ISSN: 2582-6751
Journal of Clinical Medical Research

ATHENAEUM SCIENTIFIC PUBLISHERS

Review Article

Spontaneous Coronary Artery Dissection: A Comprehensive Review

Olivia Kiffin[1*], E Hoey[2]

[1]University of Manchester Medical School, United Kingdom

[2]Honorary Senior Lecturer at University of Birmingham Medical School, United Kingdom

***Correspondence author:** Olivia Kiffin, University of Manchester Medical School, United Kingdom; Email: Livkendal04@gmail.com

Citation: Kiffin O, et al. Spontaneous Coronary Artery Dissection: A Comprehensive Review. Jour Clin Med Res. 2026;7(2):1-11.

https://doi.org/10.46889/JCMR.2026.7210

Received Date: 30-05-2026

Accepted Date: 22-06-2026

Published Date: 29-06-2026

Abstract

Spontaneous coronary artery dissection is a non-atherosclerotic cause of Acute Coronary Syndrome (ACS) that predominantly presents in middle-aged, female patients lacking traditional cardiovascular risk factors. It typically arises by one of two mechanisms: an intimal tear of epicardial arteries or by formation of an Intramural Haematoma (IMH). This can propagate to form a false lumen and subsequently result in luminal compression, leading to myocardial ischaemia and potentially infarction. The aetiology of SCAD is multifactorial and often associated with a variety of arteriopathies, such as Fibromuscular Dysplasia (FMD), hormonal factors, haemodynamic stress and pregnancy-associated SCAD.

Spontaneous coronary artery dissection accounts for a significant majority of acute coronary syndromes in this demographic; however, it is severely underappreciated and commonly misdiagnosed as anxiety, musculoskeletal pain or Gastro-oesophageal reflux disease due to the atypical profile of the patients. Acute management is mainly handled by secondary care teams, with conservative management favoured in most cases. Coronary angiography remains the diagnostic gold standard for Spontaneous Coronary Artery Dissection (SCAD).

General physicians play an essential role in managing the patient after diagnosis by prescribing medication regimens and lifestyle modifications, aiding admission to cardiac rehabilitation programmes and monitoring for potential recurrence. General physicians play an indispensable role in helping these typically healthy patients navigate this vulnerable period of their lives by providing crucial information and psychosocial support.

Keywords: Acute Coronary Syndrome; Fibromuscular Dysplasia; Spontaneous Coronary Artery Dissection

Introduction

Spontaneous Coronary Artery Dissection (SCAD) is an increasingly recognised cause of myocardial infarction, particularly in middle-aged women [1-3]. Previously presumed to be a 'rare' pregnancy-related cause of Acute Coronary Syndrome (ACS), increased clinical awareness and more frequent use of high-resolution intraluminal imaging techniques have led to wider recognition [1-3]. SCAD is the spontaneous dissection of the epicardial coronary arteries caused by non-atherosclerotic causes, excluding trauma-induced and iatrogenic origins [1,2]. SCAD is characterised by haematoma formation within the tunica media of the coronary vessels, following the formation of a false lumen, ultimately leading to occlusion of the true lumen. This limits luminal blood flow, leading to compromised perfusion and myocardial ischaemia [1-3]. This essay will outline the principal clinical features of SCAD, the demographics most affected by SCAD and discuss the challenges regarding underrecognition and misdiagnosis. It will also delve into the management of SCAD, emphasising the key role of General Physicians (GPs) who diligently work alongside patients, not only to provide acute, structured management for patients with recently diagnosed SCAD, but also to support patients with SCAD throughout the remainder of their lives. With a combination of continuity, empathy and a deep understanding of their patients' lives, GPs are able to deliver patient-centred, holistic care, profoundly influencing their patients' quality of life and long-term outcomes.

Epidemiology

SCAD was historically thought to be an extremely rare cause of Acute Coronary Syndrome (ACS) [1,2]. It was usually detected after death due to Myocardial Infarction (MI) and was therefore thought to be uniformly fatal [2]. Despite contemporary data beginning to challenge this belief, with more evidence proving that SCAD is more prevalent than initially thought, it is still underdiagnosed and its true incidence unknown. This is attributable to a variety of reasons, being low clinical suspicion of ACS due to the atypical profile of the patient, even with the presence of traditional cardiovascular symptoms [1,2]. SCAD displays a unique epidemiological pattern, with a predilection for young, seemingly healthy women, opposed to atherosclerotic cardiovascular diseases which are more prolific in men possessing multiple risk factors [1-3]. Roughly 90% of all SCAD cases are comprised of female patients, with the average age of onset being between 44-53 years old [2,3]. Conventional cardiovascular disease precipitants, such as hypertension, hyperlipidaemia and type 2 diabetes, are often not present in the cohort affected by SCAD, emphasizing the clinical distinction between SCAD and other atherosclerotic diseases [1,2]. No notable differences in SCAD prevalence between ethnicities have been discerned, although the marginally higher frequency of Caucasian patients affected by SCAD is likely due to sampling bias and cohort demographics [3]. Markedly, SCAD accounts for an estimated 35% of MIs in women <50 years of age, emphasising its clinical relevance in this demographic [3]. This highlights that it's paramount for first-contact healthcare professionals to be vigilant and have a high index of suspicion in otherwise healthy, young women presenting with ACS symptoms.

