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Spontaneous Coronary Artery Dissection: Report on 20 Cases at Multiple Centers and a Review of the Literature

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Abstract

Spontaneous coronary artery dissection (SCAD) is an uncommon etiology of Acute Coronary Syndrome (ACS); however, appropriate treatment based on early diagnosis can improve outcomes. We screened medical records of all ACS patients who were admitted to one of 8 different centers (Cardiovascular Research Consortium-8 Universities) and underwent emergent coronary angiography from January 2001 to December 2014. From these ACS patients, we selected the patients with SCAD based on a review of the results of coronary angiography. Patient demographics, treatment, and in-hospital and long-term outcomes were determined from a review of medical records and angiographic findings. Of the 9377 ACS patients, 20 (0.21%) were diagnosed with SCAD. In these 20 SCAD patients, the mean age was 48.6 ± 12.0 years old, and 19 patients were female (95.0%). In 3 patients (15%), SCAD was associated with pregnancy. Coronary spasm was associated with SCAD in 2 patients (10%). Two patients (10%) were treated conservatively, and percutaneous coronary intervention was performed in 18 patients (90%). Two patients (10.0%) received target lesion revascularization, and one patient (5%) received coronary artery bypass grafting (CABG). SCAD recurred in one patient (5%), and there was in-hospital death in one patient (5%) after CABG. The patient demographics and outcomes in this study were compatible with recent reports of SCAD, except for the trigger of coronary spasm. SCAD should be suspected in middle-aged female ACS patients, and conservative treatment is recommended when there is no ongoing ischemia or left main trunk dissection. In addition, close follow-up is important.

Keywords: Spontaneous coronary artery dissection; Marfan syndrome; Coronary spasm; Iatrogenic coronary artery dissection

Introduction

Spontaneous coronary artery dissection (SCAD) is an uncommon etiology of acute coronary syndrome (ACS); however, appropriate treatment based on early diagnosis can improve outcomes. The treatment of SCAD includes noninvasive drug therapy, Percutaneous Coronary Intervention (PCI), and Coronary Artery Bypass Grafting (CABG). The treatment strategy should be selected based on the morphology of dissection and the severity of the condition in patients with SCAD. At the present time, there are no guidelines for SCAD treatment, because SCAD per se is relatively rare and includes various morphologies and abnormalities of the vessel. The first SCAD case in the world was reported in 1931 [1]. Recently, the number of clinical studies on SCAD evaluated with intravascular imaging has been increasing, and there is more widespread recognition of SCAD [2-5]. In the present study, we reviewed 20 patients with angiographically confirmed SCAD, who were admitted to one of 8 different centers (Cardiovascular Research Consortium-8 Universities) with a diagnosis of ACS from January 2001 to December 2014. In addition, we performed a brief review of the literature.

Methods

We screened medical records of all ACS patients who were admitted to one of 8 centers (Cardiovascular Research Consortium-8 Universities) and underwent emergent coronary angiography from January 2001 to December 2014. From these ACS patients, we selected the patients with SCAD based on a review of the results of Coronary Angiography (CAG). Patient demographics, potential etiologic associations, clinical presentations, coronary distributions, treatment modalities, and in-hospital and long-term outcomes were determined through a review of medical records and angiographic findings.

The angiographic criterion for SCAD was the presence of a spontaneous separation of the coronary artery wall that was not iatrogenic or related to trauma. The contemporary use of the term SCAD is typically reserved for the nonatherosclerotic variant, and most modern series exclude SCAD due to atherosclerotic coronary artery disease. For the purpose of our study, and because this disease is

distinct from atherosclerotic disease, the term SCAD refers to "nonatherosclerotic SCAD".

This retrospective study was approved by the local institutional review boards. All SCAD patients included in the study agreed to the use of their records for research.

Results

Of the 9377 ACS patients, 20 patients (0.21%) were diagnosed with SCAD (Table 1). In these 20 SCAD patients, the mean age was 48.6 ± 12.0 years old, and 19 patients were female (95.0%). There was ST elevation myocardial infarction (STEMI) in 18 patients (90.0%), non-ST elevation myocardial infarction in 1 (5.0%), and unstable angina pectoris in 1 (5.0%).

