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Relation between Coronary Artery Ectasia Characteristics and Development of Acute Coronary Syndrome

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ABSTRACT

Background: An aneurysmal aberration of the artery known as coronary artery ectasia (CAE) is identified by significant dilatation and a luminal diameter that is 1.5 times larger than the surrounding normal segments. As opposed to coronary artery aneurysms, which only have a little extension of the arterial wall, it is different. Managing acute coronary syndromes (ACS) in isolated CAE can be difficult because there is no proven therapy and little research in this area.

Objective: To determine the predictors of ACS occurrence among patients with coronary ectasia especially ectatic segment characteristics.

Patients and Methods: The present study included 272 patients with coronary ectasia and classified as follows: Group (A): presented with ACS (n=112, 41.2%) and group (B): presented with chronic coronary syndrome (CCS) (n=160, 58.8%). Age or gender had no statistically significant impact on the development of ACS in the patients under study.

Results: The best cutoff of serum D-dimer level in diagnosis of ACS was ≥ 0.85 and those of serum uric acid level, LDL cholesterol, CRP and serum triglycerides were ≥7.3 mg/dl., ≥178 mg/dl., ≥7.6 mg/l. and ≥181.5 mg/dl, respectively. There was statistically non-significant relation between ACS development and the culprit coronary, but notably ectasia was more frequently affecting the right coronary artery (RCA). The best cutoff of ectatic segment length in predicting ACS was ≥39 mm with area under curve 0.7, sensitivity 64%, and specificity 88%. The best cutoff of ectatic segment diameter in predicting ACS was ≥6 mm with area under curve 0.8, sensitivity 74%, and specificity 65%.

Conclusions: With the aid of noninvasive conventional and laboratory risk factors, we are able to anticipate the development of ACS in patients with coronary ectasia and may then recommend the best course of action for preventing ACS recurrence.

Keywords: Ectasia, ACS, CCS, Predictors.

INTRODUCTION

An aneurysmal aberration of the artery known as CAE is identified by significant dilatation and a luminal diameter that is 1.5 times greater than the surrounding normal segments. Compared to coronary artery aneurysms, it is different, which have a limited expansion of the arterial wall. CAE, which is shown in 2.7–2.8% of angiograms, is caused by disease processes that undermine the integrity of the arterial wall [(1)]. It can also be a congenital defect (20–30%) in addition to being often associated with atherosclerotic disease (50%) and after connective tissue diseases, inflammatory illnesses, and infections (10–20%) [(2)]. The most common symptom of coronary artery ectasia, stable angina, can get worse with acute coronary syndrome (ACS) (61-66%) [(3)]. It might be challenging to manage ACS in the context of isolated CAE since there is no established strategy and limited research available [(4)].

The left circumflex (LCX) and left anterior descending (LAD) coronary arteries follow the right coronary artery (RCA), which is implicated in up to 85% of instances, in terms of coronary involvement. The involvement of the left major coronary artery is exceedingly rare (0.1%) [(5)].

CAE is classified into four Types [(6)]: Type I: There is diffuse ectasia in two or more vessels. Type II: One artery has localised disease, whereas another has broad ectasia. Type III: Diffuse ectasia affecting only one vessel. Type IV: Engagement that is regional or segmental.The process is thought to be the result of the vessel's media being destroyed, which raises the wall tension and leads to dilatation. Ectatic segments are an extreme type of expansive vascular remodeling caused by plaque development inside the artery walls, according to a remodeling theory advanced by certain experts [(7)].The severity of concurrent coronary artery disease has a direct impact on the prognosis for coronary artery ectasia [(8)]. In CAE with underlying CAD, the risk of unfavourable cardiac events is increased. Even with isolated CAE, there is a chance of myocardial ischemia and infarction. There is no evidence to support a connection between an artery's diameter and the result [(9)].

The aim of this study was to determine the predictors of ACS occurrence among patients with coronary ectasia especially ectatic segment characteristics.

PATIENTS AND METHODS

This study included 272 patients with coronary ectasia. Patients were picked up from Zagazig University Catheterization Laboratory, Egypt through the period from August 2019 to August 2022. Patients were classified as follow: Group (A) presented with acute coronary syndromes (n=112, 41.2%) and group (B) that presented with chronic coronary syndrome (n=160, 58.8%).

Inclusion criteria: Patients who presented with solitary coronary artery ectasia and acute or chronic coronary syndromes were included. When the diameter of the

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dilated segment was larger than 1.5 times that of the adjacent, healthy segment, CAE was recognised. Diagnosed by ischemic chest pain and ST-segment alterations, ACS is a set of conditions that includes unstable angina, ST-elevation myocardial infarction, and non-ST elevation myocardial infarction [(10)]. The presence of either CAD risk indicators or a typical angina pectoris history led to the diagnosis of CCS [(11)].

