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BIOACCUMULATION OF SOME HEAVY METALS AND HISTOPATHOLOGICAL, ULTRASTRUCTURE ALTERNATION IN KIDNEY, BRAIN AND LUNG OF MIGRATORY BIRDS AROUND MANZALA LAKE

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ABSTRACT

Migratory birds collected from September to March (2011-2012) from colonies on Lake of Manzala, in a variety of bird species, including mallards *(Anas Platyrhynchos),* pintails *(Anas Acuta)*, teal *(Anas Crecca)* and coor *(Atra Atra)*. Immediately, heavy metals were measured in kidneys, liver and brain. Results showed that heavy metals accumulations in birds tissue area under investigation were detected in following descending order: pb > Cd > Cu. Carcasses were in good post mortum condition, the birds was moderately emaciated. The spleen was mildly enlarged. The intestine got darker, swollen, the tissues of liver and kidney were damaged and fragile. The gross lesions mainly confined to the kidney which was dark or with haemorrhages and showed evidence of sever degeneration. The kidneys showed sever degenerative changes which was more prominent in the cortex than the medulla. The kidneys showed marked glomerular, tubular and interstitial pathological alternation. Many glomeruli were shrunked with invaginated diffuse hyalinized thickening of capillary endothelium. The tubules were dilated with accumulation of eosinophilic homogenous material in tubular lumina. Necrobiotic changes with nuclear pyknosis Kayorrhexis, karyolysis, cytolysis, interstial odema and fibrosis were observed in the kidney. Focal cellular necrosis was observed in the renal proximal tubule. There was focal haemorrhagic area in between the renal tubules, dilatation and congestion of blood capillaries and PAS positive deposits focally spread in the mesangial matrix. The tubule showed hyaline droplet degeneration and increased protein content by bromophenol blue stain. Semithin section of kidney stained by toulidine blue showed heavy metals. In the form of great number of dark granules in the apical sections of the tubular epithelial cell. Ultrastructural findings of glomeruli demonstrated proliferation of mesangial cells. Mitochondria in the cells showed some degree of swelling. Nuclear inclusion bodies and large residual bodies were present. The rough endoplasmic reticulum lost their characteristic arrangement. Lysosomes were numerous in some cells. Lysosomes in some cells contained dense bodies of varying sizes and large residual bodies were present. Marked clumping of chromatin with the aggregation of interchromatin material at the center of the nucleus with intranuclear inclusions bodies. Pericellular oedema, glioses or sattelosis in brain gray matter and demylenation or spongiosis in the white matter. Chromatolysis of the nucleus and cytoplasmic vaculation in the neuron of the cerebral cortex. Pulmonary congestion and the lung alveoli were markedly filled with erythrocytes. The intealveolar blood cappilaries. The lungs exhibited focal apoptosis at the apical portion of the cells and thickening of the interstitial connective tissue.

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Key words: *Migatory birds, Heavt metals, Light & TEM*

INTRODUCTION

Toxic heavy metals in water, air and soil are global problems representing a growing threat to humanity. Heavy metals are widely distributed in environment and some of them can cause physiological, biochemical and histological disorders. Humans are exposed to these metals from numerous sources including contaminated air, water, soil and food. Therefore, the evaluation of toxic potentials of metals is important for the risk assessment of human beings ordinarily exposed to these substances. The physiological influence of metals on organisms, humans and animals is conditioned by the nature of metal, by the type of compounds and by their amount. Moreover, different scientific studies indicated that the degree of toxic manifestation of different metals depends on dose, duration, route of administration and other physiological biochemical and histological disorders (Gochfeild and Burger 1987; Danielyan, 2010).

Seabirds possess several attributes that make them potentially useful as environmental indicators because they essentially represent the top of the food chain, (Bost and Le Maho, 1993). Marine birds are known to be an efficient 'tool' for investigating the marine environment (Cherel and Weimerskirch, 1995, Barrett and Krasnov, 1996; Furness and Camphuysen, 1997). They are widely used as bioindicators of contaminants such as metals (Monteiro and Furness 1995; Gray, 2002). Seabirds gather expressive levels of trace elements due to their position in marine food webs and their long life duration (Seebaugh *et al.,* 2005). Seabirds are good sentinel species because they are observable, sensitive to toxicants and live in different trophic positions (Ferreira and Horta, 2010).

Long term monitoring of heavy metals such as cadmium, lead, cupper and more specifically mercury are potentially harmful to most organisms even in very low concentrations and have been reported as hazardous environmental Pollutants (Leonzi *et al.,* 1986). Lead exposure can lead to chronic nephropathy (flower *et al.,* 1980 and Chang *et al.,* 1980). Cadmium and lead can lead to a verity of problems. These problems include smaller clutch sizes, reduced fertility, hatching failure, nestling mortality and behavioral abnormalities of chicks, reduced body mass and delayed fledge time (Burger and Gochfeld, 1991).

Many experimental studies showed that several heavy metals caused renal failure associated with severe histopatholgical and physiological alterations (Soudani *et al.,* 2010). Lead is associated with a wide range of adverse health effects in birds, and excessive exposures to lead are frequently lethal to waterfowl (Beyer *et al.,* 1998b). Cadmium is a highly toxic metal with a long biological half-life that targets lung, liver, kidney and bone (goering *et al.,* 1995). and lead to apoptotic cell damage in proximal tubules (Hamada *et al.,* 1994). Excessive exposure to cadmium may cause anemia, poor weight gain, and increased mortality rates in birds (Jacobs *et al.,* 1969). According to Furness and Monaghan (1987) Cd binding protein generally renders the metal harmless. Cd can cause damage to the kidney.
Histopathological have been increasingly recognized as valuable tools for the detecting adverse chronic effects of contaminant exposure and uptake on aquatic organisms (Myers *et al.,* 1998; Pietrapiana *et al.,* 2002).

The available literature data on histopathological effect of heavy metals on migratory birds is few. Therefore, the objective of this study was to determine the levels of heavy metals in kidney of migratory birds taken from around Manzala lake, studying the histopathological effect of heavy metals in different organs and ultrastructure alternation of the kidney.

MATERIALS and METHODS

- Collection of birds:
Two hundred specimens from around lake of Manzala, from September to March (2011-2012) in a variety of wild bird species were collected, the species included mallards *(Anas Platyrhynchos*), pintails *(Anas Acuta)* and teal (*Anas Crecca)* and coore *(Atra Atra)*. Immediately after collection, the birds were necropsied. Body condition and any macroscopic lesions were recorded.

- Biochemical Analysis:
Preparation of the samples for analysis:
The collected sample were kept under freezing at18°C until analyzed. Samples from kidney, liver and brain were weighted from each dried in a hot air oven (100-105°C) till the wet tissues reached to a constant weight, according to (AOAC,2006). Moisture content of each sample was calculated. One gram was taken from the dried grinded sample into a flasks. 5ml of nitric acid was added. The digestion flasks were kept over night and then putted on the hotplate at 80°C until all the tissues were dissolved and the brown fumes of NO3escaped. The contend was filtered and diluted till 25 ml with distilled water. A blank was carried out in the same way.

***Preparation of the standard solutions*:**
All metals were determined against aqueous standards. Standards prepared at dilution 0,05 and 1ppm. The target elements analyzed were Cd, Pb ,and Cu. Stock standard solution (Merck, Germany) of each element were used to prepare to obtain calibration curves. The metal analyses of samples were carried to the central laboratory of the faculty of Veterinary Medicine Assuit University using a ZEEnit 700 P Atomic Absorption spectrophotometer. The content of heavy metals are expressed by mg/kg dry weight of the tissue.

- Histopathological examination:
Tissue samples were collected after postmortem examination from kidneys, lung and brain. These samples were fixed in formalin 10% for at least 24-72hrs, then washed with water, dehydrated, cleared, and embedded in paraffin wax. The paraffin embedded tissues were blocked and microtomized. Sections of 5-7 microns were stained with Heamatoxylin and Eosin stain for histological exam, Periodic acid Schiff (PAS) stain for demonstration of glycogen content and Mercuric-Bromophenol blue stain for the total protein determination according to Bankrofts *et al.* (1996).

-Transmission electron microscopic study:
kidney tissues greater than 2 cm long were minced into smaller pieces of approximately 1 x 1 mm and were fixed in 4 percentcold buffered glutaraldehyde. Toluidine blue stained semi-thin sections were examined under light microscope. Semethin sections contrasted with uranyle acetate and lead citrate were examined and photographed with JEOL transmission electron microscope (J.E.M.- 100 Cx II) operating at 80 Kv, Assuit University Electromicroscop unit.

