

# Spontaneous coronary artery dissection by intravascular ultrasound in a patient with myocardial infarction

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Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome and sudden cardiac death. The incidence, causes, pathogenesis, and treatment have not been defined clearly, but spontaneous coronary artery dissection should be considered in young patients without major cardiovascular risk factors or in patients in the peripartum period who present with acute coronary syndrome. The treatment is often challenging. Medical treatment is usually considered, and percutaneous coronary intervention or coronary artery bypass surgery may be possible in some patients. We herein report two cases of middle-aged males with myocardial infarction who were treated with percutaneous coronary angioplasty.

**Keywords:** Coronary artery dissection, spontaneous; Myocardial infarction; Coronary angiography; Ultrasonography, interventional; Percutaneous coronary intervention

## INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and sudden cardiac death. The first case of SCAD was reported by Pretty [1] in 1931, while the first angiographic diagnosis was not made until 1978 [2]. SCAD occurs mostly in female patients during the peripartum period, but it can also occur in patients with atherosclerosis or in patients without any of these features [3,4]. Its incidence, etiology, and pathogenesis remain unclear. The clinical presentation of SCAD depends on the extent and flow-limiting severity of the coronary dissection. The condition ranges from asymptomatic state to the development of unstable angina, acute myocardial infarction, ventricular arrhythmia, and sudden cardiac

death. There is no definite guideline on how to manage patients with SCAD [5]. In this report, we describe two patients with myocardial infarction caused by SCAD who were treated with percutaneous coronary intervention (PCI).

## CASE REPORTS

### Case 1

A 60-year-old male presented with dizziness and chest discomfort. The patient had experienced severe crushing chest pain with sweating 15 years ago. However, the patient did not visit the hospital at that time because the chest pain completely resolved after several hours. He was a 20-pack-year smoker and had been taking a

calcium channel blocker, an angiotensin receptor blocker, and a hydrochlorothiazide for hypertension.

Electrocardiography showed the Q wave in the V<sub>1</sub>, V<sub>2</sub>, and V<sub>3</sub> leads. Transthoracic echocardiography showed akinesia of the mid to apical left ventricular (LV) anteroseptal wall. Cardiac enzymes were within their normal ranges and remained normal throughout the follow-up.

