CASE REPORT

Catastrophic catheter-induced coronary artery vasospasm successfully rescued using intravascular ultrasound imaging guidance

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► Additional material for this paper are available online. To view these files, please visit the journal (<http://dx.doi.org/10.1136/bcr-2017-222607>).

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Accepted 28 November 2017

SUMMARY

A 46-year-old man underwent coronary angiography for stable angina. He developed inferior ST-segment myocardial infarction during the angiography. Intravascular ultrasound (IVUS) findings suggested coronary vasospasm. Intracoronary administration of isosorbide dinitrate restored the coronary flow. This case illustrates the essential role IVUS imaging played in establishing the diagnosis of catheter-induced coronary vasospasm.

BACKGROUND

We report a rare case of severe catheter-induced coronary spasm that led to cardiogenic shock. Coronary artery spasm was first introduced by Prinzmetal *et al*¹ and later defined as a sudden intense vasoconstriction of an epicardial coronary artery leading to occlusion.² Among patients with active spasm, an episode often occurs during coronary arteriography. However, frequent coexistence of atherosclerotic coronary artery disease and coronary spasm makes the diagnosis more difficult. Moreover, it should be noted that misidentification of coronary artery spasm can lead to unnecessary or inappropriate percutaneous coronary intervention.³ Furthermore, its impact cannot be underestimated, especially since left main coronary spasm has been reported to occur in 20% of those who underwent

coronary artery bypass grafting and repeat coronary angiography in a recent report by Ilia *et al*.⁴ Therefore, our case makes a significant contribution to the literature because it shows how this disease may be recognised and diagnosed.

CASE PRESENTATION

A 46-year-old man with a medical history of active smoking and Hashimoto's thyroiditis was admitted for Canadian Cardiovascular Society Class 3 stable angina. There was no family history of heart disease. During the 3 weeks prior to admission, the patient had been experiencing chest discomfort radiating to his neck. He had denied habitual use of any kinds of drug, including cocaine.

INVESTIGATIONS

Although the transthoracic echocardiography showed normal left ventricular systolic function, the radionuclide stress test showed evidence of ischaemia of the inferior wall. Dual antiplatelet therapy, consisting of aspirin and clopidogrel, together with rosuvastatin was administered on the day of admission. At admission, his blood pressure was 112/62 mm Hg, the pulse was 60 beats per minute and regular, and the respiratory rate was 16 breaths per minute. Baseline angiography, after intra-arterial administration of 5000 U of heparin, showed a dilated right coronary artery (RCA) with no stenosis (figure 1), and we moved on to imaging the left coronary artery. Towards the end of the left coronary angiogram, the patient complained of severe chest pain, followed by bradycardia (heart rate of 36 beats per minute) and a sudden drop in blood pressure (the systolic pressure had dropped to 60 mm Hg). An ECG demonstrated inferior ST-segment elevation with reciprocal anterolateral ST-segment depressions, suggesting acute inferior myocardial infarction, after which led to ventricular tachycardia (figure 2A). We immediately proceeded to perform right coronary angiography again. To our surprise, the RCA, which had appeared normal on the previous angiogram, was completely occluded at the ostium (figure 2B).

DIFFERENTIAL DIAGNOSIS

After inserting the guiding catheter and wire, intravascular ultrasound (IVUS) imaging was performed to rule out coronary dissection. Although IVUS showed no sign of coronary dissection, luminal

Figure 1 Initial angiography of the right coronary artery.

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To cite: Kodaira M, Tabei R, Kuno T, *et al*. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-222607

Figure 2 ECG and coronary angiography after development of chest pain. ECG recorded after the patient experienced chest pain (A). ST-segment elevation was noted in leads II, III, arteriovenous fistula (aVF) (yellow arrows). Repeated angiography of the right coronary (white arrow) artery after appearance of symptoms and haemodynamic collapse (B).

narrowing at the spastic site and positive remodelling at the distal site were observed (figure 3A, B and online supplementary video). Medial thickening was observed together with luminal compromise at the occluded site.

TREATMENT

From these findings, we suspected coronary vasospasm and administered 1 mg of isosorbide dinitrate into the RCA.

OUTCOME AND FOLLOW-UP

This immediately improved the coronary flow and haemodynamic status (figure 4A). Repeat IVUS imaging revealed

Figure 3 Images after development of chest pain. Intravascular ultrasound imaging of the occluded right coronary artery at the vasospastic lesion (A) and the distal lesion showing positive remodelling (B). Yellow arrows point to the medial thickening.

dilatation of the RCA (figure 4B, C). Before leaving the catheter lab, his blood pressure had recovered to 123/76 mm Hg, and his heart rate to 56 beats per minute. The patient was discharged on isosorbide mononitrate and benidipine hydrochloride. At 6 months' follow-up, he was in good condition without symptoms.

