Original Article

Coronary angioplasty in spontaneous coronary artery dissection—Strategy and outcomes

Sanjai Pattu Valappil*, Mathew Iype, Sunitha Viswanathan, Alummoottil George Koshy, Prabha Nini Gupta, Radhakrishnan Vellikatu Velayudhan

Department of Cardiology, Government Medical College, Thiruvananthapuram, Kerala, 695011, India

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ABSTRACT

Objective: To study the clinical, angiographic and technical characteristics of patients with spontaneous coronary artery dissection (SCAD) undergoing percutaneous coronary intervention (PCI).
Methods: This was a retrospective single center study where patients with angiographically confirmed SCAD undergoing PCI over a period of 4 years (2013–2017) were analyzed. We also sought to identify the clinical and angiographic predictors of procedural failure during PCI.
Results: There were a total of 42 patients with angiographically confirmed SCAD during the study period of which 16 patients (38.1%) underwent PCI. 14 out of the 16 patients (87.5%) taken up for PCI had technical success. In all patients the lesion was initially attempted to cross with a floppy wire and if unsuccessful it was escalated to a hydrophilic wire and finally to a stiff wire The SCAD lesion was crossed with a floppy wire in 71.4% of patients, with a hydrophilic wire in 14.2% and a stiff wire in 7.1% of patients. Wire escalation was required in 5 patients (31.3%) and in 60% of cases there was a technical success after wire escalation. Presence of diabetes mellitus, hypertension, dyslipidemia, smoking, coexisting atherosclerosis, diffuse nature of the lesion, and baseline Thrombolysis in Myocardial Infarction (TIMI) ≤ 2 flow did not predict procedural failure during PCI.
Conclusion: PCI in SCAD is associated with a fair rate of technical success in our population. Choosing an initial floppy wire and then escalating to a hydrophilic wire followed by a stiff wire is an optimal revascularization strategy.

1. Introduction

Spontaneous coronary artery dissection (SCAD) is defined as the non traumatic or non iatrogenic dissection of an epicardial coronary artery.[1] SCAD is an oft-neglected cause of acute ischaemia in young women bereft of traditional atherosclerotic risk factors. History beckons us to the first documented case of SCAD by Harold Pretty in 1931, who made the diagnosis post-mortem in a 42 year old woman presenting with acute onset of vomiting after mistakenly attributing her symptoms to indigestion.[2]

SCAD is an uncommon angiographic entity with a reported prevalence of 0.1–4% among acute coronary syndrome (ACS) patients and 0.4% of patients presenting with sudden cardiac death.[3,4] SCAD should always be suspected in a young female presenting with ACS with specific predisposing factors such as fibromuscular dysplasia, multiple pregnancy, connective tissue diseases like Marfan's syndrome and Ehler Danlos syndrome, systemic inflammatory conditions, Raynaud's syndrome, coronary vasospasm and cocaine abuse.[5–7]

There are two postulated theories regarding the pathophysiology of SCAD. The traditional belief has been that SCAD occurs secondary to an intimal tear followed by dissection of blood into the intima-medial plane resulting in a false lumen.[8] This results in the formation of the type 1 SCAD as defined by contrast dye staining of the arterial wall with multiple radiolucent lumens.[9] Type 1 SCAD can easily be identified by conventional coronary angiogram. The second theory establishes that SCAD initiates following the rupture of the vasa vasorum externa which lies in the adventitia. This results in an intramural hematoma and with the expansion of the

* Corresponding author at: Bhavana No 10/28, 4th cross street East, Shenoynagar, Chennai, Tamilnadu, Pin code 600030, India.
E-mail address: sanjaiinorion@gmail.com (S.P. Valappil).

hematoma, there is compression of the true lumen. The hematoma may then extend proximally and distally resulting in a diffuse narrowing of the coronary artery and type 2 SCAD. Type 2 SCAD is difficult to recognize on conventional angiogram due to the absence of an intimal flap, nevertheless on close inspection there is an abrupt but subtle change in the luminal diameter of the vessel and is typically diffuse in nature (more than 20 mm).[1] Type 3 SCAD is an atherosclerotic mimic and is characterised by focal or tubular stenosis and cannot be easily identified by conventional angiogram. Suspicion arises due to the lack of atherosclerosis elsewhere in the coronary tree and absence of conventional cardiovascular risk markers. Intracoronary imaging shows multiple localized intramural hematomas.

