International Journal of Cardiology Sciences

**ISSN Print:** 2664-9020
**ISSN Online:** 2664-9039
**Impact Factor:** RJIF 5.63
IJCS 2025, 7(2): 80-86
www.cardiologyjournals.net
Received: 14-06-2025
Accepted: 17-07-2025

**Hany Hassan Ebaid**
Department of Cardiology, Faculty of Medicine, Benha University, Benha, Egypt

**Ghada Mohammed Hassan**
Department of Cardiology, Faculty of Medicine, Benha University, Benha, Egypt

**Mohammed Ahmed Hamoda**
Department of Cardiology, Faculty of Medicine, Benha University, Benha, Egypt

**Ahmed Mohammed Abd El Monem**
Department of Cardiology, Faculty of Medicine, Benha University, Benha, Egypt

**Corresponding Author:**
**Hany Hassan Ebaid**
Department of Cardiology, Faculty of Medicine, Benha University, Benha, Egypt

# Non-invasive predictors of coronary artery ectasia

**Hany Hassan Ebaid, Ghada Mohammed Hassan, Mohammed Ahmed Hamoda and Ahmed Mohammed Abd El Monem**

**DOI:** https://www.doi.org/10.33545/26649020.2025.v7.i2b.122

**Abstract**
**Background:** Coronary artery ectasia (CAE), defined as dilatation ≥1.5 times the adjacent normal coronary segment, is an uncommon angiographic finding with reported prevalence of 0.3-4.9%. Although often considered non-obstructive, CAE carries significant thrombotic risk and adverse outcomes, highlighting the need for reliable non-invasive predictors.
**Objectives:** This research aims to research the risk factors and non-invasive predictors of CAE among the cases presented or manifested by coronary artery disease (CAD).
**Methods:** This prospective cohort was conducted at Banha University and Al-Matria Teaching Hospital including 270 cases, 67 controls and 203 cases with acute coronary syndrome (ACS) undergoing coronary angiography were divided into: Group 1 (CAE, n=96, subgroups 1a obstructive n=20, 1b non-obstructive n=76), Group 2 (obstructive CAD, n=107), and Group 3 (controls with normal coronaries, n=67). Data collection included demographics, cardiovascular risk factors, laboratory markers (lipids, creatinine, ALT, AST, Hb), cardiac enzymes, echocardiography (LVEF), and angiographic parameters.
**Results:** Age and sex were comparable across groups, but hypertension (75.0%) and diabetes mellitus (66.7%) were significantly more frequent in CAE compared with CAD (59.8%, 57.0%) and controls (46.3%, 32.8%) ($p \leq 0.001$). ACS presentation varied ($p<0.001$): unstable angina predominated in controls (100%), non-ST elevation myocardial infarction (NSTEMI) in CAE (37.5%) and CAD (44.9%), while STEMI was largely confined to CAD (32.7%). CAE cases had severe dyslipidemia: cholesterol 296.5 vs 214.5 vs 157.5 mg/dL, triglycerides 231.0 vs 156.0 vs 98.5 mg/dL, LDL 207.5 vs 144.0 vs 97.0 mg/dL, LDL/HDL 6.4 vs 3.6 vs 2.1 (all $p<0.001$). Cardiac enzymes were highest in CAD, lowest in controls, with CAE in between, LVEF was lowest in CAD (50%) and highest in controls (59%) ($p<0.001$). Angiographically, CAE showed single-vessel predominance (84.4%) with RCA (39.6%) and LCX (21.9%) as most affected ($p<0.001$). Obstructive CAE was linked to STEMI (55.0%), longer ectasia (3.8 vs 2.3 cm), more ectatic sites, and thrombus (35.0% vs 0%, $p \leq 0.001$).
**Conclusions:** CAE is more often associated with unstable angina, whereas obstructive CAD commonly presents with STEMI and NSTEMI. CAE cases exhibit marked metabolic abnormalities as opposed to CAD, and obstructive ectasia cases show greater STEMI prevalence, diffuse and longer ectatic segments, more thrombus, and higher ejection fraction. The right coronary artery is the vessel most frequently involved.

**Keywords:** Coronary artery ectasia, LDL/HDL ratio, Non-invasive predictors, Dyslipidemia, Acute coronary syndrome

## Introduction

Coronary artery ectasia (CAE) is abnormal dilatation of a coronary artery measuring at least 1.5 times greater than the diameter of an adjacent angiographically normal segment [1]. This vascular abnormality may present either as a localized lesion or as a diffuse enlargement. The reported incidence of CAE in cases undergoing coronary angiography ranges between 0.3% and 4.9%. The aneurysmal dilatation characteristic of CAE alters hemodynamics, leading to blood stasis, increased viscosity, and turbulent flow, which may in turn precipitate acute myocardial infarction (MI) even in the absence of complete coronary artery occlusion [2].

Findings from the Coronary Artery Surgery Research indicated that cases with CAE and concomitant coronary artery disease (CAD) had a five-year mortality rate of 25-29.1% [3]. The high thrombogenic potential of ectatic arteries remains a significant clinical challenge for interventional cardiologists, despite the partial benefit achieved with anticoagulant

therapy. Moreover, indirect evidence, particularly from studies of familial hypercholesterolemia, has suggested a link between plasma lipoprotein abnormalities and CAE progression [4].

