CORONARY PERFORATION DURING PERCUTANEOUS CORONARY INTERVENTION; EXPERIENCE AT ARMED FORCES INSTITUTE OF CARDIOLOGY

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ABSTRACT

Objective: The study was conducted to find out the frequency of coronary perforation during percutaneous coronary intervention.

Study Design: Descriptive cross sectional study.

Place and Duration of Study: This study was conducted at AFIC/ NIHD Rawalpindi from Jan 2010 to Mar 2012.

Materials and Methods: We report the incidence, management, and clinical outcome of coronary perforation in 12 of 9668 patients (0.12%) undergoing percutaneous coronary interventions between Jan 2010 and Mar 2012.

Results: The most commonly involved artery was left anterior descending artery (66.7%) followed by circumflex (16.7%) and right coronary artery (16.7%). There was one type I perforation (8.3%), eight type II perforations (66.6%) and three type three perforations (25%). One fourth of the coronary perforations were guide wire related. Conventional strategies to treat perforations i.e., prolonged balloon inflations, reversal of anti-coagulation in selected cases and covered stents were used. There was one death, one emergency surgery and one Q wave myocardial myocardial infarction. Pericardial effusion occurred in 3 out of 12 patients with cardiac tamponade occurring in 2 patients requiring emergency peri-cardiocentesis.

Keywords: Coronary perforation, Myocardial perfusion, percutaneous coronary intervention.

INTRODUCTION

Coronary perforation is an uncommon but potentially life threatening complication of percutaneous coronary intervention (PCI). The incidence of coronary perforation is low occurring in less than 0.2% of patients following balloon angioplasty and seems to occur more frequently with the use of coronary devices such as high speed rotational atherectomy, directional coronary atherectomy (DCA), laser and stents. Moreover, the problem may be further complicated by use of novel anti-platelet agents, Glycoprotein IIb/IIIa inhibitors being used with increasing frequency.

PATIENTS AND METHODS

On scrutiny of records twelve patients were found to have suffered from coronary perforation. Six were males (50%) and six were females (50%). The most commonly involved artery was left anterior descending artery (66.7%) followed by circumflex (16.7%) and right coronary artery (16.7%).

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The wire most commonly used was Runthrough wire (83.3 %). Lesion classification was classified according to American heart association/ American College of Cardiology (AHA/ ACC) classification task force. All patients underwent PCI according to practice guidelines at the time of the procedure. Adjunctive therapies included Aspirin, Clopidogrel, glycoprotein (Gp) IIb/IIIa inhibitors. Conventional steerable guide wire systems were used and the operator selected the interventional devices at the time of the intervention. Patients were examined angiographically before and immediately after coronary intervention. Similar single view projections were used at each angiographic examination. Percent coronary stenosis, reference and minimal luminal diameter were determined using quantitative coronary analysis after intracoronary administration of 100 ug of Nitroglycerin using a computer-assisted automated edge detection algorithm. Absolute reference and minimal luminal diameters were determined (in millimeters) using the guiding catheter filled with contrast for calibration. For each lesion the single view showing the most severe degree of stenosis was
used for analysis. A lesion treatment was considered to be successful when there was > 50% gain in luminal diameter and < 20% residual diameter stenosis in the absence of severe complications including death, Q-wave MI or emergency bypass surgery.

RESULTS

From Jan 2010 to Mar 2012 coronary perforation occurred in 12 out of 9868 consecutive patients who underwent PCI at AFIC/NIHD. Incidence of perforation therefore was 0.12%. All these patients were planned for stent procedures including debulking as required and received Aspirin pre-procedure and Clopidogrel peri-procedure. All patients were anti-coagulated with heparin with target activated clotting (ACT) between 250-300 seconds. Two patients received Gp IIb/IIIa inhibitios prior to detection of perforation. No patient received Gp IIb/IIIa peri-procedure. There was one type I perforation, eight type II perforations and 3 type three perforations (Fig 1).

Coronary perforation due to guide wire (crossing lesion, distal wire perforation) was seen in 4 cases (33.3%). Of these cases, perforations occurred while crossing the lesion with guide wire in two cases and with the distal wire in 2 cases. Of these cases, perforations occurred with the use of hydrophilic guide wires in 83.4% of patients, with the use of medium and standard guide wires in 16.6%. In remaining cases perforation occurred during stent deployment or post-stenting ballooning.

Treatment strategy

Covered stents were used in five cases (58.3%), three with type 3 perforation and two with type 2 perforations who continued to bleed after prolonged balloon inflation and reversal of anticoagulation. In three cases after balloon inflation stents were deployed to manage associated dissection. Three patients required pericardiocentesis and one of them needed emergency surgery to seal the rent in right ventricle.

