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**Review Article**

# Barlow's Disease: Navigating Long-Term Outcomes and Modern Management Strategies

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## ABSTRACT

Barlow's disease is a primary form of mitral valve prolapse (MVP) characterized by myxomatous degeneration, leading to excessive leaflet thickening, redundancy, and elongation. This structural abnormality can result in progressive mitral regurgitation, predisposing patients to complications such as atrial fibrillation, heart failure, and infective endocarditis. While some individuals remain asymptomatic, others experience significant morbidity, often necessitating medical or surgical intervention. This review aims to (1) understand the pathophysiology of Barlow's disease, (2) evaluate the long-term outcomes in affected patients, and (3) analyze modern management strategies including advanced imaging, risk stratification tools, and minimally invasive mitral valve repair techniques. Recent advancements in diagnostic modalities and surgical approaches have contributed to better patient selection, reduced operative risks, and improved survival and quality of life. By synthesizing current evidence, this article provides a comprehensive overview to guide clinicians in optimizing long-term care and outcomes in patients with Barlow's disease.

## INTRODUCTION

Barlow's disease, a distinct and severe form of myxomatous mitral valve degeneration, is characterized by excessive thickening, elongation, and redundancy of the mitral valve leaflets, often accompanied by annular dilation and prolapse. These morphological abnormalities can lead to significant mitral regurgitation (MR), contributing to progressive left ventricular (LV) dilation, atrial fibrillation, heart failure, and increased cardiovascular morbidity and mortality. First described by John Brereton Barlow, this condition represents a unique and complex subtype of mitral valve prolapse (MVP) with distinct pathological, clinical, and surgical implications. While many patients with Barlow's disease remain asymptomatic in early stages, its natural progression can result in severe valvular

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dysfunction, necessitating timely intervention. The surgical management of Barlow's disease poses unique challenges due to the extensive leaflet involvement, annular disjunction, and multi-scallop prolapse typically seen in these patients. Repair durability and long-term outcomes heavily depend on surgical expertise, patient selection, and appropriate timing of intervention[1]. This review seeks to evaluate the long-term clinical outcomes of Barlow's disease and to explore evolving management strategies, including advanced imaging techniques, minimally invasive mitral valve repair, and transcatheter approaches. Emphasis is placed on risk stratification, optimal timing for intervention, and individualized treatment paradigms to enhance both prognosis and quality of life.

## Epidemiology and Prevalence of Barlow's Disease

Barlow's disease constitutes a subset of mitral valve prolapse (MVP), which affects approximately 2–3% of the general population. Among MVP cases requiring surgical correction, Barlow's disease accounts for a significant proportion, especially among those with severe and symptomatic mitral regurgitation.

### Prevalence Highlights:

- Barlow's disease represents 50–60% of MVP cases undergoing surgical repair.
- More commonly diagnosed in middle-aged and older adults, though it may present in younger individuals.
- Males tend to exhibit more severe mitral regurgitation and are more likely to require surgical intervention compared to females.

### Epidemiological Insights

The prevalence and clinical manifestation of Barlow's disease demonstrate significant variability across different populations, influenced by genetic, environmental, and demographic factors. While mitral valve prolapse (MVP) is estimated to affect 2–3% of the general population, Barlow's disease accounts for a more severe, though less common, subset requiring surgical intervention in 50–60% of MVP-related surgeries.

**Geographic and Ethnic Variation:** The reported prevalence of Barlow's disease may vary based on geographic location and ethnic background, potentially due to underlying genetic predispositions and environmental factors. However, comprehensive population-based studies are limited, and further research is needed to delineate these differences with greater clarity.

**Genetic Predisposition:** There is increasing evidence to suggest a hereditary component in Barlow's disease. Familial clustering of MVP has been documented, and certain cases of Barlow's disease have shown autosomal dominant inheritance patterns. Genetic investigations have identified mutations in genes associated with connective tissue integrity and extracellular matrix regulation, although definitive markers for Barlow's disease remain under investigation.

**Association with Connective Tissue Disorders:** Barlow's disease has been associated with systemic connective tissue disorders, most notably Marfan syndrome and Ehlers-Danlos syndrome. These conditions share common histopathological features, including myxomatous degeneration of the mitral valve, which may contribute to the early onset and aggressive progression of valvular pathology.

