

ASSESSMENT OF URIC ACID IN PATIENTS OF END-STAGE RENAL DISEASE WITH HYPERTENSION AND DIABETIC NEPHROPATHY AND THE RISK OF CARDIOVASCULAR DISEASES

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ABSTRACT : The relationship of hyperuricemia to kidney disease, diabetes, hypertension and the risk of cardiovascular diseases remain controversial. The aim of this study is to evaluate the use of uric acid (UA) levels to find the higher risk of cardiovascular disease (CVD) in patients with end stage renal disease that have diabetic nephropathy (DN), nephropathy with hypertension (NH) and patients with both diabetic nephropathy with hypertension (DNH). This study deals with 115 patients with end-stage renal disease under hemodialysis sub-grouped into 35 patients with (DN), 40 patients with (NH), and 40 patients with (DNH). Some biochemical parameters were determined in the serum of all participants such as HbA1c, fasting blood glucose (FBG), UA, urea, serum creatinine, total serum protein, calcium, phosphate, albumin, and globin levels. The present study revealed a significant increase ($P < 0.05$) in HbA1c, FBG, urea and creatinine in DN and DNH patients compared to NH group. However, non-significant difference was found in total serum protein, serum albumin, globulin, calcium, and phosphate levels between the groups. A positive correlation was found between UA level with FBG, HbA1c and creatinine in DN and DNH groups in comparison to NH group. Levels of UA can be considered as a reliable marker, which is less expensive and helps clinicians in controlling the progression to microvascular complications. The early detection of any complication and adopting the appropriate treatment to reduce the risk of CVD can reduce morbidity and mortality.

Key words : Hyperuricemia, diabetes mellitus, hypertension, cardiovascular diseases, end stage renal disease.

INTRODUCTION

End-stage renal disease (ESRD) is the final stage of chronic kidney disease where the kidney fails to remove the final products of metabolic pathways because of the decreased rate of glomerular filtration (GFR). This stage is caused by many factors included genetic or oxidative stress (Bellomo *et al*, 2004; Yun *et al*, 2014; Xue *et al*, 2014).

Many diseases may cause or are associated with chronic kidney diseases such as smoking, dyslipidemia, chronic inflammation, hypertension, proteinuria, and diabetes mellitus (DM). These diseases may develop to cause CVD (Mikolasevic *et al*, 2017; Yacoub *et al*, 2010; Tsuruya *et al*, 2014).

Structural kidney abnormalities are characteristic of DM although many risk factors are associated with both DM and DN; the explanation of this association is still unrevealed. The progression of DN may take a long period before the symptoms appear or increased albuminuria as a clinical DN and loss of kidney functions. Therefore, search for good biomarkers is of important

before the onset appearance of DN symptoms or earlier therapy before renal function decline (Lewis and Maxwell, 2014).

Many studies linked the elevation of uric acid (UA) in renal disease progression and diabetes complications and they considered it as a potential target (Liu *et al*, 2018; Lytvyn *et al*, 2015). The deleterious effects of UA on blood pressure and renal function has been demonstrated even when baseline UA levels are within the normal range. The UA activates the renin-angiotensin-aldosterone system (RAAS), increases oxidative stress, and promotes inflammation (Filiopoulos *et al*, 2012). Consequently, higher UA levels are associated with metabolic abnormalities, cardiovascular disease and kidney dysfunction (Lewis and Maxwell, 2014; Sochett *et al*, 2006; Stanton, 2014).

The UA has a dual role in oxidative stress status; it is considered an antioxidant since it scavenges some free radicals such as superoxide anion and hydroxyl radical. In another hand, UA plays a role in vascular cells of smooth muscle as pro-oxidant that lead to increased oxidative status (Yu *et al*, 2010). This study aimed to