

Typical coronary artery aneurysm exactly within drug-eluting stent implantation region in a patient with rheumatoid arthritis

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ABSTRACT

The information presented comes from a case report concerning a left anterior descending coronary artery aneurysm (CAA). The typical “zig-zag” phenomenon, developed exactly within the segment of the sirolimus-eluting stent (SES), and in the left anterior descending coronary artery (LAD). The patient had a previous history of rheumatoid arthritis. We speculated that the CAA could be related to the vascular inflammatory reaction caused by the rheumatoid arthritis and the drug-eluting stent implantation.

Key words: Coronary artery aneurysm, rheumatoid arthritis, sirolimus -eluting stent

INTRODUCTION

Coronary artery aneurysm (CAA) formation after stent implanted^[1-3] was reported before. However, the typical CAA which has been related to both rheumatic disease and drug-eluting stent (DES) implantation has been reported rarely. This case showed typical “zig-zag” phenomenon exactly within the segment of sirolimus-eluting stent (SES) in left anterior descending coronary artery (LAD) and had a special history of rheumatoid arthritis. Both the inflammatory rheumatic disease and DES implantation might promote the formation of aneurysms.

The patient was a 51-year-old man who had a pre-existing history of rheumatoid arthritis, diagnosed 20 years earlier, but he had never used a prescription, not even steroids.

In 2004, the patient had experienced an acute myocardial

infarction (AMI) and was admitted to the chest disease hospital as an inpatient. After one week of observation, a coronary angiogram was performed. This angiogram showed a total occlusion in proximal LAD and a SES (3.0*23 mm) was implanted [Figures 1 and 2]. During the remainder of the patient’s inpatient treatment, he was given medication including 75 mg of Clopidogrel qd and was advised to continue taking it for 6 months. He was also given 100 mg of aspirin qd and was advised to continue taking it indefinitely. Until the date of admission on October 23, 2009, he had been strictly following medical advice.

The patient was admitted into the cardiac inpatient ward on 23, October, 2009. His main complaint was a feeling of paroxysmal precordial discomfort that had lasted for about 1 month. The pain had been frequently recurring over the past 5 years.

The patient was an ex-smoker but had no other risk factors for coronary artery disease. The physical examination revealed no significant positive signs in the lungs, heart, or joints. The initial electrocardiogram (ECG) report showed a sinus rhythm, QS_{V1-V3} type, and a T_{V3-V5} inversion. The troponin T (TnT) result was negative; and the C reactive

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protein (CRP) report result was 0.64 mg/dl. A coronary angiogram was performed again on October 28, 2009, and the findings revealed the zigzag phenomenon exactly within the segment of the LAD that had been implanted with a stent in 2004 [Figure 3]. No further intervention was performed. The patient was advised to continue taking his medications, which included Clopidogrel 75 mg qd and aspirin 100 mg qd. Close long-term follow-up was requested.

DISCUSSION

CAA is characterized by an abnormal dilatation of a localized portion or diffused segment of the coronary artery tree. This is not frequently found during angiography or autopsy. CAA formation is usually reported after percutaneous transluminal coronary angioplasty (PTCA), directional coronary atherectomy (DCA), and laser angioplasty, at a frequency of 3.4% to 10% in the early stages.^[4,5] Rab *et al.* reported a 32% incidence of CAA after stent implantation when steroids and colchicine were given after the procedure.^[6] They speculated that despite the presence of the reinforcing stent, steroid-mediated impairment of vascular healing might have led to the weakening of the arterial wall and to the formation of the aneurysm. Historically, the development of CAA after stenting has rarely been reported. Recently, however, a 1.25% incidence of CAA formation after DES implantation has been reported.^[3]

Several hypotheses have been postulated to explain the mechanism of CAA development, including dissection formation, late stent malposition, hypersensitivity reactions to infectious processes, and inflammatory reactions.

Dissection formation

The use of oversized, high-pressure balloon inflation can split the intima from the media. If vascular healing was insufficient, then aneurysm formation would occur. Many reports have proven that aneurysm formation is related to the use of bailout stenting after coronary dissection due to PTCA. High-pressure balloon inflation, which is used for the complete deployment of the stent, can lead to the formation of a small dissection around the stent.^[6] Sota reported that aneurysm formation was more common in patients with a coronary dissection was higher than in patients without it (8.9% vs 4.7%).^[7] Minor dissections, which are a universal phenomenon following balloon angioplasty, usually seal off and heal on their own after the stenting procedure. The anti-proliferative action of the medication may preclude the growth of tissue intima and media. Delayed healing and weakened vessel walls can eventually lead to aneurysm.



Figure 1: Angiographic picture of 100% occlusion in proximal LAD, taken on October 21, 2004



Figure 2: Angiographic picture after DES implantation, taken on October 21, 2004



Figure 3: Angiographic picture of zigzag phenomenon exactly within stented segment, taken on October 28, 2009

Late stent malposition (LSM)

Alfonso *et al.* reported that 1,197 consecutive patients with a late angiographic evaluation after DES implantation had been analyzed.^[3] In 15 patients (1.25%), CAAs had developed by the time of the follow-up visit. All angiographic CAAs presented the IVUS definition of an

aneurysm. In all cases, the IVUS detected malaposition of the stent with a prominent distance between the DES struts and the vessel wall. This indicated that CAAs were more frequently found in patients suffering from acute myocardial infarction (6 out of 15) of whose DES had been present longer.

Hypersensitivity reaction

Virmani *et al.* demonstrated aneurysmal dilation of the stented arterial segments.^[8] The report included a severe localized hypersensitivity reaction consisting predominantly of T lymphocytes and eosinophils in a patient who eventually died of late DES thrombosis. Lack of endothelial coverage and severe DES malposition caused by aneurysmal vessel enlargement was shown. The hypersensitivity reaction was thought to have been caused by the polymer.^[8,9]

Infectious processes

Some investigators have reported exceedingly rare occurrences of mycotic CAAs after DES implantation.^[10]

Inflammatory reaction

Karas and associates reported the histo-pathological findings of the vascular responses to balloon injury and stent placement in the coronary artery in a swine model.^[11] They demonstrated that intracoronary stenting was associated with a marked inflammatory reaction around the stent wires, and the degree of intimal proliferation appeared to be greater after stenting than after balloon injury. Ivana Hollan reported that patients with inflammatory rheumatic disease had more pronounced chronic inflammatory infiltration in the media and intima than those obtained from control patients.^[12] The infiltrates might represent an inflammatory process that promoted atherosclerosis and formation of aneurysms.

For this case, the typical CAA may have been related to both rheumatic disease and DES implantation. The chronic inflammatory infiltration in the media and intima of coronary artery resulted in the rheumatic disease and DES implantation may promote the formation of aneurysms.

We suggest that DES implantation should be used with caution for patients with the history of rheumatic disease.

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