Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Scholars Research Library

Annals of Biological Research, 2015, 6 (3):12-18
(http://scholarsresearchlibrary.com/archive.html)

Annals of Biological Research
Scholars Research Library
ISSN 0976-1233
CODEN (USA): ABRNBW

Degenerating effects of potash (Kaun-K_2co_3) on the kidney: Unabated continental challenge to human health in Nigeria

[1]Bankole J. K., [2]Ngokere A. A., [3]Ajibade O. M., [4]Igunbor C. M. and [5]Eloka C. C. V.

[1,4]Department of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State
[2]Department of Medical Laboratory Science, Namdi Azikiwe University, Awka, Anambra State
[3]Department of Earth Sciences, Olabisi Onabanjo University, Ago-Iwoye, Ogun State
[5]Department of Medical Laboratory Service, Cottage Hospital Auchi Polytechnic, Auchi, Edo State

---

ABSTRACT

In recent times different injurious and terminal diseases with unknown cause and devastating effects have infringe into human lives. One of these diseases is the renal failure in humans which may be caused by edible chemicals and other edible earth materials such as potash (Kaun-K_2CO_3). In order to unravel this increasing phenomenon in humans, the effects of potash on the kidney of Wister rats was investigated. Twenty (20) Wister rats were used for the study and grouped into four categories marked A, B, C and D with 5 rats in each group. The potash (hydrated sodium carbonate - dried lake salt.) used was obtained from the regular Ekpoma market in the central part of Edo state. The rats in groups B, C, and D were fed with 3g, 6g, and 9g concentrations of potash respectively mixed in growers mash for a period of three weeks in order to investigate the effects of this compound on the kidney. The results of this study showed that at varying concentration of potash, there were progressive tubular and vascular changes, cellular necrosis and glomerular degeneration and this imply that potash is cytotoxic to the kidney tissues. Therefore, excessive consumption of this earthy material (potash-Kaun) may lead to its accumulation that could cause severe and irreparable damage to the kidney and disrupt normal body functions which may eventually lead to loss of life.

Keywords: Potash (Kaun), Renal damage. Wister rats, Progressive alterations, Kidney

---

INTRODUCTION

It is palpable from recent scientific reports that there is an increased use of geological mineral substances in human and animal foods (Aribido *et al*., 2001)[1]. In Nigeria, the renewed government interest in solid mineral exploration and exploitation may possibly explain the reason for the use of naturally occurring inorganic salts for diverse purposes (Aribido *et al*., 2001)1. Based on this, the beneficial effects of these mineral additives in plant residue and animal nutrient utilization for animal productivity increase have been reported by Harrison *et al*., 1985; Mees and Merchen, 1985) [2-3]. While some natural products such as alum were also used in water processing for home and industrial needs.

The mineral salt of interest is Potash which is a dry lake salt of sodium bicarbonate with water of crystallisation ($Na_2CO_3NaHCO_3.2H_2O$). The deposit is usually covered by shallow water, less than two feet deep. (Davidson *et al*., 1974; Oyeleke and Morton, 1981)[4-5] This earth material is described as a mixture of salts with other different components including impurities which coexist ins mineral and salts such as thermonitrite, halite, thernadite, mirabilite, and gypsum (Mankanjuola and Beetlestone, 1975)[6]. It consist of some metals like Ca, Mg, Fe, Zn, S, Cl, Si, P, K and Al as well as other micronutrients (Ekanem and Harrison, 1997; Ako, 1984; Ikwegbu *et al.*, 1984; and ILCA, 1985)[7-10]. Potash have different variety of red-white sodium carbonate (20%) and whitish sodium sulphate (80%). Both varieties contains little or no potassium and users cannot distinguish between common greyish-white

crystal soda potash and the rare yellowish-white crystal potash (Ekanem, 1977)[11]. Pearl ash is organic potash sourced from burnt wood and plant ashes, while mineral potash is industrially produced form Solvay process which chemically the treatments of common salt solution with ammonia and carbon dioxide or from Engel-Precht process (Ibeme, 2010)[12]. In Nigeria, potash occurs in abundance and it is known by various names such as akanwu, kanwa, kaun and kawe. Most importantly, because of its popularity and usage for various domestic purposes in some part of the world, it is ranked next in importance to the common table salt in Nigeria (Turner, 1989)[13].

