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Clinical and Angiographic Profile of Patients with Spontaneous Coronary Artery Dissection in a Tertiary Care Centre

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Abstract

Background: Spontaneous coronary artery dissection is one of the rare causes of Acute coronary syndrome. Clinical presentation of the disease is variable in pattern and severity related to extent and development of rate of dissection. Initial reports on this condition were scarce as they were based on postmortem examination of fatal cases. Currently, Coronary angiography is the most widely used diagnostic technique in this condition. SCD may occur as an isolated phenomenon (I-SCD) or associated with coronary artery disease (A-SCD). Management depends on the clinical presentation and the results of coronary angiography.

Methodology: Reviewed meticulously 38,462 patients consecutive coronary angiogams retrospectively which were done between august 2010 to february 2014 using SCD definition[8] as defined for the study. Patients medical records were analysed thoroughly for clinica and demographic data.

Results: 57 Patients of spontaneous coronary artery dissection were identified after review of 38,462 coronary angiograms. Fifty four (94.7%) of study population were males. Mean age was 51.2 ± 10.6years. We found 29.8% of patients with hypertension, 8.8% obesity, 29.8% diabetes mellitus, 49.1% dyslipidemia, 54.4% old IHD and 77.2% smoking history. No patients were in peripartum period in the study. 45(73.7%) of them presented with STEMI, 4(7%) of them with NSTEMI.

Conclusion: SCAD is a rare condition with prevalence rate of 0.14.It is seen commonly in middle and elderly age group male population with predisposing factors for CAD. SCD presents predominantly as ACS. The diagnosis of SCAD is made principally with invasive coronary angiography.

Keywords: Spontaneous Coronary Artery Dissection; Acute Coronary Syndrome; Coronary Angiogram.

Introduction

Spontaneous coronary artery dissection (SCAD) is a rare condition which can lead to acute coronary syndrome. The clinical presentation is wide, ranging from simple chest pain to sudden cardiac death.[1,2,3,4,5,6] Clinical presentation of the disease is variable in pattern and severity related to extent and rate of dissection.[4] Coronary artery dissections can occur spontaneously or as a result of coronary angiography, coronary angioplasty, cardiac surgery procedures, blunt chest trauma or enlargement of aorta dissection. The diagnosis of SCAD can be made after elimination of all other secondary causes of coronary artery dissection.[1] The spectrum of clinical presentation can range from chest pain symptoms alone to ST-segment–elevation myocardial infarction (STEMI), ventricular tachycardia/fibrillation, and sudden death. SCAD may involve single to multiple coronary artery.[2] The population-based incidence of SCAD is unknown. While SCAD accounts for the cause of ACS, the

real prevalence of SCAD is still unknown.[3] The diagnosis of SCAD is made principally with invasive coronary angiography, although adjunctive imaging modalities such as computed tomography angiography, intravascular ultrasound, and optical coherence tomography may increase the diagnostic yield.[2] Coronary angiography is gold standard for SCAD detection even though it underestimates the presence of small intimal tears and not ideal for multiple plane projections.[9] Retrospective registry studies have reported SCAD detection in 0.07% to 1.1% of all coronary angiograms performed. Data from SCAD case series that by and large excluded atherosclerotic dissection demonstrate a younger, female preponderance and an association with peripartum or postpartum status.. The first case was reported in 1931, which was based on autopsy findings after the sudden cardiac death of a 42 year-old female. Since the first case, SCAD had been reported mainly in autopsy series. Currently, clinicians have been paying more attention to the presence of SCAD because coronary angiography is more frequently performed for patients with coronary artery disease. It has been reported that SCAD is associated with various pathophysiological situations, such as atherosclerosis, peripartum episode, collagen diseases, cocaine abuse, severe hypertension, oral contraceptives, exercise, fibromuscular dysplasia or vasospasm. The classic angiographic appearance of SCAD is with contrast media in the two lumens separated by a radiolucent intimal flap, with persisting contrast in the false lumen after the remainder of the vessel has been washed out. An intimal tear may or may not be present, and the dissection may be obscured by significant narrowing of the true lumen with vessel irregularity.[25] Dissection of the coronary artery results in separation of the different layers of the arterial wall with the creation of a false lumen. The dissection plane can be situated between the intima and the media or between the media and the adventitia. Haemorrhage into the false lumen followed by thrombosis can compress the true lumen of the coronary artery resulting in a non-occlusive or occlusive obstruction of the blood flow causing myocardial ischaemia. Angiographically coronary dissections can be graded according to the National Heart, Lung, and Blood Institute classification system developed by the Coronary Angioplasty Registry. This system grades coronary dissections based on angiographic appearance as types A to F.[8,10] SCD may occur as an isolated phenomenon (I-SCD) or associated with coronary artery disease (A-SCD).[5] Management options range from conservative medical treatment to percutaneous intervention or a surgical approach, depending upon the localization of the dissection, number of vessels involved and clinical scenario. The prognosis varies widely in the literature, but early diagnosis and appropriate management may significantly improve outcome.[6] There is no definite guideline on how to manage patients with SCAD.[7,8] Reports have demonstrated favourable outcomes with conservative management (with documented angiographic resolution), fibrinolysis, percutaneous coronary intervention (PCI), and coronary artery bypass grafting (CABG), but there have been no comparative studies of treatment modalities. Regardless of initial treatment strategy, in-hospital and early outcomes have in general been reported to be favourable.[3]

