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A SINGLE-CENTER COMPARATIVE ANALYSIS OF STERNOTOMY AND MINIMALLY-INVASIVE APPROACH TO MITRAL VALVE SURGERY: EVALUATING OUTCOMES AND HEALTHCARE RESOURCE IMPLICATIONS

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Introduction

Mitral valve disease is a common cardiac condition affecting millions of people worldwide. Surgical intervention is often necessary to repair or replace the diseased valve, and the two main surgical approaches mostly used are: full sternotomy (STER) and minithoracotomy (MTH). Full sternotomy involves dividing the sternum to access the heart, while minithoracotomy involves making a smaller incision between the ribs. Both approaches have advantages and disadvantages, and the choice of procedure depends on various factors such as the patient's overall health, the severity of the valve disease, and the surgeon's preference and expertise.

While MTH for mitral valve repair appears to offer advantages like faster recovery and shorter hospital stays compared to traditional open-heart surgery, its role remains unclear. Concerns persist regarding potential drawbacks such as increased operative time, complication rates, and the long-term durability of the repair. Current guidelines do not yet offer definitive recommendations for MTH.

In our Center the first cases of MTH were performed in 2004 but the numbers increased after 2010 assessing for 10 to up to 40 cases per year. This thesis aims to compare the outcomes of full sternotomy and minithoracotomy in patients undergoing mitral valve surgery in our Center. The primary objective is to assess the differences in mortality, morbidity, and long-term survival between the two approaches. Secondary objectives include evaluating the impact of each approach on factors such as operative time, blood loss, length of hospital stay as well as survival at follow-up and freedom from reoperation.

1. Surgical Anatomy of the Mitral Valve Apparatus

The mitral valve apparatus is a complex and integrated "**anatomo-functional unit** " essential for unidirectional blood flow between the left atrium and left ventricle. This apparatus comprises five key components: the annulus, leaflets, chordae tendineae, papillary muscles, and the left atrium itself (1). These components function synergistically to ensure proper valve closure during systole and opening during diastole. Any disruption to the structural integrity or functional interplay of these components can impair valvular performance, potentially leading to mitral regurgitation (insufficiency), mitral stenosis, or a combination of both (2,3).

1.1 The Mitral Annulus

The ***mitral annulus***, a critical component of the mitral valve apparatus, is an incomplete ring of dense collagenous tissue that encircles the posterior leaflet. Notably, a distinct fibrous ring is absent around the anterior leaflet, which instead exhibits continuity with the aortic valve and aortic wall (4). This anatomical and functional continuity is further underscored by the close correlation between mitral annular deformation and aortic root dynamics throughout both systole and diastole (5). The inherent elasticity and distensibility of the mitral annulus are essential for its dynamic function. (2) While annular contraction during systole contributes to effective leaflet coaptation, this compliance also renders the annulus susceptible to dilation in the setting of left atrial or ventricular enlargement, potentially contributing to mitral regurgitation (4). Throughout the cardiac

cycle, the mitral annulus undergoes significant conformational changes driven by external forces originating from the atrial and ventricular musculature. These changes are primarily mediated by two types of motion: sphincteric contraction and translation. Sphincteric contraction reduces the annular area by up to 25% during ventricular mid-systole (6). Atrial contraction accounts for over 90% of this annular narrowing, with ventricular contraction contributing the remaining 10% (7). Translational motion, occurring along the long axis of the left ventricle, is a consequence of ventricular base torsion and coincides with left atrial filling. This motion serves to reduce stress on the valve leaflets while maintaining their coaptation (8). The upper limits of the systolic annular dimension are a perimeter of 125 mm and an area of 675 mm^2 (7).

1.2 Leaflets

The mitral valve comprises two ***leaflets***, anterior and posterior, each originating from the annulus and extending into the left ventricular outflow tract. The leaflets are precisely dimensioned to ensure complete and effective coaptation during systole (9). These leaflets exhibit distinct morphological characteristics. The longer posterior leaflet accounts for approximately two-thirds of the annular circumference, while the anterior leaflet occupies the remaining third (4). This differential length contributes to the unique saddle-shaped configuration of the mitral valve, optimizing leaflet apposition and minimizing stress during closure. The atrial surface of the leaflets displays regional variations in texture. The portion closest to the annulus appears smooth and translucent. Conversely, the distal portion, approximately 1 cm from the free edge, exhibits a rougher

texture due to its rich content of hydrophilic proteins. This "rough zone" plays a crucial role in ensuring secure leaflet coaptation during ventricular systole (9). The ventricular surface, particularly that of the anterior leaflet, is characterized by an intricate network of collagen fibers that originate from the chordal insertions and extend towards the annulus (9). This fibrous network provides structural support and contributes to the leaflet's mechanical properties. The posterior leaflet, also termed the "mural" leaflet, arises from the posterior two-thirds of the annular circumference. Two distinct indentations, or clefts, divide this leaflet into three scallops along its free margin (10). These clefts enhance leaflet flexibility, promoting optimal coaptation along the valve closure line (11). Although extending nearly the entire length of the leaflet, the clefts do not reach the annulus. The resulting scallops are designated P1 (laterally), P2 (centrally), and P3 (medially). While lacking distinct clefts, the anterior (or "aortic" or "septal") leaflet is similarly divided into three corresponding segments (A1, A2, A3) based on its adjacency to the posterior leaflet scallops (3). In some instances, an additional leaflet, referred to as a "commissural," "accessory," or "junctional" leaflet, may be present at the anterolateral (A1-P1) or posteromedial (A3-P3) commissures (12).

1.3 Chordae Tendineae

The ***chordae tendineae*** are fibrous structures that originate from the papillary muscle heads, exhibiting considerable variation in their branching patterns. These cords fan out and insert onto the ventricular surface of both the anterior and posterior leaflets (9) . As direct extensions of the papillary muscles, the chordae tendineae form an integral part

of the mitral subvalvular apparatus, establishing a crucial link between the mitral valve and the left ventricle. Notably, approximately half of the chordae from each leaflet attach to each of the two papillary muscles (4). Based on their leaflet insertion sites, the chordae tendineae can be classified into three main types:

- Primary ("marginal") chordae: These attach to the free edges of the leaflets, playing a critical role in preventing leaflet prolapse and ensuring proper coaptation during valve closure.
- Secondary ("basal") chordae: Thicker than primary chordae, these insert into the rough zone of the leaflets, contributing to leaflet stability and ventricular geometry.
- Tertiary chordae: These are exclusively attached to the basal portion of the posterior leaflet.

The primary and secondary chordae serve distinct functions. The thicker primary chordae are essential for maintaining leaflet apposition and facilitating valve closure. Their importance is highlighted by the observation that severing these chordae results in acute mitral regurgitation. In contrast, the secondary chordae appear less critical for preventing regurgitation, as their disruption does not typically lead to immediate valvular incompetence. Instead, these chordae are thought to play a role in maintaining the normal dimensions and geometry of the left ventricle. The precise function of the tertiary chordae remains to be fully elucidated (2).

