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Spontaneous Left Main Coronary Artery Dissection Presenting with Acute Myocardial Infarction in an Elderly Male: A Clue from the Collateral Flow

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ABSTRACT

Spontaneous Coronary Artery Dissection (SCAD) is a rare cause of Acute Myocardial Infarction (AMI), which usually occurs in young or middle-aged healthy females. Here, we reported a 62-year-old male having SCAD in the Left Main Coronary Artery (LMCA) presenting with AMI. In this case, we could diagnose a spontaneous LMCA dissection with the help of contrast dye staining at the end of the collateral flow from the Right Coronary Artery (RCA). Even a small amount of contrast dye staining at the end of the collateral flow from the RCA provides an important clue for suspecting spontaneous LMCA dissection. Meticulous catheter manipulation should be required in such cases to prevent the extension of iatrogenic catheter-dissection during the procedure.

1. Introduction

Spontaneous Coronary Artery Dissection (SCAD) has been defined as a hemorrhagic separation of the coronary artery media with the formation of a false lumen without predisposing factors (1). It is a rare cause of Acute Myocardial Infarction (AMI) and usually occurs in young or middle-aged healthy females during pregnancy and postpartum without the traditional risk factors for coronary atherosclerosis (1). The present study aimed to report a case of SCAD of the Left Main Coronary Artery (LMCA) presenting with AMI in an elderly male patient. Interestingly, the spontaneous LMCA dissection could be diagnosed based on the collateral flow from the Right Coronary Artery (RCA).

2. Case Presentation

A 62-year-old man was brought to the emergency room with ongoing chest pain and cold sweating. He had no medical history and risk factors, except for heavy smoking. On admission, his blood pressure was 121/71 mmHg. The initial electrocardiogram showed a Q wave at V1-2, a 1-mm ST elevation at V2-4 and aVL, and a 1-mm horizontal ST depression at II, III, and aVF (Figure 1A). The initial chest X-ray showed mild pulmonary congestion. The initial creatinine kinase-muscle/brain level was 1.2 ng/mL and cardiac troponin I was slightly elevated to 0.08 ng/mL. The bedside transthoracic echocardiography revealed that the left ventricular systolic function was reduced and hypokinetic motions of anterior and anteroseptal walls were observed. AMI was diagnosed and an emergent Coronary Angiography (CAG) was performed. An initial right CAG revealed that the RCA had good flow without significant stenosis, but grade 1 collateral flow toward the Left Anterior Descending (LAD) artery was observed. Interestingly, a contrast dye staining was observed during the systolic phase at the end of the LAD collateral flow, which could be indicative of a spontaneous LMCA dissection (Figure 1B, Video 1). The first shot of the left CAG revealed that there was no significant obstructive lesion at the LMCA during that time, but there was a suspected dissection in the retrospective magnified view (Figure 1C). Unfortunately, the second shot of the left CAG revealed a marked spiral dissection at the LMCA (Figure 2A). After the contrast dye injection, his blood pressure decreased and the

