Case report

A CASE OF METHANOL INTOXICATION COMPLICATED WITH ACUTE PANCREATITIS, ALCOHOLIC KETOACIDOSIS, AND INTRACEREBRAL HEMORRAGE

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ABSTRACT

Methanol intoxication occurs after accidental or suicidal ingestion occasionally and the toxicity is due to the formation of formaldehyde and formic acid. Methanol ingestion can also lead to acute pancreatitis, a sudden inflammation of the pancreas, and high metabolic acidosis as a complication. Brain injury in methanol intoxication could be ischemia or hemorrhage form. Therefore we'd like to present a report a male with methanol intoxication with several complication such, acute pancreatitis, alcoholic ketoacidosis, and CVA-ICH. The patient presented with hemodynamic instability and severe metabolic acidosis with pH 6.991. The anion gap was 30 mmol/l. Amylase was 187 U/l and CT scan showed intra-cerebral hemorrhage at right fronto temporo parietal lobe with peri-focal edema (volume 48 cc). Patient unfortunately died three days later after force discharge by his families. Brain injury in methanol toxicity is characterized by lesions affecting both basal ganglia and subcortical regions. The lesion could be ischemia/necrosis or hemorrhage, selectively affecting the putamenal bilaterally.

Keywords: Methanol intoxication, acute pancreatitis, brain injury

INTRODUCTION

Methanol also referred to as methyl alcohol is used widely in commercial and industrial. Methanol's toxicity is due to the formation of two toxic metabolites, formaldehyde and formic acid. It is well absorbed from the gastrointestinal tract; with peak levels attained 30 to 90 min after ingestion. Most incidents of toxicity occur after oral ingestion, but significant absorption may also occur through the lungs or skin. The serum half-life after mild toxicity is 14 to 20 h. With severe toxicity, this increases to 24 to 30 h. Following ingestion, highest concentrations are found in the kidney, liver, and gastrointestinal tract, but high levels are also found in the vitreous humor and optic nerve. Most methanol (90 to 95 percent) eliminated by the liver, while renal excretion accounts for 2 to 5 percent; pulmonary excretion is minimal.[1,2]

Acute pancreatitis is a sudden inflammation of the pancreas characterized by a discrete episode of abdominal pain and elevated serum amylase and lipase levels. Depending on its severity, it can have severe complications and high mortality despite treatment. While mild cases are often successfully treated with conservative measures, Methanol ingestion can also lead to acute pancreatitis.[3,4]

Methanol ingestion cause high anion gap metabolic acidosis from the production of formic and lactic acid and CNS disturbance ranging from inebriation and drowsiness to obtundation, seizure, and coma. Selective toxicity of the optic nerve and basal ganglia are well known features. We report

a young patient with methanol intoxication who presented with severe high anion gap acidosis metabolic and intra-cerebral bleeding.[5]

CASE REPORT

A 23 years old male was admitted to the emergency department because of severe vomiting since 8 hours before admission accompanied with abdominal pain that spread to the back. He also suffered from shortness of breath since 2 hours before admission. Initially he had suffered from headache since 1 day before admission and he start felt nausea since 12 hours before admission. After that he suffered from vomiting contained food and fluid residual for several times (his mother said that he suffered from vomiting more than 10 times). According to his neighbour, he had a party with his friends and drank alcohol 2 days before admission. Two of his friends has the same symptoms and was admitted to RSSA (Rumah Sakit Saiful Anwar) but unfortunately has died. His little brother said that he had habit to consume alcohol since 6 years ago with his friends. On the physical examination he look severely ill with GCS 234, pupil was isocor with light reflex positive, blood pressure was 90/60 mmHg, with pulse rate 120 bpm and look tachypnea with respiratory rate 40 times/min and axillary temperature 37.9^{0}C

Laboratory results showed leukocyte 35,100 u/l, kalium 8.24 mmol/l, blood sugar 337 mg/dl, amylase 387 U/l, urea 77 mg/dl, creatinine 2.31 mg/dl, and severe metabolic acidosis with pH 6.991, BE -24.9, and anion gap 30.

