstract

Acute methanol poisoning occurs sometimes in people who cannot afford the prices of marketed alcoholic beverages and utilize methanol as a substitute, in those who deliberately try to commit suicide, or in people using methanol for their work, like those working in the laboratories etc.

The clinical picture of the acute methanol poisoning consists of a tetrad of symptoms: Optic and central nervous system, gastrointestinal and respiratory tract symptoms. These symptoms are an expression of developing metabolic acidosis due to methanol metabolites (formate). Optic nerve damage leads in a majority of the cases to loss of vision and optic nerve atrophy. Late sequelae in the patients who survive severe intoxication, beside blindness, include parkinsonism.

We present here the clinical picture in a patient who suffered a severe methanol intoxication after a suicidal attempt, who after recovery from the acute intoxication developed a parkinsonian akinetic syndrome responsive to I-DOPA. In this case CT of the brain was normal, although MRI of the brain disclosed bilateral putaminal necrosis with some other unusual features.

Case Presentation

A 46-year-old woman working as a laboratory technician in the Department of Chemistry and because of a reactive depression related to her marital difficulties, made a suicide attempt swallowing 100 ml of methanol. Several hours later, she started to feel dizzy, had abdominal cramps and vomited. She lost consciousness and was brought to the Emergency Room in a comatose state.

On admission, she was unconscious (GCS 8) with spontaneous breathing. The pupils were large and unresponsive to light. The patient was hypotonic and no tendon reflexes were elicited. Laboratory tests disclosed plasma pH 6.76, HCO_3 2.7, pCO_2 21, pO_2 45.9, Sodium 152 mEq/ml, Potassium 6.3 mEq/ml, Chloride 95 mEq/ml, Creatinine 1.4 mg%, Urea 15 mg%, Glucose 224 mg%, Hemoglobin 17.2 g%, Hematocrit 53%, WBC 15,000/mm^3, platelets 343,000/mm^3, plasma osmolarity 379, osmolar gap 60, methanol blood level at the admission was 200 mg% and no ethanol was detected. Shortly after the admission, she developed pulmonary oedema, hemoptysis, hematuria and shortly later became anuric. An endotracheal intubation was performed. The patient received 600 cc of ethanol 10% (7.5 cc/Kg, 120 cc/h) and later on 1.5 cc/Kg; folic acid 50 ITlg SÏX tilTlQ5 ä day intravenously. She had two hemodialysis and started to pass urine. The next day the patient was alert and no abnormal neurological signs were noticed. Visual acuity was 6/6 in both eyes. On eye ground examination two small hemorrhages were found on the left retina. One week later, the patient was discharged in a fair condition.

Two weeks later was admitted to the Neurology Department because of slowness in the motor activities. Her mental status was unaffected. Visual acuity was 6/6 in both eyes, the pupils reacted well to light and accommodation and the optic discs were normal. The neurological examination revealed amimia, bradykinesia, positive glabellar tap response, rigidity of the neck muscles, slight cogwheel rigidity of the limb muscles and a mild resting tremor. While walking she did not move her arms which were kept slightly flexed and the patient had to turn with several steps. The head was slightly bent forwards. Seborrhea was evident. The Webster scale score was 12. Sinemet 125 mg I.i.d was started with a fast improvement.

A CT scan of the brain was normal. However, on mull-spin-echo MRI findings of putaminal necrosis were seen (Figure 1, 2) and after Gd DPTA injection an unusual linear enhancement at the medial limit of the necroGc putamina was found (Figure 3).

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Correspondence:
Yuval O Herishanu, Department of Neurology, Soroka University Medical Center, Goldman Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel,

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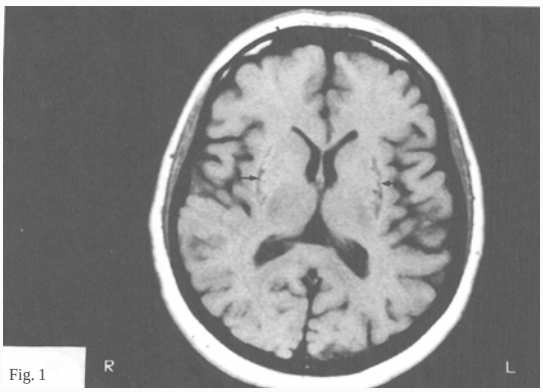

Figure 1: A-D Multi-spin echo images T1 weighted (600/15 ms) demonstrate low signal intensity lesions (arrows) corresponding to bilateral putamina.

