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Predictive value of serum uric acid and coronary artery calcium scoring for ischemic heart disease

Mohamed Ghalib Zakari[1]

[1] *Department of Medicine ,College of Medicine, University of Tikrit, Tikrit, Iraq*

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Corresponding author E mail:
Dr_mohamedghalib@tu.edu.iq

ABSTRACT

Low-dose computer tomography (CT) scanning of the heart is a non-invasive method that provides quantitative information of the calcification of the coronary vessels. High serum uric acid (SUA) level is known to be associated with various cardiovascular (CVD) risk factors and diseases such as hypertension, diabetes mellitus, and metabolic syndrome. Recently, some researches show that higher SUA levels are linked with an increased risk of hospitalizations related to chronic kidney diseases, mortality, and also CVDs .Its role as a risk factor for cardiovascular disease is studied extensively; however, whether the association is independent of conventional risk factors is controversial. The study aimed to value of CT coronary artery calcium scoring in hyperuricemic patients as a predictive risk for development of ischemic heart disease. The current study was conducted on at Ibn-Alnafees teaching Hospital, from 1st of November 2023 to the end of May 2024. The study included 168 patients, 118 male patients and 50 female patients. All patients underwent a proper history taking and thorough physical examination and were sent for the biochemical investigations. Electrocardiogram done for all patients. Echo study done for those Electrocardiography was not giving exclusive diagnosis. CT coronary artery calcium score and CT Angiography were done for all patient diagnosed as coronary artery disease. The study for coronary artery disease patients shows There is a strong correlation between hyperuricemic patients, coronary artery calcium and lipid profile in patient with ischemic heart disease and there was also a significant increase of Calcium score which is approved by non-contrast CT Angiography and then confirmed this strong correlation by contrast CT Angiography that shows the luminal arterial defects in diabetic patients group.

INTRODUCTION

Coronary Artery Disease (CAD), in other term, coronary heart disease (CHD), is one of the most killers and common silent diseases contributing high morbidity and mortality worldwide ,In the United States of America CAD is the main cause of death in adults; responsible for about one-third of all dead people, particularly, above 35 years of age [1],Atherosclerosis is a diffuse disease that affects many arteries of the body not just the coronary arteries. In the early stages, it causes changes in the walls of the arteries with increases in cholesterol content and scar tissue, while in the later stages, it can causes plaques that thicken the wall of the artery and in some cases it cause narrowing of the center of the artery so that the flow of blood is gradually reduced. At this stage, calcium is generally present in the plaques [2]. Coronary artery calcium (CAC) is typically present in direct proportion to the overall extent of atherosclerosis, although typically only a minority (approximately 20%) of plaque is calcified [3].

The relative risk associated with coronary calcification is greater than that associated with established factors, such as smoking, hypertension and diabetes mellitus[4]. The progression of coronary artery calcification is associated with a higher incidence of coronary events, even in those people who are asymptomatic at the time of initial scanning [5]. Thus, the presence of coronary artery calcification is not only indicative of atheromatous plaque disease, but its progression may correspond with cardiovascular event rates. The increasing frequency of hyperuricemia and gout are most likely caused by westernized lifestyle and environment [6]. Although the physiological solubility of uric acid occurs at 6.4 mg/dl, the presence of uric acid-binding proteins increases this solubility to 7.0 mg/dl before reaching a supersaturated state. For this reason, hyperuricemia occurs when the serum level of uric acid exceeds 7.0 mg/dl, at which point it starts to crystalize within the human body. However, an increase in the serum uric acid level is considered to accompany the increased risk of disease associated with adult lifestyle habits (lifestyle diseases) even when the serum level of uric acid is ≤7.0 mg/dl. In women, especially, the disease risk increases at even lower serum uric acid levels compared to men and requires more attention.

Hyperuricemia often accompanies metabolic syndrome, hypertension, diabetes, dyslipidemia, chronic renal disease, and obesity, and the serum uric acid level is known to vary significantly depending on meals, lifestyle, gender, and previous use of diuretics [7]. Based on these facts, it is believed that the uric acid level only partly reflects the lifestyle origins of the disease, and it merely serves as a marker of cardiovascular disease. Furthermore, since female hormones lower the serum uric acid levels, they tend to increase after menopause, and the evaluation of uric acid becomes more difficult. The number of confounding factors involved in evaluating serum uric acid levels complicates the sole analysis of uric acid; indeed, intervention tests that focused only on uric acid are rare. However, due to the advanced research methods and the discovery of new antihyperuricemic drugs such as febuxostat, uric acid studies have been attracting attention. Recently, Degli Esposti showed that higher SUA levels are linked with an increased risk of hospitalizations related to chronic kidney diseases, mortality, and also CVDs [8]. Serum uric acid plays a pivotal role in the pathogenesis of cardiovascular diseases affecting

