Cardiology Research and Cardiovascular Medicine
Asopa S and Ohri SK. Cardiolog Res Cardiovasc Med 11: 298.
www.doi.org/10.29011/2575-7083.100298
www.gavinpublishers.com

OPEN ACCESS

GAVIN PUBLISHERS

A Narrative Review

Early Bioprosthetic Valve Failure: Mechanisms, Diagnostic Pathways and Surgical Decision-Making

Sanjay Asopa and Sunil K. Ohri*

Wessex Cardiac Centre, University Hospital Southampton NHS Foundation Trust, Southampton, UK

***Corresponding author:** Sunil K. Ohri, Wessex Cardiac Centre, University Hospital Southampton NHS Foundation Trust, Tremona Road, Southampton SO16 6YD, United Kingdom.

Citation: Asopa S and Ohri SK (2026) Early Bioprosthetic Valve Failure: Mechanisms, Diagnostic Pathways and Surgical Decision-Making. Cardiol Res Cardio vasc Med 11:298. DOI:https://doi.org/10.29011/2575-7083.100298

Received Date: 29 April, 2026; **Accepted Date:** 07 May, 2026; **Published Date:** 13 May, 2026

Abstract

The use of bioprosthetic heart valves has expanded substantially over the past two decades, now accounting for the majority of valve replacements performed worldwide. While late structural valve degeneration remains the dominant mode of failure beyond a decade, early bioprosthetic valve failure (EBVF), defined as valve dysfunction necessitating reintervention, causing severe haemodynamic compromise or death within 5 years of implantation, represents a distinct and increasingly recognised clinical problem. EBVF is multifactorial in origin and reflects the interplay of three principal pathological processes: accelerated structural valve degeneration driven by host immune response and dystrophic calcification; valve thrombosis, both subclinical and clinical, including hypoattenuated leaflet thickening after transcatheter implantation; and prosthetic valve endocarditis, which carries disproportionate early mortality. Non-structural causes including patient–prosthesis mismatch, paravalvular regurgitation, and procedural malposition further contribute, particularly with transcatheter valves. Younger recipients, valves implanted in the mitral position, valve-in-valve procedures, end-stage renal disease and metabolic disorders are established risk factors. Multimodality imaging, integrating transthoracic and transoesophageal echocardiography, cardiac computed tomography, cardiac magnetic resonance and nuclear imaging, has transformed the early recognition and characterisation of EBVF. This review synthesises current understanding of the mechanisms, risk factors, diagnostic approach and therapeutic implications of early bioprosthetic valve failure, with emphasis on the surgical perspective. A temporal-mechanistic approach—linking time from implantation to likely pathology—can improve diagnostic accuracy and guide appropriate selection of anticoagulation, antimicrobial therapy, redo surgery or valve-in-valve intervention.

Keywords: bioprosthetic heart valve; structural valve degeneration; valve thrombosis; prosthetic valve endocarditis; transcatheter aortic valve replacement; valve-in-valve; multimodality imaging.

Introduction

Surgical and transcatheter bioprosthetic heart valves (BHVs) have, over the past twenty years, progressively displaced mechanical prostheses to become the substitute of choice for the majority of patients undergoing valve replacement [1,2]. The principal drivers of this shift are the favourable haemodynamic profile of biological valves, the avoidance of lifelong oral anticoagulation, and the rapid expansion of transcatheter aortic valve replacement (TAVR) into intermediate- and low-risk surgical populations [3,4]. Improvements in tissue engineering, anti-calcification treatments and stentless or sutureless designs have further broadened the patient cohort considered suitable for biological valves [5].

Despite these advances, the durability of BHVs remains finite. The probability of structural deterioration rises steeply beyond ten years, and reintervention rates between 10 and 20 years range widely depending on valve model, position, and patient age [3,6]. Of greater concern, however, is the emergence of a distinct subset of patients who experience bioprosthetic valve failure within months to a few years of implantation, an occurrence termed early bioprosthetic valve failure (EBVF). Once dismissed as anecdotal, EBVF is now increasingly recognised in the era of younger, lower-risk recipients and complex valve-in-valve (ViV) procedures [7,8]. The ten-year outcomes of the NOTION trial have confirmed

comparable clinical outcomes between TAVR and surgical aortic valve replacement in lower-risk patients but with lower rates of severe structural valve deterioration after TAVR [9], while the PARTNER 3 five-year data have demonstrated the extension of TAVR into populations whose life expectancy substantially exceeds the expected durability of a single bioprosthesis [4]. Recent data suggest that hemodynamic valve deterioration may be detectable in over 6 per cent of TAVR recipients within the first year, with smaller valve size a significant predictor [10].

