

# A case of coronary artery disease with rapid progress triggered by stent implantation

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[journals.sagepub.com/home/imr](https://journals.sagepub.com/home/imr)**Chao Feng , Liang Li and Shudong Xia**

## Abstract

Drug-eluting stents (DESs) have a low prevalence of in-stent restenosis. However, we describe a patient with coronary artery disease with rapid progress, which might have been triggered by implantation of a DES. The patient was a 72-year-old woman who was first admitted to hospital with non-ST-segment elevated myocardial infarction and had a DES implanted after coronary angiography showed severe stenosis of the left circumflex artery. However, although she kept taking dual antiplatelet therapy, her condition deteriorated and she was admitted to hospital three more times. Angiography showed that the coronary stenosis had become more severe and was more severe not just in the stent-implanted segments, but also in other coronary arteries. Another DES and drug-eluted balloon were used. However, the stent-implanted and balloon-dilated segments became severely stenosed within 1 month. Tests for auto-immune diseases and allergies were negative. We speculate that the first DES triggered an unknown response of the coronary arteries and led to severe stenosis from the stent-implanted segment to the distal segment and other arteries.

## Keywords

Coronary artery disease, drug-eluting stent, restenosis, balloon, coronary angiography, dual antiplatelet therapy

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## Introduction

Drug-eluting stents (DESs) are currently widely used for treating coronary artery disease (CAD) on the basis of their advantage of controlling restenosis over bare metal stents. For in-stent restenosis, a drug-eluting balloon and prolonged dual antiplatelet therapy can usually solve this

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problem.<sup>1-3</sup> We report here a case of CAD with rapid progress of stenosis in multiple vessels that was triggered by implantation of a DES. This condition could not be treated by further coronary intervention and medication.