**Silent Progression and Clinical Detection:** A significant number of patients with Barlow's disease remain asymptomatic during the early

stages. Diagnosis is often delayed until complications such as moderate to severe mitral regurgitation, atrial fibrillation, or left ventricular dysfunction manifest clinically. This silent progression underscores the need for vigilant screening and regular follow-up, particularly in high-risk populations or those with a family history of MVP. Given the potential for serious complications, early identification through advanced imaging techniques, risk stratification protocols, and serial monitoring is vital to ensure timely and effective intervention in patients with Barlow's disease.

**Pathophysiology and Etiology of Barlow's Disease**

Barlow's disease is a primary form of **mitral valve prolapse (MVP)** characterized by **myxomatous degeneration** of the mitral valve, leading to leaflet thickening, elongation, and redundancy. These structural abnormalities contribute to **mitral regurgitation (MR)** and potential cardiovascular complications{.3}

**1. Myxomatous Degeneration of the Mitral Valv**

Myxomatous degeneration is the hallmark of Barlow's disease, involving:

- **Excessive extracellular matrix remodeling:** Increased deposition of glycosaminoglycans (especially dermatan sulfate) leads to thickening and ballooning of the leaflets.
- **Collagen and elastin disruption:** A reduction in Type I and Type III collagen weakens the valve structure, making it more prone to prolapse.
- **Chordal elongation and rupture:** The supporting chordae tendineae become elongated and fragile, increasing the risk of leaflet prolapse and mitral regurgitation.

This progressive degeneration results in **billowing leaflets, excessive motion, and a characteristic thickened appearance** seen on echocardiography.

**2. Genetic and Environmental Factors**

**Genetic Factors:**

- Barlow's disease has a known familial association, suggesting a genetic predisposition.
- Mutations in genes encoding for extracellular matrix proteins (e.g., fibrillin-1, collagen, and elastin) have been implicated.
- Connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome, are linked to an increased risk of myxomatous degeneration.

**Environmental and Systemic Influences:**

- **Oxidative stress and inflammation** may accelerate degeneration of valve tissue.
- **Hypertension and mechanical stress** on the valve contribute to worsening mitral regurgitation.
- **Aging-related changes** increase the severity of myxomatous degeneration over time.

**3. Structural and Functional Changes in the Mitral Valve**

**Structural Abnormalities:**

- **Thickened, redundant mitral leaflets** (>5 mm in echocardiography).

- **Marked annular dilatation** and displacement of leaflet coaptation.
- **Excessive systolic motion**, leading to leaflet prolapse into the left atrium.
- **Elongated or ruptured chordae tendineae**, increasing MR severity.

**Functional Consequences:**

- **Mitral regurgitation (MR):** As the valve fails to close properly, blood leaks backward into the left atrium, increasing cardiac workload.
- **Atrial and ventricular remodelling:** Chronic MR leads to left atrial enlargement and left ventricular dilatation.
- **Increased risk of arrhythmias:** Atrial fibrillation and ventricular arrhythmias become more common due to structural changes.

## 4. Clinical Presentation of Barlow's Disease

Barlow's disease can present with a wide spectrum of symptoms, ranging from asymptomatic cases to significant cardiovascular complications due to mitral regurgitation (MR). The clinical manifestations depend on the severity of valve involvement and the extent of MR progression.[2] The clinical presentation of Barlow's disease is highly variable, ranging from asymptomatic cases detected incidentally to symptomatic patients presenting with complications such as significant mitral regurgitation (MR), arrhythmias, or heart failure. This heterogeneity is attributed to the progressive nature of the disease and the degree of mitral valve involvement. The clinical spectrum of Barlow's disease is broad and evolves over time. A high index of suspicion, especially in patients with a family history of MVP or unexplained cardiac symptoms, is essential for early detection. Comprehensive clinical evaluation, supported by echocardiographic and electrocardiographic monitoring, plays a pivotal role in identifying disease progression and guiding timely intervention.

### 1. Asymptomatic Phase:

Many individuals with Barlow's disease remain asymptomatic for years. Diagnosis during this phase often occurs incidentally during routine physical examination or echocardiographic evaluation. A characteristic mid-systolic click followed by a late systolic murmur may be auscultated in some patients, suggestive of mitral valve prolapse.

### 2. Symptomatic Phase:

As the disease progresses and mitral regurgitation becomes hemodynamically significant, patients may begin to experience symptoms including:

1. **Exertional dyspnea**
2. **Fatigue and reduced exercise tolerance**
3. **Palpitations**
4. **Atypical chest pain**
5. **Orthopnea and paroxysmal nocturnal dyspnea** in more advanced stages

### 3. Arrhythmias:

Barlow's disease is associated with a heightened risk of arrhythmias, particularly **atrial fibrillation**, due to left atrial enlargement. Ventricular arrhythmias, including non-sustained or sustained ventricular tachycardia, may also occur, increasing the risk of **sudden cardiac death** in select patients.

