JJBS

Volume 3, Number 2, March 2010
ISSN 1995-6673
65 - 68

Jordan Journal of Biological Sciences

Exposure to Potassium Carbonate Emulsion Induced Nephro-Toxicity in Experimental Animals.

Akintunde J.K [a], Opeolu B [b] and Aina O.O [c]

[a] *Federal University of Technology,Department of Biochemistry, Ondo state, Nigeria.*

[b] *Department of Veterinary physiology, Biochemistry and Pharmacology, University of Ibadan, Nigeria*

[c] *Department of Veterinary Anatomy, Faculty of Veterinary Medicine, University of Ibadan, Nigeria.*

Abstract

The study investigated the possible toxic effects of potassium carbonate emulsion on some biomarkers of tissue damage in rabbits. Exposure of rabbits to potassium carbonate (K_2C0_3) emulsion at 50mg/L and 100mg/L via oral drinking for 14 consecutive days caused significant increase in the creatinine and uric acid at 100mg/L by 48.6% and 126.3% respectively. Also, potassium carbonate (K_2C0_3) emulsion significantly increased serum blood urea nitrogen (BUN) at 50mg/L and 100mg/L concentration. The results however suggested that potassium carbonate emulsion exposure via oral drinking could precipitate kidney damage.

Keywords: Potassium Carbonate Emulsion, Nephro-Toxicity, Serum Metabolites.

1. Introduction

Potassium is an essential dietary mineral and electrolyte. Normal body function depends on tight regulation of potassium concentrations both inside and outside of cells (Peterson, 1997). A limited number of enzymes require the presence of potassium for their activity. The activation of sodium, potassium-ATPase requires the presence of sodium and potassium. The presence of potassium is also required for the activity of pyruvate kinase, an important enzyme in carbohydrate metabolism (Sheng, 2000). Severe hypokalemia may result in muscular paralysis or abnormal heart rhythms (cardiac arrhythmias) that can be fatal (Sheng, 2000; Food and Nutrition Board, 2004). The richest sources of potassium are fruits and vegetables. People who eat large amounts of fruits and vegetables have a high potassium intake (8-11 grams/day) (Sheng, 2000; Food and Nutrition Board, 2004). A recent dietary survey in the U.S. indicated that the average dietary potassium intake is about 2,300 mg/day for adult women and 3,100 mg/day for adult men (Hajjar et al., 2001). The use of potent potassium supplements in potassium deficiency requires close monitoring of serum potassium concentrations. Potassium supplements are available as a number of different salts, including potassium chloride, citrate, gluconate, bicarbonate, aspartate and orotate (Hendler & Rorvik, 2001). Abnormally elevated serum potassium concentrations are referred to as hyperkalemia. Hyperkalemia occurs when potassium intake exceeds the capacity of the kidneys to eliminate it. The most serious complication of hyperkalemia is the development of an abnormal heart rhythm (cardiac arrhythmia), which can lead to cardiac arrest (Mandal, 1997). The present study was undertaken to elucidate toxicity associated with exposure to potassium carbonate emulsion.

2. Materials and methods

Twelve California rabbits (males) weighing 1.05kg – 1.65kg were purchased from the animal house of the college of animal Science University of Agriculture, Abeokuta, Ogun State, Nigeria, housed in cages and exposed to 12hr light/ dark photoperiod cycle . The animals were left to acclimatize for two weeks prior to the administration of the emulsion. They had free access to food and water *ad libitum.*

2.1. Exposure study

The experimental animals were divided into three groups of four. Groups I and II received 50ppm and 100ppm of potassium carbonate (K_2Co_3) emulsion respectively for two weeks and group III was administered with physiological saline for the same period.

2.2. Blood Sample Collection

The animals were sacrificed by cervical dislocation after a 12hour fasting period. In all cases, blood samples

were collected by cardiac puncture into clean dry centrifuge tubes and allowed to coagulate by standing for 30minutes. The blood samples were then centrifuged for 10minutes at 3000g using a bench centrifuge. Plasma aliquots were collected and preserved at 70^0C for biochemical assays.

