Srce i krvni sudovi 2022; 41(1): 5-12

Review article

Spontaneous coronary artery dissection: What we know so far?

Svetlana Apostolović[1,2], Stefan Milutinović[2], Tomislav Kostić[1,2], Miroslav Nikolić[1], Miljan Krstović[2], Milovan Petrović[3], Ivan Ilić[4], Zlatko Mehmedbegović[5], Dejan Milašinović[5], Aleksandra Djoković[9], Vanja Miloradović[8], Nemanja Djenić[7], Vladimir Mitov[8], Nikola Jagić[6], Zoran Perišić[1,2], Aleksandar Nešković[6]

[1]Clinic for cardiovascular diseases, University Clinical Center of Niš, [2]Faculty of Medicine, University of Niš, [3]Institute of Cardiovascular Diseases of Vojvodina, Medical Faculty, University of Novi Sad, [4]Institute for Cardiovascular Diseases Dedinje, [5]University Clinical Center of Serbia, Cardiology clinic, [6]Clinical hospital center Zemun, [7]Military Medical Academy, [8]Medical Center Zajecar, [9]Clinical hospital center Bežanijska Kosa

Abstract Spontaneous coronary artery dissection (SCAD) is one of the causes of acute coronary syndrome (ACS). Although it is difficult to differentiate SCAD from other causes of ACS based on the clinical presentation, some patient characteristics can aid the clinician in considering SCAD as a diagnosis. Namely, SCAD is far more common in women, especially in the peripartum period, and patients often do not have typical risk factors associated with atherosclerosis-related myocardial infarction. Furthermore, there is an association with FMD, migraine, and inherited connective tissue disorders. The primary pathophysiologic disturbance is the separation of layers of the coronary artery, either by hemorrhage from the vasa vasorum and formation of an intramural hematoma or by blood entering the vessel wall through an endothelial disruption thus creating a false lumen. The most common presentations are myocardial infarction, cardiogenic shock, and ventricular arrhythmias, while the most common presenting symptom is chest pain. The gold standard in diagnostics is coronary angiography. Angiography findings can be classified by the Yip-Saw classification into three categories, and the procedure should be approached with special attention due to the fragility of coronary arteries in these patients. Further imaging techniques that can be of use are OCT, IVUS, CTCA.

Initial management is directed toward treating the myocardial infarction and its complications. A significant portion of SCAD can be managed conservatively, and the PCI is used to maintain a grade 3 TIMI flow with minimal intervention. In case there are clinical or anatomical high-risk features present, the patient can be referred to CABG if it is available.

Other than managing the myocardial infarction, medical management plays a role in managing chronic chest pain, preventing recurrence of SCAD, treating extracoronary vascular disorders, and improving patients' quality of life. Patients with left ventricular dysfunction and patients after PCI are managed according to the current guidelines. Dual antiplatelet therapy is prescribed to all patients following PCI, in the acute phase of SCAD, and for up to 1 year to patients who are treated medically. After the first year, it is needed to tailor the treatment to each patient individually.

Aiming to preserve and improve the quality of patients' lives, they should receive regular follow-up and timely diagnosis and treatment for possible issues, which will, along with cardiovascular rehabilitation, aid in reaching this goal.

Kew words Spontaneous coronary artery dissection, acute coronary syndrome

Introduction

Spontaneous coronary artery dissection (SCAD) is one of the acute coronary syndrome causes. Once a rare phenomenon, SCAD is now a recognizable entity thanks to the use of coronary angiography and highly-sensitive troponins in the early diagnosis of acute coronary syndrome (ACS) and greater awareness of this disease. Although randomized controlled studies are still nonexistent, thanks to national SCAD registers from the last five years, a significant advance has been made in diagnosing and treating these patients[1]. This review paper will provide a revision of all the latest findings concerning the appropriate diagnostics, as well as the current therapeutic modalities.

Frequency and cause of SCAD

Although specific symptoms and signs that would help differentiate spontaneous dissection (SCAD) from other

Address for correspondence: Svetlana Apostolovic, MD, PhD, Clinic for cardiovascular diseases, Clinical center of Nis; Medical faculty, University of Nis.
E-mail: drapostolovicgmail.com

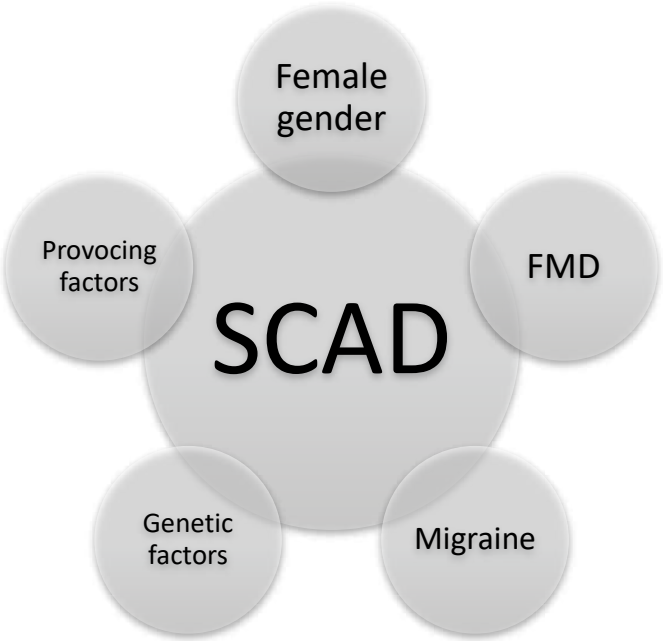

Figure 1. Causes of spontaneous dissection, including associated conditions. Provoking factors include emotional stress, certain medications, physical activity and Valsalva maneuver. SCAD (Spontaneous Dissection of Coronary Arteries); FMD (Fibromuscular Dysplasia).

