Taurine and calcium carbonate (Caco\textsubscript{3}) in gentamycin induced nephrotoxicity in albino rats

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Abstract

Gentamycin is antibiotic used against Gram –ve bacteria. It has nephrotoxic effect due to production of reactive oxygen species (ROS). The aim of this study is to determine the protective effect of Taurine and calcium carbonate in combination with gentamycin. This study was conducted on 24 animals which were classified into 4 groups, control (C group), Gentamycin treated group (G-group) which was injected by 80mg/kg/day intraperitoneal. Gentamycin and taurine treated group (G+T group) which received gentamycin as the pervious group and taurine in a dose of 7.5mg/kg/day. Calcium carbonate treated group (G+ Ca group) received caco\textsubscript{3} in a dose of 1 g/kg orally in combination with gentamycin in the same used dose. By the end of study biochemical analysis of BUN, creatinine, sodium and potassium were measured. Histopathological examination of the renal tissue was done to demonstrate necrosis of renal tissue. The results cleared nephrotoxic effect of gentamycin as BUN, creatinine and area of necrosis were increased significantly (p<0.05). The effect of both taurine and caco\textsubscript{3} was demonstrated but the protective effect of caco\textsubscript{3} is more than taurine.

Key words: Gentamycin, taurine, caco\textsubscript{3}
Introduction

Gentamycin an aminoglycoside antibiotic used for treatment of severe threatening infections caused by Gram -ve and gram +ve bacteria. One serious limitation to its use is that, it can cause ototoxicity and nephrotoxicity (1). It has been estimated that up to 30% of patients treated with gentamycin for more than 7 days show some signs of renal impairment (2). The mechanisms involved in gentamycin induced nephrotoxicity remains unclear but different theories are suspected. It has been shown that GM exerts its adverse renal effect by generation of ROS, which results in sever tissue Damage (3). ROS directly act on cell components, including lipids, proteins, and DNA, destroying their structure and cause Peroxidation of membrane lipids (4). That is supported by increased MDA levels, one of the products of lipid peroxidation. (5, 6)

Gentamicin induces lysosomal phospholipidosis. Inhibition of lysosomal phospholipases, subsequent accumulation of phospholipids, and the formation of lysosomal myeloid bodies have been implicated as direct mechanisms of nephrotoxicity (7). Furthermore, acute tubular necrosis, glomerular damage and renal inflammation are the major events implicated in gentamicin nephrotoxicity (8). The antioxidants effect in either preventing or mitigating gentamycin induced nephrotoxicity has detected (9). The role of renal mitochondria on protection against GM nephrotoxicity has detected (10).

Taurine is the major intracellular free β-amino acid present in most mammalian tissues. It is present naturally in food, especially in seafood and meat. It plays various important physiological roles including osmoregulation, bile acid conjugation, and modulation of CNS function and cell proliferation. (11). The beneficial effects of taurine as an antioxidant in biological systems have been attributed to its ability to stabilize bio-membranes, scavenge ROS to form taurine chloramine (12). Taurine chloramine has been shown to serve as an oxidant reservoir, exhibiting delayed oxidant effects or acting at a distant site (13). This phenomenon is particularly significant in phagocytes, which are a source of taurine-related antioxidants and are prevalent in an early phase of inflammation in the glomerulus and tubules (14).

Since gentamycin induces nephrotoxicity by plasma and subcellular membrane damage which appear to be critical pathogenic events. This might involve competitive displacement of Ca^{2+} from anionic phospholipids at the plasma and organelle membrane
level, decrease in Na–K–ATPase, adenyl cyclase, mitochondrial function and ATP production, protein synthesis, solute reabsorption, and overall cellular function \(^{(15)}\). The effect of Ca\(^{2+}\) on gentamicin nephrotoxicity may relate to its ability to inhibit critical gentamicin-renal membrane interactions.