Coronary Perforation During Percutaneous Coronary Intervention
Lessons From Our Experiences

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SUMMARY

Coronary perforation is an undesirable complication during percutaneous coronary intervention (PCI). We reviewed the cases of overt coronary perforation in our institute and analyzed their clinical backgrounds, the characteristics of the target lesion, management, and clinical outcomes. Between 1991 and 2005, we experienced 12 cases (0.35%) of coronary perforation in a total of 3415 PCI procedures. The perforation occurred during the use of debulking devices in 3 cases, immediately after stenting in 2, immediately after postdilatation of the stent in 2, and during wiring in 3 cases. Restoration was attempted by long inflation of a balloon in 7 cases, implantation of a covered stent graft in 1, and emergency surgical repair in 1 case. Subsequent cardiac tamponade occurred in 3 patients who required pericardiocentesis, and 1 patient died due to congestive heart failure. Administration of protamine was effective in stopping the bleeding in 6 patients, whereas continuation of antiplatelet therapy resulted in no overt rebleeding. Coronary perforation during PCI is a rare complication but is associated with significant morbidity and mortality. Intravenous administration of protamine is effective when it is used in conjunction with nonsurgical devices for initial management of perforation. (Int Heart J 2007; 48:1-9)

Key words: Percutaneous transluminal coronary angioplasty, Complication, Cardiac tamponade, Coronary stent, Drug-eluting stent, Antiplatelet therapy

During percutaneous coronary intervention (PCI), coronary perforation is one of the most undesirable complications because it is occasionally life-threatening by causing cardiac tamponade or acute myocardial infarction (AMI).1,2) Recent
reports have shown that the incidence of coronary perforation during PCI is 0.2% to 0.6%, and the use of debulking devices is highly associated with this complication. Despite a rapid increase in the incidence of this serious complication in recent years, definitive management of coronary perforation has not yet been established. Thus, we reviewed our experiences with coronary perforation during the past 15 years and analyzed the characteristics of the lesion, its management, and clinical outcomes.

METHODS

We analyzed the PCI database in which complete information on the patients who received PCI from January 1991 to December 2005 at our institute was recorded. This database also contained the details of the process during the PCI session, including the complications encountered, in the “comments” field. Possible cases that were suspected to have developed coronary perforation were selected by a computerized search task by inserting one of the following key words: “perforation,” “penetration,” “extravasation,” or “tamponade.” Further information on these patients was obtained from the medical chart and movie image of the angiograph recorded during PCI. The characteristics of the lesion were defined based on the American College of Cardiology/American Heart Association Task Force (ACC/AHA) classification. Eccentricity or the extent of calcification was determined by visual estimation during intravascular ultrasound (IVUS) examination. The type of perforation was classified according to the criteria proposed by Ellis, et al. From the information collected, we examined (1) the relationship between the occurrence of coronary perforation and the clinical background, lesion characteristics, or intervention device and (2) the relationship between management and outcome.

RESULTS

Incidence: A total of 3415 PCI procedures were performed between January 1991 and December 2005, and coronary perforation occurred in 12 cases (0.35%). All 12 patients had received antiplatelet therapy with ticlopidine 200 mg and/or aspirin 81 to 200 mg/day. At the time of PCI, heparin was administered by bolus injection or continuous infusion in order to ensure that the activated clotting time was more than 250 seconds.