1.4 Papillary Muscles

The ***papillary muscles*** are specialized appendages of cardiac muscle that project into the ventricular cavities. In the left ventricle, these typically consist of two distinct

muscular structures originating from the middle third of the ventricular wall. Based on their anatomical location, they are designated as the anterolateral and posteromedial papillary muscles. The anterolateral papillary muscle generally comprises a single muscular head and receives dual blood supply from the left anterior descending and circumflex arteries. In contrast, the posteromedial papillary muscle commonly consists of two heads and is supplied solely by the posterior descending artery, rendering it more susceptible to ischemia (8,13). As the anchoring points for the chordae tendineae, the papillary muscles play a critical role in maintaining mitral valve competence. Their coordinated contraction with the left ventricle generates tension on the chordae tendineae, preventing leaflet prolapse during systole and ensuring unidirectional blood flow.

2. Physiopathology of the Mitral Valve

2.1. Mitral Stenosis

Mitral stenosis (MS) is defined as a narrowing of the mitral valve orifice, resulting in an area less than 2 cm^2 (normal range: 4-6 cm^2). This restricted orifice creates an impediment to blood flow from the left atrium to the left ventricle, leading to elevated left atrial pressure, increased transvalvular pressure gradients, and ultimately, impaired cardiac output (14,15). While rheumatic fever remains the predominant cause of MS globally, degenerative MS due to mitral annular calcification (MAC) is increasingly prevalent, particularly in industrialized nations (14).

2.1.1. Rheumatic Mitral Stenosis

Despite its declining incidence in developed countries, rheumatic heart disease (RHD) continues to pose a significant global health burden. It is estimated that 15.6 million individuals are affected by RHD, with approximately 470,000 new cases of acute rheumatic fever occurring annually. Of these, 60% progress to RHD, and a small but significant proportion (1.5%) succumb to RHD-related complications each year. Among individuals with RHD, 25% present with isolated MS, while 40% exhibit combined MS and mitral regurgitation (16). RHD typically develops following a childhood infection with Group A beta-hemolytic Streptococcus. The initial pharyngeal infection triggers an autoimmune response, leading to the production of antibodies that cross-react with host tissues, including the heart valves. This molecular mimicry results in chronic inflammation and scarring of the mitral valve, ultimately leading to stenosis (17). Rheumatic MS is more

prevalent in developing countries and tends to progress more rapidly than other forms of MS. Recurrent episodes of acute rheumatic fever increase the risk of developing RHD, and although any cardiac valve can be affected, the mitral valve is most commonly involved (18).

Characteristic features of rheumatic MS include (13):

- Commissural fusion
- "Fish-mouth" appearance of the valve orifice
- Leaflet thickening, especially at the free edges
- Shortening and fusion of the chordae tendineae

2.1.2. Mitral Annular Calcification

Mitral annular calcification (MAC) affects an estimated 9-15% of the general population, with a higher prevalence (up to 40%) among the elderly. Notably, a significant proportion (nearly 50%) of patients undergoing transcatheter aortic valve implantation (TAVI) for aortic stenosis also exhibit MAC, with severe MAC observed in 9.5% of these cases (19).

2.1.3. Other Etiologies

Other less common causes of MS include (13):

- **MAC:** Predominantly affects elderly patients with advanced renal failure.

- **Radiation-induced valvulopathy:** Typically manifests 10-20 years after mediastinal radiotherapy.
- **Congenital MS:** Rare.
- **Systemic inflammatory diseases:** Conditions like systemic lupus erythematosus and rheumatoid arthritis can cause valve inflammation and subsequent stenosis.
- **Functional MS:** Caused by obstructive lesions such as large atrial myxomas or endocarditic vegetations.

2.1.4. Pathophysiology

A normal mitral valve area ranges from 4 to 6 cm^2. In mitral stenosis, when this area falls below 2 cm^2, a diastolic pressure gradient develops between the left atrium and left ventricle. This gradient arises from the obstruction to blood flow through the stenotic valve. Consequently, left atrial pressure rises to overcome the resistance and maintain adequate ventricular filling. Elevated left atrial pressure has several detrimental effects. Firstly, it causes left atrial enlargement, predisposing to atrial arrhythmias, particularly atrial fibrillation. Secondly, it transmits backward pressure to the pulmonary vasculature, leading to pulmonary hypertension, edema, and ultimately, right ventricular failure with tricuspid regurgitation. The increased atrial pressure also impairs atrial contractility. The stretched atrial myocytes lose their ability to generate effective systolic contractions, further compromising ventricular filling and cardiac output. This, coupled with the reduced flow through the stenotic valve, culminates in diminished cardiac output and the development of congestive heart failure (13,15). The pathophysiology of mitral stenosis explains why tachycardia is particularly deleterious in these patients. A rapid heart rate

shortens diastolic filling time, further exacerbating the transvalvular gradient and compromising ventricular filling (13).

2.1.5. Diagnosis

Echocardiography is the gold standard for diagnosing and assessing the severity of mitral stenosis. The quantitative evaluation focuses on three primary parameters (20): mean diastolic pressure gradient (21), mitral valve area, and (22) secondary alterations, including left and right atrial chamber size and pulmonary artery pressure.

- **Mean diastolic pressure gradient:** A mean gradient >10 mmHg is consistent with severe MS, 5-10 mmHg with moderate MS, and <5 mmHg with mild MS.
- **Mitral valve area:** Can be measured using two-dimensional (2D) or three-dimensional (3D) planimetry.
- **Secondary alterations:** Include left atrial enlargement, pulmonary hypertension, right heart dilation, and functional tricuspid regurgitation.

Another valuable diagnostic tool is exercise stress echocardiography. This is particularly useful when there is a discrepancy between symptom severity and the resting echocardiographic findings. For example, in asymptomatic patients with severe MS, exercise testing can help determine if they can tolerate exertion without developing symptoms. Similarly, in patients with moderate MS and significant symptoms, stress echocardiography can unmask hemodynamically significant stenosis that may not be apparent at rest (13).

2.2. Mitral Regurgitation

Mitral regurgitation (MR), also known as mitral insufficiency, is characterized by the abnormal backflow of blood from the left ventricle to the left atrium during systole (23). This retrograde flow occurs due to incomplete closure of the mitral valve leaflets. MR is one of the most prevalent valvular heart diseases globally, affecting over 175 million individuals worldwide (24). Its prevalence is rising, primarily attributed to increasing life expectancy and the aging population. Prevalence increases from less than 1% in individuals younger than 45 years to over 11% in those older than 75 years, significantly impacting survival (23). MR carries a significant burden of mortality and morbidity. The mortality rate in patients over 50 years with moderate MR receiving medical therapy is 3%, rising to 6% in those with severe MR (25). However, nearly 50% of patients with severe MR are not candidates for conventional surgical correction due to left ventricular dysfunction, advanced age, and comorbidities (23).

