

# Spontaneous Coronary Artery Dissection

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## Learning objectives:

- To recognise spontaneous coronary artery dissection as a cause of myocardial infarction in low risk, predominantly female, patients
- To understand that although spontaneous coronary artery dissection is an important cause of peripartum myocardial infarction, ~90% of incident cases are not pregnant
- To be aware that a visible dissection flap at angiography is absent in the majority of cases
- To understand the rationale for an 'as conservative as possible' approach to revascularisation and key challenges in medical treatment

## Introduction

Spontaneous coronary artery dissection (SCAD) is an increasingly recognised cause of non-atherosclerotic acute coronary syndromes leading to myocardial infarction. It is characterised by the presence of blood entering and separating the layers of the coronary arterial wall to form a false lumen. This leads to external compression of the true coronary lumen restricting coronary blood flow and leading to coronary insufficiency (Figure 1). SCAD should be distinguished from atherosclerotic dissections arising from plaque rupture events or erosions allowing blood to enter the intimal space and from iatrogenic dissections arising during coronary procedures.

## Main text

### *Epidemiology*

The true incidence of SCAD is unknown, largely because most patients fall into the lowest risk categories for conventional atherosclerotic disease leading to under-diagnosis and probably lower rates of presentation by patients.<sup>1</sup> Historically thought to be very rare, increasing use of high sensitivity Troponin with early angiography for the assessment of acute chest pain presentations has led to a greater recognition of SCAD. However, accurate angiographic diagnosis can be challenging and reported rates of SCAD diagnosis from angiographic registries (0.2% - 0.7% of angiograms; 2.0% of ACS angiograms)<sup>2, 3</sup> probably significantly underrepresent true SCAD incidence. There are currently no blood biomarkers which distinguish SCAD from other causes of ACS.

SCAD overwhelmingly affects young to middle-aged women with males accounting for less than 10% of cases in most large contemporary series.<sup>4-8</sup> Indeed around a quarter of ACS presentations in women under the age of 50 are reportedly due to SCAD.<sup>8,9</sup> The mean age of affected patients ranges from 42-53 but the condition has been reported in patients aged 18-84.<sup>4,5</sup>

### *Pregnancy*

Although historically described primarily in the context of peri-partum myocardial infarction, SCAD associated with pregnancy is now recognised to account for a small proportion of the overall disease burden (5-18%).<sup>4,8</sup> SCAD may however cause a significant proportion of peripartum myocardial infarctions, predominantly occurring late in gestation or early, late or sometimes very late in the post partum period.<sup>10-12</sup> Some evidence suggests that multiparity might be a risk for pregnancy associated SCAD.<sup>7,13</sup>

### *Mechanical stressors*

SCAD may be precipitated by Valsalva-type manoeuvres such as coughing<sup>14</sup>, vomiting<sup>15</sup>, heavy lifting<sup>16</sup> or by extreme or isometric exercise.<sup>17-19</sup> It is hypothesised that this results from transient increases in coronary wall shear stress. In some cases however, there seems to be a significant lag time between the last exercise episode and the onset of symptoms of SCAD, suggesting high exercise capacity may be part of a SCAD patient phenotype rather than a direct cause of the dissection. An association with exercise is more frequent in male SCAD cases<sup>20</sup>. Altered coronary shear stress and vasoreactivity may also explain the rare case-reports associating SCAD with cocaine,<sup>21-23</sup> amphetamine use<sup>5,24,25</sup> and a possible association with migraine.<sup>26</sup>

### *Emotional stressors*

Emotional stress and sometimes preceding physical illness are reportedly associated with SCAD events, with apparent emotional precipitants more common in female patients.<sup>5,8,20</sup>

### *Connective disorders, autoimmune and inflammatory disorders*

There are several reports of SCAD in patients with known connective tissue disorders (e.g. Marfan<sup>27</sup>, Ehler-Danlos type 4,<sup>28</sup> Loeys-Dietz syndromes<sup>29</sup> and adult polycystic kidney disease<sup>30-33</sup>). However, specific genetic testing in SCAD-survivors has a low yield.<sup>34</sup> Likewise SCAD has been rarely reported in association with autoimmune/inflammatory disorders including Behcets,<sup>35</sup> systemic lupus erythematosus,<sup>36-41</sup> polyarteritis nodosa<sup>42</sup> and inflammatory bowel disease<sup>43</sup> but again these cases represent rare associations rather than typical presentations.

### **Presentation**

Patients with SCAD present with acute coronary syndromes, usually with chest pain associated with an elevation of cardiac enzymes.<sup>44</sup> The proportion of patients presenting with ST-segment elevation (STEMI) versus non-ST-segment elevation (NSTEMI) varies between series with some reporting a dominance of STEMI<sup>4,6,8</sup> and others NSTEMI.<sup>5,7</sup> This likely reflects differences in patient selection between studies with the former series likely under-representing patients with less severe presentations. A small proportion (3-14%) present with resuscitated ventricular arrhythmia.<sup>4-6</sup> Some cases present as unexplained sudden death, although, because of the challenges of accurate post mortem diagnosis, this condition is likely under-represented in post mortem series.<sup>45,46</sup>

Patients with SCAD too frequently suffer from failed or delayed diagnosis<sup>44</sup>. This primarily results from emergency medical systems targeted to screen patients with acute chest pain presentations on the basis of traditional atherosclerotic cardiovascular risk factors. As a result some SCAD patients are not

appropriately referred for coronary angiography or referral is delayed. A high index of clinical suspicion is required for women presenting with typical chest pain and abnormalities of either ECG or cardiac biomarkers.

