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REVIEW

Angiographic patterns in spontaneous coronary artery dissection: novel diagnostic insights

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ABSTRACT

Introduction: Spontaneous coronary artery dissection (SCAD) is a rare but well-recognized cause of acute coronary syndrome, especially important in women. Invasive coronary angiography (ICA) is the fundamental diagnostic technique for the confirmation of SCAD. Knowing the angiographic patterns suggestive of SCAD is essential for the correct identification of patients with this entity.

Areas covered: In this narrative review, the main angiographic characteristics of SCAD lesions as detected by ICA are presented and discussed.

Expert opinion: In addition to the specific angiographic classification of SCAD, several authors have described complementary angiographic patterns suggestive of SCAD. Knowledge and correct identification of these angiographic patterns is essential for the correct diagnosis of patients with clinical suspicion of SCAD.

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1. Introduction

Spontaneous coronary artery dissection (SCAD) is a relatively rare cause of acute coronary syndrome (ACS). SCAD affects mostly women (90% of patients are female) and constitutes a major cause of ACS in women under the age of 50. Although its etiology is not entirely clear, in a majority of patients the initial phenomenon appears to be intraparietal bleeding leading to the development of an intramural hematoma (IMH). On the other hand, in some patients, the initial event may be the disruption of the intima and part of the media layers of the coronary artery wall, although this phenomenon may also be secondarily seen in patients with an IMH [1]. SCAD has been classically described in invasive coronary angiography (ICA) as a double arterial lumen image with contrast retention within the false lumen. Over the last 10 years, data from large series of SCAD patients have changed many of the paradigms classically associated with this entity, including the description of SCAD angiographic patterns that did not show this double arterial lumen (dissection) pattern. The publication in 2014 of the first specific angiographic classification of SCAD was an important starting point for abandoning the classical idea of the double angiographic lumen as the only synonym for SCAD on ICA [2]. The recognition and description of more subtle forms of SCAD visible in ICA, mostly corresponding to IMH, has been instrumental in increasing the diagnosis sensitivity and accuracy, as well as in advancing our understanding of this disorder. The parallel development of intracoronary imaging techniques (ICI) (both intravascular ultrasound and, especially, the near-histologic resolution of optical coherence tomography – OCT) has made it possible to reliably prove that these new angiographic patterns indeed correspond to IMH. Despite the undoubted role of ICI in the correct diagnosis of SCAD, ICA remains, along with a high index of clinical suspicion, the fundamental pillar and the most widely used diagnostic test for the identification of SCAD. In this narrative review by experts in the field, we aim to describe the ICA patterns of SCAD, including new patterns that complement the SCAD angiographic classification. A comprehensive and systematic description of these additional angiographic patterns should improve the identification of lesions suggestive of SCAD allowing an adequate management of these patients.

2. Coronary angiography in spontaneous coronary artery dissection

2.1. Classical angiographic classification of dissection

With the inception of percutaneous coronary intervention (PCI) with plain-balloon angioplasty, the need arose to classify the iatrogenic dissections produced by this therapy, as their complexity was directly associated with a higher risk of acute vessel occlusion. The National Heart, Lung, and Blood Institute (NHLBI) classification was then established [3]. This standardized grading system developed by the American College of Cardiology and the American Heart Association included six dissection types, from A through F, based on angiographic appearances. Since at that time, there was not yet awareness

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Article highlights

- Knowledge of the classical and other more subtle angiographic presentations of SCAD is required to ensure a correct diagnosis and management of this elusive clinical entity.
- The publication of the SCAD-specific angiographic classification has served to improve the recognition of angiographic patterns without the classical double-lumen.
- Intracoronary imaging techniques have been of major value in gaining further anatomic insights allowing an improved angiographic recognition and superior diagnostic accuracy of SCAD.
- Several additional angiographic findings suggestive of SCAD have been described, which are useful in identifying those lesions that are most likely to correspond to dissection/hematoma.
- A high degree of clinical suspicion together with a careful analysis of the coronary angiogram remains paramount for the correct diagnosis of patients with SCAD.

of IMH-related SCAD patterns, early works in SCAD relied on this classification for the angiographic analysis of these lesions. As non-double lumen IMH patterns were progressively described, the need arose to develop an alternative, specific classification for SCAD lesions.

