Spontaneous Coronary Artery Dissection: Clinical Characteristics, Management, and Outcomes in a Racially and Ethnically Diverse Community-Based Cohort

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ABSTRACT

Context: Spontaneous coronary artery dissection (SCAD) is a cause of acute coronary syndrome, which predominantly affects healthy women; however, few data define this vulnerable population.

Objective: To identify demographic and clinical characteristics of patients with SCAD and determine outcomes in a community-based cohort.

Design: Retrospective cohort study of patients with SCAD at Kaiser Permanente Northern California during a 10-year period. We compared 111 SCAD cases with 333 healthy, matched controls.

Main Outcome Measures: Predisposing factors, treatment modalities, and inhospital and late outcomes.

Results: Patients with SCAD had a mean age (standard deviation) of 48.1 (11) years; 92.8% were women, and 49.5% were nonwhite. Of women, 9% were peripartum. Fibromuscular dysplasia was identified in 21.8% of femoral angiograms obtained. With conditional logistic regression, only pregnancy and hyperlipidemia were associated with SCAD compared with controls. Fifty-five patients (49.5%) were successfully treated without revascularization; of the 54 who had urgent percutaneous coronary intervention, 2 required coronary artery bypass grafting for SCAD extension. During a median follow-up of 2.6 years, major adverse cardiovascular events occurred in 8.1% of patients. Pregnancy-related SCAD was not associated with worsened outcomes. However, Emergency Department visits or hospitalizations because of recurrent chest pain occurred frequently for 54% of patients with SCAD.

Conclusion: The study cohort is comparable to published SCAD cohorts, but notable for a racially and ethnically diverse population. Compared with the controls, only pregnancy and hyperlipidemia were associated with SCAD. For the SCAD cases, major adverse cardiovascular events occurred in 8.1%, and race did not influence outcomes.

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare and poorly understood cause of acute coronary syndrome and sudden cardiac death. The pathophysiology is caused by an intimal tear in the vessel wall or spontaneous hemorrhage within the vasa vasorum, leading to dissection of the vessel.[1] The prevalence of SCAD is unknown but has been reported to range from 0.1% to 4% of all acute coronary syndrome presentations.[2,3] SCAD appears to account for a larger percentage of myocardial infarction in women younger than the age of 50 years, representing 24% to 40% of cases.[4,5] It occurs predominantly in women without known risk factors for cardiovascular disease.[1] Fibromuscular dysplasia (FMD) may be diagnosed concomitantly with SCAD.[6] Another risk factor is pregnancy, especially with advanced maternal age and multiparity, which potentially involves more proximal vessels and extensive dissections.[7] Other precipitants include major emotional or physical stressors and hormonal factors, such as perimenopausal state, oral contraceptive pills (OCPs), hormonal replacement therapy (HRT), and infertility treatment.[1] Several case series of SCAD have been published, but the total number of cases and outcomes reported remains small.[8,9] This study attempts to identify demographic and clinical characteristics of patients with SCAD compared with healthy controls in a racially diverse, community-based cohort, as well as to report long-term outcomes of SCAD.

METHODS

We conducted a retrospective cohort study of all patients with a diagnosis of SCAD made at Kaiser Permanente (KP) Northern California (KPNC) between January 1, 2003, and December 31, 2012. KPNC is a large integrated health care delivery system providing comprehensive medical care to more than 4 million members (estimated to be more than one-fourth of the population of Northern California). The Kaiser Foundation Research Institute's institutional review board approved this study with a waiver of consent.

Database searches included a combination of International Classification of Diseases, Ninth Revision (ICD-9) and Current Procedural Terminology, Fourth Edition (CPT-4) codes for dissection of coronary artery (414.12), dissection of other artery (443.29), and coronary aneurysm (414.11). In addition, angiograms of pregnant women with acute coronary syndrome were reviewed to identify additional patients for the SCAD cohort. Exclusion criteria included age younger than 18 years, less than 9 months of enrollment in KPNC in the 12 months before the SCAD date, end-stage renal disease treated with dialysis, and coronary angiogram unavailable for review.

Coronary angiography and available intravascular ultrasonograms from all potential SCAD cases were independently reviewed by 2 experienced interventional cardiologists (RL, KM). Cases were included in the study when there was consensus regarding the diagnosis of SCAD.

