

## ORIGINAL ARTICLE

# Role of multi-slice CT coronary angiography in evaluating the different patterns of coronary artery disease in patients with unstable angina

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Received 1 December 2014; accepted 28 March 2015

Available online 6 May 2015

### KEYWORDS

Coronary artery disease (CAD);  
Multislice computed tomography (MSCT);  
Coronary artery calcium score (CACS);  
Unstable angina

**Abstract Objective:** To evaluate the different patterns of coronary artery disease among patients with unstable angina by the role of multislice CT coronary angiography.

**Patients and methods:** From September 2013 to May 2014, 40 patients complaining from unstable angina showing initial negative ECG and troponin enzyme underwent a multi-slice CT coronary angiography. Each patient underwent a non-contrast scan to determine the calcium score, then a contrast enhanced ECG gated scan, then the obtained axial images were reconstructed on an advanced workstation. Finally, a systematic analysis of the coronary artery lesions was performed. **Results:** 9 patients had normal CTCA, 5 had dense coronary calcification, 16 had no significant obstructive lesion and 10 patients had significant CAD. A total of 60 coronary vessels were found to have plaques. The number of patients with multi-vessel disease was significantly higher than those with single-vessel disease at the time of diagnosis.

**Conclusion:** Non-invasive multi-slice CT coronary angiography is a reliable technique of high ability to detect coronary artery disease and estimate the degree of obstruction, number of affected arteries and the pattern of their affection and can be used in workup in patients with unstable angina.

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## 1. Introduction

The danger, cost and time burden associated with coronary catheter angiography (CCA) suggests a need to develop a

noninvasive assessment for patients with suspected coronary artery disease (CAD) especially for those with low probability of disease (1).

The socioeconomic importance of heart disease provides considerable motivation for development of radiologic tools for noninvasive imaging of the coronary arteries (2).

However, during the last 10 years, progressive improvements in the spatial resolution of multi-detector CT (MDCT) allow accurate identification of CAD, thereby offering an alternative to CCA (3).

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Peer review under responsibility of Egyptian Society of Radiology and Nuclear Medicine.

<http://dx.doi.org/10.1016/j.ejrnmm.2015.03.013>

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The rapid rise of coronary computed tomographic (CT) angiography from a research application to widely embraced clinical tool over the last decade has very few parallels in medicine. We currently observe a convergence of factors that has the potential of making coronary CT angiography a pivotal cornerstone in cardiovascular disease management, deserving the highest level of attention of our field (4).

Chest pain is a nonspecific symptom that can have cardiac or non-cardiac causes. The term angina is reserved for pain syndromes arising from presumed myocardial ischemia (5).

Many conditions causing chest pain or discomfort, such as an acute coronary syndrome or angina, have a potentially poor prognosis, emphasizing the importance of prompt and accurate diagnosis (6).

Unstable angina is defined as a new onset chest pain or abrupt deterioration in previously stable angina (7).

It is a clinical syndrome between stable angina and acute myocardial infarction. It typically occurs at rest and has a sudden onset, sudden worsening and recurrence over days and weeks (5).

## 2. Patients and methods

A total number of 40 patients with unstable angina were scheduled for elective multislice CT coronary angiography between September 2013 and May 2014. All patients came complaining of recent onset of dyspnea on exertion, fatigue on mild effort or ischemic chest pain (defined as retro-sternal heaviness or squeezing sensation that may radiate to the left arm, neck, back or lower jaw, which could be at rest or precipitated by effort, and relieved by rest or sublingual nitrates).

Also the pretest probability of CAD of the American College of Cardiology (ACC) and American Heart Association (AHA) is assessed for all patients based upon the age, gender and the symptoms.

Patients included in our study are 22 males and 18 females, ranging in age between 34 and 79 years, with a mean age of 58.82 years.

The following risk factors were extracted from the medical notes and patients history:

- Hypertension (72.5%).
- Diabetes (57.5%).
- Hyperlipidaemia (57.5%).
- Smoking (30%).
- Positive family history of CAD (25%) (First degree relative suffering cardiac events under the age of 55 years).

Exclusion criteria:

- Pregnant women.
- Previous MI, CABG, and Percutaneous coronary intervention.
- Irregular heart rate.
- Contraindications to iodine contrast.
- Renal insufficiency (creatinine level  $\geq 1.5$  mg/dl).
- Inability to sustain a breath holds for 8 s.
- Inability to comply with the protocol requirements.

All patients were subjected to the following:

- (1) Full history: Including history of systemic hypertension, DM, hyperlipidaemia, smoking and family history of IHD.
- (2) Revision of previous laboratory and cardiac investigations.
- (3) Patient preparation: All patients were instructed to fast 4–6 h prior to the examination with no discontinuity of their medications.
- (4) Reassurance of the patient was done and all steps of the study were explained in detail to each patient.
- (5) To evaluate patients ability of breath holding for relatively long time, they were required to perform a deep inspiration and to continue to hold their breath without pushing (i.e. Valsalva maneuver). During this trial, the patient was observed for compliance and the electrocardiogram for significant changes.

Patients who could not withhold breathing for the presumed scanning time were instructed to hyperventilate for a period of 5 min and are re-evaluated. In some patients, this action was repeated several times till the patient can withhold breath for the aimed period.

