Case Report
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Methanol poisoning, case report

Abstract

Methanol, also known as methyl alcohol or wood alcohol, is considered the simplest alcohol, is used in industry as a component of various household substances (antifreeze, solvent and fuel). While the sporadic cases are now described, its mortality is usually very high. Its use in the production of adulterated alcoholic beverages has resulted in several outbreaks of acute poisoning. Toxic exposure occurs predominantly orally, although inhalation or transdermal absorption may lead to poisoning. The susceptibility to the toxic effects of methanol is variable, but eating a small amount can lead to severe poisoning. Both accidental poisoning for suicide has a high morbidity and mortality, largely due to the difficulty in confirming the diagnosis is that which delays treatment should be initiated at the slightest suspicion of the table without waiting for laboratory confirmation, since the precocity of the administration of therapeutic measures is crucial to limit damage and allow a potential recovery of the patient. Here is a case of methanol poisoning treated in the Emergency Room of a second level hospital in Mexico City.

Keywords: methanol, intoxication, diagnostic, treatment

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Case report

A 42-year-old male patient, veterinary occupation, a history of addiction to cocaine, which according to his relatives have not consumed for years; to the- positive coholism from the age of 18, with levels of drunkenness every 8 days. Current condition: Table of 2 days of evolution after cleaning skins at work with unspecified substance, as well as ingestion of unspecified amount of alcoholic beverages. Start with anxiety, irritability, nausea you are, vomit, blurred vision and diplopia; It is drive- Initially by private doctor without data more precise. The table increases, presenting- sudden loss of bilateral vision and attending to the first level of care unit, from where is referred On admission to the emergency room, he presented 3 tonic-chronic-generalized convulsive coughs, which are jugulated with intravenous diazepam. TO Postictal exploration: distal cyanosis, mydriasis bilateral (Figure 1), no jugular ingurgitation, cardiopulmonary and abdomen without commitment, extreme midades with bilateral Babinski. Blood pressure: 118/79, heart rate 100 x', frequency Respiratory 16 x', temperature 36.4, destrostix 180. Advanced airway management begins, crystalloid solutions, impregnation with phenytoin mg, applying for paraclinics (biometrics, blood mica, gasometry), cranial tomography neo and electrocardiogram. Entry laboratory with hb 15.0g/dL, leukocyte cough 13,000, glucose 91mg/dL, creatinine 3.0mg/dL, Na 143mEq/L, K 5.3mEq/L, chlorine 113mEq/L. BUN 18mg/dL, lipase 589U/L (Table 1).

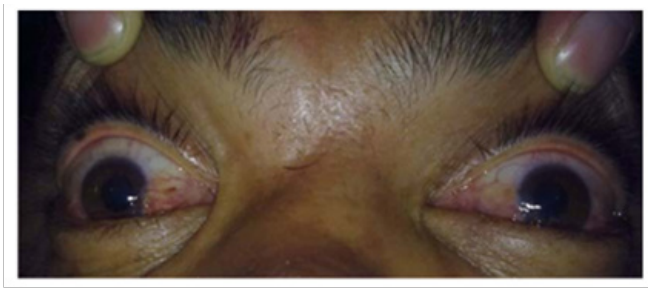

Figure 1 Bilateral mydriasis in the patient intoxicated by methanol.

The report of the skull tomography is: Edema disseminated brain, compatible with the meta Bolic and/toxic (Figure 2). The evolution continues being torpid, develop- frank hematuria and persistent metabolic acidosis; the latter despite aggressive management with crystalloid and bicarbonate (Figure 3).

Table 1 The gasometric reports are presented below

	Entry	1hour	3 hours	5 hours
pH	6.8	6.8	7.01	7.17
pCO_2	35	18	2. 3	eleven
pO_2	64	337	309	251
HCO_3	Not detectable	7.7	6.4	4
EB	Not measurable	-19.3	-25.3	-24.5

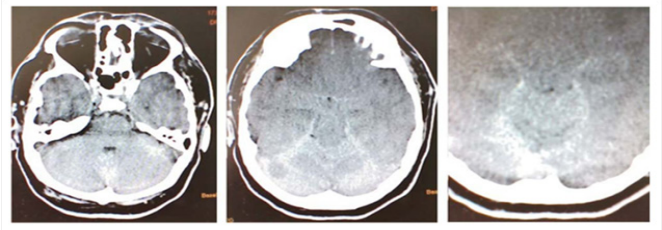

Figure 2 Three tomographic slices showing signs of severe cerebral edema.