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SCAD was confirmed to be present angiographically if clinically significant coronary atherosclerosis was absent, in addition to pathognomonic evidence of an intimal dissection with contrast retention in the arterial wall, multiple lumens, an abrupt change of arterial caliber, diffuse smooth narrowing typical of intramural hematoma, and/or confirmatory intravascular ultrasonography findings.[10]

Three controls for each case were obtained from the KPNC healthy population and matched to the SCAD case by age, sex, and follow-up time. The healthy population control group excluded patients with end-stage renal disease or any cardiovascular events occurring before the index date of the matched SCAD case. Each control had at least 9 months of enrollment in KPNC in the 12 months before the matched case date.

Demographics, clinical presentation, predisposing factors, treatment modalities, and inhospital and late outcomes were obtained through review of the medical records and analysis of subsequent coronary angiograms. FMD was defined by a "string-of-beads" appearance (ie, alternating stenoses and aneurysms) on imaging modalities of the arterial vessels, including femoral angiogram, computed tomography, or magnetic resonance imaging. Major adverse cardiovascular events (MACE), including recurrent SCAD, myocardial infarction, hospitalization because of congestive heart failure, and death, were obtained during follow-up. Death outcomes were ascertained from KPNC databases, State of California death records, and the Social Security Death Index. *Peripartum* was defined as during pregnancy or up to 3 months postpartum.

Statistical analysis was performed with statistical software (SAS 9.3, SAS Institute Inc, Cary, NC). Continuous data were summarized as means (standard deviation) and analyzed using the Student *t*-test with matched SCAD and control group data. Discrete variables were expressed as frequencies (percentages) and compared with a χ^2 test for unmatched data, and conditional logistic regression was used to evaluate predictors for SCAD in the matched case-control analysis. For patients with SCAD, a Cox proportional hazard model was used to evaluate predictors for follow-up events. A value of $p < 0.05$ was considered statistically significant.

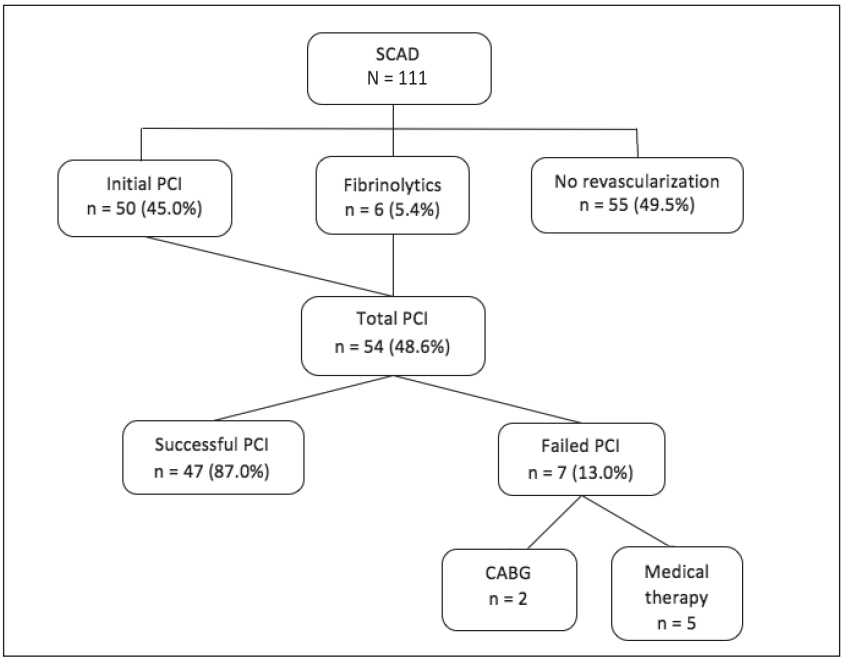

Figure 1. Initial treatment of spontaneous coronary artery dissection (SCAD)
CABG = coronary artery bypass grafting; PCI = percutaneous coronary intervention; SCAD = spontaneous coronary artery dissection.