There were a total of 23 dissected vessels in the 20 patients, and 3 patients had 2 dissected vessels. The right coronary artery (RCA) was most often dissected (12/23 vessels, 52.2%), followed by the left anterior descending artery (LAD) (8/3 vessels, 34.8%), the left circumflex artery (LCX) (2/23 vessels, 8.7%), and the left main trunk (LMT) (1/23 vessels, 4.3%).

No.	Age	Sex	Diag	Related Vessel	No. of Vessels	Preg	CSA	PA	MS	Treat	Procedure	Stent type	Final TIMI	TLR	Heal	Rec	Death	HT	DL	DM	Smoking	FH	Marfan syndrome
1	45	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	DES	2	0	+	0	0	0	0	0	0	0	0
2	52	F	STEMI	LAD	1	0	0	0	0	PCI	Stent	DES	3	0	+	0	0	+	0	0	0	0	0
3	53	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	BMS	3	0	+	0	0	0	+	0	0	0	0
4	45	F	STEMI	LMT, RCA	2	+	0	0	0	PCI	Balloon	-	3	-	-	+	0	+	0	0	+	0	0
5	55	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	DEA	3	0	+	0	0	0	+	0	0	0	0
6	47	F	NSTEMI	LAD, LCX	2	0	0	0	0	PCI	Stent	DES, BMS	3	0	+	0	0	0	0	0	0	0	0
7	51	F	STEMI	LAD	1	0	0	0	0	Med	-	-	3	-	-	0	0	0	0	0	0	0	0
8	54	F	STEMI	LAD	1	0	0	0	0	PCI	Wiring	-	3	-	-	0	0	0	0	0	0	+	0
9	50	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	DES	3	0	+	0	0	0	0	0	0	0	0
10	57	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	BMS	3	-	-	0	+	+	0	0	0	0	0
11	37	F	STEMI	LAD	1	0	+	0	0	PCI	Thrombectomy	-	3	0	+	0	0	0	0	0	+	+	0
12	74	F	STEMI	LAD, RCA	2	0	0	0	0	PCI	Stent	BMS	3	0	+	0	0	0	0	0	0	0	0
13	54	F	STEMI	RCA	1	0	+	0	0	PCI	Thrombectomy	-	3	0	+	0	0	+	0	0	0	+	0
14	47	F	STEMI	LCX	1	0	0	0	0	PCI	Stent	BMS	3	0	+	0	0	0	0	0	+	0	0
15	12	F	STEMI	LAD	1	0	0	0	0	PCI	Balloon	-	3	0	0	0	0	0	0	0	0	0	+
16	61	F	STEMI	RCA	1	0	0	0	0	PCI	Stent	BMS	3	+	+	0	0	+	0	0	+	0	0
17	43	F	STEMI	LAD	1	+	0	0	0	PCI	Balloon	-	3	+	0	0	0	0	0	0	0	0	0
18	54	M	STEMI	RCA	1	0	0	0	0	PCI	Thrombectomy	-	3	0	+	0	0	0	+	0	+	0	0

19	36	F	UAP	RCA	1	0	0	0	0	Med	-	-	3	0	+	0	0	0	0	0	0	0
20	45	F	STEMI	RCA	1	+	0	0	0	PCI	Stent	BMS	3	0	+	0	0	0	0	0	0	0

Table1: Patient characteristics, triggers, treatment, outcomes, coronary risk factors and related disease (BMS; bare metal stent, CSA: coronary spastic angina, DES; drug eluting stent, Diag; diagnosis, DL; dyslipidemia, DM; diabetes mellitus, FH; familial history, Heal.; healing, HT; hypertension, LAD; left anterior descending coronary artery, LCX; left circumflex coronary artery, LMT; left main trunk, Med; medication, MS; mental stress, NSTEMI; non ST elevation myocardial infarction, PA: vigorous physical activity, PCI; percutaneous coronary intervention, Preg; pregnancy, RCA; right coronary artery, Rec; recurrence, STEMI; ST elevation myocardial infarction, TIMI; thrombolysis in myocardial infarction grade, TLR; target lesion revascularization, Treat; treatment, UAP; unstable angina pectoris).

