Acute pancreatitis due to methanol toxicity during the COVID-19 pandemic

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Abstract

Acute methanol poisoning may be associated with a variety of symptoms, such as abdominal pain, blurred vision, loss of consciousness, and acute pancreatitis. We present a 37-year-old man with acute pancreatitis due to methanol poisoning during the COVID-19 pandemic.

K E Y W O R D S

blindness, COVID-19, methanol poisoning, pancreatitis

1 | INTRODUCTION

Methanol is an organic solvent, mainly used for industrial purposes, obtained by fermentation of wood. It is colorless, volatile, and slightly sweeter than ethanol.¹ It is used in materials such as windshield washers, antifreeze, carburetor cleaners, copier inks, perfumes, food heaters, and other fuels.² Acute methanol poisoning may be associated with a variety of symptoms, such as headache, vomiting, abdominal pain, blurred vision, and loss of consciousness. However, in more severe cases, patients may develop blindness, severe metabolic acidosis, coma, and eventually death,³ and despite advances in diagnosis and treatment, the mortality rate remains high.⁴ Pancreatitis is a disease in which pancreatic enzymes cause damage to pancreatic tissue and lead to dysfunction of the gland as well as distant organs and systems.⁵ Gallstone disease, smoking, and excessive alcohol consumption are known to be risk factors for both acute and chronic pancreatitis. However, about 20% of pancreatitis cases are idiopathic, and the basis is not yet known.^{6,7} Although ethanol drinking is one of the major causes of acute and chronic pancreatitis, little is known about the incidence of acute pancreatitis following methanol poisoning.⁸ As far as we know, acute pancreatitis following methanol poisoning is very rare and, so far, is limited to case reports and case series in two papers.^{8,9}

Misconceptions and unrealistic news about the effectiveness of different materials, such as alcohol and opium,

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in preventing the COVID-19 pandemic, has quickly spread to the virtual public media. Alcoholic beverages, on the contrary, are federally prohibited in Iran. Consequently, alcoholic beverages are frequently manufactured, repackaged in popular brand containers, and illegally sold. Following the COVID-19 pandemic in Iran, a combination of these factors resulted in a high number of methanol poisoning deaths. In Iran, only industrial alcohols (containing methanol and other toxic alcohols) have been legally marketed throughout the country.^{10,11} Here, we present a 37-year-old man with acute pancreatitis due to methanol poisoning following the COVID-19 pandemic.

2 CASE PRESENTATION

On January 21, 2021, a 37-year-old man, with dizziness, headaches, nausea and vomiting, and abdominal pain, was referred to the emergency room of a local hospital in Mazandaran Province, northern Iran. Standard treatment was initiated for the patient based on his clinical symptoms, the results of initial tests, including blood sugar (BS) >250 mg/dl, triglycerides (TG) >6000mg/dl, and amylase of 119 U/L (Table 1), and his past medical history of diabetes mellitus, with the diagnosis of diabetic ketoacidosis and pancreatitis.

On the second day, the patient's symptoms did not improve, and blurred vision, severe restlessness, followed by a decrease in the level of consciousness were added to his symptoms, and due to the progression of symptoms despite treatment procedures, he was referred to our medical center, which was better equipped. On the initial examination, the pupils were dilated and reactive to the light, and his family reported a history of alcohol consumption in the last 48 h, which had not been mentioned due to fear of legal punishment.

Because of the decreased level of consciousness, to support the airway, he was immediately intubated. Unfortunately, the patient had a cardiorespiratory arrest during intubation, for which a resuscitation procedure was performed, and his cardiac rhythm then returned to a sinus rhythm.

Following the patient's stabilization and removal of the immediate life-threatening risks, a consultation with a toxicologist was requested, and a peripheral blood sample was obtained for further examination, which revealed metabolic acidosis with high anion gap and increased triglycerides and amylase (Table 1).

On an ultrasound of the abdomen, the liver had a coarse echo, but the pancreas had a normal echo and lacked free fluid around it. The brain CT scan showed bilateral and symmetric putaminal hypodensity that probably reflects acute putaminal necrosis with associated hypoattenuation in cerebral white matter (Figure 1).

