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 counties (1).

Primary percutaneous intervention (PCI) is now classified as class I indication in STEMI in the Guidelines of the European Society of Cardiology (ESC) (2).

Reperfusion therapy is the cornerstone of the treatment of patients with acute ST elevation myocardial infarction (STEMI) (3). Many randomized clinical trials have shown that primary percutaneous coronary intervention (PCI) is superior to thrombolytic therapy in the treatment of patients with STEMI (4).

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 (Thrombosis In Myocardial

Infarction) grade 3 epicardial flow (normal) in 95% of patients(5,6).

Despite this achievement, mortality, although declining, still remains high . 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 (7).

Successful primary 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 (8).. Other studies of primary PCI have also reported improved LV systolic function compared to thrombolysis (9).

In acute myocardial infarction (MI), decreasing compliance of the left ventricle is directly associated with poor prognosis(10). In patients with ST segment elevation MI (STEMI), left ventricular filling pressure increases(11,12).

Early improvement of perfusion after MI will improve left ventricle function and decrease the infarction area, thus decreasing mortality(13,14). The efficacy of reperfusion treatment may be shown indirectly with electrocardiography (ECG), by regression of ST elevation, but there is a need for methods to

demonstrate left ventricle and microvascular function improvement[15] .

Primary percutaneous coronary intervention (PCI) is regarded as the best reperfusion model in STEMI. PCI may be used to show hemodynamic changes in the left ventricle or to measure left ventricle end-diastolic pressure (LVEDP) for evaluation of reperfusion efficacy and success.

studies have now demonstrated a robust association between BNP or NT-proBNP and the short- and long-term risk of death across the spectrum of non-ST-elevation ACS,[16-18]. including patients without myocardial necrosis or clinical evidence of heart failure. In some patients with ACS, elevated levels of BNP directly reflect the degree of left ventricular dysfunction resulting from acute myocardial infarction. However, the strong association between levels of BNP/NT-proBNP and mortality among patients without measurable myocyte necrosis (i.e., release of cardiac troponin) indicate that the level of BNP may reflect the extent or severity of the ischemic insult, even when irreversible injury has not occurred.[18]

These findings suggest that transient ischemia may induce BNP synthesis and release in proportion to the severity of myocardial ischemia. As such, BNP adds a new dimension to our ability to quantify the consequences of acute myocardial ischemia.