Research Article

Spontaneous Coronary Artery Dissection: Under Diagnosed but Fatal Cause of Myocardial Ischaemia Detected by Angiography

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Abstract

Objectives: *This study sought to assess the diagnostic value of spontaneous coronary artery dissection (SCAD) in coronary angiogram. This study also aims in SCAD's clinical implication in the patient's therapeutic & prognostic arena.*

Background: *SCAD is a rare, infrequent & often missed clinical entity in patients presenting with acute coronary syndrome (ACS). Patients of SCAD can present with different clinical presentation ranging from myocardial ischaemia to myocardial infarction, different types of fatal arrhythmias and even death. Frequently missed due to lack of angiographic recognition & documentations are the major causes for under diagnosis. With the advancement of diagnostic modalities including intracoronary imaging there has been improved diagnosis of SCAD. The aim of this study is to showcase this rare clinical entity as well as make awareness among the Cardiologists to diagnose & further management of the patients to reduce morbidities & mortality among the patients with acute coronary syndrome..*

Methods: *Following a retrospective protocol, SCAD was diagnosed by coronary angiogram in 7 patients among 2880 patients undergoing coronary angiography in the Evercare Hospital, Dhaka, Bangladesh in the span of 2 years. Conservative, interventional & surgical management strategies were obtained.*

Results: *7 patients were identified as SCAD & enrolled in the study considering inclusion & exclusion criteria. Sample was subdivided into 3 groups on the type of ACS. Group-I with UA, Group-II with NSTE - ACS & Group-III with STE - ACS. SCAD was correlated with type of ACS. Mean age 49.08±7.52, 85.7% male & 14.3% female. 57.1% hypertensive, 42.9% diabetic, 71.4% current smoker, 42.9% dyslipidaemic & 14.3% asthmatic. 14.3% UA, 28.6% NSTE-ACS & 57.1% STE-ACS. Vessel involvement in SCAD was noted. 28.6% involved left anterior descending artery (LAD), 14.3% involved left circumflex artery (LCx) & both left main (LM) coronary artery & LAD, 42.9% involved right coronary artery (RCA). Involvement of SCAD varies with type of ACS, $p<0.001$.*

Conclusion: *SCAD is an under diagnosed but may be fatal clinical entity. Its diagnosis can help the physician to take decision in the management of the patient which also has clinical & prognostic implication.*

Keywords: *Spontaneous coronary artery dissection (SCAD), acute coronary syndrome, myocardial infarction, coronary angiogram*

Introduction

SCAD is defined as a non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen. [1] This separation can occur between the intima and media or between the media and adventitia, with intramural hematoma (IMH) formation within the arterial wall that compresses the arterial lumen, decreasing ante-grade blood flow and subsequent myocardial ischemia or infarction. [1,2] Spontaneous coronary artery dissection, also termed as dissecting aneurysm, intramural haemorrhage or haematoma, is an uncommon clinical entity. [3-7]

The sign & symptoms that raise the possibility of SCAD are as follows:

Clinical features that raises suspicion of SCAD
Myocardial infarction in young women (especially age ≤50)
Absence of traditional cardiovascular risk factors
Little or no evidence of typical atherosclerotic lesions in coronary arteries
Peripartum state
History of fibromuscular dysplasia
History of relevant connective tissue disorder: Marfan's syndrome, Ehler Danlos syndrome, Cystic Medial Necrosis, Fibromuscular Dysplasia
History of relevant systemic inflammation: Systemic Lupus Erythematosus, Crohn's disease, Ulcerative colitis, Polyarteritis Nodosa, Sarcoidosis
Precipitating stress events, either emotional or physical (intensive exercise)

Table 1: Depicting the clinical features that raises suspicion of SCAD

It usually tends to involve the outer media and causes luminal occlusion by pushing the inner media against the opposing wall. Clotted blood filling the false lumen may simulate coronary thrombosis at the naked eye, masking the dissection. Thus, the accurate scenario of this clinical condition may be underestimated. A review of published reports showed that 69% of the cases were diagnosed at necropsy. [7] Among them 80% of the cases were women, and more than 25% of these were in the peripartum period. [8-22] Involvement of the right coronary artery seems to be more common in men, whereas left anterior descending coronary artery involvement is frequent in women. Coronary artery dissection affects young adults; in a series of cases that were diagnosed before death. According to De Maio et al [6] mean age was 46 years in males and 38 years in females. The clinical presentation of this includes the entire spectrum of acute coronary syndrome and is primarily related to the extent of the dissection and the vessel involved. Nonetheless, sudden death without pre- evidence of myocardial infarction is much more frequent. [23] Survival is possible if obstruction of the lumen is incomplete, or an uncomplicated myocardial infarction. [24-27] Spontaneous healing of a coronary dissection has also been shown to occur, on both clinical and histological evidence. [13, 26]

