Spontaneous coronary artery dissection

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ACUTE CORONARY SYNDROMES

Spontaneous coronary artery dissection

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Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome or sudden cardiac death. Initial reports on this condition were scarce as they were based on postmortem examination of fatal cases. Currently, the clinical recognition of SCAD has increased as coronary angiography is utilised frequently in the clinical evaluation of patients with acute coronary syndromes. Moreover, intracoronary imaging techniques such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT), which provide detailed morphological information on coronary lesions and on the location of dissection planes between the different layers of the arterial wall, have enabled a more detailed clinical assessment of SCAD. Furthermore, non-invasive coronary angiography by multidetector computed tomography (MDCT) has been used for longitudinal follow-up evaluation of patients with SCAD.

The clinical presentation of SCAD depends on the extent and the flow limiting severity of the coronary dissection, and ranges from asymptomatic to unstable angina, acute myocardial infarction, and ventricular arrhythmias to sudden cardiac death. As several diseases and conditions have been associated with SCAD it therefore probably constitutes a heterogeneous entity.

The use of coronary stents represented a breakthrough in the management of iatrogenic coronary dissections occurring during percutaneous interventions. However, in SCAD their application should be balanced against the expected outcome after a spontaneous natural course of this condition, because coronary stenting may end up in a 'full metallic jacket' covering of a long segment of the coronary artery. Current insights into the aetiology, pathogenesis, diagnostic imaging and therapeutic management of SCAD are reviewed.

DEFINITION

Coronary artery dissection can be primary or secondary. Primary dissections occur spontaneously whereas secondary dissections occur as an extension from aortic root dissection or following an insult as a consequence of coronary angiography, coronary intervention, cardiac surgery or chest trauma.

Dissection of the coronary artery results in separation of the different layers of the arterial wall with the creation of a false lumen. The dissection plane can be situated between the intima and the media or between the media and the adventitia. Haemorrhage into the false lumen followed by thrombosis can compress the true lumen of the coronary artery resulting in a non-occlusive or occlusive obstruction of the blood flow causing myocardial ischaemia.

Angiographically coronary dissections can be graded according to the National Heart, Lung, and Blood Institute classification system developed by the Coronary Angioplasty Registry.[1] This system grades coronary dissections based on angiographic appearance as types A–F. Type A dissections represent radiolucent areas within the coronary lumen during contrast injection, with minimal or no persistence of contrast after the dye has cleared. Type B dissections are parallel tracts or double lumen separated by a radiolucent area during contrast injection, with minimal or no persistence after dye clearance. Type C dissections appear angiographically as contrast outside the coronary lumen, with persistence of contrast in the area after clearance of dye from the coronary lumen. Type D dissections represent spiral luminal filling defects, frequently with extensive contrast staining of the vessel. Type E dissections appear as new, persistent filling defects. Type F dissections represent those that lead to total occlusion of the coronary artery, without distal anterograde flow.

Incidence

The first case of SCAD was described in 1931.[2] More than 300 documented cases of SCAD have been published since then. Initial cases were based on autopsy findings after sudden cardiac death (table 1).[3] More recently the syndrome has become increasingly diagnosed, as coronary angiography is more often performed in patients suspected of having an acute coronary syndrome.[4] The incidence of SCAD in angiographic series varies widely—from 0.07% up to 1.1% for patients who are referred for coronary angiography.[5–11]

The mean age at presentation is 30–45 years (range 30–70 years).[4 10 11] More than 70% of SCAD cases are women, and in approximately 30% it occurs during the peripartum period.[3 4] Among women, the incidence of SCAD was highest in women below the age of 40 years and decreased significantly with advancing age (figure 1). When only women below 50 years of age presenting with an acute coronary syndrome were considered, the prevalence of SCAD increased up to 8.7%, and reached 10.8% in the case of ST elevation myocardial infarction.[10]

The left anterior descending artery is the most frequently involved vessel—in autopsy and

Table 1 Incidence and outcome of spontaneous coronary artery dissection

Studies	Study population	Number of cases	Incidence (%)	Initial treatment, N (%)			In-hospital	Mean follow-up	1 year event
				Medical	PCI	CABG	Deaths, N (%)	(months)	Free survival (%)
De Maio *et al*[3]	–	27	–	–	–	–	2 (7.0)	41	96
Pasalados Pita *et al*[5]	2241	5	0.22	3 (60)	0 (0)	2 (40)	0 (0.0)	18	100
Jorgensen *et al*[6]	9852	10	0.10	6 (60)	0 (0)	4 (40)	0 (0.0)	21	90
Zampieri *et al*[7]	2225	5	0.22	5 (100)	0 (0)	0 (0)	0 (0.0)	27	80
Hering *et al*[8]	3803	42	1.10	3 (7)	31 (14)	8 (19)	1 (2.0)	13.5	76
Celik *et al*[9]	3750	9	0.24	1 (11)	1 (11)	7 (11)	0 (0.0)	12	89
Valnetto *et al*[10]	11605	23	0.20	10 (44)	8 (34)	5 (22)	1 (4.0)	15.6	77
Mortensen *et al*[11]	32869	22	0.0007	7 (32)	13 (59)	2 (9)	0 (0.0)	34.8	100

