Alcoholic Ketoacidosis

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ABSTRACT

Alcoholic ketoacidosis is a common disorder in chronic alcoholic persons. The Pathogenesis is complex associated with extracellular fluid volume depletion, alcohol withdrawal, abdominal pain, and acid abnormalities. The syndrome is rapidly reversible and has a low mortality. The treatment is administration with normal saline, glucose, potassium and thiamine. We report two cases of Alcoholic Ketoacidosis, first case with classically picture of AKA and the second one AKA with Decrease consciousness and concomitant illness, pneumonia. We believe this report possibly to be the first report of AKA in Indonesia.

Key words : alcoholic ketoacidosis,chronic alcoholic abusers, treatment.

Alcoholic ketoacidosis (AKA) is a common disorder in chronic malnourished alcoholic person[1,2,3]. It was first describe by Dillon and his colleagues in 1940, they reported on nine ethanol abusers who were seen at the Philadelphia general Hospital.[2,4] This syndrome has not been reported in Indonesia.It might be due to often underdiagnosed in view of rapid response to intravenous fluids and glucose, which often obscures its presence.[4,5]

The clinical findings were very similar to those in patients with diabetic ketoacidosis except for there being little or no hyperglycemia or glucosuria.[1,4] The disorder occurs in chronic ethanol abusers who have usually had a binge drinking associated with negligible intake of solid food,[5] either because of gastritis or some other intercurrent illness. Clasically, the patient begins to develop upper abdominal problems, often alcoholic gastritis or pancreatitis leading to severe vomiting and inability to take food[4]. The patients often complain of upper abdominal pain, nausea and vomiting.[1,2] The patient's mental state may vary from completely normal to severe disorientation[5]. Altered mental status is always explained by a concomitant process such as intoxication, hypoglycemia, or a cerebrovascular accident.[1,2] Some patients also had withdrawal symptoms including delirium tremens[2]. The most frequent physical findings are tachycardia, tachypnea and abdominal tenderness.[1] The acid-base abnormalities are more diverse than just a wide-gap metabolic acidosis and often include a concomitant metabolic alkalosis, hyperchloremic acidosis, or respiratory alkalosis.[1,2] Lactic acidosis is also common. Blood glucose level usually normal or slightly elevated, except in patients with Diabetes.[1,2,3] Hypoglycemia in AKA is rare and associated with irreversible encephalopathy.[6] Blood ethanol levels have often been undetectable or only mild elevated, because usually they stopped drinking one or more days before admission.[2,5] Electrolyte disorders including decrease level of sodium, potassium, phosphate, calcium, magnesium and an increase level of glucose were common on presentation.[1,2,3] Altered mental status, fever, hypothermia, or other abnormal findings were uncommon and reflected other underlying processes.[1] Wrenn KD et al reported a study from 74 patients with AKA and they found the signs and symptoms (Table 1)

There were approximately equal numbers of men and women. Although early studies suggested a female preponderance.[1,2,3,7] There also appear to be tendency for AKA to recur in certain patients. Nearly one quarter had either a prior history of AKA or a repeat episode of AKA.[1]

Treatment of patients with AKA is direct to correcting fluid, electrolytes, and metabolic abnormalities, and to seeking, diagnosing, and managing any concomitant ilness.[2] The main components on treating AKA is intravenous administration of saline, glucose, potassium salts and thiamine, which usually rapidly corrects the metabolic disturbances.[1,2,3,7] The administration of sodium bicarbonate only if there is severe metabolic acidosis (PH<7,1)[1,2,3,7] Insulin is usually not necessary, except in patients known or suspected to have diabetes.[1,2,3,7] And last but not least if there were coexisting acute illnesses must give the appropriate treatment.[1,2]

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Table 1. Presenting Symptoms and Signs in AKA[1]

Symptom	Number	%	Physical Finding	Number	%	Range
Nausea	56	76	Tachycardia (pulse > 100/minute)	39	58	100-150/mint
Vomiting	54	73	Tachypnea (RR >20/minute)	33	49	22-36/minute
Abdominal pain	46	62	Abdominal tenderness	32	43	
Shortness of breath	15	20	Heme-positive stool	7	18	
Tremulousness	15	20	Hepatomegaly	13	18	
Hematemesis	14	19	Altered mental status	9	15	
Dizziness	14	19	Hypotension (SBP < 100 mmHg)	8	12	70-93 mm Hg
Muscle pain	7	10	Abdominal distention	4	5	
Fever	6	8	Hypothermia	3	4	33.1°-34.2°C
Diarrhea	5	7	Fever	2	3	38.1°-38.5°C
Syncope	3	4	Decreased bowel sounds	1	1	
Seizure	2	3	Rebound tenderness	1	1	
Melena	1	1				

RR = respiratory rate; SBP = systolic blood pressure

There are no data of AKA in Indonesia, so by these two cases we would like to remind us to be aware if we meet chronic alcohol abusers with wide anion gap metabolic acidosis.

