

Case Report: Methanol Poisoning as a Trigger for the Presentation of Diabetes Mellitus: A Case Report



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ABSTRACT

Background: The clinical and laboratory manifestations of methanol poisoning and Diabetic Ketoacidosis (DKA) might be very similar.

Case Presentation: A 15-year-old male, who lived in Mashhad, Iran, was referred to a medical toxicology center because of the loss of consciousness following the consumption of home-made alcohol. Laboratory tests revealed metabolic acidosis, the high blood levels of methanol, and ketonuria and glucosuria in urine analysis. The treatment of DKA and methanol poisoning was started simultaneously.

Conclusion: Methanol poisoning, as physical stress or because of pancreatitis as a complication, could be a trigger for diabetes in susceptible individuals, and physicians should be aware of the similarity of presentation of these situations.

1. Introduction

Methanol poisoning often occurs because of the consumption of home-made alcohol by young people (Teenagers). On the other hand, the most common endocrine disease in young people is diabetes, and DKA is a common cause of hospital admissions in this age group. Various sources are mentioned as predisposing factors in the development of DKA, one of which is drug overdose and exposure to toxins. In this study, we evaluated the

clinical and laboratory manifestations of a patient with DKA following methanol poisoning.

2. Case Report

A 15-year-old male patient was admitted in MTC with nausea and vomiting and abdominal pain 1 day after the consumption of home-made alcohol in a party. He complained of blurred vision on the afternoon of the same day. In his family history, father suffered from insulin-dependent diabetes and mother suffered from iron deficiency anemia using ferrous sulfate. Before referring

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to the hospital, the patient had an attack of generalized tonic-clonic seizures. On admission, the patient had localized painful stimulations, verbal response as groans and non-reactive to light, and mydriatic pupils. He had also acetone odor of the breath. Physical examination revealed systolic/diastolic blood pressure:90/60 mmHg, axillary temperature:37°C, respiratory rate:30/min, and pulse rate:130/min. Electrocardiography showed sinus tachycardia and gasometry findings; PO₂:90 mmHg, O₂ Sat:90%, pH:7.1, PCO₂:15 mmHg, and bicarbonate:10mEq/L. Urine analysis findings showed blood:+++, ketone:+++, and glucose:+++. Table 1 shows other laboratory tests.

In addition to the routine treatment of DKA, the required attempts for methanol poisoning were made, including folic acid [1], bicarbonate sodium, and ethanol based on toxic serum level of methanol; he underwent 4 hours hemodialysis as well. The following findings were reported after hemodialysis: Serum Methanol:35mg/dL, Serum Ethanol:100mg/dL, Gasometry: PO₂:90 mmHg, O₂ Sat:90%, pH:6.9, PCO₂:20mmHg, and bicarbonate:8mEq/L.

The patient underwent hemodialysis twice because of the high serum level of methanol and remained metabolic acidosis; unfortunately, he died because of ventricular tachyarrhythmia during the second hemodialysis. The deceased person was autopsied on the next day and toxicology tests reported no detectable level of ethanol and methanol, while the serum level of isopropyl alcohol was 10 mg/dL.

3. Discussion

Methanol poisoning is relatively common in Iran [2] because of the consumption of home-made alcohol and a high mortality rate is anticipated if the beginning of treatment is delayed; the mortality rate of DKA with treatment is (1-3%) [3]. A patient with diabetes with the previous recurrent attacks of DKA was reported, who experienced a refractory DKA because of methanol poisoning [4]; but, there is no report of the first presentation of diabetes because of methanol poisoning. Methanol poisoning may lead to metabolic acidosis and methanol metabolites (formaldehyde and formic acid) may cause severe damage to the optic nerve and central nervous system.