When secondary causes are excluded, [11, 15, 24, 28-36] the etiology of it remains uncertain. Among

the risk factors hypertension has rarely been reported. [11] Pregnancy or oral contraceptives induced arterial wall changes have been well documented37; hormonal and haemodynamic factors also may contribute in weakening of the tunica media, thereby correlate well with higher incidence of spontaneous coronary dissection in the puerperium. [8-16,38,39]

A true pattern of cystic medial necrosis has been described in the involved coronary artery as the cause of spontaneous coronary artery dissection. [40-42] The vasa vasorum have been implicated as a possible initiating site for coronary dissection; thus, constituting the initial source of bleeding. As in aortic dissection, the source of blood could be the true lumen through an intimal tear, which has been detected in few cases. [43-46]

In many published cases, a fairly diffuse adventitial and peri-adventitial inflammatory reaction consisting mainly of eosinophils, was observed. [4,29,30,47-49] Because of the presence these "peri-arteritis like" adventitial change, it was proposed that the dissection is a result of lytic action of protease released from eosinophils. [30] However, it is likely that the peri-adventitial inflammation seen in some cases is reactive in nature, rather than causative. [49] Intense physical exercise as a precipitating factor of coronary artery dissection has already been described. [50, 51] Noteworthy is a report of cocaine induced coronary artery dissection, which adds this entity to the long list of cardiovascular complications of cocaine abuse. [52]

The in vivo diagnosis of spontaneous coronary artery dissection by selective angiography depends on the visualization of two lumina separated by a radiolucent intimal flap. [7, 53-55]

This procedure entails the risk of cardiac arrest by injection of hypertonic medium into the false lumen, thus aggravating the dissection. Some cases may not be recognized if an intimal tear does not occur or if the true lumen is severely narrowed or if the false lumen is occluded by a clot. The prognosis for these patients is poor. In a survey of 123 cases, Benham and Tillinghast found that 67% of the patients died and 33% survived, treated either surgically or medically.56 Since sudden death is the most frequent clinical presentation, the clinician is left without the time of any potential therapeutic option in the majority of cases. Medical treatment may play a palliative role. Ramamurti et al first reported the use of intravenous streptokinase in a case with left anterior descending coronary artery dissection.57 Several workers think that this treatment may be effective in lysing the clot in the false lumen and in re-establishing patency of the true lumen56; nonetheless, we wonder whether thrombolysis might aggravate the bleeding, thus worsening the coronary dissection. Although the outcome for patients with spontaneous coronary dissection is considered grim, with a high mortality rate, aggressive surgical

treatment in a recent series of 10 consecutive patients resulted in 100% survival. [7]

Forker et al carried out the first operation of for a spontaneous dissection of the right coronary artery in 1969 by performing an aorto- coronary bypass. [58] Thayer et al [5] recommended coronary artery bypass grafting for all patients with spontaneous dissection, whereas De Maio et al [6] advocated this intervention only in cases of left main disease, three vessel disease, few or recurrent ischaemia. Indeed, this technical approach may be considerably difficult in the setting of vessel wall dissection, with the false lumen underlying the site of graft anastomosis. [59] Successful surgical repair by extrusion of the intramural haematoma was reported in 1986 by Vicari et al. [60] In cases of severe heart failure following myocardial infarction caused by coronary artery dissection, cardiac transplantation has been performed and total artificial heart implantation attempted as a temporary measure. [61,62] Nowadays, new interventional techniques, such as stents and balloons, are being considered for the management of coronary dissection. [63, 64]

Methods

This retrospective cross-sectional analytical study was undertaken in the Department of Cardiology since October 2018 to October 2020. All the patients admitted in the Department of Cardiology who underwent for coronary angiography was enrolled in the study on the basis of inclusion & exclusion criteria. All the angiograms were analyzed meticulously for SCAD. Among 2880 cases total 7 were identified & diagnosed as SCAD. All the data of these 7 patients were studied. These 7 cases were sub-divided into further 3 groups on the basis of types of acute coronary syndrome. Group-I includes those patients with unstable angina, Group-II those patients with non-ST elevated ACS & Group-III was covered with those patients with ST elevated ACS. The study obtained ethical clearance from the institutional ethical clearance committee.

Selection criteria

a) Inclusion Criteria:

- All Patients admitted for coronary angiography.
- Age >20 years.
- Both males and females.

b) Exclusion Criteria:

- Age <20 years.
- Patients those who don`t fulfill the inclusion criteria.
- Chest pain due to other causes.

- Patients with valvular heart disease, congenital heart disease and cardiomyopathy.
- Patients having major non cardiovascular disorder causing elevation of Troponin-I such as severe renal impairment, prolonged immobilization, major surgery, chest trauma, myocarditis (pericarditis) & acute pulmonary embolism.
- Any systemic infection.
- Patients who were under chemotherapy on discovery of malignancy.