- (6) Mild oral sedation with diazepam (5 mg) was sometimes given 45 min prior to scan in particularly anxious patients.
- (7) Beta blockers were not administrated to all our patients; only those with heart rate above 75 bpm were given beta blockers (50 mg Atenolol) 45 min prior to the examination (contraindications to B blockers are excluded). This leads to increasing the diastolic phase of the cardiac cycle, which facilitates the acquisition process.

### 2.1. Estimating the pretest probability of coronary artery disease

Step 1: Categorize the nature of the chest pain:

- (1) Is the chest pain substernal?
- (2) Are the symptoms precipitated by exertion?
- (3) Is there any prompt relief within 10 min with rest or nitroglycerin?

- Typical angina: all three features are present
  - Atypical angina: any two features are present
  - Nonanginal chest pain: one or no features are present
- Step 2: Estimate the pretest probability of coronary artery disease based on age, gender, and character of chest pain
- Low probability (<10%)
    - Asymptomatic men and women of all ages
    - Women < 50 years with atypical chest pain
  - Intermediate probability (10–90%)
    - Men of all ages with atypical angina
    - Women  $\geq 50$  years with atypical angina
    - Women 30–50 years with typical angina
  - High probability
    - Men  $\geq 40$  years with typical angina
    - Women  $\geq 50$  years with typical angina

### 2.2. Contrast material

A bolus of 70–80 ml of non-ionic contrast (Ultravist 370 Schering, Berlin, Germany, Omnipaque 370 Daiichi Pharmaceutical, Tokyo, Japan or Iopamiro 370 Bracco

Spain), was injected through an 18-gauge canula into an upper limb vein (right ante-cubital vein is preferred when available) with a flow rate of 5–6 ml/s using a programmed dual head power injector pump. A saline chaser bolus was used (50 ml) to washout the contrast from the right side of the heart for better visualization of the middle segment of the right coronary artery.

### 2.3. Scan protocol and parameters

The MDCT datasets were acquired using Definition dual source (Siemens Medical Solutions, Forchheim, Germany) with 300 ms gantry rotation time and 64 multi-detector slices dual source of energy.

- (1) A noncontrast scan was performed to determine the calcium score ( $64 \times 3$  mm, tube current up to 200 mA at 120 kV).
- (2) The contrast-enhanced ECG gated scan was obtained within one single breath-hold (average 10 s,  $64 \times 0.6$  mm, tube current up to 430 mA at 120 kV).
- (3) The obtained axial images reconstructed using different reconstruction techniques on an advanced workstation.

### 2.4. Image acquisition

#### 2.4.1. Scanogram

The patient is positioned supine comfortably on the CT couch with no movement in order to ensure that the planned scan region matches the region actually scanned and that the entire coronary tree is imaged.

Spatial resolution is highest in the center of the scan field, and that is why the patient should be shifted slightly to the right side of the table, so that the heart is as close to the center as possible.

ECG leads are fixed at the four corners of the pericardium. All reconstructions are performed using the retrospective ECG gating. For this technique; an ECG must be recorded simultaneously throughout the duration of the scanning.

A first step in the imaging process is to delineate the limits of the acquisition. This is accomplished by obtaining the scanogram. It yields an AP view of the chest that is used to position the imaging volume of the coronary arteries that extends from the level of the carina down to about 1 cm below diaphragm. The center of the field of view is 2 cm to the left of the dorsal spine on the AP scout.

#### 2.4.2. Calcium score

Analysis and quantification of the coronary artery calcium (CAC) is performed for all patients using ECG prospective triggering with 3-mm slice collimation without contrast administration. Exact determination of the scan range required for subsequent CTCA allowed reduction in radiation exposure. Any area highlighted, which clearly represented CAC, was identified as a region of interest. Sequential axial sections were analyzed. The total calcified area (the score) was then calculated by the workstation software as equivalent Agatston score (8).

If there was extensive calcification (CAC Score > 1000), the CT examination ends at this step. Five of forty patients had

dense calcification and the examination ended for them at this step (Fig. 1).

#### 2.4.3. The acquisition for the CT coronary angiography

It was only performed for 35 patients (5 patients were excluded from the CTA due to high calcium score > 1000).

Semi-automated determination of the starting time using the “Bolus-tracing technique” utilizing the Smart prep program (Bolus tracking, Siemens medical solutions) was utilized in all patients. It entails injection of the whole volume of the utilized contrast material as a one bolus at the pre-determined rate. After a delay of about 10 s from the start of injection (time estimated for the contrast to reach the great vessels of the chest, being variable according to the site of the cannula, rate of injection, body built and heart rate), series of axial images at the aortic root (at the level of the origin of the left main coronary artery) are acquired with an interval of 2 s between subsequent images.

The density within the ascending aorta is monitored in each axial image on a real time base (scan and scan at the same level) while the region of interest (ROI) carefully avoiding the atheromatous calcifications. Time-attenuation curves were generated. When the density within the ascending aorta exceeds 100 HU (i.e. the contrast started to arrive), the scanning is triggered with a delay of further 3 s (time needed for the table movement to the cranial start position while the patient is instructed to hold breathing).

This time delay also allows for increase in the contrast concentration at the ascending aorta and coronary arteries. It is to be noted that the axial images taken are of low radiation dose with a 120 kV and 40 mA s (not of diagnostic value). This is to reduce the radiation exposure.

