Plaque morphological changes after drug-coated balloon angioplasty according to underlying plaque components

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ABSTRACT

A 52-year-old man with a short chronic total occlusion in the left superficial femoral artery underwent drug-coated balloon (DCB) angioplasty. Evaluation using integrated backscatter intravascular ultrasound revealed that the plaque volume of fibrosis was compressed just after treatment (from 494.67 mm^3 to 398.36 mm^3) and was further decreased at 1 month after treatment (to 362.07 mm^3). The plaque volume of the lipid pool was not changed at follow-up compared with that just after DCB dilation. These integrated backscatter intravascular ultrasound findings suggest that the effect of DCB angioplasty may differ depending on the type of underlying plaque components. (J Vasc Surg Cases Innov Tech 2025;11:101651.)

Keywords: Fibrous plaque; Integrated backscatter; Intravascular ultrasound; Femoral artery; Peripheral artery; Intraplaque

The efficacy of drug-coated balloon (DCB) angioplasty has been recognized widely in revascularization for femoropopliteal arteries.[1,2] Late lumen enlargement after DCB angioplasty has been reported[3,4]; however, the details of plaque morphological changes have not been elucidated.

The integrated backscatter (IB) method is an intravascular ultrasound (IVUS)-based color mapping method by which tissue characterization and quantitative analysis can be performed automatically in vivo.[5] In this color mapping, plaque properties are classified into four categories, calcification (red), dense fibrosis (yellow), fibrosis (green), and lipid pool (blue and purple), according to the preset threshold IB values. A previous study showed that the fibrosis can be compressed during endovascular treatment[6]; however, there is no information about whether the long-term efficacy of DCB angioplasty can be affected by the underlying plaque components.

We herein describe a case in which late acquired plaque morphological changes were detected by using the IB method after DCB angioplasty for a femoropopliteal arterial lesion. The patient provided written informed consent for the report of his case details and imaging studies.

CASE REPORT

A 52-year-old man with intermittent claudication was admitted to our hospital. The patient was undergoing lipid-lowering therapy for dyslipidemia and was a current smoker. The superficial femoral artery was occluded for a length of approximately 5 cm. Administration of cilostazol did not improve his symptoms, and endovascular therapy was performed. An ipsilateral approach was established, and a guidewire could be passed antegradely through the intraplaque route. Plain old balloon angioplasty was performed using a 5-mm semicomplaint balloon catheter (SHIDEN HP; KANEKA, Tokyo, Japan), and DCB angioplasty was performed using a 5-mm Ranger (Boston Scientific, Maple Grove, MN) at nominal pressure. The final angiogram showed linear dissection without any flow limitation. Evaluation using high-definition IVUS (Altaview; Terumo, Tokyo, Japan) was performed just after the guidewire passage and after the DCB dilation. One month after treatment, follow-up angiography and IVUS were performed simultaneously during coronary intervention with the patient's consent. All of the IVUS images obtained were analyzed using the IB method. Collateral channels were located just proximal and just distal to the occluded site, and quantitative and qualitative analyses were performed between the two collateral channels (Fig 1).

Fig 2 shows a representative cross section analyzed at three different time points, including after passage of the guidewire and after DCB dilation and at 1 month after the treatment. After tracing the external elastic membrane, internal elastic membrane, and luminal surface at 1-mm intervals, the plaque area (the remainder after subtracting the lumen area from the internal elastic membrane area) was analyzed by the IB method. After analysis, longitudinal accumulation was performed, and vessel and plaque volumes were calculated.[7,8] The results of quantitative and qualitative analyses using the IB method are

