Spontaneous Coronary Artery Dissection: A brief review of contemporary practice

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Spontaneous coronary artery dissection (SCAD) is becoming increasingly recognised as an important cause of myocardial infarction, especially in young women with few risk factors for atherosclerotic disease. Diagnosis has increased in recent years largely due to the increased use of coronary angiography, intra-coronary imaging and increased awareness.[1]

Definition

Spontaneous separation of the coronary artery wall architecture in a non-atherosclerotic artery that is not iatrogenic or induced by trauma. The resulting intramural haematoma compromises antegrade blood flow giving rise to myocardial ischaemia.[2]

Epidemiology

The true prevalence of SCAD is likely underestimated and was previously thought to be a rare disease frequently associated with pregnancy. SCAD can be attributed to 1.7-4% of patients presenting with acute coronary syndromes (ACS).[3] SCAD affects women in the majority of cases (>90%) with a mean age range of 44 to 55 years.[3,4]

Pathophysiology

Dissection can occur between any of the three arterial layers (intima, media, adventitia). There are two postulated pathophysiological mechanisms. The intimal tear hypothesis describes a primary disruption in the intima creating a point of entry for intramural haematoma to accumulate in the false lumen. The medial haemorrhage hypothesis describes spontaneous haemorrhage into the arterial wall from the vasa vasorum leading to intramural haematoma. Both mechanisms result in accumulation of blood within the false lumen which compromises antegrade luminal flows resulting in myocardial ischaemia.[1]

Predisposing factors

Fibromuscular dysplasia is strongly associated with SCAD.[5] Other associated factors include connective tissue disorders (Marfan syndrome, Loeys-Dietz syndrome, Ehler-Danlos syndrome, cystic medical necrosis), systemic inflammatory disease (systemic lupus, Crohn's disease, ulcerative colitis) and hormonal therapy (oral contraceptives, beta-HCG).[1] Pregnancy related SCAD only accounts for approximately 5% of SCAD cases but can still occur out to 24 months post-partum.[6]

Clinical presentation

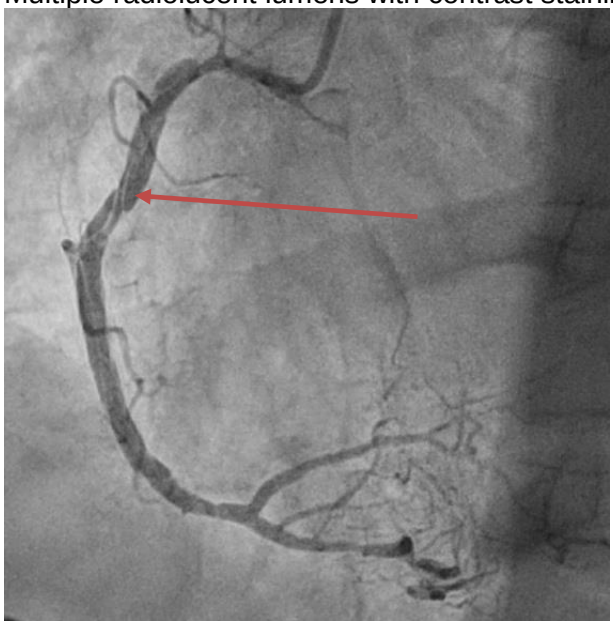
Chest pain is the most common presenting complaint. Dizziness, back pain and dyspnoea are less common presentations. Patients are often labelled as ACS early in the admission process.[7]

