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Case Report

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Brain imaging findings of methanol poisoning and detection of chronic stage nervous system damage: A case report

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Abstract

In this article, we presented the chronic neurological findings of an acute case of methanol intoxication due to the use of homemade alcohol. In the neurological examination performed 6 months after intoxication, bilateral vision loss, tremor, dysarthria, dysphagia, loss of taste sensation, and decrease in the Cognitive State Test score were detected. Peripheral polyneuropathy consistent with axonopathy was detected in the peripheral nerve conduction study, and optical neuropathy was detected in the examination of the pattern visual evoked potential. Brain magnetic resonance imaging revealed findings compatible with bilateral putaminal necrosis.

Keywords: methanol intoxication, optic neuropathy, putaminal hemorrhagic necrosis, polyneuropathy, cognition

1. Introduction

Methanol is called wood wine and obtained from the distillation of wood (1). It is contained in antifreeze, cleaning solutions, paint, and paint removers. Illegally produced alcohol or homemade alcoholic beverages may contain high methanol levels (2). Methanol itself is not very toxic, but its metabolites are. Methanol is converted to formaldehyde by the alcohol dehydrogenase enzyme, and formaldehyde is metabolized to formic acid by aldehyde dehydrogenase (2). Adverse effects due to methanol intoxication are caused by the effect of lactic acid, which increases due to formic acid and cellular hypoxia (3). There may be residual sequelae in the patients who have survived after acute methanol poisoning. We aimed to investigate the residual sequelae of methanol intoxication related to central nervous system (CNS), visual pathways, peripheral nervous system, and cognition.

2. Case

35-year-old male patient applied to the neurology outpatient clinic of our hospital with the complaints of speech disorder, difficulty in swallowing, tremor in the hands, inability to taste, and forgetfulness. In his anamnesis, it was learned that he consumed approximately 200 ml of alcohol produced at home six months ago and then applied to the hospital with the complaints of headache, nausea, vomiting, blurred vision, and tendency to sleep. The patient declared that he received treatment in the intensive care unit with the diagnosis of methanol poisoning. A detailed neurological examination was performed 6 months after the event. The right eye could count fingers at a distance of one meter and the left eye at a distance of 0.5 meters. In the fundus examination, the bilateral temporal region was pale, and the visible retina was normal. Direct and indirect light reflex was bilaterally minimal. Bilateral gag reflex could not be observed; there was no uvula movement. Sweet (1/1 diluted sugar water), salty (1/1 diluted salt water) and sour (lemon juice) taste test was performed with the patient who stated that he could not taste. The test was applied to the right and left sides of the tongue. He could not get the bilateral salty and sweet flavors, and he got the sour taste partially. He had complete muscle strength in four extremities. There was no rigidity or bradykinesia. Babinski's reflex was negative on the left and positive on the right. There was a 3-4 beat clonus on both feet. Cognitive State Test was applied upon the description of forgetfulness complaint. He scored 21/27 on the cognition test. A decline was observed in memory, recall, abstraction-judgement, attention, and language skills in the test. No hearing loss was detected in the audiometry test. Brain magnetic resonance imaging (MRI) revealed bilateral putaminal and frontal subcortical lesions. There was no evidence of myelopathy in cervical and thoracic MRI. Bilateral visual evoked potential (VEP) study revealed bilateral visual transmission disorder. Distal symmetrical polyneuropathy (PNP) findings were detected in the peripheral nerve conduction study (NCS). He was investigated for the etiology of PNP. The laboratory tests results as fallows: fasting blood glucose was 83 mg/dL, HbA1c was 5.2%, thyroid stimulating hormone (TSH) was 2.1 mIU/L, and vitamin B12 was 374 ng/L. Anti-nuclear antibody, PR3 ANCA, MPO ANCA, Anti-DsDNA, anti-Sm-D1 were negative. Monoclonal gammopathy was not detected in serum immune electrophoresis. No

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abnormal band was observed in urine immune fixation electrophoresis. Anti-human immunodeficiency virus (HIV), Anti HCV, HBsAg were negative.

