Journal of Cardiology 63 (2014) 119–122

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Clinical features of spontaneous coronary artery dissection

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ARTICLE INFO

Article history:
Received 8 April 2013
Received in revised form 14 June 2013
Accepted 1 July 2013
Available online 4 September 2013

Keywords:
Spontaneous coronary artery dissection
Acute coronary syndrome
Female
Peripartum or postpartum
Low coronary risk factors

ABSTRACT

Background: Spontaneous coronary artery dissection (SCAD) is an infrequent but increasingly recognized cause of acute coronary syndrome (ACS). Previous case reports demonstrated that this condition occurs in young females with a low atherosclerotic risk factor burden and may be associated with peripartum or postpartum status. The purpose of this study was to review patients with angiographically confirmed SCAD to provide additional insight into the diagnosis and treatment of this condition.
Methods and results: We screened medical records of all patients with ACS from March 2001 to November 2012. From these patients, we selected patients with SCAD based on coronary angiographic review. Of a total of 1159 ACS patients, 10 patients (0.86%) were diagnosed with SCAD. The mean age of these patients was 46 years, and 9 were female. ST-elevation myocardial infarction (STEMI) was observed in 9 patients and 5 patients had no coronary risk factors. One patient was treated conservatively with medication alone and 3 patients underwent thrombectomy. Balloon angioplasty was performed in 2 patients, and a bare metal stent was placed in one of these patients later. In the remaining 4 patients, bare metal stents were implanted emergently. Follow-up coronary angiography showed appropriate repair of SCAD in all 10 patients.
Conclusions: In our experience, the clinical features of SCAD appear to be similar to those reported previously. SCAD appears to be rare, but it should be considered in ACS patients, especially in younger females.

Introduction

Spontaneous coronary artery dissection (SCAD) is an infrequent but increasingly recognized cause of acute coronary syndrome (ACS). Although it is not related to atherosclerosis, its origin is unknown. Case series of SCAD excluding atherosclerotic dissection demonstrate a young, female preponderance with a low burden of atherosclerotic risk factors and an association with peripartum or postpartum status [1–7]. Other SCAD associations identified from case reports include connective tissue disorders, spasm, and excessive exercise [1,4,8–11]. This suggests some underlying vascular predisposition, although a unifying structural vessel wall abnormality has not yet been identified. Treatment of SCAD may vary from conservative management to coronary revascularization such as percutaneous coronary intervention (PCI) [12] or coronary artery bypass grafting, according to its type and severity. However, the optimal treatment strategy remains unknown. In this report, we review patients with angiographically confirmed SCAD, who were admitted to Dokkyo Medical University hospital with a diagnosis of ACS from March 2001 to November 2012.

Methods

We screened medical records for all ACS patients who were admitted to Dokkyo Medical University hospital and underwent emergent coronary angiography from March 2001 to November 2012. From these ACS patients, we selected the patients with SCAD based on coronary angiographic review. The angiographic criteria for SCAD were the presence of a dissection plane together with the absence of coronary atherosclerosis. Patients with other diagnoses such as iatrogenic coronary artery trauma and atherosclerotic plaque dissection were excluded. Patient demographics, potential etiologic associations, clinical presentation, coronary distribution, treatment modality, and in-hospital and long-term outcomes were determined through medical record and angiographic review. This

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http://dx.doi.org/10.1016/j.jjcc.2013.07.001

retrospective study was approved by the local institutional review board. All SCAD patients included in the study agreed to the use of their records for research.

Results

Of the 1159 ACS patients, including 926 (79.9%) with ST elevation myocardial infarction (STEMI), 185 (16.0%) with non-STEMI, and 48 (4.1%) with unstable angina pectoris (UAP), who underwent emergent coronary angiography, 10 patients (0.86%) were diagnosed with SCAD. In these 10 SCAD patients, STEMI was seen in 9 patients (90% of all SCAD patients and 0.97% of all STEMI patients), and the remaining case was diagnosed as UAP (10% of all SCAD patients and 2.1% of all UAP patients. Characteristics of these 10 SCAD patients are shown in Table 1. Typical coronary angiographic and intravascular ultrasound (IVUS) findings (case 7) are shown in Fig. 1. Coronary angiography showed that the left anterior descending artery (LAD) was occluded proximally, and a smooth contour of the left circumflex artery (LCX) wall indicated a low probability of atherosclerotic coronary artery disease. The IVUS findings showed that coronary artery dissection was present, where the true lumen was compressed by the hematoma of the false lumen. The 10 patients with SCAD had a mean age of 46 ± 17 years, and 9 (90%) were female. Of these, one patient (case 7) was 38 weeks pregnant and another (case 9), the only UAP patient, was post-partum. Case 5 was a 12 year-old female who had Marfan syndrome. Two patients (cases 6 and 7) developed ventricular fibrillation that required electrical defibrillation. When single-vessel dissection was present (9 cases), the location of dissection was the LAD in 3 patients (cases 1, 5, and 7), the right coronary artery (RCA) in 5 (cases 3, 6, 8, 9, and 10) and the LCX in one (case 4). The remaining patient (case 2) had multivessel dissection that included the LAD and RCA. Of the 4 coronary risk factors including hypertension, diabetes, smoking, and dyslipidemia (low-density lipoprotein cholesterol >130 mg/dl, high-density lipoprotein cholesterol <40 mg/dl, and/or triglyceride >150 mg/dl), 1 patient (case 6) had 2 risk factors, 4 patients (cases 3, 5, 7, and 8) had 1 risk factor, and the remaining 5 patients (cases 1, 2, 4, 9, and 10) had no risk factors. Thus, no patient had more than 3 risk factors.

