Ponavljajuća spontana disekcija koronarnih arterija u bolesnice srednje životne dobi

A Middle-Aged Woman with Recurrent Spontaneous Coronary Artery Dissection

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SAŽETAK: Spontana disekcija koronarnih arterija rijedak je, ali dobro poznat uzrok akutnog infarkta miokarda. Češća je u žena reproduktivne dobi i u bolesnika s fibromuskularnom displazijom. Liječenje se sastoji od kontrole čimbenika kardiovaskularnog rizika te primjene vazodilatatora i beta-blokatora. Predstavljamo slučaj 48-godišnje bolesnice s infarktom miokarda uzrokovanim spontanim disekcijom i ponovnom pojavom bolesti zbog njezina širenja u kasnijemu stadiju.

SUMMARY: Spontaneous coronary dissection is an infrequent but well-described cause of acute myocardial infarction. It is associated with women of reproductive age or patients with fibromuscular dysplasia. Treatment consists of controlling cardiovascular risk factors, vasodilators, and beta-blockers. We present a case of 48-year-old patient with myocardial infarction secondary to spontaneous dissection and recurrence due to disease extension at a later stage.

KLJUČNE RIJEČI: spontana disekcija koronarnih arterija, infarkt miokarda, liječenje.

KEYWORDS: spontaneous coronary dissection, myocardial infarction, treatment.

CITATION: Cardiol Croat. 2023;18(9-10):265-70. | https://doi.org/10.15836/ccar2023.265

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TO CITE THIS ARTICLE: Sánchez Amaya DJ, Godínez Córdova LB, Ramos Coria DA, Gopar-Nieto R, Sierra Lara-Martínez JD. A Middle-Aged Woman with Recurrent Spontaneous Coronary Artery Dissection. Cardiol Croat. 2023;18(9-10):265-70. | https://doi.org/10.15836/ccar2023.265

TO LINK TO THIS ARTICLE: https://doi.org/10.15836/ccar2023.265

RECEIVED:
May 29, 2023
UPDATED:
June 30, 2023
ACCEPTED:
July 15, 2023

Edukacijski cilj

Nisu svi infarkti miokarda uzrokovani aterosklerotskom bolesti. Postoje i drugi mehanizmi, primjerice oni vezani uz epikardijalne krvne žile, kao što je spontana disekcija koronarne arterije, iako nisu česti pa je njihova ponovna pojava manje vjerojatna. Uporaba reproducibilnog i pouzdanog oslikavanja omogućuje točnu klasifikaciju bolesnika kako bi im se odredili primjerena prognoza i praćenje nakon liječenja.

Learning objective

Not all acute myocardial infarctions are due to atherosclerotic disease. There are other mechanisms, such as those related to epicardial vasculature, like spontaneous coronary artery dissection, although they are not frequent and less likely to recur. Having reproducible and reliable imaging studies allows adequate classification of patients in order to provide appropriate prognosis and follow-up.

Uvod

Spontana disekcija koronarnih arterija (SCAD) definira se kao odvajanje slojeva stijenke epikardijalne koronarne arterije zbog intramuralnog krvarenja koje nije povezano s aterosklerozom, jatrogenim ozljedama ili traumom, a može dovesti do akutnog koronarnog sindroma (AKS), aritmija, kardiomiopatije i iznenadne srčane smrti[1,2]. Intramuralno krvarenje (IMH) uzrokuje kompresiju lumena te posljedičnu ishemiju distalnog miokarda; IMH može nastati zbog disrupcije intimalnoga sloja kao početne promjene ili zbog puknuća vase vasorum. Konvencionalna koronarna angiografija dijagnostička je metoda prvog izbora, no primjena intravaskularnog oslikavanja kao što je intravaskularni ultrazvuk (IVUS) ili optička koherencijska tomografija (OCT) te pokatkad i koronarne kompjutorizirane tomografske angiografije (CCTA) također omogućuje preciznu dijagnozu i razvoj strategije praćenja. Najčešće se preferira konzervativno liječenje, dok god nisu prisutne ishemija ili hemodinamska nestabilnost. Ponavljajuća spontana disekcija koronarnih arterija (R-SCAD) rijedak je klinički entitet, no pravodobno prepoznavanje od velike je epidemiološke i terapijske važnosti.

