Exposure to potassium carbonate emulsion induced nephro-toxicity in experimental animals.

SHORT COMMUNICATIONS

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Abstract

الملخص

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). Potassium carbonate (Pearl ash or potash) is not used as potassium supplement. It is strongly alkaline and it is mainly used in the production of soap and glass. 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.*

3. Exposure study

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

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3.1. 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.

3.2. Clinical chemistry

Serum total protein, Albumin and potassium ion (K^+) as well as biomarkers of nephro-toxicity such as urea, creatinine and uric acid were determined. The total protein was determined by Lowry et al (1951). Albumin concentrations were measured by Sigma bromocresol purple (BCP) as described by (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 using commercial kit (Randox) according to manufacturer's protocol.

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.

5. 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.

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

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

Values are expresses as mean±S.D (n=5)

*Values differ significantly from control (P < 0.05)

** Percentage change compared with the control

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 significantly but not in a 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 50mg/L by 253.8%, 38.6% and 88.8% respectively when compared with the control.

Table 2: The effects of potassium carbonate emulsion on plasma metabolites.

Parameters	Urea (mg/dL)	Creatinine (mmol/L)	Uric acid(mmol/L)
Control	0.80±0.01	0.70±0.09	0.80±0.21
50mg/L	2.83±0.25*(253.8)**	0.97±0.37(38.6)**	1.51±0.73(88.8)**
100mg/L	4.47±0.85*(458.8)**	1.04±0.16*(48.6)**	1.81±0.21*(126.3)**

Values are expresses as mean±S.D (n=5)

*Values differ significantly from control (P < 0.05)

** Percentage change compared with the control

6. 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 a result of the ever-increasing human population as well as food losses to pests and disease both on the field and in storage (Sheng, 2000; Food and Nutrition Board, 2004). Some of these activities and chemical preservatives have resulted in food poisoning and becoming 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 (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 cause kidney damage. The significant decrease in protein synthesis as recorded in the study indicated possible damages to liver causing reducing protein synthesis. The rabbits treated with 100mg/L 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_2CO_3) may precipitate nephro-toxicity and induce liver damage. This also suggests a potential risk to humans who may come in contact with this supplement since liver and kidney are the major sites of chemical and drug metabolism.

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