Long-term follow-up study of elderly patients with covered stent implantation after coronary perforation

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Abstract

Objective To evaluate the long-term efficacy of covered stent implantation in the treatment of elderly patients with coronary perforation while undergoing percutaneous coronary intervention (PCI).

Methods From June 2004 to June 2012, our center has followed ten elderly patients (age ≥ 60 years) who sustained coronary perforation during PCI. The major adverse cardiac events (MACE) were observed as well. The patients were advised to take 75 mg/day Clopidogrel for two years, and indefinite use of 100 mg/day enteric-coated aspirin.

Results Six out of the 10 patients aged from 60 to 76 years old (mean 68.6 ± 5.2 years) were male, four were female. The average diameter of the implanted stents was 3.3 ± 0.3 mm, and the average length was 22.1 ± 3.7 mm. All the ruptures were successfully sealed without intra-procedural death. The follow-up duration ranged from 0.6 to 67 months (mean 31.7 ± 24.5 months). One patient died of multiple organ failure due to lung infection in 19 days after PCI; one died of cardiac sudden death in 13 months after PCI; one had angina pectoris in 53 months after PCI; one underwent multi-slice CT examination in six months after PCI, and no in-stent restenosis was found. The other four patients received angiography follow-up, and the results showed that three patients had no intra-stent restenosis, while one had left anterior descending (LAD) restenosis in the covered stent in 67 months after PCI. The in-hospital mortality was 10% (1/10). The MACE rate in 12 months after PCI was 10% (1/10). During the entire followed-up period, the restenosis rate in target vessels was 20% (1/5), mortality was 20% (2/10), and the MACE rate was 40% (4/10).

Conclusion Treatment of coronary perforation by using covered stents can achieve favorable long-term results; a two-year dual antiplatelet therapy (DAPT) after PCI can effectively prevent intra-stent thrombosis.

Keywords: Coronary perforation; Percutaneous coronary intervention; Covered stent

1 Introduction

Coronary perforation is a rare but critical complication of percutaneous coronary intervention (PCI), which increases the risk of acute cardiac tamponade and high mortality. Covered stent implantation, with satisfactory immediate and short-term outcomes, can effectively prevent coronary hemorrhage and reduce the incidence of acute cardiac tamponade and mortality without surgery. However, there is a limited amount of long-term follow-up data on the efficacy of covered stent implantation in the current literature. Therefore, we report on a cohort of 10 patients who sustained coronary perforation during PCI at our center through a long-term follow up. The analytical reports are as follows.

2 Methods

2.1 Clinical and coronary angiographic (CAG) data

We followed 10 patients who underwent coronary perforation during PCI through a long-term follow-up from June 2004 to June 2012. Six of the 10 patients aged from 60 to 76 (mean 68.6 ± 5.2 years) were male, four were female. Three patients suffered from acute anterior myocardial infarction 7 to 12 days ago; one had an inferior old myocardial infarction; six had unstable angina pectoris. Coronary angiography showed left main/triple-vessel lesions in three cases, triple-vessel lesions in two cases, double-vessel lesions in three cases, and single-vessel in two cases. In two cases, the target lesions were found in the right coronary artery, another eight cases were in the left anterior descending artery (LAD). Four of which also had calcifications; one had chronic total occlusion (CTO) lesion (Table 1).
Table 1. Clinical and coronary angiographic data.

| Case No. | Gender | Age, yrs | Risk factors | Diagnosis          | Number of diseased vessels | Target lesion                       | Degree of target lesion stenosis (%) and features |
|----------|--------|----------|--------------|--------------------|----------------------------|------------------------------------|---------------------------------------------------|
| 1        | Male   | 65       | Smoking      | Anterior wall AMI 7 days | LM + triple-vessel          | Distal segment of LAD             | 99%                                               |
| 2        | Female | 76       | Hypertension | UAP                | LM + triple-vessel          | Proximal-middle segments of LAD   | 70%, calcifications                              |
| 3        | Female | 69       | No           | UAP                | LM + triple-vessel          | Proximal-middle segments of LAD   | 100%, CTO, calcifications                        |
| 4        | Female | 65       | Hypertension | UAP                | Single-vessel               | Proximal-middle segments of LAD   | 70%                                               |
| 5        | Male   | 70       | Hypertension | Inferior OMI       | Single-vessel               | Distal segments of RCA            | 99%                                               |
| 6        | Male   | 71       | No           | UAP                | Triple-vessel               | Proximal-middle segments of LAD   | 95%, calcifications                              |
| 7        | Male   | 63       | No           | Anterior AMI 12 days | Double-vessel               | Proximal segments of LAD          | 100%                                              |
| 8        | Male   | 75       | No           | Anterior AMI 11 days | Double-vessel               | Proximal segments of LAD          | 100%                                              |
| 9        | Female | 72       | Hypertension | UAP                | Double-vessel               | Proximal-middle segments of LAD   | 90%, calcifications                              |
| 10       | Male   | 60       | No           | UAP                | Double-vessel               | Middle segments of RCA            | 70%                                               |

AMI: acute myocardial infarction; CTO: chronic total occlusion; LAD: left anterior descending; LM: left main coronary artery; OMI: old myocardial infarction; RCA: right coronary artery; UAP: unstable angina pectoris.

