

Predictors of Procedural Complications in Coronary Interventions and their Impact on Prognosis: Mini Review

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ABSTRACT

Coronary interventions have become an integral part of the modern treatment of patients with coronary artery disease. Due to their invasive nature however, these interventions carry a potential risk of certain procedural complications such as coronary perforation, dissection, no reflow phenomenon, or even myocardial infarction. The occurrence of any complication may influence the immediate procedural result and the short/long-term morbidity and mortality. Over the last four decades, potential predictors of these complications have been identified. Despite the fact that several risk score models have been suggested, no single risk model has ever been significantly validated in large cohorts.

Coronary perforation increases substantially the mortality and MACE rate, in both acute and chronic cases. Typical risk factors include female sex, advanced age and aggressive high-pressure balloon inflation. No reflow is usually associated with advanced age, arterial hypertension, smoking, dyslipidemia, and renal failure. Among predictors of coronary dissection, factors such as female gender, multivessel disease, and complex coronary anatomy with proximal tortuosity and longer lesions, have been identified as potential risk factors. All these complications can lead to procedural myocardial infarction with negative impact on patients' outcomes.

Keywords: Coronary interventions; Bypass surgery; Myocardial infarction; Coronary dissection; Coronary perforations

Abbreviation: cTn: cardiac Troponin; CTO: Chronic Total Obstruction; EF: Ejection Fraction; ESC: European Society of Cardiology; MACE: Major Adverse Cardiovascular Events; PCI: Percutaneous Coronary Intervention; STEMI: ST Elevation Myocardial Infarction; TIMI: Thrombolysis in Myocardial Infarction; URL: Upper Reference Limit; UD: Fourth Universal Definition of Myocardial Infarction; SCAI: Society Cardiovascular Angiographic Interventions; ARC 2: Academic Research Consortium 2; ULN: Upper Limit of Normal; URL: Upper Reference Limit

INTRODUCTION

Modern treatment of patients with acute and chronic coronary disease features conservative and invasive aspects. Whereas the conservative approach focuses on the use of typical cardiac medications, it is the interventional approach that has gained increasing importance due to constant development of catheters, balloons, stents, and special dedicated devices. Since their introduction by Gruentzig in 1978, percutaneous coronary interventions have been associated with procedural complications. Throughout the last few decades however, the risk of complications has decreased substantially. The current risk of mortality, periprocedural myocardial injury or urgent transfer for bypass surgery is 1.5% to 1.7%, 1.5% and 0.2% respectively, in both elective and acute interventions [1]. The proper management of typical complications has been well established over the years and has become a significant contributor to the overall success of every interventional cardiologist [2]. As the most essential aspect of handling complications is apparently avoiding them, it is of utmost importance to detect possible predictors of complications. The patients' overall outcomes, both short and long term, may be worsened by complications [3]. This article will review selected

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coronary complications, and discuss their predictors and possible impact on prognosis. The management of complications is beyond the scope of this paper and can be found elsewhere.

RISK STRATIFICATION AND RISK SCORES

To predict in-hospital mortality and complications of coronary interventions, a lot of risk models have been developed in the past. The Mayo Clinic, for instance, suggested a predictive risk score based on eight variables (Table 1), which was able to assess the risk of in-hospital mortality and some major complications such as myocardial infarction, stroke, or transfer to urgent bypass surgery [4]. In a subsequent study, the researchers from the Mayo Clinic improved the risk model for both short mortality and complications but restricted it to lower-risk patients and no multicentre validation was performed [4]. Presently, no validated single risk score can be recommended to evaluate the risk of procedural complications in coronary interventions (Table 1) [2].

Table 1: Mayo clinic risk score (additive).

Points
6
5
4
3
2
1
5
5
3
2
2
2
2

Interpretation

0-5 very low risk (<2%*), 6-8 low risk (>2-5%*), 9-11 moderate (>5-10%*), 12-14 high risk (>10-25%*), >15 very high risk (>25%*).