The contemporary epidemiology of EBVF is best appreciated against the backdrop of these landmark trials. In the PARTNER 3 cardiac CT substudy, subclinical leaflet thrombosis was detected in 13 per cent of TAVR recipients and 5 per cent of surgical recipients at 30 days, with persistent or recurrent disease at one year [11]. Prosthetic valve endocarditis affects approximately 0.5 to 1 per cent of bioprosthetic valves per year, with the highest hazard in the first six to twelve months [12,13]. Reported rates of moderate or severe patient–prosthesis mismatch range from 20 to 70 per cent in surgical series, depending on annular size and implantation technique [14,15]. Aggregating these mechanisms, contemporary registries suggest that between 5 and 12 per cent of bioprosthetic valves develop some form of clinically relevant dysfunction within the first five years, with the proportion progressing to outright failure varying from 1 to 4 per cent [6,16,17].

EBVF is not a single pathology but a final common pathway for several distinct processes (Figure 1). Accelerated structural degeneration mediated by foreign-body immune response, dystrophic calcification, valve thrombosis (both clinical and subclinical), prosthetic valve endocarditis (PVE), patient–prosthesis mismatch (PPM), and technical procedural complications all contribute. The relative importance of each varies according to valve type, anatomical position, and recipient characteristics [16,17]. Distinguishing among these aetiologies has immediate clinical relevance, since the appropriate intervention—anticoagulation for thrombus, antibiotics and possible reoperation for endocarditis, redo surgery or transcatheter ViV for degeneration—depends entirely on accurate mechanistic diagnosis.

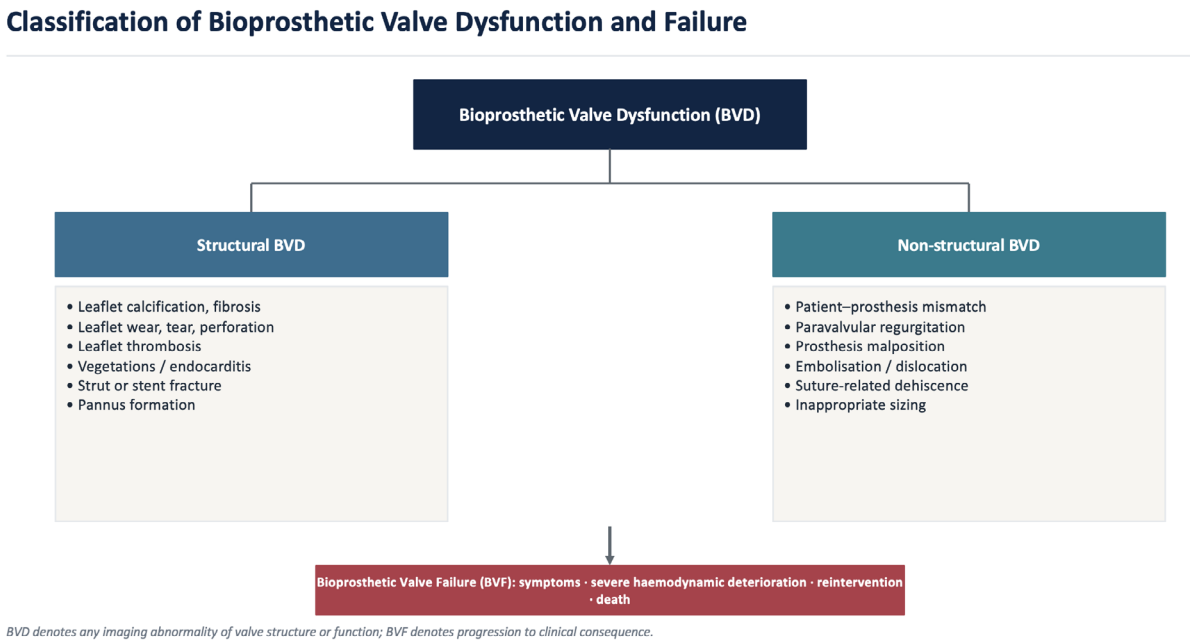

Figure 1: Conceptual classification of bioprosthetic valve dysfunction (BVD) and bioprosthetic valve failure (BVF). BVD is divided into structural (leaflet wear, calcification, thrombosis, endocarditis) and non-structural (patient–prosthesis mismatch, paravalvular regurgitation, malposition) categories. BVF represents progression to clinical consequence, including symptoms, severe haemodynamic deterioration, reintervention or death. Adapted from VARC-3 and EAPCI/EACTS consensus [18,19].

This review differs from prior reviews by integrating the temporal phase of failure, the underlying mechanism, and the treatment pathway into a single practical framework. It provides a contemporary surgical perspective on the aetiology of EBVF, summarises the definitions and terminology proposed by recent consensus documents, examines the principal mechanisms of early failure, and discusses the diagnostic and management implications. The emphasis throughout is on the practical question facing the clinician at the bedside: when a recently implanted bioprosthesis fails, what has gone wrong, and what should be done?

Definitions and Classification

Bioprosthetic valve dysfunction versus failure

A clear distinction must be drawn between bioprosthetic valve dysfunction (BVD) and bioprosthetic valve failure (BVF) (Figure 1). The 2017 European Association of Percutaneous Cardiovascular Interventions consensus and the subsequent 2021 Valve Academic Research Consortium-3 (VARC-3) document define BVD as any abnormality of valve structure or function detected on imaging, regardless of clinical consequence [18,19]. BVF, by contrast, describes BVD that has progressed to a clinically meaningful endpoint: severe haemodynamic deterioration, valve-related symptoms, reintervention, or death [16,19].

BVD is further subdivided into structural and non-structural categories. Structural BVD encompasses leaflet wear, tear, prolapse, fibrosis, calcification, strut fracture, or deformation, including changes secondary to thrombosis or endocarditis. Non-structural BVD includes patient–prosthesis mismatch, paravalvular regurgitation in the absence of leaflet pathology, prosthesis malposition and embolisation [16].

Defining “early” failure

There is no universally accepted temporal threshold for early BVF. Historical surgical series applied an arbitrary 30-day cut-off corresponding to operative mortality, while more recent consensus statements have proposed broader windows reflecting the natural history of different failure modes [19,20]. For the purposes of this review, EBVF is defined as bioprosthetic valve failure occurring within five years of implantation. This threshold is pragmatic rather than universal, chosen to capture the bulk of thrombotic and infective events as well as the rare but distinct phenomenon of accelerated structural deterioration in younger recipients and those with metabolic comorbidities [20,21].

Within this five-year window, three temporal phases can be recognised (Figure 2). Very early failure (within 30 days) is dominated by procedural and surgical complications: malposition, paravalvular leak, suture-related dehiscence and acute thrombosis. Early failure (30 days to 1 year) is increasingly characterised by infective endocarditis, subclinical and clinical leaflet thrombosis, and the haemodynamic consequences of patient–prosthesis mismatch. Intermediate failure (1 to 5 years) reflects the onset of accelerated structural degeneration in vulnerable recipients, late-onset endocarditis and persistent thrombus-mediated leaflet damage [21,22]. These phases are dominant but overlapping; more than one mechanism may be active at any given time point.