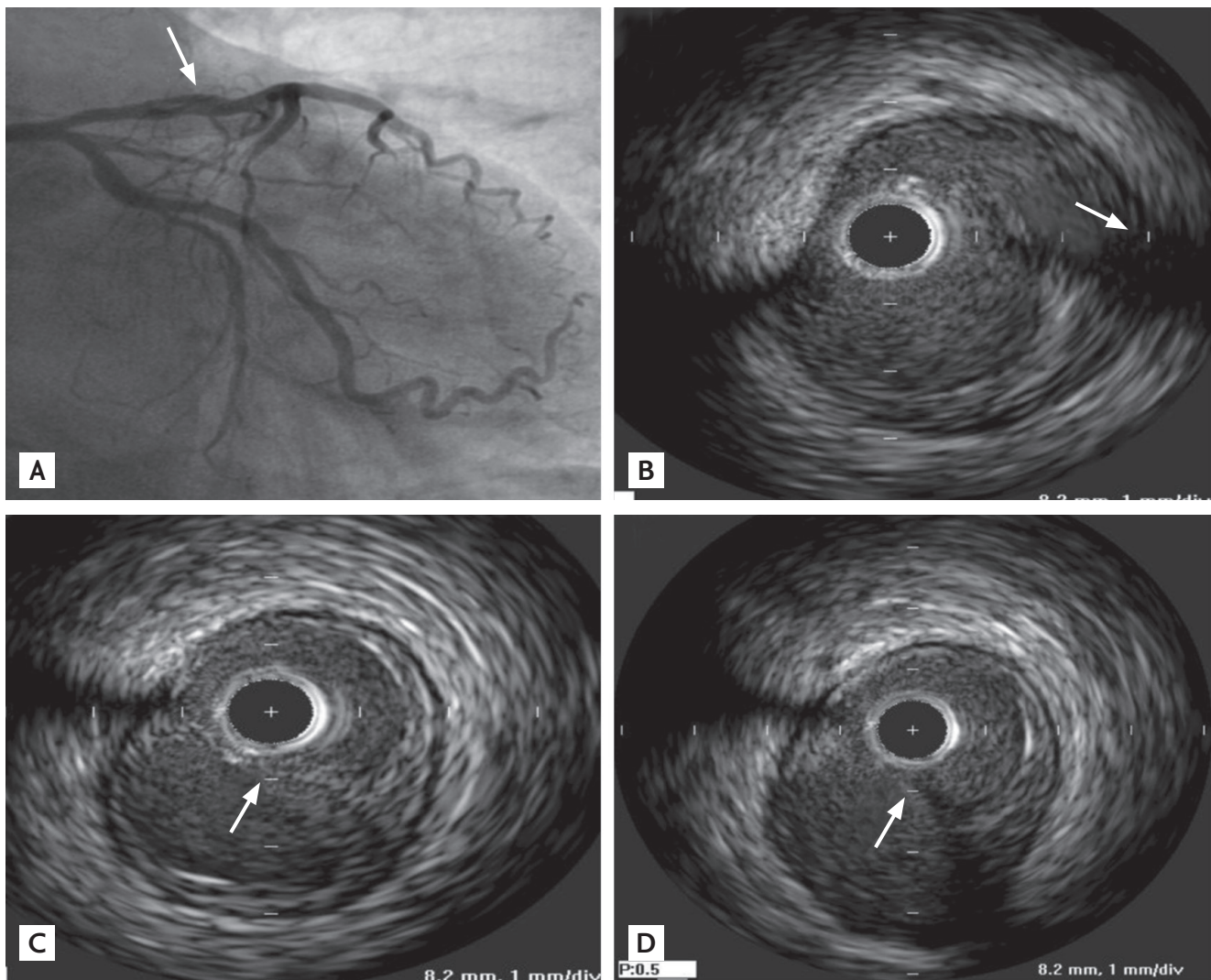
Coronary angiography revealed a dual lumen separated by a flap-like defect, indicative of intimal dissection in the proximal left anterior descending artery (LAD) (Fig. 1A). Intravascular ultrasonography (IVUS) showed an entry intimal tear proximal to the dissection site in the LAD (Fig. 1B). Fig. 1C shows an intimal

flap dividing the true lumen from the false one at the dissection site. The false lumen had a larger area than the true lumen. Fig. 1D demonstrates a re-entry intimal tear distal to the dissection site.

Percutaneous coronary angioplasty was performed, and a 4.0 × 22.0-mm everolimus-eluting stent (Xience Prime, Abbott Vascular, Santa Clara, CA, USA) was inserted at the proximal LAD lesion.

### Case 2

A 40-year-old male was admitted to our hospital because of frequent chest pain. The patient had experi-



**Figure 1.** (A) Intimal dissection (arrow) in proximal left anterior descending artery (LAD) by coronary angiography, (B) entry (arrow) of intimal tear of the proximal to dissection site in the LAD by intravascular ultrasonography (IVUS), (C) intimal flap (arrow) dividing between true lumen and false lumen in the dissection site by IVUS, (D) re-entry (arrow) of intimal tear of the distal to dissection site by IVUS.

