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Ultrasound Attenuated Coronary Plaque as a Risk Factor for Slow Flow or No-Reflow During Percutaneous Coronary Intervention: A Case Report

Shinichi FURUICHI, MD
Akira ITOH, MD, FJCC[*1]
Hatsue ISHIBASHI-UEDA, MD[*2]
Teruo NOGUCHI, MD[*3]
Masami MIYAWAKI, MD
Kei NOMURA, MD
Yukio ARITA, MD
Masato OTSUKA, MD[*1]
Takashi SHINDO, MD

Abstract

Slow flow or no-reflow is a serious complication during percutaneous coronary intervention (PCI), but little is known about the risk factors. A 64-year-old man underwent coronary angiography and PCI for stable angina. Pre-interventional intravascular ultrasound demonstrated an ultrasound attenuated coronary plaque, as a long eccentric bulky plaque with a marked decrease of the back echo without calcification. Since the lesion was highly eccentric in the large left anterior descending artery, directional coronary atherectomy (DCA) and subsequent stent implantation were planned. Serious no-reflow occurred after DCA. The DCA specimen suggested that the lipid-laden atheromatous gruel could attenuate the ultrasound reflection and cause distal embolization, resulting in no-reflow during PCI. The presence of ultrasound attenuated coronary plaque is a predictor of slow flow or no-reflow in PCI, indicating that distal protection devices may be required during the procedure.

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Key Words
■ **Angioplasty** ■ **Embolisms** ■ **Complications** ■ **Intravascular ultrasound**

INTRODUCTION

No-reflow is an independent predictor of death and myocardial infarction after percutaneous coronary intervention (PCI).[1,2] However, slow flow or no-reflow during PCI is difficult to predict. Predictors of no-reflow are not well characterized, especially in elective patients.[3–6] The present report describes a case of ultrasound attenuated coronary plaque that was complicated with no-reflow during elective PCI.

CASE REPORT

A 64-year-old man with coronary risk factors of

住友病院 循環器内科：〒530−0005 大阪市北区中之島5−3−20；[*1]大阪市立総合医療センター 循環器内科：〒534−0021 大阪市都島区都島本通 2−13−22；国立循環器病センター [*2]病理部門，[*3]内科心臓部門，大阪
Department of Cardiology, Sumitomo Hospital, Osaka; [*1]Department of Cardiology, Osaka City General Hospital, Osaka; [*2]Division of Pathology, [*3] Division of Cardiology, Department of Medicine, National Cardiovascular Center, Osaka
Address for correspondence: ITOH A, MD, FJCC, Department of Cardiology, Osaka City General Hospital, Miyakojima-hondori 2−13−22, Miyakojima-ku, Osaka 534−0021; E-mail: akiraitoh@ocgh.hospital.city.osaka.jp
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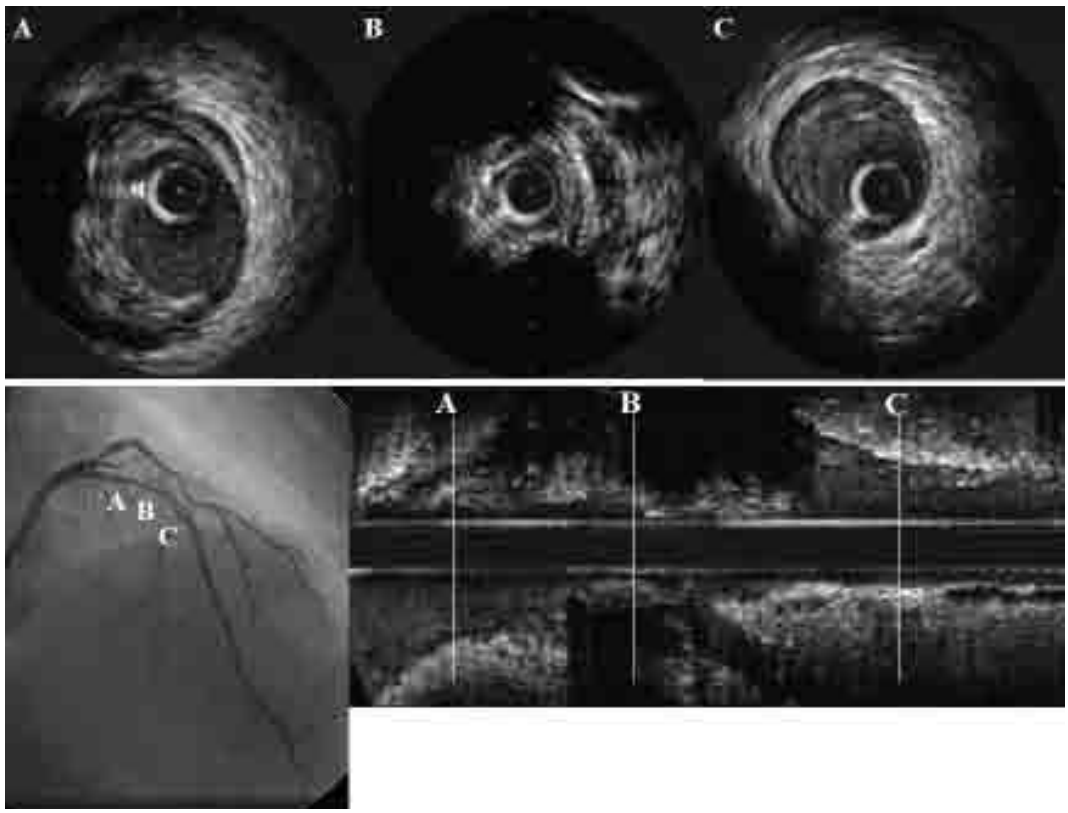

