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شبكة المعلومـــــات الجامعية

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جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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INFARCT-RELATED CORONARY ARTERY ANGIOPLASTY:

Relation between residual stenosis, contractile reserve, and functional recovery

THESIS
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ABBREVIATIONS

CAD : Coronary artery disease.

CABG : Coronary artery bypass graft.

DSE : Dobutamine stress echocardiography.

IRA : infarct-related artery.

MI : Myocardial infarction.

PTCA : Percutaneous Transluminal Coronary Angioplasty.

LV : Left Ventricle

LVEF : left Ventricular ejection fraction.

LDD : low-dose dobutamine infusion.

Chapter I

INTRODUCTION

MYOCARDIAL VIABILITY:

The definition of myocardial viability has been variable in published reports. According to Oxford English Dictionary, viable means "capable of living". Incorrectly, the term viable and capable of contracting in the presence of adequate blood flow have been used interchangeably for the myocardium. Viable myocardium may represent normal, non-ischemic myocardium or ischemic but reversibly dysfunctional myocardium. One definition of viable myocardium has been the temporal improvement in contractile function after specific therapeutic interventions, most commonly restoration of coronary artery blood flow. However, restoration of blood flow does not necessarily translate to improvement in contractile function in all regions (1). This definition also presupposes that revascularization successfully restores resting nutrient blood flow to normal levels. It ignores the occurrence of inadequate revascularization for technical reasons, poor distal runoff, or the presence of abnormal microvasculature within the revascularized bed (2). However, a reduced coronary flow at rest is not a necessary requirement for definition of viable myocardium, and that recovery of function can also be seen in segments with a normal flow at rest (3).

In the recent years, diagnostic testing to evaluate the presence and extent of viable but dysfunctional myocardium has become an important component of the clinical assessment of patients with chronic coronary artery disease and left ventricular (LV) dysfunction. It is well established that impaired LV function in such patients is not always an irreversible process related to previous myocardial

infarction, because LV function may improve considerably after myocardial revascularization procedure (4).

The mechanism for this improvement in systolic function remains a matter of uncertainty and debate because the underlying processes responsible for reversible contractile dysfunction are often difficult to ascertain in patients. Restoration of blood flow to chronically underperfused myocardium may lead to functional recovery of hibernating myocardium (5), whereas revascularization of myocardium with adequate perfusion at rest but with recurrent ischemic episodes during stress may successfully reverse persistent contractile dysfunction caused by repetitive stunning (6). It is likely that both hibernation and repetitive stunning do occur clinically and contribute to ischemic LV dysfunction. Moreover, both processes may occur in the same patient and even in the same myocardial region, some myocardial regions that are hibernating at rest may develop ischemia during exercise with a subsequent process of post ischemic stunning superimposed on the baseline hibernating state (7).

MYOCARDIAL STUNNING:

It has been over 20 years since post-ischemic myocardial dysfunction was first described by Heyndrickx et al in 1975 in conscious dogs undergoing brief coronary occlusion (8). Beginning in the 1980s and continuing to an even greater extent in the 1990s, however, post-ischemic dysfunction has become the focus of increasing interest both among experimentalists (9) and clinicians (10) because of

two major reasons. First, coronary reperfusion by means of thrombolytic therapy, percutaneous transluminal coronary angioplasty (PTCA), or coronary artery bypass graft (CABG) surgery has become a standard approach for the management of acute ischemic syndromes in patients with coronary artery disease. Second, several studies have demonstrated that many patients experience spontaneous reperfusion as a result of lysis of coronary thrombi or release of coronary spasm. Accordingly, post-ischemic myocardial stunning is a part of the natural history of coronary artery disease and contributes to the morbidity associated with this disorder.

Definition of myocardial stunning:

Myocardial stunning is a general term that describes the mechanical dysfunction that persists after reperfusion despite the absence of irreversible damage and despite restoration of normal or near normal coronary flow (9, 11).

Experimental settings of myocardial stunning:

One of the major problems in formulating a unifying pathophysiological and a pathogenic paradigm for myocardial stunning is that this phenomenon occurs in a wide variety of settings that differ from one another in several major respects (12). Indeed, myocardial stunning might be regarded not as a single entity but instead as a phenomenon.

At the experimental level, the available observations can be grouped into 6 categories (13):

1-stunning after a single, completely reversible episode of regional ischemia in vivo (e.g., coronary occlusion <20 minutes in dogs) as originally described by Heyndrickx et al (8). This does not result in any myocardial necrosis, but on reperfusion, the recovery of contractile performance in the previously ischemic myocardium is delayed for several hours. This is the "classic" model of myocardial stunning (14,15).

2-Myocardial stunning after multiple, completely reversible ischemic episodes:

Repeated brief (2 to 10 minutes) coronary occlusions depress systolic function and result in prolonged contractile impairment despite absence of irreversible damage (16), its severity is not related to collateral perfusion during ischemia, and a preconditioning effect develops during the first three ischemic episodes (17). The first 5-minute occlusion preconditions the myocardium against the next occlusions, so that the overall severity of stunning is the same after one or three occlusions; however, after the third occlusion this preconditioning effect is lost, and additional occlusions cause a cumulative depression of contractility.

3-Myocardial stunning after a single, partly irreversible ischemic episode (subendocardial infarction): When reperfusion is instituted after a period of coronary occlusion greater than 20 minutes but less than 3 hours, the subendocardial portion of the region at risk is generally found to be infarcted; resulting in an admixture of infracted subendocardium and stunned subepicardium.