CAE has also been associated with systemic and connective tissue disorders like Marfan syndrome, systemic lupus erythematosus, and Takayasu arteritis. In rare circumstances, it has been linked to congenital anomalies, trauma, cocaine abuse, or Kawasaki disease [5].

Dyslipidemia, defined as elevated plasma concentrations of total cholesterol, low-density lipoprotein cholesterol, or triglycerides, or reduced levels of high-density lipoprotein cholesterol, is recognized as a major risk factor for ischemic heart disease (IHD). Globally, elevated LDL-C is estimated to account for approximately 1/3 of deaths attributed to IHD and ischemic stroke [6].

Given these associations, the present research was designed to evaluate risk factors and identify non-invasive predictors of CAE among cases presenting with clinical manifestations of CAD.

## Patients and methods

### Design and population

This prospective cohort investigation enrolled a total of 270 participants, including 203 cases diagnosed with acute coronary syndrome (ACS) and 67 controls, who were admitted to the cardiology departments of Banha University Hospital and Al-Matria Teaching Hospital between August 2021 and August 2024. Ethical approval was granted by the Research Ethics Committee of Benha University (Approval No. …), and all enrolled subjects provided written informed consent prior to participation.

### Patient Selection

Inclusion criteria specified adult cases aged ≥18 years, admitted with ACS, and referred for coronary angiography. ACS diagnoses included ST-segment elevation MI (STEMI), unstable angina (UA), non-STEMI (NSTEMI). UA was defined as ischemic chest pain occurring at rest or with minimal exertion, lasting longer than 15 minutes, unrelieved by rest or nitroglycerin, and unaccompanied by cardiomyocyte necrosis [7]. NSTEMI was characterized by acute chest discomfort, absence of persistent ST-segment elevation, and a rise/fall of cardiac troponins exceeding the 99th percentile. STEMI was diagnosed by persistent ST-segment elevation at the J-point in two or more contiguous leads, using established age- and sex-specific criteria, in conjunction with ischemic symptoms and/or elevated troponin levels [9].

Exclusion criteria encompassed cases younger than 18 years, individuals with prior coronary artery bypass grafting (CABG), malignancy, chronic liver disease (progressive dysfunction >6 months), chronic kidney disease (eGFR <90 ml/min/1.73 m² for >3 months) [10,11], or systemic inflammatory disorders that could bias CAE evaluation.

### Groups

Cases were classified into three angiographic groups. Group 1 included 96 subjects with CAE, defined as coronary arterial dilatation ≥1.5 times the diameter of a reference normal segment [12]. This cohort was further divided into subgroup 1a, consisting of CAE cases with associated obstructive lesions, and subgroup 1b, consisting of CAE without obstruction. Group 2 consisted of 107 cases diagnosed with obstructive CAD, defined as ≥70% luminal narrowing of a major epicardial artery or branch, or ≥50% narrowing of the left main coronary artery [13]. Group 3 comprised 67 control subjects with angiographically normal coronary arteries, free from stenosis or ectasia.

## Methods

### Demographics and clinical characteristics

All participants underwent thorough history taking and clinical examination, with emphasis on ischemic symptomatology, hypertension, diabetes mellitus (DM), prior ischemic heart disease, and family history of CAD. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic pressure ≥90 mmHg, or current antihypertensive treatment [14]. DM was diagnosed based on fasting plasma glucose ≥126 mg/dl, post-prandial glucose ≥200 mg/dl, HbA1c ≥6.5%, or ongoing antidiabetic therapy [15]. A family history of CAD was considered positive when a first-degree male relative <55 years, or a female relative <65 years, was affected. Cardiovascular and systemic physical examinations were performed for all cases.

### Investigations

Investigations included 12-lead electrocardiography to detect ischemic alterations, laboratory assessments (cardiac biomarkers, renal and hepatic function tests, lipid profile, and hemoglobin concentration), and transthoracic echocardiography. Echocardiographic measurements adhered to the recommendations of the American Society of Echocardiography [16] and left ventricular ejection fraction (LVEF) was calculated using the modified Simpson's method [17]. Diagnostic coronary angiography was performed via radial or femoral access, utilizing multiple projections to fully delineate vessel morphology [18].

CAE was further classified according to angiographic criteria. Vessel diameter was categorized as small (<5 mm), medium (5-8 mm), or giant (>8 mm). Morphology was defined as diffuse when the dilatation extended across the majority of the vessel length, or focal when limited to a short segment. Shape was categorized as saccular when the transverse diameter exceeded the longitudinal dimension, or fusiform when the longitudinal diameter predominated [1].

Additional parameters documented included anatomical location (right coronary artery [RCA], left anterior descending [LAD], left circumflex [LCX], or multivessel involvement), presence of slow flow (angiographic evidence of dye stasis reflecting microvascular dysfunction) [19], and intraluminal thrombus (plaque rupture with flow-limiting obstruction) [20].

