

**Research Article****Comparison of Serum Uric Acid Level and Coronary Artery Calcium Score**Fatih Demircan<sup>1\*</sup>, Ramazan Ulu<sup>2</sup>, Ali Gürel<sup>2</sup>, Nevzat Gözel<sup>3</sup>, Banu Küçükürüm<sup>4</sup>, Refik Demirtunç<sup>1</sup><sup>1</sup> Haydarpasa Training Hospital Department of Internal Medicine, Istanbul<sup>2</sup> Firat Medical Faculty Department of Nephrology, Elazig<sup>3</sup> Firat Medical Faculty Department of Internal Medicine, Elazig<sup>4</sup> Siyami Ersek Training Hospital Department of Radiology, Istanbul**\*Corresponding author**

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**Abstract:** The aim of the study was to evaluate the interrelation between serum uric acid and artery calcification in asymptomatic coronary artery disease subjects. Dialysis patients display an increased mortality which is associated with cardiovascular calcifications. The coronary artery calcification (CAC) score as determined by multi-detector computed tomography is known to predict coronary artery disease. Serum BUN, creatinine, LDL cholesterol, uric acid, AST, ALT, LDH, sodium, potassium, chlorine, calcium, phosphorus, albumin levels and coronary calcium score results executed with multislice CT were noted to these forms. Mean serum uric acid level of patients was found to be 6.06. Female patients' mean calcium score was 220.58 (medium- high risk of ischemia), and the mean calcium score of male patients was 278.83 (medium-high risk of ischemia). In conclusion, in our cohort of haemodialysis patients, There was a no correlation between SUA level and CAC.**Keywords:** Serum uric acid, coronary artery calcification (CAC) score, Haemodialysis, Multi-detector computed tomography

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**INTRODUCTION**

Many studies have shown that serum uric acid (SUA) level is an important and independent risk factor in the development of cardiovascular diseases. This cardiovascular risk associated with SUA has been explained by several mechanisms [1, 2]. Important relationships have been reported between SUA levels, various inflammatory markers, oxidative stress, and atherosclerosis markers such as endothelial dysfunction [3-6]. Serum uric acid is a useful biomarker for mortality and an indicator of a poor prognosis in high-risk patients with several cardiovascular diseases [7, 8]. SUA plays a important role in the pathogenesis of cardiovascular diseases affecting xanthine oxidase pathway that causes active oxygen species generation with deterioration of cells membranes [9]. It has been suggested that SUA could be a marker of oxidative damage [10]. However, neither the Framingham Heart Study nor the Atherosclerosis Risk in Communities (ARIC) Study found any such associations, and a recent analysis of the ARIC database demonstrated that although higher serum uric acid concentrations were associated with increased mortality in the non-CKD population even after adjustment for metabolic syndrome, the presence of CKD weakened the association [11-13]. The inconsistency of the data was confirmed by a recently published study of a large

historical cohort of a national insurance provider that documented a stronger association between serum uric acid concentrations and cardiovascular morbidity in patients with severely decreased GFR [14]. Thus, the question of whether uric acid has a pathogenic role in the onset and progression of CKD and CVD remains unanswered.

CAC had been recognised as a strong predictor of cardiovascular disease progression and patient survival in patients without kidney disease as arterial calcium deposition is involved in creation and evolution of atherosclerotic plaque [15-17]. Computed tomography has been used to quantify CAC [18]. The calculated CAC score is important for determination of both obstructive and non-obstructive coronary artery disease [18].

Although CAC is frequently and considerably found among HD patients, the underlying mechanisms for the progression of vascular calcification in these patients have not fully elucidated. Several factors including calcium (Ca) and/or phosphate (Pi) disorder, fetuin-A, and differentiation of vascular smooth muscle cells to osteoblastic cells have been reported as the mechanisms of CAC in HD patients [19-22].

Therefore, the aim of this retrospective study was to investigate whether SUA independently risk factors for CAC at HD patients beyond traditional cardiovascular risk factors.

## MATERIALS AND METHODS

Thirty patients with chronic renal failure who were followed by polyclinics of Haydarpasa Numune Training and Research Hospital between the years 2005-2007 were included in this retrospective study. 14 patients were male and 16 were female. The mean age of patients was 52 years.