DISCUSSION

Coronary perforation is defined as persistent extravasation and accumulation of contrast medium through the vessel wall. Although the frequency of coronary perforation is not very high it is a serious complication in the setting of percutaneous coronary intervention. The reported incidence is around 0.5% in cases of balloon angioplasty and 0.5-3% in percutaneous trans-luminal coronary angioplasty. Advanced age, female gender, complex coronary anatomy (type B2 and C lesion) chronic total occlusions, intravascular ultrasound use and the use of ablative devices seem to be the key factors predisposing to this complication.

Furthermore its magnitude and management may be further complicated with the use of glycoprotein (GP) IIb/IIIa platelet inhibitors which are being used with increasing frequency during PCI. Perforations are classified into three broad groups. Type 1 comprises discrete or localized extravascular flow oozing into the myocardium or epicardium usually caused by guide wires. Type 2 perforation is defined by persistent extra-vascular flow. Atherectomy devices are usually responsible for this type of perforation. Type 3 being the most severe is defined as an active extravasation through a large breach (at least 1 mm) in the integrity of the adventitia. The present study demonstrated slightly lower incidence of coronary perforation as compared to international literature because of less use of new devices in our set up so far i.e., atherectomy devices. Other risk factors for coronary perforation are related to complex coronary anatomy (calcified lesions, chronic total occlusion, tortuosity of the vessel and ostéal lesion). Similar to previous reports, most of our patients had complex anatomy (American College of Cardiology/American Heart Association lesion class B and C) making the intervention more difficult. Similar to other studies our study demonstrated that distal migration of the guide wire is an important factor for coronary perforation as 33.3% of the coronary perforation were guide wire related in present study. Although coronary perforation can occur while attempting to cross the lesion with the guide wire, the distal wire by itself or a fractured guide wire can often cause the perforation. The importance of this finding
cannot be overemphasized as two out of four guide wire related perforation had type III perforation and required pericardiocentesis. It is also important to recognize that perforation is not always immediately evident as tamponade can present later as occurred in two of our patients. This diagnosis should remain high on the list of the differential diagnosis of post-PCI hypotension. Another form of cardiac perforation not included in the present study occurs as a consequence of the temporary pacing wire and interventional cardiologist should be aware of this cause of pericardial effusion and tamponade in patients undergoing PCI.

Major clinical outcome in coronary perforation is related to the angiographic classification of the perforation occurring more frequently in patients who experienced type III perforation. In our study one fatality had occurred because of such type of perforation. In our study we did not find a trend for an increased incidence of tamponade in patient receiving GP IIb/IIIa antagonists as reported in some studies nor their use was associated with a deleterious impact on mortality or emergency surgery rates.

Treatment of coronary perforation in the current PCI era requires early detection, angiographic classification, immediate balloon occlusion of the coronary vessel extravasation followed by use of covered stents and relief of haemodynamic compromise if needed. Keeping in mind that pericardial effusion and tamponade are more likely to occur in type II and III perforations, immediate occlusion of the perforated vessel should be accomplished by prompt and prolonged balloon catheter inflation at the site of perforation. Perfusion balloon catheters can be used for prolonged balloon occlusion maintaining myocardial perfusion and therefore avoiding myocardial ischaemia during balloon occlusion. Immediate attention should be directed to reversal of heparin anti-coagulation with Protamine sulphate to achieve a partial thromboplastin time of less than 60 sec or an ACT of less than 150 seconds. Platelet transfusion is useful in patients treated with abciximab but not with tirofiban or eptifibatide. Type III perforation is mostly dealt with covered stents. These stents have revolutionized the treatment of coronary perforation and have decreased mortality and need for emergency surgery in these patients. In our study covered stents were used in seven patients (58.3%). Other forms of sealing perforated vessels described prior to the introduction of the covered stents include the use of autologous vein graft, makeshift stent sandwich and microcoil embolization.

**CONCLUSION**

In the current era of frequent device use, the incidence of coronary perforation remains low and mostly occurs as a consequence of guidewire migration and injury and with the use of debulking devices in the presence of Gp IIb/IIIa inhibitors. Treatment requires early detection, angiographic classification, immediate occlusion of coronary vessel extravasation and relief of haemodynamic compromise, reversal of heparin anticoagulation and platelet transfusion in selected cases and the use of cover stents.

**Conflict of Interest**

This study has no conflict of interest to declare by any author.

**REFERENCES**