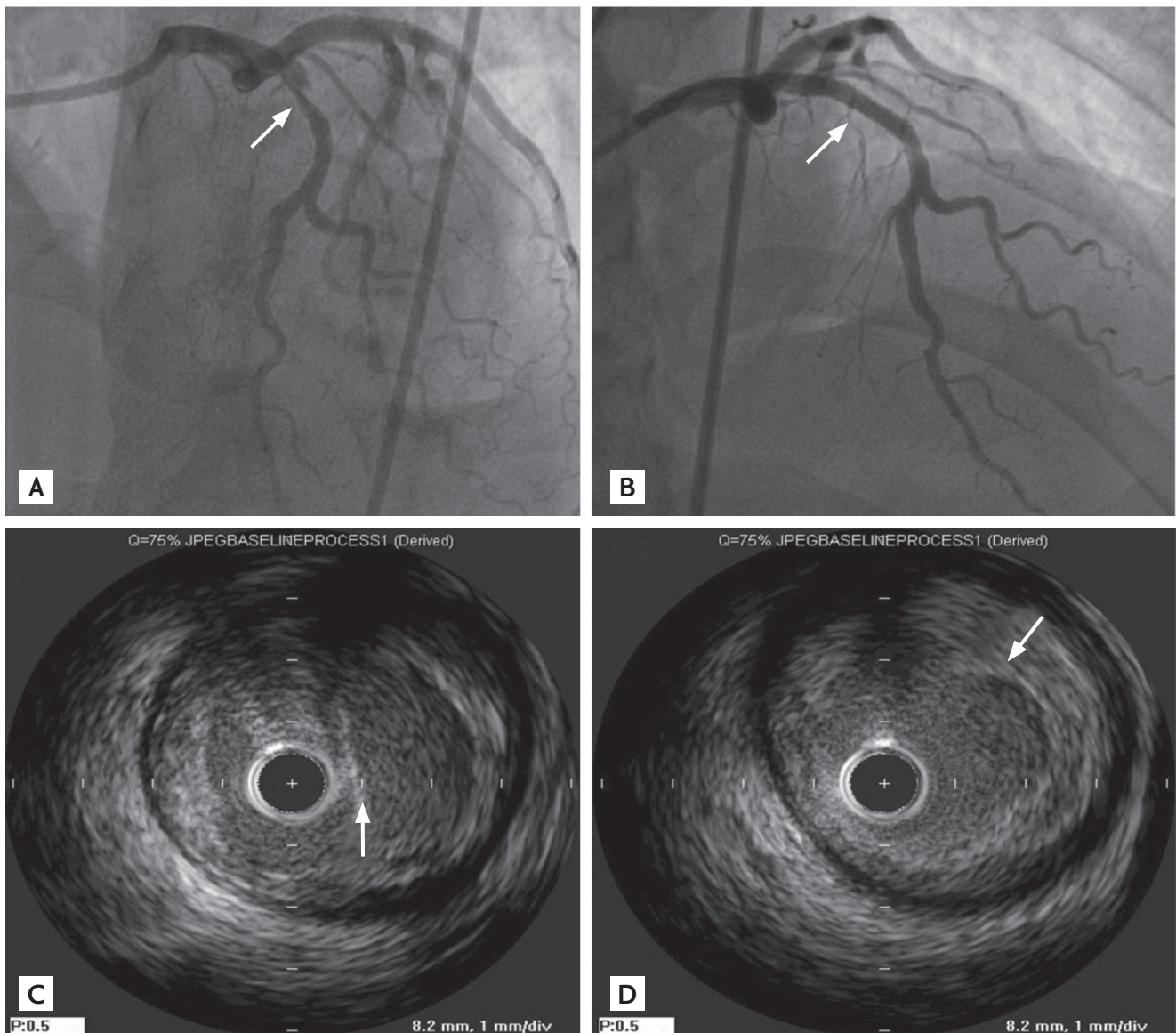
enced severe squeezing chest pain 3 months previously, and he had developed frequent chest discomfort with dyspnea since then.

He was a 20-pack-year smoker. He had been taking a calcium channel blocker, an angiotensin receptor blocker, and statin for hypertension and hyperlipidemia for the past 10 years.

Electrocardiography showed ST elevation in the V<sub>1</sub> to V<sub>4</sub> leads and reciprocal ST depression in leads II, III, and aVF. Transthoracic echocardiography showed pre-

served LV global systolic function with no definite regional wall motion abnormality. Cardiac enzymes demonstrated mildly elevated troponin I, which was initially 0.2 ng/mL and peaked at 0.3 ng/mL during the follow-up.

Coronary angiography showed a dual lumen defect, indicative of intimal dissection in the mid LAD (Fig. 2A). The other coronary arteries were angiographically normal. IVUS showed an intimal flap dividing the true lumen and false lumen at the mid LAD site (Fig. 2C and 2D).



**Figure 2.** (A) Coronary angiography revealing intimal dissection (arrow) in mid left anterior descending artery, (B) immediate poststenting coronary angiography, (C) intimal flap (arrow) dividing between true lumen and false lumen in the dissection site by intravascular ultrasonography (IVUS), (D) entry (arrow) of false lumen site in the dissection site by IVUS.

