TOXOLOGIC EFFECT OF SODIUM CHLORIDE

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ABSTRACT

The aim of this study is to point out the dangers of accumulative intake of excessive amount of Nacl either by drinking water or ration which can lead to many pathological tissue changes of body organs.

Three groups of laboratory animals mostly male (3 mices for each group); distributed as high dose fed animals, Intermediate dose fed animals, and control group and this experiment continue for six months. Taken autopsy showed may gross changes characterized by regions of pale areas in liver with enlarged kidney, while microscopic findings appeared in several body organs; kidney, liver, heart, skeletal muscles, skin but were predominately in kidney which varied as being illustrated in figures with comments.

INTRODUCTION

Salt Toxicity or Poisoning occurs when an animal consumes a diet high in salt (sodium chloride), or when the animal consumes normal amounts of sodium but does not have adequate water.

Even though the body only contains about 0.2% sodium, it is essential for life and is highly regulated. About half of the sodium in the body is in the soft tissues of the body; the other half in bones (1). Sodium makes up about 93% of the basic mineral elements in the blood serum and is the chief cation regulating blood pH. The ability of muscles to contract is dependent on proper sodium concentrations. Sodium plays major roles in nerve impulse transmission and the rhythmic maintenance of heart action (1).

In the USA, it is more common in swine (the most sensitive species), cattle, and poultry. Sheep are relatively resistant.(2).

Mortality among songbirds is reported most commonly in cardueline finches (crossbills, grosbeaks, and siskins) (3). These birds appear to be attracted to salted roads, possibly in response to a dietary sodium deficiency (4). Accidental poisoning induced by excessive sodium intake in cases of infants is often due to inadvertent preparation of food. There

was a dramatic case of mass poisoning of 14 babies in a clinical setting in England, March 1962 (5, 6,7).

Moderate levels of dietary Na+ improve weight-gain in broilers, likely by increasing water consumption (8,9). Recent research has shown that ascites related to high dietary Na+ is caused by increased hydraulic pressure in the venous sys-tem following right ventricular failure from valvular insufficiency as a result of dilation and hypertrophy of the right ventricle in response to pulmonary hypertension (10). In chickens, right ventricular hypertrophy (11)

MATERIAL AND METHODS

Animals

Laboratory mices were collected from local markets then be subjected from October to April. Mices (male spp. Only) predominately young ; mean body weight less than 150 g) were divided into three groups (three animals for each group)distributed as high dose feeding group(20 % Nacl concentration) , intermediate dose feeding group(5 % Nacl concentration) ; and control group. Animals were housed in wire mesh cages under ambient light conditions, with adding Nacl to drinking water and ration. They were acclimatized to captivity for at least two weeks prior to testing. After six months animals were killed; and autopsy had been done to histopathological technique procedure and stained using hematoxylene and eosin stains. (12)

RESULTS

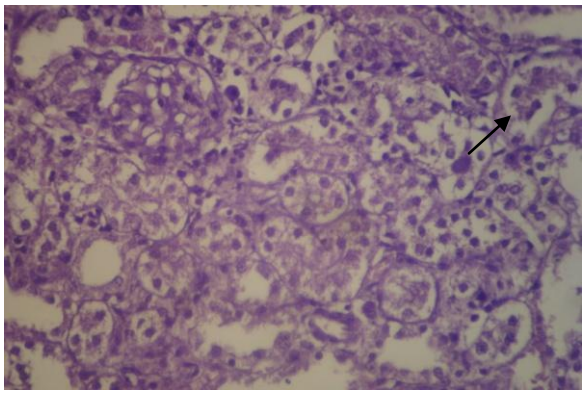
Experiment results were summarized by different body tissue pathological lesions. These lesions varied form organ to another, but were apparent and considerable in kidney but other organs like heart , liver. Skin, and muscles showed less different lesions.

The most significant pathological changes were in the kidney and those were varied from varying degrees of dilatation of cortical tubules (fig 2, 3, 9). On occasion those changes were associated with cortical tubules basophilia reflecting regenerating cortical tubules (fig 1, 5, 7) and cortical area fibrosis (fig. 4).