Contemporary evidence suggests that the prevalence of SCAD out of all ACS cases is 1.7-4%, much higher than the 0.2-1.1% that was previously concluded [3]. Although the prevalence of SCAD has increased due to greater awareness, the true burden is likely underestimated due to ongoing challenges with a lack of clinician familiarity with the disease and limitations on current coronary angiographic techniques [1,2,4]. SCAD lacks pathognomonic features, making it difficult to identify and distinguish from other vascular pathologies [1,2,4]. This can lead to SCAD being missed completely or potentially misinterpreted as coronary spasms or atherosclerotic tapering [1,2,4]. The current gold standard diagnostic technique for SCAD is coronary angiography, a technique that mainly focuses on imaging the lumen, it is unable to picture the coronary artery walls adequately [1,2,4]. Higher resolution imaging techniques such as Optical Coherence Tomography (OCT) and Intravascular Ultrasound (IVUS) are much better suited at detecting pathological features of SCAD, however are not routinely used as first-line investigation techniques [1,2].

SCAD was formerly thought to primarily occur in pregnant women, however <5-10% of SCAD cases actually occur due to these circumstances [3]. Pregnancy-associated and postpartum SCAD (P-SCAD) occurs most commonly in the third trimester of gestation or during the early postpartum period [3]. Although P-SCAD is the minority within the SCAD population, it has great clinical significance as it's the most common cause of MI in pregnant women, with a substantial 43% of MIs attributed to SCAD [1]. P-SCAD also accounts for a disproportionately high number of severe presentations of SCAD. A combination of the hormonal fluctuations, increased haemodynamic stress and altered connective tissue architecture is thought to be the mechanism for arterial wall vulnerability in pregnant women, which ultimately leads to increased sheer stress and intimal tearing [1,2].

SCAD can affect any coronary artery, but most commonly involves the LEFT ANTERIOR DESCENDING (LAD) artery [2,3]. The majority of dissections occur in the mid-distal segments, with proximal involvement seen in fewer than 10% of cases [2]. This reduces the likelihood of SCAD being identified via angiography as the smaller calibre of the vessels can make it much harder to identify subtle luminal compression [3]. Multivessel SCAD is reported in 9-23% of patients, supporting the concept of SCAD as a systemic arteriopathy, opposed to an isolated coronary event [2]. Familial clustering of SCAD is rare, accounting for only a small fraction of cases. This epidemiological pattern points to SCAD being polygenic as well as multifactorial, rather than following a Mendelian disease pattern. Most familial cases have been associated with an identifiable arteriopathy, particularly Fibromuscular Dysplasia (FMD) or Ehlers-Danlos syndrome. Both dominant and recessive patterns have been described but no single causative gene has been identified. Continuous research suggests SCAD is polygenic and the reason for familial SCAD is due to inherited vascular frailties. For this reason, screening family members of those with SCAD and an inherited arteriopathy is the main screening for SCAD, opposed to routine genetic testing for all patients [1,2].

Pathophysiology

Mechanisms of SCAD

The pathogenesis of SCAD is generally unclear however is thought to be multifactorial, with most patients having an underlying

arteriopathy, hormonal imbalances or triggered by environmental precipitants [1,2]. The mechanism underlying SCAD formation is hypothesised to be caused by via two distinct pathways. A potential cause of SCAD is the 'inside-out' mechanism, in which an intimal tear occurs in an epicardial artery allowing blood to cross the elastic lamina and accumulate in the tunica media of the artery wall [2,3]. It is uncertain if intimal tears are the initiating event of SCAD or instead the result of exceeding pressure of a false lumen which causes the subsequent rupture of the artery wall [1,2]. This is known as a 'reverse' intimal tear, the other potential causes could be iatrogenic due to intracoronary imaging equipment (Fig. 1) [2,3].