### 4. Heart Failure:

In cases with severe, chronic mitral regurgitation, progressive left ventricular volume overload can lead to **left ventricular dilation and dysfunction**, eventually resulting in **congestive heart failure** if left untreated.

**5. Infective Endocarditis:**

Though less common, the redundant and thickened mitral valve leaflets in Barlow's disease may serve as a nidus for **infective endocarditis**, particularly in individuals with significant regurgitation or prior valvular interventions.

**6. Psychological Symptoms:**

Some patients may report **anxiety, panic attacks, or dizziness**, often attributed to autonomic dysfunction or the psychological impact of chronic cardiac symptoms.

**A. Symptoms**

**1. Common Symptoms:**

- **Palpitations:** Often due to **ventricular and supraventricular arrhythmias**, including premature ventricular contractions (PVCs) and atrial fibrillation.
- **Dyspnea (Shortness of Breath):** Develops as **mitral regurgitation progresses**, leading to increased left atrial pressure and pulmonary congestion.
- **Atypical Chest Pain:** Often **non-anginal** and unrelated to exertion, possibly due to myocardial stretch or autonomic dysfunction.
- **Fatigue:** Due to reduced cardiac output, particularly in cases with severe MR.
- **Dizziness or Syncope:** May result from **arrhythmias or autonomic dysfunction**.

**2. Symptoms in Advanced Stages:**

- **Heart failure symptoms:** Orthopnea, paroxysmal nocturnal dyspnea (PND), peripheral edema in severe cases.
- **Stroke or Transient Ischemic Attacks (TIAs):** Due to embolization from **atrial fibrillation or mitral valve thickening**.
- **Symptoms of Infective Endocarditis (IE):** Fever, weight loss, new murmur in cases of valve infection.

**B. Murmurs and Auscultatory Findings**

**1. Key Auscultatory Features of Barlow's Disease:**

- **Mid-to-late systolic click:** Classic hallmark due to abrupt tension in the **elongated mitral valve leaflets or chordae tendineae**.
- **Late systolic murmur:** Due to **mitral regurgitation**, best heard at the **apex** and radiating to the **axilla or back**.
- **Dynamic Changes:**
  - **Louder with standing or Valsalva maneuver:** Decreased left ventricular volume enhances leaflet prolapse.
  - **Softer with squatting:** Increased venous return reduces prolapse severity.

**2. Severe Mitral Regurgitation Findings:**

- **Holosystolic murmur** if MR becomes more severe and persistent.
- **S3 heart sound** in cases with left ventricular dysfunction.

- **Signs of Pulmonary Hypertension:** Loud **P2 component** of the second heart sound.

**C. Differential Diagnosis**

Since Barlow's disease shares features with several other cardiac conditions, it is important to distinguish it from:

| Condition | Key Differentiating Features |
|---|---|
| **Classic Mitral Valve Prolapse (Non-Barlow's MVP)** | Less leaflet thickening, no excessive redundancy, minimal MR. |
| **Hypertrophic Cardiomyopathy (HCM)** | Harsh systolic murmur, louder with Valsalva, asymmetrical septal hypertrophy. |
| **Aortic Stenosis** | Systolic ejection murmur at the right upper sternal border, radiates to carotids. |
| **Infective Endocarditis** | New-onset murmur, fever, embolic events, vegetations on echocardiography. |
| **Dilated Cardiomyopathy (DCM)** | S3 gallop, biventricular dysfunction, secondary MR. |
| **Coronary Artery Disease (CAD)** | Exertional angina, ST-T changes on ECG, ischemic symptoms. |

## 6. Long-Term Outcomes of Barlow's Disease

Barlow's disease follows a **variable but progressive course**, primarily driven by **mitral regurgitation (MR)** and its associated complications. While some patients remain asymptomatic for years, others develop significant **cardiac remodeling, arrhythmias, and heart failure** over time.