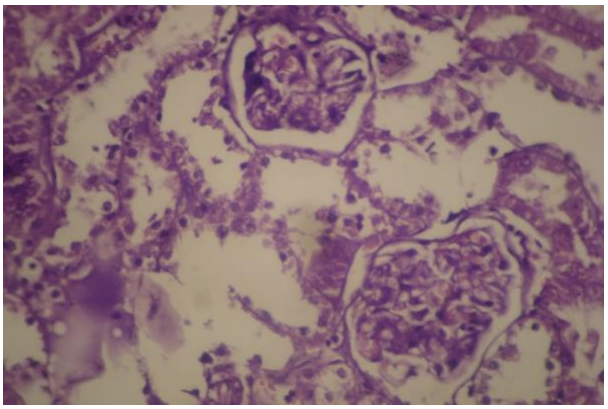
In addition further renal changes include enlarged vacuolated glomerular capillary endothelial cells (fig 6, 10, 11), cortical areas of fibrosis, cortical tubular necrosis and inflammatory cells (fig 6, 8) and atrophic glumeruli (fig 7).

Furthermore, other toxologic pathologic changes in other visceral organs and tissues were centrilobular vaculation of hepatocytes (fig 13) and septal fibrosis (fig. 16) while in heart it was vaculation of myocardial muscle cells(fig 15), atrophic skeletal muscle cells with intercellular edema and epidermal vaculation due to glycogenic infiltration and reduction of sweat glands of skin (fig. 14).

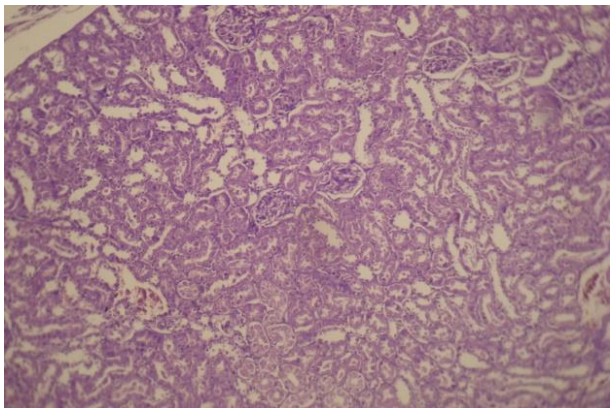
A) High dose lesions



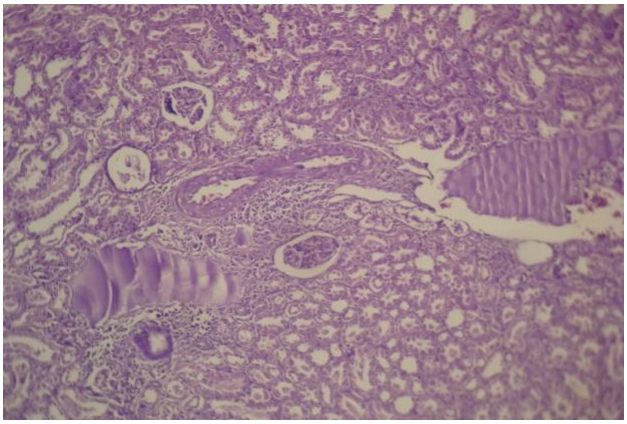
(Fig.1.) Kidney; an area of cortical tubular basophilia (indicating regeneration 0f cortical tubules)(H&E, x10)



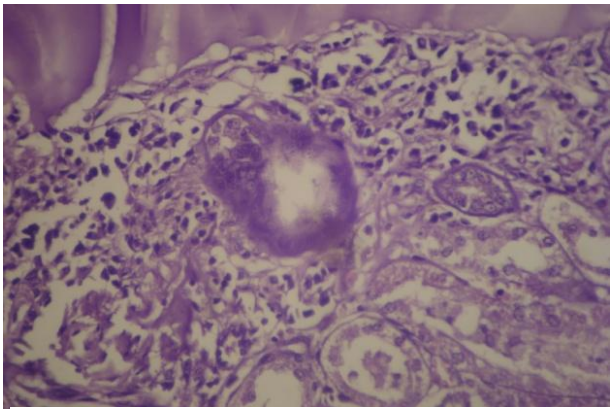
(Fig.2.) Kidney; marked dilatation of cortical tubules (H&E, x80)



