



Acute coronary syndroms due to spontaneous coronary artery dissection: A monocentric Tunisian experience

Dissection spontanée des coronaires et syndrome coronarien: Expérience tunisienne monocentrique

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SUMMARY

Introduction: Spontaneous coronary artery dissection (SCAD) remains under-diagnosed. Our aim was to describe the characteristics of patients with SCAD and to assess their prognosis.

Methods: This was a prospective monocentric observational study conducted in the cardiology department of Mongi Slim La Marsa Hospital from August 2018 to August 2020.

Results: Thirteen cases were diagnosed. The incidence was 2.6% of acute coronary syndroms (ACS) coronary angiographies. The mean age was 56 years with a sex ratio of 0.2. The majority of patients (46%) had no cardiovascular risk factors and two had hypothyroidism. No case was related to pregnancy. The predominant presentation was non-ST-elevation ACS in 61% of cases (8 patients), while five patients (39%) presented with ST-elevation ACS, three were ongoing and two were seen on day 2. We noted a predominance of type 2 SCAD (46%), with 54% involvement of left descending artery. Two patients underwent unsuccessful fibrinolysis while three patients (23%) had a percutaneous coronary intervention. Success was noted in two patients (67%), and conservative treatment was the rule for 77% of the patients. No patient underwent surgery. The rate of MACE and mortality at the median follow-up of 18 months was 0%.

Conclusion: SCAD is an particularly underdiagnosed cause of ACS, with good short and long-term prognosis.

Keywords

Spontaneous coronary artery dissection; Acute coronary syndrome; Prognosis

Résumé

Introduction : La dissection spontanée des artères coronaires (DSAC) reste sous-diagnostiquée. Notre objectif était de décrire les caractéristiques des patients présentant une DSAC et d'évaluer leur pronostic à moyen terme.

Methodes : Etude observationnelle prospective monocentrique menée dans le service de cardiologie de l'hôpital Mongi Slim La Marsa d'Aout 2018 jusqu'à Aout 2020.

Resultats : Treize cas ont été colligés. a diagnostiqué 13 cas. L'incidence était de 2,6% des coronarographies des SCA. L'âge moyen était de 56 ans avec un sex ratio de 0,2. La majorité des patients (46%) n'avaient aucun facteur de risque cardio-vasculaire et deux avaient une hypothyroïdie. Aucun cas n'était en rapport avec la grossesse. La présentation prédominante était un SCA sans susdécalage ST dans 61% des cas (8 patients), alors que cinq patients (39%) présentaient un SCA avec sus-décalage ST, trois étaient évolutifs et deux vus à J2. On a noté une prédominance des DSAC type 2 (46%) et d'atteinte de l'interventriculaire antérieure (54%). Deux patients ont été thrombolysé avec échec. Trois cas (23%) ont eu une intervention coronaire percutanée. Un succès a été obtenu chez deux patients (67%), et le traitement conservateur a été la règle pour la majorité (77%). Aucun patient n'a eu un pontage. Le taux des MACE et de mortalité au terme du suivi médian de 18 mois était de 0%.

Conclusion : La DSAC est une entité à reconnaitre du fait de ses particularités ayant un bon pronostic à court et à moyen termes.

Mots-clés

Dissection spontanée des coronaires; syndrome coronarien aigu; pronostic

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INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is an epicardial artery dissection that is neither iatrogenic nor post-traumatic. Since the first autopsy description of SCAD by Pretty in 1931 (1), our understanding of the disease has evolved. Literature data suggest that it is an underdiagnosed condition particularly in young women.

METHODS

This was a prospective, descriptive, monocentric study in the cardiology department of Mongi Slim hospital. We included acute coronary syndrome (ACS) patients with SCAD from July 2018 to August 2020. Data were collected based on patients' files and we investigated the type, clinical presentation and context SCAD as well as the therapeutic strategies. latrogenic, posttraumatic and dissection related to aortic dissection or atherosclerotic plaque rupture were excluded. Our aim wastodescribe the demographic, clinical, angiographic and therapeutic characteristics of SCAD patients, and to evaluate their immediate and long-term prognosis. The primary endpoint was the occurrence of major acute cardiovascular events (MACE) during a median follow-up period of 18 months.