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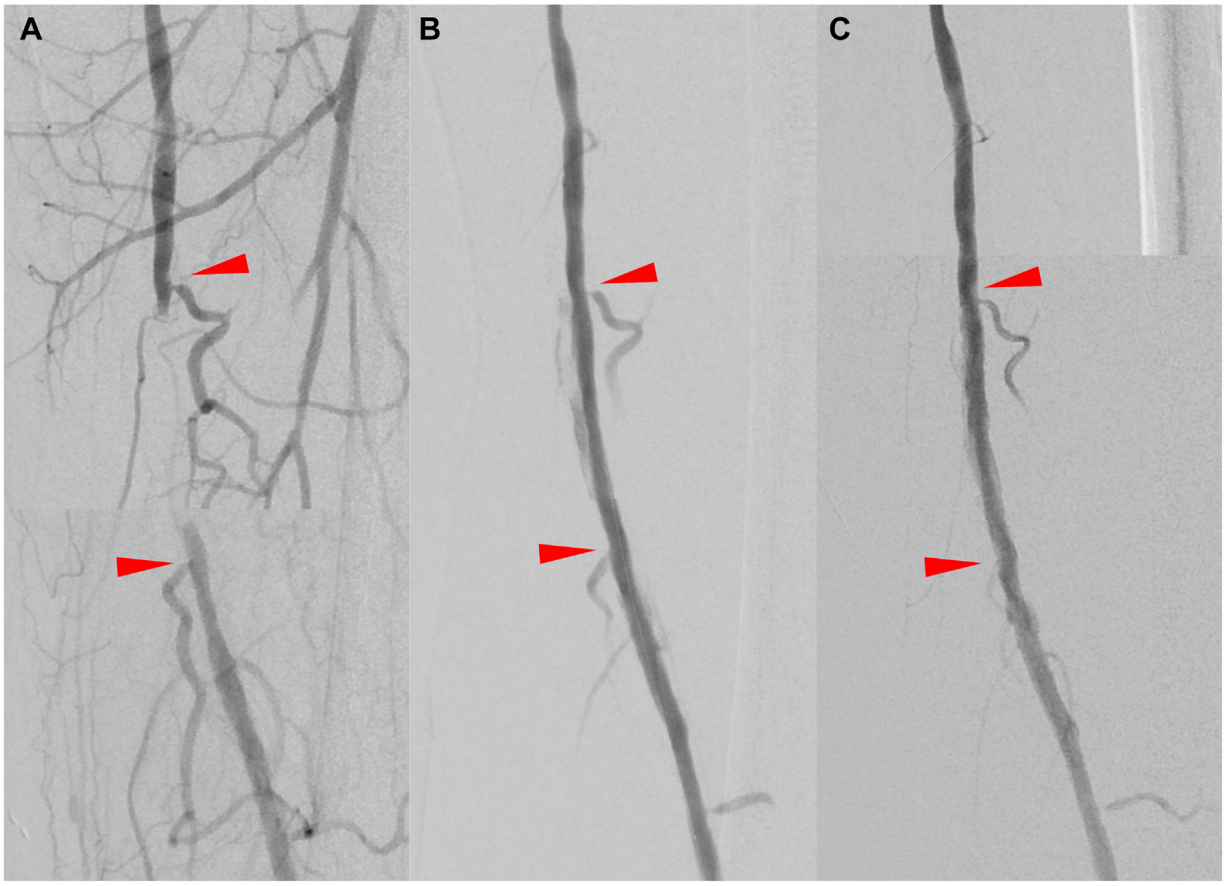


Fig 1. Angiograms **(A)** before the procedure, **(B)** after the procedure, and **(C)** 1 month after the procedure. Short chronic total occlusion was observed at the distal part of the superficial femoral artery. Collateral channels were observed just proximal and just distal of the occluded site (*red arrows*).

shown in Fig 3. At 1 month after treatment, the vessel volume and lumen volume were further enlarged compared with those just after DCB dilation. Among the plaque components, the plaque volume of fibrosis had been compressed by the procedure (from 494.67 mm^3 to 398.36 mm^3) and was further decreased at follow-up (to 362.07 mm^3), although the plaque volume of the lipid pool was not changed at follow-up compared with that just after DCB dilation (from 460.03 mm^3 to 456.37 mm^3).