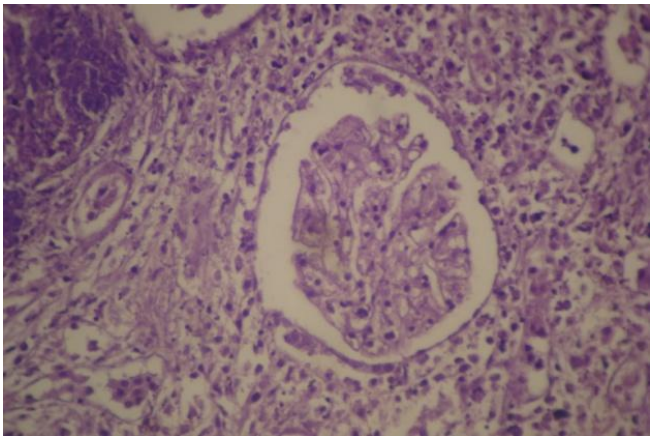
(Fig.3) Kidney; areas of moderate dilatation of cortical tubules (H&E, x10)



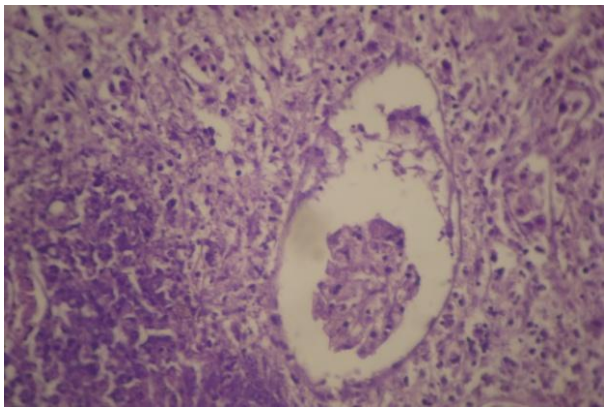
(Fig.4) Kidney; Fibrosis of cortical area with mixed inflammatory cells infiltration. (H&E, x10)



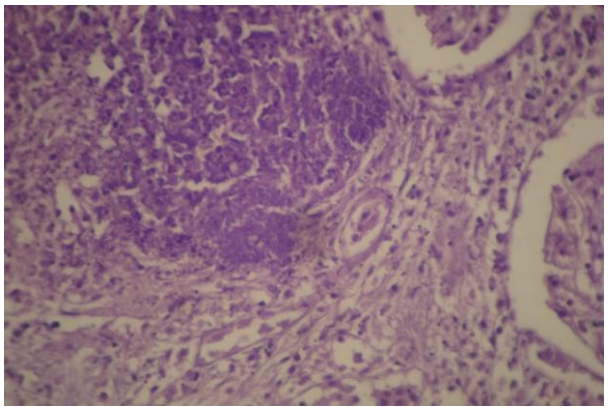
(Fig.5) Kidney; Area of mononuclear cells and cortical tubular basophilia. (H&E, 40x)



(Fig.6) kidney; A cortical area of fibrosis with monocellular cells; note swollen vacuolated glumerulous (H&E, 80x)

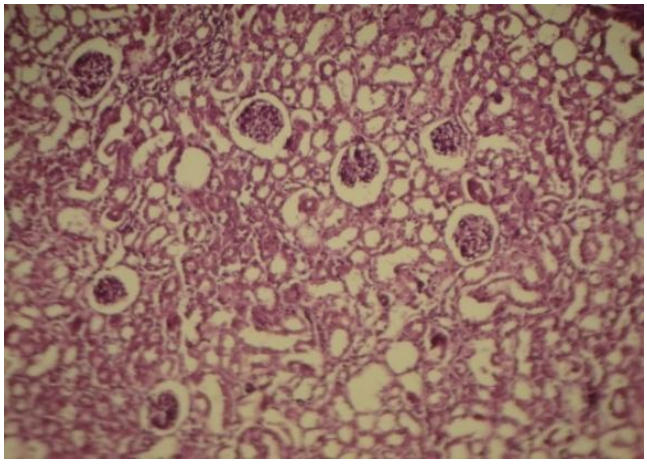


(Fig.8) kidney; High magnification of cortical area of fibrosis with focal necrosis (H&E, 160x)

