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PATIENT-PROSTHESIS MISMATCH IN AORTIC VALVE REPLACEMENT FOR AORTIC STENOSIS: IMPACT ON EARLY OUTCOME AND LEFT VENTRICULAR MASS REGRESSION FOLLOWING SURGERY

A thesis submitted for partial fulfillment of MD Degree in Cardiothoracic Surgery

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ABBREVIATIONS

Activated coagulation time	ACT
Angiotensin converting enzymes	ACE
American society of echocardiography	ASE
Aortic Peak gradient	APG
Aortic Regurgitation	AR
Aortic root enlargement	ARE
Aortic stenosis	AS
Aortic valve area	AVA
Aortic valve flow	AVF
Atrial fibrillation	AF
Aortic valve replacement	AVR
Body mass index	BMI
Body surface area	BSA
Cardiac output	COP
Canadian cardiac society	CCS
Cardiopulmonary bypass	CPB
Cerebrovascular accident	CVA
Chronic obstructive airway disease	COPD
Continous positive airway pressure	CPAP
Coronary angiography	CA
Coronary artery disease	CAD
Coronary flow reserve	CFR
Cross clamp time	CCT
Cube of diastolic dimension	$(Dd)^3$
Cube of systolic dimension	$(Ds)^3$
Effective orifice area	EOA
Effective orifice area indexed	EOAI
Electrocardiogram	ECG
End diastolic volume	EDV
Ejection fraction	EF
End systolic volume	ESV
Fractional shortening	FS
High performance	HP
3-hydroxy-3-methylglutaryl-coenzyme A	HMG-CoA
Intensive therapy unit	ITU
Intercostal chest tube	ICT

International normalized ratio	INR
Interventicular septum thickness	IVSd
Internal geometric area	IGA
Intra-aortic ballon pump	IABP
Lactate dehydrogenase	LDH
Left ventricle	LV
Left ventricular hypertrophy	LVH
Left ventricular ejection fraction	LVEF
Left ventricular end diastolic pressure	LVEDP
Left ventricular internal dimension	LVID
Left ventricular diastolic diameter	LVIDd
Left ventricular systolic diameter	LVISd
Left ventricular mass	LVM
Left ventricular mass index	LVMI
Low density lipoprotein	LDL
Low cardiac output	LCOP
Mitral regurgitation	MR
Myocardial Infarction	MI
Non significant	NS
Non steroidal anti-.inflamatory drugs	NSAID
New York Heart association	NYHA
Patient prosthesis mismatch	PPM
Posterior wall thickness in diastole	PWTd
Polytetrafluoroethylene	PTFE
Pressure gradient	PG
Red blood cels	RBCs
Renal failure	RF
Relative ventricular wall thickness	RWT
Supra-annular valve	SAV
Standrad deviation	SD
Stentless porcine aortic valve	SPV
Strok volume	SV
Transient ischeamic attack	TIA
Transvalvulat pressure gradient	TPG
Trans-thoracic echocardiography	TTE
Trans-oesophageal echocardiography	TOE
Thromboebolic	TE
Von Willibrand Factor	vWF

INTRODUCTION

In patients with aortic valve stenosis, left ventricular hypertrophy (LVH) develops as an adaptive process in response to elevated pressure in the left ventricle. Aortic valve replacement (AVR) is effective for relief of excessive afterload, but significant LVH often remains following AVR. Because severe LVH is a well-known hazard of cardiac events, its regression is a major concern. (125,189)

Valve prosthesis-patient mismatch (PPM) is present when the effective orifice area (EOA) of the inserted prosthetic valve is too small relative to body surface area (BSA). PPM is defined as a valve effective orifice area indexed for body surface area (IEOA) equal to or greater than 0.8 to 0.9 cm^2/m^2. (134,137) This is a frequent problem in patients undergoing AVR (20% to 70% prevalence), and its main hemodynamic consequence is to generate high transvalvular gradients through normally functioning prosthetic valves. (189)

The presence of left ventricular hypertrophy (LVH) is associated with a two- to threefold increase in cardiovascular-related mortality. (190) Increased LV mass is an independent predictor of mortality in patients with systemic arterial hypertension as well as in normotensive patients. (191) LV hypertrophy is a strong independent risk factor for mortality in patients undergoing aortic valve replacement (AVR). (181)

Lund and associates recently showed that the indexed left ventricular mass (ILVM) and its regression following AVR is closely linked to long-term survival. and it has been reported to be a predictor of postoperative elevated trans-valvular gradients and residual LVH. (192)

