New Lines in Management of Cardiogenic Shock due to Myocardial Infarction

An Essay

Submitted for partial fulfillment of M.sc degree

In Critical Care Medicine

Presented By

Sayed Abd Elsalam Sayed

M.B.B.Ch

Supervised By

Professor Doctor/ Mostafa Kamel Reyad

Professor of Anesthesia and Critical Care Faculty of Medicine, Ain Shams University

Doctor/ Yasser Ahmed Abd Elrahman

Lecturer of Anesthesia and Critical Care Faculty of Medicine, Ain Shams University

Doctor/ Rafik Emad Latif

Lecturer of Critical Care Medicine

Faculty of Medicine, Ain Shams University

2014

بسم الله الرحمن الرحيم

وقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ ورَسُولُهُ والْمُؤْمِنُونَ

صدق الله العظيم

سورة التوبة آية (105)

First, thanks are all due to Allah for Blessing this work until it has reached its end, as a part of his generous help throughout our life.

My profound thanks and deep appreciation to Prof. Dr. Mostafa Kamel Reyad Professor of Anaesthesia, Intensive Care and Pain Management, Faculty of Medicine, Ain Shams University for his great support and advice, his valuable remarks that gave me the confidence and encouragement to fulfill this work.

I would like also to express my deep gratitude to Dr. Yasser Ahmed Abd Elrahman, Lecturer of Anaesthesia, Intensive Care and Pain Management, Faculty of Medicine, Ain Shams University for her generous help, guidance and patience through all stages of this work.

I wish also to thank Dr. Rafik Emad Latif, Lecturer of Anaesthesia, Intensive Care and Pain Management, Faculty of Medicine, Ain Shams University for for his great help and support in co-supervising the work.

I am extremely sincere to my family who stood beside me throughout this work giving me their support.

Words fail to express my love, respect and appreciation to my wife for her unlimited help and support.

Lastly, all the love to my dear daughter for being patient, understanding and cheerful throughout this work.

Sayed Abd Elsalam Sayed

List of Abbreviations

ACEI = angiotensin- converting enzyme inhibitors.

ACS = Acute coronary syndrome.

ACT = Activated clotting time.

AMI = acute myocardial infarction.

AMP = Adenosine Monophosphate.

BiPAP = bilevel positive airway pressure.

BNP = Brain natriuretic peptide.

C.shock = Cardiogenic shock.

CABG = Coronary artery bypass graft.

CHF = chronic heart failure.

CI = cardiac index.

CO = cardiac output

COMT = catechol-O-methyl transferase.

CPAP = Continuous positive airway pressure

CUPID = Calcium Up-Regulation by Percutaneous Administration of Gene Therapy in Cardiac Disease trial

CVP = center venous pressure

DA = Dopamine

ECMO = Extracorporeal membrane oxygenation

EPI = Epinephrine

ESC = European Society of Cardiology

HCM = hypertrophic cardiomyopathy

List of Abbreviations

HIT = Heparin-induced thrombocytopenia

HR = heart rate

HTN = Hypertension

I:E = inspiratory to expiratory ratio

IABP = Intra aortic Balloon Pump Counter pulsation

IL-6 = interleukin-6

ISO = Isoprelanine.

IVC = inferior vena cava

JRE = juvenile rheumatoid arthritis

LDH = lactate dehydrogenase

L-NAME = N-Nitro L-arginine methylester

LV = left ventricle

LVOTO = left ventricular outflow tract obstruction

MAO = Monoamine oxidase.

