Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS) that mostly occurs in middle-aged women. In recent years, it has been attracting attention as a major cause of ACS in younger women. Several case series, middle-scale sample size retrospective cohort studies, and a recent large prospective study have all identified many features of this disorder along with gaps in the literature. Almost all patients with SCAD present ACS and account for 1−4% of all ACS cases. In case of hemodynamically stable patients with thrombolysis in myocardial infarction flow grade 2 to 3, conservative treatment is recommended because healing of the dissected vessel is commonly expected. However, revascularization strategies for patients with SCAD are poorly established. Intracoronary imaging modalities such as intravascular ultrasound and optical coherence tomography have a potential for preventing technical failure during angioplasty and improving the success rate of revascularization, but it has not yet fully elucidated. From existing retrospective studies, long-term mortality is low even though the recurrence of SCAD is not rare. This review will summarize the clinical characteristics, management, and prognosis of SCAD.

KEY WORDS: acute coronary syndrome, intracoronary imaging, intra-mural hematoma, non-atherosclerotic coronary artery disease, spontaneous coronary artery dissection

I. Introduction

Spontaneous coronary artery dissection (SCAD) is a non-atherosclerotic cause of acute coronary syndrome (ACS) due to flow obstruction secondary to the spontaneous dissection of the epicardial coronary trees and mostly occurs in middle-aged women. It is thought to be either underdiagnosed driven by the low suspicion of ACS in relatively young women or misdiagnosed as atherosclerotic coronary artery disease because of the lack of recognition and clinical experience of this disease. Recent advancements in medical technology allow us to recognize and understand the features of SCAD. This review will summarize the clinical characteristics, management, and prognosis of SCAD.

II. Epidemiology

In recent times, the understanding of SCAD epidemiology has improved. Although the true prevalence of SCAD remains unclear, up to 1−4% of ACS cases have been reported to be caused by SCAD. There is a clear gender difference in this disorder as it occurs predominantly in women (90%). In studies limited to women aged 50 or younger, 24−35% of acute myocardial infarction (AMI) cases were caused by SCAD. Classically, SCAD was recognized as peripartum-related ACS, but contemporary reports revealed that peripartum status as a cause of SCAD accounted for a minority of the cases (<5%). In contrast, 21−27% of AMI cases during pregnancy and 50% of post-partum coronary events were reportedly due to SCAD. A Canadian study reported that peripartum SCAD was an independent predictor of a 30-day major adverse cardiovascular event (MACE). The clinical characteristics of male SCAD patients are poorly documented, however, based on few clinical experiences, SCAD should be suspected in younger male ACS patients with low coronary risk factors. The racial difference is also unclear; however, this difference may not be significant. From the cohort of the Canadian study, the incidence of SCAD in Caucasians, Asians, and Africans was almost the same as that in the Canadian population.

III. Pathology and pathophysiology

There are two presumed mechanisms: in the first, primary intimal tear causes coronary blood flow into the internal elastic lamina; in the second, primary disruption of a vasa vasorum micro-vessel leads to hemorrhage directly into the tunica media resulting in intramural hematoma (IMH). Either the dissect-
ed flap or the IMH causes flow disturbance against the true lumen and subsequent myocardial ischemia. Classically, the former mechanism was thought to be the only cause of SCAD, however, optical coherence tomography (OCT) studies have proved the absence of intimal tear in some SCAD patients. It is unknown whether the intimal tear is a primary cause of SCAD or secondary result from the IMH, or if these two types of SCAD may be different disease entities. Saw et al. defined IMH by angiographic classification of “Type 2” that presented diffuse and smooth narrowing, which was the most dominant (60–67%) of angiographically diagnosed SCAD. Histologically, fibrin-rich hematoma is present in the false lumen with infiltration of various inflammatory cells, often with a predominance of eosinophils.

Emotional or physical stress such as unusually intense exercise and the Valsalva-type maneuver may be associated with the development of SCAD in some patients. Emotional stressors have more often been reported as precipitants of SCAD in women than in men, whereas physical stressors have more often been reported among men with SCAD. Thus, there might be a group of misdiagnosed patients (2.5%) among those with previously diagnosed takotsubo cardiomyopathy, often preceded by emotional or physical stressors. Severe vasospasm is thought to be another trigger for SCAD that involves strong shear stress to the intima and media. Even though there are few supporting case series, this mechanism seems to be important because calcium channel blockers may be considered as a secondary prevention for such patients.

