Benha Journal of Applied Sciences (BJAS) **print: ISSN 2356–9751**
Vol. (8) Issue (3) (2023), (62-66) **online: ISSN 2356–976x**

http://bjas.journals.ekb.eg

Assessment of CoronaryAtherosclerotic Plaque Characteristics by Computed Tomographic Angiography in Smokers Compared to Non-smokers

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Abstract

Background:Smoking and passive smoking have been identified as variable risk factors for AMI and CAD,smoking plays a major role in premature coronary atherosclerosis and in accelerating atherosclerosisby increasing the oxidation of LDL and damaging coronary endothelial vasodilation. The purpose of this study is toassess coronary atherosclerotic plaque characteristics including (site, extent, nature and length)by computed tomographic angiography in smokers compared to non-smokers.. Methods:This cross sectional, single center study was conducted at atkobri El koba military hospital during the period from December 2019 to July 2021This study included 200 male patients withsuspectedCAD scheduled for coronary CT angiography. They were divided into two equal groups. The first group included smokers and the second group included non-smokers. Results: The smokers' grouphad a significantly higher prevalence of CADcompared to non-smokers' group.There was no significant statistical difference between the two groups regarding number of affected vessel if it was single or multiple vessels.Middle RCA, Left Main artery, Proximal LAD and Mid LAD were more affected among smokers). Comparison of thenature of obstruction showed that therate of obstruction was higher among smokers, as well calcific plaques were the most prevalent type among smokers, however it was more common in non-smokers.Conclusion:Compared to the non-smokers, smokers had a considerably greater prevalence of CAD in our research. The severity of coronary blockage was greater in smokers than in nonsmokers. CCTA is a potential non-invasive method that may detect coronary artery stenosis and rule out coronary artery disease, as well as evaluate the features of atherosclerotic plaques.

Keywords: Coronary Atherosclerosis; Computed Tomography and Smoking.

1. Introduction

Recently, tobacco smoking is the greatest preventable cause of mortality globally. The tobacco epidemic is one of the worst public health concerns the world has ever faced, taking the lives of over 8 million people annually. A smoker's life expectancy is at least 10 years shorter than a non-smoker's [1].According to World Health Organization data, smoking is responsible for 10% of allcardiovascular disease (CVD) cases [2]. Acute myocardial infarction (AMI) and coronary artery disease (CAD) have been associated with smoking and passive smoking [3]. Smoking significantly contributes to early coronary atherosclerosis and the acceleration of atherosclerosis by increasing the oxidation of low-density lipoprotein (LDL) and impairing coronary endothelial vasodilation [4].The mechanisms by which cigarette smoking induces and promotes atherogenesis and, consequently, atherosclerosis and atherothrombosis are complex and interconnected. The key pathways are inflammation, endothelial dysfunction, prothrombosis, altered lipid metabolism, insulin resistance, and increased demand for but diminished supply of myocardial oxygen and blood (demand-supply mismatch) [5].Coronary computed tomography angiography (CCTA) recently emerged as a promising non-invasive tool able not only to identify coronary artery stenosis or to exclude CAD, but also to assess the characteristics of atherosclerotic plaques itself [6].

We assess the relationship between smokingand the presence, severity and extent of atherosclerotic plaque in patients undergoing MSCT coronary angiography with suspected CAD.

2. Aimof the study

Toassess coronary atherosclerotic plaque characteristics including (site, extent ,nature and length)by computed tomographic angiography in smokers compared to non-smokers.

3. Patients and methods

Study design

This cross sectional, single center study was conducted at atkobri El koba military hospital during the period from December 2019 to July 2021.

Patients' selection

The current study included 200 male patients withsuspectedCAD scheduled for coronary CT angiography. They were divided into two equal groups. The first group included smokers and the second group included non-smokers.

Inclusion criteria

- Patient with stable angina with an equivocalstress test.
- Patient with intermediate risk of CAD with uninterpretable ECG or unable to exercise.
- Asymptomatic patient with low risk of coronary artery disease. With a family history of CAD.
- Exclusion criteria
- Patients with acute coronary syndrome.
- Non diagnostic coronary images because of motion artifacts or inadequate contrast concentration on CCTA images.
- Patients with history of CABG.
- Patients with previous history of PTCA.
- Patient with renal impairment.
- Patient with atrial fibrillation.

A_Blood sample collection, measurement of biochemical markers and leukocyte counts

By venipuncturing the large antecubital veins, blood samples were collected. After an overnight fast, blood samples were drawn and the serum was centrifuged at 2,000 rpm for 1o minutes at 4oC. Triglyceride, total, low-density lipoprotein-cholesterol, and high density lipoprotein-cholesterol levels in the serum were measured enzymatically using a commercially available test kit (Hitachi P8oo, Holliston, Massachusetts, USA).