DISCUSSION

We present a case in which follow-up IB-IVUS enabled quantitative and qualitative analyses of morphological changes after DCB angioplasty for a superficial femoral arterial lesion. In this case, the vessel and lumen volumes were further enlarged at 1 month after DCB angioplasty, and the fibrosis volume was further decreased after the treatment, unlike the lipid pool volume. To the best of our knowledge, this report is the first on the analysis of plaque morphological changes after DCB angioplasty. The results of analyses in this case suggest that the effect of DCB angioplasty may differ depending on the type of underlying plaque components.

Plaque volume reduction and delayed stenosis regression after DCB angioplasty in a peripheral artery were previously reported,[3,4] but there is still no information about the impact of underlying plaque components. In animal experimental models, the efficacy of DCB was evaluated by assessing the changes that occurred in a media of healthy models,[9,10] and histological analysis of plaque morphological changes caused by DCB angioplasty has not been performed. Endothelial cell or smooth muscle cell loss can occur after DCB angioplasty, and the loss may occur within the plaque components; however, the mechanism of plaque volume reduction arising from intraluminal paclitaxel application through balloon dilation remains unclear, and further histological or imaging investigation is needed.

As in the lipid pool, the plaque volume seemed to be increased just after DCB dilation compared with that just after guidewire passage. This finding may be due to the increased distance from the IVUS transducer to the plaque by enlarging the vessel by balloon

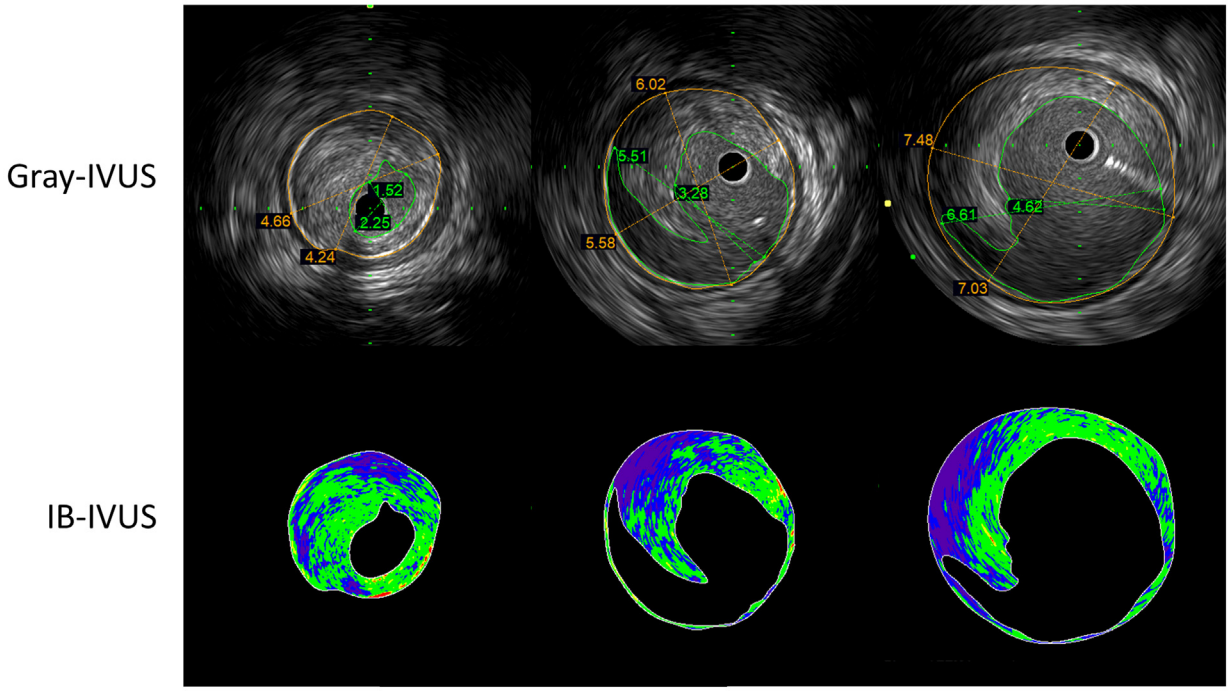
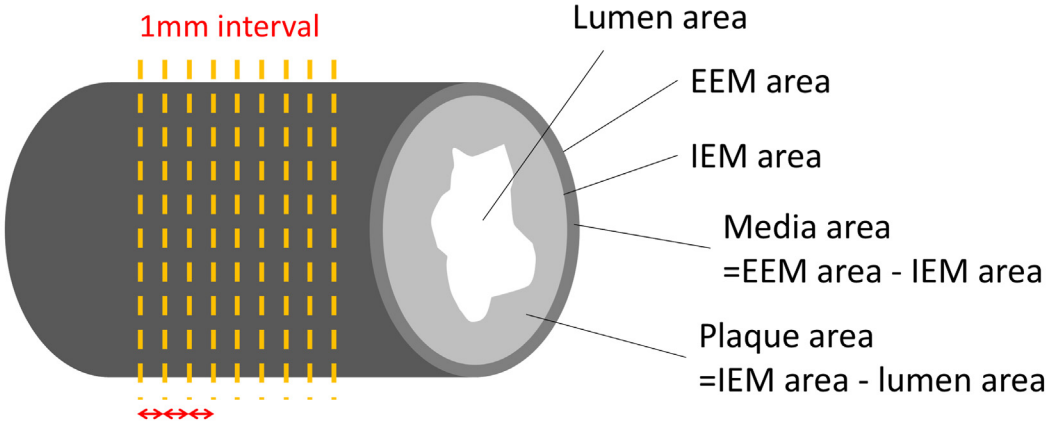