Several arteriopathies including fibromuscular dysplasia (FMD), connective tissue disorder, and systemic inflammatory disease are thought to be predisposing conditions of SCAD. Especially, a considerable number of papers have demonstrated the comorbidity of SCAD with FMD, but there is a large variation in prevalence (11–86%). The problem is the difficulty in diagnosing FMD, as this relies only on visual abnormalities of the arterial wall assessed by imaging modalities such as angiography, or ultrasonography. The overall prevalence of FMD is unknown because FMD is an asymptomatic condition.

Whether pregnancy-associated SCAD should be treated separately remains controversial because SCAD mostly affects the female population, and the rate of pregnancy-associated SCAD is low, as mentioned in the epidemiology section. Importantly, pregnancy-associated SCAD tends to present as a more severe clinical condition when compared to non-pregnancy-associated SCAD, with more cases being a proximal artery dissection. The presumed mechanism is that the estrogen and progesterone receptors present in the coronary arteries may mediate changes of the arterial wall.

IV. Clinical presentation

SCAD usually presents as ACS with or without ST-segment-elevation that depends on the degree of flow disturbance within the true lumen. It is not clear whether coronary dissection alone leads to chest pain. This rare but fatal disease can be easily overlooked if not suspected. The reason is that most of the patients with SCAD are relatively young females with a low risk for ischemic heart disease. Moreover, women often present with atypical chest pain and angina-equivalent symptoms such as dyspnea, weakness, palpitation, fatigue and indigestion. Therefore, delayed diagnosis is not rare for a patient with SCAD compared to an atherosclerotic ACS patient. Diagnostic ability may be improved with increased recognition of this disease.

Anatomically, all coronary trees including the left main trunk can be affected. There are several case series describing simultaneous multi-vessel SCAD, and one case presenting with a tandem IMH. The distal left ascending coronary artery and obtuse marginal branch are the most common segments affected by SCAD.

V. Diagnosis

SCAD is thought to be underdiagnosed. The key examination for diagnosing SCAD is coronary angiography because it usually manifests as ACS. Diagnosis is straightforward when typical angiographic patterns of SCAD are seen in patients with a typical background: young or middle-aged women, with a low risk for atherosclerotic disease. Some cases, however, require intracoronary imaging modalities such as high-resolution intravascular ultrasound (IVUS) or OCT for distinguishing SCAD with another cause of ACS and identifying the dissected flap or the IMH.

The angiographic appearances of SCAD depend on the degree of preserved flow in the true lumen and the presence of intimal disruption. In consideration of these points, Saw et al. have characterized three distinct angiographic appearances and patterns of SCAD. Type 1 shows contrast dye staining of the arterial wall with multiple radiolucent lumens that suggests the presence of intimal disruption. Type 2 shows diffuse and smooth narrowing of varying severity that suggests an IMH. Type 3 mimics atherosclerotic stenosis that is challenging to differentiate without intracoronary imaging. Interventional cardiologist should keep in mind that the non-Type 1, which shows no dissected flap angiographically, is the most dominant pattern of SCAD (71%). Representative cases of Type 1 and 2 patterns are shown in Fig. 1 and 2, respectively.

Both IVUS and OCT can provide complementary details for diagnosing SCAD. IVUS is advantageous as an initial modality in the following two respects. First, its deeper tissue penetration...
(4–8 mm) than that of compared to OCT (1–2 mm) enables visualization of the entire vessel aspect even when there is a large amount of stagnated blood in the pseudo-lumen that is hard for OCT to assess sufficiently. Another advantage of IVUS is that it is not necessary to remove red blood cells with the injection of contrast media, which is an essential step for OCT but has a potential risk of expanding coronary dissection. In contrast, OCT provides us with incomparably clear images of vessel structures that are helpful for distinguishing SCAD from other causes of ACS. The representative case of intracoronary imaging during the angioplasty is shown in Fig. 3.