CABG, coronary artery bypass surgery; PCI, percutaneous coronary intervention.

angiographic series the LAD accounts on average for 60% of the cases (range 38–77%).[3 4 6 8 10 11] Several cases of SCAD with simultaneous multiple vessel involvement have been reported. Multivessel coronary dissection was observed in about 20% of the cases and involvement of the left main coronary artery was present in 12% of the cases.[4] Spontaneous dissection of the left main stem can extend distally to involve multiple coronary branches.

PATHOGENESIS AND AETIOLOGY

SCAD remains an unclear aetiopathologic entity. The results of earlier histopathologic examinations performed during an autopsy for sudden cardiac death have recently been complemented by the findings of IVUS and OCT intracoronary examinations in patients who survived a SCAD.

SCAD results from vessel wall haematoma formation in the outer third of the media or between the media and the adventitia in the absence of traumatic or iatrogenic causes, resulting in a false lumen (figure 2). Expansion of this lumen through blood or clot accumulation leads to distal propagation of the dissection and to compression of the real lumen, causing myocardial ischaemia. An intimal tear is only seldom observed.

The most common pathologies associated with SCAD are coronary atherosclerosis and vascular changes occurring during the peripartum period. Other causes of SCAD (table 2) are connective tissue disorders, systemic lupus erythematosus vasculitis, cocaine abuse, vigorous exercise, and prolonged sneezing. However, a large number of cases must be classified as idiopathic because no underlying condition can be detected.

Atherosclerosis

Plaque inflammation leading to thinning and subsequent rupture of the fibrous cap covering the necrotic core is a key process in the pathogenesis of acute coronary syndromes. Plaque rupture may not only be complicated by intraluminal thrombosis but also by the development of deep subintimal dissection. As a result of the inflammatory process the junction between the intima and media is weakened and the media underneath an atherosclerotic plaque is thinned or sometimes even absent. After plaque rupture blood may therefore enter via the intimal tear, creating a medial dissection with intramural haematoma formation (figure 2).

In the initially published series of cases with SCAD the presence of coronary artery atherosclerosis varied from 8.5% to 28% of the patients.[3 4] However, it appears that mild atherosclerosis is more often the underlying cause than previously thought.[8 9 12 13] In a series of nine non-pregnant cases of SCAD who had no other coexistent disease that could cause dissection, atherosclerosis was observed on angiography in all cases both in the dissected and non-dissected coronary arteries.[9] When IVUS was systematically used in the evaluation of SCAD, coronary atherosclerosis was

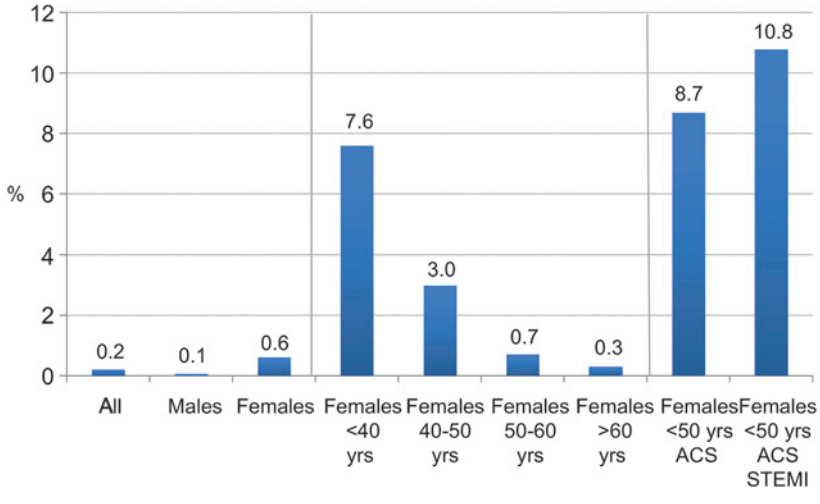

Figure 1 Incidence of spontaneous coronary artery dissection in different subsets of patients. ACS, acute coronary syndromes; STEMI, ST elevation myocardial infarction; yrs, years. Redrawn from Vanzetto *et al*.[10]

