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Histopathology of Kidney of Albino Rat Poisoned with Uranyl Nitrate

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Heavy metals input into the media either terrestrial or aquatic, is an important aspect of environmental pollution. Heavy metals are known to produce toxic effects on the different tissues of various terrestrial and aquatic animals. Some of these are highly toxic at even very low concentrations and they alter the cellular architecture of many organs including the kidney. Little has been done on the effect of rare earth metals particularly that of uranium (CARONE & SPECTOR 1960, BENCOSME et al. 1960) on the kidney of animals. In the present communication histopathological changes produced by uranium on the kidney of albino rats have been discussed.

MATERIALS AND METHODS

Male albino rats of almost similar in weight (175 to 200 g) were collected in 4 separate groups and each group was having 4 rats. Animals of all the groups were kept on standard pellet diet from Liver Brothers (India) Ltd. and water ad libitum. Out of these 4 groups, the animals of one group were kept as control and fed on the above diet. The rats of remaining 3 groups were fed on 20 mg of uranyl nitrate mixed with their pallet diet on alternate days. The rats from both control and experimental groups were sacrificed after 15, 19 and 27 days of the initial dose and the kidney from the sacrificed rats was fixed in neutral formalin to cut paraffin sections of 5 to 7 μm. The histological changes caused by uranyl intoxication were studied in contrast to the control kidney structure using haematoxilin and eosin stains.

OBSERVATIONS

At 15 days treatment preliminary pathological changes were observed (Fig. 1). The glomerular capsular wall was found to be thickened accompanied by the shrinkage in the glomerular capillary network leaving space between the wall and network. In the renal tubules the cellular epithelium showed many degenerative changes. In proximal and distal convoluted renal tubules the vacuolation of cytoplasm was noted inside the cells and nuclei occupied an excentric position. Degeneration of inner epithelial membrane (brush border) was comparatively more in proximal tubules than that of distal convoluted tubules. However, the tubules in most areas of kidney were observed to be intact.

After 19 days uranyl treatment the histological structure of kidney was considerably altered (Figs. 2, 3). Degeneration of tubular walls was more extensive and many of epithelial cells showed

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hydropic degeneration. Nowhere was karyo-pycnosis observed. Most of the tubular cells were desquamated and the lumen was obliterated. At a few sites the glomerular capsular wall was damaged. Further shrinkage in the glomerular network was noted as the intracapsular cavity widened. In perihaemal area the changes were most severe where the damage of tubular epithelium was accompanied by clumping of the nuclei at a few places. Drastic changes were observed in the interstitial hematopoietic tissue also. Most of the vessels were ruptured and red blood corpuscles invaded the tissue resulting in a hemorragic condition.

Most severe renal histopathic lesions were observed after 27 days treatment with the toxin (Figs. 4, 5). Most of the renal tubules in medullary region of kidney were completely deformed due to extensive hydropic degeneration of the cells and the nuclei were haphazardly arranged. The spindle-shaped nuclei depicted its pycnotic condition at many places. The connective tissue between the renal tubules was almost completely damaged and only scars of dead cells were visible. Further shrinkage of the glomerular capillary network widened the space between capsular wall and the glomerulus. Severe clumping of the nuclei was also observed at many places in medullary zone. Only a few alterations were observed in the cortical region after prolonged uranyl treatment. The damage of the epithelial membrane of some renal tubules and of the interstitial hematopoietic tissue, vacuolation of cytoplasm and excentric position of nuclei were specially noted in the corticomedullary region. Necrosis was occasional and the damaged tubules were seen only at a very few places. The peripheral part of the cortex was almost unaffected.

DISCUSSION

Important renal histopathic lesions produced by uranyl nitrate treatment involve the proximal convoluted tubules and glomeruli. The changes in the proximal convoluted tubules include vacuolization, loss of brush border, hydropic degeneration of the epithelium and keryopycnosis. Similar results in rabbit have been reported by BEAVER & BURR (1963) after treatment with bismuth. Necrotic findings of proximal convoluted tubules support the findings of CARONE & SPECTOR (1960). Vacuolization in the renal tubular epithelial cells has been reported by BENCOSME et al. (1960).

