Spontaneous coronary artery dissection: Case series from two institutions with literature review

Carlos Esteban Uribe[1], Juan David Ramirez-Barrera[2], Carlos Rubio[1], Cesia Gallegos[3],
Luz Adriana Ocampo[2], Clara Saldarriaga[2], Carlos Eusse[1], Carlos Tenorio[1], Nilson Lopez[4], Andres Moreno[1],
Natalia Gonzalez Jaramillo[5], Alexander Morteza Chehrazi-Raffle[6], Vikas Singh[3], Pedro Martinez-Clark[3]

[1]Clínica Cardio VID, Interventional Cardiology, Medellin, Colombia-USA
[2]Clínica Cardio VID, Cardiology, Medellin, Colombia-USA
[3]Interventional Concepts Inc; Miami FL-USA
[4]Hospital Pablo Tobon Uribe Cardiology; Medellín Colombia-USA
[5]Clínica Cardio VID Epidemiology; Medellín Colombia-USA
[6]The Chicago Medical School; North Chicago IL-USA

ABSTRACT

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS). Consequently, its presentation and optimal treatment are yet to be clearly defined. In the current literature, all case series report less than 50 patients, most of whom are either young peripartum women or women who have used oral contraceptives over long periods. All information in this study was compiled by the database service from two hospitals, the first one between 2003 and 2012 and the second one between 2007 and 2012, to include the clinical characteristics, angiography. and treatment approaches in the study population. The study population consisted in four women (50%) and four men (50%) whose ages ranged between 28 and 57 years. Two women had a history of oral contraceptive use and three women presented during peripartum. None of the patients had traditional cardiovascular risk factors or previous heart disease. In 88% of the cases, the principal diagnoses were non-ST segment elevation myocardial infarction and unstable angina. All patients underwent emergency coronary angiography and percutaneous coronary intervention. Half of them were treated with drug-eluting stents and the other half with bare metal stents. The most frequent type of dissection was NIHBL Type E, and the right coronary artery was the most frequently compromised. SCAD is a rare cause of ACS; however, its identification has improved due to the availability of angiography and new complementary techniques. Regarding treatment, PCI seems effective with adequate long-term results.
(Anatol J Cardiol 2015; 15: 409-15)
Keywords: spontaneous coronary dissection, peripartum, sudden death, acute coronary syndrome

Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome. As such, the incidence, pathogenesis, and treatment have yet to be clearly defined. The first reports were based on post-mortem studies of fatal cases. In the current literature, publications contain less than 50 patients, most of whom are either young peripartum women or women who have used oral contraceptives over long periods. In this study, we evaluated the characteristics of eight patients admitted to our institution with a diagnosis of SCAD by the virtue of the classification of the dissection, associated risk factors, clinical presentation, treatment performed, and long-term prognosis. In addition, we compiled a literature review to assess the fundamental aspects of this condition.

Study methodology

We evaluated 19,625 coronary angiograms between 2003 and 2012 from patients treated in Clinica Cardio VID, and Hospital Pablo Tobon Uribe, interventional cardiology centers. We collected the clinical characteristics, angiographic features, and treatment strategies and followed up at one year. We found a total of eight patients who were angiographically diagnosed with SCAD. In accordance with the National Heart, Lung and Blood Institute (NHLBI) scale, we defined a coronary dissection as a double lumen in an artery with a radiolucent flap (1). We conducted a demographic descriptive analysis of patients with SCAD. We determined the type of acute coronary syndrome with which the patient presented and documented the treatment received. Follow-up at one year was achieved in four of the eight patients (50%).

410 Uribe et al.
Spontaneous coronary artery dissection

Anatol J Cardiol 2015; 15: 409-15

Table 1. Baseline clinical characteristics of patients

Patient	1	2	3	4	5	6	7	8
Age (years)	35	28	57	28	55	49	41	55
Gender	Female	Female	Female	Female	Male	Male	Male	Male
Race	Black	White	Black	White	White	White	White	White
Postpartum	Yes	Yes	No	Yes	NA	NA	NA	NA
Hypertension	No	No	Yes	No	Yes	Yes	No	No
Diabetes	No	No	No	No	Yes	Yes	No	No
Dyslipidemia	No	No	No	No	Yes	Yes	No	No
Connective tissue disease	No	No	No	No	No	No	No	No
Oral contraceptive use	Yes	No	No	Yes	NA	NA	NA	NA
Previous heart disease	No	No	No	No	No	No	No	No
Presentation	UA	Non-STEMI	Non-STEMI	UA	UA	Non-STEMI	Non-STEMI	Non-STEMI
Troponins	NA	0.9	3.2	NA	MB: 16	3.39	MB: 13	2.0
Ejection fraction	0.55	0.10	0.45	0.55	0.50	0.45	0.60	0.60
Treatment	PCI+DES	PCI+DES	PCI+DES	PCI+DES	PCI+BMS	PCI+BMS	PCI+BMS	PCI+DES
Follow-up	No events	No events	ND	ND	ND	No events	ND	No events

