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Coronary Computed Tomography Angiography Criteria in Men vs Women

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Abstract:

Background: Atherosclerosis involves the accumulation of atheromas or fibrofatty plaques within the large-to-medium-sized arteries of the heart, potentially leading to rupture or erosion that obstructs blood flow, ultimately causing ischemic heart disease. This study aimed to assess gender-based criteria of coronary artery tree based on coronary computed tomography angiography (CCTA). **Methods:** This cross-section, comparative and dual center study included 1000 patients with low to intermediate risk of coronary artery disease (CAD) scheduled for CCTA at the National Heart Institute and Benha University Hospital. All patients underwent ECG, Echocardiography and CCTA. **Results:** Prevalence of atherosclerosis in males was significantly higher than females (272 patients 51.32 % vs 185 patients 39.36 %, P=0.002). Calcium score was significantly higher in males compared to females (144.71 ± 86.82 vs 133.98 ± 81.36, P =0.045), High risk plaques (118 patients 43.21% vs 66 patients 35.74 %, P=0.016) and segment involvement score (1.43 ± 0.7 vs 1.35 ± 0.64, P =0.045) were significantly higher in males than females. The positive remodeling (372 (31.3%) vs 245 (23.2%) , P-value =0.009) , Napkin ring sign (87(7.3) vs 72(6.8), P=0.004), > 1 types of HRP (304 (25.6) vs 191(18.1,P=0.004) and stenosis severity score (2.57 ± 1.39 vs 2.37 ± 1.33 , P-value =0.025) were significantly higher in males than female. Total plaque volume was insignificantly different between both groups (P<0.001). **Conclusion:** Men exhibit higher coronary atherosclerosis compared to women, these disparities persist even after adjusting for socioeconomic, lifestyle, and traditional risk factors (e.g., smoking, hypertension), suggesting biological mechanisms.

Keywords: Coronary CT Angiography Criteria; Men; Women.

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Introduction

Atherosclerosis is a disease characterized by the accumulation of atheromas or fibrofatty lesions in the large-to-medium-sized arteries of the heart. This accumulation can result in perforation or erosion, which obstructs blood flow and ultimately causes ischemic heart disease (IHD) [(1)]. Numerous critical factors contribute to the pathogenesis of atherosclerosis, such as lipid accumulation, vascular inflammation, arterial rigidity, remodeling, intimal thickening and fibrosis, and the rupture or erosion of plaques, which can result in myocardial infarction (MI) and ischemia. Prior to manifesting as clinical disease, this pathogenesis frequently advanced undetected for years, or even decades. Primary consequences of atherosclerosis include cerebral infarction (stroke), peripheral artery disease, myocardial infarction (MI), and aortic aneurysms [(2).]

Atherosclerosis, the most prevalent death reason worldwide, affects both sexes. However, men are more likely to succumb to atherosclerosis or coronary artery disease (CAD) at a younger age (40–60 years), while women typically develop atherosclerosis after menopause, resulting in a higher prevalence of the condition among women. Despite the fact that atherosclerosis is less prevalent in women, individuals with autoimmune diseases are at an elevated risk of developing CAD. This correlation is particularly strong in the context of disorders including hypertension, systemic lupus erythematosus, rheumatoid arthritis, and systemic sclerosis, underscoring the fact that men exhibit unique immune mechanisms that contribute to CAD in comparison to women [(3)].

CAD remains the primary morbidity reason and mortality in both sexes. Recent research has shown that, despite the absence of gender-specific diagnostic and treatment approaches, risk factors, clinical presentations, and ongoing symptoms may vary significantly between genders. Obstructive CAD are more common in men; however, they are more likely to experience sudden cardiac mortality, ischemia, and persistent symptoms. Furthermore, the efficacy of treatment and follow-up is frequently compromised by the diminished obstructive CAD prevalence of females [(4)].

Coronary computed tomography angiography (CCTA) is effective in detecting future cardiac events risk, guiding clinical decisions, and enhancing outcomes in those suspected of having CAD. Latest study has shown that quantitative burden of atherosclerotic plaque can be quantified using CCTA, which has a predictive value that exceeds conventional cardiovascular risk assessments, CA calcification scores, and the presence of obstructive coronary disease [(5)]. Notably, individuals with a low-attenuation plaque burden exceeding 4% had a nearly fivefold increased experiencing MI risk .Nonetheless, it remains unclear whether the distribution of different plaque types varies by sex and whether these measurements provide comparable prognostic value for all patients [(6)].

The purpose of this investigation was to assess the gender-based criteria of the CA tree through the use of CCTA.