Introduction

Spontaneous coronary dissection (SCAD) is defined as the separation of the layers of the wall of an epicardial coronary artery due to intramural hemorrhage (IMH), not related to atherosclerosis, iatrogenic injury, or trauma, and can lead to acute coronary syndrome (ACS), arrhythmias, cardiomyopathy, and sudden cardiac death[1,2]. Intramural hemorrhage causes compression of the lumen and subsequent ischemia of the distal myocardium; IMH can occur due to disruption of the intimal layer as an initial change or due to rupture of the vasa vasorum. Conventional coronary angiography (CA) is the diagnostic method of choice, but the use of intravascular imaging studies such as intravascular ultrasound (IVUS) or optical coherence tomography (OCT) and occasionally coronary computed tomography angiography (CCTA) also allows for precise diagnosis and follow-up strategy. Conservative management is typically preferred, as long as there is no ongoing ischemia or hemodynamic instability. Recurrent-SCAD (R-SCAD) is an infrequent entity, however, identifying it is important for both epidemiological and therapeutic purposes.

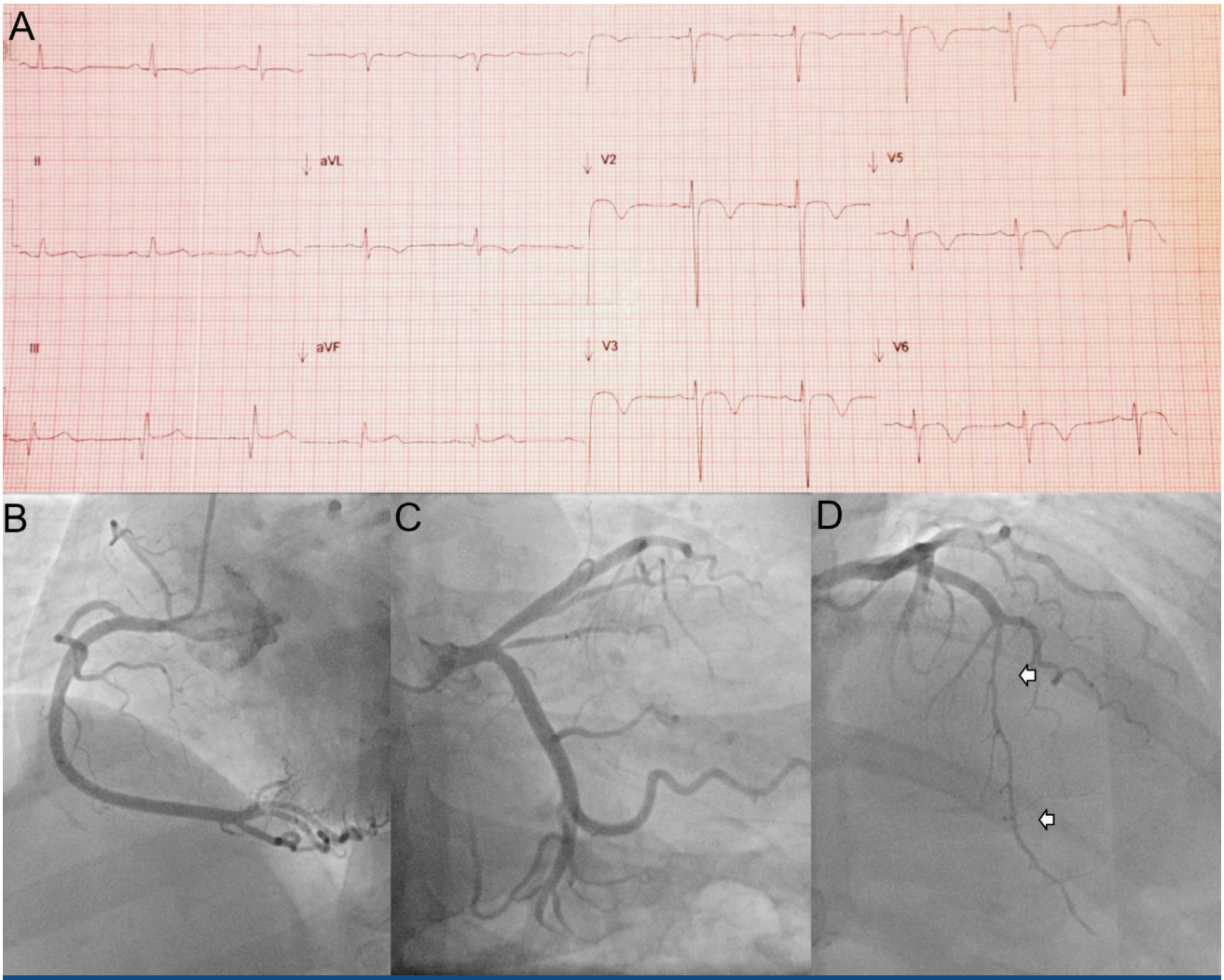

FIGURE 1. 12-lead electrocardiogram, Wellens pattern, type 2. B), C), and D) - coronary angiography: type 2 spontaneous coronary dissection along the mid distal segments of the left anterior descending artery (black arrows).

Prikaz slučaja

Četrdesetosmogodišnja bolesnica hispanskog podrijetla javila se na odjel hitne službe (HS) zbog prekordijalnih boli koje su trajale 10 sati. Nijekala je prisutnost bilo kakvih komorbiditeta. Pri dolasku vitalni su joj znakovi bili stabilni, a fizikalni je pregled bio uredan. Međutim, 12-kanalni