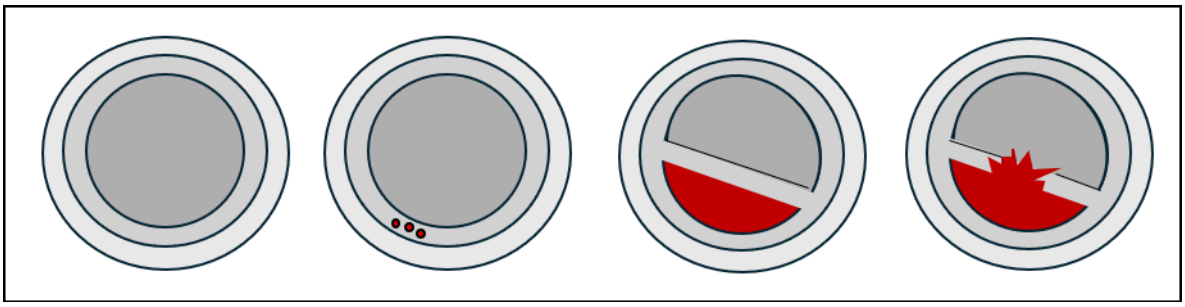

Figure 1: Depicts the process of a reverse intimal tear, with the initial rupture of Vaso vasorum leading to intramural haemorrhage in the tunica media. The haemorrhage expands over time, compressing the true lumen. Increasing pressure can cause secondary intimal tearing, allowing the false lumen and true lumen to come into contact.

Diagram Created by the Author Using Concepts from the ESC Position Paper on SCAD2

The other mechanism, named the 'outside-in' mechanism, proposes that a false lumen is formed by spontaneous haemorrhagic disruption of the vasa vasorum micro-vessels traversing the interior of the epicardial artery walls [1-3]. This causes intramural haematoma formation which can propagate false lumen formation. The extent of the haemorrhage directly affects coronary artery occlusion, with the larger the haematoma, the more obstructed the artery becomes, ultimately worsening ischaemia. Data from a recent study conclude that the obstruction and resulting ischaemia is solely attributed to the haematoma, not thrombi. Some other studies have affirmed the presence of thrombi; however are thought to have formed secondarily not the main driver of obstruction [1,2]. Optical Coherence Tomography (OCT) studies support the 'outside-in' pathophysiological theory, as imaging shows no communication between the true and false lumen [2,3].

Propagation of the dissection in SCAD behaves differently from dissections induced by atherosclerosis [2,3]. The artery walls in a SCAD patient are weakened due to predisposed arteriopathies, compromising the integrity of the artery wall, allowing extensive, axial spread [1,2]. The spread of the dissection can be extensive, with intra-coronary imaging deducing a mean dissection length of >45 mm^3. This opposes the spread of an atherosclerotic tear, in which the arterial media is usually scarred and atrophic, limiting the extent of propagation [2,3].

Histopathological Findings

SCAD is historically underdiagnosed, meaning post-mortem examination requires a high amount of suspicion and a thorough coronary histopathological examination [2]. SCAD has a predilection to the distal segments of epicardial arteries and autopsy shows the presence of periadventitial inflammation [2,3]. There is a mixed infiltrate with an eosinophilic predominance. This has been debated as some researchers think this may be a normal reaction caused by vascular injury, whereas some think it's pathognomonic. It is still debated if it's a cause for SCAD or a response. The presence of eosinophils can deduce if an intimal tear has been caused iatrogenically during post-mortem investigation or an intervention. The investigation into peripheral arteries is also required to uncover potential arteriopathies [2].

Arterial Wall Vulnerability

Fibromuscular Dysplasia

Fibromuscular Dysplasia (FMD) is one of the most frequent arteriopathies associated with SCAD patients, present in 17-86% of all SCAD patients [2]. The reason for this great variation in prevalence is due to differing imaging sensitivity, patient populations and the number of vascular beds investigated. FMD is a non-atherosclerotic, non-inflammatory vascular disease which causes

atypical growth of blood vessels, often manifesting as artery stenosis, dilation, dissection or increased tortuosity [1]. It causes a weakened arterial architecture, characterised by dysplasia and disorganisation of the smooth muscle cells comprising the tunica media [2]. The wall becomes thin and less elastic, ultimately leading to areas of weakness and subsequent dilation. This makes the artery more prone to micro tears and eventually dissection. Multifocal FMD is the most common variant of FMD seen in SCAD patients, being described as having a 'string-of-beads' pattern, with alternating areas of dilation and stenosis. Unifocal FMD is much more rarely seen with <10% of patients having this variation [2]. FMD mainly affects medium-sized arteries, including but not limited to renal, cervical-cephalic and visceral arteries [2]. In the US registry of FMD, 25.7% have had a known dissection of at least one vascular bed, with a further 10% of those patients having SCAD [2]. Although extracoronary FMD is highly prevalent in SCAD patients and some coronary arteries have close pathological features to those of coronary FMD, it remains premature to determine that SCAD is a complication of coronary FMD. This is due to a multitude of reasons, as most of the evidence linking these two diseases has been curated from studies using the same cohorts. The typical angiographic hallmarks of FMD are rarely seen in coronary arteries. Coronary dissections are also rare in FMD patients, with <3% of patients in the US registry of FMD having had a SCAD [2]. Lastly, an abundance of SCAD cases are idiopathic or connected to other arteriopathies, signifying that SCAD is likely caused by a plethora of contributing factors, as opposed to a singular pathology.

