REVIEW

Percutaneous Coronary Intervention Complications: Where do we Stand?

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Abstract

Coronary angioplasty (or percutaneous coronary intervention, PCI) is a mature and widely diffused treatment technique for coronary artery disease. Over the last decades, great evolution has been realized concerning the related technology, the pharmacologic armamentarium and operators' experience resulting in improved safety and success rates of PCI. Despite the fact that associated risks have declined over time, since PCIs are invasive procedures, complication rates have always been and still are a vexing reality. They concern the cardiologist who sets the indication, the interventionalist who performs the procedure but most importantly the patient who should be well informed for the anticipated benefits and risks before giving his written informed consent. A concise update on recent data about the most important issues regarding PCI complications is attempted herein.

Key Words: percutaneous coronary intervention; complications

Abbreviations: BARC = Bleeding Academic Research Consortium; CIN: contrast-induced nephropathy; NCDR = National Cardiovascular Data Registry; PCI = percutaneous coronary intervention; STEMI: ST-segment elevation myocardial infarction

Introduction

Any percutaneous coronary intervention (PCI) is a procedure with expected benefits but also with potential risks. As with any invasive procedure there should be a clear indication that justifies even the smallest possible risk.[1] Procedural success and complication rates are used to measure outcomes after PCI. Procedural success is defined as angiographic success without the occurrence of major complications (death, myocardial infarction or cardiac surgery) within 30 days of the procedure. Clinical success is defined as procedural success without the need for urgent repeated PCI or surgical revascularization within the first 30 days of the procedure.[2] Several clinical, angiographic, and technical variables predict the risk of procedural failure in patients undergoing PCI. Major complications include death, myocardial infarction and stroke, while minor complications include transient ischemic attacks, vascular complications, contrast-induced nephropathy (CIN) and some angiographic complications.[2] Substantial improvements in coronary devices, adjunctive antithrombotic therapy and secondary prevention after PCI have significantly improved early and late clinical outcomes after PCI over time.[3]

Morbidity and mortality after PCI undoubtedly have been decreasing during the last two decades while minor incidents were probably underestimated.[4, 5] However, complication rates remain vexing and vary among various recent reports, depending mainly on the characteristics of the population under study.

Mortality

Although mortality after PCI is generally rare (<1%), it depends on the clinical profile of each patient and thus mortality risk models have been previously developed to facilitate patient-level management (guiding therapeutic decisions and informed consent) but also to be applied for quality of care assessment and clinical research. Data from the National Cardiovascular Data Registry (NCDR) have been used to develop and validate PCI risk models including a "full" model of 21 variables with angiographic details, a pre-catheterization model without angiographic details and most importantly a simplified bedside risk score of 8 variables.[6] As has been indicated in the recently developed EuroHeart PCI score, in-hospital mortality among PCI patients can be well predicted by a risk estimation model that contains 16 variables.[4] This is the most contemporary risk score available that has been validated in a large European population (two data sets of >20000 patients each) and where 3 clinical factors have been demonstrated as the strongest predictors of peri-procedural mortality: age >80 years, hemodynamic instability and primary PCI for STEMI.[4] Results from more than one million procedures included in the NCDR contributed to an updated CathPCI Registry mortality model that was reported recently.[5] In-hospital mortality was 1.4%, ranging from 0.2% among elective cases to 65.9% among patients with shock and recent cardiac arrest. Table 1 demonstrates in detail how the NCDR (initial and newest) risk scores and the EuroHeart PCI risk score are calculated and Table 2 the above respective scores that predict several in-hospital mortality levels.