elektrokardiogram pokazao je simetrično invertirane T-valove u prekordijalnim odvodima, što je kompatibilno s Wellensovim sindromom tipa b (**slika 1A**). Inicijalni laboratorijski nalazi pokazali su vrijednost visokoosjetljivog troponina od 5808 ng/mL te NT-proBNP-a od 304 pg/mL. Nakon postavljanja dijagnoze infarkta miokarda bez elevacije ST-segmenta (NSTEMI) započeto je liječenje dvojnom antitrombotskom terapijom (DAPT) koja se sastojala od acetilsalicilatne kiseline i klopidogrela, uz intenzivnu dozu statina (atorvastatin 80 mg) te enoksaparina 1mg/kg supkutano dva puta dnevno. Rana koronarna angiografija (**slika 1B** i **slika 1C**) pokazala je naglo smanjenje promjera u srednjem segmentu lijeve prednje silazne arterije (LAD), što

Case report

48-year-old Hispanic woman presented to the emergency department (ED) with 10 hours of anginal chest pain. She denied any comorbidities. On arrival, her vital signs were stable and physical examination was unremarkable. However, the 12-lead electrocardiogram showed symmetrical inverted T-waves in precordial leads, compatible with Wellens type B pattern (**Figure 1 A**). Initial laboratory tests showed high-sensitivity troponin of 5808 ng/mL and NT-proBNP of 304 pg/mL. After diagnosis of non-ST elevation acute myocardial infarction (NSTEMI), dual antiplatelet therapy (DAPT) consisting of aspirin and clopidogrel, high-intensity statin (atorvastatin 80 mg), and enoxaparin 1mg/kg subcutaneously two times a day was initiated. An early CA (**Figure 1 B** and **Figure 1 C**) revealed an abrupt diameter reduction in the mid-segment of the left anterior descending artery (LAD), suggestive of hematoma secondary to type 2 SCAD. However, intravascular imaging

