Rheumatology International (2019) 39:1821–1827
https://doi.org/10.1007/s00296-019-04351-3

Rheumatology
INTERNATIONAL

CASE BASED REVIEW

Spontaneous coronary artery dissection in systemic lupus erythematosus: case-based review

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Received: 19 March 2019 / Accepted: 15 June 2019 / Published online: 21 June 2019

Abstract

Patients with systemic lupus erythematosus (SLE) present an increased prevalence of coronary heart disease. The majority of cases of acute coronary syndrome (ACS) in patients with SLE are due to atherosclerosis. Less common causes include thrombosis of an angiographically normal coronary artery and coronary vasculitis. Spontaneous coronary artery dissection (SCAD) is a rare cause of ACS in these patients. We report the case of a 53-year-old female diagnosed of SLE presenting with an ACS caused by SCAD. She was treated medically and her clinical course was favorable. A literature search identified seven additional cases of SCAD associated with SLE. The main clinical features found in these reports are revised. ACS caused by SCAD in SLE patients is a condition likely under-reported in literature. SCAD should be suspected in patients with SLE and ACS, especially in younger women without evident cardiovascular risk factors. An early accurate diagnosis of SCAD is key to provide specific treatment, which differs from that of usual atherosclerotic ACS.

Keywords Systemic lupus erythematosus · Coronary artery disease · Myocardial infarction · Spontaneous coronary artery dissection

Introduction

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of myocardial infarction (MI) and sudden cardiac death, which typically occurs in young patients, mostly women, without risk factors for atherosclerotic heart disease [1–3]. SCAD has been mainly associated with fibromuscular dysplasia, pregnancy and childbirth, and more rarely with connective tissue diseases (Marfan syndrome, Ehlers–Danlos type IV and Loeys–Dietz syndrome) or some inflammatory systemic diseases, such as systemic lupus erythematosus (SLE), inflammatory bowel disease, sarcoidosis and systemic vasculitis [1, 2]. However, in the majority of patients presenting with SCAD, no concomitant arteriopathy, inflammatory disease, or other obvious risk factor are found [4]. In this paper, we present a case of SCAD in a patient with SLE (SCAD-SLE) and a review of the previous cases described in the literature.

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Clinical observation

A 53-year-old woman presented to the emergency department with severe inter-scapular pain triggered by physical exertion, dizziness and nausea. She reported similar but milder episodes occurring the previous 4 days. She had no current cardiovascular risk factors, except for smoking cessation 23 years before. The patient had been diagnosed with SLE at the age of 39 years (arthritis, oral/nasal ulcers, Raynaud, lymphopenia, ANA positive and hypocomplementemia). She was treated with hydroxychloroquine (200 mg/day) and low doses of prednisone (5 mg/day). The disease remained stable, with a low activity index (SLEDAI $\leq$ 4), and without renal, cardiac, pulmonary or neurological involvement throughout the previous years.

Vital signs on admission were blood pressure 123/76 mmHg, pulse 80 beats/min, temperature 35.8 °C, respiratory rate 16 breaths/min, SpO_2 97% on room air and body mass index (BMI) 21.9 kg/m^2. Physical examination was unremarkable, and the patient remained hemodynamically

stable. The chest X-ray was normal and the ECG showed a negative T wave in V4–V6. An angio-CT scan of the aorta and pulmonary arteries ruled out an acute aortic syndrome, as well as defects of pulmonary artery repletion. High sensitivity troponin T was 193 ng/L and creatine kinase 360 UI/L, which peaked to 231 and 373, respectively, after 4 h, compatible with acute coronary syndrome (ACS). Coronary angiography showed an early first obtuse marginal branch of the circumflex artery (Cx) with reduced lumen (2 mm) and long tubular stenosis, suggestive of hematoma/dissection (Figs. 1, 2, 3). No angiographic stenosis was visible in the rest of the epicardial arteries. The echocardiogram showed a normal systolic function of the left ventricle, with posterior mid-apical hypokinesis, without pericardial effusion, or other mechanical complications.