However, potash are used for many purposes in Nigeria. The effect of potash on cooking time indicates a reduced time of 10-15mins from a prolonged time of 40-65mins in cowpea. In cooking it aids the preservation of green colour of vegetables with a concentration of 0.1-0.5% potash. Buchanan and Purgh (1961)[14] reported the medicinal use of potash for all sorts of ailments. In powdery form, when mixed with tobacco it is used as snuff. In the Northern part of Nigeria, it is also administered in large doses by the 'Hausas' in the form of guinea corn and millet porridges called 'kunun Potash' which is administered to women immediately after delivery for the purpose of increasing the quality and quantity of breast milk (Davidson *et al*., 1974)[4]. It is also used as a tenderizer, flavouring agent, food preservative, prophylactic as well as improving protein digestibility of cowpea (Omueti *et al*., 1992 and Uzogara et al., 1988)[15,16]. Reports of its use as softener for food and vegetables such as cowpea, okro and ewedu have been attested to by Ankrah and Dolvo, (1978)[17]. Apart from being taken in food, it is also used as a component by some Nigeria medicinal concoctions and sometimes it is chewed raw (Sodipo *et al*., 1992)[18] with high potency in cough, tooth and stomach aches and constipation. Although potash is widely used in Cameroon, Ghana, Nigeria and several other West African countries, no documented study has been reported of its mineralogy.

In this regard, studies have indicated that the high level of potash in foods and drinking water could be detrimental to human health (Davidson *et al.*, 1974, Bello, 1988)[19,20]. These findings with other existing facts show that potash as a mixture of various salts with other earth impurities may be toxic which raises doubt on its ingestion safety by humans. Although the biochemical and physiological effects of potash have been investigated but data paucity still exists, concerning its dietary effects in humans (Oyeleke, 1988)[21]. This work is only to determine the effects of potash on the kidney histology.

MATERIALS AND METHODS

ANIMAL TREATMENT

Ethical approval was granted from Faculty of Basic Medical Sciences, College of Medicine, Ambrose Alli University, Ekpoma to carry out the research. Albino-Wister rats were procured from the animal house, college of medicine, Ambrose Alli University Ekpoma. A total of twenty (20) albino-Wister rats were used for this experiment which were randomly assigned three test groups of 5 rats in each group and 5 as control group. Before initiation of experiment, the rats were acclimatized for a period of 2 weeks**.** Standard environmental conditions such as temperature ($26+2^0$C), relative humidity (45-55%) and 12 hours dark/light cycle were maintained. All the animals were fed with growers mash obtained from Edo Feeds and Flour Mill Limited, Ewu, Edo State, Nigeria and water under strict hygienic conditions.

Twenty adult Wister rats with average weight of 185g were randomly assigned into four groups: A, B, C and D of five in each group. Group B, C and D served as test groups while group A served as the control. The rat in each group were further tagged 1, 2, 3, 4 and 5. The rats in group A were tagged A1, A2, A3, A4 and A5 as well as in group B, C, and D. The group A (control) animals were fed with normal growers mash while those in group B, C and D were fed with graded doses of potash (3g, 6g and 9g) mixed with 97g, 94g and 91g of growers mash respectively. The potash (hydrated sodium carbonate - dried lake salt.) used was obtained from the regular Ekpoma market in the central part of Edo state and the rats in groups B, C, and D were fed two (2) times daily for three (3) weeks with 3g, 6g, and 9g concentrations of this earth mineral/salt respectively mixed in growers mash.

SAMPLE COLLECTION AND TREATMENT FOR HISTOLOGICAL STUDIES

All the animals were euthanized at the end of the third week after the feeding treatments; they were anaesthetized using chloroform as sedative. Tissue samples were collected from the kidneys and preserved in 10% neutral buffered formalin and were later processed using standard histopathology techniques. Using 70% alcohol, 80% alcohol, 90% alcohol, 90% alcohol, 95% alcohol, Absolute, Xylene 1, Xylene II, Molten paraffin wax-1, Molten paraffin Wax II, for 2hrs each (As used in OAUTHC, Ile-Ife, Osun State Nigeria).