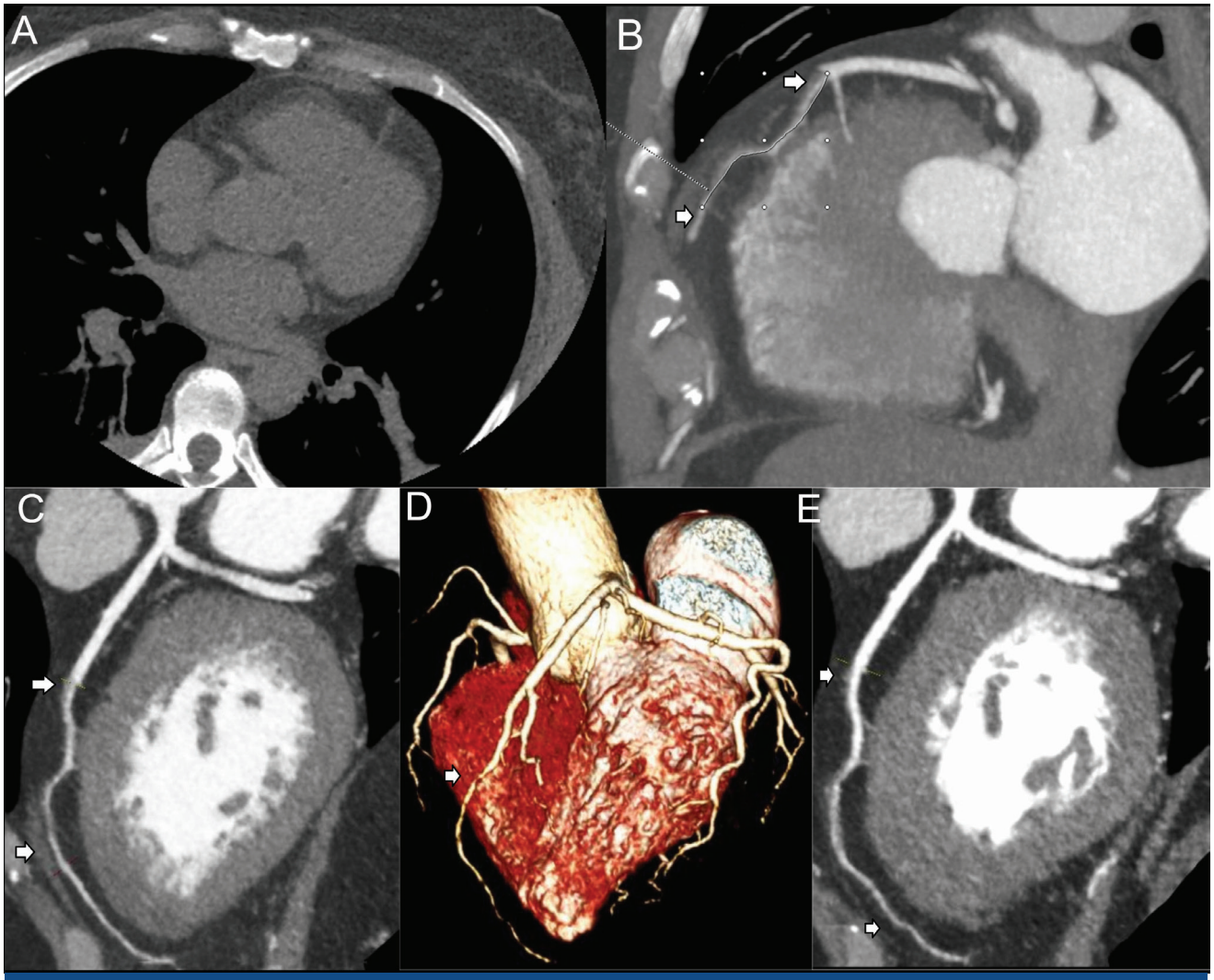

FIGURE 2. A) Computed tomography angiography – no contrasted acquisition images show absence of any coronary calcification. B) and C) coronary computed tomography angiography – sagittal view shows diffuse spontaneous coronary dissection (SCAD) in the mid and distal segment of left anterior descending artery (LAD) (white arrows), and D) volumetric reconstruction of the heart and coronary arteries, where LAD SCAD is clearly seen (white arrow). E) curved multiplanar reconstruction obtained in a second CCTA show LAD trajectory, with SCAD extension (white arrows).

upućuje na hematom uzrokovan SCAD-om tipa 2. Međutim, intravaskularno oslikavanje nije bilo moguće zbog znatnog suženja. CCTA pregledom (**slika 2A** i **slika 2B**) potvrđeni su SCAD tipa 2 dužine 48 mm i smanjenje lumena od 60 do 70 % u srednjem i distalnom segmentu LAD-a te vrijednost kalcija od 0 UA. Transtorakalna ehokardiografija pokazala je koncentričnu hipertrofiju lijeve klijetke (LV), masu LV-a od 139 g/m^2 te relativnu debljinu stijenke od 0,43, očuvanu istisnu frakciju LV-a (55 %), diskineziju apeksa te anteriornu i septalnu apikalnu akineziju kongruentnu sa zahvaćenom arterijom. Dodatne laboratorijske pretrage pokazale su vrijednost LDL-a od 1,6 mmol/L, vrijednost ukupnog kolesterola od 2,72 mmol/L te vrijednost HDL kolesterola od 0,58 mmol/L. Bolesnica je liječena enalaprilom 10 mg dvaput na dan i bisoprololom 5 mg jednom na dan, te je otpuštena nakon pet dana.