Exclusion criteria: The study excluded participants who had normal coronary angiography as well as those with proven coronary stenosis, myocardial bridge, coronary vasospasm, valvular heart disease, or previous cardiomyopathy.

All patients were subjected to: Full history taking and through clinical examination with special emphasis on age, sex, smoking, diabetes mellitus, hypertension and dyslipidemia. Also specific investigations were performed for all patients:

Electrocardiography (ECG):

Using Biocare equipment with a paper speed of 25 mm/s and an amplification of 10 mm/mv, an ECG was done at the time of admission. A 12-lead ECG with the right leads (V3R and V4R) and posterior leads (V7-V9) was also made in order to see the whole surface of the heart. Shifts from the previous TP segment were recorded 20 ms after J point and used to calculate the ST segment shifts.

Laboratory investigations including:

1. Cardiac enzymes: from ACS presenters including (CK-MB, Hs-troponin T). If the initial set of measurements of Hs- troponin T was negative, they were repeated three hours later.
2. Complete blood count (CBC) including hematocrit value, platelet count and mean platelet volume.
3. Admission random blood sugar and glycosylated hemoglobin (HbA1c).
4. D-dimer, C- reactive protein (CRP) and serum uric acid.
5. Lipogram: It uses the Cobas Integra instrument's spectrophotometry approach to measure total cholesterol, low-density lipoproteins (LDL), high-density lipoproteins (HDL), and triglycerides.

Transthoracic echocardiography (TTE):

Using a Siemens ACUSON X300 ultrasound system with a P4-2 1.8 MHZ transducer and following the recommendations of the American Society of Echocardiography (ASE) and European Association of Echocardiography, all patients had comprehensive TTE (EAE). During the echocardiogram, the patient was in a supine or left lateral position and was softly breathing. The biplane modified Simpson technique was used to calculate the left ventricular ejection fraction (LVEF).

Coronary angiography:

Trans-radial or femoral coronary angiography was preformed to define the coronary anatomy and ectatic segment diameter and length. Also, intervention to culprit vessel by balloon dilation or aspiration thrombectomy and intra-coronary glycoprotein IIb-IIIa inhibitors was done.

Ethical approval: The Research Ethical Council for the Faculty of Medicine, Zagazig University approved the study after getting written informed consent from each participant (IRB: 6072-28-4-2020). The conduct of the study was guided by the Declaration of Helsinki, the code of ethics established by the World Medical Association for research involving human subjects.

Statistical analysis

In order to organise the data, Microsoft Excel was used. The Statistical Software for the Social Sciences (SPSS) version 20.0 was then used to enter the data for analysis. Depending on the kind of data. Quantitative continuous data were represented by mean ± SD whereas qualitative data were represented by number and percentage. An independent t-test was carried out to assess variations in quantitative data between the two groups. The Chi-square test was used to look into any differences between the two groups' qualitative data. The ROC curve was used to assess the cut-off point. The P value was set at 0.05 for results that were significant, and at 0.001 for those that were highly significant.

RESULTS

The mean age in group (A) was 55.40 ± 10.57 years. The average age in group (B) was 57.78 ± 9.39 years. The age gap between the two groups' means was not statistically significant (P = 0.567). In terms of gender, group (A) had 84 men (75%) and 28 women (25%). 56 women (35% of the group) and 104 men (65%) made up group (B). Additionally, there was no statistically significant difference between the two groups (P = 0.567). Age or gender had no statistically significant impact on the development of ACS in the patients under study. In terms of hypertension, there was a statistically significant difference between the two groups (87.5% versus 45%, p=0.004). Regarding diabetes mellitus (DM), there was a statistically insignificant difference between the two groups (57.1% against 50%, p=0.67). In terms of smoking, there was a statistically significant difference between the two groups (81.3% versus 45%, p=0.014). Regarding dyslipidemia, there was a statistically significant difference between the two groups (93.8% versus 57.5%, p=0.009). There was a statistically insignificant difference between the two groups with a positive family history of CAD (25% versus 28.1%, p= 0.999). There was no statistically significant difference in obesity between the two groups (37.5% versus 45%, p=0.608), as shown in table (1).