RESULTS

Epidemiology

Among 500 coronary angiograms performed in the context of an ACS, 2.6% of the patients were diagnosed with SCAD (13 cases). The mean age was 56 years, with extremes ranging from 35 to 72 years. Patients in their fifties were the most common age group. The SCAD population was mainly feminine (85%), with a sex ratio of 0.2.

Cardiovascular risk factors and associated conditions

Almost half of our population had no cardiovascular risk factor (CVRF) (46%). Arterial hypertension was the most common CVRF (46%), followed by smoking (38%), diabetes (8%) and dyslipemia (8%). The average number of CVRFs per patient was 0.8. No case of SCAD was related to pregnancy. Intense physical stress and a hypertensive crisis were the precipitating factors in two patients each. Two patients had hypothyroidism on treatment, while one patient suffered migraine, and another was followed-up for depression.

Clinical and echocardiographic presentations

The predominant clinical presentation was non-ST elevation myocardial infarction (NSTEMI) in eight patients (61%). Of these, only one was at a very high ischemic risk. Five patients (39%) had an ST-elevation myocardial infarction (STEMI), two of which with a late presentation (seen on day 2). All of our patients ended up having a coronary angiogram as shown in Figure 1. The majority of patients (n=11, 85%) had no wallmotion abnormalities on echocardiography. One patient had apical akinesis with a left intraventricular thrombus while another patient had segmental hypokinesis.



NSTEMI: Non-ST elevation myocardial infarction; SCAD: Spontaneous coronary artery dissection; STEMI: ST elevation myocardial infarction

Angiographic characteristics

SCAD mainly involved the left anterior descending diagonal arteries (n=7) followed by the left circumflex artery - marginal arteries (n=4) and the right coronary artery (n=2). No patient had SCAD of the left main coronary artery. The majority of patients had a type 2 SCAD (46%) with a length >20 mm (62%) (Table 1). Severe tortuosity was observed in 5 patients (38%).

Table 1. Lesional characteristics in our population				
Angiographic characteristics	Number of patients(%)			
Туре 1	5 (38%)			
Туре 2	6 (46%)			
Туре 3	1 (8%)			
Туре 4	1 (8%)			
< 10mm	3 (23%)			
10-20 mm	2 (15%)			
>20mm	8 (62%)			
Thrombus	2 (15%)			
Severe tortuosity	5 (38%)			

Some angiographic presentations of our patients are shown in Figures 2, 3 and 4.



Figure 2. Type 2 dissection of a distal marginal artery

DISCUSSION

Given the potentially life-threatening presentations, all patients were initially started on dual antiplatelet therapy (Aspirin and Clopidogrel) with curative anticoagulation. Of the three patients with ongoing myocardial infarction (MI), two underwent fibrinolysis with failure (100%). In our survey,

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Figure 3. (A) Type 1 dissection of a distal circumflex artery la Cx distale; (B). Final outcome after stent angioplasty with a distal hematoma migration and artery occlusion



Figure 4. Type 4 dissection of the left anterior descending artery

only three patients (23%) underwent percutaneous coronary intervention (PCI). Successful PCI was achieved in two patients (67%). Conservative treatment was therefore the rule for the majority of patients (N=10, 77%) due to a distal anatomical location or largely extensive lesions (Table 2). No patient underwent coronary artery bypass grafting (CABG)

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	Clinical presentation	Treatment	Coronary findings	Result if PCI		
Patient 1	Ongoing STEMI	Fibrinolysis	Distal site (RCA) TIMI 3 flow	-		
Patient 2	Ongoing STEMI	Conservative treatment	Distal site (Mg) TIMI 3 flow	-		
Patient 3	Ongoing STEMI with refractory angina	Fibrinolysis / PCI	TIMI 1 flow	Failure (hematoma extension)		
Patient 4	STEMI on day 2	Conservative treatment	Distal site (LAD) TIMI 3 flow	-		
Patient 5	STEMI on day 2 with refractory angina	PCI	LAD, TIMI 0 flow	Success		
Patient 6	NSTEMI at very high ischemic risk	PCI	Proximal LAD TIMI 1 flow	Success		
Patient 7	NSTEMI	Conservative treatment	Distal site (LAD) TIMI 3 flow	-		
Patient 8	NSTEMI	Conservative treatment	Distal site (LAD) TIMI 3 flow	-		
Patient 9	NSTEMI	Conservative treatment	Distal site (LAD) TIMI 3 flow	-		
Patient 10	NSTEMI	Conservative treatment	Distal site (LAD) TIMI 3 flow	-		
Patient 11	NSTEMI	Conservative treatment	Distal site (Mg) TIMI 3 flow	-		
Patient 12	NSTEMI	Conservative treatment	Lesion length (RCA) TIMI3 flow	-		
Patient 13	NSTEMI	Conservative treatment	Lesion length (Mg) TIMI 3 flow	-		