Pathogenesis of AKA (figure 1)[2]

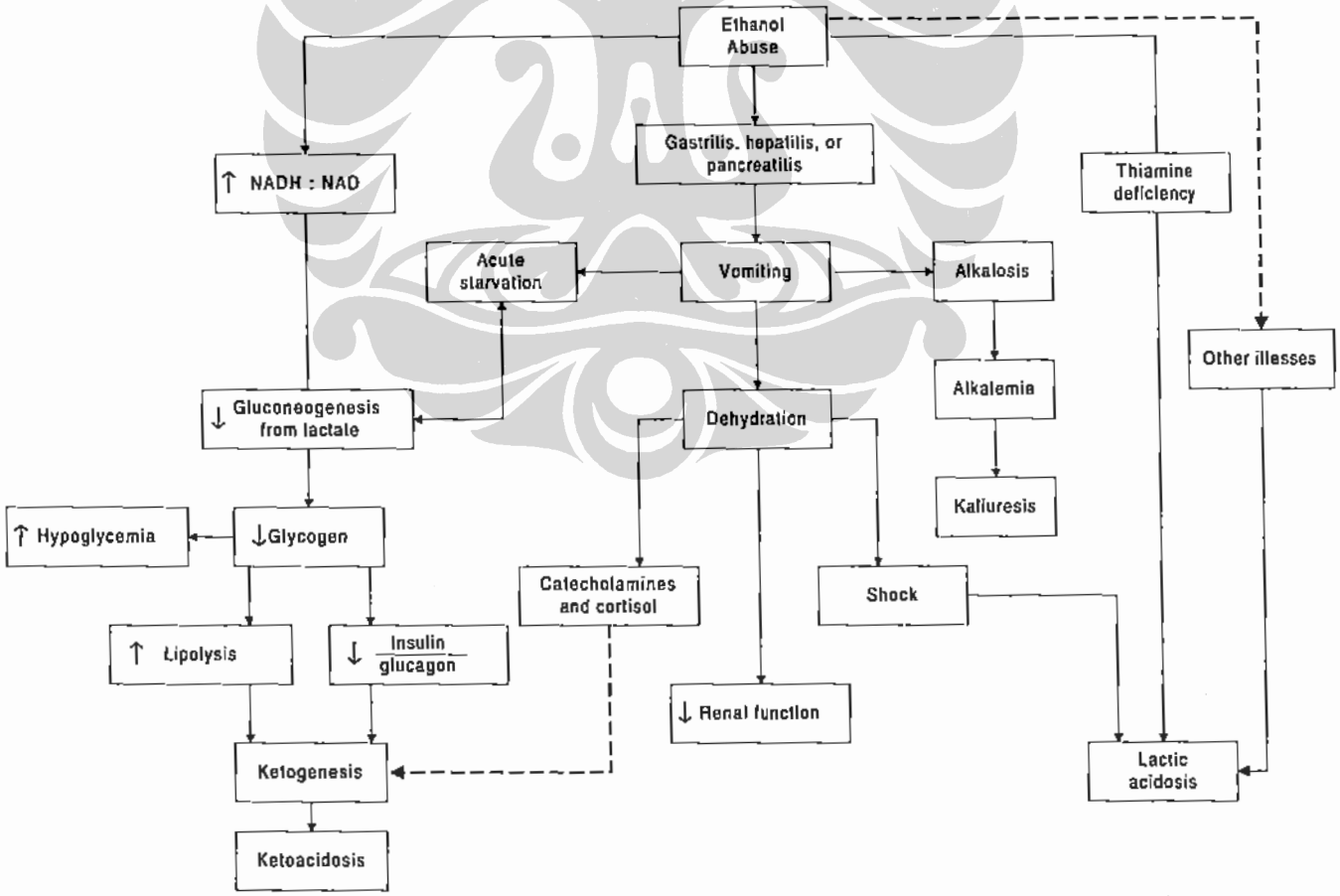

Figure 1. Flow Chart of Pathogenesis of Alcoholic Ketoacidosis[2]

CASE REPORT

Case 1

Patient F, a 37 years old man was admitted to RSUPN Cipto Mangunkusumo with chief complaint of shortness of breath 2 days before the admission. One day before the admission he had had a shortness of breath with epigastria pain, nausea and vomit. This patient no suffer from cough.