Table (1): Comparison between the studied groups regarding demographic data

	Group I (ACS) N=112 (41.2%)	Group II (CCS) N=160 (58.8%)	χ2	p value
Age (years)	55.40 ± 10.57	57.78 ± 9.39	0.327	0.567
Sex: Male	84 (75%)	104 (65%)		
Female	28 (25%)	56 (35%)	0.524	0.469
Hypertension	98 (87.5%)	72 (45%)	8.429	0.004*
DM	64 (57.1%)	80 (50%)	3.36	0.67
Dyslipidemia	105 (93.8%)	92 (57.5%)	6.885	0.009*
Smoking	91 (81.3%)	72 (45%)	6.077	0.014*
Obesity	42 (37.5%)	72 (45%)	0.263	0.608
Positive family history of CAD	28 (25%)	45 (28.1%)	0.1	0.999

* Statistically significant p value ** Statistically highly significant p value

As indicated in table (2) and figure (1), 32.1% of ACS patients had NSTEMI, 28.6% had unstable angina, 17.9% had anterior MI, 17.9% had inferior MI, and 3.6% had lateral STEMI.

Table (2): Distribution of patients according to ACS syndrome encountered

ACS subtypes	N=112	Percentage (%)
Anterior STEMI	20	17.9%
Inferior STEMI	20	17.9%
Lateral STEMI	4	3.6%
NSTEMI	36	32.1%
Unstable angina	32	28.6%

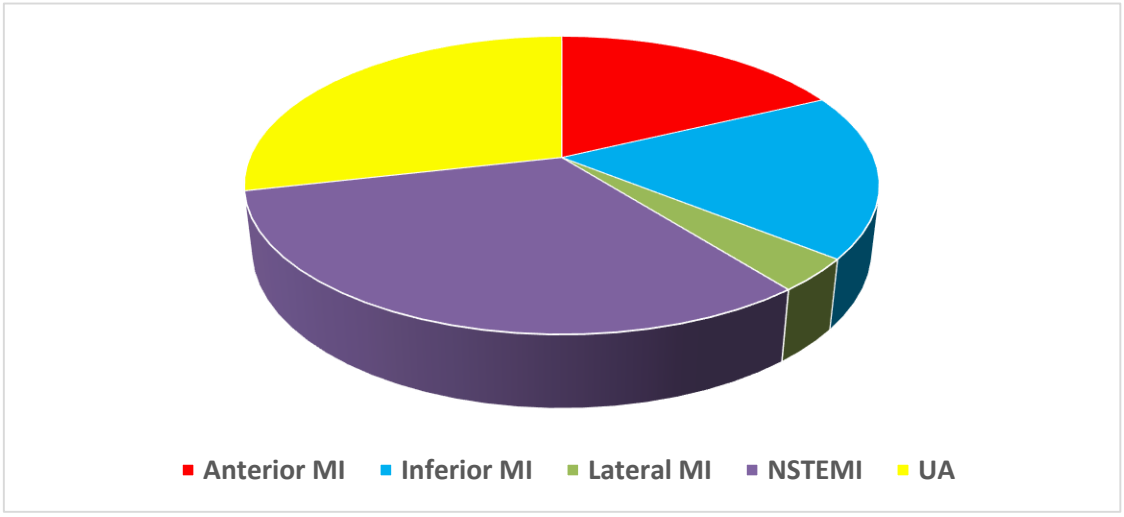

Figure (1): Pie chart showing distribution of patients according to ACS syndrome encountered

The development of ACS was statistically related to all LDL cholesterol, total cholesterol, triglycerides, mean platelet volume, uric acid, D-dimer and C-RP (all were significantly higher in those with ACS). The relationship between the onset of ACS and either HDL, platelet count, HbA1c, or hematocrit level was statistically insignificant. Additionally, the modified Simpsons derived ejection fraction measurement of systolic function revealed a highly statistically significant difference between the two groups (40.82 versus 55.07, p= 0.001**), as shown in table (3).

Table (3): Comparison between the studied groups regarding laboratory and echocardiographic data

Parameter	Group I (ACS) N=112 (41.2%)	Group II (CCS) N=160 (58.8%)	t	p value
LDL cholesterol(mg/dL)	202.75±47.93	139.65±33.23	5.192	<0.001**
Total cholesterol(mg/dL)	261.94±64.51	204.0±50.72	3.489	0.001**
Triglycerides(mg/dL)	242.19±50.86	157.93±35.6	6.06	<0.001**
HDL cholesterol(mg/dl)	34.44±4.97	35.33±8.6	-0.435	0.665
Platelet count(mcL)	262.19±62.42	237.7±56.42	-0.081	0.935
Mean platelet volume	10.09±0.36	9.21±1.1	4.467	<0.001**
HbA1c(%)	7.36±1.46	7.07±1.31	-0.088	0.93
Uric acid(mg/dl)	8.59±2.01	7.33±1.73	2.262	0.028*
CRP(mg/dl)	8.69±1.4	5.7±1.41	6.959	<0.001**
D-dimer	1.26±0.31	0.63±0.14	5.434	<0.001**
Hematocrit	37.88±3.2	37.98±2.35	-0.136	0.803
Ejection fraction	40±8.20	55±7.00	5.2	<0.001**

* Statistically significant p value. ** Statistically highly significant p value.