Diagnosis

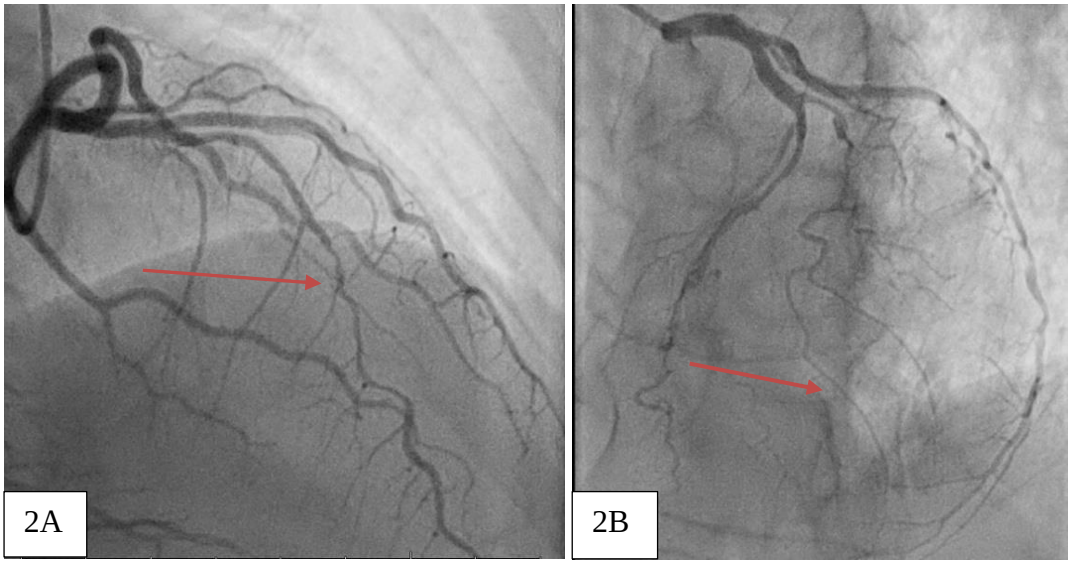
Coronary angiography is the first line investigation and quite often yields the diagnosis. Diagnostic accuracy is improved with intracoronary imaging (Optical Coherence Tomography (OCT) and IntraVascular Ultrasound). However, instrumentation of the dissected coronary artery may propagate the dissection plane, as can hydraulic forces from contrast injection to facilitate OCT. Despite this OCT is still preferred as the higher resolution corresponds to a higher diagnostic yield. Dissections are observed in multiple arteries in 9-19% cases.[8] The left anterior descending artery is the most commonly affected. Cardiac CT angiography has limited role in the initial diagnosis of SCAD due to poor spatial and temporal resolution and the tendency for SCAD to affect smaller calibre (<2.5mm) distal arteries making CT angiography difficult as a diagnostic tool. It may be useful as a non-invasive follow up assessment to ensure healing has occurred, especially in larger calibre proximal vessels.[9,10]

Angiographic classification

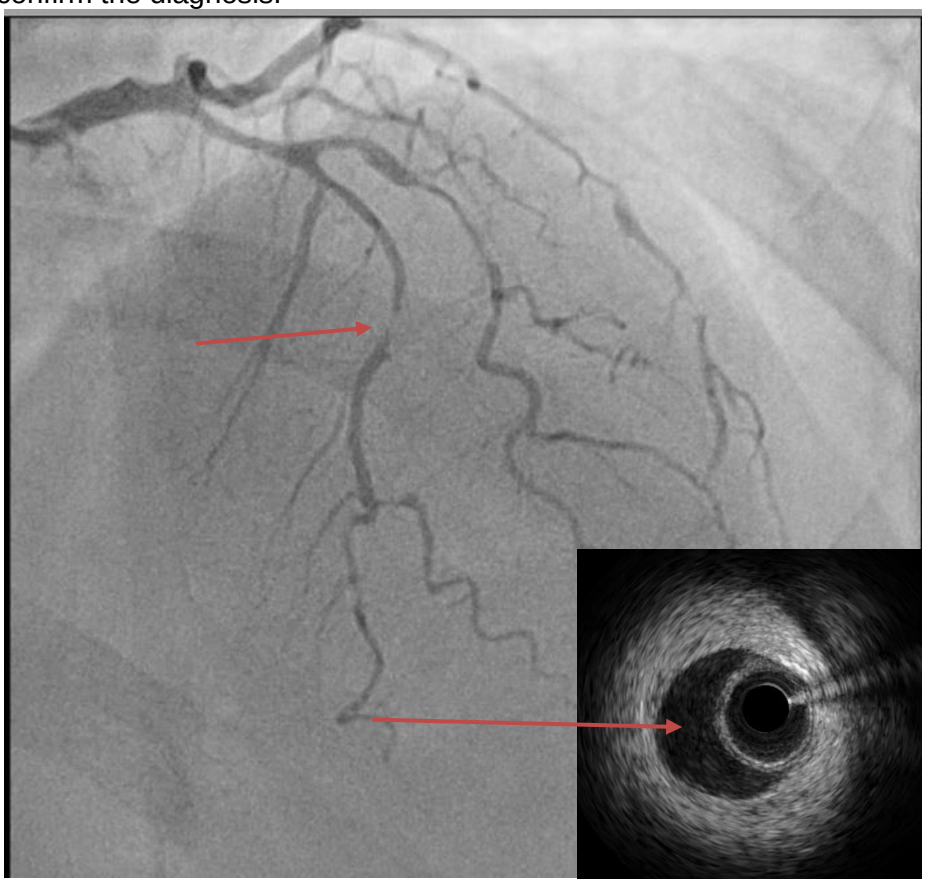
Type I
Multiple radiolucent lumens with contrast staining