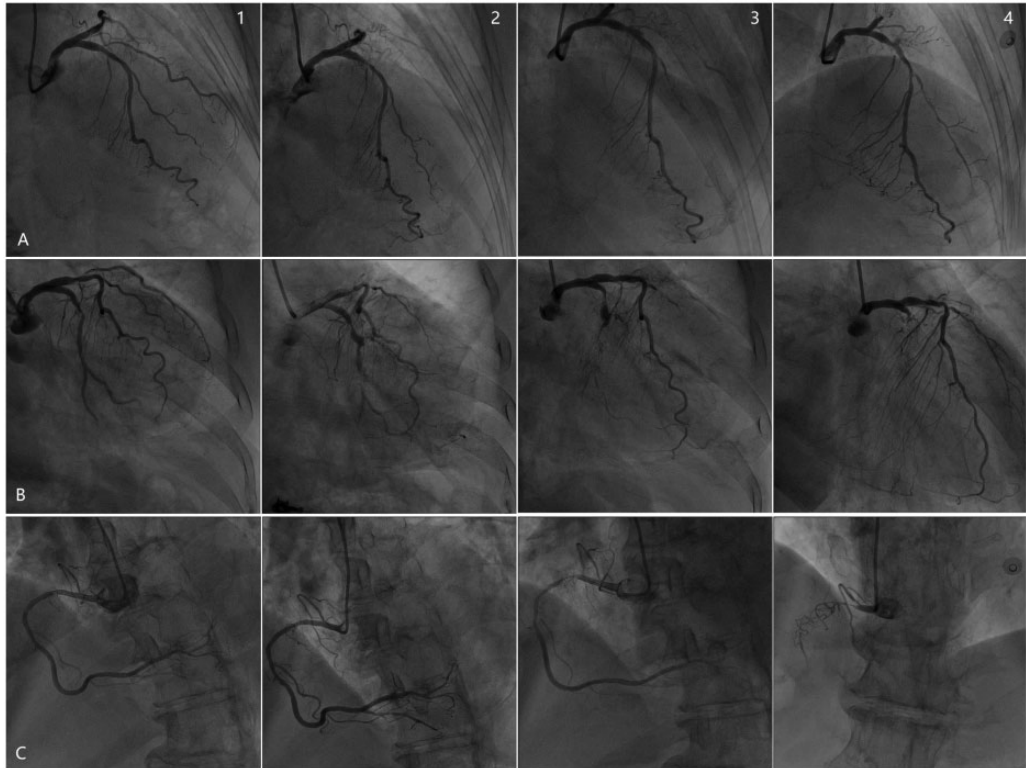
## **Case report**

A 72-year-old woman was admitted to hospital because of new onset of chest pain for 15 hours, with the first onset of pain one year previously. She had a history of hypertension for over 20 years and her blood pressure was well controlled with amlodipine. She denied any history of other chronic diseases. Her father died of stroke at 85 years and two of her siblings had hypertension. Her body mass index was 26.8 kg/m<sup>2</sup>. A physical exam showed no clinically significant signs. An electrocardiogram showed mild ST-segment depression of V2 to V5 and an abnormal Q wave in leads I and avL. The level of cardiac troponin-T was 1.49 ng/mL and the creatine kinase-MB level was 75.9 U/L. Echocardiography showed a mildly enlarged left ventricle with a normal left ventricular ejection fraction and no regional wall motion abnormality. She was diagnosed with non-ST-segment elevation myocardial infarction. She had coronary angiography performed, which showed 50% stenosis of the left anterior descending (LAD) artery, 75% stenosis of the first diagonal branch (D1), 90% stenosis of the left circumflex (LCX) artery, and 30% stenosis of the right coronary artery (RCA) (Figure 1). An everolimus-eluting stent (Everlink, 3.0 × 23 mm; Abbott, Columbus, OH, USA) was implanted into the LCX artery. After this procedure, the chest pain was obviously relieved and she was discharged three days later. She took aspirin, clopidogrel, atorvastatin, perindopril, and bisoprolol regularly.

One month later, she visited the hospital again because of recurrent chest pain for

two days. A blood test showed that the troponin-T level was 0.38 ng/mL and an electrocardiogram was similar to the previous result. Another coronary angiography was performed and showed obvious progress of coronary stenosis, with 90% stenosis of the LAD artery, total occlusion of D1, 95% in-stent restenosis of the LCX artery, and 99% stenosis of the distal LCX artery. There was also positive remodeling of the stent-implanted segment of the LCX artery, diffuse 95% stenosis of the obtuse marginal branch, and 50% stenosis of the RCA (Figure 1). Another DES was implanted into the LAD artery and the LCX artery was dilated using a paclitaxel-eluting balloon. Further examination showed that the levels of antinuclear antibody, antineutrophil cytoplasmic antibody, rheumatoid factor, complements, immunoglobulin, and C-reactive protein, and the erythrocyte sedimentation rate were within the normal limits. Ultrasound for the peripheral arteries and computed tomography for the aorta showed no obvious stenosis. Three days later, the patient was discharged with ticagrelor to replace clopidogrel.

Two months later, she was admitted to hospital again because of exertional chest pain and dyspnea. The troponin-T level was 0.24 ng/mL and the brain natriuretic peptide level was 1596.8 pg/mL. An electrocardiogram showed no obvious change and echocardiography showed an enlarged left ventricle with a left ventricular diastolic diameter of 66.9 mm, an ejection fraction of 41%, and moderate regurgitation of the mitral valve and tricuspid valve. Another angiography was performed and showed positive remodeling of the stent-implanted segment of the LAD artery with plaque ulceration, 95% in-stent restenosis of the LCX artery, and 85% stenosis of the RCA (Figure 1). Autoimmune markers were tested again and were all still negative, with no obvious stenosis identified in the



**Figure 1.** Angiography showing the progress of coronary stenosis. (A) The left anterior descending artery, (B) left circumflex artery, and (C) right coronary artery. Columns 1 to 4 show the results of four angiographies sequentially

aorta and carotid arteries. A skin patch test was conducted using an everolimus-eluting stent and was negative. A bypass surgery was recommended, but she refused and kept taking dual antiplatelet therapy together with bisoprolol, perindopril, and spironolactone.