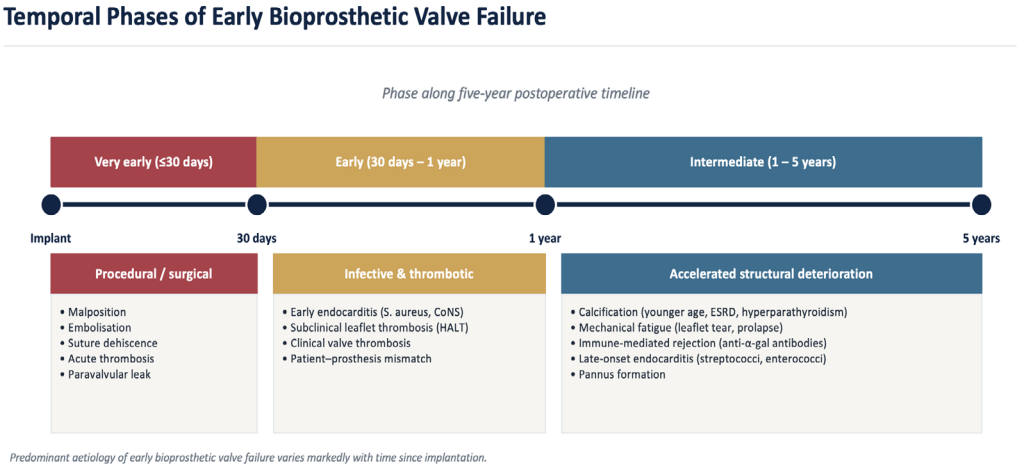

Figure 2: Temporal phases of early bioprosthetic valve failure within five years of implantation. Very early failure (≤30 days) is dominated by procedural and surgical complications. Early failure (30 days to 1 year) is characterised by infective endocarditis, leaflet thrombosis and patient–prosthesis mismatch. Intermediate failure (1–5 years) reflects the onset of accelerated structural valve degeneration. Note that these phases are dominant but overlapping; more than one mechanism may be active at any given time point.

Stages of structural valve degeneration

The standardised three-stage classification of structural valve degeneration provides a framework applicable to both early and late failure [19,23]. Stage 1 SVD denotes morphological leaflet changes (thickening, calcification or restricted motion) without haemodynamic consequence. Stage 2 represents morphological abnormality accompanied by moderate stenosis or regurgitation. Stage 3, the threshold for symptomatic reintervention, is defined by severe stenosis (mean gradient greater than 40 mmHg or effective orifice area less than 1 cm^2 for the aortic position) or severe regurgitation [23]. When any of these stages occur within five years of implantation, the patient may be considered to have early SVD, which itself constitutes one mechanism of EBVF.

Stage	Aortic bioprosthesis	Mitral bioprosthesis
Stage 1 Morphological without haemodynamic impact	Vmax <3 m/s; mean gradient <20 mmHg; DVI >0.35; EOA >1.2 cm^2 (BSA <1.6 m^2); morphological leaflet abnormality without significant gradient rise.	Mean gradient <5 mmHg; MVA >1.5 cm^2; morphological abnormality only.
Stage 2 Moderate haemodynamic dysfunction	Vmax 3–4 m/s; mean gradient 20–40 mmHg; DVI 0.25–0.35; ΔMG ≥10 mmHg vs baseline; or new moderate regurgitation.	Mean gradient >5 mmHg; ΔDVI ≥0.4 or ≥20%; ΔMVA ≥0.5 cm^2 or ≥25%; or new moderate regurgitation.
Stage 3 Severe haemodynamic dysfunction	Vmax >4 m/s; mean gradient >40 mmHg; ΔMG >20 mmHg vs baseline; EOA <1 cm^2 (BSA <1.6 m^2); or new severe regurgitation.	Mean gradient >10 mmHg; ΔDVI ≥0.8 or ≥40%; ΔMVA ≥1.0 cm^2 or ≥50%; MVA <1.0 cm^2; or new severe regurgitation.
BSA, body surface area; DVI, Doppler velocity index; EOA, effective orifice area; MG, mean gradient; MVA, mitral valve area; SVD, structural valve degeneration; Vmax, maximum velocity. Stage 3 SVD is the threshold at which reintervention is recommended in symptomatic patients.		

Table 1: Stages of structural valve degeneration for the aortic and mitral bioprosthesis (adapted from VARC-3 and EAPCI/EACTS consensus) [18,19,21].

Accelerated Structural Valve Degeneration

Pathophysiology of early structural failure

Native valves are dynamic, self-renewing structures, while bioprosthetic leaflets are biologically inert and incapable of repair, leaving them to bear cumulative mechanical, haemodynamic and immunological insults passively [5,24]. Three interrelated mechanisms underpin SVD (Figure 3): dystrophic calcification, mechanical fatigue, and immune-mediated rejection. These processes interact and amplify one another; early failure is typically observed when more than one process is active concurrently, particularly in younger recipients or those with metabolic predisposition.