2.3. Clinical chemistry

Serum total protein, Albumin and potassium ion (K^+) as well as biomarkers of nephro-toxicity such as urea, creatinine and uric acid. The total protein was determined by Lowry et al (1951). Albumin concentrations were measured by the stigma bromocresol purple (BCP) (Beale and croft, 1961). Serum K^+ was determined by flame emission spectrophotometer using UNICAL 1300. The estimation of urea was measured by the method of coloumbe and Farreaus (Hervey, 1953). Creatinine was determined by the method of Eichhorn et al (1961). Uric acid was estimated by the method of Henry using commercial kit (Randox).

2.4. Statistical Analysis

Treated groups were compared to control group by student's t- test using Microsoft excel. All data were expressed as mean ± S.D (n = 4). A value of $P < 0.05$ was considered to indicate a significant difference.

3. Results

The result of effects of potassium carbonate emulsion on plasma albumin, plasma protein and blood K^+ ion are presented in Table 1. Treatment with the emulsion of carbonate resulted in significant increase in serum potassium ion (K^+) and albumin.

Potassium carbonate emulsion in rabbits caused significant increase in total albumin in concentration dependent manner when compared with the control. The levels of total protein decreased but not in concentration dependent manner when compared with the control. Table 2 shows the result potassium carbonate emulsion on uric acid, creatinine and urea. The emulsion significantly increased uric acid, creatinine and urea by 126.3%, 48.6% and 458.8% respectively at highest concentration (100mg/L) when compared with the control ($P<0.05$). As shown in table 2.0, the results of urea, creatinine and uric acid showed that oral administration of the potassium carbonate emulsion significantly increased at concentration 100mg/L by 458.8%, 48.6% and 126.3% respectively when compared with the control.

Table 1: The effects of potassium carbonate emulsion on plasma proteins and electrolyte.

Parameter	Control	50mg/L	100mg/L
Albumin	3.64±0.03	4.07± 0.32* (11.8)**	4.95± 0.75* (36.0)**
Total Protein	9.73±0.08	8.31 ± 1.16 (14.6)**	8.39 ± 0.94* (13.8)**
Blood K^+ ion	0.78±0.01	2.99 ± 1.84* (283.3)**	2.34 ± 2.06* (200.0)**

*Values differ significantly from control ($P < 0.05$).

*Percentage change compared with the control.

Table 2: The effects of potassium carbonate emulsion on the kidney.

	Control	50mg/L	100mg/L
Urea	0.80±0.00	2.83±0.25*(253.8)**	4.47±0.85*(458.8)**
Creatinine	0.70±0.09	0.97±0.37(38.6)**	1.04±0.16*(48.6)**
Uric acid	0.80±0.21	1.51±0.73(88.8)**	1.81±0.21*(126.3)**

* Values differ significantly from control ($P<0.05$)

**Percentage change compared with the control.

4. Discussions

Civilization and technological advancement had created a great harm to human health through the introduction of food additives as chemical preservatives, flavours, seasonings, modification of food texture, nutritional quality or colorants (Timothy and Peckham, 1978). Similarly, there have been increasing efforts in agricultural activities particularly in preservation of farm products which is as a result of the ever-increasing human population as well as food losses to pests and disease both on filed and in storage (Sheng, 2000; Food and Nutrition Board, 2004). Some of these activities and chemical preservatives have resulted in food poisoning and unsafe for human consumption with significant public health implications while some are carcinogenic inducers e.g. potassium bromate (Hajjar et al., 2001; Hendler & Rorvik, 2001; Mandal, 1997).

In the present study rabbits were orally exposed to different concentrations of potassium carbonate emulsion. Early works on the study of potassium carbonate emulsion had established that preservatives are dose-dependent. It was observed that sodium and potassium salts of benzoic acid have been found to cause no deleterious effect when used in small quantity (Morris et al., 1986; Milks, 1991). Also, potassium carbonate before meals promotes the

secretion of gastric juice but large doses neutralized the free hydrochloric acid (HCL) in the stomach and render the chyme neutral or alkaline, this interferes with the secretions from the pancreas, liver and intestines and hindered digestion (Mikkelsen, 1984). It was investigated that potassium ion causes depression of the central peripheral nervous system by depressing the reflexes and paralyses by higher concentration (Welt et al., 1960; Perkins, 1984; Weisburg, 1999). It was also noted that salt poisoning causes progressive muscular weakness, inflammation of the gut, dark colour liver, inability to stand, convulsion, excessive thirst and eventually death with haemorrhage and severe congestion in gastro intestinal (GIT) tract, liver, muscle and kidney (Gordon and Andrew, 1966; Shahr and John, 1991).

