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Research paper

The presence of thrombus in spontaneous coronary artery dissection: A systematic review of autopsy findings☆

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ABSTRACT

Background: Spontaneous coronary artery dissection (SCAD) is an important cause of acute coronary syndrome in young women. There is no consensus on optimal treatment, though a conservative approach including anti-platelet agents is commonly used. We hypothesized that most cases of SCAD would not demonstrate true lumen thrombus in the dissected artery, suggesting that anti-platelet agents might not have a role in the treatment of SCAD.

Methods: We conducted a systematic review of the published literature through March 2022 to identify pathology images from individuals who died of SCAD. The images were independently reviewed by a pathologist to assess for the presence of thrombus and inflammatory cells.

Results: We identified 40 cases from 34 publications with available pathology images and found only one case of true lumen thrombus. Additionally, we found that 53% of cases involved eosinophilic inflammation.

Conclusion: The role of antiplatelet agents in the treatment of SCAD should be re-evaluated. Further studies are needed to better understand the significance and treatment implications of eosinophilic inflammation.

1. Introduction

Spontaneous coronary artery dissection (SCAD) is a cause of acute coronary syndrome and sudden cardiac death that disproportionately affects young women. In a consecutive series of angiograms in women under age 50, more than 20% of myocardial infarction (MI) cases were caused by SCAD [1]. Predisposing factors for SCAD include pregnancy, fibromuscular dysplasia (FMD), and connective tissue disorders [2]. While the pathogenesis of SCAD is not fully understood, it is thought to involve either spontaneous hemorrhage into the arterial media, resulting in an intramural hematoma which then compresses the true lumen, or an intimal tear, with intramural hematoma formation on that basis [2]. With increased availability of intracoronary imaging modalities, including intravascular ultrasound and optical coherence tomography (OCT), SCAD is more frequently recognized [2]. Despite increasing awareness and diagnosis of SCAD, there have been no randomized controlled trials and there is no consensus on optimal treatment. Our goal was to determine whether thrombus formation in the true lumen is part of the pathophysiology of fatal cases of SCAD, as anti-platelet agents are commonly used [3]. We hypothesized that the majority of SCAD cases would not demonstrate thrombus within the true lumen of the dissected artery, implying that antiplatelet agents might not have a role in the treatment of SCAD.

2. Methods

A review of electronic databases including MEDLINE, Embase, and Ovid was conducted from inception through March 2022. Search terms included "spontaneous coronary artery dissection" OR "coronary artery dissection" OR "SCAD" AND "autopsy." Citations were included if they provided pathology images of dissected coronary arteries acquired from autopsies of patients who died of SCAD. Images were independently reviewed by a pathologist who described the presence or absence of thrombus and inflammatory cells (A.V.R.). Thrombus was defined as microscopically having lines of Zahn, consisting of alternating light pale platelets and fibrin deposits with darker red cell rich layers. Eosinophilic

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inflammation was defined as identification of eosinophils in the vascular wall (intima, media or adventitia), given that eosinophils are not normally identified in the wall of blood vessels [4]. Thrombus in the false lumen was distinguished from that in the true lumen by location of thrombus relative to vascular wall layers; the true lumen is on the endothelial side of the vascular intima, internal to the vascular media. Cases with histological evidence of FMD were identified by fibrous thickening of the intima with focal destruction of the internal elastic lamina [5–7]. Location of the dissection was recorded. Cases were noted to have intimal tears if these were visible in provided histology sections or associated imaging (i.e. post-mortem coronary computed tomography). Cases were excluded if there was evidence of atherosclerotic disease, no convincing evidence of dissection in the available images, or if there was no clinical information provided. Additionally, when available, data regarding patient demographics, past medical history, vessel (s) involved, and treatments used were recorded.

Subgroup analysis was completed for pregnancy-associated SCAD, FMD, and presence of intimal tear using the Fisher exact test to compare categorical variables. Continuous variables were assessed using the Mann-Whitney *U* test. Cases were excluded from both the numerator and denominator of subgroup analyses if the necessary data for those cases was not available (i.e. inconclusive presence of thrombus). Significance level was set at a two-sided alpha level of 0.05. Statistical analysis was completed using Excel version 2201 (Microsoft, Redmond, WA).