2.2.1. Etiology

MR can be classified as acute or chronic. Chronic MR is further categorized as primary (organic) or secondary (functional). **Acute MR** often results from specific events such as:

- Trauma
- ST-elevation myocardial infarction (STEMI) with papillary muscle rupture or chordal rupture
- Infective endocarditis with leaflet perforation or chordal rupture (26)

Spontaneous chordal rupture has also been reported in degenerative mitral valves, particularly in myxomatous degeneration and Marfan syndrome (23).

Chronic Primary MR is attributed to structural abnormalities of the mitral valve apparatus itself, including the leaflets, chordae tendineae, papillary muscles, and annulus. Common causes include:

- **Mitral valve prolapse (MVP):** The most frequent cause of MR, defined echocardiographically as systolic displacement of the leaflets ≥2 mm above the mitral annular plane (13). Two main types of degenerative leaflet changes contribute to MVP:
    - Barlow's disease: Characterized by myxomatous degeneration with abnormal accumulation of mucopolysaccharides.
    - Fibroelastic deficiency: Involves abnormalities in connective tissue structure leading to loss of mechanical integrity (27).
- Other causes of primary MR include infective endocarditis, MAC, rheumatic heart disease, connective tissue disorders, congenital malformations (e.g., cleft mitral valve), and certain medications (e.g., anorexiants) (13).

While rheumatic heart disease remains a leading cause of primary MR in developing countries, myxomatous degeneration with MVP is the most common etiology in developed nations (28). Familial aggregation studies have also demonstrated a genetic predisposition to degenerative myxomatous MR (29).

Chronic Secondary MR results from alterations in left ventricular geometry that disrupt the normal function of the mitral valve apparatus. It can be further classified as:

- **Ischemic MR:** Left ventricular dysfunction due to ischemia-induced structural remodeling, often secondary to coronary artery disease. Over 10% of patients with coronary artery disease have moderate to severe MR, impacting long-term survival (30).
- **Non-ischemic MR:** Occurs in various non-ischemic cardiomyopathies, including dilated cardiomyopathy, restrictive cardiomyopathy, and hypertrophic cardiomyopathy. It can also be secondary to atrial fibrillation and diastolic heart failure, with resultant left atrial and ventricular dilation (13,23).

2.2.2. Pathophysiology of MR

The pathophysiology of MR involves impaired coaptation of the mitral valve leaflets, leading to retrograde blood flow during ventricular systole. Because the regurgitant mitral orifice is functionally in parallel with the aortic valve, MR reduces the resistance to left ventricular ejection. Consequently, MR augments left ventricular emptying. Nearly 50% of the regurgitant volume is ejected into the left atrium before aortic valve opening. The volume of MR depends on the size of the regurgitant orifice and the pressure gradient between the left ventricle and left atrium. Left ventricular systolic pressure, and therefore the ventriculoatrial gradient, is influenced by systemic vascular resistance. Increases in preload or afterload, and decreases in contractility, all increase left ventricular volume, which enlarges the mitral annulus and thereby the regurgitant orifice. Initially, the left ventricle compensates for MR by emptying more completely and by increasing preload (via the Frank-Starling mechanism). Because acute MR reduces

left ventricular end-systolic pressure and radius, left ventricular wall stress decreases substantially, allowing a reciprocal increase in the extent and velocity of myocardial fiber shortening and a reduction in end-systolic volume. When MR, especially if severe, becomes chronic, left ventricular end-diastolic volume increases and end-systolic volume returns to normal. According to Laplace's law, which relates wall stress to the product of intraventricular pressure and radius, in the chronic compensated stage of severe MR, the increased left ventricular end-diastolic volume increases wall stress to normal or supranormal levels. The resulting increase in left ventricular end-diastolic volume and mitral annular diameter creates a vicious cycle in which MR worsens. In patients with chronic MR, both left ventricular end-diastolic volume and mass are increased; that is, a typical volume-overload (eccentric) hypertrophy develops. However, the degree of hypertrophy is often not proportional to the degree of LV dilation, and thus the ratio of LV mass to end-diastolic volume may be lower than normal. Nonetheless, the reduction in afterload allows ejection fraction to be maintained in the normal to supranormal range. With the onset of decompensation, left ventricular chamber stiffness increases, causing diastolic pressure to rise. End-systolic volume, preload, and afterload increase, while ejection fraction and stroke volume decrease. Plasma atrial natriuretic peptide levels also increase in response to volume overload and are higher in patients with symptomatic decompensation.

2.2.3. Diagnosis

Similar to mitral stenosis, echocardiography is the primary imaging modality for diagnosing and evaluating mitral regurgitation. Multiple parameters are used to quantify MR severity, and these should be integrated to obtain a comprehensive assessment (13)

. An integrated approach incorporating qualitative, semi-quantitative, and quantitative measures of MR, along with assessment of left ventricular and left atrial size, is recommended (19).

2.2.3.1. Assessment of Primary MR

In primary MR, the effective regurgitant orifice area (EROA) is a key parameter routinely measured and strongly correlates with mortality risk. An EROA greater than 20 mm^2 is associated with increased mortality compared to the general population, with the risk progressively increasing above 40 mm^2 (31). Three-dimensional transesophageal echocardiography (3D TEE) provides detailed visualization of the mitral valve, akin to direct surgical inspection, and is particularly useful in evaluating primary MR (19).

Exercise echocardiography is valuable for assessing changes in MR volume and pulmonary pressures during peak exercise. It is especially helpful in patients with symptoms discordant with the severity of MR at rest (32).

In asymptomatic patients with severe primary MR and normal left ventricular and left atrial size, serial monitoring of serum B-type natriuretic peptide (BNP) can be useful. Low BNP levels are associated with a lower risk of mortality and can provide valuable prognostic information during follow-up (33).

2.2.3.2. Assessment of Secondary MR

While the echocardiographic methods and parameters used to assess secondary MR are similar to those used for primary MR, it's important to note that lower thresholds for EROA and regurgitant volume are applied to define severe secondary MR. This is

because in patients with heart failure, the total left ventricular output is reduced, potentially leading to an underestimation of regurgitant volume. Calculating the regurgitant fraction while accounting for lower flow rates has shown to have important prognostic implications. Furthermore, the crescent-shaped regurgitant orifice typical of secondary MR can lead to an underestimation of EROA (19). An EROA ≥ 30 mm^2 calculated by the 2D proximal iso-velocity surface area (PISA) method generally corresponds to severe secondary MR.

2.2.3.3. Role of Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance imaging (CMR) provides accurate measurements of regurgitant flow and is the most accurate non-invasive technique for measuring left ventricular end-diastolic volume, end-systolic volume, and mass. While echocardiography remains more reliable for detailed visualization of mitral valve structure and function, CMR offers a promising method for more precise assessment of MR severity. The combined use of 3D echocardiography, CMR, and exercise echocardiography can be particularly helpful in identifying patients with severe MR, especially when resting 2D echocardiography findings are inconclusive.