In acute or chronic renal failure, the use of potassium-sparing diuretics and insufficient aldosterone secretion (hypoaldosteronism) may result in the accumulation of excess potassium due to decreased urinary potassium excretion. This suggests that the emulsion of the carbonate might precipitate kidney damage. The significant decrease in protein synthesis as recorded in the study indicated possible damage(s) to liver however reducing protein synthesis. The rabbits treated with 100ppm of the emulsion showed symptoms such as withdrawal from food, excessive thirst, drowsiness (weakness, reduced irritability, and polyuria, an indication of potassium acute toxicity (Mandal, 1997; Liu et al., 2000).

In conclusion, this study shows that high exposure to emulsion of potassium carbonate (K_2, CO_3) may precipitate nephro-toxicity and induce liver damage. This then suggests a potential risk to humans that may come in contact with this supplement since liver and kidney are the major sites of chemical and drug metabolism.

References

Beale RN, Croft RN. A sensitive method for determination of urea. J Clin Path 1961, 418-424.

Lowry O Hm, Rosebrough NJ, Farr A L & Randall RJ. J Biol Chem, 1951; 193 265.

Eichhorn F, Zelmanowski S, Lew E, Rutenberg A, Fanias B. Improvement of the

uric acid determination by the carbonate method for serum and urine. J Clin Pathol. 1961 14(4): 450–452.

Food and Nutrition Board, Institute of Medicine: (National Academies

Press).Potassium. Dietary Reference Intakes for Water, Potassium, Sodium,

Chloride, and Sulfate. Washington, D. C.: Natl Acad Press. 2004;173-246.

Hajjar IM, Grim CE, George V, Kotchen TA. Impact of diet on blood pressure and age- related changes in blood pressure in the US population: analysis of NHANES III. Arch Intern Med 2001; 161(4):589-593.

Hendler SS, Rorvik DR. eds. PDR for Nutritional Supplements. Montvale: Medical Economics Company, Inc; 2001.

Hervey G R. determination of Creatinine by the Jaffe reaction. Nature 1953, 171 (4364): 1125

Keller A. Total serum protein. In Kaplan, L. A. and A. J Pesce (Ed) clinical chemistry Theory Analysis and correlation St. Louis Mosby company USA, 1984.

Kolthoff I.M.; Elving P.J.: Treatise in analytical chemistry. Part I and II. John Wiley and Sons. 1976, 15:15-105.

Liu S, Manson JE, Lee IM, et al. (2000). Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study. Am J Clin Nutr. 72(4):922-928.

Mandal AK. Hypokalemia and hyperkalemia. Med Clin North Am. 1997; 81(3):611-639.

Mikkelsen: Potassium secretion by distal tubule after potassium adaptation,

American Journal of physiology, 1984, 221-437.

Milks HJ. Veterinary Pharmacology material medical and therapeutics. 1991,10: 438 – 440.

Morris GK, Cliver O, Cochranne BA. Progress in food safety, food research

institute,University of Wisconsin 1986, 46: 1340.

Perkins. Potassium and sodium in the skeletal muscle Laeger Ugeskr, 1984, 15:

4007– 4010.

Peterson LN: Potassium in nutrition. In: O'Dell BL, Sunde RA, eds. Handbook of

nutritionally essential minerals. New York: Marcel Dekker, Inc; 1997:153-183.

Shahr HM, John W. Analytical methods in Toxicology, 1991, 10: 52 – 53.

Sheng H-W. Sodium, chloride and potassium. In: Stipanuk M, ed. Biochemical and Physiological Aspects of Human Nutrition. Philadelphia: W.B. Saunders Company. 2000; 686-710.

Timothy M. Peckham: General and applied toxicology 1978; 1304.

Weisburg. Release of intracellular potassium as the physiological stimulus for pain: Journal of Applied physiology, 1999, 14, 463.

Welt LG, Hollander W, Blythe W. The consequence of potassium depletion.

Journal Chronic disease 1960, 11: 213 – 214.