2.2 Coronary intervention

All patients were prescribed doses of heparin according to their weight (70–100 unit/Kg) before PCI. Nine patients had Type III coronary perforation (according to the criteria defined by Ellis [1]), one had Type I coronary perforation, which was not identified immediately and further developed a cardiac tamponade 1.5 h after the procedure. Of all the cases, two were caused by false lumen dilations; three resulted from over dilation during stent deployment; one resulted from proximal overlapping section dilation in tandem stent; four resulted from post-dilation of non-compliant balloon. Every patient had a successful implantation of a single covered stent. All the perforations were successfully sealed after the implantation and contrast streaming disappeared.

The two patients who had occlusions in proximal LAD suffered coronary perforations caused by false lumen dilation. Because of the large perforation and severe angina along with an unstable hemodynamic situation after the prolonged balloon inflation technique in the left main-left circumflex coronary artery (LM-LCX), covered stents were deployed immediately in LM-LCX, sealing the perforation in LAD at once. In the other eight cases, the covered stents were placed at the lesion site after failed closure of the perforation by prolonged balloon inflations. The patients were advised to take 75 mg/day clopidogrel for two years, long-term use of enteric-coated aspirin 100 mg/day, statins, beta-blockers, and angiotensin-converting enzyme inhibitors (ACEI), (Table 2).

2.3 Follow-up

The clinical data were obtained from inpatient observation, outpatient clinic visits, and telephone interviews. A dedicated person recorded the data and followed the patients after the implantation. Major adverse cardiac events (MACE) were defined in this study as recurrence angina, non-fatal myocardial infarction, revascularization and death. In-stent restenosis was defined as > 50% diameter narrowing by coronary angiography. Lesion segment restenosis was defined as > 50% lumen diameter narrowing at an average of 5-mm proximal and distal to the stent edges.

Table 2. Percutaneous coronary intervention data.

| Case No. | Reason for coronary perforation | Covered stent size (mm) |
|----------|---------------------------------|-------------------------|
| 1        | Stent deployment                 | 3.0 × 26                |
| 2        | Non-compliant post-dilation      | 3.0 × 19                |
| 3        | False lumen dilation             | 3.5 × 26                |
| 4        | Overlapping section dilation by stent balloon | 3.5 × 16 |
| 5        | Non-compliant post-dilation      | 3.0 × 19                |
| 6        | Stent deployment                 | 3.0 × 19                |
| 7        | Non-compliant post-dilation      | 3.5 × 26                |
| 8        | False lumen dilation             | 3.5 × 19                |
| 9        | Non-compliant post-dilation      | 3.0 × 19                |
| 10       | Stent deployment                 | 3.5 × 26                |

2.4 Statistical methods

The data are presented as mean ± SD unless other indicated. The enumeration data are presented as percent (%).

3 Results

3.1 Clinical results and PCI

Eight had multi-vessel disease, four combined with calcifications. In eight cases, the target vessels were LAD, three had total occlusion lesions. The use of heparin during the
procedure was based on common guidelines. In two cases, the coronary perforations were caused by false lumen dilation. While the other eight correlated with either over-dilated stent, or over post-dilation. Of the 10 cases, nine were type III coronary perforation, while one was type I. Only one patient did not undergo pericardiocentesis since pericardial effusion was not detected by EB-Echo. In the other cases, 100–470 mL pericardial effusion was effectively drained. The implanted covered stents were 3.3 ± 0.3 mm in diameter and 22.1 ± 3.7 mm in length on average. All the ruptures were successful sealed, without usingprotamine in any single case.