BMS - bare metal stent; DES - drug-eluting stent; MB - phosphocreatine kinase assay ; NA - non available; ND - no data; Non-STEMI - non-ST elevation myocardial infarction; PCI - percutaneous coronary intervention; UA - unstable angina

Table 2. Angiography characteristics

Patient	Artery	Double lumen	Contrast retention	Lumen compression	Occlusion	Arteriosclerosis of other vessels	NIHBL classification
1	Cx	Yes	Yes	Yes	No	No	E
2	RCA	No	No	Yes	No	No	E
3	Cx	Yes	Yes	Yes	No	No	E
4	RCA	Yes	Yes	Yes	No	No	E
5	Cx	Yes	Yes	Yes	No	Yes	E
6	RCA	Yes	Yes	Yes	Yes	No	F
7	RCA	Yes	Yes	Yes	Yes	No	E
8	RCA	Yes	Yes	Yes	No	No	E

Cx - circumflex artery; RCA - right coronary artery

Results

Table 1 summarizes the clinical characteristics and treatment. Four of the patients were women (50%) and six of the patients were Caucasian (75%). The age range was between 28 and 57 years with a mean age of 43.5 years (50 for men, 37 for women). Among women, the most prevalent risk factor was postpartum, which was present in 75% of women. Prior use of contraception was reported in two of the women patients. No patients had traditional cardiovascular risk factors or previous heart disease, and none had any history of secondary coronary dissection either. The reasons for admission were non-STEMI (63%) and unstable angina (37%). All patients underwent emergency coronary angiography and received immediate percutaneous treatment. Four patients were treated with bare metal stents (50%) and four with drug-eluting stents (50%), all of which resulted in TIMI 3 flow.

Table 2 indicates angiography characteristics. The right coronary artery was most frequently compromised (63%), and the majority had NIHBL Type E classification of dissections (88%). Only one patient had a severely impaired left ventricular systolic function with an ejection fraction of 10%. No patients died during hospitalization. Event-free survival and angina events were gathered from only four patients because of a loss of follow-up in four patients.

Literature review

SCAD is an uncommon cause of acute coronary syndrome and sudden death that is poorly understood and classically affects otherwise healthy and young population (2-5). It is a separation between the layers of the coronary artery (intima or media), which creates a fold with free communication between the true and false lumen or an intramural hematoma causing

Anatol J Cardiol 2015; 15: 409-15

Uribe et al.
Spontaneous coronary artery dissection **411**

blood flow obstruction. Furthermore, the rupture of the *vasa vasorum* may also generate vessel wall hemorrhage without communication with the lumen (2, 3). However, it is not clear whether the dissection or the hematoma happens first. Although often presenting as an ACS, the pathophysiology is completely different in the sense that SCAD does not involve atherosclerosis or plaque rupture, but is rather the result of the aforementioned mechanisms. This is corroborated by the fact that it is more common in young women without risk factors for atherosclerosis. The true prevalence is unknown; however, the advances in imaging techniques have led to new understanding of diagnosis and management, particularly with the use of IVUS, CT angiography, and optical coherence tomography (OCT) (2-4, 6, 7). The average age of incidence is 42 years, with about 80% of patients being females; 20–25% of the cases occur in the peripartum period and are more frequently diagnosed in the left coronary artery (4, 8). It is also associated with collagen diseases. Interestingly, most cases are diagnosed at autopsy (5, 7).

In terms of classification, coronary dissection can be primary or secondary. Primary dissections occur spontaneously, whereas secondary dissections can be caused by an extension of an aortic root dissection, percutaneous coronary intervention, cardiac surgery, or thoracic trauma (5). The NHLBI classifies dissections based on angiographic appearance. Type A dissections are characterized by the presence of radiolucent areas within the vessel lumen during contrast injection with little or no persistence once the dye has cleared. Type B dissections present parallel tracts or a double lumen separated by a radiolucent area during the contrast injection with little or no persistence once the dye has cleared. Type C dissections have persistent contrast extravasation. Type D dissections are spiral luminal filling defects on vessels with complete but slow distal flow. Type E dissections are persistent filling defects with slow anterograde flow. Type F dissections result in total occlusion of the vessel (1, 5).