CORONARY COMPLICATIONS

Coronary perforation

The incidence of this serious complication ranges from 0.19 to 3.0%, as it was observed in the NCDR Cath PCI Registry [5]. Lesions more complex in nature (type B or C, calcified lesions, CTOs) are more likely to sustain perforation. Female sex and advanced age have been identified as potential risk factors as well [5,6]. Basically, there are two mechanisms of perforation: Guidewire penetration and vessel rupture (usually caused by balloon or stent mismatch with oversizing). The early studies suggested that increasing the balloon: Artery ratio greater than 1.2:1 enhanced perforation risk. Implementation of debulking devices such as rotational atherectomy also increases perforation risk [6]. The special case of CTO recanalization has set a typical environment for perforation due to highly aggressive dilatation and use of devices in

the subintimal space.

Ellis classified coronary perforations into three classes (Table 2). The Grade II and III perforations are usually caused by high-pressure inflations, oversized balloon catheters, the use of debulking devices, or highly complex interventions in the subintimal space during CTO procedures [7].

Table 2: Modified Ellis classification of coronary perforations.

		~1
Class	Description	Clinics
Type I	Extra luminal crater without extravasation	Almost always benign, treated safely and effectively with a stent
Type II	Blush (pericardial or myocardial) without extravasation and without a>1 mm exit hole	Common results in late tamponade, needs close atten-tion
Type III	Extravasation with a>1 mm exit hole	High risk of tamponade
III A	Directed towards pericardium	High risk of tamponade
III B	Directed towards myocardium	Usually more benign
Type III Perforation into an isolated cavity chamber such as coronary sinus spilling (Type IV)		Often benign, may cause fistulae formation
Type V	Distal guide wire perforation	High risk of tamponade

Each perforation may influence the overall outcome. Grade II and III perforations can lead to cardiac tamponade with rapid hemodynamic deterioration and increased mortality. This is associated with a 7.1% increase rate of death and a 25.9% increase rate of Major Adverse Cardiac Events (MACEs) [5,7].

No reflow/slow reflow

Coronary no reflow has been described as the lack of myocardial perfusion after opening a previously closed or stenosed epicardial artery. According to the most common angiographic definition, it is TIMI<3 flow after interventional treatment occurring in the absence of vessel obstruction, spasm, or dissection [3]. The incidence has been reported to range from 0.6%-2.3% (among STEMI patients in the CathPCI Registry) [8]. The exact mechanism has not been fully recognized so far, but structural and functional alterations in microcirculation have been suggested. This, in turn, seems to be the result of distal embolization, ischemic and reperfusion injury, and probably individual susceptibility. Typical patient-based predictors are advanced age, arterial hypertension, smoking, dyslipidemia, renal failure, and pro-inflammatory chronic conditions [9]. In case of myocardial infarction with ST segment elevation, it is late presentation (>6 hours) that predicts the no reflow. Among the procedural findings, common predictors are TIMI<2 on arrival, high thrombotic burden, high burden lipid-rich plaque, high pressure balloon inflations, the use of debulking techniques and long lesions [9]. No reflow is generally associated with larger infarction size, decreased left ventricular ejection fraction and short/long-term mortality: An in-hospital and 6-month mortality increase of 6-fold and 10-fold, respectively, have been found in retrospective analysis [8].

Abrupt closure

Abrupt closure is defined as the cessation of coronary flow usually related to dissection or thrombosis. Vessel closures have

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become rare events in the modern practice with an incidence of 0.3% due to improved interventional techniques (mainly the use of modern drug eluting stents) and advanced pharmacology (e.g. glycoprotein IIb/IIIa inhibitors, effective dual antiplatelet therapy) [10]. Dissection and injury to the medial are the most common mechanisms of acute closure. Due to mechanical obstruction, thrombus formation and often vasoconstriction can occur. The commonly used classification of coronary dissection is presented in Table 3.

 Table 3: The National Heart, Lung and Blood Institute (NHLBI)

 classification of coronary dissections.

Type A	Minor radiolucent areas within the lumen with little or no		
	contrast persistence		
Type B	Double lumen separated by a radiolucent area, with little or		
	no contrast persistence		
Type C	Extra luminal cap with contrast persistence		
Type D	Spiral filling defect		
Type E	New persistent filling defect		
Type F	Any dissection with TIMI 0		

Patient-related predictors of abrupt closure include unstable angina, multivessel disease and female sex. Among angiographic features, proximal tortuosity, longer lesions and de novo stenosis, as well as degenerated vein grafts, high thrombus burden have been described as potential risk factors. Data regarding prognostic significance are mainly derived from the balloon angioplasty era: 6% of patients died, 36% suffered nonfatal myocardial infarction and 30% were transferred for emergent bypass surgery [10,11].

