

CLINICAL RESEARCH

Incidence, Predictors, Management, Immediate and Long-Term Outcomes Following Grade III Coronary Perforation

Rasha Al-Lamee, MA,*†‡ Alfonso Ielasi, MD,* Azeem Latib, MD,*†
Cosmo Godino, MD,*† Massimo Ferraro,† Marco Mussardo, MD,* Francesco Arioli, MD,*
Mauro Carlino, MD,* Matteo Montorfano, MD,* Alaide Chieffo, MD,*
Antonio Colombo, MD*†

Milan, Italy; and London, United Kingdom

Objectives The aim of this study was to evaluate the incidence, predictors, management, and clinical outcomes in patients with grade III coronary perforation during percutaneous coronary intervention.

Background Grade III coronary perforation is a rare but recognized complication associated with high morbidity and mortality.

Methods From 24,465 patients undergoing percutaneous coronary intervention from May 1993 to December 2009, 56 patients had grade III coronary perforation.

Results Most lesions were complex: 44.6% type B2, 51.8% type C, and 28.6% chronic total occlusions, and within a small vessel (≤ 2.5 mm) in 32.1%. Glycoprotein IIb/IIIa inhibitors were administered in 17.9% of patients. The device causing perforation was intracoronary balloon in 50%: 53.6% compliant, 46.4% noncompliant; intracoronary guidewire in 17.9%; rotablation in 3.6%; and directional atherectomy in 3.6%. Following perforation, immediate treatment and success rates, respectively, were prolonged balloon inflation 58.9%, 54.5%; covered stent implantation 46.4%, 84.6%; coronary artery bypass graft surgery (CABG) and surgical repair 16.0%, 44.4%; and coil embolization 1.8%, 100%. Multiple methods were required in 39.3%. During the procedure ($n = 56$), 19.6% required cardiopulmonary resuscitation and 3.6% died. In-hospital ($n = 54$), 3.7% required CABG, 14.8% died. The combined procedural and in-hospital myocardial infarction rate was 42.9%, and major adverse cardiac event rate was 55.4%. At clinical follow-up ($n = 46$) (median: 38.1 months, range 7.6 to 122.8), 4.3% had a myocardial infarction, 4.3% required CABG, and 15.2% died. The target lesion revascularization rate was 13%, with target vessel revascularization in 19.6%, and major adverse cardiac events in 41.3%.

Conclusions Grade III coronary perforation is associated with complex lesions and high acute and long-term major adverse cardiac event rates. (J Am Coll Cardiol Intv 2011;4:87–95) © 2011 by the American College of Cardiology Foundation

From the *Interventional Cardiology Unit, San Raffaele Scientific Institute, Milan, Italy; †Interventional Cardiology Unit, EMO-GVM Centro Cuore Columbus, Milan, Italy; and the ‡Imperial College Healthcare National Health Service Trust, London, United Kingdom. The authors have reported that they have no relationships to disclose.

Manuscript received March 31, 2010; revised manuscript received July 26, 2010, accepted August 6, 2010.

Grade III coronary perforation or coronary rupture is an extremely rare but well recognized complication of percutaneous coronary intervention (PCI) (1,2). It is defined by the Ellis criteria as a perforation resulting in extravasation of blood through a frank perforation (≥ 1 mm) or spilling into an anatomic cavity (3). Previous studies reporting the incidence, predictors, and management of coronary perforation have encompassed all 3 grades of coronary perforation, but there have been no studies specifically focusing on these aspects in the grade III subgroup alone. The incidence of grades I to III coronary perforation ranges from 0.1% to 3.0% (1,3–6).

See page 96

Studies have reported predictors of coronary perforation as: female sex (2,3), calcified and noncompliant arteries (7,8), type C coronary lesions, previous coronary artery bypass grafting (CABG), the use of atheroablative devices (2), older patient groups, hypertension and non-ST-segment elevation myocardial infarction (9).

Abbreviations and Acronyms

CABG = coronary artery
bypass graft

GPI = glycoprotein IIb/IIIa
inhibitors

IVUS = intravascular
ultrasound

MACE = major adverse
cardiac event

MI = myocardial infarction

PCI = percutaneous
coronary intervention

PTFE =
polytetrafluoroethylene

ST = stent thrombosis

TLR = target lesion
revascularization

TVR = target vessel
revascularization

Grade III coronary perforation is the most serious form of perforation and is associated with the highest mortality rates ranging from 7% to 44% of cases (9–12). It is also associated with very high rates of cardiac tamponade of up to 40% (10) and the need for emergency CABG in 20% to 40% (9,10). Treatment modalities include prolonged balloon inflation, covered stent implantation, pericardiocentesis for cardiac tamponade, CABG, and microcoil embolization (13). Often a combination of these techniques is required to achieve adequate hemostasis. Newer techniques such as covered stent deployment have meant that emergency CABG requirements have decreased (14). However, the incidence of coronary perforation with a higher proportion of grade III perforation appears to be increasing because of higher rates of PCI in complex patient and lesion subgroups and the widespread use of glycoprotein IIb/IIIa inhibitors (GPI) (5).

The aim of this study was to report the incidence, predictors, treatment, and long-term outcomes of all patients with grade III coronary perforation as a complication of PCI in the real-world setting of 2 institutions with high volumes of coronary intervention.