Three days before the admission patient drank more than one bottle and ate a little food (4-5 spoonfuls of rice). And one day before the admission patient did not eat anything. The defecation and urinate were normal. The history of drinking a lot, often urinate and frequent of starvation were denied by patient. History of heart disease, kidney disease, and hypertension, and lung disease were also denied. Patient has been drinking since when he was in high school, 20 years ago and since last 10 years he drank alcohol every day and he could drank 4 bottles in a day. Since 2-3 months before the admission, patient had drunken alcohol which has been mixed with ginseng, approximately half bottle a day. There were no family histories of diabetes mellitus or other metabolic diseases. This complaint suffered from for the first time.

On the physical examination the patient was moderately ill, fully alert. The blood pressure was 130/80, pulse rate was 112 times/min, respiration rate was 36 times/min, the temperature was not febrile. His body weight was 55 kg, and his body height was 160 cm. The conjunctiva was not pale and sclera was not jaundice. The tongue was dry, JVP 5-2 cm H_2O, lymph node was not palpable, heart and lung were normal. Spleen and liver were not palpable, there was an abdominal tenderness, the bowel sound was normal, and shifting dullness was negative. The extremity was warm, there was no edema and the skin turgor was decreased.

Laboratory results showed that the hemoglobin was 18.5 g/dl; hematocrit 54 vol%; white blood cell was 13.100/ml; the platelets was 377000/ml; ureum 30 mg/dl; creatinin 1,3 mg/dl; blood glucose 148 mg/dl. The arterial blood gas were pH 7,04; pCO_2 8,4; pO_2 155,5; HCO_3 2,2; base excess -25,9 mEq/l, O_2 saturation 98,1; natrium 134; kalium 4,6; chloride 97; blood keton +1. Urinalysis specific gravity was 1.015; pH 6; glucose - ; protein +1; keton +2; blood +1; bilirubin - ; leucocyt 5-6/hpf; eritrocyt 2-3/hpf; cylinder - ; crystal amorf. The EKG was normal, and also the CXR.

The problem of this patient was alcoholic ketoacidosis and dyspepsia. The problem of alcoholic ketoacidosis was based on the drinking habit of patient since he was 21 years old. 3 Days before the admission to the hospital, patient drank alcohol and ginseng more than 1 bottle with a little calorie intake, because of nausea and vomiting. The patient also felt epigastria pain.

On the physical examination, there was found tachypnea with kussmaul, tachycardia and abdominal tenderness. From the the blood gas analysis we found severe metabolic acidosis with high anion gap. This patient administered with normal saline and dextrose 5%. He also got bicarbonate therapy 350 mEq gradually in the emergency and oral bicarbonate 3x2 tablets in the hospital ward. The problem of dyspepsia was based on the presence of vomit, nausea and epigastria pain, and got ranitidine and antacid. The consumption of alcohol for years was presumed to be the cause of the problem.

Nine hours after he treated in the emergency room, in the hospital ward there were another metabolic acidosis which was corrected by bicarbonate 200 mEq. After the metabolic acidosis was overcome, the patient was getting better and the calorie intake was better. From the abdominal ultrasonography it was found that the condition of liver and gall were normal and there was stone in the right kidney. We plan to consult to urology department. Eighth day of treatment in the hospital, we signed out the patient.

Case 2

Patient W, a 35-year old man, was admitted to RSUPN Cipto Mangunkusumo with chief complaint of unconscious 6 hours before admission. He had a history of productive cough for 1 week. Three days before the admission he had fever, epigastria pain and nausea with vomiting more than 5 times a day. Because of his vomiting, he had not eaten any solid food for 3 days but he continued drinking liquor with ginseng and energy drink between the vomiting. One day before admission, he appeared to be nervous, his complaints were headache, weak and an increased shortness of breath. He had no trauma and he denied being exposure to toxins and the use of medications.

