



Spontaneous Coronary Artery Dissection: A Distinct Disease Repeatedly Managed as an Atherosclerotic One

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Abstract

Spontaneous coronary artery dissection (SCAD) causes up to 4% of all acute coronary syndromes overall and as much as a third of myocardial infarctions in women under 50, yet it is mechanistically unrelated to atherosclerosis: an epicardial coronary artery tears or forms an intramural haematoma without any underlying plaque. Roughly 70% of cases lack the angiographic hallmarks that make the disease easy to recognise, and case reports describe patients nearly discharged with a panic-attack diagnosis before troponin results revealed infarction. Once recognised, SCAD is usually managed conservatively -- roughly 86% of cases in a recent cohort, with only 2.3% of those needing subsequent invasive treatment -- because percutaneous intervention on a dissected, non-atherosclerotic artery carries its own procedural risk. Fibromuscular dysplasia, a systemic arteriopathy, co-occurs in a substantial but highly inconsistently reported fraction of SCAD patients across studies (21.8% to 86%), and short-term outcomes look favourable relative to conventional acute coronary syndrome, while three-year major adverse cardiac event rates and recurrence remain a real, if variably estimated, ongoing risk. This review sets out that evidence and closes with a complication distinct from the diagnostic one that opens it: SCAD survivors, overwhelmingly young and previously healthy women, show psychological symptom burden estimates that differ three- to four-fold between studies, and no standardised post-SCAD psychological screening pathway yet exists in routine cardiology follow-up.

Keywords: Spontaneous coronary artery dissection; Fibromuscular dysplasia; Myocardial infarction; Women's cardiovascular health; Post-traumatic stress

Introduction

Spontaneous coronary artery dissection (SCAD) is a tear in the wall of an epicardial coronary artery, or a spontaneous intramural haematoma within it, that obstructs blood flow without any underlying atherosclerotic plaque -- a mechanistically distinct disease that nonetheless presents with the same chest pain, ECG changes, and troponin rise as an atherosclerotic myocardial infarction (MI) [1,2]. That overlap

in presentation, combined with a patient population that looks nothing like the typical atherosclerotic-MI patient, is precisely what makes SCAD so easy to miss: it accounts for up to 4% of all acute coronary syndromes but as much as 35% of MI in women under 50, making it the single most common cause of MI in that population [5]. One case report describes a patient nearly discharged with a diagnosis of panic attack before troponin results revealed the true infarction [5] -- an illustration of a recognition problem this review returns to

directly.

This review sets out the evidence on SCAD's presentation, its association with an underlying systemic arteriopathy, its management and outcomes, and closes with a second, distinct complication: what recognising and treating the dissection itself still leaves unaddressed for the patients who survive it.

Material and Methods

This is a narrative review of the epidemiological, angiographic, management-outcome, and psychological-outcome literature on SCAD, identified through PubMed, cardiology society resources, and citation tracking of the sources above, without a pre-registered protocol or systematic search string. Where studies report differing estimates for the same statistic -- as recurs throughout this literature -- this review presents the range or the specific figures from each cited source rather than resolving them into a single pooled estimate; no numerical value in this review has been invented, estimated, or extrapolated beyond what the cited source itself reports.

Results

Most SCAD does not look like the textbook dissection, which is exactly the diagnostic problem

SCAD is angiographically classified into three types, and the distribution of these types matters clinically: Type 1, showing the classic multiple radiolucent lumen or contrast staining of the arterial wall, accounts for only 29.0% of cases; Type 2, a long, diffuse, smooth stenosis without a visible dissection plane, accounts for the majority at 60.2%; and Type 3, a focal, tubular stenosis that can closely mimic atherosclerotic disease, accounts for the remaining 10.8% [2]. Figure 1 sets out this distribution directly. Type 2 and Type 3 lesions are the ones a clinician reading an angiogram is most likely to mistake for atherosclerotic disease, coronary spasm, or a technical artefact [5], and roughly 70% of SCAD cases overall lack the classic angiographic hallmarks that make Type 1 straightforward to recognise, often requiring intravascular imaging such as optical coherence tomography or intravascular ultrasound to confirm the diagnosis [1]. The most common presenting symptom is unremarkable: chest discomfort, present in 91.5% of cases [2], indistinguishable on symptom grounds alone from any other acute coronary syndrome.

ANGIOGRAPHIC SCAD TYPE DISTRIBUTION:
TYPE 2 DOMINATES, NOT THE 'CLASSIC' TYPE 1

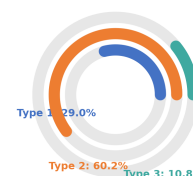


Figure 1: Angiographic classification of SCAD by type, showing Type 2 as the majority presentation rather than the more readily recognised Type 1 [2].

The scale of this recognition problem is visible in how quickly documented incidence has risen simply as awareness has caught up with the disease: a US hospital-discharge analysis found recognised SCAD incidence rising from 4.95 to 14.81 per million discharges between 2010 and 2017 [2], a change that far outpaces any plausible change in the underlying biological frequency of the disease over seven years. Figure 2 sets that trend out directly.

