International Journal of Medical and Pharmaceutical Case Reports

11(5): 1-4, 2018; Article no.IJMPCR.47253
ISSN: 2394-109X, NLM ID: 101648033

Methanol Toxicity Presenting as Acute Abdomen: Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Author YA designed the study. Author MI performed the statistical analysis and wrote the protocol. Author MS wrote the first draft of the manuscript. Authors YA and MI managed the analyses of the study. Author MHED managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJMPCR/2018/v11i530094
Editor(s):
(1) Dr. Daniel Laubitz, Assistant Research Scientist. Steele Children's Research Center, Dept. of Pediatrics / Gastroenterology and Nutrition, Arizona Health Sciences Center, University of Arizona, Tucson, USA.
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(2) Javier Rodríguez Villanueva, University of Alcalá, Spain.
Complete Peer review History: http://www.sdiarticle3.com/review-history/47253

Case Study

Received 03 December 2018
Accepted 16 February 2019
Published 13 March 2019

ABSTRACT

Background: Acute methanol poisoning is a fatal illness. Several Atypical presentations could make it difficult to suspect the diagnosis.
Case Report: A 50 years old male known chronic alcoholic presented to the emergency department with severe acute abdominal pain nausea and vomiting. He rapidly deteriorated within 2 hours to develop confusion and seizures, was found to have double gap severe metabolic acidosis and elevated serum methanol. The patient was managed with Continuous Renal Replacement Therapy (CRRT) and supportive measures. He gradually improved and was discharged with no neurological or visual complications.

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Conclusion and Recommendations: Acute methanol toxicity should be expected if the alcoholic patient develops a rapid neurological deterioration and shows double anion gap acidosis.
Health authorities should provide the serum formic acid test for diagnosis and the fomepizole as a preferable antidote. Until then, supportive treatment, intravenous ethanol and CRRT should be immediately started in these patients.

Keywords: Methanol toxicity; continuous renal replacement treatment; double anion gap acidosis.

1. BACKGROUND

Also known as wood alcohol, methanol is a component of washing fluids, antifreeze formulations, photocopying fluids, perfumes, and paint removers [1]. Methanol is a clear, colourless solvent used in anti‑freeze solutions, varnishes, paint, and fuel. Methanol poisoning may occur due to accidental or suicidal ingestion of these, or it may occur due to the consumption of adulterated alcoholic beverages. The lethal dose of methanol is around 1 g/kg [2]. Methanol by itself is largely non‑toxic and toxicity results due to its metabolism into formaldehyde and formic acid in the liver. This also accounts for the time lag between substance ingestion and clinical manifestation which is considered to be around 6‑24 h. The patient may present with nonspecific symptoms as nausea, vomiting, headache, dizziness, and weakness. Clinically, the patient may present with several complications like high anion gap metabolic acidosis, visual disturbances, and neurologic deficit. If not treated early, the patient may develop coma and death can occur [3].

2. CASE REPORT

50 years old male, a heavy smoker, and alcoholic for 10 years, has a history of hypertension and was on no regular treatments, with no other chronic illnesses or drug intake. The patient Presented toOhud hospital emergency room (ER) with a complaint of severe epigastric pain, repeated vomiting for 2 days. There was no history of fever, hematuria, seizures, weakness, decreased sensations, or visual changes. At first om presentation to ER, the patient was fully Conscious, oriented, alert, GCS 15/15, temp.: 36.7 C, pulse 84 bpm, BP 187/127 mmhg and SpO2 was 99%on room air. Chest, cardiovascular and neurological examinations were all normal. Abdominal examination revealed Moderate epigastric tenderness. His complete blood count on presentation was; WBC 9.52 RBC 12.4 HB 15.7 PLAT 256 . Biochemistry: Alp 157, Ck 219, AST 162 ALT 170 ALKP 157 GGT 256 ALB 40.1 bil11.9. Random blood sugar was RBS 227mg/dl and serum Amylase was 245 mg/dl . Kidney function test showed blood urea nitrogen of 1.9 mmol/L, creatinine of 132 mmol/L, serum sodium 144 mmol/L, and potassium 5.2 mmol/L. Arterial blood gas revealed pH of 7.17, bicarbonate of 9.9 mmol/L, and paCo2 of 19.5 mm Hg. Anion gap was 15 mmol/L, osmolal gap was 47 mmol/kg (Table 1). ECG was normal. ECHO: good systolic function, mild concentric LVH, normal right side, no pericardial effusion . Thyroid Function Test, Troponin and cardiac enzymes were normal. The Surgeon revised the patient after 2 h for the clinical possibility of acute pancreatitis by that time Patient became tachypneic and tachycardic PR: 130 B/m, RR: 30 B/m, SpO2: 100% room air, U/S abdomen was done and revealed no gallstones but pancreas was not seen, so C-T abdomen was arranged but Patient rapidly deteriorated and became confused GCS: 12/15, PR: 167 bpm, RR: 40 bpm, BP: 210/109 mmhg. He was immediately managed by IV thiamin 100 mg, Tri B amp. (4 amp) IM and Na HCO3 infusion, Intubated, ventilated and transferred to the intensive care unit. Serum methanol levels were sent which were high with the value of 54 mmol/L and Serum Ethanol level was 15 mmol/L, it was revealed later that the patient drank ethanol and methanol together. Serum formic acid test could not be done in our laboratory. 4 hours later while in ICU, the patient developed acute generalized seizure that was aborted by propofol and dormicum. He had another repeated seizure 2 h later and was also aborted by dormicum infusion. CT Brain was done and reported to be normal. Ophthalmologist reported no optic neuropathy. Venous blood gases test (VBG) showed improvement of acidosis after Continuous Renal Replacement Therapy (CRRT) pH: 7.46, pCO2: 22.5, HCO3: 15.8. The patient gradually stabilized and improved (Table 1). He was extubated on the third day of admission and transferred to the general medical word and was discharged after another 2 days. He was back to his normal status with no neurological deficits.