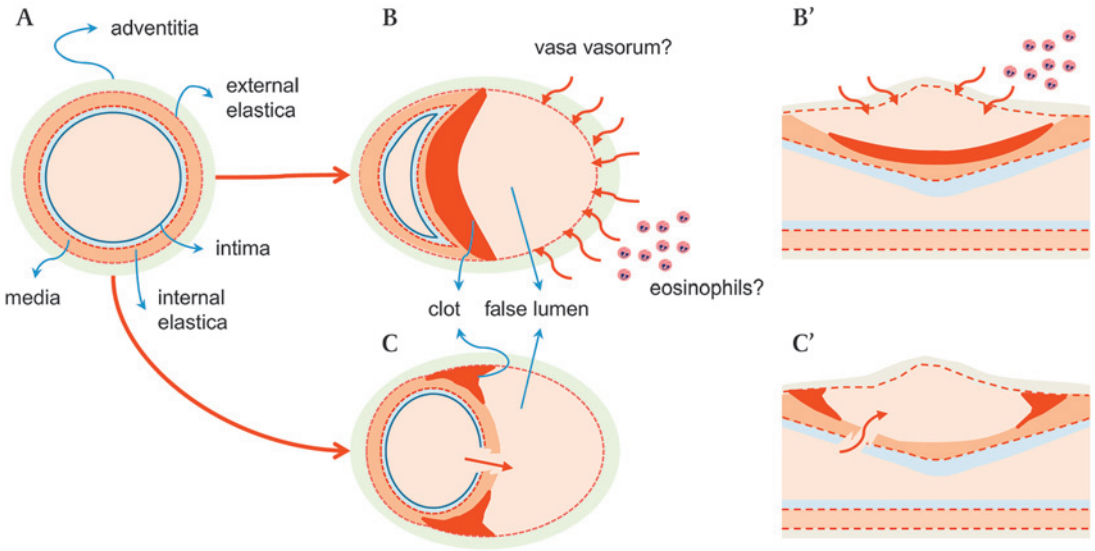

Figure 2 Different types of spontaneous coronary artery dissection (SCAD). A: cross-sectional image of a normal coronary artery. B and B′: Cross-sectional (B) and longitudinal (B′) section showing a coronary dissection resulting in medial dissection and intramural haematoma formation without an intimal tear. This type of dissection can only be diagnosed by intravascular ultrasound or optical coherence tomography. Bleeding from ruptured vasa vasorum and/or eosinophilic infiltrates may play a role in the pathogenesis of this type of SCAD. C& C′: Cross-sectional (C) and longitudinal (C′) section of a coronary artery showing a dissection resulting from an intimal tear with medial dissection and intramural haematoma formation. Redrawn from Aqel *et al*[13] and Angelini.[14]

detected in 35 of 42 patients.[8] Intriguingly, an intimal tear is not always detectable by IVUS.[12] A possible explanation for this observation is that the initial tear eventually seals under the pressure generated within the neolumen or because of spontaneous clotting within the neolumen.[14] Another explanation for the absence of a visible intimal tear in a number of patients with SCAD is the presence of a different pathophysiological mechanism—an increased density of vasa vasorum related to the inflammatory process involved in atherosclerotic plaque formation may cause intramedial haemorrhage and subsequent dissection. There is indeed increasing evidence linking intimal neovascularisation and immature microvessels originating from adventitial vasa vasorum not only with plaque destabilisation and rupture but also with the progression of atherosclerotic plaque.[15] Microvessel density in the adventitia and at the intima–media border is increased already early on in the development of atherosclerosis. It is conceivable that these changes that are associated with an increased propensity of microvascular rupture and bleeding may promote intramural dissection and haematoma formation in patients with only mild atherosclerotic lesions.

SCAD in the peripartum period

One third of all SCAD cases in women occur in the peripartum period, of which one third occur in late pregnancy and two thirds in the early postpartum period.[4 16] The peak incidence is within the first 2 weeks after delivery. The earliest reported case presented at 9 weeks of conception and the latest 3 months postpartum. Only 30% of the patients in this group have known risk factors for coronary artery disease.[16] Patients with advancing age and multiparity have an increased risk for SCAD.[16] The left anterior descending artery was involved in 78% of cases, the left main in 24%, and multivessel dissection occurred in 40%.[16]

The pathogenesis of SCAD in the peripartum period is still unclear. Haemodynamic factors together with arterial wall changes related to pregnancy, a lytic action of proteases released from eosinophils, and intimal tears are the main hypotheses presented to explain the pathophysiology involved.

