

CASE REPORT

Acute myocardial infarction caused by thrombotic occlusion at a stent site two years after conventional stent implantation

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Two cases of acute myocardial infarction caused by thrombotic occlusion at the conventional stented site two years after stenting are described. Late thrombotic stent occlusion may be caused by atherosclerotic regression, sustained inflammatory reaction, and inhibition of proliferation of neointima. Cardiologists must be aware of the potential for late thrombosis following even conventional stent implantation.

Percutaneous coronary stenting is an innovative technique that has been highly effective for reducing the incidence of restenosis.¹ One continuing problem, however, is thrombotic stent occlusion.² In many cases, acute and subacute thrombosis occurs within 14 days after stenting.³ In addition, late thrombosis after brachytherapy or the implantation of a drug eluting stent has been reported.^{4,5} We experienced two cases in which stent thrombosis developed two years after implantation of conventional stents.

CASE REPORT 1

A 38 year old woman was referred to our university hospital because of acute myocardial infarction. Coronary angiography showed complete occlusion of the proximal left anterior descending artery where a stent had been placed 27 months earlier to treat her angina pectoris (fig 1A). Notably, angiography performed on day 198 after stent placement had shown no restenosis at the stented site and ticlopidine had been withdrawn; aspirin was continued. Angioscopy performed before coronary revascularisation showed an exposed stent strut and massive white and mixed thrombi in the culprit lesion (fig 1B). Intravascular ultrasound imaging showed gaps between the stent struts and the vessel wall (fig 1C). Thrombi were aspirated from the thrombotic occlusion area in the left anterior descending artery through a thrombosuction catheter (RESCUE catheter, Boston Scientific, Watertown, Massachusetts, USA). Then the area was expanded with a balloon and successfully reperfused (fig 1D).

CASE REPORT 2

In a second case, coronary angiography in a 57 year old woman referred to our hospital with acute myocardial infarction showed complete occlusion of a mid right coronary artery segment where a stent had been placed 31 months earlier (fig 2A). The patient had a history of rheumatoid arthritis, hypertension, diabetes mellitus, and a healed myocardial infarction. Angiography on day 101 after the placement of the stent had shown no restenosis at the stented site. From then on she received no antiplatelet agent, although an adrenocortical steroid (betamethasone) was continued for treatment of her rheumatoid arthritis.

Angioscopy performed before coronary revascularisation showed exposed stent struts and a substantial amount of mixed thrombus in the culprit lesion (fig 2B). Intravascular ultrasound showed incomplete adhesion of the stent to the vessel wall (fig 2C). A new stent was partially superposed on the previous stent in the thrombotic occlusion area of the right coronary artery segment and the culprit coronary artery was successfully reperfused (fig 2D).

DISCUSSION

There have been numerous studies of early acute or subacute thrombotic complications after stent implantation.² With the exception of thrombosis after brachytherapy or implantation of drug eluting stents, however, less attention has been paid to late stent thrombosis, as clinical studies have shown that thrombotic occlusion of conventional stents develops within 14 days after stenting but rarely thereafter.³ Nevertheless, we encountered two such cases.

For over three years, Kimura and colleagues⁶ followed up with coronary angiography patients who had undergone stent implantation.⁶ They found that late improvement in luminal diameter occurred between six months and three years after stent implantation. Similarly, Asakura and colleagues⁷ used angioscopy to investigate neointimal coverage of stents and observed that the neointima became thicker for six months after stent implantation and then thinned, becoming transparent within three years. Histological findings showed this regression to be characterised by atrophic changes in smooth muscle cells, decreased cell density, and fibrotic maturation of intimal hyperplasia.^{8,9} It has also been suggested that apoptosis may contribute to such neointimal thinning.¹⁰

Coronary occlusion in the two patients described here may have been caused by regression of the neointima covering their stents, as thrombus formation is a likely response to exposure to a metallic foreign body. Moreover, in the second patient, who was taking an adrenocortical steroid for the treatment of rheumatoid arthritis, the occlusion may have developed as a result of attenuated healing, which is a known side effect of steroid treatment and may lead to excessive inhibition of neointimal proliferation. It thus appears that long term follow up after stent implantation, keeping in mind the possibility of late stent thrombosis, is necessary even with conventional stent implantation.