### ***Angiographic findings***

Whilst recognition of typical angiographic features and use of intracoronary imaging is improving accurate diagnosis, this condition remains under-recognised at angiography.<sup>9, 48</sup>

### ***Angiographic classification***

One key area of persisting misunderstanding is an expectation that the false lumen in SCAD will usually be angiographically apparent. Saw et al described an angiographic classification for SCAD which has been increasingly adopted.<sup>44</sup> Type 1 angiographic SCAD (Figure 2A) is characterised by an identifiable false lumen with a linear filling defect or dissection 'flap'. There is often contrast hold-up in the false lumen after clearance from the true lumen. This appearance is familiar to interventional cardiologists from catheter or interventional procedure induced dissections where contrast readily enters the false lumen. However, such cases make up a minority of SCAD angiograms (29-48%). More common are Type 2 lesions where contrast penetration of the false lumen is not evident. In Type 2a (Figure 2B) lesions, there is distal reconstitution of a normal vessel calibre (often preceded by a tapered critically narrowed segment at the distal extent of the dissection). In Type 2b lesions (Figure 2C), the stenosis continues to the distal extent of the affected coronary territory. Type 2 lesions account for 52-67% of presentations. Type 3 lesions (Figure 2D) are angiographically indistinguishable from atherosclerosis without recourse to intracoronary imaging but appear rare (2-3.9% of cases). We would also propose a Type 4 variant (Figure 2E) which presents with a total, usually distal, vessel occlusion where sources of coronary embolism have been excluded and there is subsequent evidence of complete vessel healing in keeping with the natural history of SCAD.

### ***Additional angiographic features***

Whilst SCAD has been reported in all coronary segments, it has a frequent predilection for more distal coronary segments (in contrast to atherosclerotic disease).<sup>4-7</sup> When it occurs, proximal disease more commonly has a Type 1 appearance and may have associated thrombus within the true lumen whilst mid to distal SCAD is more usually a Type 2 appearance. Although more discrete dissected segments do occur, long affected segments are more typical. Most<sup>4-6, 8</sup> but not all<sup>7</sup> series report the left anterior descending to be the commonest affected vessel. Coronary tortuosity is increased with a non-significant trend towards a higher risk of recurrence in those with the greatest tortuosity.<sup>26</sup> Simultaneous multivessel SCAD is well recognised.

### ***Vessel healing***

Although there are some reports of persisting dissections or coronary maladaptation following SCAD,<sup>4, 47, 48</sup> complete vessel healing in conservatively managed SCAD seems to occur in the most cases which remain stable during the acute period (Figure 3A&B) with normalisation reported to occur in the majority by 26-days.<sup>5, 7</sup>

### ***Intracoronary imaging***

Although the angiographic findings in SCAD may be characteristic, if diagnostic uncertainty remains, intracoronary imaging by intravascular ultrasound (IVUS) or optical coherence tomography (OCT) findings are usually characteristic. OCT has the advantage of higher spatial resolution (15-20µm) but limited depth penetration whilst IVUS has greater depth penetration but limited spatial resolution

(150 $\mu$ m).<sup>49, 50</sup> Because some important features (true lumen boundaries, fenestrations between true and false lumens and local thrombus) require higher resolution imaging, OCT is preferred for SCAD imaging by some authors.<sup>44</sup> A number of clinical issues should however be considered before imaging and/or deciding on the optimal imaging strategy. Firstly, in patients with TIMI3 flow and in whom the angiographic appearances are typical, there is an inherent risk that any coronary instrumentation of the dissected artery could exacerbate matters and potentially convert a case which could be conservatively managed into one in which intervention becomes necessary. Secondly, the high pressure contrast injection required for luminal blood clearance with OCT could extend the dissection (this is probably most relevant in proximal Type I dissections). Finally, given the relatively distal locations of many SCADs and especially in Type 2B dissections, it may be impossible to image the entire length of the affected segment (5/11 cases in one series<sup>51</sup>) and adequate blood clearance for OCT imaging from the true lumen may be challenging. However in expert hands, intracoronary imaging appears both safe and clinically useful.<sup>51</sup>

The classical OCT imaging findings in SCAD are described<sup>51</sup> and shown in Figure 4. The imaging catheter is sited in the true lumen with the false lumen, visible as a crescentic arc, separated by the intimal-medial membrane (Figure 4A). In some cases the true lumen may appear 'free floating' within the false lumen (Figure 4B). Fenestrations connecting false and true lumens may be evident but are not always present (an intimal tear was identified in 7/11 cases in one series<sup>51</sup>) (Figure 4A&C). Thrombus may be present either in the true or false (Figure 4B) lumens. IVUS (Figure 4D) may also be used but given the lower spatial resolution, care is required to ensure the intimal-medial membrane is not missed or misinterpreted.<sup>52, 53</sup>

## **Management of SCAD**

### *Revascularisation*

Decision-making about revascularisation in acutely presenting SCAD is more complex than for NSTEMI or STEMI of atherosclerotic aetiology for two principal reasons. Firstly, outcomes following revascularisation, whether surgical or by percutaneous coronary intervention (PCI), are less good<sup>54</sup> and secondly, there is a high likelihood of complete healing of the dissection with conservative management. This has led to a growing consensus in favour of conservative therapy where clinically possible (i.e. maintained TIMI 3 flow and haemodynamic stability) and limiting intervention to what is required to minimise myocardial injury where revascularisation is essential. All series do however report a small proportion of SCAD cases who progress with conservative management so close observation in the acute phase is necessary.

### *Percutaneous revascularisation in SCAD*

Whilst PCI in SCAD can be very successful and is certainly sometimes essential to restore blood flow in the affected vessel, there are a number of specific technical challenges. The anatomical site and extent of the dissection can be an issue as SCAD often involves longer and relatively distal small calibre coronary segments and with Type IIb dissections, there is no clear distal landing zone for a stent. Furthermore, the haematoma in the false lumen behaves very differently from fibroatheromatous plaque material following stent expansion, such that the haematoma frequently tracks proximal and/or distal to the stented segment creating new pre and/or post stent luminal restrictions which may require further stents (Figure 5C,D&E).<sup>55</sup> The overall result is that patients with SCAD undergoing PCI frequently require long stented segments to effectively restore luminal architecture. In general, where stents are deemed necessary, drug eluting stents are preferred. Bioabsorbable scaffold use has also been reported and the long term clinical results in this population are awaited.<sup>56, 57</sup> Other

interventional strategies are also described including limited balloon angioplasty to restore TIMI 3 flow followed by a conservative approach or even use of a cutting balloon to fenestrate the dissection and relieve external compression.<sup>58</sup> Where PCI is required, intracoronary imaging may be useful for procedure optimisation.<sup>51</sup> There is a report demonstrating late stent strut malapposition following resorption of the false lumen haematoma<sup>59</sup> but there is little current evidence of an increase in stent thrombosis following PCI in SCAD.

