Přehledový článek | Review article

Spontaneous Coronary Artery Dissection in Patients with COVID-19: A Systematic Review of Published Case Reports

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SOUHRN

Kontext: Spontánní disekce koronární tepny (spontaneous coronary artery dissection, SCAD) představuje unikátní medicínskou výzvu vzhledem k nutnosti rychle stanovit diagnózu a zahájit léčbu; vyžaduje přitom velmi dobrou znalost základních mechanismů. Cílem tohoto systematického přehledového čllánku je nabídnout komplexní přehled případů SCAD u pacientů s diagnózou onemocnění covid-19.
Metody: Dne 3. června 2023 jsme provedli systematický přehled článků v databázích včetně PubMed, ScienceDirect a Cochrane. Do přehledu jsme zahrnuli případy SCAD u pacientů s onemocněním covid-19, v nichž byly uvedeny údaje jednotlivých pacientů.
Výsledky: Studie analyzovala 12 kazuistik; u všech pacientů byla potvrzena diagnóza onemocnění covid-19, byli ve věku s mediánem 49 let, jednalo se převážně o ženy (58,3 %). Většina pacientů vykazovala klasické symptomy onemocnění covid-19 (66,7 %), přičemž těžké projevy byly pozorovány u pacientek s těhotenstvím v anamnéze. Často byly zaznamenány vysoké hodnoty troponinů (83,3 %) stejně jako změny na EKG záznamu, hlavně abnormality v úseku ST a vlně T. Hlavním diagnostickým nástrojem byla koronagrafie (91,7 %), přičemž nečastějším místem disekce byla přední sestupná větev levé věnčité tepny (50 %). Ve čtyřech případech byla zjištěna snížená ejekční frakce levé komory, ale po léčbě nebyly zaznamenány žádné kardiovaskulární komplikace.
Závěr: Tato studie popisuje charakteristiky SCAD u pacientů s infekčním onemocněním covid-19; zároveň byly pozorovány vhodné diagnostické postupy a léčebné strategie.

Keywords:
COVID-19
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Treatment

ABSTRACT

Background: Spontaneous coronary artery dissection (SCAD) presents a unique challenge due to its prompt diagnosis and treatment requirements, demanding an in-depth understanding of its underlying mechanisms. This systematic review aims to provide a comprehensive summary of the SCAD cases in patients diagnosed with COVID-19.
Methods: We performed a systematic search across databases, including PubMed, ScienceDirect, and Cochrane, on June 3, 2023. We included reported cases of SCAD in COVID-19 patients that described individual patient data.
Results: The study analyzed 12 case reports, revealing that all patients were confirmed COVID-19 cases with a median age of 49, predominantly female (58.3%). Most exhibited classic COVID-19 symptoms (66.7%), with severe presentations observed in those with a pregnancy history. High HS troponin levels were common (83.3%), as were ECG changes, namely ST and T abnormality. Coronary angiography was the primary diagnostic tool (91.7%), with the left anterior descending artery (LAD) being the most common site of dissection (50%). Four cases had reduced ejection fraction, but no cardiovascular complications occurred post-management.
This study elaborates on the characteristics of SCAD in COVID-19 patients, and appropriate modalities and treatments were observed.