RECOGNISED SCAD INCIDENCE NEARLY TRIPLED IN THE US,
2010-2017 -- AWARENESS, NOT JUST DISEASE FREQUENCY

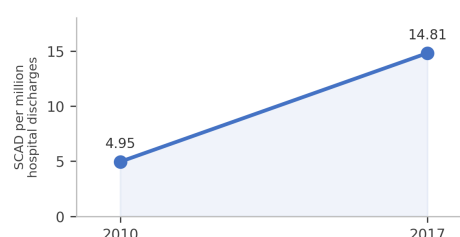


Figure 2: Recognised SCAD incidence per million US hospital discharges, 2010-2017, reflecting rising diagnostic recognition rather than a change in underlying disease frequency [2].

A systemic arteriopathy travels with SCAD in a substantial, but very inconsistently estimated, fraction of patients

Fibromuscular dysplasia (FMD), a non-atherosclerotic, non-inflammatory arteriopathy that can affect multiple vascular beds, is the condition most consistently linked to SCAD, and its presence carries prognostic weight: one study found FMD associated with a hazard ratio of 5.1 for SCAD recurrence, while another found a more modest relative risk of 2.02 (95% CI 1.03-3.94) [1,2]. What is far less consistent is how often FMD is actually found to co-occur with SCAD in the first place: reported prevalence ranges from 21.8% to as high as 86% across studies, with one series finding 62.7% overall and a marked sex difference, 52.7% in women versus 27.8% in men [1,2]. Figure 3 sets these disparate estimates out side by side on a single axis, since no single number in this literature can currently be presented as the prevalence of FMD in SCAD.

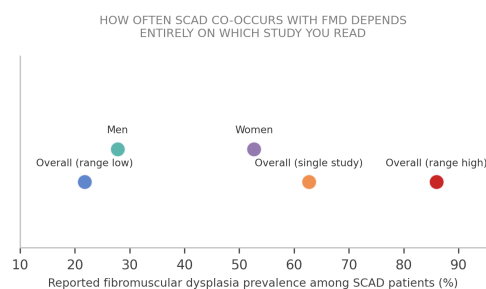


Figure 3: Reported fibromuscular dysplasia prevalence among SCAD patients across different studies and subgroups, illustrating the range rather than a single pooled estimate [1,2].

Conservative management is now the default, and short-term outcomes look favourable -- but the medium-term picture is more mixed

Because SCAD arteries are dissected and non-atherosclerotic, percutaneous coronary intervention (PCI) carries a distinct risk of propagating the dissection rather than fixing it, and conservative medical management has become the preferred default where the patient is haemodynamically stable: 86.4% of cases in one cohort were managed conservatively, and only 2.3% of those patients subsequently required invasive treatment [2], though other observational series report conservative management rates as low as 32% and PCI rates as high as 59%, reflecting differences in case mix and era [1]. Short-term outcomes for SCAD compare favourably with conventional acute coronary syndrome: in-hospital mortality of 2.5% versus 7.6% for non-SCAD ACS, and 30-day major adverse cardiac events (MACE) that improved from 8.8% in earlier cohorts to 3% in more recent ones [1]. The medium-term picture is less reassuring: 3-year MACE of 14% has been reported [1], and SCAD recurrence estimates themselves vary substantially -- one cohort reporting roughly 10.4% cumulative recurrence at 3 years, another reporting separate 3-year risks of 3.5% for dissection extension and 2.4% for outright recurrence [1,2]. Figure 4 sets out illustrative recurrence-free survival curves anchored to two of these differing cohort estimates, and Figure 5 places the mortality and MACE figures alongside each other directly. Table 1 summarises the type-specific and management-related figures from this section together.

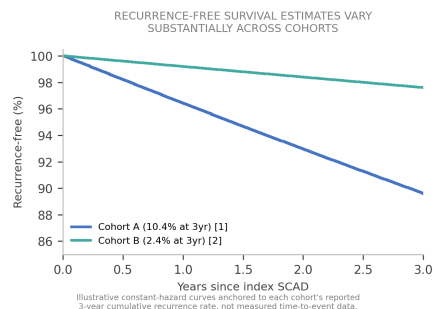


Figure 4: Illustrative recurrence-free survival curves over 3 years, anchored to two different cohorts' reported cumulative recurrence rates [1,2].

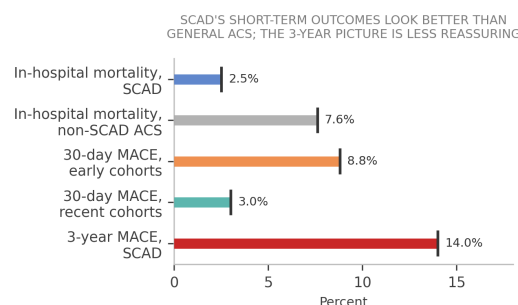


Figure 5: In-hospital mortality and major adverse cardiac event (MACE) rates for SCAD compared with non-SCAD acute coronary syndrome, short- and medium-term [1].

Metric	Estimate	Source
SCAD share of ACS	up to 4%	[1,5]
SCAD share of MI, women <50	24.2-35%	[1,5]
Conservative management	32-88% (86.4% recent cohort)	[1,2]
3-year MACE	14%	[1]

Table 1: Selected epidemiological and management figures for SCAD, presented as the range or specific figures reported by each cited source [1,2,5].