3. Results

The initial search of databases yielded 567 citations, of which 36 contained autopsy images of sufficient quality for analysis (Fig. 1). One citation was excluded due to the presence of significant atherosclerotic disease seen in the autopsy image, one was excluded due to lack of convincing dissection in the provided pathology image, and one was excluded because no clinical information was provided with pathology images, making it unclear if the histology sections provided were from unique patients. Ultimately, 34 citations including 40 cases were included in the final analysis (Table 1). Of these 40 cases, 90% were female, with an average age at death of 43 years, 55% were dead on arrival to the hospital, and 15% received antithrombotic therapy. Of the patients who were alive on arrival to the hospital, 3 (17%) underwent PCI. The most common vessel involved was the left anterior descending (70%), with 38% of cases involving multiple vessels. Most decedents had no known medical history, though 5% had systemic vasculitis and 18% were pregnant or peri-partum. On independent review of the pathology images, all had intramural hematoma, one had evidence of thrombus in the true lumen, and 13% had evidence of thrombus in the false lumen. The majority of dissections occurred in the outer media or media-adventitia junction (63%) or elsewhere in the media (38%). There was eosinophilic inflammation of the affected artery or arteries in 53% of cases (Fig. 2). Intimal tears were identified in 6 of the 40 cases. Only one, different, case with intimal tear exhibited eosinophilic inflammation (Table 2).

Subgroup analysis comparing the 7 pregnant and peripartum cases (pSCAD) to non-pSCAD cases revealed that pSCAD patients were on average approximately 10 years younger than non-pSCAD cases (33.7 vs. 43.2 years, $p = 0.009$). Patients with pSCAD were less likely to have eosinophilic inflammation compared to non-pSCAD (14% vs. 61%, $p = 0.04$). There were no significant differences between groups in the presence of false lumen thrombus (0% with pSCAD vs. 16%, $p = 0.35$, Table 2).

A total of 4 (10%) patients were identified as having evidence of FMD on histology. These patients were less likely to have eosinophilic inflammation compared to those without FMD (0% vs. 58%, $p = 0.04$, Table 2).

4. Discussion

Our systematic review of 34 publications, including 40 fatal cases of SCAD, found only one instance of true lumen thrombus in the included cases. This study is significant as it is the largest to our knowledge assessing coronary artery pathology in patients with fatal SCAD, with

