Evaluation of Pulmonary artery Pressure in Patients with Chronic Renal Failure

Thesis

Submitted for Partial Fulfillment of Master Degree in Chest Diseases and Tuberculosis

Presented by

Omnya Magdy Tawfeek Ahmed
(M.B.B.Ch. Ain Shams University)

Supervised by

Prof. Dr. Taher Abdelhamid EL-Naggar
Professor of Chest Diseases and Tuberculosis
Faculty of Medicine, Ain Shams University

Dr. Ayman Mortada Abdelmotlb
Assistant Professor of Cardiovascular Diseases
Faculty of Medicine, Ain Shams University

Dr. Ashraf Adel Gomaa
Assistant Professor of Chest Diseases and Tuberculosis
Faculty of Medicine, Ain Shams University

Faculty of Medicine
Ain Shams University
2018

قياس ضغط الشريان الرئوى لدى مرضى الفشل الكلوى المزمن

رسالة

توطئة للحصول على درجة الماجستير في الامراض الصدرية والتدرن

مقدمة من

أمنية مجدى توفيق احمد/الطبيبة

بكالوريوس الطب و الجراحة – كلية الطب جامعة عين شمس

تحت إشراف

أ.د/ طاهر عبدالحميد النجار

أستاذ الأمراض الصدرية و التدرن

كلية الطب- جامعة عين شمس

د/أيمن مرتضى عبدالمطلب

أستاذ مساعد امراض القلب والاوعية الدموية

كلية الطب- جامعة عين شمس

د/ أشرف عادل جمعه

أستاذ مساعد الأمراض الصدرية و التدرن

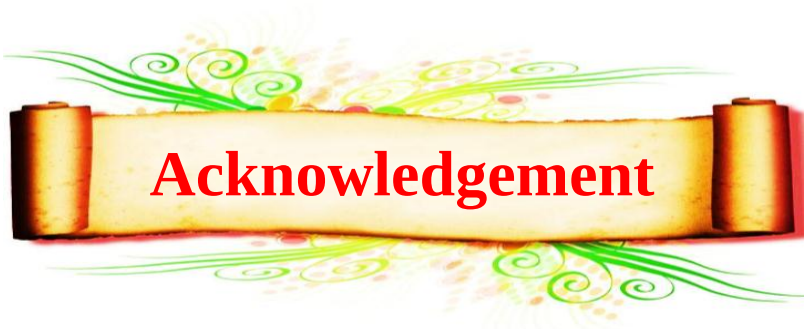
كلية الطب- جامعة عين شمس

كلية الطب

جامعة عين شمس

٢٠١٨

سورة البقرة الآية: ٣٢



Acknowledgement

First and foremost thanks to ALLAH, the Most Merciful.

I would like to express my great appreciation to ***Prof. Dr. Taher Abdelhamid EL-Naggar****, Professor of chest diseases, Faculty of Medicine, Ain Shams University; for his sincere effort, valuable advice and great confidence that he gave me throughout the whole work. His time and supreme effort are clear in every part of this work. Many thanks &gratitude for him.*

I am deeply grateful to ***Dr. Ashraf Adel Gomaa;*** *Assistant Professor of Chest Diseases and Tuberculosis- Ain Shams University; for his great directions & continuous advice all through the work.*

I would like to thank ***Dr. Ayman Mortada Abdelmotlb;*** *Assistant Professor of cardiovascular diseases, faculty of medicine, Ain Shams University; for his great help, efforts and continous advice through out the whole work..*

Contents

List of Abbreviations

6MWD	**:**	Six-minute walk distance
ALI	**:**	Acute lung injury
APAH	**:**	Assciated Pulmonary arterial hypertension
AVF	**:**	Arteriovenous fistula
BAS	**:**	Balloon atrial septostomy
CCB	**:**	Calcium Channel Blocker
CHD	**:**	Cyanotic Heart Disease
CKD	**:**	Chronic kidney disease
CO	**:**	Cardiac output
COPD	**:**	Chronic obstructive pulmonary disease
CT	**:**	Computed tomography
CTD	**:**	Connective Tissue Disease
CTEPH	**:**	Chronic thromboembolic pulmonary hypertension
DLCO	**:**	Diffusion capacity for carbon monoxide
ERA	**:**	***Endothelin receptor antagonists***
ESRD	**:**	END stage renal disease
FPAH	**:**	Familial form of idiopathic Pulmonary arterial hypertension
HIV	**:**	Human immunodeficiency virus
INR	**:**	International normalized ratio
IPAH	**:**	Idiopathic Pulmonary arterial hypertension