### Statistical methods

Statistical analyses were performed using SPSS software, version 28 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was first evaluated through the Kolmogorov-Smirnov test. Data conforming to normal distribution were summarized as mean values with standard deviations (SD), whereas skewed data were reported as median with range. Categorical variables were described in terms of frequencies and percentages. For comparisons across the three study groups, one-way analysis of variance (ANOVA) was applied to normally distributed data, while the Kruskal-Wallis test was used for non-parametric variables; significant findings were further explored with post-hoc pairwise testing. Associations

between categorical variables were assessed using either the Chi-square test or Fisher's exact test, depending on distributional assumptions. To determine independent predictors of CAE, binary logistic regression modeling was employed. A probability value of ≤0.05 was accepted as the threshold for statistical significance.

## Results

Analysis of baseline characteristics demonstrated no statistically significant differences across the three groups with respect to age distribution or gender. The prevalence of hypertension and diabetes mellitus, however, was significantly higher among Group 1 (CAE) cases compared with Groups 2 (obstructive CAD) and 3 (controls). No significant differences were observed between the groups regarding smoking status, previous ischemic heart disease, or family history of CAD.

In terms of ACS presentation, UA was most frequent in Group 3 (controls), whereas NSTEMI predominated in Groups 1 (CAE) and 2 (obstructive CAD). STEMI was encountered predominantly in Group 2 (obstructive CAD). The distribution of ACS subtypes differed significantly among the groups, with overall comparisons demonstrating high statistical significance (p <0.001).

**Table 1:** Demographics, risk factors, and clinical data of the patients.

| Parameters | Group 1 (n=96) | Group 2 (n=107) | Group 3 (n=67) | p |
|---|---|---|---|---|
| Age | 55.4 ± 8.9 | 57.5 ± 8.4 | 54.4 ± 7.8 | 0.344 |
| Gender, Male | 67 (69.8%) | 64 (59.8%) | 49 (73.1%) | 0.139 |
| HTN | 72 (75.0%) | 64 (59.8%) | 31 (46.3%) | 0.001* |
| DM | 64 (66.7%) | 61 (57.0%) | 22 (32.8%) | <0.001* |
| History of IHD | 1 (1.0%) | 4 (3.7%) | 2 (3.0%) | 0.470 |
| Family History of IHD | 31 (32.3%) | 29 (27.1%) | 25 (37.3%) | 0.361 |
| Smoking | 58 (60.4%) | 61 (57.0%) | 38 (56.7%) | 0.854 |
| Unstable Angina | 49 (51.0%) | 24 (22.4%) | 67 (100%) | <0.001* |
| NSTEMI | 36 (37.5%) | 48 (44.9%) | 0 (0%) | <0.001* |
| STEMI | 11 (11.5%) | 35 (32.7%) | 0 (0%) | <0.001* |

Data were presented as Mean ± SD or n (%), SD: Standard deviation, HTN: Hypertension, DM: Diabetes Mellitus, IHD: Ischemic Heart Disease, NSTEMI: Non-ST-Segment Elevation Myocardial Infarction, STEMI: ST-Segment Elevation Myocardial Infarction, *Statistically significant p (< 0.05).

Significant intergroup differences were observed across most laboratory parameters, including creatinine, ALT, cholesterol, triglycerides, LDL, HDL, LDL/HDL ratio, and hemoglobin, while AST showed no significant variation. Group 1 consistently demonstrated higher creatinine, ALT, lipid profile s, and hemoglobin as opposed to Groups 2 and 3, with most differences persisting after Tukey correction, whereas Groups 2 and 3 exhibited more comparable profiles. All cardiac enzymes (troponin, CPK, CK-MB) varied significantly, with Group 2 showing the highest median levels and Group 3 the lowest. Pairwise comparisons confirmed significant differences between Groups 1 and 2, and Groups 2 and 3 for all enzymes, while between Groups 1 and 3 significance was observed for CPK and CK-MB but not troponin. Left ventricular ejection fraction also differed significantly, being lowest in Group 2 and highest in Group 3, with all pairwise comparisons reaching statistical significance (p < 0.05).