### *Coronary artery bypass grafting (CABG) in SCAD*

Emergency CABG is sometimes used as a bail-out strategy in SCAD. Two typical scenarios are either a failure of PCI (usually due to an inability to track the guidewire into the true lumen) or left mainstem or very proximal SCAD where there is felt to be a high degree of jeopardy involved with conservative management or stenting. Although useful in the short term for SCAD limited to the proximal vessel, grafting is unsuitable for distal or extensive dissections. Furthermore in the medium term, graft failure rates are high (only 5 of 16 conduits remained patent at 3.5 years in one series<sup>54</sup>) as a result of dissection healing in the native coronary leading to competitive flow in the bypass conduit (Figure 5A&B).

### *Thrombolysis*

Although safe and apparently effective thrombolysis has been described<sup>60</sup>, use in the context of SCAD is generally avoided because of descriptions of dissection extension or even rupture leading to cardiac tamponade<sup>61-64</sup>.

### ***Medication after SCAD***

There are currently no clinical trials to guide optimal medical management following SCAD. However there are a number of special considerations which distinguish the therapeutic approach in SCAD from standard treatment strategies following myocardial infarction.

### *Antiplatelet therapy*

Dual antiplatelet therapy followed by antiplatelet monotherapy are clearly indicated in patients who have undergone PCI with stenting for SCAD in accordance with guidelines<sup>65</sup>. However for those without stents the indication is less clear. Furthermore, women of child-bearing age can develop significant menorrhagia with antiplatelet therapy.<sup>66</sup> There is some pathophysiological logic in initiating antiplatelet therapy in the acute phase where intracoronary imaging is reported to show that SCAD can act as a substrate for thrombus formation within the true lumen.<sup>51</sup> In conservatively managed SCAD, once healing has been confirmed, the logic of maintaining platelet inhibition for a condition whose primary pathophysiological event is a spontaneous intramural bleed seems less evident, although discontinuing antiplatelet therapy in SCAD remains controversial.

### *Anticoagulant therapies*

The risk/benefit of anticoagulant therapies either acutely (prior to or during coronary angiography, PCI or cardiopulmonary bypass) or chronically is unknown. In general, concerns about dissection expansion/extension in the acute phase and the theoretical increased risk of recurrence should minimise their use. Where strong indications exist however, (e.g. left ventricular mural thrombus or thromboembolism), a lack of clear evidence of harm probably favours their use with careful monitoring in selected cases.

### *Statins*

Patients with SCAD are frequently commenced on statins as part of the standard evidence and guideline-based cocktail of medications usually initiated following myocardial infarction in atherosclerotic patients.<sup>67, 68</sup> However, the logic of prescribing cholesterol lowering agents for a condition which has no known pathophysiological link to cholesterol seems unclear and one study has suggested a link between statins and increased recurrence risk.<sup>4</sup>

### *Other post-myocardial infarct medications*

Where patients are left with impaired left ventricular function following SCAD, evidence and guideline-based medications (e.g. ACE-inhibitors, ARBs,  $\beta$ -blockers, mineralocorticoid-receptor antagonists) are recommended.<sup>67, 68</sup> However, as the SCAD population is generally younger, female and without pre-morbid hypertension, low blood pressure can be limiting in many patients. In normotensive SCAD-survivors with preserved left-ventricular function, a more conservative approach to these medications may be sensible.

### *ICD/devices/mechanical assist/transplantation*

Unfortunately some patients suffer significant myocardial injury as a result of their SCAD event(s). Extreme cases may present with cardiogenic shock requiring mechanical ventricular assist or even extra-corporeal membrane oxygenation (ECMO) as a bridge to transplantation<sup>69</sup>. Devices (ICD/CRTD) should be considered in patients with severe impairment of left ventricular function in accordance with guidelines.<sup>67, 68</sup>

### ***Convalescent imaging***

#### *Coronary CT*

Although CT findings in SCAD have been described,<sup>70</sup> the relatively low spatial resolution of CT combined with variations in contrast penetration of the false lumen and the distal coronary location of many SCAD events, limit use for the primary diagnosis of this condition<sup>71</sup>. Where CT may have utility is in follow-up of established conservatively managed cases to non-invasively demonstrate coronary healing,<sup>48</sup> especially given reports of iatrogenic dissections occurring during follow-up angiography.<sup>72</sup>

#### *Assessment of LV systolic function*

As with myocardial infarction of any aetiology, an assessment of left ventricular function in the recovery phase following SCAD is mandatory. Echocardiography or cardiac MRI can be used.<sup>71</sup> This is of particular importance as there is some suggestion that recovery of left ventricular function following SCAD myocardial infarction may be better than that following conventional atherosclerotic events<sup>73-75</sup> and this may impact on medical treatment choices.

#### *Peripheral arterial assessment: fibromuscular dysplasia (FMD)*

SCAD is frequently a coronary manifestation of a more widespread arteriopathy. Remote dissections and aneurysms have been described with 66% of cases having some extra-coronary abnormality in one series (Figure 6).<sup>76</sup> Fibromuscular dysplasia (FMD) is the commonest remote arteriopathy found in SCAD patients,<sup>77, 78</sup> although the reported incidence varies widely (25-86%).<sup>76, 79, 80</sup> Coronary abnormalities distinct from dissections have been reported in patients with peripheral arterial FMD, (including a subgroup with previous SCAD) and it has been suggested that SCAD may be a complication of coronary FMD.<sup>81</sup> Although the long term clinical significance of peripheral arterial FMD in SCAD-survivors remains to be determined, an assessment for renal, cervico-cephalic and iliac FMD is recommended in all patients with SCAD.<sup>78</sup> This can be by CT<sup>82</sup> or MRA<sup>78, 79</sup>. CT has the advantage of

higher spatial resolution and therefore potentially better sensitivity whilst MRA avoids the radiation dose inherent to CT which may be a particular concern in this younger population.<sup>78</sup>

### **Management after SCAD**

#### *Risk of recurrence*

Recurrent SCAD is well recognised. Reports incidences range from 5 to 19% of cases,<sup>4-8, 26</sup> although repeat events may be over-estimated in registries reliant on self or even clinician referral. Prospective series are underway to assess this further. Recurrent events may occur in the same or a different (Figure 3) artery.

