Coronary perforation due to sirolimus-eluting stent's strut rupture with post-dilatation

Perforacja tętnicy wieńcowej podczas redylatacji stentu uwalniającego sirolimus

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Abstract

Coronary artery perforation is a rare and dangerous complication of percutaneous coronary intervention. Its consequences may range from minimal dye staining of the pericardial cavity to haemodynamic collapse, and it requires urgent treatment. Nearly half of cases occur due to the use of stiff and hydrophilic guidewires. Perforations due to stent deployment or strut rupture are seen more rarely, but have a more serious prognosis. We present a case of coronary perforation due to rupture of a sirolimus-eluting stent's strut. The perforated site was occluded immediately with the implantion of two consecutive bare metal stents.

Key words: perforation, sirolimus-eluting stent, strut rupture

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INTRODUCTION

Coronary artery perforation (CP) is a rare and potentially lifethreatening complication of percutaneous coronary intervention (PCI). It can lead to abrupt haemodynamic collapse. So, it requires rapid detection, classification and treatment [1]. Here we presented a case of coronary perforation due to rupture of a sirolimus-eluting stent's strut. The perforated site was occluded immediately with the implantion of two consecutive bare metal stents (BMS).

CASE REPORT

A 60 year-old female patient was admitted to our cardiac centre complaining of recurrent anginal attacks. She was diagnosed with an anterior myocardial infarction three days prior to admission and thrombolytic treatment (t-pa) was applied in the second hour of chest pain. As for the medical background of the patient, she was on medication for hypertension, dyslipidaemia and impaired glucose tolerance. She was overweight, with a body mass index of 29 kg/m². Her electrocardiography showed precordial inverted T-waves with an absence of R progression. Echocardiography revealed anterior and apical hypokinesis with a 45% left ventricular ejection fraction. After initial evaluation, coronary angiography was performed. The left anterior descending artery (LAD) was diffusely diseased at the mid-portion with multiple critical stenosis, and 75% stenosis was also obtained in the proximal circumflex artery (Fig. 1A, B). Surgical treatment was proposed for the patient.

However, the patient refused surgery and interventional therapy was scheduled. A stiff-tipped floppy guidewire was preferred for LAD intervention. The LAD lesions were predilated with a 2.0–20 mm balloon (Fig. 1C). Then, 2.5 \times 28 mm and 3.0 \times 33 mm sirolimus-eluting stents were implanted sequentially using the overlapped technique, the edges of both stents overlapping by aproximately 2–3 mm. Residual 30–35% stenosis was obtained in the distally placed stent (Fig. 2). After

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Figure 1. Long segment left anterior descending artery (LAD) lesion from left and right anterior oblique projections (A, B). Appearance of LAD lesion after predilatation with 2.0 × 20 mm balloon (C)

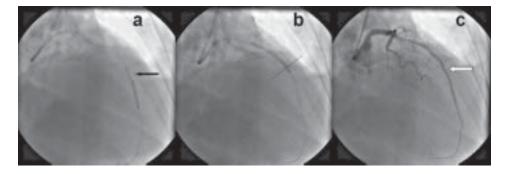


Figure 2. Sequential implantation of two sirolimus-eluting stents in an overlapped manner (**A**, **B**). Black arrow indicates residual stenosis in stent placed distally (**A**). Upper edge of distal stent is marked with black line, clearly revealing overlapping (**B**). Section **C** shows view of left anterior descending artery after implantation of stents, and white arrow indicates residual stenotic segment

that, residual in-stent stenosis was dilated with a 3.0×20 mm non-compliant balloon at high pressure. During the peak state of dilatation, a small notch appeared in the distal part of the balloon and it was extended outward through the struts of the stent. A further injection revealed Type 3 perforation of the vessel from just beneath the overlapped region, which had developed due to rupture of the stent's strut (Fig. 3).

The patient had defined chest pain immediately post--dilatation. Due to the unavailability of an appropriately sized graft stent, a 3.0×18 mm cobalt chromium BMS (driver) with relatively narrow struts was implanted over the perforated segment. But the stent implantation did not seal the perforated region. A second 3.0×18 mm cobalt chromium BMS was implanted to the perforated area and the perforation was sealed completely (Fig. 4A, B). Further injection did not show any extravasation of contrast agent. Subsequent echocardiographic examination showed posteriolateral 0.6 cm pericardial fluid. A control echocardiographic examination three days later did not show any progression in pericardial effusion. Cardiac troponin levels and CK-MB also were not increased. Anticoagulation with acetylsalicylic acid, heparin, clopidogrel and tirofiban was not reversed due to succesful intervention. The patient was discharged from hospital three days later and a control examination after one month was uneventful. Con-

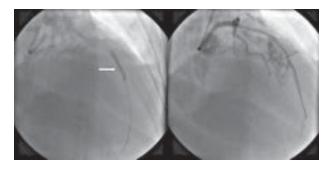


Figure 3. Post-dilation of residual stenosis with high pressure balloon (left side). White arrow reveals notch formation outward to struts which could indicate strut rupture and perforation. After post-dilation, strut rupture and coronary perforation is clearly seen just beneath the overlapping border (right side). Overlapping edge is marked with black line

trol angiography showed no evidence of stent restenosis or extravasations from the perforated site (Fig. 4C).

