

LVEDP as a Predictor for Success of Reperfusion in
Patients with Anterior STEMI Undergoing Primary PCI

Thesis

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القسطرة التداخلية الأولية على إعادة ارتواء عضلة القلب
فى مرضى الاحتشاء الحاد بالجدار الأمامى لعضلة القلب**

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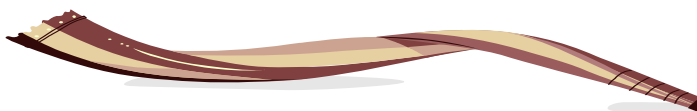
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INTRODUCTION

ST segment elevation myocardial infarction (STEMI) constitutes 40% of all acute myocardial infarctions (AMI), which continues to be a significant public health problem in both developed and developing countries (*Rogers WJ et al., 2000*), (*Hellermann JP et al, 2002*).

Primary percutaneous intervention (PCI) is now classified as class I indication in STEMI in the Guidelines of the European Society of Cardiology (ESC) (*Siler S et al., 2005*).

Reperfusion therapy is the cornerstone of the treatment of patients with acute ST elevation myocardial infarction (STEMI) (*Ryan TJ et al., 1999*). Many randomized clinical trials have shown that primary percutaneous coronary intervention (PCI) is superior to thrombolytic therapy in the treatment of patients with STEMI (*Grines CL et al., 1993*), (*Zijlstra F et al., 1993*), (*Gibbons RJ et al., 1993*), (*The GUSTO-IIb Angioplasty Substudy Investigators, 1997*).

Nevertheless, the occurrence of Thrombolysis in Myocardial Infarction (TIMI) ≤ 2 flow remains the "Achilles heel" of primary PCI occurring in 2% to 37% of patients (*Grines CL et al., 1999*), (*Antoniucci D et al., 1998*), (*STENTIM-2 Investigators Maillard L et al., 2000*), (*Suryapranata H et al., 1998*), (*Kenner MD et al., 1995*), (*Ito H et al., 1996*), (*Giri S et al., 2006*), (*Maximal Individual Therapy in Acute Myocardial Infarction (MITRA) and Myocardial Infarction Registry (MIR) Study Groups, 2001*), even in the era of the routine use of stents and newer antithrombotic and antiplatelet agents, strategies shown to improve the outcomes of patients undergoing primary PCI (*Mehta RH, 1999*), (*Brener SJ et al., 1998*), (*ADMIRAL Investigators, 2001*).

The aim of reperfusion therapy for many years has focused on achieving epicardial artery patency at the site of the occlusive thrombus. It is now possible, through advances in interventional techniques and adjunctive pharmacological treatment, to achieve TIMI (Thrombolysis In Myocardial Infarction) grade 3 epicardial flow (normal) in 95% of patients.

The myocardial blush grade (MBG) has been devised for the visual assessment of myocardial reperfusion after primary

percutaneous coronary intervention (PCI), and this score is an independent predictor for adverse outcome (*van't Hof AW, 1998*), (*Henriques JP et al., 2003*).

Despite this achievement, mortality, although declining, still remains high (*Goldberg RJ et al., 2004*). This is possibly because despite restoration of TIMI grade 3 flow, 40% of patients do not achieve microvascular flow, which should be the goal of reperfusion therapy (*Iwakura K et al., 2001*).

The no-reflow phenomenon (absence or reduced microvascular flow despite restoration of epicardial coronary artery patency) was first described by Kloner *et al* (**Kloner RA et al., 1980**). Electron microscopy showed microvascular obstruction due to endothelial blebbing, white cell infiltration, red cell stagnation, and extracellular oedema (*Kloner RA et al., 1980*).

This process may be accelerated after reperfusion as a result of liberation of oxygen free radicals. The phenomenon of no-reflow in the clinical setting is also caused by microembolism from the occlusive thrombus and downstream plaque plugging following PCI. Microvascular spasm as a result

of liberation of vaso-active amines from activated platelets is also implicated (*Kloner RA et al., 1980*).

The consequence of no-reflow phenomenon is profound in that it results in left ventricular (LV) remodeling, LV dysfunction, heart failure, and increased mortality (*Ito H et al., 1996*). Hence, the success of reperfusion therapy should be gauged not only by the rapid restoration of epicardial coronary flow but also by rapid achievement of myocardial perfusion (*Ito H et al., 1996*).

How can one rapidly assess myocardial perfusion? Clinical (resolution of chest pain and ST segment elevation) and coronary angiographic markers (TIMI flow, TIMI frame count, and myocardial blush grade) have been widely used. They are all indirect markers of myocardial perfusion (*Ito H et al., 1996*).

However, myocardial contrast echocardiography (MCE), a technique that utilizes microbubbles, which remains intravascular and accurately denotes the status of microvascular perfusion within that region. The clinical significance of no-reflow using MCE has been demonstrated previously (*Ito H et al., 1996*). Recently, it has been shown that MCE is the best

technique to assess no-reflow compared with clinical and angiographic markers (*Greaves K et al., 2003*), (*Galiuto L et al., 2008*).

Successful rescue PCI within 3–24 hours of the onset of chest pain has been associated with improved LV systolic function at a mean follow-up period of 22 months (*Fath-Ordoubadi F, 1999*). In this study, baseline and repeated ventriculograms were used to assess LV systolic function. Other studies of primary PCI have also reported improved LV systolic function compared to thrombolysis (*Hochman JS et al., 2006*).

One of the causes of diastolic dysfunction is coronary artery disease (*Parodi G et al., 2006*). Acute coronary syndromes (ACS) may present with acute heart failure purely as a result of exacerbation of diastolic dysfunction (in the absence of any systolic dysfunction) (*Henein MY et al., 1993*).

PCI does improve diastolic function in such patients but patients with hypertension (the most common subset of patients with diastolic dysfunction) and AMI have a higher propensity to develop heart failure postprimary PCI than those without hypertension (*Banerjee P, 2003*).