2.2. Specific angiographic classification of SCAD

An important point in the recognition of the angiographic patterns of SCAD was the publication by *Saw et al*. of the first dedicated angiographic classification of SCAD in 2014 [2]. In this study, three main angiographic patterns associated with SCAD were defined (Figure 1). Type 1 lesions (pathognomonic of SCAD) are defined as single or multiple radiolucent lumens with contrast dye staining of the coronary artery wall. In the largest prospective SCAD cohort described to date, the type 1 pattern was found in less than one-third of cases (29%) [4]. Type 2 lesions are defined as smooth and diffuse (usually >20 mm) lumen narrowing that can vary in severity. An abrupt change in arterial caliber can often be observed, which usually indicates the onset of the IMH. Following this, the same investigators suggested dividing type 2 lesions into subtypes 2a and 2b, depending on whether the extension of the IMH reached the most distal segments of the vessel (type 2b) or it was limited to a coronary segment, subsequently recovering the normal caliber of the distal coronary artery (type 2a). Type 2 is the most frequent type of SCAD lesion, accounting for 60% of cases in the description of the 750 patients included in the Canadian registry [4]. Finally, type 3 lesions were described as more focal lumen narrowing (<20 mm), where a differential diagnosis against atherosclerotic etiology was essential and where the use of ICI techniques is of particular interest in confirming the diagnosis of SCAD. In the previously described registry, type 3 lesions occurred in only 10.8% of SCAD cases [4].

2.3. Type 4 SCAD lesion/complete vessel occlusion

Following the publication of the first classification by *Saw et al.* several authors proposed the inclusion of a fourth type of SCAD lesion (type 4), as it was understood that it was not represented in the previous classification [5]. Thus,

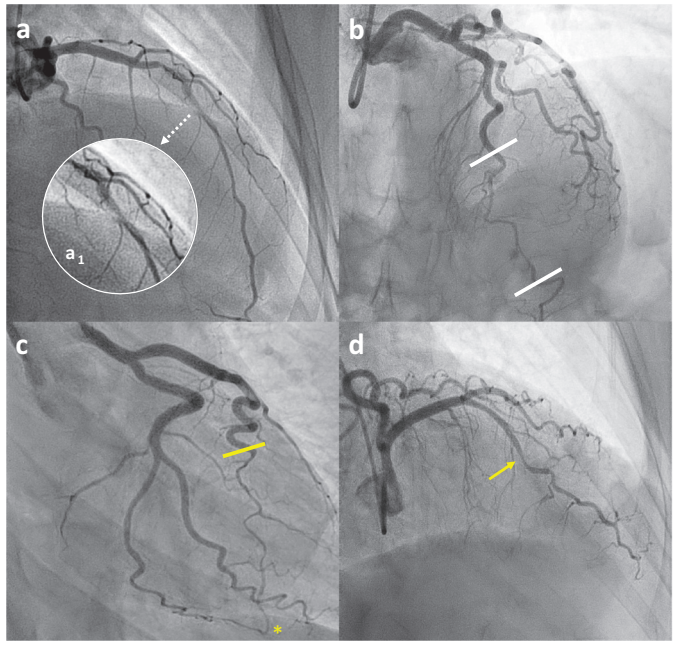

Figure 1. Specific angiographic classification of SCAD. (a) Shows a classic double arterial lumen pattern compatible with a *Saw et al.* type 1 pattern in the mid-segment of the left anterior descending coronary artery (LAD) (A1 shows detail of the lesion). (b) Shows a type 2a intramural hematoma (IMH) in the distal segment of the LAD, with recovery of the normal vessel caliber distal to the SCAD lesion. (c) Shows a type 2b IMH in the distal segment of the LAD, with the lesion reaching the most distal segment of the coronary artery (yellow asterisk). Please notice the proximal tortuosity of the healthy proximal LAD and the 'saw line' or 'broken-line' morphology of the distal affected segment of the vessel. (d) Shows the presence of a short type 3 IMH in the mid-segment of the LAD (yellow arrow), which is difficult to distinguish from a conventional atherosclerotic lesion without intracoronary imaging.

a type 4 SCAD lesion was defined as a complete occlusion of the coronary vessel (usually at the level of a distal segment or secondary branch), without initial angiographic signs of SCAD proximally to the point of occlusion, once the presence of coronary embolism has been excluded or a complete vessel recovery can be demonstrated at angiographic follow-up (Figure 2). In clinical practice, it is not uncommon to encounter complete vessel occlusion without initial signs suggestive of SCAD. Occasionally, it is only after the performance of PCI and opening the vessel that angiographic signs highly suggestive of IMH or dissection can be observed. Alternatively, the use of an ICI technique may confirm the diagnosis of SCAD in these occluded vessels. The prospective Spanish SCAD registry included 318 patients with SCAD, using this type 4 pattern as part of a predefined angiographic classification. In this registry, an occlusive type 4 angiographic pattern was described in 10% of cases [6]. Recently, *Mori et al*. analyzed those patients with type 4 lesions included in the DISCO registry [7]. In that work, those patients with a type 4 lesion had a low incidence of adverse events at long-term follow-up. In addition, patients with type 4 lesions were more frequently managed with PCI in the setting of ST-segment-elevation myocardial infarction (STEMI), left anterior descending (LAD) coronary artery, and/or proximal segment involvement. Recently, our group has also analyzed the characteristics and outcomes of SCAD patients presenting