There are some limitations in diagnosing SCAD. Imaging de-
vices such as high-resolution IVUS and OCT are not always available in the catheter laboratories of most countries. Thus, the rate of using IVUS/OCT to diagnose SCAD was very low in several cohort studies (7.6–13%)\(^9,35\), while about 90% of SCAD cases were diagnosed only by angiography; this holds a potential risk for overdiagnosis. In fact, the using rate of IVUS/OCT in those international cohort studies is lower than the reported percentage of Type 3 SCAD which imaging devices are essential for diagnosis. In contrast, based on several Japanese studies in which imaging devices are routinely used\(^3,4,36\), the distribution of initial thrombolysis in myocardial infarction (TIMI) flow grade is quite different from that reported in studies from the other countries: the initial TIMI flow grade 0–1 is higher in Japan (56–58%)\(^3,4\) than in the other international cohorts (27–32%)\(^9,35\), which suggests the potential underdiagnosis of non-Type 1 SCAD with TIMI flow grade 2–3. Fig. 4 shows two cases of ACS that were difficult to distinguish as SCAD and takotsubo cardiomyopathy without intra-coronary imaging. Moreover, most cases of SCAD involve the distal segment of the coronary trees, which makes it more difficult to assess an IMH by IVUS/OCT (Fig. 5, 6). Repeat angiography may be helpful for the diagnosis of SCAD in small vessels\(^\text{17}\) (Fig. 5).

VI. Management
The management of ACS with SCAD is different from atherosclerotic ACS. Almost all cases of atherosclerotic ACS have an indication for primary percutaneous coronary intervention (PCI), whereas this is not the case for all SCAD cases. Tweet et al.\(^35\) promoted the algorithm for acute management of SCAD classified by the initial TIMI flow grade. Conservative management is preferred for a TIMI flow grade 2 to 3 because PCI for SCAD was associated with a high rate of technical failure, while a good prognosis was seen for those treated with conservative management. Moreover, when there is preserved flow in the true lumen and no ongoing ischemia, “healing” of the dissected vessel is commonly expected (Fig. 6). Therefore, indication of PCI including stenting should be prudent especially in young female patients to avoid unnecessary long-term antplatelet therapy. The goal of PCI for SCAD is a TIMI grade 2 to 3, not defined by the acute gain nor the residual diameter stenosis. The success rate of PCI varies depending on the report (70–90%)\(^3,4,9,35,38\). However, the success rate may be affected by the use of IVUS/OCT, as the rate is higher in Japan where IVUS/OCT is routinely used (Table 1). Imaging guided PCI is reasonable for SCAD cases because
Fig. 4  Two cases of acute coronary syndrome (ACS) that are difficult to distinguish as spontaneous coronary artery dissection (SCAD) and takotsubo cardiomyopathy (TCM) without intra-coronary imaging. Upper panels show a TCM case with an angiographic Type 2 SCAD-like coronary artery. Angiogram (B) shows smooth narrowing of the distal left descending artery (LAD) and the left ventriculogram (LVG) (A, arrows) shows apical ballooning motion. Intravascular ultrasound images (C, D) show no dissected flap nor intramural hematoma (IMH) at the distal LAD. Lower panels show a SCAD case with atypical TCM-like LVG (E, arrows). IVUS images (G, H) show an IMH at the posterior descending artery despite a normal angiographic appearance (F). Abbreviations are as Fig. 1.

Fig. 5  A patient who has a previous history of spontaneous coronary artery dissection (SCAD), presented chest pain possibly due to recurrence of SCAD in distal small vessel. Angiogram (A) reveals SCAD-like narrowing (arrow) in the distal right coronary artery that is hard to confirm by the angiogram alone; however, insertion of intra-coronary imaging tools in such small vessels has a potential risk of worsening the coronary flow. In this case, we can refer to the previous angiogram (B), taken 5 years prior, suggesting SCAD in the distal right coronary artery.
ballooning or stenting in the pseudo-lumen worsens the flow of the true lumen. Furthermore, IVUS/OCT is a suitable modality for confirming the location of the guide wire in the true lumen before angioplasty. Also, IVUS/OCT clarifies the SCAD lesion despite this being a limitation of angiography. Interestingly, one recent report from the United States disclosed the high success rate of revascularization for ST-segment elevated SCAD (91%), but the percentage of IVUS/OCT use was not mentioned. A detailed analysis identifying the negative factors contributing to unsuccessful PCI has not been reported. Additionally, while several case series support the efficacy for IVUS/OCT use in PCI for SCAD, it has not been fully investigated.