RESULTS

The database search yielded 111 patients with confirmed SCAD from 2003 to 2012, and a corresponding 333 healthy controls were selected and matched by age, sex, and follow-up time. The clinical characteristics of the cases and controls are detailed in Table 1. Of the SCAD cases, the mean age (standard deviation) was 48.1 (11) years, and 92.8% of patients were women. This was a racially and ethnically diverse SCAD cohort, with 49.5% nonwhite patients, including 13.5% black, 16.2% Hispanic, and 18% Asian patients (Table 2). There were significantly more peripartum women in the SCAD cohort: 8.7% of patients with SCAD vs 1.3% of matched controls ($p = 0.0002$). With conditional logistic regression and limited to women, pregnancy was significantly associated with SCAD with an adjusted odds ratio of 7.5 (95% confidence interval = 1.9-28.1). Of note,

Table 1. Characteristics of SCAD cases and matched controls

Baseline characteristic	SCAD cases (n = 111)	Controls (n = 333)	p value
Age, mean (SD) [range], y	48.1 (11.0) [25-82]	48.2 (11.0) [25-83]	0.97
Women, no. (%)	103 (92.8)	309 (92.8)	> 0.99
Nonwhite race, no. (%)	55 (49.5)	158 (47.5)	0.69
Hypertension, no. (%)	32 (28.8)	78 (23.4)	0.24
Pregnancy, no. (%)[a]	9 (8.7)	4 (1.3)	0.0002
Diabetes mellitus, no. (%)	8 (7.2)	23 (6.9)	0.92
Hyperlipidemia, no. (%)	42 (37.8)	85 (25.5)	0.01
Tobacco use, no. (%)	14 (12.6)	41 (12.3)	0.93
Oral contraceptive pill (OCP), no. (%)[a]	12 (11.6)	45 (14.6)	0.42
Postmenopausal hormone replacement, no. (%)[a]	5 (4.8)	15 (4.8)	> 0.99
Combined OCP and HRT, no. (%)[a]	17 (16.5)	60 (19.4)	0.51

[a] Men excluded in percentage calculation (women only n = 103).
HRT = hormone replacement therapy; SCAD = spontaneous coronary artery dissection; SD = standard deviation.

there was no significant difference in OCP use or postmenopausal HRT between the groups (Table 1). There was a statistically significantly higher rate of hyperlipidemia in the SCAD cohort compared with the control group, with an odds ratio of 1.9 (95% confidence interval = 1.2-3.1). Otherwise, there was no statistically significant difference in cardiovascular risk factors. Outcomes data were obtained for all cases and included an 86% retention rate of cases as KP members at the end date of the study.

The clinical presentation and angiographic distribution of SCAD cases are summarized in Table 3. The majority (91%) of SCAD cases presented with acute coronary syndrome, with 23.4% having ST-segment elevation. Precipitating emotional or physical stress was reported in 12.6% of SCAD cases. Two patients reported illicit drug use within 24 hours of SCAD presentation, and an additional 4 patients used illicit drugs within the past year; these illicit drugs included marijuana, methamphetamine, cocaine, and heroin. The left anterior descending coronary artery was the most frequently involved (65.8%) artery. The left main coronary artery was involved in 5 (4.5%) cases. Multivessel SCAD occurred in 20.7% of patients. Femoral angiography was performed in 49.5% of SCAD cases, and FMD was identified in 21.8% of femoral angiograms obtained.

Figure 1 illustrates the initial management of SCAD cases: 55 (49.5%) of 111 cases were successfully treated conservatively without revascularization. Four of 6 patients who received fibrinolytic therapy before transfer to an angiography facility required subsequent percutaneous coronary intervention (PCI). Among 54 (48.6%) of SCAD cases treated with PCI, 87% underwent successful PCI, and 2 required coronary artery bypass grafting (CABG) because of failed PCI. In our SCAD cohort at discharge, aspirin was prescribed in 94% of patients, clopidogrel in 82%, a statin in 81%, a β-blocker in 88%, and an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker in 63%. At 1-year follow-up, 68% of patients continued to take aspirin; 48%, clopidogrel; 59%, statin; 60%, β-blocker; and 43%, angiotensin-converting enzyme inhibitor or angiotensin receptor blocker.