RESULTS

Concentrations of heavy metals in Migratory birds tissues showed that, the concentrations of Pb were in the following order: kidney >liver >brain. The Concentrations of Cd were in the following order: kidney >liver >brain. The Concentrations of Cu were in the following order :liver> kidney >brain (Table 1).

Table 1: Concentration of heavy metals (mg per kg dry weight) in different tissue of Migratory birds from lake of Manzala.

Heavy metals	Kidneys	Liver	Brain
Lead	6.416	3.482	3.274
Cadmium	1.982	0.735	0.208
C upper	0.023	0.028	0.009

The post-mortum examination:
The birds were moderately emaciated, characterized by loss of elasticity of the skin and difficulty in reflecting the skin from the underlying musculature. The pectoral muscles were mildly atrophied. There was minimal subcutaneous and no intracoelomic adipose tissue. The spleen was mildly enlarged. The intestine was darker, swollen. The liver were damaged and fragile. Kidney abnormalities included variation in size and decreasing weight with haemorrhages and severe degeneration.

Histopathological examination:
Kidney:
The kidneys showed marked glomerular, tubular and interstitial pathological alternation. Glomerular alternation revealed variation in the degree of severity. Some cases showed hypercellularity due to proliferation, accumulation of mesangial cells with thickening of the basement membrane (Fig. 1 a, b and c). Priglomerular focal area of amyloid deposit (Fig.1 d and e). Some sections figure showed expansion of Bowman's space and contraction of the glomerulus. Periglomerular fibrosis could be seen (Fig.1 g, h and i). Diffused hyaline and thickening of capillary endothelium were observed. In some glomerular tufts haemorrhagic lesions were seen. Hyperaemia of medullary and cortical vessels with mononuclear cell infiltrates were evident in some birds. Atrophy and fibrosis of the glomeruli and edema in Bowman's capsules could also be observed. Densely packed granules were observed (fig .1 j, k and i).

The most severe changes in the proximal convoluted tubules consisted of cytoplasmic vacuolation of tubular epithelial cells. The tubules showed dilatation with accumulation of eosinophilic homogenous material in tubular lumina (Fig.2 a, b and c). Focal cellular necrosis were observed as nuclear pyknosis Kayorrhexis, karyolysis and cytolysis (Fig.2 d and e). Infiltration of mononuclear cells was observed. Single epithelial cells were desquamated to their lumen with loss of brush border (Fig.2 f). Swelling in the lining epithelium of the renal tubules with narrow lumen and the presence of lymphocytes and monocytes were observed (Fig.2 f). The distal segment was lined with large, relatively clear columnar epithelial cells with central nuclei and the brush border was reduced or not present (Fig.2 g, h and i).

In some cases there was an increase in the incidence of marked severe degenerative changes in the lining epithelial cells of the renal tubules at the cortical portion and distortion the renal architecture (Fig.2 j) with fibrous peritubular necrotic tubules. Mononuclear cell infiltrates were observed in some places of the medullary part of the kidney, blurring the tubular structure (Fig 2. k). Intranuclear inclusion bodies were present in cells of the tubules (Fig 2. i). Several acid-fast appoptic bodies in cells of the tubules and eosinophilic homogenous material in tubular lumina were observed. Few renal tubules showed desquamated epithelial cells. Necrotic cells with pyknotic nuclei sloughed into the tubular lumen with a cellular or coarsely granular appearance. Focal haemorrhagic areas in between the renal tubules were demonstrated (Fig.2 i).

The kidneys showed interstial odema, dilatation and congestion of blood capillaries (Fig.3 a, b, c and d). Interstitial fibrosis and hyaline droplet degeneration with protein casts were found in the proximal tubule (Fig. 3. e, f. g and h).

On examination of some cases showed multiple granulomas. The grouloma consisted mainly of mononuclear lymphocyte, plasma cells, macrophage. Some of these undergone hyalinosis and appeared as highly acidophilic irregular structure (Fig.3 I and j). The granulomas were surrounded by connective tissue formed fibroblasts and fibrocytes. Foci of lymphocytes and pigment-laden macrophages were intermixed with strands of fibroblasts (Fig.3 k).

PAS positive deposits in the mesangium and segmental thickening of the glomerular basement membrane were observed (Fig.4. a). Varying degrees of arteriosclerotic changes were demonstrated in the efferent and afferent arterioles (Fig. 4. b). Strong PAS-reaction of the thickened basement membranes surrounding both the proximal and distal convoluted tubules which lost the brush borders were seen. Deposits of hyaline materials were seen in collecting tubules and interstitial tissues. The glomuli and

basement membranes showed a strong stainability of total protein contents in the renal glomeruli cells (Fig. 5). Strong stainability of total protein contents in the atrophied renal epithelial cells and granular casts and cell debris in the lumens of renal tubules. The inclusion bodies demonstrated inside the lumen of proximal convoluted tubule and noticed of internuclear inclusion inside the epithelial cells and granular casts and cell debris in the lumens of renal tubules (Fig. 5).

Transmission electron microscopic study:
Examination by light microscopy of the semithin transsection of the kidney stained by toulidine blue showed that the heavy metals caused greater degenerative changes in the kidney. The glomerul were contain protinacious materials in the space of Bowman's capsule. Epithelial cells of kidney tubules demonstrated vacuolar degenerative changes and dark granules were found in the apical sections of the tubular epithelial cells (Fig. 6. a, b and c). The translucent membranes of damaged cells can also be observed in the tubular lumen. The number of apoptotic bodies was increased (Fig. 6. d, e and f). Several miotitic division demonstrated in the tubular cells (Fig. 6. g and h). The renal tubules showed large areas of hemorrhage (Fig .6. i).

The mesangial cells showed extensive proliferation of the smooth endoplasmic reticulum. The rough endoplasmic reticulum underwent a progressive loss of structural integrity. The mesangial cells had numerous spinous cytoplasmic processes, numerous dense bodies and lipid inclusions. The mitochondria in all tubular lining cells showed some degree of swelling. The lysosomes increased in number within epithelial cells. Inclusion bodies were composed of densely packed granules smaller than those comprising the nucleolus or those found in the surrounding nucleoplasm (Fig.7. a and b). Scattered among recognizable mitochondria were ovoid bodies composed of dense osmophilic material and Large residual bodies. The endothelial cells of the glomerular capillaries were swollen, occasional pyknotic nuclei and lost their fenestration. The podocyte nuclei were with irregular outline. The foot processes were disorganized and in several lost leaving fragment at parts. The basement membrane become thickened from place to place with homogenous appearance (Fig.7. a and b).

The most remarkable ultrastructural findings were in proximal tubular lining cells were the pathological changes and mitochondrial proliferation. The mitochondria in all tubular lining cells showed degree of swelling (Fig. 8. a and b). It was also noticed that mitochondira could lose their characteristic shape and showed widespread damage in structure. The mitochondria1 cristae were marginated swollen. Some of the mitochondria are transected by cristae, suggesting division of the inner compartment. The cristae assumed circular shape at many instances. Most of the mitochondrial cristae fragmentation was seen at their luminal ends. Granulation in the swollen mitochondria matrix was also detected. In some cases the mitochondria were found extremely swollen and totally devoid of cristae (Fig. 8. a and b).

The rough endoplasmic reticulum lost their characteristic arrangement. They were broken down into very small bits which were distributed haphazardly throughout the cell. Total disintegration of the reticule was also noticed in some cells. Smooth Endoplasmic reticulum was swollen and in some cells appeared to be increased in amount. The reticulum in some regions enclosed electron-dense material by bending of cisternae (Fig.8. a and b).

Lysosomes were numerous in some cells contained dense bodies of varying size. Furthermore, the accumulation of electron dens material of different sizes was observed (Fig.8. a and b).

Nuclei showed marked clumping of chromatin with aggregation of interchromatin material at the center of the nucleus. In some cells, bilobed nuclei with dilated nuclear membranes. The nucleolus, instead of being similarly incorporated into the expanding masses, was displaced peripherally (Fig.8. a and d). Intranuclear electron dense inclusions of different sizes were observed. The inclusion appeared a small electron-dense zone midway between the nucleolus and the nuclear membrane. It was composed of densely packed granules considerably smaller than those comprising the nucleolus or those found in the surrounding nucleoplasm. Inclusion bodies consisting of dense masses surrounded by fibrous networks. (Fig.8. a and d).

Conspicuous thickening of the basal lamina, especially in areas associated with invaginations. The basal lamina developed characteristic folding and these folds were found to contain electron-dense materials. The entrapped materials could possibly be invagenated from the basal lamina and taken into the cell (Fig.8. c and d). The plasma membrane was also found swollen at a few places. The microvilli were shortened, disorganized and disordered. Remnants of a brush border appeared.