Fig. 1 Coronary angiogram (*lower left*) and intravascular ultrasound images (*A*−*C*) prior to directional coronary atherectomy
Intravascular ultrasound image at site *B* demonstrated the marked ultrasound attenuation. Longitudinal intravascular ultrasound reconstruction (*lower right*) indicated eccentric ultrasound attenuated plaque with positive remodeling.

hypercholesterolemia, hypertension and previous smoking underwent coronary angiography for stable angina. A baseline angiogram indicated a significant stenosis in the proximal portion of the left anterior descending artery (LAD) without calcification. Directional coronary atherectomy and subsequent stent implantation were planned under intravascular ultrasound (IVUS) guidance. An 8F ZUMA2 JCL4 guiding catheter (Medtronic) was passed into the left coronary artery following intravenous administration of 10,000 IU of heparin. A Hi-Torque UNI-CORE guide wire (Guidant) was advanced into the distal LAD and exchanged for a 300 cm Hi-Torque FLEXI-WIRE (Guidant) through a Transit exchange catheter. Pre-interventional IVUS demonstrated an ultrasound attenuated coronary plaque, as a long eccentric bulky plaque with a marked decrease of the back echo without calcification (**Fig. 1**). The vessel diameters estimated by longitudinal IVUS reconstruction were about 5.7 mm at the lesion site, 3.5 mm at the distal reference site, and 4.8 mm at the proximal reference site. Marked positive remodeling was seen at the attenuated plaque site.

To reduce plaque volume, we performed directional coronary atherectomy (DCA) using a FLEXI-CUT atherectomy device (Guidant). After DCA, angiography revealed local dissection at the target site with apparent slow-flow in the LAD. The patient complained of chest pain, which was associated with ST-segment elevation in leads I, aVL, and V_1−V_4. Since coronary dissection at the lesion site was considered to be a possible cause of slow flow, we implanted a 3.5 × 23 mm Multilink Penta stent (Guidant) to cover the lesion. After stenting, angiography showed complete absence of flow in the LAD. Intracoronary administration of nitrates and verapamil did not improve distal flow. To understand the cause of this phenomenon, we performed IVUS, which revealed no residual dissection and adequate dilation of the target site with disappearance of the ultrasound attenuation (**Fig.**