Buccheria et al has devised a scoring system for the diagnosis of this elusive angiographic entity.[10] In a prospective study of 168 SCAD patients by Saw et al type 2 angiographic SCAD was the commonest (67.0%), followed by type 1 (29.1%) and type 3(3.9%).[5] In our study we identified patients with type 1 SCAD as evidenced by angiographic evidence of an intimal flap or multiple radio-luscent lumen. As we did not use any intracoronary imaging techniques like intravascular ultrasound (IVUS) or optimal coherence tomography (OCT) it was practically difficult if not impossible to identify patients with type 2 or type 3 SCAD.[11] Intracoronary imaging techniques are currently an essential part of the management of SCAD particularly in the diagnosis of type 2 and type 3 SCAD. OCT owing to its higher spatial resolution is able to delineate the intimal characteristics of the dissection flap albeit at the cost of lower tissue penetration which prevents it from identifying the transverse extent of the intra mural hematoma. IVUS despite its lower spatial resolution has a better tissue penetration and is prefered over OCT because there is a risk of propagating the dissection by positive pressure perfusion during OCT.[12] Stable patients with SCAD are by default offered conservative management owing to the complex nature of the disease. Revascularisation is suggested only when patients have features of ongoing ischemia. PCI in SCAD is traditionally associated with high rates of technical failure. Studies examining the technical characteristics and hardware used during PCI in SCAD are limited to anecdotal to case reports. The current study was undertaken to analyze the clinical, angiographic and procedural characteristics of the patients who underwent PCI for SCAD in our centre over a period of 4 years.

2. Methodology

2.1. Materials and methods

This was a retrospective analysis of patients with SCAD undergoing percutaneous coronary intervention (PCI) in the department of cardiology, Government Medical College Thiruvananthapuram. Our cardiac catheterization registry was screened with the relevant key words 'Spontaneous coronary artery dissection' from the period of January 2013 to January 2017. The search yielded 111 results from a database of more than 12000 patients. After excluding iatrogenic dissection; the relevant angiograms were reviewed by 2 senior independent non blinded cardiologists. A diagnosis of SCAD was entertained only when the decision of the cardiologists were concordant. The final filter yielded a total of 42 patients with angiographically confirmed SCAD. Of these 42 patients; 16 underwent PCI. We analyzed the clinical; angiographic and technical characteristics of these patients. We also sought to identify the clinical and angiographic characteristics which could predict technical success or failure during PCI.

2.2. Definitions

Spontaneous coronary artery dissection: SCAD was defined by the presence of an angiographically confirmed dissection plane in a major epicardial coronary artery with or without the presence of coexisting atherosclerosis.[1]

Technical success: Technical success in PCI was defined as the ability to cross the SCAD lesion successfully followed by an improvement in the TIMI flow ≥ 2 from the baseline after balloon dilatation or stenting.

2.3. Statistical analysis

Continuous variables were expressed as minimum, maximum, mean, standard deviation (SD), and qualitative data were presented as percentages and frequencies. Continuous variables were analysed by a ' Student ' t-test and categorical variables by the Chi square test when appropriate. Univariate and multivariable logistic regression analysis was used to identify the independent risk factors associated with procedural failure during PCI. The results of this model were presented as an Odds Ratio (OR) and a 95% confidence intervals (95% CI) for OR. The statistical analyses were performed with SPSS software (version 17.0). A 2-sided probability value of 0.05 was considered to indicate statistical significance.

3. Results

3.1. Patient demographics

There were a total of 42 patients with angiographically confirmed SCAD over the study period of 4 years. All patients had type 1 SCAD. Out of these 16 patients (38.1%) underwent PCI with Drug eluting stents (DES). The primary indication for PCI was ongoing ischemia and easily provocable angina. The mean age of the patients was 51 ± 9.14 years and 87.5% were male. Majority of the patients presented as an ACS: 43.75% as ST elevation myocardial infarction and (STEMI) and 18.75% as unstable angina (UA)/non ST elevation myocardial infarction (NSTEMI). Half of the patients had a history of severe exertion prior to the onset of SCAD. Though intense valsalva like manoeuvres during parturition is said to be a common precipitating cause of SCAD, the two women in our series were postmenopausal. Among the patients presenting with STEMI, SCAD was noted in the non infarct related artery in 42.9% of cases. Screening for iliac artery dissection and renal artery involvement (in patients with hypertension) was done to rule out fibromuscular dysplasia. Unexpectedly, this did not yield any positive results. The baseline demographics of the patients undergoing PCI is shown in Table 1 below.