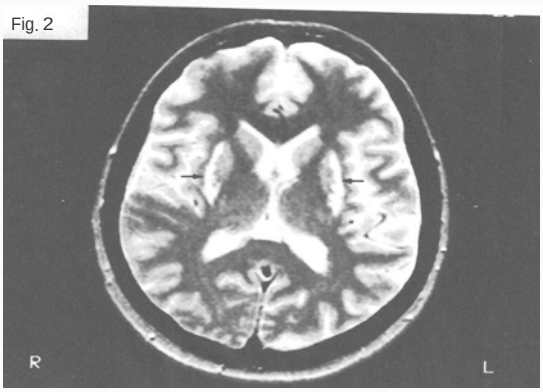

Figure 2: The lesions show very high signal on T2 weighted images (2500/90 ms) (arrows). Globus pallidum is spared.

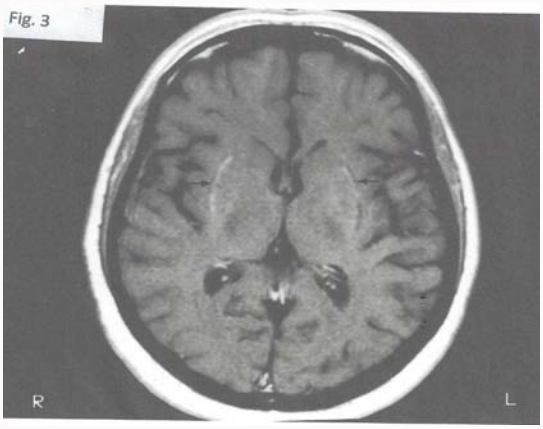

Figure 3: After GD DTPA administration, a linear enhancement is present at the medial limit of the necrotic areas on both sides.

Discussion

Methanol is a colorless and volatile liquid absorbed through the mouth, skin and by inhalation. Methanol intoxication results from ingestion of adulterated alcohol, voluntary suicidal attempt, cutaneous exposure [1] or inhalation, sometimes from perfume [2] or from vapors in laboratory [3]. Rarely, extensive use of illegal spirits may provoke a mass methanol intoxication like that which occurred in Estonia in 2001 with a mortality of more than 500/ of the patients [4]. Methanol is metabolized by the enzyme alcohol dehydrogenase in the liver to formic acid which is responsible for the effects observed in the methanol intoxication. The toxicity is due to metabolic acidosis and the formate anion [5]. The onset and course of the symptoms of acute methanol poisoning are parallel to increase of the formate levels and of severe metabolic acidosis. This process takes approximately 12 h. The clinical picture of the acute methanol intoxication consists as of a tetrad of symptoms: Optic and central nervous system, gastrointestinal and respiratory tract symptoms. Optic nerve damage leads in a majority of the cases to loss of vision and optic nerve atrophy. Late clinical sequelae in those who survive severe intoxication, beside the blindness, include Parkinsonism. Our patient had no signs of optic nerve damage but presented clinical signs of parkinsonism which reacted well to levodopa treatment as observed by others [2,6]. In this case, CT of the brain was normal, although MRI showed putaminal necrosis and after Gd DPTA an unusual linear enhancement at the medial limit of necrotic putamina. The putaminal lesions are well disclosed in the second and subsequent weeks [7-10]. Airas et al. [11], by using PET scanning with 6-[18F] Fluoro-L-dopa confirmed symmetrical impaired presynaptic dopaminergic activity in the striatum as indicative of functional impairment of dopaminergic nigrostriatal neurons in a patient who developed extrapyramidal signs after methanol intoxication. In this patient MRI disclosed putaminal injury like in our patient and in a majority of the patients surviving methanol intoxication.

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