Patients and methods

This cross-section, comparative and dual center study included 1000 age > 18 years old and both sexes patients had low to intermediate risk of CAD scheduled for CCTA. The study was carried out at the National Heart Institute and Benha University Hospital from July 2024 to May 2025.

The patients provided written consent that was informed. The purpose of the study was explained to each patient, and they were assigned a secret code number. The investigation was done with the permission of the investigation Ethics Committee at the Faculty of Medicine at Benha University.

Exclusion criteria were Individuals had CAD history, previous CABG or PCI, atrial fibrillation and atrioventricular, atrial flutter and interventricular block, severe renal impairment, cardiac pacing, severe valve dysfunction, pregnancy, and patient refusal.

All studied cases were subjected to the following: Detailed clinical history, including [patients' age, gender, smoking status, and comorbidities (diabetes mellitus (DM) [(7),] hypertension [(8)], CAD [(9)] and hyperlipidemia)]. **Full clinical examination: Clinical examination including [**diastolic and systolic Blood pressure, body mass index (BMI), heart rate, assessment of body weight, and waist circumferences]. **laboratory investigations** are: complete blood count (Hemoglobin, WBCs, platelets), liver function (and ALT, AST), kidney function tests (Urea, Serum creatinine), glycated haemoglobin (HbA1C) and glucose levels, lipid profile (high-density lipoprotein (HDL), low-density lipoprotein (LDL), cholesterol, and triglycerides), international normalized ratio, and C-reactive protein]

ECG:

12 leads ECG was done for all patients to exclude arrhythmias and to detect the presence of ischemic changes

Echocardiography:

Echocardiography was performed in the left lateral decubitus using two ultrasound systems (Epiq 7, Philips and the Vivid S6, GE, Horten, Norway). A concurrent ECG signal was employed to acquire images. Compliance with the American Society of Echocardiography (ASE) guidelines was maintained during the recording and calculation of a diverse array of parameters. The parasternal long axis, which is perpendicular to the long axis of the ventricle at the level of the mitral valve, was used to determine the interventricular septum thickness, posterior wall thickness, LV end-diastolic diameter, and LV end-systolic diameter using M-mode echocardiography. These values were employed to calculate the ejection fraction using the Teicholz method (Teicholz EF): (LV end-diastolic dimension) - (LV end-systolic dimension) / (LV end-diastolic dimension) **[(10)].**

CCTA:

Siemens DSCT scans were implemented to execute CCTA. One hour prior to their examinations, the pulse rates of all patients were assessed. Propranolol hydrochloride (Inderal at 40 mg/tablet) is taken orally for HR of 65 bpm or higher, except who had contraindications to beta-blockers. The dosage ranged from 40 to 80 mg. Nitroglycerin 0.5 mg was given sublingually just prior to the scan. A 60 mL injection of iodinated contrast mixture and 60 mL of saline solution was administered to each patient. In order to regulate the administration of contrast material, test boluses were injected into the ascending aorta. The scan was delayed by 12 seconds. The scan was conducted promptly, and the images were reconstructed to identify CA images that were devoid of motion. The reconstructed CT image data was exposed to axial, multi-planar reformat, maximum intensity projection, and short-axis, cross-sectional views. The data was subsequently transferred to a computer terminal for post-processing. Regardless of the resolution of the image, each arterial segment was evaluated with the intention of diagnosing the individual [(11)]. All images were interpreted by two specialists who were oblivious of the patients' characteristics and study design after they were scanned. The term " CA plaque " (CAP) refers to any aberrant structure within the CA wall that was visible in 2 distinct image orientations. For the purpose of CAP categorization, the coronary system was divided into 16 distinct segments in accordance with a modified AHA classification. In accordance with a modified AHA classification, the coronary system was divided into 16 distinct segments for the purpose of CAP categorization. The

following segments are identified in the following order: the left anterior descending artery (LAD), proximal, middle, and distal LAD; proximal, middle, and distal diagonal; proximal, middle, and distal circumflex arteries (Cx); proximal, middle, and distal broad marginal branches; and proximal, middle, and distal right cardiac artery (CA). The long axis of each coronary segment is orthogonal to the cross-sectional reconstructions. maximum intensity projections, thin sections, and original axial views (0.5-mm thickness) are employed to identify these segments. Following this, an assessment of each plaque was conducted in accordance with its segments [(12)].

The study precluded those having more than three segments in the distal region, as well as those with excessive calcification, excess artifact, poor image quality, and segments that could not be evaluated in the proximal or intermediate component of the coronary tree. For each segment, the CAPs were classified as follows: "none," "noncalcified plaque" (NCP; defined as a CT density less than the contrast-enhanced coronary lumen but greater than the surrounding tissue), "calcified plaque" (defined as a CT density greater than the contrast-enhanced coronary lumen), and "mixed plaque" (encompassing calcified and noncalcified elements within a single plaque) [(13)].