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Background

In late 2019, coronavirus disease (COVID-19) emerged, presenting numerous challenges to global healthcare systems. While the primary respiratory symptoms of respiratory infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) are well-documented, increasing evidence suggests that the virus can affect multiple organ systems, including the cardiovascular system. Spontaneous coronary artery dissection (SCAD) as a rare but emerging complication and potentially life-threatening condition has garnered attention. SCAD is defined as a nontraumatic separation of the layers of the coronary artery wall by intramural hemorrhage, generating restricted blood flow to the heart muscle.[1] This condition primarily affects young women without traditional coronary artery disease risk factors.[2] COVID-19-related SCAD presents a unique clinical challenge, as it requires prompt diagnosis and treatment and necessitates a comprehensive understanding of the underlying pathophysiological mechanisms. In COVID-19 patients, SCAD develops from the interaction between the pro-inflammatory state induced by the viral infection, the direct endothelial injury, and potential hypercoagulability related to COVID-19.[3] A systematic review of case reports is crucial to gain further insights into the relationship between COVID-19 and SCAD. This systematic review aims to provide an exhaustive summary of the reported cases of SCAD in patients diagnosed with COVID-19. Consolidating the available evidence will contribute to the growing knowledge surrounding this unique cardiovascular complication. The findings may help clinicians recognize and manage SCAD promptly in COVID-19 patients, improving outcomes and potentially saving lives.

Methods

The present review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline for 2020[4]. Ethical approval was not required since no patients were directly involved in this study, and only published data were utilized.

Eligibility criteria

We systematically searched for SCAD case reports in patients with COVID-19, focusing on individual patient data. SCAD is defined as an emergency condition characterized by a tear in the wall of a coronary artery. Studies reporting SCAD in patients without COVID-19 were excluded. Additionally, studies involving animals as subjects, those not available in full text, and those written in languages other than English were also excluded. Duplicate articles were resolved before the title and abstract screening.

Search strategy and study selection

Systematic studies were retrieved from PubMed, ScienceDirect, and Cochrane on June 3, 2023. A manual or bibliography search was conducted to ensure comprehensive coverage of sporadic cases. The keywords used for the search included "spontaneous coronary artery dissection" and "COVID-19". We independently screened titles and abstracts of articles to identify potentially eligible studies, followed by a full-text review. Any discrepancies during the screening process were resolved through discussion with senior authors.

Data extraction

We extracted the following information from the included studies: author, year of publication, country, age, gender, presenting symptoms and signs, comorbidities and medical history, predisposing factors, diagnostic imaging, electrocardiogram (ECG) changes, high sensitivity (HS) troponin I level, timing about COVID-19 infection and severity, corticosteroid use, location of vessel dissection, ejection fraction, cardiac motion, management, complications, and outcomes. A structured and standardized form was meticulously used for data extraction. Any disagreement observed during the extraction process was resolved through discussion with senior authors.

Quality assessment

The risk of bias in the included studies was assessed using the Joanna Briggs Institute (JBI) critical appraisal checklist for case reports. The results were presented as a checklist rather than an accumulated score.[5] In cases where there were differing assessments or judgments during the quality assessment, these discrepancies were resolved through thorough discussions involving senior authors.

Statistical analysis

A meta-analysis could not be performed because this systematic review focuses on an extremely rare disease based on published cases. Narrative synthesis was the primary data synthesis method used in this review. However, quantitative analysis of extracted data was conducted using descriptive statistics. Similar findings for variables, such as clinical presentation, ECG findings, HS troponin I level, location of vessel dissection, and mortality, were grouped to assess their frequency. For instance, we compiled reported sites of vessel dissection, such as left anterior descending artery (LAD), right coronary artery (RCA), and left circumflex artery (LCx).

Results

Study selection

Out of the initial 388 records retrieved in the search, 155 were identified as duplicates and were consequently excluded. After thoroughly screening titles and abstracts, an additional 227 articles were excluded. Six published articles were included in this systematic review following this screening process, as determined through a comprehensive full-text assessment.[6–17] The PRISMA flow diagram visually presents the study's selection process and the rationales for exclusion (**Fig. 1).**

Quality assessment

All included case reports were assessed using a JBI critical appraisal checklist for case reports. Upon summarizing the critical appraisal checklist, it became evident that the overall risk of bias in these case reports was generally low. However, it's important to note that we did not establish