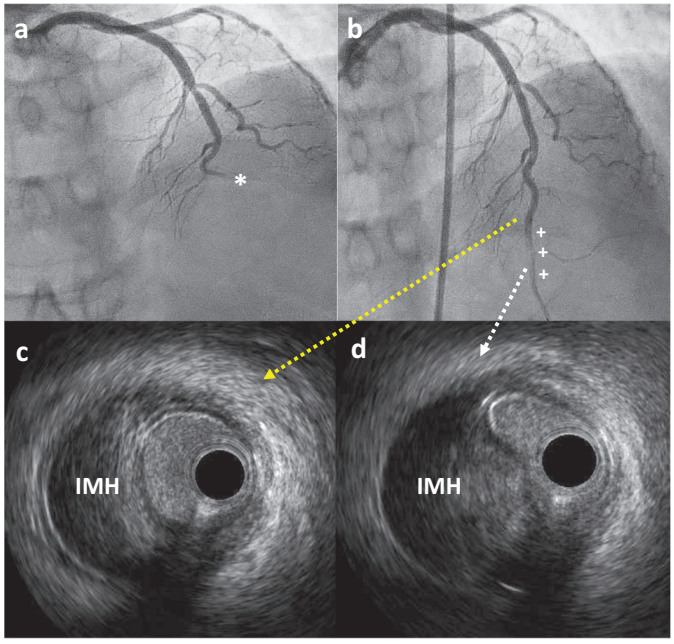

Figure 2. An example of a SCAD lesion with an initial angiographic pattern of complete occlusion (type 4) is shown. (a) Shows an area of complete occlusion in the distal segment of the LAD (white asterisk), with no obvious initial signs of SCAD. (b) After passing an intracoronary guidewire, an image of a double lumen and haziness compatible with SCAD is seen. (c and d) Intravascular ultrasound displays the presence of an intramural hematoma (IMH) (c – angiographically silent, d – causing significant lumen impingement), confirming the diagnosis of SCAD.

with an initial occlusion (Thrombolysis in Myocardial infarction – TIMI flow 0/1) versus those with flow-preserved lesions [8]. In that work, we found no differences in infarct size between groups, with TIMI 0/1 patients more frequently presenting as STEMI and requiring PCI, which generally presented good functional results and a low rate of procedure-related complications. Moreover, both in-hospital and late follow-up outcomes were similar in both groups. However, major caution is required to interpret a total vessel occlusion as SCAD, as this may be also seen, among others, in atherothrombosis or thrombus embolization. In the case of a lesion that initially presents as a complete occlusion, other additional diagnostic clues (such as evolutive changes, new findings after wiring or intervention, additional information with the use of ICI, etc.) should be considered before a final diagnosis is made.

2.4. Hybrid patterns

While the specific angiographic classification developed by *Saw et al.* helps to identify the most common patterns in SCAD, in actual clinical practice we often encounter SCAD lesions showing hybrid patterns. We can identify lesions in which a dissection (flap) zone is continued with areas of focal or diffuse lumen narrowing suggestive of adjacent IMH and even with complete vessel occlusion in the most distal part of the lesion [9]. We must be aware that the specific angiographic classification is a non-ordinal simplification, and that the relevance of these hybrid patterns, despite unclear, may not be discarded in clinical practice.

2.5. Stick insect and radish appearance

In 2017, *Motreff et al.* described in detail the angiographic findings of 55 patients with SCAD [10]. These French authors defined five common angiographic findings in patients with SCAD. Firstly, an absence of signs of atherosclerosis in other coronary arteries not affected by SCAD. Secondly and thirdly, angiographic characteristics that are superimposable to the type 1 lesions as described by *Saw et al.*, such as the presence of radiolucent flaps generating at least two lumens or contrast dye staining of the coronary artery wall. Fourthly, the onset or end of the IMH lesion coincident with the origin of a side branch. Finally, they described the presence of a long, smooth, and linear lumen narrowing, as well as two specific new angiographic appearances, the 'stick insect' and the 'radish' morphologies (Figure 3). The latter two patterns are due to extrinsic compression of the coronary lumen by the IMH, whose longitudinal extension is often interrupted by a side branch. Interestingly, the authors reported that at least three of these five signs were present in 92.7% of SCAD cases.