Methods

Patients. We analyzed data that had been prospectively collected following PCI in 2 institutions, San Raffaele Scientific Institute and EMO-GVM Centro Cuore Columbus Hospital, Milan, over a 16-year period from May 1993 to December 2009. All cases of grade III coronary perforation had been prospectively entered into the PCI database as defined by the Ellis et al. (3) criteria; a retrospective analysis of these cases was performed by 2 physicians (R.A. and A.I.) with information collection from the database. All original angiograms were reviewed by a technician (M.F.) to confirm the occurrence of grade III perforation. A detailed review with clarification of all information from clinical documentation and coronary angiography was then performed for all cases by the same physicians (R.A. and A.I.) at both institutions. Both physicians involved in this task had not taken part in the procedures analyzed.

Study definitions. Grade III coronary perforation or coronary rupture was defined by the Ellis et al. (3) criteria as extravasation of blood through a frank perforation (≥ 1 mm) or into an anatomic cavity chamber on coronary angiography (3). Cardiac tamponade was defined as the presence of 1 or more of the following: 1) systemic hypotension (systolic blood pressure <90 mm Hg) with evidence of pulsus paradoxus on clinical or invasive assessment; 2) evidence of pericardial fluid collection by angiography or by echocardiography with echocardiographic features of tamponade: significant respiratory variation in transmitral Doppler velocity, dilated inferior vena cava with failure to collapse on inspiration, or diastolic collapse of the right ventricular free wall. Periprocedural myocardial infarction (MI) was defined as a 3-fold increase in creatine kinase-myocardial band. Successful treatment of grade III perforation was defined by the absence of any angiographic evidence of contrast extravasation or clinical or echocardiographic signs of cardiac tamponade. Angiographic restenosis was defined as $>50\%$ diameter stenosis by quantitative coronary angiography within a previously stented segment. *Target lesion revascularization* (TLR) was defined as the need for any repeat revascularization for a stenosis within the stent or within the 5-mm borders adjacent to the stent. *Target vessel revascularization* (TVR) was defined as the need for any repeat revascularization on a treated vessel. *Major adverse cardiac event* (MACE) was defined as a combination of all cause mortality, MI, TLR, TVR, and need for CABG. *Stent thrombosis* (ST) was defined using the Academic Research Consortium (15) definitions and cumulative ST as a combination of all episodes of ST during follow-up.

Procedure. All patients were treated with aspirin and a loading dose of a thienopyridine before the procedure. During the procedure, all patients were then treated with intravenous heparin with an initial 100-U/kg bolus followed by further heparin as necessary to achieve a target activated

clotting time >250 s. Ten patients (17.9%) were also treated with concomitant GPI therapy at the discretion of the operator. Coronary intervention was then performed as usual with pre-dilation, and stent implantation using standard techniques via the femoral artery as have previously been described (16). At the time of perforation, an emergency echocardiogram was performed when tamponade was suspected; however, there were instances in which pericardial drainage was performed without prior imaging if the clinical scenario demanded it. Post-operatively, all patients received aspirin unless there was a specific contraindication, and those patients receiving an intracoronary stent received dual antiplatelet therapy with aspirin and a thienopyridine therapy as determined by contemporary guidelines. Following cessation of thienopyridine therapy, all patients continued to take aspirin indefinitely.

Data collection. All patients were followed-up at regular intervals with clinic visits or telephone interviews. Additional data were obtained from primary care physicians, referring cardiologists, or relatives when necessary. All repeat interventions and complications were prospectively entered into a dedicated database. Clinical follow-up was obtained in all patients, and angiographic follow-up was obtained in 56.5% of patients following successful treatment of grade III coronary perforation.

End points. Adverse procedural and in-hospital events were defined as the need for cardiopulmonary resuscitation, MI, acute ST, necessity for urgent CABG, and death. The long-term primary end points were defined as death from any cause, MI, TVR, TLR, need for CABG, and MACE at any time during the in-hospital stay or at follow-up. We defined the secondary end points as the incidence of restenosis, and ST, defined as probable, possible, or definite.

Statistical analysis. All statistical analysis was performed using SPSS statistical software (version 16.0, SPSS Inc., Chicago, Illinois). Continuous variables are expressed as mean \pm SD or median \pm interquartile range (25th percentile to 75th percentile) as appropriate. Categorical variables are expressed as counts and percentage.

Multivariable logistic regression analysis was used to determine independent predictors of grade III coronary perforation, using purposeful selection of covariates. Variables associated at univariate analysis with grade III coronary perforation (all with $p < 0.2$) and those judged to be of clinical importance from previously published literature were eligible for inclusion into the multivariable model-building process. Candidate variables included sex, circumflex artery lesion, mid-vessel lesion location, coronary occlusions, stent implanted, directional atherectomy, rotablation, calcified lesion, bifurcation lesion, intravascular ultrasound (IVUS) performed, diabetes mellitus, tortuous vessel, cutting balloon pre-dilation, and previous CABG. Model discrimination was measured by the C-statistic and

calibration by the Hosmer-Lemeshow goodness-of-fit test (17).