**Table 2:** Comparison between different groups regarding lab findings, cardiac enzymes, and EF.

| Variables | Group 1 (n=96) | Group 2 (n=107) | Group 3 (n=67) | P | P1 | P2 | P3 |
|---|---|---|---|---|---|---|---|
| Creatinine | 0.87 [0.1-1.1] | 1.0 [0.7-1.6] | 0.97 [0.7-1.5] | 0.037 | 0.026* | 0.070 | 0.914 |
| ALT | 22.0 [18-37] | 30.0 [10-40] | 27.0 [18-37] | 0.030 | 0.027* | 0.134 | 0.765 |
| AST | 30.0 [25-48] | 35.0 [23-55] | 35.0 [25-48] | 0.604 | - | - | - |
| Cholesterol | 296.5 [210-360] | 214.5 [206-232] | 157.5 [89-183] | <0.001 | <0.001* | <0.001* | <0.001* |
| TG | 231.0 [180-321] | 156.0 [130-169] | 98.5 [68-135] | <0.001 | <0.001* | <0.001* | <0.001* |
| LDL | 207.5 [143-275] | 144.0 [135-163] | 97.0 [33-112] | <0.001 | <0.001* | <0.001* | <0.001* |
| HDL | 34.5 [23-42] | 39.0 [32-46] | 42.0 [39-56] | <0.001 | <0.001* | <0.001* | <0.001* |
| LDL/HDL | 6.4 [4.0-8.5] | 3.6 [3.3-4.7] | 2.1 [0.8-2.7] | <0.001 | <0.001* | <0.001* | 0.824 |
| Hb | 16.0 [14-19] | 14.0 [12-17] | 14.0 [12-16] | <0.001 | 0.026* | 0.070 | 0.914 |
| Troponin | 0.03 [0.01-2.0] | 1.0 [0.01-6.0] | 0.02 [0.0-0.04] | <0.001* | <0.001* | 0.772 | <0.001* |
| CPK | 90 [60.0-200.0] | 150 [70.0-250.0] | 75.5 [55.0-99.0] | <0.001* | <0.001* | 0.005* | <0.001* |
| CK MB | 25.0 [15.0-70.0] | 60.0 [15.0-90.0] | 18.5 [12.0-32.0] | <0.001* | <0.001* | 0.009* | <0.001* |
| Ejection Fraction (EF) | 55 [50.0-62.0] | 50.0 [38.0-60.0] | 59 [54.0-68.0] | <0.001* | 0.002* | <0.001* | <0.001* |

s are expressed as median and interquartile range (IQR) in square brackets, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, TG: Triglycerides, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, Hb: Hemoglobin, CPK: Creatine Phosphokinase, CK-MB: Creatine Kinase-Myocardial Band, EF: Ejection Fraction, Ps <0.05 were considered statistically significant. Significant Kruskal tests were followed by Tukey correction for multiple tests to calculate the adjusted P, P1: G1 Vs G 2, P2: G 1 Vs G3, P3: G 2 Vs G 3, *indicates significant p.

LAD involvement was similar between groups, while LCX and RCA lesions were significantly more frequent in Group 1. Conversely, combined LAD_RCA lesions and multivessel disease affecting all major arteries were more common in Group 2. These findings suggest that Group 1 had more isolated vessel involvement, whereas Group 2 exhibited a higher prevalence of extensive CAD. Single-vessel disease was more prevalent in Group 1, whereas multi-vessel disease (two or three vessels) was significantly higher in Group 2. This suggests a more extensive coronary involvement in Group 2 as opposed to Group 1 (p<0.001).

**Table 3:** Comparison between different groups regarding affected vessels and number of affected vessels.

| Affected vessels | | Group 1 (n=96) | Group 2 (n=107) | p |
|---|---|---|---|---|
| LAD | | 22 (22.9%) | 25 (23.4%) | **0.94** |
| LCX | | 21 (21.9%) | 9 (8.4%) | **<0.001** |
| RCA | | 38 (39.6%) | 27 (25.2%) | **<0.001** |
| LAD_RCA | | 1 (1.0%) | 16 (15.0%) | **<0.001** |
| LAD_LCX | | 5 (5.2%) | 0 (0%) | **0.006** |
| All Vessels | | 9 (9.4%) | 21 (19.6%) | **0.001** |
| Number of Affected Vessels | Single | 81 (84.4%) | 61 (57.0%) | **<0.001** |
| | Two | 6 (6.3%) | 16 (15.0%) | |
| | Three | 9 (9.4%) | 30 (28.0%) | |
| | Total | 96 (100%) | 107 (100%) | |

Data were presented as n (%), LAD: Left Anterior Descending artery, LCX: Left Circumflex artery, RCA: Right Coronary Artery, *indicates significant p.

No significant differences were found in baseline demographics or major cardiovascular risk factors between ectasia cases with obstruction (Group 1a) and those without obstruction (Group 1b), indicating comparable background characteristics. However, ACS presentation varied significantly: STEMI was observed exclusively in the obstructive group (55.0%), while UA predominated in the non-obstructive group (60.5%). NSTEMI frequencies were similar between the two subgroups, with no statistically significant difference.

**Table 4:** Demographics, risk factors, and clinical findings of ectasia subgroups.

| Parameter | | Group 1a (n=20) | Group 1b (n=76) | p |
|---|---|---|---|---|
| Age (years) | | 57.73 ± 10.72 | 55.04 ± 8.23 | 0.227 |
| Gender (Male) | | 15 (75.0%) | 52 (68.4%) | 0.325 |
| HTN | | 14 (70.0%) | 58 (76.3%) | 0.337 |
| DM | | 14 (70.0%) | 50 (65.8%) | 0.126 |
| History of IHD | | 1 (5.0%) | 0 (0.0%) | 0.208 |
| Family History IHD | | 8 (40.0%) | 23 (30.3%) | 0.687 |
| Smoking | | 14 (70.0%) | 44 (57.9%) | 0.970 |
| ACS Type | Unstable angina | 3 (15.0%) | 46 (60.5%) | <0.001* |
| | NSTEMI | 6 (30.0%) | 30 (39.5%) | 0.436 |
| | STEMI | 11 (55.0%) | 0 (0.0%) | <0.001* |

Data were presented as mean ± SD or n (%). HTN: Hypertension, DM: Diabetes Mellitus, IHD: Ischemic Heart Disease, ACS: Acute Coronary Syndrome, NSTEMI: Non-ST-Segment Elevation Myocardial Infarction, STEMI: ST-Segment Elevation Myocardial Infarction, *indicates significant p.

