Introduction

Free radicales may cause lipid peroxidation and damage macromolecules and celluler structure of the organism e.g endothelium and erythrocytes. (Ceballos et al ., 1996).

Cells have developed antioxidant defense to prevent free radical injury including superoxide dismutase (SOD) and glutathione peroxidase (GPX) (Zima et al ., 1996).

Oxidative damage due to free radical production is increased in uremic patients and has been suggested as a possible factor contributing to the anemia of CRF, the pathogenesis of atherosclerosis and increased susceptibility to infection (Mc Grath et al ., 1995).

Shurtz et al ., (1995) stated that the activity of antioxidant enzymes (catalase, superoxide dismutase and glutathione peroxidase) is reduced in uremic patients.

Increased in local production of reactive oxygen species and reduction in antioxidant enzyme activities are responsible for the enhanced oxidant stress in many renal diseases e.g : Glomerulonephritis, lupus nephropathy, ischemic acute renal failure and chemically induced renal papillary necrosis (Nippon et al ., 1996).

It is difficult to quantitate free radicals because of their short half-lives and reactive nature. therefore indirect methods measuring products of lipid peroxidation . e.g malondialdehyde is prefered (Haklar et al., 1995).

Hassan et al ., (1997) reported that antioxidant therapy with vitamin C, vitamin A and vitamin E has a protective effect against oxidative stress associated with uremia.

Aim of the work

This work is designed to study the oxidant stress and the state of some intrinsic antioxidants enzymes in some renal diseases in order to shed light on its effect on the progression of these diseases and its relation to its complication. Also , we aim to know the possible protective effect of therapeutic antioxidants on the progress and complications of renal diseases.