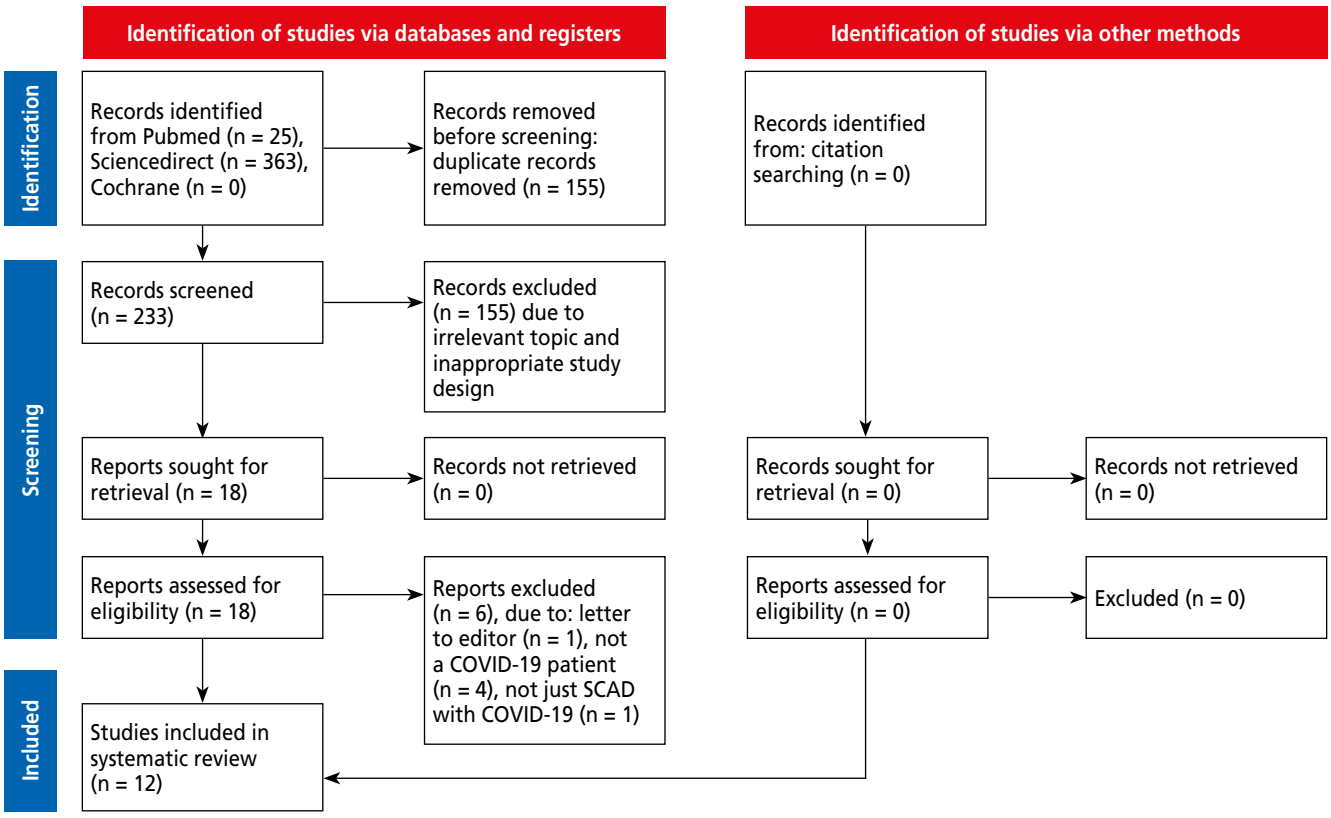

Fig. 1 – PRISMA flow of study selection.

specific criteria for categorizing a study as having a low risk of bias based solely on the total score obtained from the checklist (supplementary file).

Study characteristics

This systematic review of published cases included 12 case reports (**Table 1**).[6,7,9–18] The year of published case reports ranged from 2020 to 2022. Most published cases are from America[9,10,16,17,19] and Europe (5 cases each),[6,12–15] followed by Asia (2 cases).[7,11]

Clinical characteristics

The age of the patients ranged from 35 to 70 years, with a median age of 49. The data on age distribution suggests that SCAD in patients with COVID-19 is most common in adults and the elderly, with no instances reported in pediatric patients (**Table 1**). All cases were confirmed through COVID-19 testing, with five male patients for every seven female patients.