Materials and Methods

In this study, we Retrospectively reviewed 38,462 patients coronary angiograms. Out of which 57 cases of spontaneous coronary dissections were identified after careful angiographic review. After ruling out all secondary causes of coronary artery dissection, demographic, clinical, echocardiographic findings, angiographic profiles, treatment modality options given, in hospital outcomes of these patients are analysed and studied.

Patients and Definitions

A total of 38,462 coronary angiographies were performed, and the characteristics of individual coronary lesions were prospectively reported in a dedicated, relational database by institution cardiologists. This database specifically included the term "SCD" among the diagnostic variables. However, to avoid missing any patient with SCD, an additional retrospective search was performed, retrieving from the database of all the reports that included the description of "dissection" or "filling defects." After a careful initial screening, 2 experienced interventional cardiologists jointly reviewed all corresponding coronary angiograms. Vessels with a prior coronary intervention and those with iatrogenic coronary dissections were excluded. In addition, patients with a prior myocardial infarction (with or without previous thrombolysis) presenting linear intracoronary filling defects, compatible with residual thrombus at the culprit vessel, were also excluded. SCD was defined as a longitudinal radiolucent linear image (intimal flap) detected by angiography in

at least 2 orthogonal projections and confirmed by 2 experienced observers.[8] The diagnosis of SCD was confirmed in 57 consecutive patients that constitute the study population. Demographic, associations, clinical presentation, coronary distribution, treatment modality, and in-hospital outcomes were retrospectively collected through medical record and angiographic review. Coronary angiography was performed using a 6 Fr Judkins-type catheter via the femoral or radial approach. A dedicated database was designed for the purposes of the study. Serial electrocardiograms and cardiac markers were obtained in all patients. In the I-SCD group, a detailed clinical screening was systematically performed and same was collected. Specifically, these patients underwent a full blood analysis, including: blood count, coagulation profile, biochemistry, acute phase reactants (ultrasensitive C-reactive protein, erythrocyte sedimentation rate), lipid profile, thyroid function tests. An echocardiogram, exercise test (where indicated), and blood analyses were obtained. Myocardial infarction and TIMI flow were defined and used as per standard guidelines in the study. This prospective protocol, including the initial conservative management strategy, was approved by the Institutional Ethics Committee. Informed consent was obtained from all patients.

Coronary angiography

Coronary angiographic studies were performed by a femoral or radial approach and multiple angulated angiographic projections were obtained. SCD were classified according to the National, Heart, Lung, and Blood Institute (NHLBI) classification developed by the Coronary Angioplasty Registry[8,10,11,12] and to the presence or absence of concomitant CAD. This system grades coronary dissections based on angiographic appearance as types A to F. Type A dissections represent radiolucent areas within the coronary lumen during contrast injection, with minimal or no persistence of contrast after the dye has cleared. Type B dissections are parallel tracts or double lumen separated by a radiolucent area during contrast injection, with minimal or no persistence after dye clearance. Type C dissections appear angiographically as contrast outside the coronary lumen, with persistence of contrast in the area after clearance of dye from the coronary lumen. Type D dissections represent spiral luminal filling defects, frequently with extensive contrast staining of the vessel. Type E dissections appear as new, persistent filling defects. Type F dissection represent those that lead to total occlusion of the coronary artery,without distal anterograde flow. Associated CAD was defined as at least 1 coronary lesion (different from the SCD lesion) with a diameter stenosis 50% on visual assessment. The Thrombolysis In Myocardial Infarction (TIMI) flow grade classification was also used to characterize SCD lesions.[11] Reference segment, percent diameter stenosis, and lesions were noted.