Brain:
The most common alteration were the presence of pericellular oedema (Fig.9). Glioses or sattelosis in brain gray matter (Fig.9 a, b and c), demylenation or spongiosis in brain white matter chromatolysis of the cytoplasm with cytoplasmic vaculation in the neuron of the cerebral cortex (Fig.9. d and e). The blood vessels were congested. Their endothelial lining were hypertrophied with the presence of acidophilic deposits inside their lumens. Most of the perkinji cells showed signs of degeneration and other appeared swallen with fintaly granular cytoplasm and loss of their nuclei (Fig .9 f). Their number was reduced. Encephalitis represented by gliosis and satellatosis, congestion and perivascular oedema. Several acid-fast intranuclear inclusion bodies were present in cells of the tubules. PAS positive deposits

a Strong PAS-reaction around the cells. Staining by Mercury Bromophynol showed precipitation of the stain inside the brain tissue showing a strong stainability of total protein contents in the cells (Fig.9. J, k and i).

Lung:

The histopathological alternation of lungs of the studied migratory birds revealed varying degree of chronic inflammation. The alveoli were markedly filled with erythrocytes and fibrin threads and large number of mononuclear cells, lymphocyte and macrophage (Fig.10. a, b and c). Large areas of necrosis were seen. The inter alveolar blood capillaries were congested. The lungs exhibited focal thickening of the interstitial connective tissue which, became more extensive, encroaching upon the alveolar walls, nor was there morphologic evidence morphologic of severe circulatory impairment (Fig.10 d, e and f). All birds had hemorrhages in the lungs. A significant effect was found for pulmonary fibromuscular hyperplasia and haemorrhage in lungs. Distortion of lung structure was found in this same concentration. The lungs of severe haemorrhage and distortion of lung architecture were observed (Fig.10. d, e and f). PAS positive at the interalveolar septa, thickness of pulmonary blood vessel (Fig.10. g and h). Deposite of bromophenol at the wall of blood capillary (Fig.10. i). The groulomas appeared as chronic inflammary cellular reaction surrounded by a cellular zone which different in thickness according to the size of granuloma. It is consisted mainly of mononuclear lymphocyte. The granulomas were surrounded with connective tissue encapsulated by proliferation of fibroblasts and fibrocytes (Fig.10. j and k). Also, diffuse haemosiderosis could be observed. The lungs exhibited apoptosis (Fig.10. i).

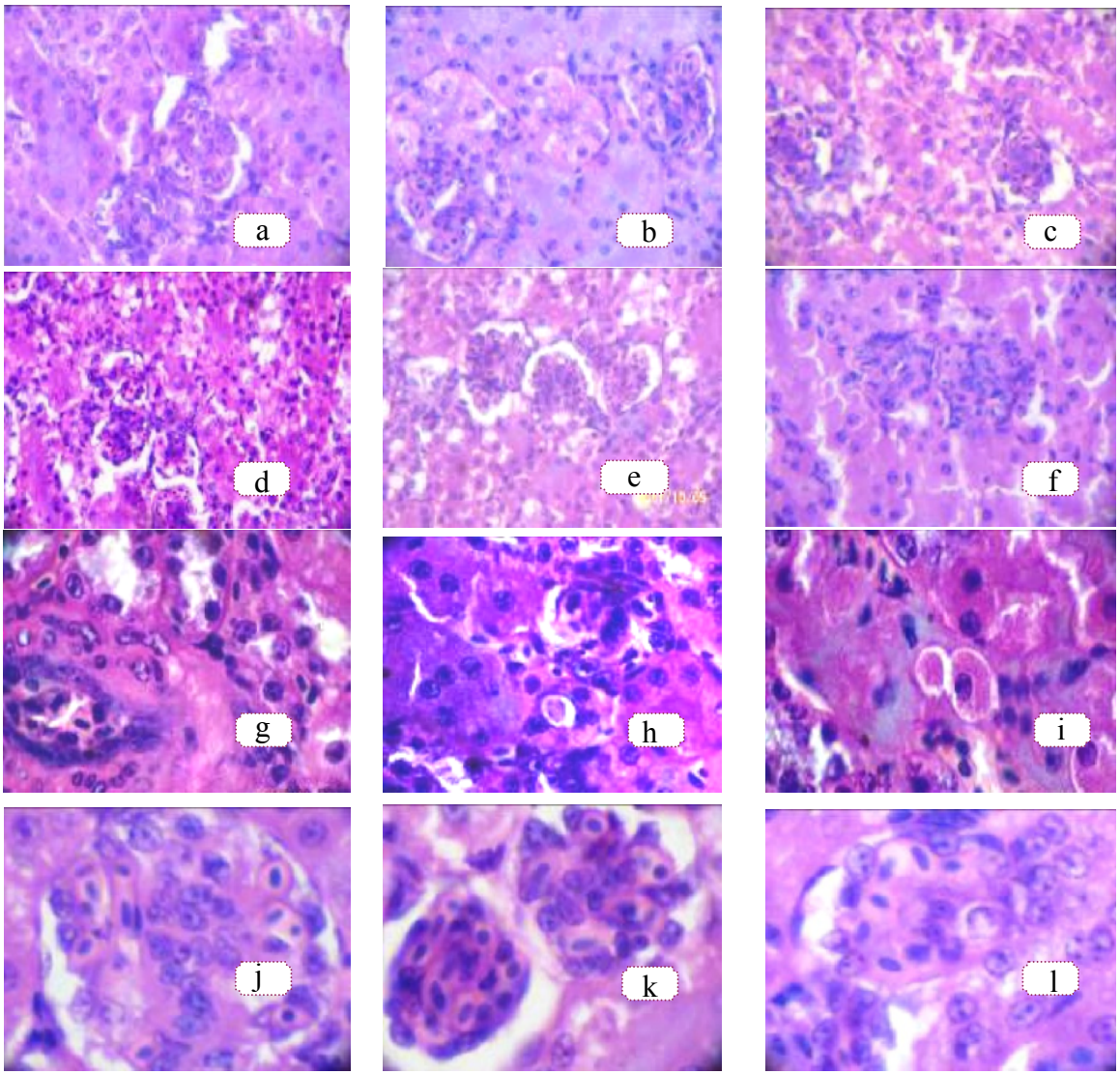

Fig. 1: Kidney of migratory birds showing priglomerular focal amyloid deposite (a,b,&c). Hypercellularity, proliferation and accumulation of mesangial cells, expansion of space inside the Bowman's capsules and contraction of the glomerulus (d,e&f). Contracted glomerular tufts with periglomerular fibrosis. Mononuclear cell infiltrates, diffused hyaline and thickening of capillary endothelium, densely packed granules. (g,h&i). Hyperaemia, glomerular tufts with haemorrhage (j,k&l).

