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ORIGINAL ARTICLE
Ischemic Heart Disease

Usefulness of Repeat Angiography to Establish Spontaneous Coronary Artery Dissection in Small Vessels

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Background: Spontaneous coronary artery dissection (SCAD) is a rare disease that is often misdiagnosed, except in typical cases. Although intracoronary imaging and multislice coronary computed tomography angiography (CCTA) are useful in establishing dissection, they may not be feasible in all instances, especially in small vessels.

Methods and Results: We describe a series of 7 patients with acute coronary syndrome secondary to small vessel SCAD that was detected only upon repeat coronary angiography (CAG). This cohort had a mean (±SD) age of 50±6 years, was predominantly female (n=6; 86%), and had few coronary risk factors. Three patients (43%) had dissection of the distal segment of the right coronary artery, 3 (43%) had distal left circumflex artery dissection, and 1 patient (14%) had a diagonal branch dissection. None of the patients required percutaneous coronary intervention, and received conservative therapy only, because the infarct area was sufficiently small. No definitive diagnosis of SCAD could be established in any of the patients at first admission because CAG alone or CCTA did not reveal the presence of a flap or intraluminal hemorrhage. However, in such patients without a definitive diagnosis, repeat CAG in the chronic stage showed enlargement of vessels, suggesting the healing of an SCAD.

Conclusions: Repeat CAG may be useful for suggesting the occurrence of SCAD.

Key Words: Coronary angiography; Diagnosis; Spontaneous coronary artery dissection

Spontaneous coronary artery dissection (SCAD) is defined as the dissection of an epicardial coronary artery that is not associated with atherosclerosis or trauma and is not iatrogenic. SCAD is a rare disease, and its reported prevalence in acute coronary syndrome (ACS) ranges between 0.1% and 4%.[1,2] ACS is defined as unstable angina and acute myocardial infarction.[3] Coronary angiography (CAG) is one of the major diagnostic modalities for SCAD. The Saw coronary angiogram classification for SCAD[4] defines 3 types of CAG findings: Type 1, which refers to the pathognomonic angiographic appearance of SCAD with contrast dye staining of the arterial wall and the presence of multiple radiolucent lumens; Type 2, which refers to the presence of diffuse stenosis of varying severity and length; and Type 3, which represents focal or tubular stenosis, usually <20 mm in length, that mimics atherosclerosis. Although Type 1 SCAD is clearly recognized as a flap during CAG, Types 2 and 3 are sometimes impossible to diagnose by CAG alone, and intracoronary imaging techniques such as intravascular ultrasonography (IVUS) or optical coherence tomography (OCT), as well as multislice coronary computed tomography angiography (CCTA), are useful for establishing SCAD. Nonetheless, diagnosing SCAD in small vessels is challenging because of technical issues, such as non-delivery of the intracoronary imaging device or insufficient resolution for detection using CCTA.

Here we describe a series of 7 cases in which SCAD in small vessels was detected only on repeat CAG in the chronic stage.

Patient Characteristics

Between 2010 and 2019, 7 patients with SCAD in the small vessels were treated at Niigata University Medical and Dental Hospital and Niigata Prefectural Shibata Hospital.

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Table. Baseline Characteristics of Patients With Spontaneous Coronary Artery Dissection

Patient no.	Sex	Age (years)	Presentation at admission	Coronary risk factors				Dissection site	TIMI flow	Therapy	Peak CK (IU/L)
				Diabetes	HT	HL	Smoking				
1	Female	41	NSTEMI	0	0	0	0	12	3	Conservative	1,070
2	Female	49	STEMI	0	0	0	0	12	3	Conservative	535
3	Female	55	NSTEMI	0	0	1	0	4PD	3	Conservative	381
4	Female	57	STEMI	0	0	1	0	4AV	3	Conservative	287
5	Male	54	STEMI	0	0	0	0	4AV	2	Conservative	180
6	Female	43	NSTEMI	0	0	0	1	9	3	Conservative	1,523
7	Female	54	NSTEMI	0	0	0	0	12	3	Conservative	135

4AV, atrioventricular branch; 4PD, posterior descending artery; CK, creatine kinase; DM, diabetes; HL, hyperlipidemia; HT, hypertension; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TIMI, Thrombolysis in Myocardial Infarction.