3. Surgical Treatment Indications

Surgical intervention should be considered in patients with functional disability and/or in those who are asymptomatic or mildly symptomatic but exhibit progressive deterioration of left ventricular (LV) function or ongoing LV enlargement. Without surgical treatment, the prognosis for patients with MR and heart failure is poor. Therefore, mitral valve repair or replacement is recommended for symptomatic patients.

The 2021 guidelines developed by the Task Force for the management of valvular heart disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS) provide specific recommendations for the treatment of primary MR, secondary MR, and mitral stenosis.

3.1. Primary MR

Urgent surgical intervention is indicated for patients with severe acute MR. In cases where papillary muscle rupture is the underlying cause, valve replacement is generally necessary. Surgery is also recommended for symptomatic patients with severe primary MR and acceptable surgical risk as determined by the heart team (Class IB recommendation). The presence of any of the following factors warrants consideration for surgery, irrespective of symptomatic status, as they are associated with worse outcomes (19):

- Left ventricular ejection fraction (LVEF) < 60%

- Left ventricular end-systolic diameter (LVESD) > 40 mm (34) (Class IB recommendation)
- Left atrial (LA) volume > 60 mL/m² or LA diameter > 55 mm (35)
- Systolic pulmonary artery pressure (SPAP) > 50 mmHg
- Atrial fibrillation (AF)

3.2. Surgical Approaches

Mitral valve repair is the preferred surgical approach when durable results are anticipated (Class IB recommendation) as it is associated with improved survival compared to valve replacement. MR due to valve prolapse can be effectively repaired with a low risk of recurrence and reoperation. However, the reparability of rheumatic lesions, extensive valve prolapse, and particularly leaflet calcification or extensive annular calcification is more challenging. When repair is not feasible, valve replacement with preservation of the subvalvular apparatus is preferred (19).

3.3. Secondary MR

For secondary MR, surgery is recommended in patients with severe disease undergoing concomitant coronary artery bypass grafting or other cardiac surgery (Class IB recommendation) (19). The surgical approach should be tailored to the individual patient. In selected patients without advanced left ventricular remodeling, mitral valve repair with a completely undersized rigid ring can restore valve competence, improve

symptoms, and lead to reverse left ventricular remodeling. In patients with echocardiographic predictors of repair failure, additional valvular/subvalvular techniques or valve replacement with chordal preservation may be considered. Valve replacement prevents MR recurrence, although this does not necessarily translate into reverse left ventricular remodeling or improved survival. Indications for isolated mitral valve surgery in secondary MR are restrictive due to significant procedural risk, high MR recurrence rates, and the lack of a proven survival benefit (19).

3.4. Transcatheter Edge-to-Edge Repair (TEER)

For patients deemed unsuitable for conventional surgery, transcatheter edge-to-edge repair (TEER) with the MitraClip™ system is an alternative treatment option. Two randomized controlled trials (COAPT and MITRA-FR (36)) have evaluated its safety and efficacy in patients with symptomatic heart failure and persistent severe secondary MR despite medical therapy (Class IB recommendation). Results indicate that the procedure is safe and effectively reduces MR for up to 3 years. However, in the MITRA-FR trial, MitraClip™ implantation did not impact the primary endpoint of all-cause mortality or heart failure hospitalization at 12 months and 2 years compared to medical therapy alone. In contrast, the COAPT trial demonstrated that MitraClip™ implantation substantially reduced the primary endpoint of cumulative heart failure hospitalizations, as well as several predefined secondary endpoints, including all-cause mortality at 2 years. The conflicting results of these two trials have generated considerable debate. Therefore, the MitraClip™ system should be considered in selected patients with severe secondary MR

who meet the COAPT inclusion criteria, are receiving optimal medical therapy, and are as close as possible to the patients actually enrolled in the study. In patients with less severe MR (EROA < 30 mm²) and advanced left ventricular dilation/dysfunction, the prognostic benefit of MitraClip™ remains unproven (37). Patients with left and/or right ventricular failure and no option for revascularization may be better served by cardiac transplantation or left ventricular assist device implantation. Valve intervention is generally not an option when LVEF is < 15%. Transcatheter mitral valve repair systems other than MitraClip™, as well as transcatheter mitral valve replacement devices, are currently under investigation, but clinical data remain limited (19).

3.5. Mitral Stenosis

In mitral stenosis, the type and timing of treatment should be determined based on clinical features, valve anatomy, and the subvalvular apparatus. Generally, intervention is reserved for patients with moderate to severe, clinically significant rheumatic mitral stenosis (valve area ≤ 1.5 cm²). Percutaneous mitral commissurotomy (PMC) has significantly impacted the management of mitral stenosis. In Western countries, where the incidence of rheumatic fever and the number of PMCs performed are low, this treatment should be limited to experienced operators in specialized centers to improve safety and procedural success rates. Efforts should be made to increase the availability of PMC in developing countries where access to treatment is limited due to economic constraints. PMC should be considered the first-line treatment for selected patients with mild or

moderate calcification or a compromised subvalvular apparatus who present with favorable clinical characteristics.

4. Mitral Valve Pathology Treatment

4.1. Surgical approaches to the Mitral Valve

Traditional surgery with *conventional sternotomy* remains the gold standard for treating most mitral valve diseases. Sternotomy provides excellent exposure to the mitral valve and allows for concomitant procedures such as aortic valve surgery, coronary revascularization, ascending aorta, and aortic arch repair. The mitral valve can be accessed through standard left atriotomy through the interatrial groove, superior left atrial approach through the dome of the left atrium between the aorta and the superior vena cava, and right atrial approach with transeptal access. Central cannulation with bicaval cannulae for the venous line and standard ascending aorta for the arterial line can be performed in case of sternotomy.

Minimally invasive mitral valve surgery encompasses techniques aimed at reducing surgical trauma and improving patient outcomes. Most minimally invasive mitral valve repair procedures utilize a *right mini-thoracotomy* approach, characterized by a 4-6 cm incision in the right submammary fold at the fourth intercostal space. In our Center, we use also 3D camera support to have a better view of the valve anatomy and to perform the operation with more accuracy. The CPB is instituted through femoral vein and arterial cannulation with retrograde perfusion. Relative contraindications to femoral cannulation and retrograde perfusion include severe peripheral vascular disease, abdominal aortic aneurysm, and central aortic atherosclerosis. Thus, patients who undergo a minimally invasive approach need to perform a preoperative Angio CT scan.

4.1.1. Mitral Valve Repair

Mitral valve repair aims to correct structural abnormalities of the valve without complete removal. A thorough understanding of the underlying pathogenetic mechanism is crucial as it guides the surgical approach and influences the prospects for successful intervention. The Carpentier classification system is widely recognized as an essential tool in evaluating mitral valve pathology. It focuses primarily on leaflet motion and effectively categorizes morphological and functional abnormalities of the mitral valve. This classification provides a framework for understanding the various pathological conditions affecting the mitral valve, allowing surgeons to plan tailored surgical interventions (23).

