Translational Medicine
ISSN: 2161-1025
OPEN ACCESS Freely available online
Mini Review

Spontaneous Coronary Artery Dissection: Mini-Review

Marek M Rogowski[1,2,3]*, Christian Eichhorn[2], Kaffer Kara[3], Matthias Frick[2,4]

[1]Department of Cardiology Clinic, Agaplesion General Hospital, Hagen, Germany; [2]Department of Medical Science, Private University in the Principality of Liechtenstein, Triesen, Liechtenstein; [3]Department of Cardiology, University of Ruhr-Universitat Bochum, Bochum, Germany; [4]Department of Cardiology, Academic Teaching Hospital, Feldkirch, Austria

ABSTRACT

Spontaneous Coronary Artery Dissection (SCAD) is a rare form of Acute Coronary Syndrome (ACS) affecting predominantly young non-pregnant females. The primary pathology features a spontaneous tearing event in the tunica media of a coronary artery wall with subsequent formation of a true and false lumen. This usually leads to non-atherosclerotic myocardial infarction. The diagnosis must be confirmed by a diagnostic angiography, preferably with intracoronary imaging such as Optical Coherence Tomography (OCT). Whereas most cases may be treated conservatively, a coronary intervention remains a safe and effective option in occlusive dissections or when a considerable amount of myocardium in jeopardy can be seen. The possible interventional techniques involve a balloon angioplasty, cutting angioplasty, limited wiring and/or stenting. Due to low mortality and good prognosis, this condition may present the most benign type of ACS. Still, the recurrence rate is up to 10% within the first five years.

Keywords: Spontaneous coronary dissection; Coronary intervention; Intracoronary imaging; Angiography; Coronary artery

INTRODUCTION

SCAD is attributed to a non-traumatic hematoma within the tunica media of the coronary wall with the typical formation of a true and false lumen [1]. When the pressure within the false lumen exceeds diastolic blood pressure, the compression of the true lumen develops and leads to acute myocardial ischemia. SCAD was first reported by Pretty in 1931 and up to 1800 cases have been reported worldwide since then [2].

LITERATURE REVIEW

Pathophysiology

Even though the exact origin of the media hematoma is not known, two mechanisms have widely been suggested. The 'inside-out' hypothesis assumes that a primary injury takes place between endothelium and intima border and this allows blood from the true lumen to enter the space between the internal and external elastic membrane (and so to create the false lumen) [3].

The 'outside-in' idea, on the other hand, highlights a spontaneous hemorrhage within the vessel wall as a primary dissection trigger [4]. Some OCT studies showed that in many cases there was no initial connection between the true and false lumen. This, in turn may suggest that in vast majority of cases, SCADs arise from rupture of the pressurized false lumen into the true lumen rather than the other way round [5].

Epidemiology

The incidence of SCAD ranges from 0.8%-4% in the general population, affecting female gender in over 90% of cases and accounts for one third of ACS events in non-pregnant women younger than 50 [6]. Pregnancy-associated SCAD occurs in up to 10% of all SCAD cases. Whereas traditional risk factors of atherosclerosis are far less common, SCAD may be associated with chronic inflammation, female sex hormones, emotional stress and extensive isometric exercise [7].

Correspondence to: Dr. Marek M Rogowski, Department of Cardiology Clinic, Agaplesion General Hospital, Hagen, Germany, E-mail: marcorogowski@gmail.com

Received: 09-Nov-2023, Manuscript No. TMCR-23-27959; **Editor assigned:** 13-Nov-2023, Pre QC No. TMCR-23-27959 (PQ); **Reviewed:** 27-Nov-2023, QC No. TMCR-23-27959; **Revised:** 04-Dec-2023, Manuscript No. TMCR-23-27959 (R); **Published:** 11-Dec-2023, DOI: 10.35248/2161-1025.23.13.306

Citation: Rogowski MM, Eichhorn C, Kara k, Frick M (2023) Spontaneous Coronary Artery Dissection: Mini-Review. Trans Med.13:306.