Statistical analysis

Statistical analysis was performed with SPSS version 18.0. Results are expressed as mean± SD for approximately normally distributed variables and median (interquartile median) for skewed variables. Qualitative data are presented as numbers (%).

Results

Demographic Characteristics

Fifty-seven consecutive patients with SCAD were identified, of whom 54(94.7%) were male and 3(5.3%) were females. Mean age was 51.2±10.6 years.

Our Study showed hypertension (29.8%), obesity (8.8%), diabetes mellitus (29.8%), hyperlipidemia (49.1%), old IHD (54.4%) and smoking (77.2%). No patients in peripartum period were found in this study.

Clinical Presentation and Angiographic Distribution

STEMI was the presenting diagnosis in 73.7%, non-STEMI in 7%, unstable angina in 7% and PAD in 3.5%. Fourty-ninepatients (86%) reported chest pain during presentation, 2 (3.5%)patients had presented with ventricular tachycardia requiring emergent defibrillation.[2] (3.5%) patients each presented with CHB, LL Claudication, 1(1.8%) patient each had breathlessness and pedal edema associated with chest pain. Of the ventricular arrhythmic episodes, both were in the setting of STEMI. 26(45.6%) patients had preserved ejection fraction (EF≥50%),22(38.5%) had mild LV dysfunction (EF40–49%) and 9(15.7%) had moderate LV dysfunction(EF30–39%). The left anterior descending (LAD) artery was the most commonly involved vessel 28(49.1%) followed by RCA 26(45.6%) and LCX 1(1.8%). Multivessel coronary dissection at presentation was evident in 2 patients (3.5%). 2 patients (3.5%) demonstrated

2-vessel and none demonstrated 3-vessel dissection. No LMCA dissections were found. In 21(36.8%) involved proximal segments of vessels, 19(33.3%) involved mid segments, 10(17.5%) involved proximal and mid segments both and 5(8.8%) involved mid and distal segments. In 11(19.3%) patients found to have insignificant CAD, 25(43.9%) had SVD, 14(24.6%) had DVD and 7(12.3%) had TVD. 31(54.4%) patients showed isolated spontaneous coronary dissection(I-SCD) where as 26(45.6%) had associated coronary artery disease with SCD(A-SCD). 37(64.9%) patients CAG were in NHLBI(National Heart, Lung and Blood institute) group A-B-C and 20(35.1%) were in group D-E-F.17(29.8%) had showed TIMI 0–1 flow and 40(70.2%) had showed TIMI 2–3 flow. (Table 1)

Initial Management Strategy and Early Outcomes

Table 1: Demographic characteristics (n=57).

		Percentage
Age (yrs)	Mean 51.6±10.2	–
Sex n(%)		
Males	54	94.7
Females	03	5.3
Diabetes mellitus n (%)	17	29.8
Hypertension n (%)	17	29.8
Smoking n (%)	44	77.2
Obesity n(%)	05	8.8
Dyslipidemian (%)	28	49.1
Old IHD n (%)	31	54.4
Presentation n (%)		
Chestpain	49	86.0
VT(vent.tachycardia)	02	3.5
LL Claudication pain	02	3.5
CHB	02	3.5
Chest pain+DOE	01	1.8
Chestpain+p.edema	01	1.8
Diagnosis n (%)		
STEMI	42	73.7
AWMI	25	43.9
IWMI	17	29.8
NSTEMI	04	7.0
UA	04	7.0
Effort angina	05	8.8
PAD	02	3.5
EF by ECHO n (%)		
≥50	26	45.6
40–49	22	38.5
30–39	09	15.7
<30	00	–

37(64.9%) patients were advised optimal medical therapy, 15(26.3%) were advised to undergo PTCA, 4(7.0%) were advised to undergo CABG and 1(1.8%) patient was advised (Table 2)

MPI followed by PTCA. All 57 patients were discharged. There were no in-hospital mortality.

