Rapid Plaque Progression in a Patient with Non-ST-Segment Elevation Acute Coronary Syndrome: A Case Report

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Abstract

Acute coronary syndrome (ACS), diagnosed by optical coherence tomography (OCT), is caused primarily by plaque rupture, plaque erosion, and calcified nodules. Plaque erosion is more common in patients with non-ST-segment elevation myocardial infarction than ST-segment elevation myocardial infarction. This study reports the details of a case of ACS caused by massive thrombus formation due to plaque erosion. The factors associated with plaque erosion and thrombosis could not be confirmed on the basis of clinical examination findings and the physicians’ experience; therefore, the initial diagnosis of an ischemic event was replaced by a diagnosis of rapid plaque progression, as microscopically confirmed by OCT. Therefore, OCT examination must be performed for blurred or “rapidly progressing” lesions identified through angiography.

Keywords: Non-ST-segment elevation acute coronary syndrome; Plaque erosion; Organized thrombus; Optical coherence tomography

Introduction

Non-ST-segment elevation acute coronary syndrome (NSTEMI) is acute thrombosis caused by severe stenosis of the coronary artery and/or rupture or erosion of a vulnerable plaque, with or without vasoconstriction or microvascular embolism, thus decreasing coronary artery blood flow and leading to myocardial ischemia [1]. Patients with non-ST-segment elevation myocardial infarction (NSTEMI) are more prone to plaque erosion than patients with ST-segment elevation myocardial infarction (STEMI). The formation of much thrombi caused by plaque erosion in the patient’s vasculature which subverts the traditional ischemic events caused by plaque rapid progression. Consequently, optical coherence tomography (OCT) examination must be performed for blurred or “rapidly progressing” lesions identified via angiography.

Case Presentation

A 60-year-old woman was admitted to our hospital on May 6, 2021, because of intermittent chest tightness and pain that had lasted for 30 years but had...
worsened for 1 day. The patient had no history of smoking or drinking. She had a family history of coronary heart disease, and her mother had died of heart disease. Her physical examination after admission indicated the following findings: temperature 36.3 °C, pulse rate 71 bpm, respiration 19 breaths/minute, and blood pressure 132/78 mmHg. She had clear respiratory sounds in both lungs, without dry or wet rales; no murmur was heard in the auscultation area of each valve; and she had a soft abdomen without tenderness. The ECG after admission showed sinus rhythm and was normal (Figure 1). Her troponin was 1.7 μg/L (reference value 0.010–0.025 μg/L). She was diagnosed with acute NSTEMI (Killip grade I). On May 10, 2021, coronary angiography (CAG) was performed, and the left main artery was normal. The degree of stenosis was 40%–50% in the proximal and middle segments of the left anterior descending branch (LAD) and 70%–80% in the middle segment (Figure 2A). The blood flow myocardial infarction thrombolysis test (TIMI) grade was level 3. The degree of stenosis in the distal segment of the left circumflex branch (LCX) was 30%–40% (Figure 2B), and the blood flow TIMI grade was level 3. The degree of stenosis in the middle segment of the right coronary artery

Figure 1  First Electrocardiogram after Admission.

Figure 2  First Coronary Angiogram.
was 20%–30%, and the blood flow TIMI grade was level 3. Because the lesions in the middle of the LAD were blurred, and the patient’s troponin level was elevated, the culprit lesion might be located in the LAD. Therefore, OCT examination was suggested for the patient, but her family refused and requested conservative management. After CAG, she took aspirin 100 mg (once per day), ticagrelor 90 mg (twice per day), atorvastatin 20 mg (once at night), metoprolol tartrate acid 6.25 mg (twice per day), and isosorbide mononitrate 20 mg (twice per day). No further symptoms of angina occurred.