Categories were established as follows in response to an evaluation of the CA disease-reporting and data system (CAD-RADS) for the most critical stenosis in any vessel: CAD-RADS 0: 0% stenosis and no plaque, CAD-RADS 1: 1%–24% stenosis or plaque with positive remodeling but no stenosis, CAD-RADS 2: (25%–49% mild stenosis), CAD-RADS 3: (50%–69% moderate stenosis), CAD-RADS 4a: (70%–99% severe stenosis in one or two vessels), CAD-RADS 4b: (70%–99% severe stenosis in three vessels or left main 50%), and CAD-RADS 5: (all 100% stenosis or total occlusion) [(14).]

Agatston scoring was implemented to determine CA extent calcification, Lesions with a CT density of >130 HU in a 1 mm2 balanced area in 2–3 adjacent pixels are interpreted as calcifications by the Agatston scoring system. The Agatston score was determined through the utilization of a proprietary software application. Consequently, axial sections were implemented to detect calcifications. The radiologist detected calcific deposits in the coronary arteries. The software calculates the calcium score by multiplying the density score by the lesion area and density, with a particular emphasis on the calcified lesion. The software automatically measures lesions area density.

After the calcium scores of the four primary CA were determined then patient's total calcium score. The following risk categories were established, as a calcium score of [>400] demonstrates very high risk of calcific atherosclerotic plaque, high risk (100–400), medium risk (10–100), low risk (1–10) and score 0 demonstrates the absence of risk. In addition, the calcium score was assessed in relation to age and gender, and patients were classified as high-risk if their calcium score exceeded 75% [(14).]

obstructive (plaques with a maximal stenosis ≥ 50%), un-obstructive (plaques with a lumen narrowing less than 50%) and as none (0% luminal stenosis) were severity of luminal diameter stenosis classification. The maximal intra-luminal stenosis in any of the segments of main epicardial CAs was used to diagnose CAD at the ≥ 50% stenosis threshold [(14)].

The diagonal branches and obtuse marginal branches of LAD artery, RCA and left circumflex (LCX) artery system were each deemed to contain obstructive CAD. The posterior descending artery was classified as either a component of the LCX or RCA system, depending on the dominance of the CA [(15).]

Subsequently, the presence of the following high-risk plaque features was

assessed for each plaque. If at least one high-risk plaque feature was observed, each patient was classified as having high-risk plaque [16]. We evaluated the presence or absence of three HRP features: positive remodeling (PR), napkin-ring sign (NRS), and scattered calcification.

Remodeling index (RI) of ≥ 1.1 was used to define PR. This index was determined by dividing the vessel size at maximal remodeling by the vessel size at the reference site [17]. NRS was defined as a central area with low attenuation that is encompassed by a peripheral area with high attenuation [18].

Spotty calcification was the term used to describe focal areas of calcification within the CA wall that were less than 3mm in maximal diameter [19]. Low attenuation plaque was not included due to inconsistent observer reproducibility

Two straightforward clinical CAP scores were implemented: the segment-involvement score and the segment-stenosis score. Segment-stenosis scores were utilized to quantify the extensiveness of CAP.

The degree of obstruction affecting the coronary luminal diameter was used to evaluate existence of plaque in each coronary segment, with a rating of 0 indicating no plaque and 3 indicating severe plaque. The cumulative score, which ranges from 0 to 48, is calculated by aggregating the scores of all 16 individual segments[20].

We calculated the segment involvement score to evaluate overall distribution of CAP. This score is based on the total number of CA segments that exhibit plaque, regardless of the extent of luminal stenosis in each segment, and ranges from 0 to 16. In addition, a three-vessel plaque score was devised, which yielded a binary outcome (0 or 1) that was not contingent upon the plaque severity in the left anterior descending, RCA and LCX [20].

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Statistical analysis

Statisticians and data managers were making use of SPSS 26 (USA, Armonk, New York, IBM) To ensure the quantitative data was normal, we used visualization tools, the Kolmogorov-Smirnov test, and the Shapiro-Wilk test. Summarizing the quantitative data required the use of medians, ranges, means, and standard deviations in accordance with the normality principle. To simplify the category data, percentages and numbers were being used. In order to compare the groups that were evaluated, for quantitative data that follows a normal distribution, the independent t-test, and for data that does not, the Mann-Whitney U test. The categorical data was compared using two independent tests: Fisher's exact and Chi-square. The succeeding studies were all driven by the availability of data. It was clear that all of the statistical tests were biased. Significance was determined by p-values lower than 0.05.

