Original Research Article

Hyperuricemia and smoking correlation in Egyptianyoung adults with coronary artery disease:ObservationalStudy

LearningObjective:

HUA is significantly associated with the occurrence of CAD in non-smokers of young adults ≤ 35years old. Unexpectedly, HUA and smoking have a contrast; the existence of these two riskfactors in a patient is not remarkably associated with CAD occurrence compared to the statistical results of non-smokers.

Abstract

Background: Young adults, especially those under 35, are often overlooked; however, previous studies have shown that coronary artery disease (CAD) incidence increases rapidly. Previous studies proved many risk factors for CAD, such as hyperlipidemia, hypertension, diabetes, andsmoking. The present study ultimately aimed to figure out the correlation betweenHyperuricemia (HUA) and smokinginCAD Patients underthe ageof35.

Methods: This observational study included 100 young adults (18–35 years of age) suspected ofCAD. We used the Gensini Score system to ascertain coronary angiography outcome and CAD;based on the number of affected vessels,localization of the segment,and the stenosisgrade.
The Gensiniscore being>0indicated the presence of CAD.

Results: In young adults under 35, there was a significant correlation between HUA and CAD.The interaction between HUA and smoking had a contrast for CAD that statistically shown theexistence of CAD increased in patients who have hyperuricemia and non-smokers compared tosmokers.

Conclusions: By monitoring various parameters in young adults ≤ 35 years old. This studystrengthens the correlation between:1) hyperuricemia and the occurrence of CAD, 2) Smoking and the CAD.Furthermore, the reisaclear association between HUA and the occurrence of CAD in non-smokers.

Keywords: Hyperuricemia,Smoking,CoronaryArteryDisease.

Introduction:

Coronary artery disease is the most frequent reason for mortality in cardiovascular diseases.Young adults, especially those under 35, are often overlooked; however, previous studies haveshown that CAD incidence increases rapidly.Smoking and cigarette smoke are among thedominant risk factors for early or accelerated peripheral, coronary and cerebral atherosclerоticvascular disease. Kamceva et al.,[1] in a study to determine whether cigarette smoking, as a riskfactor for CAD, affects antioxidant status. As a consequence, it raised oxidative stress andreduced antioxidantprotection.

Hyperuricemia is linked with cardiovascular diseases (CVD), such as coronary artery disease(CAD), stroke, and hypertension[2], but the role of serum uric acid (SUA) as an independent riskfactor for CVD remains unclear. Many previous studies have shown that hyperuricemia iscommonly recorded in patients with CVD or at a high risk of CVD, such as hypertension, CAD,stroke, heartfailure,metabolicsyndrome,andperipheral vasculardisease[3].

This study aimed to determine; a. the association between hyperuricemia and coronary arterydisease. b. Studying the association between HUA and CAD in smokers and non-smokers anddarify the conflict effect between the risk factorsof CAD.

Methods:

StudyDesignandParticipants

This observational study was carried out at the cardiology department, Tanta UniversityHospital,afterapprovalfromEthicalCommitteeandobtainingwritteninformedconsentfr omeach patient. The study involved 100 patients from October 2019 till March 2020. Eligibilitycriteria included all young adults (18–35 years of age) suspected to have CAD who underwentcoronary angiography. The exclusion criteria were as follows: 1. Renal impairment (anestimated glomerular filtration rate [eGFR] < 60 mL/min/1.73 m2). 2. Active infections,autoimmune diseases, or neoplastic disease.3. Hepatic and hemolytic disorders. 4. Patientrefusal.

Procedures/StudyProtocol

A total of 100 patients went through a complete clinical record and accurate history taking.Thorough history taking with stress on age, sex, and history of risk factors for CAD, includinghypertension, diabetes mellitus, smoking habits, dyslipidemia,and family history of ischemicheart diseases. Blood pressure under 140/90 mmHg patients described as hypertension orhaving Medication for anti-hypertension [4]. Taking insulin or oral hypoglycemic agents was areason to consider the patient with diabetes. For patients with lack awareness about theirdiabeteshistory;weusedtheWHOcriteriafor2006diabeticdiagnosticasfollows:a.under126mg /dl for fasting blood glucose, and under 200 mg /dl for oral glucose tolerance or oral glucosetolerance test 2-hour plasma glucose ≥ 200 mg/dL. Smokerpatients were defined as a patientsmoking for six months or more. Suppose serum uric acid level was more than 7.0 mg/dl inmen and 6.0 for women, defined as hyperuricemia[5]. Coronary artery disease is common inpatientswithpositivefamily history.