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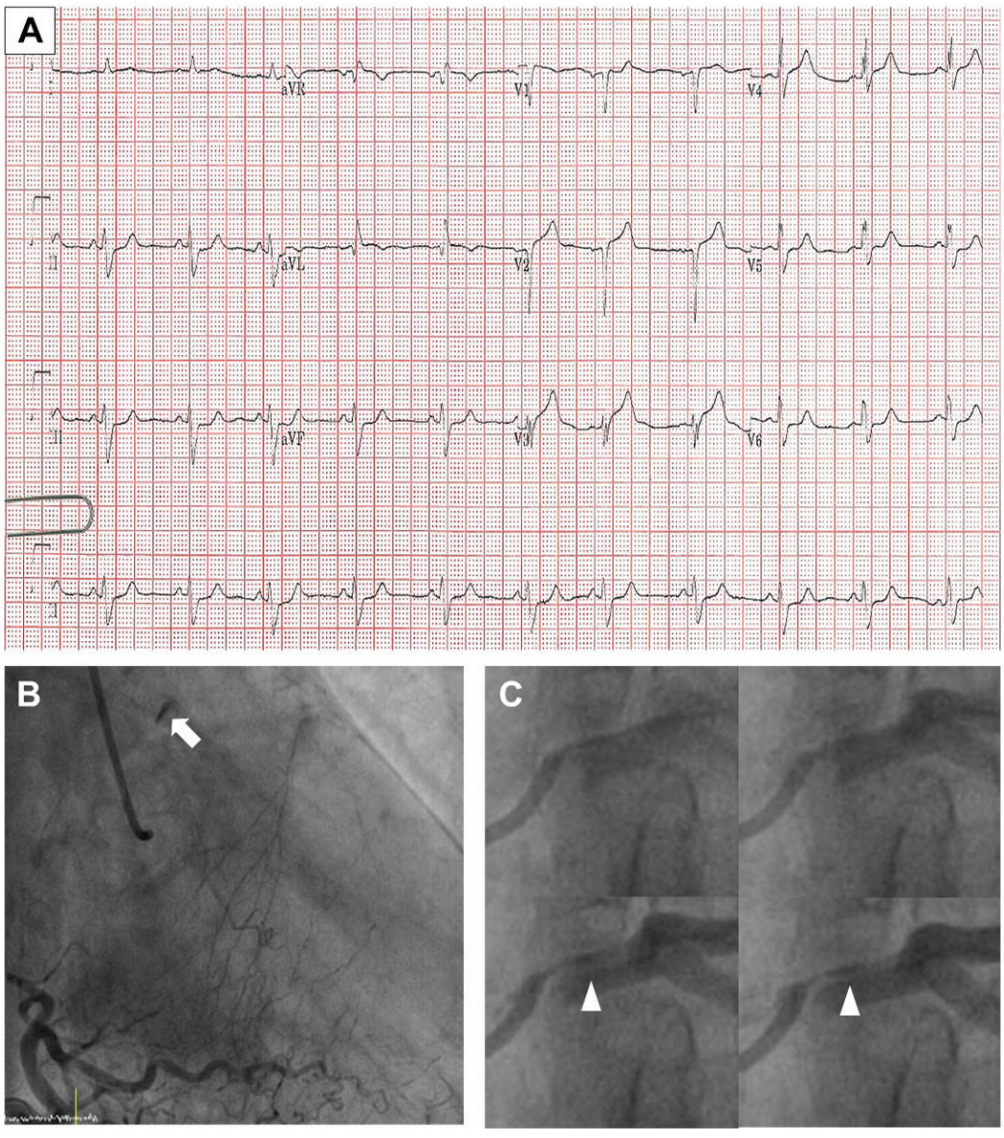

Figure 1. (A) Electrocardiography Showing ST Segment Elevation in Leads V2-4 and aVL, Q Wave in Leads V1-2, and ST Depression in Leads II, III, and aVF. (B) Left Coronary Collateral Flow from the Right Coronary Artery and Contrast Dye Staining at the End of the Collateral Flow (Arrow). (C) First Contrast Dye Injection for the Left Coronary Angiography, Which Was Suspicious of Dissection at the Left Main Coronary Artery (Arrow Head).

Video 1. Right Coronary Angiography Showing a Collateral Flow toward the Left Anterior Descending Artery and Contrast Dye Staining at the End of the Collateral Flow, Which Was Suspicious of Spontaneous Left Main Coronary Artery Dissection.
[To watch the videos, please check the html format.]

Thrombolysis In Myocardial Infarction (TIMI) flow of the left coronary artery decreased from grade 3 to 0. The Intra-Aortic Balloon Pump (IABP) was rapidly inserted and Percutaneous Coronary Intervention (PCI) was initiated. An initial Runthrough guidewire failed to pass into the true lumen. Therefore, a Fielder guidewire was used, which successfully crossed the lesion. An Intravascular Ultrasound (IVUS) revealed a dissection flap and a false lumen with an intramural hematoma at the LMCA and confirmed that the guidewire was located in the true lumen (Figure 2B, Video 2). A Xience Alpine stent (3.5 × 18 mm) was successfully implanted at the LMCA. After PCI, the TIMI flow in the left coronary artery was fully recovered and the stent was well-deployed (Figure 2C). The patient made an uneventful recovery and was discharged from the hospital.

3. Discussion

In the present case, spontaneous and iatrogenic catheter-induced LMCA dissection could not be distinguished completely. However, a spontaneous LMCA dissection was strongly suspected with the help of contrast dye staining at the end of the collateral flow from the RCA. Furthermore, a spontaneous LMCA dissection might be aggravated by a left diagnostic catheter engagement with the contrast dye injection.

