In spite of longstanding side effects, cyclosporine inhibitors (CNI) stay a cornerstone of immunosuppressive therapy in kidney transplantation, autoimmune disorders and various glomerulopathies (1,2). A maximum of efficiency with a minimum of side effects is the eventual goal of immunosuppressive treatment with this drug. These drugs are powerful immunosuppressive substances with associated long-term renal toxicity intermediated by renal tubular epithelial cell damage and arterial and renal arteriolar vasoconstriction (1-3). Their chronic administration has also other side effects, consisting hyperlipidemia, and hypertension too. Thus, prompt recognition of CNI-induced acute renal toxicity is necessary for stop or minimize renal damage, and timely detection of chronic renal toxicity is serious for discontinuing the drug and preventing irreversible renal damage (2-4). Monitoring of calcineurin inhibitor therapy is frequently handled by blood trough levels, pharmacokinetics like assessment of 2-hour peak levels, or through various areas under the curve measurements to reach a balancing safety and efficiency. Numerous studies have detected that, cyclosporine A renal toxicity has been accompanied by enhanced reactive oxygen species production in renal tubular and glomerular cells. Numerous studies have detected that, cyclosporine A renal toxicity has been accompanied by enhanced reactive oxygen species production in renal tubular and glomerular cells.

Core tip: Numerous studies have detected that, cyclosporine A renal toxicity has been accompanied by enhanced reactive oxygen species production in renal tubular and glomerular cells.

Keywords: Cyclosporine A, Nephrotoxicity, Herbal drugs, Cyclosporine inhibitors, Thrombotic microangiopathy, Vasoconstriction

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