common causes of acute coronary syndrome (ACS) do not exist, specific patient characteristics can direct us toward this diagnosis. A significantly lower prevalence of risk factors characteristic of the development of myocardial infarction associated with atherosclerosis was observed[2,3]. Hypertension is present in approximately 30% of SCAD patients, while the presence of diabetes is rare[4].

SCAD can present in both genders, with around 90% of patients being women aged 47-53[3,4,5,6], while approximately 10% of patients are men, as indicated in available literature[4,7,8]. It is a very rare occurrence in young patients (<25 years old) and older ones (>80 years old). It is a more common occurrence in patients suffering from hereditary connective tissue disorders or during pregnancy[9]. SCAD is responsible for around 23-68% of acute coronary syndromes during pregnancies, which is reflected as a common occurrence during the peripartum period[10,11]. Cohort studies have shown that SCAD might also appear in primiparous, multiparous, and pregnant women, in the postpartum and even postmenopausal period[2,4]. It remains to be determined whether the amount of estrogen and progesterone or their fluctuations have a role in the development of this entity[12]. It is assumed that estrogen's influence manifests itself through metalloproteinase activation, while progesterone's high level leads to weakening of the blood vessel wall[13].

Other provoking factors that could lead to SCAD development include strenuous physical activity in men and emotional distress in women[4,14,15]. The development of SCAD after using cocaine and after emesis was shown in various case reports[15,16,17].

Considering SCAD association with fibromuscular dysplasia (up to 50%), as well as migraine, the presence of these diseases in the patient's history can lead physicians toward SCAD[18]. Both clinical entities are more commonly seen in women. In addition, patients that suffer from connective tissue disorders (Marfan syndrome, Ehlers-Danlos, Loeys-Dietz), as well as Alport syndrome or adult polycystic kidney disease, or have a positive family history of these diseases, and experience signs and symptoms that resemble ACS, need to undergo a workup for SCAD. Gene sequencing showed that 3,5% of patients suffering from SCAD also suffer from one of the above-mentioned diseases[19].

Pathophysiology

The central pathophysiological event of spontaneous coronary artery dissection is the formation of a false lumen in the media that leads to separation of the coronary artery wall layers. As the name suggests, this disease is not associated with atherosclerosis, trauma, or iatrogenic injury but instead occurs spontaneously[1,18,21,22]. What divided SCAD into two of the largest groups is the presence of endothelial disruption and the formation of an intimal flap. Thus, the two hypotheses that explain the pathophysiology of this disease are proposed. The first is the so-called "inside-out" hypothesis, which suggests that blood enters the subintimal space through the endothelial disruption, thus creating the false lumen. However, back in 2002, with the help of intravascular ultrasound, an intramural hematoma without signs of endothelial disruption was noticed[23], leading to the creation of the second "outside-in" hypothesis that suggests that a hematoma can occur de novo, most likely as a consequence of microvasculature dysfunctions in the coronary artery wall[24,25,26]. After the initial development, ischemia and consequent necrosis due to luminal compression caused by axial propagation or the propagation of the intramural hematoma occur[1].

Clinical presentation

The clinical presentation of patients with SCAD is most similar to other causes of ACS. In over 90% of patients with SCAD, it manifests as a myocardial infarction (STEMI and NSTEMI)[4,8,27)], but its presentation can also be a ventricular arrhythmia (3-5%)[8,27-30] as well as cardiogenic shock (2%)[27]. By far, the most common symptom is chest pain, with the possibility of propagation to the neck, arms, or back, followed by dyspnea and sweating. Although certain factors can act as provocateurs for the appearance of symptoms (emotional stress, physical exertion), further differential-diagnostic measures must be taken with caution because these provoking factors are common for other clinical entities with a similar presentation (emotional stress – Takotsubo cardiomyopathy; physical exertion – atherosclerotic plaque rupture)[31,32].

Angiography

As patients with SCAD almost always show signs of ACS, the gold standard for diagnosing these patients is

Table 1. Characteristics of SCAD types[3]				
	Type I	**Type II**	**Type III**	**Type IV**
Angiographic appearance	Pathognomonic appearance of an arterial dissection with contrast entering the false lumen through the intimal flap; visible intimal flap	Narrowing of the lumen due to an intramural hematoma in abscence of an intimal tear; lesions usually long (>20mm) and characterized by sudden decrease in lumen size	Compression of the lumen by an intramural hematoma, but the lesions are usually <20mm, thus can resemble an atherosclerotic plaque	Complete oclussion of the coronary artery wall; may resemble thromboembolic oclussion
Frequency	29%	67%	4%	