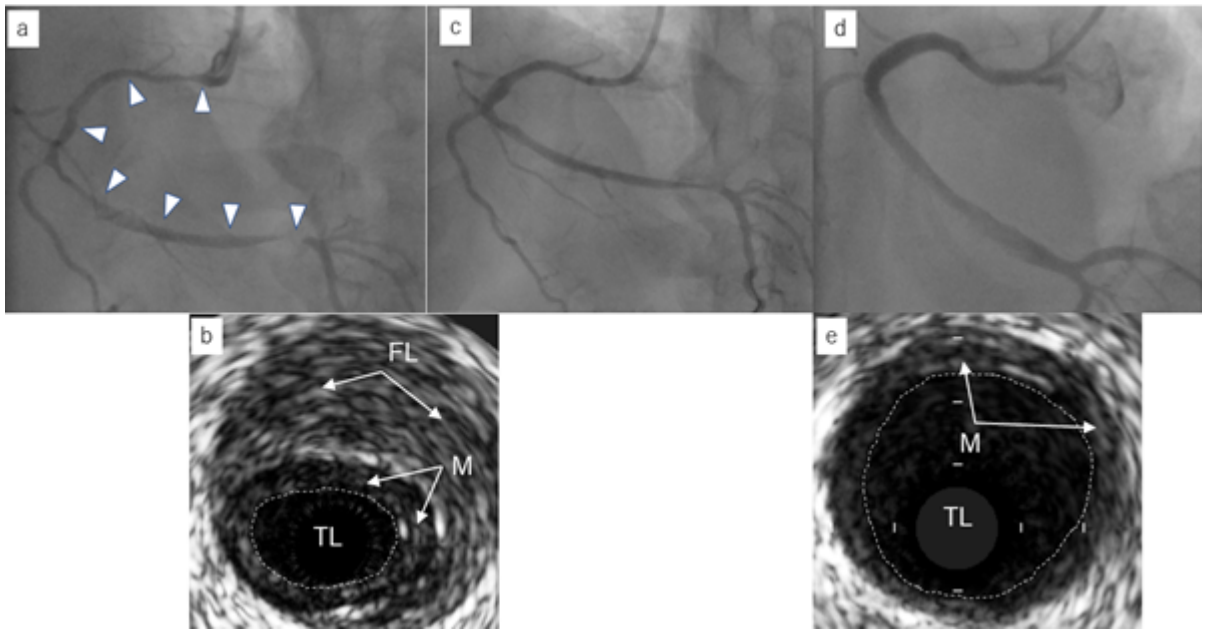

Figure 1: Case of a favorable clinical course (a) The initial CAG showed a long dissection of the right coronary artery (white arrows), (b) IVUS image shows the false lumen with intramural hemorrhage (white arrows), true lumen, and media (white arrows) at the initial CAG, (c) CAG after thrombectomy shows TIMI 3 coronary flow, (d)&(e) Follow-up CAG and IVUS after 3 months show the healing of the true lumen and media (white arrows). CAG; Coronary Angiography, FL; False Lumen, IVUS; Intravascular Ultrasound, M; Media, TIMI; Thrombolysis In Myocardial Infarction Grade, TL; True Lumen).

Regarding the trigger of onset, SCAD was associated with pregnancy in 3 patients; one was 38 weeks pregnant (No. 17), and two were 2 months and 10 years postpartum (No. 19 and 4, respectively). Coronary spasm was associated with SCAD in 2 patients (No. 11 and 13), and no patient had SCAD induced by vigorous physical activity or mental stress.