Results

This was a retrospective cross sectional analytical study conducted at the Department of Clinical & Interventional Cardiology, Evercare Hospital Dhaka, Bangladesh for a period of 24 months from October 2018 to October 2020. 2880 consecutive cases those who underwent for coronary angiogram were included in the study on the basis of inclusion and exclusion criteria. Among these cases SCAD was searched & diagnosed. Then total of 7 SCAD cases were identified & analyzed. Then these cases were subdivided into 3 groups on the basis of type of ACS. Group-I constitutes the patients diagnosed as unstable angina (UA), Group-II constitutes the patients diagnosed as non ST elevated ACS (NSTE - ACS) & Group-III constitutes the patients diagnosed as ST elevated ACS (STE - ACS).

Table II: Age distribution of the study population (n=7)

Age group (years)	**Group-I (n=1)**		**Group-II (n=2)**		**Group-III (n=4)**		p value
	Number	%	Number	%	Number	%	
20-30	0	0.0	0	0.0	1	25.0	0.292 ns
31-40	0	0.0	0	0.0	1	25.0	
41-50	0	0.0	2	100.0	0	0.0	
51-60	1	100.0	0	0.0	1	25.0	
61-70	0	0.0	0	0.0	1	25.0	
Mean ± SD	54.00±0.00		45.00±5.66		48.25±16.89		

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS

Age distribution table (Table II) of the study population showed that the individuals having unstable angina (UA) are more aged than the other groups. Most of the study population belonged to 40-60 year age group then the rest of the age groups. Significant differences could not be found between different age groups (p>0.05).

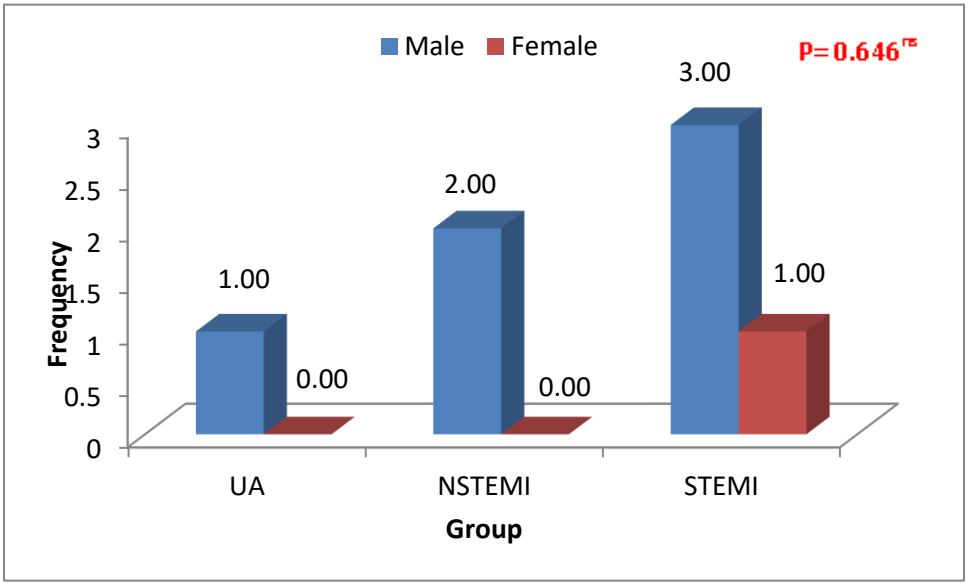

Figure 1: Bar diagram shows sex distribution of the study population (n=7).

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Sex distribution bar diagram (**Figure 1**) of the study population showed that most of the study population was male (85.7%). The rest were female. No significant difference was observed between genders (p>0.05).

Table III: Anthropometric status of the study population (n=7)

Parameters	Group-I (n=1)	Group-II (n=2)	Group-III (n=4)	p value
BMI				**0.752 ns**
Mean ± SD	25.07±0.00	25.04±0.67	26.96±3.68	
BSA				**0.802 ns**
Mean ± SD	1.63±0.00	1.71±0.17	1.73±0.11	

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Anthropometric status table (**Table III**) of the study population showed that the individuals who sustained ST elevated ACS (STE-ACS) was more obese than the other groups. No significant differences were found between different groups (p>0.05).

Table IV: Risk factors analysis of the study population (n=7)

Risk Factors		**Type of ACS**						p- Value
		Group-I (n=1)		**Group-II (n=2)**		**Group-III (n=4)**		
		No.	%	No.	%	No.	%	
Hypertension	No	1	100.0	0	0.0	2	50.0	**0.233 ns**
	Yes	0	0.0	2	100.0	2	50.0	
Diabetes	No	1	100.0	1	50.0	2	50.0	**0.646 ns**
	Yes	0	0.0	1	50.0	2	50.0	
F/H of CAD	No	1	100.0	2	100.0	4	100.0	**ND**
Smoking	No	0	0.0	0	0.0	1	25.0	**0.478 ns**
	Current	1	100.0	1	50.0	3	75.0	
	Ex	0	0.0	1	50.0	0	0.0	
Dyslipidaemia	No	1	100.0	0	0.0	3	75.0	**0.140 ns**
	Yes	0	0.0	2	100.0	1	25.0	

Unpaired t-test was done.

ns means not- significant.