The patient had been drinking 1-2 bottles of alcoholic beverages a day, regularly for 15 years. History of polyfagia, polydipsia and polyuri were denied by patient. Histories of heart disease, hypertension, renal and pulmonary disease were also denied. There were no family histories of diabetes mellitus or other metabolic disorders.

On the physical examination there was found that patient was severely ill, delirium. The blood pressure was 90/60 mmHg, pulse 120/min, fast and deep respiration with rate of 36/min, temperature 37°C. Conjunctiva was

not pale and sclera was not jaundice. JVP was normal, lymph node was not palpable. Heart sound was normal, no murmur or gallop. The lungs were sonoir, vesicular with rales at the right lung. Abdomen was not distended, he had an epigastria pain, the spleen and liver were not palpable, the bowel sound was normal and there was no ascites. The extremity was warm, there was no edema, and the skin turgor was decreased.

Laboratory results showed hemoglobine 16 g/dl, leukocyte 8000/ul, hematocrit 48 vol%, platelet 134.000/ul, urea 35 mg/dl, creatinine 1,4 mg/dl, blood glucose 176 mg/dl. Urinalysis specific gravity 1.025, pH 6, protein (-), glucose (-), aceton (+1), blood (-), bilirubin (-), urobilinogen 0.1, leukocyte 0-2/field, erythrocyte 0-1/field. Blood acetone (+), blood gas analyses: pH 6.953, pCO_2 143,3 mmHg, HCO_3 2,9 mEq/l, base excess -26,9 mEq/l, O_2 saturation 96,5%. Electrolyte sodium 131 mEq/l, potassium 2,5 mEq/l.

The problems of this patient were the decreased consciousness, alcoholic ketoacidosis, dehydration, dyspepsia syndrome and pneumonia. The problem of decreased consciousness was based on physical examination on admission that he was unconscious and responded only to painful stimuli. His breath was Kussmaul and the result of blood gas analyses and electrolyte showed severe metabolic acidosis, hyponatremia and hypokalemia. Examined by neurologist, there was no focal deficit. Based on these data we thought that the decreased consciousness was caused by metabolic imbalance. Because he was an alcoholic, it seemed that alcohol contributed this condition. Unfortunately, the blood alcohol level was not measured. This condition was treated with bicnat 200 mEq and KCl 50 mEq.

AKA were based on history of alcohol consumption preceding the patient's admission, the diminished calorie intake because of nausea-vomiting, dehydration and mild mental alteration which was followed. On physical examination, there were found a decreased of consciousness, shortness of breath with Kussmaul. Arterial blood gas showed severe metabolic acidosis, the blood acetone was positive, ketone urine (1+) and blood glucose was slightly elevated. The patient was treated with saline and 5% dextrose.

Dehydration was based on history of vomiting, tachycardia, hypotension and the decreased skin turgor. The patient was treated with intravenous saline fluid, and glucose 5%.

The dyspepsia syndrome was based on nausea, frequent vomit and epigastria pain. Chronic alcohol consumption might be the caused of this problem. The patient was treated with ranitidine and antacid. The symptoms gradually disappeared within 4 days therapy.

The problem of pneumonia was based on history of fever and productive cough. On physical examination, there were found rales at the right lung, the laboratory result showed white blood cell increase. Patient was treated with Ampicillin 4x1 g IV. The therapy was still continued. There was no rales anymore on the physical examination when patient leaved the hospital.

Five hours after therapy was started, the arterial pH was elevated. In the next 12 hours, the patient was fully alert. The patient complained of pain in his eyes, but from the consultation with ophthalmologic division, there was not found a neuritis optic. As a result of the treatment, the acid-base abnormality was corrected and became normal on the 4th hospital day. Despite our advice, the patient signed out on the 7th hospital day and he rejected to have his liver examined by ultrasonography because he has already felt better.

DISCUSSION

These two cases are proposed to be demonstration cases. Because this condition often under diagnosed.[5] Severe metabolic acidosis in alcoholic persons is an emergency case which can lead to death of a patient,[7,8] but this syndrome is rapidly reversible and has a low mortality.[1]

In the first case diagnosis of alcoholic ketoacidosis on this patient was based on history of chronic alcohol abuse, followed by low intake of calorie due to nausea and vomit. When the patient was admitted to the hospital, the complaint was shortness of breath, epigastria pain, vomit, nausea, history of alcohol consumption for years and less calorie intake since 3 days before admission. The most common symptoms of AKA were nausea, vomiting, and abdominal pain[1]. From physical examination we found tachypnea, tachycardia, abdominal tenderness and decreased of skin turgor, the most common physical findings were tachypnea, tachycardia, and abdominal tenderness.[1] The laboratory results showed an increase of white blood cell, light increased of blood glucose, light hyponatremia and mild ketonemia. There was severe metabolic acidosis with high anion gap. The patient was given normal saline fluid and glucose fluid. His condition became better after 3 days of treatment. This such a classically picture of AKA.