Case presentation:

Case 1: A 63 years old female patient hypertensive for ten years on CCB with no past history of smoking, not known to be diabetic. Her BMI is 22.5 kg/m. She was complaining of recurrent chest tightness describing it as retrosternal chest discomfort radiating to shoulders appears only when she performs excessive duties at work or climbing three floors and relieved immediately by rest. ECG showed nonspecific changes (inverted T wave in Lead III). By reviewing her laboratory values, it was found that she is dyslipidemia (total Cholesterol: 226 mg/dl, LDL: 131 mg/dl, HDL: 42 mg/dl, TG: 266 mg/dl) and her blood sugar was controlled in the last 3 months (HbA1C: 4.8%). CT angiography showed a single LAD lesion with Ca score 16.6. **Figure 1**

Case 2: A 52 years old male patient diabetic for 7 years on oral hypoglycemic drug (metformin), smoker, not known to be hypertensive. His BMI is 25.7 kg/m2. He was complaining of recurrent chest discomfort associated with slightly dyspnea appears on exercise or climbing

two floors and relieved immediately by rest. ECG showed Left Bundle Branch Block. By reviewing his lab. values it was found that he is dyslipidemic (total cholesterol.: 312 mg/dl, LDL: 152 mg/dl, HDL: 41 mg/dl, TG: 176 mg/dl) and his blood sugar is well controlled in the last 3 months (HbA1C: 4.9 %). CCTA revealed that he had 16 lesions with Ca score of 115.7. **Figure 2**