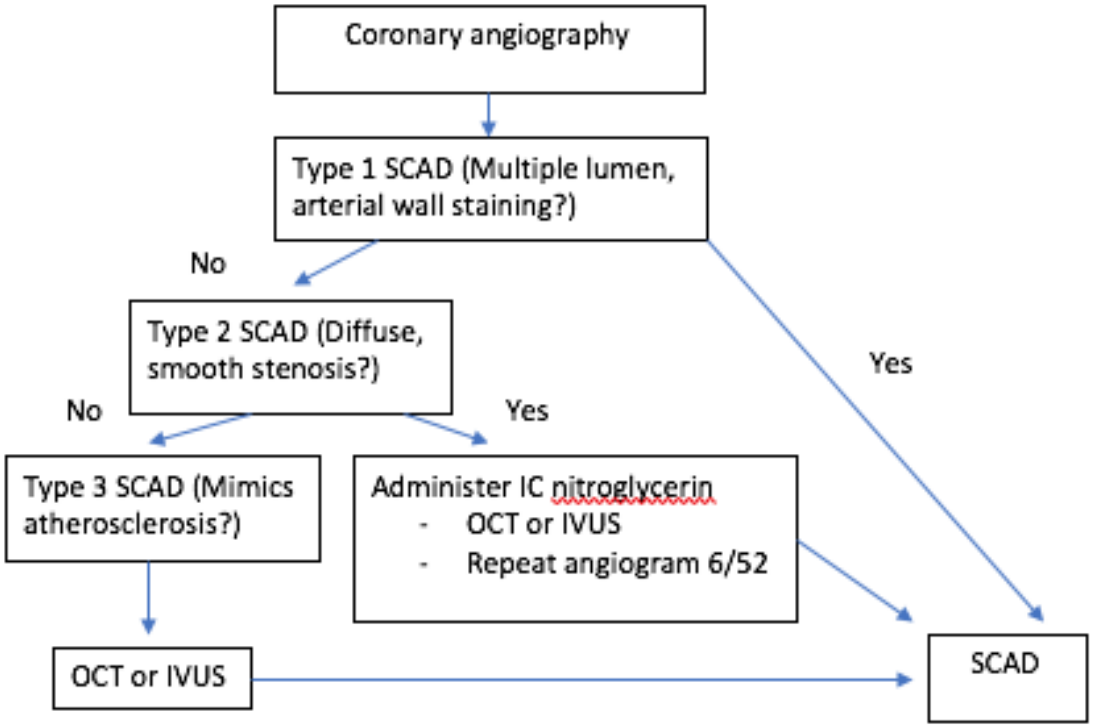
Type II
Diffuse stenosis with abrupt changes in arterial calibre from normal diameter to diffuse smooth narrowing. Stenosis is often long (>20mm) and may be bordered by normal artery segments proximal and distal to the intramural haematoma (Type 2A) or may extend to the apical tip of the artery (Type 2B).



Type III
Focal stenosis mimicking atherosclerotic disease which requires intracoronary imaging to confirm the diagnosis.



IVUS or OCT requires the visualisation of intramural haematoma or a double lumen to make the diagnosis. Image showing IVUS with wire in true lumen and crescent shaped larger false lumen with intramural haematoma.



