PLASMA ELECTROLYTES AND ENZYMES IN COUNTRY LIQUOR POISONING

K. P. MISTRY, S. C PARIKH AND R. T. DAVE

Department of Clinical Chemistry,
Medical College, Baroda - 390 001

(Received on November 2, 1981)

Summary : Plasma sodium, potassium, bicarbonate, calcium, magnesium, pseudocholinesterase, amylase and alkaline phosphatase were estimated in 43 cases of country-liquor poisoning and 29 normal controls. In the poisoned subjects, plasma potassium, magnesium and amylase levels were elevated, while plasma bicarbonate levels were diminished; and plasma pseudocholinesterase and alkaline phosphatase were not affected. Plasma calcium and pseudocholinesterase were elevated in poisoned patients who recovered; however, these were diminisihed in fatal cases. Plasma bicarbonate and amylase were affected depending upon the severity of poisoning

Key words : electrolytes enzymes country liquor poisoning

INTRODUCTION

Alcohol drinking is a very old habit. In "Dry–States", addicts are attracted to take country liquors, which are sometimes contaminated by toxic substances. These toxicants, known as congeners, are aldehydes, ketones, ethers, esters, organic acids, methanol and higher aliphatic or aromatic alcohols.

Blood samples of 43 subjects who were victims of country liquor poisoning were examined. The liquor was found to be contaminated with methanol as reported by Forensic Laboratory. The blood svmples were analysed for electrolytes and some enzymes to find out the effects of methanol poisioning on these parameters.

MATERIALS AND METHODS

For comparison 29 normal subjects were included in the study. The age of normal subjects varied from 18-43 years and of the poisoned patients between 20–60 years. One subject in each group was female, and the rest were males. Among the 43 poisoned

patients, eight expired during the course of treatment, three survived with loss of vision, and, 32 recovered with good vision.

The blood samples were collected in heparinised vials; and plasma was analyzed for sodium and potassium (by flame photometry), calcium (10), magnesium (10), bicarbonate (by Van Slyke's Volumetric Apparatus), pseudocholinesterase (9), amylase (1) and alkaline phosphatase (9).

RESULTS

Table I shows the results in normal and poisoned cases. The plasma biochemistry was studied in all poisoned subjects; and the poisoned cases were subdivided into three sub-groups according to the severity of poisoning: (1) cases who recovered with good vision, (2) those who recovered but lost vision and (3) who died during the course of treatment.

The plasma sodium was not affected in the poisoned cases, or in poisoned patients who expired. There were increases in plasma potassium concentrations in all the poisoned subjects. Plasma bicoarbonate showed a marked reduction, and, it was minimally affected in patients who recovered and was maximally affected in patients who died. There was no effect on plasma calcium in the overall poisoned group; however, it was reduced in the patients who recovered. In all the poisoned subjects there was an increase in plasma magnesium concentration.

The pseudocholinesterase activity was reduced in the patients who recovered, was increased in poisoned cases who died and was unaffected in overall poisoned cases. The amylase activity increased in all the poisoned patients depending upon the severity of poisoning. There was no significant effect on plasma alkaline phosphatase.

DISCUSSION

Both ethanol and methanol cause lactic acidosis (8) and methanol also causes formic acidosis (6) Ethanol depresses the respiratory centre, thus causing simultaneously metabolic and respiratory acidosis (8).

In the present study while plasma sodium was unaffected, plasma potassium was increased in country-liquor poisoned cases. Nicholson and Taylor (7) however, reported a decrease in plasma sodium and an increase in potassium. This increase in potassium was attributed to the efflux of cellular potassium in exchange with plasma hydrogen ions (4). Kalbfleisch (5) observed no change in plasma sodium, potassium, calcium and magnesium. Rubini *et al.* (8), Eggleton and Smith (2) and Kalbfleisch (5) showed decreases in

TABLE I : Various plasma biochemical parameters in normal and country liquor-poisoned subject*