Results

Baseline patient and lesion demographics. From 24,465 interventional procedural records, we found 56 patients who had coronary intervention complicated by grade III coronary perforation, leading to an incidence of 0.23%. The baseline clinical demographics of these patients are presented in Table 1. Most patients were men, and most patients had presented with stable angina.

Most perforations were situated in the left anterior descending artery, and 32.1% of these lesions were within a small vessel (≤ 2.5 mm). Ninety-six percent of lesions were complex with type B2 lesions in 44.6% and type C lesions in 51.8% of patients. In addition, 28.6% of lesions were chronic total occlusions. The baseline lesion characteristics are outlined in Table 2.

Procedural characteristics. All patients were treated with intravenous heparin and a further 17.9% received GPI during the procedure as either a bolus dose or infusion. Multivessel PCI was performed in 42.9%. Most patients received an intracoronary stent; directional atherectomy was performed in 5.4%; and cutting balloon pre-dilation was carried out in 10.7%. In addition, IVUS was used to guide PCI in 50.0% of cases. The baseline procedural characteristics are shown in Table 3.

The device causing perforation was an intracoronary balloon in 50.0% ($n = 28$) of patients with a compliant balloon used in 53.6% ($n = 15$) and a noncompliant balloon used in 46.4% ($n = 13$). Perforation occurred during pre-dilation before stent implan-

Table 1. Baseline Clinical Characteristics (n = 56)

Age, yrs	66.5 \pm 12.1
Male sex	44 (78.6)
Ejection fraction	55.8 \pm 9.1
Prior myocardial infarction	25 (44.6)
Prior PCI	22 (39.3)
Prior CABG	6 (10.7)
Unstable angina (CCS IV)	4 (7.1)
Stable angina (CCS I–III)	46 (82.2)
Silent ischemia (CCS 0)	6 (10.9)
Multivessel disease	42 (76.4)
Renal impairment (plasma creatinine ≥ 1.4 mg/dl)	4 (7.1)
Cardiovascular risk factors	
Family history of coronary artery disease	22 (39.3)
Hypertension	35 (62.5)
Hypercholesterolemia	38 (67.9)
Current smoker	5 (8.9)
Diabetes mellitus	8 (14.3)

Data presented as percentages and absolute numbers or mean \pm SD.

CABG = coronary artery bypass graft; CCS = Canadian Cardiovascular Society; PCI = percutaneous coronary intervention.

Table 2. Lesion Characteristics (n = 56)

Vessel	
Left anterior descending	25 (44.6)
Circumflex	7 (12.5)
Right coronary artery	13 (23.2)
Intermediate	1 (1.8)
First diagonal	3 (5.4)
Second diagonal	1 (1.8)
Obtuse marginal	3 (5.4)
Septal	1 (1.8)
Saphenous vein graft	2 (3.6)
Lesion location	
Ostial	4 (7.1)
Proximal	22 (39.2)
Mid	26 (46.4)
Distal	4 (7.1)
Lesion and vessel morphology	
Type A	0
Type B1	2 (3.6)
Type B2	24 (44.6)
Type C	29 (51.8)
Chronic total occlusion	16 (28.6)
Significant calcification	13 (23.2)
Small vessel ≤ 2.5 mm	18 (32.1)

Data presented as percentages and absolute numbers or mean \pm SD.

tation in 39.3% (n = 11) and during post-dilation following stent deployment in 60.7% (n = 17) of patients. The mean balloon artery ratio was 1.3 ± 0.2 mm in the compliant balloon group and 1.3 ± 0.3 mm in the noncompliant balloon group. The balloon delivering an intracoronary stent caused vessel perforation in 17.8% (n = 10) of patients, whereas this complication was caused by an intracoronary guidewire in 17.9% (n = 10) with most of these wires being non-hydrophilic (n = 8, 80%).

Management and procedural outcomes. Following grade III coronary perforation, pericardiocentesis for cardiac tamponade was performed in 28.6% (n = 16) and an intra-aortic balloon pump was implanted as an emergency in 19.6% (n = 11) of patients. The overall success rate following treatment of perforation was 87.7% (n = 50). The perforation was treated with prolonged balloon inflation in 58.9% (n = 33); however, this technique was only successful in 54.5% (n = 18) of those treated. Covered stent implantation was performed to treat the grade III coronary perforation in 46.4% (n = 26) and had a higher success rate with hemostasis achieved in 84.6% (n = 22) of patients (Fig. 1). Of the cases treated with covered stent implantation, 96.1% (n = 25) had polytetrafluoroethylene (PTFE) stents implanted, and in 1 case, a custom-made saphenous vein graft-covered stent was implanted in the era before the availability of covered stents. From 25 PTFE stents implanted, 22 were Jostent coronary graft stent (Abbott Vascular Laboratories, Redwood City, California) and 3 were Direct-Stent stent

grafts (InSitu Technologies Inc., Minneapolis, Minnesota) with 3-, 3.5-, and 4-mm diameters and 12-, 16-, and 19-mm lengths. Implantation of a standard stent was performed in 17.9% (n = 10) of cases but was only successful in treating the perforation in a minority of patients (n = 3, 30.0%). Coil embolization was performed in 1 patient and was successful in sealing the perforation. An emergency CABG was performed in 16.0% (n = 9) but was only successful in sealing the rupture in 44.4% (n = 4). Multiple methods of treatment were required in an attempt to achieve hemostasis in 39.3% (n = 22). During the procedure, cardiopulmonary resuscitation was required in 19.6% (n = 11), and the overall intraprocedural mortality rate was 3.6% (n = 2). The characteristics of grade III coronary perforation and treatment are outlined in Table 4.