Creatinine was significantly higher in the obstruction group (Group 1a) as opposed to the non-obstruction group (p = 0.022). No significant differences were observed between groups for ALT, AST, lipid profile (cholesterol, TG, LDL, HDL, LDL/HDL ratio), or hemoglobin.

**Table 5:** Lab findings of ectasia subgroups.

| Variable | Group 1a (n=20) | Group 1b (n=76) | p |
|---|---|---|---|
| Creatinine (mg/dL) | 0.65 (0.45-1.00) | 0.60 (0.30-0.80) | 0.022* |
| ALT (U/L) | 28.50 (20.65-30.65) | 24.80 (21.10-28.75) | 0.189 |
| AST (U/L) | 32.75 (28.55-39.40) | 33.90 (28.60-39.00) | 0.942 |
| Cholesterol (mg/dL) | 288 (273.10-308.50) | 282.7 (244.90-318.10) | 0.191 |
| TG (mg/dL) | 268.40 (217.60-284.65) | 243.20 (215.00-283.85) | 0.334 |
| LDL (mg/dL) | 207.35 (188.55-232.85) | 198.90 (171.15-237.85) | 0.323 |
| HDL (mg/dL) | 34.50 (30.05-39.50) | 35.30 (31.15-41.55) | 0.144 |
| LDL/HDL Ratio | 6.60 (5.00-7.50) | 6.10 (5.20-7.50) | 0.882 |
| Hb (g/dL) | 15.80 (14.65-17.90) | - | 0.711 |

s are presented as median (interquartile range). ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, TG: Triglycerides, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, Hb: Hemoglobin, *indicates significant p.

Diffuse ectasia was significantly more common in the obstruction group (70.0%) as opposed to the non-obstruction group (39.5%; p = 0.015). No significant differences were observed for size, focal morphology, or shape classification.

**Table 6:** Morphological characteristics of coronary artery ectasia in patients with occlusion (group 1a) and without occlusion (group 1b).

| Parameter | | Group 1a (n=20) | Group 1b (n=76) | p |
|---|---|---|---|---|
| Size Classification | Small (< 5 mm) | 3 (15.0%) | 18 (23.7%) | 0.403 |
| | Medium (5-8 mm) | 5 (25.0%) | 28 (36.8%) | 0.321 |
| | Giant (> 8 mm) | 12 (60.0%) | 30 (39.5%) | 0.397 |
| Length/Morphology | Diffuse (long segment dilation) | 14 (70.0%) | 30 (39.5%) | 0.015* |
| | Focal (localized dilation) | 6 (30.0%) | 46 (60.5%) | 0.124 |
| Shape Classification | Saccular (transverse > longitudinal) | 8 (40.0%) | 24 (31.6%) | 0.595 |
| | Fusiform (longitudinal > transverse) | 12 (60.0%) | 52 (68.4%) | 0.803 |

Data are n (%). ps: t-test for continuous, Chi-square/Fisher's exact for categorical, *indicates significant p.

Vessel involvement patterns were largely comparable between groups, except for combined LAD + RCA affection, which was observed only in the obstruction group and reached borderline significance (p = 0.050). The distribution of single-, two-, or three-vessel involvement showed no significant differences. However, cases in the obstruction group demonstrated significantly longer mean ectasia length (p < 0.001), a greater number of ectatic sites per patient (p < 0.001), and a higher prevalence of thrombus (p < 0.001). No significant differences were noted regarding aneurysm formation or vessel tortuosity.

**Table 7:** Affected Vessels, number of vessels, and ectasia parameters of ectasia subgroups.

| | | Group 1a (n=20) | Group 1b (n=76) | p |
|---|---|---|---|---|
| Vessel(s) | LAD | 5 (25.0%) | 17 (22.4%) | 0.803 |
| | LCX | 4 (20.0%) | 17 (22.4%) | 0.820 |
| | RCA | 8 (40.0%) | 30 (39.5%) | 0.966 |
| | LAD + RCA | 1 (5.0%) | 0 (0.0%) | 0.05* |
| | LAD + LCX | 1 (5.0%) | 4 (5.3%) | 0.96 |
| | All vessels | 1 (5.0%) | 8 (10.5%) | 0.45 |
| Number of Affected Vessels | Single-vessel | 17 (21.0%) | 64 (79.0%) | 0.93 |
| | Two-vessel | 2 (33.3%) | 4 (66.7%) | 0.601 |
| | Three-vessel | 1 (11.1%) | 8 (88.9%) | 0.680 |
| Mean length of ectasia (cm) | | 3.80 ± 1.10 | 2.35 ± 0.74 | <0.001* |
| Number of ectatic sites per patient | | 2.38 ± 1.10 | 1.58 ± 0.82 | <0.001* |
| Number with thrombus | | 7 (35.0%) | 0 (0.0%) | <0.001* |
| Number with aneurysm | | 1 (5.0%) | 9 (11.8%) | 0.682 |
| Other key features (e.g., tortuosity) | | 4 (20.0%) | 7 (9.2%) | 0.233 |

Data are presented as mean ± SD or n (%), LAD: Left Anterior Descending artery, LCX: Left Circumflex artery, RCA: Right Coronary Artery, *indicates significant p.

