Introduction

Diabetic nephropathy (DN) occurs as a consequence of interaction between metabolic and hemodynamic factors and characterized by macroalbuminuria that is, a urinary albumin excretion of more than 300 mg in a 24-hour collection or macroalbuminuria and abnormal renal function as represented by an abnormality in serum creatinine, calculated creatinine clearance, or glomerular filtration rate (GFR) ***(Zelmanovitz et al., 2009).***

Uric acid (UA) is produced by xanthine oxidase from xanthine and hypoxanthine, which in turn are produced from purine ***(Baillie et al., 2007).***

In humans, about 70% of daily UA disposal occurs via the kidneys decreased to 5-25% in chronic kidney disease (CKD), leads to hyperuricemia ***(Aringer and Graessler, 2008).***

UA in the blood is saturated at 6.0-6.8 mg/dL at ambient conditions, with the upper limit of solubility placed at 7 mg/dL. Urate is freely filtered at the glomerulus, reabsorbed, secreted, and then again reabsorbed in the proximal tubule ***(Enomoto et al., 2002).***

Although UA can act as an antioxidant, excess serum accumulation is often associated with cardiovascular disease. It is not known whether this is causative (e.g., by acting as a prooxidant)

or a protective reaction taking advantage of urate's antioxidant properties ***(Tausche et al., 2006).***

CKD, also known as chronic renal disease, is a progressive loss of renal function over a period of months or years ***(Levin et al., 2008).***The most common causes of CKD are DN, hypertension, and glomerulonephritis ***(Tonelli et al., 2006).***

Increased UA levels were associated with increased risk of the development of hypertension and cardiovascular disease ***(Fang et al., 2000).*** Increased UA levels also have a pathogenic role in the progression of CKD. Potential mechanisms include endothelial dysfunction, vascular smooth cell proliferation, increased synthesis of interleukin 6, insulin resistance, and impaired endothelial nitric oxide (eNO) production ***(Siu et al., 2006).***