The tissues were removed from their plastic cassettes after the last timing, placed at the centre of the metallic tissue mould and then filled with molten paraffin wax. These were left to solidify and placed in the refrigerator at 5^oC for 15 minutes. The blocks were cooled in the refrigerator for the stated time, and removed from the metallic case; they are trimmed and cut serially at 3nm on a rotary microtome. The sections were floated in water bath at 55^oC, picked

up with a clean frosted end slides and placed on the hot plate for 40 minutes for adequate attachment hence, the sections were de-waxed, hydrated, air dried and stored in a slide box ready for staining process.

HAEMATOXYLIN AND EOSIN STAINING PROCEDURE

Sections for general tissue structure were stained by Haematoxylin and Eosin technique. The sections were dewaxed in three (3) changes of xylene for 5 minutes and hydrated through descending grades of alcohol (absolute, 95%, 80% and 70%). These were stained in Harris haematoxylin for 5 minutes and rinsed in a running water to remove excess stains. The sections were differentiated in 1% acid alcohol for 3 seconds and were blued in running water for duration of 10 minutes. They were counterstained with 1% eosin, for 1 minute and finally rinsed in water and dehydrated in ascending grades of alcohol (70%, 80, 95% and absolute). The sections were cleared in xylene, air-dried and mounted with dibuthylphthalate propylene xylene (DPX) and they were examined under a light microscope and photomicrographs were taken. (As used in Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife, Osun State Nigeria). All the necessary clinical quality control were ensured with the standard precautions in terms of proper timing, pH test of the solutions, accurate measurements of reagents and prevention of particulate contaminants.

RESULTS AND DISCUSSION

The results of the Haematoxylin and Eosin staining (H & E) reactions showed that potash consumption induced marked abnormalities in the kidney initiated with disruption of tubular and glomerular organisation. The potash resulted in progressive tubular, glomerular and interstitial alterations as seen in the cytoarchitectural distortion and reduction in the number of renal corpuscle in the treated groups (B, C and D) which was at variance with that of the control group A. The micrograph of the kidney section in the group A showed normal histological features. The section indicated a detailed cortical parenchyma and the renal corpuscles appeared as dense rounded structures with the glomeruli surrounded by a narrow Bowman's spaces (Fig. 1, Group A). This could be as result of the fact that experimental animals in this group were fed with normal feed (growers mash) and as such no observable effects were visibly seen.

However, the kidney sections B2, B3 and B5 in figure 2, group B animals fed with 3g of potash showed that the potash induced marked abnormalities in the kidney which include disruption of the tubular organization such as tubular perforations, deformation of brush borders as well as mesangial hypercellularity, mesangial proliferation, atrophy of basal cytoplasm, focal necrosis of tubular cells and pyknosis of nuclei. Pathological effects also observed were exudations from degenerated cells, vacuolations, inflammatory cells infiltration as well as slight glomerular degeneration. The potash administered appears to have caused considerable kidney damage.

Also, the kidney sections C1, C2, C4 and C5 in figure 3, group C experimental animals which were fed with 6g of potash revealed similar distortion as in group B although more intense. The more severe effects observed in these slides are suggestive that as the concentration of potash increased, the defects induced became more severe. The kidney demonstrated hyperplasia, tubular perforations, deformation of brush borders, atrophy of basal cytoplasm, focal necrosis of tubular cells and pyknosis of nuclei, exudations from degenerated cells, vacuolations, and inflammatory cells infiltration as well as necrosis and shrinkage of the glomeruli. From the present results, it may be inferred that higher dose of potash administration resulted in more degenerative and atrophic changes observed in the renal corpuscle and tubules which are symbolic signs of cell death. The necrosis observed could probably be due to the high concentration of the potash on the kidney that must have been toxic to the kidney tissues. It could be inferred therefore from the result of this study that the cell death was due to the nephrotoxic effects of the potash on the kidney.

Furthermore, the kidney sections D1, D2, D4, and D5 in figure 4, group D experimental animals revealed enlargement of tubular lumen, tubular perforations, tubular hyperplasia, mesangial hypercellularity, mesangial proliferations, deformation of brush borders, atrophy of basal cytoplasm, and focal necrosis of tubular cells/pyknosis of nuclei, exudations from degenerated cells, vacuolations, and inflammatory cells infiltration. Other defects observed were severe glomerular necrosis, glomerular shrinkage, cloudy swelling and vascular changes (damage) with haemorrhage. The results are in line with observed effects noticed in group C which indicated that the effects of potash on kidney is dosage dependent.