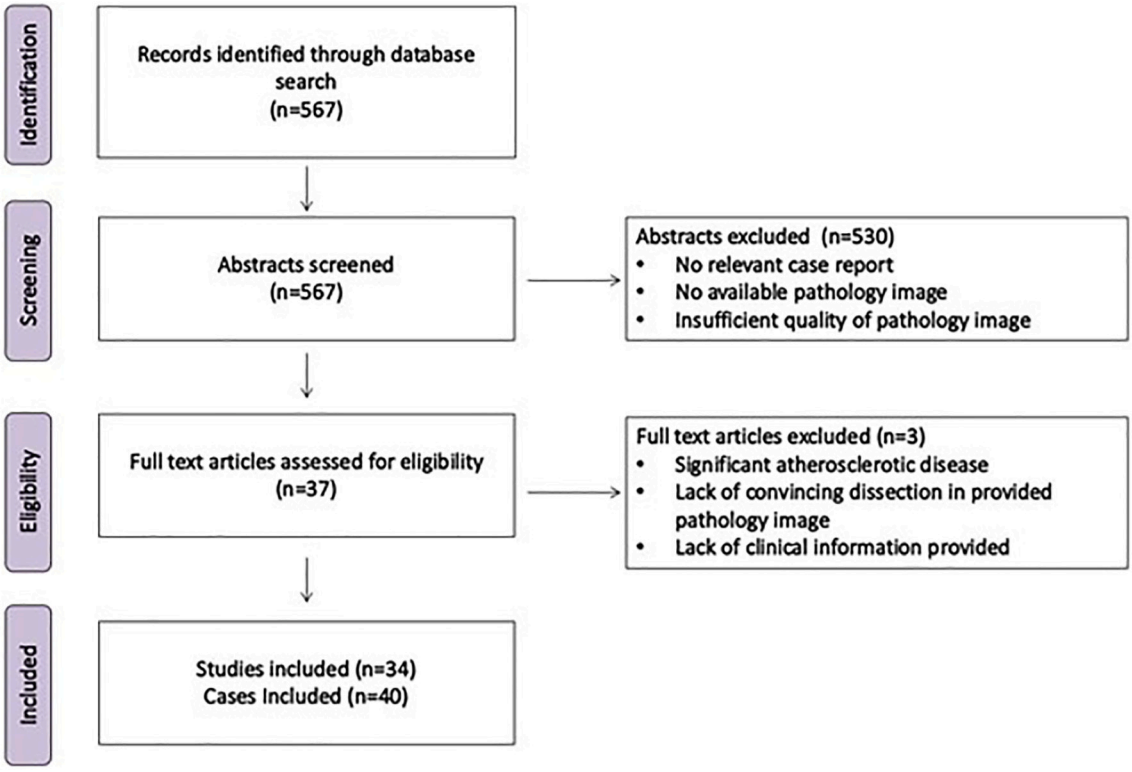

Fig. 1. Record selection for systematic review.

Table 1
Cases included in the systematic review.

Case #	Citation	Age/sex	Known medical history	Pregnant or peri-partum	Vessel(s) involved	Thrombus	Eosinophilic inflammation	Intimal tear	Location of dissection	Dead on arrival	Use of anti-thrombotics	FMD on histology
1	Pisano 1979 [15]	37/F	-	+	RCA	-	-	-	Outer media	+	n/a	-
2	Pisano 1979 [15]	49/F	Hypertension	-	LAD	-	+	-	Media	+	n/a	-
3	Lie 1987 [16]	24/F	-	-	RCA, LAD	False lumen	-	+	Media	-	-	+
4	McDonald 1989 [17]	41/F	Hypertension	-	LAD	-	+	-	Media-adventitia border	+	n/a	-
5	Hartman 1990 [18]	59/F	Hypertension	-	RCA	-	-	+	Media	-	-	-
6	Siegel 1994 [19]	36/F	-	-	LAD	-	+	-	Outer media	+	n/a	-
7	Basso 1996 [20]	43/F	-	-	LAD	-	+	-	Outer media	+	n/a	-
8	Conraads 1999 [21]	36/F	-	-	LAD, RCA	-	-	+	Outer media	-	+	-
9	Ropponen 1999 [7]	42/F	Psoriasis	-	LM, LAD	-	-	-	Media	-	Unknown	+
10	Zupan 2001 [22]	42/F	-	-	LM, LCx	-	-	-	Media-adventitia border	-	-	-
11	Salmo 2002 [23]	55/F	-	-	LAD	-	+	+	Outer media	+	n/a	-
12	Kavalar 2003 [24]	47/F	-	-	RCA	-	-	-	Media-adventitia border	+	n/a	-
13	Lepper 2005 [25]	43/F	Obesity, hypertension	-	LAD, LCx, RCA	-	+	-	Media-adventitia border	-	+	-
14	Muretto 2006 [26]	32/F	-	-	LAD	-	-	-	Media-adventitia border	-	-	-
15	Brodsky 2007 [5]	38/F	-	-	RCA	-	-	-	Media	-	-	+
16	DeGiorgio 2007 [27]	45/F	-	-	LM, LAD, LCx	-	-	-	Media-adventitia border	-	-	-
17	Lunenbourg 2008 [28]	55/F	-	-	LM, LAD, RCA	Inconclusive	+	-	Media-adventitia border	+	n/a	-
18	Wei 2008 [13]	49/M	High cholesterol, asthma, obesity	-	OM1	False lumen and true lumen	+	-	Media	+	n/a	-
19	Stoukas 2009 [29]	43/M	-	-	LAD	-	+	-	Media	+	n/a	-
20	Stoukas 2009 [29]	37/F	-	-	LAD	False lumen	+	-	Media	+	n/a	-
21	Pabla 2010 [30]	35/F	-	+	LAD, LCx	-	-	-	Media-adventitia border	-	-	-
22	Fengping 2011 [31]	53/F	-	-	LAD	-	+	-	Media-adventitia border	-	-	-
23	Omalu 2011 [32]	64/F	Hypertension	-	LAD	-	+	-	Media-adventitia border	+	n/a	-
24	Desai 2012 [33]	Unknown	Unknown	-	Unknown	-	+	-	Media	+	n/a	-