Changes in the concentrations of sex hormones are thought to alter the normal arterial wall architecture, resulting in an increased susceptibility to spontaneous dissections. The changes in the vascular wall include smooth muscle cell proliferation, impaired collagen synthesis, and alterations in the protein and acid mucopolysaccharide content of

Table 2 Aetiology of spontaneous coronary artery dissection

Atherosclerosis
Peripartum episode
Other causes and conditions
Connective tissue disorders
Type IV Ehlers–Danlos syndrome
Marfan syndrome
Idiopathic cystic medial degeneration
Vasculitis
Systemic lupus erythematosus
Polyarteritis nodosa
Exercise
Prolonged sneezing
Oral contraceptive use
Idiopathic

the media. The very high percentage of multivessel involvement observed in pregnant women with SCAD supports the hypothesis that arterial wall changes during pregnancy under hormonal influence are an important causal factor in the pathogenesis of SCAD in the peripartum period.

During pregnancy total blood volume and cardiac output are increased. This may lead to augmented shear forces on the luminal surface and an increased wall stress in pregnancy and particularly during labour. Both vascular and haemodynamic changes occurring during pregnancy and labour therefore predispose the coronary arteries to the development of intramural dissections.

A periadventitial infiltrate composed of eosinophilic granulocytes involving the vasa vasorum has been observed in patients with SCAD.[17] In autopsied peripartum cases an eosinophilic infiltrate was reported in over 50% of the dissections.[17] During labour and the peripartum period eosinophils infiltrate the cervix and uterus where they are possibly involved in remodelling of the cervix during parturition and in the postpartum uterine involution. The infiltrate of eosinophils that can be observed in dissected coronary arteries could be a systemic manifestation of this process. Eosinophil granules contain lytic enzymes including collagenase and major basic protein and other substances that have a cytotoxic effect. It was postulated that spontaneous dissection could result from the breakdown of medial–advential wall layers by the substances released by activated eosinophils.[17] Of note, eosinophils possibly also play a role in the pathogenesis of postpartum cardiomyopathy since focal subendocardial infiltrates with eosinophils have been observed in the myocardium of patients with this condition.[17] Others, however, suggest that the eosinophilic inflammation along the dissection plane is reactive and not the cause.

Since an intimal tear is found only infrequently at autopsy, disruption of the vasa vasorum leading to intramedial haemorrhage and subsequent dissection without an intimal tear also has been proposed as a possible mechanism in this subset of patients with SCAD.[18] It has been suggested that normal coronaries may be more susceptible to luminal compression by intramedial haemorrhage in the absence of the stenting effect of atheroma, providing a potential explanation for predilection of this disorder in young women with otherwise normal coronary arteries.

On the other hand, SCAD is not the exclusive cause of peripartum acute myocardial infarction.[19] In a population based study of more than 12 million deliveries in the USA, the incidence of acute myocardial infarction was 6.2 per 100 000; 45% of the women diagnosed with acute myocardial infarction underwent coronary angiography and 37% have undergone a revascularisation procedure. A recent review of the literature revealed a high incidence of risk factors for ischaemic heart disease in patients with pregnancy associated myocardial infarction. Evaluation of coronary artery morphology (angiographically or at autopsy) revealed a dissection only in 28% of the patients, whereas a coronary stenosis was observed in 40%. Coronary dissection was the primary cause of infarction in the peripartum period (50%) and was found more commonly in postpartum compared with antepartum cases (34% vs 11%).[19]

Other causes and conditions

A number of connective tissue disorders have been associated with SCAD such as type IV Ehlers–Danlos syndrome and Marfan syndrome. In these disorders dissection arises from medial degeneration of the coronary arteries. Non-Marfan idiopathic cystic medial degeneration has been reported as cause of a single or multiple vessel SCAD. This disorder is characterised as in Marfan syndrome by focal fragmentation of elastic fibres and loss of smooth muscle cells of the media associated with deposits of varying amounts of acid mucopolysaccharides.

Vasculitis, including systemic lupus erythematosus and polyarteritis nodosa, has been associated with the occurrence of SCAD.

Several case reports have described an association of SCAD with exercise, prolonged sneezing or cocaine abuse. Cocaine induced systemic hypertension or coronary vasospasm is thought to cause coronary dissections.

Finally, SCAD has also been associated with oral contraceptive use; the pathogenesis is probably related to the same mechanisms as those implicated in the peripartum period.