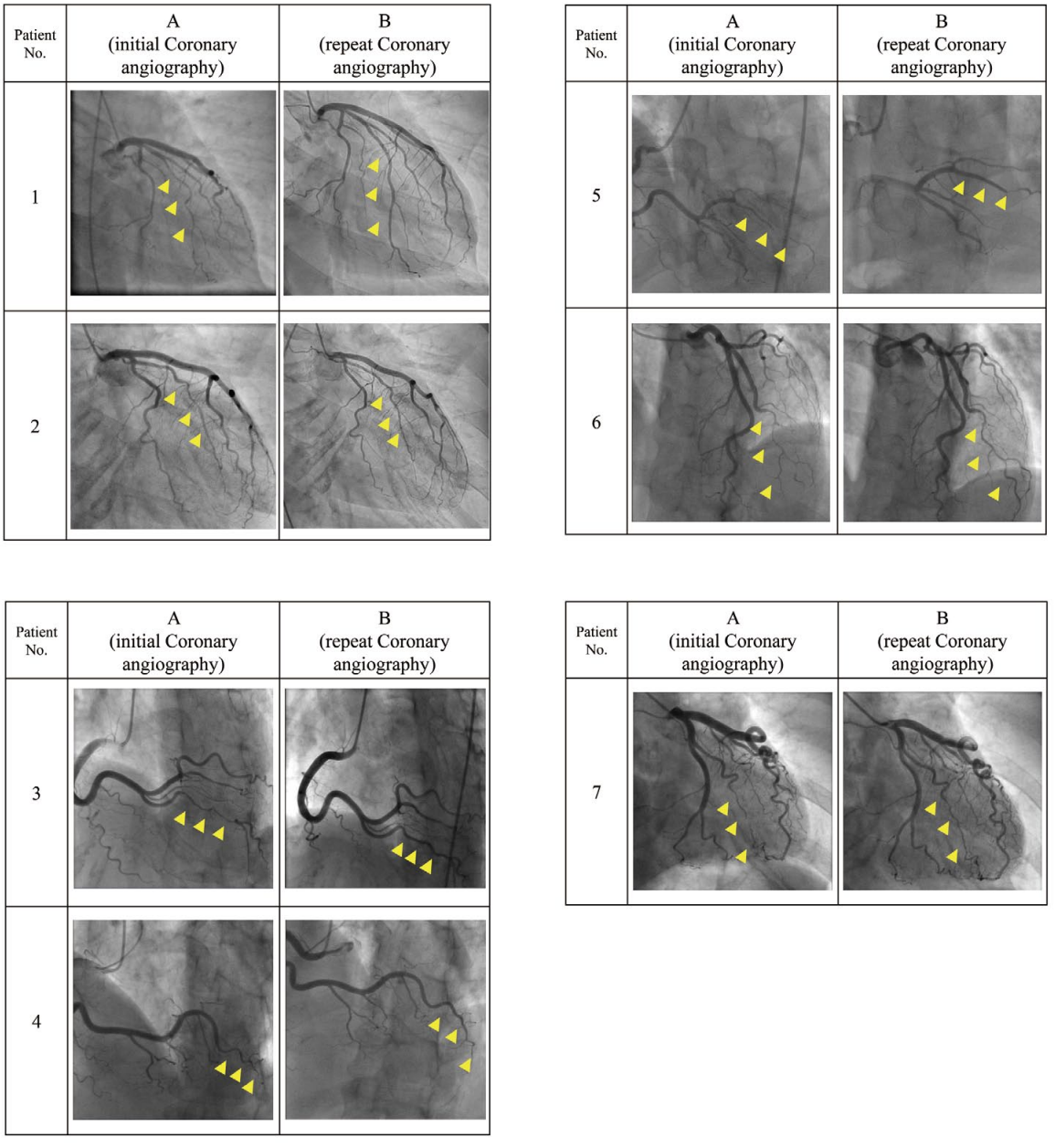

Figure 1. (**A**) Initial and (**B**) repeat coronary angiographies in all 7 patients. Arrowheads indicate the location of the dissection.