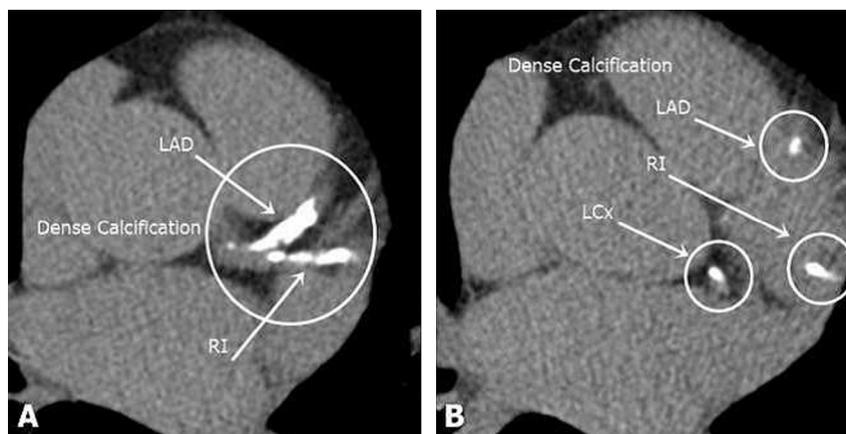
Finally, the volume data set for coronary artery visualization is acquired in a spiral mode. During the helical scan, the ECG signal was recorded digitally. Patients were automatically instructed to maintain an inspiratory breath hold while the CT data and the ECG trace were acquired. The Distance between the level of the carina and the base of the heart about (120 mm) was covered in approximately 10 s.

Bolus timing procedures and main acquisitions were performed in all patients with no clinically important adverse reactions to contrast material. Despite that the CT scan is completed within a couple of minutes, the total examination time (including the patient preparation and instruction) was around 20 min.

#### 2.4.4. Image reconstruction

The reconstructed axial images at different points of the cardiac cycles are sent to an off-line workstation (Advanced workstation syngo via Siemens medical solutions, Germany; for images acquired via the definition dual source 64-multichannel system).

For analysis of the small and tortuous coronary arteries, it is of utmost importance to keep the reconstructed slice thickness for the coronary axial slices as thin as possible. A slice thickness of 0.6 mm reconstructions was used. The reconstruction of images at the workstation is a time-consuming process, aimed to select the most appropriate set of images for coronary artery visualization, avoiding those reconstructions with artifacts due to cardiac motion.



**Fig. 1** Non-enhanced CT image (A and B) Axial Ca Scoring CT image revealed: The total calcium score for all coronary arteries is 1499, represented by multiple calcific plaques along the territories of all coronary arteries, especially in LT system, the CTCA ended at this step.

As a general rule in the CT definition of Siemens, the optimum phase of the cardiac cycle when all the coronary arteries are best visualized is *the best diastole phase* (usual range is 70–80% of the R–R interval of the cardiac cycle) in case of heart rate less than 75 b/m while it is *the best systole phase* (usual range is 40–50% of the R–R interval) in case of heart rate more than 75 b/m.

The ECG recorded during the acquisition examined to look for exaggerated cycle variability and/or premature beats that could introduce artifacts in the images. For the aim of visualization of a lesion like/artifact in any segment of the coronary arteries, the whole cardiac phases are examined in an attempt to prove artifact and exclude true lesion.

Multiple post-processing techniques were used as automatic (and manual) volume rendering techniques (VRT, which were generally used to ascertain the morphology and course of the vessels), Multiplanar Reconstructions (MPR) and Maximum Intensity Projections (MIP).

Identification of coronary artery segments was based on the model suggested by the American Heart Association (AHA) (Fig. 2), but the obtuse marginal branches of LCx (segment 14 & 15) are not included in statistics of this study owing to their usual small size.

### 2.5. Analysis of coronary artery lesions

Systematic analyses of a coronary artery MDCT study took into consideration the following steps:

- (1) Analysis of images reconstructed from different phases of the cardiac cycle, in order to choose those where the coronary arterial tree is best filled with contrast and where movement artifacts are the least.
- (2) A complete review of axial images that constitute the cardiac volume, paying attention to cardiac anatomy, degree of opacification of chamber and walls of the heart, and aspect of extracardiac structures.
- (3) Optimization of images aimed to improve the visualization of coronary arteries, by using specific post-processing protocols.
- (4) Analysis of the coronary artery tree, for which is fundamental the following systematization:

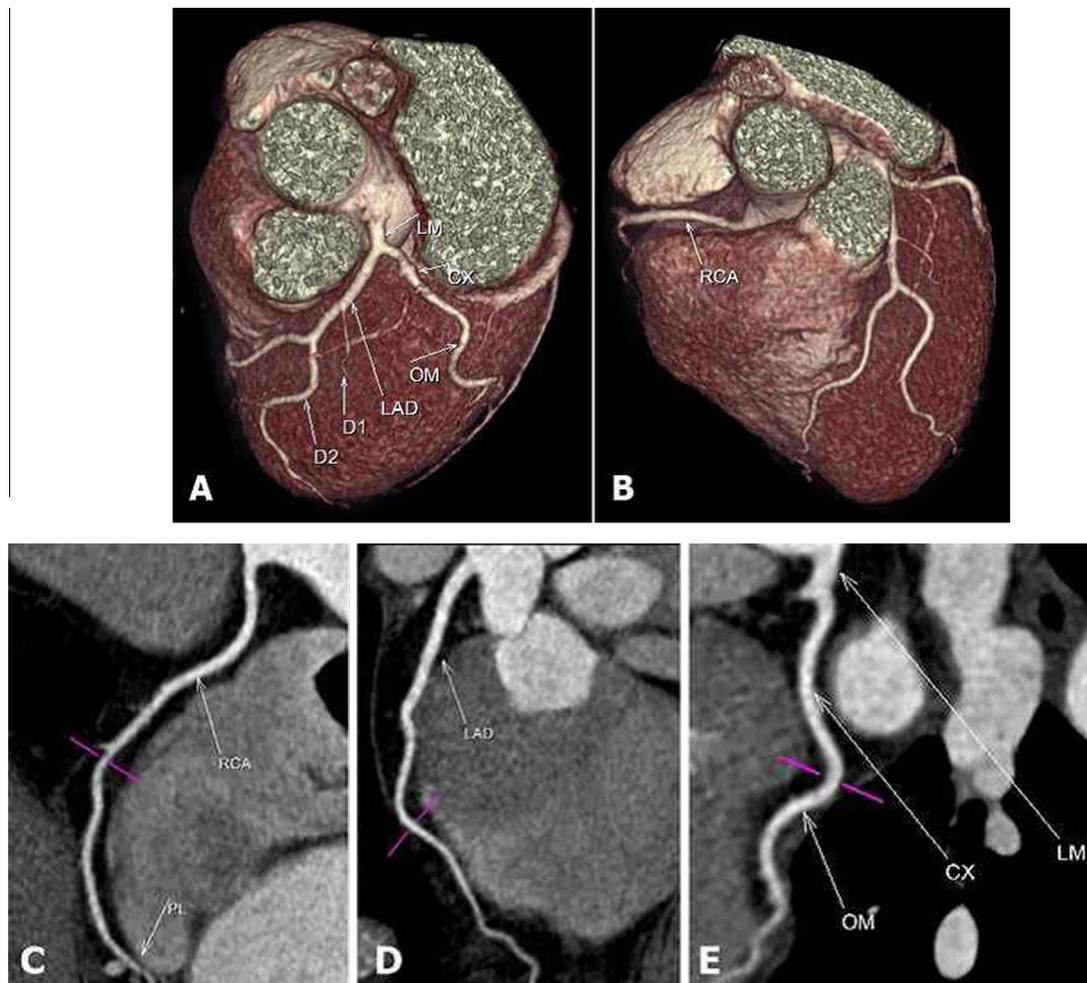
- Examination of the anatomical distribution of coronary arteries, aimed to identify normal variants and congenital abnormalities of the origin of vessels.
- Detection and localization of coronary artery lesions, carefully avoiding sections and angulations or interposed structures with potential image artifacts.
- Evaluation of composition and morphology of the lesion. In regard to the composition of the plaque, a distinction was made between calcified and non-calcified plaques. Plaques with a mean attenuation of 130 HU or greater were graded as calcified, whereas plaques with a mean attenuation of less than 130 HU were graded as non-calcified. Calcified plaques were identified on nonenhanced scans, and non-calcified plaques were identified on contrast-enhanced scans.
- Qualitative and quantitative assessment of obstruction of the vessel caused by the lesion.

A classification of atherosclerotic coronary artery lesions is possible by applying this systematic analysis of MDCT. This classification can be made according to the following aspects:

- The number of vessels involved.
- The location: proximal, middle or distal portions of the vessel.
- The extension of the lesion: focal or diffuse.
- The degree of obstruction.
  - a. Non-significant stenosis (less than 60 % of the vessel lumen, including mild and moderate degrees of obstruction).
  - b. Significant stenosis (equal or more than 60 %, including critical subocclusive and occlusive lesions).
- The components of the lesion:
  - a. Non-calcified, mixed, or “soft” lesions.
  - b. Calcified lesions: The calcium component of the lesion can be focal, diffuse, eccentric or concentric. (Fig. 3).

### 3. Results

A total number of 40 patients with unstable angina (recent onset chest pain) (22 males and 18 females) were referred to



**Fig. 2** (A and B) Colored 3D VRT image, (C–E) Curved MPR images showing normal distribution of the different coronary artery segments. LM: Left Main Trunk, CX: Circumflex artery, OM: Obtuse Marginal artery, LAD: Left Anterior Descending artery, D1, D2: Diagonal branches, RCA: Right Coronary Artery.

perform CTCA. A 72.5% of patients were hypertensive, 57.5% were diabetic, 57.5% had hyperlipidaemia, 30% were smokers, and only 25% had positive family history of ischemic heart disease (IHD).

### 3.1. Frequency of CAD among patients complaining from unstable angina (recent onset chest pain)

Among 40 patients, 9 (22.5%) had normal CTCA, 5 (12.5%) had dense coronary calcification, 16 (40%) had no significant obstructive lesion, and 10 (25%) had significant CAD (Table 1).

There is a strong association and concomitance of CAD with risk factors.

□ 23 (74.2%) of CAD were hypertensive (60% of them had significant obstructive lesion), while 8 (25.8%) are normotensive (40% of them had significant stenosis),  $P$  value < 0.05.

□ 18 (58%) were diabetic (70% of them had significant lesion), while 13 (42.8%) are not diabetic (30% of them had significant stenosis),  $P$  value < 0.05.

□ 7 (22.6%) had positive family history of IHD (20% of them had significant stenosis), while 24 (77.4%) had negative family history of IHD (80% of them had significant obstruction),  $P$  value > 0.05. (Fig. 4).