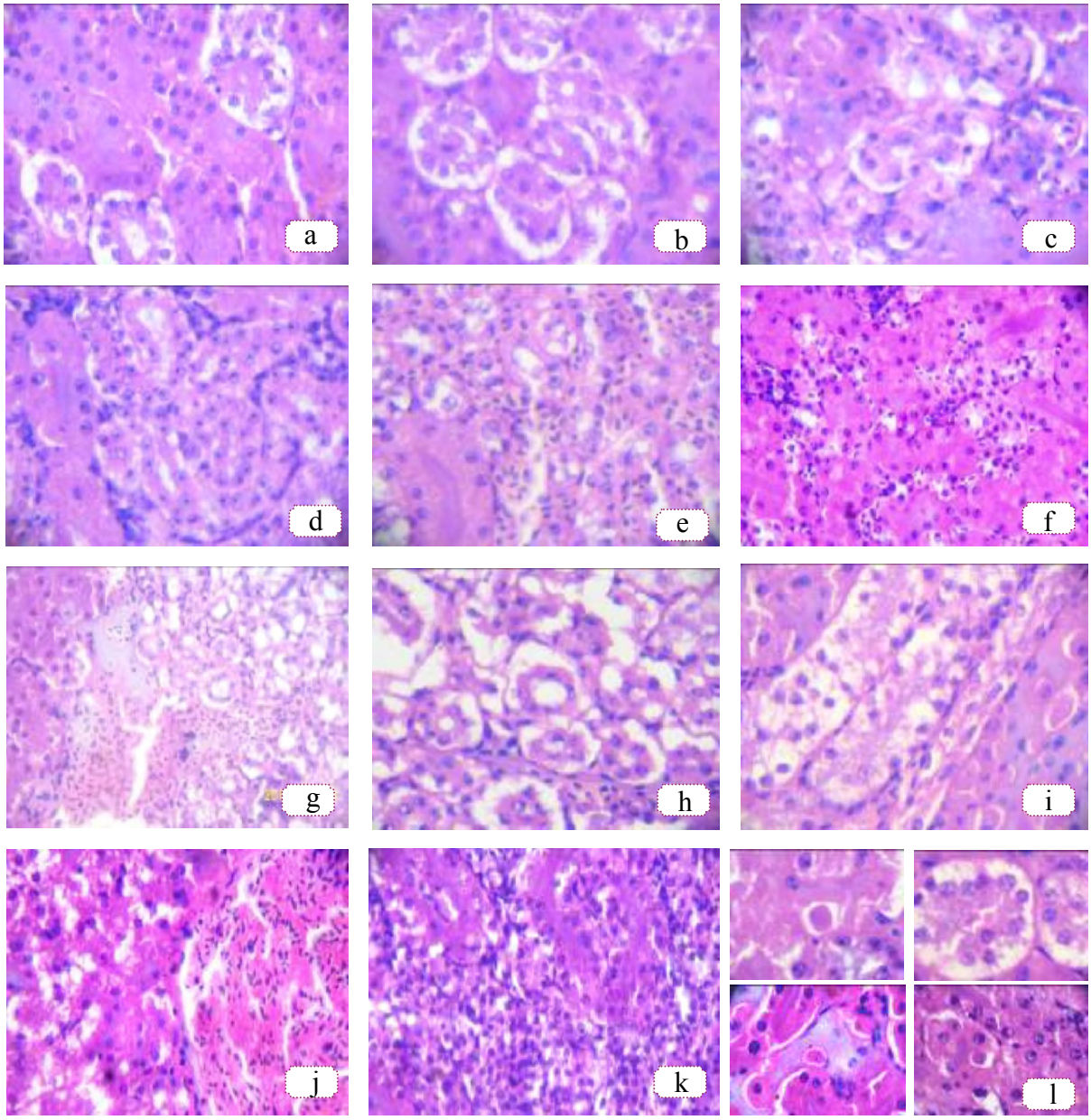

Fig. 2: Kidney of Migratory birds showing cytoplasmic vacuolation of tubular epithelial cells of the proximal convoluted tubules, dilatation with accumulation of eosinophilic homogenous material in tubular lumina ,hypertrophy of epithelial cells and degeneration of epithelia of renal tubules with infiltration of mononuclear cells (a,b,&c). Focal cellular necrosis in the renal proximal tubule, nuclear and cytoplasmic pleomorphism, nuclear pyknosis Kayorrhexis, karyolysis, cytolysis and fibrosis (d&e). Inflammatory cellular infiltration, amyliod deposite (f). Cytoplasmic vacuolation of tubular epithelial cells of the distal convoluted tubule, focal haemorrhagic area in between the renal tubules and collecting tubules Collecting tubules are lined with the relatively low simple cubic epithelium(g&h&i). Severe vascular degenerative changes in the lining epithelial cells of the renal tubules at the corticomedullary portion and distortion the renal architecture j). Fibrous peritubular necrotic tubules (k). Several appoptic bodies in cells of the tubules, eosinophilic homogenous material in tubular lumina, renal tubules showed single epithelial cells desquamated to their lumen necrotic cells with pyknotic nuclei sloughed into the tubular lumen (l).

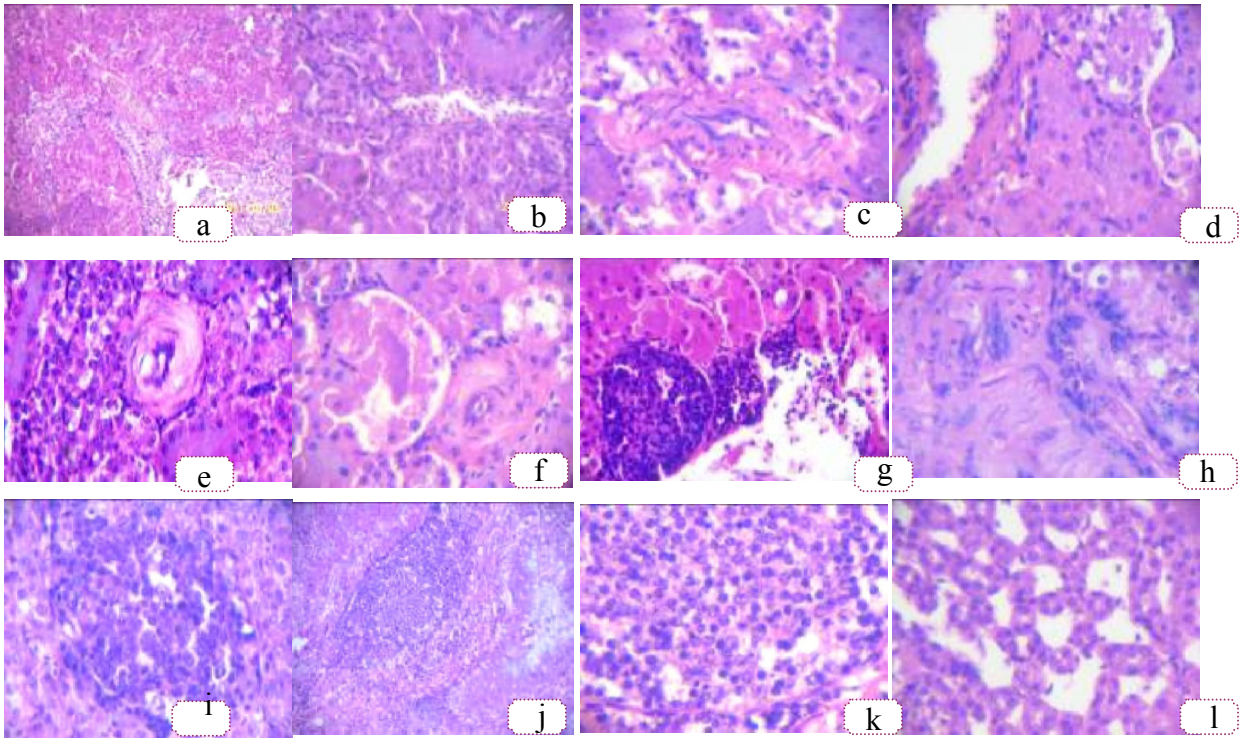

Fig. 3: Kidneys showing interstitial odema, dilatation and congestion of blood capillaries , hemorrhage between the degenerated renal tubules at the corticomedullary portion, degeneration in the epithelial cells lining the renal tubules (a&b). Hyperemic capillaries, thickening of the capillary wall (c&d). Degenerayed renal tubule ,thickening the wall of blood vessels infiltration of chronic inflammatory cells(e&f). Multifocal to coalescing amyloidosis with lymphoid cells aggregation (g). Strands of fibroblasts (h). Granulomas. cellular reaction of mononuclear, hyalinosis and encapsulated by proliferation of fibroblasts and fibrocytes (l,j&k). Regenerated renal tubules (l).

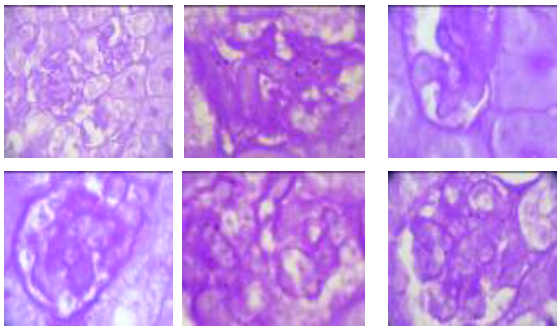

Fig.4 a: PAS positive deposit in the glomerular basement membrane, intense PAS-positive deposits in Bowmann's basal membrane. Depositst in the mesangium and thickening of the basement membrane.

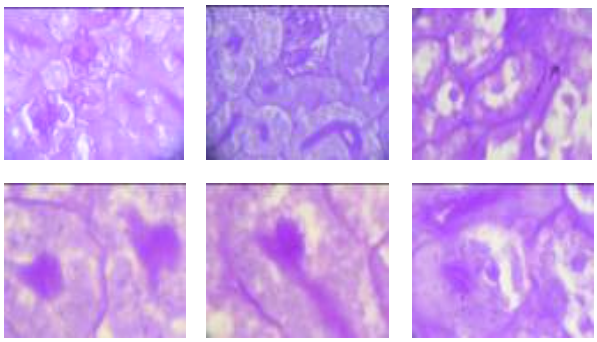

Fig.4 b: Dilatation and atrophy of the proximal tubules with luminal hyaline deposits . kidney section revealing destruction of brush borders of renal tubules and increased glycogen content. (PAS).