Table 2: Angiographic characteristics (n=57).

	Numbers	Percentage
Territory n(%)		
LMCA	0	–
LAD	28	49.1
LCX	26	45.6
RCA	01	1.8
LAD + RCA (Multi vessel)	02	3.5
Segments involved n(%)		
Proximal	21	36.8
Mid	19	33.3
Distal	01	1.8
Prox+mid	10	17.5
Mid+distal	05	8.8
Side branches	01	1.8
CAG Impression		
Insignificant	11	19.3
SVD	25	43.9
DVD	14	24.6
TVD	07	12.3
Associated lesions		
I-SCD	31	54.4
A-SCD	26	45.6
NHLBI Classification		
A-B-C	37	64.9
D-E-F	20	35.1
TIMI flow		
0 and 1	17	29.8
2 and 3	40	70.2
Advice given		
OMT	37	64.9
PTCA	15	26.3
CABG	04	7.0
MPI followed by PTCA	01	1.8
Early outcome		
In-hospital mortality	00	–
Discharged from hospital	57	100.0

Discussion

The incidence of SCAD in angiographic series varies widely from 0.07% up to 1.1% for patients who are referred for coronary angiography.[8,12–18] Vanzetto et al. reported that the prevalence of SCAD was 0.2%.[17] In our study the prevalence of SCAD is 0.14% among refered for angiography in our centre. In many previous studies, SCAD was thought to be a very rare cause of coronary

artery disease, mainly occurring in young females without classical coronary risk factors.[8] The mean age at presentation is 30–45 years (range 30–70 years).[8,17,20,21] More than 70% of SCAD cases are women. And in approximately 30% it occurs during the peripartum period.[8,19,20] Among women, the incidence of SCAD was highest in below the age of 40 years and decreased significantly with advancing age.[8] The majority of patients with SCAD diagnosed in vivo are young adults, with a mean age of 46 years for males and 38 years for females.[8,21] Women are commonly more affected than men.[6,19] De Maio et al in their study found a mean age of 46 yrs in males and 38 years in females.[30] Celik et al[33] proposed in his study that spontaneous coronary artery dissection is not a disease just of middle-aged women but can also be found in older patients with risk factors for atherosclerosis. They described a series of 9 patients with spontaneous coronary artery dissection. The mean age in this group was 55.7 years, and 7 patients were males. All these patients had multiple risk factors for atherosclerosis. In our study, Mean age was 51.2± 10.6 years with minimum age 30 yrs. and maximum age 74 yrs and with significant sex differences (Males 94.7%). In multiple prevous studies, One third of all SCAD cases in women occur in the peripartum period, of which one third occur in late pregnancy and two thirds in the early postpartum period.[8,20,23] The peak incidence is within the first 2 weeks after delivery. The earliest reported case presented at 9 weeks of conception and the latest 3 months postpartum. Only 30% of the patients in this group have known risk factors for coronary artery disease.[8,23] Patients with advancing age and multiparity have an increased risk for SCAD.[8,23] The left anterior descending artery was involved in 78% of cases, the left main in 24%, and multivessel dissection occurred in 40%.[20] In our study, only 3(5.3%) of patients were females and none of them were in the peripartum period.

Marysia. S. Tweet et al in their study showed a low proportion of hypertension and diabetes mellitus without significant sex differences but statistically higher rates of hyperlipidemia and tobacco use in men. They documented 19% of HTN, 3% of DM, 14% dyslipidemia, 28% of patients with smoking history and 10% contraceptive use.[2] Tsuyoshi Nishiguchi et al showed dyslipidemia in 30.8%, smoking in 7.7%, HTN in 54%, DM in 31% and obesity in 23% in their study.[3] In our study, we documented hypertension in 29.8%, obesity in 8.8% and diabetes mellitus in 29.8%, hyperlipidemia in 49.1%, old IHD in 54.4% and smoking in 77.2%. None of our patient were on oral contraceptives.