**A. Natural History of Barlow's Disease**

- **Early Stages (Asymptomatic Phase):**
  - Many patients have **mild or no mitral regurgitation** and remain asymptomatic for years.
  - Some experience **palpitations or atypical chest pain**, often due to **autonomic dysfunction** or **ventricular arrhythmias**.
- **Progressive Phase:**
  - With time, **myxomatous degeneration worsens**, leading to **progressive MR** and **left atrial (LA) and left ventricular (LV) remodeling**.
  - Increasing **ventricular volume overload** can result in **atrial fibrillation (AF), heart failure (HF), and pulmonary hypertension (PH)**.
- **Late/Advanced Phase:**
  - **Severe MR** leads to symptomatic heart failure, requiring **surgical intervention (mitral valve repair/replacement)**.
  - **Complications such as stroke, endocarditis, or irreversible LV dysfunction** can occur in untreated cases.

**B. Risk of Mitral Regurgitation (MR) Progression**

**Factors Contributing to MR Severity**

The progression of mitral regurgitation in Barlow's disease is influenced by multiple structural and functional changes. Progressive elongation or rupture of the chordae tendineae further compromises leaflet support, leading to worsening regurgitation. Additionally, mitral annular dilatation impairs effective leaflet coaptation, intensifying MR severity. Enlargement

and dysfunction of the left ventricle contribute to adverse remodeling, while the development of atrial fibrillation increases left atrial pressure and volume overload, compounding the regurgitation.

**Clinical Impact of Severe MR**

Severe MR leads to significant hemodynamic consequences. Chronic volume overload causes left atrial enlargement, which in turn increases the risk of atrial fibrillation. Persistent regurgitation also impairs left ventricular function, eventually leading to systolic dysfunction and heart failure. If left untreated, severe MR can further progress to pulmonary hypertension, significantly affecting long-term morbidity and mortality.

**Predictors of Rapid MR Progression**

Several echocardiographic and clinical parameters have been identified as predictors of rapid MR progression. An ejection fraction (EF) less than 60% may indicate early ventricular dysfunction, even in asymptomatic patients. A left atrial diameter greater than 40 mm suggests chronic pressure overload and atrial remodeling. Additionally, a regurgitant volume exceeding 60 mL is a hallmark of severe MR and is associated with adverse outcomes, warranting timely surgical intervention.

**C. Complications of Barlow's Disease**

1. **Atrial Fibrillation and Arrhythmias:** One of the most common complications associated with Barlow's disease is atrial fibrillation (AF), which results from left atrial (LA) enlargement and stretch due to chronic mitral regurgitation (MR). The structural and electrical remodeling of the atrium predisposes to AF and atrial flutter. These arrhythmias significantly increase the risk of thromboembolic events, such as stroke and systemic embolism. In patients with a $CHA_2DS_2$-VASc score of 2 or more, long-term anticoagulation is generally recommended.

2. **Heart Failure:** mProgressive MR leads to chronic volume overload and eventual left ventricular (LV) dysfunction, culminating in heart failure (HF). Patients typically present with clinical features such as dyspnea on exertion, fatigue, orthopnea, pulmonary congestion, and peripheral edema. Early surgical intervention is crucial, particularly when LV ejection fraction falls below 60% or there is progressive LV dilatation, to prevent irreversible myocardial damage.

3. **Infective Endocarditis:** The myxomatous degeneration of the mitral valve in Barlow's disease creates a substrate for bacterial colonization, increasing the risk of infective endocarditis (IE). This risk is especially pronounced in patients with severe MR or thickened valve leaflets. Clinically, IE may manifest as fever, embolic events, and new or changing cardiac murmurs. While routine antibiotic prophylaxis is not advised for most patients, it may be indicated in those with a history of endocarditis or prosthetic heart valves.

4. **Stroke and Systemic Embolism:** Patients with Barlow's disease are at elevated risk for stroke and systemic embolism, particularly in the presence of AF or spontaneous echo contrast within a dilated LA. Valve thickening and calcification may also promote embolic events independently. In high-risk patients, including those with left atrial thrombus or persistent AF, anticoagulation therapy is essential to mitigate the risk.

**D. Prognostic Indicators**

The prognosis in Barlow's disease with mitral regurgitation (MR) is influenced by several clinical and echocardiographic factors. Severe MR significantly raises the risk of developing left ventricular (LV) dysfunction and heart failure. A left ventricular ejection fraction (LVEF) below 60% is a marker of early ventricular decompensation. An enlarged left atrium (diameter > 40 mm) is associated with a higher likelihood of atrial fibrillation (AF) and stroke. The presence of pulmonary hypertension further worsens outcomes and increases surgical risk. Additionally, arrhythmias such as AF, ventricular tachycardia (VT), and premature ventricular contractions (PVCs) are linked to poorer survival. Age and comorbidities like hypertension and coronary artery disease (CAD) also negatively impact overall prognosis.