Moreover, the results obtained in this group (D) showed that in higher concentration of potash, there was reduction in renal cell numbers of proximal and distal collecting tubules which have resulted in tubular enlargement. The tubular cells may have undergone hypertrophy and some renal cells have lost their normal shape. The cuboidal epithelial cell lining the tubules showed complete vacuolization with degenerating cytoplasm and more nuclear and their disorderly scattering nature kidney sections noticed in all the rats. Changes like vacuolation of epithelial cells

of renal tubules and pronounced enlargement of the tubules were observed at higher concentration in section C4 and D1. Vacuolization on the other hand due to degeneration of the cytoplasm is quite obvious as well as perforations of the kidney. It may therefore be inferred from the present results that higher dose administration of potash resulted in severe glomerular degeneration observed in the renal corpuscle and atrophic changes of the renal tubules as remarkably shown in section D1, D4, D5 and D2 in figure 4, Group D. The results of this study revealed that the glomeruli were shrinken and some of them were vacuolated and there was also cloudy swelling in this group (D) fed with 9g of potash which were absent in other groups B and C slides from experimental animal tissues fed with potash at lower concentration. It was also observed that those fed with 9g of potash, the glomeruli undergoes complete necrosis and tubular enlargement as indicated in section D1 and D4. The inflammatory cells infiltration may have been as a result of immune inflammatory response to potash.

Finally, the process of cellular necrosis involves disruption of membranes, as well as structural and functional integrity (Eweka, 2007)[22]. Cellular necrosis is not induced by stimuli intrinsic to the cells as in programmed cell death, but by an abrupt environmental perturbation and departure from the normal physiological conditions (Belluardo et al., 1990)[23]. In cellular necrosis, the rate of advancement depends on the severity of the environmental insults. The greater the severity of the insults, the more rapid the progression of cellular injury which could possibly explains the increase in distortion and degeneration of the kidney as the concentration of potash increased in this study (Belluardo et al., 1990)[23]. There was no documented report found to backup these findings at the time of this research. Since the kidney is responsible for the regulation of mineral salts, high level of potash in foods and drinking water could be detrimental to human health as proofing in this study as well as the finding of Oyeleke (1988)[21] who reported that the increase in potash intake leads to decreased food and water intake and the presence of wrinkled skin with some hair losses.

PHOTOMICROGRAPHS (HISTOLOGICAL OBSERVATIONS)
NORMAL KIDNEY

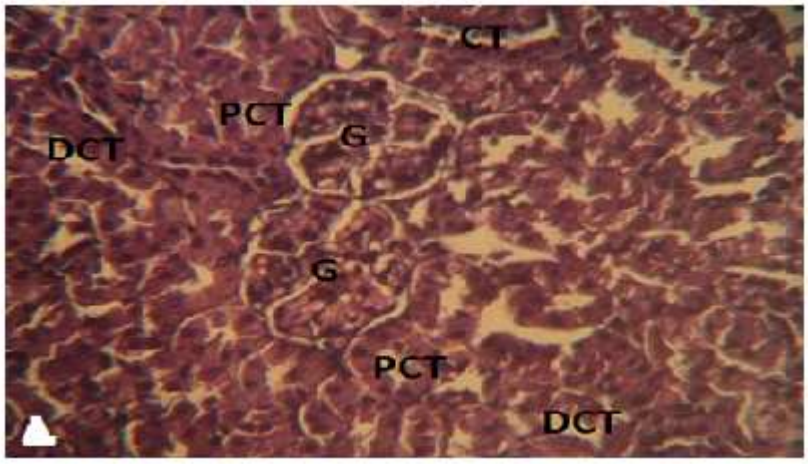

Figure 1: (H & E × 400) Control Kidney sections (A4) showing normal pictures. The Bowman's capsule, glomerulus (G), proximal convoluted tubules (PCT), distal convoluted tubules (DCT) and collecting tubule (CT) showed intact architectural integrity