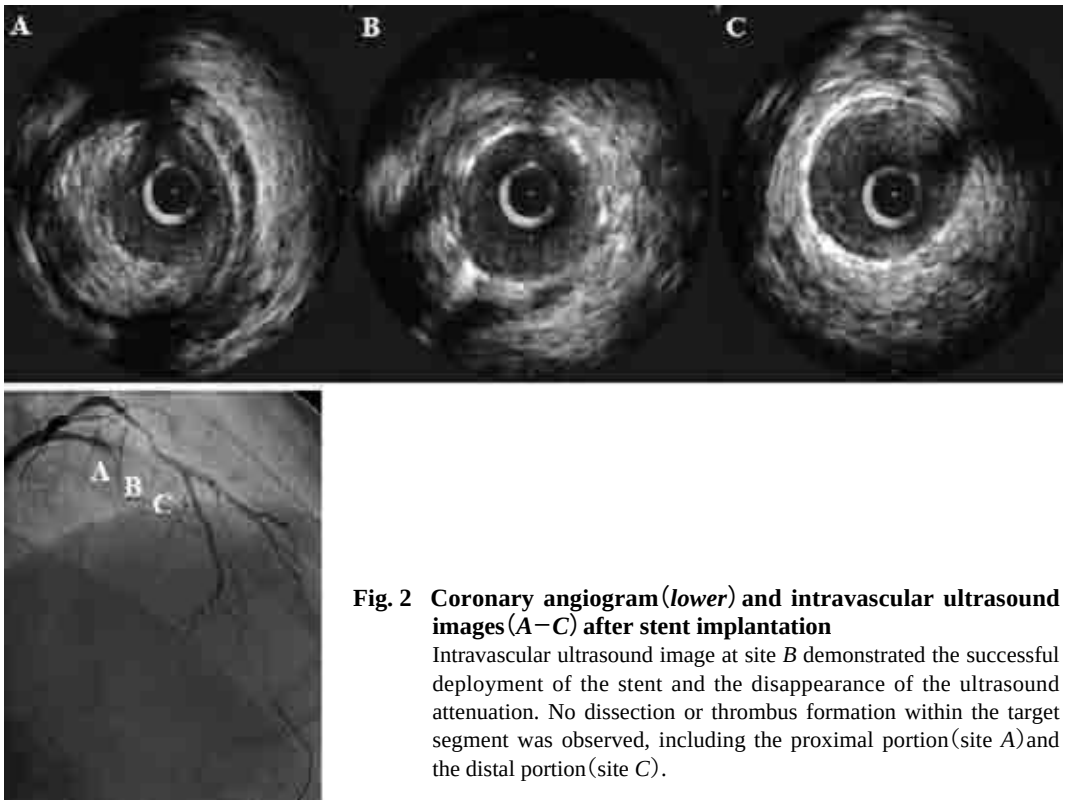

Fig. 2 Coronary angiogram (*lower*) and intravascular ultrasound images (*A*–*C*) after stent implantation
Intravascular ultrasound image at site *B* demonstrated the successful deployment of the stent and the disappearance of the ultrasound attenuation. No dissection or thrombus formation within the target segment was observed, including the proximal portion (site *A*) and the distal portion (site *C*).

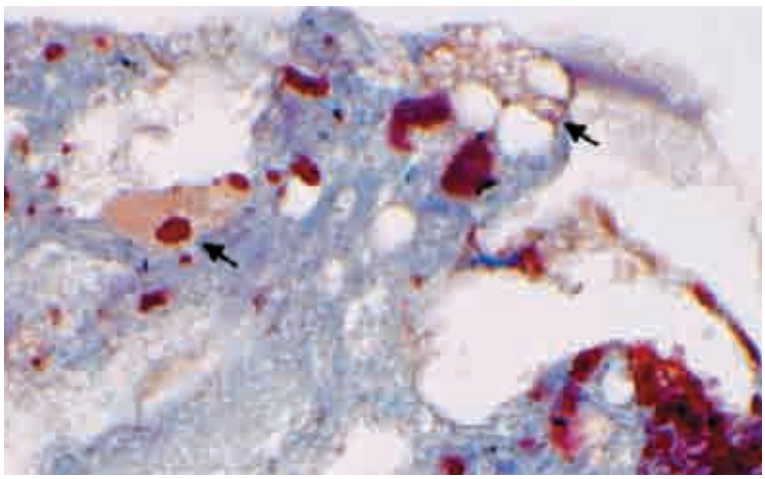

Fig. 3 Photomicrograph revealing cholesterol crystals and foam cells (*arrows*) within the atheromatous core retrieved by directional coronary atherectomy (Masson's trichrome stain, × 200)