### 3.2. Coronary artery calcium (CAC)

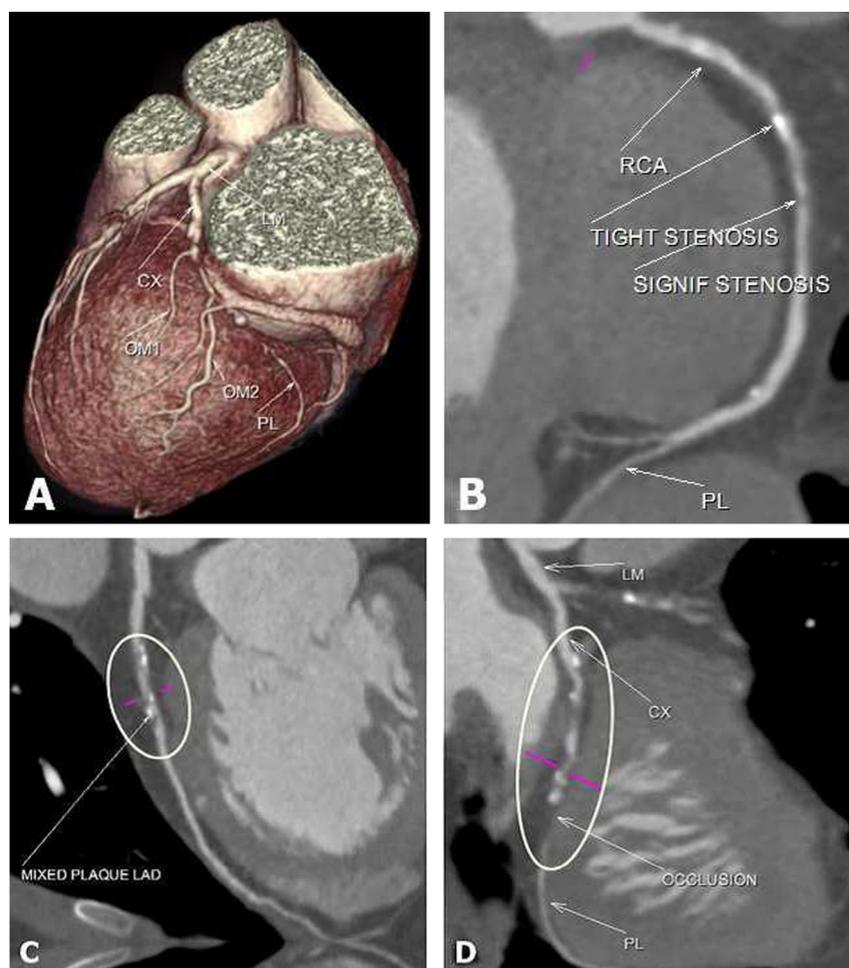
Among 9 patients had CAC Score = 0, there was one patient (11.2%) had CAD (not significant), while 8 (88.8%) had normal CTCA.

A 26.5% had significant CAD in CACS = 1–99, 55.6% had significant CAD in CACS = 100–399, and 100% had significant CAD in calcium score  $\geq 400$  (Fig. 5).

Male patients show higher level of CAC compared to female patients (average is 469.3 in males vs. 232.3 in females,  $P$  value < 0.05) (Table 2).

Patients  $\geq 60$  years old had a strong statistical significance of higher CAC than those < 60 years (Mean 444.4 Vs 66.2,  $P$  value < 0.05) (Table 3).

Male patients < 60 years old also have statistically significant higher CAC compared to females of the same age group



**Fig. 3** Contrast-enhanced CT image: (A) Colored 3D VRT image, (B–D) Curved MPR images revealed: o LMT: Normal trunk that bifurcates into LAD and CX. o LAD: Proximal segment diffusely affected by multiple mixed atherosclerotic plaques that cause variable degree of stenosis reaching to significant stenosis distally, producing more than 70% lumen reduction. o CX: The middle segment diffusely affected by multiple mixed (predominantly soft) plaques that cause variable degree of stenosis reaching to subtotal occlusion at its most distal segment. It supplies two patent OM branches, free of atherosclerotic changes. o RCA: Dominant artery, the proximal and middle segments show multiple plaques; some of them are soft while the other are mixed, and they caused mild irregularity at middle segment, while its middle segment shows two focal significant stenoses reaching more than 70% lumen reduction. The distal segment appears patent and free of atherosclerotic changes.

**Table 1** The findings of CTCA.

Patients	Findings			
	Normal CTCA	Dense coronary calcifications	Significant CAD	No significant obstructive lesions
Number	9	5	10	16
%	22.5	12.5	25	40

(mean 105.47 in male Vs 26.9 in female,  $P$  value < 0.05) (Table 4).

No significant differences in CAC between male and female  $\geq 60$  years old (Mean 414.66 Vs 489,  $P$  value > 0.05) (Table 5).

Hypertensive patient shows similar CAC of normotensive patients, while Diabetic patients show statistically significant

higher CAC compared to Nondiabetic patients (328 Vs 179.9,  $P$  value > 0.05) (Table 6).

### 3.3. Finding of CTCA in relation to pretest probability

Patients were classified into three groups based on pretest probability regarding age, gender, and character of chest pain to Low, Intermediate, and High probability of CAD.

15% of patients had low probability, 67% had intermediate probability, and 18% had high probability for CAD and 34% of patients with low pretest probability had CAD (17% significant and 17% nonsignificant), while 66% of patient had normal CTCA. 81.5% of patients with intermediate pretest probability had CAD (37% significant and 44.5% nonsignificant), while 18.5% had normal CTCA.

