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Sub-acute effect of ingesting potassium iodide salt on some visceral organs of domestic mice (*Mus musculus*)

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ABSTRACT: The effect of ingesting potassium Iodide (KI) salt was determined using sixteen(16) male domestic mice (*Mus musculus*), aged 2-3 months and of weight range 27.0gm to 38.6gm. KI salt of 500, 1000, and 1500mg/kg body weight doses dissolved in distilled water were given *ad libitum* to rats to determine both physical and histological changes induced by the different doses of the salt on the experimental animals. Physical changes observed include loss of appetite, sluggishness, weight loss, altered body conditions, lethargy, abdominal enlargement and death. These changes were marked in the group that received higher dosages of KI. The liver and kidney processed for microscopy showed remarkable histological lesions of varied intensity. The kidney exhibited tubular hyalinization, while the liver revealed chronic venous congestion, intraportal ulceration and portal hypertrophy among other effects. These lesions are indicative of potassium iodide induced toxicity. From this study, it is evident that ingesting KI salt in high doses has deleterious effects on some visceral organs of albino rats.

Key Words **:** Toxicology**;** Potassium iodide, Rat Kidney, Rat Liver.

Introduction

Potassium iodide is a white crystalline salt used in photography and radiation treatment. It also finds widespread application as an iodide source because it is less hygroscopic than sodium iodide, making it easier to work with. It has been reported that, one third of the world's population lives in areas of I deficiency [1], and this has been documented as the world's leading cause of intellectual deficiency . Iodine/iodide has been successfully used in the treatment of so many illnesses [2]. Some of these conditions include fibromyalgia, thyroid disorders, chronic fatigue immune deficiency syndrome, autoimmune disorders as well as cancer.

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Apart from inducing goitre and reduced thyroid hormone synthesis [3] , iodine deficiency sets up the immune system to malfunction which can lead to many of the above named disorders developing. Other associated iodine disorders documented in laboratory animals include decreased egg production in hens [4], decreased haemoglobin level in pigs [5], necrotic lesion in the liver of dogs [6]. Indiscriminate use of various iodine preparations may result to many cases of poisoning [7]. Though various species can be seen to differ widely in their susceptibility to iodine toxicity, all animals can tolerate iodine levels far in excess of their requirements for this element [5]. As a result of the treatment and prevention of goitre and hyperthyrodism, a recommended daily intake of Iodine for adults of both sexes in North America and Western Europe varies from 150 to 300 µg [1].

Iodine toxicity is rare in animals and human, but nuclear explosions that give off radioactive iodine and the ingestion of excessive stable iodine in parts of the world where seaweeds are heavily consumed represent specialized iodine toxicity concerns [8]. Also in the treatment of some disease conditions such as fibrocystic disease of the breast, cancer etc, various concentrations of the iodides of the element very much higher than what ideally would be required for normal thyroid function, have been administered to patients with these needs in highly varying concentrations by the various clinicians and also in the various parts of the world.

Since it is possible for potassium iodide to find its way into human ecosystem or the food chain of biologic organisms, in such concentration as to lead to unprecedented bio-accumulation of the substance, which can constitute a hazard. This work was designed; to ascertain the adverse sub-acute effect of potassium Iodide on the glandular epithelial cell of the liver and the Kidney of domestic mice (Mus musculus) and to characterize grossly and microscopically, the degree of its effect on these selected organs.

Materials and Method

Experimental Animals

Sixteen (16) inbred domestic mice (*Mus musculus*), all males, aged 2-3 months old, and of weight range 7.0 gm to 38.6 gm were used for this study. The animals used were obtained from the Animal House of Faculty of Veterinary Medicine, University of Nigeria, Nsukka. The animals were kept in Animal House of Anatomy Department, College Of Medicine, University of Nigeria, Enugu Campus. They were housed in cages and fed with commercial diet and water ad libitum for 28 days to acclimatize to the new environment. At the end of the period of acclimatization the Animals were divided into four (4) groups: A, B, C, and D. Individual identification of the animals was done by metal ear tags. All rats were maintained and utilized in accordance with the National Institute of Health (NIH) guideline for care and use of laboratory animals.