ND means not-detected.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Risk factor analysis table (**Table IV**) of the study population showed that smoking remained the major risk factor followed by hypertension, diabetes & dyslipidaemia. But no statistical significant difference among the study groups (p>0.05) was noted.

Table V: Lipid distribution of the study population (n=7)

Lipid Profile	Group-I (n=1)	Group-II (n=2)	Group-III (n=4)	p value
Total Cholesterol	120.00±0.00	150.00±36.77	146.50±33.17	**0.736**^{ns}
LDL - C	85.00±0.00	67.00±1.41	83.00±29.50	**0.758** ^{ns}
HDL - C	34.00±0.00	42.50±4.95	42.00±9.63	**0.706** ^{ns}
TG	99.00±0.00	201.00±213.55	101.00±45.26	**0.612** ^{ns}

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Lipid distribution analysis table (**Table V**) of the study population showed that total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) & triglyceride (TG) were high in non ST elevated ACS group but low density lipoprotein cholesterol (LDL-C) was higher in unstable angina (UA) group. But no statistical significant difference among the study groups (p>0.05) was noted.

Table VI: Sugar profile of the study population (n=7)

Sugar Profile	Group-I (n=1)	Group-II (n=2)	Group-III (n=4)	p value
RBS	6.80±0.00	7.45±2.33	7.43±1.02	**0.924**^{ns}

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Sugar profile analysis table (**Table VI**) of the study population showed that non ST elevated ACS group had higher blood sugar level followed by ST elevated ACS group & then unstable angina group. But no statistical significant difference among the study groups (p>0.05) was noted.

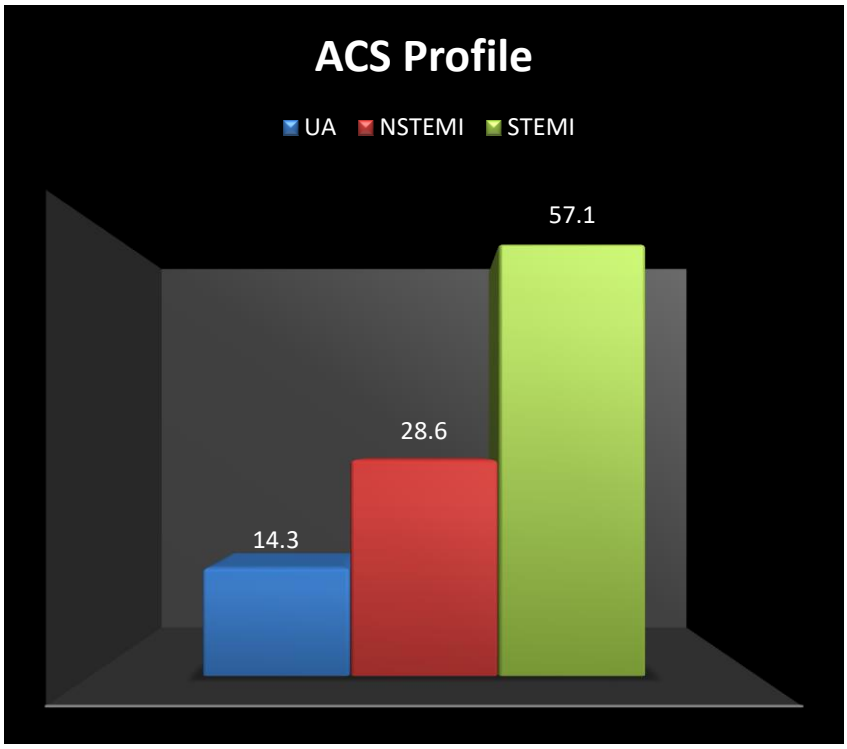

Figure 2: ACS profile of the study population (n=7).

ACS profile table (Figure 2) showed the different categories of ACS in which the study populations belong to. Majority (57.1%) sustained ST elevated ACS, and then non ST elevated ACS (28.6%) & lastly unstable angina group (14.3%).

Table VII: Cardiac Biomarkers profile of the study population (n=7)

Cardiac Biomarkers Profile	**Group-I (n=1)**	Group-II (n=2)	Group-III (n=4)	p value
Troponin-I	0.01±0.00	1.63±1.88	17.51±24.74	**0.388^{ns}**
BNP	12.00±0.00	16.25±7.50	17.50±3.54	**0.805^{ns}**

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

Cardiac biomarkers profile analysis table (Table VII) of the study population showed that ST elevated

ACS group topped first with both higher troponin-I & BNP levels followed by non ST elevated ACS group & then unstable angina group. But no statistical significant difference among the study groups (p>0.05) was noted.