It might be more useful though to consider cardiovascular complications and importantly all-cause mortality early after PCI, extending at the post-discharge period, which by consensus is measured at 30 days after intervention. In the New York state PCI registry the overall complication rate was 3.36% with a mortality rate of 0.6% at one month and 0.047% in the catheterization laboratory.[7] Cardiogenic shock and procedure urgency were the most predictive of inpatient mortality, whereas the presence of a chronic total occlusion, subacute stent thrombosis and left main lesion location were significant

angiographic predictors.[5] Generally prognosis worsens with increasing clinical acuity: in-hospital mortality is higher in the setting of STEMI, in cardiogenic shock and in patients who develop an occlusion with prior poor left ventricular function, while patients of advanced aged and acute coronary syndromes are particularly vulnerable.[2] Data from the British Columbia Cardiac Registry have led to the identification of several risk factors for 30-day mortality prediction after PCI in order to construct a risk score model.[8] In recent randomized controlled studies early mortality is low and for patients with stable angina it varies between 0.3 and 1%.[9-12]

The evolution towards death after PCI is not necessarily of cardiac origin since the mechanisms that lead to major events are multiple and interrelated. In a recent report coming from a registry of >5000 patients, in-hospital mortality was found at 1.5%. Left ventricular failure was the most common cause of death (35.3%), followed by neurological compromise (16.5%) and arrhythmia (12.1%). Of note, procedural complications were responsible for a small fraction of deaths (7.1%) and reviewers determined 93% of deaths to be mostly or entirely unpreventable.[13]

Myocardial infarction

The most frequent mechanisms are the embolization of atherothrombotic fragments and the occlusion of side branches or collateral branches after stent implantation. The most serious complication is an occlusive coronary dissection with failure to cross for stent delivery.

Myocardial infarction is probably the most imperfectly determined complication among PCI registries since definitions have been varying for the last two decades and comparison between studies would therefore be futile. According to the latest third universal definition of myocardial infarction the diagnosis of a myocardial infarction related to PCI (type 4a) is arbitrarily made in case of a rise of troponin more than five times above the upper 99th percentile of the normal values in patients with normal baseline values or a rise of troponin values more than 20% if the baseline values have been initially elevated and are stable or falling.[14] In addition, either (i) symptoms suggestive of myocardial ischemia, or (ii) new ischemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.[14] According to this latest definition a recent study reported rates of PCI related myocardial infarction around 4%, which seems a correct estimation.[15] However it should be noted that a threshold for a worsening prognosis, related to an asymptomatic increase of cardiac biomarker values in the absence of procedural complications is not yet determined due to still unconvincing data.[16, 17]

Stent thrombosis

A subcategory of PCI-related myocardial infarction is stent thrombosis, as documented by angiography and/or at autopsy and a rise and/or fall of troponin values (myocardial infarction type 4b).[14] According to the Academic Research Consortium recommendation the occurrence of stent thrombosis should be stratified in relation to the timing of the PCI procedure as “early” (0 to 30 days), “late” (31 days to 1 year), and “very late” (>1 year) to distinguish likely differences in the contribution of the various pathophysiological processes during each of these intervals.[18]Early stent thrombosis is very often related to technical PCI issues, such as imperfect stent deployment or residual dissection (example in Figure 1). Stent thrombosis risk remains high especially during the first 15 days following stent implantation having as predisposing risk factors a context of acute coronary syndrome (especially STEMI), premature antiplatelet treatment discontinuation and clopidogrel resistance (genetic or other).[19]

The most potent available antiplatelet drugs - ticagrelor and prasugrel - have contributed in the reduction of acute stent thrombosis rates, which is estimated between 0.3% and 0.8% in recent studies.[9-11] For the moment according to the guidelines they are preferentially used in acute coronary syndrome cases, while clopidogrel should be prescribed for elective PCI.[20]

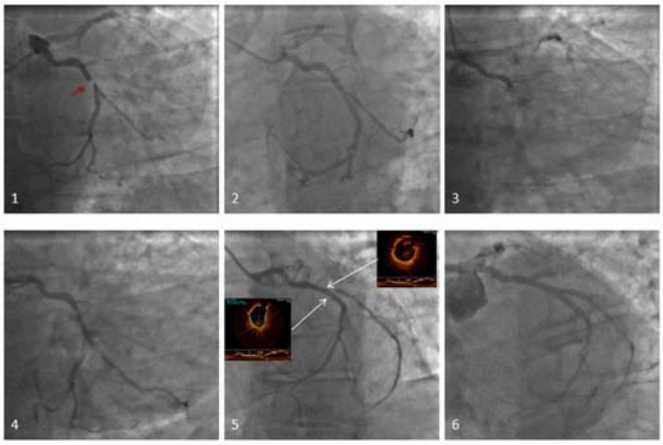

Figure 1. A subacute stent thrombosis case explained by technical issues regarding PCI. 1) A subocclusive, thrombotic lesion of a dominant circumflex artery at the level of the first marginal branch is the culprit lesion for a non ST-elevation acute coronary syndrome. The marginal branch has also significant ostial-proximal disease, 2) PCI was performed with direct stenting using a 3x15 mm stent and no bifurcation technique while the disease at the origin of the first marginal was not