Table 1. Patient serum biochemistry and venous peripheral gases progress throughout his hospital course

	Normal reference range	ER 0 Time	ER after 1H	ICU 4H	ICU 2ED Admission date	ICU 3ED Admission date	On discharge from the hospital
PH	7.35-7.45	7.17	7.15	7.18	7.28	7.46	7.46
Pco2	34-45mmHg	19.5	29.9	35	38.6	22.5	35.2
Hco3	22-26mmol/L	9.9	11.6	13.7	18.2	19.6	24.8
RBS	2.5-6.4mmol/L	12.2	8.8	6.4			6.1
AMYLASE	25-115U/L	245	165		96		65
BUN	2.5-6.4mmol/L	1.9	1.9	1.5	0.5	1.01	0.4
CRE2	49-115Umol/L	132		69.1	90.4	59	58
Na	136-145 mmol/L	144	148	140	136		135
K	3.5-5.1 mmol/L	5.2	5.6	4.2	4.1	2.5	3.6
Cl	98–107 mmol/L	112					103
Calculated osmolality		312.5					
Measured osmolality	285–295 mmol/kg	360					
Anion gap	10–14 mmol/L	15					
Osmolal gap	5–10 mmol/kg	47.5					
ALT	1-37U/L	170		69	56		62
AST	15-41U/L	162	143	79	65		86
GGT		256	204	209			
BILI	5.13-17.1Umol/L	11.9	13.9				
ALB	35-41U/L	40.1		34.09			

3. DISCUSSION

Methanol toxicity effect is caused by its metabolite formic acid. Formic acid inhibits the cytochrome oxidase system resulting in reduced ATP production which eventually causes tissue hypoxia and increased serum lactate [4]. The main source of methanol in our patient was ingestion of cologne with a methanol concentration of 65%. As there is a latent period between methanol ingestion and clinical manifestations which is the time needed for accumulation of formic acid, our patient did not show typical presentation of methanol toxicity on arrival to ER. The main presentation of this patient was unusual, as he presented with acute severe abdominal pain that seemed to be secondary to severe acidosis. Similarly, Kavalcı et al. put gastrointestinal tract complaints and vision problems to the forefront [5]. Neurological deficits in the form of confusion and later seizures were delayed, owing to the time of accumulation of the toxic metabolite. Ophthalmic electrophysiological tests such as ERG and ECP have been reported to be more sensitive indicators of retinal toxicity [6], so their use, even if the funduscopic examination is normal, should be considered. In our patient, both ECP and visual fields were tested and were normal. Despite the high level of serum methanol, the patient did not develop ocular toxicity and was discharged eventually with no visual disturbances or any neurological deficit. This is in agreement with Mathieu et al. as they found that serum methanol concentration was not correlated with visual damages or mortality [7]. Preferable out come in our patient might be due to the early Continuous Renal Replacement Treatment (CRRT), contradictingthe Recent recommendationsthat limit hemodialysis indications [8]. Our findings are in agreement with Jelle L Epker and et al. who believed that in a hemodynamic instable situation high methanol concentrations and methanol-induced derangements of homeostasis are safely and effectively treated with continuous venous hemodiafiltration (CVVH-DF) [9]. The fact that the patient ingested ethanol together with the methanol may playeda role asa self-anti-dote and contributed to the complete recovery. Another study reported a case of a patient who consumed an excessive mixture of methanol and ethanol however he survived the acute methanol poisoning without anylong-term neurological sequelae [10]. We believethat Continuous renal replacement treatment (CRRT)might be better than the usual hemodialysis interrupted cession in removing methanol and its metabolite in a

continuous manner. There is also a case report of a patient who survived acute methanol poisoning without long-term sequelae despite a high serum level of formic acid upon admission after been treated with 10% solution of ethanol, intravenous folinic acid and two 8-hour sessions of intermittent hemodialysis [10]. Intravenous ethanol infusion was not given to our patient nor fomepizole, because they were not available.

4. CONCLUSION

Acute methanol poisoning is a fatal illness. Several Atypical presentations could make it difficult to suspect diagnosis and is related to the time it takes to form the toxic metabolite. It should be expected if an alcoholic patient develops a rapid neurological deterioration and shows double anion gap acidosis.

5. RECOMMENDATIONS

There should be a public education campaign about the sources and consequences of methanol ingestion. Health authorities should provide the serum formic acid test for the proper diagnosis. Fomepizole as a preferable antidote should also be available. Until then, supportive treatment and CRRT should be immediately started in patients with acute methanol toxicity.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline participant consent and ethical approval has been collected and preserved by the authors.

ACKNOWLEDGEMENT

The authors would like to thank Enago for the English language review.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sdiarticle3.com/review-history/47253