Idiopathic SCAD

In many cases no condition can be detected that may cause SCAD or that has been associated with it, and such episodes must therefore be classified as idiopathic. Most if not all of the patients with SCAD are otherwise healthy premenopausal women. Conventional coronary risk factors do not seem to be related to idiopathic SCAD, even if a weak association with smoking and hypertension has been reported.

CLINICAL PRESENTATION AND DIAGNOSIS

The clinical presentation ranges from unstable angina, acute myocardial infarction, ventricular arrhythmias to sudden cardiac death.[8–11 16] In rare instances it can be asymptomatic and discovered incidentally on coronary angiography.

Whenever a young patient without major coronary risk factors or a woman in the postpartum period presents with an acute coronary syndrome or sudden cardiac death, the possibility of a SCAD should be suspected and an urgent coronary angiography considered.

Coronary angiography

Intubation of the coronary ostium and dye injection should be done with great caution in patients suspected of having a spontaneous coronary dissection, as forceful actions during coronary

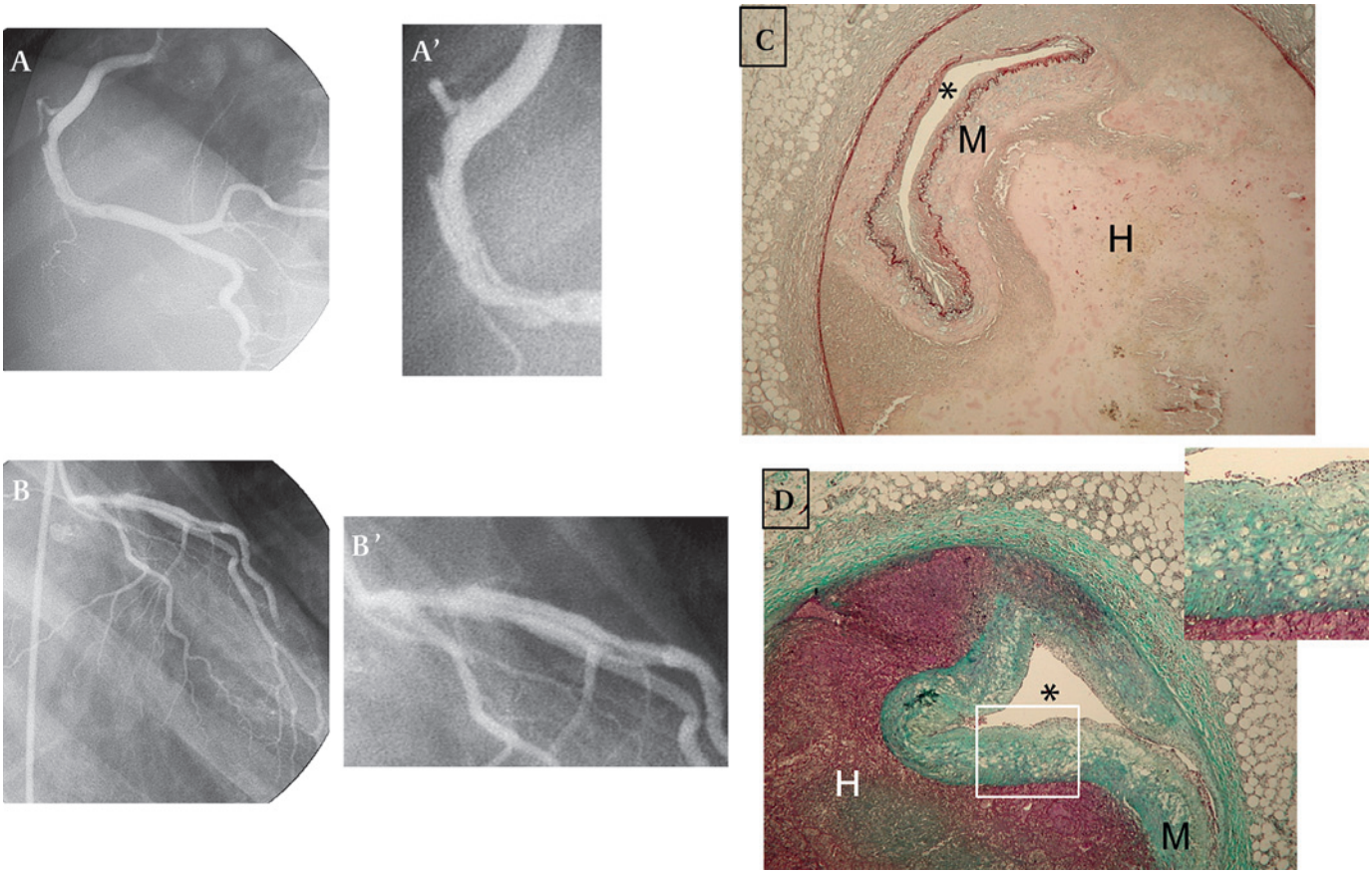

Figure 3 Spontaneous coronary artery dissection of the three major coronary arteries in a 36-year-old woman subsequent to cystic media necrosis. A & A′ (zoomed): Coronary angiography showing a spiralling type D dissection with extraluminal staining of the vessel wall in the mid segment of the right coronary artery. A discrete retrograde filling of the left anterior descending artery can also be discerned. B & B′ (zoomed): Long type D dissection originating from the left main coronary artery and extending into the proximal and mid segments of the left anterior descending artery. C: Cross-sectional section of a coronary artery with dissecting haematoma (H) compressing the lumen (*). The haematoma is situated between the media (M) and the external elastic membrane. There is only a slight thickening of the intima. Orcein-Alcian blue staining. D: Similar findings as in C. The media shows severe cystic medial necrosis (see magnified part). Trichrome Masson staining.

angiography may lead to extension of the dissection. The typical appearance of a coronary dissection on coronary angiography is the presence of a thin longitudinal radiolucent line representing an intimal medial flap with flow in two or more separate lumens (figure 3). The intramural lumen may fill and empty slowly and may distally end in a cul de sac with stasis of dye in between the injections. The intraluminal dissection flap may show haziness and intraluminal filling defects indicative of the presence of thrombi may be observed. The distal lumen of the coronary artery may be narrowed due to compression by the intramural haematoma. Eventually the coronary artery shows a long eccentric narrowing without the presence of a visible intimal flap. This may