Cardiovascular symptoms, such as chest pain, palpitation, and dyspnea, are present in all cases.[6,7,9–18] Classic symptoms of COVID-19, such as shortness of breath, fever, coughing, and anosmia, were found in 6 cases.[6,9,11–13,15] One case presented with a syncopal episode due to ventricular fibrillation and required a defibrillation.[16] The time when SCAD symptoms appear varies. It can be at the same time as the diagnosis of COVID-19 or weeks after recovering from COVID-19. Five cases[9,13,14,17,19] were diagnosed with COVID-19 on the same day as the SCAD diagnosis, and two cases[12,15] were diagnosed two days after the SCAD diagnosis. Meanwhile, the other five cases[6,7,10,11,16] have a history of or have been hospitalized due to COVID-19, varying from 7 days to 3 months post-COVID-19.

The comorbidities of the patients varied among the cases. Five cases[10,11,13,14,17] did not have special medical terms, and five cases[6,7,12,15,19] had comorbidities or risk factors related to coronary artery disease, such as smoking, overweight, hyperlipidemia, hypertension, diabetes, and peripheral artery disease (PAD). There are two comorbidities related to pregnancy: pregnancy itself and postpartum cardiomyopathy.[9,16] A history of corticosteroid use was also reported in two cases,[6,10] with one of them in a case that had no risk factors for coronary artery disease or comorbidities except being a woman.

The HS troponin I levels were elevated in most cases (9 out of 10) that underwent examination. At the same time, there was no mention of HS troponin I levels in the other 2 cases.[11,17] Abnormal ECG changes were consistently observed in all cases reporting ECG examination results (11 of 12 cases), commonly presenting ST elevation and T inversion (**Table 1**).[6,7,9–14,16–18] However, the study by Courand et al. did not provide information on ECG examinations.

Coronary angiography is the most common primary imaging modality to diagnose SCAD (11 of 12 cases), followed by echocardiography,[7,9,11,13–16,19] coronary magnetic resonance (CMR),[7,14] CT angiography,[9] and myocardial perfusion imaging (MPI).[11] The location of the vessel's dissection is frequently found in the LAD (6 cases),[9,10,12–14,19] followed by the RCA (4 cases)[6,11,15,17] and LCx (2 cases).[7,16] The echocardiographic examination that was carried out showed that four patients experienced a decrease in the ejection fraction below 50%.[10,12,16,19] Meanwhile, the most

Table 1 – Demographics, presentation, comorbidities, COVID-19 infection status, and diagnosis of SCAD

