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Early Bioprosthetic Valve Failure: A Mini Review

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Abstract

Early Bioprosthetic Valve Failure (EBVF)-defined as valve dysfunction necessitating reintervention, causing severe haemodynamic compromise, or death within five years of implantation-is an increasingly recognized clinical syndrome of growing importance as Bioprosthetic Heart Valves (BHVs) are extended to younger, lower-risk populations. Unlike late Structural Valve Degeneration (SVD), EBVF encompasses a spectrum of distinct mechanisms including accelerated calcification, immune-mediated SVD, bioprosthetic valve thrombosis, Prosthetic Valve Endocarditis (PVE) and prosthesis-patient mismatch. The temporal phase of failure provides critical diagnostic guidance: procedural and anatomical causes dominate within 30 days, infective and thrombotic mechanisms predominate within the first year and accelerated structural degeneration emerges thereafter. A multimodality diagnostic strategy integrating transthoracic and transesophageal echocardiography, cardiac computed tomography, cardiac magnetic resonance and nuclear imaging (18F-FDG PET/CT) is essential. Treatment is mechanism-directed and planning for re-intervention-whether redo surgery or transcatheter Valve-In-Valve (ViV)-must incorporate lifetime valve management strategy, including future re-intervention feasibility. Anti-calcification tissue platforms, decellularized xenograft scaffolds and immune biomarker surveillance offer the prospect of earlier identification and risk-stratified follow-up. Systematic identification of EBVF and structured heart team decision-making are essential as the global BHV population expands.

Review Methods

This narrative mini-review was conducted in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) framework. A systematic search of PubMed/MEDLINE, EMBASE and the Cochrane Library was performed from inception to May 2025 using the following MeSH terms and free-text keywords: "bioprosthetic valve failure", "early structural valve degeneration", "bioprosthetic valve thrombosis", "prosthetic valve endocarditis", "TAVR durability", "valve-in-valve TAVR", and "patient-prosthesis mismatch". Reference lists of retrieved articles were hand-searched for additional relevant studies. Included sources comprised randomised controlled trials, prospective and retrospective observational studies, registry analyses, systematic reviews, meta-analyses and relevant clinical practice guidelines published in peer-reviewed journals. Case reports were included only where they described index adverse events of regulatory significance (e.g., the Trifecta safety communication). No formal quality assessment scoring was applied given the narrative design; however, study relevance, sample size and impact factor of the source journal were used to prioritise cited evidence. This review does not constitute a registered systematic review and no PROSPERO registration was undertaken.

Introduction

Bioprosthetic Heart Valves (BHVs) now account for the majority of valve replacements worldwide, driven by favorable hemodynamics, avoidance of lifelong anticoagulation and the expansion of Transcatheter Aortic Valve Replacement (TAVR) into intermediate- and low-risk populations [1,2]. Improvements in anti-calcification treatments, stentless and suture less designs, and tissue engineering have further broadened the cohort considered suitable for biological valves [3]. While late Structural Valve Degeneration (SVD) beyond ten years is the

dominant mode of failure, Early Bioprosthetic Valve Failure (EBVF)-defined as valve dysfunction necessitating reintervention, causing severe haemodynamic compromise, or death within five years of implantation-represents a distinct and increasingly recognized clinical syndrome [4]. The ten-year outcomes of the NOTION trial and the PARTNER 3 five-year data have confirmed the extension of TAVR into populations whose life expectancy substantially exceeds the expected durability of a single bioprosthetic, lending greater urgency to understanding early failure [2,5]. Contemporary registries suggest that 5-12% of BHVs develop clinically relevant dysfunction within five years, with 1-4% progressing to overt failure [4].

Mechanisms of early failure

EBVF is not a single pathology but a final common pathway for several distinct processes. Three interrelated mechanisms underpin accelerated SVD: dystrophic calcification, mechanical fatigue and immune-mediated rejection. Glutaraldehyde fixation, while reducing immunogenicity, paradoxically generates aldehyde groups that bind calcium ions and promote hydroxyapatite nucleation; macrophage and red blood cell infiltration further accelerates this process through iron-mediated oxidative injury [3,6]. Modern anti-calcification strategies-including alpha-amino oleic acid treatment (Mosaic), ethanol pretreatment (Hancock II), surfactant treatments (Trifecta) and the RESILIA tissue platform (Edwards Lifesciences)-address specific aspects of the calcification cascade, with the COMMENCE trial demonstrating freedom from SVD exceeding 99% at five years and encouraging seven-year data from the aortic cohort [7]. Whether these treatments fundamentally alter the trajectory of EBVF in the youngest recipients, where calcification is most aggressive, remains to be established.