Figure (2) showed that the optimal cutoff of D-dimer level for the diagnosis of ACS was 0.85 with an area under the curve of 0.885, a sensitivity of 81.3%, and a specificity of 77.5% (p=0.001). In order to diagnose ACS, the optimal mean platelet volume cutoff was 9.9 with an area under the curve of 0.784, a sensitivity of 81.3%, and a specificity of 67.5% (p=0.001) (figure 3). According to figure (4), 7.3 mg/dl of blood uric acid was the optimum cutoff value for diagnosing ACS with an area under curve of 0.672, a sensitivity of 68.8%, and a specificity of 60% (p=0.046).

With an area under the curve of 0.851, sensitivity of 81.3%, and specificity of 80%, a serum LDL cholesterol level of 178 mg/dl was the optimum cutoff for diagnosing ACS (p = 0.001). Regarding serum total cholesterol, 218.5 mg/dl was the optimum cutoff for diagnosing ACS with an area under curve of 0.755, a sensitivity of 75%, and a specificity of 65% (p=0.001), as shown in figure (5). According to figure (6), a serum C-RP level of 7.6 mg/l with an area under curve of 0.92, a sensitivity of 87.5%, and a specificity of 87.5% was the optimum cutoff for diagnosing ACS. According to figure (7), the optimum cutoff for diagnosing ACS was 181.5 mg/dl with an area under curve of 0.9, a sensitivity of 75%, and a specificity of 75% (p=0.001).

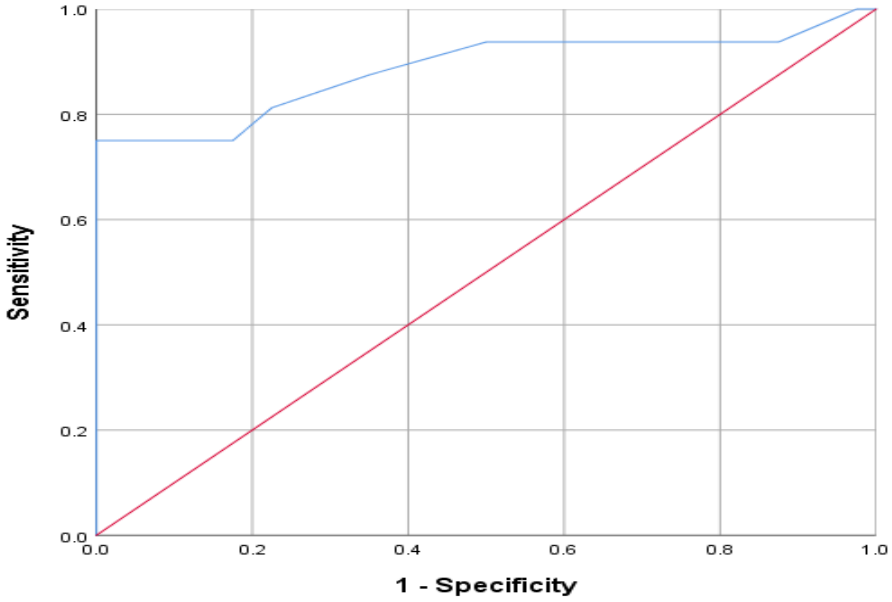

Figure (2): ROC curve demonstrating the D-dimer's diagnostic efficacy in the diagnosis of ACS in the individuals under study

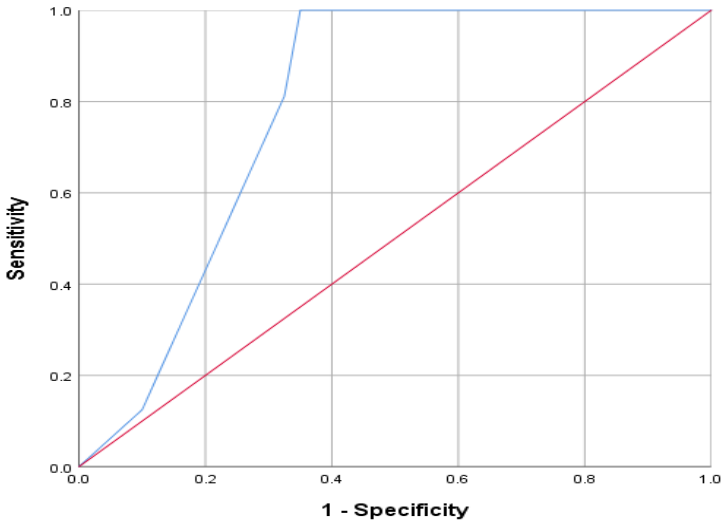

Figure (3): ROC curve demonstrating the ability to detect ACS in individuals under study using mean platelet volume