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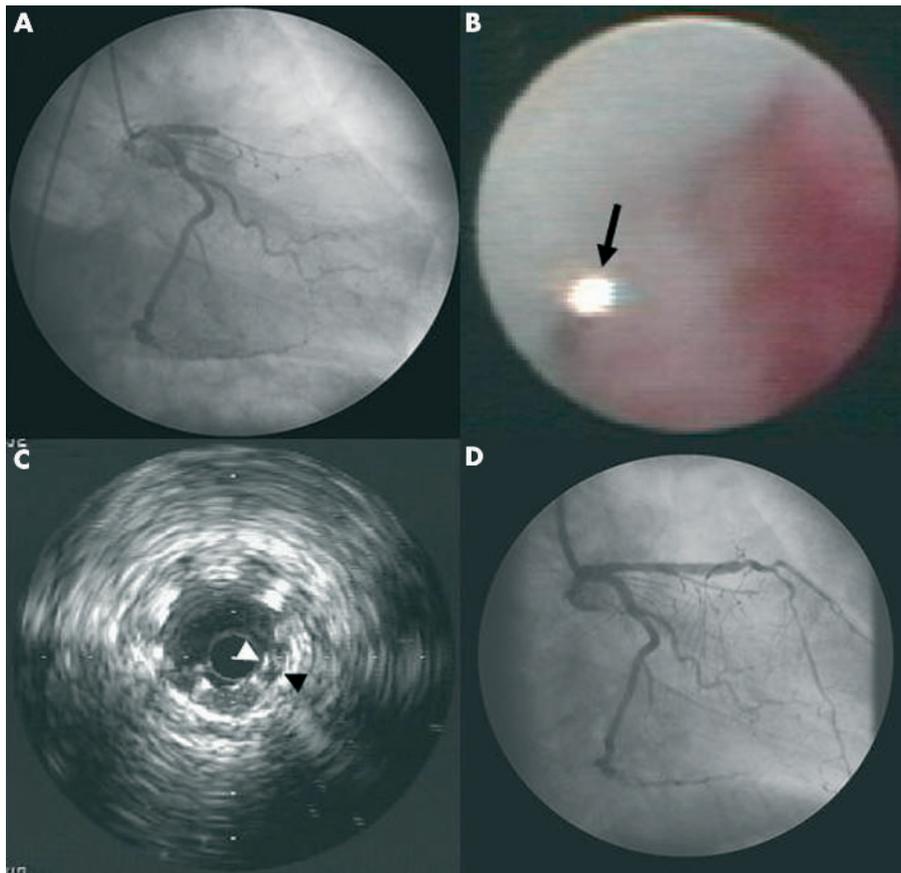


Figure 1 (A) Coronary angiogram showing complete occlusion in the proximal left anterior descending artery segment, where a stent had been placed 27 months earlier. (B) A stent strut (black arrow) and a substantial amount of white and mixed thrombi can be seen in another angioscopic image. (C) Intravascular sonography shows the presence of gaps between stent struts (white arrowhead) and the vessel wall (black arrowhead). (D) Conventional balloon angioplasty successfully opened the occluded lesion.

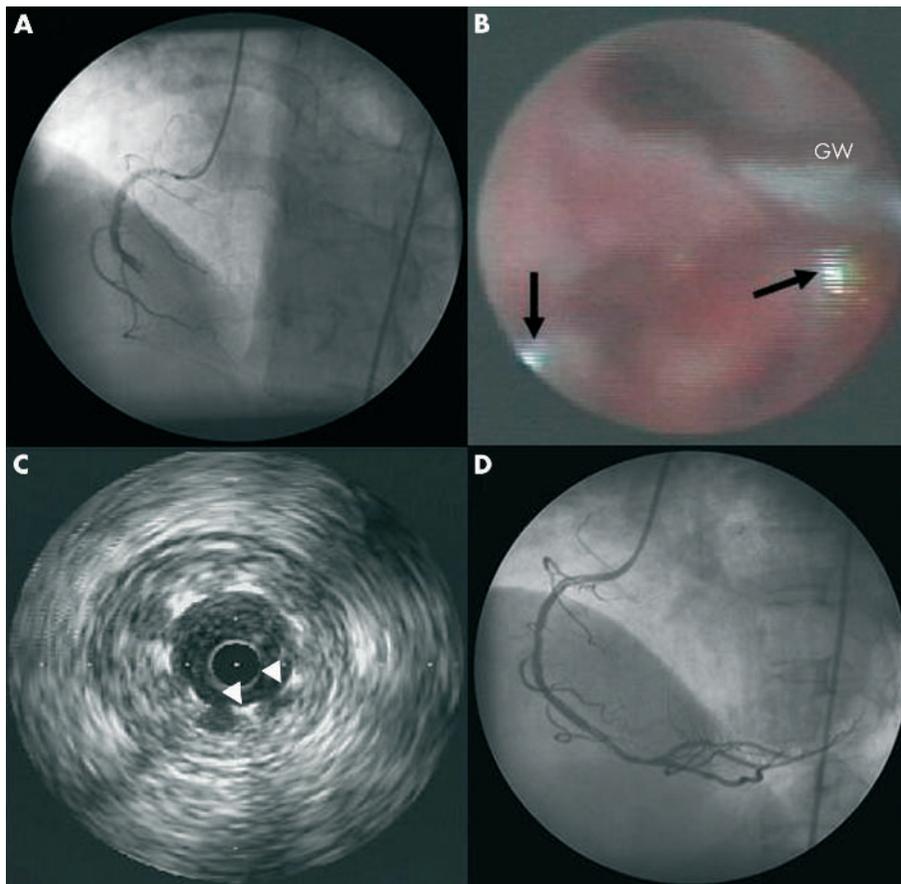


Figure 2 (A) Coronary angiogram showing a right coronary artery occluded in the mid segment, where a stent had been placed 31 months earlier. (B) Stent struts (black arrows) and a substantial amount of mixed thrombus can be seen in another angioscopic image. GW, guidewire. (C) Intravascular sonography shows a malapposed stent (white arrowheads) in the culprit lesion. (D) The culprit coronary artery was successfully reperfused with a new stent superposed on the previous stent.

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