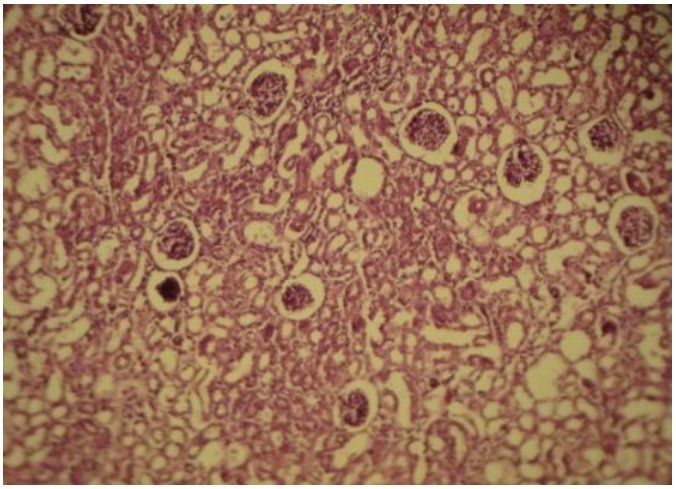


(Fig.7) Kidney; Atrophic glomeruli, an area of cortical tubular basophilia (indicating regeneration of cortical tubules) (H&E, 40x)

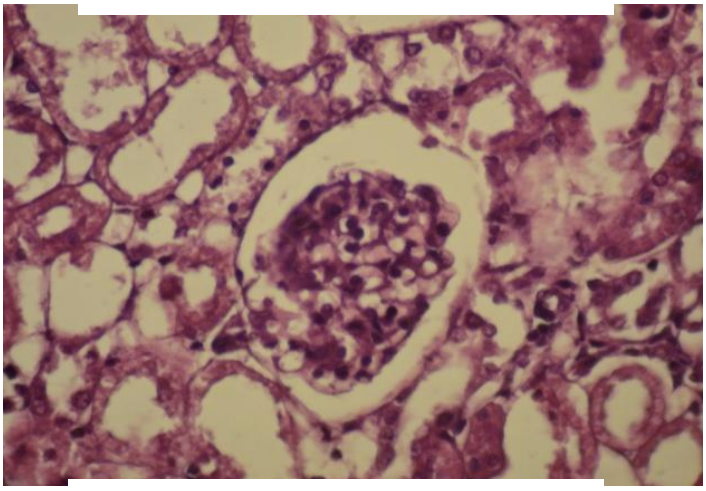
B) Intermediate dose lesions



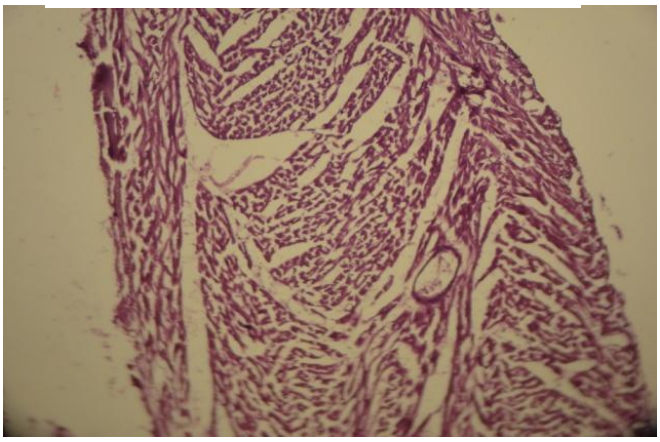
(Fig.9) Kidney; vaculation of glomeruli possibly of endothelial cells and dilated cortical tubules (H&E, 10x)