Female Sex Hormones and Pregnancy

P-SCAD is SCAD that occurs anywhere from 5 weeks of gestation to 6 weeks postpartum. It typically presents at a lower age with an average age of onset is between 33-36 years of age [2]. P-SCAD is correlated to significantly worse prognosis compared to non-pregnant women with SCAD. A multitude of cohort studies have demonstrated that P-SCAD cases are more likely to involve proximal vessels, affect multiple vessels, lead to worsened left ventricular function, as seen by a significantly reduced ejection fraction [2].

The majority of pregnant women do not have SCAD, with 1.81 cases per 100,000 pregnant women2, from which we can deduce that individual predisposing factors play a major role in SCAD propagation, by weakening the vessel walls. Hormonal changes brought about by pregnancy act on arterial walls, effectively weakening them and making them more susceptible to damage [1,2]. High progesterone concentrations impair collagen production and reduce the elasticity of the tunica media, whilst increased oestrogen levels induce a hypercoagulable state [1,3]. A weakened vascular wall in conjunction with a prothrombotic state is highly conducive to SCAD and intraluminal thrombus formation. Moreover, an increased circulatory volume increases haemodynamic stress on the already compromised vessels. The combination of these factors amplifies sheer stress and microstructural changes, bringing about the onset of SCAD [3].

Notably, multiparous women are more susceptible to SCAD due to the repeated hormonal fluctuation and haemodynamic stress during each pregnancy. This chronic repetitive impairment across multiple pregnancies progressively weakens the coronary vasculature predisposing multiparous women to SCAD2.

Mechanical and Emotional Stressors

Mechanical stressors can also play a role in precipitating SCAD, particularly in patients with a predisposing arteriopathy. Physical exertion, Valsalva manoeuvre inducing activities, heavy-lifting and extreme emotional distress, such as bereavement, can rapidly increase shear stress exerted on the already frail arteries, provoking intimal tearing or Vaso vasorum disruption [2]. Around 50% of SCAD events are induced via an identifiable mechanical or emotional stressor and 10-20% during the postpartum period, when the added haemodynamic stress and hormonal changes further weaken the tunica media [1-3]. These factors emphasise that SCAD is driven by the interplay between structural fragility and acute mechanical stress, explaining why it frequently arises in young, healthy patients during periods of heightened physiological or emotional strain (Fig. 2).