On June 28, 2021, the patient experienced sudden chest pain during intense activity, which was relieved after resting for several minutes. Starting at 09:00 on June 29, 2021, the patient’s intermittent chest pain was relieved within several minutes. At 22:00, the symptoms of sudden chest pain were more

Figure 3  Electrocardiograms.  
(A) Second electrocardiogram after admission-resting electrocardiogram. (B) Second electrocardiogram after admission-seizure electrocardiogram.
severe than before, and were accompanied by pain in the left upper limb, which lasted for 30 minutes. She subsequently visited the emergency department. The ECG showed a sinus rhythm and was generally normal (Figure 3A). The patient was admitted to the hospital on June 30, 2021, and was diagnosed with unstable angina. During hospitalization, several episodes of chest pain symptoms occurred. During the attacks, dynamic changes were observed in the electrocardiogram coinciding with the onset of chest pain, thus indicating that her sinus rhythm and the ST segment of leads I, II, aVF, and V4–V6 were slightly depressed (<0.05 mV) (Figure 3B). After she took a “Suxiao Jiu xin pill,” her symptoms were relieved. (The patient could not tolerate nitrate drugs.) On July 2, 2021, CAG examination showed that the left main artery was normal, the degree of stenosis in the proximal and middle segments of the LAD was 40%–50%, and the degree of stenosis in the middle segment of the LAD was 70%–80% (Figure 4A), with level 3 TIMI blood flow. The degree of stenosis in the distal segment of the LCX was 99% (Figure 4B), and the TIMI blood flow was at level 2. The degree of stenosis in the middle segment of the right coronary artery was 20%–30%, and the TIMI blood flow was at level 3. After the patient’s family consented, an OCT examination was performed. Because of severe LCX lesions, balloon predilation was performed before the OCT examination. The results indicated fibrous plaques, a torn intima, and suspected organized thrombi. The minimum lumen area was 1.28 mm² (Figure 5A–C). After redilation with a predilated balloon, a 3.0 × 23 mm drug-coated stent (MicroPort) was delivered through the guidewire to the distal lesion of the LCX, and the stent was released at a rate of 8 atm (1 atm = 101.325 kPa). CAG (Figure 6) and OCT examination were performed after noncompliant high-pressure balloon dilation. The OCT examination showed satisfactory stent expansion and good adhesion, with a minimum stent internal area of 4.86 mm² (Figure 6A–C). The OCT examination of the LAD showed fibrous plaques, organized thrombi, and a minimum lumen area of 1.36 mm² (Figure 7A–C). Therefore, a 2.75 × 18 mm drug-eluting stent (Medtronic) was delivered through the guidewire to the middle lesion of the LAD at a rate of 12 atm. CAG (Figure 8) and OCT examination were performed after noncompliant high-pressure balloon dilation. The OCT examination results indicated satisfactory stent expansion, good adhesion, and a minimum lumen area of 4.19 mm² (Figure 8A, B). After PCI, the patient regularly took 100 mg aspirin (once per day), 75 mg clopidogrel (once per day), 10 mg rosuvastatin (once at night), and 47.5 mg metoprolol succinate (once per day). Because she was unable to tolerate the associated suffocation symptoms, ticagrelor was replaced with oral clopidogrel. The aggregation rates of AA and ADP in platelet function tests during hospitalization were 7.62% and 23.06%, respectively (clopidogrel was replaced by ticagrelor during the second hospitalization). In the follow-up to date, no further symptoms of angina had been observed.

Figure 4  Second Coronary Angiogram.
**Figure 5**  LCX-OCT Imaging before PCI.

**Figure 6**  LCX-Coronary Angiography and OCT Imaging after PCI.
**Figure 7**  LAD-OCT Imaging before PCI.

**Figure 8**  LAD Coronary Angiography and OCT Imaging after PCI.
Discussion

NSTE-ACS is acute thrombosis caused by severe stenosis of the coronary artery and/or rupture or erosion of a vulnerable plaque, with or without vasoconstriction or microvascular embolism, thus decreasing coronary artery blood flow and leading to myocardial ischemia [1]. Jia et al. [2] have found that the main manifestations of ACS on OCT imaging are plaque rupture, plaque erosion, and calcified nodules. Patients with NSTEMI are more prone to plaque erosion than patients with STEMI. Plaque erosion is characterized by an abnormal or discontinuous surface of the vascular endothelium with attached thrombi, and no damage to the fiber cap. Thrombosis organization refers to thrombus formation, wherein endothelial cells and fibroblasts grow from the vascular wall toward the thrombus. The granular tissue extends into and gradually replaces the thrombus, thus leading to mechanization. Mechanized thrombus adheres to the blood vessel wall and is not easily detached. The newly formed endothelial cells in the organized thrombus cover the cracks generated by the drying up of the thrombus, thus forming a labyrinthine but communicable channel, allowing for partial communication of blood flow upstream and downstream of the thrombus. The phenomenon known as “recanalization” has been found in several studies and case reports [3–5]. The occasional vascular lesions with a “woven” appearance found on CAG images are actually organized thrombus recanalization, as observed in OCT images. Multiple recanalization channels are referred to as “honeycomb vasculature,” “Swiss cheese,” or “lotus root lesions.” Our patient was diagnosed with NSTE-ACS during both hospitalizations. OCT examination did not reveal plaque rupture but instead indicated an organized thrombus, which adhered to the surfaces of fibrous plaques. Plaque erosion led to thrombus formation, thus indicating that severe stenosis of the lumen did not originate from the plaque itself but from the erosion of the plaque, thus forming thrombus that did not completely occlude the lumen. Through thrombus organization, the effective area of the lumen was decreased; therefore, the thrombus might have been a recanalized thrombus rather than a multichannel organized thrombus. Because of the large volume of the thrombus, the effective area of the lumen (single) decreased, thereby leading to the occurrence of NSTE-ACS.