The seminal work of Senage and colleagues in Nature Medicine (2022) identified circulating IgG antibodies against residual xenoantigens-notably galactose-α-1,3-galactose (α-gal) and N-glycolylneuraminic acid (Neu5Gc)-in the great majority of recipients, with antibody titres at three months post-implantation independently predicting subsequent SVD [8]. This finding has reframed SVD from a purely degenerative phenomenon into one with a substantial immunological dimension and has motivated efforts to develop α-gal-deficient porcine donors and decellularised scaffolds with reduced antigenicity. Younger age at implantation is the strongest demographic risk factor, with patients under 60 experiencing SVD at substantially greater rates due to higher cardiac output, more vigorous immune response and longer exposure. End-stage renal disease, hyperparathyroidism, diabetes mellitus, mitral position and valve-in-valve implantation all confer additional risk [4,6].

The influence of prosthetic design on EBVF is exemplified by the Trifecta valve experience. The externally mounted leaflet geometry-while providing a larger effective orifice area-exposed leaflets to direct contact and frictional abrasion against the stent posts, resulting in an unexpectedly high rate of early SVD within five to seven years, particularly in smaller sizes. The FDA issued a safety communication in February 2023, and Abbott subsequently withdrew the Trifecta and Trifecta GT valves from the US market in July 2023 [9]. This episode underscores the principle that hemodynamic optimization and durability are not independent goals, and that surveillance of new valve designs through the first five to seven years of clinical use is essential. In the transcatheter setting, crimping induces irreversible micro-injury to leaflet collagen, while asymmetric deployment distributes stress unevenly and creates regions of stagnation that predispose to thrombosis [10].

Bioprosthetic valve thrombosis ranges from subclinical leaflet thrombosis (hypoattenuated leaflet thickening, HALT)-detected in 10-15% of TAVR and up to 7% of surgical AVR recipients on cardiac CT-to clinically significant obstruction [11]. All three components of Virchow's triad contribute: surface damage through incomplete endothelialisation and residual glutaraldehyde, stasis in neo-sinus regions of flow recirculation and hypercoagulable states related to atrial fibrillation, malignancy, or recent surgery. Up to half of subclinical episodes resolve spontaneously without specific intervention, but chronic organized thrombus may evolve into fibrosis and calcification, converting what began as a thrombotic process into irreversible structural valve damage. Vitamin K antagonist therapy generally reverses haemodynamic abnormalities in clinically significant cases; however, the GALILEO trial demonstrated that routine rivaroxaban after TAVR does not improve clinical outcomes and increases bleeding, supporting a targeted rather than universal approach to anticoagulation [12].

Prosthetic Valve Endocarditis (PVE) accounts for up to one-third of early reinterventions, with a cumulative incidence of approximately 1-3% at one year for both surgical and transcatheter prostheses [13]. Early PVE (within 12 months) is most commonly attributable to perioperative contamination, with Staphylococcus aureus, coagulase-negative staphylococci and Enterococcus species predominant. The infective process typically involves the suture line and sewing ring, producing perivalvular abscess, pseudoaneurysm, fistula and dehiscence. In-hospital mortality remains 15-25%, with one-year mortality approaching 40% in some series; surgery, when indicated, should not be delayed in suitable candidates [13,14]. Non-structural causes include patient-prosthesis mismatch (severe PPM with indexed effective orifice area <0.65 cm^2/m^2, reported in 20-70% of surgical series depending on annular size), paravalvular regurgitation and procedural malposition, particularly within the first 30 days of implantation [15].

Temporal-mechanistic framework

Within the five-year EBVF window, three temporal phases can be recognized. Very early failure (≤30 days) is dominated by procedural complications: malposition, paravalvular leak, and acute thrombosis. Early failure (30 days-1 year) is increasingly characterized by endocarditis, subclinical and clinical leaflet thrombosis and patient-prosthesis mismatch. Intermediate failure (1-5 years) reflects the onset of accelerated structural degeneration, late-onset endocarditis and persistent thrombus-mediated leaflet damage. These phases are dominant but overlapping; more than one mechanism may be active at any given time point (Table 1).

Table 1: Temporal-mechanistic guide to investigation of suspected EBVF.

Time from Implant	Most Likely Causes	First Escalation Test
≤30 days	Malposition, PVL, dehiscence, acute thrombosis	TEE ± CT
30 d-1 year	PVE, HALT/thrombosis, PPM	TEE, CT, blood cultures
1-5 years	Accelerated SVD, pannus, late PVE	CT ± PET/CT

CT: Computed Tomography; HALT: Hypoattenuated Leaflet Thickening; PET: Positron Emission Tomography; PPM: Patient-Prosthesis Mismatch; PVE: Prosthetic Valve Endocarditis; PVL: Paravalvular Leak; SVD: Structural Valve Degeneration; TEE: Transoesophageal Echocardiography.