Table 2. Differential diagnosis of SCAD	
Differential diagnosis	Comment
Atherosclerosis	May angiographically resemble SCAD types 1, 2 and 3. The presence of a thrombus distally to the stenosis, presence of calcifications or a lipid plaque lean toward the diagnosis of atherosclerosis.
Vasospasm	May resemble SCAD type 2; however, injecting intracoronary nitrates may confirm the diagnosis.
Embolization	Sudden oclussion may resemble SCAD type 4. Caution is needed during the differential diagnosis. The presence of an arteficial valve, proximal atherosclerotic plaque, coronary ectasis, shunt, hypercoagulable states lean toward the diagnosis of embolization.
Takotsubo cardiomyopathy	Apical akinesia is characteristic. Careful evaluation of terminal parts of LAD on angiography is needed.
Iatrogenic dissection	Rarely seen as an isolated phenomenon, usually traumatic in origin. It is well known that patients with SCAD have an increased tendency toward iatrogenic dissections (28).

coronary angiography. The dissection most commonly occurs in the left anterior descending artery (LAD), more specifically in the mid and distal segments. Multi-vessel SCAD appears in 5-13% of cases[1]. Angiographic findings can be classified into three groups according to the Yip-Saw classification (Table 1). However, certain classification deficiencies are appreciable as it encompasses only the most common angiographical characteristics. For that reason, some authors suggest a fourth group that would account for all the changes that do not fulfill the criteria for the first three, but lead to coronary artery occlusions[33].

A greater coronary artery tortuosity found during coronarography was observed in patients suffering from SCAD (78% as opposed to 17% with controlled subjects), and a clear coronary tortuosity can be used as a marker of recurrent SCAD[34]. Caution is needed when performing coronary angiography on patients with SCAD as their coronary artery walls are considered to be more fragile. Because of that, deep catheter cannulation should be avoided, and a careful injection of intravenous contrast is advised to avoid the risk of further propagation of the dissection.

Although the angiographic findings of SCAD are characteristic, there are certain challenging clinical scenarios in the sense of differential diagnosis that should be noted. Some of them are represented in Table 2.[35]

Post-angiography diagnostics (OCT, IVUS, CTCA)

These diagnostic techniques can be used when the diagnosis is not determined via an angiography[36,37]. Although superior, and with a better resolution, these diagnostic techniques carry the risk of certain complications, such as extensions of the current dissection, iatrogenic dissection, and false lumen cannulation — thus should be performed with additional caution. According to the current data, these complications can occur in 8% of patients with SCAD[38]. For this reason, routine use of these techniques is not recommended. However, if SCAD is suspected but its presence cannot be determined through an angiography, the use of these techniques is justified.

IVUS is superior compared to OCT. It enables deeper penetration and a complete assessment of the degree of blood vessel involvement[35]. A pathognomonic sign of SCAD is the so-called triple-band of the intimal-medial membrane. The limitation of this method is the inability to differentiate SCAD and lipid-rich atheromas due to its lower resolution (which is not the case with OCT)[35].

Despite the shallow penetration, OCT can confirm a SCAD diagnosis due to its excellent resolution and visualization of the real and false lumen, dissection flap, and intramural hematoma. During the intervention, closure of the flap is necessary to ensure favorable long-term results. An OCT is the only technique that can show whether, after stent placement and/or tracing, *restitutio ad integrum* of the coronary wall is present after spontaneous dissection[40].

The use of CCTA in the initial SCAD diagnosis is still a controversial topic. CCTA can be of use in the visualization of dissection flaps, intramural hematomas, and stenoses, especially in the proximal segments of coronary arteries[18,41]. However, its specificity and sensitivity are still unknown, and the few available case reports provide information about its false-negative results[42-44].

The primary role of this diagnostic technique can be in the follow-up of patients with SCAD, especially those with recurrent chest pain without evidence of

