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Acute stent thrombosis due to stent underexpansion managed with rotational atherectomy☆,☆☆

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ABSTRACT

Stent underexpansion is a well known risk factor for stent thrombosis. We report a case of acute stent thrombosis which occurred after primary percutaneous coronary intervention to the right coronary artery for an inferior ST-elevation myocardial infarction. Minutes after completion of the procedure, the patient had acute stent thrombosis, manifested by new chest pain and new ST-elevation. IVUS showed the stent to be underexpanded. Despite high pressure balloon inflation, the stent remained underexpanded. This was then managed with rotational atherectomy within the underexpanded stent, followed by high-pressure balloon dilatation and deployment of another stent within it.

1. Introduction

Stent underexpansion is a well recognized risk factor for stent thrombosis [1,2]. Chronic hard and calcified lesions can prevent optimal stent expansion. However with advent of rotational atherectomy, cutting balloons, non-compliant balloons and increasing skill of operators, such lesions can now be treated. Difficulty however may arise on occasions when such lesions are overlooked. In other instances, the operator may not have enough time to prepare the lesion adequately before deploying the stent in an unstable patient with acute coronary syndrome (ACS). Underprepared lesions in such cases can lead in sub-optimal stent expansion and mal-apposition. We describe a case of stent underexpansion in a patient who had presented with an acute inferior ST-elevation myocardial infarction (STEMI). He developed acute stent thrombosis even as he was being shifted out of the catheterization lab. This case report highlights some of the technical challenges of dealing with such a clinical scenario.

2. Case report

A 64 year-old gentleman presented to our emergency department with acute inferior STEMI. He had hypertension, dyslipidemia and was known to have coronary artery disease having undergone coronary stenting in the past. On presentation, the patient had a systolic blood pressure of 90 mmHg, albeit without clinical signs of hypoperfusion. Electrocardiogram (ECG) revealed inferior STEMI with evidence of right ventricular infarction. The patient was already on aspirin and was loaded with ticagrelor in the emergency room. He was transferred to the cardiac catheterization laboratory for primary angioplasty. Right femoral arterial access was obtained with a 6 French (Fr) sheath. Coronary angiography revealed 50% in-stent restenosis in the proximal left anterior descending (LAD) stent with another 50% lesion in mid LAD, and a 40% lesion in the circumflex artery. Septal collaterals were noted from LAD to the right posterior descending artery (PDA). The right coronary artery (RCA) was found to have an acute thrombotic occlusion in the proximal segment (Fig. 1). Primary percutaneous coronary intervention (PCI) was performed. Intravenous heparin to a target ACT of >250 s was given. The RCA was engaged with a 6Fr Judkin's right 4 guide catheter. A BMW Universal 0.014" (Abbott Vascular, Illinois, USA) guidewire was manoeuvred across the thrombotic lesion without much difficulty. Thrombus aspiration with 6Fr Thrombuster II (Kaneka, Tokyo, Japan) was performed after which some antegrade flow was seen. The lesion was then predilated with Ottimo-Ex (Kaneka, Tokyo, Japan) 2.0 mm balloon. Despite inflating at a pressure of 20 atm, a small waist persisted. However considering the borderline hemodynamics and the clinical scenario, it was decided to stent the lesion. A bare metal stent (BMS), Azule (Orbus Neich, Wanchai, Hong Kong), 3.0×18 mm was deployed and post dilated with a non-compliant Fortis (Kaneka, Tokyo, Japan) 3.0×18 mm balloon. A 20% focal residual stenosis persisted despite further post-dilations with 4.0×12 mm non-compliant balloon Quantum Apex (Boston Scientific, Marlborough, Massachusetts) (Fig. 2). TIMI 3 flow was achieved. The result was felt to be acceptable and the guide catheter was removed. At the end of the procedure, the operator unscrubbed but noted that on the monitor that the ST segments appeared more elevated than before. The patient

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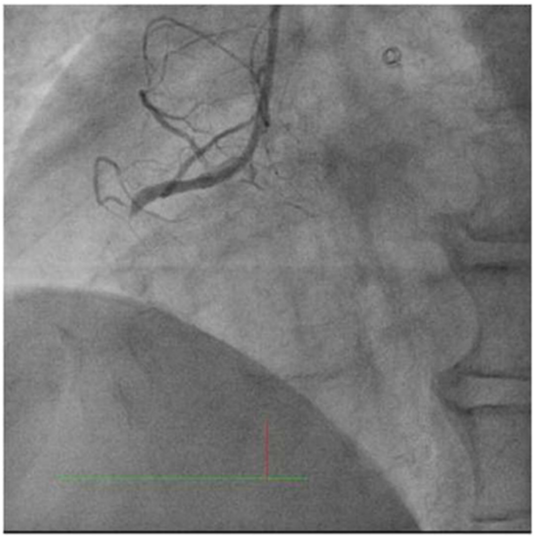

Fig. 1. Occluded proximal RCA on presentation.