The patient was diagnosed with MI provoked by SCAD of the first obtuse marginal branch of Cx. In the presence of a long SCAD with preserved distal flow (TIMI 3) in a hemodynamically stable patient, medical treatment was recommended. She was treated with vasodilators (intravenous nitroglycerin), beta-blockers (bisoprolol 5 mg/day), single antiplatelet therapy with acetylsalicylic acid (100 mg/day), and statins (atorvastatin 20 mg/day). Lipids, glucose and HbA1c were ordered to further risk stratify her; results were total cholesterol 209 mg/dL, LDL 93.3 mg/dL, HDL 91.0 mg/dL, triglycerides 126.0 mg/dL, glucose 88 mg/dL, and HbA1c 5.3%. The clinical and laboratory assessment of SLE during admission was unremarkable except for mild lymphopenia (1240/mm^3). Urine analysis was normal. Determination of ANAs was positive at 1/160 dilution, and anti-dsDNA, Sm, SS-A and SS-B test were all negative. Complement levels were normal. Antiphospholipid antibodies (aPL), including anti-cardiolipin, anti-β2 glycoprotein 1 and lupus anticoagulant, were negative.

Her subsequent clinical course was favorable. The patient was discharged 7 days after admission, with recommendation for a cardiac rehabilitation program (physical activity, secondary prevention education, and psychosocial support), and treatment with bisoprolol (5 mg/day), acetylsalicylic acid (100 mg/day) and pitavastatin (2 mg/day). After 6 months of follow-up, the patient remains asymptomatic, and free of cardiac events.

Review of the literature

In addition to the case presented, reports of SCAD associated with SCAD-SLE with some clinical information were identified using MEDLINE/PubMed, Scopus and Web of Science databases. The following MeSH search terms were used: “lupus”, “systemic lupus erythematosus”, “SLE” [AND] “spontaneous coronary artery dissection”, “coronary dissection”, “acute coronary syndrome”, and “coronary artery disease”. The search was conducted in May 2019. A