		Sodium (mEq/L)	*Potassium (mEq/L)*	*Bicarbonate (mEq/L)*	*Calcium (mg/100 ml)*	*Magnessium (mg/100 ml)*	*Pseudocholinesterase (IU/ml)*	*Amylase (Somegyi Units/100 ml)*	*Alkaline phosphatase (KA Units/100 ml)*
	Normal (NR)	139.3 ±5.7 (26)	3.5 ±0.3 (26)	21.5 ±2.4 (30)	9.61 ±0.49 (29)	1.37 ±0.59 (28)	175.4 ±42.4 (29)	89.4 ±38.0 (26)	3.6 ±1.4 (22)
	Overall (PO)	140.1 ±7.6 (22)	4.8 ±0.9 (22)	11.2 ±4.4 (41)	9.21 ±0.92 (39)	2.11 ±0.78 (37)	190.3 ±58.3 (39)	199.3 ±35.3 (20)	2.4 ±1.2 (12)
POISONED	Recovered with good vision (PR)	138.8 ±7.3 (19)	4.8 ±0.9 (19)	11.7 ±4.6 (29)	9.02 ±0.96 (31)	2.05 ±0.80 (30)	136.4 ±63.4 (29)	197.0 ±32.9 (16)	—
	Recovered but lost vision (PL)	—	—	8.8 ±1.4 (3)	9.81 ±0.66 (2)	2.70 ±0.54 (2)	166.7 ±73.9 (3)	—	—
	Died (PD)	148.1 ±6.7 (3)	4.6 ±0.8 (3)	8.7 ±1.0 (7)	9.75 ±0.44 (6)	2.41 ±0.71 (6)	219.0 ±48.1 (5)	243.3 ±19.0 (3)	
'P' VALUES	NR Vs PO	>0.1	<0.005	<0.005	>0.1	<0.005	>0.1	<0.005	>0.1
	NR Vs PR	>0.1	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	—
	NR Vs PD	<0.05	<0.01	<0.005	>0.1	<0.005	>0.05	<0.005	—
	PR Vs PD	<0.025	>0.1	<0.005	<0.005	>0.1	>0.1	<0.005	—

*±S.D., Number of observations are given in parentheses

urinary excretion of sodium and potassium in alcoholics. Thus it is possible that these ions are reabsorbed at kidney level in exchange with hydrogen ions, causing increases of plasma sodium and potassium. Kalbfleisch (5) also reported an increase in urinary calcium and magnesium due to diuretic effect of alcohol.

The present study confirms the reduction in plasma bicarbonate reported by Rubini *et al.* (8) in alcoholics.

Goldberger (4) suggested that severe acidosis might cause dissolution of bone minerals, enabling calcium and magnesium to come out to neutralise acidity. This may be the cause of increased plasma magnesium reported in the present study.

In the poisoned cases who recovered with good vision, plasma psuedocholinesterase was reduced, while it was elevated in poisoned cases who died. It is difficult to explain this observation.

The variations in the effects reported by different workers may be due to factors like amount of alcohol consumed, the way it was consumed, nutritional status of the consumer and long-term or short-term effects.

The main cause of death in the present study was respiratory failure, as ethnol causes depression of respiratory centre (3) It has been suggested that formic acid, an intermediary metabolic product of methanol, is responsible for blindness (6)

ACKNOWLEDGEMENTS

The authors express deep gratitude to late Dr. P.T. Acharya, who guided this research work.

REFERENCES

1. Carrawa, T.C. A stable starch substrate for determination of amylase in serum and other body fluids. *Am. J. Clin. Pathol.*, **32** : 97-99, 1957.
2. Eggleton, M.G. and I.G Smith The effect of ethyl alcohol and some other diuretics on chloride excretion in man. *J. Physiol. (London)*, **104** : 435–442, 1946.
3. Gibbins, RJ., Y. Israel, H. Kalant, R.E. Popharo, W. Schmidt and R.G. Smart. Research advances in alcohol and drug problems, Vol. I, A Willeg-Biomedical Health Publication, pp 93–198, 1974.
4. Goldberger, E. A primer of water electrolyte and acid-base syndromes., 5th Ed., K.M. Varghese Co., Bombay, pp 343, 1975.
5. Kalbfleisch, J.M., R.D. Lindenmann, H. B. Ginn and W. O. Smith. Effects of ethanol administration on urinary excretion of magnesium and other electrolytes in alcoholics and normal subjects. *J. Clin. Invest.*, **42** : 1471–1475, 1963.
6. Martin, A.G., K.E. McMartin, S.S. Hayrch, M.S. Hayrch and T.R. Tebby. Methanol poisoning : Occular toxicity by formate. *Toxicol. Appl. Pharmac.*, **45** : 201–208, 1978.
7. Nicholson, W.M. and H.M. Taylor. The effect of alcohol on the water and electrolyte balance in man. *J. Clin. Invest.*, **17** : 279–282, 1938.
8. Rubini, M.E., C.R. Kleeman and E. Lamdin. Studies on alcohol diuresis. I. The effect of ethanol ingestion on water, eectrolyte and acid-base metabolism. *J. Clin. Invest.*, **34** : 439–447, 1955.
9. Varley, H. Practical Clinical Biochemistry 4th Ed., Arnold Heinemann Publisher's (India) Pvt. Ltd., pp. 300–301, and 455–456, 1978.
10. Wilkinson, R.H. A Micromethod for serum calcium and serum magnesium. *J. Clin. Pathol.*, **10** : 126–135, 1957.