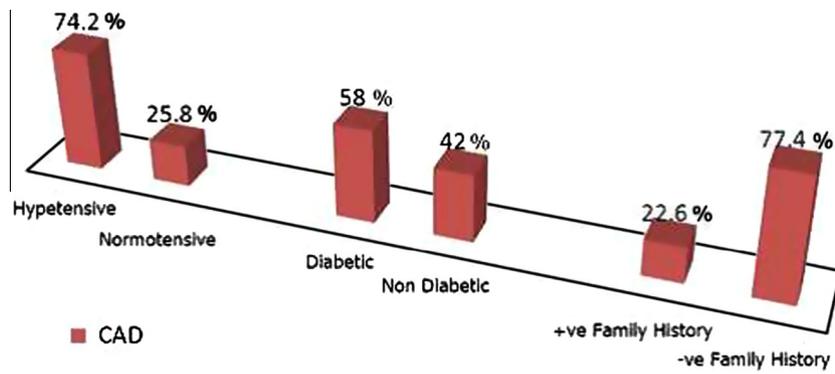


Fig. 4 Represents CAD in correlation to Risk factors.

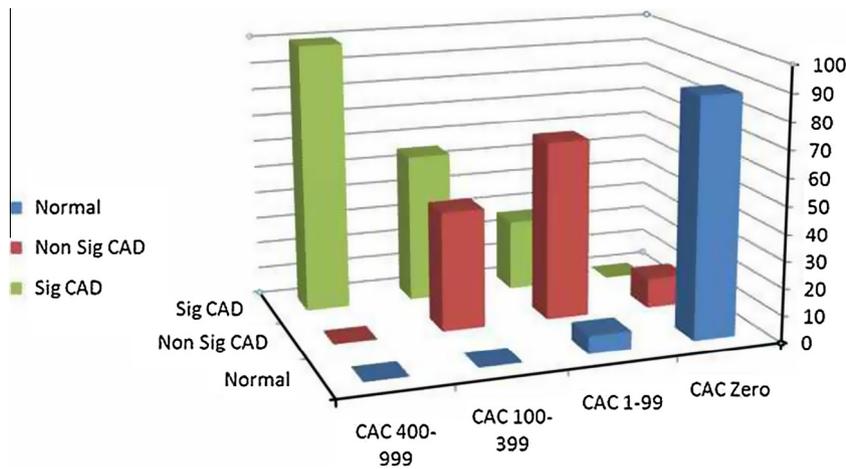


Fig. 5 Represents the frequency of CAD in patients regarding CAC score.

Table 2 Ca scoring in relation to sex.

Sex	Number	CAC
Male	22	469.3
Female	18	232.3

Table 3 Ca scoring in relation to age.

Age group	Number	Mean CAC	P value
< 60	20	66.2	< 0.05
≥ 60	20	444.4	

Table 4 Ca scoring in relation to sex below 60 years old.

Sex	Number	Mean CAC	P value
Male	10	105.47	< 0.05
Female	10	26.9	

All patients with high pretest probability had CAD, and 57% of them had significant CAD, while 43% had no significant CAD (Fig. 6).

3.4. CTCA findings in relation to coronary arteries

CT Angiography was performed in only 35 patients, because the other 5 patients had dense coronary calcification (CAC > 1000 that impedes the technique).

Among the 10 patients who had significant stenosis (≥ 60%), the number of affected arteries was 33 (2 in LM, 10 in LAD, 7 in LCX, 9 in RCA and 5 in smaller branches) as shown in Table 7.

Among the 16 patients who had no significant CAD, the number of affected arteries was 27 (14 in LAD, 2 in LCX, 9 in RCA and 2 in smaller branches).

A total of 60 coronary vessels (1.7 ± 1.3 per patient; range, 1–4) were found to have plaques. The most commonly affected vessel was the LAD artery [40%] (LAD 24/60) vs. 30% (RCA 18/60), 15% (LCX 9/60) and 3.4% (LM 2/60); all P value < 0.05.

Patients with multivessel affection have a statistically significant higher incidence of significant stenotic lesions compared to single vessel affection (P value < 0.05) (Table 8).

No significant relation was seen between the numbers of affected vessels and type of affection (P value > 0.05) (Table 9).

Diabetic patients show a statistically significant higher incidence of multivessel affection than nondiabetic patients (85.7% vs. 75%, P value < 0.05), while no such significance

**Table 5** Ca scoring in relation to sex  $\geq 60$  years old.

Sex	Number	Mean CAC	P value
Male	12	414.66	< 0.05
Female	8	489	

**Table 6** CAC in relation to risk factors.

	Mean CAC	P value
Normal patients	0.3	
Normotensive patients	275.73	> 0.05
Hypertensive patients	237.00	
Diabetic patients	328.00	< 0.05
Nondiabetic patients	179.90	

was seen between hypertensive and normotensive patients (*P* value > 0.05) (Table 10).

### 3.5. Type of plaques

Among 60 lesions, calcific plaques were the most common which were 31 (51.5%), then mixed plaques were 36 (43.5%), while soft plaques represented only 3 (5%).

Among 33 significant lesions (causing  $\geq 60\%$  luminal reduction), there were 19 (57.5%) mixed plaques, 11 (33.4%) calcific plaques, and 3 (9.1%) soft plaques, with strong statistical significance (*P* value < 0.05) (Fig. 7).