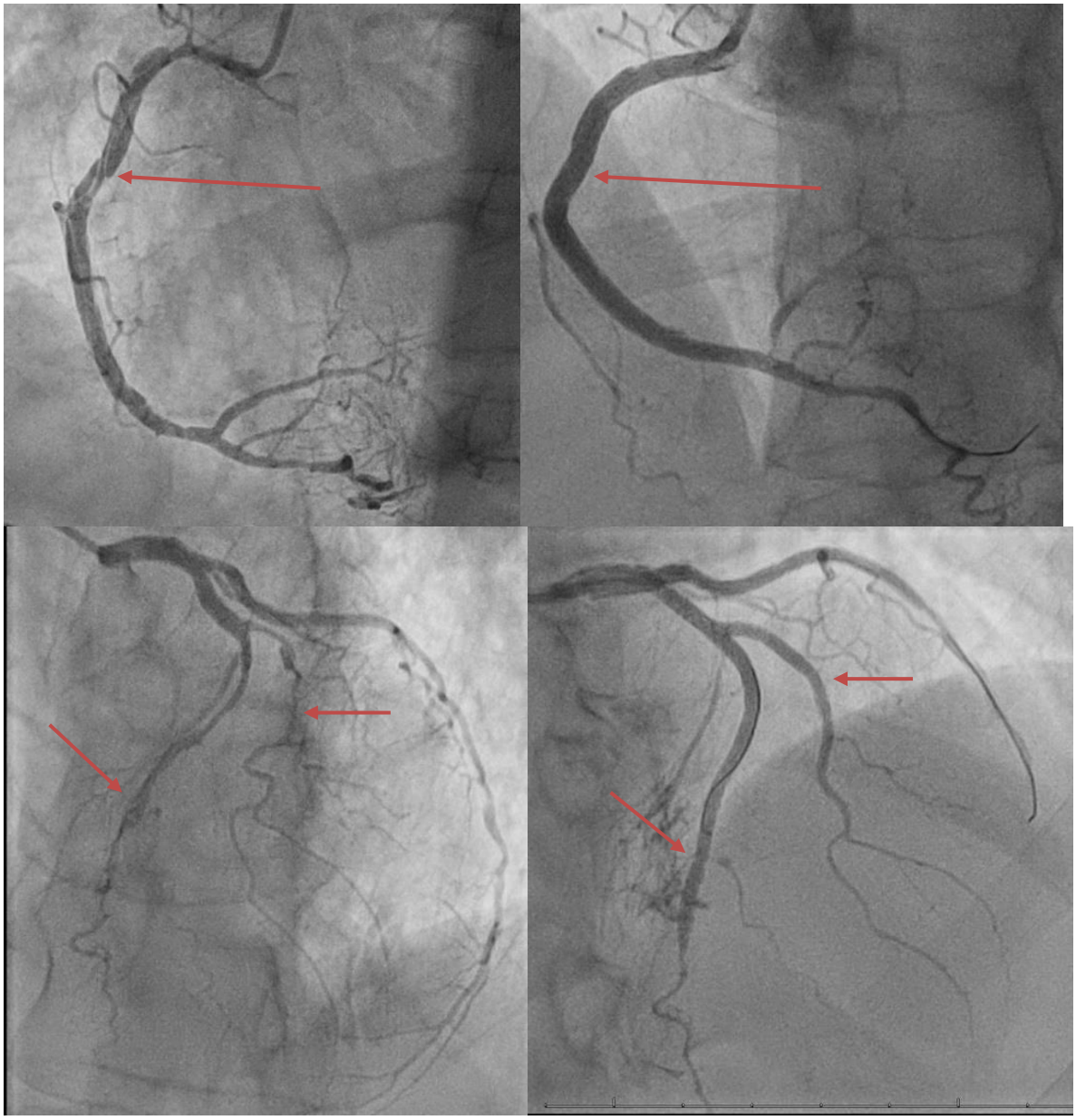
Diagnostic algorithm adapted from Contemporary review on spontaneous coronary artery dissection by Saw et al.