3.2 Follow-up results

The follow-up duration ranged from 0.6 to 67 months (mean 31.7 ± 24.5 months), with a 100% follow-up rate. In case No. 8, the covered stent was deployed in LM-LCX. The patient died of multiple organ failure due to lung infection 19 days after PCI. In case No.6, the patient died 13 months after PCI from an unknown cause, which could have been sudden cardiac death. In case No.4, the patient had chest pain at 53 months after PCI. The syndrome was alleviated by medication treatment without CAG after the patient was hospitalized. In case No.5, the patient underwent multi-slice CT examination six months after PCI. No intra-stent restenosis was found. In case No.9, the patient did not complain of chest pain at the 28 month follow-up, therefore CAG was not performed. In case No.10, the patient was hospitalized at our hospital 14 days after PCI because of high fever. Pericardial effusion was detected by EB-Echo. Pericardiocentesis was therefore performed, and 200 mL pericardial fluid thereafter was effectively drained. The patient’s fever subsided and he was discharged after anti-infection treatment. The recovery was favorable at four months of follow-up. The other four patients underwent coronary angiography which confirmed LAD in-stent restenosis in case No.1 at 67 months after PCI, and who thus received target vessel revascularization (TVR) and a drug eluting stent implantation. Fifteen to 54 months after PCI, the angiographic result suggested that the remaining three patients had no restenosis within the stent (Figure 1 & 2). The in-hospital mortality rate was 10% (1/10). The MACE rate was 10% (1/10) 12 months after PCI. During the entire followed-up, the restenosis rate in target vessels was 20% (1/5), mortality rate was 20% (2/10) and MACE rate was 40% (4/10).

4 Discussion

As has been reported, the incidence of coronary perforation ranges from 0.1% to 3.0%. The incidence of acute cardiac tamponade ranges from 17% to 24% when coronary perforation occurred. The emergency operative repair rate ranges from 24% to 36%. The mortality might be as high as 10%, or even higher. Women and the elderly are at the highest risk of coronary perforation. Rates of perforation may be potentially higher when atherectomy devices are used, or complex calcified lesion occurred. Over 96% of the lesions are type B2 or type C lesions. They were prone to coronary perforation, as most of them had multi-vessel lesions; some also had calcification or chronic total occlusion.

Conventional management options would include substitution of heparin with protamine, prolonged balloon inflation across the dissection in the coronary vessel, discontinuing glycoprotein IIb/IIIa inhibitors, coil embolization (spring coil, gelfoam, autologous fat, thrombin, etc.), placement of covered stents and surgical repair. However, heparin substitution might not be able to seal the ruptures effectively. As PCI shall be performed within coronary arteries, heparin substitution may potentially lead to focal or proximal embolization, or more severe complications.
Therefore, discontinuing or substituting heparin might not be a proper management for coronary perforation. Taking immediate measure to seal the rupture would be a better option. Providing temporary hemostasis across the dissection in the coronary vessel would better fit the distal small vessels. Surgery is unquestionably the last option, since it is a less-preferred alternative by both doctors and patients. Prolonged balloon inflation will result in intra-coronary thrombosis, which increases the risk of myocardial infarction and death. Of the 10 cases, only one was type I coronary perforation, covered stent was placed over the perforation as cardiac tamponade occurred. The remaining nine cases were type III. All the ruptures were successful sealed, without the use of protamine in any single case. Only one patient died in hospital from multiple organ failure due to lung infection, all the others survived and were finally discharged. This suggests that covered stent implantation is an effective way for management of large coronary perforations.

The covered stent, with a unique structure, consists of an ultrathin, biocompatible, and expandable polytetrafluoroethylene layer sandwiched in between two coaxial stainless steel, slotted tube, balloon expandable stents, without a mesh structure inside. Al-Mukhaini, et al. has claimed that it is more difficult for endothelialization due to its structural differences than conventional stent. And as a result, there is a-high rate of in-stent restenosis, which is however not seen in current research. Nevertheless, the stated potential issue has been taken into account by this study, oversized covered stents were thus excluded and the average length is 22.1 mm. The implantations were undertaken with caution to avoid critical side branch coverage which might cause subsequent massive myonecrosis. Moreover, the patients received dual antiplatelet therapy for two years after the implantation.

There was no intra-procedural death. The post-operation and in-hospital mortality was 10% (1/10). The MACE rate was 10% (1/10) 12 months after PCI. During the entire follow-up, the restenosis rate in target vessels was 20% (1/5), mortality was 20% (2/10), MACE rate was 40% (4/10).

There was no evidence of definite thrombosis within the covered stent. The outcome was satisfactory, when compared with an overall restenosis rate ranged from 20% to 40% at six months after treatment using a conventional metallic uncoated stent. This suggests that treatment of coronary perforation using covered stents can achieve favorable long-term results. A dual antiplatelet therapy for two years after PCI can effectively prevent intra-stent thrombosis.

Given the low overall incidence of coronary perforation at our hospital, there were consequently fewer patients underwent PCI. However, this study is a small sample, single-centered, retrospective, follow-up study. Data based on the application of covered stents in the treatment of coronary perforation may need to be analyzed from a larger sample size in any future study.

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