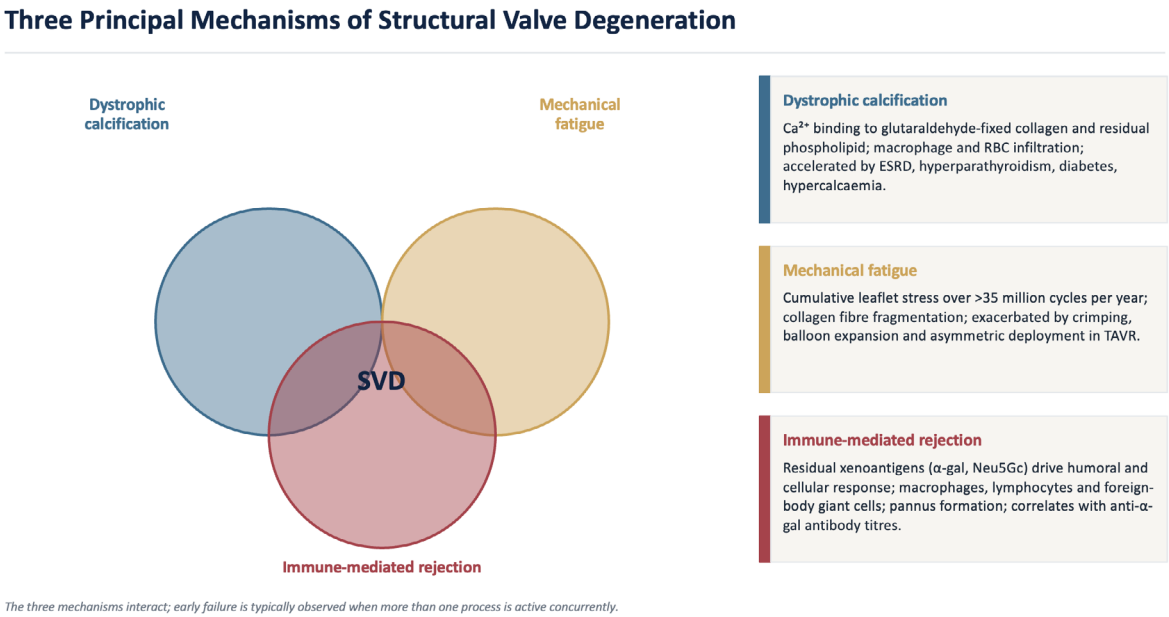

Figure 3: The three principal pathological mechanisms of structural valve degeneration: dystrophic calcification, mechanical fatigue, and immune-mediated rejection. These processes interact and amplify one another, particularly in younger recipients and patients with metabolic predisposition. Interaction arrows between the three pathways illustrate that calcification, fatigue and immune activation are not independent but synergistic.

Calcification is initiated by the binding of calcium to residual phospholipid cell membranes and to glutaraldehyde-treated collagen within the leaflet [5,24,25]. Glutaraldehyde fixation, while reducing immunogenicity and providing tensile strength, also generates aldehyde groups that bind calcium ions and promote nucleation of hydroxyapatite. Macrophage and red blood cell infiltration accelerates this process by liberating intracellular calcium and contributing iron-mediated oxidative injury [24]. In recipients with end-stage renal disease, hyperparathyroidism or markedly elevated calcium–phosphate products, calcification can progress at a strikingly accelerated rate [20,25].

Several anti-calcification strategies have been incorporated into modern bioprostheses. These include alpha-amino oleic acid treatment (Mosaic, Medtronic), ethanol pretreatment (Hancock II, Medtronic), surfactant treatments (Trifecta, Abbott), and the more recent RESILIA tissue platform (Edwards Lifesciences), which uses a glycerolisation step to cap residual aldehyde groups and enable dry storage. The COMMENCE trial and its extension cohorts have demonstrated promising mid-term durability, with freedom from structural valve deterioration exceeding 99 per cent at five years and encouraging seven-year data from the aortic cohort, although direct comparative long-term evidence with conventional pericardium in randomised trials remains limited, and the durability advantage in younger recipients requires further follow-up [26,27]. Whether these treatments fundamentally alter the trajectory of EBVF in the youngest recipients, where calcification is most aggressive, remains to be established.

Mechanical fatigue results from the repetitive flexion of leaflets during more than 35 million cardiac cycles per year. Fibres of collagen progressively fragment and elastic components disorganise, predisposing to leaflet tear, perforation, and prolapse. In transcatheter heart valves, additional stresses are imposed by crimping, balloon expansion, and post-deployment leaflet asymmetry, all of which may accelerate mechanical deterioration [17,28].