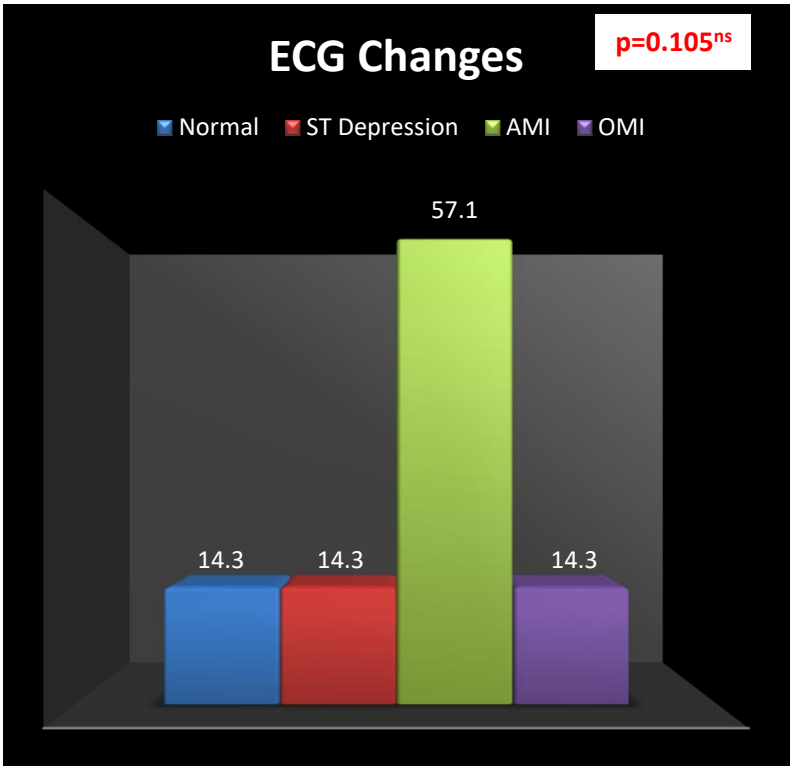

Figure 3: ECG changes of the study population (n=7).

ECG changes table (Figure 3) of the study population showed different ECG changes encountered among the study groups. But no statistical significant difference among the study groups (p>0.05) was noted.

Table VIII: LVEF profile of the study population (n=7)

LVEF Profile	**Group-I (n=1)**	Group-II (n=2)	Group-III (n=4)	p value
LVEF	65.00±0.00	50.00±14.14	32.50±6.46	**0.059ns**

Unpaired t-test was done.

ns means not- significant.

Group-I: Individuals with UA.

Group-II: Individuals with NSTE-ACS.

Group-III: Individuals with STE-ACS.

LVEF profile table (**Table VIII**) of the study population showed ST elevated ACS group had more depressed LV function followed by non ST elevated ACS group & unstable angina group. But no statistical significant difference among the study groups (p>0.05) was noted.

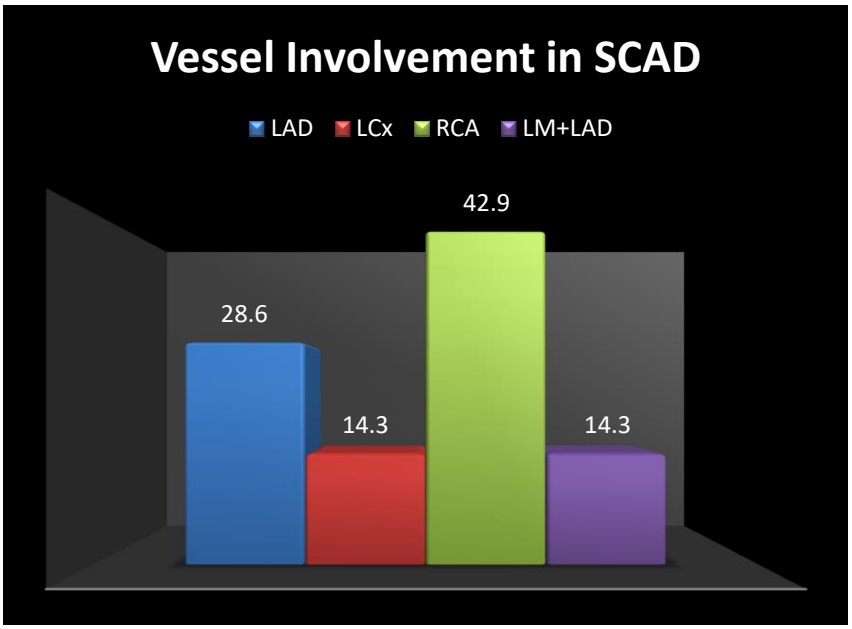

Figure 4: Vessel involvement in SCAD of the study population (n=7).

Vessel involvement in SCAD (Figure 4) of the study population showed majority had right coronary artery (RCA) involvement, followed by left anterior descending (LAD), left circumflex (LCx) and left main with left anterior descending (LM + LAD) involvement.

