Coronary artery spasm: mimicry, misdiagnosis and misfortune

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DESCRIPTION

A 51-year-old man with a history of ischaemic heart disease (IHD), previous percutaneous coronary intervention (PCI) and rheumatoid disease, presented with chest pain and inferoposterior ST segment elevation in August 2013. He had continued to smoke after his diagnosis of IHD and PCI.

In 2006, he presented with an acute coronary syndrome (ACS) and had a significant left anterior descending artery (LAD) stenosis, which was treated with one stent (figure 1A, B). He presented again in 2007 with in-stent restenosis, and a second stent was implanted (figure 1C, D). In April 2013, he had an emergency presentation

Figure 1 Coronary angiogram showing significant stenosis of the proximal portion of the left anterior descending (LAD) at initial presentation in 2006 with ST elevation myocardial infarction (A) and, in 2007, with in-stent restenosis (C), both showing good angiographic results after stent implantation (B and D). In-stent thrombosis in early 2013 showing completely occluded LAD (E), again treated with another stent implantation (F). Left coronary artery in the most recent angiogram from August 2013 showing a widely patent stent but widespread spasm in other vessel including the branches (G), which returned to normal calibre after intracoronary nitrate (H).

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with stent thrombosis and a third stent was implanted in the LAD (figure 1E, F).

When the patient presented again in August 2013, coronary angiography revealed widely patent proximal stents, but the remainder of the LAD and all of its branches were in spasm (figure 1G).

The images from the previous interventions were reviewed; the calibre of the coronary arteries at the end of the procedures had dramatically increased compared with baseline (figure 1B, D, F). A diagnosis of coronary artery spasm was made and intra-coronary nitrate administered. The vasospasm resolved and the arteries returned to their full size with no intervention required (figure 1G).

Review of the patient's medication history revealed that in 2013, following stent thrombosis, his regular calcium channel blocker was stopped and substituted with a β -blocker. He was restarted on a vasodilating calcium channel blocker during this admission and to date no further problems have ensued.

Focal coronary artery spasm due to coronary artery smooth muscle hyperactivity has been well described, especially following delivery of a drug-eluting stent.¹ A more diffuse spasm in all the epicardial arteries is less common and likely to be a consequence of diffuse endothelial dysfunction from several factors,² in this case smoking and rheumatoid arthritis.

A diffuse spasm in all the epicardial arteries is less common than focal coronary artery spasm, and is likely to be a consequence of diffuse endothelial dysfunction from several factors,¹ in this case smoking and rheumatoid arthritis. Although β -blockers are used in coronary artery spasm, their use can be detrimental,² so early recognition of this phenomenon is important to ensure appropriate treatment. Calcium channel blockers remain the mainstay in the treatment of coronary artery spasm.³

Learning points

- ▶ Coronary artery spasm can mimic coronary artery stenosis due to atherosclerotic plaque, and may result in unnecessary stent implantation.
- ▶ Endothelial dysfunction, in this case a pre-existing rheumatoid disease, is likely the pathophysiology of the widespread coronary artery spasm despite stent implantation in the affected segment at initial presentation.
- ▶ Calcium channel blockers should be continued in patients with previous history of diffuse coronary artery spasm.

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Patient consent Obtained.

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