Test Substance

Analar potassium iodide salt obtained from Conraw Hospital Equipment and Chemical Company, Nigeria Limited was used for the study.

Experimental Design

Cage A served as control, while cages B, C, D, constituted the tests. Potassium Iodide (KI) salt was weighed out, dissolved in l litre of distilled water and reconstituted to obtain
required concentrations in milligram (mg) per kilogram (kg) body weight (b.w.) based on derived value of L.D50 as follows: 500 mg KI/kg b.w., 1000mg KI/kg b.w., 1,500mg KI/kg b.w. Animals in Group A, received distilled water only.
Group B received 500mg KI/kg b.w.
Group C received 1000mg KI/kg b.w.
Group D received 1,500mg KI/kg b.w.

All groups were fed with commercial diets (Standard Top Feed) and water ad libitum throughout the duration of the experiment which lasted for 21 days.

Weights of the animals were taken and recorded just before commencement of administration of KI and at regular weekly intervals throughout the duration of the experiment. The animals were daily monitored for signs of distress such as weight loss, alopecia, lethargy or other behavioural changes

including disorientation, dullness, loss of appetite or death. Concentrations of KI at which such signs or changes occurred were clearly recorded. On the 22nd day, 2 animals randomly selected from each group were painlessly sacrificed by exposure to inhalation of chloroform anaesthetic vapour. Some visceral organs namely kidney and liver were dissected out from each animal. They were all observed grossly, before being processed histologically for microscopic studies.

Results

Morphological observations:

The animals were observed throughout the duration of the experiment. Weight drop was evident in treated groups and most pronounced in the highest dosage group. The drop in weight continued until the third week. The most significant weight loss was recorded in group D. The animals in the control group continued increasing in weight through out the duration of the experiment. The most consistent physical observation noted mainly in cage D, were sluggishness, weight loss, and loss of appetite, altered body conditions such as ruffled hairs, loss of hair, lethargy, abdominal enlargement and death. Samples of liver and kidney obtained from control rats showed no evidence of anatomical abnormalities of size, colour, or consistency. In group D haemorrhage was seen in the liver and kidney. These organs were diminished and shrunken.

Histological Findings

Observed in the kidney of the animals in group D were hyalinization and hyaline droplets in the tubules. The most significant lesion observed in the liver of the rat in the same group D were chronic venous congestion (stasis) as well as intraportal ulceration and portal hypertrophy. Partial vascular thrombosis of the central vein and intralobular necrosis are evident. Some of the hepatocytes showed perinuclear haloes and are also necrotic. The other animals in the rest treated groups maintained normal histological features. See plates 1-7 for Histopathology.

Table1. Mean weight values and the standard deviations over the period of experiment.

Cages	Doses in mg/kg b.w.	Baseline weight Mean S.D.	Weight in Week 1 MSD	Week 2	Week 3
A	0	33.1 ± 5.0	33.3 ± 5.5	35.5 ± 4.0	37.8 ± 2.5
B	500	32.0 ± 0.6	31.8 ± 0.4	31.5 ± 2.5	34.4 ± 1.7
C	1,000	35.8 ± 4.4	33.8 ± 4.2	32.3 ± 3.9	32.9 ± 4.0
D	1,500	29.9 ± 2.9	29.6 ± 2.1	25.6 ± 2.0	23.0 ± 4.2

Doses of KI in mg/kg B.W.

Table 2: Result of Acute Toxicity.

Cages	No. of Rats	Dose of KI Mg k/g b.w.	Log dose	No. dead	% Mortality
A	4	0.00	0.00	0/4	0
B	4	500	2.70	0/4	0
C	4	1000	3.00	0/4	0
D	4	1500	3.18	2/4	50%

The LD 50 of potassium iodide is 1500 mg/kg body weight.