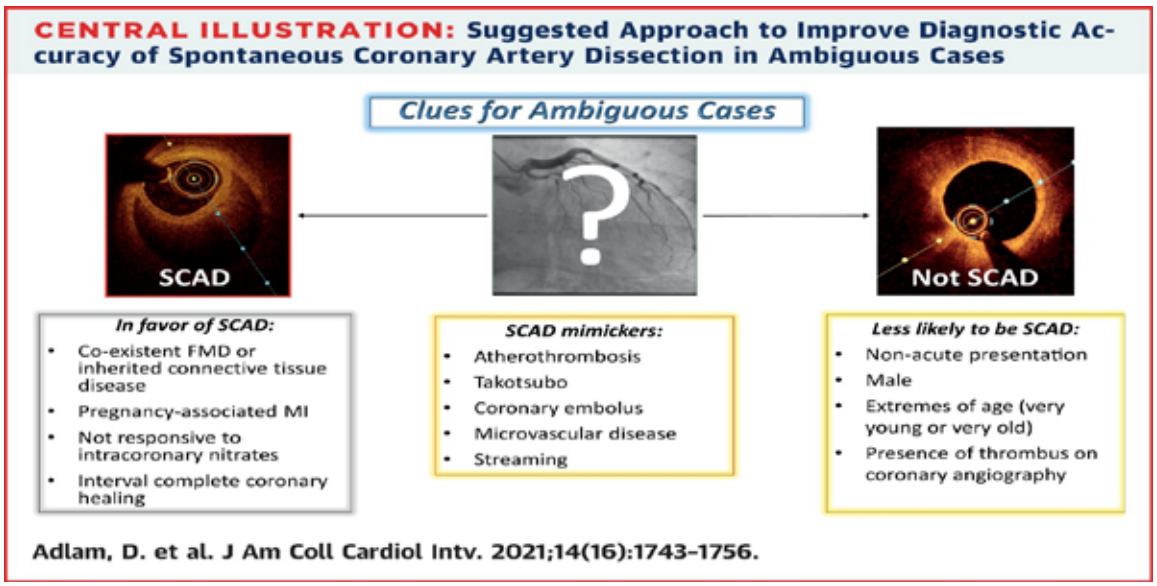

Figure 2. Recommended approach in unclear cases of SCAD. SCAD (Spontaneous Dissection of Coronary Arteries); FMD (Fibromuscular Dysplasia); MI (Myocardial Infarction). From: Adlam D, Tweet MS, Gulati R, Kotecha D, Rao P, Moss AJ, et al. Spontaneous coronary artery dissection. JACC: Cardiovascular Interventions. 2021;14[16]:1743–56.[35]

myocardial ischemia or necrosis. Nevertheless, more research and experience are needed in this direction[44]. In some cases, angiography and intracoronary imaging techniques can yield negative results while the presentation is still leaning toward SCAD. In such cases, some clinical and demographic characteristics can favor SCAD, and follow-up angiography (invasive or CCTA) can help discover the cause of acute coronary syndrome[1].

Therapy
Myocardial infarction treatment

Initial treatment of patients suffering from SCAD focuses on preserving or restoration of coronary blood flow and myocardial function. Thrombolytic therapy should be avoided, considering the possibility of dissection propagation or hematoma. Any decisions concerning the patient's treatment are made in regards to the clinical findings, such as the patient's hemodynamic stability along with the coronary angiography results which can show the extensiveness of the ischemic myocardium, as well as the degree of the distal flow in the affected blood vessel. High risk clinical characteristics are persistent chest pain with proof of ischemic changes, hemodynamic instability, shock, or clinically significant ventricular arrhythmias, whereas high risk anatomical features are more affected blood vessels with more significant proximal dissections or affected left coronary or LAD artery[1,18,45]. Due to a significant occurrence of SCAD in pregnant women, it is important to mention that any risk to the fetus is negligible with appropriate preventive measures; thus, the standard treatment of myocardial infractions is also recommended for pregnant women[46].
Due to pathophysiological differences in the mechanisms of SCAD and myocardial infractions due to atherosclerotic plaque rupture, along with the fact that all medically treated SCAD cases show an angiographically proven flow restitution with decreasing stenosis levels[7,47,48], the majority of patients suffering from SCAD can be conservatively treated, especially hemodynamically stable ones[1,18].

Percutaneous coronary intervention with SCAD

PCI outcomes in SCAD patients are less predictable and associated with a higher risk of complications compared to patients with atherosclerotic myocardial infarction[1,7,18,27], as well as a higher risk of iatrogenic dissections, sudden blood vessel occlusions, or hematoma propagation[49]. Therefore, when the SCAD diagnosis is confirmed, further instrumentalization is not necessary in case of minimal ischemia or distally affected coronary artery. The aim is to sustain TIMI flow grade 3 with minimal intervention, while revascularization is reserved for patients with STEMI, ongoing ischemia, proximal occlusions, unstable heart rate or hemodynamic status, heart failure, as well as those that progress to occlusion after the initial conservative treatment[18,24,50].
When high-risk features are present, it is necessary to consider whether the patient is a candidate for revascularization through PCI or coronary artery bypass grafting (CABG). It is important to emphasize that stent placement with a PCI procedure carries the risk of subsequent stent malposition due to resorption of the intramural hematoma over time[51].

Surgical myocardial revascularization in SCAD

This type of intervention is typically applicable to patients where the PCI procedure was unsuccessful or it was estimated that the risk would be too severe (for example, left coronary artery dissection with ongoing