Patients who had attacks of angina during hemodialysis, with electrocardiographic changes, elderly patients who have multiple risk factors and patients who require advanced cardiological assessment were included in this study.

Patients under 18 years of age, despite of renal failure without regular dialysis patients, gout, malignancy, and patients with known coronary artery disease were excluded from the study.

In order to standardize data a form was filled for each patient in this study. Age, gender, family history of coronary artery disease, the presence of concomitant disease (e.g. hypertension, diabetes), serum BUN, creatinine, LDL cholesterol, uric acid, AST, ALT, LDH, sodium, potassium, chlorine, calcium, phosphorus, albumin levels and coronary calcium score results executed with multislice CT were noted to these forms. In addition, findings on physical examination of patients, arterial blood pressure (mmHg), pulse rate (per minute), ECG samples were analyzed and added to the form.

For coronary calcium score assessment, 16- detector multidetector computed tomography axial volumetric slices with 1 mm thickness without contrast medium have been studied in synchronization with the ECG. Left main coronary artery, left anterior descending artery, circumflex artery and right coronary artery were evaluated with Aganston score by using "Siemens calcium scoring Software". According to this scoring system, patients with low risk do not require further cardiological examination, but moderate or high- risk patients require advanced cardiological assessment such as classic angiography.

## Statistical Analysis

In order to evaluate the findings obtained in this study, NCSS 2007 & PASS 2008 Statistical Software (Utah, USA) statistical analysis programmes were used. In addition to descriptive statistical methods (mean, standard deviation, frequency), chi- square test was used for comparison of qualitative data. Spearman's correlation test was used to examine the correlation

between the parameters. Confidence interval was 95% and p value <0.05 was considered significant.

## RESULTS

Thirty patients (16 (53.3%) women and 14 (46.7%) men) with chronic renal failure who were followed by polyclinics of Haydarpasa Numune Training and Research Hospital between the years 2005- 2007 were included in our study. Biochemical parameters of patients on the same term have been evaluated. The mean age of patients was  $52.76 \pm 16.48$ . Female patients' mean calcium score was 220.58 (medium- high risk of ischemia), and the mean calcium score of male patients was 278.83 (medium-high risk of ischemia). The mean calcium scores according to gender were not significant different ( $p > 0.05$ ). Coronary calcium scores were found to be at moderate to high risk groups for both men and women.

The mean age of patients was 52 years. There was no significant correlation between calcium score and age ( $p > 0.05$ ). Mean serum uric acid level of patients was found to be 6.06. In our study, there was no significant correlation between calcium score and serum uric acid levels ( $p > 0.05$ ). Mean LDL level was 101.8 mg/dl. There was no significant correlation between calcium score and serum LDL levels ( $p > 0.05$ ).

## DISCUSSION

The results of our study didn't show the relationship between serum uric acid level and CAC. We also failed to prove the association between dyslipidemia and CAC. Elevated serum uric acid level has been associated with both the presence of intrarenal arteriolar lesions [23, 24] and with an increased risk for cardiovascular mortality in subjects with CKD [25, 26]. We support a hypothesis that serum uric acid can contribute to influence of inflammatory activation, metabolic disorders, and calcium deposition in plaques and in vasculature. The evidence of microinflammation as an independent risk factor for the progression of CAC in HD patients in a prospective cohort study was first and only reported by Jung *et al.* [27].

Although it is reported that impaired renal function has association with increasing coronary calcium score, it still unclear whether this association is independent from traditional cardiovascular risk factors [28]. Some studies found an independent association [29-31], while others studies did not [32-34]. The pathophysiology of coronary artery disease in chronic kidney disease (CKD) is multifactorial including, next to traditional risk factors like older age, diabetes mellitus, systolic hypertension, left ventricular hypertrophy and hyperlipidemia, chronic inflammation, oxidative stress, abnormal bone and mineral metabolism, hyperhomocysteinemia, malnutrition and anemia [35, 36]. In conclusion, this study showed that CAC is commonly problem of HD patients and our

findings didn't show any relationship between SUA and CAC.

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