Immune-mediated rejection has emerged as a third, increasingly recognised, mechanism. Despite glutaraldehyde fixation, residual xenoantigens (notably galactose-α-1,3-galactose and N-glycolylneuraminic acid) elicit a sustained adaptive immune response in human recipients [29,30]. The resulting infiltration by lymphocytes, macrophages, and foreign-body giant cells generates pannus, an inflammatory tissue overgrowth comprising immune cells, endothelial cells and myofibroblasts that progressively encroaches on the leaflet base and commissures [30,31].

The seminal work of Senage and colleagues, published in Nature Medicine in 2022, identified circulating IgG antibodies against α-gal and Neu5Gc in the great majority of patients undergoing bioprosthetic valve replacement, with antibody titres rising sharply in the months following implantation [29]. In their cohort, the magnitude of the antibody response to α-gal at three months post-implantation was an independent predictor of subsequent SVD over follow-up. 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 [30].

Risk factors for accelerated SVD

Younger age at implantation is the single strongest demographic risk factor (Figure 4). Patients aged under 60 years' experience SVD at substantially greater rates than older recipients, a phenomenon ascribed to higher cardiac output, greater leaflet stress, more vigorous immune response, and longer expected exposure [3,20]. End-stage renal disease, hyperparathyroidism, diabetes mellitus and chronic inflammatory states all predispose to premature calcification [20,25]. Bovine pericardial valves tend to fail through stenosis driven by leaflet calcification, while porcine valves more often present with regurgitation from leaflet tear [5]. Mitral position bioprostheses degenerate more rapidly than aortic, reflecting greater mechanical loading and higher prevalence of atrial fibrillation with associated stasis [16,18].

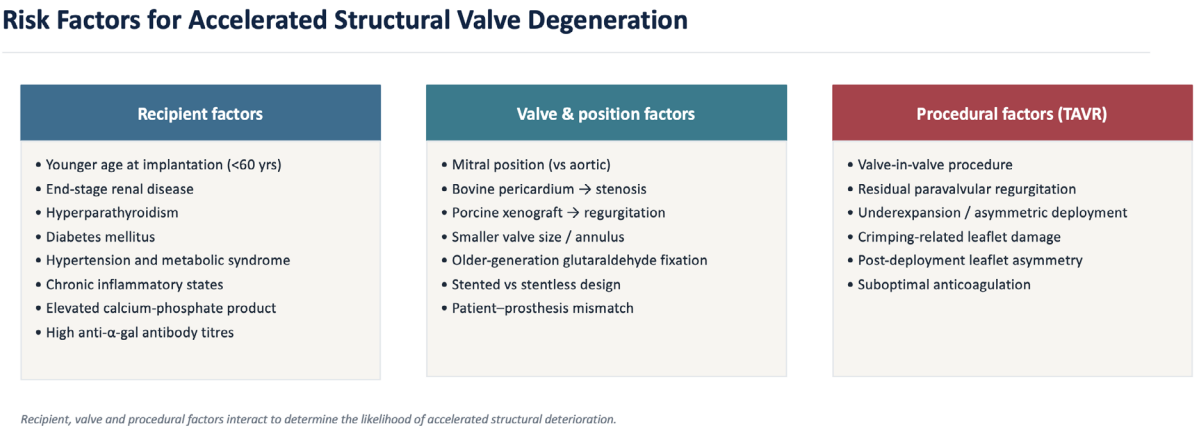

Figure 4: Risk factors for accelerated structural valve degeneration. Recipient factors (younger age, end-stage renal disease, hyperparathyroidism, diabetes), valve factors (mitral position, bovine pericardium for stenosis, porcine for regurgitation), and procedural factors (valve-in-valve, paravalvular leak, underexpansion) all contribute. Surgical and TAVR-specific risk factors are presented separately for clarity.

In the transcatheter setting, valve-in-valve implantation, residual paravalvular regurgitation, underexpansion and asymmetric deployment have all been associated with accelerated leaflet degeneration [17,28,32]. Prospective imaging studies using ^{18}F-sodium fluoride positron emission tomography have demonstrated active microcalcification at the leaflet level within months of TAVR, with baseline tracer uptake predicting subsequent haemodynamic deterioration [33,34].