LAD: left anterior descending artery; Mg: marginal artery; NSTEMI: non ST-elevation myocardial infarction; PCI: percutaneous intervention; RCA : right coronary artery ; STEMI: ST-elevation myocardial infarction; TIMI: thrombolysis in myocardial infarction

At discharge, beta-blockers (92%), Aspirin (85%) and Clopidogrel (77%) were the most prescribed molecules, followed by statins (69%). Antihypertensive treatment with converting enzyme inhibitors or angiotensin II receptor blockers (61%) was also prescribed. Acenocoumarol was prescribed for one patient with an apical thrombus.

Follow-up

The median length of hospital-stay was 5 days, with extremes ranging from 4 to 17 days. No MACE were noted during hospitalization. No patient was lost to follow-up. The median follow-up duration was of 18 months. No patient was readmitted to hospital or had an angiographic check-up. Mortality and the rate of MACE at the end of follow-up in our survey was 0%.

DISCUSSION

Physiopathology

SCAD results from the development of a hematoma in the tunica media leading to separation of the intima or intima-media complex from the underlying vessel, resulting in compression of the true lumen. Recent optical coherence tomography (OCT) imaging studies show that the false lumen is pressurized and that the fenestrations observed may result from rupture of the false lumen into the true lumen rather than vice versa (2).

Epidemiology

SCAD was historically considered a very rare angiographic finding, but contemporary series report an incidence of 0.07 to 0.2% of all coronary angiograms and 2 to 4% of coronary angiograms performed for ACS (3–5). These findings are similar to those observed in our population. Large contemporary series report an average patients age ranging from 44 to 53 years (6–10). The median age in a recent Tunisian serie was 10 years lower than ours (11). The majority of SCAD patients (90%) are female (5,10,12,13), suggesting apathophysiological role for estrogen and progesterone. Male patients are slightly younger and the onset is favoured by intense physical exercise rather than emotional stress (14).

Cardiovascular risk factors and associated conditions:

Atherosclerosis is rare in typical SCAD. Patients with SCAD have fewer CVRF (10). However, many patients have certain risk factors for ischemic heart disease,

including hypertension, smoking and dyslipidemia, although there is no evidence that these contribute directly to the risk of SCAD. In our survey, only two patients (15%) had more than one CRF. (7–10,14–16).

Pregnancyandperipartumcasesrepresentaminority of SCAD (around 10% in most contemporary series). However, 21-27% of MI during pregnancy and 50% of post-partum coronary events are thought to be due to SCAD. The precise nature of this association is still to be elucidated, but it may be linked to hormonal influences on vascular connective tissue or microvascularisation. In our survey, there were no cases of CASD associated with pregnancy.

SCAD was also associated in literature to fibromuscular dysplasia (FMD). It is a non-atherosclerotic, noninflammatory disease of the arterial wall, which also occurs mainly in middle-aged women with few risk factors, and that could lead to dissections and aneurysms of medium-sized arteries. Although FMD is the most common extra-coronary vascular anomaly in patients with SCAD, patients without imaging evidence of FMD have been reported to have other arterial anomalies, including coronary or extra-coronary dissections, aneurysms or tortuosity (78%). Some of these patients may in fact have a typical FMD, non-identified due to insufficiently sensitive imaging techniques. Inoursurvey, no case of MFD has been reported.

SCAD was also associated with systemic inflammatory diseases in 8.9% (15). A clear link between systemic inflammation and SCAD remains incetain. In our survey, twowomenhadhypothyroidismonreplacement therapy, an association reported by lonescu et al (17) in 2009.

