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15th MID TERM UPCSI

INTERVENTION CONFERENCE

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The Centrum Hotel, Lucknow

NEWSLETTER

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From the Desk of the Editor

Dr. Roopali Khanna

Dear Readers,

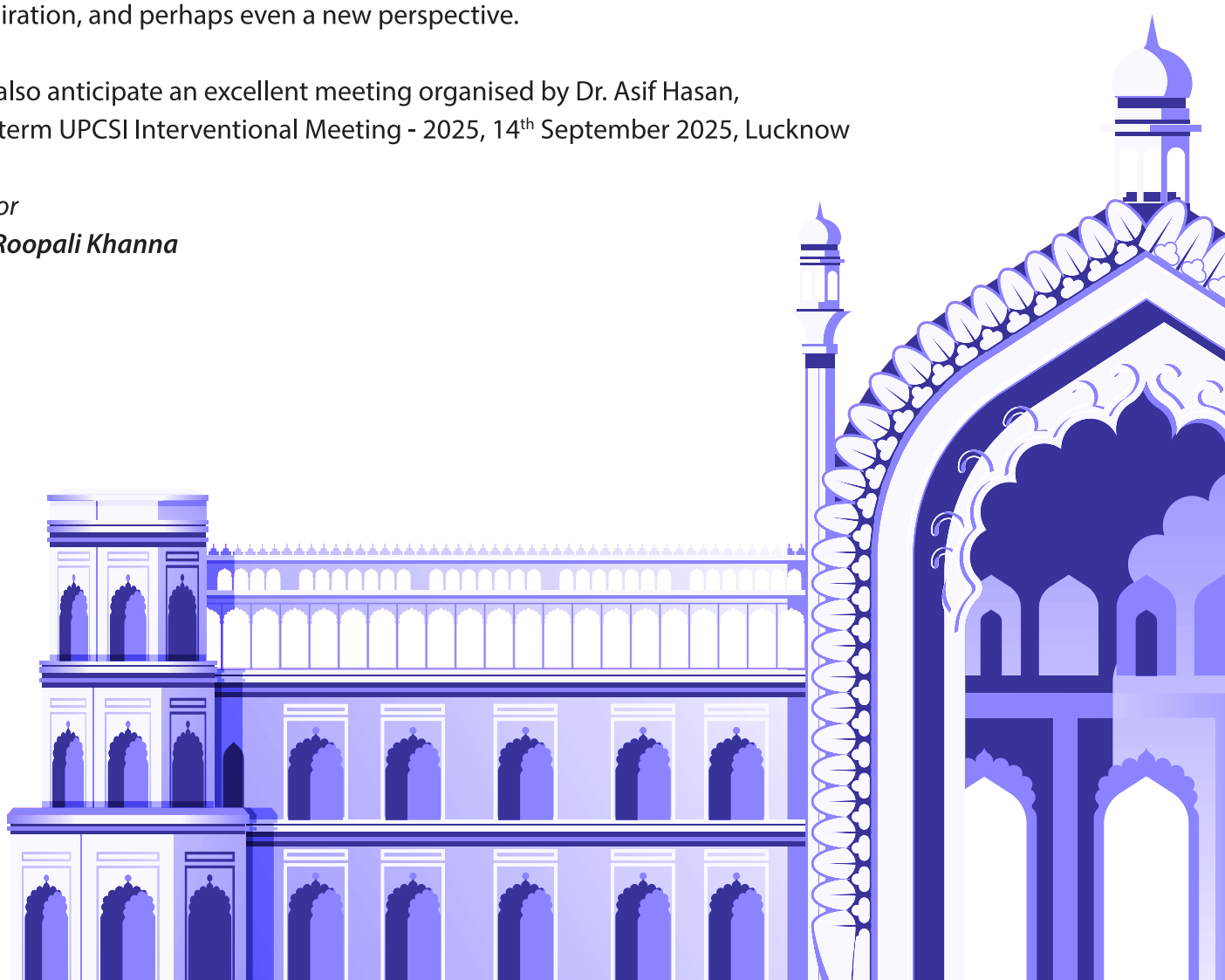
We are pleased to launch the new issue of the Newsletter in September 2025. This issue provides an overview of recent topics in the field of cardiology.

This issue features a diverse range of topics, including arrhythmias, advanced structural interventions, and challenges in coronary and pediatric interventions. Our contributors have worked tirelessly to bring you relevant and resonant content.

Thank you for your continued support. I hope this issue provides you with knowledge, inspiration, and perhaps even a new perspective.

We also anticipate an excellent meeting organised by Dr. Asif Hasan,
Midterm UPCSI Interventional Meeting - 2025, 14th September 2025, Lucknow

Editor
Dr. Roopali Khanna



ATRIAL FIBRILLATION IN HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFPEF): MANAGEMENT INSIGHTS

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Introduction

Atrial fibrillation (AF) is common in patients with heart failure with preserved ejection fraction (HFpEF; LVEF ≥50%), with prevalence around 40–60%. Both conditions often coexist, share risk factors (age, diabetes, ischemic heart disease), and worsen each other. Dyspnea without obvious volume overload can pose diagnostic challenges, as AF complicates interpretation of echocardiographic indices and natriuretic peptides.

AF in HFpEF is linked to poor atrial compliance, higher pulmonary pressures, and reduced exercise capacity. Presence of AF increases morbidity, stroke, and mortality, especially in persistent or permanent AF compared to paroxysmal AF. Optimal management of this overlap is crucial for improving outcomes.

Lifestyle and Comorbidity Management

Non-cardiac comorbidities contribute significantly to poor outcomes in HFpEF–AF. Addressing them improves rhythm control and disease progression.

• RACE 3 trial: Targeted therapy of comorbidities (HTN, obesity, hyperlipidemia, HF) improved sinus rhythm maintenance in AF patients (75% vs 63%).

• Weight loss: The LEGACY study showed ≥10% weight reduction led to a six-fold lower AF recurrence.

• Physical activity: Exercise improves cardiorespiratory fitness (meta-analysis) and reduces AF risk (TOPCAT trial: 42.8% lower risk with higher activity).

• Sleep apnea: Highly prevalent, but CPAP has not consistently reduced AF or HF events.

• Iron deficiency: The DAMOCLES trial showed anemia worsens quality of life and exercise capacity in HFpEF.

Medical Therapy

•SGLT2 inhibitors: PRESERVED-HF and EMPEROR-Preserved trials showed benefits in HFpEF regardless of AF status, with reduced HF hospitalizations, improved quality of life, and possibly lower AF events. SGLT2 inhibitors lead to weight loss , epicardial fat reduction and natriuresis that may reduce AF events. On the basis of these data, the ACC/AHA/HFSA guidelines gave SGLT2i as class 2a recommendation for the management of HFpEF. For the composite endpoint of cardiovascular death or first hospitalisation for HF, the effect was consistent for those with AF (HR 0.77, 95% CI 0.69–0.87) and those without (HR 0.83, 95% CI 0.72–0.95)

• ACEI/ARBs/ARNIs/β-blockers: Generally less effective in HFpEF; excessive rate control can blunt exercise tolerance.

• Anticoagulation: Direct oral anticoagulants (DOACs) are indicated to reduce high thromboembolic risk in HFpEF–AF.

• LAA occlusion (LAAO): Non-inferior to anticoagulation for stroke prevention but with higher device failure rates in HF patients.

Rhythm vs Rate Control

• Rate control: β-blockers or non-dihydropyridine CCBs are first line agents. The RATE-AF, an open-label trial compared the use of bisoprolol to digoxin in elderly HFpEF-AF patients and showed comparable primary outcome of QoL between the two groups at 6 months but the secondary outcome parameters such as functional ability and decrease in NT-proBNP were better in digoxin arm at 12 months, with comparable decrease in heart rate in both groups.

• Rhythm control with antiarrhythmics: Limited evidence; amiodarone may worsen outcomes in HFpEF.

• Catheter ablation: AF ablation incorporating (pulmonary vein isolation) PVI, is now a class I indication for treatment of individuals with symptomatic paroxysmal AF that is refractory to at least one anti-arrhythmic medication, and a class IIa recommendation for individuals with recurrent paroxysmal AF even prior to initiation of therapeutic trials of anti arrhythmic drug therapies

• EAST-AFNET 4 showed early rhythm control (mostly drugs, some ablation) reduced CV outcomes in AF with HF, especially in HFpEF. The primary outcome (composite endpoint of death from cardiovascular causes, stroke, hospitalization with worsening of HF or acute coronary syndrome) occurred less frequently in HF patients randomly assigned to early rhythm control vs those assigned to usual care (hazard ratio [HR] 0.74, 95% CI 0.56–0.97, p = 0.03)

• The exploratory analyses of AF patients in the EAST-AFNET 4 study suggested that amiodarone, but not treatment with flecainide, propafenone, or dronedarone, was potentially associated with early HF hospitalizations in patients with HFpEF

• CABANA trial (79% patients in the trial had HFpEF): Ablation reduced death, disabling stroke, or cardiac arrest by 36% in HFpEF–AF, though HF hospitalization effects were less robust.

• AV node ablation + pacing: The APAF-CRT trial showed reduction in all-cause mortality, irrespective of ejection fraction in hospitalized patients with permanent AF and heart failure with narrow QRS hospitalised for HF, irrespective of their baseline EF

ABC Pathway

The "Atrial Fibrillation Better Care (ABC)" approach offers a holistic

framework:
• A: Avoid stroke with anticoagulation.
• B: Better symptom management with tailored rate/rhythm control.
• C: Cardiovascular/comorbidity risk management, including lifestyle and psychosocial care.
Adherence improves outcomes in AF with HFpEF.
Conclusion
AF and HFpEF frequently coexist and significantly worsen quality of life, hospitalizations, and mortality. Diagnosis is often delayed due to overlapping symptoms. Evidence supports:
• Aggressive management of comorbidities (HTN, obesity, diabetes, sleep apnea, anemia).
• Use of SGLT2 inhibitors to improve prognosis in HFpEF, with possible benefits on AF.
• Anticoagulation for stroke prevention.
• Early rhythm control strategies (per EAST-AFNET 4) and catheter ablation (CABANA) show promise, though dedicated HFpEF trials are needed.
• AV node ablation with pacing remains a fallback option when other strategies fail.
In summary, comorbidity control, anticoagulation, early rhythm strategies, and patient-centered care are the cornerstones of managing AF in HFpEF.

Suggested Reading

1. Rienstra M, Hobbelt AH, Alings M, Tijssen JG, Smit MD, Brügemann J, Geelhoed B, Tieleman RG, Hillege HL, Tukkie R, Van Veldhuisen DJ. Targeted therapy of underlying conditions improves sinus rhythm maintenance in patients with persistent atrial fibrillation: results of the RACE 3 trial. European heart journal. 2018 Aug 21;39(32):2987-96.
2. Aminian A, Zajichek A, Arterburn DE, Wolski KE, Brethauer SA, Schauer PR, Kattan MW, Nissen SE. Association of metabolic surgery with major adverse cardiovascular outcomes in patients with type 2 diabetes and obesity. Jama. 2019 Oct 1;322(13):1271-82.
3. Zhu W, Liang W, Ye Z, Wu Y, He X, Xue R, Wu Z, Zhou Y, Zhao J, Dong Y, Liu C. Association of physical activity and risk of atrial fibrillation in heart failure with preserved ejection fraction. Nutrition, Metabolism and Cardiovascular Diseases. 2021 Jan 4;31(1):247-53.
4. Anker SD, Butler J, Filippatos G, Ferreira JP, Bocchi E, Böhm M, Brunner–La Rocca HP, Choi DJ, Chopra V, Chuquiure-Valenzuela E, Giannetti N. Empagliflozin in heart failure with a preserved ejection fraction. New England Journal of Medicine. 2021 Oct 14;385(16):1451-61
5. Solomon SD, McMurray JJV, Claggett B, de Boer RA, DeMets D, Hernandez AF, et al. Dapagliflozin in heart failure with mildly reduced or preserved ejection fraction. N Engl J Med. 2022;387:1089–1098. doi: 10.1056/NEJMoa2206286.
6. Vaduganathan M, Docherty KF, Claggett BL, Jhund PS, de Boer RA, Hernandez AF, et al. SGLT-2 inhibitors in patients with heart failure: a comprehensive meta-analysis of five randomised controlled trials. Lancet Lond Engl. 2022;400:757–767. doi: 10.1016/S0140-6736(22)01429-5.
7. Kotecha D, Bunting KV, Gill SK, Mehta S, Stanbury M, Jones JC, Haynes S, Calvert MJ, Deeks JJ, Steeds RP, Strauss VY. Effect of digoxin vs bisoprolol for heart rate control in atrial fibrillation on patient-reported quality of life: the RATE-AF randomized clinical trial. Jama. 2020 Dec 22;324(24):2497-508.
8. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC, Conti JB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. Journal of the American College of Cardiology. 2014 Dec 2;64(21):e1-76.
9. Rillig A, Magnussen C, Ozga A-K, Suling A, Brandes A, Breithardt G, et al. Early rhythm control therapy in patients with atrial fibrillation and heart failure. Circulation. 2021;144:845–858. doi: 10.1161/CIRCULATIONAHA.121.056323
10. Packer DL, Piccini JP, Monahan KH, Al-Khalidi HR, Silverstein AP, Noseworthy PA, Poole JE, Bahnson TD, Lee KL, Mark DB, CABANA Investigators. Ablation versus drug therapy for atrial fibrillation in heart failure: results from the CABANA trial. Circulation. 2021 Apr 6;143(14):1377-90.
11. Brignole M, Pentimalli F, Palmisano P, Landolina M, Quartieri F, Occhetta E, Calò L, Mascia G, Mont L, Vernooy K, Van Dijk V. AV junction ablation and cardiac resynchronization for patients with permanent atrial fibrillation and narrow QRS: the APAF-CRT mortality trial. European Heart Journal. 2021 Dec 7;42(46):4731-9.
12. Fauchier L, Bisson A, Bodin A. Heart failure with preserved ejection fraction and atrial fibrillation: recent advances and open questions. BMC Med. 2023; 21: 54.

CORONARY STENT INFECTION: A RARE AND UNFORESEEN COMPLICATION

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Introduction

Percutaneous coronary intervention (PCI) with stent implantation is one of the most commonly performed procedures in modern cardiovascular medicine. While generally safe and effective, complications may arise, ranging from in-stent restenosis to stent thrombosis. Among these, coronary stent infection (CSI) is one of the rarest and least expected adverse events. With fewer than a hundred reported cases in literature, CSI represents a critical yet under-recognized condition. This article provides a detailed review of its pathogenesis, clinical manifestations, diagnosis, management, and outcomes.