## 4. Discussion

In the present study we found that the frequency of CAD among all patients complaining from recent chest pain was 77.5%. Among 40 patients complaining of unstable angina we found 9 (22.5%) having normal CTCA, 5 (12.5%) having dense coronary calcification, 16 (40%) having no significant obstructive lesions, and 10 patients (25%) having significant CAD.

Zero calcium score was detected in 9 cases (22.5%) including one case (11.2%) showing CAD.

**Table 7** Distribution of the lesions through coronaries and their degree of stenosis, SD: Standard Deviation.

	Degree of stenosis		Total	%	SD
	$\geq 60\%$	< 60%			
Number of patients	10	16	26		
Artery	33	27	60	100	1.7 $\pm$ 1.3
LM	2	0	2	3.4	
LAD	10	14	24	40	
LCX	7	2	9	15	
RCA	9	9	18	30	
Others	5	2	7	11.6	

**Table 8** The relation between number of vessel affection and the degree of stenosis.

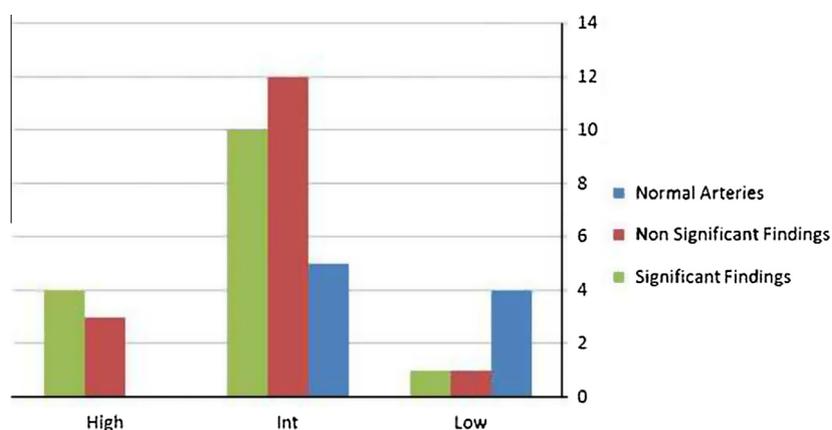
	Single V. Affection	Multi V. Affection	P value
Stenosis < 60%	5	11	< 0.05
Stenosis $\geq 60\%$	0	10	

**Table 9** The relation between number of vessel affection and the type of affection.

	Single V. Affection	Multi V. Affection	P value
Focal	3	13	> 0.05
Diffuse	2	8	

A 26.5% significant CAD in CACS = 1–99, 55.6% significant CAD in CACS = 100–399, and 100% significant CAD in calcium score  $\geq 400$ .

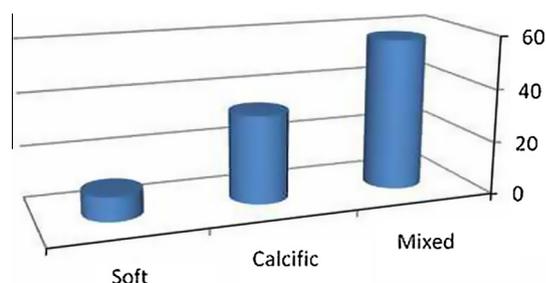
In 2012, Koulaouzidis et al. in their study “CTCA as Initial Work-Up for Unstable Angina Pectoris” found that among 43 patients there were 26 (60.5%) having CAD. Among 43 patients, 17 (39.5%) had normal CTCA, 22 (51.1%) had no significant lesion (ranging from minimal to moderate degree



**Fig. 6** Represents the percentage of CTCA finding in respect to pretest probability based on age, gender, and character of pain.

**Table 10** The number of vessel affection in diabetic/nondiabetic and hypertensive/normotensive patients.

	Single V. Affection	Multiple V. Affection	P value
Diabetic	2(14.3%)	12(85.7%)	<0.05
Nondiabetic	3(25%)	9(75%)	
Hypertensive	5(26.3%)	14(73.7%)	>0.05
Normotensive	0(0%)	7(100%)	

**Fig. 7** Represents the types of plaque that caused significant stenosis.

of luminal reduction (<60%), and only 4 (9.3%) had a significant obstructive lesion (7).

In 2011, Villines et al. found in their study about the prevalence and severity of CAD and adverse events among symptomatic patients with CAC scores of zero that 13% of their patients had nonobstructive stenosis, 3% had  $\geq 50\%$  stenosis, and 84% had normal CTCA (9).

In the present study we found 2 (34%) patients with low pretest probability having a CAD (17% significant and 17% nonsignificant), while 4 (66%) patients having normal CTCA. Twenty-two (81.5%) patients with intermediate pretest probability had CAD (37% significant and 44.5% nonsignificant), while 5 (18.5%) had normal CTCA.

All patients with high pretest probability had CAD, and 4 (57%) of them had significant CAD, while 3 (43%) had no significant CAD. This is exactly within the range of the *National Institute for Health and Clinical Excellence (NICE) guidelines for "Chest pain of recent onset"* published in 2010: low (1–30% probability), intermediate (31–70%), or high (71–99%) (6).

In 2012, Koulaouzidis et al. also found that among 21 patients with low pre-test probability, 14 (66.7%) patients had normal CTCA and 7 (33.3%) patients had no significant CAD. Fourteen patients had an intermediate pre-test probability of which 3 (21%) had normal CTCA, 9 (64%) had evidence of no significant CAD and 2 (15%) had obstructive CAD. Finally, among the 15 patients with a high pre-test probability, all patients had atherosclerotic disease but 13 patients (86.5%) had no significant obstructive lesions, while 2 patients (13.5%) had significant lesions (7).