occur when there is no flow of dye into the false lumen or when there is a deep intramural haematoma without any communication with the true lumen of the coronary artery. In this case, the presence of a dissection may not always be recognised clinically and the eccentric narrowing caused by the intramural haematoma impinging on the lumen can be misinterpreted as an atherosclerotic stenosis. If a (very) long eccentric stenosis with protruding convex smooth borders is observed in a young or middle aged woman presenting with an acute coronary syndrome, and who otherwise does not have other signs of atherosclerosis on coronary angiography, the clinician should suspect a SCAD with a deep intramural haematoma and should proceed to an examination with either IVUS or OCT.

Intravascular ultrasound

IVUS can diagnose SCAD, especially those that are angiographically inapparent. Typical IVUS features of SCAD are the presence of an intramural haematoma in the outer third of the media compressing the true lumen with minimal or no atherosclerosis present (figure 4).[12 20] IVUS also enables assessment of the length and morphology of the intramural dissection, and the detection of an intimal tear if present. Moreover, during interventional treatment IVUS guidance will ensure correct wire placement and will allow geographically accurate positioning and an optimal deployment of coronary stents.

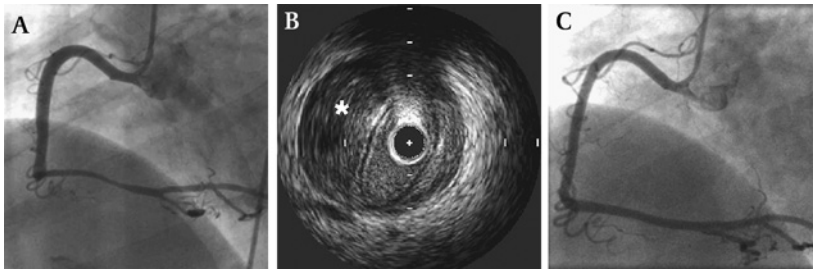

Figure 4 A: Left anterior oblique (LAO) view showing long stenosis of the right coronary artery. B: Corresponding intravascular ultrasound examination showing haematoma (asterisk) compressing the true lumen. C: LAO projection showing a favourable angiographic appearance at follow-up. Reproduced with permission from Arnold *et al*.[20]

Optical coherence tomography

OCT is a new, light based imaging modality that provides in vivo very high resolution images of the coronary artery. In cases of SCAD the unique spatial resolution of OCT provides a very precise visualisation of the intimal tear and an assessment of the localisation and length of the intramural haematoma unparalleled by existing IVUS imaging systems[21] (figure 5).

Coronary CT angiography

Multidetector row CT coronary angiography allows non-invasive imaging of coronary artery disease, albeit with lower spatial resolution and somewhat less accuracy than invasive coronary angiography. Since coronary CT angiography provides a direct view of the coronary plaque composition and wall thickness it may be useful in the diagnosis and assessment of SCAD. Several reports have demonstrated the ability of CT coronary angiography to visualise the presence of intimal dissections and the extent of intramural haematomas in cases of SCAD.[22] Furthermore, given its non-invasive nature, CT coronary angiography may be the optimal imaging method for the follow-up of patients with SCAD in whom a conservative management is initially chosen.