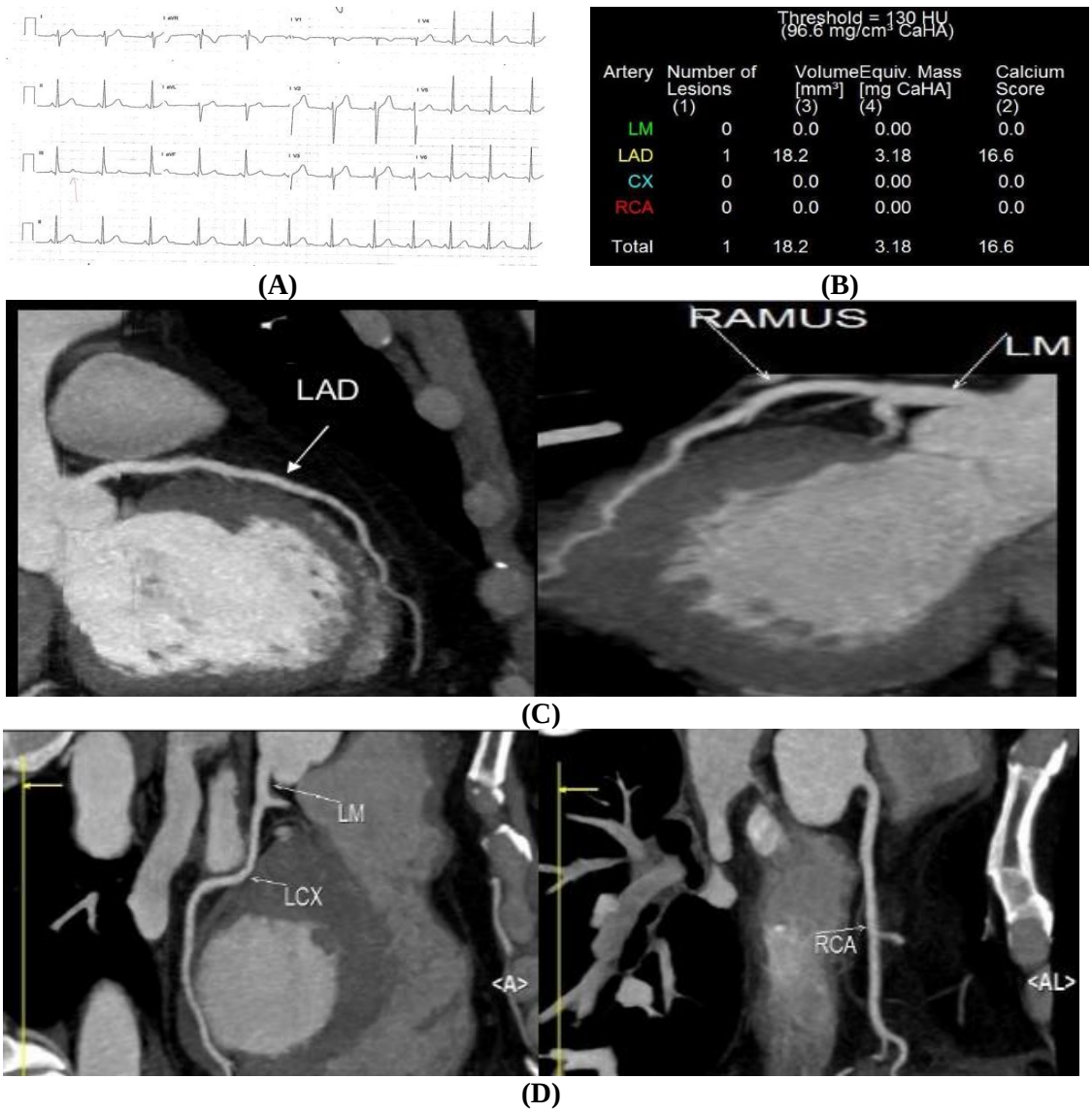

Figure 1: CCTA findings in case 1; A: LAD shows non-significant plaque B: Sizable ramus branch without lesions C: LCX shows no significant lesions. It gives a sizable normal OM branch D: RCA is dominant normal

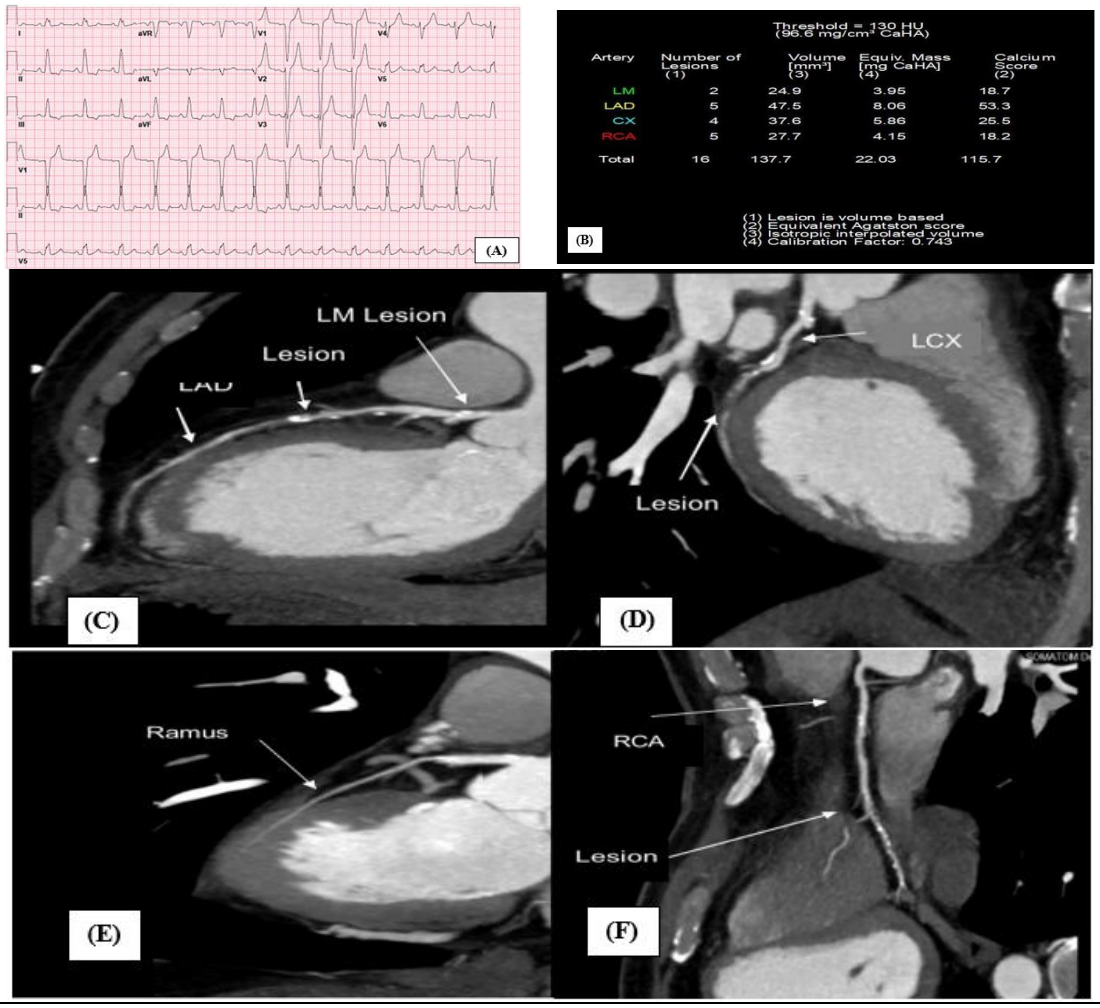

Figure 2: CCTA findings in case 2; C: LAD shows proximal significant mixed plaque followed by mid segment tight lesion D: LCX shows mid segment significant mixed plaque after giving OM br E: Ramus branch without lesions F: RCA is dominant shows mid and distal significant mixed lesions.