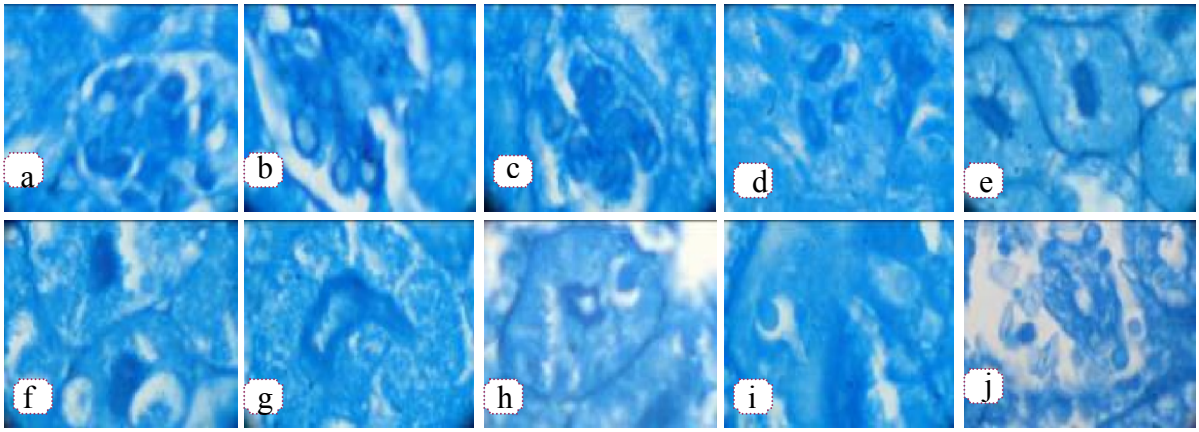

Fig. 5: Kidney section illustrating Bromophenol blue stain positive deposits in Bowmann's basal membrane, glomerul, Depositst in the mesangium, thickening of the basement membrane (a,b,c&d). dilatation and atrophy of the proximal tubules with luminal deposits and increased with improvement of brush borders of rena ltubules (e,f&g). kidney sections showed appoptic bodies increased protein content(h,i&j).

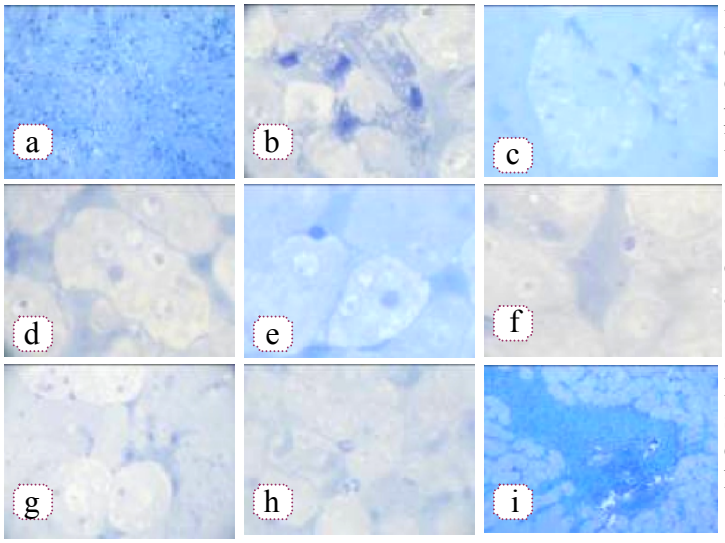

Fig. 6: Semithin section Epithelial cells contain small dark granules in the cytoplasm(a&b). The glomeruli with proteinaceous content in the space of Bowman's capsule .displays hyperplasia(c).

Epithelial cells of kidney tubules with degenerative changes and dark granules in the apical sections of the tubular(d,e&f).

Epithelial cells showed presence of several stages of cell mitosis(g&h Tubular cells show degeneration cortical and medullary haemorrhage(i). (Toluidine blue staining).

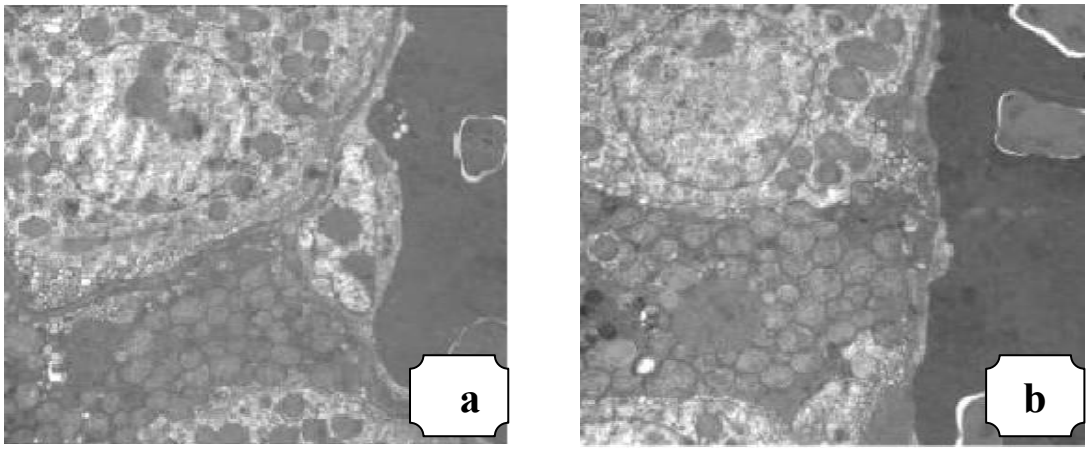

Fig. 7a&b: Demonsterated transverse section of the gromuli, the mesangial cells showed the presence, well developed Golgi apparatusan and SER. numerous spinous cytoplasmic processes, mitochondria proliferation, RER lost their characteristic carrengement, Increased number of lysosomal, abundancy of osmophilic bodies of different sizez, well developed peroxosome. Nuclear chromatin is extremely dense. inclusion body in the center of the nucleus. The endothelial cells cells of the glomerular capillaries were swallen and lost their fenestrace.

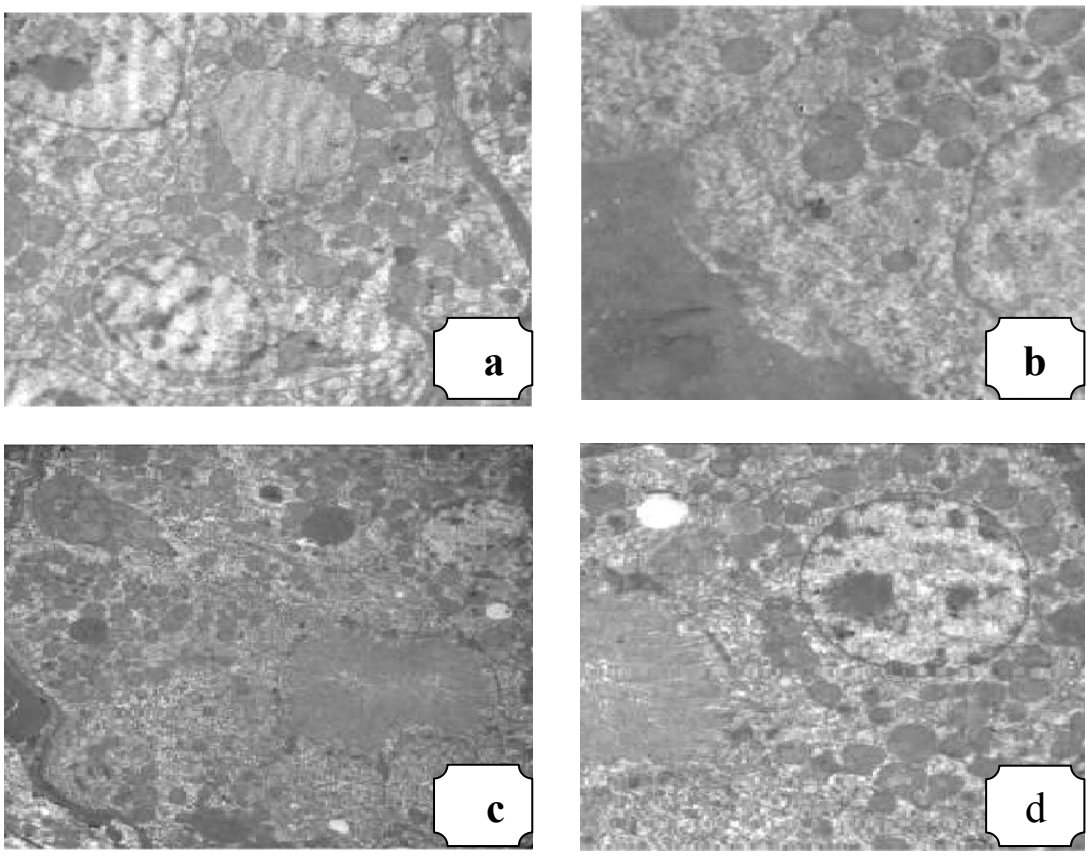

Fig. 8 a&b: Demonstrated transverse section of the tubule with degenerative changes of the epithelial cells. well developed SER. RER lost their characteristic arrangement, mitochondrial proliferation, Increased number of lysosomal and abundancy of osmophilic bodies of different sizez, well developed peroxosome. Nuclear chromatin is extremely dense. inclusion body in the center of the nucleus. The nucleolus is displaced peripherall. Microvilli Remnants of a brush. Fig. 8c&d demonstrated abundancy of osmophilic bodies of different sizez, mitochondrial proliferation. The basal membrane is varies in thickness, with erythrocyte, Microvilli Remnants of a brush bordre.