Fig 2. Representative gray and IB-IVUS images of the same cross section at each timing. *IB*, Integrated backscatter; *IVUS*, intravascular ultrasound.



	Pre	final	1M later
EEM volume, mm^3	1510.59	1969.88	2308.18
IEM volume, mm^3	1141.02	1657.49	1896.75
Media volume, mm^3	369.57	312.39	411.43
Lumen volume, mm^3	224.02	783.85	1063.51
Plaque volume, mm^3	917	873.67	833.29
- Calcification, mm^3	2.52	2.31	1.89
- Dense fibrosis, mm^3	14.98	12.98	12.94
- Fibrosis, mm^3	494.67	398.36	362.07
- Lipid pool, mm^3	404.78	460.03	456.37

Fig 3. Quantitative and qualitative volume analyses by using IB-IVUS at each timing. The EEM, IEM, and luminal surface were traced in all of the cross sections at 1-mm intervals and volumes were calculated by performing longitudinal accumulation. *EEM*, External elastic membrane; *IB-IVUS*, integrated backscatter intravascular ultrasound; *IEM*, internal elastic membrane.

angioplasty that resulted in stronger attenuation and/or that the possibility of the unclear area behind the dissection after balloon angioplasty being included as part of the lipid pool.

A previous study revealed that plaque area compression was likely to be achieved in fibrosis by balloon angioplasty.[6] Given that DCB is more likely to be effective when a larger MLA is achieved,[11] fibrosis may be a plaque component that is more likely to benefit from DCB therapy. The imaging analysis in this case showed that fibrosis was decreased further and the lipid pool was not changed after DCB angioplasty, indicating that fibrosis may also be a plaque component that is more likely to achieve the additional lumen enlargement effect that occurs in the chronic phase after DCB angioplasty. It may be better to deploy a stent if the lesion containing the lipid pool has residual stenosis. Further clinical investigation focusing on the patency after DCB angioplasty according to the underlying plaque components is needed.

The limitations of this report are that this is a case report on one patient and that the possibility of a measurement error cannot be ruled out. Further investigation in a larger number of patients is needed to determine the exact behavior after DCB angioplasty according to each underlying plaque component.

CONCLUSIONS

We herein described a case in which the plaque volume of fibrosis was regressed after DCB angioplasty in a femoropopliteal arterial lesion at 1 month after the treatment unlike that of the lipid pool according to IB-IVUS analysis. These IB-IVUS findings suggest that the effect of DCB angioplasty may differ depending on the type of underlying plaque components.

DISCLOSURES

None.

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