**Table 8:** ROC analysis of LDL/HDL to diagnose coronary artery ectasia

| | LDL/HDL |
|---|---|
| AUC | 0.968 |
| 95% CI | 0.949 - 0.987 |
| Cut-off | 3.12 |
| Sensitivity | 99.0% |
| Specificity | 54.6% |
| PPV | 65.6% |
| NPV | 98.6% |
| p | <0.001 |
| Accuracy | 74.8% |

Significant P; AUC: Area under the curve; PPV: Positive predictive; NPV: Negative predictive

LDL/HDL ratio is a strong diagnostic marker for coronary artery ectasia, with an AUC of 0.968, indicating excellent discriminative ability. At a cut-off of 3.12, the test achieved very high sensitivity (99.0%) and negative predictive (98.6%), suggesting it is highly reliable in ruling out the disease. However, specificity was modest (54.6%), reflecting a higher false-positive rate. Overall diagnostic accuracy was 74.8%, with a statistically significant p (<0.001), supporting the robustness of the test in this clinical setting.

**Table 9:** Binary logistic regression of predictors of coronary artery ectasia

| Variables | Coronary artery ectasia | | | |
|---|---|---|---|---|
| | OR | 95% CI | | |
| | | Lower bound | Higher bound | p |
| Age | 0.99 | 0.94 | 1.05 | 0.754 |
| **Gender (RF=females)** | | | | |
| Male | 1.17 | 0.45 | 3.01 | 0.750 |
| **DM (RF=no)** | | | | |
| Yes | 3.14 | 1.21 | 8.17 | 0.019* |
| **HTN (RF=No)** | | | | |
| Yes | 2.41 | 0.93 | 6.25 | 0.072 |
| **Smoking (RF=No)** | | | | |
| Yes | 1.15 | 0.47 | 2.8 | 0.763 |
| Cholesterol | 1.17 | 1.04 | 1.31 | 0.009* |
| TG | 34.96 | 0.02 | 70.47 | 0.998 |
| LDL/ HDL | 76.74 | 4.1 | 143.66 | 0.004* |

RF=Reference category, OR=Odds ratio, CI=Confidence interval, Significant p

The binary logistic regression analysis of CAE predictors showed significant independent predictors included diabetes mellitus (OR 3.14, 95% CI 1.21-8.17, p = 0.019), elevated cholesterol (OR 1.17, 95% CI 1.04-1.31, p = 0.009), and LDL/HDL ratio (OR 76.74, 95% CI 4.1-143.66, p = 0.004). Other factors like age, gender, hypertension, smoking, and triglycerides were not statistically significant. These findings suggest that lipid profile, particularly LDL/HDL ratio, along with diabetes and total cholesterol, strongly predict the presence of CAE.

## Discussion

CAE is an under-recognized ACS phenotype with thrombotic risk. In this prospective two-center cohort (CAE n=96; obstructive CAD n=107; normal n=67), we compared clinical profiles, laboratory markers, cardiac enzymes, echocardiography, and angiography, and evaluated lipid-based predictors, including the LDL/HDL ratio to identify non-invasive predictors of CAE among consecutive ACS cases.

In the present research comparing cases with CAE, obstructive CAD, and angiographically normal controls, both overlapping and distinct features were observed. Age, sex, smoking, and family history of ischemic heart disease exhibited comparability between groups. Though, HTN and DM were significantly more common in CAE cases compared with CAD and controls, emphasizing the role of metabolic and vascular risk factors in ectasia development. These findings are consistent with Xi and co-authors [21] and Zografos and co-authors [22], who identified hypertension, smoking, and male sex as strong predictors of CAE. In contrast, Shereef and Kandeel [23] reported no significant demographic variation, suggesting that conventional risk factors alone cannot fully explain CAE pathogenesis and that additional mechanisms like vascular remodeling, chronic inflammation, or genetic predisposition may contribute.

Biochemical analysis revealed striking differences across groups. Cases with CAE exhibited significantly higher creatinine, ALT, total cholesterol, triglycerides, LDL, LDL/HDL ratio, and hemoglobin as opposed to CAD and controls, while AST did not differ. Elevated creatinine suggests an underrecognized interaction between renal dysfunction and coronary remodeling, as impaired renal clearance and endothelial dysfunction may promote ectatic changes. The marked dyslipidemia and altered metabolic profiles reinforce the concept that systemic metabolic derangements influence the course of CAE. Previous studies have largely focused on inflammatory pathways [24,25]; however, our results suggest that ectasia may result from a multifactorial process combining both metabolic and inflammatory mechanisms.

