Acute Intervention in Cardiogenic Shock Due to MI
Impact of IABP

Thesis Submitted by

Amr Mohamed Esmat, MSc.

In Partial Fulfillment of
MD in Critical Care Medicine

Supervisors

Prof. Dr. Sherif Mokhtar, MD
Professor of Cardiology
Professor of Critical Care Medicine
Critical Care Medicine Department
Cairo University

Dr. Mohamed Ashraf, MD
Asst. Prof. of Critical Care Medicine
Critical Care Medicine Department
Cairo University

Dr. Tarek El Gohary
Asst. Prof. of Critical Care Medicine
Critical Care Department
Cairo University

Dr. Hossam El Sherif
Lecturer of Critical Care Medicine
Critical Care Department
Cairo University

Cairo University

2008

ABSTRACT

Cardiogenic shock (C. shock) following acute myocardial infarction (MI) still carries a high mortality rate despite advances in pharmacotherapy including thrombolytic reperfusion, the prompt availability of diagnostic angiography and therapeutic interventions i.e. (primary PCI) with or without the support of circulatory augmentation using intra aortic balloon counter pulsation (IABC). The clinical benefits of the latter procedure are related to the augmentation of the coronary diastolic perfusion and the unloading effect of the pre-systolic sink. The purpose of the present study was to assess in an objective way the positive effects (if any) of the IABC on real cardiac haemodynamics as measured by the tissue Doppler technique in cardiogenic shock. Twenty patients with C. shock complicating acute MI not more than 48 hrs after MI constituted the material of the present study (12 males and 8 females, mean age 55.95±13.12 yrs). Pts were divided on alternate bases into two groups, with and without IABC. Following clinical and laboratory evaluation, all pts were started on vasoactive and/or inotropic therapy and rolled into the cath laboratory for diagnostic coronary angiography. Primary PCI without circulatory augmentation was performed in 10 pts and followed by IABC in 10 pts. Tissue Doppler imaging (TDI) and M-mode to measure cardiac dimensions and function, were performed before and within one hour following PCI. A non-significant difference was observed in almost all echo parameters assessed between the two groups of patients on admission. The diastolic blood pressure (BP) of group I pts showed significant improvement from admission readings. Systolic BP of group II pts showed correspondingly higher readings in follow up. Serum lactate decreased significantly with IABC, whereas for group II a non-significant difference was observed between admission and follow up readings. A significant improvement was observed between the admission and follow up readings in group I pts in terms of LVEDd, LVESd, IVSTd, %FS, and %EF. Higher % ↑ in Sm and Em and higher % ↓ in Am was observed in group I in comparison to group II. There was no difference in survival between pts treated with IABC and those who were not. Fifty *per cent* of pts from each group died by the end of the study. Logistic regression analysis has reported the following as final predictors of unfavourable outcome with specificity and sensitivity of 100%: follow up systolic BP ≤ 80, CK ≤1700 and pH ≤ 7.18. In conclusion, despite the obvious immediate haemodynamic benefits of IABC, the short term outcome might not be correspondingly significantly better.

Key word: Cardiogenic shock, myocardial infarction (MI), intra aortic balloon counter pulsation (IABC)

Contents

Acknowledgement

For **ALLAH** the merciful, the compassionate, I kneel to express my gratitude for all the countless gifts I have been offered, including those who gave their hands to enable me to fulfill this work.

No words are sufficient to express my deep appreciation and profound gratitude to ***Prof. Dr. Sherif Mokhtar***, Professor of Critical Care Medicine, for offering all his students an inspirational role model, for showing us the excitement and joy of critical care medicine, for his dedication to education and for his magnificent assistance and sincere supervision throughout the work of this thesis. I am truly indebted to him with all what I learned and still learning in critical care medicine.

I would like to send my deepest gratitude to ***Prof. Dr. Allia Abd el- Fattah,*** Professor of Critical Care Medicine and Cheif of Critical Care Medicine Department for her abundant encouragement, continuous support and endless giving.

My true appreciation is due to ***Dr. Mohamed Ashraf,*** Assistant Professor of Critical Care Medicine for his meticulous supervision, for his kind guidance, valuable instructions and generous help

I am deeply thankful to ***Dr. Tarek el-Gohary,*** Assistant Professor of Critical Care Medicine for his great help, outstanding support and active participation.

Special thanks are due to ***Dr. Hossam el-Sherif,*** Lecturer of Critical Care Medicine for his sincere efforts, fruitful encouragement and contributions in performing the echocardiographic part of this thesis.

I wish to thank all the cath lab team, for their great efforts and important contributions.

I am very thankful to ***Dr. Lamiaa Hamed,*** Medical informatics specialist, Critical Care Medicine Department, for her efforts in performing the statistical work of this thesis.

I wish to thank deeply Mrs. ***Manal Youssef***, Critical Care Medicine Department for her patience in performing the computer work of this thesis.