Mjesec dana poslije u bolesnice se ponovno razvila prekordijalna bol, uz arterijski tlak od 160/90 mmHg. Ponovljeni EKG nije pokazao nikakve promjene u usporedbi s prethodnim. Međutim, primijećena je dinamična promjena u troponinu, a bolesnici je ponovno dijagnosticiran NSTEMI. Novi CCAT pokazao je produženje SCAD-a tipa 2 u LAD-u s prethodnih 48 na 60 mm (**slika 2C**). S obzirom na to da nije primijećena angina ili hemodinamska nestabilnost, preporučena je optimalna farmakološka terapija. Kako bi se eliminirala fibromuskularna displazija (FMD), obavljena je kompjutorizirana angiotomografija (**slika 3**) koja je bila uredna. Bolesnica je bila podvrgnuta ambulantnom praćenju, uz benevolentnu evoluciju bolesti.

Diskusija

SCAD se više ne smatra rijetkom i nedovoljno istraženom bolesti; procjenjuje se da je njezina zastupljenost 0,78 – 0,98 %, a 1 – 4 % slučajeva AKS-a posljedica je tog stanja[3]. Do 90 % slučajeva SCAD-a pojavljuje se u žena mlađih od 65 godina[1,4,5]. Čimbenici rizika uključuju mlađu životnu dob, tjelesne i emocionalne stresore te upalne bolesti i bolesti vezivnoga tkiva[1].

could not be performed due to significant narrowing. CCTA examination (**Figure 2 A** and **Figure 2 B**) confirmed type 2 SCAD with 48 mm length and lumen reduction of 60-70% in the mid and distal segments of LAD, in addition to a calcium score of 0 UA. Transthoracic echocardiography showed left ventricular (LV) concentric hypertrophy, LV mass of 139 g/m^2 and relative wall thickness of 0.43, preserved LV ejection fraction (55%), apex dyskinesia, and anterior and septal apical akinesia, congruent with the culprit artery. Additional blood tests revealed LDL 1.6 mmol/L, total cholesterol 2.72 mmol/L, and HDL 0.58 mmol/L.

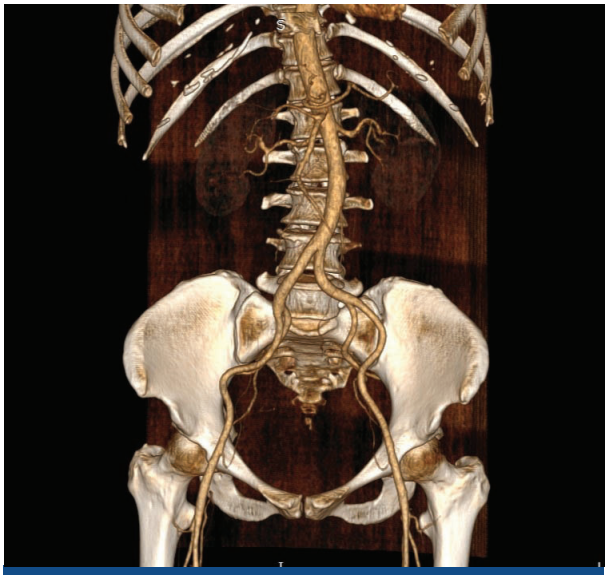

FIGURE 3. Computed tomography angiography shows no thoracic, abdominal, or pelvic arterial alterations.

The patient was treated with enalapril 10 mg two times a day and bisoprolol 5 mg once a day, and was discharged after five days.

However, one month later, the patient developed anginal chest pain once again, and her blood pressure was 160/90 mmHg upon arrival. A new 12-lead electrocardiogram showed no changes compared with the previous one. Nevertheless, a dynamic change of cardiac troponin was recorded, and the patient was once again diagnosed with NSTEMI. A new CCTA showed LAD type 2 SCAD extension from the previous 48 mm to 60 mm (**Figure 2 C**). Since no angina or hemodynamic instability was observed, optimal medical therapy was selected. Computed angio-tomography performed to investigate for fibromuscular dysplasia (FMD) (**Figure 3**) was reported as normal. The patient underwent ambulatory monitoring, with a benevolent evolution.

