

# Severe Coronary Spasm in Systemic Lupus Erythematosus Resulting in Recurrent Occlusions and Guide Wire Fracture

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Middle-aged female patients with systemic lupus erythematosus (SLE) have an increased risk of coronary artery disease and myocardial infarction (MI). We report a case of left anterior descending coronary artery (LAD) MI associated with severe coronary spasm in both the LAD and left circumflex artery, complicated with fracture of the distal wire within the microcatheter which was successfully removed by manual aspiration using an inflation device. From this series of rare complications of SLE with MI, severe coronary spasm and guide wire fracture, we underscore that clinicians performing coronary intervention should be aware of an elevated chance of possible severe coronary spasms in SLE patients.

**Key Words:** Coronary spasm • Myocardial infarction • Systemic lupus erythematosus

## INTRODUCTION

Patients with systemic lupus erythematosus (SLE) have a five-fold increased risk of coronary artery disease with a 50-fold increased risk of myocardial infarction (MI) in middle-aged female patients with SLE.<sup>1</sup> We report an interesting case of left anterior descending coronary artery (LAD) MI associated with suspected recurrent coronary spasm in both LAD and left circumflex artery (LCX).

## CASE REPORT

A 44-year-old female with a history of SLE and lupus nephritis was admitted to our hospital due to a 3-hour episode of angina. The patient's electrocardiogram showed ST elevation in V1-V3. Emergency transcatheter coronary angiography was performed with the suspicion of anterior wall ST elevation myocardial infarction. Angiography showed total occlusion of the LAD due to acute thrombus (Figure 1A); therefore, thrombus aspiration by manual thrombus aspirator (Thrombuster II, Kaneka, Japan) was immediately performed and two thrombi were aspirated. However, severe coronary spasm (Figures 1B and 1C) was noted, which was subsequently treated with intracoronary nitroprusside injection. A repeated angiography showed persistent stenotic lesions in the LAD, and for this reason two stents were placed in the LAD lesions (Figure 1D).

However, a sudden occlusion of the proximal LCX was found immediately after the stents were placed (Figures 1D). Initially, we believed that the proximal LCX occlusion was secondary to the thrombus migration. Due to the difficulty we experienced in progressing the thrombus aspirator, we first separately advanced two