The first reported case was described in 1931 in the autopsy of a 42-year-old woman (5). To date, no single publication has presented more than 50 cases. Although the first reports were from autopsy studies, the advent and availability of coronary angiography has enabled early diagnosis and evaluation of the possible pathophysiological mechanisms. The incidence of SCAD by angiography is highly variable, typically ranging from 0.07% to 1.1% of patients referred for cardiac catheterization (9, 10). Our study found a prevalence of 0.035% in a sample population of more than 19.000 patients. Risk factors for SCAD include African descent, age over 35 years, multiparity, hypertension, thrombophilia, diabetes mellitus, smoking, and pre-eclampsia. Our results had similar mean ages for men and women (38 and 48 years, respectively). Women comprised 57% of our sample population, which is a smaller proportion than previous studies (9, 10). However, the proportion of our cases associated with peripartum was 70%, which is consistent with the literature.

In our population, the most common clinical presentation is a non-STEMI. Contrary to previous studies, our study found that the circumflex artery was most commonly affected among women, and the RCA was most compromised among men, which is different from what other series have reported. Additionally, two risk factors that occur in our registry with a higher frequency than previous literature are Caucasian descent and hypertension (5).

The pathogenic mechanisms for SCAD are still under investigation, and many of the hypotheses described are based on speculation or causal associations. The results of pathologic descriptions have come from autopsies of patients who had presented with sudden death due to cardiac arrest, and are now being complemented with the use of the aforesaid intravascular imaging techniques. Contrary to aortic dissections, SCAD is typically circumferential and located within the external third of the tunica media or between the tunica media and tunica adventitia which creates a false lumen, all of this in the absence of traumatic or iatrogenic causes. Furthermore, this lumen expands because of blood or clot accumulation, leading to the distal propagation of the dissection, compressing the real lumen, and resulting in myocardial ischemia.

The most common conditions associated with SCAD are atherosclerosis and vascular changes during the peripartum period. One third of all SCAD cases occur in women in the peripartum period, possibly as a result of hormonal and hemodynamic changes that ensue during pregnancy and early after delivery. The peak incidence is within the first 2 weeks after delivery, and it may take up to six months to return to the pre-pregnancy baseline (5, 11, 12). Pregnancy increases cardiac output and increases plasma volume, which in turn increases ejection fraction and blood pressure. These increases lead to a higher shear force on the artery walls that can lead to intimal tears. In addition, hormonal and biochemical changes (decreased collagen synthesis and increased production of progesterone) weaken the middle layer of the vessel, thereby increasing the likelihood of SCAD during peripartum. After a patient has developed SCAD, pregnancy is not recommended (2, 13).

Other causes include systemic inflammatory conditions, such as periarteritis nodosa, lupus erythematosus, and eosinophilia. In the latter, the proposed mechanism is related to adventitial infiltration by eosinophils. This infiltration is described in up to 43% of SCAD cases and suggests a relationship between the eosinophilic infiltration of the cervix during pregnancy and eosinophil infiltration of the coronary artery adventitia (18). Cytotoxin release results in the lysis of the tunica media and damage to the arterial collagen that predisposes dissection. Some studies propose that eosinophils are also a part of the mechanism for peripartum cardiomyopathy; however, more studies are needed to support this hypothesis (14, 15). Our study reported the peripartum period as an important risk factor present in 42% of the cases in our registry; however, this could be due to selection bias because all subjects were referred to a tertiary reference center.

The smaller percentage of coronary dissections is due to spontaneous ruptures of atherosclerotic plaques, although the

