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Case Reports

Plaque Rupture as a Cause of Apparent Coronary Aneurysm Formation Following Directional Coronary Atherectomy

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Late coronary aneurysm formation was observed following treatment by directional coronary atherectomy. Intravascular ultrasound disclosed that the mechanism involved was plaque rupture. The cause of coronary aneurysm may be multifactorial after coronary interventions. Intravascular ultrasound imaging is useful for understanding the pathologic mechanism of coronary aneurysm production. *Cathet. Cardiovasc. Diagn.* 41:48–50, 1997. © 1997 Wiley-Liss, Inc.

Key words: intravascular ultrasound; directional coronary atherectomy; contrast echocardiography; coronary aneurysm; Albunex

INTRODUCTION

A coronary aneurysm has been defined as an area of localized coronary dilatation whose diameter is one-half more than the diameter of the adjacent normal segment [1]. However, it is impossible to distinguish angiographically a true aneurysm from a pseudoaneurysm. Recently, intravascular ultrasound (IVUS) has been reported to be useful for determining the mechanism of aneurysm formation [2,3]. We report a case of coronary aneurysm following directional coronary atherectomy (DCA), which was subsequently found by IVUS to involve plaque rupture instead of medial disruption.

CASE REPORT

A 53-year-old male patient presented with an anterior wall myocardial infarction in February 1995. Cardiac catheterization performed in April demonstrated an occlusion of the proximal portion of the left anterior descending coronary artery (LAD) and a 90% stenosis in the left circumflex coronary artery (LCX). The right coronary artery (RCA) exhibited mild irregularities and showed good collateral flow to the distal segment of the LAD. The left ventricular ejection fraction was 53%. IVUS imaging using a 30 MHz transducer catheter (3.2 Fr., Cardiovascular Imaging Systems, Sunnyvale, CA) was performed in the LAD after initial dilatation with a 2.0

mm conventional balloon catheter. The IVUS demonstrated a marked eccentric plaque with deep calcification. Quantitative measurements of the lesion revealed a lumen cross-sectional area (L-CSA) of 1.1 mm², total arterial area (TAA) of 29.1 mm², and the plaque area (PA) of 28.0 mm². The TAA of the lesion was larger than that of the left main artery (28.2 mm²). This observation indicated the presence of compensatory enlargement of the coronary artery at the proximal portion of the LAD (Fig. 1). Based on these angiographic and IVUS findings, we elected to perform DCA with a 7 Fr. GTO Simpson coronary atherocath (Devices for Vascular Intervention, Santa Clara, CA) and achieved excellent angiographic results. IVUS still showed a large residual plaque but an acceptable lumen size (L-CSA, 6.5 mm²; TAA, 31.7 mm²; and PA, 25.2 mm²). The residual plaque echo composition was homogeneous and there was no evi-

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Fig. 1. Left coronary arteriogram and corresponding intravascular ultrasound images obtained before directional coronary atherectomy: (a) left main coronary artery, (b) stenosis site, (c) distal site. Marked eccentric fibrous plaque was evident at the proximal portion of the left anterior descending coronary artery. The imaging catheter was surrounded by hypodense echoes suggesting thrombus formation. Concentric plaque was present immediately distal to the stenosis. The left main artery was almost normal.

Fig. 2. Left coronary arteriogram and corresponding intravascular ultrasound images obtained immediately after directional coronary atherectomy: (a) left main coronary artery, (b) dilated site, (c) distal site. The lesion was successfully enlarged on the arteriogram. However, there was significant residual plaque on IVUS. No evidence of dissection or deep arterial damage was recognized.

dence of dissection at the lesion on ultrasound (Fig. 2). The resected pathologic specimen displayed fibrous plaque and organized thrombus formation, but there was no evidence of media resection or necrotic tissue.

