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Histopathological Changes In Two Models Of Acute Kidney Injury

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Two major challenges in management of Acute Kidney Injury, which cause and develop renal disturbances, are inflammation and oxidative stress. Acute Kidney Injury represents an important health problem, since an effective therapy to treat it is not available yet (1). During Acute Kidney Injury renal morphophysiology is affected as a consequence of renal ischemia/reperfusion (I/R) or nephrotoxicity (2, 3). The objective of this work was to identify renal histopathological changes in two experimental models of Acute Kidney Injury in Wistar rats.

Wistar rats (male, 300 g) were used in two experimental models of Acute Kidney Injury: a model of renal I/R and a model of nephrotoxicity. In each model, rats were distributed in two groups (n=8/group): a Negative control group and a Positive control group (rats submitted to renal I/R (30 min of ischemia and 24 h of reperfusion) in I/R model and rats treated with kanamycin (500 mg/kg, intraperitoneally, during 7 days) in nephrotoxicity model). Kidneys were sagittally sectioned, fixed in buffered formaldehyde 4 % and processed until paraffin embedding. Three µm thick sections were obtained, which were stained with hematoxylin/eosin and PAS. Histological kidney slides were analyzed, and the morphology of glomeruli, proximal tubules and interstitium was evaluated by means of a Zeiss Primo Star light microscope.

Normal morphology of renal cortex parenchyma (glomeruli, tubules and interstitium) was observed in Negative control animals corresponding to the two models of Acute Kidney Injury (Fig. 1 A, B). Renal I/R provoked tubular damage in wide areas, related to dilation of tubular lumens with detached cells (Fig. 1 C). These changes were accompanied by increase of cellularity and capillary enlargement in glomeruli and proliferation of inflammatory cells in the interstitium. Kanamycin treatment induced patchy tubular necrosis in the middle cortex, which was classified as moderate or intense (Fig. 1 D). Additionally, retraction of glomerular tufts with obliterated capillary lumens was noticed, as well as inflammatory infiltrate associated to areas of necrosis.

Light microscopy histopathological study of renal structures allowed performing the accurate diagnosis of AKI induced by I/R and kanamycin nephrotoxicity.

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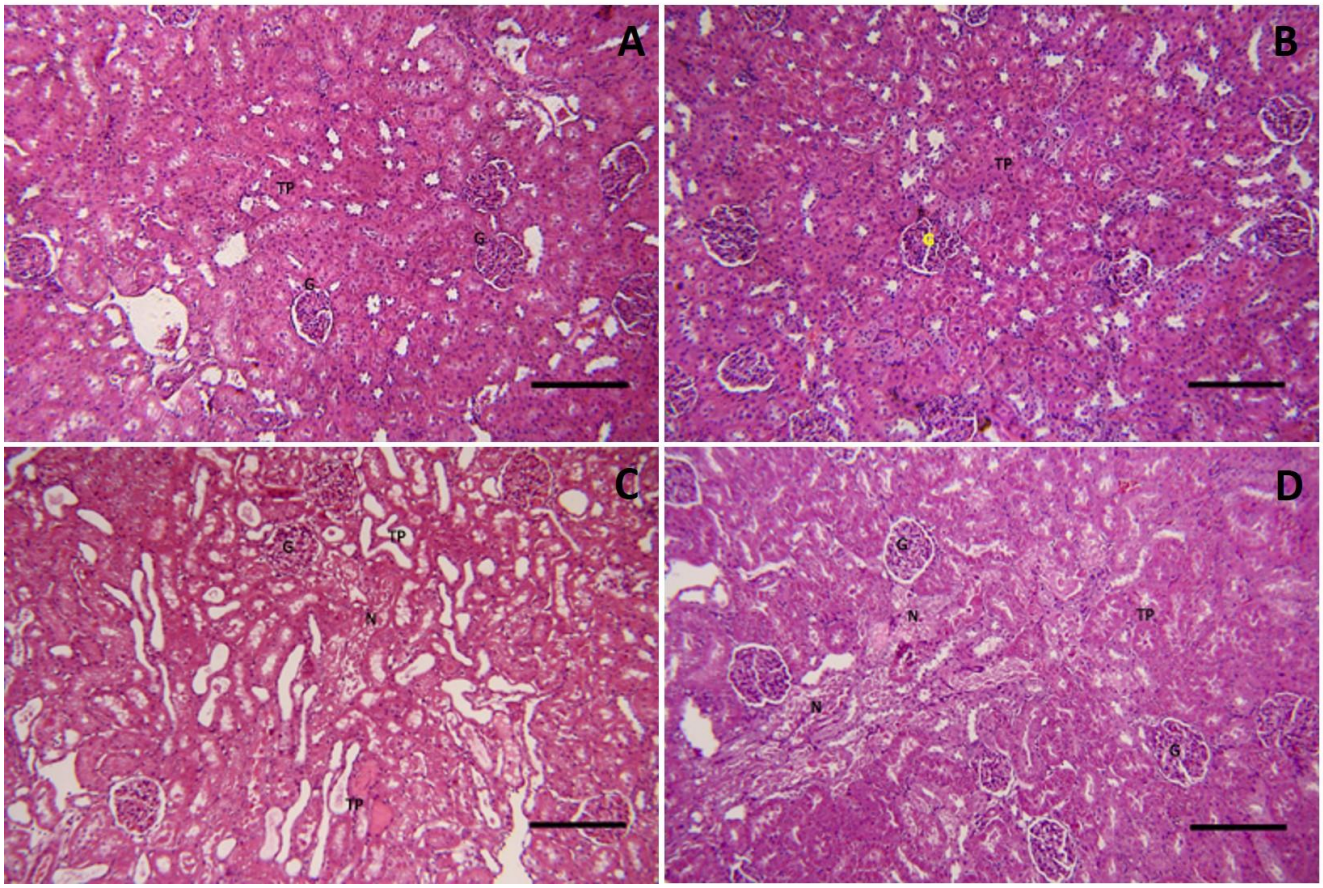

Figure 1. Wistar rats´ renal cortex. Hematoxylin/eosin staining. A) Negative control group (Ischemia/Reperfusion model), B) Negative control group (Kanamycin nephrotoxicity model), C) Ischemia/Reperfusion group, D) Kanamycin nephrotoxicity group. Glomeruli (G), Proximal tubule (TP), necrosis areas (N). Bars = 50 µm.

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