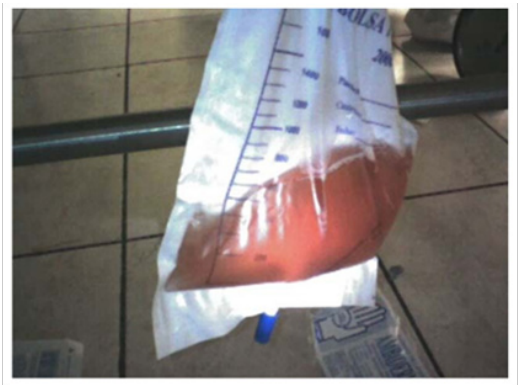

Figure 3 Hematuria developed by the patient intoxicated by methanol.

Given the antecedents of being exposed recently to chemicals and alcoholic beverages unknown, associated with neurological deterioration with sudden visual conditions and metabolic acidosis

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with persistent high anion gap, the possibility is considered Methanol intoxication. By virtue of not to perform hemodialysis or count on the antidote of election, femepizol, it is decided to start infusion by son- nasogastric dose of 40% ethyl alcohol at doses 0.6g/kg in bolus, with a maintenance of 100mg/kg/h, as well as dosage adjustment of bicarbonate and Adjuvant treatment with folic acid. Previously to At the beginning of the ethanol a sample had been sent to

blood determination of methanol to a laboratory private. The patient is admitted to the Inpatient Care Unit tensivos maintaining hydric management, ethanol by nasogastric tube and mechanical ventilatory support. He

establishes an APACHE score of 40 points, with a potential mortality of 91.1%. The evolution is torpid, with poor response to management, control gasometry with persistent acidosis te and EB of -24.5. At 24 hours after admission, the patient presents cardio respiratory arrest that does not reverse after 20 minutes of advanced resuscitation maneuvers, giving by deceased. The *post-mortem* report delivered after a week mentions blood methanol levels of 79mg/dL (Recommended level: Not detectable).

Discussion

Methanol (CH_3OH) is an aliphatic alcohol, liquid colorless and volatile at room temperature. By itself It is harmless, but its metabolites are extreme toxic. Its use is usual in industrial products laboratories and in the home itself, existing also a clandestine use as a substitute in drinks alcoholic. The most common route of acute intoxication da is the oral. There is a great variability in the dose that is considered toxic and lethal, although most the authors consider the latter as 30mL of pure methanol.[1–8] Methanol is rapidly absorbed from the digestive tube, giving plasma peaks at 30-90minutes The serum half-life ranges between 14 and 30hours; is distributed freely and its volume of distribution is 0.6-0.7L/kg, does not bind to proteases. A small amount of methanol is found in the exhaled air of normal subjects by average production endogenous In untreated patients, the elimination renal nation is less than 5% and the rest is eliminated by hepatic biotransformation, oxidizing through the alcohol dehydrogenase and generating formaldehyde, the which is converted, by the aldehyde dehydrogenase, into formic acid and, subsequently, through an oxy-folate-dependent production, in charcoal anhydride co and water. In the toxokinetics of methanol, it is interesting Consider that ethanol has an affinity for

the alcohol dehydrogenase between 10-20 times higher to methanol, hence its effectiveness as an antidote. The toxic effects of methanol overdose should be to the formation of these two metabolites, being the Formica is the main responsible for toxicity ocular and high anion gap metabolic acidosis. In advanced stages of intoxication, it can marse lactate as a consequence of the inhibition Toxoplasma by format and tissue hypoxia, exacerbated stopping metabolic acidosis. Methanol by itself it is not toxic; the toxic action depends on the amount of toxic metabolites that form.[9–12] The characteristic data of metastatic poisoning nol is the metabolic acidosis with anion gap> 16 mmol /L, so it should be suspected in all the situations in which, inexplicably, there is acid- Metabolic syndrome with anion gap increase. Own Acidosis can be caused by, through oxalic acid, myocardial depression and acute tubular necrosis. The alcoaldehyde, and the glycolic and glyoxylic acids contribute and in both the depression of the Central Nervous System as to renal toxicity, with focal hemorrhage, cortical necrosis, dilation of proximal tubules and formation of calcium oxalate crystals.[13–15]

The onset of symptoms is extremely variable and depends on the dose of methanol, the of incorporation and of the route of entry, starting between 30 minutes and 72hours, although the usual is that they appear in the first

mere 12-24hours that is the time needed for the biotransformation of methanol.[16]

The symptoms and signs of poisoning can be different repercussions and grouped into:

A. Central Nervous System: in mild intoxication or moderate headache, dizziness, lethargy, ataxia or a state similar to alcohol intoxication. In cases severe seizures, coma and edema may appear cerebral. Selective neurotoxicity is the result of the hypoxia that formsic acid produces after cytochrome oxidase inhibition.[17,18]

B. ocular involvement: there is sudden loss of visual acuity and papilledema with irreversible blindness

C. Versible by atrophy of the optic nerve. They can appear hear nystagmus and alterations of pupillary reflexes lares. In addition, loss of life may develop ion, mydriasis with loss of photomotor reflex and papilla edema.[17–19]