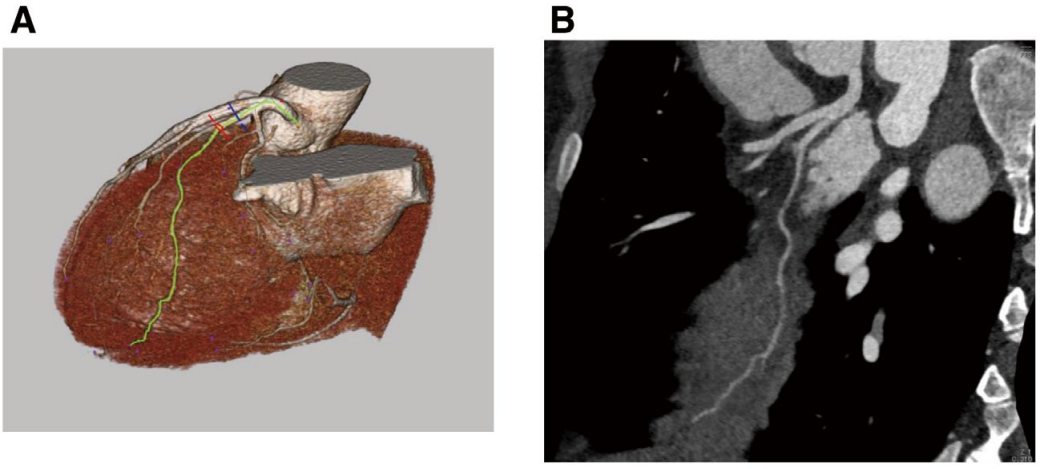

Figure 2. Multislice coronary computed tomography angiography of Patient no. 1. (**A**) Volume rendering image. (**B**) Curved multiplanar reformation of the circumflex artery.

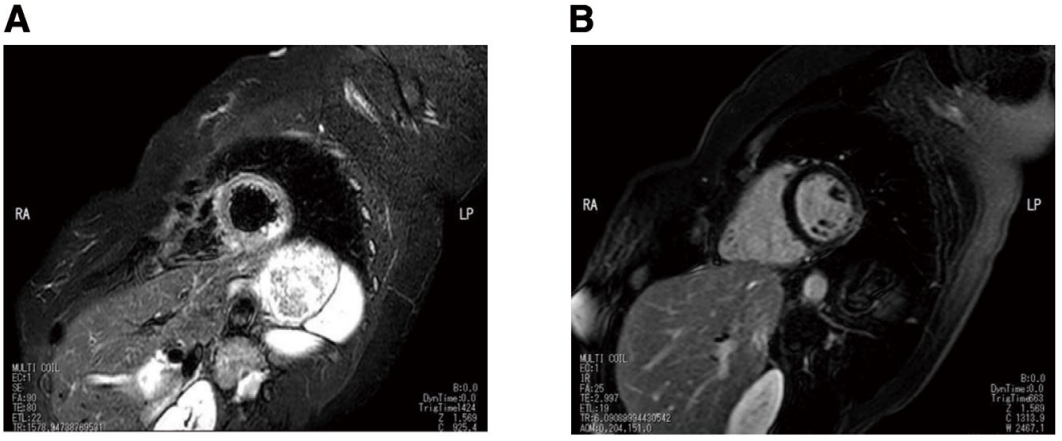

Figure 3. Magnetic resonance imaging of Patient no. 1 showing (**A**) a T_2-weighted image and (**B**) a late gadolinium-enhanced image.

The baseline characteristics of these patients are given in **Table**. The mean (±SD) patient age was 50±6 years, 6 (86%) were female, and they had few coronary risk factors. ST-elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI) at admission were seen in 3 (43%) and 4 (57%) patients, respectively, and none of the patients exhibited cardiogenic shock. The location of the dissection was the distal segment of the right coronary artery (RCA) in 3 patients (43%), the distal segment of the left circumflex artery (LCX) in 3 patients (43%), and the diagonal branch in 1 patient (14%). None of the patients required percutaneous coronary intervention and all were managed using conservative therapy because the infarct area was small.

Median peak creatine kinase (CK) levels were 381 U/L (interquartile range [IQR] 234–803 U/L), reflecting the presence of only a small infarction. The patients were discharged without any complications. All patients underwent repeat CAG a median of 4 months (IQR 0.75–8 months) after the initial admission. Written informed consent was obtained from all patients before repeat CAG.

Initial and repeat CAGs of all patients are shown in **Figure 1**. No definitive diagnosis of SCAD could be established at the time of initial admission because neither CAG alone nor CCTA showed the presence of a flap or intraluminal hemorrhage. Nonetheless, in these patients, repeat CAG in the chronic stage showed enlargement of vessels, suggesting healing after SCAD.