We did not give antibiotics for the patient because from the clinical data there was no sign of infection. In the USG examination the liver was normal, there was stone in the right kidney. So the patient's problem was

renal stone in the right kidney. We planned to consult the case to urology department after the patient leave the hospital.

In this patient, there was repeatedly metabolic acidosis. It is presumed that the cause was low calorie intake. This patient should administered by glucose liquid and normal saline to replace volume depletion and glucose to interrupt ketogenesis and replace glycogen stores,[1,2,3,4,7] feeding the patient as soon as that is possible[4]. This patient administered by intravenous sodium bicarbonate because the acidosis metabolic was severe (<7,1). Intravenous Sodium bicarbonate administration is not necessary, except in severe academia PH below 7,0 to 7,1.[1,2,3,4,7]

The patient has been given education not to drink alcohol and explanation about the impact of drinking alcohol and the possibility of death because of recurrent AKA with other concomitant disorder.

In second case, besides AKA the patient suffer from decreased of consciousness. Altered mental status reflects other underlying processes such as serious infectious illness, hypoglycemia, ethanol intoxication or withdrawal.[1,2] Blood glucose routine during hospitalization within normal limits, showed the patient was not diabetic.

AKA usually responds rapidly within 6 to 18 hours. AKA on this patient responded to treatment with volume repletion and glucose alone within 17 hours, was signed by repair of consciousness and increased of pH values.[4] The acid base abnormality was corrected 4 days after therapy.

Electrolyt measured showed hypokalemia (2,5 mEq/l), that was common on AKA because of poor intake and excess losses.[1,2,7] After treated with KCl, potassium level elevated to be normal.

Alcohol when chronically ingested may increase free fatty acid production in the liver. Some studies showed extremely elevated free fatty acid levels. It probably closely related to the ketonemia with acidosis[2,4]. On this patient we can not analysis free fatty acid level because the laboratory facility was not ready yet. This patient also suffered from pneumonia. Patients with AKA are prone to develop to aspiration pneumonia and bacterial infection.[4] Five days after antibiotics therapy, rales at right lung disappeared and clinically pneumonia was cured.

In these two cases we did not administered thiamine because of our lack of experience in treating patient with AKA. Thiamine is recommended to prevent wernick encephalopathy that could happened in patient with ethanol abuse.[1,2,7]

We believe this report possibly to be the first report of alcoholic ketoacidosis in Indonesia. We Hope this illustration could increase our awareness of this Syndrome when we meet a patient with chronic alcohol abuse with wide anion gap metabolic acidosis and such symptoms and signs.

REFERENCES

1. Wrenn KD, Slovis CM, Minion GE, Rutkowski R. The syndrome of alcoholic ketoacidosis. Am J Med. 1991; 91: 119-28.
2. Fulop M. Alcoholic ketoacidosis. Endocrinol Met Clin North Am. 1993; 22: 209-20.
3. Oster JR, Epstein M. Acid base aspects of ketoacidosis. Am J Nephrol. 1984; 14: 137-51.
4. Williams HE. Alcoholic hypoglycemia and ketoacidosis; Med Clin North Am. 1984; 68:33-8.
5. Thompson CJ, Johnston DG, Baylis PH, Anderson J. Alcoholic ketoacidosis: an underdiagnosed condition?; Br Med J. 1986; 292: 463-5.
6. Jain H, Beriwal S, Singh S. Alcohol induced ketoacidosis, severe hypoglycemia and irreversible encephalopathy. Med Sci Monit. 2002; 8: CS77-9.
7. Alson R. Alcoholic ketoacidosis. Emergency medicine.2001 May 4. Available from: URL : http://www.e-medicine.com. Accesed on February, 5,2003.
8. Kadis P, Balazie J, Marolt VF. Alcoholic ketoacidosis : a cause of sudden death of chronic alcoholics. Forensic Sci. Int. 1999;103:S53-9.