Two patients (10%) were treated conservatively, and PCI was performed in 18 patients (90%) to treat 21 lesions. In the patients treated with PCI, wire passage only, thrombectomy alone, balloon dilation alone, and stent implantation were performed in 1 (4.8%), 3 (14.3%), 4 (19.0%), and 13 (61.9%) of the 21 lesions, respectively. In the 13 lesions that were treated with stents, a bare-metal stent (BMS) was implanted in 8 lesions (61.5%), and a drug-eluting stent (DES) was implanted in 5 lesions (38.5%). TIMI 3 flow was achieved in 19 patients (95%), and TIMI 2 flow was achieved in 1 patient (No. 1).

Follow-up CAG was performed in 16 patients, and the healing of SCAD was shown in 14 patients (87.5%). Two patients (10.0%) had target lesion revascularization due to stent thrombosis (No. 16) or an extension of the dissection area (No. 17). None of the patients had restenosis due to intimal proliferation. In one case with Marfan syndrome (No. 15), the LAD was totally occluded after balloon dilation alone; however, conservative treatment was continued because there was good collateral flow (Rentrop grade III) (see case presentations). There was recurrence of SCAD in one patient (No. 4) (5%), and in-hospital death in one patient (No. 10) (5%) (See case presentations).

The prevalence of coronary risk factors was determined including hypertension, diabetes, smoking, a familial history of coronary artery disease and dyslipidemia (low-density lipoprotein cholesterol >130 mg/dl, high-density lipoprotein cholesterol <40 mg/dl, and/or triglyceride >150 mg/dl). Hypertension was present in 5 patients (25%), smoking in 5 (25%), a familial history of coronary artery disease in 3 (15%), dyslipidemia in 3 (15%), and there was no patient

with diabetes. In addition, no patient had more than 3 risk factors, and 8 patients had no risk factors.

There was one patient (No. 15) (5.0%) who had Marfan syndrome, and the remaining 19 patients had no other vascular disease, such as fibromuscular dysplasia (FMD), Ehlers-Danlos syndrome or vasculitis of any kinds.

A favorable clinical course was shown in the case No. 13 (Figure 1). This case was a 54-year-old female, who had SACD that presented as STEMI with coronary spastic angina as a trigger. Initial CAG showed TIMI 2 coronary flow with an extensive coronary dissection from the ostium to distal portion of the RCA (Figure 1a). After successful wire passage, intravascular ultrasound (IVUS) showed narrowing of the true lumen due to compression by a massive intramural hematoma in the false lumen (Figure 1b). Thrombectomy using an aspiration catheter caused true lumen dilation and TIMI 3 coronary flow, and further dilation of the true lumen was shown 15 minutes later (Figure 1c). Therefore, the procedure was completed without stent implantation. Follow-up CAG and IVUS after 3 months showed complete healing of SCAD (Figure 1d and Figure1e).

The case No. 15 (Figure 2) was a 12-year-old female with Marfan syndrome who had SCAD that presented as STEMI in the hospital after an operation for scoliosis (Figures 2a-2d). The initial CAG showed total occlusion of the LAD (Figure 2a). After successful wire passage, IVUS showed narrowing of the true lumen due to compression by a massive intramural hematoma in the false lumen (Figure 2b), and the false lumen had a diameter of more than 3.0 mm. Dilation using a 2.0 mm balloon (Figure 2c) led to TIMI 3 flow (Figure 2d), and the coronary dissection extended to the LMT. After confirmation of TIMI 3 flow for more than 15 minutes, the procedure was terminated without stent implantation because of concern that the dissection would extend from the LMT to the aorta. Follow-up CAG after 3 months showed total occlusion; however, there was good collateral flow from the RCA (Rentrop grade III), and she had no symptoms of angina or heart failure. Therefore, conservative treatment was continued.