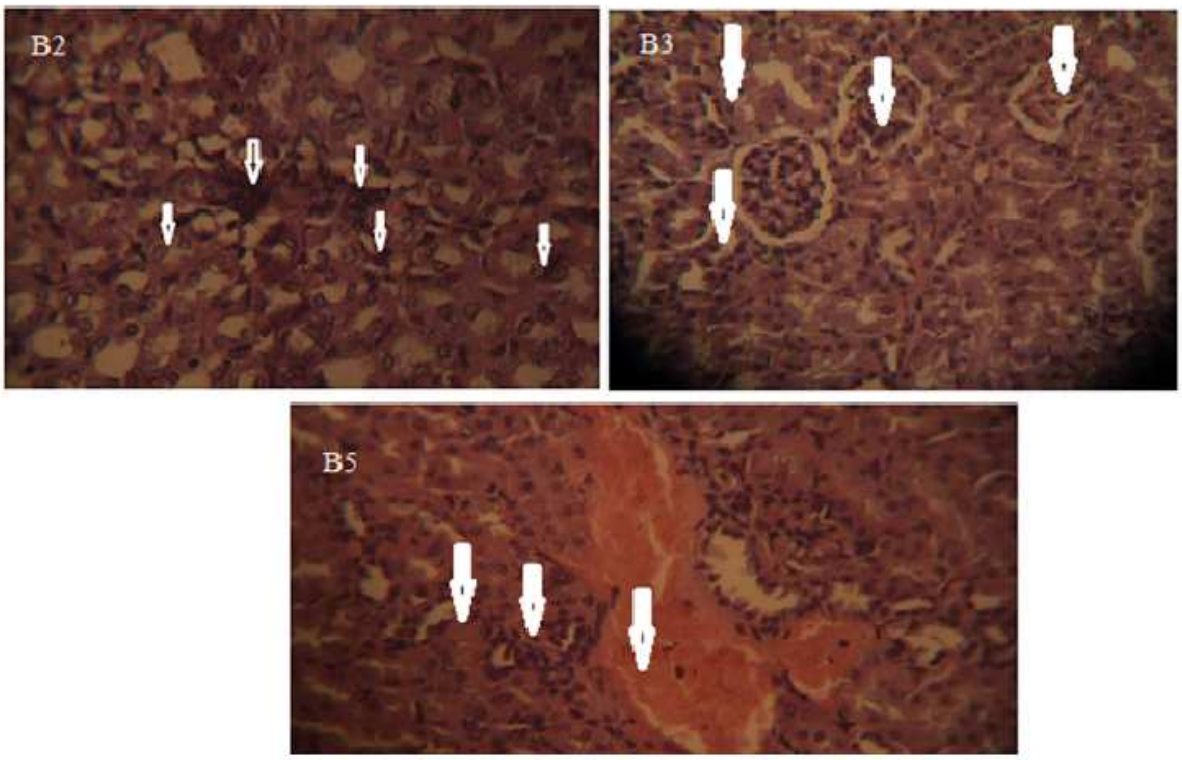

Figure 2: (H and E x400) Test (B2, B3 and B5) kidney sections shows vacuolations, infammatory cell infiltration, Pyknosis of th nuclei, atrophy of basal cytoplasm, mesangial proliferation, mesangial hypercellularity, focal necrosis of tubular cells and exudations from degenerated cells as indicated with allow