Carpentier Classification: (38)

- **Type I: Normal leaflet motion**
    - Caused by annular dilation or leaflet perforation
    - Regurgitant jet directed centrally
- **Type II: Excessive leaflet motion**
    - Caused by papillary muscle rupture, chordal rupture, or redundant chordae
    - Eccentric jet directed away from the involved leaflet
    -
- **Type IIIa: Restricted leaflet motion in systole and diastole**
    - Commonly caused by rheumatic heart disease
    - Normal papillary muscles
    - Jet may be directed centrally or eccentrically
- **Type IIIb: Restricted leaflet motion in systole**

- Caused by papillary muscle dysfunction or left ventricular dilation
- Abnormal papillary muscles
- Jet may be directed centrally or eccentrically

Surgical Technique

There are several techniques for repairing the mitral valve depending on the physiopathology. Considering that the lateral scallop of the posterior leaflet (P1) is usually not affected by prolapse, it is used as a reference point to compare all other segments. The specific repair technique applied depends primarily on the site of the prolapse. All leaflet and chordal repair techniques are accompanied by the incorporation of an annuloplasty.

In case of posterior prolapse (approximately 75% of patients with degenerative MR) the repair can be performed through quadrangular or triangular resection of the prolapsing part of the posterior leaflet (usually P2), leaflet plication, artificial chordae positioning. When the prolapse involves both leaflets an edge-to-edge repair by suturing both the prolapsing parts of the opposite leaflets can be performed. This technique is also a very valuable adjunct in patients who have the potential for Systolic Anterior Motion of the mitral valve.

4.1.2. Mitral Valve Replacement

Mitral valve replacement involves removing the damaged valve and replacing it with a prosthetic valve. This procedure is indicated when the native mitral valve is severely damaged and cannot be repaired effectively. Prosthetic valves are broadly categorized as mechanical or biological and are selected based on the patient's specific needs and surgeon's assessment. The European Society of Cardiology recommends mechanical valves for patients younger than 65 years and biological valves for those older than 70 years. The American Heart Association suggests mechanical valves for patients under 50 years and biological valves for those over 70 years (39).

Choosing a Prosthetic Valve

The decision regarding prosthetic valve choice requires careful consideration of the benefits and risks associated with each option.

- **Mechanical valves:** Offer greater durability and lower lifetime risk of reoperation, making them a preferred choice for younger patients or those at high risk for reintervention. However, the lifelong anticoagulation required with these valves carries an increased risk of bleeding and stroke, which can impact quality of life.
- **Bioprosthetic valves:** Recommended for patients with difficulties managing long-term anticoagulation. These valves do not require lifelong anticoagulation, which can improve quality of life. However, they are prone to structural deterioration over time, potentially leading to the need for reoperation (40).

In clinical practice, patient age plays a key role in prosthetic valve selection. Older patients, who may have a lower tolerance for lifelong anticoagulation and a lower lifetime risk of reoperation, may benefit more from bioprosthetic valves. Ultimately, the final decision should be individualized based on patient characteristics, personal preferences, and consultation with a multidisciplinary team of specialists. This personalized approach ensures the patient receives the most appropriate treatment for their specific needs, maximizing quality of life and long-term prognosis.

5. Study design

5.1 Materials and Methods

From January 2004 to December 2022, 1,184 patients underwent mitral valve surgery at the Cardiac Surgery Department, IRCCS Azienda Ospedaliero-Universitaria di Bologna, Policlinico di Sant'Orsola.

The study population includes all patients who underwent surgery on the mitral valve, with the exception of those who received combined procedures involving the aortic valve, aortic root, and ascending aorta. The only additional procedures considered were electrode implantation and permanent pacemaker placement, atrial fibrillation surgery (radiofrequency, MAZE technique), patent foramen ovale closure, and tricuspid valve repair.

Regarding the surgical approach, the study population includes 812 (68.6%) patients who underwent surgery via median sternotomy (STER) and 372 (31.4%) patients who received a minimally invasive approach (MTH).

The follow-up of the patients alive and not reoperated was the longest available. Death or reoperation ended the follow-up for the other patients.

5.1.1 Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version 26.0 (IBM-SPSS Inc., Armonk, NY) and STATA/SE 18.1 (StataCorp 4905 Lakeway Dr College

Station TX 77845 USA). Categorical variables are presented as counts (percentages), while continuous variables are presented as mean ± standard deviation. Descriptive statistics was performed by using the Chi-square test to compare categorical variables and the Student's T-test for continuous variables, between MTH and STER groups.

The logistic regression expressed as ODDs ratio (OR) with a Confidence Intervals of 95% (CI 95%) was used to identify the risk factors for intrahospital mortality, mortality at follow-up and reoperation at follow-up.

Kaplan-Meyer curve was used to assess overall survival and freedom from reintervention at follow-up.

5.2 Results

5.2.1 Study Population

The study population was divided into two groups based on the surgical approach: median sternotomy (STER) and right anterolateral minithoracotomy (MTH). Sex distribution was similar in the two groups (57.9% in STER and 62.1% in MTH, p=0.171). Statistical analysis showed that the STER group had a higher incidence of cardiovascular risk factors, including systemic hypertension (p<0.001), diabetes mellitus (p<0.001), smoking (p=0.001), peripheral vascular disease (p<0.001), stroke (p=0.044), non-critical coronary artery disease (p=0.030), critical coronary artery disease (p<0.001), atrial fibrillation (p<0.001), moderate to severe pulmonary hypertension (p<0.001), and tricuspid regurgitation (p<0.001). Additional preoperative characteristics of the population are reported in Table 1.

Table 1. Preoperative Characteristics

Table 1	**Overall (1184)**	**STER (812)**	**MTH (372)**	***p***
Male sex	701 (59.2%)	470 (57.9%)	231 (62.1%)	*0.171*
Hypertension	637 (53.9%)	468 (57.7%)	169 (45.7%)	***<0.001***
Diabetes	69 (5.8%)	61 (7.5%)	8 (2.2%)	***<0.001***
Dislipidemia	456 (38.6%)	317 (39.1%)	139 (37.6%)	*0.619*
Smoking	405 (34.2%)	302 (37.2%)	103 (27.7%)	***0.001***
Dialysis	4 (0.3%)	2 (0.2%)	2 (0.5%)	*0.421*
Peripheral Arteriopathy	101 (8.6%)	87 (10.7%)	14 (3.8%)	***<0.001***
Stroke	37 (3.1%)	31 (3.8%)	6 (1.6%)	***0.044***
TIA	21 (1.8%)	15 (1.9%)	6 (1.6%)	*0.781*
Coronaropathy				
Non-critical	162 (13.7%)	123 (15.1%)	39 (10.5%)	***0.030***
Critical	66 (5.6%)	63 (7.8%)	3 (0.8%)	***<0.001***
Atrial Fibrillation	378 (31.9%)	325 (40.0%)	53 (14.2%)	***<0.001***
Reoperation	22 (1.9%)	14 (1.7%)	8 (2.2%)	*0.601*
Active IE	34 (2.9%)	28 (3.4%)	6 (1.6%)	*0.206*
Pulmonary Hypertension	534 (45.1%)	405 (49.9%)	129 (34.7%)	***<0.001***
Tricuspid Regurgitation	263 (22.2%)	222 (27.3%)	41 (11.0%)	***<0.001***