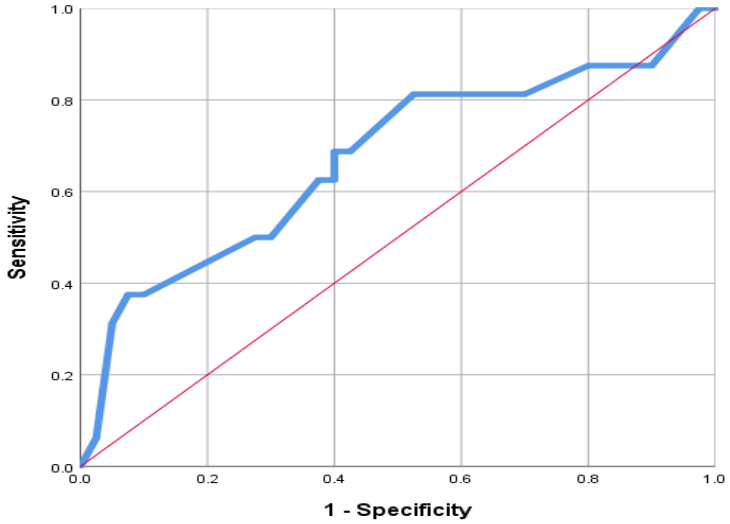

Figure (4): ROC curve demonstrating the effectiveness of uric acid in the diagnosis of ACS in the individuals under study.

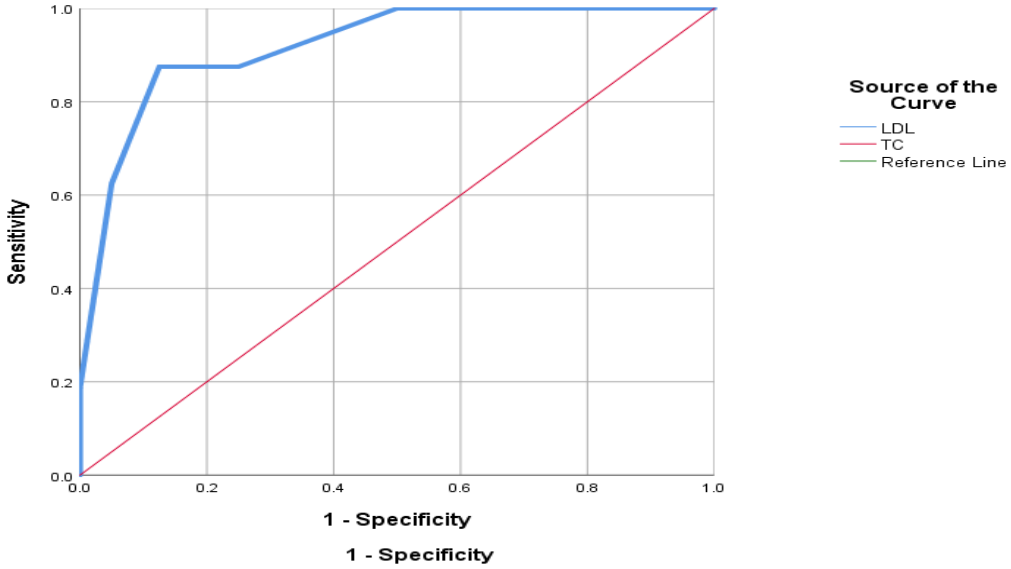

Figure (5): ROC curve demonstrating the effectiveness of LDL and total cholesterol in the diagnosis of ACS in the sample of patients

