Stent thrombosis is rare with anti-platelet therapy, which consists of aspirin and ticlopidine as a post-stenting administration. A 77-year-old man had repeated stent thrombosis, which was not predicted by coronary angiography, despite using contemporary periprocedural anti-platelet therapy. Only intravascular fiberscopy was able to detect the cause of the stent thrombosis. (Jpn Circ J 2001; 65: 232–235)

Key Words: Coronary angioscopy; Stent; Subacute thrombosis

Case Report

A 77-year-old man with class 3 angina since May 1999 was referred in June 1999 for coronary evaluation. Smoking was the only relevant clinical history. His electrocardiogram showed T wave inversion from the V2 to V6 leads with poor R wave progression. The antero-septal left ventricular wall motion was reduced on echocardiography, but without left ventricular chamber dilatation or evidence of significant valvular disease. Coronary angiography (CAG) 2 days after beginning isosorbide dinitrate 40 mg/day, calcium antagonist (diltiazem 90 mg/day), aspirin (162 mg/day) and ticlopidine (200 mg/day) demonstrated total occlusion of the proximal left anterior descending (LAD) coronary artery with retrograde filling via collateral vessels from the right coronary artery. Because of persistent angina despite medical therapy, surgical intervention for the LAD coronary artery was deemed necessary, but balloon angioplasty had a suboptimal result because of a long intimal dissection. A NIR stent (length: 25 mm; diameter: 3 mm) was implanted at 12 atm to correct the intimal dissection with an optimal result on CAG (Fig 1).

However, routine angiography 24 h later showed that the stent had silently occluded at the mid portion. Tissue plasminogen activator (6,400,000 units) and heparin (6,000 units/day) were given intravenously after repeat intervention with higher inflation pressure because of haziness in the stent on the post-procedural CAG and to avoid thrombogenic occlusion of the stent. The next day the LAD was patent (Fig 2) and the patient was discharged 2 days later. Significant creatine kinase (CK) elevation was not detected throughout.

Eight days after discharge he had a sudden severe chest pain that prompted emergency re-evaluation: ST elevation in leads V2–6 was seen on the ECG, with severe hypokinesis of the anteroseptal wall. CAG showed re-occlusion of the LAD artery at the same site (mid portion of the stent); repeat percutaneous transluminal coronary angioplasty (PTCA) appeared to give an optimal result (Fig 3).

After an uneventful 11 days of observation, CAG, IVUS and intravascular fiber-angioscopy (IVFS) were performed to evaluate the reasons of the repeated subacute stent thrombosis. CAG showed the LAD as widely patent and on IVUS the stent also appeared to be well-dilated (about 3 mm diameter) without evidence of thrombus or dissection flaps (Fig 4). However, on IVFS the stent did not appear to be adequately dilated; there was a partial protrusion of the stent-struts into the lumen, and a compressed red and white thrombus between the intima and the stent-strut, but not protruding into the lumen, was also seen (Fig 5).

Cylostazol (200 mg/day) was added and the patient was discharged without evidence of re-occlusion, although the maximum CK reached 6,830 IU/L.

Discussion

Coronary stent implantation has become the major method of myocardial revascularization and its indications have been expanded even to patients with small vessels, diffuse lesions, restenotic lesions, total occlusions and saphenous vein graft stenoses. Because the stent itself is thrombogenic, the presence of pre-existing thrombus and ruptured or eroded coronary plaques, which frequently occur in patients with acute myocardial infarction (AMI), initially excluded the use of this technique for AMI because of the justifiable concerns of a higher risk of thrombotic occlusion. However, with improvements in the stent deployment technique and the availability of more effective antithrombotic therapy, several trials of stenting for AMI have reported its feasibility and efficacy. The new antithrombotic therapy of combining aspirin and ticlopidine was reported by Colombo et al to reduce stent thrombosis event rates as low as 0.6% (acute stent thrombosis) and 0.3% (subacute stent thrombosis).
Coronary Angioscopy in Stent Thrombosis

Even lower rates of thrombosis, bleeding or vascular complication with the same regimen have been reported by other investigators.\textsuperscript{12,13}

IVUS studies have shown that more than 80% of stents are insufficiently dilated despite an apparently successful deployment angiographically.\textsuperscript{14,15} Colombo et al hypothesized that stent thrombosis was likely to be caused more by incomplete deployment of the stent and impaired rheologic conditions in the stented artery than by the thrombogenic properties of the stent.\textsuperscript{11} Multiple stents, bail-out implantation, small vessel size (<3.0 mm), the presence of unstable angina or AMI, an impaired left ventricular ejection fraction,
the existence of intimal dissection or intramural thrombus and inadequately dilated stent have all been reported as predictors of subacute occlusion. In addition, initial total occlusion with retrograde filling via collateral vessels, such as in the present case, is also thought to be a high risk for subacute occlusion.

In the present case, stent thrombosis occurred twice despite acceptable angiographic results and adequate preventive therapy. Further therapeutic strategies, such as anti-GP2b/3a, were not economically available at that time. Angiography could not predict the stent thrombosis, but angiography identified the defect in the geometric configuration: the stent not fully covering the damaged intima and the presence of an intramural thrombus. However, the type of stent might be implicated in the protruding stent-struts because it has been reported that the NIR stent is less flexible than other contemporary flexible stents in the expanded state. The protruding stent-struts, which could not be detected by angiography or IVUS, might have promoted platelet adhesion and stent thrombosis. In addition, other pathophysiological factors in the acute coronary syndrome might have been involved in the repeated stent thrombosis.

In the present case, angiography was not only more efficient in detecting intracoronary thrombus than angiography and IVUS but also allowed detailed observation of the degree of dilatation of the implanted coronary stent. Only intracoronary angiography could detect the cause of the repeat subacute thrombosis after stenting that resulted in AMI.

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**References**