5.2.2 Intraoperative Characteristics

The Mini group had a lower incidence of urgent/emergency procedures (4.9% vs. 1.1%, p=0.001). Concerning the number of valve repairs and replacements, the MTH group exhibited a higher frequency of mitral valve repairs (p<0.001), while the STER group had more tricuspid valve repairs (p<0.001) and permanent pacemaker implantations (p=0.006). Operative times were longer in the MTH group, both for cardiopulmonary bypass time and aortic cross-clamp time. Further details on intraoperative variables are presented in Table 2.

Table 2. Intraoperative characteristics

Table 2	**Overall (1184)**	**STER (812)**	**MTH (372)**	*p*
Emergency	44 (3.7%)	40 (4.9%)	4 (1.1%)	***0.001***
MV Plastic	808 (68.2%)	473 (58.3%)	335 (90.1%)	***<0.001***
MV Replacement	376 (31.8%)	339 (41.7%)	37 (9.9%)	***<0.001***
TV Plastic	195 (16.5%)	181 (22.3%)	14 (3.8%)	***<0.001***
PMK Implant	16 (1.4%)	16 (2.0%)	0 (0)	***0.006***
CPB time	144.0 ± 49.9	124.4 ± 36.9	188.2 ± 46.9	***<0.001***
ACC time	101.6 ± 31.5	91.4 ± 26.0	124.7 ± 30.5	***<0.001***

5.2.3 In-Hospital Outcomes

Regarding in-hospital mortality, 22 patients (1.9%) died during hospitalization, with 19 deaths (2.3%) in the STER group and 3 (0.8%) in the MTH group (p=0.071). Among postoperative complications, significant differences were observed in the incidence of re-intubation, which was higher in the STER group, in the incidence of new-onset atrial fibrillation, which was higher in the MTH group, and in the incidence of permanent pacemaker implantation, which was higher in the STER group.

Hemodynamically, low cardiac output in the postoperative period was observed in 10.3% of patients in the STER group, and intra-aortic balloon pump (IABP) support was required in 30 patients (3.7%) in the STER group (p=0.005). The incidence of thoracic wound dehiscence was similar between the two groups (2.0% in the STER group and 3.5% in the MTH group, p=0.111).

A significant difference was also found in the length of intensive care unit (ICU) stay, which was longer for the STER group (3.1 ± 5.7 days for the STER group vs. 2.4 ± 2.7 days for the MTH group, p=0.002). However, the difference in length of hospital stay

was not statistically significant (11.6 ± 10.5 days for the STER group vs. 10.9 ± 7.7 days for the MTH group, p=0.198). Further information on postoperative outcomes is provided in Table 3.

Table 3. Postoperative Outcomes

Table 3	**Overall (1184)**	**STER (812)**	**MTH (372)**	*p*
Intrahospital death	22 (1.9%)	19 (2.3%)	3 (0.8%)	*0.071*
MV>24h	70 (6.0%)	55 (6.8%)	15 (4.1%)	*0.066*
Re-IOT	33 (2.8%)	29 (3.6%)	4 (1.1%)	***0.016***
Bleeding	49 (4.2%)	34 (4.2%)	15 (4.1%)	*0.916*
Stroke	9 (0.8%)	7 (0.9%)	2 (0.5%)	*0.551*
TIA	5 (0.4%)	4 (0.5%)	1 (0.3%)	*0.584*
Delirium	25 (2.1%)	14 (1.7%)	11 (3.0%)	*0.166*
AF	271 (23.0%)	167 (20.7%)	104 (28.2%)	***0.004***
PM implant	34 (2.9%)	34 (4.2%)	0 (0)	***<0.001***
MI	11 (0.9%)	9 (1.1%)	2 (0.5%)	*0.344*
Low CO	95 (8.1%)	83 (10.3%)	12 (3.3%)	***<0.001***
ECMO	8 (0.7%)	6 (0.7%)	2 (0.5%)	*0.700*
IABP	33 (2.8%)	30 (3.7%)	3 (0.8%)	***0.005***
Dialysis	31 (2.6%)	25 (3.1%)	6 (1.6%)	*0.146*
Wound dehicence	29 (2.5%)	16 (2.0%)	13 (3.5%)	*0.111*
Trasfusions (n. pts)	320 (27.3%)	212 (26.3%)	108 (29.5%)	*0.254*
RBC units (n.)	0.7 ± 2.5	0.8 ± 2.8	0.6 ± 1.6	*0.379*
ICU stay days	2.9 ± 5.0	3.1 ± 5.7	2.4 ± 2.7	***0.002***
Hospital stay days	11.3 ±9.7	11.6 ± 10.5	10.9 ± 7.7	*0.198*
MOF	21 (1.8%)	17 (2.1%)	4 (1.1%)	*0.226*
ACC	20 (1.7%)	19 (2.4%)	1 (0.3%)	***0.011***

5.2.4 Univariate and multivariate analysis

To evaluate the intrahospital mortality a univariate and multivariate analysis was performed including the clinical preoperative variables considered to impact the outcome (Table 4). At the univariate analysis age, female sex, diabetes, low eGFR, high NYHA class, LVEV, increased LVEDV, increases PAPs were linked with intrahospital mortality. These significant variables were included in the multivariate model which reported a higher risk for mortality linked to diabetes (OR 4.06 (1.12 - 14.85) p=0.033) and NYHA class (OR 3.84 (1.48 - 9.95) p=0.006).