Several revascularization techniques with PCI for SCAD have been identified in case reports. To date, there have been no randomized studies investigating this technique and no recommended strategies. However, based on theoretical and clinical experience, adjusted sized balloon angioplasty and adjusted length direct stenting, which are usually chosen for atherosclerotic lesions, should be avoided for IMH (non-Type 1), as longitudinal extension of the IMH, both proximally and distally, is highly expected. To avoid such complications, several revascularization techniques are recommended, including: 1) using a cutting bal-

Table 1  Acute percutaneous coronary intervention in spontaneous coronary dissection

| Reference | Nation     | Year | Patients (n) | Age       | Female (%) | PCI (%) | Successful PCI (%) |
|-----------|------------|------|--------------|-----------|------------|---------|-------------------|
| Saw et al. | North America | 2019 | 750          | 52 ± 10   | 89         | 14      | 70                |
| Lobo et al. | America | 2019 | 53           | 49 ± 10   | 93         | 62      | 91                |
| Nishiguchi et al. | Japan | 2017 | 12           | 63 ± 12   | 58         | 100     | 92                |
| Nakashima et al. | Japan | 2016 | 63           | 46 ± 10   | 94         | 56      | 91                |
| Lettieri et al. | Italy | 2015 | 134          | 52 ± 11   | 81         | 38      | 73                |
| Tweet et al. | America | 2014 | 189          | 44 ± 9    | 92         | 46      | 70                |
| Saw et al. | Canada | 2014 | 168          | 52 ± 9    | 92.3       | 33      | 63                |

PCI: percutaneous coronary intervention
loon to fenestrate the intima-media membrane and decompress the IMH as a stand-alone strategy or prior to stenting\(^{[4,35-41]}\), 2) targeting an intimal tear or “flap” for focal stenting or stenting just the proximal extent of the dissection to prevent proximal propagation and to enlarge the true lumen\(^{[42-43]}\), 3) extend stent lengths to reduce the chances of proximal or distal hematoma propagation, 4) minimal size balloon angioplasty to restore flow followed by a conservative strategy\(^{[44]}\), and 5) use of biodegradable coronary scaffolds\(^{[45]}\). Coronary artery bypass grafting (CABG) is considered in patients with left main trunk and proximal dissection when ischemia is refractory despite an attempted conservative approach. Data on CABG are limited in case reports, small case series, and retrospective observational studies with small sample sizes.

Unfortunately, there is no evidence-based medical management for both the acute and chronic phases of SCAD. Only \(\beta\)-blockers appear to be protective against recurrent SCAD\(^{[46]}\).

VII. Prognosis

The long-term mortality of SCAD is low (0–8%), but the recurrence of SCAD is not rare. The reported rate of recurrent SCAD varies in the literature (10–30%), likely due to differences in study design, duration of follow-up, definition of recurrence, and the limitations of diagnostic accuracy as mentioned in the previous section\(^{[2, 5, 9, 47, 48]}\). That is, the recurrence rate varies based on whether studies include early phase (<30 days) recurrence, whether they include only de novo dissection unrelated to the index dissected coronary tree, or if they include extension of the index dissection resulting from expansion of the unhealed SCAD lesion in the acute phase. When limited to subsequent de novo dissection, incident recurrent SCAD rates are more consistent (11–17%)\(^{[2, 25, 49]}\).

VIII. Conclusion

SCAD is a major cause of ACS in middle-aged or younger women and is diagnosed by coronary angiography. Existing literature has identified an association between SCAD and FMD. Improvements are necessary in the diagnostic accuracy, revascularization techniques, and medical therapy for secondary prevention, though the long-term mortality rate is low. We recommend that middle-aged or younger women with ACS are carefully examined via angiography, and that intracoronary imaging if suspected. The role of intracoronary imaging in the diagnosis and revascularization of SCAD requires further research.

Disclosure statement

None declared.

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