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Case Report

A Case of Recurrent Coronary Artery Restenosis Associated with Suspected Arteritis in a Young Woman

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Abstract

Approximately 5% of patients with Acute Myocardial Infarction (AMI) do not have atherosclerotic Coronary Artery Disease (CAD) but present other factors responsible for luminal narrowing. A 24-year-old woman presented with angina and AMI without any known risk factors and recurrent coronary restenosis in the 7 year-follow-up period. Thorough tests revealed no suggestive factors besides increased amounts of tumor necrosis factor-α and interleukins. A diagnosis of arteritis was suspected. Therefore, glucocorticoid and immunosuppressor administration was performed, and the patient's condition was relieved. Severe coronary artery disease with smooth distal coronary vessels in a young female without apparent risk factors could suggest a non-atherosclerotic etiology.

Keywords: Coronary artery disease; Acute myocardial infarction; In-stent restenosis; Arteritis; Glucocorticoid; Immunosuppressor

Abbreviations

AMI: Acute Myocardial Infarction; CABG: Coronary Artery Bypass Grafting; CAD: Coronary Artery Disease; CAG: Coronary Angiography; DES: Drug-Eluting Stent; ECG: Electrocardiograph; ISR: Intra-Stent Restenosis; IVUS: Intravascular Ultrasound; LAD: Left Anterior Descending Artery; LCX: Left Circumflex Artery; LM: Left Main Coronary Artery; OCT: Optical Coherence Tomography; TA: Takayasu Arteritis

Introduction

Atherosclerosis is the leading cause of Coronary Artery Disease (CAD) and Acute Myocardial Infarction (AMI). Approximately 5% of AMI patients do not have atherosclerotic CAD but present other factors causing luminal narrowing [1]. CAD and AMI are rarely diagnosed in young females. The diagnosis of severe CAD with smooth distal coronary vessels in a young woman without apparent risk factors could suggest a non-atherosclerotic etiology, e.g., congenital abnormality, coronary artery spasm, embolism, spontaneous dissection, drug abuse, syphilis, chest radiotherapy, Marfan syndrome, or autoimmune diseases [2].

Case Presentation

A 24-year-old woman presented in October 2012 with progressive tightening chest pain, with onset after walking fast about 50 meters and relief following a brief rest. She was otherwise healthy. Family history was unremarkable. She was admitted to a local hospital in November 2012 for persistent chest pain for 3h, accompanied by sweating, nausea and vomiting. Physical examination was unremarkable. Electrocardiograph (ECG) showed slightly elevated ST segment in chest leads V1-4, and limb leads I, avL and avR; poor R wave progression in chest leads V1-3; and high amplitude T wave in leads V2-4 (Figure 1A). T wave amplitude in leads V2-4 decreased 2h post-admission (Figure 1B). Laboratory tests showed slightly increased troponin and myocardial enzymes (cTNI, 1.2 ng/ml; CK-MB, 27 IU/L; CK, 331 IU/L). Blood lipids, ESR, CRP, D-dimer, C3, rheumatoid factor and anti-streptolysin O were normal (Table 1). AMI was diagnosed until the next day with significantly increased troponin and myocardial enzymes; ECG showing QS wave in leads V1-3, I and avL, inverted T wave in leads V2-5, I and avL (Figure 1C); and segmental abnormal left ventricle anterior wall movement on echocardiogram. AMI was immediately treated with anticoagulation, antiplatelet and lipid lowering therapy with statin. Serological tests were negative for antinuclear antibodies, extractable nuclear antigens, anti-double stranded DNA antibodies, antineutrophil cytoplasmic or antiphospholipid antibodies, syphilis, hepatitis B and C virus, and human immunodeficiency virus.

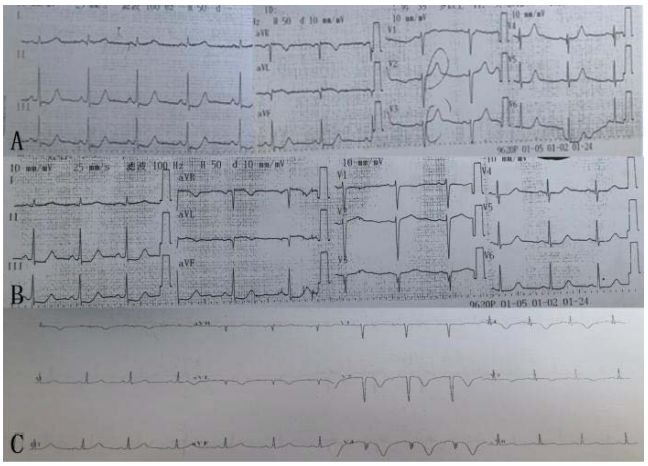

Figure 1: A: ECG at admission (8h after attack). ST segment was elevated in leads V1-4, I, avL and avR, depressed in leads II, III and avF. R wave show poor progression in leads V1-3. T wave amplitude was high in leads V2-4. **B:** ECG 2h after admission (10h after attack). T wave amplitude decreased in leads V2-4 compared with ECG at admission. **C:** ECG on the second day. ST segment returned to the equipotential line. R wave disappeared in V1-3, I and avL. R wave amplitude decreased in V4-6, II, III and avF, and QS amplitude was lower in avR. There were inverted T wave in V2-5, I and avL, and flat T wave in V6.
ECG: electrocardiogram.

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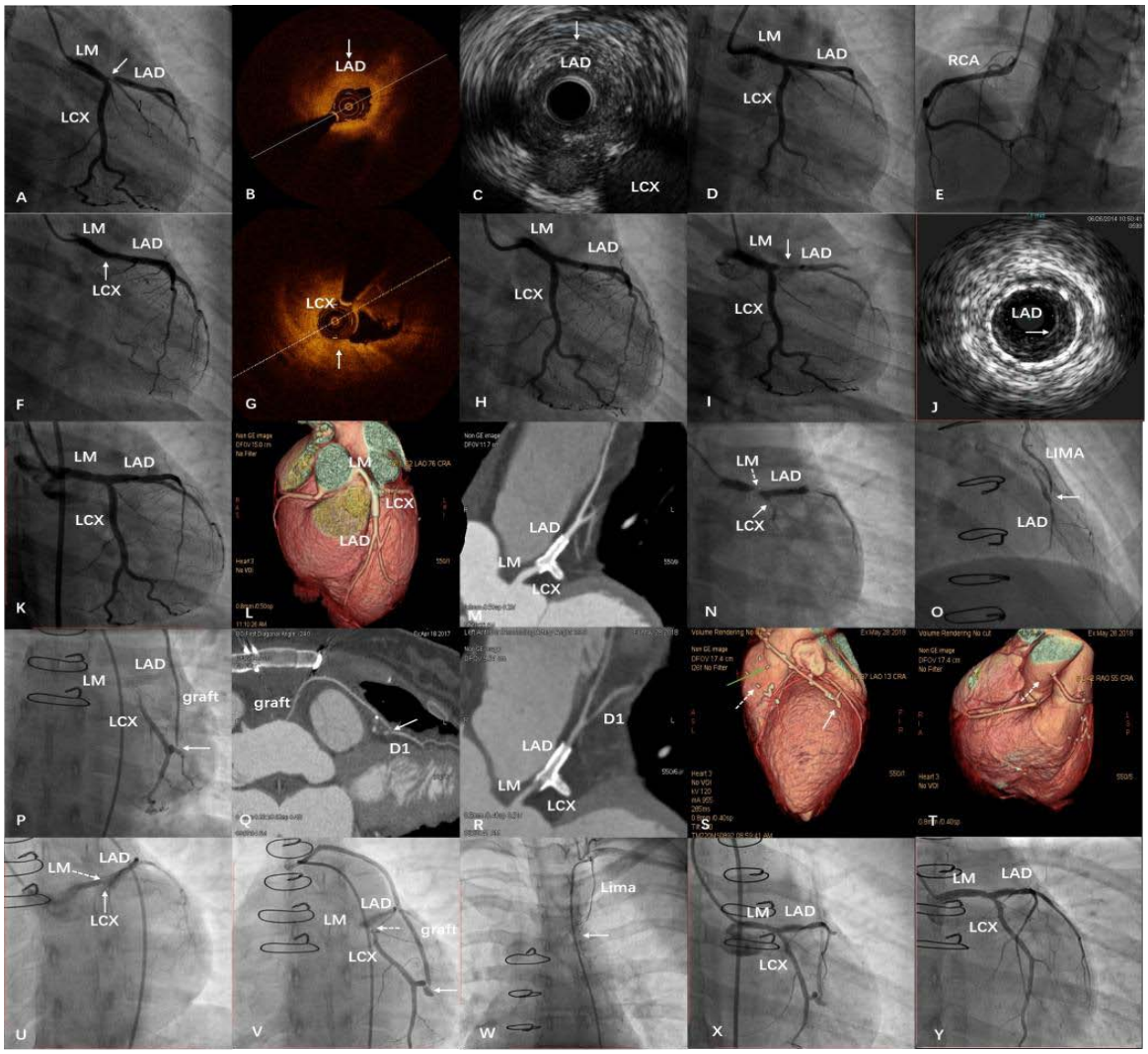

Figure 2:
A: Severe stenosis at the orifice of the LAD (arrow) by CAG in November 2012.
B: Fibrosis plaque in the orifice of the LAD (arrow) by OCT.
C: Fibrosis plaque in the orifice of the LAD (arrow) by IVUS.
D: CAG after stent implantation.
E: Normal right coronary artery.
F: Totally occluded LCX at the orifice (arrow) by CAG in December 2013.
H: CAG after stent implantation.
I: Severe ISR in the LAD (arrow) in June 2014.
K: CAG after stent implantation.
L: Enhanced CT showing patent stents in the left coronary artery in November 2015.
M: Enhanced CT showing patent stents in the left coronary artery in November 2015.
N: Severe ISR in the LCX (solid arrow) and LM (dotted arrow) in March 2017.
O: Severe stenosis at the anastomotic stoma of LIMA with the LAD (arrow) in April 2017.
P: Severe stenosis at the anastomotic stoma of the graft with obtuse marginal branch (arrow).
Q: Severe stenosis at the anastomotic stoma of the graft with the first diagonal branch in enhanced CT (arrow).
R: Enhanced CT showing a patent stents in the left coronary artery in May 2018.
S: Occluded obtuse marginal branch distal to the anastomotic stoma (solid arrow); totally occluded grafts to the LAD (green arrow) and the first diagonal branch (dotted arrow) by enhanced CT.
T: Occluded graft to the first diagonal branch from the origin (dotted arrow).
U: Intrastent occlusion in the LCX (solid arrow) and LM (dotted arrow) and in November 2018.
V: Occluded obtuse marginal branch distal to the anastomotic stoma (arrow).
W: Occluded LIMA at the middle segment (arrow).
X: CAG after balloon dilation.
Y: CAG showing patent left coronary artery in December 2018.
CAG: Coronary Angiography; CT: Computed Tomography; ISR: Intrastent Restenosis; IVUS: Intravascular Ultrasound; LAD: Left Anterior Descending Artery; LCX: Left Circumflex Artery; LIMA: left internal mammary artery; LM: Left Main Artery; OCT: Optical Computed Tomography.

One week post-admission, Coronary Angiography (CAG) showed severe stenosis at the orifice of Left Anterior Descending Artery (LAD) (Figure 2A). Optical Coherence Tomography (OCT) (Figure 2B) and Intravascular Ultrasound (IVUS) (Figure 2C) showed fibrous plaque in the LAD after thrombus aspiration. A Resolute Drug-Eluting Stent (DES, 3.5×15 mm; Medtronic, USA) was implanted from the LAD to the Left Main Coronary Artery (LM) (Figure 2D). The patient was administered daily aspirin (100 mg), clopidogrel (75 mg) and rosuvastatin (20 mg) for CAD prevention.