Markers of myocardial injury also demonstrated significant variation. Troponin, CPK, and CK-MB were highest in CAD, lowest in controls, and intermediate in CAE, reflecting the gradient of ischemic burden. Pairwise comparisons confirmed that CAE cases had significantly higher cardiac enzymes than controls but lower than obstructive CAD. Similarly, left ventricular ejection fraction (EF) differed significantly, with CAD cases showing the lowest s, controls the highest, and CAE cases in between ($p < 0.05$). These findings indicate that CAE, although frequently classified as “non-obstructive,” is not benign, as it is associated with subclinical myocardial injury and functional impairment. Comparable reductions in EF among CAE cases were previously reported by Shereef and Kandeel [23] and Zografos [22], supporting the concept that ectatic vessels may compromise myocardial perfusion even in the absence of critical stenosis.

Angiographic features further delineated differences between the groups. In CAE, single-vessel disease predominated, while CAD cases more frequently exhibited two- or three-vessel involvement, consistent with the diffuse atherosclerotic burden of CAD. The RCA and LAD were the most commonly affected arteries in both groups, a distribution repeatedly reported in prior studies [22,23,26]. Diffuse ectasia was characteristic of CAE, whereas obstructive CAD typically presented with multi-vessel narrowing, suggesting distinct vascular remodeling trajectories: dilatation and ectatic change in CAE versus progressive stenosis and occlusion in CAD.

Dyslipidemia emerged as one of the most distinctive hallmarks of CAE. Total cholesterol, triglycerides, LDL, and LDL/HDL ratios were all markedly elevated as opposed to CAD and controls. Regression analysis confirmed diabetes mellitus, total cholesterol, and LDL/HDL ratio as independent predictors of CAE, highlighting the central role of lipid imbalance in ectasia pathogenesis. The LDL/HDL ratio, in particular, demonstrated strong predictive, underscoring its relevance not only in atherosclerosis but also in the vascular remodeling unique to ectasia. These findings mirror those of Xi and co-authors [21] and Manginas [26], although Shereef and Kandeel [23] reported less pronounced lipid abnormalities, pointing to possible genetic, ethnic, or environmental variability across populations.

To further substantiate these results, predictive modeling analyses were performed. The ROC analysis of the LDL/HDL ratio confirmed its excellent diagnostic performance, with very high sensitivity and strong negative predictive, making it reliable for ruling out CAE. Despite modest specificity, the overall diagnostic accuracy supports the robustness of this parameter as a simple, non-invasive tool.

Complementary logistic regression analysis further emphasized the role of lipid imbalance, identifying diabetes mellitus, total cholesterol, and LDL/HDL ratio as independent predictors of CAE, while traditional risk factors like age, sex, hypertension, smoking, and triglycerides were not significant. The particularly strong association of the LDL/HDL ratio highlights its dominant role in risk stratification compared with other lipid markers. Collectively, these findings confirm that dyslipidemia, particularly LDL/HDL imbalance, plays a central role in the development of CAE, with diabetes mellitus acting as an important synergistic contributor.

Taken together, these results indicate that CAE cases occupy a distinct position between obstructive CAD and normal coronary anatomy. While sharing some demographic and risk factor similarities with both groups, CAE is characterized by a unique profile of severe dyslipidemia, metabolic and renal disturbances, diffuse single-vessel involvement (particularly RCA and LAD), and intermediate myocardial injury and dysfunction. This highlights CAE as a separate clinical entity whose pathogenesis likely extends beyond conventional atherosclerotic mechanisms to include systemic metabolic derangements and vascular remodeling pathways.

This research has several limitations. It was conducted in two hospitals and included only cases already diagnosed with CAD, which may limit generalizability and exclude broader at-risk populations. The design assessed associations rather than causality, without accounting for lifestyle, genetic, or inflammatory factors like CRP, IL-6, and TNF. Coronary angiography may miss subtle cases, comorbidity severity was not detailed, long-term follow-up was lacking, and prevalence of CAE could not be determined.

## Conclusion

Cases with CAE more frequently presented with UA, whereas obstructive CAD cases predominantly had STEMI and NSTEMI. As opposed to obstructive CAD, CAE cases showed significantly higher creatinine, ALT, cholesterol, triglycerides, LDL, HDL, LDL/HDL ratio, and hemoglobin. Within CAE, obstructive cases were more often associated with STEMI, higher ejection fraction, and lower creatinine, but also demonstrated diffuse ectasia, longer ectatic segments, more ectatic sites, and a greater prevalence of thrombus. The RCA was the most commonly affected vessel in CAE.