The mean length of follow-up (standard deviation) for cases was 3 (2.2) years, and the median (interquartile range) was 2.6 years (25th-75th percentiles = 1.4-4.3 years). Among SCAD cases, MACE occurred in 9 (8.1%). There were 8 cases with recurrent myocardial infarction, and all underwent repeated coronary angiography. Three angiograms showed new SCAD, 2 additional angiograms were without a new angiographic abnormality in the setting of early recurrent chest pain and troponin elevation, 1 showed early SCAD extension and hemopericardium, and 2 showed late recurrent myocardial infarctions, which were angiographically unexplained. Early recurrent symptoms or troponin elevation or both occurred within 30 days and typically represented propagation of the index dissection from unhealed SCAD, whereas late recurrence after 30 days was more likely because of de novo SCAD.[1] All cases of late recurrent de novo SCAD occurred in women and in a different vessel location than the index episode. Hospitalization because of congestive heart failure was observed in 1 case. No deaths were observed among SCAD cases. Among controls, MACE occurred in only 1 patient with a noncardiovascular death (0.3%). A Cox proportional hazard model did not show

Table 2. Racial or ethnic distribution

Race or ethnicity	SCAD (n = 111), no. (%)	Controls (n = 333)
White	56 (50.5)	175 (52.6)
Nonwhite	55 (49.5)	158 (47.4)
Black	15 (13.5)	38 (11.4)
Hispanic	18 (16.2)	59 (17.7)
Asian	20 (18.0)	46 (13.8)
Other	2 (1.8)	15 (4.5)

SCAD = spontaneous coronary artery dissection.

Table 3. Clinical presentation and angiographic distribution of SCAD cases (n = 111)

Parameter	Value[a]
Clinical presentation	
ST-elevation myocardial infarction	26 (23.4)
Non-ST-elevation myocardial infarction	75 (67.6)
Unstable angina pectoris	4 (3.6)
Ventricular fibrillation or ventricular tachycardia	6 (5.4)
Maximum troponin-I level, ng/mL, mean (SD)	15.1 (20.6)
Left ventricular ejection fraction, mean (SD), %	55.4 (11.1)
Emotional stress triggering event	10 (9)
Physical stress triggering event	4 (3.6)
SCAD angiographic distribution	
Left main coronary artery	5 (4.5)
Left anterior descending coronary artery	73 (65.8)
Ostium and proximal	14 (12.6)
Mid and distal	67 (60.4)
Left circumflex coronary artery	42 (37.8)
Ostium and proximal	5 (4.5)
Mid and distal	39 (35.1)
Right coronary artery	20 (18)
Ostium and proximal	3(2.7)
Mid and distal	20 (18)
Ramus intermedius coronary artery	1 (0.9)
> 1 SCAD lesion	23 (20.7)
Femoral angiogram	55 (49.5)
Fibromuscular dysplasia, no. (% of femoral angiogram)	12 (21.8)

[a] Value = no. (%) unless otherwise indicated.
SCAD = spontaneous coronary artery dissection; SD = standard deviation.

age, sex, race, PCI, number of SCAD lesions, or lesion site to be significantly associated with an increased risk of MACE. Limiting the model to women only, we found that pregnancy was not associated with MACE. During follow-up, 5 pregnancies occurred among SCAD cases with no recurrent SCAD episodes.

After the initial SCAD hospitalization, 60 (54.0%) of the SCAD cases had at least 1 Emergency Department visit or hospitalization because of chest pain; of these, 34 had repeated coronary angiography. Clinically driven repeated angiography was performed more than 20 days after the initial SCAD episode in 19 SCAD cases, with 15 (78.9%) of those showing complete healing of the initially dissected artery and new SCAD in 3 cases (as mentioned earlier regarding recurrent SCAD).

DISCUSSION

The baseline characteristics of this study cohort are comparable to those of other published SCAD cohorts.[8,9,11-14] Our SCAD cohort was predominantly women (92.8%) with an average age of 48.1 years.[8,9,12-14] This cohort was notable for a racially and ethnically diverse population, with 49.5% being nonwhite, higher than other cohorts in the literature, which reported 5% to 18% nonwhite patients.[8,9] Outcomes do not differ between white and nonwhite patients. This study has comparable rates of cardiovascular risk factors, specifically hypertension, hyperlipidemia, and cigarette smoking compared with those in the literature (eg, published rates are 27% to 51% for hypertension, 23% to 52% for hyperlipidemia, and 0.6% to 34% for smoking).[8,9,12-14] The rate of diabetes is higher in this study (7.2% of patients with SCAD in this study compared with 0% to 4.6% in the literature).[8,9,12-14]