Recognising the dissection is not the end of the disease for the patient

The complication this review closes on concerns what happens after SCAD is correctly diagnosed and managed. SCAD survivors are overwhelmingly young, previously healthy women, and two studies measuring their psychological outcomes report strikingly different pictures. A clinic-based case-control study of 172 women found depression in 9% and anxiety in 12% -- rates the authors note were comparable to age-matched Dutch general-population women (12% and 7% respectively) and notably lower than typical ischaemic heart disease patients (29% and 39%) [4]. A separate patient-registry survey of 512 respondents (97.5% women) found depression in 32%, anxiety in 41%, and post-traumatic stress disorder symptoms in 28%, with lower psychological resilience associated with worse outcomes on every measure [3]. Figure 6 sets these two studies' estimates side by side; the three- to four-fold difference between them is not easily resolved by this review, and likely reflects real differences between a clinic-recruited cohort and a self-selected registry-survey sample, rather than either estimate being simply wrong.

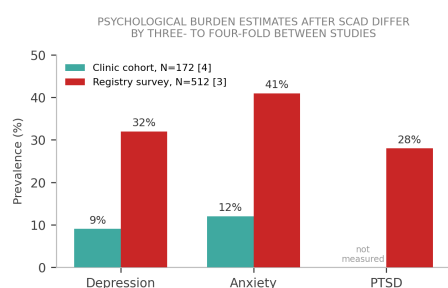


Figure 6: Depression, anxiety, and post-traumatic stress disorder symptom prevalence among SCAD survivors, from two studies using different sampling approaches [3,4].

Discussion

The evidence reviewed here supports treating SCAD as what it mechanistically is -- a distinct arteriopathy, not an atherosclerotic disease -- at every stage from diagnosis through management. The diagnostic problem this review opens with (a majority Type 2 presentation, 70% lacking classic angiographic hallmarks, symptoms indistinguishable from any other acute coronary syndrome) and the management principle it documents (conservative treatment preferred specifically because PCI on a non-atherosclerotic, already-dissected artery carries its own risk) both follow directly from that mechanistic distinction, and departing from either -- reading a Type 2 lesion as atherosclerotic disease, or reflexively stenting it -- risks real harm. The wide variation in FMD co-prevalence and recurrence-rate estimates across studies reviewed here likely reflects genuine differences in screening completeness (not every cohort systematically imaged for FMD) and case ascertainment, rather than the biology varying that much between populations; that heterogeneity is a limitation of the current evidence base, not a reason to discount FMD's relevance to individual patient risk assessment.

The psychological-outcome discrepancy this review closes on deserves equal weight to the better-established diagnostic and management questions, precisely because it currently receives far less clinical attention. A three- to four-fold difference in reported depression, anxiety, and PTSD prevalence between two reasonably conducted studies is not a reason to dismiss the question as unresolved noise; it is a reason to systematically screen for these outcomes in every SCAD survivor rather than relying on either study's estimate to decide who needs attention. No cardiology society guideline reviewed for this article specifies a standardised post-SCAD psychological screening protocol, in notable contrast to the increasingly detailed guidance on FMD screening, antiplatelet duration, and follow-up imaging that has developed for the disease's cardiac management. A population that is disproportionately young, previously healthy, and facing a sudden, poorly explained cardiac event -- often while managing pregnancy, young children, or a career -- is a population for whom this gap plausibly matters as much as any unresolved question about recurrence risk.

Study limitations

This is a narrative review, not a systematic review with a pre-registered protocol, and it deliberately presents conflicting estimates from different studies rather than resolving them into pooled figures, which limits this review's ability to state a single definitive number for FMD prevalence, recurrence risk, or psychological symptom burden. The psychological-outcome studies discussed differ in recruitment method (clinic-based versus registry-survey), response rate, and outcome-measurement instrument, which likely explains much of the discrepancy between them but cannot be fully disentangled from this review alone. Both psychological studies reviewed are drawn from predominantly Western, majority-female cohorts, and their applicability to male SCAD patients or non-Western populations is not established by the evidence reviewed here.

Conclusion

Spontaneous coronary artery dissection is not atherosclerotic disease, and every stage of its evidence base reviewed here -- its majority non-classic angiographic presentation, its association with an underlying systemic arteriopathy in a substantial but inconsistently measured fraction of patients, and its distinct, conservative-first management approach -- follows from that fact. Recognising SCAD as its own disease, rather than a variant of atherosclerotic MI, is the precondition for managing it correctly, and the clinical literature reviewed here shows real, if uneven, progress on that front over the past decade. What this review's evidence does not show is equivalent progress on what happens to the patient after the dissection itself is managed: psychological symptom burden that two studies estimate at three- to four-fold different rates, and no standardised screening pathway in routine follow-up to catch whichever estimate turns out to be closer to true for a given patient. Closing the diagnostic gap this review opens with was a genuine advance in cardiology practice; closing the psychological-follow-up gap it closes with has not yet had the same attention.

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