addressed, 3) Stent thrombosis 5 days later, 4) PCI for stent thrombosis with only thromboaspiration (10 runs) and adjunctive abciximab intracoronary bolus and IV infusion with restoration of TIMI III flow but still visible residual thrombus fragments, 5) After 10 days of intensified dual antiplatelet (clopidogrel replaced by prasugrel) and antithrombotic therapy there is no angiographic thrombus. With optical coherence tomography a small proximal residual thrombus is discovered and the stent is found underexpanded and undersized (3mm stent for a 3.5 mm artery diameter), 6) Final result after PCI for the circumflex stent optimization and additional stenting for the marginal ostium-proximal disease using a bifurcation technique.

Coronary perforation

It is a very rare but life-threatening complication with an incidence that is not well determined. In a study of more than 7000 procedures the incidence of perforation was 0.93% and almost two thirds of them occurred during PCI for chronic total occlusion.[21] The two possible mechanisms are well identified:

- Coronary artery rupture, usually upon lesions calcified and resistant. It can be caused by oversized balloons or stents, balloon rupture, aggressive stent post-dilatation or excessive rotablation.
- Distal perforation, usually in cases of chronic occlusions and/or severe coronary tortuosity. It is caused by guidewire exit, especially with the use of stiff and hydrophilic guidewires.

Both mechanisms can lead to rapidly accumulating pericardial effusion with cardiac tamponade and require emergency pericardiocentesis. The most important countermeasures are graft stents in case of coronary rupture and distal embolization with various interventional radiology techniques (usually with thrombogenic coils) in case of distal perforation. Necessary material should be readily available in the catheterization laboratory. Emergency cardiac surgery is the ultimate solution in case of failure of the above.

Emergency cardiac surgery

The need for emergency surgery to bail-out a complication has decreased dramatically over the years and it is quite rare nowadays, in 0.61% of PCI procedures according to a recent report from a large US registry (>18000 procedures, between 1992 and 2000).[22] It is indicated mainly for an extensive coronary dissection (61%), perforation/tamponade (20%) or a recurrent coronary artery occlusion (20%). In even rarer cases (1-2%) it may be needed because of iatrogenic aortic dissection or the impossibility to retrieve devices and material used during PCI.[22]

Cerebrovascular accident

Stroke is a rare but possibly dramatic complication of PCI because of functional consequences. In the PCI Registry of the Euro Heart Survey periprocedural stroke was observed in 0.4% of all PCI procedures, in 0.3% of PCIs in elective patients and in 0.6% in PCIs performed for ACS. The overall in-hospital mortality was 19.2% for patients who developed stroke compared with 1.3% for those without stroke. Hemodynamic instability, age ≥ 75 years, history of stroke, and congestive heart failure were found to be independent predictors for periprocedural stroke in acute coronary syndromes, whereas only PCI of a bypass graft and renal failure could be identified as independent predictors in elective patients. That means that most predictors for periprocedural stroke are not modifiable and cannot be diminished before PCI.[23] This makes sense since catheter manipulation is inevitable while at the same time risk factors for coronary, cerebrovascular and aortic atherosclerotic disease overlap.

Hemorrhagic complications

They are divided in two categories according to their timing.

Immediate, in-hospital hemorrhagic complications. They largely depend on the arterial access site, since the radial approach reduces by 50% major hemorrhagic complications, especially in acute coronary syndromes.