No	Author/Year of publication	Country	Age (year), sex	Signs and presenting symptoms	Medical history and comorbidities	Imaging diagnosis	ECG abnormalities	Troponin I level	Timing according to COVID-19 infection, severity	History of corticosteroid use
1	Kireev et al./2020	Russia	35, M	Chest pain, weakness, fever, nasal congestion, anosmia, dry cough, chest congestion	Smoker, overweight, no autoimmune disease	Coronary angiography	Right precordial leads: slight ST elevations	Increase	Approximately 18 days post COVID-19, mild	Methylprednisolone
2	Kumar et al./2021	USA	48, F	Chest pain	Migraine, hyperlipidemia, FMD was ruled out	Coronary angiography, echocardiography, renal ultrasonography	Lead III and aVF: submillimeter ST elevations Lead V_3–V_5: biphasic changes	Increase	Covid test (+) at admission, NA	None
3	Alemzadeh et al./2022	Iran	58, F	Chest pain,	Severe thrombocytopenia, hyperlipidemia, hormone replacement therapy and physical/emotional stress was ruled out	Echocardiography, CMR, coronary angiography	Lead I and aVL: Q waves; lead V_5–V_6: inverted T	Increase	2 months post COVID-19, NA	None
4	Pettinato et al./2022	USA	43, F	Chest pain, palpitations, nausea, hypotensive, tachycardic	None	Coronary angiography	Lead V_2–V_4: ST elevation and T inversion	Increase	2 months post COVID-19, NA	Yes
5	Emren et al./2021	Turkey	50, M	Cough, fever, chest pain	None	Echocardiography, MPI, coronary angiography	Inferior leads: Q and inverted T waves	NA	7 days post COVID-19, Mild	None
6	Albiero and Seresini/2021	Italy	70, M	Chest pain, fever	Smoker, hypertension, diabetes	Coronary angiography	Precordial leads: ST-T abnormalities	Increase	1 day after coronary angiography, mild	None
7	Gasso et al./2020	Spain	39, M	Fever, cough, myalgia, chest pain, dyspnea	None	Echocardiography, coronary angiography	Lead II, III, aVF, and V_4–V_6: mild ST-elevation; lead V_1–V_4: appearance of a dominant R wave; lead II, III, aVF, and V_5–V_6: Q wave	Increase	Covid test (+) at admission, severe	None
8	Cannata et al./2020	UK	45, F	Chest pain	FMD was ruled out	Coronary angiography, echocardiography, CMR	Anterolateral leads: ST elevation	Increase	IgG covid test (+) at admission, NA	None

Pokračování na další straně

Table 1 – Demographics, presentation, comorbidities, COVID-19 infection status, and diagnosis of SCAD *(Dokončení)*

No	Author/Year of publication	Country	Age (year), sex	Signs and presenting symptoms	Medical history and comorbidities	Imaging diagnosis	ECG abnormalities	Troponin I level	Timing according to COVID-19 infection, severity	History of corticosteroid use
9	Ahmad et al./2021	USA	43, F	Cardiac arrest, hypothermia, bilateral decreased breath sounds	AF during pregnancy	Echocardiography, coronary angiography	AF with rapid ventricular	Normal	12 weeks post COVID-19, mild	None
10	Courand et al./2020	France	55, M	Cough, febrile, dyspnea, chest pain	Peripheral artery disease	Echocardiography, coronary angiography	NA	Increase	Moderate crazy pavy pattern in the lung in 48 hours after test result	None
11	Lewars et al./2022	USA	51, F	Chest pain, dyspnea	Anxiety, postpartum cardiomyopathy	Echocardiography, CT angiography	T wave inversion	Increase	COVID test (+) at admission, NA	None
12	Ali et al./2022	USA	53, F	Chest pain	NA	Coronary angiography	Inferior leads: ST elevation	NA	COVID test (+) at admission, NA	None

Table 2 – Artery dissection location, cardiac function, complication, management, and outcomes