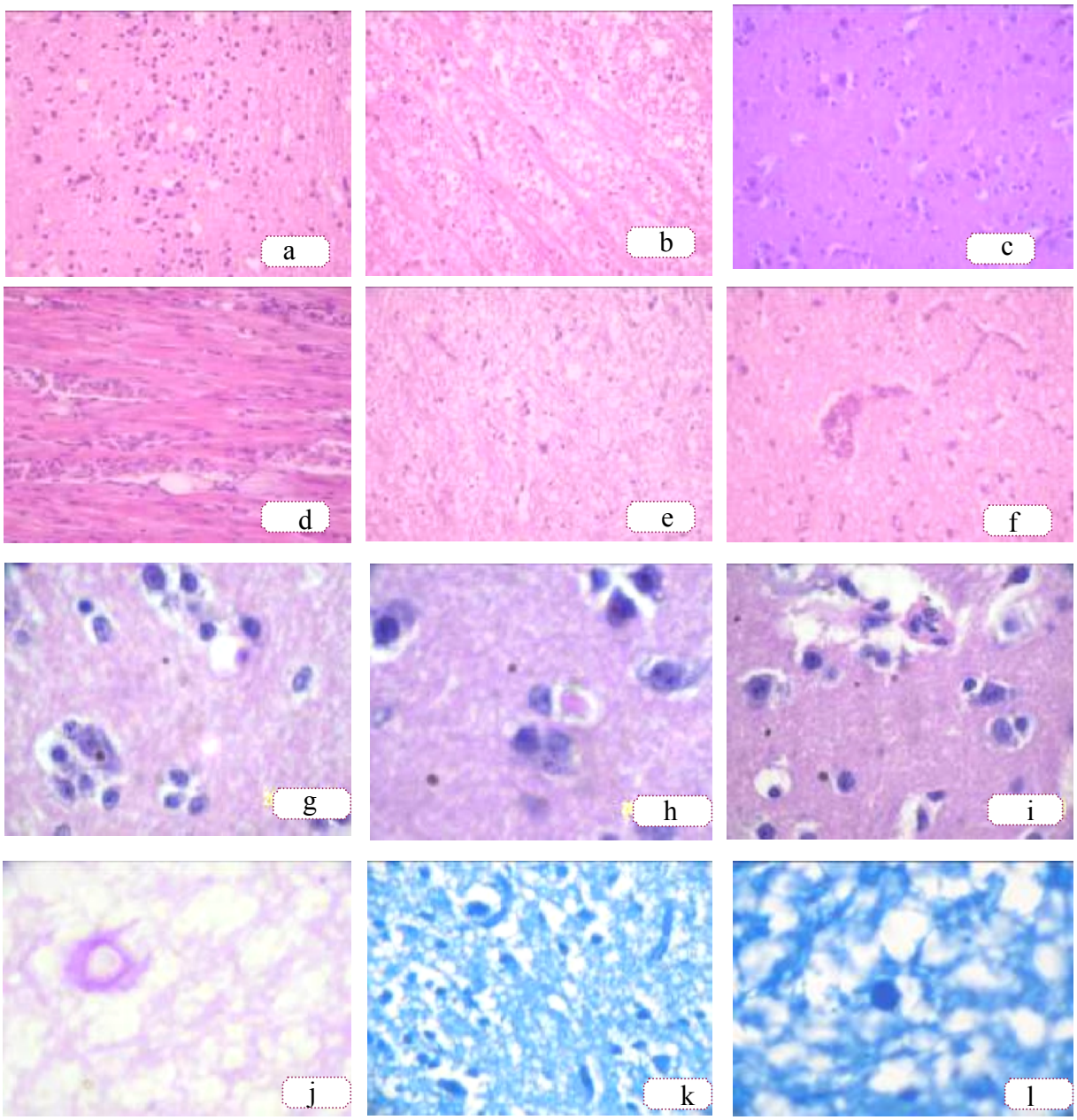

Fig. 9: Brain of migratory birds showing pericellular oedema, glioses or sattelosis in brain gray matter (a,b&c)). Demylenation or spongiosis in brain white matter chromatolysis of the nucleus and cytoplasmic vaculation in the neuron of the cerebral cortex (d&e). The blood vessels were congested dilated blood capillaries and hypertrophy of their endothelial lining with the precence of acidophilic deposits inside their lumens (f). Perkinji cells showed signs of degeneration and other appeared swollen with faintly granular cytoplasm and loss of their nuclei, cerebellar Purkinje cells were apparently reduced in number, several acid-fast intranuclear inclusion bodies (g,h&i). PAS positive deposits a Strong PAS-reaction around the cells. (j) Mercury Bromophynol showed precipitation of the stain inside the brain tissue the inclusion bodies(K&l).