SCAD is a rare, but often fatal cause of acute ischemic coronary events with a complex pathogenesis. It has been most commonly indicated in young or middle-aged females in the peripartum period or under oral contraceptive use,

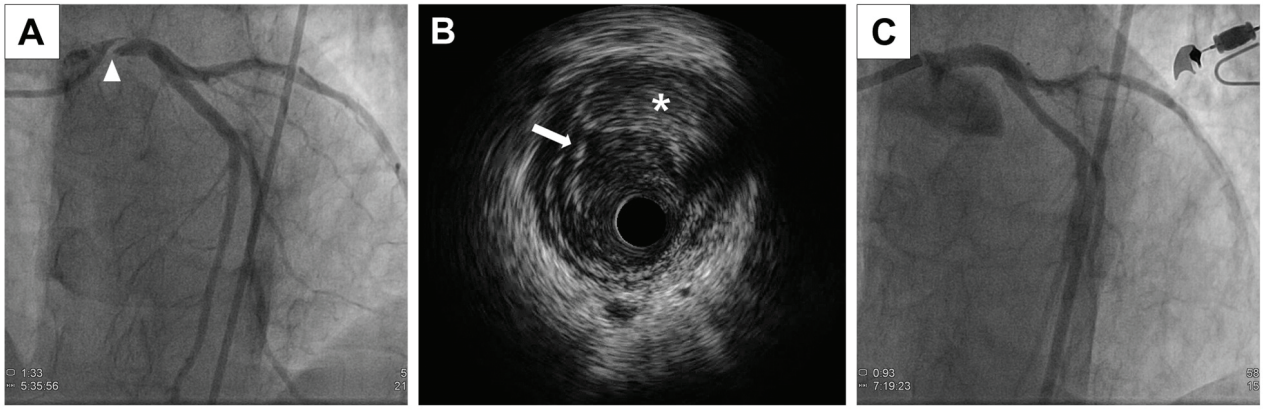

Figure 2. (A) Second Contrast Dye Injection for the Left Coronary Angiography and Spiral Dissection at the Left Main Coronary Artery (Arrow Head). (B) Intravascular Ultrasound Image Showing Dissection Flap (Arrow) and False Lumen with Intramural Hematoma (Asterisk). (C) Final Coronary Angiography after Stent Implantation at the Left Main Coronary Artery.

Video 2. Intravascular Ultrasound Showing a Soft Atherosclerotic Plaque at the Proximal Left Anterior Descending Artery and a Dissection Flap and False Lumen with Intramural Hematoma at the Left Main Coronary Artery.
[To watch the videos, please check the html format.]

without coronary atherosclerosis and its risk factors (1). It has also been associated with intense physical exercise and connective tissue disorders (1). However, the present case occurred in an older male unlike previously reported SCAD cases. Furthermore, the IVUS findings of the case showed a soft atherosclerotic plaque at the proximal LAD artery. Previous reports demonstrated that SCAD could be seen in older patients with risk factors for coronary atherosclerosis (2). In a series of 3750 consecutive CAG, nine patients had SCAD. The mean age of this group was 55.7 years and seven patients were male (2). All patients had atherosclerotic burden and their clinical courses were more benign compared to the non-atherosclerotic SCAD patients (2).

There are many challenges for CAG and PCI of SCAD, such as the risk of iatrogenic catheter-induced dissection and the difficulty of advancing the coronary wire into the distal true lumen and propagating an intramural hematoma (1). These arteries are structurally weakened by the underlying arterial pathology and are prone to iatrogenic dissections and aggravation of dissections during CAG and PCI. In a retrospective review of 348 CAG studies in SCAD patients, iatrogenic catheter-induced dissections occurred in 3.4% of the patients in addition to their presenting of SCAD arteries, with a much higher incidence (14.3%) in ad hoc PCI cases (3). The current case also showed that a spontaneous LMCA dissection might be extended by diagnostic catheter engagement with a contrast dye injection. In this case, a contrast dye staining at the end of the collateral flow from the RCA was an important clue for suspecting a spontaneous LMCA dissection. Therefore, meticulous catheter manipulation should be required in such cases to prevent the extension of iatrogenic catheter-dissection during the procedure.

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Authors' Contribution
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