Predictors of grade III coronary perforation. Multivariable logistic regression was used to assess predictors of grade III coronary perforation. The c-statistic for the propensity score model was 0.78, indicating excellent discrimination. The Hosmer-Lemeshow goodness-of-fit test p value was 0.99, confirming good calibration and fit of the multivariable model that estimated the propensity score. The significant predictors of perforation are presented in Table 5.

In-hospital outcomes. During the in-hospital period, 1 patient had an acute ST following covered stent implantation and 2 patients required urgent CABG and surgical repair of perforation following presentation pericardial effusion 2 to 3 days following the procedure. The combined procedural and in-hospital MI rate was 42.9% (n = 24). In addition, 14.8% (n = 8) died during the index hospitalization period leading to a total mortality rate of 17.9% (n = 10) and a combined procedural and in-hospital MACE rate of 55.4% (n = 31).

Table 3. Baseline Procedural Characteristics (n = 56)

Glycoprotein IIb/IIIa inhibitors	10 (17.9)
Unfractionated heparin	56 (100)
Multivessel stenting	24 (42.9)
Devices used during procedure	
Compliant balloon	26 (46.4)
Noncompliant balloon	21 (37.5)
Intracoronary stent	44 (78.6)
Cutting balloon	6 (10.7)
Directional atherectomy	3 (5.4)
Rotablation	9 (16.1)
Intravascular ultrasound	28 (50.0)
Type of guidewire used	
Standard (nonhydrophilic)	38 (67.9)
Light support (nonhydrophilic)	7 (12.5)
Intermediate support (nonhydrophilic)	16 (28.5)
High support (nonhydrophilic)	17 (30.3)
Hydrophilic	12 (21.4)

Data presented as percentages and absolute numbers or mean \pm SD.

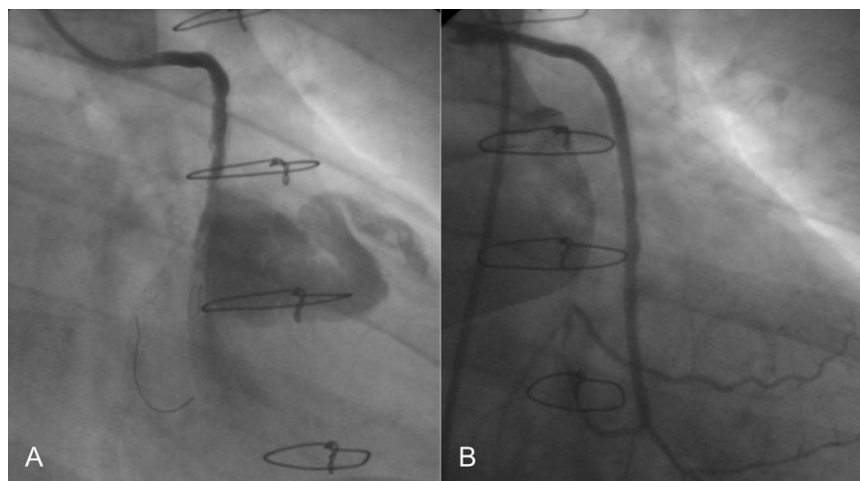


Figure 1. Example of Grade III Coronary Perforation Before and After Treatment

(A) Grade III coronary perforation following percutaneous coronary intervention to saphenous vein graft. **(B)** Successful treatment of coronary perforation achieved following covered stent implantation.

A subgroup analysis of the 10 patients who had GPI and heparin therapy is summarized in Table 6. In this group the proportion of patients who needed multiple treatment methods was higher than the overall group at 50.0% (n = 5), and overall rate of successful treatment of perforation was 80.0% (n = 8). In addition, the procedural and in-hospital MACE rate was 90.0% (n = 9) with 1 death during the procedure and 1 death during the in-hospital period.

Follow-up clinical outcomes. Clinical follow-up was achieved in all patients with median follow-up of 38.1 months (7.6 to 122.8). Most patients (n = 44, 95.7%) were asymptomatic at follow-up with symptoms of stable angina in the remainder (n = 2, 4.3%). The in-stent restenosis rate was 38.5% (n = 10)

with 60.0% (n = 6) of these being occlusive. The MACE rate at follow-up was 41.3% (n = 19), which was composed of MI in 4.3% (n = 2), all-cause mortality in 15.2% (n = 7), need for CABG in 4.3% (n = 2), TLR in 13.0% (n = 6), and TVR in 19.6% (n = 9). Definite ST occurred in 4 patients (8.6%), all of whom had covered stent implantation to treat grade III perforation. One of these patients experienced recurrent episodes of subacute ST following PTFE stent implantation at 9, 11, and 30 days after the index procedure despite maximal antiplatelet and anticoagulant therapy; on each of these occasions, he had repeat angiography and repeat PCI and went on to have CABG but subsequently died of cardiogenic shock. One patient experienced subacute ST on dual antiplatelet therapy 7 days following the index PCI procedure with implantation of a PTFE stent; he subsequently had repeat PCI with drug-eluting stent implantation. One patient presented with late ST 3 months following PTFE implantation at the index procedure while taking dual antiplatelet therapy; he was treated with conventional balloon angioplasty and made a good recovery. One patient presented with late ST 2 months after PTFE stent implantation while taking dual antiplatelet therapy and was managed conservatively. Long-term clinical and an-