Results

The aged of the studied groups was markedly lower in males than in females (62.1±7.58 vs 63.13±7.6 years) (P=0.032). The prevalence of smoking (282 patients 53.21% vs 16 patients 3.4%, P < 0001) and hyperlipidemia (281 patients 53.02% vs 201 patients 42.77%, P =0.002) was significantly increased in males. However, females were considerably more likely to have hypertension than males (303 patients, 64.47% vs 247 patients, 46.6%, P <0.001). the medications used (statins, antiplatelets, betablockers, calcium channel blockers, and OAD), and the family history of CAD, DM, and PAD were comparable. The HR was significantly higher in males (85.54±8.94 vs. 84.25±9.09 bpm, P=0.024). Although there was no significant difference between the two groups in terms of SBP, DBP, weight, height, BMI, and BSA. Males had increased HbA1C level than females (5.78±1.88% vs 5.43±1.92%, P=0.004). Males exhibited significantly lower HDL levels than females (53.08±7.48 vs 60.16±9.2mg/dL, P<0.001). Hb, WBCs, platelets, electrolytes (sodium and potassium), total cholesterol, LDL, TG, liver, and kidney function were insignificantly different. **Table 1**

Female had significant raised LVEF than males (62.91±2.02 vs 61.03±1.95, P-value <0.001) where LVESD and LVEDD were comparable. **Table 2**

The prevalence of atherosclerosis was considerably higher in males than in females (272 patients, 51.32 % vs 185 patients, 39.36 %, P=0.002). In comparison to females, males exhibited a significantly higher calcium score (144.71 ± 86.82 vs 133.98 ± 81.36, P =0.045), a significantly higher number of high-risk plaques (HRP) (118 patients 43.21% vs 66 patients 35.74 %, P=0.016), and a significantly higher segment involvement score (1.43 ± 0.7 vs 1.35 ± 0.64, P =0.045). The positive remodelling (372 (31.3%) vs 245 (23.2%), P-value =0.009), Napkin ring sign (87(7.3) vs 72(6.8), P=0.004), >1 forms of HRP (304 (25.6) vs 191(18.1, P=0.004), and stenosis severity score higher in males (2.57 ± 1.39 vs 2.37 ± 1.33, P-value =0.025) than in females. The total plaque volume did not differ significantly between the two groups (P<0.001).**Table 3**

Table 1: Demographic data, clinical data, laboratory investigations of studied groups

		Male (n=530)	Female (n=470)	P-value
Demographic data	**Age (years)**	62.1±7.58	63.13±7.6	**0.032***
	Smoking	282 (53.21%)	16 (3.4%)	**<0.001***
	HTN	247 (46.6%)	303 (64.47%)	**<0.001***
	DM	237 (44.72%)	190 (40.43%)	0.170
	Hyperlipidaemia	281 (53.02%)	201 (42.77%)	**0.002***
	PAD	51 (9.62%)	58 (12.34%)	0.168
	Family history of CAD	196 (36.98%)	183 (38.94%)	0.524
	Statins	260 (49.06%)	235 (50%)	0.765
	Antiplatelets	252 (47.55%)	243 (51.7%)	0.189
	Betablockers	29 (5.47%)	14 (2.98%)	0.052
	Calcium channel blocker	261 (49.25%)	244 (51.91%)	0.399
	OAD	4 (0.75%)	6 (1.28%)	0.408
clinical data	**Weight (kg)**	80.32±11.49	79.67±11.93	0.385
	Height (m)	1.64±0.06	1.63±0.06	0.077
	BMI (kg/m^2)	29.92±4.81	29.94±5.04	0.960
	BSA (m^2)	1.92±0.03	1.92±0.03	0.699
	HR (beats/min)	85.54±8.94	84.25±9.09	0.024*
	SPB(mmHg)	133.81±14.15	132.38±13.52	0.104
	DPB(mmHg)	78.62±13.17	77.38±11.69	0.118
laboratory investigations	**Hb (g/dl)**	12.14±1.04	12.03±1.14	0.110
	WBCs ($*10^9/L$)	8.03±2.09	8.02±2.06	0.899
	Platelets ($*10^9/L$)	303.88±59.02	298.02±57.34	0.113
	HbA1C (%)	5.78±1.88	5.43±1.92	**0.004***
	Na^+ (mEq/L)	140.62±2.3	140.54±2.39	0.560
	K^+ (mEq/L)	4.28±0.49	4.31±0.5	0.218
	Serum creatinine (mg/dl)	1.01±0.2	0.99±0.2	0.066
	Urea (mg/dl)	46.32±14.69	45.2±15.29	0.240
	eGFR ($mL/min/1.73m^2$)	109.77±11.56	110.73±12.06	0.198
	Cholesterol (mg/dL)	216.25±44.07	215.99±44.76	0.926
	TG (mg/dL)	286.5 ± 67.93	284.21 ± 67.3	0.592
	HDL (mg/dL)	53.08±7.48	60.16±9.2	**<0.001**
	LDL (mg/dL)	147.16±42.39	144.55±43.02	0.336
	AST (U/L)	29.83±6.1	29.93±5.83	0.784
	ALT (U/L)	31.54±6.58	30.93±5.97	0127