Table IX: Correlation analysis of the risk factors of the study population (n=7)

Dependent Variable (Y)	Independent Variable (X1.....X14)	*Coefficient (r) value*	p-value	Sig. Level
Vessel involved with SCAD	Age	0.089	0.85	NS
	Sex	0.611	0.145	NS
	BMI	0.348	0.445	NS
	Hypertension	-0.746	0.050	**S**
	Diabetes	-0.629	0.131	NS
	Smoking	-0.509	0.243	NS

	Troponin-I	-0.493	0.261	NS
	Total Cholesterol	-0.711	0.073	NS
	LDL Cholesterol	-0.323	0.48	NS
	HDL Cholesterol	0.569	0.182	NS
	Triglyceride	-0.675	0.096	NS
	ECG Change	-0.013	0.978	NS
	LVEF	0.133	0.776	NS
	Type of MI	0.133	0.775	NS

s means significant

ns means non-significant

Above table (Table IX) showed the regression analysis of the variables of the study population which depicted that among all study variables hypertension was the most important for developing SCAD in different types of ACS.

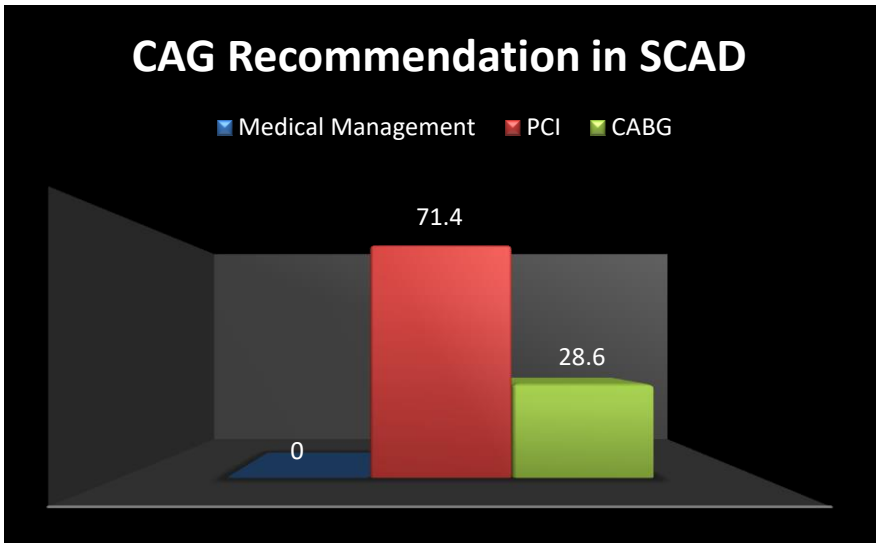

Figure 5: CAG recommendation in SCAD of the study population (n=7).

Coronary angiogram (CAG) recommendation in SCAD (Figure 5) of the study population showed majority (71.4%) were recommended for angioplasty (PCI). On the other hand 28.6% population was recommended for coronary artery bypass grafting (CABG).

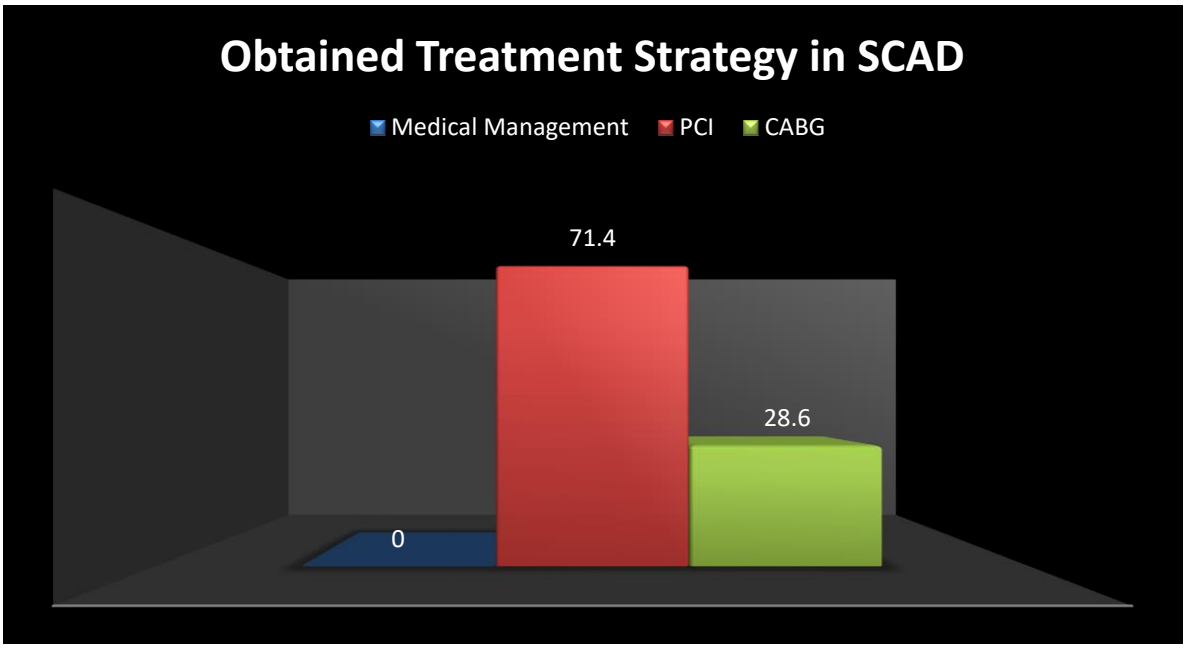

Figure 6: Obtained treatment strategy in SCAD of the study population (n=7).