Myocardial infarction

Based on the reported data, procedural myocardial injury is very common, occurring in up to 47% of interventions [12]. It may result directly from periprocedural aspects, or result from device complications such as stent thrombosis or in-stent restenosis. Periprocedural myocardial injury is defined by the increase of cTn values (>99th percentile URL) in patients with normal baseline values (<99th percentile URL), or an increase of cTn values>20% of the baseline value when above the 99th percentile URL [12]. The impact of the injury on the prognosis is still a matter of debate.

The various definitions of peri-procedural myocardial infarctions are presented in Table 4. The incidence of type 4a myocardial infarction ranges from 4.3% to 7.1% [1].

 Table 4: Peri-procedural myocardial infarction-current definitions.

Definition	Biomarker criteria	Required evidence
SCAI	CK-MB >10 × ULN, or	None
	CK-MB >5 × ULN	New pathological Q-waves
UD: PCI	Troponin >5 × 99th percentile URL	ECG: New ischemic changes or new pathological Q-waves; or angiographic: flow-limiting complication (dissection, occlusion, etc); or imaging: Loss of viable myocardium in consistent pattern
UD: CABG	Troponin >10 × 99th percentile URL	ECG: New pathological Q-waves; or angiographic: New graft or native vessel occlusion; or imaging: Loss of viable myocardium or new wall motion abnormality

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ARC 2 (for	Troponin>35	ECG: New significant Q-waves or
PCI and	times URL	equivalent; or flow-limiting angiographic
CABG)		complication; or new substantial loss of
		myocardium on imaging

Common causes of myocardial infarction can be classified into three groups:

- Procedure-related such as side branch occlusion, dissection, distal embolization, or no-reflow
- Lesion-related such as large thrombus burden, high plaque volume and plaque instability
- Patient-related such as genetic predisposition, pro-inflammatory states, and aspirin/ADP receptor resistance.

Whether myonecrosis is associated with late adverse events and impacts the overall prognosis or is just a symptom of coronary disease has been controversly debated in the last years. In many multicentre studies and meta-analyses of multiple clinical trials, it has been established that troponin elevation is directly related to adverse events, included death and myocardial infarction [3]. Worse long-term outcomes have been observed in large-scale side-branch loss from subintimal dissection or stenting of the false lumen [13].

DISCUSSION

Coronary complications have been reported and described thoroughly over the last four decades. Some procedural issues such as coronary perforations with cardiac tamponade and obstructive shock may have prompt devastating impact on in-hospital mortality. Other complications such as abrupt close may result in non-fatal procedural infarction with the worsening of the left ventricular ejection fraction, development of heart failure and adverse longterm mortality [2].

Risk stratification in coronary interventions is a very important issue in both planning procedures and counselling patients. Despite the fact that some specific predictors for certain complications have been described, there are a few risk factors that have been repetitively identified in most complicative cases [14,15].

CONCLUSIONS

From the practical and methodical point of view, it seems reasonable to divide the predictors into following groups:

- Patient-related factors such as advanced age, female sex, multimorbidity;
- Procedure-related factors such as multivessel disease, proximal tortuosity, complex lesions, high pressure dilatation, the use of debulking devices;
- Operator-related such as overall interventional experience.

It should be noted that the individual aspect (i.e. single operator's performance) may play a crucial role in developing and managing complications, especially in treating patients at night and over the weekend. On the intuitive basis, an inexperienced fellow would probably encounter more complications than an experienced interventionalist. Still, no large randomized data exist. Last but not least, kidney function and reduced ejection fraction have often been identified to predict overall complications. In the recent cross-sectional study, the author was able to demonstrate the significance of reduced ejection fraction in predicting complications in urgent and emergent interventions. This highlights the necessity of pre-

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interventional echocardiography in both stable and unstable settings.

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DECLARATION

Nothing to declare.

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