2.6. Broken line morphology

Some years ago, our group described for the first time the presence of a broken line (BKL) (zigzag or 'saw line' appearance) angiographic pattern as suggestive of SCAD [11,12]. The BKL pattern was defined as a long lesion (usually >20 mm in length) consisting of serial contiguous straightened or rectified segments in an otherwise smooth and tortuous coronary vessel (Figure 3). The striking contrast between the saw-line appearance and the smooth, rounded, tortuous adjacent segments was considered very characteristic. Recently, our group comprehensively analyzed the role of this specific angiographic pattern with data from the Spanish SCAD registry [13]. In that work, the BKL pattern appeared in 16% of SCAD lesions. Moreover, the presence of a BKL pattern was associated with a lower risk of major adverse cardiovascular events at follow-up, even after adjustment for potential confounders. Our hypothesis to explain these results is that the BKL pattern might represent the evolution of a stabilized long IMH, making its late further progression more unlikely. This would explain the better risk profile compared to lesions without this BKL pattern, although this should be interpreted with caution in the absence of confirmatory data from other cohorts.

2.7. Coronary tortuosity

Classically, the presence of significant coronary tortuosity has been associated with an increased risk of developing SCAD. Some authors relate the presence of coronary tortuosity as a marker of arteriopathy, including fibromuscular dysplasia (FMD) [14]. However, findings in this regard are controversial, as pathological studies do not seem to show any signs of FMD in the coronary arteries of patients with SCAD [15]. A previous study by the Mayo Clinic group analyzed the role of coronary tortuosity in patients with SCAD [16]. In this work, SCAD patients had more frequent coronary tortuosity compared to age- and sex-matched controls. In addition, severe coronary tortuosity (defined in this study as the presence of ≥ 2

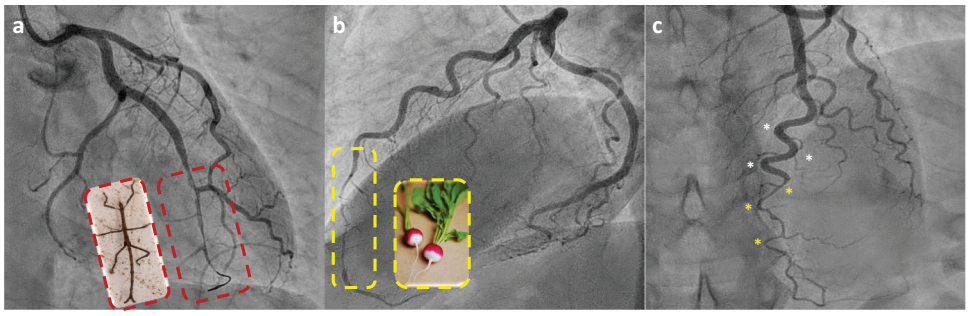

Figure 3. Additional angiographic patterns highly suggestive of SCAD. (a) Shows an intramural hematoma (IMH) in the distal segment of the circumflex artery, involving a segment with the onset of several side branches, generating a 'stick insect' angiographic pattern. (b) Shows the presence of a long IMH in the distal segment of the left anterior descending coronary artery (LAD), with an abrupt change in caliber both proximal and especially distal to the hematoma, generating the so-called 'radish' pattern. (c) Shows the presence of an IMH in the distal segment of the LAD. The IMH generates a change in the smooth normal bends of the coronary artery, as can be seen proximally (white asterisks), shifting to a diseased segment with a narrowed lumen and abrupt, rectified angles, generating a broken line pattern (yellow asterisks).

consecutive curvatures of ≥ 180° in a coronary artery ≥2 mm in diameter) was associated with an increased risk of SCAD recurrence and with the presence of extra coronary vascular abnormalities, including FMD. The 'corkscrew' sign (a ≥ 360° helical twist of a coronary artery), as an expression of severe tortuosity, may help in the identification of patients more prone to SCAD [16]. It is not clear whether marked tortuosity could be a sign of structural alteration of the coronary artery wall (making it more prone to SCAD), and/or that these areas of extreme tortuosity generate abnormal shear stress forces that eventually may lead to the occurrence of classical dissection or IMH.