Table 4. Characteristics of Grade III Coronary Perforation (n = 56)

Device causing rupture	
Compliant balloon	15 (26.8)
Mean balloon artery ratio	1.3 ± 0.2
Noncompliant balloon	13 (23.2)
Mean balloon artery ratio	1.3 ± 0.3
Stent delivery system	10 (17.8)
Cutting balloon	4 (7.1)
Directional atherectomy	2 (3.6)
Rotablation	2 (3.6)
Hydrophilic wire	2 (3.6)
Nonhydrophilic wire	8 (14.3)
Action following rupture	
Pericardiocentesis	16 (28.6)
Emergency intra-aortic balloon pump	11 (19.6)
Heparin reversal	24 (42.9)

Data presented as percentages and absolute numbers or mean ± SD.

Table 5. Logistic Regression Analysis for Predictors of Grade III Coronary Perforation

	OR	95% CI for OR	p Value
Type B2/C lesions	3.75	1.47–9.60	0.006
Coronary occlusion	1.91	1.02–3.60	0.045
Rotablation performed	3.47	1.59–7.58	0.002
Intravascular ultrasound–guided procedure	5.36	3.10–9.25	<0.001

CI = confidence interval; OR = odds ratio.

Table 6. Treatment and Procedural Outcomes Following Grade III Coronary Perforation

Lesions	Overall Group	Patients Not Treated With GPI	Patients Treated With GPI
Treatment of rupture	n = 56	n = 46	n = 10
Prolonged balloon inflation	33 (58.9)	27 (58.7)	6 (60.0)
Successful	18 (54.5)	16 (59.3)	2 (33.3)
Covered stent implantation	26 (46.4)	20 (43.4)	6 (60.0)
Successful	22 (84.6)	17 (85.0)	5 (83.3)
Standard stent implantation	10 (17.9)	10 (21.7)	0
Successful	3 (30.0)	3 (30.0)	0
Coil embolization	1 (1.8)	1 (2.2)	0
Successful	1 (100)	1 (100)	0
CABG and surgical repair of perforation	9 (16.0)	6 (13.0)	3 (30.0)
Successful	4 (44.4)	3 (50.0)	1 (33.3)
Multiple treatment methods used	22 (39.3)	17 (37.0)	5 (50.0)
Overall successful treatment of rupture	50 (87.7)	42 (91.3)	8 (80.0)
Procedural complications	n = 56	n = 46	n = 10
Cardiopulmonary resuscitation	11 (19.6)	4 (8.7)	3 (30.0)
Death	2 (3.6)	1 (2.2)	1 (10.0)
In-hospital complications	n = 54	n = 45	n = 9
Acute stent thrombosis	1 (1.9)	1 (2.2)	0
Necessity for CABG	2 (3.7)	1 (2.2)	1 (11.1)
Death	8 (14.8)	7 (15.5)	1 (11.1)
Combined procedural and in-hospital events	n = 56	n = 46	n = 10
Myocardial infarction	24 (42.9)	17 (37.0)	7 (70.0)
Major adverse cardiac event	31 (55.4)	22 (47.8)	9 (90.0)

Data presented as percentages and absolute numbers or mean \pm SD.
GPI = glycoprotein IIb/IIIa inhibitors; other abbreviations as in Table 1.

geographic outcomes during the follow-up period are presented in Table 7.

Discussion

The main findings of this paper are: 1) the incidence of grade III coronary perforation was extremely rare; 2) predictors of grade III coronary perforation were complex coronary lesions, coronary occlusions, and the use of rotablation and IVUS; 3) multiple methods of treatment are available, but prolonged balloon inflation and covered stent implantation were successful in a reasonable proportion of cases; 4) despite improvements in the treatment of grade III coronary perforation, rates of MII and mortality remained high; 5) the occurrence of grade III coronary perforation following administration of GPI was associated with increased procedural and in-hospital MACE rates; and 6) from 1993 to 2009, the incidence of grade III coronary perforation remained relatively unchanged, but there was an improvement in procedural and in-hospital MACE rates (Fig. 2).