The patient was transferred to the ICU and initiated rehydration with NS 2 liters in an hour and after the hypotension was successfully treated with volume supplementation, we give load insulin 10 International Unit (IU) intravenously and continued with short-acting human insulin (Actrapid®) 5 IU/hour. We planned to perform emergency hemodialysis, but it cannot be done because of family refusal. We also give natrium bicarbonate 100 mg intravenously (slowly) and intravenous calcium gluconas to manage the metabolic acidosis and hyperkalemia. Folic acid, pantoprazole and thiamin were also administered.

Following these measures the hemodynamic condition of the patient improved markedly. The pH and kalium levels normalized. Although the hemodynamic parameters improved, the patient remained unresponsive and became unconscious. Because of the initial high level of methanol and the severity of the acidosis, severe neurological damage was to be expected.

We performed head CT scan and the result showed intra-cerebral hemorrhage at right fronto temporo parietal lobe with peri-focal edema (volume 48 cc). The neurologist also decides to join care this patient and give citicholin, vitamin K, mecobalamin, and mannitol infusion. After 8 days in ICU, he moved to the Stroke Unit for management the CVA.

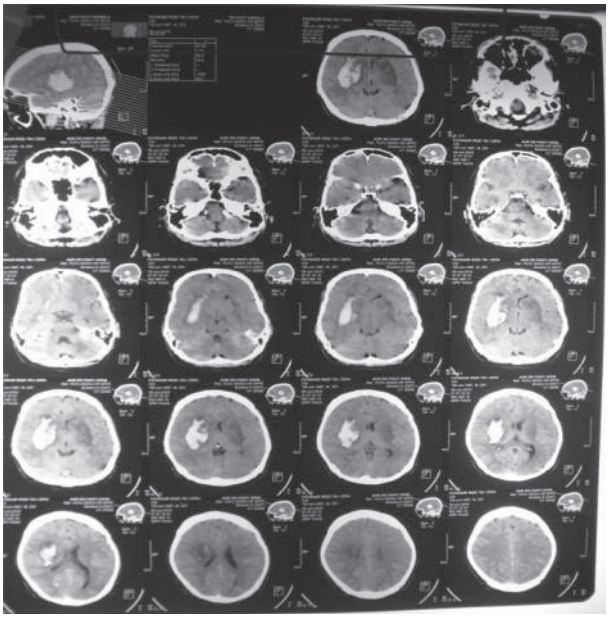

Figure 1. Head CT scan result of the patient showed intra-cerebral hemorrhage at right fronto temporo parietal lobe with peri-focal edema (volume 48 cc)

Unfortunately, his family decided to took him at home because of financial problem and we also perform follow up to his house and he died after 3 days treated at home.

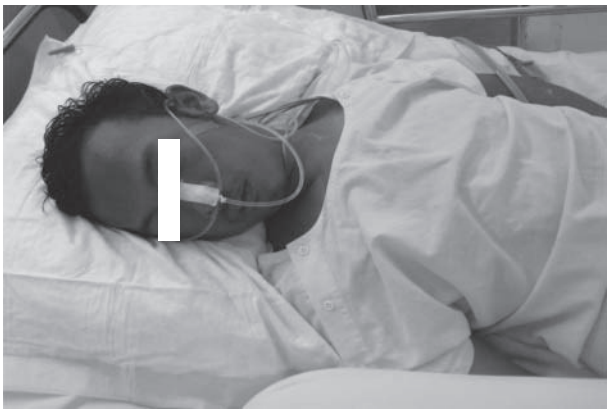

Figure 2. Picture of the patient

DISCUSSION

Criteria for initiation of therapy in patients with known or suspected methanol poisoning are: (1). Plasma methanol > 20 mg/dl *or* (2). Recent history of ingestion of toxic amounts of methanol and an osmolar gap > 10 mOsm/l *or* (3). Suspected methanol ingestion with at least 2 of the follows: arterial pH < 7.3; serum bicarbonate < 20 mmol/l; osmolar gap > 10 mOsm/l.[6]

