Percutaneous coronary intervention in acute myocardial infarction: from procedural considerations to long term outcomes
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Part I General Introduction
GENERAL INTRODUCTION

Patients presenting with AMI are treated with primary percutaneous coronary intervention (PCI). Across the broad spectrum of patients with acute myocardial infarction (AMI), short-term (in-hospital or 30-day) mortality has decreased dramatically over the past 30 years, concomitantly with the increasing use of mechanical reperfusion by PCI and pharmacological therapies such as beta blockers, angiotensin-converting-enzyme (ACE)-inhibitors and statins. The current in-hospital mortality of ST-elevation myocardial infarction (STEMI) is below 5%. However, morbidity is still high due to peri-procedural events as well as events that occur during the natural course of infarct healing.

A devastating complication of PCI is ischaemic stroke which occurs in 0.07% to 1.3% of patients undergoing a PCI procedure. However, the frequency of silent cerebral infarction is much higher, ranging from 2% to 35%. Moreover, bleeding complications after PCI are associated with an increased risk of mortality and morbidity. Therefore, considerable effort has been made to develop novel treatment strategies directed at minimizing bleeding complications. One such strategy, performing PCI via the radial artery, has been shown in prospective, randomized trials to result in a reduction in bleeding complications arising at the arterial puncture site. Unfortunately, although access site bleeding represents a common source of bleeding in patients undergoing PCI, as many as 50% to 60% of major and minor bleeding complications are not related to the arterial access. Moreover, there have been concerns regarding the possible increase in radiation exposure for patients when the radial artery is used as an access site.

After the performance of primary PCI during the acute phase of myocardial infarction, an adequate healing response is pivotal for preserving left ventricular (LV) function and geometry. The initial post-MI phase includes fibrotic repair of the necrotic area with scar formation with subsequent elongation and thinning of the infarct zone. During this initial phase, myocyte necrosis, edema and inflammation are localized to the infarcted region. This complex and dynamic process of infarct healing is critically mediated by monocytes, that may lead after uncontrolled monocyte response to impaired post-AMI healing.

Adequate post-AMI healing is crucial for the prevention of adverse left ventricular remodeling. Adverse left ventricular remodeling refers to alterations in ventricular architecture involving both the infarcted and non-infarcted zones leading to progressive increase in systolic and diastolic left ventricular volumes. This increase of LV volumes can be considered adaptive as it is an attempt to augment stroke volume and to maintain cardiac output. However, in patients with progressive post-infarction dilation, the end-systolic volume index increases progressively and LV ejection fraction (LVEF) declines.
These changes are important predictors of mortality. Also, in these patients there is a risk of left ventricular thrombus formation and subsequent thromboembolic complications.

The magnitude of adverse remodelling and subsequent mortality is related to infarct size and the presence of microvascular injury. Cardiac magnetic resonance (CMR) is considered the non-invasive standard for these parameters as it provides a detailed evaluation of cardiac function and anatomy. Contrast-enhanced CMR with late gadolinium enhancement allows assessment of myocardial viability and transmural extent of viable myocardium providing the potential to identify those patients who are at highest risk for adverse remodeling.

Moreover, CMR enables the determination of the size of the infarct’s surrounding border zone, the so-called penumbra. This area consists of a heterogeneous mass of necrotic, ischemic and viable myocardium, as well as edema and fibroblasts which hypothetically provides a substrate for the development of ventricular arrhythmias.

Beyond the acute phase of myocardial infarction, CMR can monitor the process of long term left ventricular remodeling. There is accumulating evidence that left ventricular remodeling is a dynamic process occurring not only in the first weeks to months after myocardial infarction, but also in the long period thereafter. Pathological Q waves on the electrocardiogram are considered the classic ECG sign of necrosis, but these Q waves may partially or completely disappear during the evolution of myocardial infarction. CMR can be used to analyze whether such Q-wave regression is related to shrinkage in infarct size and/or improvement of LV function.

However, the ultimate goal is reversal or attenuation of adverse LV remodeling with therapeutic interventions in addition to standard pharmacotherapeutic regimens. Bone marrow cells have the capacity to proliferate, migrate, and also differentiate into various mature cell types. It therefore has the potential for tissue repair. Animal studies demonstrated a beneficial effect of local administration of progenitor cells. Several clinical trials rapidly followed to translate these exciting preclinical results with the aim to prevent deterioration of LV ventricular function and thereby reducing the large burden of heart failure.
REFERENCES