Epidemiology

Coronary stent infection is extremely rare, with an estimated incidence of <0.01% of PCI procedures. Most cases occur within 2 to 4 weeks after stent implantation, though delayed infections have been reported. The rarity of this complication has made it difficult to establish standardized diagnostic and management protocols.

Pathogenesis

The pathophysiology of CSI typically involves bacterial seeding of the stent either during the intervention or through hematogenous spread from a remote source. The most frequently isolated organism is Staphylococcus aureus, followed by coagulase-negative staphylococci, Pseudomonas aeruginosa, and occasionally gram-negative bacilli.

Risk Factors

- Breach of aseptic technique during PCI
- Prolonged or complex PCI procedures
- Multiple catheter exchanges
- Immunosuppression or diabetes mellitus
- Presence of indwelling catheters or intravenous lines

Once bacteria adhere to the metallic stent surface, biofilm formation occurs, rendering the infection resistant to antibiotics and host immune defences. This may lead to endothelial disruption, mycotic aneurysm, abscess formation, or even stent thrombosis.

Clinical Presentation

The clinical manifestations of CSI can be nonspecific and may mimic post-PCI complications such as pericarditis or recurrent angina. Common features include:

- Fever and chills
- Chest pain (often recurrent or persistent after PCI)
- Sepsis or septic shock in severe cases
- Elevated inflammatory markers (CRP, ESR, leukocytosis)

Complications may include:

- Coronary artery pseudoaneurysm
- Stent thrombosis leading to myocardial infarction
- Pericardial effusion or tamponade
- Systemic embolization

Diagnosis

Diagnosing CSI requires a high index of suspicion, especially in patients with unexplained fever and chest pain following stent implantation.

Diagnostic Tools

1. Blood Cultures: Often positive for Staphylococcus aureus or other pathogens.
2. Imaging:
   - CT Coronary Angiography: Identifies pseudoaneurysms, abscesses, or perivascular inflammation.
   - Transthoracic/Transesophageal Echocardiography: Detects pericardial involvement and complications.
   - Invasive Coronary Angiography: Reveals aneurysmal changes or stent-related abnormalities.
   - PET scan: Increased uptake at the site of infection in the vicinity of stent
3. Laboratory Tests: Elevated inflammatory markers (ESR, CRP, Procalcitonin) and cardiac enzymes.

Management

Treatment strategies are individualized, depending on severity and presence of complications.

Medical Therapy

- Prolonged Intravenous Antibiotics (typically 6–8 weeks)
- Empiric broad-spectrum coverage, later tailored to culture results
- Interventional and Surgical Therapy
- Stent removal with surgical revascularization (CABG) in severe cases
- Covered stent placement in selected patients with pseudoaneurysm
- Drainage of abscess or pericardial effusion if present
- Failure rate and mortality after medical therapy alone is quite high. Combination therapy with both antibiotics and revascularization often provides the best outcomes in complicated cases. In early cases medical therapy can be tried.

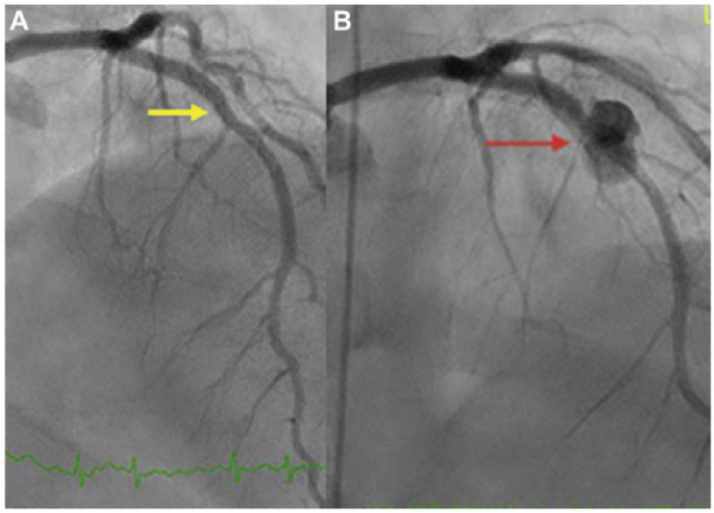
Prognosis
Despite advances in imaging and therapy, mortality from CSI remains high (30–40%). Outcomes depend on:
Early recognition
Appropriate antimicrobial therapy
Timely surgical or percutaneous intervention

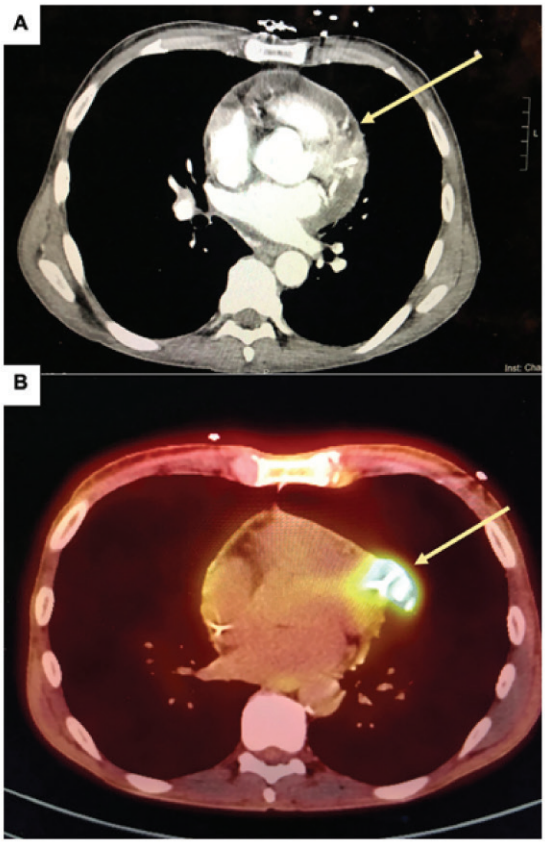
Conclusion

Coronary stent infection, though exceedingly rare, represents a potentially fatal complication of PCI. A high degree of clinical suspicion, combined with advanced imaging and microbiological testing, is essential for timely diagnosis. Early initiation of prolonged antibiotic therapy and consideration of surgical or interventional management remain the cornerstones of therapy. Increasing awareness among clinicians is crucial to improving outcomes in this unforeseen and life-threatening complication.

Suggested Readings

Robert S Dieter. Coronary artery stent infection. Clinical Cardiology 23, 808-810 (2000)
Francesco Bendandi et al. Coronary Stent Infection and Subsequent Abscessualization Causing Dislocation in Extravascular Position. JACC: Cardiovascular Interventions, Volume 15, Number 16 (July 2022)
Venkatakrishnan Ramakumar et al. Coronary Stent Infections — A Systematic Review and Meta-Analysis. Volume 54, September 2023, Pages 16-24
Nicole M. Robertson et al. Coronary Stent Infection Complicated by Persistent Methicillin-Sensitive Staphylococcus aureus Bacteremia Myocardial Abscess and Pericardial Effusion. Annals of Internal Medicine: Clinical Cases e221363 (2023)
Suryawan, I Gde Rurus et al. Coronary stent infection: a systematic review. Coronary Artery Disease 33(4):p 318-326, June 2022.
Sathish Kumar Rentapalla et al. Stent Infection Leading to Coronary Aneurysm and Coronary Cameral Fistula. JACC Journals › JACC: Case Reports.





Cardiac imaging to characterize coronary stent infection complicated by myocardial abscess on chest computed tomography with contrast (A) and cardiac positron emission tomography scan (B) with high uptake surrounding the stent consistent with any inflammatory pattern designated by yellow arrows.

CORONARY ARTERY CANNULATION AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION: CHALLENGES, DETERMINANTS, AND FUTURE PERSPECTIVES

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Introduction

TAVI has revolutionized the management of severe aortic stenosis. While initially limited to inoperable or high-risk patients, it is now routinely performed in younger, low-risk groups as well. This demographic shift highlights new priorities: not only valve durability, but also the capacity to perform safe and effective coronary angiography (CA) and percutaneous coronary intervention (PCI) in the years following TAVI.

Coronary Artery Disease In Aortic Stenosis

CAD is reported in up to 80% of patients undergoing TAVI . Registry data suggest that CAD progression is common, with up to 10% of patients experiencing acute coronary syndrome within two years post-TAVI. This reinforces the importance of guaranteed coronary accessibility.

Factors Influencing Coronary Re-Access

Coronary re-engagement after both patient anatomy and prosthesis design determine TAVI. Four major factors have been identified:

1. Stent frame–aortic wall relationship – Narrow sinuses of Valsalva (SoV) limit catheter maneuverability.
2. Leaflet position – Supra-annular leaflets may obstruct coronary ostia.
3. Commissural alignment – Random commissural positioning can block access.
4. Valve design – Frame height, cell size, and skirt design affect accessibility.

The RE-ACCESS study of 300 patients demonstrated an overall 7.7% rate of failed coronary cannulation, almost exclusively associated with Evolut self-expanding valves .

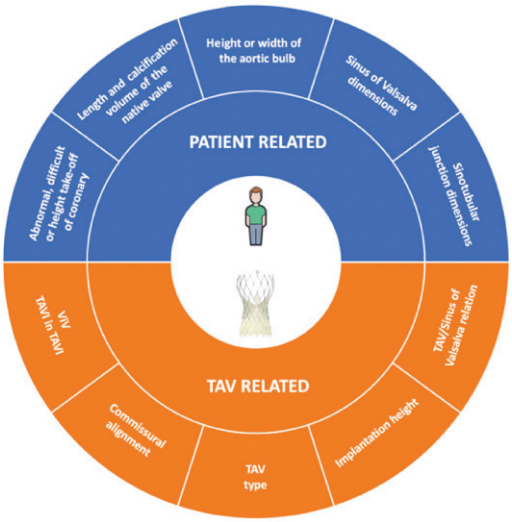


Figure 1. Factors influencing coronary re-access after TAVI.

Valve-Specific Considerations

Self-Expanding Valves

• **Evolut R/PRO/PRO+** – Nitinol, supra-annular design; prone to commissural overlap and coronary obstruction. Commissural orientation techniques (e.g., cusp overlap deployment) may mitigate risk.

• **Evolut FX+** - Enhanced coronary access: The valve is designed with three large, open windows in its frame, making it easier for physicians to access the coronary arteries if needed for future interventions. These windows are four times larger than in previous Evolut models.

• **ACURATE neo/neo2** – Features open-cell frames but supra-annular leaflet position increases overlap with coronaries.

• **ALLEGRA** – Large diamond-shaped cells designed to facilitate re-access, though clinical experience is limited.

• **PORTICO** – Intra-annular leaflet design and larger frame cells generally ease re-engagement.

Balloon-Expandable Valves

Balloon-Expandable Valves

• **SAPIEN 3 / Ultra** – Shorter cobalt-chromium frame, intra-annular leaflets, and large open upper cells provide a favorable profile for re-access.
• **Myval** – Similar to SAPIEN, with additional intermediate sizes; early data suggest excellent re-accessibility.

TECHNICAL STRATEGIES FOR CORONARY CANNULATION

With Self-Expanding Valves

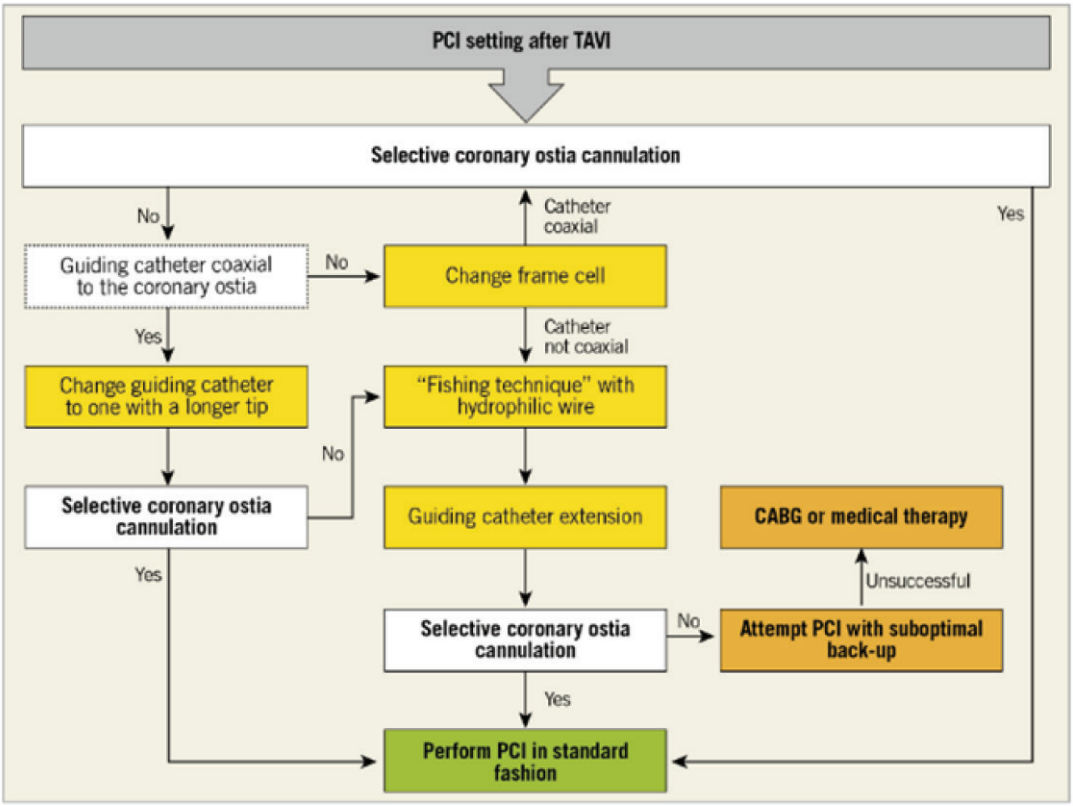
• Prefer femoral or left radial approach for coaxiality.
• Downsize diagnostic catheters (JL 3.0–3.5, JR 4.0).
• Engage ostia through cells aligned with the coronaries; if obstructed, use adjacent cells.
• Employ adjunctive tools: coronary guidewires, extension catheters, anchoring balloons.

With Balloon-Expandable Valves

• Standard catheter selection usually suffices.
• Cannulation achievable above the frame when SoV is tall; otherwise through large stent cells.
• Wire-assisted techniques may be required if commissural posts block direct engagement.