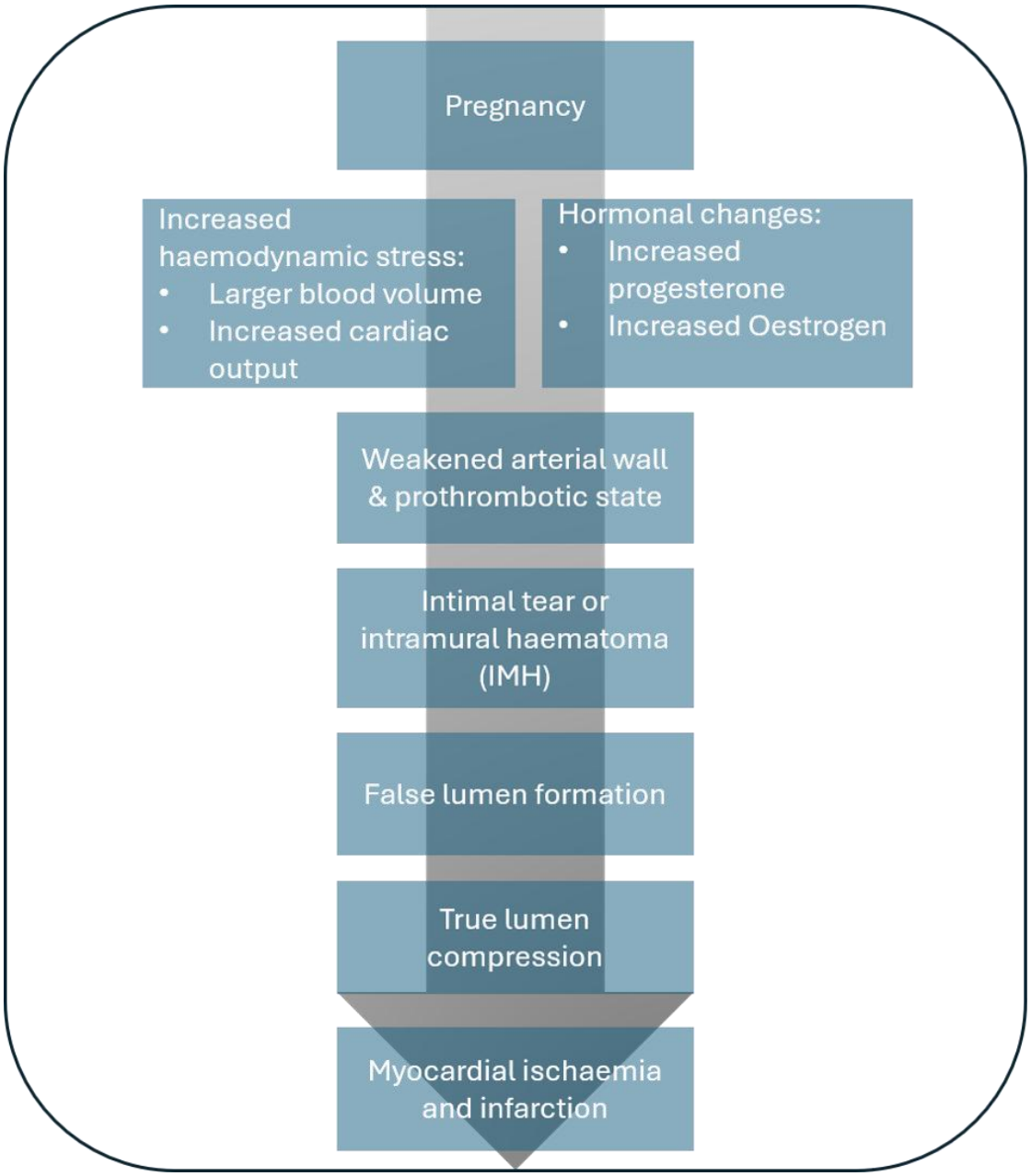

Figure 2: Depicts a visual pathway of the pathophysiology of pregnancy-associated SCAD (P-SCAD). Diagram created by author using concepts from the ESC position paper on SCAD [2].

Clinical Presentation

SCAD patients have a broad clinical presentation but most commonly manifest as ACS with elevated myocardial necrosis markers. Chest pain is the predominant symptom, occurring in 60-90% of patients [2]. Associated features such as dyspnoea, nausea and vomiting, diaphoresis or radiation to the arms or neck may accompany. Presenting symptoms are often synonymous with atherosclerotic ACS, with 26-87% presenting as STEMI and 13-69% with NSTEMI [2]. A minority of patients can develop ,ventricular arrhythmias, cardiogenic shock or on rare occasions, sudden cardiac death. Troponin concentration is highly variable and may look unremarkable initially, making SCAD patients appear deceptively well [2].

This creates a high likelihood of mislabelling symptoms as benign causes. Clinical suspicion must take precedence over ECG and troponin results, which early on, may look completely unremarkable. It is imperative that GPs maintain a low threshold for urgent referral, in addition to recognising red flags and understanding that SCAD occurs in atypically presenting patients. Improved awareness in primary care can reduce diagnostic delay, ultimately improving long-term outcomes (Fig. 3).

Presenting Symptom	Percentage (%) of Patients Presenting with Symptom
Chest pain	60-90
Radiation to arm	49.5
Radiation to neck	23.4
Nausea and vomiting	23.4
Diaphoresis	20.9
Dyspnoea	19.3
Back pain	12.2

Figure 3: Prevalence of the presenting symptoms in SCAD patients. Data extracted from ESC position paper on SCAD [2].

Diagnosis of SCAD

Diagnosis of SCAD can be challenging due to the lack of pathognomonic features. The differential diagnoses for SCAD are varied, some examples being atherosclerotic ACS, Takotsubo cardiomyopathy, coronary thromboembolism or coronary wall spasm [2]. There are no SCAD-specific blood biomarkers that can be used in diagnosis, however elevated troponin levels indicate myocardial ischaemia, in addition to abnormal ECG readings and clinical presentation, which would prompt further investigation [1,2]. Coronary angiography is typically the first-line assessment requested when SCAD is suspected, with intracoronary imaging used only when diagnostic uncertainty is present. Coronary angiography is the gold standard diagnostic technique for SCAD. It is widely available and is able to discern the pathognomonic features of SCAD, such as the presence of a double lumen or dye extravasation from the true lumen [2]. Despite being first-line, it does have various limitations. The pathognomonic appearance of SCAD is seen in only one subtype of SCAD, equating to 29.1% of all patients [5]. 1Relying solely on these features, >70% of SCAD patients would be missed [5]. In order to tackle this discrepancy, an official angiographic classification model has been developed, aiding diagnosis of SCAD with more subtle presentations (Fig. 4).