No.	Author/Year of publication	Location of vessel's dissection	Ejection fraction	Cardiac motion	Management	Complication	outcome (survived/death)
1	Kireev et al./2020	RI, RCA	NA	NA	PCI, dual antiplatelet anticoagulation	None	Survived
2	Kumar et al./2021	LAD	45–50%	Distal anteroseptal and apical segments akinesis	Dual antiplatelet, nitroglycerin, fentanyl, beta blocker, amiodarone	None	Survived
3	Alemzadeh et al./2022	LCx	50%	Akinesia in the mid to apical lateral wall	Dual antiplatelet, metoprolol, lisinopril, statin	None	Survived
4	Pettinato et al./2022	LAD	45–49%	Basal to apical anterior, septal, and apical lateral segments of the left ventricle (LV) hypokinesis	Dual antiplatelet, nitroglycerin, heparin, morphine, ondansetron, atorvastatin, beta blocker, metoprolol, spironolactone, lisinopril, warfarin	None	Survived
5	Emren et al./2021	RCA	55%	Inferior and inferoseptum abnormality	PCI, dual antiplatelet anticoagulation, atorvastatin, metoprolol	None	Survived
6	Albiero and Seresini/2021	LAD	40–45%	Akinesis in LCx, severe hypokinesis in LAD	PCI, clopidogrel, bisoprolol, ASA, atorvastatin, metformin, pantoprazole	None	Survived
7	Gasso et al./2020	OM, LAD	50–55%	Hypokinesis of the severe basal, middle inferoseptal, inferolateral, and inferior wall; and hypokinesis of the basal inferior and lateral right ventricle	Conservative	None	Survived
8	Cannata et al./2020	LAD	NA	Hypokinesia of the anterior wall with moderate left ventricular systolic impairment	Dual antiplatelet, beta blocker, ACE inhibitor	None	Survived
9	Ahmad et al./2021	LCx	20%	New-onset global hypokinesis	Impella device, methylprednisolone	None	Survived
10	Courand et al./2020	RCA	60%	None	Conservative, ASA, statin, beta blocker	None	Survived
11	Lewars et al./2022	LAD	Normal	Dyskinetic apex	Nitroglycerine, aspirin, heparin, PCI	None	Survived
12	Ali et al./2022	RCA	NA	NA	PCI	None	Survived

common abnormal heart wall movements were hypokinesis (5 cases)[10,12–14,16] and akinesis (3 cases).[7,12,19] No complications related to the heart and blood vessels were found after management in all cases (**Table 2**).

A total of 5 cases underwent percutaneous coronary intervention (PCI) as the main therapy,[6,9,11,12,17] and no cases underwent coronary artery bypass graft (CABG). The remaining cases were administered conservative therapy with drugs.[7,10,13–16,19] Most cases reported receiving dual-antiplatelet therapy,[6,7,10–12,14,19] and two cases received aspirin alone.[9,15] Anticoagulant therapy was also given in 4 cases[6,9–11] as heparin or warfarin. Some cases also received beta-blocker therapy, ACE inhibitors, and statins. Statins are administered regardless of whether there is a history of hyperlipidemia. Methylprednisolone was administered in one case due to myocarditis as a late inflammatory sequela of COVID-19.[16] Due to an ejection fraction of 20% with new onset global hypokinesis, the patient in that case also received an impella device, which was then replaced with veno-arterial extra corporeal membrane oxygenation (VA-ECMO).[16] Despite all the characteristics and therapy provided, no cases experienced complications or mortality.

Discussion

Epidemiology and pathophysiology

Spontaneous coronary artery dissection, or SCAD, frequently occurs in young females under 50. SCAD was also reported in older and postmenopausal women and less than 10 % to 15 % of cases in men.[20] In this study, women younger than 50 have been reported by Kumar et al., Pettinato et al., Cannata et al., and Ahmad et al. Meanwhile, Alemzadeh et al., Lewars et al., and Ali et al. have reported SCAD cases in women older than 50. On the other hand, SCAD in men was only reported by Kireev et al., Emren et al., Albiero and Seresini, Gasso et al., and Corand et al.

Several studies have found that one of the risk factors for SCAD is pregnancy (pregnancy-related spontaneous coronary artery dissection or P-SCAD), and its incidence is approximately 1.81 per 100,000 pregnancies.[21,22] P-SCAD cases were found during pregnancy and three months of the postpartum period, and the most common incidences occur during the first month of the postpartum period and in the first week of the postpartum period.[22,23] Ahmad et al. reported atrial fibrillation in pregnancy as a comorbidity in a 43-year-old woman suffering cardiac arrest, whose ECG showed ventricular fibrillation and cardiogenic shock. Lewars et al. reported a 51-year-old woman with postpartum cardiomyopathy.