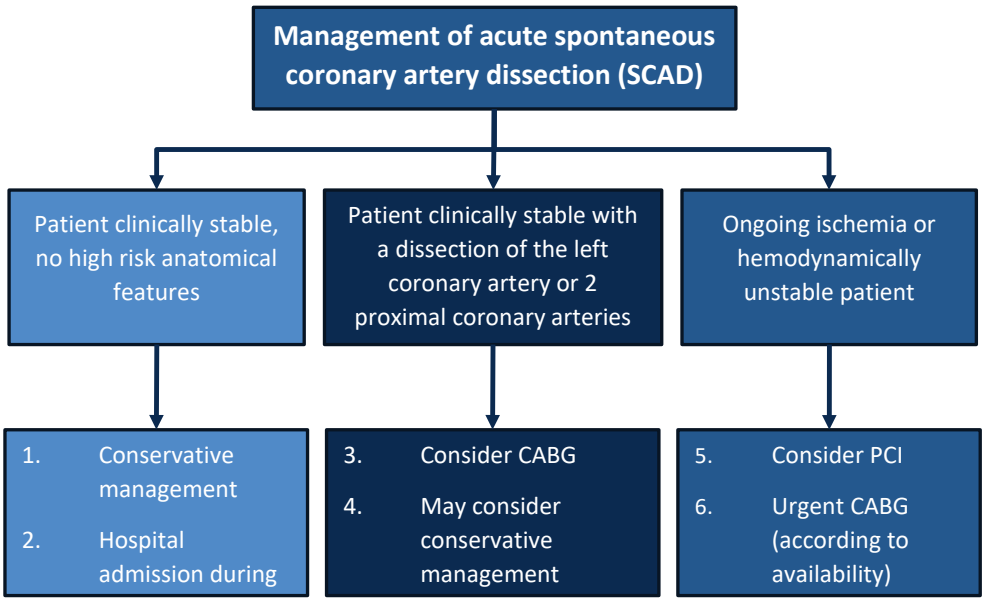

Figure 3. Management algorithm for SCAD. SCAD (Spontaneous Dissection of Coronary Arteries); CABG (Coronary Artery Bypass Grafting); PCI (Percutaneous Coronary Intervention) From: Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, et al. Spontaneous coronary artery dissection. Journal of the American College of Cardiology. 2020;76[8]:961–84.[26]

infarction). The procedure could be technically demanding due to the fragility of the tissue affected by the dissection, especially in patients with hereditary connective tissue disorders. Although this procedure's success rate is high, long-term maintenance of graft patency is not common[7] due to the recanalization of the coronary artery and consequent competitive flow that leads to graft occlusion. For this reason, using venous grafts is recommended for this type of intervention, with the aim of preserving arterial grafts for possible future uses[18].
Patients with severe forms of the disease, including cardiogenic shock and acute myocardial infarction complications due to SCAD, can benefit from measures of advanced mechanical circulatory support with intra-aortic balloon pumps or devices for extracorporeal membrane oxygenation until the patient becomes stable or receives a heart transplant[52,53].

Medical management of acute coronary syndrome caused by SCAD

Except for its role in the treatment of acute myocardial infarction, drug therapy has a role in treating chronic chest pain, preventing SCAD recurrence, treating extracoronary vascular disorders, and improving patients' quality of life.
Patients with left ventricular dysfunction are treated according to current recommendations for treating heart failure (beta-blockers and ACE inhibitors or angiotensin receptor blockers)[18,54]; whereas, patients who underwent a stent placement receive therapy in accordance with recommendations for treatment after PCI[55]. It is also necessary to treat hypertension as it is associated with recurrent SCAD[28].
According to the latest recommendations for the treatment of acute myocardial infarction[55,56], anticoagulant and dual antiplatelet therapy are commonly prescribed to patients before being diagnosed with SCAD. However, based on expert consensus, anticoagulant therapy should be ceased upon diagnosing SCAD on angiography[1,18]. The use of thrombolytic therapy for treating acute SCAD is not recommended[1].
Dual antiplatelet therapy is prescribed to all patients following PCI[55,56]. Grounded on expert recommendations, this therapy can be used during the acute phase of SCAD**,** as well as during the following year in patients treated conservatively[18]. Nevertheless, a recent study demonstrated a significantly higher frequency of major adverse cardiovascular events (MACE) in conservatively treated patients that were on dual antiplatelet therapy compared to those prescribed a single antiplatelet medication. As a result, further research and possible reconsideration of this therapeutic modality is necessary[57].
Due to insufficient evidence, the length of antiplatelet therapy after the first year should be adjusted according to individual patient characteristics. For instance, aspirin could be prescribed to patients with FMD to prevent thrombotic and thromboembolic complications. On the other hand, perimenopausal women suffering from menorrhagia could benefit from a shorter duration of antiplatelet therapy in lack of other indications for this treatment[58].
No evidence favors routine use of statins after SCAD, leading to this medication being reserved for patients with hyperlipidemia. As for the antianginal medications, only nitrates, calcium channel blockers, and ranolazine can be considered[18]. To prevent SCAD recurrence, beta-blockers can be used as there are indications of their benefits in this regard[28]. Factors related to recurrent SCAD are hypertension[28], FMD, migraine[3], and coronary artery tortuosity[34]. For the purpose of preventing SCAD recurrence, patients are recommended avoiding

strenuous physical activity, along with activities that involve the prolonged Valsalva maneuver[1,18].
To preserve and improve the patients' quality of life, special attention should be given to the signs and symptoms of migraines, anxiety, depression, and post-traumatic stress disorder, which are common occurrences after SCAD and significantly influence the quality of life[60]. That being said, screening with the goal of early detection of these conditions and treating patients can tremendously affect the patient's recovery. Cardiovascular rehabilitation is also of importance and is recommended for all patients following SCAD events[18].

Conclusion

SCAD is a rare but significant cause of acute myocardial infarction (AMI), which affects women more frequently. It differs pathophysiologically from the acute myocardial infarction associated with atherosclerosis, and the basis of acute coronary artery stenosis is a hematoma rather than a thrombus. It is important to think about this cause of AMI, especially in women under the age of 55, who are without risk factors for atherosclerotic disease. Myocardial revascularization is the treatment of choice in case of proximal coronary artery occlusion, ongoing ischemia, or cardiogenic shock. Drug treatment is preferred over percutaneous intervention. Patients with SCAD-induced AMI who are treated conservatively, primarily with short-term anticoagulation therapy and a single antithrombotic agent as a long-term treatment, had a better prognosis, according to recent observational studies. In over 85% of SCAD patients treated with medication, control angiography after 30 days shows spontaneous healing of the coronary artery.