In a study by George L. Higgins et al,[34] all patients presented shortly after experiencing chest pain, which was described uniformly with classic ischemic modifiers suggestive of acute coronary syndrome. Associated symptoms and signs were reported in 44 cases and most frequently included dyspnea (34% of patients) and diaphoresis (30% of patients). Seven women (16%) had experienced previous self-limited chest pain. Marysia. S.Tweet et al[2] in their study showed Seventy-nine patients (91%) reported chest pain during presentation, and 12 (14%) experienced ventricular fibrillation or ventricular tachycardia requiring emergent defibrillation. Of the ventricular arrhythmic episodes, 9 were in the setting of STEMI and 3 with non-STEMI. Chest pain was the leading symptoms of patients in our study (86%), 3.5% of patients each presented with CHB, ventricular tachycardia and lower limb claudication. 1.8% have presented with chestpain with breathlessness and another 1.8% chestpain with peripheral edema. Both VT cases were associated with STEMI.

Tsuyoshi Nishiguchiet al[3] in their study showed that 62% of their patients of SCAD were found in STEMI. Marysia. S. Tweet et al[2] in their study showed STEMI was the presenting diagnosis in 49%, non-STEMI in 44%, and unstable angina in 7%. Fernando Alfonso et al[5] showed that 40% their study showed STEMI as the presenting diagnosis, non-STEMI in 36%, and unstable angina in 9%. In our study, STEMI was the presenting diagnosis in 73.7%, non-STEMI in 7%, and unstable angina in 7% and PAD in 3.5%.

Marysia. S. Tweet et al[2] in their study showed EF of 51±14% in total 87 patients and in a study by Fernando Alfonso et al[5] in their study showed EF of 57±16% in their total 45 patients. In our study, 26(45.6%) patients had preserved ejection fraction (≥50%),22(38.5%) had mild LV dysfunction and 9(15.7%) had moderate LV dysfunction.

Tsuyoshi Nishiguchi et al[3] in their study found LAD involvement in 62%,RCA involvement in 38% of patients. Marysia. S. Tweet et al[2] in their study showed LMCA involvement in 9%, LAD involvement in 71%, RCA involvement in 31%, LCX18%, multivessel in 23% of patients. Fernando Alfonso et al5 in their study showed LMCA involvement in 2%, LAD involvement in 53%, RCA involvement in 29%, LCX in 16%.

In a study by Verma PK et al,[27] The pattern of arterial involvement is similar to that of atherosclerotic disease, with 66% of cases affecting the LAD, 25% affecting RCA and 7% affecting the the left circumflex artery. Less than 10% of

cases report multivessel dissection and/or left main coronary artery involvement. In a study by Jorgensen MB et al,[32] among patients the dissection occurs in 70% of cases in the LAD, 20% in the right coronary artery (RCA), 4% in the circumflex artery (Cx) and in less than 1% in the left main coronary artery (LMCA). In our study, the left anterior descending (LAD) artery was the most commonly involved vessel 28(49.1%) followed by RCA 26(45.6%) and LCX 1(1.8%). Multivessel coronary dissection at presentation was evident in 2 patients (3.5%). 2 patients (16%) demonstrated 2-vessel and none demonstrated 3-vessel dissection. No LMCA dissections were found.

Fernando Alfonso et al[5] showed involvement of proximal segment in 13%, mid segment in 58%, distal segment in 18%,diffuse in 11% and bifurcation in 11% in their study. In our study, 21(36.8%) involved proximal segments, 19(33.3%) involved mid segments, 10(17.5%) involved proximal and mid segments both and 5(8.8%) involved mid and distal segments. In our study we also showed 11(19.3%) patients found to have insignificant CAD, 25(43.9%) had SVD, 14(24.6%) had DVD and 7(12.3%) had TVD.

Fernando Alfonso et al[5] in their study showed 60% of their total cases were isolated SCD(I-SCD) and 40% as SCD associated with coronary artery disease(A-SCD).They also showed NHLBI class A-B-C in 49%, class D-E-F in 31% and unclassified in 20%. In our study, 31(54.4%) patients showed isolated spontaneous coronary dissection (I-SCD) where as 26(45.6%) had SCD associated coronary artery disease (A-SCD) and 37(64.9%) in to NHLBI (National Heart, Lung and Blood institute) group A-B-C and 20(35.1%) in to group D-E-F. Fernando Alfonso et al[5] showed 11% of their patients in TIMI 0–1 flow group and 78% in TIMI 2–3 flow group. One more study by Tsuyoshi Nishiguchiet al[3] showed 39% of patients with TIMI 0–1 FLOW and 61% with TIMI 2–3 flow. In our study, 17(29.8%) had TIMI 0–1 flow and 40(70.2%) had TIMI 2–3 flow.

