

Vitamin D attenuates gentamicin-induced acute renal damage via prevention of oxidative stress and DNA damage

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Abstract

Background: Despite being one of the most nephrotoxic drugs, gentamicin (GM) remains a mainstay as a first-choice agent in a vast variety of clinical situations owing to its superlative efficiency as a broad-spectrum antibiotic in treating several life-threatening bacterial infections. This urgently calls for the need for in-depth analysis of the mechanisms governing GM-induced nephrotoxicity and entails the necessity of presenting novel protective agents capable of ameliorating those renal deleterious effects. The reactive oxygen species and redox-sensitive transcription factors in GM-induced nephrotoxicity have recently called attention. **Purpose:** This study has been designed to shed light on the possible mechanisms of GM-induced nephrotoxicity and to provide a consensus set of histopathological, immunohistochemical, genetic and biochemical parameters elucidating the protective role of vitamin D against this nephrotoxicity. **Methods:** Twenty-four adult male albino rats were equally divided into four groups: group I (control group), group II (GM), group III (GM þ vitamin D) and group IV (vitamin D only). Kidney function tests, histopathological examination, gene expression of nuclear factor 2, nuclear factor kappa beta (NF-kB) and western blot of NF-kB p65, assessment of glutathione peroxidase and nicotinamide adenine dinucleotide phosphate oxidase (NADPH) oxidase by ELISA, as well as immunohistochemical evaluation of inducible nitric oxide, malondialdehyde, 8-hydroxy 2 deoxyguanine and vitamin D receptor, have been carried out. **Results:** The kidney function deterioration, tissue oxidative stress development and the histopathological changes induced by GM were significantly attenuated by vitamin D administration. **Conclusion:** Vitamin D attenuates GM nephrotoxicity through its antioxidant properties and prevention of DNA damage.