She re-developed exertional angina in December 2013. CAG showed totally occluded Left Circumflex Artery (LCX) (Figure 2F). OCT revealed severe intimal fibrous proliferation (Figure 2G). Another DES (Xience Prime, 3.5×15 mm; Abbott Laboratories, USA) was implanted (LCX to LM) (Figure 2H). Tests for autoantibodies and inflammatory factors were still negative. CYP2C19, PON1 and ABCB1 genes (clopidogrel absorption and metabolism) were all wild type. Thromboelastography showed good response to aspirin and clopidogrel.

She had recurrent exertional angina in June 2014. CAG showed intra-stent restenosis (ISR, 99%) in the LAD (Figure 2I). IVUS showed severe intimal fibrous proliferation (Figure 2J). A DES (Promus Element, 4×12 mm; Boston Scientific Corporation, USA) was implanted (Figure 2K). CRP and ESR were both normal, and LDL-C was 1.59 mmol/L. Coronary enhanced CT showed patent stents in the left coronary artery in November 2015 (Figure 2L and 2M).

Exertional angina was re-diagnosed in February 2017. CAG showed severe ISR in LM and LCX (Figure 2N). She underwent Coronary Artery Bypass Grafting (CABG) in March 2017 (left internal mammary artery to LAD, right internal mammary artery from aorta to obtuse marginal branch, and external femoral artery from aorta to diagonal branch). Pathology showed no aortic wall inflammation or atherosclerosis. Unfortunately, angina occurred again 3 weeks after CABG. Enhanced CT and CAG revealed severe anastomotic stenosis (90%) in three grafts (Figure 2O, 2P and 2Q). Autoantibodies, proteins C and S, anti-thrombin III, ESR and CRP remained negative. However, TNF-α, IL-6, IL-8 and IL-10 amounts were increased (Table 2). Rheumatologists highly suspected arteritis, and prednisone (10 mg Qd, add to 30 mg Qd) and mycophenolate mofetil (1g Bid) were administered. TNF-α and interleukin amounts gradually decreased, and angina disappeared with daily activities one month later. Glucocorticoid and immunosuppressor administration were continued and adjusted according to the levels of inflammatory factors. Enhanced CT in May 2018 showed severe ISR in the LM, LAD and LCX, occluded bypass grafts in the LAD and diagonal branch, and severe distal anastomotic stenosis in the obtuse marginal branch (Figure 2R, 2S and 2T).

Angina exacerbated with daily activity in December 2018. CAG showed severe ISR in the LM, LAD and LCX (Figure 2U, 2V and 2W). Angioplasty was performed with two drug-eluting balloons (Bingo, 3.5*15 mm; Yinyi, China) (Figure 2X). Prednisone dose was increased from 10 mg to 20 mg QD. Another CAG was performed one month later, revealing patent stents (Figure 2Y and Figure 3). Prednisone dose was gradually decreased to 10 mg QD in April 2019. Ultrasound showed slightly increased intima-media thickness in right subclavian artery and abdominal aorta in May 2019, which was 12 mm and 14 mm, respectively. She was well with no angina, and secondary CAD prevention plus prednisone/mycophenolate mofetil were continued.

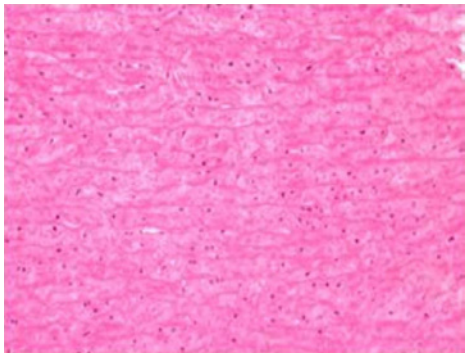

Figure 3:

Discussion

This young female patient had repeated occlusion of coronary arteries without apparent risk factors. Atherosclerotic and non-atherosclerotic causes other than autoimmune diseases were easily ruled out. The commonest autoimmune diseases affecting coronary arteries include polyarteritis nodosa, giant cell arteritis, systemic lupus erythematosus, Kawasaki's disease and Takayasu Arteritis (TA) [1].

