Serum Magnesium and Other Electrolyte Levels in Chronic Alcoholic Patients in a Tertiary Mental Care Centre in North Coastal Andhra Pradesh India

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Abstract:
Background: In this study we sought to investigate the effects of chronic alcoholism on the body leading to changes in levels of serum magnesium and other serum electrolytes. METHOD: A total of fifty (50) patients with chronic alcoholism without treatment were included in the study. Twenty five (25) healthy non alcoholic controls were also included in the study for comparison. RESULTS: Magnesium levels were decreased in patients of chronic alcoholism with 1.5±0.09 mg/dl (mean±SD) when compared to controls with 2.2±0.21mg/dl (mean±SD) with a statistically significant 'p' value of <0.001. Serum electrolytes i.e sodium levels increased in cases with a (mean±SD)145 ±1.23 mEq/L when compared to controls with138±3.13 mEq/L(mean ±SD) with a statistically significant 'p' value of <0.001. Potassium levels decreased in cases with 3.2±0.14 mEq/L (mean±SD) when compared to controls with 3.7±0.25 mEq/L (mean±SD) with a statistically significant 'p' value of <0.001. CONCLUSION: In chronic alcoholism the renal tubular defects contribute to the abnormalities of serum electrolytes and magnesium metabolism.

Key Words: chronic alcoholism, magnesium, electrolytes.

I. Introduction

Chronic alcoholism is an important health and social problem that is prevalent all over the world. In India it is one of the leading causes for morbidity and consequent loss of man hours. Chronic alcoholics are at a risk for multiple system failure because of micronutrient deficiency and toxic effects of alcohol on all tissues. Hence there is a potential for exaggerated risk for morbidity and mortality.

Chronic alcoholism is a disease, which progresses overtime resulting in loss of control over alcohol consumption, but can return to normal life only as long as drinking is completely stopped. A typical adult chronic alcoholic will have 10 to 15 years of drinking and lots of instigating factors which contribute to the complications due to chronic alcoholism. Alcohol abuse may result in a wide range of electrolyte and acid–base disorders, including hypophosphatemia, hypomagnesemia, hypocalcemia, hypokalemia, metabolic acidosis and respiratory alkalosis.

Alcoholics have a buildup of a substance tetrahydroisoquinoline (THIQ) in their brain, which is highly addictive and resembles morphine. Most of the chronic effects of alcoholism are due to
(a) Accumulation of fat in liver cells leading to fatty liver.
(b) Accumulated toxic effects of acetaldehyde.

Both result in cellular death, followed by replacement with fibrous tissue mainly of liver leading to cirrhosis, when liver functions are reduced, hepatic coma results. Excess may lead to cell death. Five percent of all deaths in India are due to liver diseases for which the most important culprit is alcohol.

Ethanol is readily absorbed when consumed as an aqueous solution. Some quantity is absorbed by the stomach itself, but most of it is absorbed by intestine. Only 1% of the ingested alcohol is excreted through the lungs, urine or sweat in the unoxidized form. Major fraction of ingested alcohol is oxidized in the liver by various enzymes. It is converted to acetaldehyde by the action of the enzyme alcohol dehydrogenase. It generates species of oxygen free radicals leading to lipid peroxidation and mitochondrial damage. The cell membranes are damaged, resulting in generalized reduction in the reabsorptive ability of the proximal tubular cells of the kidney. As a result serum magnesium is decreased and the electrolytes are also affected.

Present study aims to estimate the serum magnesium and serum electrolyte levels. Magnesium is essential for all enzymatic processes involving ATP in the body. It plays an important role in oxidative phosphorylation. Electrolytes like sodium, potassium and chloride are essential to the body as they are
responsible for maintaining the body's electrolyte and water balance. They play an important role in nerve conduction, muscle contraction and transport of substances across the membranes. Ethanol is responsible for derangement of monovalent and divalent cation metabolism and acid-base homeostasis.\(^4\)

II. Materials And Methods

The present study was conducted on fifty patients with chronic alcoholism. Patients were from the Psychiatry Department of King George Hospital, Andhra Medical College, Visakhapatnam, Andhra Pradesh, India. Their ages ranged from 30-70 years, all are men. Study also included 25 non alcoholic healthy controls who are age matched.

Inclusion Criteria
1. Duration of alcohol consumption of more than 10 Years, 15 drinks/week.
2. Age group between 30-70 years.

Exclusion Criteria
1. Patients with associated liver disease and pancreatic diseases.

Method: Estimation of serum magnesium levels was done by Calmagite method (colorimetric).