CORONARY RE-ACCESS STRATEGY AFTER TAV

It is essential to understand the potential challenges and technical implications of CA and PCI in patients who have undergone TAVI, particularly for quick and selective coronary ostia cannulation, especially in the setting of acute coronary syndromes (central illustration). Recently, the TAVRCathAID application was released on both the Google Play Store and the Apple Store. It is an educational mobile app with illustrations of the relationship between the coronary ostia and TAV in different anatomies, and a step-by-step guided approach to performing CA and PCI after TAVI (Bhatheja et al. TAVR Cath AID: an educational mobile application to learn coronary access after transcatheter aortic valve replacement. Presented at ACC. 2019, New Orleans, USA, 16-18 March 2019)



Central illustration. Algorithm for coronary ostia cannulation after TAVI in case of PCI.

CORONARY ACCESS AFTER TAVI-IN-TAVI

As TAVI expands, repeat procedures for degenerated prostheses (TAVI-in-TAVI) are becoming more frequent. These scenarios pose additional challenges:

- Presence of two valve frames
- Displaced bioprosthetic leaflets
- Commissural overlap
- Risk of coronary occlusion, especially with narrow SoV

CT studies suggest more than half of patients may have impaired access after TAVI-in-TAVI, particularly with supra-annular self-expanding valves. Intra-annular balloon-expandable valves (e.g., SAPIEN) offer more favorable conditions.

Novel strategies such as BASILICA leaflet laceration may preserve coronary flow and access in redo procedures, though experience with newer valves is limited.

Future Directions

- **Commissural alignment** at the time of implantation to reduce overlap.
- **Valve design innovations** prioritizing intra-annular leaflets and larger frame cells.
- **Operator guidance** tools such as the TAVRCathAID mobile app.
- **Prospective trials** to evaluate long-term coronary outcomes post-TAVI.

Conclusion

TAVI is an established therapy across risk groups, but coronary re-access remains a key issue, especially in younger patients and redo procedures. Balloon-expandable valves generally allow easier re-access, while supra-annular self-expanding valves are more challenging. Redo TAVI further complicates cannulation. Future valve technologies must balance durability with guaranteed coronary accessibility.

References

Stefanini GG, Stortecky S, Meier B, Windecker S, Wenaweser P. Severe aortic stenosis and coronary artery disease. EuroIntervention 2013;9:S63-8

Patterson T, Clayton T, Dodd M, Khawaja Z, Morice MC, Wilson K, Kim WK, Meneveau N, Hambrecht R, Byrne J, Carrié D, Fraser D, Roberts DH, Doshi SN, Zaman A, Banning AP, Eltchaninoff H, Le Breton H, Smith D, Cox I, Frank D, Gershlick A, de Belder M, Thomas M, Hildick-Smith D, Prendergast B, Redwood S, and ACTIVATION Trial Investigators. ACTIVATION (PercutAneous Coronary inTervention prIor to transcatheter aortic VAlve implantaTION): A Randomized Clinical Trial. JACC Cardiovasc Interv 2021;14:1965-74

Barbanti M, Costa G, Picci A, Criscione E, Reddavid C, Valvo R, Todaro D, Deste W, Condorelli A, Scalia M, Licciardello A, Politi G, De Luca G, Strazzieri O, Motta S, Garretto V, Veroux P, Giaquinta A, Giuffrida A, Sgroi C, Leon MB, Webb JG, Tamburino C. Coronary Cannulation After Transcatheter Aortic Valve Replacement: The RE-ACCESS Study. JACC Cardiovasc Interv 2020;13:2542-55

Zajarias A, Eltchaninoff H, Cribier A. Successful coronary intervention after percutaneous aortic valve replacement. Catheter Cardiovasc Interv 2007;69:522-4

Ochiai T, Chakravarty T, Yoon SH, Kaewkes D, Flint N, Patel V, Mahani S, Tiwana R, Sekhon N Nakamura M, Cheng W, Makkar R. Coronary Access After TAVR. JACC Cardiovasc Interv 2020;13:693-705

Khan JM, Greenbaum AB, Babaliaros VC, Rogers T, Eng MH, Paone G, Leshnower BG, Reisman M, Satler L, Waksman R, Chen MY, Stine AM, Tian X, Dvir D, Lederman RJ. The BASILICA Trial: Prospective Multicenter Investigation of Intentional Leaset Laceration to Prevent TAVR Coronary Obstruction. JACC Cardiovasc Interv 2019;12:1240-52

TRANSCATHETER AORTIC VALVE REPLACEMENT: EXPANDING INDICATIONS, CURRENT STATUS, AND FUTURE HORIZONS

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Transcatheter aortic valve replacement (TAVR) has revolutionized the treatment of aortic stenosis (AS) over the past two decades. Initially restricted to inoperable or high-risk surgical patients, its indications now encompass patients across the surgical risk spectrum. Technological advances, growing operator experience, and robust randomized controlled trial (RCT) evidence have driven this paradigm shift. Recent focus extends beyond severe symptomatic AS to bicuspid valve anatomy, small annuli, asymptomatic severe AS, moderate AS with heart failure, and pure aortic regurgitation. Device durability, coronary access, valve-in-valve procedures, and lifetime management strategies remain crucial considerations, particularly in younger patients.

Introduction

Since the landmark first-in-human TAVR in 2002, the procedure has emerged as one of the most significant advances in interventional cardiology. Severe symptomatic AS, once treated solely with surgical aortic valve replacement (SAVR), now has a less-invasive alternative that is safe, effective, and widely adopted. TAVR is currently recommended across all surgical risk categories by the ACC/AHA and ESC guidelines.

The expansion of TAVR into younger and lower-risk patients, together with exploration of novel indications such as bicuspid anatomy, valve-in-valve (ViV) therapy, and even moderate AS, represents a major shift in cardiovascular care.

Current Indications and Guideline Recommendations

Evidence from Randomized Controlled Trials

Multiple landmark trials (PARTNER, SURTAVI, Evolut Low Risk, NOTION) have demonstrated non-inferiority or superiority of TAVR compared with SAVR across surgical risk categories. Benefits include shorter recovery time, fewer perioperative complications, and comparable long-term survival.

Guideline Recommendations

- ACC/AHA 2020:
- TAVR recommended in patients >80 years or with life expectancy <10 years (Class I)1.
- Reasonable option in patients aged 65–80 years with suitable anatomy (Class I).
- ESC 2021:
- TAVR recommended in patients >75 years or those at high/prohibitive surgical risk2.
- Choice between TAVR and SAVR in others depends on anatomy and heart team decision-making.

Technological Advances in TAVR

Valve Design Evolution

- Self-expanding valves (SEV): e.g., Evolut FX, Acurate Neo 2, Portico/Navitor.
- Balloon-expandable valves (BEV): e.g., Sapien 3 Ultra Resilia, MyVal Octacor.
- Mechanically expandable valves (MEV): Lotus Edge (withdrawn but foundational).
- Innovations Enhancing Outcomes
- Lower-profile delivery systems for transfemoral access.
- Improved sealing skirts to minimize paravalvular leak.
- Commissural alignment features to preserve coronary access.
- Anti-calcification technologies such as RESILIA tissue.
- Biomimetic valves (DurAVR) designed for enhanced durability and laminar flow.

Long-Term Durability and Valve Performance

Durability is the most pressing concern as TAVR expands to younger patients.

- RCT follow-ups: PARTNER-3 and Evolut-LR confirm sustained hemodynamic improvements at 5 years.
- NOTION trial: At 10 years, TAVR had lower structural valve deterioration (20.2%) compared with SAVR (37.3%).
- Bench data: Simulations suggest BEV and SEV platforms may function effectively for up to 20–25 years.

Despite encouraging data, real-world durability beyond a decade is not fully established.

Expanding Indications Beyond Severe AS

Bicuspid Aortic Valve (BAV)

- Historically excluded from trials due to anatomic complexity.
- Improved outcomes with new devices and imaging.
- Registry data (PARTNER 3 BAV low-risk cohort) show outcomes comparable to tricuspid AS in selected

anatomies.
- Caution: heavily calcified raphes and large aortas remain high-risk.

Valve-in-Valve (ViV) Therapy
- Effective alternative to redo-SAVR for failed bioprosthetic valves.
- Success depends on prior valve type, size, and the feasibility of valve fracturing.
- Concerns include coronary obstruction and high residual gradients.

Pure Native Aortic Regurgitation (AR)
- Absence of annular calcification historically limited TAVR feasibility.
- Dedicated devices (JenaValve Trilogy, J-Valve) show promising results in early trials.
- May address a large unmet need in nonsurgical AR patients.

Asymptomatic Severe and Moderate AS
- Early intervention in asymptomatic severe AS may prevent irreversible remodeling.
- Ongoing RCTs: EARLY-TAVR, EASY-AS, PROGRESS, TAVR-UNLOAD.
- Preliminary results suggest benefits in reducing heart failure hospitalizations and improving quality of life.

Procedural Considerations
- Coronary access: Important for younger patients needing future PCI; commissural alignment techniques are evolving.
- Conduction abnormalities: Pacemaker implantation rates remain higher after TAVR, particularly with SEVs.
- Antithrombotic therapy: Current data favor single antiplatelet therapy in most patients; anticoagulation reserved for leaflet thrombosis or other indications.

Challenges and Future Horizons

1. Durability: Definitive >15-year data are lacking.
2. Patient selection: Identifying which bicuspid, moderate AS, or AR patients benefit most.
3. Redo-TAVR and lifetime management: Sequencing interventions over decades will be vital in younger patients.
4. Economic impact: Expanding indications to younger populations raises cost-effectiveness concerns.
5. Device innovation: Designs balancing durability, low gradients, and preserved coronary access will shape the future.

Indication	Key Evidence	Outcomes	Current Status
Severe symptomatic AS	PARTNER, SURTAVI, Evolut Low Risk	Comparable/superior to SAVR	Standard of care
Bicuspid valve AS	Registries, PARTNER 3 BAV registry	Similar outcomes in select anatomies	Emerging, not guideline standard
Valve-in-Valve	Global registries, VARC-3	High success, avoids redo-SAVR	Established option
Pure AR	JenaValve, J-Valve trials	Promising safety/efficacy	Investigational
Asymptomatic severe AS	EARLY-TAVR, EASY-AS	Reduced CV hospitalization	Trials ongoing
Moderate AS with HF	TAVR-UNLOAD	QoL improvement, trend to benefit	Investigational

Conclusion

TAVR has transcended its original role as a therapy for inoperable AS to become the treatment of choice across surgical risk profiles. Its boundaries are expanding to encompass bicuspid anatomy, valve-in-valve, pure AR, and possibly moderate AS with heart failure. Technological innovation continues to improve safety, durability, and deliverability, while long-term durability remains the key unanswered question. The next decade will determine whether TAVR becomes not only the preferred therapy for AS but also a solution for a wider range of valvular pathologies.

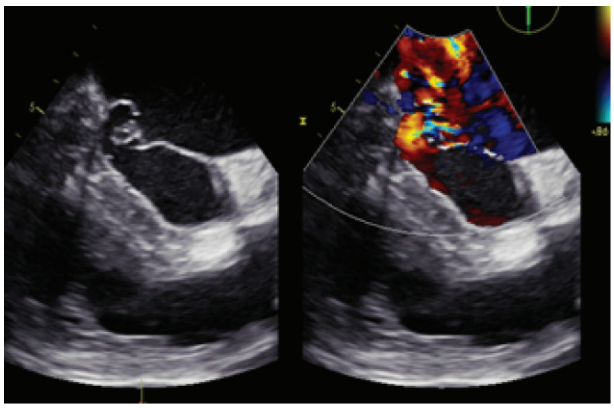
References

1. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines
2. 2021 ESC/EACTS guidelines for the management of valvular heart disease
   Eur Heart J, 43 (2022), pp. 561-632Frans J. Beerkens,
3. Gilbert H.L. Tang, Annapoorna S. Kini, Stamatios Lerakis, George D. Dangas, Roxana Mehran, Sahil Khera, Martin Goldman, Valentin Fuster, Deepak L. Bhatt, John G. Webb, Samin K. Sharma,Transcatheter Aortic Valve Replacement Beyond Severe Aortic Stenosis: JACC State-of-the-Art Review

TRANSCATHETER EDGE TO EDGE REPAIR (TEER): TO CLIP OR NOT TO CLIP- PATIENT SELECTION HOLDS THE KEY

DR. ROOPALI KHANNA, DR. HARSHIT KHARE
SGPGIMS, LUCKNOW

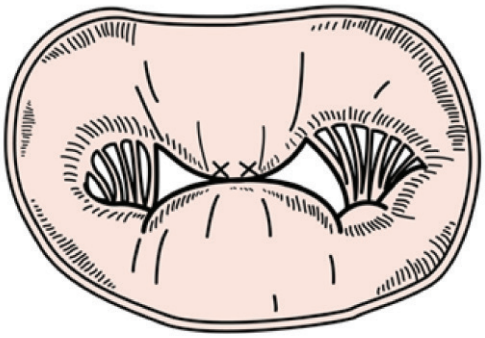
A 70-year-old gentleman visits a cardiac specialty OPD at a tertiary care centre for his symptoms of dyspnea, along with orthopnoea and paroxysmal nocturnal dyspnea. He was suffering from severe degenerative mitral valve regurgitation (MR) with dilated and remodelled left ventricular chamber and left atrium, along with the presence of other co-morbidities like COPD and Diabetes. After an initial evaluation by a senior resident, the resident has a dilemma in choosing the right therapeutic strategy for him: whether to continue with medical therapy or send him for Mitral Valve repair, or can he be considered for transcatheter mitral valve repair (TMVR) or Mitral Transcatheter Edge to Edge repair (M-TEER)?

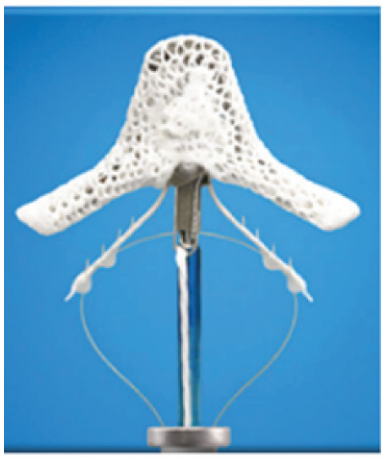


Transoesophageal echocardiography showing - Degenerative flail mitral leaflets with prolapsing P2, P3 leading to severe Mitral regurgitation

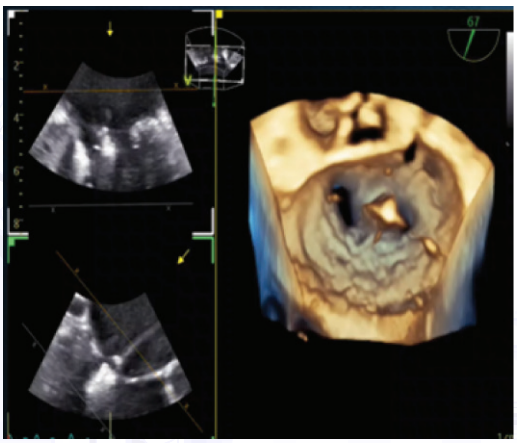
As he scrutinises his case and weighs the evidence in favour and against the M-TEER, he asks his senior to clear his doubt. The discussion that follows is summarized below and offers learning insights for all the young cardiologists who would be making this decision in the future.