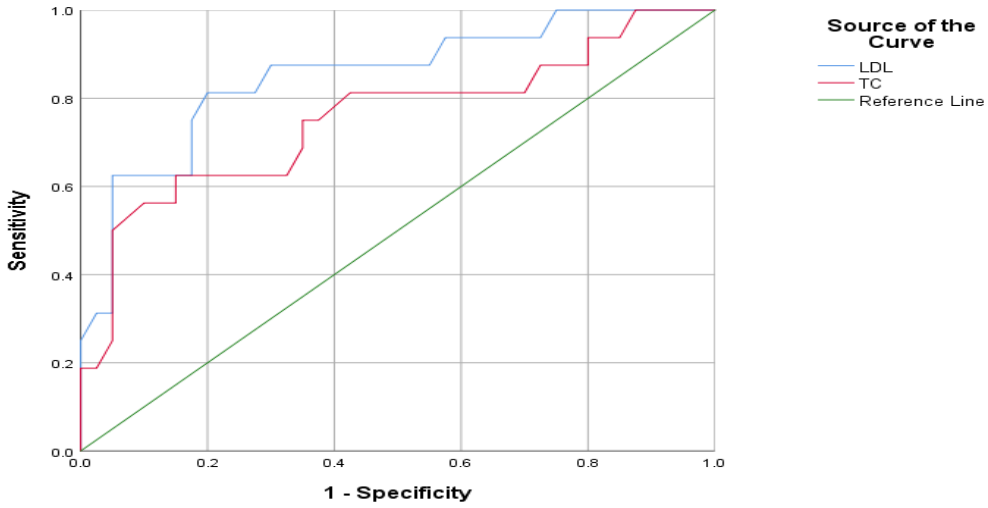

Figure (6): ROC curve demonstrating C-RP ability to accurately diagnose ACS in the individuals under study

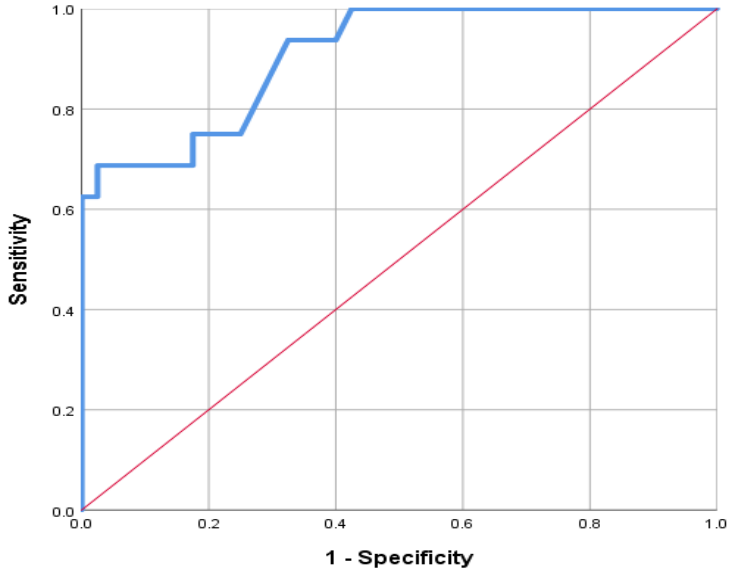

Figure (7): ROC curve demonstrating the accuracy of triglycerides in the diagnosis of ACS in the patients under investigation.

Although there was no statistically significant correlation between the development of ACS and the offending coronary, it should be noted that RCA ectasia was more common, as shown in table (4).

Table (4): Comparison between the studied groups regarding affected coronary

Coronary affected	Group I (ACS) N=112 (41.2%)	Group II (CCS) N=160 (58.8%)	χ^2	p value
Left main (LM)	1 (0.9%)	1 (0.6%)	Fisher	>0.999
Left anterior descending (LAD)	41 (36.7%)	59 (36.9%)	0.065	0.799
D1	0 (0%)	0 (0%)	Fisher	>0.999
LCX	6 (5.4%)	9 (5.6%)	Fisher	0.365
OM1	2 (1.7%)	1 (0.6%)	Fisher	>0.999
RCA	54 (48.2%)	80 (50%)	2.326	0.127
2 vessel CAD	6 (5.4%)	7 (4.4%)	Fisher	0.235
3 vessel CAD	2 (1.7%)	3 (1.9%)	Fisher	0.122

The best cutoff of ectatic segment length in predicting ACS is ≥ 39 mm with area under curve of 0.7, sensitivity of 64%, and specificity of 88%, as shown in figure (8). The best cutoff of ectatic segment diameter in predicting ACS is ≥ 6 mm with area under curve of 0.8, sensitivity of 74%, and specificity of 65%, as shown in figure (9).