Copyright: © 2023 Rogowski MM, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Diagnosis

The diagnosis of SCAD must be confirmed by an invasive angiography, usually supported by intracoronary imaging. As SCAD is associated with an increased risk of catheter-induced dissection (3.4% *vs.* 0.2% in the general population), the optimal co-axial technique and avoidance of aggressive catheter maneuvers are of utmost importance [5]. Non-selective injections allow the proper evaluation of ostial and proximal lesions prior to selective intubation. SCAD usually occurs in the mid-distal arteries, especially of the tortuous Left Anterior Descending Artery (LAD). The yip-saw angiographic classification is presented in Table 1 [1].

Class	% of cases	Description
Type 1	30 %	Dual lumen appearance due to contrast penetration of the false lumen.
Type 2 -2 a -2 b	50-70%	long smooth stenosis. -with recovery of a normal vessel diameter. -without recovery of a normal vessel architecture.
Type 3	2-4%	Shorter dissections with no possible distinction between SCAD and fibrotic atheroma without intracoronary imaging.
Type 4	1%	Any coronary occlusion.
Multi vessel	1%	More than one discontiguous segment.

Table 1: Angiographic yip-saw classification.

From the practical point of view, the only essential difference is the one between dissections with and without fenestrations between true and false lumen. The type 1 cases have a lower risk of Percutaneous Coronary Intervention (PCI) complications and better outcome [8].

SCAD: Differential angiographic diagnosis

Following conditions may mimic SCAD and should always be considered as potential differential diagnosis: It includes

- Contrast streaming like type 1, usually disappears after increasing the injection pressure/volume,
- Coronary vasospasm relieved by intracoronary administration of nitrates,
- Iatrogenic dissection, especially catheter-induced proximal dissection highly challenging to differentiate; previous symptoms and Electrocardiogram (ECG)/ Echocardiogram (Echo) results must be considered,
- Coronary embolism like type 4, very challenging, needs careful evaluation,
- Plaque rupture and thrombus generation,
- Takotsubo cardiomyopathy.

Intracoronary imaging

Due to its high spatial resolution and ability to visualize the lumen-intimal interface, OCT is usually recommended for SCAD, apart from the proximal type 1 dissection where some concern must be raised about the use of contrast injection due to possible hematoma extension [9]. The pathognomonic sign of a dissection (white-black-white) can be seen with IVUS in 40% of cases. In 60% of cases, however IVUS is not able to distinguish SCAD from a fibrotic plaque and brings no further diagnostic benefit [10].

The pros and cons of both Intravascular Ultrasound (IVUS) and OCT are presented in Table 2.

	Advantages	Disadvantages
IVUS	Greater depth of imaging, no contrast injection.	Lower spatial resolution.
OCT	Lower depth of imaging, contrast injection.	Higher spatial resolution.

Table 2: The pros and cons of both IVUS and OCT.

Management

Conservative approach: Most cases of SCAD have a positive outcome and heal spontaneously with a complete normalization of coronary arteries within six months. Multiple studies confirmed this observation (e.g. saw at, in 74 cases a follow-up angiography showed no dissection after 26 days or in a series by Rogowski et al., 29 out of 30 cases healed spontaneously after 6-months) [4,7]. A regular invasive follow-up is not recommended due to risk of iatrogenic dissection, contrast use and radiation exposure. In asymptomatic cases, the benefit of Computed Tomography Coronary Angiogram (CTCA) to confirm the healing process must be weighed against the X-radiation (X-ray) exposure.

PCI: Interventional treatment of atherosclerotic patients with ACS has demonstrated many advantages and especially in case of ST Elevation Myocardial Infarction (STEMI) it has been showed to improve patient's overall prognosis. In case of SCAD, however, this benefit may not be so visible. A PCI is necessary when there is a significant amount of myocardium in jeopardy, which usually assumes proximal and mid-vessel acute occlusions [1]. Contrary to treating an atherosclerotic lesion, the primary revascularization goal of SCAD-PCI is to restore the normal coronary flow (and not normal coronary anatomy) [10]. In case

of uncovering the whole dissection segment, for instance it must be assumed that provided Thrombolysis in Myocardial Infarction 3 (TIMI 3), coronary flow is the uncovered part heals over time, which was demonstrated in a study with 215 SCAD patients [11]. Furthermore, as PCI in SCAD is associated with a significantly increased complication risk, this should encourage a treating cardiologist to accept the TIMI 3 flow and deny prolonged interventions with aggressive and complex maneuvers. The possible interventional options are

- Limited wiring or Plain Old Balloon Angioplasty (POBA) only,
- Undersized stenting,
- Upstream and/or downstream stenting,
- Cutting balloon angioplasty.