Table 1
Baseline clinical characteristics of patients undergoing PCI for SCAD.

Baseline Demographics	Percentage/Mean
Mean Age	51 ± 9.14
Male, n(%)	16 (87.5)
Mean LVEF,%	52 ± 12.97
Hypertension, n(%)	5(31.3)
Diabetes Mellitus, n(%)	6(37.5)
Dyslipidemia, n(%)	5(31.3)
Smoker, n(%)	6(37.5)
Family History of CAD, n(%)	2(12.5)
Chronic Stable angina, n(%)	6(37.5)
Unstable angina/NSTEMI, n(%)	3(18.75)
STEMI, n(%)	7(43.75)

3.2. Angiographic characteristics

3.2.1. Associated atherosclerosis

Isolated SCAD (SCAD without coexisting atherosclerosis) was seen among 50% of the cases and the remaining 50% patients had SCAD with associated atherosclerosis.

3.2.2. Distribution of SCAD

14 out of the 16 patients 87.5% had single vessel SCAD, whereas in two patients SCAD was noted in more than one major epicardial vessel. The LAD (Left anterior descending) and the RCA (Right coronary artery) were the commonly involved vessels and in 38.9% of patients PCI was done to each of the vessels. SCAD was limited to the proximal vessel in 27.7% of cases and in another 27.7% of cases the dissection extended from the proximal to the mid segment. Isolated distal vessel segment involvement was seen in 16.6% of cases. Majority of the patients (61.1%) had diffuse (more than 20 mm) SCAD and this was reflected in the mean stent length which was 41.62 ± 14.51 mm.

3.2.3. Tortuosity of the vessel

Tortuosity which has been noted as an independent angiographic marker of SCAD was also analyzed.[13] Severe tortuosity (>3 consecutive segments with >90° bends) was noted in 43.8% and mild tortuosity (>3 consecutive segments with >45–90° bends) was noted in 18.75% of patients. Nearly 72.2% of patient had TIMI 3 flow at baseline angiography. The baseline angiographic characteristics of patients undergoing PCI for SCAD are shown in Table 2 below.

3.3. Percutaneous Coronary intervention

3.3.1. Successful PCI

Fourteen out of the 16 patients (87.5%) taken up for PCI had a technical success as defined by the ability to cross the SCAD lesion successfully followed by an improvement in the TIMI flow ≥ 2 from the baseline after balloon dilatation or stenting, out of these 1 patient (6.25%) underwent plain old balloon angioplasty (POBA) and 13 patients (81.25%) underwent PCI with DES. In all patients the lesion was initially attempted to cross with a floppy wire and if

unsuccessful it was escalated to a hydrophilic wire and finally to a stiff wire The SCAD lesion was crossed with a floppy wire in 71.4% of patients, with a hydrophilic wire in 14.2% and a stiff wire in 7.1% of patients. Wire escalation was required in 5 patients (31.3%) and in 60% of cases there was a technical success after wire escalation. Among the patients who underwent a successful PCI, none of them had any in hospital events (periprocedural myocardial infarction, contrast induced nephropathy, local site complications) and all patients were stable at discharge. Fig. 1 illustrates a successful PCI done to a patient with SCAD of the RCA.

3.3.2. Unsuccessful PCI

Among the two patients with failed PCI,the first patient presented with chronic stable angina with low ejection fraction of 38% and regional wall motion abnormality (RWMA) of the LAD and the left circumflex (LCX) territory. His angiogram showed a diffuse SCAD of the LAD with TIMI 2 flow and SCAD of the LCX also. Despite wire escalation upto Hi-Torque Progress 200 XT (Abbott Vascular Co., Santa Clara, California, USA) the lesion was unable to be crossed. Final check shot showed TIMI 1 flow due to partial closure of the true lumen. The second patient presented as an inferior wall with right ventricular myocardial infarction who was thrombolyzed with streptokinase and subsequent elective coronary angiogram showed a discrete eccentric 90% lesion in the proximal RCA which was the infarct related artery. He also had a discrete SCAD of the ostium of the Major obtuse marginal (OM) vessel. The Proximal RCA was stented and then it was decided to tackle the OM SCAD as it was a large 3.5 mm vessel subtending a significant area of the myocardium with TIMI II flow distally. After the initial failure with a floppy wire, the OM was crossed with a Hi-Torque Whisper Extra Support guidewire (Abbott Vascular Co., Santa Clara, California, USA); however, despite repeated attempts it was unable to track even a 1 mm balloon across the lesion, hence it was decided to abandon the procedure and keep him on optimised medical management.