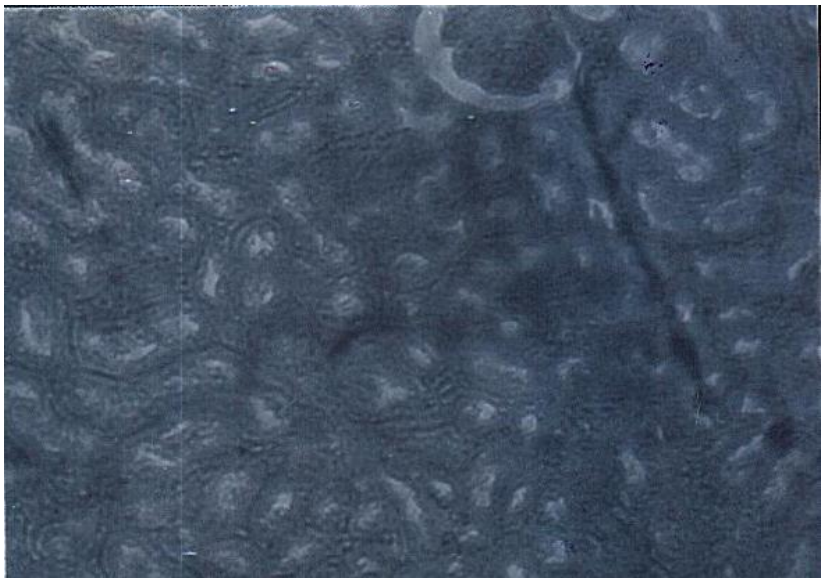

Plate 1: Kidney section of the rat in the control group displaying normal architecture stained by H&E technique. X100

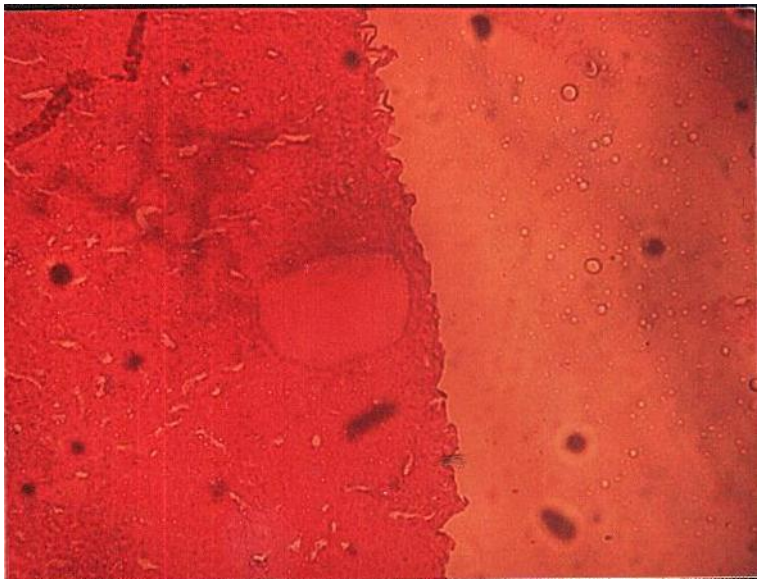

Plate 2: Renal necropsy section of the rat fed 1500mg/kg body weight of KI displaying features of hyalinization at the extreme right and the hyaline droplet in the tubules. X100.

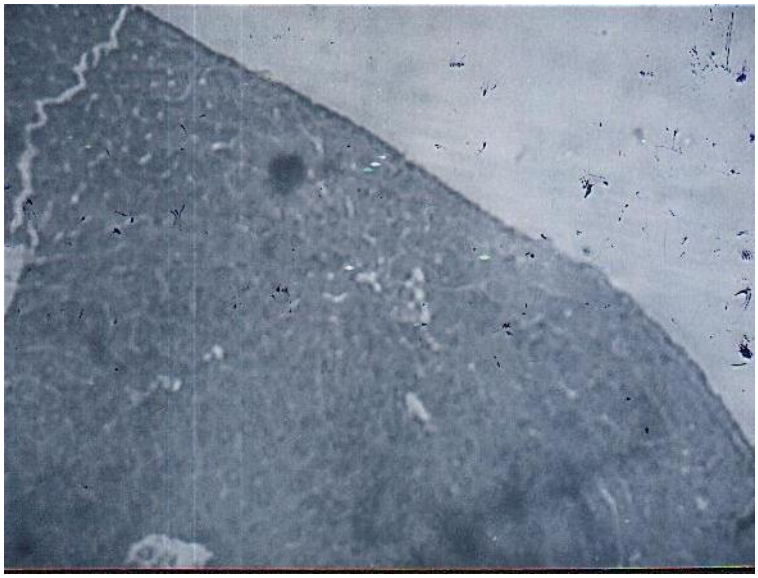

Plate 3: Liver section of the rat in the control group showing normal architecture. Stained by H&E. X100

