direct angioplasty is the most common strategy for treatment of acute coronary syndromes, but fails to secure TIMI-3 flow in 12–30% of cases mainly because of the occurrence of the 'no-reflow'. Acute coronary syndromes are believed to be associated with secondary intracoronary thrombus formation, and it is this thrombus burden that may cause distal thrombo-embolism when performing interventional therapy. A case of no-reflow following balloon dilatation occurred in the absence of thrombus burden, which had been removed in advance by intravascular ultrasound (IVUS)-guided thrombectomy.

**Case Report**

A 62-year-old man with a past history of anteroseptal myocardial infarction (MI) and who was being treated with anti-platelets, long acting nitrate, nicorandil and calcium antagonists was hospitalized with continuous chest oppression of 5 h duration. Physical examination on admission showed Killip-I status, normal blood pressure (127/80 mmHg) and regular pulse rate (68 beats/min). Electrocardiogram showed sinus regular rhythm, but ST elevation in leads III and aVF and ST depression in leads I, aVL and V2-6, and thus he was diagnosed as having an acute inferior MI. Emergency cardiac catheterization revealed total occlusion (TIMI-1) of the distal segment of the right coronary artery (RCA). Based on the angiography findings, we attempted thrombectomy with the Rescue™ thrombectomy catheter (Boston Scientific Corp) because we considered thrombi were the cause of the obstructive lesion. We also inserted a temporary pacing catheter before angioplasty. The Rescue™ catheter is made of several components. The 4.5 Fr shaft has a semi-monorail lumen, which is compatible with the usual 0.014 inch percutaneous transluminal coronary angioplasty (PTCA) guidewire. A console box is attached to the aspiration catheter and the maximum aspiration force is 0.8 atmosphere. The collection bottle is

**Key Words:** Atheromatous plaque; Lipid core; No-reflow; Percutaneous transluminal coronary angioplasty (PTCA)

Radiolucent findings of coronary angiogram are believed to usually represent intracoronary thrombus, but in the present case, were atheromatous plaque with a large lipid core. A 62-year-old man who suffered from an inferior acute myocardial infarction was admitted to hospital 6 h after onset. The initial cine angiograms showed TIMI-1 flow in the distal portion of the right coronary artery, so thrombectomy was initially carried out and TIMI-2 flow achieved. However, the radiolucent lesion did not disappear and so adjunctive mechanical dilatation of the lesion was performed, which resulted in ‘no-reflow’ (TIMI-0). Finally, aspiration of the material from the stagnated lesion was attempted and immediately obtained TIMI-3 flow. The retrieved materials were macrophages (foam cells) and many cholesterol crystals, both of which are considered to be atheromatous gruel. Therefore, the sudden flow reduction following percutaneous transluminal coronary angioplasty was caused by mechanical disruption of an atheromatous plaque with a large lipid core. 

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Fig 1. Left anterior oblique views of right coronary artery (RCA).
(A) Contrast dye showing the totally occluded distal segment of the RCA. (B) Reperfusion succeeded and intra-coronary filling defects appeared at the previously occluded and distal sites of the RCA after thrombectomy (arrows). (C) Angiographical no-reflow occurred following balloon angioplasty. (D) Coronary filling delay improved immediately after aspiration from the no-flow coronary region.
equipped with a nylon mesh filter (400μm interval), and its capacity is 150 ml. Red thrombus clots were aspirated, which improved coronary flow to TIMI-2, but severe stenosis with a large filling defect persisted (Fig1). IVUS imaging of this mass revealed that part of the lesion was echo-lucent, suggesting a large lipid core (Fig 2), so balloon angioplasty was performed at the site; however, the coronary angiogram showed no-reflow of the coronary vasculature. Repeated thrombectomy using the Rescue™ catheter improved the filling delay. The aspirated sample from the site of no-reflow in the coronary artery contained cholesterol crystals and macrophages (Fig 3A, B). During the no-reflow, the electrocardiogram showed pacing rhythm only, but the patient's sinus rhythm re-appeared when coronary flow was improved by the second aspiration.

**Discussion**

The principal finding of this case is that the cause of the no-reflow immediately after balloon angioplasty in the patient with acute coronary syndromes is not thrombus burden, but the plaque components of atheromatous lesions. The incidence of no-reflow after percutaneous coronary intervention for acute MI has been reported to be 11.5–30%, but as those reports were defined by the final angiogram, they did not include transient flow reduction during intervention. Therefore, the incidence of flow reduction during intervention may be greater.

The mechanism of the classic no-reflow phenomenon in the animal laboratory model has been described as increased microvascular impedance to flow (neutrophil plugging of capillaries, myocyte contracture and edema, and endothelial blistering), but it is not clear whether these findings are associated with the no-reflow phenomenon observed in humans after mechanical intervention to restore epicardial patency. Acute coronary syndromes usually occur at atherosclerotic lesions in humans, so we consider that the pathophysiological mechanisms of no-reflow differ between animal and human subjects. The cause of no-flow following angioplasty is not distal embolism with thrombus clots, but plugging of coronary beds with plaque gruel (ie, foam cells, tissue factors, cholesterol crystals). The most effective therapy for no-reflow is unknown.

Sakata et al reported that treatment with nicorandil was effective after successful coronary revascularization, and Piana et al used verapamil for no-reflow and reported good results. Wainstein et al reported that verapamil can improve TIMI flow, but does not have clinical efficacy. In the present case, oral administration of nicorandil did not prevent no-reflow and these findings suggest that the mechanism of no-reflow associated with reperfusion therapy is complex. We consider that direct aspiration using the Rescue™ catheter may be a new strategy for improving the stagnated flow caused by no-reflow. Both the microscopic and IVUS findings of the no-reflow observed in this case suggest that it was caused by intracoronary debris containing lipid-rich atheroma, which is direct evidence of one of the mechanisms of coronary no-reflow during reperfusion.

The clinical implication of this case is that it is necessary to assess the lesion vulnerability before intervention and determine the reperfusion strategy carefully.

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