3.3.3. Stents

The mean stent diameter was 3.17 ± 0.34 mm. 11 out of the 13 stented patients required only a single stent. In 2 patients (12.5%) a second stent was required due to proximal extension of the dissection during stenting. The mean dye volume used was 189.38 ± 69.3 ml and the average flouroscopic time was 10.42 ± 6.88 min. The procedural characteristics of the patient are shown in Table 3 below.

3.3.4. Predictors of PCI procedural failure

The presence of diabetes mellitus, systemic hypertension, dyslipidaemia, smoking, coexisting atherosclerosis, diffuse lesion, and baseline TIMI ≤ 2 flow did not predict procedural failure during PCI. This is shown in Table 4.

3.3.5. Follow up of patients

The current cohort of patients underwent clinical follow-up. The period of follow up ranged from eight months to three years post PCI. Out of the 14 patients who underwent PCI, none of the patients had cardiac or non cardiac death, non fatal myocardial infarction, target vessel revascularization (TVR), target lesion revascularization (TLR), stent thrombosis or angina at follow-up. The two patients who faced technical failure during PCI were kept on optimised medical management and did not face any adverse cardiac events.

4. Discussion

The current study was undertaken to analyze the technical strategies and procedural outcomes of patients with SCAD

Table 2
Baseline Angiographic characteristics of patients undergoing PCI for SCAD.

| Angiographic characteristics	Percentage/Mean
Isolated SCAD,n(%)	8(50)
Atherosclerosis associated SCAD, n(%)	8(50)
STEMI with SCAD in IRA, n(%)	4(57.1)
STEMI with SCAD in non IRA, n(%)	3(42.9)
Single vessel SCAD, n(%)	14(87.5)
Two vessel SCAD, n(%)	2(12.5)
LAD, n(%)	7(38.9)
DIAGONAL, n(%)	0
LCX, n(%)	2(11.1)
OM, n(%)	2(11.1)
RCA, n(%)	7(38.9)
RAMUS, n(%)	0
Proximal vessel only, n(%)	5(27.7)
Proximal to mid vessel, n(%)	5(27.7)
Mid vessel only, n(%)	2(11.1)
Mid to distal, n(%)	3(16.6)
Distal vessel only, n(%)	3(16.6)
Discrete, n(%)	2(11.1)
Tubular, n(%)	5(27.8)
Diffuse, n(%)	11(61.1)
Tortuosity > 90%, n(%)	7(43.8)
Tortuosity < 90%, n(%)	3(18.75)
No Tortuosity, n(%)	6(37.5)
TIMI 3 flow, n(%)	13(72.2)
Chronic Total Occlusion, n(%)	1(5.6)
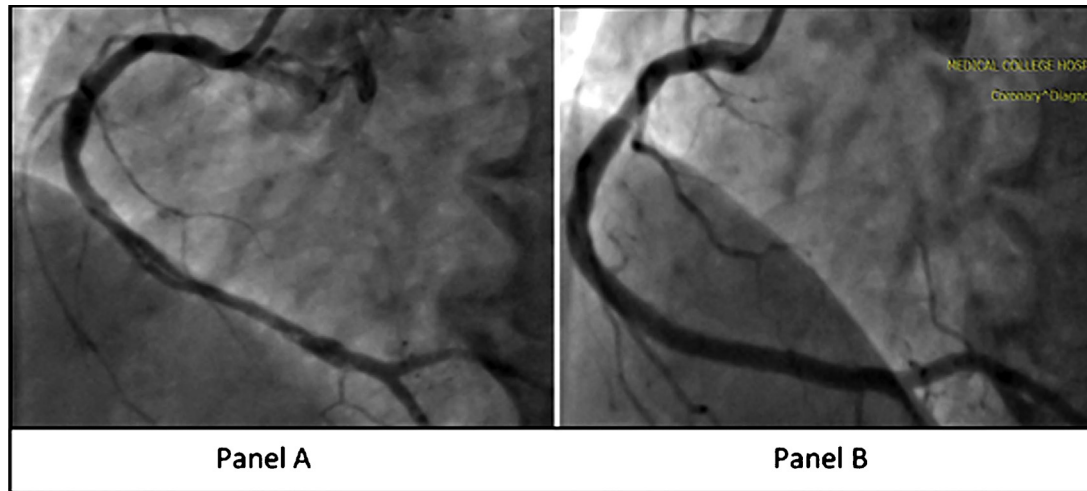
Fig. 1. Panel A- Left anterior oblique (LAO) view shows a spiral coronary artery dissection extending from the mid RCA upto the precrux RCA with TIMI 3 flow distally. The lesion was crossed successfully with Hi-Torque Balance Middleweight (BMW) Universal II guidewire (Abbott Vascular Co., Santa Clara, California, USA). Panel B −Final check shot after stenting the mid to precrux RCA with 3 * 48 Xience Xpedition (Abbott Vascular Co., Santa Clara, California, USA) shows the complete scaffolding of the dissected segment.