Although other studies have compared patients with SCAD to patients with acute coronary syndrome, this study is the first known case-control study comparing SCAD cases with a healthy population without known cardiovascular disease.[15] There is a low prevalence of coronary artery disease risk factors in patients with SCAD compared with a control group with acute coronary syndrome. However, the prevalence of hyperlipidemia is higher in our SCAD population compared with healthy controls.[8,9,12-14]

Although pregnancy was more associated with SCAD compared with the control group, most SCAD events did not occur peripartum. Our study showed peripartum status in 9 (8.7%) of 103 women, compared with 2% to 17% reported in other studies.[8,9] There was no recurrent SCAD in 5 pregnancies after the initial SCAD event. None of the women who subsequently became pregnant had a pregnancy-associated index SCAD event. Tweet et al[16] reported that 8 of 363 women in their registry became pregnant after SCAD; 7 did not experience recurrent SCAD, but 1 had recurrent SCAD involving the left main coronary artery requiring CABG. Our study also did not show a significant difference between patients with SCAD and controls with comparable rates of OCP and postmenopausal HRT.

As reported in other series, our study found FMD to be common in the SCAD cohort. Our iliofemoral artery FMD rate of 21.8% underestimates FMD prevalence because we did not perform routine screening studies of other vessels, nor did all patients have iliofemoral arteriography. Tweet et al[17] detected FMD of the iliac artery in 50% of those who underwent limited angiography. Saw et al[18] screened for FMD in 3 vascular territories and observed FMD in 72% of SCAD cases. There were no patients in the control cohort with a diagnosis of FMD, although the prevalence of FMD in the population is 3% to 4% based on renal transplant donor studies.[19] The clinical manifestation of FMD is often asymptomatic, and as many as 25.7% of patients may present with dissection before receiving a diagnosis.[20] In our SCAD cohort, most patients received a diagnosis of FMD after their index SCAD presentation; only 1 patient had a preceding diagnosis of FMD.

Optimal acute management of SCAD is not known, but most groups recommend conservative management for stable patients with low-risk anatomy and no evidence of ongoing ischemia or hemodynamic compromise.[1,8,9,21] The reported technical failure rate with PCI is 9% to 31%,[8,9,12-14] and the rate of bypass graft occlusion is high (> 70% at follow-up angiography).[17,18] In our study, 49.5% of patients were successfully treated with a conservative approach. We experienced a low technical failure rate, including 13% of all PCIs, with 2 patients requiring CABG after failed PCI. We observed no deaths occurring inhospital or during comprehensive follow-up in our SCAD cohort.

Overall, the MACE rate (8.1%) was low compared with that reported in other studies. Our recurrent myocardial infarction rate was 7.2%, with an angiographically proven SCAD recurrence rate of 2.7% during a median follow-up of 3.2 years (25th-75th percentiles = 1.6-4.3 years). In comparison, Tweet et al[17] reported a recurrent SCAD rate of 17% with a median follow-up of 48.1 months (interquartile range = 18-106 months). Saw et al[18] reported a recurrent SCAD rate of 13% with a much longer median follow-up of 6.9 years in their retrospective cohort. Their respective updated cohorts reported a recurrence rate of 16.8% to 18% with a median 2.3- to 3.1-year follow-up.[8,9] Our lower recurrence rate could be because of high β-blocker prescription of 88% at discharge and 60% at 1-year follow-up. In one series, β-blockers were associated with a hazard ratio of 0.36 for recurrent SCAD.[9] This cohort is more racially and ethnically diverse than those described in the literature, and more research is needed to assess whether MACE outcomes vary by race or ethnicity. In this study, most recurrent infarction with new troponin-I elevation occurred early within 30 days after the presenting SCAD without angiographic evidence of new SCAD, and it may be because of subtle extension of the index SCAD vessel. Failure to angiographically identify the cause of recurrent infarction may also reflect the insensitivity of angiography for detecting small-vessel involvement or microvascular mechanisms including endothelial dysfunction.