He is told that the principle of M-TEER is based on the "Alfieri stitch of edge-to-edge repair" and represents a percutaneous variant of its surgical counterpart. Since its FDA approval in 2013, not only has the number of patients undergoing M-TEER increased, but also the spectrum of their usage.





Alfredi surgical repair, Pictorial representation of Mitra clip



Trans-oesophageal echocardiography showing clipping of mitral leaflets leading to a double orifice mitral valve

The first clinical evidence in favour of M-TEER came from the EVEREST-II trial, which showed that M-TEER was comparable to MV surgery in patients deemed high risk for surgery and having symptomatic functional MR.3,4 Later COAPT trial demonstrated the superiority of the MitraClip device over guideline-directed medical therapy (GDMT) in patients of both ischemic and non-ischemic HF with symptomatic HF with severe secondary MR.5 While it was clear that MitraClip would benefit severe secondary MR patients who were on GDMT, the conflict of opinion occurred when the results of Mitra-FR came. The Mitra-FR trial showed no difference in death or heart failure hospitalization (HHF) in patients with severe secondary MR.6

The postulated reason for this difference was perhaps the patient selection. Unlike the Mitra-FR trial, the patients in the COAPT trial had more MR per unit of ventricular volume (disproportionate mitral regurgitation) with lesser dilated chambers and were already on GDMT. More recently, the RESHAPE-HF-2 trial further consolidated the evidence for MitraClip use in symptomatic moderate to severe secondary MR for reducing HHF, improving quality of life, as well as reducing all-cause mortality.7 Based on the results of these trials (Summarized in Table 1) and recent meta-analysis8, the European Society of Cardiology (ESC) recently released valvular heart disease guidelines, upgrading the recommendation for M-TEER from previous class IIb to Class I now. It advocates that TEER should be offered to patients with severe secondary MR with symptomatic HF with an LVEF of <50% provided they are already on optimized GDMT and CRT when indicated, and fulfilling specific clinical and echocardiographic criteria.9 They further outlined the following echocardiographic criteria to look for when making patient selection:

a) Suitable anatomy for M-TEER.
b) NYHA class ≥II.
c) LVEF 20%–50%.
d) LVESD ≤70 mm.
e) At least one HHF hospitalization in the previous year or raised cardiac biomarkers (BNP ≥300 pg/mL or NT-proBNP ≥1000 pg/mL).
f) Systolic Pulmonary Artery Pressure ≤70 mmHg.
g) No severe RV dysfunction.
h) No Stage D or advanced HF. No significant coronary artery disease requiring intervention.
j) No significant Aortic valve and/or Tricuspid Valve disease.
k) No cardiomyopathies.

If, however, these above-mentioned criteria are not fulfilled, the M-TEER becomes lesser indicated with a recommendation of class IIb.

Only a few trials have evaluated the benefit of M-TEER in primary MR patients with intermediate and high surgical risks.10,11 In primary MR with suitable valve anatomy, guidelines give a class IIa recommendation for M-TEER.9

More recently, a newer system has evolved, named Transcatheter Mitral Valve Implantation (TMVI), replicating surgical MV implantation for MV anatomy unsuitable for M-TEER, but the evidence is still limited.12

As the consultant concluded the discussion, enumerating the guideline-specified criteria, he asked his resident, who was still assimilating all the evidence presented, to review the clinical status and echocardiographic findings of the patient to see if he could benefit from M-TEER.

What do you think, was this patient a good candidate for surgery?

TRIAL	STUDY DESIGN	PRIMARY OUTCOME	RESULTS	COMMENTS
EVEREST-II (2011)	279 patients - severe (grade 3 or more) MR symptomatic with an LVEF of >25% & LVESD <55mm, asymptomatic with an LVEF of 25-60% & LVESD > 40mm, or had significant PAH or new onset Atrial Fibrillation (AF). Mean age- 67 years Mean LVEF - 60% Degenerative MR–73%, Functional mitral regurgitation – 27%. Follow-up – 5 years The baseline characteristics of the two groups were comparable except for the prevalence of congestive heart failure, seen in 91% of the TMVR group compared to 78% in the surgical therapy group.	The primary outcome is a composite of freedom from surgery, death, reoperation, or the development of grade 3 or more MR. The primary safety outcome -occurrence of either MACE or renal failure or prolonged requirement (>48hours) for a ventilator, or new onset AF or requirement for blood transfusion > 2 units or deep wound sepsis or gastrointestinal complications following surgery at 30 days. Secondary endpoints - quality of life (QOL) and NYHA functional class.	Primary effectiveness outcome was 55% in the M-TEER group and 73% in the surgical group ($p = 0.007$). The primary safety outcome was a 15% M-TEER group and 48% surgical group (repair/replacement) ($p < 0.001$), primarily driven by a lower need for blood transfusions in the M-TEER group. No significant differences between the two groups in terms of improvement in the LVEF, LVESD, QOL, and the NYHA class.	M-TEER significantly reduced the grade of MR and the related symptoms. In the functional MR group, the results were comparable to those of surgery at the 1-year follow-up. In contrast, surgery performed better than TMVR for degenerative MR in terms of reoperation rates. The 5-year follow-up data showed that M-TEER patients more commonly required surgical correction during the first year of follow-up, but during the later years of follow-up, there were lower rates of conversion from TMVR to surgical correction.4
COAPT (2018)	614 patients- symptomatic HF and grade 3 or more of secondary MR on GDMT MitraClip plus GDMT group - 302 GDMT group - 312 patients. Open-label design trial F/U of 24 months. These patients had a history of recurrent hospitalisation for HF (HHF) and had raised cardiac biomarkers of HF (BNP ≥300 pg/ml or a NT-proBNP ≥1500 pg/ml) and were not appropriate for MV surgery.	The primary effectiveness outcome was HHF at 24 months. The primary safety outcome was freedom from device complications at one year. The secondary endpoints included MACE, stroke, requirement for LVAD or heart transplant, all-cause mortality alone and in combination with HHF, changes in LV dimensions, and severity of MR and worsening of tricuspid regurgitation (TR).	Primary effectiveness endpoint- 35.8 % Mitraclip group compared to 67.9% in GDMT ($p < 0.001$). The primary safety endpoint, 96.6% for MitraClip ($p < 0.001$). Secondary outcomes were significantly better for the Mitraclip group compared to GDMT, except for TR.	The use of MitraClip in a patient with severe secondary MR was superior to GDMT.

TRIAL	STUDY DESIGN	PRIMARY OUTCOME	RESULTS	COMMENTS
MITRA-FR (2018)	304 patients with severe secondary MR with symptomatic HF were randomized either to MitraClip (n = 152) or medical therapy (n = 152). Mean LVEF was between 15 and 40%	The primary outcome was a composite of death or HHF. The secondary outcomes included death or HHF alone and in combination.	The composite primary endpoint - 54.6% of the MitraClip group compared to 51.3% of the medical therapy group (p = 0.53). The secondary outcomes were also not significant.	In severe secondary MR with symptomatic HF, MitraClip was not better to GDMT. The lack of benefit was likely due to the poor selection of patients who were not on GDMT and had severe underlying cardiomyopathy scoring a poor prognosis in them.
RESHAPE-HF-2 (2024)	621 patients with moderate to severe MR with symptomatic HF with a history of recent worsening in the form of either HHF or raised cardiac biomarkers and LVEF between 20-50%, were enrolled and randomized to receive either MitraClip + GDMT or GDMT only in an open-label design.	The primary endpoint included a composite of the rate of first or recurrent HHF or death at 2 years of follow-up. Secondary endpoints included all-cause mortality, cardiovascular (CV) mortality, 6-minute walk distance (6MWD), quality of life, and less than grade 2-MR at 12 months.	The primary outcome was reduced by 36% in the MitraClip + GDMT group compared to the GDMT alone group (p <.001); secondary endpoints were also significantly improved in the MitraClip group for each of them.	This trial not only extended the benefit of M-TEER to the moderate-grade secondary MR group but also demonstrated a mortality benefit at 2 years compared to GDMT alone.

References

1. Zhou S, Egorova N, Moskowitz G, et al.: Trends in MitraClip, mitral valve repair, and mitral valve replacement from 2000 to 2016. J Thorac Cardiovasc Surg. 2021, 162:551-562.e4
2. Suradi HS, Kavinsky CJ, Hijazi ZM: Percutaneous mitral valve repair: the MitraClip device. Glob Cardiol Sci Pract. 2016, 2016:e201617.
3. Whitlow PL, Feldman T, Pedersen et al; EVEREST II Investigators. Acute and 12-month results with catheter-based mitral valve leaflet repair: the EVEREST II (Endovascular Valve Edge-to-Edge Repair) High Risk Study. J Am Coll Cardiol. 2012 Jan 10;59(2):130-9. doi: 10.1016/j.jacc.2011.08.067. PMID: 22222076.
4. Feldman T, Kar S, Elmariah S, et al; EVEREST II Investigators. Randomized Comparison of Percutaneous Repair and Surgery for Mitral Regurgitation: 5-Year Results of EVEREST II. J Am Coll Cardiol. 2015 Dec 29;66(25):2844-2854. doi: 10.1016/j.jacc.2015.10.018. PMID: 26718672.
5. Stone GW, Lindenfeld J, Abraham WT, et al; COAPT Investigators. Transcatheter Mitral-Valve Repair in Patients with Heart Failure. N Engl J Med. 2018 Dec 13;379(24):2307-2318. doi: 10.1056/NEJMoa1806640. Epub 2018 Sep 23. PMID: 30280640.
6. Obadia JF, Messika-Zeitoun D, Leurent G, et al; MITRA-FR Investigators. Percutaneous Repair or Medical Treatment for Secondary Mitral Regurgitation. N Engl J Med. 2018 Dec 13;379(24):2297-2306. doi: 10.1056/NEJMoa1805374. Epub 2018 Aug 27. PMID: 30145927.
7. Anker SD, Friede T, von Bardeleben RS, et al; RESHAPE-HF2 Investigators. Transcatheter Valve Repair in Heart Failure with Moderate to Severe Mitral Regurgitation. N Engl J Med. 2024 Nov 14;391(19):1799-1809. doi: 10.1056/NEJMoa2314328. Epub 2024 Aug 31. PMID: 39216092.
8. Praz F, Borger MA, Lanz J, et al; ESC/EACTS Scientific Document Group. 2025 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2025 Aug 29:ehaf194. doi: 10.1093/eurheartj/ehaf194. Epub ahead of print. PMID: 40878295.
9. Makkar RR, Chikwe J, Chakravarty T, Chen Q, O'Gara PT, Gillinov M, et al. Transcatheter mitral valve repair for degenerative mitral regurgitation. JAMA 2023;329:1778–88. 10.1001/jama.2023.7089

BASICS OF IVUS FOR FELLOWS

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Principle of IVUS

IVUS operates on the same basic principles as conventional ultrasound. IVUS catheters typically emit ultrasound waves in the 20–45 MHz range with high-definition (HD) catheters of frequencies up to 60 MHz, offering axial resolution of 100–200 micrometers and tissue penetration depths of up to 10 mm.3

Catheter Designs

There are two main IVUS catheter designs:

Mechanical Transducer: Utilizes a single rotating transducer at the catheter tip to generate a 360-degree image. These catheters are generally smaller and offer higher resolution (up to 40 MHz), making them the preferred design in current practice.

Solid-State (Phased Array) Transducer: Comprises multiple transducer elements arranged circumferentially at the tip, which are activated sequentially. Although typically lower in resolution (around 20 MHz), these catheters offer structural durability and rapid image acquisition.3,7

Coronary Artery Measurements by IVUS

The following is a brief list of many of the commonly used measurements and terms that are thought to be important for the practicing interventionalist.4

Proximal Reference Vessel:

The segment with the largest lumen proximal to the stenosis, within the same vessel segment, typically located within 10 mm of the lesion and free of side branches.

Distal Reference Vessel:

The segment with the largest lumen distal to the stenosis, also within 10 mm and without intervening branches.

Maximal Lumen Diameter:

The greatest lumen diameter measured between the leading edges of the intima on opposite sides.

Minimal Lumen Diameter:

The smallest lumen diameter measured between the leading edges of the intima on opposite sides.

Reference Lumen Diameter:

Diameter of the vessel lumen as measured from the leading edge of the external elastic membrane (EEM) on both sides.

Lumen Eccentricity:

A measure of shape asymmetry: (Maximal Lumen Diameter-Minimal Lumen Diameter)/ Maximal Lumen Diameter

Luminal Cross-Sectional Area (CSA):

The circumferential area enclosed by the luminal border.

External Elastic Membrane CSA (EEM CSA) or Reference Luminal CSA:

The cross-sectional area enclosed by the EEM, representing the full vessel area.

Area Stenosis (%):

Quantifies the degree of lumen narrowing: {(Luminal CSA-Minimum Vessel CSA)/Luminal CSA}×100

Plaque or Atheroma Area: Plaque Area= EEM CSA – Luminal CSA

Plaque Burden (%):

Indicates the extent of atherosclerosis relative to the total vessel area: (Plaque area/EEM CSA)*100

Remodeling Index (RI):

Reflects positive or negative remodeling at the lesion site: (Lesion EEM CSA/Reference vessel EEM CSA)*100

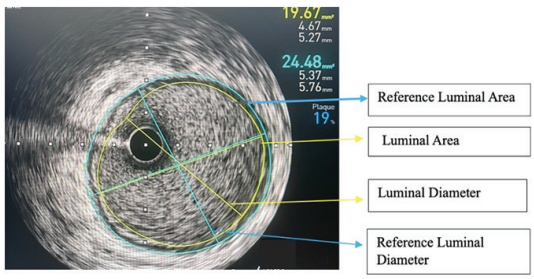
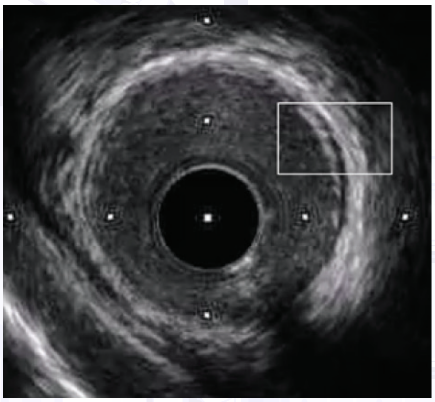


Figure 1. IVUS image of normal coronary artery showing measurements

Endovascular Anatomy on IVUS-

Arterial Wall

A typical IVUS image of a coronary arterial segment shows a three-layered arterial wall. The inner layer is relatively bright compared to the blood speckle surrounding the catheter and represents the intima and internal elastic lamina-including plaque in diseased arteries. The middle layer usually is a dark echo-lucent band and represents the media. The outer layer is quite echogenic (bright) and represents the external elastic lamina, adventitia and periadventitial tissue.



