Study of Serum and Tissues Angiotensin Converting Enzyme (ACE) Activity in Rat with Gentamicin Induced Renal Toxicity

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ABSTRACT

The angiotensin I-converting enzyme (ACE) converts the inactive angiotensin I molecule to the active angiotensin II. ACE is rich in epithelium, endothelium, and neuroepithelial cells and it found largely on the brush border of intestine and kidney proximal tubules. ACE also presents in the serum. Some pulmonary and renal toxic drugs change the serum and tissue ACE contents. In this research ACE activity was studied in rats with gentamicin induced renal toxicity. Rats were sacrificed 1, 3, 5 and 7 days after gentamicin injection. ACE activity was measured in serum, kidney and lung. These data were compared with normal saline-treated rats. Treatment of rats with gentamicin results in renal damage evidenced by proteinuria, polyuria, and decreased creatinine clearance. The damage to the kidney proximal tubule was evident by (a) the histological analysis at light microscopy and (b) the augmentation in the urinary excretion of N-acetyl-β-D-glucosaminidase (NAG). Kidney ACE activity decreased and lung and serum ACE activity didn't change until day 7. Then lung ACE activity increased significantly at day 7 and kidney and serum ACE activity increased, too. Blood pressure increased significantly on the day 7 that corresponded with the lung ACE activity increment. These data suggest that kidney ACE activity decrease are due to renal damage and an universal signal increases the ACE contents in the body probably to increase systemic blood pressure and subsequently increase glomerular filtration rate (GFR).

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