#### *Recurrent (including cyclical) chest pain*

Many patients continue to experience episodes of significant chest pain long after healing of the primary lesion.<sup>83</sup> In some pre-menopausal cases pains may be cyclical, usually occurring premenstrually. Anecdotally these chest pains respond to conventional treatments for coronary spasm (i.e. cessation of  $\beta$ -blockers and initiation of high dose vasodilators such as Diltiazem). Cyclical cases respond well to implantation of a Mirena<sup>®</sup> (Bayer, Whippany, NJ.) coil.

#### *Rehabilitation and exercise risk*

In general and according to guidelines for post MI patients, cardiac rehabilitation and a return to full activity is recommended and has been validated as safe for SCAD-survivors.<sup>84, 85</sup> Isometric or extreme exercise is not recommended.

#### *Contraception after SCAD*

The predilection of SCAD for female patients and the association with pregnancy and the peri-partum period strongly suggests a role for sex hormones in the pathogenesis. This has led to some concerns over the safety of hormonal contraceptives in women post-SCAD.<sup>1</sup> Clearly barrier methods are safe and an intrauterine contraceptive either hormone free or with local hormone delivery may be useful in some cases (e.g. for cyclical chest pain or menorrhagia).

#### *Pregnancy after SCAD*

Data from one small series has reported nine pregnancies in SCAD-survivors with one recurrence occurring in a patients whose first event was not peri-partum.<sup>86</sup> The degree of left ventricular impairment post-SCAD will also contribute independently to the risk. Pregnancies which do occur should be deemed high-risk and managed by an appropriate multi-disciplinary team.

### **Key points**

- SCAD is an under-diagnosed cause of non-atherosclerotic acute coronary syndromes
- Peripartum cases account for <10% of SCAD patients
- There is frequently not a visible dissection flap on coronary angiography
- A conservative approach to revascularisation is favoured where possible

### **References**

1. Tweet MS, Gulati R, Hayes SN. Spontaneous coronary artery dissection. *Current cardiology reports*. 2016;18:60
2. Vanzetto G, Berger-Coz E, Barone-Rochette G, Chavanon O, Bouvaist H, Hacini R, Blin D, Machecourt J. Prevalence, therapeutic management and medium-term prognosis of spontaneous coronary artery dissection: Results from a database of 11,605 patients. *European*

- journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery.* 2009;35:250-254
3. Mortensen KH, Thuesen L, Kristensen IB, Christiansen EH. Spontaneous coronary artery dissection: A western denmark heart registry study. *Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions.* 2009;74:710-717
4. Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, Gersh BJ, Khambatta S, Best PJ, Rihal CS, Gulati R. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation.* 2012;126:579-588
5. Saw J, Aymong E, Sedlak T, Buller CE, Starovoytov A, Ricci D, Robinson S, Vuurmans T, Gao M, Humphries K, Mancini GB. Spontaneous coronary artery dissection: Association with predisposing arteriopathies and precipitating stressors and cardiovascular outcomes. *Circulation. Cardiovascular interventions.* 2014;7:645-655
6. Lettieri C, Zavalloni D, Rossini R, Morici N, Etti F, Leonzi O, Latib A, Ferlini M, Trabattini D, Colombo P, Galli M, Tarantini G, Napodano M, Piccaluga E, Passamonti E, Sganzerla P, Ielasi A, Coccato M, Martinoni A, Musumeci G, Zanini R, Castiglioni B. Management and long-term prognosis of spontaneous coronary artery dissection. *Am J Cardiol.* 2015;116:66-73
7. Rogowski S, Maeder MT, Weilenmann D, Haager PK, Ammann P, Rohner F, Joerg L, Rickli H. Spontaneous coronary artery dissection: Angiographic follow-up and long-term clinical outcome in a predominantly medically treated population. *Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions.* 2015
8. Nakashima T, Noguchi T, Haruta S, Yamamoto Y, Oshima S, Nakao K, Taniguchi Y, Yamaguchi J, Tsuchihashi K, Seki A, Kawasaki T, Uchida T, Omura N, Kikuchi M, Kimura K, Ogawa H, Miyazaki S, Yasuda S. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: A report from the angina pectoris-myocardial infarction multicenter investigators in japan. *International journal of cardiology.* 2016;207:341-348
9. Saw J, Aymong E, Mancini GB, Sedlak T, Starovoytov A, Ricci D. Nonatherosclerotic coronary artery disease in young women. *The Canadian journal of cardiology.* 2014;30:814-819
10. Elkayam U, Jalnapurkar S, Barakkat MN, Khatri N, Kealey AJ, Mehra A, Roth A. Pregnancy-associated acute myocardial infarction: A review of contemporary experience in 150 cases between 2006 and 2011. *Circulation.* 2014;129:1695-1702
11. Faden MS, Bottega N, Benjamin A, Brown RN. A nationwide evaluation of spontaneous coronary artery dissection in pregnancy and the puerperium. *Heart.* 2016
12. Regitz-Zagrosek V, Jaguszewska K, Preis K. Pregnancy-related spontaneous coronary artery dissection. *Eur Heart J.* 2015;36:2273-2274
13. Vijayaraghavan R, Verma S, Gupta N, Saw J. Pregnancy-related spontaneous coronary artery dissection. *Circulation.* 2014;130:1915-1920
14. Sivam S, Yozghatlian V, Dentice R, McGrady M, Moriarty C, Di Michiel J, Bye PT, Rees D. Spontaneous coronary artery dissection associated with coughing. *Journal of cystic fibrosis : official journal of the European Cystic Fibrosis Society.* 2014;13:235-237
15. Velusamy M, Fisherkeller M, Keenan ME, Kiernan FJ, Fram DB. Spontaneous coronary artery dissection in a young woman precipitated by retching. *The Journal of invasive cardiology.* 2002;14:198-201
16. Yiangou K, Papadopoulos K, Azina C. Heavy lifting causing spontaneous coronary artery dissection with anterior myocardial infarction in a 54-year-old woman. *Texas Heart Institute journal.* 2016;43:189-191
17. Balakrishnan K, Scott P, Oliver L. A confluence of circumstances: A case of ivf, extreme exercise and spontaneous coronary artery dissection. *International journal of cardiology.* 2016;203:76-77