2). These findings indicated that no-reflow might have resulted from distal shower embolization of atherosclerotic debris from the ultrasound attenuated coronary plaque. A final angiogram revealed Thrombolysis in Myocardial Infarction-2 flow without residual stenosis. After placement of an intraaortic balloon pumping, the patient was transferred to the coronary care unit. Although he had a Q-wave myocardial infarction with peak creatine kinase levels of 2,694 IU/*l*, he was eventually discharged in stable condition. The histopathological specimen retrieved by DCA contained cholesterol-rich gruel without calcification (**Fig. 3**).

DISCUSSION

Slow flow or no-reflow is a strong independent predictor of in-hospital mortality and postprocedural myocardial infarction.[1,2] However, whether slow

flow or no-reflow might occur during the procedure is difficult to predict. Various predictors of no-reflow are known in patients with acute myocardial infarction.[3−6] Angiographic evidence of thrombus has been implicated with no-reflow in the setting of PCI for acute myocardial infarction.[3,4] IVUS findings of positive remodeling and lipid pool-like image might be risk factors of no-reflow during primary PCI for infarct-related arteries.[5,6] However, the risk factors for slow flow or no-reflow during elective PCI are unknown.

The present case suggests that the presence of an ultrasound attenuated coronary plaque with marked positive remodeling may be a risk factor associated with slow flow or no-reflow even in elective PCI. Although the precise mechanisms responsible for the formation of the ultrasound attenuated coronary plaque remain unknown, the specimen retrieved by DCA indicated that the lipid-laden atheromatous gruel mixed with foam cells could attenuate the ultrasound signals. Micro-calcification inside a plaque may attenuate ultrasound.[7,8] However, we could not detect micro-calcification in the plaque obtained by DCA in this patient. This may due to the technical limitation of DCA, *i.e.* only a part of plaque could be resected and examined. However, the main cause of plaque attenuation may not be micro-calcifications.

The incidence of no-reflow or slow flow is more frequent in patients undergoing PCI for acute myocardial infarction(8.9−11.5%)or for the treatment of saphenous vein grafts(4.0−10.7%), but this phenomenon does occur during elective PCI (1.5%).[1,9] Slow flow or no-reflow is most common during rotational atherectomy(5.1−7.7%),[10,11] followed by stenting or DCA(1.7−3.0%)rather than conventional balloon angioplasty(0.3−1.7%).[9,11] The occurrence of slow flow or no-reflow should be predicted and avoided even in elective PCI. Further studies are needed to determine the more specific features of atheromas causing distal embolization of plaque debris, leading to slow flow or no-reflow.

In conclusion, ultrasound attenuated coronary plaque with marked positive remodeling may be a risk factor for slow flow or no-reflow during elective PCI, and distal protection devices may be necessary. IVUS may help to identify plaques at high risk of slow flow or no-reflow in PCI.

要　約

超音波減衰性プラーク：冠動脈インターベンションにおける冠血流低下の危険因子

古市　晋一　伊藤　彰　植田　初枝　野口　輝夫　宮脇　昌美
野村　圭　有田　幸生　大塚　雅人　新藤　高士

経皮的冠動脈形成術(PCI)中に生じる冠血流遅延や血流途絶は重大な合併症である．しかしながら，血流遅延や血流途絶の危険因子については不明な点が多い．64歳，男性の安定狭心症患者に対して冠動脈造影とPCIを施行した．PCI前の血管内エコー所見では超音波減衰性プラーク(容積が大である偏心性プラークで石灰化を伴わずに後方の超音波信号が減衰している)がみられた．血管径の大きい左前下行枝の偏心性病変であったため，方向性アテレクトミー(DCA)とそれに引き続いてステント留置を施行する方針とした．DCA施行後に血流途絶が発生した．DCAで切除した標本の病理組織学的検討からは，脂質に富むプラーク内容物が超音波の減衰をきたし，末梢塞栓を生じたものと推定された．超音波減衰性プラークの存在は，PCI時の血流遅延や血流途絶の予測因子の一つであり，予防のためには末梢保護器具の併用が必要と推察される．

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