Table 4. Univariate vs multivariate analysis for intrahospital mortality

Table 4	Univariate Analysis		Multivariate Analysis	
	OR (CI 95%)	*p*	OR (CI 95%)	*p*
MTH vs STER	0.33 (0.09 - 1.13)	*0.077*		
Age	1.12 (1.05 - 1.18)	*< 0.001*	1.08 (0.98 - 1.18)	*0.089*
Female vs Male	3.17 (1.28 - 7.85)	***0.012***	0.75 (0.18 - 3.11)	*0.69*
BMI	1.02 (0.97 - 1.06)	*0.45*		
HTN	1.86 (0.75 - 4.59)	*0.18*		
Smoke	1.09 (0.46 - 2.64)	*0.84*		
Diabetes	5.12 (1.83 - 14.3)	***0.002***	4.08 (1.12 - 14.85)	***0.033***
Dyslipidemia	1.33 (0.57-3.11)	*0.51*		
eGFR	0.95 (0.93-0.97)	***< 0.001***	0.97 (0.93 - 1.01)	*0.12*
Peripheral vasculop	1.7 (0.49 - 5.86)	*0.39*		
Stroke preop	1.44 (0.19 - 11.3)	*0.7*		
Subcritical Coronarop	1.87 (0.68 - 5.16)	*0.22*		
PCI preop	1.05 (0.14 - 7.99)	*0.96*		
NYHA class	4.91 (2.68 - 9.02)	***< 0.001***	3.84 (1.48 - 9.95)	***0.006***
Atrial fibrillation preop	2.18 (0.93 - 5.06)	*0.071*		
LVEF	1.01 (1.01 - 1.02)	***0.027***	1.01 (0.99 - 1.02)	*0.079*
LVEDV	0.98 (0.97 - 0.99)	***0.003***	0.99 (0.97 - 1.01)	*0.41*
PAPs	1.03 (1.001 - 1.05)	***0.045***	1.01 (0.97 - 1.03)	*0.98*

The risk factors for death at follow-up were then analyzed (Table 5). In the univariate analysis STER, Age, Female, high BMI, systemic hypertension, diabetes, low eGFR, peripheral vasculopathy, preoperative stroke, subcritical coronaropathy, preoperative PCI, high NYHA class, preoperative atrial fibrillation, low LVEF, LVEDV, increased PAPs were linked to a higher risk of death at follow-up. These variables were used to perform the multivariate analysis wich reported MTH, low age, low BMI, high eGFR, absence of peripheral vasculopathy as protective factors for death at follow-up.

Table 5. Univariate vs multivariate analysis for death at follow-up

Table 5	Univariate Analysis		Multivariate Analysis	
	OR (CI 95%)	*p*	OR (CI 95%)	*p*
MTH vs STER	1.18 (0.12 - 0.29)	***< 0.001***	0.51 (0.23 - 0.96	***0.036***
Age	1.1 (1.08 - 1.12)	***< 0.001***	1.06 (1.03 - 1.09)	***< 0.001***
Female vs Male	1.59 (1.19 - 2.14)	***0.002***	0.38 (0.64 - 1.69)	*0.88*
BMI	1.09 (1.05 - 1.12)	***< 0.001***	1.05 (1.01 - 1.11)	***0.04***
HTN	2.08 (1.52 - 2.85)	***< 0.001***	0.96 (0.6 - 1.51)	*0.85*
Smoke	1.02 (0.75 - 1.39)	*0.88*		
Diabetes	3.99 (2.39 - 6.66)	***< 0.001***	1.98 (0.99 - 3.93)	*0.051*
Dyslipidemia	0.85 (0.63 - 1.16)	*0.31*		
eGFR	0.96 (0.95 - 0.97)	***< 0.001***	0.98 (0.97 - 0.99)	***0.004***
Peripheral vasculopathy	4.75 (3.09 - 7.3)	***< 0.001***	2.73 (1.52 - 4.92)	***0.001***
Stroke preop	2.46 (1.23 - 4.94)	***0.011***	1.39 (0.52 - 3.77)	*0.51*
Subcritical Coronaropathy	2.63 (1.82 - 3.81)	***< 0.001***	1.16 (0.68 - 1.98)	*0.58*
PCI preop	3.14 (1.76 - 5.61)	***< 0.001***	1.21 (0.54 - 2.71)	*0.64*
NYHA class	1.88 (1.54 - 2.29)	***< 0.001***	1.25 (0.93 - 1.66)	*0.13*
Atrial fibrillation preop	2.82 (2.09 - 3.81)	***< 0.001***	1.34 (0.88 - 2.06)	*0.18*
LVEF	0.97 (0.95 - 0.99)	***0.002***	0.99 (0.97 - 1.02)	*0.86*
LVEDV	0.99 (0.98 - 0.99)	***< 0.001***	1.01 (0.99 - 1.01)	*0.4*
PAPs	1.02 (1.01 - 1.02)	***< 0.001***	1.01 (0.99 - 1.03)	*0.057*

The univariate analysis for the risk of reoperation at follow-up included the variables with a clinical significance in term of reintervention (Table 6). At the univariate the MTH access was the only protective variable for freedom from reoperation at follow-up with a statistical significance. For this reason the multivariate analysis was not needed.

Table 6. Univariate for freedom from reoperation at follow-up

Table 6	Univariate Analysis	
	OR (CI 95%)	*p*
MTH vs STER	0.48 (0.25 - 0.94)	***0.003***
Age	1.01 (0.98 - 1.02)	*0.92*
Female vs Male	1.10 (0.65 - 1.88)	*0.71*
BMI	1.01 (1.98 - 1.05)	*0.34*
HTN	1.42 (0.83 - 2.45)	*0.21*
Smoke	1.28 (0.75 - 2.19)	*0.37*
Diabetes	0.88 (0.27 - 2.87)	*0.83*
Dyslipidemia	1.13 (0.66 - 1.93)	*0.66*
eGFR	0.99 (0.98 - 1.01)	*0.15*
Peripheral vasculopathy	1.01 (0.39 - 2.58)	*0.98*
Stroke preop	1.28 (0.75 - 2.19)	*0.37*
Subcritical Coronaropathy	0.45 (0.16 - 1.27)	*0.13*
PCI preop	1.22 (0.37 - 4.05)	*0.74*
NYHA class	1.06 (0.75 - 0.49)	*0.74*
Atrial fibrillation preop	1.43 (0.83 - 2.45)	*0.19*
LVEF	0.99 (0.97 - 1.02)	*0.78*
LVEDV	1.01 (0.99 - 1.01)	*0.62*
PAPs	1.01 (0.99 - 1.027)	*0.27*

5.2.5 Long-term Outcomes

As regards long-term outcomes, the mean follow up time was 76.8 ± 61.0 months for the entire study population, with a mean follow up time of 91.0 ± 61.4 months in the STER group and 46.5 ± 47.8 months in the MTH group ($p<0.001$). Death at follow-up were

globally 221 (18.7%) with a major incidence in STER group over MTH (200 (25.6%) STER vs 21 (5.8%) MTH p<0.001). The number of patients who underwent a reoperation during follow-up was higher for STER than MTH group (47 (5.85%) vs 11 (2.89%), respectively. p=0.028).

The overall survival Kaplan Meyer curve is showed in Figure 1.