AStandardtwelve-leadelectrocardiogramwasdoneforallpatientsandanalyzedforheartrate,rhythm, conduction abnormalities, ST-segment changes, and voltage criteria. By following theECHO recommendations of American society, we have calculated the Left ventricular ejectionfraction; besides,mitral pulsed-wave Doppler measurements with the transducer in the apicalfour-chamber view. Three consecutive beats were measured and averaged for each parameter[6]. Venous blood samples were obtained by the venipuncture of the large antecubital veins ofthe patients. Clinical examinations included blood sugar, serum creatinine, fasting blood sugar,bloodurea, liver enzymes,lipidprofile,cardiacenzymes, and serumuricacid.

All young patients with documented coronary angiography were selected. The coronaryangiography was performed under local anesthesia via the femoral artery using the retrogradepercutaneous transfemoral technique (Judkins's technique) by two experienced cardiologistsblinded to the patients' clinical characteristics and laboratory results. We evaluated epicardialcoronary arteries, including the left main, left anterior descending, left

circumflex, rightcoronaryartery,andthevessels'mainbranches.Ascertainment of outcome coronary

angiography and CAD were performed using (Gensini Score system) based on the number ofaffected vessels, localization of the segment, and the grade of the stenosis. These scores are multiplied by the coefficient define d fore achcoronary artery and segment,and the result sare then added.The presence of CAD has been defined as the Gensini scorebeing>0[7].

StatisticalAnalysis

In this study, SPSS version20.0was used for the statisticalanaly s is and presentation.the mean and standard deviation present the continuous variables and data using t-test for comparing in normally distributed data; the median used fornon-normally distributed data.

Results:

Study Population:

Between October 2019 and March 2020 in Tanta university Hospital, 100 patients were dividedintotwogroupssuspectedofCADand non-CADpatients.Allpatientsunderwentanangiography scoring system that divided them into two groups; if the Gensini score was zero,the patients were non-coronary artery disease. This group included 40 male and ten femalepatientswithamean age(30±3.5).IftheGensiniscorewashigherthanzero,thepatientsweredefinedascoronaryarterydisease patients.Thisgroupincluded34malesand16females(mean age31±3).

Each group was subdivided according to sex into males and females with a mean age of 31.06years. there was a nonsignificant statistical difference between the two groups regarding age(p=0.885)&sex(p=0.171).Regardingriskfactors,smokingwasthemostfrequentbetweenthetwo groups Table (1). According to univariate analysis has shown a significant correlationbetween Hyperuricemia and CAD existence. This analysis results support the previous studiesonCADriskfactors.Allthedatahasbeenillustratedandrecorded inTable 2.

HUAandSmokingCorrelationinCADpatients

We have adjusted many factors present in table 2. We found an unexpected differencebetween HUA and CAD correlation in smokers and non-smokers. In table 3, The Data presenteda statistically significant association between HUA and CAD in non-smokers (p=0.045) comparedwith smokers (p=0.110).

Discussion:

In this study, regarding risk factors, 48% of the patients were smokers, 23% diabetic patients,14% patients have a positive family history of CAD, and 46% hypertensive patients among thestudied patients. The most affected artery was LAD (56%), followed by RCA (40%). There is asignificant correlation between HUA and CAD in young adults ≤ 35 years old. In addition, HUAand smoking have statistical significance for CAD in young adults ≤ 35 years old. HUA issignificantlyassociatedwiththeoccurrenceofCADinnon-smokersofyoungadults≤35years

old. Unexpectedly, HUA and smoking have a contrast; the existence of these two risk factors ina patient is not remarkably associated with CAD occurrence compared to the statistical resultsofnon-smokers.

A similar conclusion was reached by Lv et al. [8] that there is a contrast between smoking andHUA Patients for the existence of CAD; statistically, the non-smokers' patients with HUA werehighly associated with CAD Compared to smokers in patients under35. In addition, Serum uricacid level was higher in patients with CAD [9]. In contrast to our study, Cheong et al. [10]concluded that Serum Uric Acid showed no significant association with all-cause andcardiovascular mortality. A large sample size in Cheong et al. [10] may be the cause as 356 960patientswereincludedvs.100patientsinourstudy.

According to our results, there is an unexpected contrast between HUA and the cigarette effecton patients suspected of CAD. Different studies reached a similar conclusion that makes it avitalpointforfutureresearcherstofocuson.

Studylimitation

Thepresentstudyhasasignificantlimitationasfollows;thisobservationalstudyhasalimitednumber of patients (100 patients), so it will be a must to apply it to a broader number ofpatients. The results were applied only to Egyptian patients and opening the sight for otherresearchers to apply it in different populations and races. Future research on smoking andhyperuricemia might explain the explanations of this contrast between them. Further studieson a large geographical scale and larger sample size. The effect of anti-HUA drugs on theprogression of CAD should be investigated further in large randomized trials, which maypotentiallyprovidenewtherapeuticapproachesfortheprevention andtreatmentofCAD.

