

Ain Shams University
Faculty of Medicine
Department of Anesthesia
And intensive care

ACUTE HEART FAILURE IN ICU

Essay

Submitted for partial fulfillment of master degree of critical care medicine

Presented By

Alaa Abd EL-ALeem Kenawy

M.B.B.Ch

Faculty of Medicine - Menoufia University

Under Supervision of

Prof. Dr. Samia Ibrahim Sharaf

Professor of Anesthesia & intensive care Faculty of Medicine, Ain Shams University

Dr. Sanaa Farag Mahmoud

Lecturer of Anesthesia & intensive care Faculty of Medicine, Ain Shams University

Dr. Hend Youssef Mohammed Ali

Lecturer of Anesthesia & intensive care Faculty of Medicine, Ain Shams University

Ain Shamas University Faculty of Medicine 2013



فشل عضلة القلب الحاد داخل العنابة المركزة

مقدمة رسالة

توطئة للحصول على درجة الماجستير في العناية المركزة

مقدمه من

الطبيب / علاء عبد العليم عبد العزيز قناوي بكالوريوس الطب والجراحة - جامعة المنوفية

تحت إشراف

الأستاذ الدكتورة / ساميه إبراهيم شرف

كلية الطب – جامعة عين شمس

الدكتورة / سناء فرج محمود

مدرس التخدير والعناية المركزة

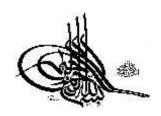
كلية الطب – جامعة عين شمس

الدكتورة / هند يوسف محمد على

مدرس التخدير والعناية المركزة

كلية الطب - جامعة عين شمس

كلية الطب - جامعة عين شمس



قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

صدق الله العظيم

سورة البقرة - آية ٣٢

CONTENTS

Contents

		Page
1	Introduction and Aim of the work	1
2	Pathophysiology of acute heart failure in ICU	4
3	Risk factors & causes	22
4	Investigations and Diagnostic Modalities	35
5	Management of acute heart failure in ICU	64
6	summary	106
7	References	109
	Arabic Summary	

LIST OF FIGURES & TABLES

LIST OF FIGURES

	Page	
Pathogenesis of HF		
Compensated and decompensated HF		
Activation of the sympathetic nervous system	12	
Activation of the renin-angiotensin system	14	
The pattern of cardiac and cellular remodeling	18	
Ventricular Remodeling: Gross and Microscopic Architecture	19	
Effect of changes in LV shape on LV wall stress	20	
Echocardiographic images from a patient with cardiac		
amyloidosis and severe diastolic dysfunction		
Cardiac MRI		
Cardiac MRI of two patients with heart failure		
Goals of treatment of the patient with AHF		
Initial management of patients presenting with AHFS		
Algorithm for assessment and management of patients admitted with AHFS and CAD	92	

LIST OF FIGURES & TABLES

LIST OF TABLES

	Page	
Mechanical Disadvantages Created by Left Ventricular		
Remodeling		
Causes and precipitating factors in AHF	33	
Clinical Presentations of AHF	36	
Grading of Congestion	38	
Goals of treatment of the patient with AHF	65	
Pharmacologic Agents Used in Acute Heart Failure		
Syndromes		
Inotropic Agents Used in Acute Heart Failure		
Syndromes	82	

ACC American College of Cardiology

ACE Angiotensin-converting enzyme

Ach Acetylcholine

ACS Acute Coronary Syndrome

AF Atrial fibrillation

AHA American Heart Association

AHFS Acute Heart Failure Syndrome

AMI Acute Myocardial infarction

ANP A trial Natriuretic Peptide

AT₁ Angiotensin type 1

AT₂ Angiotensin type 2

ATP Adenosine triphosphate

BNP Brain Natriuretic Peptide

BP Blood pressure

CAD Coronary artery disease

CCU Coronary care unit

cGMP Cyclic guanosine monophosphate

CMA Cardiac myosin activators

CNP C-type natriuretic peptide

CNS Central nervous system

CPAP Continuous positive airway pressure

CSA Central sleep apnea

CT Computed tomography

CT Computed tomography

CXR Chest x-ray

DBP Diastolic blood pressure

E Epinephrine

ECG Electrocardiogram

ECM Extracellular matrix

ED Emergency Department

EF Ejection fraction

ESC European Society of Cardiology

ETT Endotracheal intubation

Fio₂ Fraction of inspired oxygen

HF Heart Failure

h-UCN2 peptide human urocortin 2

ICDs Implantable cardiac defibrillators

IVC Inferior vena cava

JVP Jugular venous pressure

LA Left atrium

LV Left Ventricle

LVH Left Ventricle hypertrophy

MIBG Metaiodobenzylguanidine

MRI Magnetic resonance imaging

NE Norepinephrine

Noninvasive intermittent positive-pressure

ventilation

NIV Non-invasive ventilation

NO Nitric oxide

NOS Nitric oxide synthase

NP Natriuretic Peptide

NT-

N-terminal pro Brain Natriuretic Peptide

proBNP

NYHA New York Heart Association

O₂sat Oxygen saturation

PAC Pulmonary artery catheters

PCWP Pulmonary capillary wedge pressure

PEEP Positive end-expiratory pressure

RA Right atrium

RAAS Renin-angiotensin-aldosterone system

RAS Renin-angiotensin system

ROS Reactive oxygen species

RV Right Ventricle

S3 Third heart sound

S₄ Fourth heart sound

SBP Systolic blood pressure

SERCA2a | Sarcoendoplasmic reticulum Ca2+

SERCA-2a | Sarcoendoplasmic reticulum Ca²⁺-ATPase type 2a

sGC Soluble guanylate cyclase

SL Sublingual

SNS Sympathetic (adrenergic) nervous system

UF	Ultrafiltration
-AR	-adrenergic receptors
c	Circumferential wall stress
m	Meridional wall stress
	l

ACKNOWLEDGEMENTS

First of all my deepest gratitude and extreme thanks to Allah the Most Greatful, the Most Merciful.