Grade III coronary perforation is a feared and dramatic complication of PCI with poor immediate outcomes and very high mortality rates. It remains a rare event with an incidence of 0.23% in our centers in the context of high volumes of PCI. Over time, the number of cases of grade III coronary perforation has remained constant, as a likely

reflection of the increased complexity of procedures. However, even though the combined procedural and in-hospital MACE rate is high at 55%, there has been some improvement over time since the introduction of techniques such as covered stent implantation. Despite the available treatments, these methods are often unsuccessful in achieving hemostasis and multiple modalities are frequently required. Notably, these outcomes were achieved in centers with experienced operators, and success rates may be even less favorable in the hands of less experienced operators. From our study, the commonest cause of grade III coronary perforation was inflation of an intracoronary balloon, with no change in the incidence based on the balloon compliance. Grade III coronary perforation was caused by rotablation, directional atherectomy, and cutting balloon inflation in only a small number of cases; however, this figure may be influenced by the low rates of use of these adjunctive techniques in our interventional practice. Interestingly, in cases of perforation due to an intracoronary guidewire, the wire was nonhydrophilic in the majority, in contrast to previous reports (18). However, as the total numbers of cases were relatively small this finding may be a reflection of the fact that our first-line choice of guidewire is uncoated. An assessment of the predictors of grade III coronary perforation showed that this adverse event was associated

Table 7. Long-Term Outcome During the Follow-Up Period

Follow-up	46
Months	38.1 (7.6–122.8)
Months of dual antiplatelet therapy	1.0 (0–6.0)
Angiographic follow-up obtained	26 (56.5)
Angina CCS class	
Unstable angina (CCS IV)	0
Stable angina (CCS I–III)	2 (4.3)
Asymptomatic (CCS 0)	44 (95.7)
Restenosis	10 (38.4)
Death following discharge	7 (15.2)
Cardiac death	3 (6.5)
Myocardial infarction	2 (4.3)
Need for CABG	2 (4.3)
Target lesion revascularization	6 (13.0)
Target vessel revascularization	9 (19.6)
Stent thrombosis	4 (8.6)
Major adverse cardiac event	19 (41.3)

Data presented as percentages and absolute numbers, mean \pm SD, or median (interquartile range).
Abbreviations as in Table 1.

with complex coronary lesions (type B2 or C lesions), coronary occlusions, and the use of rotablation or IVUS during the procedure. In addition, the incidence of definite ST was relatively high at 8.6%; all of these cases were associated with covered PTFE stent implantation and illustrates the thrombogenic nature of these stents, in combination with the increased risk of ST conferred by coronary perforation. This finding highlights the need for less thrombogenic stents for the treatment of coronary perforation.

Previous studies have focused on an analysis of a combined group of all 3 grades of coronary perforation and have suggested that this complication is more common during PCI to type C lesions (2,9); this was confirmed by our study, with a predominance of complex type B2 or C lesions. It has also been suggested that coronary perforation is more prevalent during coronary intervention in females (2); however, sex was not a predictor of grade III coronary perforation in our study. Shimony et al. (9) reported that the incidence of coronary perforation may be associated with intervention to the right coronary artery and attributed this to the tortuous course of this vessel; however, in our study, anatomic lesion location was not a predictor of perforation. This may be a result of differences between the characteristics of vessels with grade III coronary perforation compared with those with less severe forms of perforation. Both the use of rotablation and IVUS were predictors of grade III coronary perforation in our study, reflecting the association between this adverse event and a more complex lesion subset. Notably, the use of IVUS was shown to be associated with coronary perforation in a previous study by our group and is likely to be a reflection of the fact that IVUS is more likely to be used in complex lesions or those in which PCI is complicated, for example, by balloon underexpansion during pre-dilation (19). Regarding treatment of grade III coronary perforation, it appears that we still do not have an ideal modality to treat this dramatic complication despite reported promising outcomes following covered stent implantation and microcoil embolization (20,21). Even with newer treatment methods, multiple treatment modalities were still required in 39% and although successful hemostasis was achieved in 88%, our combined procedural and

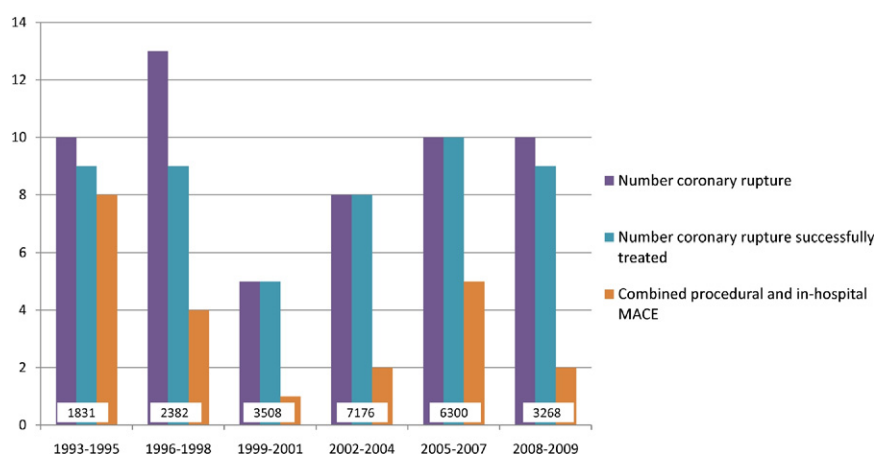


Figure 2. Incidence, Treatment, and Outcomes of Grade III Perforation From 1993 to 2009

Graph showing number of cases of grade III coronary perforation, number of cases of successfully treated, and combined in-hospital and procedural major adverse event rates over each 2- to 3-year period from 1993 to 2009. **Figures in white boxes** represent total number of percutaneous coronary intervention procedures performed within each period. MACE = major adverse cardiac event.

in-hospital mortality rate remained high at 18%, in line with previous reported mortality rates of 7% to 44% in grade III perforation (9–12). Furthermore, the rates of in-hospital MI remained very high at 43%. Based on our experience of the treatment of grade III coronary perforation, we present a proposed treatment flowchart that can be considered in the instance of this adverse event (Fig. 3).