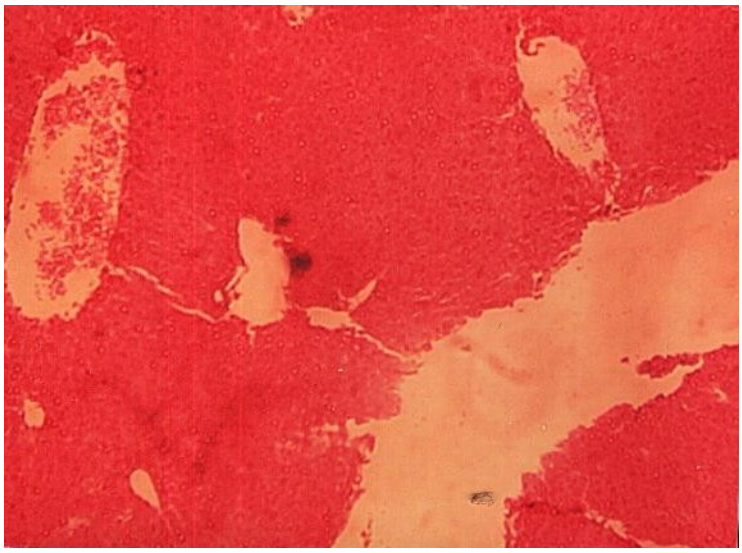

Plate 4: Liver section of the rat fed with 1,500mg/kg body weight of KI showing evidence of chronic venous congestion (stasis) as well as intraportal ulceration and portal hypertrophy. Stained by H&E method. X400

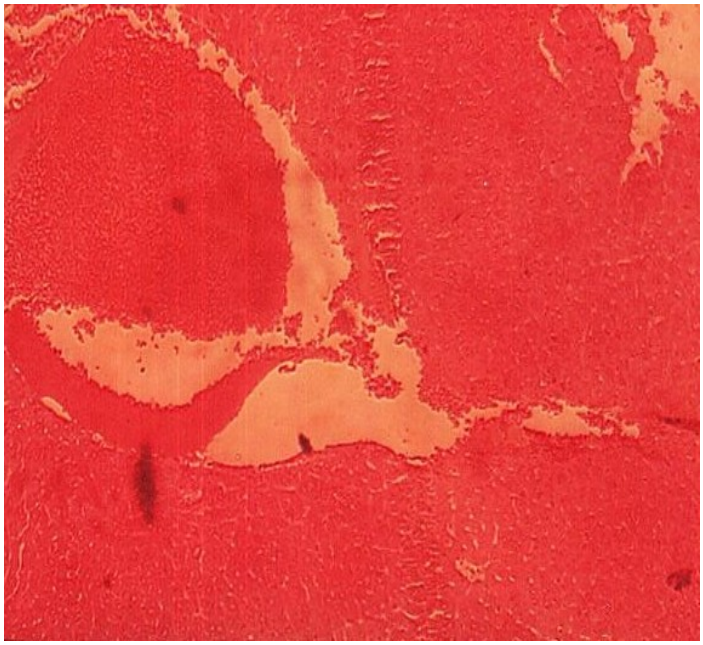

Plate 5: Liver section of the rat fed with 1,500mg/kg body weight of KI showing evidence of partial vascular thrombosis of the central vein.