In our cohort, there was a high rate of Emergency Department visits or hospitalization (54%) because of chest pain after the initial SCAD episode; of these visits, 42% were within 30 days of the index SCAD presentation. Most of these episodes were troponin-negative, and more than half underwent repeated coronary angiography. Saw et al[18] reported that 7.7% of their cohort required repeated hospitalization because of troponin-negative chest pain.

The strengths of this study include the sizable KPNC population, which includes at least one-fourth of the population of Northern California. We attempted to identify every case of SCAD in the KPNC

population via database query, which should reduce sampling bias. In addition, this study reflects a community-based setting and a racially and ethnically diverse population including 49.5% of nonwhite patients. The KP integrated electronic medical record system allows for a thorough assessment of long-term outcomes, and follow-up data were available for all our SCAD cases. There is a high retention rate of KP patients. Of note, 11 patients were excluded from this study because of nonmembership, but more than half of the patients enrolled for follow-up care. Further research is needed to determine if the lower MACE rate in our cohort correlates with continuity of care in the KP system. Whereas other studies have compared patients with SCAD and patients with acute coronary syndrome and atherosclerosis, this is the first case-control study of which we are aware that compares SCAD cases with a healthy population without known cardiovascular disease to ascertain potential predictors for SCAD.

There are a few limitations of this study because of its retrospective nature, including improvement in diagnostic recognition and treatment of SCAD during our study period from 2003 to 2012. In our study, the diagnoses of hyperlipidemia and diabetes were obtained by chart review, not from specific lipid or glucose levels measured before the SCAD episode; thereby, ascertainment bias is possible. Pregnancy-related elevation of lipid levels may have resulted in the diagnosis of hyperlipidemia in the SCAD cohort. Not all patients with SCAD were systematically screened for FMD, which likely underestimates the prevalence of FMD in our population. Compliance with medication prescription was not confirmed. Triggering events, genetic and connective tissue disease screening, and autoimmune disease were not routinely ascertained.

CONCLUSION

This case-control cohort study of SCAD included a racially diverse community population. Traditional cardiovascular risk factors, aside from hyperlipidemia, did not predict SCAD. There was a significantly higher rate of pregnancy in the SCAD cohort compared with controls. However, the rates of OCP and HRT prescription were similar. The SCAD recurrence rate was low, and the mortality rate was zero in this study. No variables, including subsequent pregnancy, were predictive of recurrent SCAD or other adverse events. Five patients had successful pregnancies without complications after their index SCAD event. However, there was a high rate of Emergency Department visits or hospitalization related to chest pain subsequently found to be troponin-negative. Further study is needed to elucidate the predictors of SCAD, determine optimal acute and preventive therapies, and define the risk of pregnancy in the SCAD population. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