Men younger than 50 years or women younger than 70 years are at high risk of plaque erosion [6]. A multicenter retrospective study has shown that patients with plaque erosion are often younger than 68 years, and have a history of angina pectoris, no history of diabetes, hemoglobin >150 g/L, and normal renal function. Patients with NSTE-ACS with these five clinical characteristics are prone to plaque erosion, which occurs with an incidence as high as 73.1% [7]. Some studies have shown that the lesions in patients with NSTE-ACS caused by plaque erosion occur primarily in the middle segment of the LAD [8]. From a pathological perspective, in plaque erosion, the fiber cap on the surface of the plaque is intact and damage occurs to endothelial cells. Studies have shown that, in addition to endothelial damage, changes in the extracellular matrix, particularly elevated levels of proteoglycans and hyaluronic acid, can be observed on the surfaces of eroded plaques [9, 10]. Simultaneously, many neutrophils aggregate, thus causing endothelial cell damage, activating platelets and coagulation systems, and leading to thrombus-induced ACS. During our patient’s two hospitalizations, laboratory examinations revealed neutrophil levels within the upper limit of the normal range. The C-reactive protein level (9.4 mg/L) and erythrocyte sedimentation rate (25 mm/h) were slightly elevated. Immunological indicators and tumor markers were normal. From a clinical perspective, we were unable to confirm factors related to plaque erosion and thrombosis. However, the formation of much thrombi caused by plaque erosion in the patient’s vasculature which subverts the traditional ischemic events caused by plaque rapid progression. OCT confirmed this diagnosis from a microscopic perspective. Consequently, OCT examination must be performed for blurry or “rapidly progressing” lesions found via CAG.

In addition, our patient experienced rapid plaque progression, caused primarily by plaque instability – particularly that of vulnerable plaques, which are likely to rupture – as well as plaque erosion accompanied by thrombosis or intraplaque bleeding. Inflammatory infiltration, lipid accumulation, and positive vascular remodeling of plaques are all involved in the rapid progression of plaques [11]. Some studies have shown that the vascular adventitia, the connective tissue of the adventitia and adipose tissue, affects the vulnerability of atherosclerotic plaques [12]. In one case report, a 41-year-old man showed noncriminal lesions progressing from
30% stenosis to occlusive lesions after 10 days. However, this patient did not undergo intracavitary imaging examination. This rapidly progressing plaque, which is considered to be related to chronic inflammation caused by long-term smoking, dyslipidemia, etc. [13]. Intraplaque hemorrhage was found in our patient’s OCT images, but no significant microvessel formation was observed within the plaques. However, in the early stages of neovascularization, incomplete maturation and fragility of the vessels can lead to bleeding within plaques, thus increasing plaque instability. Plaques accompanied by intraplaque hemorrhage often have thin fiber caps, macrophage infiltration, and necrotic cores, which together lead to plaque rupture [14]. This case report has several limitations, such as the lack of testing for inflammatory mediators such as high sensitivity C-reactive protein and interleukin-6. The exact reason for the patient’s susceptibility to plaque erosion and organized thrombosis remains unclear.

Data Availability Statement

The data underlying this article are available in the article.

Conflict of Interest

The authors have no conflicts of interest to declare.

Patient Consent

The patient consented to the use of his medical information and data.

Author Contributions

Zengming Xue performed the main research. Yachao Li wrote the main article. Mengjie Lei and Jingyao Wang analyzed the data. Yanli Yang and Zhigang Zhao prepared figures. ZX critically reviewed and revised the article.

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