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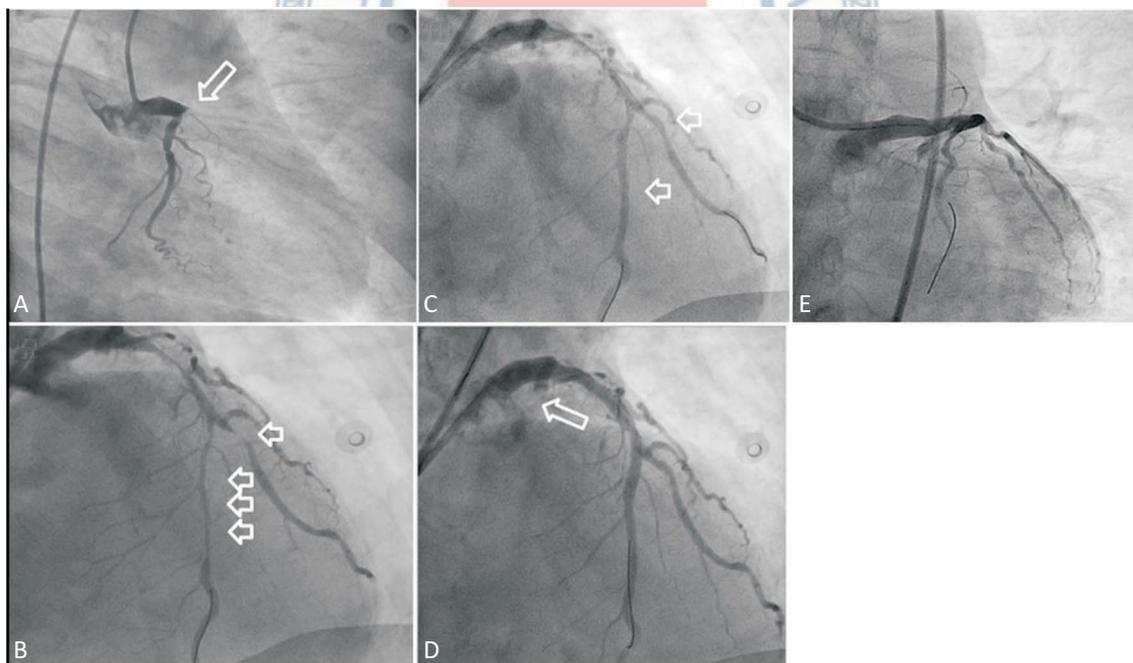
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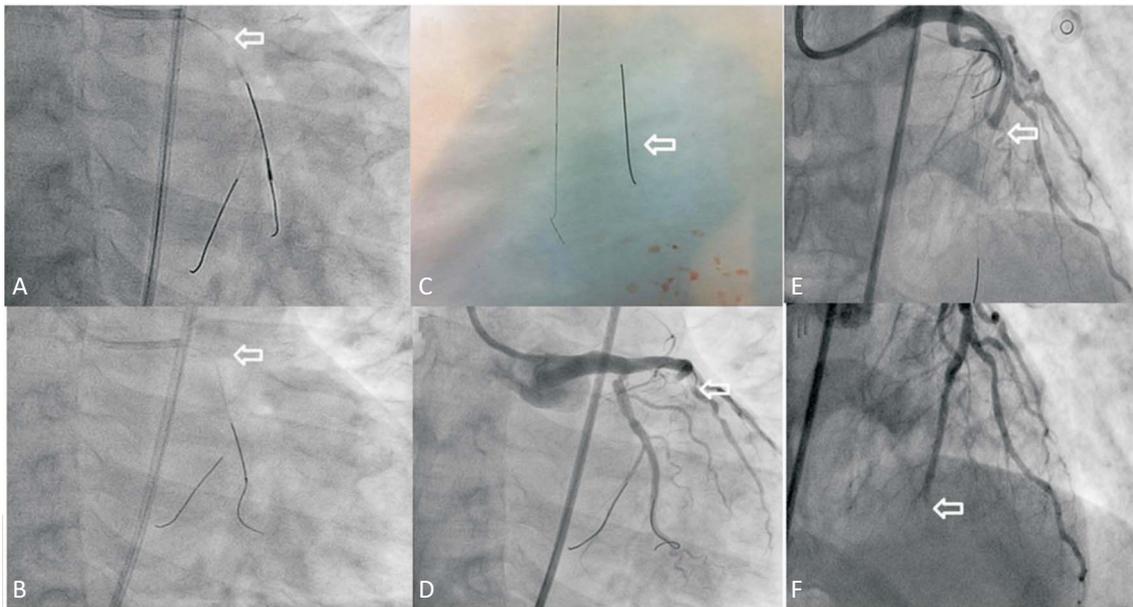
wires into the distal LCX and obtuse marginal (OM) branch of LCX. We also used intravascular ultrasound (IVUS, Volcano Therapeutics, Rancho Cordova, CA, USA) to further evaluate the cause of the proximal occlusion in the LCX (Figures 1E and 2A), but the IVUS revealed no thrombus in the LCX. Therefore, coronary spasm was suspected to be the cause of the total occlusion. We then advanced the microcatheter (Finecross, Terumo, Japan) to the LCX-OM in order to perform a selective injection of contrast medium.

However, when we attempted to pull back the LCX-OM wire (Grand Slam, Asahi Intecc, Japan) through the microcatheter, the distal portion of the wire fractured (Figure 2B). Since the distal wire fragment was still in the microcatheter, we connected the microcatheter to the inflation device, aspirated the wire fragment into the microcatheter and successfully removed the fractured wire using negative pressure (Figure 2C). Subsequent distal angiography through the microcatheter showed a patency of the distal vessel and confirmed that there was no distal thrombus embolization by thrombus migration.

Despite the injection with the vasodilator and calcium channel blocker, the total occlusion persisted. We then dilated the proximal LCX by balloon inflation. Although the flow of LCX was restored after balloon dilatation (Figure 2D), total occlusion of distal stent edge of LAD occurred after the inflation procedure (Figure 2E). Suspecting suspected coronary spasm, we repeated the same procedure in the LAD as in LCX, including thrombus aspiration (no thrombus was found), selective distal angiography (patent distal vessel without distal embolization), and distal vasodilator injection, but we failed to regain blood flow to the very distal segment of LAD despite repeated balloon dilations (Figure 2F). Because the symptoms were relieved, the patient was moved to the critical care unit under continuous medical treatment with intravenous calcium channel blocker and nitroprusside. Cardiac enzymes reached a peak value soon after the procedure. The patient thereafter remained asymptomatic and was discharged uneventfully one week later for regular clinical follow-up. Follow-up angiography three months later showed patency of both LAD and LCX.