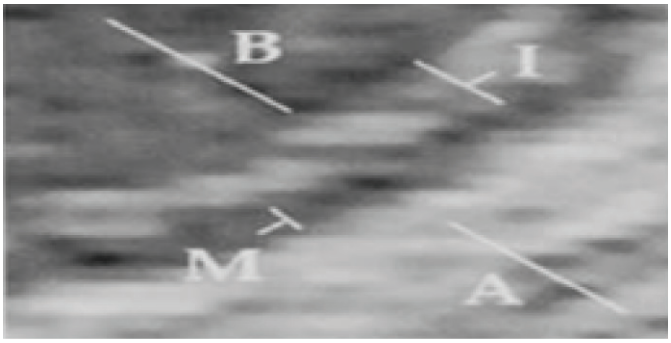


Figure 2: The typical three-layer architecture of the arterial wall. B= blood; I = intima;M = media; A = adventitia.

Blood

Blood is typically easy to identify on IVUS images due to its characteristic speckled or grainy appearance, which fluctuates with the cardiac cycle.

Perivascular Structures

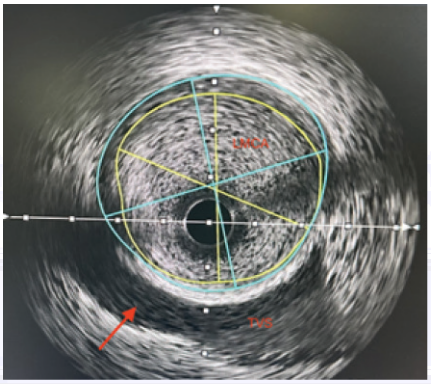
With advancements in IVUS image quality and depth of penetration, it is now possible to see structures beyond the vessel wall. These perivascular landmarks help identify the exact position and orientation within the artery.

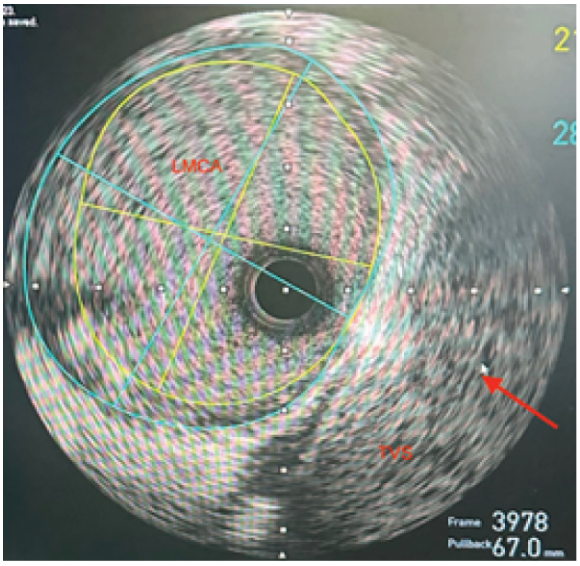
Coronary Veins

Coronary veins follow variable paths, with the great cardiac vein–coronary sinus system being the largest and primarily responsible for draining the left ventricular myocardium. The anterior interventricular vein runs alongside the left anterior descending (LAD) artery within the anterior interventricular sulcus. It gradually increases in diameter from distal to proximal before merging into the coronary sinus. The great cardiac vein travels parallel to the left circumflex artery (LCX), enlarging as it approaches the coronary sinus.

Transverse Sinus

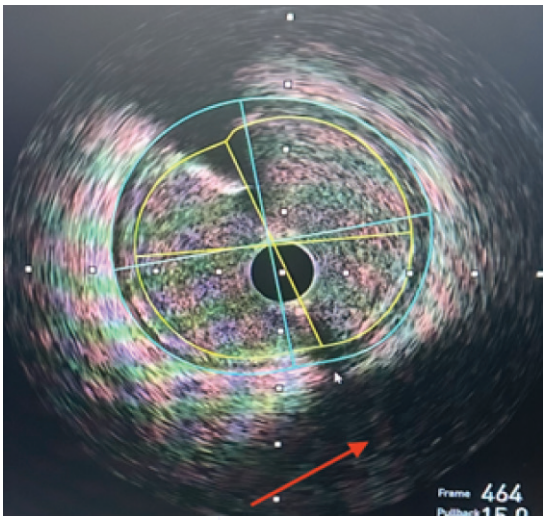
After pullback from the left anterior descending artery (LAD) to the confluence with the left circumflex artery (LCX), an echo-free space may be observed outside the left main trunk (LMT). This space lies in the epicardial direction, approximately 90° clockwise from the proximal portion of the LCX. It is referred to as the transverse sinus, representing a pericardial effusion localized within the pericardium at the transition zone between the aorta and pericardium.





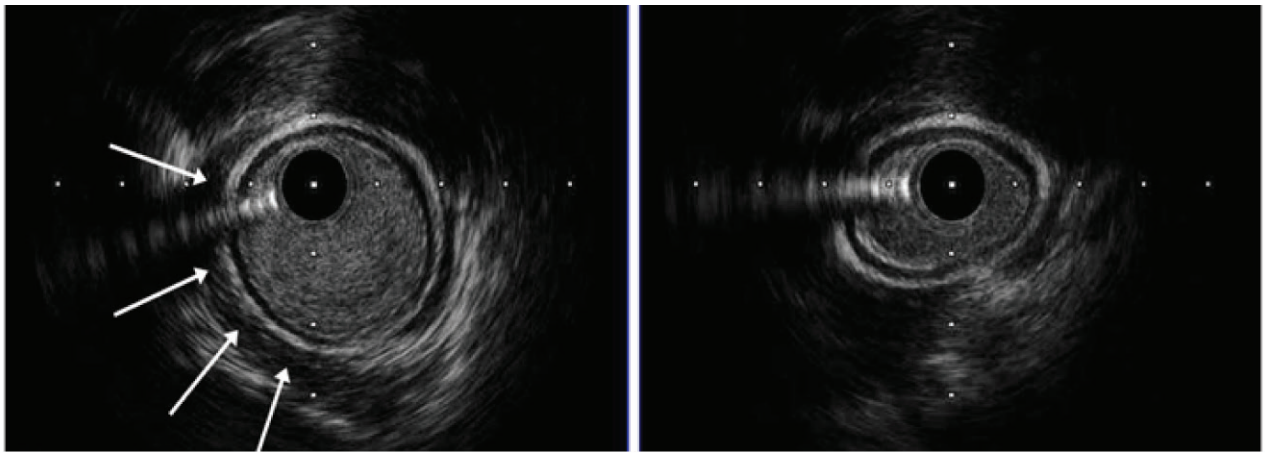
Triangle of Brocq-Mouchet

In the proximal segment of the LAD, a triangular echo-free space may be visualized outside the vessel, adjacent to the LCX. This region corresponds to a physiologic pericardial recess, anatomically defined by the LAD, the LCX, and the anterior interventricular vein, and is known as the triangle of Brocq–Mouchet.



Myocardial Bridge

When a segment of a coronary artery, most commonly the mid LAD, courses intramurally beneath the myocardium, dynamic narrowing may occur during systolic contraction. This phenomenon, termed "squeezing" or "myocardial bridging" on coronary angiography (CAG), results in functional stenosis. The overlying myocardial fibers that encase the arterial segment are referred to as a myocardial bridge.

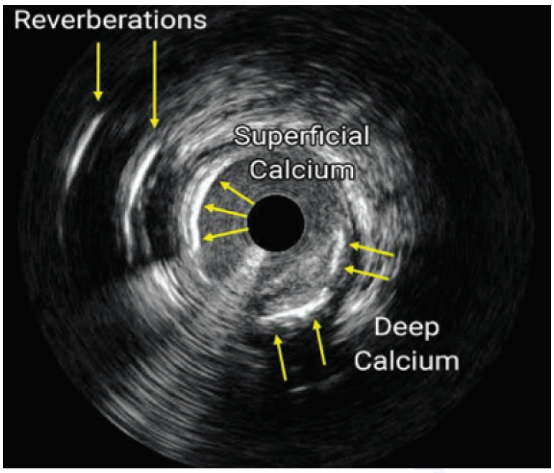


Myocardial bridge. White arrows showing the muscle bundle overlying the artery. The lumen is clearly narrowed during systole and the intimal thickening may be seen. (Left) Diastole. (Right) Systole.

Plaque morphology on IVUS-

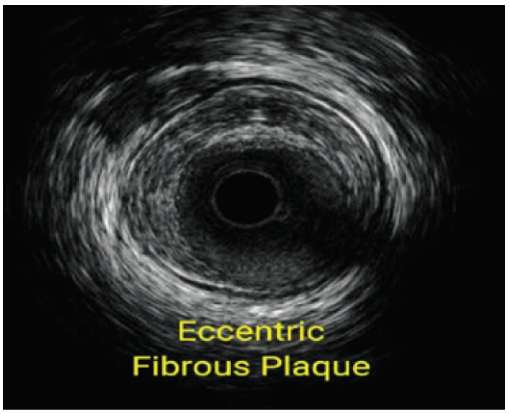
Calcific Plaque

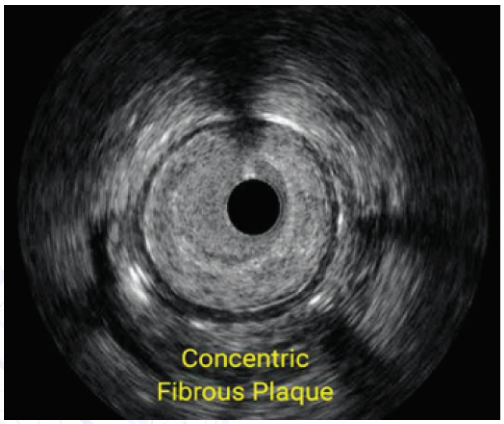
A distinctive feature of calcification is the presence of bright, concentric "ghost arcs" or reverberations within the shadowed region, which appear at equidistant intervals corresponding to multiples of the transducer–calcium distance.



Fibrous Plaque

Fibrous plaque appears moderately bright on IVUS, with a brightness comparable to that of the adventitia. Because it contains collagen and elastin, the ultrasound beam is gradually weakened as it passes through, sometimes creating a shadow behind the plaque called as progressive signal attenuation.





Fatty Plaque

Fatty plaque appears relatively echolucent on IVUS, with a soft gray-scale texture. Its homogeneous composition allows ultrasound waves to traverse the tissue with minimal impedance mismatch, resulting in reduced signal attenuation compared with fibrous plaque.

IVUS Optimization in PCI (1,2,3 Rule)

The IVUS 123 Essentials during percutaneous coronary intervention (PCI) refers to a simplified approach by following three pre-stent and three post-stent steps—collectively known as the "1-2-3 rule." This method simplifies IVUS-guided decision-making and improves outcomes by ensuring comprehensive lesion evaluation and stent optimization.7,8

Pre PCI

1 – Establish lesion length
2- Assess the plaque morphology
3 - Measure the vessel size

Post PCI

1 – Look for edge dissections and geographical miss
2 – Look for malapposition
3 – Look for optimal stent expansion

References

1. Kimoto S, Omoto R, Tsunemoto M. Ultrasonic tomography of the liver and detection of heart atrial septal defect with the aid of ultrasonic intravenous probes. Ultrasonics. 1964;2:82.
2. Kossoff G. Diagnostic application of ultrasound in cardiology. Aust Radiol. 1966;10:101–6.
3. Fitzgerald PJ, Yock PG: Mechanisms and outcomes of angioplasty assessed by intravascular ultrasound imaging. J Clin Ultrasound.1993;21:579.
4. Mintz GS, Douek P, Pichard AD, et al: Target lesion calcification in coronary artery disease: An intravascular ultrasound study. J Am Coll Cardiol.1992;20:1149.
5. Tuzcu EM, Berkalp B, De Franco AC, Ellis SG, Goormastic M, Whitlow PL, Franco I, Raymond RE, Nissen SE. The dilemma of diagnosing coronary calcification: angiography versus intravascular ultrasound. J Am Coll Cardiol. 1996 Mar 15;27(4):832-8.
6. Hiro T, Leung CY, de Guzman S, et al: Are "soft echoes" really soft? Ultrasound assessment of mechanical properties in human atherosclerotic tissue [abstr]. Circulation 1995;92:I-649.
7. Honye Junko. Basics of Comprehensive IVUS-Guided PCIJ. 2nd edition. Springer.
8. Truesdell AG, Alasnag MA, Kaul P, Rab ST, Riley RF, Young MN, Batchelor WB, Maehara A, Welt FG, Kirtane AJ; ACC Interventional Council. Intravascular Imaging During Percutaneous Coronary Intervention: JACC State-of-the-Art Review.J Am Coll Cardiol. 2023 Apr 18;81(15):1550

PLUGGING THE GAP: WHAT'S NEW IN PERCUTANEOUS VSD CLOSURE?

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Ventricular septal defect (VSD) represents one of the most common congenital heart malformations, characterized by an abnormal opening in the septum dividing the heart's lower chambers. This defect allows oxygenated and deoxygenated blood to mix, potentially leading to heart failure, pulmonary hypertension, or recurrent infections if untreated. Traditionally, surgical repair via open-heart procedures under cardiopulmonary bypass has been the gold standard, offering reliable closure but at the cost of invasiveness, longer recovery times, and risks like infection or sternotomy scars. Over the past two decades, percutaneous VSD closure has emerged as a minimally invasive alternative, using catheter-based devices to seal the defect without major surgery. This approach, first pioneered in the late 1980s, has gained traction due to advancements in device design and imaging, reducing procedural risks and hospital stays. As of 2025, innovations in devices, techniques, and hybrid methods are expanding its applicability, even to complex cases like post-myocardial infarction (MI) VSDs, plugging the gap in treatment options for high-risk patients.