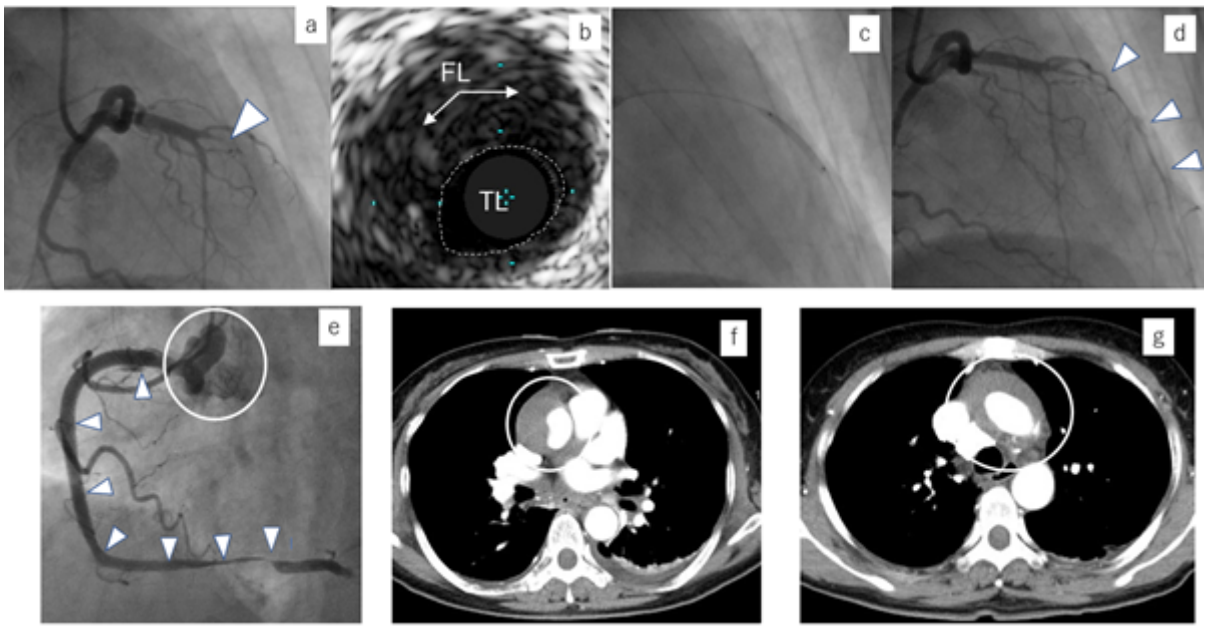

Figure 2: Cases of Marfan syndrome and in-hospital death (Marfaran syndrome; (a) The initial CAG shows the total occlusion of left anterior descending artery (white arrow); (b) The IVUS image at the initial CAG shows compression of the true lumen by hematoma within the false lumen (white arrows); (c) PCI using a 2.0-mm balloon; (d) CAG after PCI shows TIMI 3 flow (white arrows), In-hospital death; (e) CAG after stent implantation shows bidirectional extension of the dissection to the aorta (white circle) and right coronary artery distal area (white arrows). (f) & (g) Computed tomography shows the dissection from the Valsalva sinus to around the arch (white circles). CAG; Coronary Angiography, FL; False Lumen, IVUS; Intravascular Ultrasound, PCI; Percutaneous Coronary Intervention, TIMI; Thrombolysis In Myocardial Infarction Grade, TL; True Lumen).

The case No. 10 (Figures 2e-2g) who resulted in an in-hospital death was a 57-year-old female who had SACD that presented as inferior STEMI. Initial CAG showed total occlusion with dissection from the ostium to the mid-portion of the RCA. After successful wire passage, two long BMSs were implanted to cover the dissection area. Nevertheless, the dissection extended bidirectional to the aorta and RCA distal area (Figure 2e). Computed tomography showed dissection from the Valsalva sinus to around the arch (Figures 2f and 2g). In addition, there was ST elevation in leads II, III, and AVF. Emergent ascending aorta replacement and CABG were performed. Although the aortic dissection and coronary flow were stabilized, multiple organ failure occurred due to infection, and this led to in-hospital death.

Discussion

The estimated incidence of SCAD in ACS patients was 0.1–1% in classical papers from 1998 to 2009 and increased to 1.7–4% in studies after 2016 [6,7]. This change seems to be due to better recognition of SCAD and the development of intravascular imaging modalities. At the same time, the background of patients with SCAD has been changing from a peripartum status to middle-aged women with low coronary risk [8,9].