Management

Arteries affected by SCAD tend to heal spontaneously thus a conservative approach is preferred if possible. Observational data suggests that arteries are healed by 26 days post-dissection.[2,6] Recurrent myocardial infarction rate is approximately 4.5% therefore a period of inpatient monitoring is required. If there is ongoing ischaemia, ventricular arrhythmia or cardiogenic shock then revascularisation should strongly be considered.[1]

Revascularisation by PCI

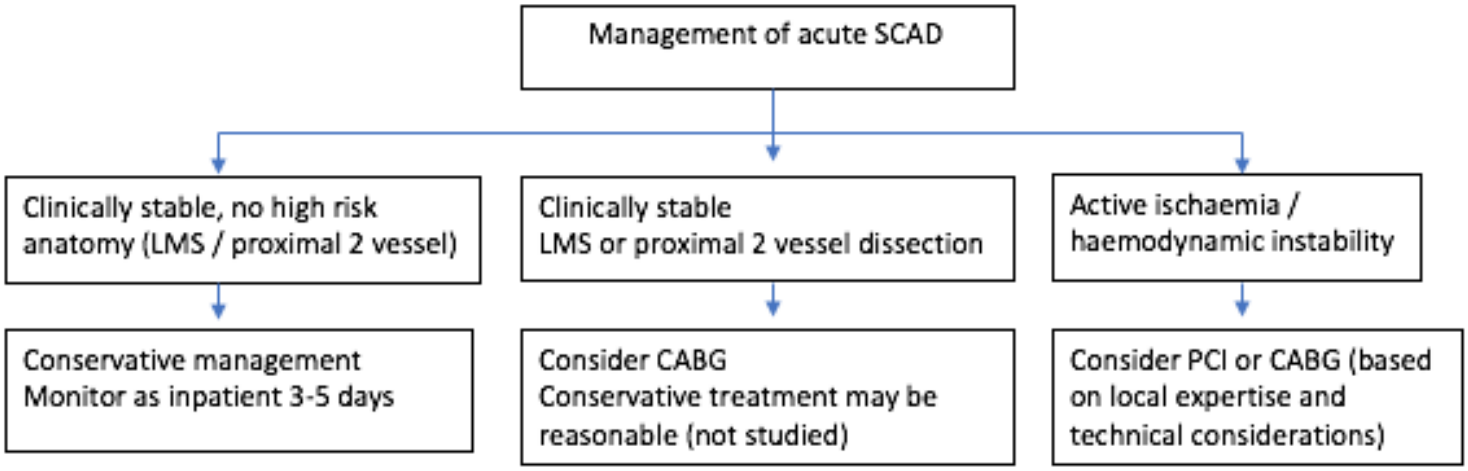
SCAD arteries are architecturally weakened and thus instrumentation can propagate the dissection. Intramural haematoma can be displaced antegradely or retrogradely compromising blood flow in the true lumen. Long stents are recommended to accommodate extension of the haematoma. There are case reports of cutting balloons being used to decompress the intramural haematoma.[11] The natural response to intramural haematoma over time is resorption which can lead to stent strut malapposition which increases the risk of late stent thrombosis therefore follow up coronary angiography and intracoronary imaging should be considered.



SCAD images before and after revascularisation. Right coronary artery reconstructed with three overlapping drug-eluting stents. Left main stem, left circumflex and left anterior descending artery reconstructed with seven overlapping drug-eluting stents.

Revascularisation by CABG

Emergent surgery should be considered in patients with extensive multivessel dissections and where revascularisation by PCI is not possible. Long term results with graft patency however are poor as the native vessel heals and restoration of competitive antegrade flow encourages graft thrombosis. Long term graft patency is as low as 27%.[12]



Invasive management algorithm adapted from the scientific statement from the American Heart Association 2018

Medical therapy

Dual antiplatelet therapy should be considered for 1 – 12 months post SCAD as the antithrombotic effect may reduce the size of the intramural haematoma.[2] There is little evidence to support the use of angiotensin receptor blockers or beta blockers in the absence of left ventricular systolic dysfunction. Rate of SCAD recurrence is approximately 15%.[6]

Research

The team at Glenfield Hospital in Leicester are prospectively recruiting SCAD survivors into their national research program. This can also act as an important support network for patients and referral is encouraged. More information can be found on their website: https://scad.lcbru.le.ac.uk. Other collaborative efforts on a wider scale include the European Observational Research Platform (EORP) SCAD study, which will open for recruitment in the summer of 2018, and aims to establish evidence based best treatment for this challenging disease process.[10]

Conclusion

SCAD is an uncommon cause of acute coronary syndromes but should be suspected in young women without traditional risk factors for IHD. A conservative approach is preferred but ongoing high-risk features may necessitate revascularisation.

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