DISCUSSION

Coronary artery perforation is a rare complication of PCI. It can lead to simple dye staining of the pericardium, to pericar-

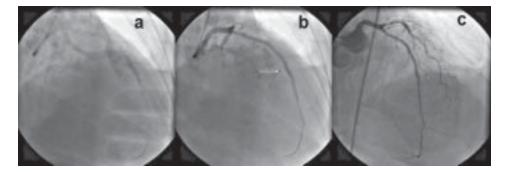


Figure 4. Sealing of perforation with implantation of two consecutive cobalt chromium bare metal stents (**A**). Complete sealing of perforation is seen from right anterior oblique projection (**B**). Section **C** shows appearance of left anterior descending artery in control angiography performed one month later. There is no evidence of perforation and all the stents are patent

dial tamponade, or to complete haemodynamic collapse [2]. Its incidence has been reported as 0.1–3.0% of PCI procedures [3]. Immediate treatment is crucial in CP cases. Prolonged balloon inflation with perfusion balloons, pericardiocentesis, embolisation of the perforated vessel, haemodynamic support and reversal of heparin anticoagulation are the first line management methods [4]. The implantation of covered stents over perforated segments is feasible and associated with a high success rate: it has beenreported as high as 91% in the literature [5]. But especially in tortuous and calcified vessels, it can be difficult to advance a covered stent through the vessel's lumen due to lack of flexibility of the stent. Emergency surgery remains the last resort of therapy [6].

Predisposing risk factors for CP formation include advancing age, female gender, kidney failure, heavy calcification, chronic total occlusion, tortuous and bending vessels, complex and type C lesions, target lesions in the circumflex and right coronary arteries, long target lesions (> 10 mm) and eccentric lesions [3, 7].

The use of an atheroablative device, laser angioplasty, cutting balloon, hydrophilic and stiff guidewires post-dilatation with high-pressure balloons can lead to CP formation [8]. Ellis et al. [9] have classified CP according to angiographic appearance: Type 1 CP is described as a crater extending outside the lumen only; Type 2 CP results in pericardial or myocardial blush without a > 1 mm exit hole; Type 3 results in streaming of contrast through a > 1 mm exit hole and the cavity spills as a perforation into an anatomic chamber. Type 1 and 2 perforations are usually caused by hydrophilic and stiff wires and do not require pericardial drainage or surgical intervention [3]. However, perforation with guidewire may cause delayed pericardial effusions or late pseudoaneurysm formation [10]. So, such cases should be observed carefully after intervention. Type 3 perforations are more often associated with stent and device use. They usually cause haemodynamic instability and nearly half of the cases require pericardial drainage. Most Type 3 perforations can be managed by percutaneous methods. Surgical repair constitutes the last resort

of treatment and is required less and less as new interventional techniques are developed. Without interventional or surgical treatment, the mortality rate for Type 3 perforation has been reported as 3-10% [1, 3, 4, 6].

Predisposing risk factors for our patient were her relatively advanced age, female gender, calcified and long target lesions. We had used stiff non-hydrophilic guidewire. But, the perforation occurred due to strut rupture after post-dilatation in our case. Perforations associated with stents usually occur from the edges or the overlapped portions. Isolated strut rupture is extremely rare and has not been previously reported for a sirolimus-eluting stent.

During post-dilation, a small notch appeared outward through the struts of the stent. Further injection showed perforation from this site and the overlapped border was seen clearly just over the perforated segment. The perforation was classified as Ellis Type 3, which has the worst prognosis. Due to the width and seriousness of the perforation, prolonged balloon dilatation and perfusion balloon had not been tried. A decision was made between surgical and interventional therapy and stent implantation was performed immediately. The rapid decision and sealing of the perforation with stent implantation prevented the formation of a pericardial tamponade.

Polytetrafluoroethlene-covered stents constitute first line management of CP cases [11, 12]. Recently, more flexible pericardial covered stents have also been used successfully in CP cases [8]. Venous covered stent usage has also been reported for venous graft perforation [10]. We used BMS due to the unavailability of the covered stent. We chose a stent with narrow struts so that we could seal the perforation easily. After implantation of two consecutive stents, the perforation was sealed. So, it is logical to use a stent with narrow struts for perforation when covered stents have failed or are unavailable. The inflation pressure of the stent also should not be higher than the optimal threshold to prevent further opening of the stent struts. Embolisation of the perforation with coil, collagen or glue, thrombin, alcohol and coagulated blood from the patient is usually preferred in perforations of distal segments [13–18]. We did not choose such a technique in this case due to the location of the perforation in the middle LAD segment.

CONCLUSIONS

CP is a rare complication of PCI which requires urgent treatment. Although covered stents are approved as first line therapy, BMS with narrow struts can also be used when covered stents have failed. A second or third stent implantation to the perforated area can be carried out, as in our case, if the first stent could not seal the perforation. Also, post-dilation of stents should be made consciously in those patients at risk of coronary perforation. Even a stent with a high radial force such as a sirolimus-eluting stent can cause a perforation via rupture of struts after post-dilatation, and this can be a life-threatining coronary perforation.

Conflict of interest: none declared

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