412 Uribe et al.
Spontaneous coronary artery dissection
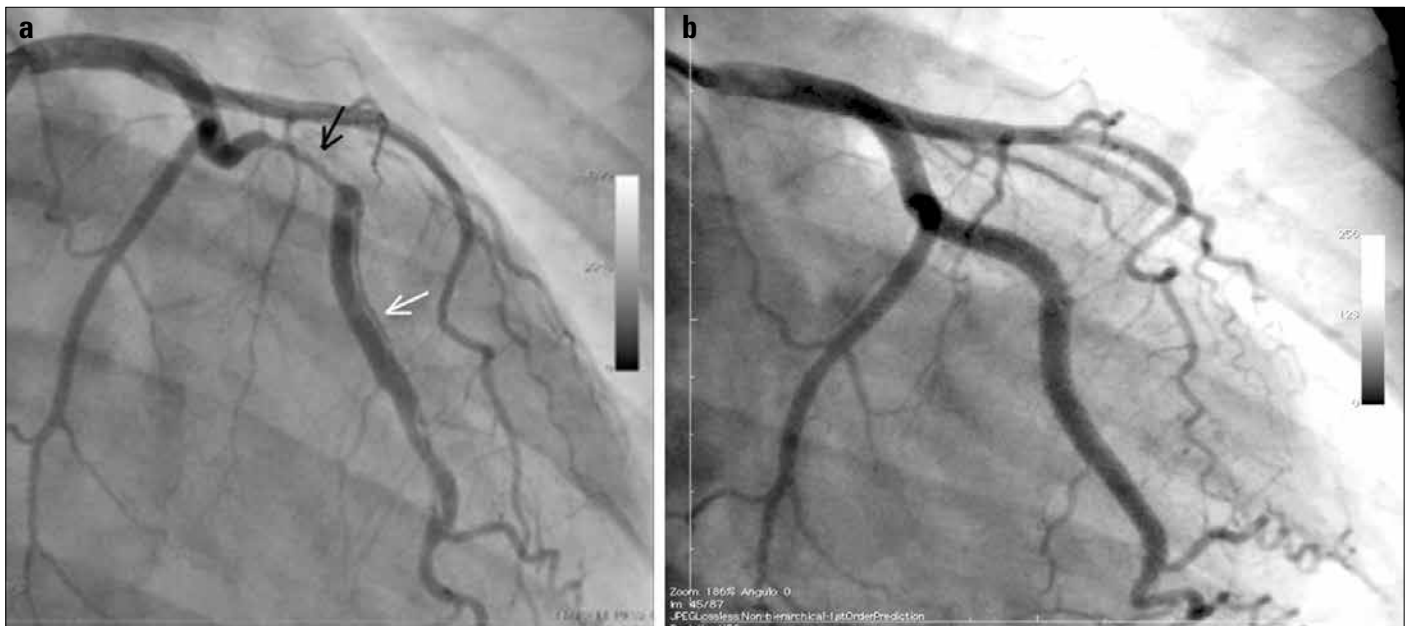
Anatol J Cardiol 2015; 15: 409-15

Figure 1. a, b. a-Pre-intervention, baseline coronary angiography. An intramural hematoma (black arrow) was found in the left circumflex coronary artery that extends distally as a spiral dissection (white arrow). b-Post-intervention, final result after treatment with two overlapping drug-eluting stents (star)

diagnoses of this type of SCAD have been increasing with the availability of IVUS (5). This atherosclerotic inflammatory process could lead to intramedial hemorrhage, which could further extend the dissection. Other conditions associated with SCAD are Marfan syndrome, Ehlers-Danlos syndrome, cystic medial degeneration, vasculitic processes, substances such as cocaine (by inducing systemic hypertension or coronary vasospasm), and other idiopathic causes in which the condition cannot be detected (5).

The clinical presentation of SCAD is similar to that of acute coronary syndromes (4, 12). Symptoms may present as unstable angina, acute myocardial infarction, ventricular arrhythmias, or sudden death. SCAD should be considered in differential diagnosis for a young patient with no risk factors, a postpartum woman with acute coronary syndrome, or a presentation of sudden death. Coronary angiography is recommended to rule out SCAD (5, 12, 16). The presentation of our patients who had SCAD was either non-STEMI (63%) or unstable angina (37%).

Coronary angiography remains the most common method for clinical diagnosis; however, requires a high degree of suspicion (2, 4). Particular care must be taken when performing coronary angiography in a patient with suspected SCAD because the procedure itself is a risk factor for increasing the dissection. A fluoroscopic diagnosis requires the presence of a thin longitudinal radiolucency representing the dissection flap, creating two or more lumens. This is exceptionally important because adequate differentiation of ACS due to SCAD from atherosclerosis is vital because the treatment approaches are completely different (2). Although angiography is the gold standard, it cannot be used to visualize the coronary wall; hence, its accuracy for diagnosis is limited (3). In this sense, complementary imaging techniques can

aid in identifying the lesion (3). IVUS, for instance, can aid in identifying intramural hematomas without flap dissection, particularly those caused by atherosclerosis. It has been described to be diagnostically helpful for navigating the guidewire into the true lumen, despite angiography yielding a good appearance (7). Optical coherence tomography (OCT) is a new tool that provides a higher resolution to visualize the intimal tear and evaluate the length of hematomas, providing valuable insights on atherosclerosis and results of coronary interventions. In prospective study of OCT performed in 17 consecutive patients out of 5,002 undergoing coronary angiography, OCT was able to rule out the diagnosis of SCAD in 6 patients and confirmed the presence of SCAD in the rest (3). Other lower resolution tools, such as magnetic resonance imaging (MRI) and cardiac computed tomography angiography (CCTA) have been used more for monitoring purposes (17, 18). Angiography allowed us to only document the dissection flap with the visualization of the true and false lumen (Fig. 1). Therefore, we may have underestimated the true incidence of SCAD because we did not have IVUS or OCT as diagnostic tools for most of our patients (Fig. 2).

