



Evaluation of Management Of Prosthetic Mitral Valve Thrombosis

Thesis

*Submitted for Partial Fulfillment of Master
Degree in Cardiothoracic Surgery*

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2019

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ

سَبِّحْكَ لَا إِلَهَ إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

صدق الله العظيم

سورة البقرة الآية: ٣٢

Acknowledgment

*First and foremost, I feel always indebted to **ALLAH**, the Most Kind and Most Merciful.*

*I'd like to express my respectful thanks and profound gratitude to **Prof. Dr. Mostafa Abdel-Azim**, Professor of Cardiothoracic Surgery Faculty of Medicine -Ain Shams University for his keen guidance, kind supervision, valuable advice and continuous encouragement, which made possible the completion of this work.*

*I am also delighted to express my deepest gratitude and thanks to **Prof. Dr. Ayman Mahmoud Ammar**, Assistant Professor of Cardiothoracic Surgery Faculty of Medicine - Ain Shams University, for his kind care, continuous supervision, valuable instructions, constant help and great assistance throughout this work.*

*I am deeply thankful to **Dr. Ahmed Ahmed Fouad Abdelwahab**, Lecturer of Cardiothoracic Surgery Faculty of Medicine - Ain Shams University, for his great help, active participation and guidance.*

And a special gratitude and appreciation goes to my kind supportive wife for continuously having my back and raising my spirit.

MOHAMED ABDEL BASSET

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List of Abbreviations

Abb.	Full term
ACE.....	Angiotensin-converting enzyme
ACS.....	Acute coronary syndrome
AF	Atrial fibrillation
APSAC	Anistreplase (anisoylated plasminogen streptokinase activator complex
aPTT	Activated partial thromboplastin time
AT3	Antithrombin III
AVR.....	Aortic valve replacement
AVR.....	Aortic valve replacement
CKD	Chronic kidney disease
COR	Class of recommendation
CPB.....	Cardiopulmonary bypass
DTIs	Direct thrombin inhibitors
ECMO	Extracorporeal membrane oxygenation
ECT.....	Ecarin clotting time
ED	Emergency departments
FDA.....	Food and Drug Administration
FFP	Fresh frozen plasma
HIT.....	Heparin-induced thrombocytopenia
INR	International normalized ratio
IV	Intravenous
LA	Left atrial
LD	Limited data
LMWH	Low Molecular Weight Heparin
LOE.....	Level of evidence
LV	Left ventricle
MVR.....	Mitral valve replacement
NACs.....	Novel anticoagulants
NOPVT	Non-obstructive prosthetic valve thrombosis

NR.....	Nonrandomized
NYHA	New York Heart Association
OPVT	Obstructive prosthetic valve thrombosis
PCC.....	Prothrombin complex concentrate
PO	Oral administration
PT.....	Pro-thrombin time
PT.....	Pro-thrombin time
PVE.....	Prosthetic valve endocarditis
PVT	Prosthetic valve thrombosis
R.....	Randomized
RAO	Right anterior oblique
rfVIIa	Recombinant factor VIIa
SJM.....	St. Jude Medical
SK	Streptokinase
TA	Tranexamic acid
TAVI	Transcatheter aortic valve implantation
TAVR	Transcatheter aortic valve replacement
TE	Thromboembolism
TEE.....	Transesophageal echocardiography
TT.....	Thrombin time
TTE	Transthoracic Echocardiography
UFH	Unfractionated heparin
UFH	Unfractionated heparin
UK.....	Urokinase
VKA	Vitamin K antagonist
VKOR.....	Vitamin K1, 2,3- epoxide reductase complex
VKORC1	Vitamin K1 2,3-epoxide reductase complex, subunit 1
VTE.....	Venous thromboembolisms

Abstract

Background: Recent decades showed steady increase in the number of cases referred for redo cardiac surgery, which are associated with increased risk of morbidity and mortality compared to the first-time operations. We aimed to investigate the risk factors for hospital mortality and morbidity in patients who underwent mitral valve replacements for previous mechanical mitral valve thrombosis.

Methodolgy: Fifty patients underwent the study from Jan. 2014 till Dec. 2017 at Cardiothoracic Department, Ain Shams University. Preoperative, operative, and postoperative data were analyzed and evaluated for risk factors affecting hospital mortality and morbidity.

Results: The hospital mortality was 22%. New York Heart Association functional class, pulmonary hypertension, preoperative ejection Fraction, postoperative neurological event, total bypass time, cross clamp time, and postoperative counseling regarding anticoagulation were found to be the most important risk factors for hospital mortality.

Conclusion: Once significant valve dysfunction is first noted, re-operation should be undertaken to minimize operative risk to avoid mortality and post operative morbidities. Also, The best way to avoid morality and morbidity associated with valve thrombosis, is to avoid it happening in the first place. This can occur by improved patient education and follow up, making PT test affordable and following up the results.

Key words: mechanical valve, mitral, redo operation, functional class

INTRODUCTION

All foreign bodies (including PVs) implanted within the human cardiovascular system are thrombogenic, potentially implying the need for short-or long-term anticoagulation to prevent thrombosis, which can lead to disabling or fatal stroke. PV thrombosis is a pathological entity characterized by thrombus formation on the prosthetic structures, with subsequent PV dysfunction with or without thromboembolism (TE) ⁽¹⁾.

PV dysfunction is a complication of mechanical or biological prostheses, which can cause reduced leaflet motion or impaired leaflet coaptation, leaflet thickening, reduced or increased effective prosthesis orifice area (leading to either stenosis or insufficiency as the primary valve defect, respectively), increased transvalvular gradient or transvalvular regurgitation, with or without development of valve-related symptoms ⁽²⁾.

The risk of PV thrombosis and TE events is higher with MHVs than with BHVs, higher for PVs implanted in the mitral position versus the aortic position and higher for right-sided PVs than left-sided PVs ⁽¹⁾.

The annual rate of PV thrombosis with MHVs ranges from 0.1% to 5.7%, with higher rates observed with specific valve types, in the early perioperative period, with MHVs

implanted in the mitral and tricuspid position, and in association with sub therapeutic anticoagulation ⁽³⁾.

Certain degrees of thrombosis are commonly observed in patients with fibrotic pannus ingrowth, prosthesis degeneration, or prosthesis endocarditis ⁽²⁾.

Patients with PV dysfunction with or without thrombosis may present with progressive dyspnea and signs of heart failure or systemic embolization. Alternatively, PV thrombosis may be an incidental finding at the time of echocardiographic follow-up ⁽⁴⁾.

PV dysfunction should be suspected in patients with symptoms of acute or sub-acute onset associated with an increase in transprosthetic gradient compared with the last echocardiographic follow-up ⁽⁴⁾.