A serum methanol level was also requested based on clinical signs and symptoms and laboratory findings, with suspicions of methanol poisoning. The serum level of methanol was 60 mg/dl. Thus, the diagnosis of

| Parameter | Normal range | Blood sample in the first center | Initial blood sample in our emergency room | Blood sample 2 days after treatment |
|-------------------|---------------------------|----------------------------------|---|-------------------------------------|
| Na | 135 – 145 mEq/L | 143 | 134 | 138 |
| K | 3.5 – 5.5 mEq/L | 4.5 | 3.4 | 4 |
| BS | ^{<} 200 mg/dl | 291 | 301 | 198 |
| BUN | 13 – 43 mg/dl | 18 | 38 | 34 |
| Creatinine | 0.6 – 1.2 mg/dl | 1.2 | 0.9 | 1 |
| Amylase | ^{<} 90 U/L | 119 | 191 | 108 |
| Lipase | 10 – 140 U/L | 150 | 185 | 147 |
| AST | 50 – 40 IU/L | 30 | 50 | 43 |
| ALT | ^{<} 45 U/L | 112 | 30 | 31 |
| ALP | 80 – 306 U/L | 150 | 156 | 161 |
| HGB | 14–18 g/dl | 18.5 | 11.4 | 12.4 |
| TG | 50 – 200 mg/dl | 6480 | 1107 | 484 |
| Total cholesterol | ^{<} 200 mg/dl | N/A | 721 | 351 |
| pH | 7.35 - 7.45 | 7.1 | 7.01 | 7.47 |
| PCO ₂ | 35 – 45 mmHg | 23.6 | 20.4 | 34 |
| HCO ₃ | 20 – 28 mmol/L | 8.4 | 5.2 | 25.3 |

TABLE 1 Results of initial tests and comparison with measured values in the first center and 2 days after treatment

Abbreviations: BS, blood sugar; HGB, hemoglobin; N/A, not available; TG, triglycerides.

FIGURE 1 Bilateral and symmetric putaminal hypodensity that probably reflects acute putaminal necrosis (left) with associated hypoattenuation in cerebral white matter (right)



methanol poisoning was definitive, and the severity of his intoxication was considered severe, so the patient underwent dialysis for 4 h and then was transferred to the intensive care unit (ICU). According to increased serum levels of triglycerides and pancreatic enzymes, and the clinical signs and symptoms of acute pancreatitis, a nasogastric tube was inserted to rest the gastrointestinal tract, then the treatment continued with sodium bicarbonate infusion of 1–2 meq/kg, methylprednisolone 500 mg q 12 h/IV up to 3 days, then prednisolone 1 mg/kg up to 2 weeks, erythropoietin (Eprex) amp 10000 IU Bid up to 3 days, vitamin B₁ 300 mg /daily (tablet), folic acid 1mg/kg up to 50 mg every 4 h up to 24 h, then 10 mg daily up to 1 month (tablet), and pantoprazole amp 40 mg Bid.

The patient had a history of consuming alcoholic beverages regularly (more than once a week) in the last few years, but no history of similar poisonings. The metabolic acidosis was corrected 2 days after starting treatment (Table 1), and he was admitted to the ICU for 5 days and then to the poisoning ward for 2 weeks. Finally, the patient was discharged in a relatively good general condition with visual impairment in the form of reduced visual acuity to the point of hand motion from a distance of 1.5 m, allowing him to walk with a cane on February 9, 2021. This research was carried out in accordance with the principles outlined in the Helsinki Declaration. Also, CARE guidelines and methodology have been followed in this study.