also complained that the chest pain had worsened. A 12-lead ECG was repeated on table which showed ST segment elevation in the anterior leads (Fig. 3). Repeat coronary angiography was performed immediately. The LAD and circumflex arteries remained unchanged. However collaterals from the septal branches of the LAD to the PDA were noted, which was unexpected considering that the patency of RCA had been recently restored. Angiography of the RCA revealed a hazy thrombus within the just implanted stent causing significant stenosis although antegrade flow was present (Fig. 4). Thrombus aspiration was repeated, with aspiration of small amounts of white thrombus. Intracoronary Eptifibatide boluses (2 doses of 180 mcg/kg given 10 min apart) followed by infusion therapy according to standard guidelines were given. Intravascular ultrasound (IVUS) with Volcano Eagle Eye (Volcano, San Diego, California, USA) excluded the possibility of edge dissection. Further dense eccentric calcification in a focal area in the mid RCA overlying the underexpanded stent was noted. Incomplete expansion was thought to be the cause of acute stent thrombosis (Fig. 5). The minimum lumen diameter was 2.8 mm with a minimum luminal area of 7.5 mm^2 (reference minimum luminal diameter was 3.6 mm with a minimum

Fig. 2. Residual 20% defect (arrow) even after multiple high pressure post dilations.

luminal area of 10.9 mm^2). High pressure dilation (up to 24 atm) within the stent was done with a non-compliant 4.0 mm balloon, cutting balloon Flextome 3.0 × 10 mm (Boston Scientific, Marlborough, Massachusetts) and scoring balloon Angiosculpt 4.0 × 10 mm (Fremont, California, USA) but the waist remained. It was then decided to perform rotational atherectomy within the stent. A 1.5 mm burr was used over 0.009" Rota extra support wire for rotational atherectomy at 150,000 rpm (Fig. 6). We considered upsizing femoral access to 7 F and exchanging for a larger system in order to use larger burr sizes but decided to repeat balloon dilation first. With further post-dilations were with the Angiosculpt 4.0 × 10 mm and Quantum Apex 4.0 × 8 mm non-compliant balloons there was an improvement of the stenosis. A Prokinetic Energy (Biotronic, Berlin, Deutschland) 3.5 × 40 mm BMS was deployed covering the previous stent and post-dilated with the 4.0 × 00208 mm non-compliant balloon at 22 atm (Fig. 7). IVUS showed a good stent apposition and better expansion although there was still a 5% residual stenosis (Fig. 8), with a minimum luminal diameter of 3.3 mm and a minimum luminal area of 9.5 mm^2. The result was felt to be acceptable. The patient was discharged well after 5 days and has since returned well for a 6-month visit.

3. Discussion

Rotational atherectomy within an underexpanded stent has been reported in literature, as summarized in Table 1 [3–7]. However, this is the first report where suboptimal expansion of a stent resulted in acute stent thrombosis in a STEMI setting which was managed by rotablation within the underexpanded stent. Although the acute stent thrombosis may also be related to the inadequate antiplatelet inhibition, this was felt to be less likely given the presence of an underexpanded stent.

Our case illustrates the technical challenges of addressing stent underexpansion resulting in stent thrombosis. If the stent underexpansion had not been 'fixed', then the patient may have possibly suffered from another stent thrombosis. In this case, despite aggressive post-dilatation, we could not adequately expand the stent. Laser atherectomy [8] has been described in the literature as a possible treatment option for underexpanded stents, but this was not available in our centre. Our only real remaining option was rotational atherectomy. However, rotablation within a stent does carry significant risks such as burr-entrapment. We discuss some important technical considerations that may lessen the risks. First, the operator needs to consider the appropriate burr size. As in de novo lesions, the burr size would be determined by the vessel size with a burr/artery ratio approximately 0.7 [9] and the degree of underexpansion. In 3 cases, the authors started with a smaller burr but upsized it to achieve a better polishing. The final burr should be large enough to ablate the stent struts. Indeed, we would have considered using a larger burr had we not obtained satisfactory expansion with balloon dilation after rotablation with the 1.5 mm burr. Like Kobayashi and colleagues [6], we used an extra support rota wire. In our opinion, the greater wire bias with the extra-support wire may allow more contact with the stenotic area for ablation. However, it is likely that wire selection may simply be one of operator preference. Second, it remains unclear if a cutting or sculpting balloon after rotablation will be useful. Cutting balloons are technically more useful treating fibrotic lesions instead of calcified lesions. As the lesion did not budge with a regular non-compliant balloon, even at high pressures, the operator felt that a cutting or sculpting balloon may provide additional radial strength against the stented calcific lesion. A third consideration is whether or not to stent over the original stent. This would depend on the angiographic and IVUS findings. If there is adequate expansion with minimal recoil, no damage to the implanted stent and no edge dissection, then post rotablation high pressure balloon dilation may be accepted without deploying another stent as in the previous reports [5–7]. However some operators may not be comfortable with not stenting after rotablation as the structural integrity of the initial stent may be compromised. Fourth, adjunctive imaging such as with IVUS