Principle: Magnesium ions react in an alkaline medium with the metallochrome dye calmagite to form a chromophore which absorbs at 520 nm. Calcium is excluded from the reaction by complexing with EDTA.

Procedure: 3ml of blood is taken in a test tube, serum is separated by centrifugation. Test done by Calmagite method. The optical densities were measured colorimetrically. Normal range of magnesium is 1.7 -2.7 mgs/dl.

Serum sodium and potassium were estimated by ion selective electrode method by using Dalko Elyte Electrolyte Analyzer. Normal Range of serum sodium is 136-145mEq/L. Normal range of serum potassium is 3.5-5mEq/L.

Results And Observations

In the present study mean value of magnesium among cases is 1.5± 0.09 mg/dl (mean ± SD) and that of controls is 2.2 ± 0.21mg/dl (mean ± SD ). The decrease in the serum magnesium levels among the cases is significant with a ‘p’ value of <0.0001.

Mean value of sodium among the cases is 145±1.23 mEq/L (mean ± SD) compared to that of controls 138± 3.13mEq/L (mean ± SD). The increase in the serum sodium levels among cases is significant with a ‘p’ value of 0.001.

Mean value of potassium among cases is 3.2 ± 0.14mEq/L (mean ± SD) compared to that of controls 3.7± 0.25mEq/L (mean ± SD). The decrease in the levels is significant with a ‘p’ value of 0.001.

Mean value of chloride among cases is 101.7±3.18mEq/L (mean ± SD) in comparison to healthy individuals 99.36±10.86 mEq/L (mean±SD). Serum chloride did not show any change in comparison to controls, ‘p’ value <0.629 not significant.

IV. Discussion

Fifty (50) patients of chronic alcoholism were studied for serum magnesium and other electrolytes. Twenty five (25) healthy controls were taken for comparison. In all cases and controls serum magnesium and other electrolyte levels are measured by appropriate methods. In chronic alcoholism the toxic effects of acetaldehyde which is formed during the metabolism of ethanol, result in a generalized reduction in the reabsorptive ability of the proximal tubular cells of the kidney. The oxidation of ethanol also generates species of oxygen free radicals which are capable of damaging cells’ These changes are reversible with abstinence of alcohol. Magnesium is involved in many of the biochemical reactions that take place in the cell, particularly in those processes involving the formation and utilization of ATP. It is the activator of many enzymes like kinases requiring ATP. It also helps maintain normal muscle and nerve function. It keeps the bones strong and present...
as apatites in the bones. Alcohol may interfere with the formation of new bone. This can lead to osteoporosis of bones and increased risk of fractures.

In the above study it was found that serum magnesium levels have decreased in cases 1.5±0.09 mg/dl (mean ±SD) when compared to controls 2.2±0.21mg/dl (mean ± SD) with a ‘p’ value of <0.0001 which is statistically significant. The values in the present study are in consistent with the study of De Marchi S, Cecchin E, Basile A et al. in December 1993 with a ‘p’ value of <0.008. Ingestion of ethanol causes a marked increase in urinary excretion of magnesium due to defective tubular reabsorption. This renal loss may account for hypomagnesemia as observed in this study. The magnesium deficiency is further exacerbated by lack of magnesium in the regular diet because of micro nutrient deficiency due to chronic alcoholism.

Alcohol consumption has major effects on absorption, elimination and serum concentration of many physiological important electrolytes. It can induce excessive urinary excretion of magnesium, calcium and phosphorus. Sodium and potassium assist in the maintenance of the body’s electrolyte and water balance, electrolyte disturbance may lead to severe and even life threatening metabolic abnormalities.

In the present study sodium levels increased significantly in cases 145±1.23 mEq/L (mean+ SD) compared to controls 138±3.13mEq/L (mean±SD) and decreased potassium levels were observed in cases 3.2±0.14mEq/L(mean+SD) compared to controls 3.7±0.25mEq/L (mean+SD), whereas Chloride levels were not significantly altered.

Hypokalemia is relatively a common electrolyte abnormality observed in alcoholic patients. It may be due to impaired reabsorption of potassium by the damaged renal tubular cells, along with co-existent hypomagnesemia. This study indicates that ethanol is responsible for the derangement of monovalent and divalent cation metabolism and acid-base homeostasis independently of malnutrition, liver diseases, pancreatitis and other intercurrent illness.

V. Conclusion

In patients of chronic alcoholism the renal tubular defects contribute to the abnormalities of magnesium and other electrolytes metabolism. The defects are transient and are reversible by abstinence of alcohol. These defects should be taken care of to lessen morbidity and mortality in such patients.

References