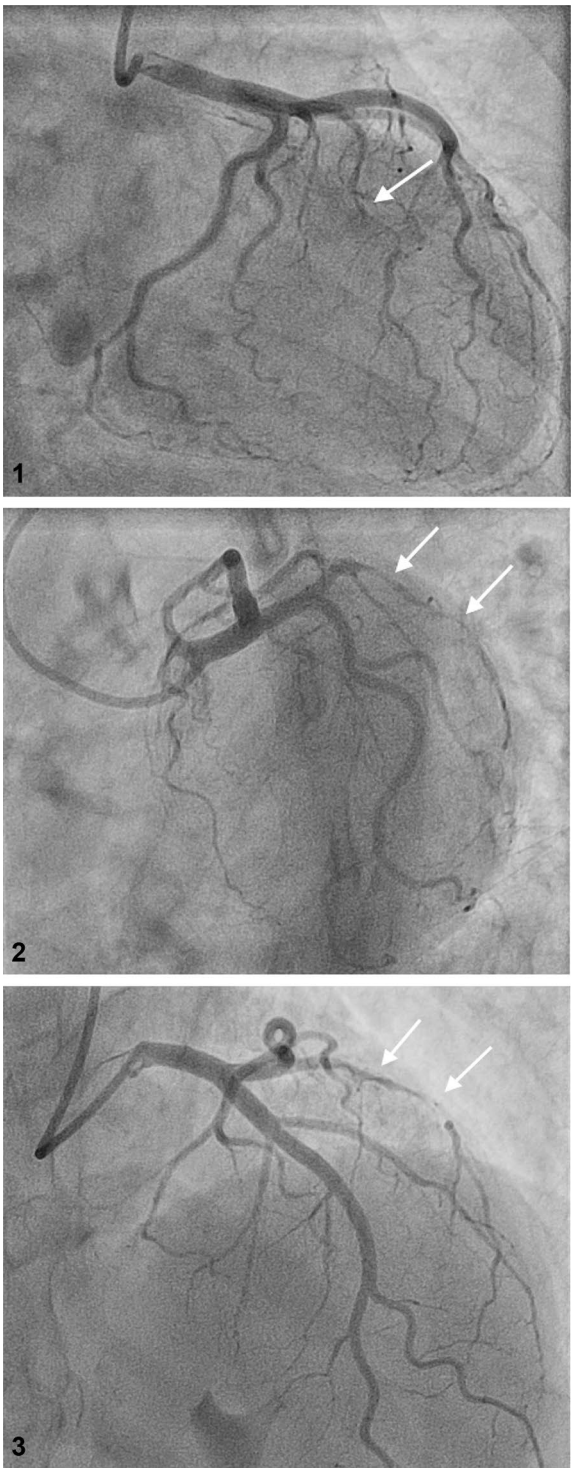

Fig. 1–3 Coronary angiography: Fig. 1 (right oblique caudo-projection). Figure 2 (left oblique caudo-projection). Figure 3 (cranial projection). A sharp intraluminal diameter loss was observed in the first marginal obtuse with images of tubular stenosis compatible with hematoma/dissection (white arrows)

total of eight cases of SCAD-SLE with clinical information were identified [5–12]. Table 1 shows the main clinical features found in these reports. According to the data extracted from previous cases, and including our report, 7 were females and 2 were males, with an average age of 36.2 years (range 17–53). In four patients, SCAD was the first manifestation leading to the diagnosis [5, 8, 9, 11]. Chest pain was the most common symptom, lasting at least one entire day in four cases. In our patient, pain was located in the inter-scapular region. Four patients reported dyspnea, three nausea, two diaphoresis and two dizziness. Cough, and headache occurred in 1 case each. Three patients presented ST elevation (STEMI), and four patients did not (NSTEMI). In two cases, data about ST status were not provided. All patients underwent confirmatory coronary angiography, and in one case intracoronary ultrasound was also performed. Confirmation of diagnosis was achieved at necropsy in one patient. In this cohort of SCAD-SLE patients, the left anterior descending coronary artery (LAD) was dissected in four cases, the posterior descending artery in one, the right coronary artery (RCA) in one, and the Cx in the remaining four, with involvement of the obtuse marginal branch in two cases. Two patients presented more than one coronary artery involvement. Medical treatment was administered in two patients. Four patients underwent percutaneous coronary intervention (PCI) with stent placement. In two cases, concomitant thrombolysis and PCI was performed. In one patient, placement of the stent was not possible during the PCI procedure, and conservative treatment was then administered. In one case, the treatment followed was not mentioned. The clinical course was favorable in eight cases, and one patient died.

Discussion

SCAD is defined as the dissection of an epicardial coronary artery not associated with atherosclerosis, trauma or iatrogenesis [1–3]. SCAD is a rare but potentially fatal entity described mainly in young women. It is considered the cause of MI in one-third of women under 50 years of age [1, 2]. The average age of SCAD patients at the moment of diagnosis varies within the different series varies from 45 to 55 years, although cases have been reported in patients under 20 and over 80 years [1, 2].

Patients with SCAD usually present as an ACS, along with laboratory findings compatible with myocardial necrosis [3]. Chest pain is the most common presentation symptom (95%), followed by nausea or vomiting (24%), diaphoresis (21%), dyspnea (20%), back pain (14%) and dizziness (9%) [1]. A certain delay in diagnosis is rather common due to atypical pain presentation [3]. The proportion of patients with SCAD who have a STEMI (26–87%) versus NSTEMI (13–67%) is variable according to previous reports [1, 3]. Coronary angiography, albeit some limitations, is the first-line diagnostic tool for SCAD [1–3]. Other diagnostic techniques (complementary to coronary angiography and definitive for diagnosis) include intracoronary ultrasound, optical coherence tomography and computed tomography coronary angiography (CTCA) [1, 3].