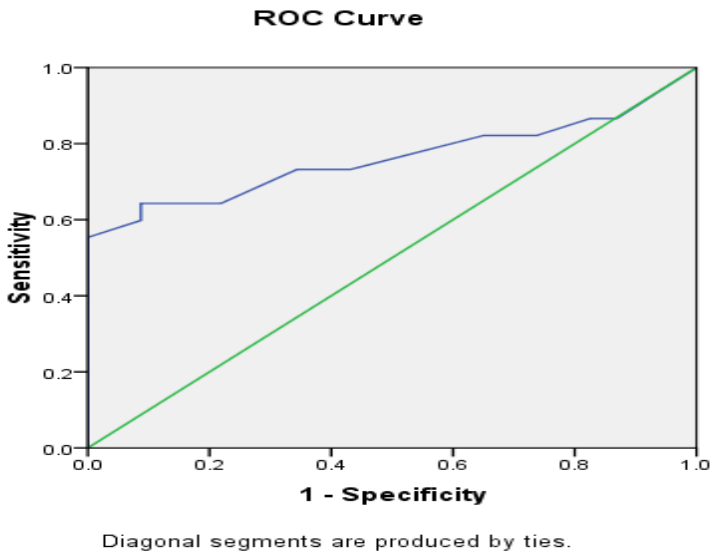

Figure (8): ROC curve demonstrating the accuracy of ectatic segment length in identifying individuals who may develop ACS.

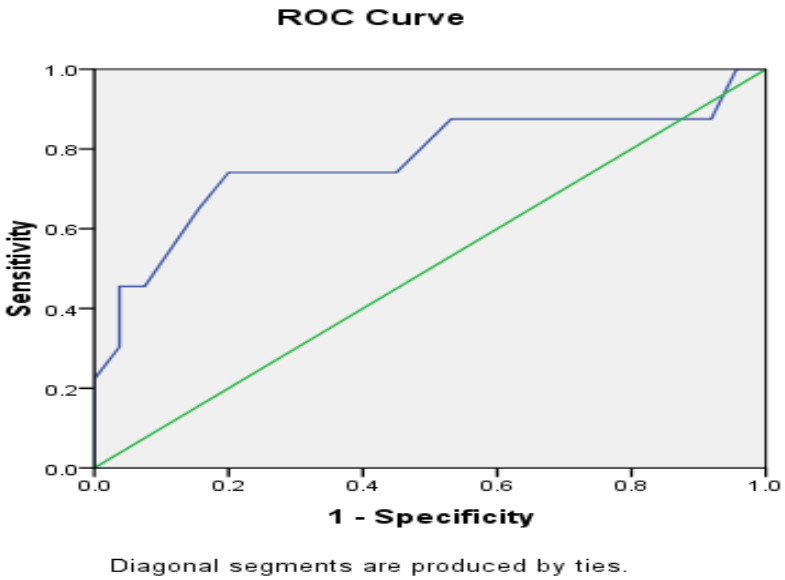

Figure (9): ROC curve demonstrating the accuracy of ectatic segment diameter in identifying individuals who may develop ACS.

DISCUSSION

Inappropriate coronary vascular dilatation is a characteristic of the somewhat common disorder known as coronary artery ectasia. The relatively common disorder known as coronary artery ectasia is characterised by unnecessary dilatation of the coronary arteries. In certain ACS instances without coronary stenoses, coronary ectasia alone is to blame. Evidence suggests to a hereditary vulnerability, typical coronary artery disease risk factors, and abnormal vessel wall metabolism, even if the exact origin of its beginning is unknown [(12)]. CAE commonly presents as an ACS due to the slow flow that promotes thrombus buildup in CAE, and eliminating this considerable amount of thrombus with percutaneous coronary intervention (PCI) may be challenging [(13)]. According to certain coronary angiographic investigations, the prevalence of CAE is estimated to be between 3 and 5% [(14)].

Our study aimed to find out the predictors of ACS development among coronary ectasia patients. The present study included 272 patients with coronary ectasia classified as follow: Group (A) presented with acute coronary syndrome (n=112, 41.2%) and group (B) presented with chronic coronary syndrome (n=160, 58.8%).

In line with **Mavrogeni *et al.*** [15], the current study found no statistically significant variation in the demographic data (age and sex) between the two groups, which made both groups well cross-matched. Moreover, we discovered no connection between demographic information and the prevalence of ACS in individuals with coronary ectasia. Since that ACS presenters had a greater frequency of the illness, there was a statistically significant difference in hypertension between the two groups (P=0.004). Contrarily, in their analysis of 3263 people, **Bahremand *et al.*** [16] observed no statistically significant difference in the prevalence of hypertension across the groups (P= 0.171).

Regarding diabetes mellitus, there was a statistically insignificant difference between the two groups (p=0.67). According to **Bermdez *et al.*** [17] and **Androulakis *et al.*** [18], diabetes mellitus and CAE have a significant, independent, and inverse connection. Diabetes mellitus is known to cause unfavourable remodeling in the arterial wall during the atherogenesis process and obstruct compensatory arterial expansion [19]. So, it could be logical to anticipate such a negative relationship between DM and CAE. This inverse association also implies that the pathophysiology of CAE could not just be a subtype of coronary atherosclerosis [20]. Contrary to **Rashid *et al.*** [21] who found no correlation between smoking and the occurrence of an ACS in individuals with coronary ectasia (P=0.1), there was a statistically significant difference between the two groups with respect to smoking (p=0.014).