xanthine oxidase pathway that contributes to the production of active oxygen species generation with deterioration of cells membranes [9]. Reactive oxygen species contribute to vascular oxidative stress and endothelial dysfunction, which are associated with the risk of atherosclerosis, damages of both cardiomyocytes and vascular endothelium inducing disturbances of myocardial contractility and vasoconstriction also [10]. Some studies shows the independent positive correlation between uric acid and the intensity of arterial wave reflection in the hypertensive but not normotensive subjects [11] .The availability of a noninvasive technique to detect coronary calcification makes it possible to obtain direct information on the presence of atherosclerosis in the coronary arteries. The sum of the area and density weightings across the coronary arteries is the unit less calcium score originally defined by Agatston and colleagues.[12] Other quantification methods are available, including a calcium volume determination and mass score .As a non-invasive measure of overall coronary artery disease burden CAC testing is a clinically useful screening test for coronary atherosclerosis [13].CT easily identified the coronary calcium because the roentgen graphic attenuation of calcium is much higher compared with that of the surrounding tissues [14] . Histologic studies have shown that a CT tissue density of greater than or equal to 130 HU is highly correlated with calcified coronary plaques [15].

PATIENTS AND METHODS

The study included 168 patients who visited the internal and cardiac clinic at Ibn al-Nafis Hospital. The study include 118 male and 50 female. They were selected according to the clinical signs and symptoms (all patients selected were suffering from chest pain), whether they had a history of illness or risk factors or without. Blood tests were conducted as well as the work of ECG for all patients. Some patients were excluded because of the lack of the required standards for study (non-cardiac, musculoskeletal, Gastro esophageal reflux, pain in the thoracic vertebrae). These selected patients were divided into many groups according to the risk factors.

All patients underwent a case history questionnaire included sociodemographic data (age, gender, smoking, wieght) and medical history (hypertension, diabetes mellitus), and were sent for the biochemical investigations venous sampling of 5 cc that was send for biochemical investigations for determination of fasting blood sugar, high-density lipoprotein (HDL) (fasting patient), Triglycerides (TGs)(fasting patient), LDL and serum VLDL. Determination of serum uric acid. Measuring of weight for BMI, blood pressure and Electrocardiography(ECG) done for all patient. Treadmill test (TMT)and Echocardiographic study done for those ECG was not giving exclusive diagnosis. CT coronary artery calcium score and CT angiography was done for all patient diagnosed as Coronary artery disease. The exclusion criteria include patient with history of coronary artery bypass graft or prior stent placement, and patient unable sustain a breath holds for at least 15 to 20 seconds. (Because CAC measurement required breathe holding for at least 15 sec).and patient with history of renal impairment(contrast induced nephrotoxicity) patient with history of any allergic reaction to contrast agent. CT coronary angiography was performed with a 64-slice scanner (Aquillon 64, V4.51 ER 010, Toshiba Medical Systems, Tochigi,

Japan) with retrospective ECG gating. Before Multi-Slice CT angiography, a non-contrast CT was acquired to measure calcium score according to the Agatston method. Slice thickness was 0.3- 0.5 mm.

STATISTICAL ANALYSIS

Data were introduced in to personal computer (IBM SPSS V. 23) were used in statistical management of the collected data . Descriptive statistics were displayed using tables .Analytic statistics were presented through usage of Chi square test , Fisher Exact and Spearman correlation.

Kappa agreement test was used to find out significance of association between CAC and the diseased arteries and the elevated level of SUA P. value < 0.001 was considered as significant level marker

CONFLICT OF INTEREST

There are no known conflicts of interest, financial or otherwise for the author of this manuscript which would interfere with the integrity of this research.

STATEMENT OF ETHICS

Informed consent was obtained from all patients for being included in the study. Ethical approval was obtained from the institutional ethical and scientific committee before commencing this study

RESULTS

This comparative cross sectional study included 168 patients, with 96 (57.1 %) of study sample suffer from IHD (case group, and 72 (42.9 %) of them with no history or sign or symptoms of suggestive IHD (control group). Regarding CAC score , 102 (60.7 %) of studied sample had high risk CAC score and 100 (59.5 %) of studied sample had abnormal CTA . The age of 96 subject (57.1 %) aged 55 years or more , while 72 (42.9 %) aged less than 55 years .Male formed (70.2%) of study group . Regarding weight status (32.7 %) of the studied sample had elevated BMI (BMI more than 25) . Smoking history was declared by (23.8 %) , (40.5 %) had HT , (29.8 %) had DM , hyperuricemia was found in (42.3 %) , and elevated TG was found in (37.5 %) , decrease HDL was found in (36.9 %), finally elevated of both LDL and VLDL were found (36.9 %) and (36.3 %) respectively , as shown in table (1).