No single strategy can be recommended due to missing high quality data [3]. It has been demonstrated that longer lengths of smaller caliber stents are usually necessary. Even though some stent malapposition was reported, no increased risk for stent thrombosis or in-stent restenosis was observed. Drug-Eluting Stents (DES) are usually recommended [12].

Coronary artery bypass graft surgery: In case of PCI failure or if conservative strategy is not possible, open surgery remains a potential option. This may be, however a highly challenging scenario in distal and extensive dissections, complicated by the fact that distinction between true and false lumen may not always be easy. Of note, due to high rates of spontaneous healing of SCAD, in the bypassed patients, high mid-term graft failure rates were observed [2].

Cardiogenic shock: Although highly uncommon, cardiogenic shock influences badly the overall prognosis. About 5% of SCAD patients suffered from cardiac arrest [1]. Any form of bridging assistance has been described: Intensive care unit, percutaneous mechanical support such as Intra-Aortic Balloon Pump (IABP), impella or Extracorporeal Membrane Oxygenation (ECMO).

Optimal medical treatment

In the acute setting, standard PCI anticoagulation with heparin should be used. As intraluminal thrombus formation is a very uncommon SCAD feature, the use of thrombolytics or glycoprotein IIb/IIIa inhibitors is not recommended [9]. In PCI patients treated with stenting, Dual Antiplatelet Therapy (DAPT) should follow according to the current guidelines. In patients treated conservatively, the proper use and duration of antiplatelet drugs is a matter of debate. Some clinicians favor a short DAPT therapy for 2-4 weeks, followed by a prolonged monotherapy with aspirin (up to 12 months) [6]. According to the patient's ejection fraction, home medication may include beta-blocker, angiotensin-converting enzyme inhibitor/Ataxia-Telangiectasia and Rad3 (ATR) and diuretics. The role of angiotensin receptor/neprilysin inhibitor and new generation Sodium-Glucose Linked Transporter-2 (SGLT2) inhibitors has not been addressed so far.

Prognosis

SCAD mortality is as low as 1.2% and the overall prognosis seems to be better than the general ACS population [1]. Still, Major Adverse Cardiovascular Events (MACE) rates are high (19.9% over the 3-year follow-up), mainly due to risk of recurrent SCAD [8].

DISCUSSION

Other vessels

Of note, due to certain genetic predisposition, a single cross sectional imaging study from brain to pelvis by Computed Tomography Angiography (CTA) or Magnetic Resonance Angiography (MRA) is recommended in all SCAD patients, extra-coronary vascular abnormalities are usually found in the cervical, renal and iliofemoral arteries [13]. Interestingly, the vascular events in the extra-coronary vasculature are extremely rare. The lesions, as demonstrated by CTA or MRA are usually stable and predisposed to conservative management (especially lifelong aspirin should be considered) [11].

Recurrent SCAD

Up to 10% of patients experience recurrent SCAD within the following five years [9]. A very late presentation (over 10 years) is uncommon, but the follow-up data are clearly missing. A recurrence usually affects a different coronary artery. In observational studies, some predictors were discovered, among them migraine, uncontrolled hypertension and non-use of beta-blockers. The prognosis remains good [1].

Post SCAD syndrome

In females with migraine and prior psychological and emotional stress, non-ischemic retrosternal pain with hospital admissions is common. In the past, vasospasm or microvascular dysfunction were suggested but the proper mechanism is not known. In most cases, treatment can take up to two years as there is no single medication available [8]. Possible options are progesterone-based contraceptives (in post-menstrual females), vasodilators (especially diltiazem) and sometimes ranolazine. Non-medical options such as cardiac rehabilitation or training program may also be helpful.

Life after SCAD

As most SCAD patients are young females, pre-conception counselling should be advised, with risk estimation on an individual basis. Based on current data, a recurrent SCAD during pregnancy occurs in 1:10 cases [3]. Pregnancy after SCAD is therefore neither absolutely contraindicated nor risk free.