18. El-Sherief K, Rashidian A, Srikanth S. Spontaneous coronary artery dissection after intense weightlifting ucsf fresno department of cardiology. *Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions*. 2011;78:223-227
19. Ellis CJ, Haywood GA, Monro JL. Spontaneous coronary artery dissection in a young woman resulting from an intense gymnasium "work-out". *International journal of cardiology*. 1994;47:193-194
20. Fahmy P, Prakash R, Starovoytov A, Boone R, Saw J. Pre-disposing and precipitating factors in men with spontaneous coronary artery dissection. *JACC. Cardiovascular interventions*. 2016;9:866-868
21. Katikaneni PK, Akkus NI, Tandon N, Modi K. Cocaine-induced postpartum coronary artery dissection: A case report and 80-year review of literature. *The Journal of invasive cardiology*. 2013;25:E163-166
22. Wickremaarachchi C, Olinga J, Ooi SY, Cranney G. Complete angiographic resolution of cocaine induced coronary artery dissection within eight days without coronary stenting--a case report. *Heart, lung & circulation*. 2016;25:e24-28
23. Ijsselmuiden A, Verheye S. Cocaine-induced coronary artery dissection. *JACC. Cardiovascular interventions*. 2009;2:1031
24. Afzal AM, Sarmast SA, Weber NA, Schussler JM. Spontaneous coronary artery dissection in a 22-year-old man on lisdexamfetamine. *Proceedings*. 2015;28:367-368
25. Kanwar M, Gill N. Spontaneous multivessel coronary artery dissection. *The Journal of invasive cardiology*. 2010;22:E5-6
26. Eleid MF, Guddeti RR, Tweet MS, Lerman A, Singh M, Best PJ, Vrtiska TJ, Prasad M, Rihal CS, Hayes SN, Gulati R. Coronary artery tortuosity in spontaneous coronary artery dissection: Angiographic characteristics and clinical implications. *Circulation. Cardiovascular interventions*. 2014;7:656-662
27. Sato C, Wakabayashi K, Suzuki H. Natural course of isolated spontaneous coronary artery dissection in marfan syndrome. *International journal of cardiology*. 2014;177:20-22
28. Nakamura M, Yajima J, Oikawa Y, Ogasawara K, Uejima T, Abe K, Aizawa T. Vascular ehlers-danlos syndrome--all three coronary artery spontaneous dissections. *Journal of cardiology*. 2009;53:458-462
29. Fattori R, Sangiorgio P, Mariucci E, Ritelli M, Wischmeijer A, Greco C, Colombi M. Spontaneous coronary artery dissection in a young woman with loeys-dietz syndrome. *American journal of medical genetics. Part A*. 2012;158A:1216-1218
30. Grover P, Fitzgibbons TP. Spontaneous coronary artery dissection in a patient with autosomal dominant polycystic kidney disease: A case report. *Journal of medical case reports*. 2016;10:62
31. Klingenberg-Salachova F, Limburg S, Boereboom F. Spontaneous coronary artery dissection in polycystic kidney disease. *Clinical kidney journal*. 2012;5:44-46
32. Basile C, Lucarelli K, Langialonga T. Spontaneous coronary artery dissection: One more extrarenal manifestation of autosomal dominant polycystic kidney disease? *Journal of nephrology*. 2009;22:414-416
33. Itty CT, Farshid A, Talaulikar G. Spontaneous coronary artery dissection in a woman with polycystic kidney disease. *American journal of kidney diseases : the official journal of the National Kidney Foundation*. 2009;53:518-521
34. Henkin S, Negrotto SM, Tweet MS, Kirmani S, Deyle DR, Gulati R, Olson TM, Hayes SN. Spontaneous coronary artery dissection and its association with heritable connective tissue disorders. *Heart*. 2016;102:876-881
35. Buccheri D, Piraino D, Andolina G. Behcet disease and spontaneous coronary artery dissection: The chicken or the egg? *International journal of cardiology*. 2016;215:504-505
36. Reddy S, Vaid T, Ganiga Sanjeeva NC, Shetty RK. Spontaneous coronary artery dissection as the first presentation of systemic lupus erythematosus. *BMJ case reports*. 2016;2016