Table (2) shows the association between age, gender, weight and smoking with studied group, CAC and CTA using. It revealed that significant association were found between gender, weight and smoking with studied group, CAC and CTA using , apart from the age that are not associated with group that getting IHD , and the gender that not associated with each of the presented variable (CAC , CTA and group) . that are represented by p – value =<0.001.

Table(3) shows the association between chronic diseases (hypertension and DM) with studied group, CAC and CTA. It revealed that significant association were found between chronic diseases (hypertension and DM) with studied group, CAC and CTA that are represented by p – value =<0.001.

Table (4) shows the association between hyperuricemia and studied group, CAC and CTA. It revealed that significant association were found between hyperuricemia and studied group, CAC and CTA that are represented by p – value =<0.001.

Table(5) shows the association between lipid profile measurements and studied group, CAC and CTA. It revealed that significant association were found between, lipid profile (TG , HDL , LDL ,

VLDL) and studied group, CAC and CTA. that are represented by p – value =<0.001.

Table (6) Regarding agreement between CTA and CAC in diagnosis of IHD < it was found that 85 % of cases had high risk CAC , at the same time 83.3% of patients had abnormal CTA result , while 75 % of patients had normal CTA in correspond with 77.2 % had low risk CAC . this mean that kappa agreement between CTA ana CAC was 0.603 , p value 0.001 (high significant but intermediate agreement).

Table (7) Regarding agreement between SUA and CAC in diagnosis of IHD , it was found that 94.2 % of cases had high risk CAC , at the same time 63.7 % of patients had high SUA , while 75 % of patients had normal SUA in correspond with 94 % had low risk CAC . this mean that kappa agreement between SUA and CAC was 0.530 , p value 0.001 (high significant but acceptable agreement)

DISCUSSION

In order to prevent mistakes caused by the stenosis's advancement over time, CT scans and catheterization were carried out by the several operators in our study within a few days. But according to reports, there was no discernible difference between a mean of 18 months of delays and referral within a few days. (17).

Men's mean calcium scores in our study did not differ substantially from women's (p =0.119) between the IHD and Control groups. Many of other researches demonstrate a substantial rise in CAC with participant age (p 0.001) between groups. Numerous other researches have also demonstrated the relationship between high risk and low risk (18). The number of coronary arteries and the degree of stenosis discovered after coronary catheterization were found to be closely correlated with coronary calcium, according to numerous earlier researches (19). Many of the researches conducted over the past fifty years have demonstrated the significant gender variations in the presentation, diagnosis, and treatment of cardiovascular disease. When compared to age-matched males over 55, premenopausal women in particular have far reduced incidence of cardiovascular disease. However, there is still no clear explanation for the recognized disparities in cardiovascular disease between men and women. Over the past 50 years, many ideas have been proposed in an effort to explain these disparities. However, the gender difference cannot be explained by a single, comprehensive theory. Differences in vascular beds, hormonal factors, and lifestyle concerns are only a few of the many elements that have been demonstrated to individually contribute to observed disparities. These factors are likely the reason of these reported differences. In addition to the previously mentioned discrepancies, our research shows that men and women differ significantly in the location, distribution, and composition of intracoronary lesions, which may account for some of the observed variances in clinical presentation (20). Even in patients with low CAC scores, smoking and weight/obesity are strong, independent predictors of early atherosclerosis and high-risk plaque, according to research on Coronary Artery Calcium (CAC) and Coronary CTA (CCTA). Regarding weight, there is a high correlation between the examined group, CAC and CTA, and obesity or being overweight, which is consistent with the study of (21). Regarding smoking, the study found a strong correlation between the VLDL, TC, and calcium score, which explains how smoking has a significant impact on CAD by playing a significant role in the deposition of Ca++ in the