In patients with SLE, a high degree of suspicion is needed for the diagnosis of ACS, especially when they present with atypical chest pain, nausea and vomiting, dyspnea, decreased tolerance to exercise, diaphoresis or other atypical symptoms. The differential diagnosis of such symptoms in the patient with SLE is extensive and also includes pleuritis, pericarditis, pulmonary embolism, pneumonia, interstitial lung disease, aortic dissection, reflux esophagitis, pulmonary hypertension, myocarditis, as well as other cardiac manifestations of SLE. Coronary artery disease (CAD) is a common complication of SLE [13]. It is estimated that the pooled risk is three times higher in patients with SLE than in their control group [14, 15]. Moreover, women with SLE in the 35–44 age subgroup carry an increased risk of MI 50 times higher compared to non-SLE women [13]. It is believed that such adverse cardiovascular events are the result of an accelerated atherosclerotic process, that ultimately leads to the development of occlusive CAD [16–18]. It has been hypothesized that the systemic inflammation state occurring in SLE somehow promotes atherosclerosis of the coronary vessels [19]. Alternatively, the causative agent may not be SLE itself, but the use of corticosteroids and other immune-suppressive agents that influence CAD risk factors such as diabetes or hypertension [18, 20]. More rarely CAD may be attributable to coronary vasculitis, thrombosis of an angiographically normal coronary artery [11, 17, 21] or coronary artery aneurysm [22, 23]. SCAD is a less common cause of ACS in this subset of patients.

A rupture of the intima or an intramural hematoma is the pathogenic mechanisms underlying SCAD [1–3]. These injuries occur as a consequence of changes within the vascular structure that promote weakening of the vessel walls. Episodes occurring during pregnancy and post-partum suggest the potential role of hormonal changes causing alterations of the connective tissue, as well as hemodynamic stress [1–3]. Eosinophilic infiltration of the vessels followed by the release of lytic enzymes seems to trigger dissection [24]. In patients with SLE, there seems to be an increased susceptibility to spontaneous dissection attributed to chronic inflammation of the vessels [8]. Recent studies point to inflammatory cells and their mediators, especially tryptase, chymase and metalloproteinase, as the origin of SCAD and aortic dissection in SLE [25, 26]. The presence of aPL may also be another causative agent, as reported in several patients with antiphospholipid syndrome [27, 28], and in a patient with SCAD occurring in the post-partum [29], as well as

Table 1 Reported cases of SCAD-SLE

Ref	Age/sex	Years SLE	SCAD clinical features	ECG	SLE clinical features	aPL	Diagnosis	Coronary artery affected	Treatment	Outcome
5	39/F	Debut	NA	NA	Renal Haematol	NA	Coronary angiography	LAD	Thrombolysis PCI	Good
6	48/F	15	Chest pain Diaphoresis Dyspnea	STEMI	Arthritis Cutaneous	NA	Coronary angiography Necropsy	LAD Cx	PCI	Dead
7	17/M	NA	NA	NA	Multisystemic	NA	Coronary angiography	Cx (OMB)	NA	NA
8	31/M	Debut	NA	NSTEMI	Arthralgias Ulcers Raynaud Pleuritis Pericarditis	+	Coronary angiography Intracoronary ultrasound	Cx	Conservative	Good
9	27/F	Debut	Chest pain Epigastric Pain Nausea Cough Dyspnea Myalgia Headache	STEMI	Myalgias	–	Coronary angiography	Posterior descending	PCI Conservative	Good
10	35/F	5	Chest pain	NSTEMI	Neurological Cutaneous	+	Coronary angiography	LAD Diagonal	PCI	Good
11	33/F	Debut	Chest pain Dyspnea	NSTEMI	NA	–	Coronary angiography	LAD	PCI	Good
12	43/F	NA	Chest pain Dyspnea Nausea Diaphoresis Dizziness	STEMI	NA	NA	Coronary angiography	RCA	Thrombolysis PCI	Good
Present case	53/F	14	Chest pain Dizziness Sickness	NSTEMI	Arthritis Ulcers Raynaud	–	Coronary angiography	Cx (OMB)	Conservative	Good

aPL antiphospholipid antibodies, *Cx* circumflex artery, *F/M* female/male, *LAD* left anterior descending artery, *NA* not available, *NSTEMI* non-ST elevation myocardial infarction, *OMB* obtuse marginal branch, *PCI* percutaneous coronary intervention, *PCI uns* percutaneous coronary intervention unsuccess, *RCA* right coronary artery, *SCAD* spontaneous coronary artery dissection, *SCAD-SLE* spontaneous coronary artery dissection associated with systemic lupus erythematosus, *SLE* systemic lupus erythematosus, *STEMI* ST elevation myocardial infarction

some other cases of carotid dissection associated with these antibodies [8]. However, aPL antibodies were present only in two SCAD-SLE patients out of five in which such laboratory tests were performed (Table 1).