2.8. Absence of coronary atherosclerosis

In the presence of a lesion suggestive of SCAD, the absence of angiographic atheromatosis (completely smooth coronary vessels) in the rest of the coronary arteries points to SCAD as the pathogenic origin of the culprit lesion. Despite the above, as in patients with Takotsubo syndrome, it is also possible to find mild atheromatosis in patients with SCAD. The current profile of patients affected by SCAD (as opposed to the classic profile before the large prospective series era), refers more frequently to middle-aged postmenopausal women in whom the presence of classic cardiovascular risk factors (excluding diabetes) is not so infrequent [4,6,17,18]. Because of that, the presence of atheromatosis in other vessels should not exclude SCAD from the differential diagnosis. However, the fact is that the presence of signs of coronary atherosclerosis is very rare among patients with a confirmed diagnosis of SCAD. In the Spanish SCAD registry, the presence of any degree of coronary atheromatosis was reported in only 5% of cases by the angiographic core lab [6,18]. Therefore, major care should be paid to prevent overdiagnosis of SCAD. In this regard, it is important to keep in mind that some atherosclerotic plaque ruptures and plaque ulcers with a cavity or even complicated plaques with associated intraluminal thrombus may mimic a double-lumen morphology. In cases with marked atheromatosis and a lesion suggestive of SCAD, the use of ICI allows the diagnosis of SCAD to be confirmed or excluded. Moreover, recent data from genetic studies also seem to show that some genetic pathways appear antagonistic between atherosclerotic coronary artery disease (CAD) and SCAD. In the study that described the first genetic variant associated with both SCAD and FMD, the complementary allele (rs9349379-G) to the SCAD-related allele (rs9349379-A), was associated with an increased risk of atherosclerotic CAD and myocardial infarction [19].

2.9. Left anterior descending (LAD) coronary artery preference

Since the beginning of modern series of patients with SCAD, it has been consistently shown that the most frequently affected coronary vessel in SCAD is the LAD. The reasons for this preferential involvement of the anterior interventricular artery are unknown. Both mechanisms related to the distinct composition of the coronary artery wall and/or to blood flow patterns in the different coronary arteries have been proposed. In the largest modern series, the percentage of lesions with LAD involvement is between 44–59% [4,6,17,18,20,21]. In those cases that affect the LAD, the differential diagnosis of Takotsubo syndrome is especially important. It has been shown that, occasionally, in the presence of a recurrent LAD artery that wraps around the left ventricular apex, SCAD can generate a pattern of regional left ventricular wall motion abnormalities that may mimic that of the classic Takotsubo syndrome. In this setting, a careful review of ICA becomes essential, as well as the use of ICI techniques in doubtful lesions, given that both the treatment and prognosis of these two entities are different in comparative studies [22–24]. Interestingly, recently, *Gulati et al.* proposed acute mechano-cardiac coronary artery disruption as a potential mechanism that would link Takotsubo syndrome with SCAD [25]. This theory could explain the preference of SCAD to affect the mid-distal segments of the LAD, and the occasional presence of multivessel SCAD involvement. Despite the above, this description remains purely speculative, providing an alternative etiopathogenic theory to the primary intima-medial rupture (*inside-out hypothesis*) and intraparietal bleeding (*outside-in hypothesis*). Interestingly enough, some patients with SCAD had relatively small myocardial infarctions and left ventricular angiograms closely resembling those of Takotsubo

patients. In other patients diagnosed as Takotsubo subtle minor angiographic irregularities resulting from an IMH may have been overlooked.

2.10. Distal coronary segment involvement

Large series of patients with SCAD have shown a clear predominance of cases affecting distal coronary segments or secondary branches, as opposed to proximal coronary vessel involvement. In the Spanish national registry of SCAD, lesions affected distal coronary segments in 38% of cases, with only 13% of lesions affecting proximal coronary segments [6]. In the Canadian prospective registry, only 8% of SCAD lesions involved the left main stem or proximal segments of the LAD or left circumflex artery [4]. The reason for a distal segment predominance in SCAD remains unknown. However, a selection bias should also be considered, since cases with proximal involvement, with a larger myocardial territory at risk, are likely to present more aggressively, with a greater number of patients presenting as sudden cardiac death before the diagnosis of SCAD can be confirmed.