Historically, percutaneous closure began with rudimentary umbrella-like devices, such as the Rashkind double umbrella, tested in small cohorts with variable success. Early challenges included device embolization, residual shunts, and high rates of atrioventricular (AV) block, particularly in peri membranous VSDs (PmVSDs), which comprise about 70-80% of congenital cases. The Amplatzer family of occluders, introduced in the early 2000s, marked a turning point with self-expanding nitinol meshes that conformed to defect anatomy. However, initial trials, like those for the Amplatzer Membranous VSD Occluder, were halted due to AV block rates exceeding 5-10%. This setback spurred device refinements, focusing on softer materials, asymmetric designs, and reduced radial force to minimize conduction tissue compression.

Recent years have seen a surge in novel devices tailored for percutaneous deployment. The KONAR-Multifunctional (MF) VSD occluder, developed by Lifetech Scientific, stands out as a versatile, CE-marked implant made from nitinol with a PTFE membrane to promote rapid endothelialisation. Its multifunctional design allows for closure of various VSD types, including PmVSDs, muscular defects, and even post-MI ruptures, with a low-profile sheath (6-9 Fr) suitable for paediatric patients. Studies from 2023-2025 report success rates of 95-100% in cohorts of 8-40 patients, with no major complications like embolization or permanent AV block in short-term follow-up. For instance, a 2025 case series demonstrated its efficacy in eight patients, highlighting ease of retrograde deployment and minimal residual shunts. Similarly, the Occlutech VSD occluder, with its braided nitinol structure, has shown promise in PmVSD closure, achieving 96.4% success in a 2025 trial of 28 children, with only trivial residual leaks at six months.

Abbott's Amplatzer line continues to evolve, with the Post-Infarct Muscular VSD Occluder featuring wider discs and a longer waist for friable post-MI tissue. A 2025 registry analysis of 18 post-MI cases reported 88% procedural success, though 30-day mortality remained high at 22% due to underlying cardiogenic shock. For congenital muscular VSDs, the Amplatzer Vascular Plug II (AVP-II) has been repurposed in double-device strategies, as seen in a 2023 study where it was nested inside a muscular occluder for large defects, yielding complete closure without embolization. Off-label use of patent ductus arteriosus (PDA) occluders, like the Amplatzer Duct Occluder II (ADO-II), has also expanded, particularly for small PmVSDs, with a 2024 meta-analysis confirming low AV block rates (1-2%) and high efficacy in infants.

Technique innovations are equally transformative, addressing limitations in access and radiation exposure. Traditional antegrade approaches via arteriovenous loops have given way to retrograde methods, which simplify deployment by accessing the defect from the left ventricle through the aortic valve. A 2025 Ukrainian study compared retrograde versus antegrade in 50 patients, finding shorter procedure times (120 vs. 180 minutes) and fewer vascular complications with the former. Trans jugular approaches offer an alternative for complex post-MI ventricular septal ruptures (VSR), as evidenced by a 2025 case report of successful closure in a high-risk elderly patient unsuitable for surgery. Hybrid strategies combine percutaneous elements with mini-thoracotomy or periventricular access, ideal for infants under 10 kg where femoral vessels are too small. A 2024 trial of 20 infants reported 95% success with hybrid closure, minimizing fluoroscopy and enabling direct visualization via transoesophageal echocardiography (TEE).

Radiation-free techniques are gaining ground, with zero-contrast methods guided solely by TEE or intracardiac echocardiography. A 2025 Frontiers study on doubly committed sub arterial VSDs (DCSA-VSD) demonstrated feasibility in adults, avoiding nephrotoxic contrast and reducing procedural risks in renal-impaired patients. Balloon calibration, once routine, is now

selective, reserved for irregular defects to size devices accurately without overinflation risks.

Clinical outcomes from recent studies underscore percutaneous closure's viability. A 2025 meta-analysis of 273 post-MI VSD closures showed 89% device implantation success, but 32% in-hospital mortality, highlighting the procedure's role as a bridge in unstable patients. For congenital cases, a 2025 Frontiers trial on peri membranous VSDs in 28 children reported 100% success with no vascular complications or mortality, and rapid left ventricular normalization post-procedure. Long-term data from a 2023 BMC Paediatrics study on 75 paediatric closures using various devices (Nit-Occlud, Hyperion, Amplatzer) revealed 100% survival at 5 years, with residual shunts resolving in 84% by one year. Comparative trials favour percutaneous over surgery in select groups; a 2023 Egyptian study found similar efficacy but shorter ICU stays (1.5 vs. 4 days) and fewer transfusions with transcatheter methods.

Despite progress, challenges persist. Post-MI VSDs carries high mortality due to tissue fragility, with device embolization in 5-10% of cases. AV block remains a concern, though newer soft devices have reduced it to <1% in PmVSDs. Residual shunts occur in 10-15% initially but often close spontaneously. Patient selection is crucial: defects >15 mm or with inadequate rims are poor candidates. Future directions include bioabsorbable occluders to eliminate long-term foreign body risks and AI-assisted imaging for precise deployment. Ongoing trials, like those evaluating KONAR-MF in multi-centre settings, aim to refine indications for adults and post-MI scenarios.

In conclusion, percutaneous VSD closure is evolving rapidly, offering safer, less invasive options that "plug the gap" left by surgery. With devices like KONAR-MF and techniques minimizing radiation and access trauma, outcomes are improving across age groups. While not universal, these advancements promise broader adoption, potentially shifting paradigms in structural heart interventions. Continued research will solidify its role, ensuring better quality of life for VSD patients worldwide.

Suggested Readings

1. Sadiq M, Qureshi AU, Younas M, Arshad S, Hyder SN. Percutaneous closure of ventricular septal defect using LifeTechTM Konar-MF VSD Occluder: initial and short-term multi-institutional results. Cardiology in the Young. 2022;32(5):755-761.
2. Haddad RN, Daou LS, Saliba ZS. Percutaneous closure of restrictive-type peri membranous ventricular septal defect using the new KONAR multifunctional occluder: Midterm outcomes of the first Middle-Eastern experience. Catheter Cardiovasc Interv. 2020;1;96: E295-E302.
3. Tanidir IC, Baspinar O, Saygi M, et al. Use of Lifetech™ KONAR-MF, a device for both peri membranous and muscular ventricular septal defects: A multicentre study. Int J Cardiol. 2020;1; 310:43-50.
4. Penny DJ, Vick GW. Ventricular septal defect. Lancet. 2011; 377:1103–1112.

PEDIATRIC HEART FAILURE – 'AN OVERVIEW'

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Introduction

Pediatric heart failure (HF) is a complex, multifaceted clinical syndrome characterized by the heart's inability to meet the metabolic demands of a growing child due to structural or functional cardiac abnormalities. Unlike in adults, where heart failure predominantly results from ischemic cardiovascular disease and hypertension, pediatric HF is most often attributable to congenital heart defects (CHDs), cardiomyopathies, myocarditis, and, less frequently, acquired conditions such as rheumatic heart disease or chemotherapy-induced cardiotoxicity.

Children undergo continuous somatic growth, neurodevelopment, and cardiovascular maturation. As a result, the underlying etiologies, pathophysiology, diagnostics, therapeutic approaches, and prognosis of pediatric HF are unique. Recognition of these distinctions has fostered an increasing awareness among healthcare providers about the need for pediatric-specific strategies in diagnosis and management, as well as family-centered, multidisciplinary care approaches that support both the child and their caregivers.

This report explores the epidemiology and prevalence, etiology and pathophysiology, clinical characteristics, diagnostic approaches, therapeutic options, challenges, and future directions for pediatric heart failure. It highlights the vital differences between pediatric and adult populations, with a focus on the implications for clinical care and support systems.

Epidemiology and Prevalence

Global burden of pediatric heart failure is challenging to estimate due to variations in definitions, underreporting, and divergent healthcare infrastructures. Nevertheless, estimates suggest that roughly 35,000 children under the age of 19 are affected annually in the United States, with 11,000–14,000 hospitalizations for HF per year. In high-income nations, the reported prevalence varies between 0.87 and 7.4 per 100,000 children. Recent analyses indicate a global prevalence of 56.2 per 100,000 in 2019, with burden highest in children under 10 years, males, and those with congenital heart defects or cardiomyopathy. Hospitalization for HF in children is associated with longer hospital stays, higher costs, and greater risk of mortality and readmission compared to their non-HF peers.

Etiology varies with geography and socioeconomic status. In high-income countries, most HF cases are linked to congenital cardiac anomalies or primary myocardial disorders, while lower- and middle-income countries continue to report significant numbers secondary to rheumatic heart disease and infections like myocarditis. These differences have direct implications for prevention, early detection, and care strategies.

Etiological Spectrum

Pediatric heart failure is etiologically diverse, with causes often age-dependent:

Infants and Early Childhood

- Congenital heart defects (CHDs): CHDs are the leading cause of pediatric HF, accounting for over half of cases in the first year of life. Lesions such as large ventricular septal defects (VSD), patent ductus arteriosus (PDA), atrial septal defects (ASD), coarctation of the aorta, transposition of the great arteries (TGA), and more complex structural anomalies can precipitate HF via pressure and volume overload, pulmonary over-circulation, or inadequate systemic output.
- Myocarditis: Viral or immune-mediated myocarditis is a significant cause, especially in infants and toddlers, with potential for rapid decompensation.

Older Children and Adolescents

- Cardiomyopathies: These include dilated (DCM), hypertrophic (HCM), restrictive, and arrhythmogenic right ventricular cardiomyopathy (ARVC). In many cases, cardiomyopathies have genetic underpinnings.
- Acquired causes: These are rarer, including rheumatic heart disease, chemotherapy toxicity, endocarditis, arrhythmias, metabolic or mitochondrial disorders, and systemic conditions leading to high-output or demand-induced failure (e.g., severe anemia or sepsis).

Age-dependent variations are reflected in studies indicating CHDs predominate until the age of five, myocarditis is more frequent in toddlers and young children, while rheumatic carditis and inherited cardiomyopathies are more common in older children and adolescents.

Pathophysiological distinctiveness

The pediatric myocardium differs at molecular, cellular, and physiological levels:

- Immature cardiomyocytes: Children's hearts, especially neonates, rely on glucose metabolism, are calcium-dependent rather than reliant on sarcoplasmic reticulum cycling (as in adults), and maintain a high regenerative capacity in the first year of life.
- Less fibrosis and remodelling: Pathological cardiac remodelling, including interstitial fibrosis, is less pronounced before adolescence, which may impact response to therapies conventionally successful in adult

HF.

- Cardiac regeneration: Neonatal and infant hearts possess demonstrable endogenous regenerative capacities via resident cardiac progenitor and stem cells; a feature rapidly lost with age.
- Neurohormonal milieu: The expression, regulation, and downstream signalling of β-adrenergic and angiotensin pathways differ, influencing pharmacotherapeutic responses.

Clinical Characteristics and Presentation

Spectrum of Presentation

Clinical manifestations are highly variable and can be subtle, especially in nonverbal patients. The classic adult symptoms—peripheral edema, orthopnea, overt pulmonary congestion—are often absent or difficult to recognize in infants and young children. The most commonly reported features:

Neonates and Infants:

• Poor feeding or prolonged feeding time ("suck-rest-suck cycles")
• Failure to thrive or poor weight gain
• Tachypnea, laboured breathing, retractions
• Diaphoresis, especially during feeding
• Lethargy, irritability
• Recurrent chest infections, cough
• Hepatomegaly

Toddlers and Older Children:

• Exercise intolerance or breathlessness with activity
• Fatigue, decreased endurance
• Swelling of the face, eyelids, abdominal distension, peripheral edema
• Frequent respiratory infections
• Growth retardation

Adolescents:

• Symptoms may more closely mimic adult presentations: overt dyspnea, palpitations, orthopnea, decreased exercise tolerance, and signs of end-organ dysfunction.

Red flag signs include reduced urine output, cold extremities, altered sensorium, and evidence of end-organ dysfunction—suggesting advanced or decompensated states. Importantly, "compensated" HF may manifest only with subtle findings (e.g., feeding difficulties, poor growth). Thus, maintaining a high index of suspicion in at-risk populations is essential.

Classification Tools

Adult-oriented tools like the New York Heart Association (NYHA) classification inadequately capture pediatric HF severity. Pediatric-specific systems include:

• Modified Ross Classification: Quantifies symptoms such as tachypnea, feeding tolerance, diaphoresis, and growth failure. Useful for infants and young children.
• New York University-Pediatric Heart Failure Index (NYU-PHFI): Utilizes weighted physiologic and therapy-based scoring.
• Staging based on hemodynamic status: Categorization as "wet/dry" and "cold/warm" is increasingly advocated for advanced management and transplant listing.

Clinical Evaluation

• Physical examination: Assessment for signs of respiratory distress, hepatomegaly, peripheral edema, abnormal heart sounds (gallop rhythms), murmurs, and signs of poor perfusion.

Diagnostic Challenges

Pediatric diagnostic cut-offs and normal reference values are often lacking or based on adult studies, leading to diagnostic uncertainty. Interpretation of many laboratory results and imaging findings must be age-, sex-, and context-specific.

Laboratory Investigations

Biomarkers:

• Brain natriuretic peptide (BNP) and N-terminal proBNP (NT-proBNP):
o Elevated in heart failure, correlate with severity and risk stratification. Age-specific thresholds and reference ranges are essential due to higher baseline levels in infants and varying normal ranges with age and sex.
o Helpful in distinguishing cardiac from primary respiratory causes in patients with dyspnea or respiratory distress.
• Cardiac troponins (cTnI and cTnT):
o Useful for identifying ongoing myocardial injury, especially in myocarditis or perioperative settings. Cut-offs are lower in children, with age- and assay-specific ranges.

Additional Tests:

• Complete blood count, electrolytes, renal/liver function: For detecting contributing or complicating systemic conditions (e.g., anemia, renal or hepatic dysfunction).
• Inflammatory markers (CRP), thyroid function, viral panels: Where indicated by clinical context.

Electrocardiogram (ECG):

• Essential for detection of arrhythmias, chamber enlargement patterns, evidence of ischemia or conduction defects (e.g., Wolff-Parkinson-White, heart block).

Imaging

Chest X-ray: Evaluates cardiac size (cardiomegaly), pulmonary vasculature, and evidence of pulmonary edema.

Echocardiography:

• It is the cornerstone test for structural and functional diagnosis. Identifies CHDs, assesses systolic and diastolic function, valve disease, chamber dimensions, shunt lesions, pericardial effusion, estimation of pulmonary artery pressures, and evaluation of ventricular synchrony.
• Special consideration for challenging geometries (e.g., single ventricle physiology or after surgical repairs) may require 3D echo and cardiac MRI for additional information.