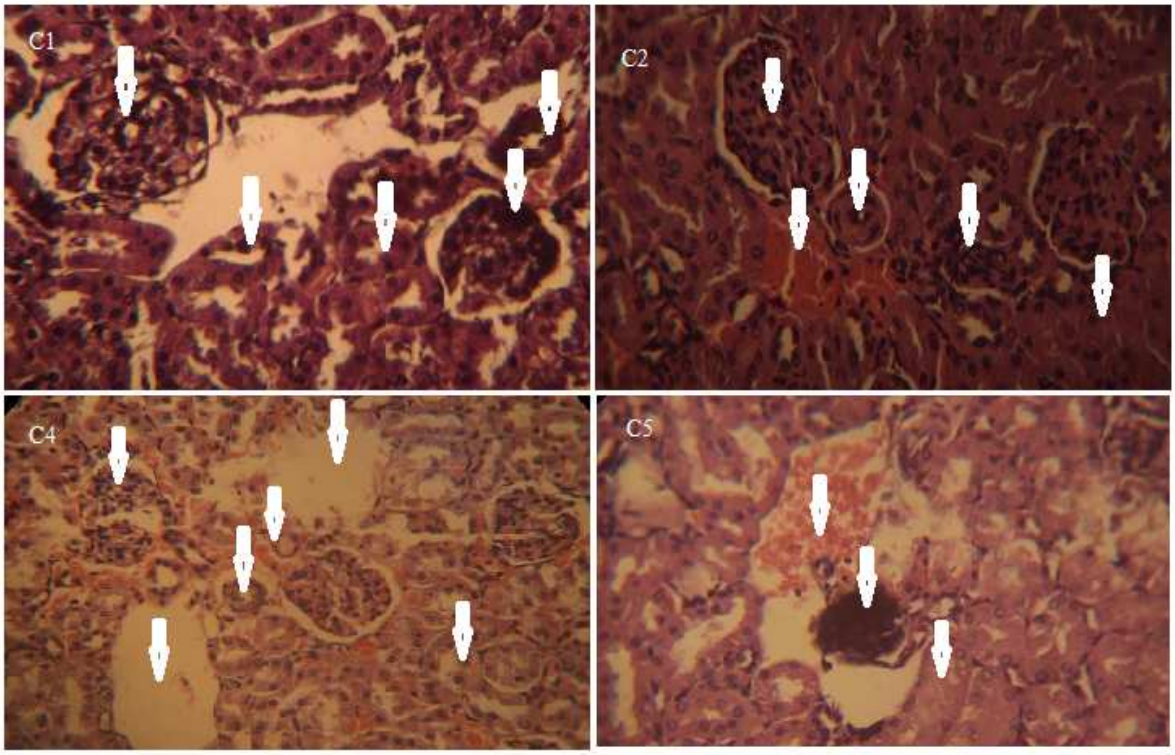

Figure 3: (H and Ex400) Test (C1, C2, C4 and C5) kidney sections shows deformation of brush border, mesangial hypercellularity, glomerular degeneration, mesangial proliferation, inflammatory cell infilteration, pyknosis of the nuclei, tubular necrosis, glomerular necrosis as well as vascular damage with haemorrhage and necrotic proximal tubules as indicated with allow