The details of the characteristics of 12 patients are described in Table I in relation to the clinical background, characteristics of the lesion, and PCI device used. Of these 12 cases, 6 were patients with acute coronary syndrome (2 with AMI and 4 with unstable angina) and 6 had chronic ischemic heart disease (4 with
stable angina and 2 with silent myocardial ischemia). The age of 7 patients was more than 70 years, and 6 patients had diabetes. The target lesions were located in the left anterior descending artery in 8 patients, in the left circumflex artery in 3, and in the right coronary artery in 1 patient. The angiographic characteristic of
the target lesion was type B1 in 1 patient, type B2 in 6 patients, and type C in 4 patients. Of the 12 patients, IVUS was carried out during the PCI sessions of 6 patients. Significant calcification was observed in 3 patients, while 2 patients had eccentric calcification. Coronary perforation occurred when a directional coronary atherectomy (DCA) device was used in 2 cases and when a rotational ablation device was used in 1 case. In 4 patients, it occurred during wiring that is
Management and outcome: The management and outcome after coronary perforation in each patient are described in Table II. Type I, II, III, and IV perforations occurred in 3, 4, 3, and 2 patients, respectively. When perforation was confirmed, heparin was immediately discontinued in 10 patients and reversal of heparin was additionally attempted by administering protamine, an antagonist, to 6 patients, whereas 2 patients continued to receive heparin. After systemic administration of an appropriate dose of protamine to the 6 patients, we confirmed the activated clotting time was less than 150 seconds. In 3 patients whose perforation type was II or IV, the bleeding stopped spontaneously after discontinuation of heparin or additional administration of the antagonist. For the remaining patients, the following procedures were carried out: plain balloon with low-pressure inflation in 4 patients, perfusion balloon with low-pressure inflation in 3 patients, covered stent in 1 patient, and direct surgical repair in 1 patient. Three patients developed cardiac tamponade requiring pericardiocentesis. As a result of uncontrollable heart failure, we lost 1 patient a few days after the PCI procedure, although we confirmed that bleeding had stopped after implantation of the covered stent. One patient underwent elective CABG because the PCI results were inadequate. All patients continued to receive antiplatelet therapy, but none showed any sign of rebleeding from the perforation site afterward.
DISCUSSION

Factors that increase the risk of coronary perforation: Between 1991 and 2005, the incidence of coronary perforation at our institute was 0.35%, which was similar to that reported in previous articles.\(^1\)\(^-\)\(^8\) Fasseas, et al reported the incidence to be 0.58% in an analysis of 16,298 procedures carried out between 1990 and 2001.\(^3\) The present study showed that there has been a rapid increase in the incidence of coronary perforation in the last few years. Of the 12 patients who developed coronary perforation, 9 experienced this complication after 2003. This was probably because in recent years, as various PCI devices have been developed, more complex lesions or smaller vessels were included as the target lesions for PCI. The development of debulking devices, eg, DCA or rotational atherectomy, has enabled the treatment of calcified and bifurcation lesions.\(^3\),\(^12\) The recently developed stiff guidewires have increased the success rate for crossing chronic total occlusion lesions.\(^13\) As a result, the number of PCI conducted for difficult and fragile lesions, which were previously considered to be unsuitable for PCI, has increased. This alteration in the type of lesion targeted by PCI may be associated with the rapid increase in the incidence of coronary perforation over the time period of this study. Previous reports have demonstrated that the increase in the risk of perforation is highly associated with the use of oversized balloons,\(^9\) stenting against eccentric, extensive, or tortuous calcified lesions,\(^5\),\(^7\),\(^9\) and the use of atherectomy devices.\(^2\),\(^3\),\(^6\),\(^8\),\(^9\) In the patient series analyzed in this study, in addition to the other risk factors, the use of the sirolimus-eluting stent (SES) was also found to increase the incidence of coronary perforation. The SES has been available in Japan since 2004, and all 5 perforation events (2.69%) in 2004 and 2005 occurred after deployment of the SES or during wiring for crossing the lesion that was to be treated by the drug-eluting stent.

We speculate that the main reason for the increased risk of perforation by the SES was due to the characteristics of both the device and the target vessel. BX Velocity, the platform stent of Cypher\(^\text{TM}\) (Cordis, a Johnson & Johnson Co., Miami Lake, FL), which is the sole drug-eluting stent currently available in Japan, is known to be one of the stiffest stents and requires high-pressure inflation for sufficient expansion.\(^14\) On the other hand, using the Cypher\(^\text{TM}\) is preferable when the possibility of restenosis is considered to be high due to the presence of a small target vessel, heavily calcified target lesion, or CTO lesion.\(^15\) It is inevitable that there will be an increased risk of perforation when PCI is carried out with a stiff stent in cases with such fragile lesions. Therefore, the risk of perforation should be recognized in advance, particularly when PCI using the SES against difficult lesions is scheduled.