D. Gastrointestinal: methanol is slightly irritant tante, which conditions nausea, vomiting and pain abdominal; If the picture progresses, they can be llar clinical and enzymatic data of pancreatitis acute twenty Methanol does not cause pulmonary toxicity, except to in case of inhalation. Dyspnea and tachypnea pre in these patients usually translate a disorder metabolic or neurological, or secondary aspiration to alterations in the level of consciousness.[20–22]

The findings of the cranial tomography of this case (massive cerebral edema) were not the usual this type of intoxication, where they are often tell the hemorrhagic necrosis of the basal ganglia them (fundamentally in the putamen and caudado); is- These areas are particularly sensitive to hypoxia for its high demand.[23–25]The diagnosis will be confirmed by the determination in blood or urine of methanol, condition with which in the majority of hospitals in our area is counted routinely, so it must prevail Clinical suspicion of metabolic acidosis

of anion gap high.[26–28] The severity of the poisoning is classified according to methanol levels in the blood, which is corrected they link with the clinic in the following ways:[29–32]

Mild poisoning

methanolemia (methanol in blood) less than 10mg/dL. Feeling fatigue, nausea, epigastralgia, headache and visual perception accommodation or accommodation.

Moderate intoxication

Metanolemia between 10-50mg/dL. Vomiting, expressions of drunkenness, especially if the intoxication is mixed (ethanol-metanol), cold and sweaty skin, blurred vision and presents tachypnea, trying to make breathing compensation of metabolic acidosis.

Severe poisoning

Metholaemia greater than 50mg/100mg/dL. Comatose arrest, rapid breathing and superficial, seizures, peripheral and central cyanosis, hypotension, papilledema. Methanolemias greater than 100mg/dL are consider an lethal.

Within the differential diagnosis, we must consider all those entities that also produce an increased anion gap metabolic acidosis (in- poisoning by salicylates, ethylene glycol, ketoacidosis alco-wind, etc.). Currently there are commercial kits based on the detection of alcohol dehydrogenase and alcohol oxidase, which can help make a diag-rapid assessment of the intake of pure methanol (without ethanol).[33–36] Evolution correlates better with the serious of acidosis than with the serum concentration of methanol The prognosis is better if the dose ingested has been fractioned in time, if simultaneously alcohol has been ingested or if it is applied early- the proper treatment. Given the high lethality of these cases, it is recommended an intensive approach, delaying as little as possible the attention Therapeutic measures include treatment Symptomatic treatment of complications, correction of acidosis, the administration of ethanol to reduce the transformation of methanol into its metabolites

toxic and the extraction of them with dialysis.[37,38] Adequate respiratory support must be provided with advanced airway management and ventilation mechanical if they are necessary. They should be administered intravenous procedures to maintain a balanced balance electrolyte and adequate diuresis. Gastric lavage is only effective within two first hours postingestion. Activated carbon, laxatives or cathartics are not effective in intoxicating methanol.[38,39]

Initially it is advisable to start the administration of its antidote (ethanol) when we suspect a significant methanol poisoning (>30 mL in adults and> 0.4 mL/kg in children) and/or in the presence of a metabolic and /or clinical acidosis, although not let's not know the dose ingested nor the methanol levels. Ethanol is considered of choice, since it is also metabolized by the alcohol dehydrogenase, although that with an affinity 10 times higher, produces a competitive inhibition blocking the formation of two metabolites (formaldehyde and formic acid), responsible for toxicity. So that this effect tera- be exercised in an appropriate manner, they must be maintained in plasma ethanol levels of 1-1.5mg/mL (100-150mg/100mL). This treatment requires the monitoring of plasma ethanol values, since there are interindividual variations in their tabolism and there is some difficulty in maintaining the adequate standards, especially when hemoglobin is dialysis together with the administration of ethanol.[40–42] Indications for the administration of methanol are: plasma methanol levels above 0.2 g/L, intake greater than 0.4 mL/kg of weight, or any symptomatic patient with metabolic acidosis with AGAP increased, until the confirmation is confirmed toxication Intravenous administration of ethanol is more selective It is oral, although it is irritating venous and may produce superficial thrombophlebitis. The optimal solution it must contain 10% ethanol and 5% dextrose. Can administer 20-30% oral ethanol; although they have used higher doses, these can produce gastritis Ethanol can be administered orally or intravenous (centrally because of its high osmolarity) dad). For either of the two ways it is necessary dilute absolute ethyl alcohol; if it is orally it dilute with water until reaching a concentration of 20-30%, and in the case of the intravenous route is diluted in glucose serum until reaching 5-10%. There are vaformulas to calculate this handling; a pattern quite practical could be this: for a average adult of 70 kg. Impregnation dose: 1 mL of absolute ethanol per kg.