Type	Angiographic Appearance	Key Features	Percentage (%) of Total SCAD Population	Clinical Relevance
1	Numerous radiolucent lumens with contrast leaking into the arterial wall	Classical double lumen finding pathognomonic of SCAD	25-30	Most easily recognisable on coronary angiography, usually with no need for intracoronary imaging
2A	Long, diffuse smooth narrowing in mid-to-distal segments, with normal vessel calibre proximally and distally	Intramural haematoma causes long lengths of stenosis (>20mm)	60-70 (combined with 2B)	Mistaken for vasospasm therefore requires high amount of suspicion.
2B	Diffuse narrowing extending distally to the tip of the artery	No distal normalisation of artery therefore remains tapered	Included In type 2 prevalence	Typically affects smaller vessels which makes hard to detect.
3	Focal stenosis (<20mm) often mimics atherosclerotic plaques	Short areas of narrowing resembling an atherosclerotic plaque	5-10	Requires intracoronary imaging to confirm the presence of an intramural

Figure 4: SAW angiographic SCAD classification model. Data acquired from ESC position paper on SCAD [2].

Coronary angiography is described as a two-dimensional luminography, specialised to visualise the contrast-filled lumen, as opposed to the coronary artery wall [2]. Intracoronary imaging such as OCT and IVUS is much better suited to visualising pathologies associated with the vessel wall. They are used when diagnostic uncertainties are present, the vessel calibre is sufficient and when any ambiguity or subtle lesions, providing a much clearer image of the affected artery [1,2]. Intracoronary imaging is also used in revascularisation procedures, guiding stents into the correct position [2].

Despite the clear benefits to utilising Intracoronary imaging, there is a serious risk of worsening the dissection due to the inherently weakened state of the arteries. The dissection may be propagated either by a guide wire or an imaging probe. OCT carries additional risks during the contrast injection where the false lumen can be hydraulically extended [1,2]. Additional drawbacks are the cost, the lack of availability and the need for specialist expertise, where diagnostic accuracies are dependent on the skill of the doctor performing the procedure [1]. For these reasons, coronary angiography remains the gold standard [2].

Definitive diagnosis of SCAD relies entirely on coronary angiography, placing further emphasis on early recognition in non-specialist settings to prevent diagnostic delay. Patients with atypical or intermittent symptoms may initially present in a primary care setting, as opposed to emergency services, which requires a high index of suspicion. A thorough assessment of the patients clinical presentation alongside demographic factors, such as sex, age and the absence of cardiovascular risk factors may support early suspicion of SCAD and allows for the appropriate escalation. Misdiagnosis of SCAD patients is common, often getting mistaken for anxiety, musculoskeletal issues or Gastro-Oesophageal Reflux Disease (GORD). This can result in diagnostic delay, potentially worsening ischaemia and leading to worse outcomes. Misdiagnosis for other cardiovascular diseases can be just as detrimental, as the management of SCAD differs widely from that of atherosclerotic ACS.

Acute Management of SCAD

Conservative management is favoured in a large proportion of patients with SCAD as most dissections naturally stabilise and heal spontaneously, with only 3.3% of patients requiring in-hospital revascularization [2]. Methods traditionally used in ACS such as Percutaneous Coronary Intervention (PCI) or Coronary Artery Bypass Graft (CABG) are often contraindicated due to the fragility of the coronary artery walls and the potential to cause iatrogenic extension of the dissection. In haemodynamically stable patients with no evidence of ongoing myocardial ischaemia, conservative management is preferred [1,2].

Acute Medical Therapy

Subsequent to discharge, GPs play a pivotal role in ensuring the safe continuation and monitoring of medications initiated in hospital. Although the GP does not determine the primary regimen, they have the responsibility of checking patient adherence to medications, monitoring for bleeding and reviewing antiplatelet therapy [1,2]. Beta-blockers are commonly prescribed, reducing arterial shear stress and lowering blood pressure. These medications are especially beneficial in patients with hypertension [2]. The GPs follow-up appointments to allow monitoring of blood pressure, heart rate and side-effects, whilst reinforcing adherence at a time when patients often feel anxious about new medications and may discontinue them prematurely [3].

GPs also review the suitability of statins, which are not routinely indicated in SCAD unless dyslipidaemia is present [2]. GPs are able to have a conversation with their patient explaining that SCAD is not caused by hypercholesterolemia unlike atherosclerotic ACS, preventing long-term statin therapy when its not clinically indicated [1,2]. Patients with left-ventricular dysfunction may be prescribed ACE inhibitors or ARBs. GPs arrange continuous renal function assessments, closely monitor electrolyte levels and ensure follow-up imaging is carried out [1,2]. They also provide safe analgesia for persistent chest discomfort related to arterial healing and educate patients to differentiate expected post-SCAD pain from more concerning symptoms that warrant urgent review [3]. By delivering ongoing attentive medication management, GPs are the essential bridge between hospital-derived care and patients' individual long-term care [3].