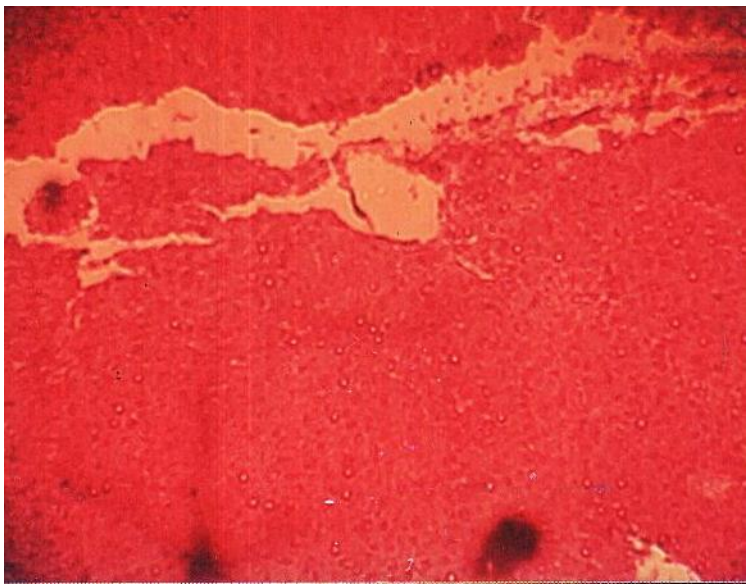

Plate 6: Liver section of the rat fed with 1500mg/kg body weight of KI displaying showing evidence of intraportal degeneration. Stained by H&E method. X100

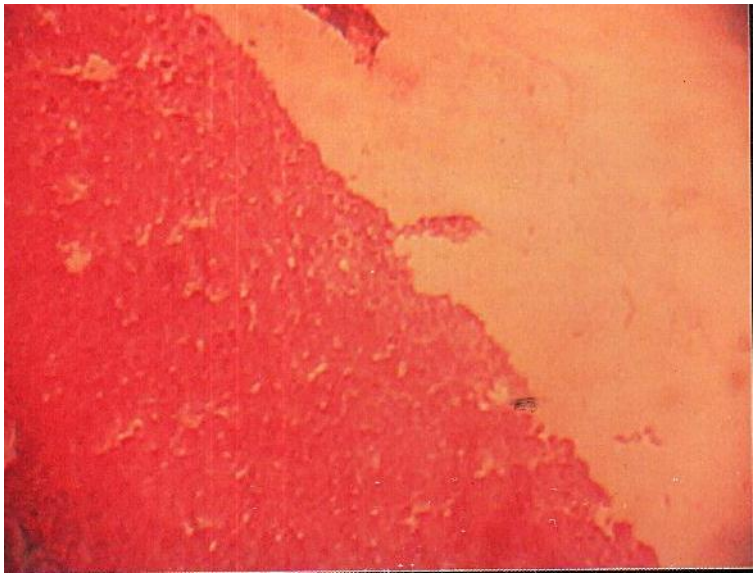

Plate 7: Liver section of rats fed with 1500mg/kg body weight of KI displaying features of nuclear eosinophilia. Some of the hepatocytes show perinuclear haloes and are also necrotic. Stained by H&E method. X100

Discussion

The results obtained in the present study show that potassium iodide salt in high concentrations is toxic to animal population. A few days after the commencement of the work there was evidence of potassium iodide induced loss of appetite, sluggishness, weight loss, altered body condition such as ruffled hairs, alopecia, lethargy, abdominal enlargement and in few of the cases death. These clinical manifestations were most profound among the group fed with 1500mg/kg body weight.

Histological analysis of the kidney showed tubular hyalinization indicative of potassium iodide toxicity. This is because excess iodide is excreted by kidney in urine (Nordic Working Group on Food Toxicity and Risk Evaluation [9]. From this study, the greatest adverse effect of potassium iodide was on the liver. Liver sections revealed chronic venous congestion (stasis), intraportal vascular necrosis, intraportal degeneration, and eosinophilia and perinuclear haloes. All these adverse effects are indicative of hepatotoxicity. Necrotic lesion in the liver of dogs fed on high doses of KI was reported by Webster et al [6] . These findings are in agreement with the work of Wolff [10], which revealed that at very high doses of oral iodate given to experimental animals hemolysis, nephrotoxicity, hepatic injury and corrosive effects in the gut occur. The high dose treated animals showed exceeding weight loss which may be due to drug induced loss of appetite [8].

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