## 7. Modern Management Strategies for Barlow's Disease

Management of Barlow's disease focuses on symptom control, prevention of complications, and timely intervention to prevent irreversible cardiac dysfunction. The choice of therapy depends on the severity of mitral regurgitation (MR), presence of symptoms, and risk of complications such as atrial fibrillation (AF) and heart failure.[6] Barlow's disease, a form of mitral valve prolapse (MVP) marked by excessive leaflet tissue, myxomatous degeneration, and multisegmental prolapse, requires a tailored and multidisciplinary management approach. The therapeutic strategy hinges on the severity of mitral regurgitation (MR), symptomatology, risk of complications, and patient-specific surgical risk.

### 1. Medical Management

Although medical therapy does not alter the natural progression of valvular degeneration in Barlow's disease, it plays a vital role in symptom control and management of associated conditions:

- Beta-blockers: Used to reduce adrenergically mediated symptoms such as palpitations, anxiety, and arrhythmias.
- Antiarrhythmics: Considered in patients with significant ventricular ectopy or atrial fibrillation (AF).
- Anticoagulation: Indicated in patients with AF, left atrial thrombus, or embolic events to prevent stroke.
- Diuretics: Help alleviate dyspnea or peripheral edema in patients with volume overload or early heart failure.
- ACE inhibitors/ARBs: Occasionally used in patients with afterload-dependent MR or hypertension, although data is limited in MVP-specific settings.

**2. Surgical Management:** Surgical intervention remains the cornerstone of long-term management in patients with severe mitral regurgitation (MR) due to Barlow's disease. Mitral valve repair is the preferred approach, offering superior outcomes in terms of ventricular function preservation, reduced mortality, and lower risk of thromboembolic events compared to valve replacement. Common techniques include quadrangular resection, artificial chordae implantation using materials such as Gore-Tex, annuloplasty, and leaflet remodeling. Evidence supports early surgical repair—even in asymptomatic patients with preserved ejection fraction and significant MR—as it improves long-term outcomes and minimizes postoperative complications. Mitral valve replacement (MVR) is reserved for cases where repair is not feasible, such as those involving severe leaflet calcification or destruction. While

mechanical valves necessitate lifelong anticoagulation, bioprosthetic valves, though free from this requirement, have limited durability, especially in younger patients.

**3. Transcatheter and Minimally Invasive Therapies:** Advances in cardiac technology have introduced less invasive therapeutic options for patients with Barlow's disease who are at high surgical risk. The Mitra Clip device, which replicates the surgical Alfieri stitch by approximating the anterior and posterior mitral leaflets, is a widely adopted edge-to-edge repair technique. Although it reduces mitral regurgitation effectively in select high-risk patients, its use in Barlow's disease remains limited due to the complex and redundant leaflet anatomy, making it less suitable as a substitute for complete surgical repair. Transcatheter mitral valve replacement (TMVR) is another emerging therapy, currently under investigation for primary MR. While promising, its application in Barlow's disease is restricted by anatomical challenges such as large mitral annuli and excessive leaflet tissue. Additionally, robotic and minimally invasive mitral valve surgery is gaining popularity in specialized centers, providing outcomes comparable to open-heart surgery with the added benefits of reduced postoperative pain, quicker recovery, and improved cosmetic results.

**4. Regular Echocardiographic Monitoring**

Timely detection of progression is critical. Patients with Barlow's disease need periodic transthoracic echocardiograms to monitor:

- Mitral valve morphology and MR severity
- Left ventricular (LV) size and ejection fraction
- Left atrial size and pulmonary artery pressures
- Chordal integrity and prolapse extent

Three-dimensional echocardiography and transesophageal echocardiography offer improved spatial resolution and are often employed preoperatively.

**5. Electrophysiological and Arrhythmia Management**

**Barlow's disease is associated with a higher risk of arrhythmias, particularly:**

- Supraventricular arrhythmias like AF
- Ventricular arrhythmias, especially in the presence of mitral annular disjunction (MAD)

Holter monitoring, cardiac MRI, and electrophysiological studies may be warranted in patients with palpitations or syncope. Management includes antiarrhythmic drugs, catheter ablation, and in some cases, implantable cardioverter-defibrillator (ICD) for secondary prevention.