Refrences

1. Adlam D, Alfonso F, Maas A, et al. 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):3353-68.
2. Gad MM, Mahmoud AN, Saad AM, et al. Incidence, clinical presentation, and causes of 30-day readmission following hospitalization with spontaneous coronary artery dissection. JACC Cardiovasc Interv 2020;13:921-32.
3. Clare R, Duan L, Phan D, et al. Characteristics and clinical outcomes of patients with spontaneous coronary artery dissection. J Am Heart Assoc 2019;8(10): e012570.
4. Saw J, Starovoytov A, Humphries K, et al. Canadian spontaneous coronary artery dissection cohort study: in-hospital and 30-day outcomes. Eur Heart J 2019;40:1188–97.
5. García-Guimaraes M, Bastante T, Macaya F, et al. Spontaneous coronary artery dissection in Spain: clinical and angiographic characteristics, management, and in-hospital events. Rev Esp Cardiol (Engl Ed) 2020 May 14
6. Kok SN, Hayes SN, Cutrer FM, et al. Prevalence and clinical factors of migraine in patients with spontaneous coronary artery dissection. J Am Heart Assoc 2018;7(24):e010140.
7. Tweet MS, Eleid MF, Best PJ, et al. Spontaneous coronary artery dissection: revascularization versus conservative therapy. Circ Cardiovasc Interv. 2014;7:777–86.
8. Sharma S, Kaadan MI, Duran JM, et al. Risk factors, imaging findings, and sex differences in spontaneous coronary artery dissection. Am J Cardiol. 2019;123:1783–7.
9. Russo V, Marrozzini C, Zompatori M. Spontaneous coronary artery dissection: role of coronary CT angiography. Heart 2013; 99:672–3.
10. Elkayam U, Jalnapurkar S, Barakkat MN, et al. Pregnancy-associated acute myocardial infarction: a review of contemporary experience in 150 cases between 2006 and 2011. Circulation 2014;129: 1695–1702.
11. Smilowitz NR, Gupta N, Guo Y, et al. Acute myocardial infarction during pregnancy and the puerperium in the United States. Mayo Clin Proc 2018;93:1404–14.
12. Kaadan MI, MacDonald C, Ponzini F, et al. Prospective cardiovascular genetics evaluation in spontaneous coronary artery dissection. Circ Genom Precis Med 2018;11:e001933.
13. Slight R, Behranwala AA, Nzewi O, Sivaprakasam R, Brackenbury E, Mankad P. Spontaneous coronary artery dissection: a report of two cases occurring during menstruation. N Z Med J 2003; 116:U585.
14. Tweet M, Hayes S, Pitta S, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012;126: 579–88.
15. 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;7:645–55.
16. Tofler GH, Kopel E, Klempfner R, et al. Triggers and timing of acute coronary syndromes. Am J Cardiol 2017;119:1560–5.
17. Steinhauer JR, Caulfield JB. Spontaneous coronary artery dissection associated with cocaine use: a case report and brief review. Cardiovasc Pathol 2001;10:141–5.
18. Hayes SN, Kim ESH, Saw J, et al. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. Circulation 2018;137(19): e523- e557.
19. Carss KJ, Baranowska AA, Armisen J, et al. Spontaneous coronary artery dissection: insights on rare genetic variation from genome sequencing. Circ Genom Precis Med 2020;13: e003030.
20. Amrani-Midoun A, Adlam D, Bouatia-Naji N. Recent advances on the genetics of spontaneous coronary artery dissection. Circulation: Genomic and Precision Medicine. 2021; in press
21. Basso C, Morgagni GL, Thiene G. Spontaneous coronary artery dissection: a neglected cause of acute myocardial ischaemia and sudden death. Heart 1996; 75:451-4.
22. Vrints CJM. Spontaneous coronary artery dissection. Heart 2010; 96:801-8.
23. Maehara A, Mintz GS, Castagna MT, et al. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol 2002;89:466-8.
24. Waterbury TM, Tweet MS, Hayes SN, et al. Early natural history of spontaneous coronary artery dissection. Circ Cardiovasc Interv 2018;11: e006772.
25. Waterbury TM, Tarantini G, Vogel B, Mehran R, Gersh BJ, Gulati R. Non-atherosclerotic causes of acute coronary syndromes. Nat Rev Cardiol 2020; 17:229–41.
26. Hayes SN, Tweet MS, Adlam D, et al. Spontaneous coronary artery dissection. Journal of the American College of Cardiology. 2020;76(8):961–84.
27. Lettieri C, Zavalloni D, Rossini R, et al. Management and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 2015;116:66-73.
28. Saw J, Humphries K, Aymong E, et al. Spontaneous coronary artery dissection: clinical outcomes and risk of recurrence. J Am Coll Cardiol 2017;70:1148-58.
29. Phan D, Clare R, Duan L, Kim C, Moore N, Lee MS. Characteristics and outcomes of patients with spontaneous coronary artery dissection who suffered sudden cardiac arrest. J Interv Card Electrophysiol 2020 February 11 (Epub ahead of print).
30. Cheung CC, Starovoytov A, Parsa A, et al. In-hospital and long-term outcomes among patients with spontaneous coronary artery dissection presenting with ventricular tachycardia/fibrillation. Heart Rhythm 2020;17:1864-9.
31. Ghadri JR, Wittstein IS, Prasad A, et al. International expert consensus document on takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J 2018;39:2032–46.
32. Franklin BA, Thompson PD, Al-Zaiti SS, et al. Exercise-related acute cardiovascular events and potential deleterious adaptations