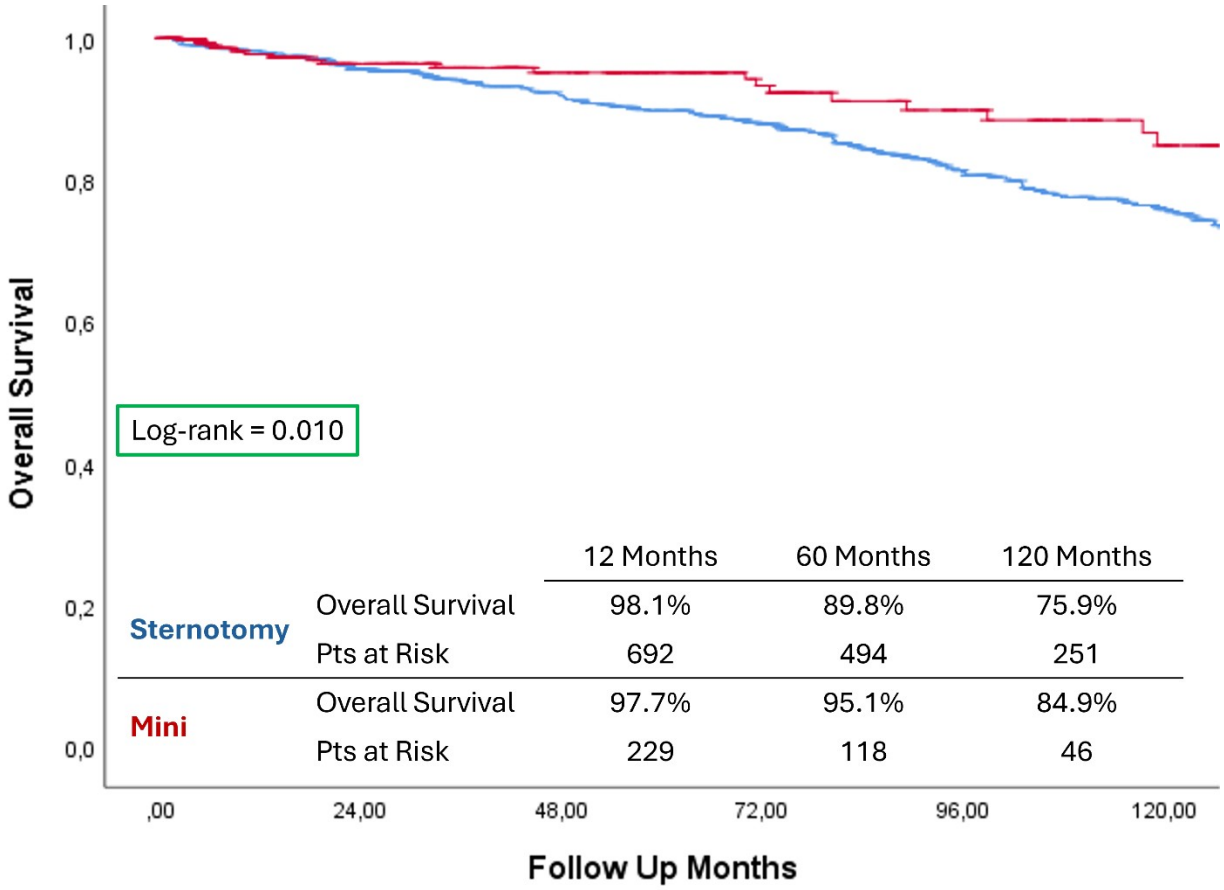

Fig.1 Kaplan Meyer curve for Overall Survival between STER and MTH groups.

5.3 Discussion

Minimally invasive mitral valve surgery with mini-thoracotomy approach (MTH) was introduced 25 years ago (41). Previous meta-analyses (42-45) failed to detect any positive impact of MTH on the occurrence of postoperative major adverse cardiac events

compared with classic sternotomy approach (STER). Nowadays, MTH is becoming a new standard for mitral valve surgery and the surgical community is far from the learning curve with this minimally invasive technique; the proponents argue its utility for treating even the most complex mitral valve disease without any additional risk of potential complications despite prolonged operation times.

However, it is still being determined if MTH benefits truly outweigh any drawbacks. Older studies, which often included data from surgeons still learning the MTH technique, mainly showed differences in procedure time and resource use between MTH and STER (42-45). These factors are often used to debate the merits of each approach. (46)

From an economic point of view, studies coming from Centers with important MTH experience, instead, reported an overall mean total cost of both procedures (47-51) as significantly lower after MTH compared to STER thanks also to less blood use and reduction of intensive care unit stay time. Furthermore, the shift towards earlier surgical intervention in mitral valve disease, particularly in cases of mitral regurgitation (52-53), has contributed to the widespread adoption of MTH. This trend favors a patient population characterized by low surgical risk, often presenting with minimal or no symptoms, who are increasingly inclined towards minimally invasive surgical options. Moreover, it has been also observed that MTH was more likely than the conventional approach in teaching hospitals and hospitals with more than 600 beds (47). One might hypothesize that larger hospitals have funds and personnel enough to support MTH. Teaching hospitals, being the ones where specialized personnel is trained and cutting-edge surgical science is ubiquitous, are expected to go for more innovative techniques and push the bounds of new technologies in MTH.

In our University Center MTH started to be performed 20 years ago with few cases. Numbers started to grow after 2010 with a range of 15 to 40 cases per year, and the cases were initially performed by one surgeon only. On one side this guaranteed one learning curve, on the other side, considering this Center always focused on aortic surgery, the MTH program slowed to ramp up. Since 2019 two other surgeons started to be trained for MTH but after three years the senior surgeon went to work for another Hospital and this contributed to another slowering of learning curve.

In our analysis, in fact, the intrahospital outcomes of MTH patients were similar to the STER patients where conventional surgical access has been taught through the years. Notably, the MTH patients underwent a more strict preoperative selection either for comorbidities either for repairability of the valve. This could explain the better outcomes in the follow-up for survival and freedom from reoperation. The ICU stay time was lower for MTH patients even if the total hospital stay was similar. This observation could be explained by the everyday clinical practice of transferring patients to other healthcare facilities rather than discharging them.

Improving the management of operative and perioperative MTH patients, as well as increasing the number of cases, will smooth out the differences between the two groups and will let the advantages of MTH emerge. Our experience is growing and this will have a positive impact on offsetting the costs and resources during the surgical hospitalization period. Moreover, MTH patients report a faster recovery after dismission, an earlier return to work, less pain, and a more positive psychological experience of surgery. However, the greatest benefits could be achieved only by implementing a fast-track extubation protocol to reduce intensive care unit stay time and allow patients to

begin rehabilitation earlier. Patients selection remains the most important factor to consider when screening patients for STER or MTH and the pre-admission clinic is fundamental for performing the necessary exams.

5.4 Limitations

This is a single-center retrospective study. The percentage of patients undergoing MTH instead of STER remains low. This reflects the challenges in selecting the surgical approach, particularly due to economic and resource constraints. Performing angio-CT scans on all patients who could potentially benefit from MTH proves difficult, despite the introduction of a pre-operative cardiac assessment clinic in recent years. Furthermore, the two populations under consideration represent two distinct eras. The introduction of the minimally invasive approach occurred gradually, with a consequent learning curve. This undoubtedly contributes to selection heterogeneity, which should diminish in the future due to improved surgical and anesthesiological expertise in MTH.

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