LHD	**:**	Left Heart Disease
MPAP	**:**	Mean Pulmonary artery Pressure
NDD	**:**	Non dialysis dependant
NIH	**:**	National Institutes of Health
NO	**:**	Nitric oxide
NOSII	**:**	Nitric oxide synthase II
PAOP	**:**	Pulmonary artery occlusion pressure
PCH	**:**	Pulmonary capillary hemangiomatosis
PGI2	**:**	Prostaglandin **I2**
PHT	**:**	Pulmonary arterial hypertension
PoPH	**:**	Porto-pulmonary hypertension
PPH	**:**	Primary Pulmonary arterial hypertension
PPHN	**:**	Persistent pulmonary hypertension of the newborn
PVOD	**:**	Pulmonary veno-occlusive disease
PVR	**:**	Pulmonary vascular resistance
RAP	**:**	Right atrial pressure
RCT	**:**	Randomized controlled trial
RHC	**:**	Right heart catheterisation
RV	**:**	Right ventricule
SPAP	**:**	Systolic pulmonary artery pressure
TAPSE	**:**	Tricuspid Annular Plane Systolic Excursion
TR	**:**	Tricuspid regurgitation

TRV	**:**	Tricuspid regurgitation Velocity
US	**:**	United States
WHO	**:**	World Health Organization
WHO FC	**:**	World health organization functional class

List of Table

List of Figures

INTRODUCTION

Pulmonary hypertension is a progressive disorder complicating heart, lung or systemic diseases with increased morbidity and mortality regardless its etiology. Pulmonary arterial hypertension (PHT) is a newly recognized disease in patients with renal disease ***(Unal et al., 2010).***

In a recent review, the prevalence of PHT in end stage renal disease (ESRD) patients was reported to be around 40-50%. Pulmonary hypertension defined as systolic pulmonary artery pressure (SPAP) > 35 mmHg at rest as estimated by Doppler echocardiography has been repeatedly reported in patients with chronic renal failure both predialysis and during regular renal replacement therapy with a high but variable prevalence ***(Havlucu et al., 2007)***.

Its presence has been recently suggested to be associated with a worse outcome. A number of causative factors have been related to this pathological finding: pulmonary artery calcifications secondary to hyperparathyroidism and hemodynamic modification related to the creation of an arteriovenous fistula (AVF) caused by a reduced ability of pulmonary vessels to accommodate the AV access-mediated elevated cardiac output possibly because of a derangement of nitric oxide-endothelin metabolism but its pathogenesis has not been completely elucidated ***(Yigla et al., 2009)***.

AIM OF THE WORK

To study pulmonary artery pressure among patients with end stage renal disease.

PULMONARY HYPERTENSION

Pulmonary hypertension (PH) is defined as a haemodynamic and pathophysiological condition characterised by an increase in mean pulmonary arterial pressure (P_{pa}) to ≥25 mmHg at rest as measured by right heart catheterisation (RHC) ***(Hoeper et al., 2013).***

Because this definition is based on hemodynamic criteria, pulmonary hypertension can be the result of a variety of diseases of different causes. Pulmonary arterial hypertension (PAH), however, should be distinctly differentiated from pulmonary venous hypertension resulting from left heart disease. PAH is characterized by elevations in the pulmonary arterial pressure and pulmonary vascular resistance (PVR) leading to right ventricular failure and premature death ***(Ghamra and Dweik, 2003).***

Thus, the definition of PAH also requires normal pulmonary artery occlusion (or wedge) pressure to exclude elevations of pulmonary artery pressure simply as a compensation for elevated pressures in the left heart. PAH is commonly caused by or associated with an underlying pulmonary, cardiac, or systemic disease (associated PAH [APAH], previously known as (secondary pulmonary hypertension). Rarely, PAH is present in the absence of an identifiable cause or associated underlying disease and is referred to as idiopathic PAH (IPAH) or primary PAH

(PPH). A familial form of IPAH (FPAH) accounts for about 6% of cases ***(Ghamra and Dweik, 2003).***

The 1973 conference provided a pathology-based classification of the disease. Pulmonary hypertension was previously classified into two categories: primary or secondary, depending on the absence or presence of identifiable causes or risk factors. The diagnosis of PPH was one of exclusion after ruling out all causes of pulmonary hypertension. The second WHO conference on pulmonary hypertension, held in Evian, France, in 1998, classified pulmonary hypertension based on similarities in the clinical features and was revised in Venice, Italy, in 2003 to reflect a treatment-based approach to pulmonary hypertension classification ***(Simonneau et al., 2004).***

Most notably, familial PAH is now referred to as heritable, with further breakdown into the genetic abnormality identified, if any. Schistosomiasis and chronic hemolytic anemia are now part of category 1 disease as associated conditions to reflect their unique importance as causative factors of PAH. Chronic thromboembolic pulmonary hypertension is no longer divided into proximal and distal, as improvements in surgical technique make this partitioning obsolete ***(Simonneau et al., 2009).***

Finally, the miscellaneous category is expanded, and now includes many conditions previously included in the "others" category of associated PAH. The latter change