Prevention of coronary perforation: Identifying the risk of perforation is impor-
tant in preventing coronary perforation during PCI. Since the occurrence of perforation is highly associated with the selection of an appropriate device for the lesion, the characteristics of the lesions should be fully evaluated in advance. The use of IVUS is helpful in such cases. The location of calcification cannot be evaluated by an angiogram, while IVUS can provide not only the severity but also the depth, eccentricity, and/or extent of calcification, which are thought to influence the risk of perforation. In particular, when IVUS shows the presence of eccentric calcification at the target lesion and indicates the noncalcified wall on the opposite side is thin, the possibility of perforation would be considerably high since an integrated pressure force directly localizes on the thin wall. In such cases, lesion modification with rotational atherectomy prior to stenting and a gradual increase in the pressure for inflating the stent is recommended. Although debulking devices are known to increase the risk of perforation, we believe that this is not the case when optimal-sized rotational atherectomy is used as a pretreatment for stenting. The selection of an undersized ablation device would be optimal in such cases since the main purpose of debulking is not to obtain sufficient lumen area but to modify the calcification, which causes difficulty in stent expansion. More importantly, if concerns still persist, the procedure should be abandoned and the therapeutic strategy should be altered.

Management of coronary perforation: In this analysis, cardiac tamponade occurred in 3 cases. Of these 3 cases, we lost 1 patient and another patient underwent emergency CABG. Therefore, cardiac tamponade was found to be closely associated with mortality. The outcome depends on how soon the bleeding can be stopped when perforation occurs. To achieve this, heparin should be immediately discontinued if it is being administered continuously. Systemic administration of protamine, which can antagonize the effect of heparin, should also be attempted unless the situation prohibits it.

Next, the blood flow of the perforated branch should be blocked by using a plain or perfusion balloon. If the perforation was created immediately after balloon inflation or stent implantation, the blood flow of the branch should be temporally blocked by inflating the plain balloon that was used for the procedure. It will take a few seconds to inflate the balloon and block the blood flow if it is still inside the guiding catheter. If myocardial ischemia caused by balloon inflation is negligible, long-term inflation can be performed until restoration of the vessel is confirmed. However, in most cases, the plain balloon has to be exchanged with the perfusion balloon that enables the blood to perfuse to the distal portion of the vessel. When the perfusion balloon is ready for use, the inflated balloon should be quickly deflated and withdrawn. The perfusion balloon should be delivered to the perforated site and inflated with low pressure, beginning with 1 atm and increasing gradually until the disappearance of dye leakage is confirmed.
In the meantime, transthoracic echocardiography (TTE) should be performed to check if cardiac tamponade develops. If TTE shows the presence of a certain amount of pericardial fluid and sufficient pericardial space for safe centesis, pericardiocentesis must be immediately carried out irrespective of the success in stopping bleeding. In such a case, the speed of hydration should be increased. Since subsequent hypotension can potentially cause greater complication, prevention of cardiac tamponade or hypovolemic shock is important.

If the above-mentioned methods fail to restore perforation, emergency CABG should be considered.1,2) It is important to realize the limitations of non-invasive devices if the damage to the vessel is substantial. Before sending the patient to the operating room, delivery of the autologous covered stent might be attempted as an optional treatment,21) although the restenosis rate of this device is known to be high.22,23) In particular, when the risk associated with the surgical procedure is considered to be higher than the subsequent cardiac event related with the restenosis or reocclusion of the covered stent, or when a certain condition does not allow an emergency operation to be performed, this method is considered to be valuable.

We continued administering antiplatelet agents to all patients after coronary perforation. In contrast to discontinuing or antagonizing the anticoagulant, discontinuation of administration of antiplatelet agents does not rapidly affect hemostasis since these agents irreversibly disturb platelet function. Thus, once the bleeding is successfully stopped, the advantage of discontinuing the use of antiplatelet agents is considered to be minimal, although the risk of thrombosis is increased.

Conclusions: Coronary perforation is a rare complication but is associated with significant morbidity and mortality. The incidence of this complication has been recently increasing in parallel with the increased use of SES or debulking devices. To restore perforation, intravenous administration of protamine, a heparin antagonist, is effective in conjunction with prolonged inflation of the perfusion or plain balloon.

REFERENCES