coronary arteries. Our findings are consistent with a study conducted by (22). Two well-known, complementary risk factors for cardiovascular disease (CVD) are diabetes mellitus (DM) and hypertension (HTN). Certain, progressive patterns of vascular damage are shown by their correlation with clinical outcomes, Coronary Artery Calcium (CAC), and Coronary Computed Tomography Angiography (CTA) results. The degree of coronary atherosclerosis as determined by CAC has previously been demonstrated to be predicted by hypertension. It was discovered that smoking and diabetes were better indicators of coronary atherosclerosis than hypertension. Additionally, a wealth of epidemiologic evidence has shown that hypertension is a separate risk factor for coronary atherosclerosis and subsequent cardiac events (23), However, compared to smoking and diabetes, it is a weaker predictor. Our results are consistent with previous research on coronary atherosclerosis, including a correlation with rising levels of obstructive CAD and a tendency toward connection with MACE occurrences. Many studies have been conducted on the relationship between hypertension and an increase in coronary atherosclerosis. The primary underlying pathophysiologic mechanism is thought to be mechanical and connected to pulse pressure (24). Increased cardiac events have been linked to wide pulse pressure. Increased systolic and pulse pressure both lead to endothelial dysfunction, which makes it easier for low density lipid cholesterol to enter the blood vessel wall and start the atherosclerotic process. Left ventricular hypertrophy (LVH), which has been linked to coronary atherosclerosis, myocardial infarction, arrhythmia, cardiac failure, or cardiac mortality, is another consequence of hypertension (25). Higher levels of subclinical atherosclerosis (measured by CAC) and coronary artery disease (measured by CTA) are substantially correlated with hyperuricemia. Even after controlling for traditional risk variables, elevated serum uric acid remains an independent biomarker for cardiovascular risk. The current data demonstrate that the prevalence of IHD was much higher in hyperuricemic patients (69.80%) than in normouricemic patients (2.80%), corroborating earlier research showing a link between elevated blood uric acid levels and CAD (26). These results highlight the possible involvement of uric acid in oxidative stress, inflammatory processes, and endothelial dysfunction—all of which are crucial in the development and course of atherosclerosis (27). He et al. (28) and Kim et al. (29) offer scientific proof that high uric acid levels cause vascular inflammation and endothelial dysfunction, two processes intimately linked to the onset and severity of CAD. Additionally, even after controlling for prevalent cardiovascular risk variables such age, gender, hypertension, diabetes mellitus, dyslipidemia, and smoking status, our analysis demonstrated that hyperuricemia remained an independent predictor of CAD. The results of Kuo et al. (30) and Akhtar et al. [31], who found hyperuricemia to be a substantial risk factor for CAD in their meta-analyses, are consistent with this independent predictive ability. Additionally, He et al. (28) shown that uric acid is a useful biomarker in cardiovascular risk stratification and that hyperuricemia significantly predicts cardiovascular mortality.

CONCLUSION

1. The physiological study for CAD patients shows a significant increase of Calcium score which is predicted

by non-contrast CTA and then confirmed this strong correlation by contrast CTA that shows the luminal arterial defects in hyperuricemic patients group.

2. Lipid profile and diabetes mellitus shows significant correlation with the Calcium score in CAD patient's also strong correlation with each other that explains the mechanism of the atherosclerosis process.
3. Apart from the age that are not associated with group that getting IHD
4. Gender that not associated with each (CAC, CTA and group)
5. kappa agreement between CTA and CAC was high significant but intermediate agreement and between SUA and CAC was high significant but acceptable agreement.

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32.

TABLES

Table 1. distribution of studied subjects according to essential variables

		N	%
Group	IHD	96	57.1%
	Control	72	42.9%
CAC	High risk	102	60.7%
	Low risl	66	39.3%
CTA	Abnormal	100	59.5%
	Normal	68	40.5%
Age	=>55 year	96	57.1%
	<55 year	72	42.9%
Gender	Male	118	70.2%
	Female	50	29.8%
Weight	Increased	55	32.7%
	Normal	113	67.3%
Smoking	Smoker	40	23.8%
	Not smoker	128	76.2%
HT	Hypertensive	68	40.5%
	Normotensive	100	59.5%
DM	DM	50	29.8%
	Not dm	118	70.2%
Uric acid	Hyperurecemic	69	38.7 %
	Normal UA	99	61.3%
TG	Increased	63	37.5%
	Normal	105	62.5%
HDL	Decreased	62	36.9%
	Normal	106	63.1%
LDL	Increased	62	36.9%
	Normal	106	63.1%
VLDL	Increased	61	36.3%
	Normal	107	63.7%

Table 2. association between age, gender, weight and smoking with studied group, CAC and CTA using Chi square test