MACNIDER (1939) reported glomerular abnormalities induced by uranium. Glomerular damage, principally haemorrage in Bowman's space and focal necrosis of tufts, have been described in frogs by OLIVER & SMITH (1930) and in rabbits by HUNTER & ROBERTS (1932) after treatment with mercuric chloride. The finding of thickening of Bowman's capsular wall in the present study is in consonance with those of HACKEL et al. (1959) in rats treated with triodine.

BAUER et al. (1951) suggested that the nephrotoxin produces a disturbance in the fat metabolizing mechanisms. The nephrotoxic effect of uranyl nitrate is probably due to an interaction of the toxin with various enzymes such as succinoxidase, hexose monophosphate oxidase, cytochrome oxidase, amine oxidase, urease and esterase (CATER & PETERS 1961). According to LINDQUIST & FELLERS (1966), it is also possible that heavy metals are found to membrane sites. As a consequence the reabsorption mechanism may be blocked.

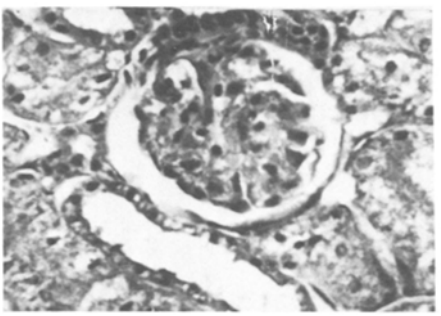

Fig. 1. After 15 days treatment X 240

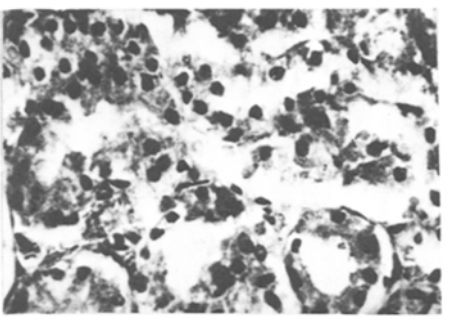

Fig. 2. After 19 days treatment X 240

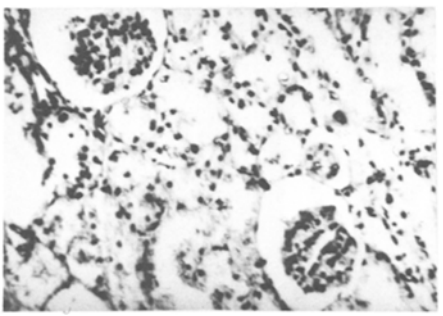

Fig. 3. After 19 days treatment (perihaemal area) X 150

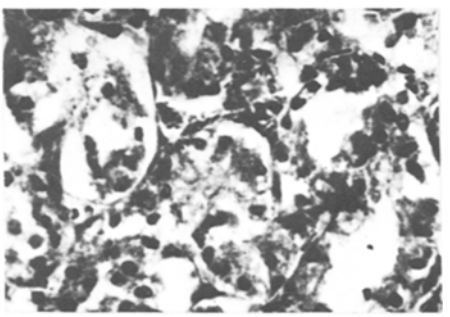

Fig. 4. After 27 days treatment X 240

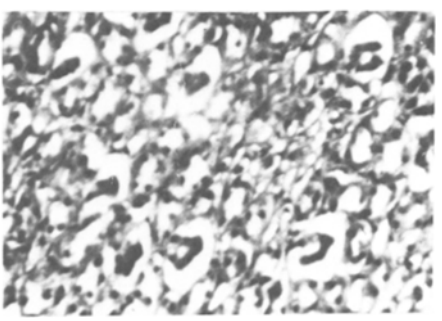

Fig. 5. Corticomedullary region after 27 days treatment X 150

However, the observations suggest that acute uranyl nitrate administration initially effects segments of the proximal convoluted tubule. Later subtle changes are produced effecting all parts of tubules which may be due to acute renal failure. It, therefore, appears that uranyl nitrate causes a disturbance in the energy metabolism of the cells at very early stages leading to other cellular disturbances and eventual cell death.

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