MANAGEMENT

There is no specific guideline on how to manage patients with SCAD. Treatment options for SCAD include medical therapy, percutaneous coronary intervention (PCI), or coronary artery bypass graft surgery (CABG). The decision to manage SCAD conservatively with medication or to perform PCI or CABG must be individualised based on both clinical and angiographic factors (figure 6).

When there is no evidence of ongoing ischaemia or haemodynamic instability, SCAD can be managed successfully with medical treatment alone.[7 23] With conservative management partial or even complete angiographic resolution of coronary artery dissections has been observed after a follow-up period of 2 months to 1 year.[3 13 20 22] Coronary CT angiography can be used as an alternative imaging method for the assessment of angiographic resolution after medical treatment of SCAD.

Medical management of SCAD is similar to the treatment of acute coronary syndromes. It includes antithrombotic therapy with heparin or low molecular weight heparin, aspirin, clopidogrel and glycoprotein IIb/IIIa inhibitors, and anti-ischaemic therapy with β-blockers and nitrates. However, the use of a potent antithrombotic therapy in SCAD may be a double edged sword: on one hand it will help decrease thrombus formation in the false lumen, allowing for more normal blood flow through the true lumen; on the other hand it may increase bleeding in the false lumen causing an expansion of the intramural haematoma, resulting in a decreased flow through the true lumen. Treatment with fibrinolytics should be avoided since their use has been reported to lead to a deterioration of the clinical condition, presumably due to a further extension of the coronary dissection

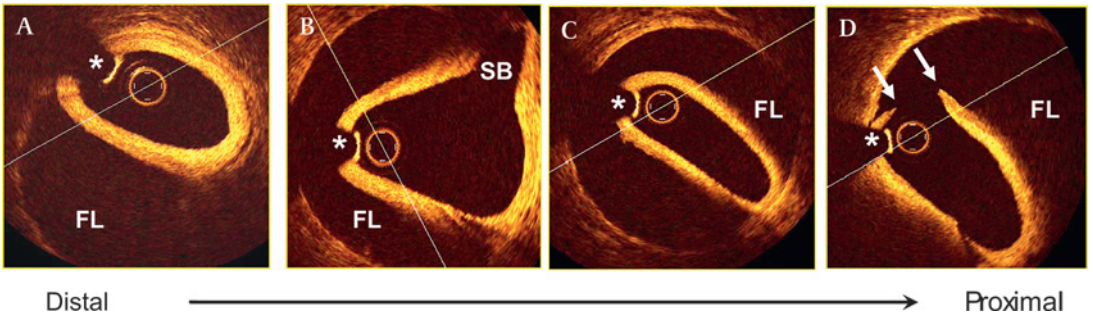

Figure 5 Optical coherence tomography (OCT) imaging of a spontaneous coronary artery dissection in a 50-year-old man. A–D from distal to proximal vessel. *Represents wire artefact. OCT imaging provides a clear view on the slit-like true lumen compressed by a large intramural haematoma (FL) caused by an intramedial dissection and also of the intimal tear in the proximal part of the vessel (arrows). Reproduced with permission from Alfonso *et al*.[21]