Medical history: No known abnormality.

Family history: The mother was a type-2 diabetes mellitus patient.

3. Discussion:

Most cases of acute methanol toxicity result from oral intake. Ingestion of up to 30 mL of pure methanol results in permanent blindness. 30-240 mL is potentially fatal, but individual sensitivity varies greatly (4). Nausea, vomiting, dizziness, headache, and visual disorders are the most common complaints in the acute phase (5). Impaired consciousness, memory loss, Parkinsonism, convulsions, coma, and death may occur in severe poisonings (6). Extrapyramidal symptoms are typical neurological sequelae in the patients recovering from poisoning. These include dystonia and Parkinsonism (7). Our patient had mild-moderate essential tremor. He had no Parkinson's tremor and no dystonia. There was Babinski positivity and clonus. No epileptic seizures were diagnosed.

Characteristic brain imaging findings of methanol toxicity include bilateral putaminal hemorrhagic necrosis, cerebral and intraventricular hemorrhage, cerebellar necrosis, and diffuse cerebral edema. Cystic necrosis may occur in the chronic stage of the affected areas (8). Bilateral putaminal necrosis can also be observed in Wilson's disease, Kearns-Sayre syndrome, and Leigh's disease. Carbon monoxide, cyanide, trichloroethane poisoning, or diffuse hypoxia may also cause similar radiological findings and should be considered in the differential diagnosis (8). Our patient did not have any previously known metabolic or hereditary diseases. In addition, the history of vision loss and homemade alcohol use was characteristic for methanol intoxication.

The case had typical bilateral putaminal hemorrhagic necrosis images consistent with methanol intoxication in acute brain tomography (CT) (Fig. 1) and acute-subacute flair MRI (Fig. 2). In the chronic period, putaminal volume loss was observed (Fig. 3).

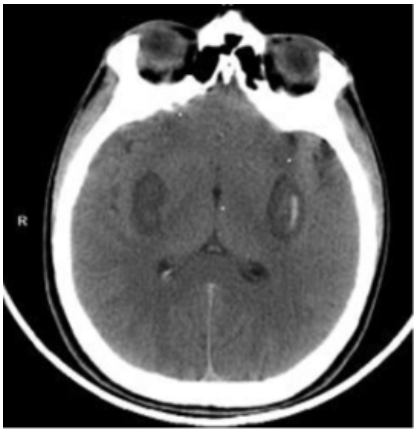

Fig. 1. Bilateral putaminal hemorrhagic necrosis accompanied by edema is observed in acute period brain CT

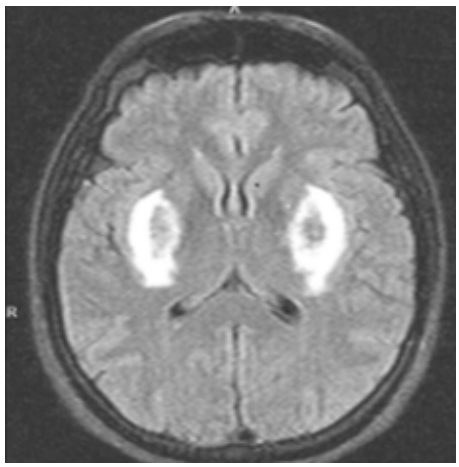

Fig. 2. Bilateral putaminal hemorrhagic necrosis areas in acute-subacute period flair MRI

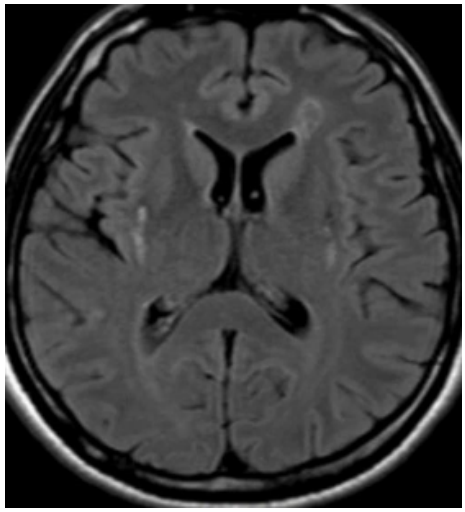

Fig. 3. Bilateral putaminal volume loss in chronic period flair MRI, lesions accompanied by hemosiderin pigments and gliosis, and hyperintense lesion in the left frontal subcortical white matter

