

# Stent-edge vasospasm after bare metal stent implantation: a case report and review of the literature

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## SUMMARY

It has been shown that coronary stenting is associated with long term abnormal endothelial dysfunction causing pronounced vasoconstrictor response to different stimuli. Stent edge vasospasm after coronary stenting may mimic stent edge restenosis, which may lead to an unnecessary intervention. This case report reemphasizes the importance of recognizing coronary vasospasm after stent implantation which may otherwise mislead the interventional cardiologist.

**Key words:** Endothelial dysfunction, stent implantation, vasospasm

## ÖZET

**Çıplak metal stent implantasyonundan sonra stent ucunda vazospazm: bir olgu sunumu ve literatürün gözden geçirilmesi**

Koroner stentlemenin değişik stimuluslara yanıt olarak belirgin vazokonstriktör yanıtı neden olan uzun süreli endotelial disfonksiyonla birlikte olduğu gösterilmiştir. Koroner stentlemeden sonra stent ucunda oluşan vazospazm, gereksiz girişime neden olabilecek stent ucu restenozunu taklit edebilir. Bu olgu girişimsel kardiyoloji yanlış yönlendirebilecek stent implantasyonu sonrası gelişen koroner vazospazmın farkına varılmasının önemini tekrar vurgulamaktadır.

**Anahtar kelimeler:** Endotel disfonksiyonu, stent implantasyonu, vazospazm

## Introduction

Percutaneous coronary interventions with adjunctive coronary stenting are widely used for the treatment of coronary artery disease. It has been shown that coronary stenting is associated with abnormal endothelial dysfunction, which may persist up to 6 months (1). Coronary spasm has been reported to complicate 1-5% of balloon angioplasty procedures (2). However the exact incidence of spasm associated with coronary stenting is not clear. In this case we illustrate a stent edge spasm, which may be mistaken for stent edge stenosis and lead to an unnecessary intervention.

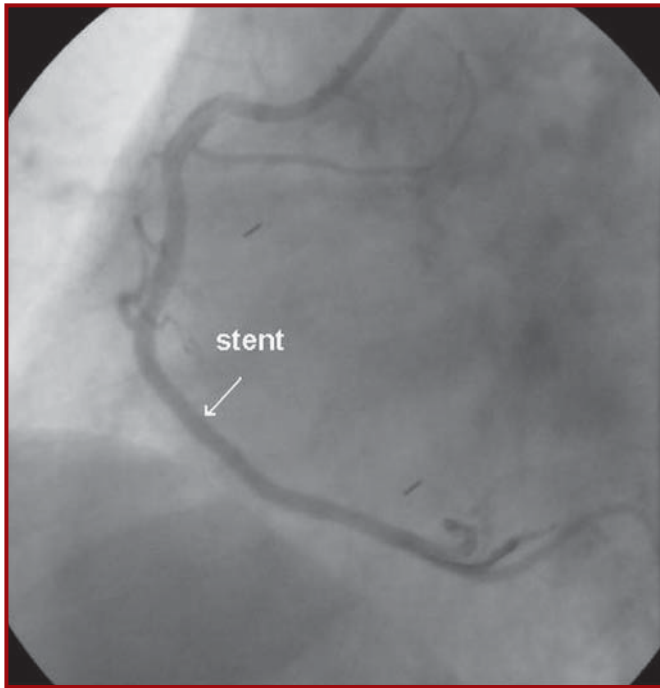
## Case Report

A 63-year-old male patient was admitted to the cardiac catheterization laboratory for patency control of a bare metal stent (BMS), which was implanted 13 months ago in his right coronary artery. A BMS with a size of 3.0x18 mm had successfully been implanted without predilation. The patient was suffering from stable angina pectoris till the stent implantation and, after the procedure he was almost completely asymptomatic except for sudden short attacks of anginal chest pain. He quitted smoking just after the stent implantation. His LDL level was 127 mg/dl and had no other risk factors for coronary artery disease. He was taking acetyl salisilic acid 300 mg/day, clopidogrel 75 mg/day and atorvastatin 20 mg/day. The coronary angiography revealed severe stenosis at the distal edge of the stent (Figures 1,2). Therefore, we decided to perform an angioplasty and subsequently a BMS implantation. However after we engaged the coronary artery with a guiding catheter, we noticed that the lesion surprisingly disappeared (Figure 3). We added a long acting nitrate and a calcium channel antagonist to his current medication. The patient is asymptomatic since this treatment.