This study also showed that grade III coronary perforation in conjunction with GPI administration was associated with an even greater risk of adverse events. The combined procedural and in-hospital MACE rate in this group was higher than that of the overall group at 90%. In addition, multiple treatment modalities were required in 50% in an attempt to achieve hemostasis with successful treatment of the rupture in 80%. Previous studies have also suggested that the use of GPI may be associated with worse short-term outcomes in patients with coronary perforation and that these agents should be used with caution in high-risk procedures (5).

Study limitations. There are some limitations of this study: 1) it was a retrospective study; 2) the population size was

relatively small; 3) angiographic follow-up was not performed in all patients; and 4) it was a descriptive study with no control group, but an attempt to match according to patient or lesion characteristics to such a specific population may be inaccurate and misleading. Previous reports of coronary perforation during PCI have also assessed relatively small numbers of patients, as it is a rare event.

Conclusions

Fortunately, grade III coronary perforation remains a rare complication of coronary intervention, with an incidence that may be rising due to increased complexity of cases in current practice and widespread use of GPI. Predictors of grade II coronary perforation are complex coronary lesions, coronary occlusions, and the use of rotablation and IVUS. An interventional cardiologist must be prepared for this iatrogenic event; all teams should be equipped with the necessary skills and technology required for treatment and should be prepared to react quickly and efficiently in the event of perforation. Despite treatment measures, this

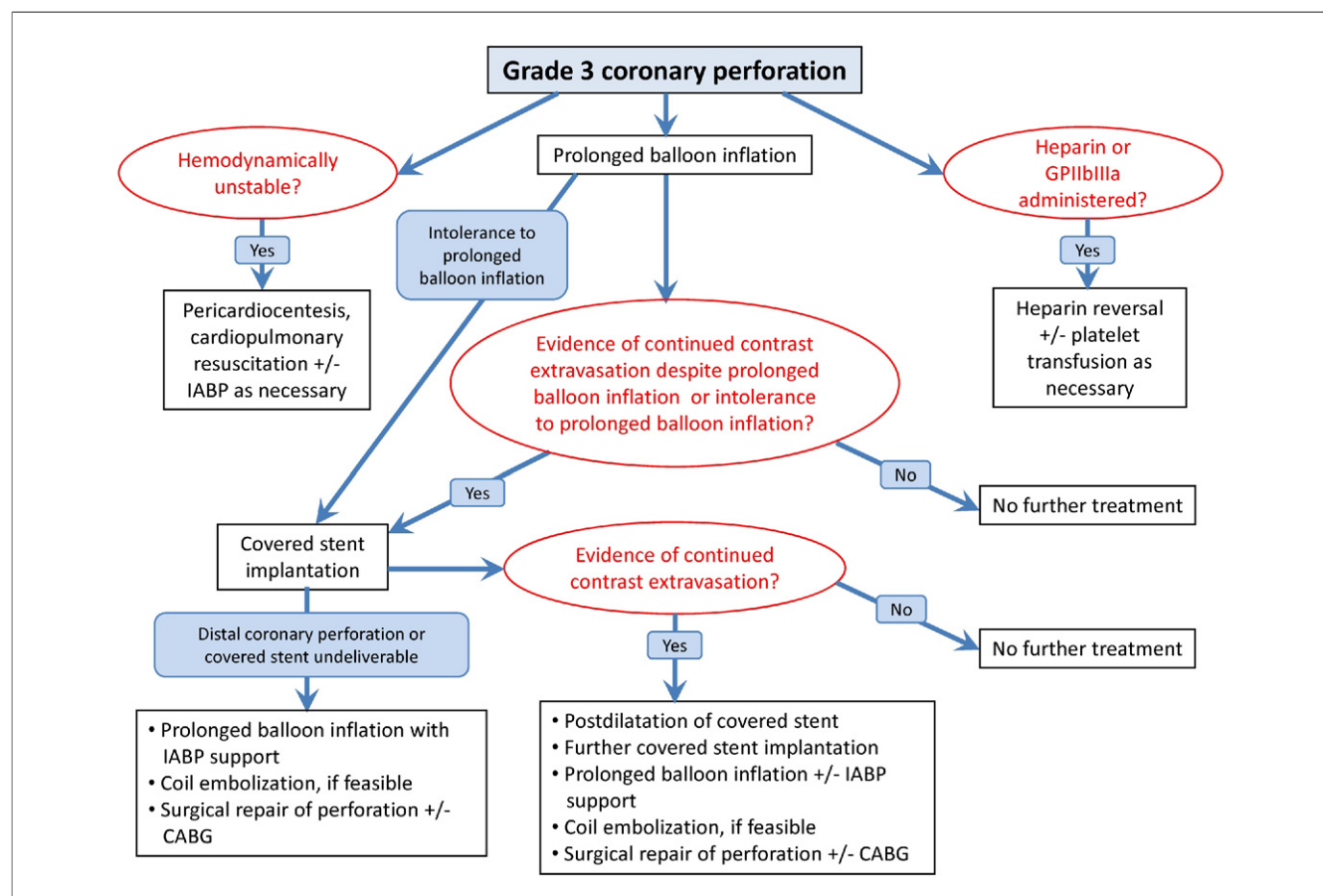


Figure 3. Flowchart for the Treatment of Grade III Coronary Perforation Based on Our Experience

An algorithm for the management of grade III coronary perforation based on our experience of the most successful and appropriate methods of treatment. CABG = coronary artery bypass graft; GPIIb/IIIa = glycoprotein IIb/IIIa inhibitors; IABP = intra-aortic balloon pump.

complication is still associated with poor adverse outcomes and there remains a need for improved technology to treat this dreaded complication.