undergoing PCI. Interestingly our study had a high rate of procedural success of 87.5% in contradistinction to the previous studies where PCI in SCAD had a failure rate of 50%.[14] There are many technical impediments during PCI in SCAD. The foremost is the crossing of the lesion and parking the wire into the distal true lumen. In reality the challenge begins even before this critical step. Utmost care should be taken even at the outset during cannulation of the coronary ostium. It is imperative to use delicate manoeuvres and injection of iodinated contrast should be done with minimum force. Extension of the dissection has been described during overzealous injection of contrast media.[8]

4.1. Coronary wires used

In all our patients we attempted to cross with a floppy wire (Hi-Torque Balance Middleweight (BMW) Universal II guidewire (Abbott Vascular Co., Santa Clara, California, USA)) and escalated to a hydrophilic wire (Hi-Torque Whisper guidewire (Abbott Vascular Co., Santa Clara, California, USA)) and then to a stiff wire in case of failure. This was an institutional protocol that was adopted in view of its congruity and practicality. Using a non traumatic

workhorse wire as the initial wire allowed for successful PCI in 71.4% of our cases. Escalating to a fully hydrophilic wire permitted an additional success of 14% because of the wire's ability to insinuate itself across the dissection flap into the true lumen. However utmost caution should be exercised since the hydrophilic wire can just as easily enter the false lumen and extend the dissection. It is imperative to ensure the wire is in the true lumen by attempting to enter into the side branches along the course of the vessel or by using intracoronary imaging techniques. The final arrow in our quiver was to use a stiff wire; this can help to cross the lesion by way of subintimal tracking through the false lumen and re-entry into the true lumen. Coincidentally as many as 50% of our SCAD patients had associated atherosclerosis; non passage of a workhorse or hydrophilic wire could very well be due to the tightness of the lesion and in such instances escalating to a stiff wire achieved technical success in one of the three patients in whom this strategy was adopted. Among the two patients in whom technical failure was met, in one patient it was unable to cross the wire into the distal true lumen and in the other patient despite crossing the lesion it was unable to pass a balloon across the lesion.

4.2. Stent optimization

The next most important step is optimizing the stent size and length. Many a times it is possible that the stent is sized without

Table 3
Procedural characteristics of patients undergoing PCI for SCAD.

PCI characteristics	Percentage/Mean
Successful PCI, n(%)	13(81.25)
POBA, n(%)	1(6.25)
Wire in false lumen/Failed PCI, n(%)	2(12.5)
Lesion crossed with Floppy wire, n(%)	10(71.4)
Lesion crossed with Hydrophilic wire, n(%)	2(14.2)
Lesion crossed with Stiff wire, n(%)	1(7.1)
Wire escalation required, n(%)	5(31.3)
Success after wire escalation, n(%)	3(60)
Overlapping Stenting, n(%)	2(12.5)
Stented segment 10–20 mm, n(%)	1(6.25)
Stented segment 20–30 mm, n(%)	1(6.25)
Stented segment 30–40 mm, n(%)	5(31.25)
Stented segment 40–50 mm, n(%)	4(25)
Stented segment 50–60 mm, n(%)	1(6.25)
Stented segment 60–70 mm, n(%)	1(6.25)
Mean stent length, mm	41.62 ± 14.51
Mean Stent size, mm, mm	3.17 ± 0.34
Mean Dye Volume, ml	189.38 ± 69.3
Mean Flouro Time, mt	10.42 ± 6.88

Table 4
Clinical and Angiographic predictors for technical failure during PCI.