Data presents as mean ± SD or frequency (%). HTN: Hypertension, DM: Diabetes mellitus, PAD: Peripheral artery disease, OAD: oral antidiabetic medications, BMI: Body mass index, BSA: body surface area, Hb: Haemoglobin, WBCs: white blood cells, HbA1C: glycated haemoglobine, GFR: estimated glomerular filtration rate, TG: Triglycerides, HDL: high-density lipoprotein, LDL: low-density lipoprotein, AST: aspartate aminotransferase, ALT: Alanine aminotransferase, *: statistically significant as p value <0.05.

Table 2: Echocardiography of the studied groups

	Male (n=530)	Female (n=470)	P-value
LVEF (%)	61.03±1.95	62.91±2.02	**<0.001***
LVESD (mm)	31.96±3.14	32.04±3.14	0.509
LVEDD (mm)	46.29±4.19	46.41±3.86	0.495

Data presents as mean ± SD or frequency (%). LVEDD: left ventricular end-systolic diameter, LVEF: Left Ventricular Ejection Fraction, LVESD: left ventricular end-systolic diameter, *: statistically significant as p value <0.05.

Table 3: Coronary computed tomography angiography (CCTA) of the studied groups

		Male (n=530)	Female (n=470)	P-value
Affected segments	**Normal segment**	2118(48.68%)	1795(60.64%)	0.278
	Obstructive lesion	1232(28.30%)	503(17.0%)	
	Non-obstructive lesion	1002(23.02%)	662(22.36%)	
Atherosclerosis %		272(51.32%)	185(39.36%)	**0.002***
Number of Vessels affected	**Single**	92(33.96%)	68(36.81%)	0.494
	2 vessels	90(33.03%)	62(33.4%)	
	Multi vessels	90(33.01%)	55(29.79%)	
Vessel affected	**LM**	15 (5.5%)	3 (1.2)	0.215
	LAD	169(62.1%)	107(48%)	
	LCX	65(23.9%)	35(16%)	
	RCA	76(27.9%)	77(34.5%)	
Calcium score		144.71±86.82	133.98±81.36	**0.045***
Lesion type	**Soft**	88(32.44%)	61(33.14%)	
	Mix	82(30.18%)	92(49.74%)	**<0.001***
	Calcific	102(37.38%)	32(17.12%)	
High risk plaques (HRP)	**present**	965(43.21%)	416(35.74%)	**0.016***
only one type of HRP	**Spotty calcification**	508 (52.6%)	191 (45.9%)	0.06
	Postive remodelling	302 (31.3%)	97 (23.2%)	**0.009***
	Napkin ring sign	70 (7.3%)	28 (6.8%)	**0.004***
II)> 1 types of HRP		247 (25.6%)	75 (18.1%)	**0.004***
Segment involvement score		1.43±0.7	1.35 ± 0.64	**0.045***
Segment stenosis score		2.57 ± 1.39	2.37 ± 1.33	**0.025***
Total plaque volume (mm3)		159.72±49.48	154.22±51.28	0.0٥٠

Data presents as mean ± SD or frequency (%). CAD: coronary artery disease. *: statistically significant as P value <0.05

Discussion

CCT is now considered a class I indication for non-invasive examination of patients suspected of having CAD as 2019, according to the recommendations of the European Society of Cardiology (ESC). The American Heart Association's protocol for treating chest discomfort in 2021 is comparable to this. The basis for this upgrading is that CCT has the same health outcomes as invasive coronary angiography and a high accuracy rate in diagnosing stenosis beyond 50% [(21)]. Furthermore, CTA facilitates the identification of vulnerable HRP, which in turn facilitates the stratification of cardiovascular risk. Currently, CCTA is recommended for cases had pre-test probability (PTP) ranging from low (5–15%) to intermediate (15–75%) levels [(22).]