2.11. Multivessel SCAD involvement

In the largest prospective cohorts published to date, the percentage of multivessel SCAD was 11–13% [4,6]. Multivessel SCAD involvement might suggest the presence of a generalized or even systemic process as a trigger for these episodes. A recent study showed that those patients with multivessel SCAD involvement presented with some distinctive clinical and angiographic features [26]. Patients with multivessel SCAD were more likely to have a prior history of hypothyroidism and anxiety disorder, presented mostly as non-ST segment elevation myocardial infarction, and less commonly displayed a type 1 angiographic pattern as compared with the single-vessel group. Moreover, a reduced initial TIMI flow of 0–1 was less frequently seen in multivessel SCAD. Although no significant differences were found in the incidence of composite adverse events at follow-up between groups, the incidence of stroke was markedly higher in patients with multivessel SCAD in this study.

2.12. Lack of significant intraluminal thrombus

In most SCAD lesions, the initial mechanism appears to be intraparietal bleeding with the formation of an IMH [27–29]. There is already evidence that, in some cases, there is also a generation of intimal-medial flaps that may expose components of the coronary artery wall to the bloodstream. Exposure to these intraparietal components could favor, as occurs in atherosclerotic plaque rupture, platelet activation, and the secondary formation of intraluminal thrombus. However, angiographic images highly suggestive of intracoronary thrombus are extremely rare in SCAD. An initial work by our group analyzed the role of OCT in 11 patients with SCAD [30]. In this study, thrombus was systematically found in the false lumen but only in a minority of cases in the true lumen. Clinical practice suggests that the amount of intraluminal thrombus in SCAD lesions is very low, and can only be visualized by OCT, with no clear angiographic signs of luminal thrombosis. A more recent study by *Jackson et al*. analyzed a larger cohort of SCAD patients with OCT [31]. In this study, only 15 of the 68 lesions (22%) showed true lumen thrombus by OCT. Again, the amount of thrombus in these lesions was very low and could not be assessed by ICA. Due to all of the above, in the presence of a lesion suggestive of SCAD, the concomitant presence of an image suggestive of a large thrombus burden by ICA should be a warning sign to look for an alternative diagnosis, such as plaque rupture/erosion or even coronary embolism.

On the other hand, a chronically recanalized thrombus of atherosclerotic origin may offer an angiographic image similar to the classic pattern of multiple lumens present in type 1 SCAD lesions. However, it has recently been described that some lesions suggestive of recanalized chronic thrombus by ICA may correspond to a late phase of the SCAD healing process. In these rare cases, complete healing of the affected segment does not occur and multiple lumens or images of fenestration (generating a lotus or honeycomb-like pattern) are detected by OCT [32].

2.13. Myocardial bridge

A myocardial bridge occurs when a coronary artery segment travels deep into the myocardial wall instead of following its usual route through the epicardial fat. Bridges may or may not be associated with the classical angiographic dynamic systolic compression phenomenon (*´milking´*). The presence of myocardial bridges has been previously related to the potential development of myocardial ischemia, coronary spasm, and sudden cardiac death [33]. Some case reports describe a close relationship between SCAD and the presence of an intramyocardial bridge with an angiographic *´milking´* phenomenon [34–36]. Furthermore, both entities affect predominantly mid-to-distal segments of the LAD. Although the cases described are anecdotal, it seems reasonable to support the idea that the systolic stress produced by myocardial bridging might be a mechanism promoting dissection in a weaker coronary artery wall susceptible to bleeding/wall rupture (Figure 4).

2.14. Angiographic evolution of the SCAD lesion – spontaneous vessel healing

The natural history in a vast majority of SCAD lesions is for complete spontaneous healing at follow-up. The study by *Hassan et al*. showed that in SCAD lesions with available late (≥30 days after the initial event) ICA surveillance, 95% of cases showed complete healing with restoration of a normal vessel appearance [37]. *Roura et al*. also showed a high percentage of complete healing (83%) by noninvasive coronary computed tomography angiography (CCTA), performed between 3 and 6 months after the index event, in patients with SCAD [38]. In addition, *Waterbury et al*. showed that the angiographic pattern of SCAD can vary depending on the time course of the lesion [1]. The study analyzed the presence of SCAD lesion progression in the first 14 days after the initial event, showing