Cardiac MRI:

• Valuable for tissue characterization (fibrosis, edema), quantification of chamber function, and complex anatomy. May require sedation in young children.

Cardiac Catheterization:

• Reserved for complex or unclear diagnoses, precise hemodynamic assessment, or therapeutic interventions.

Next-generation diagnostics:

• Genetic testing: Next-generation sequencing panels are being used for cardiomyopathies and arrhythmia syndromes,

informing both individualized care and family counselling.

Emerging biomarkers

• Emerging biomarkers: Soluble ST2, galectin-3, growth differentiation factor-15, atrial natriuretic peptide, and MR-proADM present diagnostic or prognostic promise, especially in single ventricle physiology and complex CHD, but data are inconclusive in large, diverse pediatric populations.

Accordingly, future large-scale pediatric studies are needed to standardize cut-offs and refine biomarker-based risk stratification.

Medical Treatment Options and Pharmacotherapy

Despite clear pathophysiologic rationales, most pharmacologic therapies for pediatric heart failure are extrapolated from adult trials, as robust pediatric data are lacking. Nonetheless, contemporary management utilizes a combination of symptom-relieving and disease-modifying agents. Dosing is highly individualized, guided by age, renal and liver function, and developmental pharmacodynamics.

Cornerstones of Pediatric HF Pharmacotherapy

1. ACE Inhibitors (ACEIs)

• Used to reduce afterload, modulate RAAS, improve symptoms/growth in volume/pressure overload lesions, idiopathic DCM, and after surgical palliation or repair.

• Dosing and susceptibility to adverse effects (hypotension, renal dysfunction, hyperkalemia) require careful titration, particularly in infants.

2. Beta-Blockers

• Bisoprolol (β1-selective) is preferred for children; carvedilol and metoprolol are used, though pediatric trials (e.g., carvedilol in DCM) yielded mixed results.

• Beneficial for inhibition of chronic sympathetic activation, but require thoughtful titration and are best applied once the patient is volumetrically optimized.

3. Mineralocorticoid Receptor Antagonists (MRA)

• Spironolactone and eplerenone provide anti-fibrotic properties and potassium-sparing diuresis.

4. Diuretics

• Loop (furosemide) and thiazide diuretics relieve congestion, reduce preload, and are part of acute management. Long-term use is minimized given risk for neurohormonal activation and electrolyte disturbances.

5. Cardiac Glycosides (Digoxin)

• Beneficial for symptom control and contractility augmentation, especially in infants and those with atrial arrhythmias. Use is limited by narrow therapeutic window.

6. Advanced Pharmacotherapies

• Ivabradine: If-channel inhibitor approved for use in pediatric DCM.

• Angiotensin Receptor-Neprilysin Inhibitor (ARNI): Sacubitril/valsartan is emerging as a promising therapy in pediatric patients.

• SGLT2 inhibitors: Initial safety and efficacy data in older children are promising, but widespread pediatric use awaits more robust evidence.

7. Inotropes and Vasodilators

• Medications such as dobutamine, milrinone, dopamine, adrenaline, and nitroprusside are reserved for acute or decompensated HF.

Mechanical Circulatory Support and Surgical Interventions

Some children progress despite optimal medical therapy, necessitating mechanical or surgical interventions:

Mechanical Circulatory Support (MCS)

Ventricular Assist Devices (VADs):

• Major advancements include Berlin Heart EXCOR, HeartMate III, and the Syncardia Total Artificial Heart, enabling bridge-to-transplant, bridge-to-recovery, or, rarely, destination therapy.

• VADs are increasingly used as a bridge to transplantation or even as an alternative for children ineligible for transplantation.

• These devices are now available in sizes suitable for neonates to adolescents. Berlin Heart EXCOR has shown survival rates of approximately 80% to transplant, dramatically improving outcomes for children with end-stage heart failure.

• Major complications include bleeding and thrombosis requiring personalized anticoagulation protocols, infection, neurologic injury, and device malfunction.

Extracorporeal Membrane Oxygenation (ECMO):

• Used in acute, severe decompensated heart failure or perioperatively, although outcomes are generally inferior to VADs for longer-term support.

Cardiac Transplantation:

• Remains the definitive therapy for end-stage, medically refractory heart failure in children.

• 1-year survival is now >90% in pediatric cardiac transplant recipients, although median waitlist times and mortality remain significant, especially for infants and those with congenital heart disease.

Surgical Interventions

• Direct surgical correction or palliation of CHDs (e.g., VSD, coarctation, transposition) can cure or substantially ameliorate heart failure when timely performed.

Regenerative Therapies

• Stem cell and regenerative medicine approaches hold unique promise in pediatrics due to developmental regenerative reserves. Early clinical studies using cell-based therapy (e.g., cardiac progenitor cells, bone-marrow mononuclear cells) suggest transient improvement in cardiac function, primarily in hypoplastic left heart syndrome and DCM, but wider data are needed before routine adoption.

Prognosis and Outcomes in Pediatric HF

Outcomes in pediatric HF are highly variable, determined mainly by cause, age at presentation, comorbidities, and access to specialized care:

• Congenital Heart Disease: Prognosis has improved substantially due to advancements in surgical and perioperative care, but late HF remains a risk, especially in single ventricle physiology or post-palliation (e.g., Fontan).

• Dilated Cardiomyopathy: Mortality remains high (as much as 20–30% at one year), with limited long-term survival

improvement despite modern pharmacotherapy.

Evidence Gaps and Therapeutic Limitations

• Most pharmacologic therapies are based on adult data and used off-label in children. Only a handful of randomized controlled trials exist in pediatric HF, typically underpowered due to small, heterogenous populations.

• Lack of clear evidence for optimal dosing and long-term benefits.

• Difficulties in recruiting patients and standardizing outcome measures.

Pediatric-Specific Considerations

• Age-, size-, and development-appropriate drug formulations and device sizes are often unavailable or limited.

• Complex and variable underlying anatomy post-CHD repair or palliation complicates treatment strategies.

• Growth and development necessitate continuous adjustment of therapy.

• Adverse event risks for devices (bleeding, thrombosis, infection) are higher; unique hemostatic profiles in children interfere with direct extrapolation of adult protocols.

Multidisciplinary Care and Healthcare Provider Implications

The standard of care for pediatric heart failure is shifting toward integrated, multidisciplinary, family-centered team approaches. Programs orient toward early discharge, family/caregiver education, rehabilitation, and transition planning for survivors.

• Pediatric Cardiology and Cardiac Surgery: Oversee diagnosis, advanced management, post-surgical care.

• Heart Failure Nurses and Advanced Practitioners: Guide symptom monitoring, medication titration, and family education.

• Pharmacy: Ensure safe, age-appropriate, compliant prescribing and monitoring.

• Dietitians and Feeding Specialists: Address nutritional support crucial for recovery and developmental progress.

• Physical, Occupational, and Speech Therapists: Aid in maintaining or restoring function and developmental milestones.

• Social Work and Psychology: Provide critical support for chronic illness adaptation, adherence, and psychological resilience.

• Genetic counselling: Vital for cases of familial or syndromic disease.

Such teams improve detection, early intervention, and adherence, as well as health-related quality of life, coordination of care, and rapid response to decompensation.

Family-centered Care and Caregiver Support

Pediatric heart failure places a unique and enduring burden on families and caregivers, demanding substantial ongoing involvement in care, symptom monitoring, **medication management, and emotional support.**

Challenges for families and caregivers include:

• **Emotional stress, uncertainty, and anxiety associated with** chronic illness management.

• Intensive at-home care needs, including feeding, medication administration, monitoring for signs of decompensation, and frequent medical appointments.

• Navigating the healthcare system for complex, multidisciplinary care.

• Financial stressors due to medical costs and time away from work.

• Impact on siblings and overall family function.

Future Directions and Emerging Research

The field of pediatric heart failure is rapidly evolving, with several promising avenues:

• Precision Medicine and Genomics: Next-generation genetic testing is identifying pathogenic mutations, enhancing diagnosis, risk stratification, and family screening, especially in cardiomyopathies.

• Regenerative Medicine: Progress in stem cell therapies and myocardial recovery, particularly in younger patients with retained regenerative ability, may reduce reliance on transplantation in select populations.

• Pediatric-Specific Drug and Device Development: Ongoing work is focused on formulating age-appropriate medications, advancing pediatric VAD design, and integrating technology for remote monitoring and early intervention.

• Biomarkers and Imaging: Research aims to refine the utility of biomarkers (NT-proBNP, troponins, GDF-15, galectin-3) and advanced imaging for earlier detection, risk stratification, and personalized management.

Conclusion

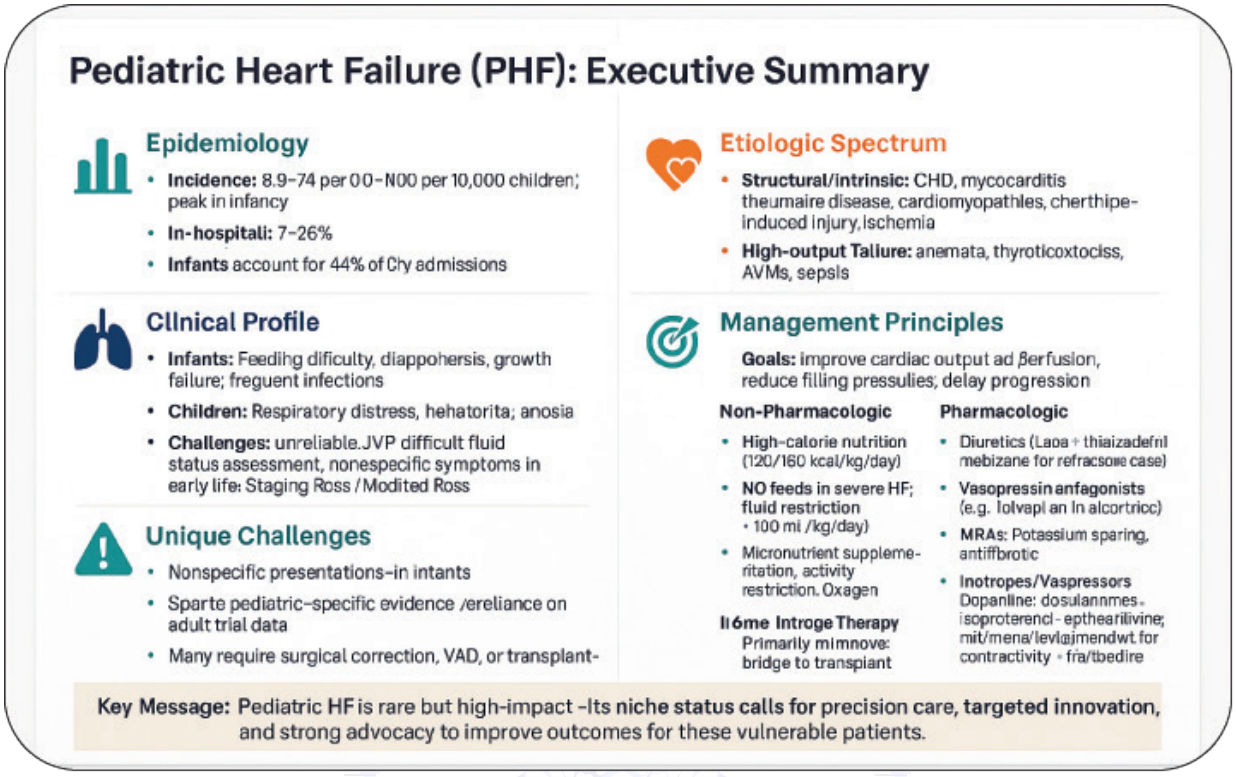
Pediatric heart failure remains a distinct and highly challenging domain within cardiology. The etiologies, presentations, diagnostic strategies, prognostic indicators, and therapeutic responses differ substantially from adult HF. These differences necessitate dedicated research, tailored clinical care, and policy interventions. Recognizing children as a unique population—rather than "small adults"—is essential, both for advancing science and for providing compassionate, effective family-centered care.

Healthcare providers and caregivers must be alert to age-specific presentations, advocate for multidisciplinary and personalized care, and support the child's holistic development and psychosocial well-being. Continued investment in pediatric-specific science, caregiving support, and innovative therapies will be vital to realizing the promise of a healthier future for this most vulnerable population.

Suggested reading

1. Yuan Y, Pan B, Wang F, et al. Age-Based Classification and Outcomes in Pediatric Heart Failure: Findings From a Retrospective Multicenter Cohort Study. J Am Heart Assoc. 2025;14(4):e038129. doi:10.1161/JAHA.124.038129
2. Rossano, J., Lin, K.Y., Edwards, J.J. (2023). Diagnosis and Management of Heart Failure in Children. In: Abdulla, Ri., et al. Pediatric Cardiology. Springer, Cham. https://doi.org/10.1007/978-3-030-42937-9_80-1

3. Ishigami S, Sano T, Krishnapura S, Ito T, Sano S. An overview of stem cell therapy for paediatric heart failure. Eur J Cardiothorac Surg. 2020;58(5):881-887. doi:10.1093/ejcts/ezaa155
4. Lai ZH, Liu ZY, Xie J, et al. Global burden of heart failure in children and adolescents from 1990 to 2019: an analysis from the Global Burden of Disease Study 2019. World J Pediatr. 2025;21(1):90-99. doi:10.1007/s12519-024-00868-1
5. McGinn C, Casey FA, Watson C, Morrison L. Paediatric heart failure - understanding the pathophysiology and the current role of cardiac biomarkers in clinical practice. Cardiol Young. 2023;33(4):503-513. doi:10.1017/S1047951123000331
6. Recla S, Schmidt D, Logeswaran T, Esmaeili A, Schranz D. Pediatric heart failure therapy: why β1-receptor blocker, tissue ACE-I and mineralocorticoid-receptor-blocker?. Transl Pediatr. 2019;8(2):127-132. doi:10.21037/tp.2019.04.08
7. Kitko L, McIlvennan CK, Bidwell JT, et al. Family Caregiving for Individuals With Heart Failure: A Scientific Statement From the American Heart Association. Circulation. 2020;141(22):e864-e878. doi:10.1161/CIR.0000000000000768
8. Das BB. Current State of Pediatric Heart Failure. Children (Basel). 2018;5(7):88. Published 2018 Jun 28. doi:10.3390/children5070088
9. Momma K. ACE inhibitors in pediatric patients with heart failure. Paediatr Drugs. 2006;8(1):55-69. doi:10.2165/00148581-200608010-00005
10. Shin YR, Park YH, Park HK. Pediatric Ventricular Assist Device. Korean Circ J. 2019;49(8):678-690. doi:10.4070/kcj.2019.0163



CORONARY ARTERY DISEASE IN YOUNG

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Coronary artery disease (CAD) in the young is an emerging and challenging field in our country. Unlike in Western populations, where CAD typically presents later in life, Indian patients often manifest the disease 10–15 years earlier, and the overall prevalence in the young population is estimated at 15–20%. Premature CAD is defined when a patient presents before the age of 45 years. The spectrum of clinical presentations is wide, ranging from sudden cardiac death (SCD) to diffuse triple-vessel disease, where coronary artery bypass grafting (CABG) can be a technically demanding and high-risk procedure. The consequences of premature CAD are not only personal but societal, as they affect individuals in the prime of their productive lives, causing a significant socio-economic burden.