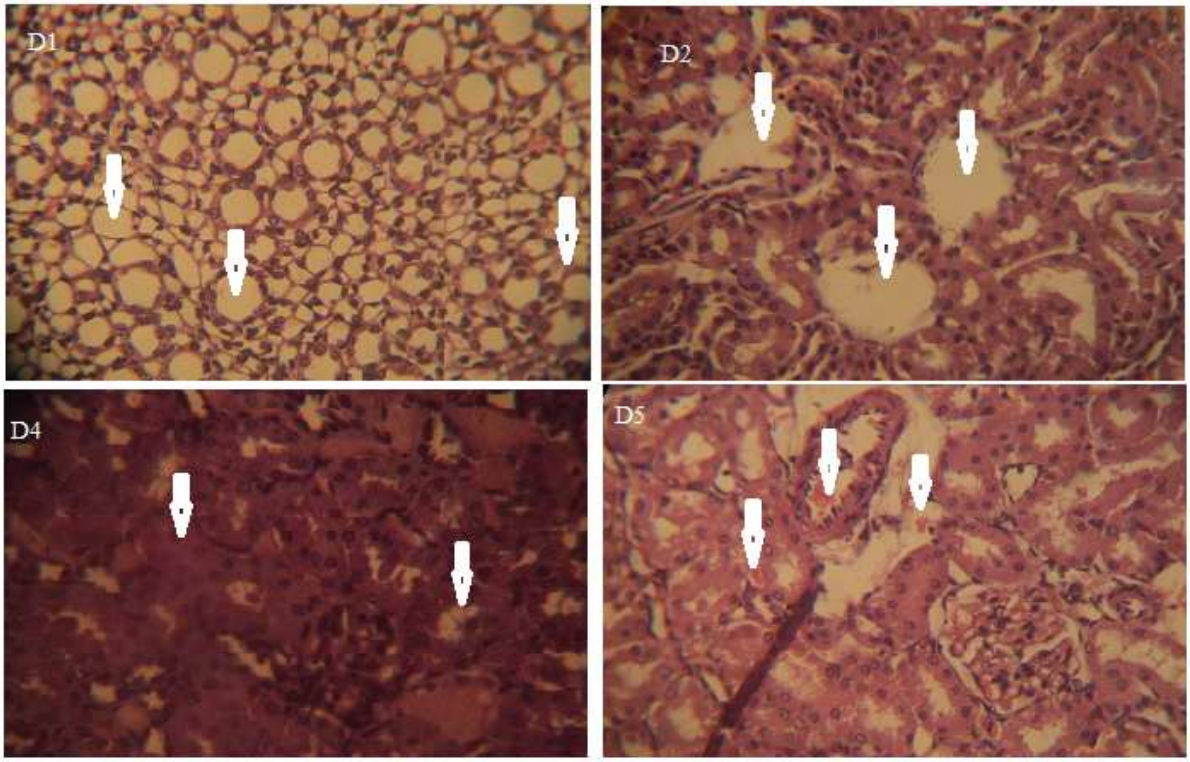

Figure 4: (H and E x400) Test (D1, D2, D4 and D5) kidney sections shows enlargement of tubular lumen, vacuolations, tubular hyperplasia, severe glomerular (degeneration) damage, cloudy swelling, tubular necrosis and vascular damage with haemorrhage as indicated with allow

Acknowledgement

We appreciate the College Of Medical Sciences, Ambrose Alli University, Ekpoma, Edo state, for permission granted to use their facility for this work. Also we thank Anthonio research centre, Ekpoma, for helping in the photomicrography and The Management and Staff of Health affair ventures, Ekpoma. Our gratitude also goes to Dr Nwaopara Tony, Anatomy department, Ambrose Alli University, Ekpoma. Dr Avwioro O.G, Delta State University ,Abraka. Proffessor G.R.A Okogun, Medical Laboratory Department, Ambrose Alli University, Ekpoma, Mr Charles Idehan, Histopathology Department Ambrose Alli University, Ekpoma. Dr Oladele A.A. of Obafemi Awolowo University Teaching Hospital Complex, Ile- Ife , Osun State for their contributions in various ways to this work.

REFERENCES

[1] S O Aribido; B K Ogunmodede; C A M Lakpini. *Nigerian Journal of Chemical Research* **2001**, 6, 3, 27-30.
[2] D G Harrison; D E Beever; D J Thamson; D E Osbourn. *Jour. Agric. Sci*. Cambridge **1985**, 85, 93.
[3] (3)D C Mees; N R Merchen, N R. Nutri. Report Internl **1985**,32, 1067.
[4] N M Davidson; L Trevitt; E H O Parry. *Bulletin WHO* **1974**, 51, 203.
[5] O A Oyeleke; I A Morton. *Nig. J. Nutri. Sci*ence **1981**,1, 2- 123.
[6] A A Makanjuola; J G Beetlestone. *J. Mining and Geology* **1975**,10 (1&2), 31-41.
[7] E J Ekanem; G F S Harrison. *Nigerian Journal of Chemical Research,***1997**, 2, 33 - 38.
[8] P A E Ako. *Journal of Chem. Res,***1984**, 6, 3, 27-88.
[9] O A Ikwuegbu; T A Gbodi; I Umo. *NURI*, **1984**, 103-120.
[10] ILCA. International Livestock Centre for Africa bulletin Addis Ababa, **1985**, 22.
[11] E J Ekanem. *J. Biochemical Analysis*,**1977**, 2, 1, 25 - 43.
[12] I U Ibeme. **2010**, *http://www.priscaquila.ucoz.com.*
[13] F M Turner. Chapman and Hall Ltd, **1989**, 1177.
[14] K M Buchanan,; J C Purgh, J.C. London, University of London Press, **1961**,44-62.
[15] O Omueti; I D Morton; W Emery. *Int. J. food Sci. and Nutrition*, **1992**, 43, 147-153.
[16] S G Uzogara; I D Morton; J W Daniel. *J. Food Chemical*, **1988**, 30, 2, 1-18.
[17] E K Ankrah; F E Dovio. *J. Sci Food Agri*culture,**1978**, 29, 950- 952.
[18] O A Sodipo; A E Akpan; O O Ajayi; M A Akanji. Abstracts from the 5th International Chemistry Conference in Africa, Gaborone, Botswana, **1992**, 36.

[19] N M Davidson; L Trevitt; E H O Parry. London, British Medical Association, **1974**, 37, 1251.
[20] M O Bello. *Journal of Chem. Res*, **1988**, 2, 21-33.
[21] O A Oyeleke. *Nig. J. Nutri. Sci*ence, **1988**, 4, 137-140.
[22] A O Eweka. *The Internet Journal of Health*, **2007**, 6, 2, 2.
[23] M Belluardo; G Mudo; M Bindoni. *Brain Res*, **1990**, 534, 225-233.