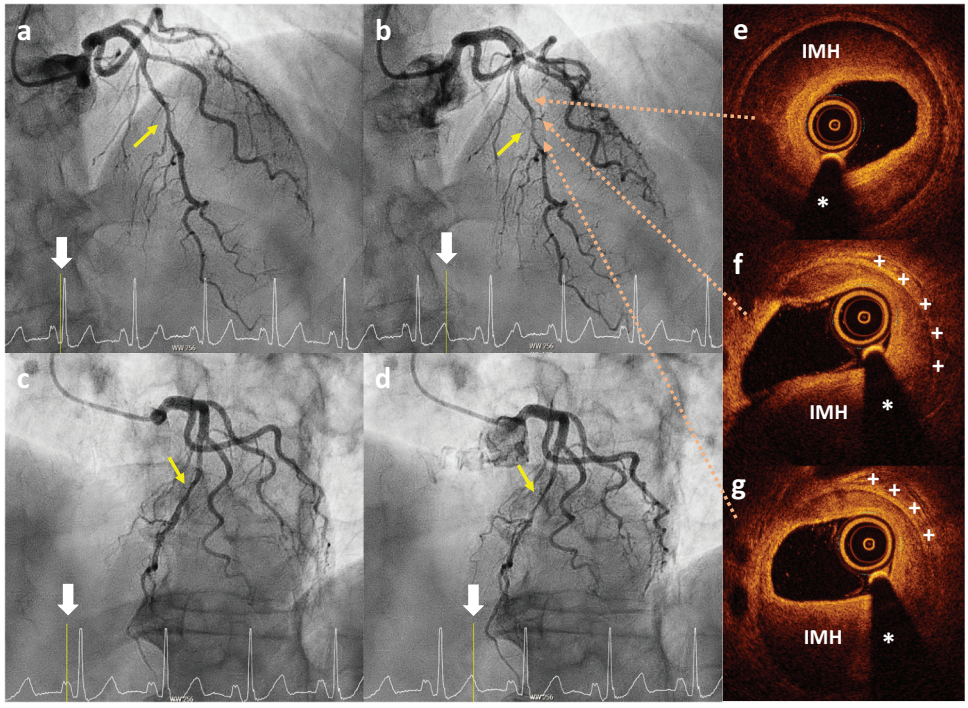

Figure 4. A 72-year-old woman was admitted for an episode of typical chest pain with raised levels of cardiac troponins. An invasive coronary angiogram (panels a-d) depicted the presence of a focal dynamic systolic-diastolic lumen narrowing (yellow arrows) at the mid-segment of the left anterior descending coronary artery (LAD). There was a significant change in lumen caliber at the point of the lesion between end-diastole (panels a and c) and end-systole (panels b and d) with '*milking*' phenomenon, suggesting the presence of a SCAD lesion over a zone of myocardial bridging. Optical coherence tomography (OCT) was performed (panels e-g) confirming the presence of a long intramural hematoma (IMH) and, at its distal part, the presence of a myocardial bridge (+). * denotes wire artifact. White arrows denote electrocardiogram to highlight end-systolic and end-diastolic phases.

that a percentage of lesions that initially showed as type 2 IMH on initial ICA evolved to a type 1 double lumen pattern at angiographic follow-up [1]. Furthermore, in this study, the presence of an IMH-type lesion was associated with an increased risk of SCAD extension.

The described tendency to spontaneous and complete vessel healing is useful in doubtful SCAD lesions. Evidence of lesion healing at follow-up, either invasively by ICA or noninvasively by CCTA, points toward a diagnosis of SCAD. However, we must bear in mind that lesions produced by coronary embolism also 'heal' on follow-up, once the emboli are dissolved. In these cases, it is extremely important to rule out the presence of specific risk factors for thromboembolism (such as valvular prostheses, atrial fibrillation, coagulation disorders, etc.).

2.15. Role of coronary computed tomography angiography (CCTA)

Although it is beyond the scope of this work to describe the imaging patterns found in CCTA in the context of SCAD, it is important to describe the usefulness and limitations of CCTA (versus ICA) in the diagnosis of this entity. The role of CCTA in SCAD is attractive given its noninvasive nature in a setting like SCAD where the coronary artery is already damaged and is also more prone to iatrogenic dissections. As explained above, several authors have described the usefulness of CCTA in the follow-up of patients with an established diagnosis of SCAD, allowing confirmation of complete arterial healing of the affected coronary segment [38]. However, data on the usefulness of CCTA versus ICA in the diagnosis of acute SCAD is scarce. We analyzed the role of CCTA versus ICA in 18 SCAD lesions [39]. In our work, CCTA identified lesions suggestive of SCAD in 78% of the cases, with the most frequent finding being a diffuse lumen narrowing accompanied by 'sleeve-like' wall thickening, which corresponded with areas of IMH on OCT. Similarly, *Tweet et al*. described CCTA findings in 14 patients with an acute episode of SCAD [40]. The most frequent patterns were abrupt and/or tapered luminal stenosis, as well as identification of zones of IMH or arterial dissection. The authors describe the similarity between SCAD lesions with noncalcified atherosclerotic plaques using this technique, which undoubtedly hinders the adoption of CCTA as the standard technique for the diagnosis of acute SCAD. Recently, several authors have suggested that the latest advances in CCTA may allow better identification and differential diagnosis of SCAD in the acute setting [41]. These authors also suggest that, in stable patients without high-risk features, an initial CCTA strategy might reduce the need for ICA in a significant proportion of patients, although this strategy has not been properly evaluated in clinical studies.