B_Coronary MSCT angiography

All subjects had coronary angiography imaging using a dual-source MSCT scanner (Somatom Definition, Siemens, Erlangen, Germany). The coronary arteries were widened by administering nitrate sublingually 2–4 minutes before to image acquisition. The coronary angiographic scan was obtained with the injection of 60 mL of nonionic contrast medium (35o mg I/mL iohexol, Amersham Health omnipaque) at a flow rate of 6 mL/s, followed by the injection of 60 mL of saline solution at the same flow rate to flush the contrast material from the right ventricle.

C_MSCT evaluation

All images were interpreted immediately after scanning by an experienced radiologist unaware of
the clinical presentation of the patients. Coronary plaque was defined as any clearly discernible structureattributable to the coronary artery wall in at least two independent image planes.The severity of luminal diameter stenosis was scored as normal (0% luminal stenosis), minimal obstruction (plaques with a lumen narrowing less than25%), mild obstruction (plaques with stenosis 25 to 49%), moderate obstruction (plaques with stenosis from 50 to 69%), severe obstruction (plaques with stenosis from 70 to 99%) and totally occluded lumen (100%). For categorization of coronary plaques, the coronary system was separated into 16 distinct segments utilizing original axial images, thin slice, maximum intensity projections, and cross-sectional reconstructions orthogonal to the long axis of each coronary segment (0.75 mm thickness) [7]. For each section, coronary plaques were classified as lipid (<30HU), fibrous (30-150HU), calcified (>350HU), and lumen (150-150HU) [7]. The extent of coronary atherosclerosis was calculated as the sum of the affected coronary segments.All plaque components and significant stenosis
were assessed per segment.

Statistical methods

Statistical analysis was done by SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analysed utilizing the Chi-square test or Fisher's exact test when appropriate. A two tailed P value < 0.05 was considered statistically significant.

4. Results

This research comprised 200 male patients with chest discomfort who had MDCT coronary angiography.

General characteristics

Smokerswere significantly older than non-smokers (54±6 vs.50±6 years, p = 0.0001). Diabetes and Hypertension were significantly prevalent in smokers group (40 patients ″40%″ vs. 15 patients ″15%″ ,p = 0.0001and39patients ″39% ″vs. 21 patients ″21%″ , p=0.008).However, there was no significant statisticaldifference between the two groups regardingfamily history(p = 0.75)**(Error! Reference source not found.&Error! Reference source not found.)**

Table (1) Demographics and comorbidities among groups.

	Group		P value
	Smoker (n=100)	Non-smoker (n=100)	
Age (years)	**53 ± 6**	**51± 6**	**0.0001***

Table (1) Continue			
Diabetes mellitus	**41(41%)**	**15(15%)**	**0.0001****
Hypertension	**39(39%)**	**21(21%)**	**0.008****
Family history	**6(6%)**	**5(5%)**	**0.75****

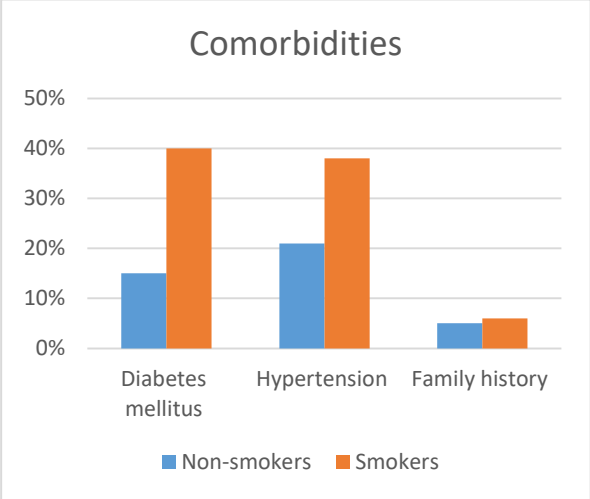

Fig. (1) : Bar chart showing comorbidities among groups.

Laboratory findings

LDL was significantly higher among smokers versus non-smokers (89.8 ± 15.8 vs. 87.6 ± 22.6 mg/dL, p =0.004) As well, HDLwas significantly lower in smokers (39.3 ± 4.2 vs.41.9 ± 6.2 mg/dL,p=0.0001).There was an insignificant differencesbetween the two groups regarding serum cholesterol, triglycerides and serum creatinine **Table (2) and Fig (2).**

Table (2) Lab findings in the studied patients.