Clinical and Angiographic predictors		Patient%	Technical failure,%	p
Diabetes Mellitus:	Yes	37.5	33.3	0.889
	No	62.5	30	
Hypertension:	Yes	31.3	40	0.611
	No	68.7	27.3	
Dyslipidemia:	Yes	31.3	40	0.611
	No	68.7	27.3	
Smoker:	Yes	37.5	16.7	0.330
	No	62.5	40	
Atherosclerosis SCAD:	Yes	50	37.5	0.590
	No	50	25	
Diffuse lesion:	Yes	61.1	22.2	0.377
	No	38.9	42.9	
Baseline ≤ TIMI 2 flow:	Yes	27.8	66.7	0.94
	No	72.2	11.1	

taking into account the prescence of an intramural hematoma and subsequent resolution of the hematoma can result in stent strut malapposition and stent thrombosis. Nonetheless oversizing the stent can result in the disastrous consequence of a proximal edge dissection and extension of the dissection flap into the aorta, owing to the delicate nature of the coronary artery in such patients. Thus stent sizing should be carefully decided after considering the proximal and distal reference vessel beyond the dissection. Similarly it is recommended that the stent length is chosen after considering an additional 5–10 mm beyond the margins of the dissection to prevent extension of the hematoma and vessel flow compromise. Despite these measures in two of our patients there was proximal shift of the hematoma requiring overlapping with a second stent. IVUS and OCT may immensely help in proper sizing of the stent and choosing a stent of adequate length. In extensive SCAD lesions where a single stent may not cover the dissection it is recommended that we stent the distal segment first followed by the proximal segment and finally the mid part to prevent hematoma extension. In one of our cases balloon dilatation resulted in optimal flow and we chose defer stenting owing to the small size of the vessel (Obtuse marginal). Yamanaga et al suggest the novel concept of providing diastolic augmentation of coronary blood flow after balloon dilatation to preserve the patency of the true lumen.[15] In guide catheter induced iatrogenic dissections, where there is an unequivocal single proximal propagation point of the dissection, reports suggest that it is possible to seal the dissection entry point with a single stent meticulously placed in the aorto ostial location.[16,17]

We tried to analyze the factors that could predict technical failure among the patients undergoing PCI. Presence of diabetes mellitus, hypertension, dyslipidemia, smoking,coexisting atherosclerosis, diffuse lesion, and baseline TIMI $\leq$ 2 flow did not predict procedural failure during PCI. These findings were corroborated in the study by Tweet et al.[14]

5. Limitations of the study

This was a single centre study with a limited number of patients. We run the small risk of misinterpreting a partially recanalised thrombus as SCAD as intracoronary imaging techniques was not employed in our patients.

6. Conclusions

6.1. Management of SCAD

The optimal management of patients with SCAD has been clearly delineated with the emergence long term follow up studies. Despite the absence of any randomized clinical trials comparing the various treatment modalities in SCAD, it is reasonable to offer conservative treatment in stable patients and in patients with extensive dissection or dissection involving distal vessels where PCI or CABG may not be feasible. Masterly inactivity offers the best result in such patients as a good number of vessels show spontaneous healing at follow-up.[18] Revascularization should be attempted only when patients present with ongoing ischemia. The revascularization strategy depends on a multitude of clinical and angiographic factors including initial presentation, hemodynamic stability, ongoing ischemia, site of dissection, extent of dissection, landing zone for stent placement, distal graftable vessels.

6.2. CABG in SCAD

Coronary artery bypass grafting (CABG) as a revascularization strategy has been advised only in patients with Left main SCAD or multivessel SCAD. The problems in CABG are twofold, as many of the dissections undergo spontaneous healing over time studies have shown high rates of graft occlusion. Secondly owing to the diffuse nature of the disease, there might not be adequate distal targets for the grafts. This is reflected in the seminal study by Tweet et al which showed a 73% bypass graft occlusion (11 out of 15 bypass grafts) at the end of 10 years.[19]

6.3. PCI in SCAD

PCI has traditionally been associated with a high rate of technical failure. In our cohort of patients we achieved a high rate of technical success (87.5%) with a reasonable fluoroscopic time and dye usage. To the best of our knowledge, the current study is the first study to focus on the technical aspects, strategies employed and procedural outcomes during angioplasty in SCAD.

It is imperative to understand that even after a dissection has been scaffolded with a stent, the patient still runs the risk of recurrent SCAD in other vessels and higher rates of target vessel revascularisation (TVR) due to the long stents placed.[14]

In summary, SCAD is an enigmatic disease and the patients should be informed regarding the complexity of their condition. The decision to revascularize a patient with SCAD is always challenging and should be individualized.

Conflict of interest

None.

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