The physical activity after SCAD has been discussed as well [6]. There are no data suggesting that exercise after SCAD increases the recurrence risk, but the role of isometric exercises is not fully understood. Therefore, a pragmatic approach with avoidance of isometric activities can be recommended [9].

CONCLUSION

SCAD is a rare form of ACS, affecting mainly young non-pregnant females presenting with chest pain but without traditional atherosclerotic risk factors. The condition has not yet been thoroughly examined in male patients. Even though most cases can be treated conservatively and the overall prognosis seems to be good, the high rates of major adverse cardiac events in the long term raise some concern. In SCAD-survivor's close follow-up is highly recommended as well as avoidance of extensive exercise and unplanned pregnancy.

FUNDING

To conduct this study, the author did not receive any special funding.

DISCLOSURE STATEMENT

The authors declare that they have no competing interests.

AVAILABILITY OF DATA AND MATERIAL

On special request, all the collected data can be available.

AUTHOR'S CONTRIBUTIONS

Marek M. Rogowski: Main conception of the paper, wrote the paper, performed the literature search.

Matthias Frick: Contributed to the main theoretical background, verified the literature search, supervised the project.

All authors discussed the results and contributed to the final manuscript.

REFERENCES

1. Alfonso F, Bastante T, Rivero F, Cuesta J, Benedicto A, Saw J, et al. Spontaneous coronary artery dissection: From diagnosis to management. Circ J. 2014;78(9):2099-2110.
2. Akhigbe EJ, Ezeh E, Mansoor K, Mader J, Okhumale PI, Lester M, et al. The self sabotaging vessel: A case report and literature review of spontaneous coronary artery dissection. Cureus. 2021;13(12):e20835.
3. Waterbury TM, Tweet MS, Hayes SN, Eleid MF, Bell MR, Lerman A, et al. Early natural history of spontaneous coronary artery dissection. Circ Cardiovasc Interv. 2018;11(9):e006772.
4. Yip A, Saw J. Spontaneous coronary artery dissection: A review. Cardiovasc Diagn Ther. 2015;5(1):37-48.
5. Kim ES. Spontaneous coronary-artery dissection. N Engl J Med. 2020;383(24):2358-2370.
6. Adlam D, Alfonso F, Maas A, Vrints C, Writing Committee. European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection. Eur Heart J. 2018;39(36):3353-3368.
7. Rogowski S, Maeder MT, Weilenmann D, Haager PK, Ammann P, Rohner F, et al. Spontaneous coronary artery dissection: Angiographic follow-up and long-term clinical outcome in a predominantly medically treated population. Catheter Cardiovasc Interv. 2017;89(1):59-68.
8. Motreff P, Malcles G, Combaret N, Barber-Chamoux N, Bouajila S, Pereira B, et al. How and when to suspect spontaneous coronary artery dissection: Novel insights from a single-centre series on prevalence and angiographic appearance. EuroIntervention. 2017;12(18):2236-2243.
9. Chien DV, Hai PD, Nhung LT, Son PT. Multiple spontaneous coronary artery dissections associated with intravenous daunorubicin treatment for acute myelocytic leukaemia: A case report. Eur Heart J Case Rep. 2020;5(1):ytaa427.
10. Nishiguchi T, Tanaka A, Ozaki Y, Taruya A, Fukuda S, Taguchi H, et al. Prevalence of spontaneous coronary artery dissection in patients with acute coronary syndrome. Eur Heart J Acute Cardiovasc Care. 2016;5(3):263-270.
11. Sharma S, Kaadan MI, Duran JM, Ponzini F, Mishra S, Tsiaras SV, et al. Risk factors, imaging findings and sex differences in spontaneous coronary artery dissection. Am J Cardiol. 2019;123(11): 1783-1787.
12. Adlam D, Cortese B, Kadziela J. Autoimmune disease and spontaneous coronary artery dissection: Causation versus coexistence. J Am Coll Cardiol. 2020;76(19):2235-2237.
13. Hayes SN, Tweet MS, Adlam D, Kim ES, Gulati R, Price JE, et al. Spontaneous coronary artery dissection: JACC state-of-the-art review. J Am Coll Cardiol. 2020;76(8):961-984.