**Figure 1.** Initial transcatheter coronary angiography of the patient with systemic lupus erythematosus showing total occlusion of the left anterior descending coronary artery (LAD) associated with severe coronary spasm of the LAD and left circumflex artery (LCX). (A) Right oblique caudal view of the left coronary artery shows total occlusion of the ostium of LAD. (B) After thrombus aspiration, angiography showed a diffuse spasm of distal and diagonal branch of LAD (arrows). (C) After injection of vasodilator, relief of the spasm was noted (arrow). (D) After LAD stenting, total occlusion of proximal LCX was noted (arrow). (E) Right oblique caudal view of LCX showed total occlusion of proximal LCX with a wire advanced into the distal LCX.



**Figure 2.** Fracture of the distal guide wire with removal and subsequent occlusion of distal LAD due to spasm and failure of balloon dilatation. (A) IVUS was pulled back from LCX-obtuse marginal junction. (B) Fracture of distal part of the guide wire in the LCX-obtuse marginal (OM). Only one body of wire could be recognized (arrow), compared with (A) (arrow). (C) Photography of distal fragment (arrow) and the rest body of wire after successful removal using negative pressure aspiration from inflation device. (D) Restoration of the flow of LCX because of sudden proximal LCX occlusion caused by coronary spasm, but distal stent edge occlusion of LAD was also noted (arrow). (E) Anterior cranial view of LAD showed distal stent edge total occlusion (arrow). (F) Despite repeated balloon dilations and vasodilator injections, there was still no-reflow in the very distal LAD (arrow). Abbreviations are in Figure 1.

## DISCUSSION

Our presented case provides two interesting findings. The first is the recurrent coronary spasm and the other is the treatment for the fractured wire. Initially, the LAD occlusion was caused by thrombus, but coronary spasm developed just after thrombus aspiration, suggesting a high risk of recurrence. After the stenting of the LAD, the LAD flow was good and there was no residual thrombus in the distal LAD. Thus, the recurrent distal occlusion of the LAD was caused by coronary spasm rather than thrombus. Furthermore, subsequent distal injection also confirmed patency of the distal LAD. Likewise for the LCX occlusion, the IVUS also showed no thrombus and the distal injection also confirmed the patency of distal vessel. Hence, coronary spasm was also the most likely cause of the LCX occlusion. In addition, wire fracture is quite rare during this kind of intervention, and it would be difficult to confirm whether the wire fracture was caused by equipment failure, severe spasm, or both.

Cardiac complications of the SLE involved the coro-

nary artery, pericardium, myocardium, endocardium, valvular apparatus, and the conducting system; these complications are the important causes of death in middle-aged female patients, similar to our reported case.<sup>1</sup> There are several possible mechanisms of early MI in the SLE, with the most common one being premature atherosclerosis,<sup>2</sup> but others have reported causes including coagulopathy, coronary arteritis, aneurysm, and rarely, and less frequently coronary spasm.<sup>3</sup> The recurrent severe coronary spasm in both LAD and LCX seen in our patient is quite rare. Despite the early MI event and recurrent severe coronary spasm, the outcome of these patients seems to be good, with no apparent complications.

There were at least two possible causes of wire fracture during our procedure. First, the wire fracture was caused by severe coronary spasm. Second, a manufacturing defect accompanied by repeated manipulation of the wire and a change of devices may also have been possible causes. Clinicians should be aware of the possibility of wire fracture during repeated manipulation of the wire and the changing of devices for critical lesions.

Despite the rarity of guide wire fracture during coronary intervention, a safe and effective method for removal of the fractured wire is important, as it may serve as a nidus for endothelial injury and platelet deposition, which can result in life threatening complications such as coronary thrombosis, dissection, emboli, and perforation. There are several methods recommended for the percutaneous management of fractured guide wires, including loop snare removal, multiple wires rotation, and stenting over the retained wire.<sup>4</sup> In our case, because the partially fractured remnant was within the microcatheter, we could simply aspirate the fractured wire via the microcatheter by applying negative pressure from the inflation device. This easy and effective method should be recommended in the future, if the fractured wire is still at least partially within the microcatheter.

## CONCLUSIONS

Clinicians performing coronary intervention should be aware of the probability of a higher chance of severe coronary spasms in SLE patients, which could increase the risk of guide wire fracture. However, if the wire is

still partially within the microcatheter lumen, there may be an opportunity to remove the fractured wire using inflation device aspiration.

## CONFLICT OF INTEREST OR EXTERNAL SOURCE OF FUNDING

None.

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