Case Report

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Lactic Acidosis: A Diagnostic Conundrum

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Abstract

Lactic acidosis is due to increased serum lactate (>5.0mmol/L) leading to decrease in blood pH. Alcoholic keto-acidosis is one of the several etiologies where serum lactate level rarely exceeds 3mmol/L. Here we present a case of 59yr old male presented to ED with c/o abdominal pain and multiple episodes of vomiting following consumption of alcohol. On examination, patient was tachypneic, tachycardic with BP of 90/60mmHg. Arterial blood gas showed severe metabolic acidosis with a pH of 6.96, pCO2 of 21.4, pO2 of 57.1, HCO3 of 4.7 and Lactate of 14.48. On probing history, attenders revealed consumption of illicit liquor by patient on previous night giving rise to suspicion of methanol toxicity.

Keywords: Lactic acidosis; Methanol intoxication.

INTRODUCTION

Lactic acidosis is a common metabolic acidosis characterized by increased serum lactate (>5.0mmol/L) leading to decrease in blood pH.[1] Alcohol interferes with the breakdown of sugars and fats for energy production, which causes an accumulation of lactic acid in blood leading to lactic acidosis. Alcoholic keto-acidosis is one of the several etiologies where serum lactate level rarely exceeds 3mmol/L.[6] Very few cases of alcoholic keto-acidosis with lactate level >5mmol/L have been reported so far.

CASE REPORT

A 59 Yrs old male patient presented to ED with c/o abdominal pain for the past 1 day. It was associated with multiple episodes of vomiting. Abdominal pain was diffuse in nature and vomiting was non-projectile and non-bilious. On examination, the patient was groaning in pain in a supine position with SpO2 level of 98% in room air, RR-24 cpm, HR-140 bpm, BP-90/60 mm of Hg. Per abdomen examination showed a distended abdomen with diffuse tenderness. Siemens Healthineers co-oxymeter arterial blood gas (Table 1) showed a pH of 6.96, pCO2 of 21.4, pO2 of 57.1, HCO3 of 4.7 and Lactate of 14.48, giving rise to strong suspicion of bowel perforation or mesenteric ischemia.

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Patient was treated with Inj. Paracetamol 1g IV, Inj. Sodium Bicarbonate 50ml IV, Inj. Piperacillin Tazobactam 4.5g IV, Inj. Metronidazole 500mg IV, Inj. Pantoprazole 40mg IV, Inj. Ondansetron 4mg IVand Intravenous fluids 500ML normal saline bolus.

Table 1: Abg Parameters

Abg Parameters	Values
pH	6.96
pC02	21.4
pO2	57.1
HCO3	4.7
LACTATE	14.48

ECG (fig. 1) showed sinus tachycardia. The chest X-ray was normal (fig. 2), with no air under the diaphragm. USG Abdomen and pelvis, as well as CT Abdomen (plain) showed normal studies. Ryles tube was inserted, and approximately 1.5L of gastric content was aspirated (fig. 3).