(*continued on next page*)

Table 1 (*continued*)

Case #	Citation	Age/sex	Known medical history	Pregnant or peri-partum	Vessel(s) involved	Thrombus	Eosinophilic inflammation	Intimal tear	Location of dissection	Dead on arrival	Use of anti-thrombotics	FMD on histology
25	D'Ovidio 2015 [34]	35/F	-	+	LAD, LCx	-	-	-	Media	-	+	-
26	Kanaroglou 2015 [35]	47/M	Cardiac sarcoid	-	PDA	-	-	-	Media	+	n/a	-
27	Makino 2015 [6]	28/F	-	-	LALD, LCx	False lumen	-	+	Media-adventitia border	+	n/a	+
28	Mandal 2015 [36]	41/F	-	-	LAD	-	+	-	Media-adventitia border	+	n/a	-
29	Mandal 2015 [36]	63/M	-	-	LAD, LCx	-	+	-	Media-adventitia border	+	n/a	-
30	Melez 2015 [37]	38/F	-	-	LAD	-	-	-	Media-adventitia border	+	n/a	-
31	Melez 2015 [37]	39/F	-	-	LAD	-	+	-	Media-adventitia border	+	n/a	-
32	Melez 2015 [37]	49/F	-	-	RCA	-	+	-	Media-adventitia border	+	n/a	-
33	Mori 2016 [38]	32/F	-	+	LAD, LCx, RCA	-	-	+	Media	-	Unknown	-
34	Mori 2016 [38]	40/F	-	+	LM, LAD	-	-	-	Media	-	Unknown	-
35	Bitting 2017 [39]	30/F	-	+	LAD, RCA	-	+	-	Media-adventitia border	+	n/a	-
36	Munguti 2017 [40]	62/F	-	-	PDA	False lumen	+	-	Media	-	-	-
37	Rose 2017 [41]	27/F	-	+	LM, LAD, LCx	-	-	-	Outer media	-	-	-
38	Moulson 2018 [42]	57/F	FMD	-	LAD	-	-	-	Media-adventitia border	-	Unknown	-
39	Izadpanah 2020 [43]	35/F	-	-	LM, LAD	-	+	-	Media-adventitia border	-	-	-
40	Fukuta 2021 [44]	45/F	-	-	LAD	-	+	-	Media	+	n/a	-

N/a = not applicable.
- = none or not present.
+ = present.
FMD = fibromuscular dysplasia.