		Group				CAC				CTA			
		IHD		Control		High risk		Low risk		Abnormal		Normal	
		N	%	N	%	N	%	N	%	N	%	N	%
Age	=>55 year	61	63.5 %	35	48.6 %	70	68.6 %	26	39.4 %	65	65.0 %	31	45.6%
	<55 year	35	36.5 %	37	51.4 %	32	31.4 %	40	60.6 %	35	35.0 %	37	54.4%
P value		.053				0.001				.013*			
Gender	Male	72	75.0 %	46	63.9 %	66	64.7 %	52	78.8 %	73	73.0 %	45	66.2%
	Female	24	25.0 %	26	36.1 %	36	35.3 %	14	21.2 %	27	27.0 %	23	33.8%

P value		.119				.051				.342			
Weight	Increased	45	46.9 %	10	13.9 %	40	39.2 %	15	22.7 %	44	44.0 %	11	16.2%
	Normal	51	53.1 %	62	86.1 %	62	60.8 %	51	77.3 %	56	56.0 %	57	83.8%
	P value	.000*				.026*				.000*			
Smoking	Smoker	35	36.5 %	5	6.9%	32	31.4 %	8	12.1 %	35	35.0 %	5	7.4%
	Not smoker	61	63.5 %	67	93.1 %	70	68.6 %	58	87.9 %	65	65.0 %	63	92.6%
		0.001				0.004				0.001			

Table 3. association between chronic diseases (hypertension and DM) with studied group, CAC and CTA using Chi square test

		Group				CAC				CTA			
		IHD		Control		High risk		Low risk		Abnormal		Normal	
		N	%	N	%	N	%	N	%	N	%	N	%
HT	Hypertensive	63	65.6%	5	6.9%	53	52.0%	15	22.7%	65	65.0%	3	4.4%
	Normotensive	33	34.4%	67	93.1%	49	48.0%	51	77.3%	35	35.0%	65	95.6%
		0.001				0.001				0.001			
DM	DM	44	45.8%	6	8.3%	40	39.2%	10	15.2%	47	47.0%	3	4.4%
	Not dm	52	54.2%	66	91.7%	62	60.8%	56	84.8%	53	53.0%	65	95.6%
P value		0.001				0.001				0.001			

Table 4. association between hyperuricemia and studied group, CAC and CTA using Chi square test

		Group				CAC				CTA			
		IHD		Control		High risk		Low risk		Abnormal		Normal	
		N	%	N	%	N	%	N	%	N	%	N	%
Uric acid	Hyperuricemia	67	69.80%	2	2.80%	65	63.70%	4	6.06%	67	67.00%	2	0.03%
	Normal UA	29	30.20%	70	97.20%	37	36.30%	62	93.94%	33	33.00%	66	97.06%
P value		0.001				0.001				0.001			

Table 5. association between lipid profile measurements and studied group, CAC and CTA using Chi square test

		Group				CAC				CTA			
		IHD		Control		High risk		Low risk		Abnormal		Normal	
		N	%	N	%	N	%	N	%	N	%	N	%
TG	Increased	57	59.4%	6	8.3%	46	45.1%	17	25.8%	56	56.0%	7	10.3%

	Normal	39	40.6%	66	91.7%	56	54.9%	49	74.2%	44	44.0%	61	89.7%
P value		.000*				.011*				.000*			
HDL	Decreased	57	59.4%	5	6.9%	45	44.1%	17	25.8%	55	55.0%	7	10.3%
	Normal	39	40.6%	67	93.1%	57	55.9%	49	74.2%	45	45.0%	61	89.7%
P value		.000*				.016*				.000*			
LDL	Increased	57	59.4%	5	6.9%	50	49.0%	12	18.2%	57	57.0%	5	7.4%
	Normal	39	40.6%	67	93.1%	52	51.0%	54	81.8%	43	43.0%	63	92.6%
P value		0.001				0.001				0.001			
VLDL	Increased	56	58.3%	5	6.9%	49	48.0%	12	18.2%	56	56.0%	5	7.4%
	Normal	40	41.7%	67	93.1%	53	52.0%	54	81.8%	44	44.0%	63	92.6%
P value		0.001				0.001				0.001			

*Table 6.cross tabulation of CTA * CAC with Kappa agreement*

		CAC		Total
		High risk	Low risk	
CTA	Abnormal	85	15	100
	Normal	17	51	68
Total		102	66	168
Kappa agreement =0.603, pv=0.001				

*Table 7. cross tabulation of SUA * CAC with Kappa agreement*

		CAC		Total
		High risk	Low risk	
Uric acid	Hyperurecemic	65	4	69
	Normal UA	37	62	99
Total		102	66	168
Kappa agreement =0.530, pv=0.001				