Obtained treatment strategy in SCAD **(Figure 6**) of the study population showed majority (71.4%) took angioplasty (PCI) as there modality of revascularization. On the other hand 28.6% went for coronary artery bypass grafting (CABG).

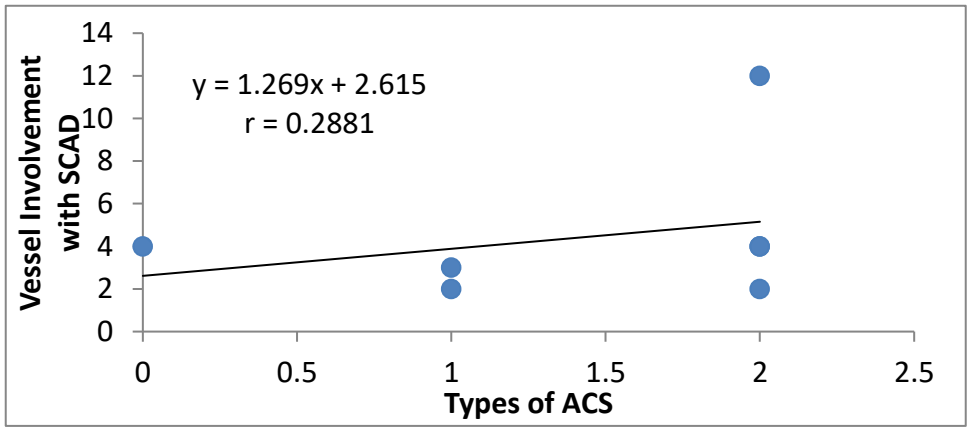

Figure 7: Correlation between Type of ACS and Vessel involvement with SCAD (n= 7).

Above figure (Figure 7) showed the Correlation between type of ACD and vessel involvement with SCAD (n= 7). It shows statistically significant mild negative correlation with low strength of association-correlation coefficient between type of ACD and vessel involvement with SCAD (R = 0.2881**, p=<0.001**) which means as the type of ACS worsens then the vessel involvement with SCAD deteriorates.

Discussion

Age and sex incidence

The mean age of presentation with SCAD is 30-45 years in many published papers (range 30-70 years) 67, 73, 74. In this study the maximum incidence of SCAD was in the age range of 40-60 years (57.1%) with the mean age approximately 49 years (Table I). 28.6% of the cases were below 40 years of age. Our study compares well with those findings. The incidence of SCAD in angiographic series varies widely from 0.07% up to 1.1% for patients who are referred for coronary angiography 68-74. The incidence rate of SCAD in our study was 0.24% which correlates well with other papers.

More than 70% of SCAD cases are women, and in approximately 30% it occurs during the peripartum period 66, 67. Among women, the incidence of SCAD was highest in women below the age of 40 years and decreased significantly with advancing age (figure 1). When only women below 50 years of age presenting with an acute coronary syndrome were considered, the prevalence of SCAD increased up to 8.7%, and reached 10.8% in the case of ST elevation myocardial infarction 65. In our study, SCAD was detected in only 1 female case. She was 30 years of age and also in her puerperium. She also presented with acute ST elevated ACS. So our study correlates well with the other papers. Majority of our study population (85.7%), were male. Here the difference with other papers probably due to our social structure. Females cannot easily avail the health care facilities in the subcontinent due to familial & social limitations and hence the discrepancy.

Risk factors

Incidence of diabetes was 42.9 % (Table IV) in our study and incidence of hypertension was 57.1% (Table IV) which again was higher. These higher incidences can be attributable to poor life style of the people in Indian subcontinent. We found that a portion of our SCAD patients had traditional cardiovascular risk factors including hypertension, hyperlipidaemia, smoking, and obesity. Other SCAD registries have shown similar co-morbid cardiovascular condition prevalence76 but SCAD is often described as affecting patients “with few or no traditional cardiovascular risk factors.”75 The basis of this description likely originates from observations that SCAD patients have a lower prevalence of traditional cardiovascular risk factors than the national, age-matched average.77–81 However, it is important to note that these risk factors are not absent and some risk factors such as hypertension are on par with age matched national prevalence.78

SCAD

We found that arterial abnormalities exist in SCAD patients beyond the dissection. We noted coronary vessel tortuosity was frequently present, with most cases demonstrating severe tortuosity. Similar to

previously published cohorts, extra-coronary fibro-muscular dysplasia (FMD) was present in slightly over 50% of patients.82-85 Even among patients without the classic beading of FMD, no patient had completely normal vasculature. These observations suggest that vessel fragility may play a role in the pathogenesis of SCAD.