Conclusion

By monitoring various parameters in young adults ≤ 35 years old, this study strengthens thecorrelation between:1) Hyperuricemia and the occurrence of CAD, 2) Smoking and the CAD.Furthermore, there is a clear association between HUA and the occurrence of CAD in non-smokers.

References:

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Table1:Demographicandclinicalcharacteristicsofthestudypopulation:the units for the tested parameters given in table 1 should be added; the type of statistical method in dependence of the type data distribution should be given

	(Group I) Non-CAD(n=50)	(Group II) CAD(n=50)	P
Sex(Male%)	40(80%)	34(68%)	0.171
Age	30.96±3.50	31.06±3.38	0.885
DiabetesMellitus	7(14%)	16(32%)	0.032*
Hyperlipidemia	9(18%)	18(36%)	0.043*
Smoking	18(36%)	30(60%)	*0.016
FamilyhistoryofCAD	3(6%)	11(22%)	0.021*
ECG(Abnormal%)	8(16%)	38(76%)	<0.001*
Echo(Abnormal%)	8(16%)	39(78%)	<0.001*
FastingBloodGlucose	98.12±16.30	107.16±23.07	0.026*
RBS	95.0(81.0–100.0)	94.50(84.0–110.0)	0.358
Totalcholesterol	171.0(160.0–230.0)	176.50(160.0–236.0)	0.997
TG	107.0(90.0–128.0)	100.0(83.0–160.0)	0.624
LDL	85.0(75.0–114.0)	114.0(85.0 –132.0)	0.002*
HDL	50.0(41.0–51.0)	48.50(42.0–50.0)	0.687
Serumcreatinine	0.77±0.19	0.85±0.21	0.035*
Uric Acid	3.90(3.40–4.50)	4.55(3.60–6.50)	0.001*

Echo:Echocardiography,ECG:Electrocardiogram,TG:Triglycerides,RBS:Randombloodsugar,

Table2:Univariateanalysisofcoronaryarterydisease(CAD)riskfactors

	MEAN±SD	NO.(%)	P	OR (95%C.I)
AGE	31.01±3.42		0.883	1.009(0.899–1.132)
SEX				
MALE		74(74.0%)	1.0	1.882(0.756–4.690)
FEMALE		26(26.0%)	0.174	
SMOKING				
YES		48(48.0%)	0.017*	2.667(1.188–5.985)
NO		52(52.0%)	1.0	

YES		18(18.0%)	0.014*	4.472(1.355–14.755)
NO		82(82.0%)	1.0	
HYPERTENSION				
YES		46(46.0%)	0.046*	2.263(1.013–5.052)
NO		54(54.0%)	1.0	
DIABETESMELLITUS				
YES		23(23.0%)	0.037*	2.891(1.068–7.823)
NO		77(77.0%)	1.0	
FAMILYHISTORYOFCAD				
YES		14(14.0%)	0.030*	4.419(1.151–16.966)
NO		86(86.0%)	1.0	
SERUM	0.81±0.20		0.039*	8.738(1.112–68.645)
CREATININETOTALC				
HOLESTEROL		32.0(32.0)	0.392	1.446(0.621–3.368)
≥200		68(68.0%)	1.0	
≥150		26(26.0%)	0.363	1.519(0.617–3.745)
<150		74(74.0%)	1.0	
LDL				
≥130		23(23.0%)	0.037*	2.891(1.068–7.823)
<130		77(77.0%)	1.0	
HDL				
<40		15(15.0%)	0.404	0.621(0.203–1.899)
≥40		85(85.0%)	1.0	

OR:Odd`sratio,C.I:Confidenceinterval,LL:Lowerlimit,UL:UpperLimit

Table 3: Association between hyperuricemia and coronary artery disease (CAD) in smokers and non-smokers

	NO. OF PATIENTS	OR(95%C.I)			
		P	Crude	P	Modell[a]
NON-SMOKERS					
NORMOURICEMIA	41		Reference		Reference
HYPERURICEMIA	11	0.014*	6.444*(1.46–28.53)	0.045*	5.017*(1.83–30.31)
CURRENTSMOKERS					
NORMOURICEMIA	41		Reference		Reference
HYPERURICEMIA	7	0.199	4.250(0.47–38.60)	0.110	9.409(0.60 –147.29)

OR:Odd`sratio,C.I:Confidenceinterval,LL:Lowerlimit,UL:UpperLimit