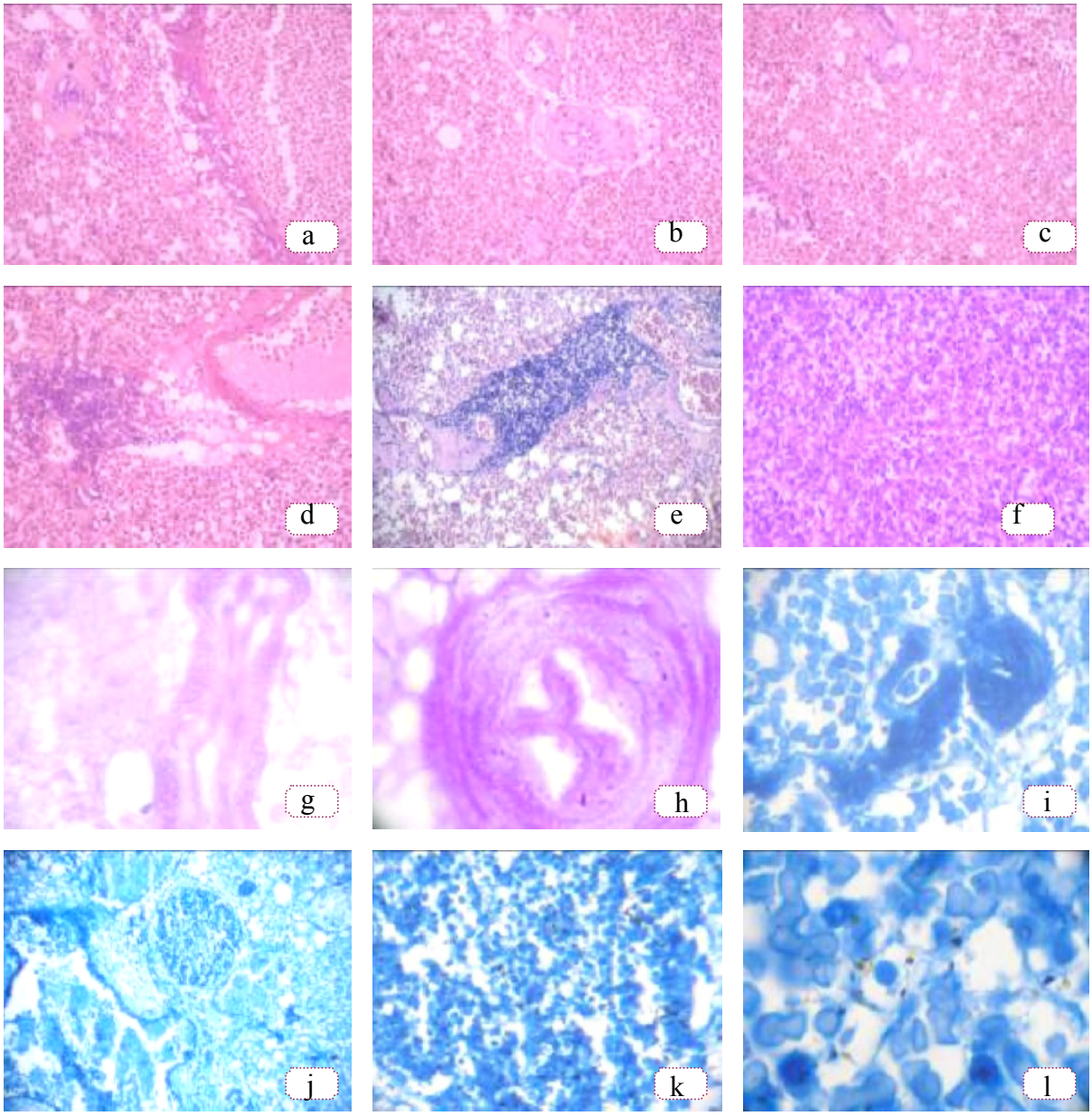

Fig. 10: Lung of migratory birds showing chronic inflammation, haemorrhages, congestion, interstitial oedema, serous exudation and cellular infiltrations in the lungs, fibriniod necrosis of blood vessel, vacuolar degeneration, the alveoli were markedly filled with erythrocytes and fibrin threads pulmonary fibromuscular hyperplasia (a,b&c). Focal area of necrosis, aggregations of inflammatory cells between the lung tissue, heamolysis and thrombosis, haemorrhages, congestion, dilation and congestion in blood sinusoids, large areas of heamorrage in blood vessels, fibrin deposit and the lung lost the architecture (d,e,&f). PAS positive at the interalveolar septa, thickness of pulmonary blood vessel. (i) deposite of bromophenol at the wall of blood capillary (g&h).) granuloma, cellular reaction surrounding by cellular reaction of mononuclear, hyalinosis and encapsulated by proliferation of fibroblasts and fibrocytes(j&k). diffuse haemosiderosis, The lungs exhibited apoptosis.(l).

DISCUSSION

The concentrations of the Pb .Cd, and Cu were detected in the four species of migratory birds were higher than the permissible level. Heavy metals are known to be comulated in birds. Low Pb concentrations have been reported for a range of seabird species sampled from oceanic or remote locations (Norheim, 1987 and Honda *et al.,* 1990). Several workers have found somewhat elevated Pb concentrations in species (Turner *et al.,* 1978; Lee *et al.,* 1989; Lock *et al.,* 1992). In contrast, Cd concentrations in the kidney were higher than those in the liver. Lacunas *et al.* (2004) stated that seabirds tend to exhibit high accumulations of cadmium, in their livers and kidneys.

High Cu levels occur in other species of waterfowl without any signs of toxic effects (Clausen and Wolstrup, 1978). High Cu levels in waterfowl are likely to have arisen from species-specific bioaccumulation and unlikely to reflect pollution or higher natural background of Cu in their habitats (Kim *et al.,* 1996). Muirhead and Furness (1988) demonstrated significant differences seem to exist in the levels of essential trace elements, Cu between the liver and kidney.

The present work showed that the kidney cortex is more affected than medulla due to long-term exposures to heavy metals. The kidneys showed marked glomerular, tubular and interstitial pathological alternation. Nicolson and osborn (1983) examined the kidneys of three species of pelagic seabird which had high tissue cadmium level showed that the seabirds had patchy nephrotoxic kidney lesions. Pathological features included necrosis and degeneration of the proximal tubular epithelium together with the direct observation of obstruction of the more distal neprhon segments by necrotic cellular debris. According to Tanimoto *et al.* (1999) cadmium is responsible for the pathological changes of kidney tubules, and these findings were also confirmed by the results of our investigation related to the kidneys of migratory birds. Nicolson and Osborn (1983) demonstrated abnormalities of the glomerular podocytes and Bowman's capsule cells also Geeth *et al.* (2005) reported that cadmium is a potent nephrotoxin that has been shown to induce apoptosis in some cells. In several clinical situations and experimental models of injury to the renal glomerulus, pathological proliferation of mesangial cells was followed by resolution involving mesangial cell apoptosis. Similar alterations in and kidney were observed and these alterations were described by Thophon *et al.* (2003) and Mansoure *et al.* (2008).

The results showed that the tubular damages were more prominent in the proximal convoluted tubules in comparison to that in the distal ones. Burce *et al.* (1980) in the kidney as evidenced by cytomegaly, karyomegaly nuclear inclusion body formation and increased numbers of iron-positive granules within renal proximal tubule cells. (Karmakar *et al.,* 1986 and Colle *et al.,* 1980) discussed that tubular alterations might be as a result of a hydrolic changes in the renal tissue and suggest that intoxication yields to a partial failure in the ion pump transport of tubules cells which in turn produces tubular swelling and causes necrosis and vacuolization of the tubules. These changes might indicate incapability of the renal cells to deal with the accumulated residues resulting from metabolic and structural disturbances caused by lead. Histopathological changes were typically desquamation and atrophy of the epithelium, dilatation of the proximal tubules with luminal hyaline casts, interstitial and tubular fibrosis, leucocyte infiltration of the interstitium, fusion of the parietal part of Bowmann's capsule and the glomerulus, and glomerular sclerosis (Scott *et al.,* 1977; Squibb *et al.,* 1979; WHO, 1992; Yasuda *et al.,* 1995 and Liu *et al.,* 1998). These findings are in agreement with our results and the previous data reported by Mansour *et al.* (2008) and Lin *et al.* (1993). They recorded alterations in renal histopathology due to environmental exposure to lead.

The presence of hyaline casts in the lumen of the damaged tubules might be an indication of glomerulonephritis and or partial failure of tubular reabsorption and intoxication. Sonne (2002) reported hyaline droplet degeneration was found in the proximal tubules and protein casts, primarily in the medulla of the kidney. The histopathological findings in the glomeruli were compared to the occurrence of hyaline droplet degeneration and protein casts. But no significant connection was found between histopathological findings and hyaline droplet degeneration or between histopathological findings and protein casts. A possible explanation is that the tubular protein casts can be found before the glomerular lesions can be observed. Goyer *et al.* (1970) predicted that lead would have a profound effect in the nucleus because of the ability of metals to alter nucleic acid conformations. Further, lead is the most active metal in the formation of intranuclear inclusion bodies which is the consistent feature in lead-intoxicated animals. Goyer (1971) has proposed that the intranuclear inclusion bodies function to bind lead and to minimize its effects. Several observations support this puroposal. The appearance of the inclusion bodies, following lead exposure, appears to depend on protein synthesis.

The main interstitial alterations in the kidney of examined birds were mainly interstitial edema together with blood capillaries congestion. Similar findings were reported by previous studies on mammals model (Jarrar, 1999). The histochemical results reported in the present study revealed a marked increased in PAS-positive materials in the brush borders of the renal tubular epithelial cells

suggesting the damage of cell membrane including its microvilli. These results were in agreement with the results of Friberg *et al.* (1986) who reported that Cd affects the carbohydrates metabolism resulting in the depletion of tissue glycogen as well as the changes in renal function involves abnormalities of tubular reabsorption manifested by such conditions as low molecular weight proteinuria, glucosuria, aminoaciduria and phosphaturia. According to Cheville (1994) extensive degeneration of the endoplasmic reticulum, as noticed in the present study lead, cadmium and cupper are able to reduce the rate of protein synthesis by influencing the attachment of polyribosomes to the rough endoplasmic reticulum and probably damaging the ribosomes themselves. The occurrence of fatty droplets and dark granules in the cytoplasm of tubular epithelial cells, the accumulation of these fatty droplets and dark granules caused the tear of cytoplasmic organelles and sporadic tubular necrosis along with a degeneration of tubule. A general negative effect exerted by metals on the membranes of both the rough and smooth endoplasmic reticulum is due to lipid peroxidation (Buss and Gibson, 1979). Bubel (1976) reported that cadmium had particularly adverse effects on the mitochondrial membrane, by means of crystal accumulation that greatly reduced the synthesis of ATP. In our electron microscopic investigation the mitochondrial proliferation was noticed, but crystal sediments on mitochondria were not observed. George (1982) reported active accumulation of metal cations by mitochondria, the organelles responsible for aerobic ATP production. George (1982) stat that metals accumulate in lysosomes by a combination of autophagy and generation of acidic groups within aging secondary and tertiary lysosomes. The formation of pathologically enlarged lysosomes was associated with membrane destabilization or increased permeability resulting in the release of degradative hydrolytic enzymes into the cytosol and also in lysosomal fusion with other intracellular vacuoles.