Many SCAD patients with no previous comorbidities may struggle with the transition to long-term medication, especially if multiple drugs are introduced at once. GPs can help patients with these adjustments, offering guidance and simplifying regimens where possible, ultimately building patients confidence and making SCAD management more accessible for patients' [1,2].

Chronic Management of SCAD

GPs are uniquely positioned, able to have continuous contact with their patients. They have more insight into a patient's home life, support systems and lifestyle, which is highly useful regarding chronic management. It gives them the ability to cater to the specific, differing needs of each patient, offering the necessary support for long-term recovery [1,3].

Recurrence, Safety Netting and Follow Up

For many SCAD patients, recurrence is an anxiety-inducing, yet very feasible possibility. The rate of recurrence can be as high as 10-15% [2]. This possibility combined with the unpredictable nature of SCAD and limited long-term preventative strategies, does little to placate patients. Patients have shown to feel helpless and vulnerable to future episodes, causing feelings of uncertainty, anxiety and potentially depression [1]. A study has shown that rates of anxiety and depressive symptoms were 20.7% (GAD-7 ≥ 10) and 20.9% (PHQ-9 ≥ 10), respectively [6]. With many of these patients also possessing risk factors such as the lack of a close confidant, financial strain, being in unpaid employment and not knowing another SCAD patient [7]. GPs are in a close position with patients, their familiarity allowing patients to open up freely about their worries and mental health problems. The GP is able to refer patients to the appropriate services, such as talking therapies or Cognitive Behavioural Therapy (CBT), as well as offer reassurance [1,3].

Many patients experience recurrent chest pain, however not all pain is indicative of ischaemia. Dissections in themselves are painful and the healing process of the artery can take months [2,8]. GPs must be able to make this distinction between ischaemic pain and post-dissection pain, ultimately knowing when to reassure and redirect patients to support services and when it's necessary to escalate [1,8]. Mistaking non-ischaemic pain for ischaemic can be detrimental as unnecessary hospital referrals can potentially expose patients to further iatrogenic harm, particularly from intracoronary imaging [2]. It's paramount that GPs manage their patients' symptoms and give timely assessments. They must also work to inform their patients, helping them to differentiate between 'normal' pain and when they should be seen or when to seek urgent care1.

Cardiac Rehabilitation

Cardiac rehabilitation is vital in the process of SCAD recovery, offering psychological support, education about recurrence and exercise guidance [1,2]. Attending cardiac rehabilitation makes patients more informed about their condition, ultimately relieving stress and building patients' confidence [1]. Referral to Cardiac rehabilitation is enforced by the GP who oversees the patients is attendance [9]. Rehabilitation allows patients to talk to others with similar experiences and emotionally process the event they have endured [1]. GP endorsement is essential to legitimise rehabilitation and ensure ongoing engagement [3,9].

Lifestyle and Risk Modification

SCAD is not atherosclerotic in nature; however maintaining good health and exercising regularly is a necessary part of secondary preventative strategies [2]. Many patients may be uncertain of what activities they are able to engage in, in fear of invoking another event, such as enduring vigorous exercise [1]. The GP is able to utilise their knowledge and give tailored advice to patients on appropriate exercise regimens, avoiding heavy-lifting and sudden unaccustomed high-intensity workouts [1,2]. Providing guidance to patients aids to allow patients to live a healthy lifestyle while reducing strain and likelihood of recurrence.

In addition to exercise regulation, helping patients to manage any current comorbidities such as hyperlipidaemia, hypertension or smoking cessation either by prescribing medications, exploring lifestyle modifications or enrolling patients on a behaviour-change course [2]. Many SCAD patients also endure chronic stress and lack of sleep, ultimately exacerbating symptoms and causing psychological distress [3]. The personal relationship GPs have with their patients allows them to explore occupational stressors, caring responsibilities and other precipitants specific to that patient. They are able to refer patients to occupational and psychological support overall reducing psychological strain [3,9]. GPs are able to offer a holistic approach to their patients' treatment, not only reducing the risk of SCAD recurrence but also helping with stress management and improving overall quality of life [3].

Pregnancy and Contraception Counselling

The demographic for SCAD being young and 90% female, many SCAD patients will be of child-bearing age and may be considering future pregnancies. This is a complex issue as the additional vascular stress from hormonal fluctuations and increased haemodynamic volume puts additional stress on the already weakened arteries [1,2]. GPs are able to initiate an open

conversation with their patients, discussing pregnancy planning and balancing the patient's desire to be pregnant with potential complications. Informing the patients in a clear comprehensible manner allows the patient to make the most suitable decision for their health [1]. The GP is able to offer pre-conception guidance and coordinate referral to the appropriate multidisciplinary teams, involving cardiologists and obstetricians [2].