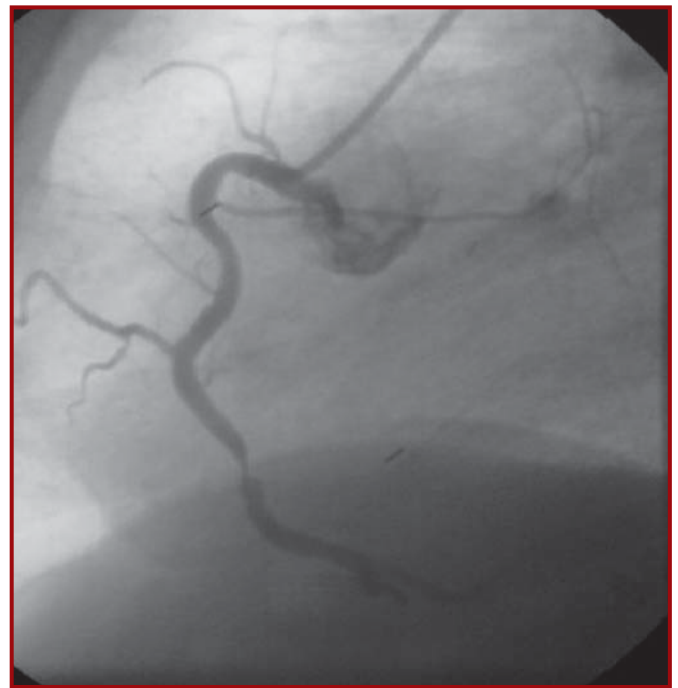
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**Figure 1.** Left anterior oblique view demonstrating distal stent edge vasospasm



**Figure 2.** Left anterior oblique view demonstrating distal stent edge vasospasm

### Discussion

This case illustrates that stent edge spasm can mislead the invasive cardiologist to consider it as in-segment restenosis. There is ongoing debate about the incidence, cause and clinical importance of coronary artery spasm after percutaneous coronary interventions. There are reports of stent edge spasm with both BMS and drug eluting stents and aborted sudden death associated with coronary spasm soon after drug elut-

ing stent implantation (3,4). The exact mechanism of spasm induced by stenting is not known. Coronary stenting appears to cause arterial injury and inflammatory response within the vessel wall (5). Caramori et al. have found persistent endothelial dysfunction in the intervened coronary artery several months after the intervention (1). More severe endothelium-dependent vasoconstriction was observed in stented patients than in patients who had undergone either balloon angioplasty or directional atherectomy. In contrast to that finding, Togni and coworkers have reported that the implantation of sirolimus eluting stent is associated with endothelial dysfunction in proximal and distal regions of treated coronary segment, whereas in the control group, BMS did not affect physiologic response to exercise (6). Toxic effects of the sirolimus and paclitaxel to endothelial cells cause delayed vascular healing, which may lead to more severe and prolonged endothelial dysfunction.

Although clinical relevance of these findings are unknown, reported cases of life-threatening coronary spasms and thrombosis with both drug eluting and BMS should be taken into account when performing an coronary intervention. When evaluating a stent edge stenosis, coronary vasospasm should also be kept in mind to prevent any further unnecessary intervention. This case illustrates such a vasospastic lesion. The observation that coronary stent implantation is associated with adverse consequences on vascular endothelium other than restenosis warrants further studies.



**Figure 3.** The relief of stent edge vasospasm several minutes later

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