Sujatha *et al.* (2011) reported accumulation of the metal within the residual bodies located near the luminal surface of the epithelial cells and suggested exocystosis of the metal into the lumen. In the present investigation the residual bodies takes several location with different sizes inside the the cytoplasm. Vacuolation of the tubular cells may be due to the formation of membrane-bound vesicles containing accumulated heavy metals as electron-dense granules or perhaps be related to vesiculation of endoplasmic reticulum as a result of the failure of ion pump. The incidence of secondary and tertiary lysosomes as a conspicuous feature in the tubular cells suggests xenobiotic induced cellular pathology disturbing both structure and function (Moore, 1985).

Ultrastructural studies revealed mitochondrial swelling and the presence of increased numbers of lysosomes within renal proximal tubule cells (Burce *et al.,* 1980). They showed the highest intracellular lead concentrations in nuclear inclusion bodies within renal proximal tubule cells and Inhibition of renal mitochondrial respiration. By electron microscopic examination, the osmophilic granules and particles found in the cytoplasm of the kidneys epithelial cells can be linked to heavy metal poisoning. Walther *et al.* (2003) asserted that zinc endangers protein metabolism in the cell and this feature of zinc could account for the occurrence of granules observed in the tubules as well as for the damaged cells in the tubular system of the kidneys. The increase of zinc resumption in tubules can cause in a very short time lasting changes related to osmotic nephrosis, possibly through the stabilization of lisosomal membrane which has been affected by zinc. Nuclear alterations were the early histological alterations developed due to long-term intoxication. This confirms that the nucleus is the key factor in most cell disturbances to injurious agents (King *et al.,* 1983) and undergoes morphologic lead poisoning. The development of the nuclear alterations might indicate the metabolic and structural disturbances as a result of lead detoxification mechanism.

Ezemonye and Enuneku (2011) reported the histopathological changes in the lungs were pulmonary haemorrhage and distortion of lung structure. These changes could lead to loss of lung elasticity and capacity to carry out gaseous exchange. The lung is the organ that ensures gaseous exchange and in addition, regulation of immune responses to inhaled antigens. In the present study, the lung showed haemorrhage and distortion of lung structure. These alterations could lead to loss of lung elasticity and capacity to carry out gaseous exchange. Toxic substances can injure the lungs thereby disrupting gaseous exchange and impairing immunological responses. The lungs have been identified as a target organ of heavy metal toxicity (Roberts, 1999 and Egwurugwu *et al.,* 2007). Similar results were observed by Ezemonye and Enuneku (2011) who found that heavy metal exposure to the lungs could cause haemorrhage and distortion of lung structure. These alterations could lead to loss of lung elasticity and capacity to carry out gaseous exchange.

The brain lesion consisted of demeylionization with neural atrophy in hippocampus were due to free radical production and pb induced oxidative stress (Abbas *et al.,* 2000). Encephalitis represented by diffuse mononuclear cells infiltration, congestion and perivascular oedema and cerebral infarction were also noticed. The sever neurodegenerative change demonstrated that the toxic effects of the lead principally were manifested in the central nervous system leading to destruction of the blood brain barrier, which leads to edema of neurons and reactive gliosis. In the present study, the most common alterations were the presence of pericellular oedema, glioses and sattelosis in brain gray matter.

Light microscopic examination, revealed oedema. Similar findings were recorded by Zheng (2001) who proved that lead is selective choroid plexus toxicant. It does not directly disrupt the morphology of brain barriers rather than alter their regulatory functions, bringing about neurotoxicity due to disorder cerebral homeostasis. Degeneration as well as necrosis of the purkinji cell layer of cerebellum showed disarrangement of the cell layers of cerebeillum with more than one space in between its granular layer layers and purkinji. The blood vessels were congested and dilated with hypertrophy of their endothelial lining with the presence of acidophilic deposits inside their lumens. Most of the perkinji cells showed signs of degeneration and other appeared swollen with fintaly granular cytoplasm and loss of their nuclei. Zheng, 2001 reported that lead toxin can access aparticular region and spare others. Struzynska et al., (1997) proved that lead appeared to have unique affinity to cerebral endothelial cells where it accumulates in great concentration explaining the vascular theory.

CONCLUSIONS

Birds contaminated by heavy metals suffered pathological alterations, with consequent inhibition of metabolic processes and hematological changes. Migratory birds has the tendency to bioaccumulate heavy metals in a polluted environment. Due to the feeding behavior of the some people (eat migratory birds) government should intact laws that will ensure that industries should make use of standard waste treatment before they are being discharged into water bodies.

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التراكم البيولوجى لبعض المعادن الثقيلة مع دراسة التغيرات الهستوباثولوجية والتركيب الدقيق فى الطيور المهاجرة المتواجدة حو ل بحيرة المنزلة

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استهدف البحث تقييم المعادن الثقيلة فى انسجة الطيور المهاجرة المتواجدة حو ل بحيرة المنزلة ومدى الارتباط بين تركيز هذة المعادن فى الانسجة والتغيرات الهستوباثولوجية في الفترة من سبتمبر حتي مارس (2011-2012) من تجمعات الطيور المتواجدة على | بحيرة المنزلة ، في مجموعة متنوعة من أنواع الطيور وصنفت الى البط الخضارى ، البلبول , الشرشير والغر. وتم قياس المعادن الثقيلة في الكبد والكلى والمخ. أظهرت النتائج عن تراكمات المعادن الثقيلة في انسجة الطيور قيد الفحص في الترتيب التنازلي: الرصاص> الكادميوم> النحاس.بالفحص العينى كانت الطيور هزيلة وصغيرة الحجم اما الاعضاء الداخلية فكانت محتقنة الكبد متهتك والكلى وبهما بعض الانزفة والرئتين داكنة اللون بها انزفة تظهرعلي السطح الخارجى والامعاء منتفخة داكنة وتضخم الطحال. وأظهرت الكلى تغيرات واضحة بارزة في القشرة عن النخاع. وتشتمل التغيرات الكبيبات والانابيب والنسيج البيني ، اظهرت مثل هذه التغييرات وجوداختلاف واضح في الشكل العام للكبيبات فكانت منبعجة ومنكمشة مع سماكة الغشاء المبطن للظهارة الكبيبة. وأظهرت الانابيب تمدد واضح مع احتواءتجويفها علي مواد متجانسة . تتكرز خلوى في الانبيبات الملتوية الكلوية وقد لوحظت التنكرز مع تعدد الأشكال النووية تغلظ النووية، انحلال النواة، انحلال خلوي وتليف ، اظهرت الكلى ارتشحات في النسيج البيني مع وجود احتقان وارتشاح . الكبيبات والانابيب انزفة بين الأنابيب الكلوية والتهاب مزمن بين والنبيبات البولية وارتشاح للخلايا الالتهابية مع حدوث تغييرات ملحوظة في الاوعية والشعيرات الدموية في النسيج البيني مع وجود احتقان وارتشاح. الكبيبات والانابيب اظهرت الخلايا إيجابية لصبغة PAS في الكبيبات و الانابيب ، مع زيادة في البروتين وضحت عند الصبغة Mercury bromophenol. واظهر فحص semithin sections بصبغةtoulidine blue ان المعادن الثقيلة أظهرت عدد كبير من الحبيبات الموجبة للصبغةفى الجزء الاعلي للخلايا. اظهرت الفحوصات بالميكروسكوب الالكتروني تغيرات واضحة فى خلايا الكبيبات و الأنابيب تتمثل فى تضخم الميتوكوندريا في جميع الخلايا اما الشبكة الاندوبلازمية فقد فقدت تركيبها كليا مع وجود العديد من اليسوزومات في بعض الخلايا متفاوتة الحجم ، وزيادة عدد المواد الليزوزومية المتبقية مع وجودجسيمات متبقية كبيرة. وأظهرت النواة تكتل واضح للكروماتين فكانت تحتوى على جسيمات داخل النواة .الفحص لقطاعات المخ وجدت التغيرات على هيئة ارتشاحات مع وجود تليف للمادة الرمادية ونزع الغلاف حول الخلايا العصبية للمادة البيضاء وتحلل كروماتيني للنواة وفجوات سيتوبلازمية في الخلايا العصبية للقشرة . احتقانات رئوية وامتلأت الحويصلات الهوائية بشكل ملحوظ بكرات الدم الحمراء واحتقان رئوي و سماكة في النسيج الضام بين الحويصلات الهوائية .