The patient experienced no further ischemic symptoms, but a repeat cardiac catheterization was performed in November 1995, because of an abnormal thallium test. Coronary angiography revealed that the LAD had developed restenosis with a 90% diameter narrowing and an interposed cauliflower-like saccular aneurysm at the site

Fig. 3. Left coronary arteriogram and corresponding intravascular ultrasound images obtained 7 months after directional coronary atherectomy: (a) left main coronary artery, (b) restenosis site, (c) aneurysm site, (d) distal site. Restenosis and a large saccular aneurysm were noted at the prior DCA site on the arteriogram. IVUS demonstrated significant plaque accumulation at the restenosis site (b). There was a less echogenic area within the plaque at the aneurysm site (c, arrow)

Albunex(+)

Fig. 4. Intravascular ultrasound images before and after intracoronary Albunex injection. The extent of evacuated plaque was clearly documented after intracoronary injection of Albunex (right). The opacified area was limited within the plaque. Deep calcification was noted at the base of the plaque (arrow).

of the prior DCA. The dimension of aneurysmal area were 5.2 mm on quantitative coronary angiographic measurement (QCA). IVUS using a 30 MHz transducer catheter (3.5 Fr., Boston Scientific, Watertown, MA) demonstrated significant plaque re-accumulation at the site of restenosis (Fig. 3b). In addition, there was a less echogenic area within the large plaque (Fig. 3c). It was difficult to recognize this area by standard ultrasound imaging, because it was filled with speckled reflections from blood due to slow coronary flow. The lumen outline was enhanced after intracoronary injection of Albunex solution diluted 50% with human albumin (Molecular Biosystems, San Diego, CA) (Fig. 4). Quantitative measurements at the aneurysm site revealed a marked in-

crease in L-CSA and a decrease in PA with no appreciable change in TAA (L-CSA, 13.4 mm²; PA, 16.5 mm²; and TAA, 29.9 mm²). The diameters of the aneurysmal site were 4.9 mm × 4.5 mm and nearly equal to the measurements from QCA. From these observations, we inferred that the coronary aneurysm seen in our patient was caused by plaque evacuation of the inner core. The restenotic segment was treated again with DCA. The stenosis was reduced to 20%, although the aneurysm appearance remained unchanged.

DISCUSSION

The reported incidence of PTCA-induced coronary aneurysm is ~ 4% [4]. Deep arterial injury is considered as the main cause of aneurysm formation after coronary interventions. Injury or resection of media could lead to gradual dilatation and thinning of the vessel wall, with the reduced wall thickness and increased stress ultimately resulting in aneurysm formation. Especially after DCA, deep arterial components such as media and adventitia are frequently seen in resected specimens [5] and coronary aneurysms occur relatively frequently [6,7]. However, it was impossible to establish the precise mechanism of aneurysm formation in vivo until recently, when IVUS became available [2,3]. Employing IVUS, Gerrand et al. [2] reported a case of pseudoaneurysm of the coronary artery, which was initially interpreted as a true aneurysm by angiography. In addition, Ge et al. [3] studied coronary aneurysms with IVUS and noted that plaque rupture might be one of the possible causes of this morphology.

Our case is unusual in that serial IVUS studies clearly showed the mechanism underlying the angiographic appearance of the aneurysm. The IVUS images indicate that the aneurysm was not due to new dilatation of the vessel wall, but was created by evacuation of the core of the large original plaque. The diameter of the aneurysm on angiography corresponded to compensatory arterial dilatation that had slowly evolved with the original plaque formation. The evacuation of the central core of the plaque could have developed gradually or was due to spontaneous rupture of the residual plaque during the follow-up period. Although it was initially assumed based on angiography that aneurysm was caused by deep artery injury during DCA, this was refuted by IVUS and the histologic findings of the resected specimen. Plaque rupture is usually seen at the culprit lesion in patients with acute coronary syndromes [8]; however, it has never been reported as a cause of late coronary aneurysm formation

after coronary intervention. The reason why there were no clinical symptoms associated with the plaque rupture might be explained by the presence of an old anterior myocardial infarction and collaterals from the RCA, or perhaps no occlusive thrombus was formed.

Sonicated albumin microspheres (Albunex) proved helpful for detection of the ruptured area in our patient. This material was originally developed as a myocardial contrast agent for intravenous use, and the safety and efficacy of intracoronary use have been described [9]. Adjunct utilization of contrast material may be beneficial for improving the lumen-intima boundary during IVUS examinations. In conclusion, our experience indicates that the mechanisms of aneurysm formation on angiography may be multifactorial even after coronary interventions. IVUS imaging can be useful for elucidating the underlying pathologic mechanisms of angiographic findings.

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