Clinical presentations

There is clear evidence that SCAD remains underdiagnosed (18). Some patients do not pay attention to their symptoms or present with sudden death. For those who consult a physician, amissed or delayed diagnosis of SCAD is common (19,20) since most medical and cardiology departements are focused on identifying patients at high ischemic risk whereas patients with SCAD generally have a low ischemic risk. The initial clinical presentation is usually an ACS associated with positive MI biomarkers. The proportion of cases with STEMI compared to NSTEMI varies between series, probably reflecting differences in selection for these registries. In our study, five patients (39%) presented with STEMI. Of these, two (40%) were seen on the 2nd day.

Therapeutical approach

In our survey, two STEMIs were had a fibrinolysis treatment with failure. There is strong evidence that the majority of SCAD will initially stabilize and then heal completely over time if managed conservatively (10,15,16,21-23). Revascularization in patients with CAD is very difficult due to the presence of a disrupted and friable underlying coronary vessel wall. This explains the poorer results of PCI in SCAD compared with atherosclerotic stenosis (7,9,10,16). For this reason, a conservative strategy should be favored whenever revascularization is not mandatory. In our study, conservative treatmentwasfavored in the majority of cases (77%). Published studies show an increased risk of PCI complications during SCAD. In the Canadian series by Saw et al (15), procedural success was achieved in only 64% of patients. In the large Mayo Clinic series (Tweet et al.) (10), procedural success was achieved in only 57% of cases. Furthermore, revascularization was not associated with a reduced long-term risk of repeat revascularization or recurrent SCAD.

In the presence an infarction requiring intervention, interventional cardiologists need to be aware of the specific additional risks associated with interventions in SCADs such as the increased risk of secondary i atrogenic dissection, a possible coronary guidewire passage through the false channel, and the occlusion of major collateral branches by hematoma propagation. Given the increased risk of PCI failure in SCAD, a number of interventional approaches have been reported such as a minimal balloon angioplasty to restore flow followed by a conservative strategy (24) or covering the proximal and distal ends of affected segments with short stents to limit hematoma extension before stenting the intermediate segment (25,26). Targeting the intimal tear with a focal stent (16,27) was also reported.

In our survey, three PCIs were performed in the context of SCAD, with a success rate of 67% comparable to the literature findings. There was one failure due to arterial occlusion by distal migration of the hematoma and one persistent distal dissection successfully managed by a second stent.

CABG in SCAD is generally used as a bailout strategy after failed PCI with ongoing ischemia or because of the site and the extent of the dissection (usually involving the LMCA or the presence of multiple dissections). Successful CABG can be difficult when the dissection extends beyond the graft anastomosis site and great care must be taken to perform the anastomosis to the true lumen. The data regarding CABG in SCAD are limited to small case series (5 to 23 cases). High failure rates have been reported possibly due to healing of the native network leading to competitive flow and secondary graft thrombosis (10). In our study, there were no cases of CABG.

To date, there are no randomized controlled trials comparing different pharmacological treatment strategies for SCAD. Current practice is therefore based on observations of cases and registries and extrapolation of guidelines for the treatment of ACS unrelated to SCAD. Anticoagulation should be limited to revascularization procedures, while chronic use should be restricted to situations where there is an unequivocal clinical indication (such as left ventricular thrombusorthromboembolicevents) (19). Inoursurvey, only one patient was maintained on anticoagulant therapy due to the presence of an apical thrombus on echocardiography.

The use of antiplatelet agents and the duration of treatment remains controversial. This arises from an apparent conflict between existing efficacy data in non-SCAD ACS versus an inherent (albeit unproven) concern about the use of these drugs extending bleeding time in a disease where the primary pathophysiology may be intramural hemorrhage (19). Patients undergoing PCI should receive DAPT for 12 months and extended or lifelong monotherapy (usually with Aspirin) in accordance with ACS recommendations. In conservatively treated patients, there is evidence from OCT studies of significant stenosis sometimes associated with thrombus in SCADs (28). This justifies antiplatelet therapy in the acute phase and most authors advocate DAPT in the acute phase (generally with Aspirin and Clopidogrel rather than the newer P2Y12 inhibitors and avoiding intravenous antiaggregants) (29–31). The optimal duration of subsequent monotherapy remains unknown.