We describe in detail the case of 1 typical patient in this cohort (Patient no. 1), in whom a definitive diagnosis of SCAD could not be made at the time of initial admission. Patient no. 1, a 41-year-old woman, presented to the emergency room at Niigata University Medical and Dental

Hospital with a 1-h history of chest pain. The electrocardiogram showed no significant ST-T change, and transthoracic echocardiography showed no apparent asynergy. The next day, the patients CK and CK-MB concentrations increased to 1,070 and 101 U/L, respectively, but CCTA using 64-slice multidetector computed tomography (**Figure 2**) revealed no significant stenosis. However, because T_2-weighted and late gadolinium-enhanced magnetic resonance imaging (MRI; **Figure 3**) showed endocardial infarction in the distal segment of the LCX, cardiac catheterization was performed. CAG revealed diffuse narrowing of the distal segment of the LCX (**Figure 1A**, Patient no. 1), but with sufficient flow, and vasospasm was not provoked by acetylcholine administration[5] (i.e., the vasospasm test was negative). Importantly, the narrowing of the LCX was not enlarged by the administration of intracoronary isosorbide dinitrate and, based on MRI findings, this distal segment of the LCX was determined to be the etiological agent of the myocardial infarction. Although the LCX lesion was suspected to be a SCAD, it was impossible to definitely establish the same because pertinent imaging devices could not be delivered. Repeat CAG was performed 9 months later, showing that the LCX lesion had healed and enlarged (**Figure 1B**, Patient no. 1). These repeat CAG findings are suggestive of the occurrence of SCAD in the distal segment of the LCX at the time of initial presentation.

Discussion

SCAD is a rare disease, with a reported prevalence in ACS of 0.1–4%.[1,2] Female sex, fibromuscular dysplasia, vascular tortuosity, a peripartum state, physical and emotional stress, and connective tissue disorders are factors known to be related to SCAD.[6–8] The major cause of myocardial injury in SCAD is insufficient coronary flow due to compression by an intramural hematoma or a false lumen. The most commonly involved vessel is the left anterior descending artery and its branches (45–61% of cases), followed by the LCX and its branches (15–45%), the RCA and its branches (10–39%), and the left main trunk in up to 4% of cases.[7,9–11] In most cases, the mid to distal segments of the coronary arteries are affected, with proximal site involvement seen in only 8.3% of cases.[7] According to the Saw coronary angiogram classification system,[4] SCAD can be classified into 3 types. Type 2 (diffuse smooth stenosis) is the most common angiographic manifestation of SCAD, occurring in up to 67.5% of cases. This is followed by Type 1 (evident arterial wall stain) in 29.1% of cases and Type 3 (mimic atherosclerosis) in 3.4% of cases.[7]

SCAD Types 2 and 3 can be impossible to diagnose by CAG alone. Intracoronary imaging devices such as IVUS and OCT are useful for diagnosing SCAD because they can detect intimal tears, the presence of a false lumen, and intramural hematomas. However, in cases of distal lesions or SCAD in small vessels, intracoronary imaging devices may not be delivered to the lesion site or they may exacerbate SCAD. CCTA is also a useful and non-invasive diagnostic tool; however, small artery dissections (especially those <2.5 mm in diameter) may not be detected due to limitations in CT resolution.[12–14]

In this report we present details of 7 patients with SCAD in the small vessels, which were evident only upon repeat CAG. Saw proposed a stepwise algorithm to diagnose non-atherosclerotic SCAD,[4] according to which early invasive CAG is recommended if SCAD is suspected. OCT or IVUS is considered useful for arriving at a definitive diagnosis and, if there are concerns about compromised arterial flow on intracoronary imaging, a repeat angiogram should be performed. However, few studies have reported initial angiogram images and those after healing.

Previous studies have reported that SCAD lesions heal in most patients (70–97%) without any intervention, but that this could take anywhere between weeks and months.[4,9,10,15,16] Therefore, conservative therapy is currently recommended for SCAD and percutaneous coronary intervention is not indicated, except if there is evidence of ongoing ischemia, shock, or left main dissection.[7,17] Further, if SCAD cannot be established beyond doubt at initial CAG or with other diagnostic techniques, repeat CAG may reveal the healing of the vessels, which suggests initial SCAD occurrence.

A limitation of this study is that it was not possible to prove whether SCAD actually occurred, even if the artery appeared to be healed upon repeat angiography, because the latter does not prove the existence of a dissection. Nonetheless, in cases where SCAD actually occurs in the small vessels but cannot be established at initial admission, repeat angiography to confirm artery healing is useful, because it is suggestive of SCAD.

Conclusions

In cases where SCAD is suspected but cannot be established at initial CAG or by other diagnostic techniques, repeat CAG may reveal vessel healing, which is suggestive of SCAD occurrence.

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Disclosures

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IRB Information

The local ethics committee of the Niigata University School of Medical and Dental Sciences determined that ethics approval for this report was unnecessary.

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