## Conflict of Interest

Not available

**Financial Support**
Not available

**References**

1. Devabhaktuni S, Mercedes A, Diep J, Ahsan C. Coronary artery ectasia-a review of current literature. Current Cardiology Reviews. 2016;12(4):318-323.
2. Doi T, Kataoka Y, Noguchi T, Shibata T, Nakashima T, Kawakami S, *et al*. Coronary artery ectasia predicts future cardiac events in patients with acute myocardial infarction. Arteriosclerosis, Thrombosis, and Vascular Biology. 2017;37(12):2350-2355.
3. Li Y, Wu C, Liu W. Coronary artery ectasia presenting with thrombus embolization and acute myocardial infarction: A case report. Medicine (Baltimore). 2017 Jan;96(4):e5976.
4. Connelly MA, Shalaurova I, Otvos JD. High-density lipoprotein and inflammation in cardiovascular disease. Translational Research. 2016 Jul;173:7-18.
5. Newburger JW, Takahashi M, Burns KC. Kawasaki Disease. Journal of the American College of Cardiology. 2016 Apr 12;67(14):1738-1749.
6. Abera A, Worede A, Hirigo AT, Alemayehu R, Ambachew S. Dyslipidemia and associated factors among adult cardiac patients: a hospital-based comparative cross-sectional study. European Journal of Medical Research. 2024;29(1):237.
7. Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, *et al*.; Task Force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. European Heart Journal. 2021;42(14):1289-1367.
8. Shah SR, Alweis R. Acute Coronary Artery Dissection: A Review of the Literature and Current Evidence. Cardiology Review. 2018 Sep/Oct;26(5):274-276.
9. Elendu C, Amaechi DC, Elendu TC, Omeludike EK, Alakwe-Ojimba CE, Obidigbo B, *et al*. Comprehensive review of ST-segment elevation myocardial infarction: Understanding pathophysiology, diagnostic strategies, and current treatment approaches. Medicine. 2023;102(43):e35687.
10. Heidelbaugh JJ, Bruderly M. Cirrhosis and chronic liver failure: part I. Diagnosis and evaluation. American Family Physician. 2006 Sep 1;74(5):756-762.
11. Webster AC, Nagler EV, Morton RL, Masson P. Chronic kidney disease. Lancet. 2017;389(10075):1238-1252. UGent Library+2The Lancet+2
12. Mavrogeni S. Coronary artery ectasia: from diagnosis to treatment. Hellenic Journal of Cardiology. 2010 Mar-Apr;51(2):158-163.
13. Serruys PW, Morice MC, Kappetein AP, Colombo A, Holmes DR, Mack MJ, *et al*. Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease. New England Journal of Medicine. 2009;360(10):961-972.
14. Carey RM, Whelton PK. Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Synopsis of the 2017 American College of Cardiology/American Heart Association Hypertension Guideline. Annals of Internal Medicine. 2018 Mar 6;168(5):351-358.
15. Selph S, Dana T, Blazina I, Bougatsos C, Patel H, Chou R. Screening for type 2 diabetes mellitus: a systematic review for the US Preventive Services Task Force. Annals of Internal Medicine. 2015;162(11):765-776.
16. Nagueh SF, Sanborn DY, Oh JK, Anderson B, Billick K, Derumeaux G, *et al*. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography and for Heart Failure With Preserved Ejection Fraction Diagnosis: An Update From the American Society of Echocardiography. Journal of the American Society of Echocardiography. 2025;38(7):537-569.
17. Foley TA, Mankad SV, Anavekar NS, Bonnichsen CR, Morris MF, Miller TD, Araoz PA. Measuring left ventricular ejection fraction-techniques and potential pitfalls. 2012.
18. Di Mario C, Sutaria N. Coronary angiography in the angioplasty era: projections with a meaning. Heart. 2005 Jul;91(7):968-976.
19. Wang X, Nie SP. The coronary slow flow phenomenon: characteristics, mechanisms and implications. Cardiovascular Diagnosis and Therapy. 2011 Dec;1(1):37-43.
20. Badimon L, Padró T, Vilahur G. Atherosclerosis, platelets and thrombosis in acute ischaemic heart disease. European Heart Journal Acute Cardiovascular Care. 2012 Apr;1(1):60-74.
21. Xi Z, Qiu H, Guo T, Wang Y, Dou K, Xu B, *et al*. Prevalence, predictors, and impact of coronary artery ectasia in patients with atherosclerotic heart disease. Angiology. 2023;74(1):47-54.
22. Zografos TA, Korovesis S, Giazitzoglou E, Kokladi M, Venetsanakos I, Paxinos G, *et al*. Clinical and angiographic characteristics of patients with coronary artery ectasia. International Journal of Cardiology. 2013;167(4):1536-1541.
23. Shereef NT. Coronary artery ectasia: Prevalence and clinical characteristics. Journal of the Indian College of Cardiology. 2019;9(3):128-1321.
24. Li JJ, Nie SP, Qian XW, Zeng HS, Zhang CY. Chronic inflammatory status in patients with coronary artery ectasia. Cytokine. 2009;46(1):61-64.
25. Esposito L, Di Maio M, Silverio A, Cancro FP, Bellino M, Attisano T, *et al*. Treatment and outcome of patients with coronary artery ectasia: current evidence and novel opportunities for an old dilemma. Frontiers in Cardiovascular Medicine. 2022;8:805727.
26. Manginas A, Cokkinos DV. Coronary artery ectasias: imaging, functional assessment and clinical implications. European Heart Journal Acute Cardiovascular Care. 2012;1.

**How to Cite This Article**
Ebaid HH, Hassan GM, Hamoda MA, Abd El Monem AM. Non-invasive predictors of coronary artery ectasia. International Journal of Cardiology Sciences. 2025,7(02):80-86