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References

1. Hayes SN, Kim ES, Saw J, et al; American Heart Association Council on Peripheral Vascular Disease; Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; and Stroke Council. Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. Circulation 2018 May 8;137:e523-57. DOI: https://doi.org/
2. Mortensen KH, Thuesen L, Kristensen IB, Christiansen EH. Spontaneous coronary artery dissection: A Western Denmark Heart Registry study. Catheter Cardiovasc Interv 2009 Nov 1;74(5):710-7. DOI: https://doi.org/10.1002/ccd.22115.
3. Nishiguchi T, Tanaka A, Ozaki Y, et al. Prevalence of spontaneous coronary artery dissection in patients with acute coronary syndrome. Eur Heart J Acute Cardiovasc Care 2016 Jun;5(3):263-70. DOI: https://doi.org/10.1161/CIR.0000000000000564.
4. Saw J, Aymong E, Mancini GB, Sedlak T, Starovoytov A, Ricci D. Nonatherosclerotic coronary artery disease in young women. Can J Cardiol 2014 Jul;30(7):814-9. DOI: https://doi.org/10.1016/j.cjca.2014.01.011.
5. Grosseto D, Santarelli A, Carigi S, et al. Incidence of spontaneous coronary artery dissection in all comers patients referred for acute coronary syndrome [abstract]. Eur Heart J Acute Cardiovasc Care 2012;1(1 Suppl):61.
6. Michelis KC, Olin JW, Kadian-Dodov D, d'Escamard V, Kovacic JC. Coronary artery manifestations of fibromuscular dysplasia. J Am Coll Cardiol 2014 Sep 9;64(10):1033-46. DOI: https://doi.org/10.1016/j.jacc.2014.07.014.
7. Saw J. Pregnancy-associated spontaneous coronary artery dissection represents an exceptionally high-risk spontaneous coronary artery dissection cohort. Circ Cardiovasc Interv 2017 Mar;10(3):e005119. DOI: https://doi.org/10.1161/CIRCINTERVENTIONS.117.005119.
8. Tweet MS, Hayes SN, Codsi E, Gulati R, Rose CH, Best PJ. Spontaneous coronary artery dissection associated with pregnancy. J Am Coll Cardiol 2017 Jul 25;70(4):426-35. DOI: https://doi.org/10.1016/j.jacc.2017.05.055.
9. Saw J, Humphries K, Aymong E, et al. Spontaneous coronary artery dissection: Clinical outcomes and risk of recurrence. J Am Coll Cardiol 2017 Aug 29;70(9):1148-58. DOI: https://doi.org/10.1016/j.jacc.2017.06.053.
10. Yip A, Saw J. Spontaneous coronary artery dissection—A review. Cardiovasc Diagn Ther 2015 Feb;5(1):37-48. DOI: https://doi.org/10.3978/j.issn.2223-3652.2015.01.08.
11. Tweet MS, Kok SN, Hayes SN. Spontaneous coronary artery dissection in women: What is known and what is yet to be understood. Clin Cardiol 2018 Feb;41(2):203-10. DOI: https://doi.org/10.1002/clc.22909.
12. Lettieri C, Zavalloni D, Rossini R, et al. Management and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 2015 Jul 1;116(1):66-73. DOI: https://doi.org/10.1016/j.amjcard.2015.03.039.
13. Rogowski S, Maeder MT, Weilenmann D, et al. Spontaneous coronary artery dissection: Angiographic follow-up and long-term clinical outcome in a predominantly medically treated population. Catheter Cardiovasc Interv 2017 Jan;89(1):59-68. DOI: https://doi.org/10.1002/ccd.26383.
14. Nakashima T, Noguchi T, Haruta S, et al. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: A report from the Angina Pectoris-Myocardial Infarction Multicenter Investigators in Japan. Int J Cardiol 2016 Mar 15;207:341-48. DOI: https://doi.org/10.1016/j.ijcard.2016.01.188.
15. Adams H, Paratz E, Somaratne J, et al. Different patients, different outcomes: A case-control study of spontaneous coronary artery dissection versus acute coronary syndrome. J Interv Cardiol 2018 Feb;31(1):41-7. DOI: https://doi.org/10.1111/joic.12447.
16. Tweet MS, Hayes SN, Gulati R, Rose CH, Best PJ. Pregnancy after spontaneous coronary artery dissection: A case series. Ann Intern Med 2015 Apr 21;162(8):598-600. DOI: https://doi.org/10.7326/L14-0446.
17. Tweet MS, Hayes SN, Pitta SR, et al. Clinical features, management and prognosis of spontaneous coronary artery dissection. Circulation 2012 Jul 31;126(5):667-70. DOI: https://doi.org/10.1161/CIRCULATIONAHA.112.105718.
18. Saw J, Aymong E, Sedlak T, et al. Spontaneous coronary artery dissection association with predisposing arteriopathies and precipitating stressors and cardiovascular outcomes. Circ Cardiovasc Interv 2014 Oct;7(5):645-55. DOI: https://doi.org/10.1161/CIRCINTERVENTIONS.114.001760.
19. Shivapour DM, Erwin P, Kim ES. Epidemiology of fibromuscular dysplasia: A review of the literature. Vasc Med 2016 Aug;21(4):376-81. DOI: https://doi.org/10.1177/1358863X16637913.
20. Kadian-Dodov D, Gornik HL, Gu X, et al. Dissection and aneurysm in patients with fibromuscular dysplasia: Findings from the US registry for FMD. J Am Coll Cardiol 2016 Jul 12;68(2):176-85. DOI: https://doi.org/10.1016/j.jacc.2016.04.044.
21. Alfonso F, Paulo M, Lennie V, et al. Spontaneous coronary artery dissection: Long-term follow-up of a large series of patients prospectively managed with a 'conservative' therapeutic strategy. JACC Cardiovasc Interv 2012 Oct;5(10):1062-70. DOI: https://doi.org/10.1016/j.jcin.2012.06.014.