The management of SCAD survivors without left ventricular dysfunction is controversial. Beta-blockers appear to reduce the risk of recurrence (30). Vasodilators such as nitrates and calcium channel blockers are reserved for the empirical treatment of chest pain during the acute phase and following the index event. Statins use is controversial in a condition whose pathophysiology has no known association with high cholesterol levels.

Prognosis

In patients surviving DSAC, long-term mortality is low. In the American Mayo Clinic survey (15), 10-year survival estimated by the Kaplan Meier curve was 92%. In our survey, during 18 months of follow-up, mortality and MACE rates were of 0%.

Survey limitations

Few limitiations are to be acknowledged, such as the small size of the study and its monocentric nature. We are also ought to acknowledge the absence of a comparison group withatherosclerosis-related ACS and the absence of longerterm follow-up of patients to detect MACE. Due to the lack of endo-coronary imaging techniques some SCADs might have been underdiagnosed. We also didn't perform angiographic control to confirm anatomical healing

CONCLUSION

SCAD is a frequently underdiagnosed entity. It predominantly affects women in their fifties, with preferential involvement of the distal left anterior descending artery. In-hospital and long-term mortality after a median follow-up of 18 months was 0%. Fibrinolysis is ineffective in this setting, and PCI is less likely to be successful. Conservative treatment remains so far the first-line treatment in these particular forms of MI.

REFERENCES

- 1. PRETTY, Harold C. Dissecting aneurysm of coronary artery in a woman aged 42. 1931;vol.1; p 667.
- 2. Jackson R, Al-Hussaini A, Joseph S, Van Soest G, Wood A, Macaya F, et al. Spontaneous Coronary Artery Dissection. JACC Cardiovasc Imaging. 2019 Dec;12(12):2475–88.
- Mortensen KH, Thuesen L, Kristensen IB, Christiansen EH. Spontaneous coronary artery dissection: A Western Denmark Heart Registry Study. Catheter Cardiovasc Interv. 2009 Nov;74(5):710–7.
- 4. Nishiguchi T, Tanaka A, Ozaki Y, Taruya A, Fukuda S, Taguchi H, et al. Prevalence of spontaneous coronary artery dissection in patients with acute coronary syndrome. Eur Heart J Acute Cardiovasc Care. 2016 Jun;5(3):263–70.
- 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 Feb;35(2):250–4.
- Rashid HNZ, Wong DTL, Wijesekera H, Gutman SJ, ShanmugamVB,GulatiR,etal.Incidenceandcharacterisation of spontaneous coronary artery dissection as a cause of acute coronary syndrome — A single-centre Australian experience. Int J Cardiol. 2016 Jan;202:336–8.
- Rogowski S, Maeder MT, Weilenmann D, Haager PK, Ammann P, Rohner F, et al. Spontaneous Coronary Artery Dissection:

Angiographic Follow-Up and Long-Term Clinical Outcome in a Predominantly Medically Treated Population. Catheter Cardiovasc Interv. 2017 Jan;89(1):59–68.