3 | DISCUSSION

Methanol, commonly known as methyl alcohol, is a colorless, flammable liquid compound composed of carbon monoxide and hydrogen ions that is produced by the distillation of damaged wood particles.¹² Methanol poisoning usually occurs after ingestion, but poisoning is also possible through pulmonary respiration and absorption through the skin.² In most cases, methanol poisoning by swallowing occurs accidentally in children and rarely as a suicidal act. Methanol is not toxic in itself, and the real toxin is the metabolite produced in the human body, formic acid, which has a half-life of about 30 h.¹³ And because of this slow rate of formic acid metabolism, the onset of clinical and laboratory symptoms takes about 6 to 24 h.²

Although methanol poisoning due to illicit and domestic alcohol consumption is a major medical problem worldwide,^{14,15} common misconceptions about the protective and therapeutic role of alcohol consumption for COVID-19 have unfortunately added to this public health problem in Iran. As a result, due to the ban on alcohol consumption in Iran and the availability of home alcohol, which is sometimes contaminated with methanol, its consumption has increased, which leads to an increase in the prevalence of methanol poisoning.¹⁶

When swallowed or inhaled, methanol initially has a narcotic effect, followed by an asymptomatic period of approximately 10–15 h.¹⁷ After this period, methanol may produce abdominal pain, nausea, vomiting, headaches, dizziness, lethargy, chest discomfort, dyspnea, visual disturbances, and metabolic acidosis.¹⁸ Visual disturbances can range from blurred vision to complete blindness.¹⁹ Methanol toxicity can lead to coma and death from respiratory or cardiac arrest. In one study, symptoms of blurred vision, headaches, dizziness, nausea, and skin problems were reported in teachers' aides who were exposed to duplicating fluid containing 99% methanol while working with "spirit duplicators".²⁰

Our reported patient was a 37-year-old man who was referred to our hospital 24–48 h after consuming methanol with clinical symptoms of abdominal pain, nausea, and vomiting, followed by blurred vision and a decreased level of consciousness. Appropriate treatment was started for him by examining the serum level of methanol and diagnosing methanol poisoning. His clinical signs and symptoms, and serum levels of amylase and lipase enzymes also suggested methanol-induced pancreatitis. Finally, the patient was hospitalized for 5 days in the ICU and 2 weeks in the poisoning ward, before being discharged with partial recovery and ocular complications.

Acute pancreatitis is a common gastrointestinal cause of hospital admission.⁶ Approximately 8% of acute pancreatitis leads to chronic pancreatitis, which reduces patients' quality of life and is also associated with an increased risk of metabolic disorders, such as diabetes and osteoporosis, as well as pancreatic cancer.²¹ Therefore, identifying modifiable risk factors for pancreatitis is thus of great importance to preventing pancreatitis and reducing related diseases and subsequent economic burdens. Since the mechanisms of ethanol-induced acute (and chronic) pancreatitis have not yet been fully elucidated,⁸ it is not surprising that there is very little data on the pathophysiology of pancreatitis following methanol poisoning.

The presence of metabolic acidosis with high anion gap, confusion, and visual disturbances should lead the physician to consider methanol poisoning.²² The fact that early complaints are similar to those of other diseases and that access to laboratory tests used in diagnosis (serum methanol, formate level, and osmolality analysis) is not always possible make diagnosis difficult.²³

Dialysis and ethanol or fomepizole are frequently used in treatment, and if treatment is delayed or inadequate, the mortality rate can be as high as 40%, and even if patients survive, poisoning can lead to permanent blindness and long-term effects on their central nervous system.⁴

4 | CONCLUSION

Although little is known about the incidence of acute pancreatitis following methanol poisoning, according to this report and similar reports cited in the text,^{8,9} acute pancreatitis can also occur following methanol poisoning. Therefore, it is important to consider acute pancreatitis in the process of diagnosis and treatment of patients with methanol poisoning in order to prevent its complications by timely diagnosis and treatment of patients, particularly following the COVID-19 pandemic in our country.

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CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

AUTHOR CONTRIBUTIONS

ZZ involved in the interpretation and collecting of data and editing of the manuscript. MS involved in writing, editing, and preparing the final version of the manuscript. MF and ESB involved in critically revising the whole manuscript. HA and RT were responsible for collecting data and submitting the manuscript. All authors reviewed the paper and approved the final version of the manuscript.

CONSENT

Informed consent for publication of this case report was taken verbally from the patient.

DATA AVAILABILITY STATEMENT

The data are available to the corresponding author and can be obtained upon request.

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