The radial approach reduces bleeding complications and is especially advantageous in certain patient subgroups (those with obesity, anticoagulation, haemostasis disorders, chronic pulmonary disease).[24] Moreover, the results from the recently reported RIVAL study did not demonstrate a mortality advantage for an all-comer radial approach among patients undergoing PCI, except for the primary PCI subgroup which did demonstrate a mortality benefit.[25] This is why in the latest European myocardial revascularization guidelines the radial approach holds a class IIa indication for primary PCI provided that it is performed by an experienced radial operator.[20]

The other half of major bleeding complications is not related to the access site and is associated with 3 times higher 1-year mortality when the Bleeding Academic Research Consortium (BARC) universal classification is applied.[26] The causal relationship as a mechanism linking major bleeding complications and mortality remains for the moment elusive.

Late hemorrhagic complications. They are underreported because they appear after hospitalization. They are related to oral anticoagulants and the more potent antiplatelet agents. According to current data and guidelines only clopidogrel, but not the more potent ticagrelor or prasugrel, should be used when a triple association of a vitamin K antagonist, aspirin and a P2Y12 inhibitor is indicated after stenting for patients with atrial fibrillation or other indication for chronic oral

anticoagulation, which makes sense considering the bleeding risk.[20] The abovementioned triple association should be of the most limited period possible since it doubles major bleeding events rates that are related to increased mortality.[27, 28] Actually some emerging data support the discontinuation of clopidogrel at three months after newer generation DES implantation for patients with stable coronary artery disease or low-risk acute coronary syndrome.[10]

Vascular access site complications

Regarding the femoral approach one should have in mind the most dreaded complication which is the retroperitoneal hemorrhage. Recently reported data from a large registry (>110000 patients) inform us that retroperitoneal hemorrhage is rare (0.4%) after PCI. It is usually related to high femoral puncture but it can occur even with an ideal puncture site due to movement of blood along fascial planes. The vast majority is treated medically (>90%) but there is significant mortality (6-7%) associated with this complication.[29] The other two important femoral access complications that sometimes need to be treated by vascular surgery, are the pseudoaneurysm and the arteriovenous fistula. Ischemic complications such as arterial dissection, thrombosis or distal embolism are also possible but rare and related to preexisting peripheral arterial disease. The predictive factors of femoral access related complications are female gender, advanced age, very low body surface area, diabetes mellitus and anticoagulant treatment.

The radial approach has been gaining popularity among interventionalists since it is practically free of the above complications and furthermore the risk of significant hematoma is very low. The most frequent radial access related complication is radial artery thrombotic occlusion (3-5% at 30 days) which is usually clinically silent. Such advantages have led to an increasing adoption of the technique by interventional cardiologists. According to a recent report from the NCDR the proportion of transradial PCI procedures has increased from 1.2% in 2007 to 16.1% in 2012 and accounted for 6.3% of total procedures from 2007 to 2012.[30]

Contrast-induced nephropathy

Contrast-induced nephropathy (CIN) is a common complication among patients undergoing PCI and is related to increased morbidity, mortality, and healthcare costs. Several efforts have been made to develop risk prediction tools to identify patients most likely to develop CIN. The most commonly used risk score was described by Mehran et al. and is based on the presence of 8 factors which are mainly pre-procedural but also two procedural (hypotension, use of intra-aortic balloon pump, congestive heart failure, chronic kidney disease, diabetes, age >75 years, anemia and volume of contrast).[31] A novel risk prediction algorithm including only pre-procedural clinical and laboratory variables has been recently developed and proposed to calculate the risk of CIN (and new requirement for dialysis) among patients undergoing PCI.[32] This risk score may prove useful for clinical decision making as well as quality of care assessment, and can be reliably calculated at bedside and before the procedure using a novel easy-to-use computational tool available online (https://bmc2.org/calculators/cin) and in a smartphone application (named SCAI PCI Risk Calculator).