Currently, there are few treatment management guidelines for patients with SCAD. Treatment options include medical therapy, percutaneous coronary intervention (PCI), or coronary artery bypass surgery (CABS). Therapeutic decisions are based on the individual evaluation of each case. There are reports of cases in which non-interventional treatment adequately resolved SCAD. As can be seen in the algorithm proposed by Vrints et al. (5), CCTA or MRI can be used as imaging methods for monitoring SCAD (Fig. 3). Medical therapy is similar to acute coronary syndromes, including antithrombotic therapy with unfractionated or low molecular weight heparin, aspirin, clopi-
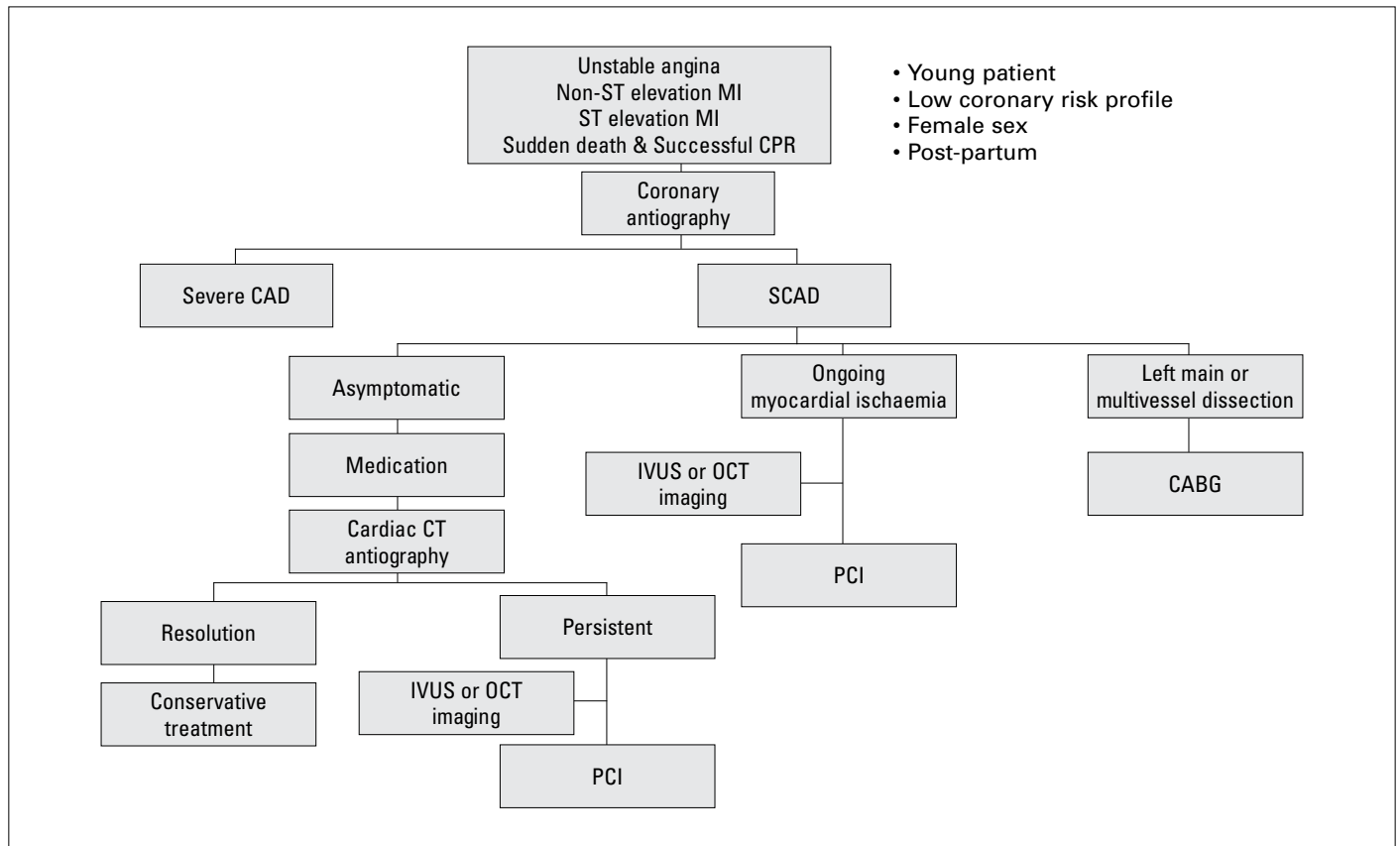
Anatol J Cardiol 2015; 15: 409-15

