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## Case Report on Acute Complication of Alcoholism.

A Mary Chandrika\*, AJ Manjula Devi, and K Sumathi.

Department of Biochemistry, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.

### ABSTRACT

The alcohol consumption can cause major health problems, including cirrhosis of the liver and injuries sustained in automobile accidents. Drinking too much on a single occasion or overtime causes serious consequences on health. A 61 year old man diabetic for 5 years on irregular treatment with oral anti-diabetic drugs was found to have alcohol induced hypoglycemia with lactic acidosis. Resuscitation continued in the intensive care unit with remarkable improvement and satisfactory outcome. Metabolic acidosis was corrected and patient was advised to control alcohol consumption and follow regular diabetic management. In this patient, the severe lactic acidosis and associated abnormalities were all attributed to acute and chronic effects of ethanol. A brief summary of the proposed mechanism by which these metabolic derangements developed and an outline of her management follows.

**Keywords:** alcohol induced hypoglycemia, lactic acidosis.

*\*Corresponding author*

## INTRODUCTION

Alcohol consumption is one of the major health problems affecting male and female. Chronic alcoholism causes serious health problems, affecting multi-organs. Increased consumption of alcohol with poor dietary intake leads to hypoglycemia.

### Case Report

A 61 year old man brought to hospital casualty in an unconscious state. He is a known diabetic for 5 years on irregular treatment with oral anti- diabetic drugs. He is an occasional alcoholic for 18 years but started consuming increased quantity of alcohol with improper food intake for 1 month due to business loss. On examination he could not be aroused and had a deep and noisy breathing. Breath had alcohol smell.

### Lab investigation reports

Blood glucose 48mg/dl, Arterial blood gas analysis- pH-7.1, pCO<sub>2</sub>-34mmHg, pO<sub>2</sub>- 80mmHg, Lactate - 9.2mmol/L, Blood urea- 48mg/dl, Serum creatinine – 1.3mg/dl ,Liver function test –Total bilirubin-2.8mg/dl, Direct bilirubin-1.5mg/dl, indirect bilirubin-1.3mg/dl , SGOT-120 U/L , SGPT-99 U/L , total protein 5.5g/l , albumin -2.5g/l, Globulin 3.0, Gamma GT- 300U/L. Lipid profile – Total cholesterol -169 mg/dl , TGL-307mg/dl, HDL-30mg/dl.

USG abdomen done showed Fatty liver.

## DISCUSSION [1-7]

Alcohol consumption is very common. Increased alcohol intake with poor appetite results in alcohol induced hypoglycemia. In this case low blood glucose (48mg/dl) shows hypoglycemia most probably after alcohol consumption. Metabolic acidosis is noted by low pH (7.1), is mainly due to lactic acidosis ( Blood lactate – 9.2mmol/L ).

Alcohol induced hypoglycemia is due to hepatic glycogen depletion combined with alcohol – mediated inhibition of gluconeogenesis. It is very common in alcohol abusers with poor nutrition but can occur in anyone who is unable to ingest food after an acute alcoholic episode. The primary pathway for alcohol metabolism involves alcohol dehydrogenase (ADH), a cytosolic enzyme that catalyzes the conversion of alcohol to acetaldehyde. This enzyme is located mainly in the liver, but small amounts are found in other organs such as the brain and stomach. During conversion of ethanol by ADH to acetaldehyde, hydrogen ion is transferred from alcohol to the cofactor nicotinamide adenine dinucleotide (NAD<sup>+</sup>) to form NADH. Much of the acetaldehyde formed from alcohol is oxidized in the liver in a presence of mitochondrial NAD<sup>-</sup> dependent aldehyde dehydrogenase (ALDH). The product of this reaction is acetate, which can be further metabolized to CO<sub>2</sub> and water, or used to form acetyl-CoA. As a result, alcohol oxidation generates an excess of reducing equivalents in the liver, chiefly as NADH. The excess NADH production appears to contribute to the metabolic disorders that are present along with chronic alcoholism.

- The NADH produced in cytosol by ADH must be reduced back to NAD<sup>+</sup> either by the malate- aspartate shuttle or by the glycerol –phosphate shuttle. Thus, the ability of an individual to metabolize ethanol is dependent upon the capacity of hepatocytes to carry out either of these 2 shuttles, which in turn is affected by the rate of the TCA cycle in the mitochondria whose rate of function is being impacted by the NADH produced by the ALDH reaction.
- The reduction is NAD<sup>+</sup> impairs the flux of glucose through glycolysis at the glyceraldehyde-3-phosphate dehydrogenase reaction, thereby limiting energy production.
- There is an increased rate of hepatic lactate production due to the effect of increased NADH on direction of the hepatic lactate dehydrogenase (LDH) reaction. This reversal of the LDH leading to a reduction in the capacity of the liver to deliver glucose to the blood.

- Similar to the lactate formation, malate is also produced from oxaloacetate. Deficiency of oxaloacetate negatively affects gluconeogenesis as well as the functioning of TCA cycle.
- In addition to the negative effects of the altered NADH/NAD<sup>+</sup> ratio on hepatic gluconeogenesis, fatty acid oxidation is also reduced as this process requires NAD<sup>+</sup> as a co factor
- Fatty acid synthesis is increased and there is an increase in triglyceride production by the liver. In the mitochondria, the production of acetate from acetaldehyde leads to increased levels of acetyl- CoA. Since the increased generation of NADH also reduces the activity of the TCA cycle, the acetyl- Co A is diverted to fatty acid synthesis.
- The reduction in cytosolic NAD<sup>+</sup> leads to reduced activity of glycerol-3-phosphate dehydrogenase resulting in increased level of glycerol 3- phosphate which is the backbone for the synthesis of the triglycerides. Both of these two events lead to fatty acid deposition in the liver leading to fatty liver syndrome.
- Increased lactate/pyruvate ratio, results in hyperlacticacidemia. Lactate accumulation causes lactic acidosis (metabolic acidosis )

### Treatment

This patient with alcohol induced hypoglycemia and metabolic acidosis was treated with sodium, chloride, potassium, phosphorus, magnesium and thiamine replacements along with attention to concomitant clinical problems. Hypoglycaemia present was corrected with dextrose and normal saline. Lastly, alcohol need not be detected on admission to make the diagnosis of this metabolic disturbance. However, when present, it could contribute directly to the lactic, acetic and B-hydroxy butyric acidoses.

### CONCLUSION

It's best to monitor blood glucose levels regularly when having alcohol. Here are some tips to help avoid this problem. Never drink on an empty stomach. Start with nonalcoholic beverages to satisfy your thirst and continue to have one available while you consume alcohol. Limit the amount that you drink.

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