Reprint requests and correspondence: Dr. Antonio Colombo, EMO-GVM Centro Cuore Columbus Hospital, Via Buonarroti 48, 20145 Milan, Italy. E-mail: info@emocolumbus.it.

REFERENCES

1. Gruberg L, Pinnow E, Flood R, et al. Incidence, management, and outcome of coronary artery perforation during percutaneous coronary intervention. *Am J Cardiol* 2000;86:680–2, A8.
2. Fasseas P, Orford JL, Panetta CJ, et al. Incidence, correlates, management, and clinical outcome of coronary perforation: analysis of 16,298 procedures. *Am Heart J* 2004;147:140–5.
3. Ellis SG, Ajluni S, Arnold AZ, et al. Increased coronary perforation in the new device era. Incidence, classification, management, and outcome. *Circulation* 1994;90:2725–30.
4. Bittl JA, Ryan TJ Jr., Keaney JF Jr., et al. Coronary artery perforation during excimer laser coronary angioplasty. The percutaneous Excimer Laser Coronary Angioplasty Registry. *J Am Coll Cardiol* 1993;21:1158–65.
5. Gunning MG, Williams IL, Jewitt DE, Shah AM, Wainwright RJ, Thomas MR. Coronary artery perforation during percutaneous intervention: incidence and outcome. *Heart* 2002;88:495–8.
6. Ramana RK, Arab D, Joyal D, et al. Coronary artery perforation during percutaneous coronary intervention: incidence and outcomes in the new interventional era. *J Invasive Cardiol* 2005;17:603–5.
7. Hennessy TG, McCann HA, Sugrue DD. Fatal stent thrombosis following successful treatment of coronary artery rupture in an octogenarian. *Cathet Cardiovasc Diagn* 1997;42:434–6.
8. Reimers B, von Birgelen C, van der Giessen WJ, Serruys PW. A word of caution on optimizing stent deployment in calcified lesions: acute coronary rupture with cardiac tamponade. *Am Heart J* 1996;131:192–4.
9. Shimony A, Zahger D, Van Straten M, et al. Incidence, risk factors, management and outcomes of coronary artery perforation during percutaneous coronary intervention. *Am J Cardiol* 2009;104:1674–7.
10. Javadi A, Buch AN, Satler LF, et al. Management and outcomes of coronary artery perforation during percutaneous coronary intervention. *Am J Cardiol* 2006;98:911–4.
11. Roguin A, Beyar R. Coronary perforation 2006—watch for the wire. *J Invasive Cardiol* 2005;17:606–8.
12. Fukutomi T, Suzuki T, Popma JJ, et al. Early and late clinical outcomes following coronary perforation in patients undergoing percutaneous coronary intervention. *Circ J* 2002;66:349–56.
13. Dorros G, Jain A, Kumar K. Management of coronary artery rupture: covered stent or microcoil embolization. *Cathet Cardiovasc Diagn* 1995;36:148–54, discussion 155.
14. Reinecke H, Fetsch T, Roeder N, et al. Emergency coronary artery bypass grafting after failed coronary angioplasty: what has changed in a decade? *Ann Thorac Surg* 2000;70:1997–2003.
15. Cutlip DE, Windecker S, Mehran R, et al., for Academic Research Consortium. Clinical end points in coronary stent trials: a case for standardized definitions. *Circulation* 2007;115:2344–51.
16. Serruys PW, Strauss BH, Beatt KJ, et al. Angiographic follow-up after placement of a self-expanding coronary-artery stent. *N Engl J Med* 1991;324:13–7.
17. Hosmer DW, Hosmer T, Le Cessie S, Lemeshow S. A comparison of goodness-of-fit tests for the logistic regression model. *Stat Med* 1997;16:965–80.
18. Wong CM, Kwong Mak GY, Chung DT. Distal coronary artery perforation resulting from the use of hydrophilic coated guidewire in tortuous vessels. *Cathet Cardiovasc Diagn* 1998;44:93–6.
19. Stankovic G, Orlic D, Corvaja N, et al. Incidence, predictors, in-hospital, and late outcomes of coronary artery perforations. *Am J Cardiol* 2004;93:213–6.
20. Briguori C, Nishida T, Anzuini A, Di Mario C, Grube E, Colombo A. Emergency polytetrafluoroethylene-covered stent implantation to treat coronary ruptures. *Circulation* 2000;102:3028–31.
21. Gaxiola E, Browne KF. Coronary artery perforation repair using microcoil embolization. *Cathet Cardiovasc Diagn* 1998;43:474–6.

Key Words: coronary intervention ■ coronary perforation ■ management ■ percutaneous.