Percutaneous coronary angioplasty was performed, and a 4.0 × 22.0-mm zotarolimus-eluting stent (Resolute Integrity, Medtronic, Santa Rosa, CA, USA) was inserted at the mid LAD lesion.

After stent insertion, resolution of the initial dissection area was confirmed angiographically (Fig. 2B).

## DISCUSSION

This report illustrated two cases of myocardial infarction caused by SCAD in middle-aged males who were treated with percutaneous coronary angioplasty.

The incidence of SCAD is low, reportedly 0.07% to 1.10% among patients referred for coronary angiography. It occurs most often in young females (aged < 40 years) and frequently in the peripartum period. Although our cases were more unusual in that they involved SCAD in the LAD of middle-aged males, SCAD is reported to predominantly involve the LAD in females and right coronary artery in males. It has rarely been associated with connective tissue disease, vasculitis, inflammatory disorders, oral contraceptive use, exercise, prolonged sneezing, and cocaine abuse. Thus, it should be suspected in a young patient without major cardiovascular risk factors or in a patient in the peripartum period presenting with acute coronary syndrome or sudden cardiac death [6-10]. Our patients had no underlying conditions, and their SCAD can be considered idiopathic. In addition, SCAD seemed to persist for more than 10 years after myocardial infarction in the first patient. While a weak association between smoking and hypertension has been reported, classic risk factors seem unlikely to be related to SCAD [6]. Coronary atherosclerosis is known to be the most common pathology associated with SCAD. In atherosclerotic arteries, plaques play a central role in the pathogenesis of dissection. Plaques may bleed or rupture, leading to dissection of the adventitia from the media and subsequent rupture of the media. They may also cause intimal tears that progress into the media.

Coronary angiography is essential in the diagnosis of SCAD. The typical appearance is the presence of a thin, longitudinal radiolucent line representing an intimal medial flap with flow in two or more separate lumens. Additional IVUS and optical coherence tomography

imaging may provide detailed information on the location and extent of the dissection. Typical IVUS features of SCAD are the presence of an intramural hematoma in the outer third of the media compressing the true lumen with minimal or no atherosclerosis [5,10]. There are two types of SCAD. One is an intimal tear and propagation of medial dissection that can be recognized angiographically, as shown in our cases. The other is medial dissection with hematoma formation, but no intimal tear. Computed tomography coronary angiography can be useful in the follow-up of conservatively managed patients.

Treatment of SCAD is often challenging and may include medical therapy, PCI, or coronary artery bypass graft surgery (CABG). Ongoing ischemia and the disease extent are important considerations when making a decision regarding the most appropriate treatment modality. Medical treatment is a reasonable option in patients with stable hemodynamics, single-vessel SCAD affecting a limited territory, or Thrombolysis In Myocardial Infarction (TIMI) of grade 2 or 3 flow. In patients with completed infarctions without residual ischemic symptoms, medical therapy is reportedly associated with a good long-term outcome. Coronary intervention can be considered as the first-line approach for SCAD complicated by occlusion or significantly slowed flow, especially in cases of proximal dissection or TIMI of grade 0 or 1 flow [3,4,6,10]. CABG may be an optimal treatment choice in multivessel disease or left main vessel disease. In our patients, PCI was performed because both had chest pain suggestive of ongoing ischemia and flow-limiting LAD lesions. During coronary intervention, the surgeon should be cautious about wiring the false lumen because vessel perforation or distal propagation of the dissection may occur; it is important to confirm wiring of the true lumen under intravascular imaging such as IVUS or optical coherence tomography [4,5]. In the present cases, we performed percutaneous coronary angioplasty and stent deployment without complications in patients presenting with myocardial infarction under IVUS. Recent developments in stenting and PCI techniques will make the role of PCI more important in treating SCAD.

In conclusion, SCAD should be considered in patients presenting with acute coronary syndrome with few conventional coronary risk factors. Prompt diag-

nosis and patient-tailored management may reduce morbidity and mortality in this population.

### Conflict of interest

No potential conflict of interest relevant to this article is reported.

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