Uribe et al.
Spontaneous coronary artery dissection   *413*

Figure 2. Algorithm for the Diagnosis and Treatment of Spontaneous Coronary artery Dissection. Reproduced with permission (5)
CAD - coronary artery disease; CPR - cardio pulmonary resucitation; ST - ST segment at EKG; MI - myocardial infarction; SCAD - spontaneous coronary artery dissection; IVUS - intravascular ultrasound; OCT - optic coherence tomography; CT - computed tomography; PCI - percutaneous coronary intervention; CABG - coronary artery bypass graft
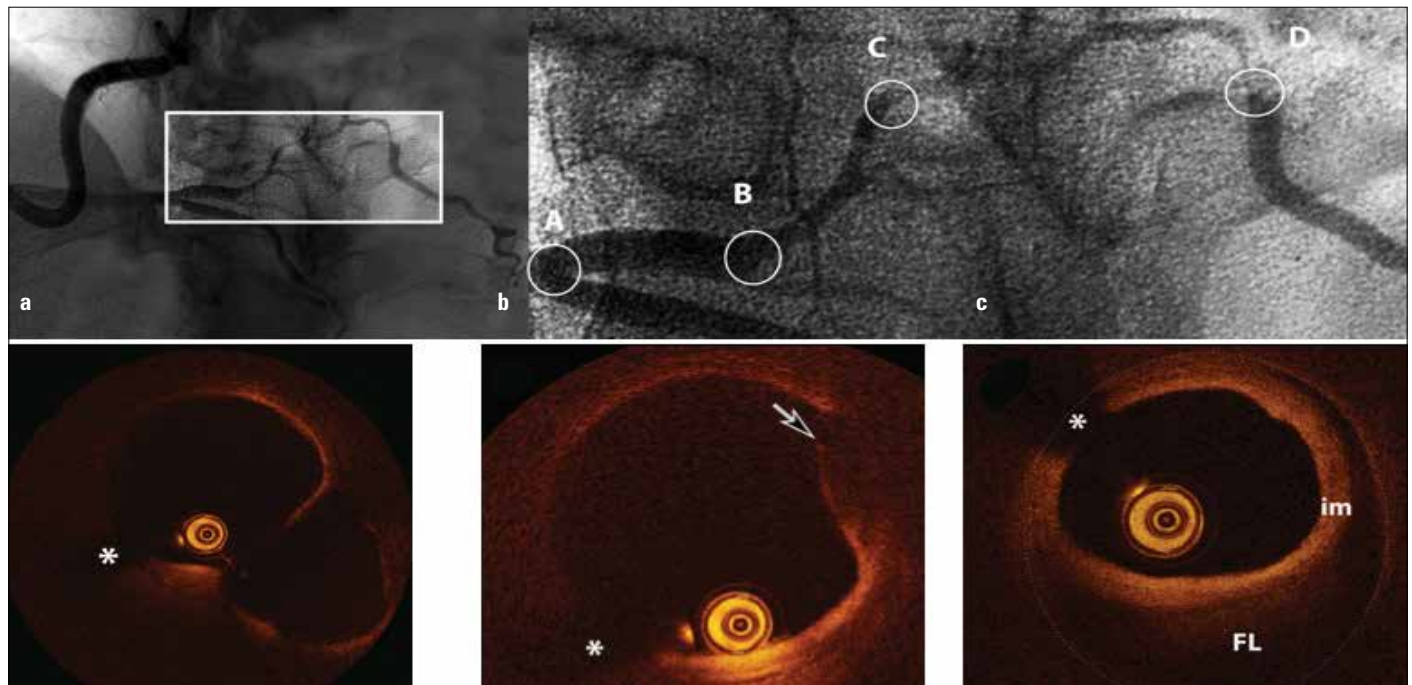

Figure 3. a-c. SCAD in large posterolateral branch of right coronary artery visualized with (OCT). (a) IntracoronaryOCTof the bifurcation of theposterior descendingandposterolateralbranches of the right coronaryartery. Nosigns ofdissectionat this level. (b) IntracoronaryOCT of a large posterolateral artery. A dissection flap is observed. (c) Intracoronary OCTof a large posterolateral artery. The dissection in the intima-media (im) separates the true lumen from the falselumen (FL). (arrow)
* shadow artifact caused by the OCTguide wire. SCAD - spontaneous coronary artery dissection, OCT - optical coherence tomography

414 Uribe et al.
Spontaneous coronary artery dissection

Anatol J Cardiol 2015; 15: 409-15

dogrel and GP IIb/IIIa inhibitors, anti-ischemic therapy with beta-blockers, and nitrates (5, 19). However, other studies warn about the use of antithrombotic therapy. Although antithrombotic therapy can help reduce the thrombus in the false lumen, allowing normal flow in the true lumen, doing so could increase the risk of bleeding, which expands the intramural hematoma and can cause the collapse of the true lumen. We believe that the use of anticoagulants is not recommended because fibrinolysis has been associated with extending the coronary dissection extension and increased mortality. In cases where the dissection causes significant compromised blood flow, percutaneous intervention or surgery should be considered. In our series, all patients were treated with percutaneous intervention, four with drug-eluting stents, and four with bare metal stents.