Although only eight cases with some clinical information have been previously described, SCAD is probably a more common cause of ACS in patients with SLE than reported in the literature. In a recent study on the pathologies associated with 66,360 patients diagnosed of SCAD, using the NIS database [4], a total of 280 patients had SLE. Interestingly, the percentage of patients with ACS plus SLE caused by SCAD was higher than the percentage of patients with SLE plus non-SCAD ACS [4].

A more conservative and individualized treatment is recommended for SCAD patients with SLE compared to ACS of atherosclerotic origin [1–3]. Non-invasive treatment, with antiplatelet aggregation agents, beta-blockers, angiotensin-converting enzyme inhibitors, statins and/or vasodilators [1–3], is preferred when blood flow is normal, there is no progression of symptoms and the patient stays hemodynamically stable. The use of heparin and dual antiplatelet therapy in SCAD remains controversial [1] and some authors have suggested than statins can increase the risk for recurrent SCAD [4, 30]. Percutaneous coronary intervention (PCI) is recommended in cases of marked damage of the epicardial coronary flow, or when hemodynamic instability or progressive ischemia ensue [1–3]. However, SCAD often affects small distal segments of the coronary arteries, making it difficult to treat with stents. Coronary bypass surgery is usually indicated for dissections that affect the left main coronary artery or cases with multiple vessel involvement. Surgery is also performed in cases of technical failures and/or complications related to PCI, and when ischemia is refractory to conservative therapy [1–3]. Finally, thrombolysis is contraindicated in the treatment of acute SCAD according to the recommendations of the European Society of Cardiology [3]. Although, some patients receiving thrombolytics may recover, the risk of hematoma and worsening of the dissection are worrisome concerns. Whether patients with SCAD-SLE, undergoing conventional treatment of SCAD, need to increase their immune-suppressive therapy or receive drugs that inhibit the release of proteases by inflammatory cells, is still under research [25, 26].

Although more data are needed on the mid- and long-term follow-up of these patients, the rate of recurrence of SCAD has been reported to vary between 17% at 4 years and 30% at 10 years [3]. Given the SCAD recurrent risk, patients presenting with recurrent chest pain require careful assessment with serial electrocardiography (ECG) and high sensitivity troponin measurement [3]. Invasive coronary angiography should be reserved for patients with hard evidence of ischaemia or myocardial necrosis. Stress echocardiography is highly valuable in assessment of SCAD patients with recurrent chest pain [31]. A role for CTCA to rule out recurrent SCAD [32], although potentially attractive, remains to be clearly elucidated [3]. SCAD involvement of distal coronary arteries or side branches is not usually well visualized on CTCA, so this technique is of limited value in many SCAD cases [1, 31]. However, CTCA can be quite useful in the follow-up when the culprit artery is previously recognized by coronary angiography [32].

In conclusion, we report a new case of SCAD-SLE. Although coronary atherosclerotic disease is the main cause of ACS in patients with SLE, SCAD is probably a more common cause of ACS in patients with SLE than previously reported in the literature. Early recognition of SCAD is important because the therapeutic approach, generally a less-invasive scheme, differs from the usual treatment of atherosclerotic ACS. A possible SCAD should be suspected in patients with SLE and ACS, especially in younger women without conventional cardiovascular risk factors.

Acknowledgements The authors wish to thank the Research Unit of Hospital Universitario de Burgos (HUBU) for their help in the preparation of the English version of the manuscript.

Author contributions The case was diagnosed and followed up by SMG, BAL and JMGG. BAL and LAR conceived and planned the study. BAL and LAR wrote the manuscript. Literature data were searched and analyzed by all the authors. The final version was read, corrected and approved by all the authors.

Funding The authors received no financial support for the research, authorship, and/or publication of this article.

Compliance with ethical standards

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval All the procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. All the authors fulfilled the ICMJE authorship criteria.

Informed consent Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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