INTRODUCTION

MYOCARDIAL INFARCTION, A PARADYGM OF SUCCESS IN MODERN MEDICINE

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Although myocardial infarction still represents the major cause of mortality in Western countries, there has been a sharp decrease of its occurrence and fatal complications (1). Since the ‘60s, when cardiovascular diseases overcame the threshold of 50% as cause of death and myocardial infarction was truly a nightmare, cardiovascular mortality declined and life-expectancy increased by 6 years, mostly thanks to prevention, early diagnosis and treatment of cardiovascular disease. These excellent achievements were the result of understanding and care of myocardial infarction, which can be summarized as follows:

a) Myocardial infarction is precipitated by acute coronary occlusive thrombosis in nearly 90% of cases, in the setting of coronary atherosclerosis (2). Prompt recanalization by thrombolysis and primary angioplasty may restore myocardial perfusion.

b) Ventricular fibrillation with cardiac arrest may occur in the early time course of coronary occlusion, however it may be rescued by defibrillation. Prompt intervention and early recovery in Coronary Care Units (CCU) with ECG monitoring and alarm system in case of life-threatening arrhythmias may prevent fatal outcome.

c) Ischemic myocyte necrosis develops as a wave front phenomenon, from endocardium to epicardium, with a time course according to which 40% of myocardium may be saved if reperfusion is accomplished within 3 hours (3).

d) Early reopening of infarcted related artery may reduce infarct size limiting life-threatening complications like transmural extension, aneurysm, cardiogenic shock, cardiac rupture and pericarditis.

e) Long term complications of myocardial infarction, like sudden arrhythmic death, may be prevented with pharmacologic and non-pharmacologic therapies, the latter including pace-maker and implantable cardioverter defibrillator (ICD) for life-threatening brady- or tachyarrhythmias respectively. Control of serum cholesterol by statins play a major role in secondary prevention by decreasing propensity to atherosclerosis and recurrence of myocardial infarction.
f) Cardiogenic shock should be no longer considered with a fatalism, thanks to ventricular assistance devices and mechanical hearts, supporting circulation as a bridge to cardiac transplantation. Repair of infarcted heart has been demonstrated to be possible by engrafting the myocardium with embryonic human stem cells (4).

g) Overall mortality by acute myocardial infarction declined by 30 to 10% in the last forty years, mostly by the accomplishments of clinical cardiology in monitoring and treating arrhythmias in CCU and limiting infarct extension by early coronary recanalization. Long term survival was also greatly improved, with mortality declining from 10% to 2%/year in the early 5 years, using ICD and putting cholesterol under control.

Pathologists played a not so minor role in these achievements, with the discovery of vulnerable plaque and plaque rupture precipitating coronary thrombosis, of time course of myocardial infarction, as well as of arrhythmic sudden death in the long term related to infarct scarring.

However, there are a series of still pending questions:

1) Identification of patients bearing vulnerable plaques at risk of rupture. Coronary thrombosis usually occurs as a “bolt from the blow” in asymptomatic patients. Developments of in vivo imaging to detect coronary plaques with impending rupture are in progress.

2) Instantaneous death by ventricular fibrillation or AV block may complicate coronary thrombosis, well before hospitalization. This represents 50% of mortality by myocardial infarction and it is out of the control, because early intervention for resuscitation within 5-7 minutes before irreversible cerebral damage is not feasible. External defibrillators should be implemented.

3) 30% of coronary thrombosis occurs through the “plaque erosion” mechanism, often in the setting of plaque inflammation. However, serum markers for a focal inflammation are questionable.

4) Although accomplishing early reperfusion and keeping open the infarcted related coronary artery are beneficial, there are some unresolved issues whether reperfusion may aggravate myocardial injury and trigger arrhythmias as well as favour cardiac rupture.

5) Heart failure is a frequent sequela and repair by stem cells has to be proven in terms of mechanical efficacy and electrical stability.

6) Coronary recanalization is carried out with stent implantation, with the inherent risk of early restenosis by intimal proliferation. The introduction of drug-eluting (in place of metal bare) stents, certainly prevent intimal cell hyperplasia, but concern was raised on the potential of late thrombosis by endothelial damage.

With these limitations and challenges in mind, certainly myocardial infarction does not represent anymore a nightmare as in the past, and the
achievements in its prevention, diagnosis and treatment should be considered as a pride of cardiovascular medicine.

BULLET POINTS:

• Hospital mortality for acute myocardial infarction (AMI) declined from 30% to 10% in the last 30 years, thanks to Coronary Care Units and early revascularization with thrombolysis, angioplasty and stent implantation.

• The pathologists played a major role by establishing plaque rupture and coronary thrombosis as a cause of AMI and by discovering that ischemic myocardium necrosis progresses from endo- to epicardium as a wave front phenomenon, with reversible injury if reperfusion is achieved within 3 hours, thus saving 40% of the myocardium at risk.

• Long term mortality following myocardial infarction is mostly due to sudden electric death, which may be prevented by pharmacologic (antiarrhythmic drugs) and non pharmacologic (ICD, pace-maker) therapy. Ventricular assistance device may support the left ventricle as a bridge to transplantation. Mortality at distance declined from 5% to 2%/year.

• Despite these indisputable achievements, there are still pending questions: in vivo identification of vulnerable plaque, mechanisms of thrombosis by plaque erosion, prompt treatment of instantaneous cardiac arrest by external defibrillation following coronary thrombosis, adverse effect of myocardial reperfusion, fate of bare and drug-eluting coronary stents.

REFERENCES:


