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Case Report

Coronary Artery Ectasia Presenting With Recurrent Inferior Wall Myocardial Infarction

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Abstract

Coronary ectasia presenting as a recurrent inferior myocardial infarction has rarely been reported in the literature. Herein, we report a 61-year-old man who presented with persistent chest pain accompanied by ST segment elevation in the inferior ECG leads. Coronary angiography showed ectasia of the right coronary artery (RCA) and total occlusion from the middle RCA. Two stents were implanted separately in the middle and distal RCA. The patient was readmitted due to recurrent inferior wall infarction 15 months after discharge. He underwent primary percutaneous coronary intervention again, and coronary angiography showed massive thrombosis and in-stent re-stenosis. The thrombosis and re-stenosis were successfully treated using balloon angioplasty. The patient was discharged under medical therapy with aspirin and clopidogrel. There were no anginal symptoms during the 3 years of follow up. (*Tzu Chi Med J* 2010;22(2):119–122)

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1. Introduction

Coronary artery ectasia (CAE) is a coronary anomaly that results in significant dilatation of one or more of the major coronary arteries. The angiographic diagnostic criteria of CAE is a segment of the artery with a diameter >1.5 times the normal segment (1). The prevalence of CAE is 0.3–12% at autopsies and during coronary angiography or multidetector computed tomography (2,3). Recurrent acute myocardial infarction (AMI) in the presence of coronary ectasia is uncommon, comprising <1% of all cases of coronary ectasia. Therefore, we report a case of recurrent AMI in the presence of coronary ectasia from our institution.

2. Case report

A 61-year-old man was admitted to Dalin Tzu Chi General Hospital because of the sudden onset of severe substernal chest pain with radiation to the left arm and jaw 2 hours prior to admission. The associated symptoms included nausea, vomiting, cold sweating, and dyspnea.

He had a history of angina that was relieved by nitroglycerin prior to this admission, but the pain had become progressively more frequent and was occurring while at rest. He was diagnosed with hypertension 10 years prior to this admission and had a history of hyperlipidemia. On physical examination, his blood

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pressure was 140/94 mmHg. His lungs were clear and sinus rhythm was present. There were no murmurs, gallop sounds, or friction rubs. The pulses were equal in the upper and lower extremities.

The resting electrocardiogram showed ST segment elevation in leads II, III and AVF, and AMI was diagnosed. Emergent coronary catheterization was performed and revealed total occlusion from the middle right coronary artery (RCA). The left main coronary artery, anterior descending and circumflex arteries were normal. Mild posterior and basal hypokinesis was noted on left ventriculography.

Primary percutaneous transluminal coronary angioplasty (PTCA) of the total RCA occlusion was performed using a 7-F guiding catheter and a 3.0-mm Maverick balloon dilation catheter over a 0.014-inch steerable wire. We chose a 3.0-mm balloon because the normal segment of the RCA (p-RCA) was 3.0 mm. Following PTCA, there was an intima flap from a longitudinal plane of dissection without significant obstruction of flow. Coronary ectasia was noted because of the dilated middle and distal RCA (4.53 mm in maximal diameter, but not the stenotic site). Two stents (bare metal stents, 3.5×20 mm) were implanted in the middle and distal RCA. The patient was asymptomatic and stable hemodynamically. Intravenous heparin was infused continuously for 24 hours, and he had an uneventful recovery. Atrioventricular block present at the time of admission had disappeared after several days (Figs. 1 and 2). Dual antiplatelet therapy (aspirin and clopidogrel) was given for 3 months after stent implantation.

The patient remained asymptomatic for 15 months after discharge, but then he noted the return of chest pains. Repeat coronary angiography showed total occlusion from the proximal RCA. The left coronary artery system remained normal. Primary PTCA was successfully performed; however, some thrombus remained

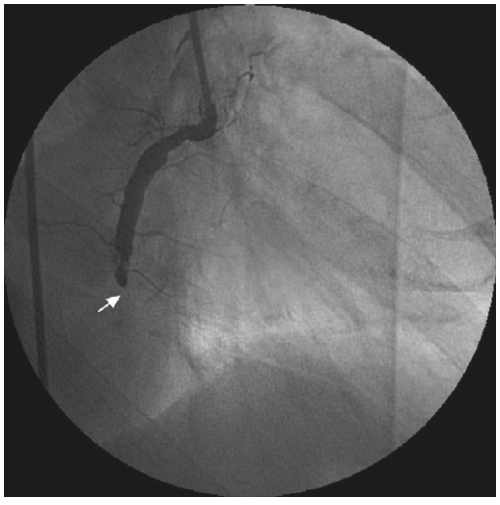

Fig. 1 — Pre-catheterization.

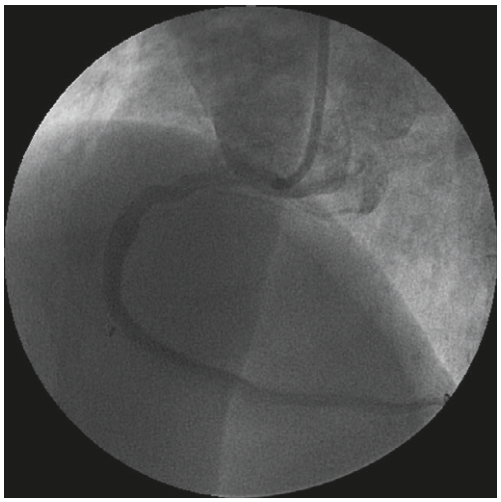

Fig. 2 — Post-catheterization.

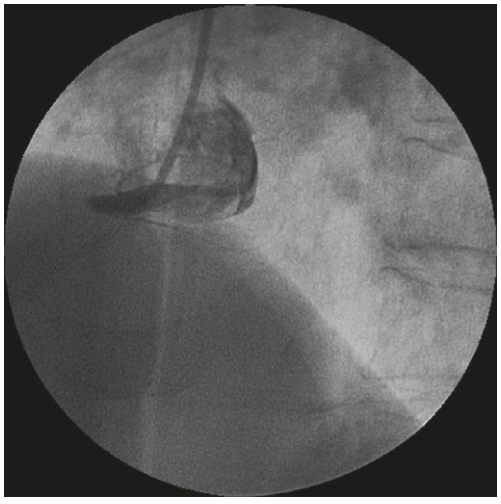

Fig. 3 — Pre-catheterization (15 months later).

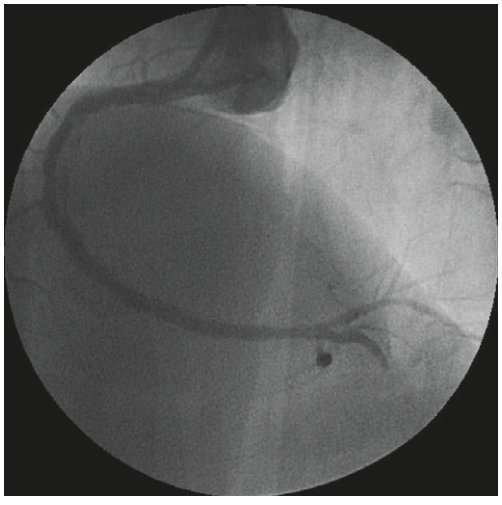

Fig. 4 — Post-catheterization (15 months later).

in the RCA (Figs. 3 and 4). Heparin was administered for 5 days. The patient performed well under medical treatment with clopidogrel, diltiazem, aspirin, and lovastatin. Dual antiplatelet therapy (aspirin and clopidogrel) was given for 1 year. There were no further anginal symptoms during 3 years of follow-up.

3. Discussion

CAE has been observed by pathologists and cardiologists for more than two centuries. This coronary anomaly was first described by Morgagni in 1761 [4]. Bourgon was the first to describe the postmortem findings of RCA dilatation in a patient who experienced sudden death in 1812 [5]. The term "ectasia" was first used by Bjork in 1966 to describe dilated coronary arteries [6]. The proximal and middle parts of the RCA are most commonly affected by ectasia, although the reasons for this are not clear. Involvement of the left anterior descending artery and left circumflex artery is variable. The left main coronary artery is less commonly involved. Most cases of CAE involve only a single vessel.

More than 50% of CAE cases were reportedly caused by atherosclerosis [7–9]. Compensatory vessel enlargement in the presence of coronary atherosclerotic plaques is a common phenomenon that is viewed as positive remodeling [10]. Progressive overcompensation leading to ectasia may be caused by an inadequate extent of media atrophy and fracture of the internal elastic lamina, as well as atypical rearrangement of smooth muscle cells [11,12]. Virmani et al [13] provided a detailed pathologic characterization of CAE, including lipid deposition with foam cells, fibrous caps, and significant loss of smooth muscle and elastic components of the vascular wall as the main histologic abnormalities. The reason for why stenosis develops in some individuals with atherosclerosis, and dilatation occurs in others is unknown.

In this report, we described a patient with CAE and recurrent thrombus resulting in AMI. While this patient had a history of diabetes mellitus, hyperlipidemia and hypertension, coronary angiography revealed only mild atherosclerosis in the left coronary artery. However, significant diffuse ectasia was noted in the RCA which was accompanied by recurrent thrombosis and AMI. It is likely that the patient had recurrent myocardial infarction due to very late in-stent thrombosis and coronary ectasia. Reduced flow within the dilated segments has been demonstrated and might predispose a patient to thrombotic occlusions of the affected artery [14]. We discovered a massive thrombus in the proximal and middle RCA during the second intervention. However, coronary flow was still reduced after suction of the thrombosis. Coronary flow reached TIMI-3 until the fully dilated distal RCA in-stent re-stenosis was done.

CAE is not a benign coronary anomaly. Sorrel et al [15] suggested treating CAE patients with: (1) chronic warfarin therapy with the maintenance of an international normalized ratio of 2.0–2.5 to reduce the risk of thrombosis; (2) aspirin (80–360mg/day) to minimize platelet aggregation; and (3) calcium-channel blockers to prevent coronary vasospasm. Therapy should be tailored to each individual patient because of the serious bleeding complications associated with warfarin use. Heparin and thrombolytic therapy have been successfully used for recanalization. For patients with co-existing obstructive lesions and refractory angina despite medical treatment, percutaneous coronary intervention (PCI) can be performed. However, stents may be difficult to deploy because the ectatic arteries are usually much larger than normal arteries. Selection of a stent of adequate size and its expansion are very important, and can be determined by intravascular ultrasound. Coronary artery bypass grafting (CABG) has been used for many years for the treatment of significant coronary artery disease coexisting with CAE, and the postoperative outcome has been uniformly good. Valente et al [16] reported that the prognosis of CAE was good with a low mortality rate (2%), possibly because their patients with ST-elevation AMI and acute coronary syndrome all underwent PCI, and the most severe cases with coexisting coronary stenosis underwent CABG. Our patient received primary PCI twice with good results. However, continuous double antiplatelet therapy may be the main reason underlying the good prognosis and symptom-free survival for 3 years.

CAE is worthy of closer study. Treatment should be tailored according to clinical manifestations and the number and involvement of the coronary arteries. Medical therapy is recommended for patients with symptomatic non-obstructive CAE. PCI may be performed in some patients, but the choice of stent size is a challenging issue. Surgery (CABG, aneurysm ligation or aneurysmectomy) is a better choice for patients who are refractory to medical therapy and not suitable for PCI.

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