Polyarteritis nodosa affecting medium and small vessels is mainly found in middle aged and elderly people, especially men [3]. About 62% patients have epicardial coronary artery involvement and myocardial infarction. The involved artery may dilate to form small berry-like aneurysms, become occluded, or rupture. Here, the coronary artery was the only affected target, with no wall destruction.

Lupus affects coronary arteries, with antiphospholipid antibodies increasing cardiovascular events. However, lupus was unlikely in this patient with no autoantibodies.

Giant cell arteritis usually affects temporal and vertebral arteries in the elderly. The coronary artery is rarely affected, and only few AMI cases have been reported in giant cell arteritis. The symptoms of this patient did not reflect giant cell arteritis.

Kawasaki disease mainly affects children <2 years old. The vasculitis of the coronary vasa vasorum could cause coronary artery aneurysm, thrombosis and dissection, leading to AMI in about 20% of patients [1]. Therefore, Kawasaki disease was also excluded.

TA, a granulomatous vasculitis, mainly affects the aorta and multiple major branches, causing arterial stenosis, occlusion or dilation in young females [4]. The onset age is 5-45 years, and individuals less than 30 years old account for about 90% of all patients [5]. Coronary artery is involved in 10-30 % patients, with 3 types of lesions: type 1, stenosis of the coronary ostia or proximal segments of the coronary arteries; type 2, diffuse or focal coronary arteritis; and type 3, coronary aneurysm [6]. The orifice is the most affected site [7]. There are several case reports of isolated coronary ostial stenosis in TA [8].

TA was highly suspected for this patient: 1) onset age <40 years;

2) recurrent stenosis in the coronary artery, intra-stent and bypass grafts; 3) increased TNF-α and ILs; 4) thickened intima-media in right subclavian artery and abdominal aorta during follow-up; 5) glucocorticoid and immunosuppressor administration alleviating the condition. However, according to the American College of Rheumatology criteria for TA [9], this case could not be confirmed as TA for the following reasons: 1) aorta and its primary branches other than the coronary artery not affected; 2) normal ostia of the left and right coronary arteries; 3) no aortic wall inflammation. Noma et al. [10] reported a 21-year-old woman with isolated left coronary ostial stenosis who had CABG with CRP negative and no confirmed aortitis. Severe stenosis of the major branches of the aortic arch and elevated CRP and ESR occurred about fifteen years post-surgically, and TA was diagnosed. Therefore, the primary aortic branches may be affected in the future. This case was diagnosed with coronary arteritis, and a glucocorticoid/immunosuppressor regimen alleviated the condition.

A few cases similar to the present one have been described. A 24-year old female TA case successfully underwent percutaneous coronary angioplasty, implanting a DES with further prednisolone and cyclophosphamide treatment for 5 months; however, high-grade ISR caused recurrent angina [11]. Despite high-dose prednisolone and tocilizumab, the high-grade ISR persisted, prompting for aortocoronary CABG, which was successful [11]. Another TA case with recurrent restenosis after bifurcation stenting of proximal LAD and first diagonal arteries successfully underwent CABG [12]. In addition, a 46-year-old man with polyarteritis nodosa and multiple AMI administered repeatedly percutaneous coronary interventions showed atypical angina again, and was treated by four-vessel CABG [13]. Furthermore, a TA case with carotid stenosis initially treated with bare metal stents was administered coronary DES and drug-eluting balloon with long-term positive effects [14].

Conclusion

A non-atherosclerotic etiology should be considered for differential diagnosis in young patients with CAD and AMI. Autoimmune diseases should be taken into account, especially in patients with recurrent coronary restenosis. Correct diagnosis and timely treatment could improve patient prognosis.

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