- 8. Faden MS, Bottega N, Benjamin A, Brown RN. A nationwide evaluation of spontaneous coronary artery dissection in pregnancy and the puerperium. Heart. 2016 Dec 15;102(24):1974–9.
- Lettieri C, Zavalloni D, Rossini R, Morici N, Ettori F, Leonzi O, et al. Management and Long-Term Prognosis of Spontaneous Coronary Artery Dissection. Am J Cardiol. 2015 Jul;116(1):66–73.
- Tweet MS, Eleid MF, Best PJM, Lennon RJ, Lerman A, Rihal CS, et al. Spontaneous Coronary Artery Dissection: Revascularization Versus Conservative Therapy. Circ Cardiovasc Interv. 2014 Dec;7(6):777–86.
- 11. Saadi, Mohamed. Gestion et pronostic à long terme de la dissection spontanée des artères coronaires : Etude multicentrique nationale. Faculté de médecine de Tunis; 2018.
- Alfonso F, Paulo M, Lennie V, Dutary J, Bernardo E, Jiménez-Quevedo P, et al. Spontaneous Coronary Artery Dissection. JACC Cardiovasc Interv. 2012 Oct;5(10):1062–70.
- 13. Elkayam U, Jalnapurkar S, Barakkat MN, Khatri N, Kealey AJ, Mehra A, et al. Pregnancy-Associated Acute Myocardial Infarction: A Review of Contemporary Experience in 150 Cases Between 2006 and 2011. Circulation. 2014 Apr 22;129(16):1695–702.
- Fahmy P, Prakash R, Starovoytov A, Boone R, Saw J. Pre-DisposingandPrecipitatingFactorsinMenWithSpontaneous Coronary Artery Dissection. JACC Cardiovasc Interv. 2016 Apr;9(8):866–8.
- Saw J, Aymong E, Sedlak T, Buller CE, Starovoytov A, Ricci D, et al. Spontaneous Coronary Artery Dissection: Association With Predisposing Arteriopathies and Precipitating Stressors and Cardiovascular Outcomes. Circ Cardiovasc Interv. 2014 Oct;7(5):645–55.
- Nakashima T, Noguchi T, Haruta S, Yamamoto Y, Oshima S, Nakao K, et al. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: A report from the Angina Pectoris– Myocardial Infarction Multicenter Investigators in Japan. Int J Cardiol. 2016 Mar;207:341–8.
- Gerede DM, Yüksel B, Tutar E, Küçükşahin O, Uzun Ç, Atasoy KÇ, et al. Spontaneous Coronary Artery Dissection in a Male Patient with Takayasu's Arteritis and Antiphospholipid Antibody Syndrome. Case Rep Rheumatol. 2013;2013:1–4.
- Tweet MS, Gulati R, Hayes SN. Spontaneous Coronary Artery Dissection. Curr Cardiol Rep. 2016 Jul;18(7):60.
- 19. Al-Hussaini A, Adlam D. Spontaneous coronary artery dissection. Heart. 2017 Jul;103(13):1043–51.
- 20. SawJ,HumphriesK,AymongE,SedlakT,PrakashR,Starovoytov A, et al. Spontaneous Coronary Artery Dissection. J Am Coll Cardiol. 2017 Aug;70(9):1148–58.
- Rogers JH, Lasala JM. Coronary artery dissection and perforation complicating percutaneous coronary intervention. J Invasive Cardiol. 2004 Sep;16(9):493–9.
- 22. Alfonso F, Paulo M, Lennie V, Dutary J, Bernardo E, Jiménez-

Quevedo P, et al. Spontaneous coronary artery dissection: long-termfollow-upofalargeseriesofpatientsprospectively managed with a 'conservative' therapeutic strategy. JACC Cardiovasc Interv. 2012 Oct;5(10):1062–70.

- Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023 Oct 12;44(38):3720–826.
- 24. Arrivi A, Milici C, Bock C, Placanica A, Boschetti E, Dominici M. Idiopathic, serial coronary vessels dissection in a young woman with psychological stress: a case report and review of the literature. Case Rep Vasc Med. 2012;2012:498465.
- 25. WalshSJ,JokhiPP,SawJ.Successfulpercutaneousmanagement of coronary dissection and extensive intramural haematoma associated with ST elevation MI. Acute Card Care. 2008;10(4):231–3.
- 26. Dashwood AM, Saw J, Dhillon P, Murdoch D. Use of a Three-Stent Technique for a Case of Spontaneous Coronary Artery Dissection. Can J Cardiol. 2017 Jun;33(6):830.e13-830.e15.
- 27. Alfonso F, Bastante T, García-Guimaraes M, Pozo E, Cuesta J, Rivero F, et al. Spontaneous coronary artery dissection: new insights into diagnosis and treatment. Coron Artery Dis. 2016 Dec;27(8):696–706.
- 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 Mar 20;59(12):1073–9.
- Liang M, Bian B, Yang Q. Characteristics and lon-term prognosisofpatients with reduced, mid-range, and preserved ejection fraction: A systemic review and meta-analysis. Clin Cardiol. 2022 Jan;45(1):5–17.
- 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):1148–58.
- Saw J, Ricci D, Starovoytov A, Fox R, Buller CE. Spontaneous coronary artery dissection: prevalence of predisposing conditions including fibromuscular dysplasia in a tertiary center cohort. JACC Cardiovasc Interv. 2013 Jan;6(1):44–52.