High osmolar gap is expected in this poisoning; on the other hand, the increased metabolism of fatty acids and ketoacids (acetoacetate and beta-hydroxybutyrate) accumulation lead to diabetic ketoacidosis [5]. This situation is more common in type 1 diabetes because of the discontinuation of insulin or physical stress such as pancreatitis [6], gastroenteritis [7], and myocardial infarction [8], which a transient increased need to insulin occurs in these conditions. Clinical manifestations (for example, nausea and vomiting, abdominal pain, impaired visual acuity, dyspnea, tachycardia, and tachypnea) in these situations are very similar; there are also several similarities in paraclinical manifestations such as hyperglycemia, metabolic acidosis, and hyperamylasemia [9].

The history of home-made alcohol use, the high serum concentration of methanol, in addition to metabolic

Table 1. Biochemical tests of the patient with DKA and methanol poisoning

Variable Name	Amount
Na	130 (meq/L)
K	5.2 (meq/L)
Ca	9 (meq/L)
pH	2.5 (mg/dL)
Serum methanol concentration	70 (mg/dL)
Serum Iron	150 (mcg/dL)
Serum amylase	450 (U/L)
Blood sugar	350 (mg/dL)
BUN	40 (mg/dL)
Cr	1.8 (mg/dL)
CPK	15000 (U/L)

Table 2. Comparison of clinical signs and laboratory of methanol poisoning and DKA

Variable Name	Methanol Poisoning	DKA
Abdominal pain, nausea, vomiting	+	+
Visual impairment	+	+
Hyperglycemia	+	+
Ketonuria	+	+
Glycosuria	-	+
Elevated serum amylase	+ (Pancreatic origin)	+ (Salivary gland origin)
Metabolic acidosis (high anion gap)	+	+

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acidosis, seizure, and mydriasis [10] strongly suggested methanol poisoning, while the presence of hyperglycemia, metabolic acidosis, glucosuria, and ketonuria was consistent with DKA. Hypotension and tachycardia were explained in the context of dehydration in DKA and metabolic acidosis in both situations.

Relatively high potassium levels could be as a result of progressive metabolic acidosis as well as an acute renal failure, while the amount of total body potassium might decrease. The rise of Blood Urea Nitrogen (BUN) and Cr of serum was consistent with dehydration in DKA and also could be considered a complication of methanol poisoning. The seizure could explain high Creatine Phosphokinase (CPK) levels; although these amounts could not explain the occurrence of renal failure because of rhabdomyolysis.

Hyponatremia may be linked to increased blood sugar [11], as measured sodium of serum reduces in hyperglycemia. While the normal sodium of serum could be a sign of severe dehydration. The simultaneous treatment of these serious conditions can lead to unexpected problems. Although bicarbonate therapy is a verified treatment of metabolic acidosis in methanol poisoning [12], it is not recommended routinely in DKA because cardiac dysfunction, decreased tissue oxygenation [13], worsening of hypokalemia, and increased risk of cerebral edema, especially in children, are anticipated theoretically and practically. While the high serum concentration of methanol (70mg/dL), the presence of metabolic acidosis, coma, and convulsion indicated hemodialysis in this case, in the context of DKA, it could result in the worsening of dehydration as a risk factor for the deterioration of acidosis [14]. Ethanol-induced Ketoacidosis [15] was another differential diagnosis although it can be rejected because of the presence of hyperglycemia.

It is noticeable that a detectable amount of isopropyl alcohol in blood sample taken from the femoral vein is the result of reverse production of this compound of blood ketone; this interaction would continue for several hours after death [16]; likewise, it is worth mentioning that isopropyl alcohol intoxication does not lead to metabolic acidosis [17] and this compound is eliminated well by hemodialysis [18].

4. Summery

Physicians must be aware of the similar symptoms and signs of these conditions; DKA and methanol poisoning and, in suspicious cases, appropriate diagnostic tests must be performed. Given some interactions between the treatment methods of these common conditions, precise follow-up is recommended. These conditions are compared in Table 2.

Ethical Considerations

Compliance with ethical guidelines

The study protocol was in conformity with the ethical guidelines of the 1975 Declaration of Helsinki, revised in 1983. Private information, including name, surname, and burial permit was removed from the data sheet to comply with ethical concerns.

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Author's contributions

All authors contributed in designing, running, and writing all parts of the research.

Conflict of interest

The authors declared no conflict of interest.

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