	Group				P value
	Smokers (n=100)		Non-Smokers (n=100)		
	Mean	Standard Deviation	Mean	Standard Deviation	
Serum cholesterol (mg/dl)	203.3	34.6	200.4	40.2	0.36
Serum Triglycerides (mg/dl)	118.2	42.4	114.2	38.9	0.62
LDL (mg/dl)	89.8	15.8	87.6	22.6	0.004
HDL (mg/dl)	39.3	4.2	41.9	6.2	0.0001
Serum Creatinine (mg/dl)	0.9	0.3	0.9	0.2	0.36

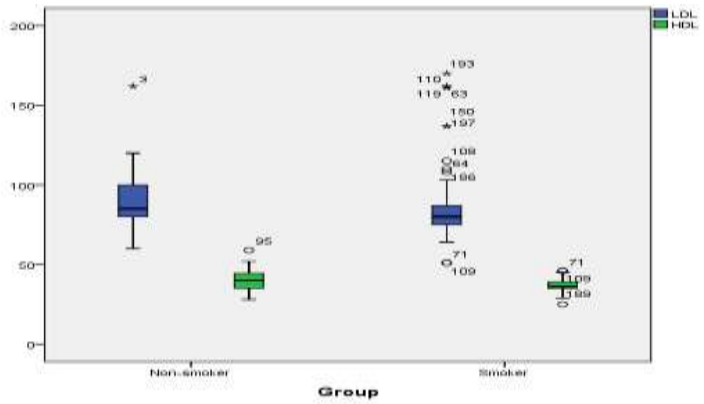

Fig. (2): Box plot showing

Comparison of LDL and HDL levels among groups.

Computed tomography

The smokers grouphad a significantly higher prevalence of CADcompared to non-smokers group (83% vs. 37% ,p =0.0001).

The number of affected vessel was insignificantly different between both studied groups whether if it was single or multiple vessel (p=0.064) **Table (3).**

Table (3) Prevalence of vessels lesion among study groups.

	Group				P value
	Smoker (n=100)		Non-smoker (n=100)		
	Count	Column N %	Count	Column N %	
Normal	17	17.0%	63	63.0%	0.0001
Obstructing lesion	83	83.0%	37	37.0%	
single	59	٧٠.٢%	26	%68.4	٠.٠٦٤
multiple	22	٢٦.٢%	3	%7.8	
2 vessels	3	٣.٦%	9	%23.8	

5. Discussion

Smoking is a risk factor for CAD and a known factor influencing the severity and pattern of CAD. The risk of developing CVD is twice as high among smokers compared to nonsmokingindividuals [8].

MSCT is a recently developed technique for assessing the function of the heart and coronary arteries non-invasively. Cardiac MSCT uses X-ray beams and a liquid dye to form a 3-D image of the heart and vessels. The scanning machine used is very sophisticated and scans the heart very quickly. This provides sharp, detailed images that can't be achieved with other tests [9].

This trial assessedcoronary atherosclerotic plaque characteristics by computed tomographic angiography in smokers compared to non-smokers.by dual-source multi-slice computed tomographic coronary angiography.

This cross sectional study included about 200 patients with chest pain and presented to MSCT coronary angiography for evaluation. Then, patients were divided into:

- Group I:100 patients smokers
- Group II: 100 patients non smokers

All patients were subjected all full history taking, laboratory investigations, 12-leads surface electrocardiogram, and MSCT.

In the present study, it was found that smokers were significantly older than non smokers with higher prevalence of DM and hypertension in smokers group. In consistent with our results, [10]**Joshi *et al.,* 2020**who evaluated effect of cigarette smoking on selected antioxidant enzymes and oxidative stress biomarkers. They found that hypertension and diabetes were more prevalent among smokers.

on the contrary, [11]**Hou *et al.,*2019** who studied coronary atherosclerotic plaque volume quantified by computed tomographic angiography in smokers compared to nonsmokers found that non smokers were older than smokers.

In the present study, it was found that LDL was significantly higher among smokers versus non-smokers and HDL was significantly lower in smokers In consistent with our results, [12]**Sun *et al.,* 2015**who assessedinfluence of cigarette smoking on the burden and characteristics of coronary artery plaques in chinese men. They found that smokers have higher level of LDL and lower level ofHDL compared to non smokers.

Also, [12]**Sun *et al.,* 2015** stated that smokers have higher level of LDL and lower level ofHDL compared to non-smokers .

In our study, the smokers grouphada significantly higher prevalence of CADcompared to non-smokers group .

In consistent with our results, [13]**Van Werkhoven*et al.,* 2011** whowho studied influence of smoking on the prognostic value of cardiovascular computed tomography coronary angiography. Theyfound that smokers with CAD by CTA were 4 fold higher than non smokers.

Also, [12] **Sun *et al.,* 2015**stated that stated that coronary atherosclerosis was more prevalent in smokers compared to non smokers.

In the present study there was no significant statistical difference between the two groups regarding number of affected vessel if it single or multivessel disease.

Some authors [4]**Salehi*et al.,* 2021** who researched the effect of cigarette smoking on coronary arteries and the pattern and severity of coronary artery disease reached this conclusion. Eight included studies demonstrated a correlation between smoking and the number of blocked coronary arteries in CAD patients. The findings of six investigations revealed no correlation between smoking and coronary artery damage. There is a correlation between smoking and coronary artery damage, according to two research.

6. Conclusion

The smokers group had a significantly higher prevalence of CAD compared to non-smokers group.Coronaries obstruction were more severe in smokers than non-smokers. CCTA is apromising non-invasive tool which canidentify coronary artery stenoses andexclude CAD, also to assess the characteristics of atherosclerotic plaques itself.

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