**6. Multidisciplinary and Patient-Centric Care**

- Coordination among cardiologists, cardiac surgeons, imaging specialists, and electrophysiologists ensures optimal care.
- Patient education is crucial for recognizing early symptoms (palpitations, exertional dyspnea), adherence to medications, and regular follow-up.
- Lifestyle advice includes avoidance of stimulants (caffeine, alcohol), maintaining a healthy weight, and exercise within tolerated limits.

**Recent Clinical and Surgical Advances**

***Totally Endoscopic Mitral Valve Repair***

A 2024 single-center study evaluated 21 patients with Barlow's disease who underwent totally

endoscopic mitral valve repair using leaflet folding, multiple neochordae, and annuloplasty. Over a median follow-up of 24 months, there were no operative deaths or strokes, and durable valve function was maintained without significant MR or systolic anterior motion[8]

***Long-Term Durability of Minimally Invasive Non-Resectional Repair***

In a cohort of 98 patients treated by minimally invasive, non-resectional techniques (neochordae ± annuloplasty), the procedure proved safe with no conversions or in-hospital complications. Over an average follow-up of 4.1 ± 3.1 years, survival at 7 years was 87%, with 93% freedom from stroke and myocardial infarction, and only 4.1% experienced moderate or worse MR recurrence[9]

***Robotic Repair Outcomes***

A larger registry including 924 patients (111 with Barlow's disease) undergoing robotic mitral repair showed excellent results: conversion to replacement was under 1%, and freedom from moderate MR at median 5.5-year follow-up remained high. Barlow's patients tended to be younger but achieved comparable outcomes to non-Barlow patients[10]

***Long-Term Outcomes of Conventional Repair***

Data from over 180 patients treated over 16 years via conventional approaches demonstrated 10-year freedom from MR-related reintervention at ~79.8%, with a low recurrence rate of >2+ MR. Recurrent MR was predominantly linked to technical repair factors rather than intrinsic disease progression, underscoring the importance of meticulous surgical technique [11].

***Annuloplasty Ring Type Does Not Alter Long-Term Outcomes***

An analysis of 296 Barlow's patients compared outcomes following edge-to-edge repair using either complete rings or posterior flexible bands. At 14 years, survival rates and recurrence rates of MR were virtually identical between groups, suggesting that optimal immediate results, rather than ring selection, determine long-term durability[12].

**Implications for Clinical Practice**

1. Minimally invasive and robotic techniques—including totally endoscopic, non-resectional repair—are increasingly feasible and durable in Barlow's disease, offering excellent mid-term outcomes.

2. Early surgical intervention in specialized centers delivers lasting repair success and low late recurrence, reinforcing the value of timely referral before onset of LV dysfunction.

3. Technical precision in repair, especially chordal reconstruction and annuloplasty, remains critical to long-term valve durability—more so than the specific annuloplasty device chosen.

These findings support a shift toward earlier repair using advanced minimally invasive approaches in experienced centers and reinforce that long-term durability hinges on surgical expertise rather than valve design.

## CONCLUSION

Barlow's disease, characterized by myxomatous degeneration of the mitral valve, is a common cause of degenerative mitral regurgitation (MR). While some patients remain asymptomatic, others develop complications such as atrial fibrillation, heart failure, and stroke. Clinically, it presents with palpitations, dyspnea, chest discomfort, and mid-systolic clicks or murmurs. Disease

progression is driven by mitral annular dilation, chordal elongation or rupture, and left ventricular volume overload, leading to atrial fibrillation and ventricular dysfunction. Medical therapy—including beta-blockers, anticoagulants, and diuretics—offers symptom control but does not halt progression. Surgical mitral valve repair is the treatment of choice in severe MR, providing better outcomes than valve replacement. For high-risk surgical candidates, newer transcatheter approaches like MitraClip and TMVR show promising results.

**Implications for Patient Care:**

- **Early risk stratification** is essential to identify patients at risk of deterioration before the onset of irreversible cardiac remodeling.
- **Routine echocardiographic surveillance** facilitates timely surgical referral and helps prevent complications such as pulmonary hypertension and LV dysfunction.
- **Multidisciplinary management**, involving cardiologists, cardiac surgeons, and electrophysiologists, is vital to develop individualized care plans and optimize clinical outcomes.
- **Patient education** on symptom awareness, lifestyle modifications, and adherence to follow-up is crucial for improving long-term prognosis and quality of life.

In conclusion, a proactive, multidisciplinary, and patient-centered approach is fundamental to navigating the long-term outcomes and optimizing the management of Barlow's disease in contemporary clinical practice.

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