I would like to express my thanks, appreciation and profound gratitude to Professor *Dr.* Samia Ibrahim Sharaf, Professor of Anaesthesiology & Intensive Care, Faculty of Medicine, Ain Shams University, for her kind supervision, careful guidance, endless patience, great effort and continuous help throughout the course of this work.

I would like also to express my everlasting gratitude and sincere thanks to *Dr.* Sanaa Farag Mahmoud , Lecturer of Anaesthesiology & Intensive Care, Faculty of Medicine, Ain Shams University for her valuable advice, endless support, kind supervision, continuous encouragement and generous help throughout the whole work.

I wish to thank *Dr.* Hend Youssef Mohammed Ali

Lecturer of Anaesthesiology & Intensive Care, Faculty of Medicine, Ain Shams University, for her great help, suggestions, efforts, encouragement and support helped to put this work in its final shape.

Finally, my deepest thanks and gratitude are due to my father and mother for the considerable patience they have shown and the great care they have given so as to smooth the rough edge of this work.

Wishing this work be beneficial in the medical field, I hope it will satisfy you all.

Introduction

Acute Heart Failure Syndrom (AHFS) is defined as gradual or rapid change in heart failure signs and symptoms resulting in a need for urgent therapy. These symptoms are primarily the result of severe pulmonary congestion due to elevated left ventricular filling pressures (with or without low cardiac output). AHFS can occur in patients with preserved or reduced ejection fraction .

Concurrent cardiovascular conditions such as coronary heart disease, hypertension, valvular heart disease, atrial arrhythmias, and/or noncardiac conditions (including renal dysfunction, diabetes, anemia) are often present and may precipitate or contribute to the pathophysiology of this syndrome (*Remme WJ and Swedberg K.*,2001).

The combination of the aging of the population in many countries, and improved survival after acute myocardial infarction has created a rapid growth in the number of patients currently living with chronic heart failure, with a concomitant increase in the number of hospitalizations for decompensated heart failure.

Coronary heart disease is the aetiology of AHF in 60–70% of patients, particularly in the elderly population. In younger subjects, AHF is frequently caused by dilated cadiomyopathy, arrhythmia, congenital or valvular heart disease, or myocarditis (*McCullough PA et al.*,2002).

Advanced heart failure and related acute decompensation have become the single most costly medical syndrome in cardiology. Patients with AHF have a very poor prognosis. Mortality is particularly high in patients with acute myocardial infarction accompanied by severe heart failure, with a 30% 12 month mortality. Likewise, in acute pulmonary oedema a 12% in-hospital and 40% 1 year mortality have been reported. About 45% of patients hospitalized with AHF will be rehospitalized at least once (and 15% at least twice) within twelve months (*Berry C et al.*, 2001).

AHF can present itself as acute de novo (new onset of acute heart failure in a patient without previously known cardiac dysfunction) or acute decompensation of chronic heart failure(*Cleland JG et al.*,2003).

The patient with acute heart failure may presentAcute decompensated heart failure (de novo or as decompensation of chronic heart failure), Hypertensive AHF, Cardiogenic shock, High output failure, Right heart failure.

AHF is a clinical syndrome, with reduced cardiac output, tissue hypoperfusion, increase in the pulmonary capillary wedge pressure (PCWP), and tissue congestion. The underlying mechanism may be cardiac or extra-cardiac, and may be transient and reversible with resolution of the acute syndrome, or may induce permanent damage leading to chronic heart failure (*Fox KF et al.*,2001).

The clinical AHF syndrome may be classified as predominantly left or right forward failure, left or right backward failure, or a combination of these.

The diagnosis of AHF is based on the symptoms and clinical findings, supported by appropriate investigations such as ECG, chest X-ray, biomarkers, and Doppler echo cardiography (*Krumholz HM et al.*,1998).

The immediate goals of treatment are to improve symptoms and to stabilize the haemodynamic condition. An improvement in haemodynamic parameters only may be misleading, however, and a concomitant improvement in symptoms (dyspnoea and/or fatigue) is generally required. These short-term benefits must also be accompanied by favourable effects on longer-term outcomes. This is likely to be achieved by avoidance, or limitation, of myocardial damage.

Mangment include medical therapy like :Oxygen and ventilatory assistance, diuretics, vasodilators, Anticoagulation, Angiotensin converting enzyme Inhibitors, *b*-blocking agents, Inotropic agents.

Or Mechanical assist devices and heart transplantation (Intra-aortic balloon counterpulsation, Ventricular assist devices, Heart transplantation) (*Krumholz MH P et al.*,2001).