Mortality varies greatly between studies, ranging from 48% to 82%. Recurrent dissections may occur in the months following the initial event. About 50% of patients develop a second episode of SCAD within 2 months (20). Those who survive the initial event have a survival rate of 80% at 25 to 30 months, and men (93%) have better survival rates than women (73%). This disparity can be attributed to the low rates of comorbidity found during peripartum. In our series, the free survival rate was available in four of eight patients, all of whom survived (100%).

Conclusion

SCAD is a rare cause of acute coronary syndrome and sudden death. Currently, its identification has improved due to the availability of coronary angiography and new complementary techniques, such as IVUS or OCT. This condition should always be considered in the differential diagnoses for young or peripartum women who present with acute coronary syndrome. Percutaneous stenting is an effective treatment with satisfactory long-term results.

Conflict of interest: None declared.

Peer-review: Externally peer-reviewed.

Authorship contributions: Concept - C.E.U., J.D.R.B., P.M.C.; Design - C.S., C.R., L.A.O.; Supervision - C.E.U., J.D.R.B., P.M.C.; Resource - P.M.C.; Materials - C.E.U., P.M.C.; Data collection &/or processing - C.E.U., J.D.R.B., C.R., L.A.O., C.S., C.E., C.T., N.L., A.M., N.G.J., A.M.C.R., Analysis&/or Interpretation - C.S., C.E., C.T., N.L., C.G., V.S.; Literature search - L.A.O., C.G., V.S.; Writing - C.E.U., C.G., V.S., C.S., C.E., A.M., N.L.; Critical review - C.E.U., J.D.R.B., C.R., L.A.O., C.S., C.E., C.T., N.L., A.M., N.G.J., A.M.C.R., C.G., V.S., P.M.C.

References

1. Huber MS, Mooney JF, Madison J, Mooney MR. Use of a Morphologic Classification to Predict Clinical Outcome After Dissection from Coronary Angioplasty. Am J Cardiol 1991; 68: 467-71. **[CrossRef]**
2. Hayes SN. Spontaneous coronary artery dissection (SCAD): new insights into this not-so-rare condition. Tex Heart Inst J 2014; 41: 295-8. **[CrossRef]**
3. Alfonso F, Paulo M, Gonzalo N, Dutary J, Jimenez-Quevedo P, Lennie V, et al. Diagnosis of spontaneous coronary artery dissection by optical coherence tomography. J Am Coll Cardiol 2012; 59: 1073-9. **[CrossRef]**
4. Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012; 126: 579-88. **[CrossRef]**
5. Vrints CJ. Spontaneous coronary artery dissection. Heart 2010; 96: 801-8. **[CrossRef]**
6. Paulo M, Sandoval J, Lennie V, Dutary J, Medina M, Gonzalo N, et al. Combined use of OCT and IVUS in spontaneous coronary artery dissection. JACC Cardiovasc Imaging 2013; 6: 830-2. **[CrossRef]**
7. Miki K, Fujii K, Nakata T, Shibuya M, Fukunaga M, Kawai K, et al. The utility of intravascular ultrasound for the diagnosis and management of spontaneous coronary artery dissection in a middle-aged woman with acute inferior myocardial infarction. J Cardiol Cases 2012; 6: e78-e80. **[CrossRef]**
8. Verma PK, Sandhu MS, Mittal BR, Aggarwal N, Kumar A, Mayank M, et al. Large spontaneous coronary artery dissections: A study of three cases, literature review, and possible therapeutic strategies. Angiology 2004; 55: 309-18. **[CrossRef]**
9. Mortensen KH, Thuesen L, Kristensen IB, Christiansen EH. Spontaneous coronary artery dissection: a Western Denmark Heart Registry study. Catheter Cardiovasc Interv 2009; 74: 710-7. **[CrossRef]**
10. Vanzetto G, Berger-Coz E, Barone-Rochette G, Chavanon O, Bouvaist H, Hacini R, et al. Prevalence, therapeutic management and medium-term prognosis of spontaneous coronary artery dissection: results from a database of 11.605 patients. Eur J Cardiothorac Surg 2009; 35: 250-4. **[CrossRef]**
11. Rajab TK, Khalpey Z, Kraemer B, Resnic FS, Gallegos RP. Recurrent post-partum coronary artery dissection. J Cardiothorac Surg 2010; 5: 78. **[CrossRef]**
12. Alfonso F, Paulo M, Lennie V, Dutary J, Bernardo E, Jimenez-Quevedo P, 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; 5: 1062-70. **[CrossRef]**
13. Tweet MS, Hayes S, Gulati R. The risk of pregnancy after spontaneous coronary artery dissection. J Am Coll Cardiol 2014; 63: 12. **[CrossRef]**
14. Kajihara H, Tachiyama Y, Hirose T, Takada A, Saito K, Murai T, et al. Eosinophilic coronary periarteritis (vasospastic angina and sudden death), a new type of coronary arteritis: report of seven autopsy cases and a review of the literature. Virchows Arch 2013; 462: 239-48. **[CrossRef]**
15. Robinowitz M, Virmani R, McAllister HA JrU. Spontaneous coronary artery dissection and eosinophilic inflammation: a cause and effect relationship? Am J Med 1982; 72: 923-8. **[CrossRef]**
16. Ito H, Taylor L, Bowman M, Fry ETA, Hermiller JB, Van Tassel JW. Presentation and therapy of spontaneous coronary artery dissection and comparisons of postpartum versus nonpostpartum cases. Am J Cardiol 2011; 107: 1590-6. **[CrossRef]**
17. Wessely R, Botnar RM, Vorpahl M, Schwaiger M, Schomig A, Ibrahim T. Images in cardiovascular medicine. Subacute thrombotic occlusion and spontaneous recanalization of the right coronary artery after percutaneous coronary intervention for ST-elevation myocardial infarction visualized by coronary angiography and cardiac magnetic resonance imaging. Circulation 2007; 116: e78-80. **[CrossRef]**