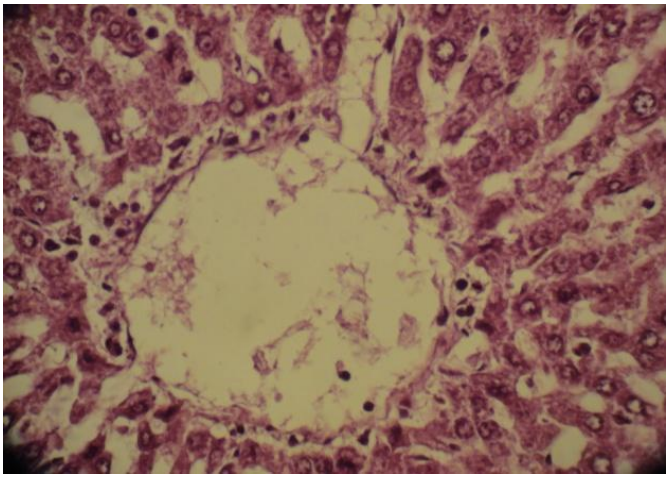
(Fig.10) Kidney; vaculation of glomeruli capillary endothelial cells (H&E, 10x)



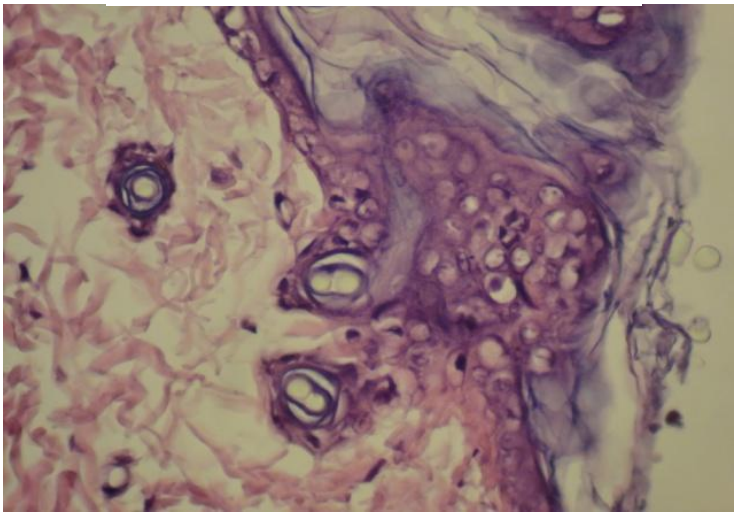
(Fig.11)) Kidney; vaculation of glomeruli capillary endothelial cells (H&E, 160x)



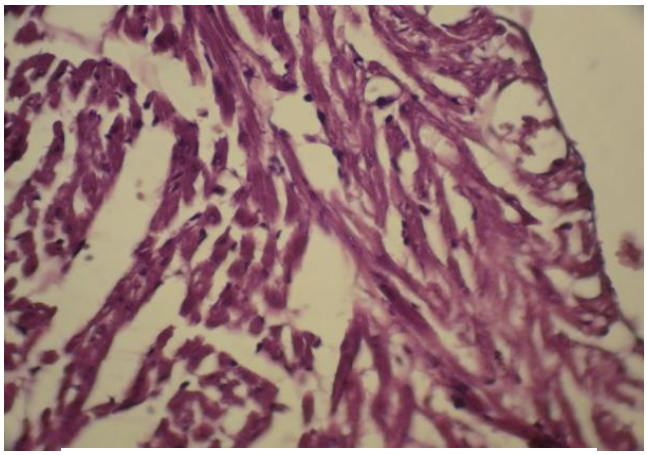
(Fig.12) skeletal muscles; atrophic skeletal muscle cells with intercellular edema. (H&E, 10x)