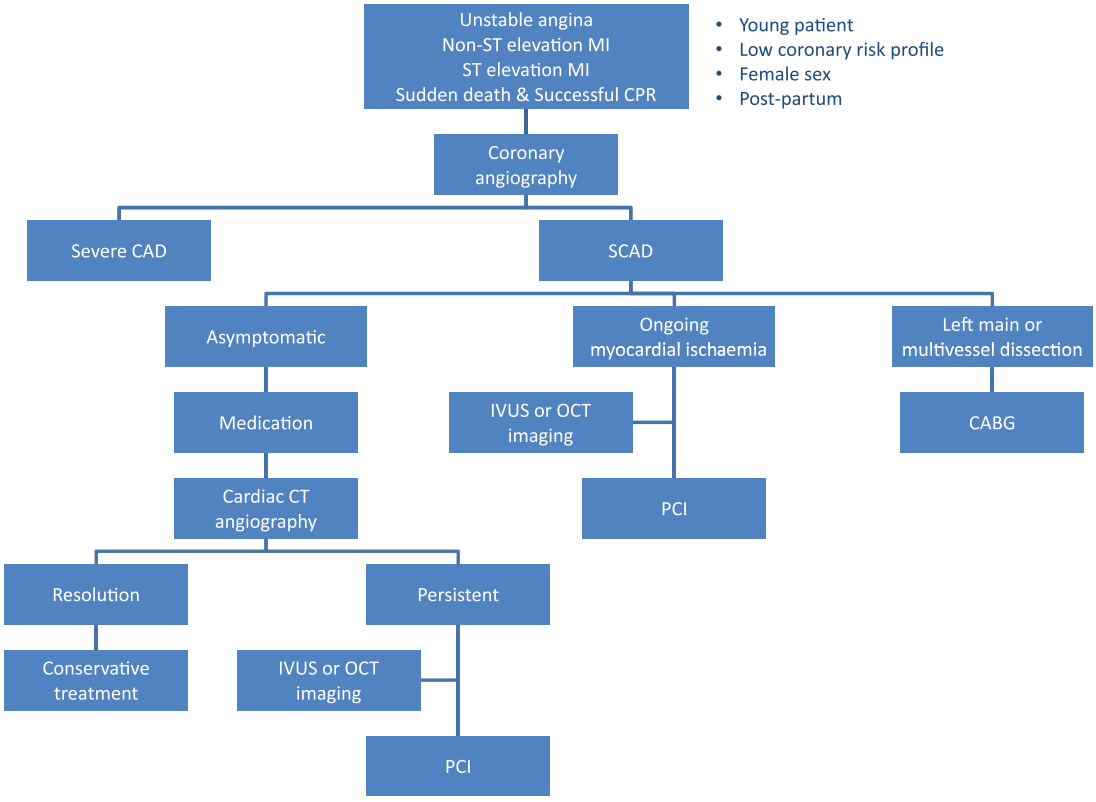

Figure 6 Diagnosis and treatment of spontaneous coronary artery dissection. CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; CPR, cardiopulmonary resuscitation; CT, computed tomography; IVUS, intravascular ultrasound; MI, myocardial infarction; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; SCAD, spontaneous coronary artery dissection.

caused by the fibrinolytic induced bleeding into the dissected vessel wall.

If a pronounced dissection persists in a major vessel after prolonged medical treatment, or in SCAD causing marked epicardial coronary flow impairment and/or ongoing ischaemia, PCI or CABG should be considered. PCI with stenting can restore flow in the true lumen, relieving ischaemia, and seal the dissection, preventing further expansion. Technical difficulties during PCI include advancing the guidewire in the true lumen rather than in the false lumen, and avoiding distal propagation of the intramural haematoma and dissection during stent delivery. The latter can be prevented by deploying the first stent with sufficient coverage of the distal border of the dissection. IVUS or OCT imaging can be used to confirm guidewire placement in the true lumen, evaluate the length of dissection and vessel size, assist in the correct positioning the first stent to deliver and assess stent apposition, and to seal the dissection at the end of the intervention. The clinical success rate of stenting in patients with SCAD is over 90%.

Single vessel dissections of major coronary arteries are usually managed with PCI with stenting, while left main dissection, multivessel involvement, or failed PCI procedures are treated by CABG. In cases of spontaneous dissections involving a long coronary artery segment, CABG can be very challenging.[10] The vessel wall may be fragile due to the underlying condition predisposing to dissection.

PROGNOSIS

In-hospital mortality of SCAD is relatively low, with a mean rate of around 3% (0–4%).[3 5–10] Patients who survive the acute phase have a good long term prognosis, with a very low recurrence rate of SCAD[8 10] or acute coronary syndrome,[8 10] and a 95% 2 year survival rate.[4] Although outcome

Spontaneous coronary artery dissection (SCAD): key points

- Incidence of SCAD is very low.
- SCAD occurs most often in young women (age <40 years).
- It occurs frequently in the peripartum period.
- Left anterior descending coronary artery is the most frequently involved vessel.
- Multivessel dissection is present in 20% of the cases.
- SCAD results from haematoma formation in the outer third of the vessel wall.
- An intimal tear is only seldom observed.

is in general good, the overall mortality in reported cases of the peripartum group is 38%.[16] Multivariate analysis of 222 patients from several publications showed that the strongest predictors of death included female sex (odds ratio (OR) 4.27) and absence of early treatment (OR 35.5).[4]

Diagnosis and management

- SCAD presents as acute coronary syndrome or sudden cardiac death.
- Early coronary angiography remains essential in the diagnosis of SCAD.
- Additional IVUS and OCT imaging provides very detailed information on the location and extent of the dissection.
- Patients with ongoing myocardial ischaemia must be treated with early revascularisation (PCI or CABG).
- Patients without flow limiting dissection should be treated conservatively as spontaneous healing is possible.
- CT coronary angiography is useful in the follow-up of conservatively managed patients.

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