37. Rekik S, Lanfranchi P, Jacq L, Bernasconi F. Spontaneous coronary artery dissection in a 35 year-old woman with systemic lupus erythematosus successfully treated by angioplasty. *Heart, lung & circulation*. 2013;22:955-958
38. Nisar MK, Mya T. Spontaneous coronary artery dissection in the context of positive anticardiolipin antibodies and clinically undiagnosed systemic lupus erythematosus. *Lupus*. 2011;20:1436-1438
39. Kothari D, Ruygrok P, Gentles T, Occleshaw C. Spontaneous coronary artery dissection in an adolescent man with systemic lupus erythematosus. *Internal medicine journal*. 2007;37:342-343
40. Sharma AK, Farb A, Maniar P, Ajani AE, Castagna M, Virmani R, Suddath W, Lindsay J. Spontaneous coronary artery dissection in a patient with systemic lupus erythematosus. *Hawaii medical journal*. 2003;62:248-253
41. Aldoboni AH, Hamza EA, Majdi K, Ngibzadhe M, Palasaidi S, Moayed DA. Spontaneous dissection of coronary artery treated by primary stenting as the first presentation of systemic lupus erythematosus. *The Journal of invasive cardiology*. 2002;14:694-696
42. Canpolat U, Dural M, Atalar E. Acute inferior myocardial infarction in a young female patient with polyarteritis nodosa. *Herz*. 2012;37:461-463
43. Srinivas M, Basumani P, Muthusamy R, Wheeldon N. Active inflammatory bowel disease and coronary artery dissection. *Postgraduate medical journal*. 2005;81:68-70
44. Saw J, Mancini GB, Humphries KH. Contemporary review on spontaneous coronary artery dissection. *Journal of the American College of Cardiology*. 2016;68:297-312
45. Desai S, Sheppard MN. Sudden cardiac death: Look closely at the coronaries for spontaneous dissection which can be missed. A study of 9 cases. *The American journal of forensic medicine and pathology*. 2012;33:26-29
46. Hill SF, Sheppard MN. Non-atherosclerotic coronary artery disease associated with sudden cardiac death. *Heart*. 2010;96:1119-1125
47. Alfonso F, Paulo M, Lennie V, Dutary J, Bernardo E, Jimenez-Quevedo P, Gonzalo N, Escaned J, Banuelos C, Perez-Vizcayno MJ, Hernandez R, Macaya C. Spontaneous coronary artery dissection: Long-term follow-up of a large series of patients prospectively managed with a "conservative" therapeutic strategy. *JACC. Cardiovascular interventions*. 2012;5:1062-1070
48. Roura G, Ariza-Sole A, Rodriguez-Caballero IF, Gomez-Lara J, Ferreira JL, Romaguera R, Teruel L, de Albert M, Gomez-Hospital JA, Cequier A. Noninvasive follow-up of patients with spontaneous coronary artery dissection with ct angiography. *JACC. Cardiovascular imaging*. 2016;9:896-897
49. Paulo M, Sandoval J, Lennie V, Dutary J, Medina M, Gonzalo N, Jimenez-Quevedo P, Escaned J, Banuelos C, Hernandez R, Macaya C, Alfonso F. Combined use of oct and ivus in spontaneous coronary artery dissection. *JACC. Cardiovascular imaging*. 2013;6:830-832
50. Poon K, Bell B, Raffel OC, Walters DL, Jang IK. Spontaneous coronary artery dissection: Utility of intravascular ultrasound and optical coherence tomography during percutaneous coronary intervention. *Circulation. Cardiovascular interventions*. 2011;4:e5-7
51. Alfonso F, Paulo M, Gonzalo N, Dutary J, Jimenez-Quevedo P, Lennie V, Escaned J, Banuelos C, Hernandez R, Macaya C. Diagnosis of spontaneous coronary artery dissection by optical coherence tomography. *Journal of the American College of Cardiology*. 2012;59:1073-1079
52. Haraki T, Uemura R, Masuda S, Lee T. Progressed multivessel spontaneous coronary artery dissection that naturally healed in a male patient with non-st segment elevation myocardial infarction. *Case reports in cardiology*. 2016;2016:4109496
53. Arnold JR, West NE, van Gaal WJ, Karamitsos TD, Banning AP. The role of intravascular ultrasound in the management of spontaneous coronary artery dissection. *Cardiovascular ultrasound*. 2008;6:24

54. Tweet MS, Eleid MF, Best PJ, Lennon RJ, Lerman A, Rihal CS, Holmes DR, Jr., Hayes SN, Gulati R. Spontaneous coronary artery dissection: Revascularization versus conservative therapy. *Circulation. Cardiovascular interventions*. 2014;7:777-786
55. Adlam D, Cuculi F, Lim C, Banning A. Management of spontaneous coronary artery dissection in the primary percutaneous coronary intervention era. *The Journal of invasive cardiology*. 2010;22:549-553
56. Watt J, Egred M, Khurana A, Bagnall AJ, Zaman AG. 1-year follow-up optical frequency domain imaging of multiple bioresorbable vascular scaffolds for the treatment of spontaneous coronary artery dissection. *JACC. Cardiovascular interventions*. 2016;9:389-391
57. Sengottuvelu G, Rajendran R. Full polymer jacketing for long-segment spontaneous coronary artery dissection using bioresorbable vascular scaffolds. *JACC. Cardiovascular interventions*. 2014;7:820-821
58. Ito T, Shintani Y, Ichihashi T, Fujita H, Ohte N. Non-atherosclerotic spontaneous coronary artery dissection revascularized by intravascular ultrasonography-guided fenestration with cutting balloon angioplasty. *Cardiovascular intervention and therapeutics*. 2016
59. Lempereur M, Fung A, Saw J. Stent mal-apposition with resorption of intramural hematoma with spontaneous coronary artery dissection. *Cardiovascular diagnosis and therapy*. 2015;5:323-329
60. Jovic Z, Obradovic S, Djenic N, Mladenovic Z, Djuric P, Spasic M, Tavicovski D. Does thrombolytic therapy harm or help in st elevation myocardial infarction (STEMI) caused by the spontaneous coronary dissection? *Vojnosanitetski pregled*. 2015;72:536-540
61. Andreou AY, Georgiou PA, Georgiou GM. Spontaneous coronary artery dissection: Report of two unsuspected cases initially treated with thrombolysis. *Experimental and clinical cardiology*. 2009;14:e89-92
62. Shamloo BK, Chintala RS, Nasur A, Ghazvini M, Shariat P, Diggs JA, Singh SN. Spontaneous coronary artery dissection: Aggressive vs. Conservative therapy. *The Journal of invasive cardiology*. 2010;22:222-228
63. Zupan I, Noc M, Trinkaus D, Popovic M. Double vessel extension of spontaneous left main coronary artery dissection in young women treated with thrombolytics. *Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions*. 2001;52:226-230
64. Goh AC, Lundstrom RJ. Spontaneous coronary artery dissection with cardiac tamponade. *Texas Heart Institute journal*. 2015;42:479-482
65. Authors/Task Force m, Windecker S, Kolh P, Alfonso F, Collet JP, Cremer J, Falk V, Filippatos G, Hamm C, Head SJ, Juni P, Kappetein AP, Kastrati A, Knuuti J, Landmesser U, Laufer G, Neumann FJ, Richter DJ, Schauerte P, Sousa Uva M, Stefanini GG, Taggart DP, Torracca L, Valgimigli M, Wijns W, Witkowski A. 2014 ESC/EACTS guidelines on myocardial revascularization: The task force on myocardial revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-thoracic Surgery (EACTS) developed with the special contribution of the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur Heart J*. 2014;35:2541-2619
66. Maas AH, Euler M, Bongers MY, Rolden HJ, Grutters JP, Ulrich L, Schenck-Gustafsson K. Practice points in gynecardiology: Abnormal uterine bleeding in premenopausal women taking oral anticoagulant or antiplatelet therapy. *Maturitas*. 2015;82:355-359
67. Task Force on the management of STEMI, Steg PG, James SK, Atar D, Badano LP, Blomstrom-Lundqvist C, Borger MA, Di Mario C, Dickstein K, Ducrocq G, Fernandez-Aviles F, Gershlick AH, Giannuzzi P, Halvorsen S, Huber K, Juni P, Kastrati A, Knuuti J, Lenzen MJ, Mahaffey KW, Valgimigli M, van 't Hof A, Widimsky P, Zahger D. ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J*. 2012;33:2569-2619

68. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, Bax JJ, Borger MA, Brotons C, Chew DP, Gencer B, Hasenfuss G, Kjeldsen K, Lancellotti P, Landmesser U, Mehilli J, Mukherjee D, Storey RF, Windecker S, Baumgartner H, Gaemperli O, Achenbach S, Agewall S, Badimon L, Baigent C, Bueno H, Bugiardini R, Carerj S, Casselman F, Cuisset T, Erol C, Fitzsimons D, Halle M, Hamm C, Hildick-Smith D, Huber K, Iliodromitis E, James S, Lewis BS, Lip GY, Piepoli MF, Richter D, Rosemann T, Sechtem U, Steg PG, Vrints C, Luis Zamorano J, Management of Acute Coronary Syndromes in Patients Presenting without Persistent STSEotESoC. 2015 esc guidelines for the management of acute coronary syndromes in patients presenting without persistent st-segment elevation: Task force for the management of acute coronary syndromes in patients presenting without persistent st-segment elevation of the european society of cardiology (esc). *Eur Heart J*. 2016;37:267-315
69. Knapp KE, Weis RA, Cubillo EI, Chapital AB, Ramakrishna H. Spontaneous, postpartum coronary artery dissection and cardiogenic shock with extracorporeal membrane oxygenation assisted recovery in a 30-year-old patient. *Case reports in cardiology*. 2016;2016:1048708
70. Torres-Ayala SC, Maldonado J, Bolton JS, Bhalla S. Coronary computed tomography angiography of spontaneous coronary artery dissection: A case report and review of the literature. *The American journal of case reports*. 2015;16:130-135
71. Tweet MS, Gulati R, Williamson EE, Vrtiska TJ, Hayes SN. Multimodality imaging for spontaneous coronary artery dissection in women. *JACC. Cardiovascular imaging*. 2016;9:436-450
72. Prakash R, Starovoytov A, Heydari M, Mancini GB, Saw J. Catheter-induced iatrogenic coronary artery dissection in patients with spontaneous coronary artery dissection. *JACC. Cardiovascular interventions*. 2016;9:1851-1853
73. Yip A, Saw J. Spontaneous coronary artery dissection-a review. *Cardiovascular diagnosis and therapy*. 2015;5:37-48
74. Bergen E, Huffer L, Peele M. Survival after spontaneous coronary artery dissection presenting with ventricular fibrillation arrest. *The Journal of invasive cardiology*. 2005;17:E4-6
75. Lempereur M, Grewal J, Saw J. Spontaneous coronary artery dissection associated with beta-hcg injections and fibromuscular dysplasia. *The Canadian journal of cardiology*. 2014;30:464 e461-463
76. Prasad M, Tweet MS, Hayes SN, Leng S, Liang JJ, Eleid MF, Gulati R, Vrtiska TJ. Prevalence of extracoronary vascular abnormalities and fibromuscular dysplasia in patients with spontaneous coronary artery dissection. *Am J Cardiol*. 2015;115:1672-1677
77. Persu A, Giavarini A, Touze E, Januszewicz A, Sapoval M, Azizi M, Barral X, Jeunemaitre X, Morganti A, Plouin PF, de Leeuw P, Hypertension ESHWG, the K. European consensus on the diagnosis and management of fibromuscular dysplasia. *Journal of hypertension*. 2014;32:1367-1378
78. Persu A, Van der Niepen P, Touze E, Gevaert S, Berra E, Mace P, Plouin PF, Jeunemaitre X. Revisiting fibromuscular dysplasia: Rationale of the european fibromuscular dysplasia initiative. *Hypertension*. 2016
79. Toggweiler S, Puck M, Thalhammer C, Manka R, Wyss M, Bilecen D, Corti R, Amann-Vesti BR, Luscher TF, Wyss CA. Associated vascular lesions in patients with spontaneous coronary artery dissection. *Swiss medical weekly*. 2012;142:w13538
80. Saw J, Ricci D, Starovoytov A, Fox R, Buller CE. Spontaneous coronary artery dissection: Prevalence of predisposing conditions including fibromuscular dysplasia in a tertiary center cohort. *JACC. Cardiovascular interventions*. 2013;6:44-52
81. Saw J, Bezerra H, Gornik HL, Machan L, Mancini GB. Angiographic and intracoronary manifestations of coronary fibromuscular dysplasia. *Circulation*. 2016;133:1548-1559
82. Liang JJ, Prasad M, Tweet MS, Hayes SN, Gulati R, Breen JF, Leng S, Vrtiska TJ. A novel application of ct angiography to detect extracoronary vascular abnormalities in patients with

- spontaneous coronary artery dissection. *Journal of cardiovascular computed tomography*. 2014;8:189-197
83. Bhatt DD, Kachru R, Gupta S, Kaul U. Recurrent chest pain after treatment of spontaneous coronary artery dissection: An enigma. *Indian heart journal*. 2015;67 Suppl 3:S18-20
84. Chou AY, Prakash R, Rajala J, Birnie T, Isserow S, Taylor CM, Ignaszewski A, Chan S, Starovoytov A, Saw J. The first dedicated cardiac rehabilitation program for patients with spontaneous coronary artery dissection: Description and initial results. *The Canadian journal of cardiology*. 2016;32:554-560
85. Krittanawong C, Tweet MS, Hayes SE, Bowman MJ, Gulati R, Squires RW, Hayes SN. Usefulness of cardiac rehabilitation after spontaneous coronary artery dissection. *Am J Cardiol*. 2016;117:1604-1609
86. Tweet MS, Hayes SN, Gulati R, Rose CH, Best PJ. Pregnancy after spontaneous coronary artery dissection: A case series. *Annals of internal medicine*. 2015;162:598-600

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### Statement

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### Figure legends

Figure 1: **Pathophysiology of SCAD.** A spontaneous haematoma forms in the outer media of a coronary artery forming a false lumen (FL). This then compresses the artery from the outside restricting blood flow in the true lumen (TL) with typical histological and angiographic appearances