Anatol J Cardiol 2015; 15: 409-15

Uribe et al.
Spontaneous coronary artery dissection   *415*

18. Chopard R, Jehl J, Dutheil J, Genon VD, Seronde MF, Kastler B, et al. Evolution of acute coronary syndrome with normal coronary arteries and normal cardiac magnetic resonance imaging. Arch Cardiovasc Dis 2011; 104: 509-17. [CrossRef]

19. Sarmento-Leite R, Machado PR, Garcia SL. Spontaneous coronary artery dissection: stent it or wait for healing? Heart 2003; 89: 164. [CrossRef]

20. Kamineni R, Sadhu A, Alpert JS. Spontaneous coronary artery dissection: report of two cases and a 50-year review of the literature. Cardiol Rev 2002; 10: 279-84. [CrossRef]

21. DeMaio SJ Jr, Kinsella SH, Silverman ME. Clinical course and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 1989;64:471-4. [CrossRef]

22. Jorgensen MB, Aharonian V, Mansukhani P, Mahrer PR. Spontaneous coronary dissection: a cluster of cases with this rare finding. Am Heart J 1994;127:1382-7. [CrossRef]

23. Hering D, Piper C, Hohmann C, Schultheiss HP, Horstkotte D. [Prospective study of the incidence, pathogenesis and therapy of spontaneous, by coronary angiography diagnosed coronary artery dissection]. Z Kardiol 1998;87:961-70. [CrossRef]

24. Çelik SK, Sağcan A, Altıntığ A, Yüksel M, Akın M, Kültürsay H. Primary spontaneous coronary artery dissections in atherosclerotic patients. Report of nine cases with review of the pertinent literature. Eur J Cardiothorac Surg 2001;20:573-6. [CrossRef]

25. Butler R, Webster MW, Davies G, Kerr A, Bass N, Armstrong G, et al. Spontaneous dissection of native coronary arteries. Heart 2005;91:223-4. [CrossRef]

26. Motreff P, Souteyrand G, Dauphin C, Eschalier R, Cassagnes J, Lusson JR. Management of spontaneous coronary artery dissection: review of the literature and discussion based on a series of 12 young women with acute coronary syndrome. Cardiology 2010;115:10-8. [CrossRef]

27. Kansara P, Graham S. Spontaneous coronary artery dissection: case series with extended follow up. J Invasive Cardiol 2011;23:76-80.

28. Romero-Rodriguez N, Fernandez-Quero M, Villa Gil-Ortega M, Urbano del Moral JA, Ballesteros Prada S, Diaz de la Llera L, et al. Spontaneous coronary dissection and its long-term prognostic implications in a cohort of 19 cases. Rev Esp Cardiol 2010;63:1088-91. [CrossRef]