UAP in case 9 was treated conservatively with a calcium channel blocker alone, because blood flow in the dissected RCA was maintained and there were no symptoms thereafter. Follow-up coronary angiography on day 20 showed that the dissection was spontaneously and completely repaired. Three (cases 1, 3, and 8) of 9 patients diagnosed with STEMI underwent thrombectomy by catheter aspiration alone. These patients were also asymptomatic thereafter, and coronary angiography at long-term follow-up (mean 7.3 months) showed that the dissection was completely repaired. Two STEMI patients (cases 5 and 7) underwent PCI with balloon angioplasty alone. One of these 2 patients (case 7) was 38 weeks pregnant and underwent Cesarean section surgery just after balloon angioplasty. Follow-up coronary angiography on day 21 showed that the culprit dissection in the LAD remained and there was a new dissection in the LCX. Therefore, the dissections in both LAD and LCX were repaired with bare metal stents. In case 5, a 12-year-old female with Marfan syndrome, balloon angioplasty was performed under IVUS guidance for total occlusion of the LAD caused by SCAD. Although the dissection progressed after balloon angioplasty because of the fragile vessel wall, there was sufficient blood flow after reperfusion so a stent was not placed. Thereafter, the patient was symptom free. Follow-up coronary angiography on day 28 showed that the LAD was occluded again. However, collateral flow to LAD area had developed from the RCA. In the remaining 4 STEMI patients (cases 2, 4, 6, and 10), bare metal stents were placed during the acute phase. Of these 4 patients, case 6 developed stent thrombosis on day 6 and again underwent PCI. The remaining 3 patients (cases 2, 4, and 10) had a stable course after stenting. Case 6 also had a stable course after the second PCI. In all of these 4 patients undergoing emergent stent implantation, long-term follow-up coronary angiography (mean 7.3 months) showed appropriate repair of the SCAD without stent thrombosis or in-stent stenosis. In 9 SCAD patients, who received catheter revascularization, IVUS was conducted for dissected vessels before and/or after treatment, and consequently, no apparent atherosclerotic plaques were observed in any of the target vessels.

Discussion

Based on the features of the 10 patients with SCAD in this study, we propose that SCAD occurs predominantly in females and younger individuals, and is independent of coronary risk factors. Additionally, thrombus aspiration alone is one of the possible strategies in selected patients with SCAD.

Although the population-based incidence of SCAD is unknown, retrospective registry studies have reported SCAD detection in 0.07–1.1% of all patients undergoing coronary angiography [13–18]. Recently, Tweet et al. [19] reported the results of a retrospective single-center cohort study identifying 87 patients with angiographically confirmed SCAD. In that report, they describe several issues as follows. First, SCAD affects a young, predominantly female population, and frequently presents as STEMI. Next, in-hospital mortality is low regardless of initial treatment, but PCI is associated with a high rate of complications. Third, the risks of SCAD recurrence and major adverse cardiac events in the long term indicate the need for close follow-up. Finally, fibromuscular dysplasia is a novel association and potentially causative factor. Our experience with 10

Table 1
Characteristics of the patients with SCAD.

No	Age	Gender	Onset	Location	HT	DM	Dyslipidemia	Smoking	LDL-C	HDL-C	TG	Treatment	Note
1	37	F	STEMI	LAD	–	–	–	+	93	40	148	Thrombectomy	History of toxicosis
2	74	F	STEMI	LAD/RCA	–	–	–	–	74	54	50	Stenting	History of cerebral infarction
3	54	F	STEMI	RCA	+	–	–	–	79	84	37	Thrombectomy	
4	47	F	STEMI	LCX	–	–	–	+	127	43	219	Stenting	
5	12	F	STEMI	LAD	–	–	–	–	67	13	241	Ballooning	Marfan syndrome, reocclusion
6	61	F	STEMI	RCA	+	–	–	+	37	25	122	Stenting	Vf, Complicated with aortic dissection
7	43	F	STEMI	LAD	–	–	–	–	113	55	254	Ballooning	Vf, Pregnant 38 W, Cesarean section
8	54	M	STEMI	RCA	–	–	+	+	129	64	78	Thrombectomy	
9	36	F	UAP	RCA	–	–	–	–	90	51	89	Medication	Postpartum 2 M
10	45	F	STEMI	RCA	–	–	–	–	70	52	59	Stenting	
Mean	46								88	48	130		
SD	17								29	20	82		

SCAD = spontaneous coronary artery dissection, F = female, M = male, STEMI = ST-elevation myocardial infarction, UAP = unstable angina pectoris, LAD = left anterior descending artery, RCA = right coronary artery, LCX = left circumflex artery, HT = hypertension, DM = diabetes mellitus, LDL-C = low density lipoprotein cholesterol, HDL-C = high density lipoprotein cholesterol, TG = triglyceride, Vf = ventricular fibrillation