Clinically, we often encounter missing links when we compare Indian patients to Western literature. Traditional risk factors such as smoking, diabetes, hypertension, and dyslipidemia certainly play a role, but they do not fully explain the occurrence of acute coronary syndrome (ACS) in many young patients. While many of these patients are smokers or hypertensive, only a few have diabetes at the time of presentation, and for those who are diabetic, the disease duration is often less than five years. This suggests that additional factors, including lifestyle, environmental exposures, and genetic predispositions, may contribute significantly to the early onset of CAD in this population.

One emerging consideration is the impact of occupational stress and sleep disruption.Studies have highlighted the role of obstructive sleep apnea and disturbed sleep patterns in promoting ACS. In India, many young professionals work in international companies or call centers with irregular hours and night shifts, which can disturb circadian rhythms. Chronic sleep deprivation and circadian misalignment can result in increased sympathetic activity, insulin resistance, and endothelial dysfunction, all of which contribute to early atherosclerosis. The psychological burden of stress, job insecurity, and pressure to meet deadlines also manifests clinically. Young patients frequently present with mood irritability, palpitations, and tachycardia, reflecting the physiological consequences of chronic stress on the cardiovascular system.

Post-COVID cardiovascular effects represent another emerging concern. A subset of young COVID survivors exhibits chronic low-grade inflammation, which can induce endothelial dysfunction, platelet activation, and a pro-thrombotic state. Identifying these patients and monitoring them for future cardiac events is crucial, as they may be at heightened risk for ACS despite appearing healthy otherwise.

Family and genetic history remain fundamental components of risk assessment in young CAD. Every young patient should be evaluated for a family history of premature CAD, specifically in the father (before 55 years), mother (before 65 years), and siblings. Clinical examination should include careful observation for xanthomas, xanthelasmas, and corneal arcus, which may indicate underlying dyslipidemia. The typical Indian pattern of dyslipidemia includes elevated triglycerides (TG) and very-low-density lipoprotein (VLDL) levels, often accompanied by mildly increased low-density lipoprotein (LDL). However, conventional lipid panels fail to differentiate the qualitative fractions of LDL. Small dense LDL particles are particularly atherogenic and should be identified, along with evaluating the apoB/apoA1 ratio. Screening family members, including parents and siblings, can help identify clusters at risk for premature CAD or sudden cardiac events such as NSTEMI/STEMI.

Substance abuse is another underestimated contributor in young CAD due to social stigma. Drug addiction, including use of cocaine, LSD, and other recreational drugs, is increasingly observed among the middle-class and B-grade population. These substances can precipitate coronary vasospasm, acute MI, and SCD due to malignant ventricular arrhythmias. Beyond drugs, lifestyle factors such as metabolic syndrome, physical inactivity, mobile addiction, and irregular late-night meals exacerbate genetic vulnerabilities to CAD.

Genetic thrombophilic disorders are also relevant. Hyperhomocysteinemia, protein C and protein S deficiencies, and elevated fibrinogen levels increase the risk of arterial and venous thrombosis. Hyperhomocysteinemia, in particular, can result in deep vein thrombosis, stroke, and MI. Another important hereditary risk factor is lipoprotein(a) [Lp(a)], which is often elevated in young CAD patients and resistant to conventional therapy, including diet, exercise, and statins. The recent availability of PCSK9 inhibitors, such as Inclisiran, offers a therapeutic option for patients with elevated Lp(a).

Environmental and lifestyle exposures further contribute to early CAD. Air pollution, particularly PM2.5 particles, has

been shown to induce systemic inflammation and endothelial injury. Young individuals commuting through heavily trafficked areas inhale particulate matter and carbon particles regularly, which can accelerate atherosclerosis. Interestingly, both physical inactivity and excessive exercise may increase cardiovascular risk through pro-thrombotic mechanisms, highlighting the importance of balanced activity.

The rise of electronic smoking is another modern concern. Many young people perceive e-smoking as trendy or safer than conventional smoking. However, e-cigarettes introduce heavy metals, such as tungsten and cadmium, from heated batteries into the lungs, which can be carcinogenic and harmful to the cardiovascular system. Furthermore, e-smoking may serve as a gateway to traditional cigarette use, establishing a dual pathway of risk.

Inflammatory processes play a critical role in the pathogenesis of young CAD. Elevated levels of interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hsCRP) are commonly observed in young patients and contribute to endothelial dysfunction, plaque instability, and acute coronary events. Diet is another important modifiable factor. Reuse of cooking oils and repeated reheating lead to high concentrations of trans fatty acids, which are abundant in Indian street foods such as samosas, bhaturas, and bread pakoda. Frequent consumption of such foods significantly increases cardiovascular risk in the young population.

These are angiograms of a few of my patients who presented with ACS:

1. Increased Lp(a)
2. Increased Homocysteine
3. Familial Hypercholesterolemia

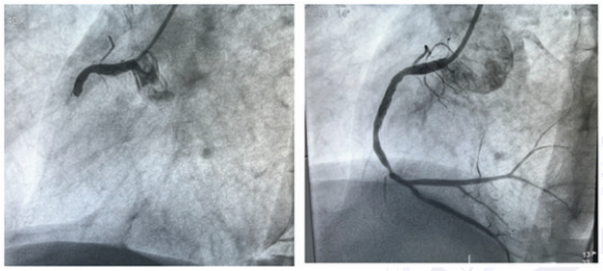

Figure 1: Angiogram of a patient with increase Lp(a):

A 35 year old male diagnosed as CAD/IWMI/CHB. The angiogram (LAO cranial view) on the left shows proximal RCA total occlusion. And the angiogram (LAO cranial view) on the right shows post PTCA in proximal RCA with TIMI flow 3.

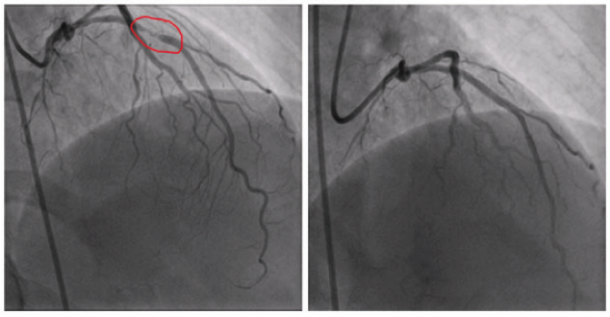

Figure 2: Angiogram of a patient with Hyper homocysteinemia:

A 30 year old male diagnosed as CAD/NSTEMI. The angiogram (RAO cranial view) on the left shows proximal LAD 90% stenosis. And the angiogram (RAO cranial view) on the right shows post PTCA in LAD with TIMI flow 3.

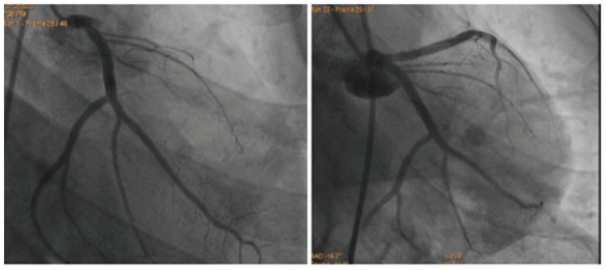

Figure 3: Angiogram of a patient with Familial Hypercholesterolemia:

A 22 year old male presented with ACS/NSTEMI and DVD. The angiogram (RAO caudal view) on the left shows osteal LAD total occlusion. And the angiogram (RAO caudal view) on the right shows post PTCA in LAD with TIMI flow 3.

In our clinical experience, angiographic findings reinforce these observations. Among young ACS patients, we have identified cases with increased Lp(a), elevated homocysteine, and familial hypercholesterolemia, underscoring the multifactorial nature of early CAD in India. Through the forum of mid-term UP CSI, I want to emphasize that we should have our own registry to study young CAD patients so as to identify novel risk factors and treat them better, rather than being wholly dependent on Western literature. We cannot imagine a young and apparently healthy person dying suddenly due to MI in the prime of his life and career. This is a huge loss to the nation. We cannot imagine the anguish of the family that loses a son/husband and a father.

Our department is planning a comprehensive project on Young CAD. The study aims to investigate this unique cohort systematically, identifying novel genetic, metabolic,

environmental, and lifestyle-related risk factors. These young individuals, although apparently healthy, may unknowingly harbor significant cardiovascular risks—a "cobra" in their hearts—posing a threat of sudden and catastrophic events. By creating awareness, performing detailed evaluations, and initiating targeted interventions, we hope to reduce premature mortality, improve quality of life, and mitigate the national burden of young CAD.

Suggested readings

1. Singh M, Kumar R, Kansal V, Singhal A, Kapoor A, Singh G. Young onset coronary artery disease in India: A descriptive observational study. Clinical Epidemiology and Global Health. 2024 Nov 1;30:101851.
2. Juan-Salvadores P, Jiménez Díaz VA, Iglesia Carreño C, Guitián González A, Veiga C, Martínez Reglero C, Baz Alonso JA, Caamaño Isorna F, Iñiguez Romo A. Coronary artery disease in very young patients: analysis of risk factors and long-term follow-up. Journal of Cardiovascular Development and Disease. 2022 Mar 11;9(3):82.
3. Michos ED, Choi AD. Coronary artery disease in young adults: a hard lesson but a good teacher. Journal of the American College of Cardiology. 2019 Oct 15;74(15):1879-82.
4. Kher M, Sathyanarayan BC. Coronary artery disease in young females: current scenario. Indian Journal of Cardiovascular Disease in Women WINCARS. 2017;2(03):039-43.
5. Guevara A. CORONARY ARTERY DISEASE IN A YOUNG ADULT: UNVEILING THE INTRICATE INTERPLAY OF RISK FACTORS. American Journal of Preventive Cardiology. 2024 Sep 1;19:100838.

HEART FAILURE IN PREGNANCY: A FOCUSED UPDATE

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Heart failure complicates 11% of pregnancies in women with pre-existing heart disease and has an in-hospital maternal mortality rate of 9%. Various risk scoring systems may be used for risk assessment in pregnant patients with heart disease. For women with HF, the 2 most relevant risk scores are the Cardiac Disease in Pregnancy II risk score7 and the mWHO (modified World Health Organization Classification of Maternal Cardiovascular Risk). The recent 2025 ESC guidelines recommend using the modified WHO classification for risk stratification.

mWHO ranks women from classes I to IV, where class I patients are expected to have similar risk as the general population, and class IV patients are at such high risk for complications that pregnancy is contraindicated. Mild impairment in left ventricular systolic function, hypertrophic cardiomyopathy (HCM), and history of HT fall into classes II and III, whereas patients with severe LV dysfunction, defined as left ventricular ejection fraction (LVEF) < 30%, and those who are in NYHA functional class III or IV are considered mWHO class IV. Additionally, women with known PPCM from prior pregnancies with any residual impairment of LV systolic function are similarly classified as mWHO class IV.

Symptoms and signs of HF during pregnancy may be difficult to appreciate because shortness of breath and lower extremity oedema are commonly present in normal pregnancy. However, if accompanied by chest pain, cough,

orthopnea, paroxysmal nocturnal dyspnea, hypertension or hypotension, tachycardia, or syncope, prompt cardiovascular evaluation is recommended. When HF is suspected, usual confirmatory testing should be done, including electrocardiography, echocardiography, and laboratory assessment, including natriuretic peptide levels. Higher N-terminal pro–brain-type natriuretic peptide levels are expected during pregnancy even in the absence of hypertension and cardiac disease. However, N-terminal pro–brain-type natriuretic peptide levels >200 pg/mL should raise concern for combined HF and pre-eclampsia.

Two peaks of HF deterioration occur in pregnancy: at 23–30 weeks and peri-delivery. Pre-conception counselling should include a discussion of management if there is a clinical deterioration during the first peak. Early delivery due to maternal cardiac deterioration will impact foetal outcomes. Patients with mild ventricular dysfunction may tolerate pregnancy with no increase in symptoms. However, those with worse than mild ventricular dysfunction (mWHO 2.0 class >II) require expert care from the Pregnancy Heart Team, with additional input from the advanced HF team, including transplant and mechanical circulatory support experts.

Women with pre-existing severe HF (LVEF <30%, mWHO 2.0 class IV) are at high risk of maternal morbidity and mortality and account for up to 15% of maternal deaths globally. Assessment of patients with pre-existing HF includes regular assessment of symptoms, echocardiography, and NP at intervals determined by the severity of HF and other non-cardiac issues.

HF in pregnancy can be treated with oral diuretics, β 1-selective beta-blockers (bisoprolol, metoprolol succinate), hydralazine, and oral nitrates. Diuretics (loop diuretics and thiazides if required) should be used with caution due to a potential reduction in uterine blood flow, but may be necessary in pulmonary congestion or echocardiographic signs of high LV end-diastolic pressure.

Loop diuretic agents can safely be used for decongestion. More real-world data exist regarding furosemide and bumetanide compared with torsemide and metolazone, which should be used cautiously. Digoxin is generally considered safe during pregnancy, but the physiological changes of pregnancy can affect its pharmacokinetics. Digoxin does cross the placenta, leading to lower maternal drug exposure; however, it is safe and has been used for the treatment of fetal tachyarrhythmias.

Beta-blockers should be initiated and gradually up-titrated to the maximum tolerated dose. Among the cardioselective beta-blockers that are effective in HF, metoprolol and bisoprolol are favoured. Less is known about carvedilol in pregnancy, although anecdotally, clinical experience has increased. Although not typically used in patients with HF, atenolol has been associated with low birth weight and is not recommended during pregnancy. The ROPAC study demonstrated no detrimental effects in women treated with beta-blockers for structural heart disease in pregnancy. ACE-Is, ARBs,. ARNIs, MRAs, ivabradine, SGLT2 inhibitors, and atenolol are contraindicated during pregnancy due to adverse effects on the foetus. Hydralazine and nitrates combinations are safer to use in pregnancy.

CARDIOLOGICAL SOCIETY OF INDIA

U.P. CHAPTER