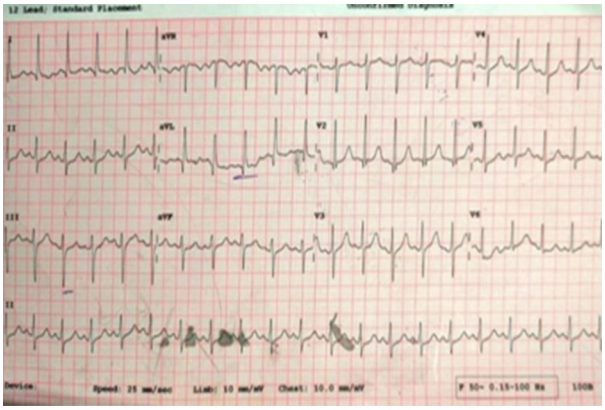


Fig. 1: ECG showed Sinus Tachycardia

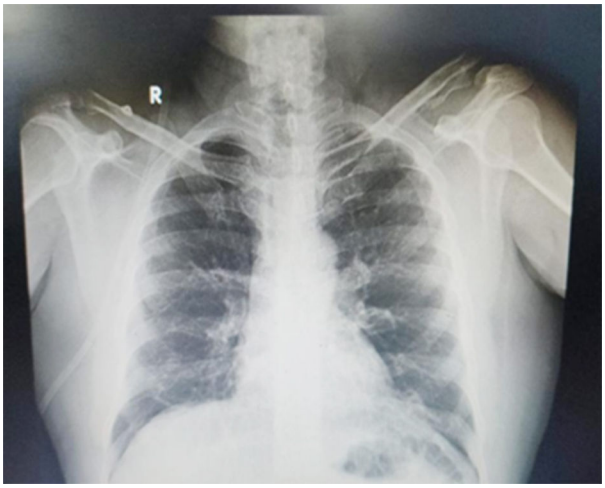


Fig. 2: The Chest X-ray

Fig. 3: 1.5 L of Gastric Content

On probing history, attenders alleged consumption of 200ml of illicit liquor followed by consumption of 500ml of ethanol by patient, giving rise to a high index of suspicion of methanol toxicity. Intravenous fluids of 2 liters of normal saline with Optineuron (thiamine 100mg, pyridoxine 100 mg, Cyanocobalamin 1000 mcg, vitamin B2 5 mg, nicotinamide 100 mg, D-Panthenol 50 mg) was given.

The patient was symptomatically better after 1GM of paracetamol infusion. 2-D ECHO was done, which showed normal LV systolic function with EF-56% and No RWMA. Serial arterial blood gas analysis showed pH of 7.48, pCO_2 of 28.7, pO_2 of 83.5, HCO_3 of 21.5 and Lactate of 1.2 (Table 2).

Table 2: Abg Parameters

Abg Parameters	Values
pH	7.48
pCO_2	28
pO_2	83.5
HCO_3	21.5
Lactate	1.2

DISCUSSION

Lactic acid is an organic compound produced by fermentation by different microorganisms that are

able to use different carbohydrate sources. Lactic acid bacteria are the most important bacteria used to produce lactic acid.[14]

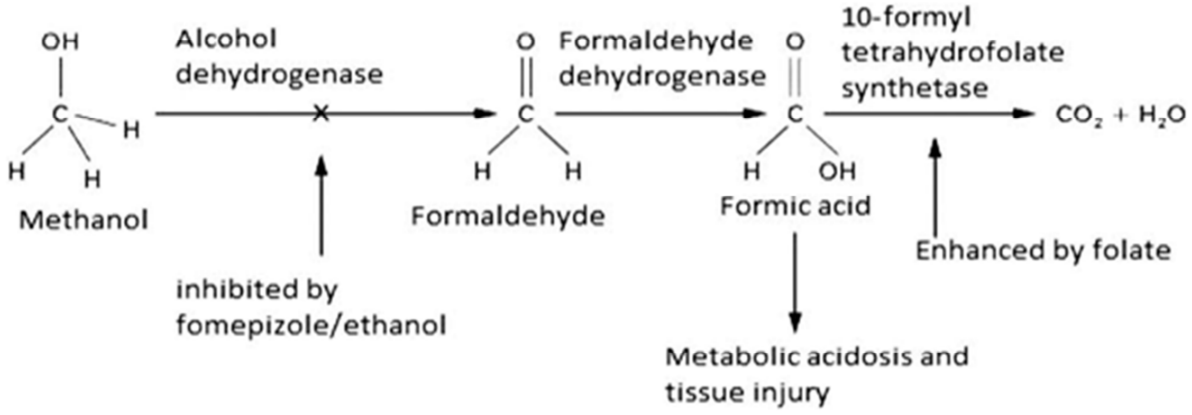
Under physiological conditions, approximately 1,500 mmol of lactate is formed daily in various organs, including muscles, intestines, red blood cells, brain, and skin. Lactate is metabolized in the liver (about 60%), kidneys (about 30%) and other organs. The normal concentration of lactate in the blood is about 1mmol/l. One of the most important metabolic changes caused by a lack of oxygen is the Pasteur effect. Due to a lack of oxygen, the pyruvate produced by the anaerobic conversion of glucose cannot enter the Krebs cycle via acetyl coenzyme A to produce energy. Converting pyruvate to lactate allows energy to be produced without oxygen. This is the most important adaptive mechanism for surviving hypoxia.[9]

Lactic acid exists in two optically isomeric forms, L-lactate and D-lactate. L-lactate is the most commonly measured level because it is the only form produced by human metabolism. Its excess means increased anaerobic metabolism due to tissue hypoperfusion. D-lactate is a byproduct of bacterial metabolism and can accumulate in patients with short bowel syndrome or patients who have had gastric bypass or small bowel resection.[13]