The prognosis of patients with SCAD depends on the clinical presentation and extent of dissection. Those with multivessel involvement or left main dissection usually carry a bad prognosis and present with extensive infarction or sudden death. On the other hand, those presenting with focal dissections localized to 1 vessel have generally a favourable prognosis.

In general, patients with SCAD who survive their initial event are reported to have good prognosis.[19] The optimal treatment for SCAD remains undetermined. However, it is notable that all patients who were treated with an initial conservative strategy experienced a benign in hospital course. These data support a conservative strategy in otherwise stable patients with normal flow in the affected coronary artery. A challenge may be that some continue to experience chest pain in the absence of ischemia, perhaps relating to perturbation in the vessel wall or alteration in vascular tone. Due consideration should be given to this possibility before revascularization is pursued on symptomatic grounds alone. In this regard, PCI was associated with elevated rates of technical failure relating to passage of coronary wire into the false lumen of the dissected vessel or loss of coronary flow through propagation of dissection and displacement of intramural hematoma by stent placement. This underscores the different mechanism of coronary obstruction in SCAD compared with atherothrombotic occlusion. It also highlights a major challenge in management, particularly in situations of on-going ischemia and infarction when time to restoration of coronary flow is critical in limiting myocardial injury.[2,24,29] It could be argued that the minimum intervention necessary to restore coronary flow is undertaken. Because complications from SCAD intervention were frequently related to the placement of stents (with resulting propagation of hematoma), the risk of stent placement should be weighed against the expectation of a favourable clinical outcome and of resolution of dissection with conservative management in suitable patients. Third, due consideration should be given to the periprocedural use of adjunctive imaging technologies such as intravascular ultrasound and optical coherence tomography both to determine extent of vessel wall disruption (which may be under appreciated at angiography) and to provide real-time guidance of interventional strategy. It is noteworthy that patients who underwent CABG as an initial strategy fared well in the short term despite left main involvement in 50%. However, it should be emphasized that the high rate of late bypass graft occlusion demonstrated in this study suggests that bypass surgery may not provide long-term protection against the effects of recurrent native coronary artery dissection.[2,26,27]

In the study by Marysia. S. Tweet et al[2] 50.5% of patients were managed conservatively, 44.8% underwent PCI, 4.5% were underwent CABG and there was 1(1.1%) in-hospital mortality. According to Kamineniet al,[31] men have higher survival rates than women (93% vs. 74%) and among women, those presenting in the peripartum period seem

to have a better outcome (mortality of 15% vs. 34%), probably due to the fact that non-pregnant women are older and have more risk factors31. In a study by Zampieri et al showed In-hospital mortality of SCAD is relatively low, with a mean rate of around 3%. Patients who survive the acute phase have a good long term prognosis, with a very low recurrence rate of SCAD or acute coronary syndrome, and a 95% 2 year survival rate.[14,19] In our study, 37(64.9%) were advised optimal medical therapy, 15(26.3%) were advised to undergo PTCA, 4(7.0%) were advised to undergo CABG and 1(1.8%) patient was advised MPI followed by PTCA. All 57 patients were discharged. There were no in-hospital mortality.

Conclusion

SCAD is a rare condition with prevalence rate of 0.14. It is seen commonly in middle and elderly age group male population with predisposing factors for CAD. SCD presents predominantly as ACS. The diagnosis of SCAD is made principally with invasive coronary angiography. Early recognition of SCAD and assessment of its severity by CAG helps in selection of treatment modality. Early conservative management and revascularisation where it is indicated carries better in- hospital outcome. Presence of associated CAD does not appear to bear clinical or prognostic implications (although the small size of the subgroups should be acknowledged). One should be aware of this diagnosis during the evaluation of patients with classic risk factors for coronary artery disease.

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