A tear of the tunica intima wall made blood enter and separate the coronary artery wall's layer, creating a false lumen filled with intramural hematoma in the medial layer. The external compression caused by the enlarging hematoma restricted coronary blood flow, which increased the pressure of the false lumen, leading to coronary artery insufficiency.[24–26] Another hypothesis is intramural hematoma accumulation from a spontaneous hemorrhage in vasa vasorum without a tear in the tunica intima.[27] Furthermore, more research about the pathophysiology of SCAD, specifically in patients with COVID-19 and its complications, is still required.

Clinical presentation and diagnostic approach

Phenotypically, SCAD exhibits consistent similarities with the atherothrombotic of acute coronary syndrome (ACS).[28] SCAD's primary manifestations, reported by the prior studies, were chest pain followed by an increase in specific cardiac enzymes, closely resembling ACS in general.[29–31] Hence, a definitive diagnosis of SCAD presents significant challenges and requires advanced diagnostic modalities, such as invasive intracoronary angiography, performed by experienced operators.[28] Presumptive diagnosis, however, can be considered by observing typical ACS clinical presentations in middle-aged women without traditional atherothrombotic risk factors like diabetes, hypertension, and dyslipidemia.[28,32] Additionally, additional comorbidities such as connective tissue diseases, arteriopathy, genetic predisposition, pregnancy, systemic inflammation, and precipitating factors like emotional stress and intense exercise in patients presenting with ACS support the suspicion of SCAD.[28]

COVID-19-associated SCAD's clinical manifestations did not appear significantly different from those in the general population. Importantly, SCAD was observed to occur in patients with varying temporal associations with COVID-19, ranging from during active infection to several months after recovery. Our study population primarily consisted of middle-aged females (median age of 49 years old), consistent with the demographic profile of non-COVID-19-associated SCAD patients.[28,33,34] Most patients had no pre-existing cardiovascular comorbidities. Nevertheless, we did observe a patient, a 70-year-old man, with risk factors of atherothrombosis, including hypertension, diabetes, and smoking, a 55-year-old male patient with peripheral artery disease, and another patient presenting with anxiety and postpartum cardiomyopathy. Chest pain was reported in all cases, with several patients also experiencing symptoms such as dyspnea and palpitations and one patient suffering from cardiogenic shock and arrest (the patient had a history of atrial fibrillation during pregnancy). This further confirms the similarity in clinical presentation between SCAD and other entities of ACS. Distinguishing factors may rely on coinciding signs and symptoms of associated comorbidities, such as COVID-19, which in our cases included flu-like syndrome and respiratory symptoms.

Furthermore, nine of the ten patients who underwent high-sensitivity troponin testing exhibited increased levels of this cardiac enzyme.[6,7,9,10,12–15,19] Six of the reported eleven EKG findings were characterized by ST elevation.[6,10,13,14,17,19] As mentioned earlier, differentiating atherothrombotic ACS from SCAD poses a distinct diagnostic challenge. For a definitive diagnosis of SCAD, the test of choice remains by invasive coronary angiography. Invasive coronary angiography remains the test of choice for the definitive diagnosis of SCAD.[28] Angiographic findings indicated that the majority of SCAD cases in COVID-19 patients was observed in LAD (50%), followed by RCA and LCX. These findings align with previous research highlighting the predisposition of SCAD in the LAD.[35,36] Moreover, coronary tortuosity and the absence of intraluminal thrombus were suggested as notable clues favoring the diagnosis of SCAD.[37,38] However, our findings noticed a case where SCAD diagnosis was made using non-invasive coronary computed tomography angiogra-

phy (or CTA), suggesting the potential role of noninvasive diagnostic modalities in detecting SCAD, especially when invasive coronary angiography was not feasible.[9]

