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Tenofovir-induced nephrotoxicity: Incidence, mechanism, risk factors, prognosis and proposed agents for prevention

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Objective: In this study, we reviewed the data regarding epidemiology, risk factors, pathogenesis and outcome of Tenofovir-induced nephrotoxicity, and discussed current and future approaches for prevention.

Method: The data were collected by searching Scopus, PubMed, Medline, Science direct, Clinical trials and Cochrane database systematic reviews.

Results & Conclusions: Several predisposing factors including elevated baseline SCr, concomitant nephrotoxic medications, low body weight, advanced age, Tenofovir disoproxil fumarate (TDF) dose and duration of treatment and lower CD4 cell count were identified as risk factors for the development of TDF-induced nephrotoxicity. Cellular accumulation through increased entry from the human organic anion transporters and decreased efflux into the tubular lumen is the main mechanism of nucleotide analog antiviral-induced nephrotoxicity. Renal function assessment and monitoring at baseline and during TDF treatment are the main approaches to prevention of TDF-induced nephrotoxicity. Rosiglitazone may be helpful in patients presenting with TDF-induced nephrotoxicity. Pretreatment with melatonin prevented all known histological changes in proximal tubular mitochondria induced by TDF. Use of antioxidants with mitochondrial-targeted properties such as MitoQ or Mito-CP may prevent proximal tubular mitochondrial against TDF damage. Vitamin E, ebselen, lipoic acid, plastoquinone, nitroxides, SOD enzyme mimetics, Szeto-Schiller peptides, and quercetin are other potential agents for prevention of TDF-induced nephrotoxicity. However, data regarding the effectiveness of nephroprotective agents against TDF-induced nephrotoxicity are not conclusive. Before extrapolation of the preclinical evidence to clinical practice, this evidence should be confirmed in future human studies.

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