There are three types of lactic acidosis. Type A is caused by hypoperfusion and hypoxia, which occur when oxygen consumption/delivery is mismatched, resulting in anaerobic glycolysis. Examples of type A lactic acidosis include all types of shock (septic, cardiogenic, hypovolemic, obstructive), regional ischemia (extremity, mesenteric), seizures, and severe chills. Type B occurs with normal tissue perfusion and without hypoxia. Examples of type B lactic acidosis include liver disease, malignancy, drugs (metformin, epinephrine), total parenteral nutrition, HIV, thiamine deficiency, congenital lactic acidosis, trauma, excessive exercise, diabetic ketoacidosis, and ethanol intoxication.[2] Type D lactic acidosis is seen primarily in people with malabsorption disorders such as short bowel syndrome.[3]

Alcohol consumption is the seventh largest risk factor for death and disability adjusted life years.[7] Ethanol is mainly metabolized in the liver by alcohol dehydrogenase. After being oxidized to acetaldehyde, it is oxidized to acetic acid by aldehyde dehydrogenase. Both enzymatic steps result in the formation of the reduced form of nicotinamide adenine dinucleotide dehydrogenase (NADH). This increase in the NADH/NAD ratio promotes the metabolism of pyruvate to lactate.[4]

Methanol is also called methyl alcohol, wood alcohol or wood spirit. It is a volatile, colorless, flammable, toxic liquid produced by the distillation of decaying wood particles and composed of



carbon monoxide and hydrogen ions (CH_3OH). This compound was illegally used to make cheap and adulterated ethanol.[10]

Methanol is successively oxidized: the enzyme alcohol dehydrogenase catalyzes methanol to formaldehyde, which is then catalyzed to formic acid by the action of the formaldehyde dehydrogenase enzyme. Folic acid given to the patient accelerates its conversion to carbon dioxide (CO_2) and water (H_2O).[11]

Methanol is found in many household cleaners and dyes, jet fuel, windshield washer fluid, gas line antifreeze, and illegally produced alcoholic beverages. Poisoning usually occurs through ingestion, but can also result from inhalation or absorption through the skin. Symptoms of poisoning may include shortness of breath, nausea, vomiting, abdominal pain, decreased sensitivity and impaired vision. Ophthalmological examination can reveal papillitis of the optic nerve

(found in 10% of cases). If the putamen is damaged, muscle stiffness and masked plants may be seen. Most clinical abnormalities are due to the effects of formic acid. Formate inhibits cytochrome oxidase, and can cause tissue hypoxia and lactic acidosis.[11] A byproduct of methanol oxidation by alcohol dehydrogenase, when ingested, formic acid causes a number of toxic manifestations, including optic neuropathy, cerebral edema, acute renal failure and severe metabolic acidosis. Symptoms may appear a few hours or even 2 days after ingestion. The lethal dose is said to be 1.2 ml/kg. Mortality rates associated with methanol consumption range from 18 to 44%.[10] When methanol is metabolized to organic acid anions, the anion gap increases while the osmolar gap decreases. Thus, patients who present to the ED mainly after ingestion may have high osmolality and a normal anion gap, while patients who present later may have the opposite.[15]

Fomepizole, a competitive alcohol dehydrogenase inhibitor, prevents the formation of alcohol metabolites and is the antidote of choice. However, fomepizole is often very expensive, making ethanol a common alternative treatment. Sodium bicarbonate and hemodialysis have been shown to help correct acid-base disturbances.[10] Management of methanol poisoning requires a multidisciplinary approach involving emergency physicians, toxicologists, and nephrologists. The first step in managing methanol poisoning is to ensure that the patient's airway, breathing and circulation are stable. This may include giving oxygen, administering intravenous fluids, and monitoring the patient's vital signs. In severe cases, mechanical ventilation and vasopressors may be required to support the patient's respiratory and cardiovascular systems.[12]

The strong ion difference (SID) is the sum of the strong cations (Na+, K+, Ca2+, Mg2+) minus strong anions (mainly Cl− but also lactate−). Lactated Ringer's solution is a balanced salt solution containing sodium (130 mmol/L), chloride (109 mmol/L), lactate (28 mmol/L), and small amounts of potassium and calcium. Lactated Ringer's SID is 28 mEq/L, closer to the normal value of 40 mEq/L than 0.9% saline, which has an SID of 0 mEq/L. Rapid administration of Ringer's lactate may decrease plasma SID and cause transient metabolic acidosis. However, lactate is usually rapidly metabolized in the liver, altering its effect.[9]

The treatment of alcoholic ketoacidosis is based on the exclusion of serious pathology and the specific treatment of alcoholic ketoacidosis, if present.[5] People can recover from alcoholic ketoacidosis if they get a timely diagnosis and proper treatment.[8]

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