The clinical feature of methanol intoxication usually occurs after a latent period of 12 – 24 hours following methanol ingestion. The latent period corresponds to the time period in which methanol is converted by alcohol dehydrogenase in the liver to formic acid, the metabolite that is responsible for the acidosis and the toxic effects. These clinical features also happen in this patient, when the patients had clinical symptoms 24 hours after drinking alcohol.[7,8]

Alcoholic ketoacidosis is a metabolic complication of alcohol use and starvation characterized by hyperketonemia and high anion gap metabolic acidosis without significant hyperglycemia. Alcoholic ketoacidosis causes nausea, vomiting, and abdominal pain. Diagnosis is by history and findings of ketoacidosis without significant hyperglycemia. Alcoholic ketoacidosis is attributed to the combined effects of alcohol and starvation on glucose metabolism. These features also happen in this patient, he came with hyperketonemia, high anion gap, metabolic acidosis, but without significant hyperglycemia.[9,10]

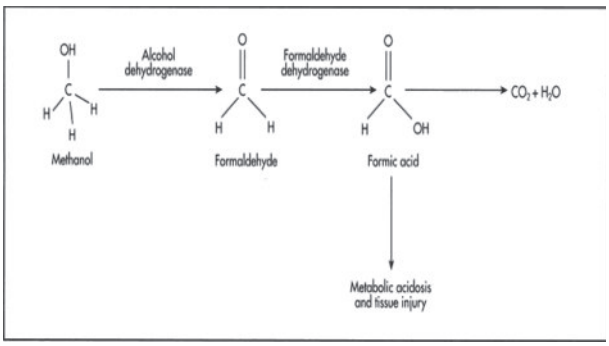

Figure 3. Methanol metabolism

Clinical features of pancreatitis acute (abdominal pain and vomiting) together with elevation of plasma concentrations of pancreatic enzymes (amylase, lipase) are the cornerstones of diagnosis. Pancreatic enzymes released into the circulation during an acute attack. Levels peak early, and decline over 3 – 4 days. Alcohol use (approximately 35%) is a major cause of acute pancreatitis. Most commonly, the disease develops in patients whose alcohol ingestion is habitual over 5 – 15 years. Alcoholics are usually admitted with an acute exacerbation of chronic pancreatitis. But several case reports have described a sole large alcohol load precipitating a first attack. This features also happen in this patient, Patient had history drink alcohol since 6 years ago and came with abdominal pain and severe vomiting and the pancreatic enzymes (amylase) was increased. Acute necrotizing pancreatitis often follows acute methanol poisoning.[8,10]

Brain injury in methanol toxicity is characterized by lesions affecting both basal ganglia and subcortical regions. The lesion could be ischemia/necrosis or hemorrhage, selectively affecting the putamenal bilaterally. It has been suggested that the putamen is at particular risk because of its high metabolic demand and location in a end zone of vascular perfusion. The precise mechanism of necrosis and hemorrhage in the case of methanol toxicity remains a matter of debate. It may represent a direct toxic effect of methanol and its metabolite "formic acid" as well as injury which is secondary to anoxia and acidosis. However , more recently a study in the state.[9,10]

These results suggest that methanol induced cerebral vasospasm may be a consequence of a large rise in intracellular calcium. These events could play a crucial role in methanol-induced cerebral edema, brain ischemia/necrosis and hemorrhage. University of New York demonstrated that methanol elevates calcium ions in cerebral vascular muscle cells.[5,6]

SUMMARY

We present a case of a male with methanol intoxication and several complication such, acute pancreatitis, alcoholic ketoacidosis, and CVA-ICH. The patient presented with hemodynamic instability and severe metabolic acidosis. Amylase was 187 U/l and CT scan showed intra-cerebral hemorrhage at right fronto temporo parietal lobe with peri-focal edema (volume 48 cc). Patient unfortunately died three days later after force discharge by his families.

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