Finally I am so thankful and honored to belong to the critical care medicine department, the land of imagination, innovation and fruitful research.

List of Abbreviation

2-D: two dimensional

3-D: Three dimensional

AMI: Acute myocardial infarction

AR: Pulmonary venous peak arterial contraction reversed velocity

AV: Atrioventricular

BP: Blood pressure

C. Shock: Cardiogenic shock

CABG: Coronary artery bypass graft

CAD: Coronary artery disease

CK: Creatine kinase

CVP: Central venous pressure

DMI: Doppler myocardial imaging

DT: Early left ventricular filling deceleration time

ECG: elctrocardiography

EF: Ejection fraction

GUSTO trial: Group italiano per Lo studio della streptochinasi nell infarcto Miocardico trial.

GUSTO: Global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries.

HA: Hospital admission

HCM: Hypertrophic cardiomyopathy

HR: Heart rate

IABC: Intra-aortic balloon counterpulsation

ICU: Intensive care unit

IHD: Ischaemic heart disease

IRA: Infarct related artery

K+: Potassium

LAD: Left anterior descending artery

LBBB: Left bundle branch shock

LDH: Lactic dehydrogenase

LV: Left ventricular

LVADs: Left ventricular assist devices

MR: mitral regurgitation

Na+: Sodium

PAMI trial: Primary angioplasty in myocardial infarction trial

PC: Prothrombin concentration

PCI: Percutaenous coronary intervention

Pt: Patient

PTCA: Percutaneous trnasluminal coronary angioplasty

PV: pulmonary venous

RV: Right ventricular

S2: Tissue velocity wave in the ejection period at the level of the mtiral annulus

SGOT: Serum glutamic oxaloacetic transaminase

SGPT: Serum glutamic pyruvic transaminase

SHOCK trial: Should we emergently revascularize occluded coronaries for shock trial

SMASH trial: Swiss multicenter evaluation of early angioplasty for SHOCK trial.

SR: Strain rate

SRI: Strain rate imaging

TACTICS trial: Thrombolysis and counterpulsation to improve cardiogenic shock Survival (TACTICS) trial

TD: Time delay

TDE: Tissue Doppler echocardiography

TDI: Tissue Doppler imaging

TIMI: Thrombolysis in myocardial infarction

Vp: Color M-mode flow propagation velocity

VSR: ventricular septal rupture

List of Tables

List of Figures

Introduction

Cardiogenic shock (C.shock) as a complication of acute myocardial infarction (AMI) continues to be an unfortunately too very serious problem that carries a high mortality rate [1]. The syndrome of C.shock has been defined as the inability of the heart-as a result of impairment of its pumping function-to deliver sufficient blood flow to the tissues to meet resting metabolic demands [2]. Thus, the purest clinical definition of C.shock includes poor cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. The diagnosis is indicated by the combination of low systolic blood pressure (BP <90 mmHg or a value 30 mmHg below basal levels for at least 30 minutes), an elevated arteriovenous oxygen difference (>5.5 ml per deciliter) , and a depressed cardiac index (<2.2 liters per minute per square meter of body-surface area) in the presence of an elevated pulmonary capillary wedge pressure (>15mm Hg) [2].

Most patients are initially evaluated at the bedside, where a reasonably accurate clinical diagnosis of C. shock may be made according to the following criteria: hypotension as defined above; evidence of poor tissue perfusion, including oliguria, cyanosis, cool extremities, or altered mentation; and persistence of shock after the correction of non-myocardial factors.

The progressive deterioration that occurs in the absence of intervention in cases of C.shock can be seen as a vicious circle [3]. Initial compensatory mechanisms includes activation of the sympathetic nervous

system, effects on renal and neurohormonal regulation, and local vasoregulation, leading to an increase in heart rate, arterial and venous vasoconstriction, an increase in myocardial contractility, and shifting of fluid into the vascular compartment. Eventually, decreased perfusion pressure, especially in the presence of the multivessel obstructive coronary disease, leads to further depression of myocardial contractility, and the compensatory mechanisms are overwhelmed by the progressive deterioration of cardiac function [(2)].

As compared with patients who have AMI without C. shock, patients who have shock are older, more frequently have anterior MI , more often have had a previous infarction, and more commonly have a history of angina or congestive heart failure [(4,5)]. Several studies have found a higher prevalence of diabetes among patients with C. shock, found a greater prevalence of occlusion of the left anterior descending artey, multivessel coronary artery disease, and persistent occlusion of the infarct-related artery among patients with C.shock [(2)].

Resussitative and supportive efforts should be initiated immediately, at the time as the diagnostic evaluation, followed by thrombolytic therapy, intra-aortic balloon pumping and revascularization.

Critical elements include adequate oxygenation and ventilation, correction of electrolyte and acid-base abnormalities, relief of pain, and restoration of sinus rhythm. In patients with inadequate tissue perfusion and adequate intravascular volume, infusion of inotropic or vasopressor drugs should be begun immediately. Vasodilators can be beneficial for