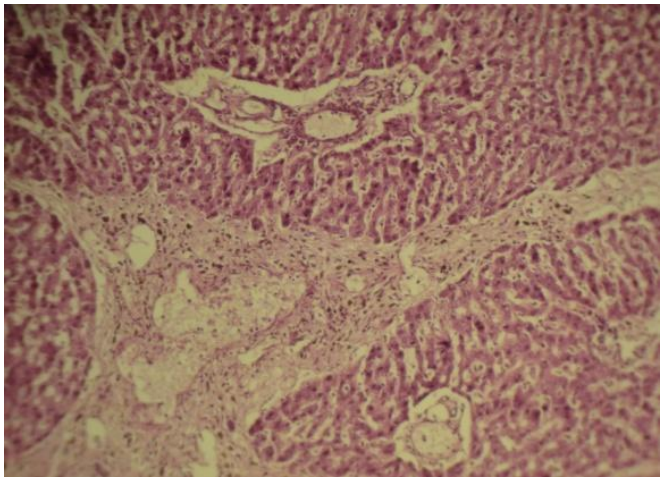
(Fig.13) Liver; centrolubular vaculation of hepatocytes (H&E, 160x)



(Fig.14) Skin; vaculation of epidermis probably glycogenic infiltration and reduction of sabecous glands (H&E, 80x)



(Fig.15) Heart ; note vaculation of myocardial muscle cells (H&E, 40x)



(Fig.16) Liver; septal fibrosis (H&E, 10x)

DISCUSSION

Present study showed different microscopic findings varied to be in many body organs like kidney, heart, liver, muscles and skin. These findings either familiar and match with other research that showed mild multifocal myocardial degeneration with mineralization and infiltration of a few mononuclear cells randomly scattered throughout the heart in birds (13).

On other hand; several research regarded that Nacl toxication inconsequential rather depend on what showed by (10)who mention that apart from some degenerative changes in the heart muscle and the kidneys, which are normal at a certain age, other pathological conditions in the internal organs have not been found which unlike to our marked changes happened in many organs especially extensive changes that showed in kidney in high dose group.

Other papers mentioned that sodium chloride poisoning showed prominent changes in kidney as in (14) the kidneys showed that nephrosis and glomerular changes were very prominent. These included glomerular hypertrophy, formation of epithelial crescents, fibrous adhesions, lobularity, shrinkage and collapse of the glomerular while salt toxicosis in commercial turkeys showed bilaterally symmetrical areas of necrosis within the cerebral hemispheres accompanied by vascular congestion and edema, as well as hyalinization of the glomerular capillary walls of the kidney and eosinophilic granular casts in the renal tubules (15).

Microscopic lesions in the more severely affected birds included liquefaction of ocular lens cortex with lens fiber swelling and multifocal to diffuse ulcerative conjunctivitis with severe granulocytic inflammation, edema, and granulocytic vasculitis resulting in

thrombosis as mentioned by (16) which explain that the kidneys were most sensitive organ to poisoning which may consequence to dysfunction of vital organ of urinary system and result in severe body damage while other body organs still healthy.

السمية المرضية لكلوريد الصوديوم

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الخلاصة

الهدف من هذه الدراسة هو أن نشير إلى مخاطر التناول التراكمي لكميات زائدة من كلوريد الصوديوم إما عن طريق مياه الشرب أو الغذاء والذي يمكن أن يؤدي إلى كثير من التغيرات النسيجية المرضية في أعضاء الجسم.
شملت التجربة استخدام ثلاث مجموعات من الحيوانات المختبرية ومعظمهم من الذكور (3 فئران في كل مجموعة) ، موزعة على النحو التالي : حيوانات تتغذى على جرعة عالية من تركيز املاح الصوديوم (20%) والمجموعه التي تتغذى على جرعة متوسطة التركيز من املاح الصوديوم (5%) ، ومجموعة حيوانات السيطرة التي تغذت على عليقة ومياه شرب طبيعية بدون اي كميات اضافية لاملاح الصوديوم، واستمرت هذه التجربة لمدة ستة أشهر.
أظهر تشريح الحيوانات المختبرية تغيرات مرضية عيانية التي تتميز بزيادة حجم الكلية وشحوب عام بالاعضاء المأخوذة ، في حين ظهرت النتائج المجهرية في عدد من أعضاء الجسم متضمنة الكلية و الكبد والقلب والعضلات والجلد ولكنها كانت في الغالب في الكلى والتي تنوعت كما سيتم تفصيله في الاشكال النسجية وتعليقاتها.

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