In this study, the incidence of SCAD in ACS patients was 0.21%, which is lower than that in recent reports. The data in this study included cases from 2001, and some patients might have been misdiagnosed, or the incidence of SCAD was low in our study population. In our study, 17 patients (85%) were middle-aged females (36-61 years) with low coronary risk, and these findings are compatible with recent reports.

Several triggers and exacerbating factors have been suggested by many investigators [10-14]. In the present study, SCAD was associated with pregnancy in three patients and coronary spastic angina in two patients. The rate of both of these triggers was relatively higher in our series than in other modern series. In one patient with pregnancy-associated SCAD, the onset was ten years after pregnancy, and this might have been due to pregnancy-induced vessel fragility. In addition, since coronary spastic angina is more common in Japan than other countries, there might be a similar tendency in SCAD patients.

Connective tissue disorders including type IV Ehler-Danlos and Marfan syndromes can be a cause of SCAD [15]. It was reported that only a minority of patients with SCAD who underwent genetic evaluation met the criteria for a connective tissue disorder (3 of 116 patients, 5.1%) [16]. The present study included only one (5%) Marfan syndrome patient: a 12-year-old female whose clinical course demonstrated an extreme vulnerability of the coronary vessel wall.

An association between SCAD and FMD has frequently been found in studies that systematically screened patients for FMD [17,18]. Like SCAD, FMD is an elusive condition of unknown etiology. In the US FMD registry of 447 patients, pathological manifestations included transient ischemic attack or stroke in 19%, arterial dissection in 20%, and aneurysms in 17%. Only 1.9% of patients had a previous myocardial infarction [19]. Although FMD is frequently found in patients with SCAD, the reverse is not true. These findings suggest that SCAD might be the result of the progression of FMD.

No FMD was observed in 20 SCAD patients in the present study; however, it is possible that not all patients were screened carefully for FMD at each center.

Precise and early diagnosis is very important because management strategies significantly differ from those used in patients affected by atherosclerotic coronary artery disease. The first-line diagnostic tool in ACS patients is CAG. The angiographic hallmark of SCAD is a longitudinal filling defect generating a double coronary lumen. Dye staining indicates the presence of slow flow in the false lumen.

An intramural hematoma sometimes shows smooth lumen narrowing, abrupt changes in arterial caliber, and focal or diffuse lesions. Since these lesions sometimes mimic atherosclerotic lesions [7,15], it is important to keep in mind that CAG may overlook SCAD in middle-aged female ACS patients with low coronary risk. Multivessel involvement is not rare, with an estimated prevalence rate of 9-19% [9,20-23]. There were 3 patients (15%) with two-vessel SCAD in this study, and the vessels included were the LMT+RCA, LAD+LCX, and LAD+RCA.

IVUS and, more recently, optical coherence tomography provide high-resolution tomographic images of the arterial wall allowing an accurate diagnosis and management of patients with SCAD [24-26]. IVUS provides a picture of the vessel wall that includes the normal 3-layered appearance along the unaffected segments and separation of the true and false lumens. When PCI is performed for SCAD, especially when stent implantation is needed, IVUS can play an essential role by showing both the start and end of SCAD, because the stent should prevent extension of dissection. In addition, the depth of penetration of IVUS into the vessel wall is sufficient to provide a full image of the arterial wall, even in the presence of a large hematoma. Thus, IVUS can give information about the appropriate length and diameter of the stent that should be implanted in PCI.

The optimal management of patients suffering from SCAD including conservative or invasive therapy remains undetermined. The rarity, diversity and complexity of the disease including related conditions make it difficult to conduct a randomized trial or to establish a guideline. Thus, current recommendations for the treatment of SCAD are based on expert opinion or limited scientific evidence.

