Case Report

Regression of coronary plaque after coronary artery bypass graft

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Summary A 62-year-old woman complained of sudden chest pain and 64-multidetector row computed tomography (MDCT) was performed. The volume-rendered image showed severe stenosis of the left main coronary trunk artery (LMT). The mean density of the plaque was 32.4 hounsfeld units (HU), which indicated soft plaque. Coronary angiography (CAG) showed significant focal stenosis of the LMT. Since the patient had experienced chest pain, and since focal stenosis of the LMT was demonstrated, lipid-lowering therapy using statin and coronary artery bypass graft (CABG, right internal mammary artery-left anterior descending branch, left internal mammary artery-obtuse marginal branch) were applied. Three years after treatment, 64-MDCT showed mild stenosis and a regression of plaque in the LMT. The mean density of the plaque was 73.1 HU (intermediate plaque). CAG showed a degradation of CABG flow, in addition to mild stenosis of the LMT. In conclusion, lipid-lowering therapy with statins may stabilize soft coronary plaque. In addition, non-invasive MDCT is a useful tool for diagnosing coronary artery disease, and for evaluating the size and properties of coronary plaque.

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Introduction

Atherosclerotic plaque is usually considered to build up slowly. Some cases of regression of plaque have been reported worldwide [1]. Lipid-lowering therapy using statins has been shown to reduce the risk of coronary artery disease and other complications [2]. In addition, coronary artery plaque can be imaged noninvasively and assessed by multidetector row computed tomography (MDCT).

MDCT has been recognized as an important resource for the evaluation of known or suspected coronary artery disease, especially for non-calcified lesions, and can also be used to assess anomalies of the aorta, pulmonary artery, other vascular structures, and cardiac chambers [3,4]. We report here a case of regression and a change in the vulnerability of coronary plaque in the left main coronary trunk artery (LMT) after statin treatment and coronary artery bypass graft (CABG).

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Case report

A 62-year-old woman was brought to an emergency room complaining of sudden chest pain at night. She had never experienced chest pain before. This symptom disappeared with the perlingual administration of nitroglycerin 0.3 mg. However, this symptom increased the same night, and she was brought to our emergency room the next day. She had a history of dyslipidemia, but no history of hypertension, diabetes mellitus, or smoking. With regard to the lipid profile (Table 1), total cholesterol (TC) was 204 mg/dL, triglyceride (TG) was 247 mg/dL, high-density lipoprotein cholesterol (HDL-C) was 44 mg/dL, low-density lipoprotein cholesterol (LDL-C) was 133 mg/dL, and the ratio of LDL-C to HDL-C

<table>
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<th>Characteristics of coronary plaque and lipid profile before CABG and 3 years later.</th>
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CABG, coronary artery bypass graft; MDCT, multidetector row computed tomography; LMT, left main coronary trunk artery; HU, hounsfield units; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol L/H ratio, the ratio of LDL-C to HDL-C.

![Image](image)  
**Figure 1** Volume-rendered (A) and multi-planar reconstructed (B) multi-detector row computed tomography images before coronary artery bypass graft and 3 years later. RCA, right coronary artery; LMT, left main coronary trunk artery; LAD, left anterior descending artery; LCX, left circumflex artery.
(L/H) was 3.02. Her blood pressure was 110/68 mmHg without any laterality. Her heart rate was 72 beats/min, regular. She had no cardiac murmur. Electrocardiogram showed normal sinus rhythm and ST depression in leads I and V5-6. Chest X-ray showed a normal cardiac silhouette (cardiothoracic ratio 50.0%). Transthoracic echocardiogram showed normal left ventricular systolic function (left ventricular ejection fraction 58%) and no asynergy. Further investigation was performed using 64-MDCT (Aquilion 64, TOSHIBA, Tokyo, Japan) and a workstation (ZIO STATION, ZIO SOFT, Tokyo, Japan) [5]. The volume-rendered image showed severe stenosis of the LMT (Fig. 1A). Multi-planar reconstruction revealed a similar finding (Fig. 1B), and a cross-sectional image showed severe stenosis with plaque in the LMT (Table 1). The mean density of the plaque was 32.4 Hounsfield units (HU). Coronary angiography showed significant focal 90% stenosis of the LMT (Fig. 2). Since focal stenosis of the LMT was demonstrated, lipid-lowering therapy (pravastatin 5.0 mg/day), antiplatelet therapy (aspirin 100 mg/day), and CABG (right internal mammary artery — left anterior descending branch, left internal mammary artery — obtuse marginal branch) were applied. The use of diltiazem hydrochloride (100 mg/day) as a calcium channel blocker was stopped after CABG, and pitavastatin (2 mg/day) was started after CABG.

Three years after CABG, she experienced chest pain again. TC was 198 mg/dL, TG was 144 mg/dL, HDL-C was 51 mg/dL, LDL-C was 109 mg/dL, and L/H ratio was 1.98 after lipid-lowering therapy. 64-MDCT showed mild stenosis and regression of plaque in the LMT. The mean density of the plaque was 73.1 HU. Coronary angiography showed degradation of CABG flow, in addition to 25% stenosis of the LMT detected by MDCT (Fig. 2).

**Discussion**

This patient represents a case of regression of coronary plaque in the LMT after CABG and statin treatment. Generally, the regression of plaques is caused by lipid-lowering therapy. The levels of L/H ratio are independent risk factors for coronary artery disease. Lower values for the ratio of L/H in addition to a greater percentage increase in HDL-C and lower levels of LDL-C in patients during treatment with statins have been associated with the regression of atheroma [6,7]. Few reports have discussed whether L/H ratio or non-HDL-C (TC minus HDL-C) is a risk factor [6,8], although we previously determined, among the levels of LDL-C, HDL-C, non-HDL-C, and L/H ratio, which is most closely related to the presence of coronary artery disease [9]. There have been some reports of the regression of plaque in patients with L/H ratio under 1.5 [6,7]. The value of L/H ratio in the present case did not fall below 1.5 (from 3.02 to 1.98). Since this
case has no other coronary risk factors, including hypertension, diabetes mellitus, or smoking, and since this case had soft plaque, we thought that lipid-lowering therapy may be significant.

MDCT has been previously used to diagnose atherosclerotic plaque [10, 11]. It has been reported that the mean density of soft plaque was 14 ± 26 HU (−14 to +47 HU), that of intermediate plaque was 91 ± 21 HU (61—112 HU), and that of calcified plaque was 419 ± 194 HU (126—736 HU) [12]. In this case, the mean density of plaque by MDCT before treatment was 32.4 HU. This plaque was classified as soft plaque that is prone to acute coronary syndrome and more vulnerable to rupture [13]. Three years after treatment, the mean density of plaque by MDCT increased to 73.1 HU, which is considered to be intermediate plaque. This increase shows that the plaque vulnerability was stabilized after treatment. MDCT may become a useful tool for studying the course of coronary plaque size and vulnerability, and for appraising the size and vulnerability of coronary plaque.

Although we thought that chest pain 3 years after CABG may have been caused by coronary spasm, we did not perform Holter electrocardiography, treadmill test, and/or provocation test. She may have had coronary spasm because the complaint “sudden chest pain at night” was relieved by nitroglycerin 0.3 mg at first visit and she had never experienced effort chest pain.

In conclusion, lipid-lowering therapy using statin may stabilize soft coronary plaque. Non-invasive MDCT is a useful tool for diagnosing coronary artery disease, and for appraising the size and vulnerability of coronary plaque.

References