Figure 2: **Angiographic appearances of SCAD.** Adapted from the Saw classification. A - Type 1 dissection with visible linear 'flap' or dual lumen in the mid left anterior descending coronary artery. B – Type 2a dissection with a long segment of narrowing in the right coronary artery with no visible 'flap' and distal reconstitution of normal vessel architecture. C – Type 2b dissection with a long segment of narrowing in the distal third of the left anterior descending coronary artery with no visible 'flap' and no distal reconstitution. C – Type 3 dissection in the mid left anterior descending coronary artery. Angiographically difficult to distinguish from an atherosclerotic stenosis. Type 4 dissection with the appearances of an abrupt vessel occlusion in the apical segment of the left anterior descending coronary artery with subsequent vessel healing and no cardiac embolic source

Figure 3: **Conservative management of SCAD.** Type 2a SCAD in the left anterior descending coronary artery (A) healed completely at repeat angiography (B) following presentation with a further acute coronary syndrome found to be due to a second Type 2b SCAD in the right coronary artery (C)

Figure 4: **Intracoronary imaging appearances of SCAD.** Typical optical coherence tomography image of SCAD with fenestration connecting true and false lumen (arrow) (A). True lumen 'floating' within the false lumen with thrombus evident in the false lumen (arrow) (B). Intramural haematoma with

no connection between true and false lumen limiting optical penetration but with the outer limit of the false lumen still identifiable (arrows) (C). IVUS image of healing proximal left anterior descending coronary dissection (D)

Figure 5: **Complications of SCAD revascularisation.** Left mainstem SCAD (A) treated by CABG with left internal mammary bypass graft to the left anterior descending artery. Follow up angiography demonstrating a healed dissection and graft failure (B). Type 2a SCAD with TIMI 3 flow in the left anterior descending coronary artery (C). Initial stenting (D – distal arrow) led to proximal migration of the haematoma. An attempt to seal the proximal extent of the dissection with a second stent (D - proximal arrow) led to further proximal extension of the dissection causing occlusion of the left anterior descending coronary artery (D) with dye hang up indicating dissection propagation into the left mainstem and circumflex (E)

Figure 6: **Extracoronary manifestations of SCAD.** Renal aneurysms (A), Iliofemoral fibromuscular dysplasia (B), a thoracic saccular aneurysm (B) and an iliac saccular aneurysm and gross tortuosity (D) found on screening in SCAD – survivors.

### MCQs

**1. Spontaneous coronary artery dissection most commonly presents in**

- A Men
- B Pregnant women
- C Patients with Marfan's syndrome
- D Young women

**2. Coronary angiography in patients with spontaneous coronary artery dissection**

- A. Usually shows short focal narrowings
- B. Most commonly shows disease in the proximal coronary arteries
- C. Often does not show a clear dual lumen or dissection 'flap'
- D. Is dangerous and should not be performed

**3. Revascularisation in patients with spontaneous coronary artery dissection**

- A Is mandatory even when there is TIMI 3 flow
- B CABG is preferable for Type 2b dissections
- C Bare metal stents are preferable because of the risk of bleeding with drug eluting stents
- D Is necessary to restore coronary perfusion in the presence of ongoing ischaemia or infarction

**4. Intracoronary imaging**

- A By OCT will show high resolution features such as fenestrations connecting the true and false lumens
- B Is not required in Type 3 SCAD
- C By IVUS is useful to image the distal extent of Type 2b SCAD
- D Is contraindicated

**5. Angiographic classification of spontaneous coronary artery dissection**

- A In Type 2a dissections the luminal restriction extends distally without reconstitution of normal vessel
- B In Type I SCAD a dissection flap is only detectable with intracoronary imaging
- C In Type 3 SCAD intracoronary imaging should be considered
- D In Type 2b SCAD a dissection flap is always visible with angiography

**6. Management after SCAD**

- A Lifelong dual antiplatelet therapy is mandatory in conservatively managed SCAD
- B Patients should be restricted from exercise and discouraged from cardiac rehabilitation
- C Patients can be reassured that recurrent SCAD is very rare
- D Patients should be offered imaging for remote arteriopathies

**Answers to MCQs**

**1. D**

SCAD most commonly afflicts young women with men accounting for <10% of prevalent cases. Historically often associated with pregnancy and connective tissue disorders, these cases make up a small proportion of the overall disease burden.

**2. C**

Angiographically often a classical dissection 'flap' or dual lumen is not present. Typically lesions are long and more commonly affect the mid to distal coronary territories (although all coronary segments can be affected).

**3. D**

Managed conservatively SCAD will usually heal completely. However where there is ongoing ischaemia and reduced flow in the infarct related artery revascularisation is necessary. Where PCI with stenting is required, drug eluting stents are preferred to bare metal stents. CABG is usually reserved for proximal disease where either PCI has failed or a PCI/conservative approach is felt to be high risk. Type 2b SCAD extends to the distal coronary extreme and is not suitable for surgical revascularisation

**4. A**

Intracoronary imaging should be considered where there is diagnostic uncertainty and to guide PCI where this is required. OCT is better at showing the high resolution features than IVUS although both can be helpful. Type 3 SCAD by definition cannot be distinguished from atherosclerotic disease without recourse to intracoronary imaging. Intracoronary imaging by any modality cannot demonstrate the distal extent of a Type 2b dissection as this extends to end of the visible coronary territory

**5. C**

Type 1 SCAD has a visible dissection flap or double lumen. Type 2a SCAD is typified by a long narrowing which tapers to a severe stenosis before reconstituting a normal distal vessel. Type 2b SCAD extends to the distal extent of the coronary without reconstitution. Type 3 SCAD cannot be distinguished from an atherosclerotic stenosis without intracoronary imaging, Type 4 SCAD is an abrupt distal vessel occlusion in a phenotypically typical patient with no cardiac source of embolus and subsequent vessel healing

### **6. D**

SCAD is frequently associated with extracoronary abnormalities and patients should be offered screening for this. Lifelong dual antiplatelet therapy is not indicated for SCAD. Discontinuation of monotherapy is controversial. Patients should be encouraged to return to full activities including cardiac rehabilitation but should avoid extreme and isometric exercise. SCAD recurrence is well recognised and appears to be more common than previously recognised.