It has been reported that most arteries with SCAD heal spontaneously (70%–97%), and this process may take 3 months to complete. 7 Additionally, in PCI for SCAD, the success rate is lower and the serious complication rate is higher than in PCI for typical ACS that is due to atherosclerosis (see the section on PCI). Accordingly, considering all of this information, a 'watchful waiting' strategy has been proposed for stable patients with SCAD, and revascularization is indicated only in cases with ongoing or refractory ischemia [27,28]. In this study, conservative therapy was used in two patients (10%) whose outcomes were favorable with the healing of SCAD.

Revascularization of a vessel with SCAD may be highly complex. The presence of a disrupted and fragile arterial wall complicates the procedure due to catheter-induced iatrogenic dissection. Both wiring and stent deployment may be associated with adverse clinical events.

All these technical difficulties may lead to a lower PCI success rate (only 43–80% in recent series) [4,8,9,20-23]. Improving the outcomes of PCI in SCAD requires meticulous angiographic and instrumentation techniques, including the avoidance of deep catheter engagement, non-coaxial positioning of the catheter tip, catheter dampening, and forceful injection of contrast agent.

The PCI procedure should be performed in sequential steps that consist of wiring alone, thrombectomy with an aspiration catheter, vessel dilation with a small balloon, and stent implantation; then, at the point that stable coronary flow is maintained for more than 15 or 20 minutes, the procedure should be finished. In a situation like this where there may be diverse and serious clinical changes, a wealth of experience and extensive knowledge is needed.

A scoring balloon may also be useful to avoid stent implantation, since this balloon can create a re-entry site at the distal potion of a dissected coronary artery. The re-entry can reduce the intravascular pressure and may lead to early healing of the dissection [29-31].

Of note, physicians should not hesitate to implant stents when they are necessary. In such cases, a reasonable aim of revascularization is to avoid the long-term consequences of a full-metal jacket strategy in a coronary artery that will spontaneously heal at follow-up [32]. Another option proposed in patients with long and severe lesions is a multistep

approach that involves stenting the distal edge, then the proximal edge, and finally the middle segment [7]. This strategy also aims to prevent hematoma propagation, and it should be kept in mind that late stent malposition might be observed in the future due to hematoma resorption [33].

In this study, conservative therapy was used in two patients (10%). In the other 18 patients who underwent PCI, wiring alone was performed in one patient (5.6%), thrombectomy alone in 3 (16.7%), balloon dilation alone in 4 (22.2%), and stent implantation in 13 (72.2%). Of the 13 patients with stent implantation, a bare-metal stent (BMS) was implanted in 8 patients (61.5%) and a drug-eluting stent (DES) was implanted in 5 (38.5%).

The PCI strategies used in SCAD patients seem to cause less vessel wall injury than the strategies used in patients with ACS due to atherosclerosis, and this tendency might be based on a concern that the fragility of the vessel wall could induce a critical hemodynamic status.

In addition, DES use was less frequent in the present study compared with usual PCI, because the operators in this study might have understood that neo-intimal proliferation after PCI in SCAD patients is much less frequent than after PCI in patients with ACS due to atherosclerosis. In the 18 patients who underwent PCI in the present study, wiring was successful in all patients, and TIMI 3 and TIMI 2 flow were achieved in 17 patients (95%) patients and one patient, respectively.

These results are relatively excellent compared with the recent reports mentioned above [4,8,9,25-27]. The healing of SCAD was observed in 14 of the 16 patients (87.5%) who underwent follow-up CAG, and there was no restenosis due to neointimal proliferation. These findings suggest that conservative therapy should be recommended in patients without ongoing ischemia, and that a BMS might be better than a DES.

The development of SCAD can be attributed to both vessel fragility and triggers, and when the sum of these two factors rises to a certain point, SCAD might occur (Figure 3). For example, in SACD triggered by coronary spasm, the onset may depend on the triggers rather than vessel fragility; however, the opposite may be true in patients with connective tissue disorders, such as Marfan syndrome.

In this study, SCAD due to coronary spasm completely healed (first case presentation); however, in the patient with Marfan syndrome (second case presentation), the dissection site remained totally occluded. Therefore, SCAD mainly derived from vessel fragility should be treated with stent implantation in the early stage to prevent extension of the dissection.