Discussion

SCAD is no longer considered a rare and understudied disease; it has an estimated prevalence of 0.78-0.98%, and up to 1-4% of all ACS cases are secondary to this condition[3]. Up to 90% of SCAD cases occur in women <65 years of age[1,4,5]. Risk factors include young age, physical and emotional stressors,

Stoga je uobičajeni profil bolesnika žena srednje dobi s niskim kardiovaskularnim rizikom i AKS-om klasificiranim kao tip 2 prema četvrtoj univerzalnoj definiciji infarkta miokarda[6]. Iako su velika većina pacijenata bjelkinje, pokazano je da su ishodi slični u žena hispanskog i afroameričkog podrijetla[4]. Interakcija između različitih okolnosti kao što su emocionalni stres, intenzivno vježbanje i hormonalni status (tj. trudnoća) mogu dovesti do predispozicije prema razvoju SCAD-a[3,4]. Značajno je da sistemska hipertenzija prisutna u do 45 % slučajeva SCAD-a[4], kao što je bio slučaj i u opisane bolesnice.

Konvencionalna koronarna angiografija zlatni je standard za SCAD, no lezije mogu biti teško primjetne, pa se preporučuje revizija koju provodi iskusni intervencijski kardiolog, ako je to moguće. SCAD obično zahvaća srednje do distalne segmente LAD-a, s difuznim suženjem i pravilnim granicama, tj. SCAD tipa 2 prema Yip-Saw klasifikaciji, kao u ove bolesnice. Prisutnost IMH-a s teškim slučajem stenoze >80 % također dovodi do predispozicije za znatnije kliničko pogoršanje pri konzervativnom liječenju[4]. Primjena intravaskularnog oslikavanja (IVUS i OCT) može pomoći u diferencijalnoj dijagnozi i pri izboru intervencije[3,4]. Iako još nije standardizirana, primjena CCTA-a pokazala se korisnom i za dijagnozu i za naknadno praćenje bolesnika.

Za SCAD su na raspolaganju slične metode liječenja kao za AKS. Međutim, primjena trombolitičkih lijekova tijekom akutne faze ne preporučuje se zbog njihovih negativnih učinaka[4]. Do trećine IMH-a proširuje se nakon manipulacije u sklopu perkutane koronarne intervencije (PCI), pa se stoga preporučuje konzervativno liječenje dok nisu prisutne hemodinamska nestabilnost, potpuna okluzija ili ponavljajuća ishemija[3,4,7]. Angiografsko poboljšanje primijećeno je nakon 30 dana u do 95 % bolesnika koji su liječeni konzervativnom terapijom[3,4]. Farmakološka se terapija preporučuje za kontrolu simptoma i sekundarnu prevenciju. Ako je postavljen stent, primjena DAPT-a svakako je opravdana, no još uvijek ne postoji konsenzus za slučajeve s konzervativnom kontrolom bolesti, u koje se ubraja i opisana bolesnica. Neki autori zagovaraju primjenu iste strategije kao kod klasičnog AKS-a, preporučujući dugoročnu monoterapiju acetilsalicilatnom kiselinom[2,3]. Ventrikularne aritmije ili primjena mehaničke cirkulacijske potpore opisane su u 6 – 11 % slučajeva[4], no, nasreću, do njih nije došlo u prikazane bolesnice, s obzirom na njihovu povezanost s lošim ishodima.

Učestalost R-SCAD-a je 17 % do 27 % tijekom prvih 30 dana te do 27 % u sljedećih 4 – 5 godina[1,6]. Žene čine 74 – 100 % slučajeva[6]. Saraswat *i sur.*[7] objavili su seriju slučajeva SCAD-a u sveučilišnoj bolnici u Australiji, pri čemu je zamijećeno 25 % ponavljajućih slučajeva, od kojih je 36 % bilo R-SCAD tipa 2 te je većinom zahvaćalo područje LAD-a, što je slično slučaju ove bolesnice. S druge strane, Tweet *i sur.*[8] objavili su da, iako se prvi SCAD obično pojavljuje u LAD-u, ponavljajući SCAD očituje se u lateralnim granama te u posterolateralnoj arteriji. Prisutnost IHM-a, ozbiljnost lezije te uključenost većega broja žila također stvaraju predispoziciju za R-SCAD[3].