Contraception counselling is also offered, with GPs advocating for progesterone-only methods, opposed to oestrogen-containing contraception due to its prothrombotic and vascular effects [2,3]. SCAD patients who become pregnant are continuously monitored by the GP, measuring blood pressure, escalating care swiftly if chest pain or dyspnoea presents and ensuring meticulous postpartum follow-up [1]. This continuity throughout the stages of pregnancy and post-pregnancy, underlines the GPs role in supporting SCAD patients through complex, emotionally demanding decisions [3].

Psychosocial Impact of SCAD on Patient

The psychological burden of SCAD on patients is profound, mainly stemming from fear of recurrence, isolation and uncertainty surrounding the disease. Serious mental health disorders are prolific in SCAD patients, with at least 15% having clinically significant anxiety and depression [6,12,13]. Many patients feel worried about exercising or being alone in fear of another event. Even 5+ years prior to the SCAD event, 21% of patients have moderate to severe anxiety or depression emphasising the importance of ongoing psychological support [6,12]. The GP can facilitate this by providing continuous psychological screening and early intervention. Additionally, the lack of a close confidante is shown to worsen mental health outcomes by a 4-fold increase, stressing the importance of having a strong social support network [6]. GPs are able to identify SCAD patients facing social isolation and can signpost them to support groups or counselling.

SCAD is a rare disease, which can contribute to the isolation some SCAD patients may experience. An amalgamation of lack of support and understanding from others about their condition can ostracise patients and emphasise feelings of loneliness. 409 SCAD patients part of a qualitative study were asked what singular word they would use to describe their experience of SCAD. 30.24% of patients chose the word 'scary', 10.35% chose the word 'traumatic' [7]. This outlines that many patients are blindsided by this sudden alarming diagnosis and find it difficult to parse all the information and why this has happened to them [10]. Many patients have major uncertainty about the disease due to lack of information given to them, with 82% of patients reporting they 'received inadequate information' about the disease subsequent to their diagnosis [7]. GPs are able to work in close confidence with their patient to bridge this knowledge gap and have conversations with their patient in a comprehensible, jargon-free manner, creating a comfortable environment and allowing patients to ask as many questions as they wish. This allows GPs to provide an outlet for stressed patients, allowing them to emotionally navigate this difficult period of their lives. ultimately comforting their patients and helping them to feel less alone.

SCAD is also a highly traumatic event for patients, with evidence in some studies stating that 35% of patients meet the criteria for PTSD [1,11,13]. Many patients are often alarmed at this sudden, serious diagnosis, often amplified in patients with no previous major comorbidities or who thought they were otherwise 'healthy' [11,13]. The symptoms experienced such as chest pain, risk of recurrence and ongoing uncertainty can have a further psychological burden on patients [1,11-13]. GPs can offer psychological support and pick up on distress early, guiding patients to appropriate mental health resources. The GP's constant presence in their patients lives helps them to rebuild their confidence and navigate the emotional aftermath of SCAD [3]. The significant psychological burden of SCAD calls for additional support services and earlier interventions in future care pathways [13]. To minimise the proportion of patients with mental health issues attributed to SCAD, future management strategies must extend beyond the current cardiac care provided. The integration of routine mental health checkups and psychological support into long-term management would reduce persistent stress, promoting ongoing recovery and overall wellness [12].

Conclusion

Although awareness of SCAD is increasing, it remains an underdiagnosed ACS in young, female patients, lacking typical cardiovascular risk factors. GPs have an indispensable role throughout the patient's journey with the disease. As the first point of contact with some SCAD patients, GPs must be able to identify irregular presentations, initiate urgent referral, overall preventing diagnostic delay. In the post-acute phase, GPs provide the continuity of care required to manage SCAD patient's significant psychosocial burden. High rates of anxiety, depression, fear and trauma-related behaviours are evident and are compounded by various social factors. GP's role as a primary healthcare professional allows them to identify psychological

distress, address modifiable psychosocial factors and ensure attendance to cardiac rehabilitation. By doing so, GPs can significantly improve long-term outcomes. Greater GP awareness of SCAD is vital, not only enhancing early diagnosis but also ensuring patients receive holistic, sustained support for a lifelong condition.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Funding Statement

This research did not receive any specific grant from funding agencies in the public, commercial or non-profit sectors.

Acknowledgement

The authors have no acknowledgments to declare.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement

The project did not meet the definition of human subject research under the preview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement

Not applicable.

Authors' Contributions

All authors contributed equally to this paper.

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