3. Conclusion

SCAD is an entity that sometimes presents a complex and elusive diagnosis. In other cases, with the characteristic clinical setting, a ready diagnosis becomes evident. Alternatively, in some other scenarios, a high index of clinical suspicion should

prompt to scrutinize the angiogram looking for subtle diagnostic clues. ICA remains the most important tool to establish a certain diagnosis of SCAD. Experience gained with ICI has permitted a better understanding of the meaning of some subtle features characteristic of IMH. ICI should be performed to guide PCI (only indicated in selected cases) and in cases where angiography remains ambiguous. It is important to keep in mind that conservative medical management is recommended as the initial strategy for most patients with SCAD. PCI should be only considered for selected SCAD patients presenting with STEMI and occluded arteries or flow-limiting lesions and persisting symptoms and those with high-risk anatomy (i.e. left main stem involvement). Accordingly, experience in the recognition of the different SCAD angiographic patterns is paramount to allow an adequate diagnosis and management.

4. Expert opinion

Spontaneous coronary artery dissection (SCAD) is a relatively rare but well-recognized cause of acute coronary syndrome. SCAD is of particular importance in young and middle-aged women, and in the context of pregnancy, where it accounts for a significant percentage of patients presenting with acute myocardial infarction. Although the pathophysiology of SCAD is not fully elucidated, two main hypotheses implicate either the presence of intraparietal bleeding or the development of an intima-medial rupture flap as promoting mechanisms of the SCAD event. Since its initial description in the 1930s, the diagnosis of SCAD has been based on the recognition of specific patterns in invasive coronary angiography (ICA). At first, the recognition of SCAD was based solely on the identification of angiographic lesions with multiple arterial lumens, similar to the images produced in iatrogenic dissections following plain old balloon angioplasty. Despite advances in our understanding of this fascinating clinical entity in the last 10–15 years, ICA remains the fundamental diagnostic tool in SCAD. The publication a decade ago of the angiographic-specific classification of SCAD was an important step in the recognition of the angiographic patterns related to intramural hematoma (IMH). This has undoubtedly served to spread the message about the importance of recognizing angiographic patterns related to IMH. In addition, data from modern large prospective cohorts of patients with SCAD show that lesions characterized by the presence of an IMH may be associated with a worse acute prognosis and an increased risk of late extension. The dissemination of this specific angiographic classification among operators worldwide has undoubtedly led to an increase in the diagnosis of this type of SCAD lesion and, therefore, in the number of patients with a final correct diagnosis of SCAD. However, since the publication of this classification in 2014, several authors have described additional angiographic features in patients with SCAD, which are also helpful in the diagnosis of potential SCAD lesions. In-depth knowledge and correct identification of these angiographic patterns can benefit a large number of additional patients. The latter can lead to relevant changes in lesion management and even patient prognosis in the long-term follow-up of SCAD patients. This paper includes a thorough review of the angiographic patterns described in SCAD so that the reader will be able to identify those angiographic features most clearly related to SCAD.

Technological evolution within the healthcare field has been a continuous process over the last decades. Thus, the progress of X-ray equipment has resulted in a significant improvement in image quality, associated with a reduction in radiation doses and contrast volume in ICA studies. Given that on many occasions, SCAD lesions are subtle in ICA, the improvement of X-ray equipment has been a fundamental point for the advances in the diagnosis of this entity. Looking to the near future, advances in the field of artificial intelligence are of particular interest to improve the angiographic diagnosis of SCAD. Thus, the development of algorithms based on artificial intelligence might soon constitute a relevant aid to improve the cost-effectiveness of ICA in the diagnosis of this elusive and challenging clinical entity.

Abbreviations

ACS	Acute coronary syndrome
BKL	Broken line angiographic pattern
CAD	Coronary artery disease
CCTA	Coronary computed tomography angiography
ICA	Invasive coronary angiography
ICI	Intracoronary imaging
IMH	Intramural hematoma
LAD	Left anterior descending coronary artery
SCAD	Spontaneous coronary artery dissection
OCT	Optical coherence tomography
PCI	Percutaneous coronary intervention
STEMI	ST-segment-elevation myocardial infarction
TIMI	Thrombolysis in Myocardial infarction

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