There was a statistically significant difference between the two groups with regard to dyslipidemia (p=0.009). This is in contrast to **Rashid *et al.*** [21] conclusion that there was no discernible difference in the two groups of patients with coronary ectasia in terms of their dyslipidemia (p=0.4).

The difference between the two groups' positive family histories of CAD was statistically insignificant (p= 0.999). In contrast, **Dastgir *et al.*** [22] observed that the two groups of CAE patients differed significantly in terms of having a positive CAD history (p= 0.001). In terms of obesity, there was a statistically insignificant difference between the two groups (p=0.608). There are a number of risk factors for ACS, but according to **Dastgir *et al.*** [22] and **El-Menyar *et al.*** [23], smoking, diabetes, hypertension, dyslipidemia, and obesity are the main risk factors ACS. In our study, the most significant risk factor for ACS caused by coronary artery ectasia was hypertension, may be due to excessive stretching of the arterial wall.

Previous research found that risk variables for CAE include younger age, male gender, obesity, smoking, hypertension, hyperlipidemia, peripheral vascular disease, and higher levels of inflammatory markers [24]. However, the majority of the data come from limited investigations or case studies.

In line with **Jafari *et al.*** [25]**,** all LDL cholesterol, total cholesterol, triglycerides, mean platelet volume, uric acid, D-dimer, and C-RP were statistically substantially correlated with the development of ACS (all were significantly higher in those with ACS). There was no statistically significant correlation between the beginning of ACS and either HDL, platelet count, HbA1c, or hematocrit levels. This is consistent with the **Rashid *et al.*** [21], who found a significant difference of C-RP level between both groups (p=0.002), which was explained by that patients with ACS had higher Hs-CRP levels than those without. Additionally, **Bermdez *et al.*** [17] discovered that all risk variables, with the exception of diabetes mellitus predispose to the development of ACS in coronary ectasia patients. Also, the fact that CAE is more common in younger, male, and smoker patients.

Concerning echocardiographic data, this study found that ACS group had lower ejection fraction compared to CCS group ($p \leq 0.001$). This is attributed to systolic dysfunction caused by MI development among ACS group in contrast to chronic patients who had enough time for collateralization and ischemia preconditioning. Regarding coronary angiographic data, the present study found no statistically significant difference between both groups concerning the affected coronary. But notably ectasia is more frequently affecting the RCA, as previously reported [26-28]. The RCA, followed by LAD and LCX were the vessels that are most commonly involved. The hemodynamic and mechanical aspects of this phenomenon can help to partially explain it [29].

Local coronary flow conditions including endothelial shear stress, near wall velocity, and static pressure have been linked to considerable lipid buildup, inflammation, internal elastic lamina deterioration, and excessive expansive remodeling in individuals with concomitant CAD, which may result in CAE [7]. Thus, variations in the occurrence of CAE could be brought on by variations in the local coronary flow conditions between the RCA, LCX, and LAD [30].

Identifying coronary ectasia lesion characteristics that predict ACS occurrence in the future is important to well follow-up those patients and use more aggressive regimens like dual antiplatelet therapy or anticoagulant to protect them. To our knowledge, no previous study described the coronary ectasia characteristics, which make the patient more liable to have ACS during his life. According to our research, the best cutoff of ectatic segment length and diameter in predicting ACS was ≥ 39 mm and ≥ 6 mm, respectively.

The patients in the current study were all from the same region, which limited the applicability of our findings to people of different races or ethnicities. The small number of patient subgroups was another drawback. Another drawback of our study was non including other risk factors for ACS, such as genetic variables and inflammatory precipitants. We need further large-scale trials comparing various antithrombotic regimes to reduce the occurrence of

ACS in the coronary ectasia group while weighing the risk of ischemia and bleeding.

CONCLUSION

With the aid of noninvasive conventional and laboratory risk factors, we are able to anticipate the development of ACS in patients with coronary ectasia and may then recommend the best course of action for preventing ACS recurrence. As noninvasive risk factors, hypertension, smoking, dyslipidemia (LDL, total cholesterol and triglycerides), mean platelet volume, uric acid, D-dimer and C-RP can predict ACS in patients with coronary ectasia. The best cutoff of ectatic segment length and diameter in predicting ACS is ≥ 39 mm and ≥ 6 mm respectively.

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Competing interests: Nil.

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