Our study shows that the age of the studied patients was significantly lower in males compered to females, while weight, height, BMI and BSA were insignificantly different between both groups.

Furthermore, Feuchtner et al., [(21)] According to the ESC 2019 guidelines, 1882 consecutive patients (age, 58.9 ± 11 years, 42.5% females) with low-to-intermediate PTP were referred for CCTA

and had complete datasets. Male patients were considerably younger than their female counterparts (P <0.001).

In terms of our findings, males exhibited significantly higher levels of smoking and hyperlipidemia than females, while males exhibited significantly lower levels of HTN than females (P<0.001). Regarding DM, IHD, PAD, and family history of CAD, there was no statistically significant difference between the two groups.

Additionally, Erdoğan et al., [23] founded that males had significantly higher rates of smoking than females (P<0.001), while males had significantly lower rates of hypertension (P<0.001). Nevertheless, the study contradicted our findings, as it demonstrated that hyperlipidemia was indifferent among both sexes.

The male and female categories did not exhibit significant difference in terms of DM or family history of CAD (P=0.16, 0.73, respectively).

The findings of our investigation indicate that males had a significantly higher HR than females (P=0.024), while there was different insignificantly in SBP or DBP between both groups.

In alignment with our results, Pagidipati et al,[24] demonstrated that SBP and DBP were different insignificantly between both groups in 8966 PROMISE trial patients who were tested as randomized (4466 stress testing, 53% female;4500 CTA, 52% female) (P=0.32, 0.83, respectively).

Males had increased HbA1C level than females (P=0.004), according to the current study. While Hb, WBCs, platelets, and electrolytes (sodium and potassium) were indifferent. Males exhibited a significantly lower HDL level than females (P<0.001), while total cholesterol, TG, LDL, AST, and ALT were equal. [25]

These findings are like Şener et al., [25] who demonstrated that males had increased HbA1C level than females (p < 0.001) particularly at 30-49 age group.

These results are in accordance with Erdoğan et al., [23] notified that HDL was significantly lower in males (46 ± 21) in contrast to females (50 ± 11) (P<0.001).

Erdoğan et al., [23] reported in 1496 patients evaluated, 52.1% were male, and 47.9% were female, stress ECG positivity between both groups was indifferent (P=0.616).

In the present study, males exhibited a significantly higher segment involvement score, segment stenosis score, and atherosclerosis prevalence than females (P=.002, 0.045, and 0.025), respectively. However, there was different insignificantly in CAD severity between both groups.

In alignment with the study, Paiva et al., [26] In contrast to females, males demonstrated a significantly higher segment involvement score, segment stenosis score, and atherosclerosis prevalence in the current study (P=.002, 0.045, and 0.025, respectively). In contrast, the severity of CAD did not exhibit any discernible variation between the two groups.

The atherosclerosis (any plaque present), high-risk plaque, and stenosis severity score of males were significantly higher than those of females, according to our research (P <0.05). The males having significantly more calcific plaques than females (P<0.001).

In the same line of our results, Erdoğan et al., [23] found in 1496 patients evaluated, 52.1% were male, and 47.9% were female. 35.4% of female patients and 52.9% of male patients had atherosclerosis (any plaque presence) (P <0.001). When plaque morphology was analyzed, we found that calcific plaque type was dominant in both genders compared to mixed and soft plaque types, and all plaque types were detected more frequently in males Regarding our outcomes, CAD lesion, CACS, remodelling index and plaque vulnerability was significantly increase in males compared to females (P= 0.001, 0.045, 0.001, <0.001 respectively).

Furthermore, Fairbairn et al.[ssssss] Women exhibited a lower prevalence of obstructive

CAD (65.4% vs. 74.7%; $p < 0.0001$) during the CCTA procedure.

Therefore, single-center study making the results less generalizable, lack of studying complications after CTA and its difference regarding gender and lack of evaluation sensitivity of CTA for CAD diagnosis in different gender.

Conclusion

Men exhibit higher coronary atherosclerosis compared to women, these disparities persist even after adjusting for socioeconomic, lifestyle, and traditional risk factors (e.g., hypertension, smoking), suggesting biological mechanisms.

Therefore, multi-center study is recommended, future studies assessing complications after CTA in both sex and future studies evaluating sensitivity of CTA for CAD diagnosis in different gender.

Sources of funding

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Conflicts of interest

No conflicts of interest

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