Left ventricular mass (LVM) is calculated with the corrected American Society of Echocardiography (ASE) formula:

$$LVM = 0.8[1.04(IVSd+LVDd+PWTd)^3-LVID^3]-13.6$$

Where IVS*d* is the end-diastolic interventricular septum thickness, LVID*d* is the LV end-diastolic internal diameter, and PWT*d* is the LV end-diastolic posterior wall thickness. Residual LV hypertrophy was defined as a LV mass index more than 131 g/m^2 in males and more than 100 g/m^2 in females. (190)

Normalization of LV mass is therefore a crucial goal of AVR. Unfortunately, the extent of LV mass regression may vary extensively from one patient to the other and it is often incomplete. These findings underline the importance of identifying and, whenever possible, avoiding risk factors for persisting LV hypertrophy following valve replacement. Residual transprosthetic pressure gradients are important to consider because an increased gradient will evidently result in an increased LV workload, thus potentially jeopardizing the regression of LV mass after AVR. (121, 123)

REVIEW OF LITERATURE

HISTORY OF AORTIC VALVE REPLACEMENT

In 1898, Samway was the first to forecast intervention for the treatment of obstructed heart valves. (1)

In 1948, Smithy and coworker reported a method for performing transaortic and transventricular aortic valvotomy in experimental animals. But unfortunately, Smithy died in the same year at the age of 34 years from aortic stenosis. (2)

In 1950, Bailey and associates performed a retrograde aortic valve dilatation using a triangular expansile instrument through the carotid artery, but difficulties with arterial dissection led them to approach this operation through the ventricular apex and with this revised approach they were able to perform aortic valve commissurotomy with reasonable success.(3)

On September 1952, Hufnagel implanted the first artificial valve into the descending thoracic aorta and started a new era in cardiac surgery. This pioneer work was followed by replacement of the aortic valve with an artificial caged ball valve in the subcoronary position by Harken and associates in 1960 in the same year that Star replaced the mitral valve with a caged ball valve. (4) Figure 1

Figure 1: Hufnagel Valve

In 1965 Binet and Carpentier performed the first aortic valve replacement using a specially prepared aortic valve xenograft, this was followed by a series of xenograft implantations throughout world. (5)

In 1967, Donald Ross introduced the use of a pulmonary autograft to replace the aortic valve. It is now most commonly used in the pediatric population. (6)

In 1980s, surgeons increasingly encountered patients requiring re-operations who had previously received bioprostheses and the durability of these valves was questioned. At the same time, mechanical prostheses enjoyed a resurgence of popularity because of new materials and designs with low thrombogenencity and better flow characteristics which were comparable to the bioprostheses valves and unquestionably higher durability and structural integrity. (5, 7)

In 1990s, surgeons were still searching for the ideal valve substitute. Although there has been no significant break through, bioprostheses valves treated with calcium retardation agents and non stented xenografts both are promising. Additionally, new preservation techniques and better distribution have made homograft valves more attractive as a valve substitute. (7)

As is true in all areas of surgery, minimally invasive approaches to valve replacement are gaining in interest. In 2002, the first human percutaneous aortic valve replacement (PAVR) became a reality. (8)

Over the last 5 years improvements in percutaneous approaches to implantation of prosthetic aortic valves have made it a potential therapeutic option for patients with severe symptomatic aortic valve stenosis. Technical and device issues are being refined, and percutaneous aortic valve replacement is showing promise in ongoing clinical trials. (9)

EMBRYOLOCICAL DEVELOPMENT OF THE AORTIC VALVE

The primitive heart tube consists of 5 segments which are: the venous sinus, the atrial segment, the inlet component of ventricular segment, the outlet component of the ventricular segment and the arterial segment. The outlet component of the ventricular segment is the conus which becomes continuous with the major artery "the truncus arteriosus". At an early stage, together they may be called the trunco-conal channel. The aortic root and the pulmonary trunk are derived from partitioning of the trunco-conal channel by fusion of the lining of the trunco-conal ridges "trunco-conal septum". Theses ridges follow a spiral course; therefore the septum that results from fusion of the two ridges has a spiral shape. (10)

The lower most part of the trunco-conal septum fuses with the developing ventricular septum to share in dividing the ventricular outflow into right and left ventricular components. At the junction of the cardiac and arterial ends of the trunco-conal canal channel, the aortic and the pulmonary semilunar valves are formed. The right and left cusps of the aortic valve are derived from the trunco-conal septum. The non septal cusp is derived from non specialized tissue in the lining of the trunco-canal channel. (11, 12) Figure 2