Diagnostic approach

The investigation of suspected EBVF begins with transthoracic echocardiography compared with a baseline study performed prior to discharge or at six weeks. New or progressive elevation of transvalvular gradients, new regurgitation, leaflet thickening, restricted leaflet motion and evidence of valve rocking should prompt further evaluation. Transesophageal echocardiography achieves sensitivity for vegetations approaching 90% (versus approximately 70% for transthoracic imaging) and for abscess detection approximately 90% (versus ~50%), and is essential in suspected endocarditis or mitral prosthesis dysfunction [16].

Cardiac CT distinguishes thrombus from pannus (a Hounsfield unit threshold of approximately 145, though clinical correlation remains essential), identifies HALT and reduced leaflet motion, characterizes perivalvular complications, and evaluates coronary obstruction risk during ViV planning [17]. Cardiac magnetic resonance provides accurate quantification of regurgitation severity and ventricular remodeling [18]. F-FDG PET/CT is now incorporated as a major Duke criterion for PVE beyond three months post-implantation in the 2023 ESC guidelines, though interpretive caution is required early postoperatively; WBC SPECT/CT provides complementary specificity [16-19]. F-sodium fluoride PET has demonstrated active microcalcification within months of TAVR, with baseline tracer uptake predicting subsequent haemodynamic deterioration [18].

Management considerations

Treatment is mechanism-specific: observation or VKA therapy for thrombosis (recurrence after withdrawal is well described); prolonged targeted antibiotics with timely surgery for endocarditis when indicated by severe regurgitation, perivalvular extension, large vegetations, uncontrolled infection, or recurrent embolism; and redo surgery or transcatheter Valve-In-Valve (ViV) for symptomatic structural failure [14,16]. Redo surgical AVR carries 5-13% in-hospital mortality, while ViV achieves approximately 95% procedural success in selected high-risk patients with one-year mortality of 15-20% [20]. The choice between redo surgery and ViV is guided by patient age and life expectancy, surgical risk (STS score), original valve label size (risk of severe ViV PPM with ≤21mm), coronary ostial height on CT (<10mm conferring high obstruction risk), the need for concomitant cardiac procedures, and predicted neo-LVOT adequacy for mitral ViV. Younger patients (<65 years) with good life expectancy and low surgical risk generally favour redo surgery, while older or higher-risk patients with adequate anatomy are better served by ViV. These decisions are best made within a structured heart team framework. An important consideration in long-term planning is the feasibility of future transcatheter re-intervention after the index procedure. A multicenter CT analysis by Sheth et al. [21] evaluated 245 patients across four groups-TAVR using SAPIEN 3 at ≤90% implant depth, TAVR at >90% implant depth, SAVR, and SAVR with Aortic Root Enlargement (ARE)-and demonstrated that TAVR with SAPIEN 3 at implant depth ≤90% was the most repeatable initial intervention, with the simulated neo-skirt positioned below both coronary arteries in 47.0% of cases and challenging anatomy for future TAVR identified in only 9.8%. By contrast, SAVR with ARE offered no improvement in future TAVR feasibility compared with standard SAVR (0% below both coronary arteries; 29.0% challenging anatomy). These findings reinforce the importance of optimizing implant depth at the time of initial TAVR to preserve future re-intervention options, and highlight that ARE at the time of surgical AVR does not facilitate subsequent transcatheter approaches [19-21].

Future directions

Several developments offer the prospect of reducing the incidence of EBVF. Anti-calcification treatments such as the RESILIA tissue platform have demonstrated promising mid-term durability, with the Indure Durability Registry currently evaluating outcomes in younger recipients (≤60 years) [7]. Tissue engineering aimed at decellularization, removal of α-gal antigens using GTKO porcine donors, and improved fixation chemistries may diminish the immune contribution to structural failure [8]. Surveillance strategies incorporating [18] F-sodium fluoride PET to detect early microcalcification and serial cardiac CT for HALT detection may permit risk-stratified follow-up tailored to individual patient profiles [18]. Refinements in transcatheter valve design, including improved sealing skirts and durable polymer leaflet valves, hold promise for both primary and ViV procedures. Prospective validation of anti-α-gal and anti-Neu5Gc antibody titres as immune biomarkers of early SVD risk could enable personalized surveillance and potentially guide the timing of prophylactic reintervention.

Conclusion

EBVF is a multifactorial syndrome distinct from late SVD. The temporal phase of failure provides critical diagnostic guidance: procedural causes dominate within 30 days, infective and thrombotic mechanisms within the first year and accelerated SVD thereafter. No single imaging modality is sufficient; multimodality

assessment integrating echocardiography, cardiac CT, CMR and nuclear imaging is essential. Treatment decisions extend beyond the immediate intervention to the patient's lifetime valve replacement trajectory and are best made within a heart team framework. As BHVs are increasingly implanted in younger, lower-risk patients, systematic identification and management of EBVF will assume growing clinical importance.

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