Amikacin (AK) is frequently used on the treatment of Gram-negative infections on neonates, but its usage is restricted because of nephrotoxicity. In this study, on neonatal rats, we aimed to investigate the effects of erythropoietin and vitamin E on AK induced nephrotoxicity. A total of 35 newborn Wistar Albino rats were divided into four groups: (1) injected with saline (serum physiological was administered to placebo controls), (2) injected with AK (1200 mg/kg), (3) injected with AK + vitamin E (150 mg/kg), (4) injected with AK + erythropoietin (EPO) (300 IU/kg/day). In renal tissue, AK levels were significantly high in all groups except the control. Tissue malondialdehyde (MDA) and nitric oxide (NO) levels were statistically higher in AK -treated group than the control. MDA and NO levels were significantly decreased with the administration of vitamin E and EPO. Glutathione peroxidase (GPX) levels were statistically low in AK group compared with the controls. The levels of GPX, in vitamin E group, were increased significantly. However, superoxide dismutase and catalase levels were not significantly different in none of the groups. Insulin-like growth factor-1 values in AK, EPO and vitamin E groups were significantly higher than the control group. Histomorphological changes such as tubular epithelial necrosis were seen in AK treated group. Histopathological improvements observed with EPO and vitamin E administration. AK nephrotoxicity is related to oxidative stress and is supported with biochemical and histopathological findings. Vitamin E and EPO, as antioxidants, can be useful renoprotective agents for ameliorating AK induced nephrotoxicity in neonates.

KEYWORDS:
Amikacin; erythropoietin; neonatal rats; oxidative injury; vitamin E

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