DISCUSSION

Catheter-induced coronary artery vasospasm has been reported previously.^{5 6} Although the RCA is the most common site, it has also been documented in the left main coronary artery.⁴ Its incidence varies greatly in past reports (0.26%–3%),^{7 8} but it is not infrequent. However, complete occlusion causing acute myocardial infarction and cardiogenic shock, as observed in our patient, is rare. Although provocation testing using acetylcholine has been recognised as the standard diagnostic test for coronary spastic angina,⁹ we considered it exceedingly risky in this case. Therefore, we reached the diagnosis of coronary spastic angina from the initial angiogram, which showed no stenosis, and his typical symptoms. IVUS imaging also showed positive remodelling at the distal coronary artery⁵ and intimal thickening,¹⁰ both of which have been linked to vasospastic angina. The two mechanisms that underlie coronary artery spasm are endothelial dysfunction^{11–13} and hyperactivity of vascular smooth muscle cells,^{14–16} both of which are also responsible for catheter-induced coronary artery vasospasm.¹⁷ The specific triggers responsible for catheter-induced coronary spasm include a mechanical irritation-induced myogenic reflex,^{18 19} stress receptor activation²⁰

Figure 4 Results after restoration of the coronary flow. Angiography of the right coronary artery just after administration of isosorbide dinitrate (A). Intravascular ultrasound imaging of the right coronary artery after restoring flow showing the vasospastic lesion being dilated (B), and the distal lesion showing more remarkable signs of positive remodelling (C).

and platelet aggregation at the catheter tip after release of vasoactive agents.²¹ The patient in this report was a cigarette smoker, which is a clinical risk factor present in 75% of all patients with coronary spasm.²² Smoking has been reported to cause oxidative modification of vascular smooth muscle cells, leading to spasm.²³ Therefore, in the present case, hyper-reactivity of the coronary artery to a mechanical stimulus may have exacerbated the coronary spasm and caused this extraordinary event.

Nitrates play a major role in the treatment of angina. The end-product of nitrate metabolism is nitric oxide, which stimulates the enzyme guanylate cyclase to catalyse the production of cyclic guanosine monophosphate. The ultimate effect of nitrates is dependent on their dose: very low doses cause venodilation, low to moderate doses cause arterial dilation and higher doses lead to arteriolar dilation.²⁴ It is noteworthy that simple administration of isosorbide dinitrate was sufficient to resolve the occlusion in our case. Administration of isosorbide dinitrate or nitroglycerine could have been a primary option immediately after recognition of the occlusion of the RCA since routine use of these vasodilators during coronary angiography is recommended.³ Intracoronary administration of nitroglycerine has been proven to promptly reverse spasm, whereas the effects of systemic nitroglycerine emerge only secondary to reduction in left ventricular wall tension and pressure.²⁵ However, even local administration of nitroglycerine may also have systemic effects leading to hypotension,³ which was a major concern in our haemodynamically unstable patient. Thus, we hesitated to immediately administer vasodilators without confirmation of the diagnosis. IVUS was chosen as the imaging modality to clarify the nature of RCA occlusion. Various studies have proven that IVUS has the potential to characterise plaque type and assess the severity of stenosis.^{26–28} This case demonstrates the role of IVUS imaging, which served to guide us in the diagnosis of coronary spasm and to avoid unnecessary stenting. To the best of our knowledge, this is the first report of IVUS images of a completely occluded vasospastic coronary artery.

Learning points

- We present our management strategy in a patient who suffered from severe right coronary artery occlusion leading to acute myocardial infarction.
- The results of the intravascular ultrasound imaging suggested that we should avoid unnecessary stenting and simply administer isosorbide dinitrate to restore the coronary flow.
- Physicians should be aware of catheter-induced coronary artery spasms, as it remains a problem that can be faced by every cardiologist who performs coronary angiography.

Contributors All the authors contributed in patient care. MK, the corresponding author, was the primary surgeon and drafted the manuscript. YN was the assistant surgeon, who supervised the procedure, critically revised the manuscript to ensure the important intellectual content was appropriate, and provided the final approval. RT and TK also critically revised the manuscript for the important intellectual content.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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