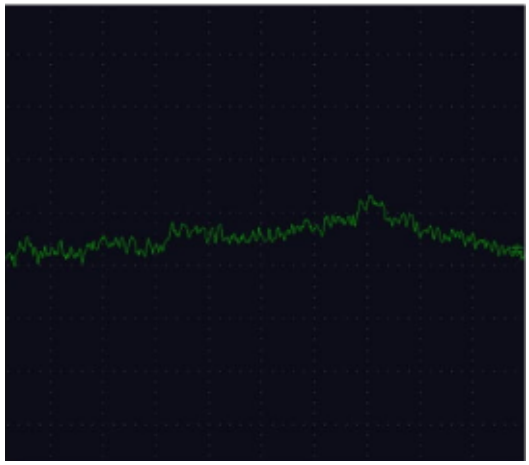

Fig. 4. Pattern VEP study showed there was no P100 wave on the left

In the literature, 5th, 7th and 9th cranial nerve involvement findings after methanol poisoning have been diagnosed (9). In our case, dysarthria, dysphagia, and loss of taste sensation (cranial 7th and 9th nerve involvement) were detected in accordance with cranial nerve involvement. Cognitive State Test was applied for the complaint of forgetfulness. He scored 21/27 on the cognition test. Although it is not common in the

literature, there are papers stating that there is a decline in cognition (6).

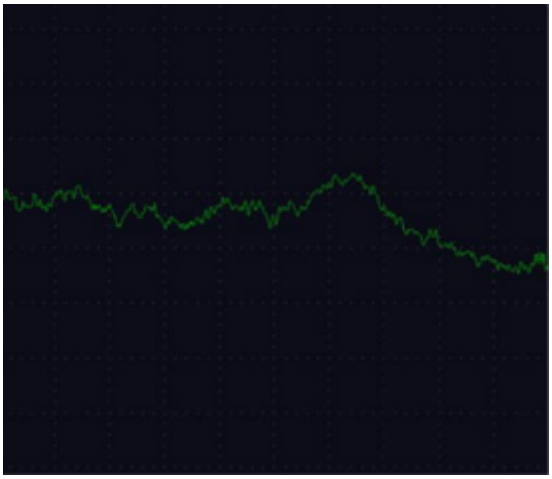

Fig. 5. Pattern VEP study showed the P100 wave on the right was torsioned, and its wave latency was prolonged

Methanol intoxication can typically cause damage to pigmented retinal epithelial cells, chiasm, and optic tract. Visual symptoms usually start within 12-48 hours and occur in approximately 50% of cases (10). In our case, visual symptoms emerged in the acute period and progressed to blindness. Pattern VEP study showed severe visual conduction disorder in both eyes (Fig. 4 and Fig. 5).

Distal symmetrical PNP findings were detected in the NCS performed on the case. Laboratory values for PNP etiology were within normal limits. He had no alcohol addiction and no diabetes. For this reason, it was thought that PNP may be due to methanol intoxication. Cases of polyneuropathy due to methyl alcohol intoxication have been reported in the literature (5).

This case showed classical, clinical and imaging findings consistent with methanol intoxication. Contrary to the literature, findings consistent with loss of sense of taste and decreased cognition were observed.

Conflict of interest

The authors declared no conflict of interest.

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None to declare.

Authors' contributions

Concept: F.Y.C., M.F.Ö., Design: F.Y.C., M.F.Ö., Data Collection or Processing: F.Y.C., Analysis or Interpretation: F.Y.C., M.F.Ö., Literature Search: F.Y.C., Writing: F.Y.C.

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