Iako je zabilježen uzročni odnos između hormonalnih stanja, kao što je trudnoća, i pojave SCAD-a, hormonska nadomjesna terapija nije se pokazala učinkovitom u sprječavanju SCAD-a[4]. Terapija beta-blokatorima jedina je farmakološka strategija za koju je dokazano da smanjuje rizik od ponovnog pojavljivanja SCAD-a[3,9]. Statini zasad nisu validirani te se stoga najčešće ne preporučuju kao redovita terapija. U opisanom slučaju došlo do ponovne pojave bolesti usprkos uvođenju terapije beta-blokatorima, no treba naglasiti da liječenje antihipertenzivima nije bilo toliko učinkovito, kao što se moglo uočiti pri otpustu, a da kardiovaskularna rehabilitacija[9], koja

and inflammatory and connective tissue disease[1]. Therefore, the typical profile is that of a middle-aged woman, with a low cardiovascular risk burden presenting with ACS, classified as type 2 according to the 4th universal definition of myocardial infarction[6]. While the vast majority of patients are white women, it has been shown that outcomes are similar in Hispanic and African American female patients[4]. The interaction between different circumstances such as emotional stress, intense exercise and hormonal status (i.e., pregnancy) might predispose towards SCAD development[3,4]. Remarkably, systemic hypertension is present in up to 45% of SCAD cases[4], as in our patient.

CA is the gold standard for SCAD, however, lesions can be difficult to spot, and a revision by an experienced interventional cardiologist is therefore recommended, if feasible. SCAD usually affects the mid to distal segments of the LAD, with a diffuse narrowing and regular borders, i.e., type 2 according to the Yip-Saw classification; as in our patient. Additionally, the presence of IMH with severe stenosis of >80% predisposes to greater clinical deterioration if treated conservatively[4]. Furthermore, the use of intravascular imaging (IVUS and OCT) may be helpful for differential diagnosis and possible interventional treatment[3,4]. Although not yet standardized, the use of CCTA has been found to be useful for both diagnosis and follow-up.

Treatment options are similar to ACS. However, the use of thrombolytics during the acute phase is not recommended due to reported negative effects[4]. Up to a third of IMH propagate just after manipulation during percutaneous coronary intervention (PCI), and thus conservative treatment is preferred as long as there is no hemodynamic instability, total occlusion, or recurrent ischemia[3,4,7]. It has been observed that up to 95% of patients treated conservatively will present angiographic healing after 30 days[3,4]. Medical treatment is recommended for symptomatic management and secondary prevention. If stent placement took place, DAPT is clearly justified, however, there is still no consensus for cases with conservative management as in our patient. Some authors advocate following the same strategy as a classic ACS, advising long-term aspirin monotherapy[2,3]. Ventricular arrhythmias or mechanical circulatory support have been described in 6-11% of cases[4], but fortunately our patient did not develop them, due to their association with poor outcomes.

R-SCAD rates ranges from 17% to 27% during the first 30 days and up to 27% in the following 4-5 years[1,6]. Female patients may account for up to 74-100% of cases[6]. Saraswat *et al.*[7] reported a series of SCAD cases in a university hospital in Australia, which found a 25% recurrence rate, of which 36% had type 2 R-SCAD mainly affecting the LAD territory, which resembles our patient. However, Tweet *et al.*[8] reported that while index SCAD usually occurs in the LAD, recurrences appeared in the marginal obtuse artery as well as posterolateral artery. Additionally, the presence of IHM, severity of the lesion, and multivessel involvement all predispose to R-SCAD[3].

Although a relationship has been observed between hormonal states such as pregnancy and the occurrence of SCAD, hormone replacement therapy has not demonstrated to be effective to prevent it[4]. Beta-blocker therapy is the only pharmacological strategy proven to ameliorate recurrence risk[3,9]. Statins have not yet been validated and are therefore not routinely recommended. In this case, recurrence was observed

čini još jednu učinkovitu preventivnu mjeru, nije bila primijenjena. Preporučljivo je provođenje dijagnostičkih nalaza za arteriopatiju, jer ona može biti prva manifestacija[4], pogotovo za R-SCAD, s obzirom na to da se u do 68 % slučajeva s vremenom postavi dijagnoza FMD-a te, iako manje često, također i Takayasuov arteritis. Preporučuje se dugoročno praćenje, a učestalost smrtnosti tijekom prvih 5 godina iznosi 0 – 5 %[4].