Prakash et al. [34]reported iatrogenic coronary artery dissection in 12 of 348 SCAD patients (3.4%), and the incidence of dissection in PCI patients was significantly higher than that in CAG-only patients (14.3% *vs.* 2%; 6 of 42 *vs.* 6 of 300; p=0.001). This may have been due to the use of a more bulky and harder guiding catheter, and more aggressive movement of the guiding catheter around a coronary ostium compared with CAG only. Furthermore, coronary artery dissection could be caused by a mistake in the operation of the guide wire. In this study, one patient died in the hospital due to iatrogenic dissection that extended from the RCA to the aorta (third case presentation). In PCI for SCAD, further dissection seems to be due mainly to vessel fragility. Furthermore, the operation of the guiding catheter and guide wire should be performed very meticulously, and it should be kept in mind that a normal PCI procedure could possibly cause iatrogenic coronary artery or aortic dissection.

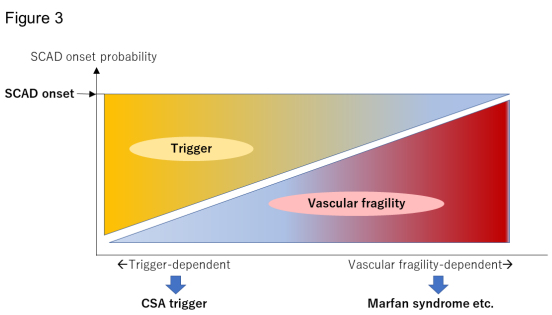

Figure 3: SCAD onset probability based on vascular fragility and triggers. (SCAD may occur at a certain point due to the summation of vascular fragility and triggers. CSA; coronary spastic angina, SCAD; spontaneous coronary artery dissection).

CABG will be needed in the patients with a high risk for PCI; however, CABG is also very challenging in patients with SCAD. The major pitfall of CABG is the risk of performing the anastomosis into the false lumen. Furthermore, the vessel wall may be too fragile due to the underlying condition, making the procedure impossible. Despite a successful CABG operation, the long-term patency of these coronary bypass grafts has been shown to be very low (4 of 15, 26.7%) bypass grafts were patent at late follow-up in the Mayo Clinic series) [4].The high rate of late bypass graft occlusion may be related to spontaneous healing of the native coronary artery resulting in normal anterograde coronary flow that competes with that provided by the bypass grafts. Although the outcomes of CABG in SCAD patients are not desirable, CABG should always be considered after PCI failure in patients with refractory symptoms and good distal vessels.

The in-hospital death rate of SCAD patients is globally low in recent series (0-2.2%), and studies with long-term follow up show a low mortality rate (0-3.1%, with up to 7 years of follow-up) [2,7,25-27]. On the other hand, the number of patients who suffer SCAD recurrence is high in some studies. Recurrence of SCAD has been observed in 1 of 4 patients during a median follow-up period of 2.3 years (12.2-27.0%) [28,35]. Notably, the predictors of worse prognosis in SCAD patients are peripartum period, larger infarcts, depressed left ventricular ejection fraction and multivessel involvement [36,37]. Close clinical follow-up of SACD patients should be advised. Life-style counseling should address the importance of avoiding apparent triggers, discontinuation of hormonal therapy, and a healthy life style. Recently, good outcomes have been reported with dedicated cardiac rehabilitation programs [38,39].

Conclusion

The patient demographics and outcomes in this study were compatible with recent reports of SCAD, except for the trigger of coronary spasm. The differential diagnosis of SCAD is essential, especially in middle-aged women with low coronary risk.

In the absence of ongoing ischemia or LMT dissection, SCAD should be treated conservatively. When PCI or CABG is needed, a

strategy that causes the least amount of vessel wall injury is desirable, considering that most dissections heal spontaneously and that iatrogenic dissection may frequently occur during an invasive procedure. In addition, SCAD should be closely followed-up, because the recurrence rate is not low.

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