MI = myocardial infarcation

MIL = Milrinone

MR = mitral regurgitation

NE = Nor-epinephrine

NO = nitric oxide

PCI = percutaneous coronary intervention

PCWP = Pulmonary capillary wedge pressure

PDE = phosphodiesterase

PEEP = Positive end-expiratory pressure

PDE = phosphodiesterase

List of Abbreviations

PVADs = Percutaneous Ventricular Assist Devices

RCP = Reitan Catheter Pump

RV = right ventricle

RVEDP = Right ventricular end-diastolic pressure

SBP = Systolic blood pressure

SERCA 2A activators = Sarcoplasmic reticulum calcium ATPase isoform-2

SLE = systemic lupus erythematosus

STEMIs = ST-segment elevation myocardial infarctions

SV = stroke volume

SVI = stroke volume index

SVR = systemic vascular resistance

SVT = supraventricular tachycardia

SWI = stroke work index

TACTICS =Treat angina with Aggrastat and determine Cost of Therapy

TnC = Treponin .

TNF = tumor necrosis factor

VA = venoarterial

VASO = Vasopressin

VV = venovenous

List of Contents

List of figures

List of figures

List of figures

List of tables

Introduction

Cardiogenic shock is a serious complication of acute myocardial infarction and occurs as a consequence of acute left ventricular failure and subsequent inappropriate tissue perfusion. Incidence of this problem has been reported to be as high as 10% in late 1990s.It has been steadily declining to be currently less than 6% since the implementation of early revascularization therapy as a class I indication in current guidelines. Despite recent advances in the treatment of cardiogenic shock, mortality is still as high as 50 % approximately ***(Jeger et al., 2012).***

Early revascularization should be strongly considered for patients with acute myocardial infarction complicated by cardiogenic shock (CS). Emergency revascularization did not significantly reduce overall mortality at 30 days. However, after six months there was a significant survival benefit ***(Hochman et al., 1999).***

Mechanical support; Intra-aortic balloon pumping (IABP) is a mature technology used in CS. By diastolic inflation and rapid systolic deflation in the aorta, it improves peak diastolic pressure and lowers the end-systolic pressure translating into an afterload reduction, improved coronary perfusion, and reduction in myocardial oxygen consumption ***(Thieie et al., 2010).***

Because of limited hemodynamic benefits inherent in IABP therapy, new technologies have been developed as left ventricular assist devices (LVAD).Which have focused on improved hemodynamic support of the failing ventricle to bridge patients to recovery ***(Seyfarth et al., 2008).***

Unfortunately vasopressors and current inotropic drugs like cardiac glycosides, β-adrenoceptor agonists, phosphodiesterase (PDE) inhibitors have consistently failed to show beneficial effects beyond short-term hemodynamic improvement in patients with cardiogenic shock ***(Hasenfuss et al., 2011).***

To address these limitations, new agents targeting novel mechanisms are being developed, namely intravenous

levosimendan, which is a new calcium sensitizer and K-ATP channel opener. It is able to achieve profound increase of cardiac index and cardiac work index in combination with reduced systemic and pulmonary resistance reduction compared to conventional therapy ***(Buerkem et al., 2010).***

Istaroxime has been developed as a non-glycoside inhibitor of the sodium-potassium-ATPase with additional stimulatory effects on the sarcoplasmic reticulum calcium pump (SERCA) and has shown lusitropic and inotropic properties in experimental and early clinical studies ***(Buerkem et al., 2010).***

Cardiac myosin activators directly activating the acto-myosin cross-bridges, are most appealing with improved cardiac performance in both animal and early clinical studies.

Gene therapy approaches have been successfully employed to increase myocardial SERCA2a.

Nitroxyl donors have been developed and have shown evidence of positive lusitropic and inotropic, as well as potent vasodilator effects in early studies.

Ryanodine receptor stabilizers reduce pathological leak of calcium from the sarcoplasmic reticulum with initial promising pre-clinical results ***(Buerkem et al., 2010).***

Finally, ***metabolic energy modulation*** may represent a promising means to improve contractile performance of the heart. There is an urgent clinical need for agents that improve cardiac performance with a favourable safety profile. These current novel approaches to improve cardiac function provide the hope that such agents may soon be available ***(Hasenfuss et al., 2011).***

Aim of Work

Aim of this study is to highlight the role of left ventricular assisted devices and new inotropes in management of cardiogenic shock due to myocardial infarction.