Zaključak

SCAD je ne pretjerano čest, no značajan uzrok akutnog infarkta miokarda. Osim u klasičnoj populacijskoj skupini, SCAD treba uzeti u obzir u svakom slučaju koji uključuje abnormalnosti koronarnih arterija bez indikacija aterosklerotske bolesti. Sveobuhvatan klinički pristup i pravilna primjena liječenja i prevencije ključne su u ograničavanju ponovnog pojavljivanja bolesti i omogućivanja uspješnog oporavka.

despite implementation of beta-blocker therapy, but it should be noted that the antihypertensive treatment was not as effective as demonstrated at discharge and that another preventive effective measure, cardiac rehabilitation[9], was not employed. Diagnostic workup for any arteriopathy is prudent, as it may be the first manifestation[4], especially in R-SCAD, since up to 68% are ultimately diagnosed with FMD[3] and, albeit not as frequent, also with Takayasu arteritis. Long-term follow-up is advised, with a mortality rate around 0-5% during the first 5 years[4].

Conclusion

SCAD is a non-common but relevant cause of acute myocardial infarction. In addition to the classic population group, it should be considered in any situation with coronary artery abnormalities and no evidence of atherosclerotic disease. A comprehensive clinical approach and adequate implementation of treatment and prevention are key to limiting its recurrence and also providing a successful recovery.

Acknowledgements: To Arias-Mendoza, Alexandra, MD for her enthusiasm and cooperation with the author in order to gather the clinical data. To Arenas-Fabbri Vincenzo, Valadés-Camaño Miguel Ángel and Castillo-Castellón Francisco, MD from the Cardiovascular Imaging Department, Instituto Nacional de Cardiología Ignacio Chávez, for their cooperation with the acquisition of imaging data.

Conflict of interest: The authors declare that there is no conflict of intertest.

LITERATURE

1. García Miranda HR, Valdovinos P, Tajes H, Alegret JM. A Case of an Elderly Patient With Recurrent Spontaneous Coronary Artery Dissection. Cureus. 2022 Mar 29;14(3):e23633. https://doi.org/10.7759/cureus.23633
2. Weber AG, Elliott J, Gopalakrishnan P. Recurrent spontaneous coronary artery dissection leading to recurrent sudden cardiac arrests: Who is at risk? Journal of Cardiology Case Reports. 2020;3(3). https://doi.org/10.15761/JCCR.1000142
3. Kok SN, Tweet MS. Recurrent spontaneous coronary artery dissection. Expert Rev Cardiovasc Ther. 2021 Mar;19(3):201-210. https://doi.org/10.1080/14779072.2021.1877538
4. Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, et al. Spontaneous Coronary Artery Dissection: JACC State-of-the-Art Review. J Am Coll Cardiol. 2020 Aug 25;76(8):961-984. https://doi.org/10.1016/j.jacc.2020.05.084
5. Krittanawong C, Kumar A, Virk HUH. Wang Z, Johnson KW, Yue B, et al. Recurrent spontaneous coronary artery dissection in the United States. Int J Cardiol. 2020 Feb 15;301:34-37. https://doi.org/10.1016/j.ijcard.2019.10.052
6. Saw J, Starovoytov A, Aymong E, Inohara T, Alfadhel M, McAlister C, et al. Canadian Spontaneous Coronary Artery Dissection Cohort Study: 3-Year Outcomes. J Am Coll Cardiol. 2022 Oct 25;80(17):1585-1597. https://doi.org/10.1016/j.jacc.2022.08.759
7. Saraswat A, Marashi HA, Raffel O. Recurrent spontaneous coronary artery dissection: a single-centre case series. Heart Lung Circ. 2018;27 Suppl 2: S474. https://doi.org/10.1016/j.hlc.2018.06.978
8. 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 Jul 31;126(5):579-88. https://doi.org/10.1161/CIRCULATIONAHA.112.105718
9. Saw J, Humphries K, Aymong E, Sedlak T, Prakash R, Starovoytov A, et al. Spontaneous Coronary Artery Dissection: Clinical Outcomes and Risk of Recurrence. J Am Coll Cardiol. 2017 Aug 29;70(9):1148-1158. https://doi.org/10.1016/j.jacc.2017.06.053