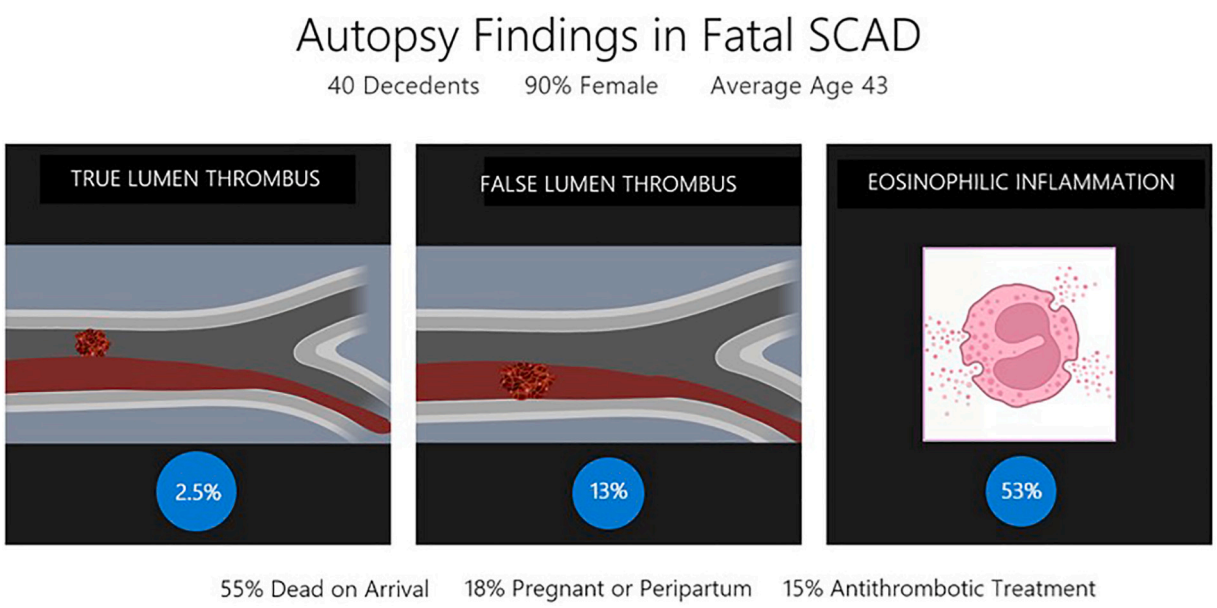

Fig. 2. Autopsy findings of fatal cases of SCAD.

Table 2
Subgroup analysis: Presence of eosinophilic inflammation and false lumen thrombus.

pSCAD			
	pSCAD $N = 7$ (%)	Non-pSCAD $N = 33$ (%)	P-value
Eosinophilic inflammation	1 (14%)	20 (61%)	0.04*
Thrombus	0 (0%)	5 (16%)	0.35

FMD			
	FMD $N = 4$ (%)	No report of FMD $N = 36$ (%)	P-value
Eosinophilic inflammation	0 (0%)	21 (58%)	0.04*
Thrombus	2 (50%)	3 (8.6%)	0.99

Intimal tear			
	Intimal tear $N = 6$ (%)	No report of intimal tear $N = 34$ (%)	P-value
Eosinophilic inflammation	1 (17%)	20 (59%)	0.07
Thrombus	2 (33%)	3 (9%)	0.98

pSCAD = pregnancy associated SCAD.
FMD = fibromuscular dysplasia.
* = $p<0.05$.

most existing literature consisting of case reports and small case series.

The population of patients with SCAD included in our study, predominantly women under age 50 without traditional cardiovascular risk factors, is consistent with that observed in the published literature [3,8]. Additionally, 18% of cases included in our study were pregnant or recently peri-partum, similar to published estimates of 5–17% of SCAD cases being associated with pregnancy [3]. Despite all cases in our study being fatal, the demographics of these cases were similar to those of the general SCAD population.

Our findings are also quite similar to those of the largest case series published to date of autopsy and OCT findings of SCAD, which included 36 autopsy-diagnosed cases of SCAD and 359 SCAD survivors [8]. Of the autopsy-diagnosed cases in that series, 81% were female, with an average age of 49, and 10% were post-partum. Interestingly, none of the fatal cases in their study involved multiple vessels, compared to 38% of cases in the present study. Additionally, 70% of cases in our study involved the LAD, compared to 33% of fatal cases and 65% of cases detected by OCT in the same series. While no cases in their study had evidence of FMD [8], 4 (10%) of cases in our present study did. The majority of dissections in the case series ($n = 31$, 86.1%) occurred near the outer media, which is also similar to our study ($n = 25$, 63%).

Another study using OCT images in 65 SCAD cases (68 vessels) found that lack of intimal flap was associated with increased false lumen pressure, resulting in compression of the true lumen [9]. Similar to the present study, the majority of cases in that study (63%) did not have a visible intimal tear. The vascular distribution of dissections in this study was also similar to that in our study, with 66% of cases involving the LAD [9]. Of note, this latter OCT study reports true lumen thrombus in 36% of fenestrated and 14% of non-fenestrated cases, as compared to 2.5% in our systematic review, but representative OCT images were not provided.

SCAD can be treated with a conservative approach or with coronary revascularization. A conservative approach is usually preferred, as percutaneous coronary intervention (PCI) is associated with high rates of technical failure and does not prevent long-term recurrence of SCAD [10]. Furthermore, conservative therapy is associated with fewer in-hospital adverse cardiac events and similar long-term outcomes as coronary revascularization [11]. The majority of cases in our systematic review (55%) died prior to receiving any medical intervention. Of the remaining cases, 3 (17%) underwent PCI and 6 (33%) received antithrombotic therapy.

Given that the pathogenesis of SCAD relates to intramural hematoma formation and not the atherothrombosis observed in typical MI, the use of antiplatelet agents in the treatment of SCAD is controversial. This is particularly important, as antiplatelet agents could theoretically be harmful in SCAD by allowing for further hemorrhage into the false lumen of the vessel [2] and preventing reabsorption of intramural hematoma [12]. This could lead to propagation of the dissection and extension of the intramural hematoma, resulting in worsening myocardial ischemia or infarction. However, if there is thrombus located in the true lumen, antiplatelet agents could theoretically be helpful in reducing ischemic complications, especially in the short-term. A comprehensive

review on the topic recommended dual antiplatelet therapy for 2–4 weeks following SCAD, followed by low-dose aspirin for 3–12 months [3]. However, a recent observational study found that SCAD patients treated with dual anti-platelet agents had increased 12-month incidence of major adverse cardiovascular events compared to those treated with a single anti-platelet agent [12].

Our study identified only one instance of true lumen thrombus, in a 49 year old male with dissection of an obtuse marginal branch of the left circumflex artery [13]. Given that only one of the included pathology images in the present study revealed true lumen thrombus, our findings indicate that the routine prolonged use of anti-platelet agents in the treatment of SCAD should be re-evaluated.

Eosinophilic inflammation was observed in the majority of cases, including those without known systemic inflammatory disorders. Some speculate that the inflammatory infiltrate is in response to the dissection, not the cause of the dissection [8]. However, these cases may represent isolated eosinophilic coronary periarteritis, which has been associated with SCAD [14]. The prevalence of eosinophilic inflammation in this systematic review is consistent with the existing literature, which demonstrates an association between eosinophilic inflammation and SCAD, but not with iatrogenic or traumatic coronary artery dissection [4]. Eosinophils release cytotoxic products, which can cause vessel wall injury, leading to dissection [4]. If eosinophils are part of the underlying pathophysiology of SCAD, immunosuppressive therapies could potentially be investigated for prevention of recurrent SCAD. Notably, patients with pSCAD or evidence of FMD were significantly less likely to have eosinophilic inflammation. This could suggest that perhaps these patients have a different underlying pathophysiology than other patients, but conclusions are limited by the small sample size.

This study has several limitations. Most notably, we were limited by the pathology sections included in the published literature. We could not examine consecutive sections to identify whether thrombus was located proximal or distal to the dissection. Additionally, findings are inherently biased as all included cases were fatal.

In conclusion, there was evidence of thrombus in the true lumen in only one of 40 cases of fatal SCAD for whom autopsy images were available in the published literature from inception to June 2021. While this study is inherently biased, as all included cases were fatal, the lack of true lumen thrombus in 39 of 40 cases suggests that the use of anti-platelet agents in the treatment of SCAD should be re-evaluated. Clinical trials are necessary to determine whether antiplatelet therapy is beneficial for patients with SCAD. Additionally, as the majority of cases in this analysis exhibited eosinophilic inflammation, further research is necessary to better understand the role of eosinophilic inflammation in the pathophysiology of SCAD, which could also have treatment implications.

Declaration of competing interest

Vita Jaspan and Amy Rapkiewicz have no competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. Harmony Reynolds receives non-financial support from Abbott Vascular, SIEMENS, and BioTelemetry Inc.

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