Multiple strategies have been demonstrated to be successful for prophylaxis of CIN and are recommended in the latest European revascularization guidelines, including adequate hydration with isotonic saline (class I-A) and the use of iso-osmolar or certain low osmolar contrast media with minimization of contrast dose at <350ml or <4ml/kg or total contrast volume/glomerular filtration ratio <3.4 (class I-A).[20] The implementation of high-dose statin before catheterization has been shown to reduce the incidence of CIN and should be considered as an additional preventive measure in patients without contraindications (class IIa-A).[20, 33] Of note, the effectiveness of other pharmacological preventive measures such as N-acetylcysteine, sodium bicarbonate 0.84% infusion (class III-A for both) or prophylactic pre-procedural hemodialysis -remains unproven (class III-B).[20]

Preparedness for complications

Mortality related to PCI complications is low and is practically unchanged for the last decade. Despite the improvement of techniques and devices this could be explained by the parallel increasing complexity of cases that are attempted. Patients should be well informed for the benefits and risks of each procedure and give written informed consent. Each operator should acknowledge his limits, evaluate the risks, anticipate technical difficulties and plan every single intervention accordingly. The catheterization laboratory should have all the necessary equipment (i.e. graft stents, coils, pericardiocentesis kit, etc.) readily available to confront any possible complication. All the above are necessary in order to be able to prevent complications and save the day when they arrive.

Table 1. NCDR and EuroHeart PCI risk scores for in-hospital mortality.

NCDR PCI score[6]		NCDR PCI score (newest)[5]		EuroHeart PCI score[4]	
Variable	**Points**	**Variable**	**Points**	**Variable**	**Points**
Age		**Age**		**Age**	
<60	0	<60	0	<60	0
60-69	4	60-69	4	60-69	2
70-79	8	70-79	9	70-79	3
≥80	14	≥80	15	≥80	6
Cardiogenic shock*	25	**STEMI***	6	**Valvular heart disease***	2
Prior CHF*	5	**CVD***	2	**Prior stroke***	2
PAD*	5	**PAD***	3	**Female gender***	2
Chronic lung disease*	4	**Chronic lung disease***	3	**Ever smoker***	1
GFR		**GFR**		**CAD severity**	
<30	18	<30	16	Left main disease	3
30-60	10	30-45	11	3-vessel disease	1
60-90	6	45-60	7	Proximal LAD disease	2
>90	0	60-90	3	Bifurcation lesion	2
		>90	0	Type-C lesion	2
		NYHA class within 2 weeks			
NYHA class IV*	4	NYHA IV	7		
		NYHA <IV	3		
		No heart failure	0		
		CS/PCI status			
PCI status (STEMI)		Sustained shock and salvage	54	**PCI indication**	
Elective	12	Sustained shock or salvage	43	Ongoing STE-ACS	8
Urgent	15	Transient shock, no salvage	37	Ongoing non STE-ACS	6
Emergent	20	Urgent (no shock/salvage)	22	Stabilized after ACS	4
Salvage	38	Emergent (no shock/salvage)	11		
		Elective (no shock/salvage)	0		
		LVEF			
PCI status (no STEMI)		<30	9	**Hemodynamic instability**	
Elective	0	30-39	4	Yes	11
Urgent	8	40-49	2	No	0
Emergent	20	≥50	0		
Salvage	42				
		Prior PCI*	3		
				No prior CABG*	4
		Cardiac arrest (<24 hours)*	13	**TIMI flow 0/1 before PCI***	2
		Diabetes mellitus			
		Insulin	3	**Diabetes mellitus**	
		No insulin	2	Yes	3
		No	0	No	0
		BMI (kg/m^2)			
		<20	5	**BMI (kg/m^2)**	
		20-29	1	<25	2
		30-39	0	≥25	0
		≥40	3		

STEMI, ST-elevation myocardial infarction; CHF, congestive heart failure; CVD, cardiovascular disease, PAD, peripheral arterial disease, GFR, glomerular filtration ratio; CAD, coronary artery disease; NYHA, New York Heart Association; LVEF, left ventricular ejection fraction; CABG, coronary artery bypass grafting; BMI, body mass index. *No points (0) if characteristic not present.

Table 2. NCDR and EuroHeart PCI respective scores that predict certain in-hospital mortality levels.

In-hospital mortality (%)	NCDR PCI score[6]	NCDR PCI score (newest)[5]	EuroHeart PCI score[4]
1	29	35	14
3	38	47	20
5	43	53	24
10	49	61	26
15	53	66	28
20	56	69	30
40	64	78	33
>50	>68	>84	>35

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