In the present study, a total of 60 coronary vessels ( $1.7 \pm 1.3$  per patient; range, 1–4) were found to have plaques. The most common affected vessel was the LAD artery [40% (LAD 24/60) vs. 30% (RCA 18/60), 15% (LCX 9/60) or 3.4% (LM2/60); all  $p < 0.05$ ]. The number of patients with multi-vessel ( $\geq 2$ ) disease was significantly higher than that

with single-vessel disease at the time of diagnosis in our patients [21 of 26 (80.7%) vs. 5 of 26 (19.3%),  $p < 0.05$ ].

Also we found among 33 significant lesions (cause  $\geq 60\%$  luminal reduction), there were 19 (57.5%) with mixed plaques, 11 (33.4%) with calcific plaques, and 3 (9.1%) with soft plaques, with strong statistical significance ( $P$  value < 0.05).

In 2010, Chu et al. also found that a total of 287 coronary vessels ( $2.5 \pm 1.1$  per patient; range, 1–4) and 470 segments ( $4.2 \pm 2.8$  per patient; range, 1–13) were found to have plaques. The number of patients with multi-vessel ( $\geq 2$ ) disease was significantly higher than that with single-vessel disease [85 of 113 (75.2%) vs. 28 of 113 (24.8%),  $p < 0.001$ ]. The most common diseased coronary vessel was the LAD artery [35.9% (LAD) vs. 27.2% (RCA), 22.6% (LCX) or 14.3% (LM); all  $p < 0.001$ ]. Furthermore, its proximal segment was the most commonly diseased coronary segment (90/470, 19.1%,  $p < 0.001$ ), followed by the proximal segment of RCA (63/470, 13.4%) and the middle segment of the LAD artery (62/470, 13.2%) (10).

In the present study we found a strong association of CAD with hypertension as 23 patients (74.2%) were hypertensive (60% of them had significant obstruction), while 8 patients (25.8%) were normotensive (40% of them had significant obstruction),  $P$  value < 0.05. Similarly, Zeina et al. in 2009 found that 82% of their patients with CAD were hypertensive and 72% were normotensive. Obstructive CAD was as twice as common in hypertensive patients (11).

In the present study, CAD was found more in diabetic patients 18 (58%) than nondiabetic patients 13 (42.8%). Also significant stenotic lesions were more in diabetic (70%) than nondiabetic patients (30%). Exactly as Bartnik et al., in their prevalence study of patients with CAD across Europe in 2004, they found that 58% of patients had DM, 36% of them had impaired glucose regulation and 22% were newly diagnosed (12).

In the present study we found no relation between positive family history of IHD and CAD as 7 patients (22.6%) had positive family history of IHD; this seems to be unreal, mainly due to improper information by the patients. Otaki et al. in 2013 found that young patients with positive family history have higher presence, extent, and severity of CAD, which are associated with increased risk for myocardial infarction. Compared with other clinical CAD risk factors, positive family history is the strongest clinical predictor of future myocardial infarction (13).

In the present study, we found that patients  $\geq 60$  years old had a statistically significant higher CAC score than those < 60 years old (Mean 444.4 vs. 66.185, and  $P$  value < 0.05), and also male patients < 60 years old had a statistically significant higher CAC score compared to female patients of the same age group (mean 105.47 in males vs. 26.9 in females, and  $P$  value < 0.05), while no difference in CAC score between males and females  $\geq 60$  years old (Mean 414.66 vs. 489 and  $P$  value > 0.05). In 2010, Chu et al. found that CT findings of CAD between men and women were almost similar in all aspects except that men had more calcified plaques ( $p < 0.05$ ) (10).

In the present study, we found that diabetic patients show a statistically significant higher CAC score compared to Nondiabetic patients (328 vs. 179.9,  $P$  value < 0.05).

In 2005, Chahal et al. found overall 40.4% of patients were aged < 55 years and 53.7% between 55–70 yrs. In both age

groups the prevalence of elevated CAC score was significantly greater in diabetic patients than non-diabetes patients (14).

In 2011, Maffei et al. also found that diabetics had a higher prevalence of coronary artery atherosclerosis (per-patient) compared to non-diabetics: 80% ( $n = 118$ ), vs. 58% ( $n = 567$ ), respectively ( $p < 0.0001$ ); and a higher prevalence of obstructive coronary disease: 37% ( $n = 55$ ), vs. 18% ( $n = 173$ ), respectively,  $p < 0.0001$  (15).

In the present study, we found that diabetic patients show a statistically significant higher incidence of multivessel affection than nondiabetic patients (85.7% vs. 75%,  $P$  value  $< 0.05$ ), while no such significance was seen between hypertensive and normotensive patients ( $P$  value  $> 0.05$ ).

In 2010, Van Werkhoven et al. also found that diabetic patients showed a higher average number of diseased coronary segments (5.6 vs. 4.4,  $P = .001$ ), with either obstructive (1.7 vs. 1.2,  $P = .01$ ) or non-obstructive (3.9 vs. 3.1,  $P = .005$ ) CAD (16).

## 5. Conclusion

Non-invasive multi-slice CT coronary angiography is a reliable technique of high ability to detect CAD and estimate the degree of obstruction, number of affected arteries and the pattern of their affection and can be used in workup in patients with unstable angina.

## Conflict of interest

None.

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