Management and outcomes

Conservative therapy is preferred over other treatments in stable SCAD patients without ongoing ischemia and patients with occlusion of distal vessels or distal branches for which PCI is not routinely performed.[32] In a cohort study at Vancouver General Hospital, 79 patients who underwent repeat CTA or angiogram after 26 days showed complete healing from SCAD. In nine patients who did a repeat examination for less than 20 days, recovery did not occur. This indicates that the SCAD healing process depends on time.[39] Compared with conservative therapy, patients treated with PCI are at higher risk of complications and suboptimal outcomes. PCI is also at risk of requiring emergency CABG because of the failure of the procedure.[32,40] Coronary angiography causing iatrogenic dissection is rare (0.02-0.09%)[41,42]. However, in SCAD, the affected coronary arteries have a weak structure, which puts them at risk for iatrogenic dissection and dissection expansion due to PCI. The PCI procedure's wire can get into the false lumen, and the true lumen can be clogged. Additionally, SCAD frequently affects the distal coronary segment, which is not large enough for stent placement – field.[32,43] The incidence of iatrogenic dissection during diagnostic angiography and PCI was 4.7% in SCAD patients, according to a retrospective observational study.[44]

In SCAD cases, CABG is another option for therapy in patients with proximal or left main stem dissection, patients with failed PCI, patients with complications due to PCI, or refractory ischemia despite attempted conservative treatment.[32] In a retrospective study of 15 bypass grafts, 11 were occluded on follow-up angiography. This emphasizes that long-term protection against the effects of recurrent native coronary artery dissection is not provided by the CABG.[40] In high-risk patients with ongoing ischemia, left main artery dissection, or hemodynamic instability, conservative therapy is inappropriate. Therefore, the treatment for each SCAD patient is left to interventionists because there is no guideline for managing SCAD. Interventionists must consider coronary anatomy, operator skill, and facility availability in determining the best therapy.[32,45]

Relieving symptoms, improving short-term and long-term outcomes, and preventing recurrent SCAD are the main therapy goals for SCAD. Evidence of the benefit of using dual-antiplatelet in SCAD patients without coronary intervention is still limited.[32] The advantage of aspirin in large ACS populations is likely greater than the risk, which is why low-dose aspirin is considered.[46] No studies have compared the risk of bleeding or short-term and long-term outcomes using dual-antiplatelet or aspirin alone in the SCAD.[32] Since most SCAD involves prothrombotic intimal tears, empirically dual antiplatelet administration would be helpful. By using antiplatelet agents to reduce the false lumen thrombus burden, the true lumen compression could be reduced.[27] A retrospective Italian series examining long-term outcomes among patients with SCAD treated with dual-antiplatelet therapy in both PCI and conservative therapy patients found no bleeding complications and similar long-term outcomes for the two groups.[47]

Anticoagulation for SCAD is still controversial, with the risk of dissection extension balanced by the potential benefit of resolving overlying thrombus and improving true lumen patency, so heparin administration must be discontinued when SCAD is proven by angiography to prevent intramural hematoma extension. Beta-blockers can reduce arterial shear stress, which is expected to reduce coronary arterial wall stress.[27] Some experts recommend the routine use of SCAD based on its benefits for atherosclerotic MI or aortic dissection. In contrast, others recommend selective use because of the effect of vasospasm or symptomatic hypotension.[32]

Angiotensin-converting enzyme inhibitors are routinely administered to MI patients complicated by LV systolic dysfunction. This drug is also an option for patients with hypertension.[27,32] Statins should not be given routinely to SCAD patients but only to patients with hyperlipidemia as primary prevention of atherosclerosis and patients with concomitant atherosclerotic disease or diabetes mellitus[32,46] because less than 50% of SCAD patients had a prior diagnosis of hyperlipidemia.[45] In addition, statins were reported to be higher in patients with recurrence of SCAD.[32]

Conclusion

This study explained that SCAD in patients with COVID-19 had similar clinical presentation and complications to ACS, and they had different comorbidities and medical histories. Appropriate modalities to diagnose and treatments were chosen. Further research on SCAD with COVID-19 is still required.

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Conflict of interest
All the authors declare that there are no conflicts of interest.

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Ethical statement
Not required.

Underlying data
All data are available in the manuscript and separated files as supplementary.

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