Six months later, she was admitted to our hospital for the fourth time because of exertional dyspnea and swelling in her legs. The natriuretic peptide level was 1387.6 pg/mL and the troponin-T level was 0.016 ng/mL. An electrocardiogram showed no obvious ST abnormality. Echocardiography showed a left ventricular diastolic diameter of 65.2 mm and an ejection fraction of 40%. Coronary angiography showed 60% in-stent stenosis of

the LAD artery with positive remodeling and plaque ulceration, occlusion of D1, 80% stenosis of D2, and occlusion of the LCX artery and RCA (Figure 1). A bypass surgery was recommended, but was declined by the patient again. After several days' injection of diuretics, she demanded to be discharged.

This is a case report with no animal and human studies included. Therefore, no ethical permission was applied for according to the statements of the Ethics Committee of The Fourth Affiliated Hospital of Zhejiang University. No human studies with non-routine procedures were performed in this study. Written consent was obtained from the patient and her family members for publication of her medical data.

## Discussion

We report a case of coronary artery disease, which rapidly progressed and led to severe ischemic cardiomyopathy. Based on the risk factors, typical symptoms of angina, and the results of the first angiography, the initial stenosis was most probably caused by atherosclerosis and was managed with a DES followed by dual antiplatelet therapy. A drug carried by a stent can inhibit inflammation and smooth muscle cell proliferation, thus lowering the chance of restenosis. However, in this case, the stent did not solve the problem. After implantation of the stent, rapid stenosis progressed not only in the stented segment, but also in other coronary arteries. The cause of the rapid progress of coronary stenosis in this case was puzzling. Although in-stent restenosis is reduced after the use of a DES, it is still a major concern for cardiologists. Different from restenosis in bare metal stents, which is mainly caused by neointimal proliferation,<sup>1</sup> restenosis in DESs is characterized by development of neoatherosclerosis, which occurs in months to years after stent implantation.<sup>4,5</sup> Histologically, in-stent neoatherosclerosis is characterized by accumulation of lipid-rich foamy macrophages either in the peri-strut area or close to the luminal surface.<sup>4</sup> However, the rapid progress of coronary stenosis in our case was far more extreme than the consequence of in-stent neoatherosclerosis<sup>6</sup> because it was beyond the stented segment and involved nearly all of the coronary arteries.

We considered other possibilities beyond atherosclerosis as the causes of coronary stenosis after the second coronary angiography. These possibilities included arteritis and fibromuscular dysplasia, both of which are rare causes of systematic arterial stenosis, especially in young and middle-aged women. Takayasu arteritis typically affects the aorta and the ostial and proximal branches of the aorta, especially the

subclavian and carotid arteries.<sup>7,8</sup> Fibromuscular dysplasia is characterized by a “string-of-bead” appearance on angiography and renovascular hypertension and stroke as the typical manifestation.<sup>9</sup> However, in our case, the patient was senile and did not have any symptoms and signs of arterial stenosis, such as limb claudication, stroke, and renovascular hypertension. Additionally, all of the tests for autoimmune markers were negative and no stenosis of the peripheral arteries and aorta was identified.

We also considered the possibility of a stent allergy,<sup>10</sup> but the result of a skin test was negative. Additionally, stenosis caused by a stent allergy should be restricted to the stented segment and the other segments should be unaffected. However, we still doubted that the stent might be the cause of the rapid progress of coronary stenosis. We speculate that the stent triggered or enhanced vascular inflammation, which was undetected in this case, and led to atherosclerosis or some other vascular changes. These changes could then have rapidly progressed from the stented segment to distal non-stented arteries, and then resulted in diffuse coronary stenosis. Additionally, after restenosis of the first stent, which was identified 1 month after implantation of the stent, we used a paclitaxel-eluting balloon. Paclitaxel and everolimus that resided in the stent might have interacted, decreased the effect of anti-atherosclerosis, and contributed to progress of restenosis. Unfortunately, we did not have the opportunity to perform an intravascular study and pathological study to identify the characteristics of the stenosis. Therefore, the specific cause of the rapid progress remains undetermined.

In conclusion, we experienced a case of progressed coronary stenosis after implantation of a stent, which was intriguing, but the cause is unclear. Our findings suggest that physiological changes in coronary

stenosis and a reaction to implantation of a stent might be even more complicated than expected. Therefore, more studies on this issue are required.


### Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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