following longterm exercise training: placing the risks into perspective-an update: a scientific statement from the American Heart Association. Circulation 2020;141:e705–e736.
33. Al-Hussaini A, Adlam D. Spontaneous coronary artery dissection. Heart 2017;103:1043–51.
34. Eleid MF, Guddeti RR, Tweet MS, et al. Coronary artery tortuosity in spontaneous coronary artery dissection: angiographic characteristics and clinical implications. Circ Cardiovasc Interv, 2014, Oct 7 (5): 656-62.
35. Adlam D, Tweet MS, Gulati R, et al. Spontaneous coronary artery dissection. JACC: Cardiovascular Interventions. 2021;14(16): 1743–56.
36. Arnold JR, West NE, van Gaal WJ, Karamitsos TD, Banning AP. The role of intravascular ultrasound in the management of spontaneous coronary artery dissection. Cardiovasc Ultrasound 2008;6:24.
37. Alfonso F, Paulo M, Gonzalo N, et al. Diagnosis of spontaneous coronary artery dissection by optical coherence tomography. J Am Coll Cardiol 2012;59:1073-9.
38. Jackson R, Al-Hussaini A, Joseph S, et al. Spontaneous coronary artery dissection: pathophysiological insights from optical coherence tomography. JACC Cardiovasc Imaging 2019;12:2475-88.
39. De Maio SJ, Kinsella SH, Silvermann ME. Clinical course and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 1989;64:471-74.
40. Apostolović S, Pavlović M, Šalinger S, et al. Spontaneous dissection of coronary arteries as a cause of acute coronary syndrome. Srce i krvni sudovi 2013;32(1): 2-13.
41. Pozo-Osinalde E, García-Guimaraes M, Bastante T, et al. Characteristic findings of acute spontaneous coronary artery dissection by cardiac computed tomography. Coron Artery Dis 2020;31: 293-9.
42. Eleid MF, Tweet MS, Young PM, Williamson E, Hayes SN, Gulati R. Spontaneous coronary artery dissection: challenges of coronary computed tomography angiography. Eur Heart J Acute Cardiovasc Care 2017; 2048872616687098.
43. Torres-Ayala SC, Maldonado J, Bolton JS, Bhalla S. Coronary computed tomography angiography of spontaneous coronary artery dissection: a case report and review of the literature. Am J Case Rep 2015;16:130–135.
44. Alzand BS, Vanneste L, Fonck D, Van Mieghem C. Spontaneous coronary artery dissection undissolved using cardiac computed tomography. Int J Cardiol 2016; 222:1040–1041.
45. Saw J, Mancini GBJ, Humphries KH. Contemporary review on spontaneous coronary artery dissection. J Am Coll Cardiol 2016; 68:297-312.
46. Cauldwell M, Baris L, Roos-Hesselink JW, Johnson MR. Ischaemic heart disease and pregnancy. Heart 2019;105:189–95.
47. Hassan S, Prakash R, Starovoytov A, Saw J. Natural history of spontaneous coronary artery dissection with spontaneous angiographic healing. JACC Cardiovasc Interv 2019;12:518-27.
48. Rogowski S, Maeder MT, Weilenmann D, et al. Spontaneous coronary artery dissection: angiographic follow-up and longterm clinical outcome in a predominantly medically treated population. Catheter Cardiovasc Interv 2017;89:59-68.
49. Prakash R, Starovoytov A, Heydari M, Mancini GB, Saw J. Catheter-induced iatrogenic coronary artery dissection in patients with spontaneous coronary artery dissection. J Am Coll Cardiol Intv 2016;9:1851–3.
50. Krittanawong C, Gulati R, Eitzman D, Jneid H. Revascularization in patients with spontaneous coronary artery dissection: Where are we now? JAHA 2021;10(13).
51. Lempereur M, Fung A, Saw J. Stent mal-apposition with resorption of intramural hematoma with spontaneous coronary artery dissection. Cardiovasc Diagn Ther 2015;5:323-9.
52. Cox J, Roberts WC, Araj FG, et al. Acute isolated coronary artery dissection causing massive acute myocardial infarction and leading to unsuccessful coronary bypass, extracorporeal life support, and successful cardiac transplantation. Am J Cardiol 2020;125:1446-8.
53. Sharma S, Polak S, George Z, et al. Management of spontaneous coronary artery dissection complicated by cardiogenic shock using mechanical circulatory support with the Impella device. Catheter Cardiovasc Interv 2019 December 26 (Epub ahead of print).
54. Bolen MA, Brinza E, Renapurkar RD, Kim ESH, Gornik HL. Screening CT angiography of the aorta, visceral branch vessels, and pelvic arteries in fibromuscular dysplasia. J Am Coll Cardiol Img 2017;10:554–61.
55. Amsterdam E, Wenger N, Brindis R, et al. 2014 AHA/ACC guidelines for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;130:2354–94.
56. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61(4): e78-e140.
57. Cerrato E, Giacobbe F, Quadri G, Macaya F, Bianco M, Mori R, et al. Antiplatelet therapy in patients with conservatively managed spontaneous coronary artery dissection from the multicentre disco registry. Eur Heart J 2021;42(33):3161–71.
58. Maas AHEM, Euler M, Bongers MY, et al. Practice points in gynecardiology: abnormal uterine bleeding in premenopausal women taking oral anticoagulant or antiplatelet therapy. Maturitas 2015;82: 355-9.
59. Kim ESH. Spontaneous coronary-artery dissection. New England Journal of Medicine. 2020;383(24):2358–70.
60. Johnson AK, Hayes SN, Sawchuk C, et al. Analysis of posttraumatic stress disorder, depression, anxiety, and resiliency within the unique population of spontaneous coronary artery dissection survivors. J Am Heart Assoc 2020;9(9):e014372.