SCAD has classically been viewed as a predominantly single-vessel disorder which is also noted in our study (85.7%) most commonly affecting the right coronary artery (42.9%) in contrast to left anterior descending artery (28.6%) which was more frequently identified as the involved vessel in most studies. Dissections in small, distal and branch vessels may be overlooked when angiographers focus only on the obvious infarct-related artery. Furthermore, this finding may reflect the better appreciation of SCAD.

Conclusion

SCAD is an infrequent condition that is under-diagnosed among patients presenting with ACS. Risk factors for SCAD are multifold, including young women, fibromuscular dysplasia, systemic inflammation, connective tissue disorders and pregnancy, and often compounded by precipitating stressors. The long-term outcome of patients who survived their initial SCAD presentation is good; however, recurrent events are frequent and these patients should be followed closely by cardiovascular specialists. Treatments typically entail conservative medical management for stable patients with ischemia resolution; however, revascularization with PCI or CABG may be necessary in a small proportion of patients.

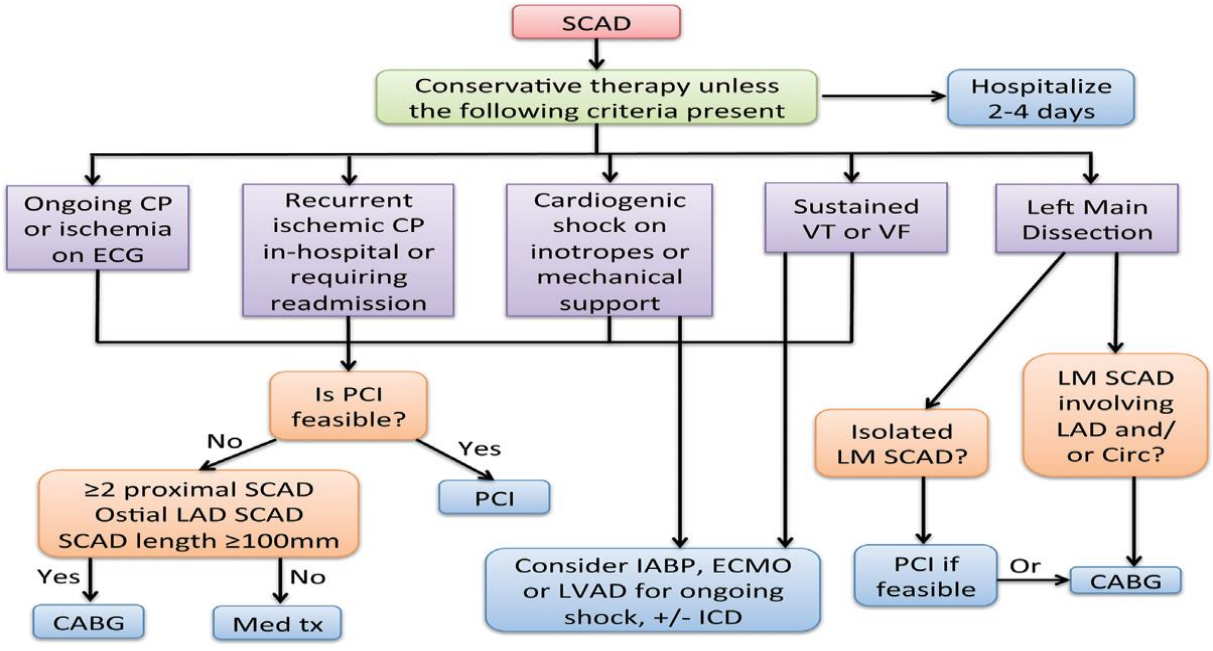

Figure 8: Management algorithm including revascularization for acute presentation of SCAD86.

In conclusion, the possibility of a spontaneous coronary artery dissection should be considered in any young adult woman who presents with an unexpected circulatory collapse, myocardial ischaemia, or infarction, without a previous history or risk factors, and not necessarily in puerperium. Suspicion of the diagnosis may then lead to emergency investigation of the coronary arteries, and surgical or interventional treatment. However, the high incidence of sudden death without premonitory symptoms casts doubt on the real possibility of prompt diagnosis and life saving treatment in the majority of cases. Limitation:

To the best of our knowledge, this is the novel retrospective study among patients with SCAD in Bangladesh. Although some limitations were acknowledged by the study team.

1.This was a single center study. By incorporating multiple centers will give more insight about the SCAD as a rare clinical entity and its clinical significance.

2. Interpretation of our findings might be limited by the sample size due to single center involvement. However, by including all adult patients in multiple centers for SCAD, we believe our study population would be representative of cases diagnosed and treated in Bangladesh.

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