Oral route: 40% whiskey: 175 mL of whiskey plus 175 of water so that the final solution has a 20% ethanol concentration.

Intravenous route: 70 mL of 100% ethanol + 630 cc of glucose serum so that the concentration of Ethanol is 10%, to pass for 15 minutes.

Maintenance dose 0.16 mL/kg/hour. This dose must be multiplied (0.20-0.40 mL/kg/h) during the time that keep the hemodialysis, if applicable. Whether it's about a chronic alcoholic, the maintenance dose. The concentration is 0.2 mL/kg/h. The maintenance dose should be followed until the concentrations Methanol levels are below 20 mg / 100 mL, and if this data is not available, until that the patient has a pH>7.3 without help from the baking soda. Another alternative could be: 0.16mL / kg / hour x 70 kg=11.2 mL/hour of 100% ethanol.

Oral route: 40% whiskey: 28mL / hour of whiskey+28 mL/hour of water. (Ethanol 20%).

Intravenous route: 11.2mL of 100% diluted ethanol two in 100.8mL of glucosate, giving 112mL of that mixture at the time (10% ethanol).

A more practical option is observed in Table 2 Today another antidote is available, the fomepizol (4-methylpyrazole), a medicine approved by the *Food and Drug Administration* (FDA) and that acts competitively inhibit ADH, with an affinity for it 80,000 times higher than methanol and 8,000 times higher than that of ethanol. It has no hepatotoxicity and has some advantages over ethanol: no increases sedation on the patient, has less risk of hypoglycemia, less excess problems of fluids, fewer problems in unstable patients hemodynamically, easy handling and administration, can be administered both orally and intravenous The great disadvantage of fomepizol is its ele-cost, since a treatment costs between 3,000 and 6,000 euros. The impregnation dose is 15 mg/kg and the maintenance of 10mg/kg every 12 hours for 2 days and then 15mg/kg every 12hours, until the normalization of the patient. It will be administered diluted in serum and pass in 30 minutes each dose.[43–51] The use of bicarbonate not only improves the acimetabolic dose, but it prevents the formation of formic acid. Your infusion should be started when bicarbonate is less than 18mEq / L. In ocasions, the necessary amount of bicarbonate is elevada (500-1,000mEq / day), since with relative often patients have a pH lower than 7.0, which does not respond to treatment with bicarbonate- to Clinical studies have shown improvement in vision after correction of acidosis, due to movement of formic acid outside the cell: The do- sis are 1-2meq/kg and its use will continue according to the base deficit.[52,53] Folic acid supplements are required, the they increase the degradation of formic acid in H_2O and CO_2 , thus reducing the severity of the ocular sions. Folate is a cofactor of catabolism formic acid and, therefore, is of special importance to provide folate supplements, so all to alcoholic patients, where they can there is a previous deficit. It has been shown that it is effective if administered up to 10 hours after the ingestion of methanol. Its administration is recommended in all patients, at a rate of 50mg per intravenous, every 4-6hours IV diluted in glucocorticoid serum sado.[41–54] Among the measures aimed at activating the extrac-of methanol, hemodialysis is the most useful, because that purifies both methanol and its metabolites.

Table 2 Management with ethyl alcohol orally for the patient with methanol poisoning

Load	Alcohol al 43%1.8 mL/kg	Alcohol al 10% 7.6 mL/kg
Maintenance in non-drinker patient	0.2 mL/kg/hour	0.83 mL/kg/hour
Maintenance in patient drinker	0.46 mL/kg/hour	1.96 mL/kg/hour

Forced diuresis is not effective and peritoneal dialysis It hardly has eliminator effect. Its indications are:

Methanol concentrations>50mg/100mL (0.5g/L), metabolic acidosis (pH<7.25) refractory to, visual manifestations, depression of the of consciousness and acute renal failure. The he Modiálisis should be followed until the methanolemia is lower than 29mg/100mL or the pH is maintained without bicarbonate help above 7.3. In case of not having this information, it must be continued during 10-12 hours. During hemodialysis, follow-up administering ethanol, although the maintenance dose is superior. (0.2-0.4 mL/kg/h).

Conclusion

Methanol poisoning is still present a problem of great toxicological interest, given severe metabolic acidosis with elevated anion gap what it causes and how quickly it can lead to complications and even death. This acidosis, as well as the sudden manifestations neurological, should guide us the suspected diagnosis It is crucial to establish it in an early na, in order to establish an early intensive treatment, since both the antidotes used and the handling hemodialysis lose much of their effectiveness when Most of the methanol has been metabolized to its toxic products.

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Conflicts of interest

The author declares no conflict of interest.

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