Sažetak

Spontana disekcija koronarnih arterija: Šta znamo do sada?

Svetlana Apostolović[1,2], Stefan Milutinović[2], Tomislav Kostić[1,2], Miroslav Nikolić[1], Miljan Krstović[2], Milovan Petrović[3], Ivan Ilić[4], Zlatko Mehmedbegović[5], Dejan Milašinović[5], Aleksandra Djoković[9], Vanja Miloradović[8], Nemanja Djenić[7], Vladimir Mitov[8], Nikola Jagić[6], Zoran Perišić[1], Aleksandar Nešković[6]

[1]Klinika za kardiovaskularne bolesti, Univeryzitetski Klinički centar Niš, [2]Medicinski fakultet, Univerzitet u Nišu, [3]Institut za kardiovaskularne bolesti Vojvodina, Medicinski fakultet, Univerzitet u Novom Sadu, [4]Institut za kardiovaskularne bolesti Dedinje, [5]Univerzitetski Klinički centar Srbije, Klinika za kardiologiju, [6]Kliničko bolnički centar Zemun, [7]Vojno Medicinska akademija, [8]Opšta bolnica Zaječar, [9]Kliničko bolnički centar Bežanijska Kosa

Spontana disekcija koronarne arterije (SCAD) je jedan od uzroka akutnog koronarnog sindroma (AKS). Iako je teško razlikovati SCAD od drugih uzroka AKS na osnovu kliničke slike, neke karakteristike pacijenata mogu pomoći kliničaru da SCAD razmotri kao dijagnozu. Naime, SCAD je daleko češći kod žena, posebno u peripartalnom periodu, a pacijentkinje često nemaju tipične faktore rizika povezane sa infarktom miokarda izazvanom aterosklerozom. Štaviše, postoji povezanost sa FMD, migrenom i naslednim poremećajima vezivnog tkiva.

Primarni patofiziološki poremećaj je odvajanje slojeva koronarne arterije, bilo krvarenjem iz vasa vasorum i formiranjem intramuralnog hematoma ili ulaskom krvi u zid suda kroz endotelni poremećaj stvarajući lažni lumen.

Najčešći simptomi su infarkt miokarda, kardiogeni šok i ventrikularne aritmije, dok je najčešći simptom bol u grudima. Zlatni standard u dijagnostici je koronarna angiografija. Nalaz angiografije se prema Iip-Sav klasifikaciji može klasifikovati u tri kategorije, a proceduri treba pristupiti sa posebnom pažnjom zbog krhkosti koronarnih arterija kod ovih pacijenata. Dodatne tehnike snimanja koje mogu biti od koristi su OCT, IVUS, CTCA.
Početni tretman je usmeren na lečenje infarkta miokarda i njegovih komplikacija. Značajnim delom SCAD-a može se upravljati konzervativno, a PCI se koristi za održavanje protoka TIMI stepena 3 uz minimalnu intervenciju. U slučaju da postoje kliničke ili anatomske karakteristike visokog rizika, pacijent se može uputiti na CABG ako je dostupan.
Osim lečenja infarkta miokarda, posebnu ulogu ima i lečenje hroničnog bola u grudima, sprečavanju ponavljanja SCAD-a, lečenju ekstrakoronarnih vaskularnih poremećaja i poboljšanju kvaliteta života pacijenata. Pacijenti sa disfunkcijom leve komore i pacijenti posle PCI leče se u skladu sa važećim smernicama. Dualna antitrombocitna terapija se propisuje svim pacijentima nakon PCI, u akutnoj fazi SCAD-a i do 1 godine pacijentima koji se leče medicinski. Nakon prve godine potrebno je prilagoditi tretman svakom pacijentu pojedinačno.
U cilju očuvanja i poboljšanja kvaliteta života pacijenata, pacijenti treba da budu redovno praćeni i blagovremeno dijagnostikovani što će, uz kardiovaskularnu rehabilitaciju, pomoći u postizanju ovog cilja.

Ključne reči: Spontana disekcija koronarnih arterija, akutni koronarni sindrom