Tissue Preservation Technologies and Bioprosthetic Valve Design

The aetiology of EBVF cannot be considered in isolation from the engineering of the prosthesis itself. The biological substrate, the fixation chemistry, the stent or frame design, and the manner in which leaflets are configured all influence the trajectory toward early failure. Understanding these design considerations is essential for the surgeon counselling patients on prosthesis selection.

Biological substrate and fixation

Three principal xenograft tissues have been used clinically: porcine aortic valve leaflets, bovine pericardium, and equine pericardium. Porcine valves preserve native valvular architecture but tend to fail through leaflet tear and regurgitation. Bovine pericardium provides excellent tensile strength but tends to fail through diffuse calcification and stenosis [5,24]. The intrinsic immunogenicity of these tissues is dominated by xenoantigens that survive fixation, notably α-gal and Neu5Gc [29,30].

Glutaraldehyde has been the standard fixative since its introduction by Carpentier in the late 1960s. The phenomenon known as the "Carpentier paradox"—that the same fixation responsible for tissue durability is also responsible for its eventual calcific failure—has driven decades of research into modifications and alternatives [24]. Successive anti-calcification treatments have targeted specific aspects of the calcification cascade, and the most substantial advance over the past decade is the RESILIA platform, which has demonstrated promising mid-term durability in the COMMENCE trial [26,27]. Importantly, RESILIA addresses calcification but does not directly modify xenoantigenicity, and so the contribution of immune-mediated rejection to failure is not obviously attenuated.

Stent design and the Trifecta experience

Among stented surgical valves, the geometric relationship between leaflets and the stent frame is a critical determinant of durability. In internally mounted designs (e.g. Carpentier-Edwards Perimount), the leaflets open within the lumen of the frame. In externally mounted designs (e.g. the original Trifecta, Abbott), the leaflet tissue is wrapped around the outside of the stent, providing a larger effective orifice area but exposing leaflets to direct contact and frictional abrasion against the stent posts. These features have been implicated in the accelerated structural deterioration observed with the original Trifecta valve, where post-market surveillance identified an unexpectedly high rate of early failure within five to seven years of implantation, particularly in smaller sizes [31,35,36].

In February 2023, the United States Food and Drug Administration issued a safety communication to healthcare providers citing published literature suggesting a higher cumulative incidence of early SVD for the Trifecta valve compared with other commercially available surgical bioprostheses [37]. Subsequently, in July 2023, Abbott announced the discontinuation and market withdrawal of the Trifecta and Trifecta GT valves in the United States [37]. This episode serves as a case study of design-related EBVF and underscores the principle that haemodynamic optimisation and durability are not independent goals. Surveillance of new valve designs through the first five to seven years of clinical use is therefore essential, since the failure modes of any novel mounting geometry may not be apparent in the early post-implantation period.

Transcatheter valve design and emerging technologies

Transcatheter heart valves face engineering constraints that surgical valves do not. Crimping induces irreversible micro-injury to leaflet collagen, potentially predisposing to mechanical fatigue and accelerated calcification [17,28,38]. Asymmetric deployment distributes leaflet stress unevenly and creates regions of stagnation that predispose to leaflet thrombosis [39]. Current-generation designs including the SAPIEN 3 Ultra and Evolut FX/PRO+ have substantially improved sealing and reduced paravalvular regurgitation [17,40]. Polymer leaflet transcatheter valves represent an emerging direction that may circumvent the immunogenicity and calcification limitations of biological tissue, though long-term human data are awaited [4].

Bioprosthetic Valve Thrombosis

Incidence and clinical spectrum

Valve thrombosis represents one of the most important and potentially reversible causes of EBVF. Symptomatic bioprosthetic valve thrombosis was historically considered rare, with an estimated incidence of approximately 0.03 per cent per year [41]. Subclinical leaflet thrombosis, however, identified by hypoattenuated leaflet thickening (HALT) on cardiac computed tomography, is far more prevalent, occurring in 10 to 15 per cent of patients after TAVR and in up to 7 per cent after surgical aortic valve replacement [11,42]. It is important to emphasise that HALT may resolve spontaneously and does not automatically mandate anticoagulation; the clinical relevance of subclinical thrombosis remains debated, but accumulating evidence suggests an association with later structural deterioration and a small but measurable increase in cerebrovascular events [11,43].

Mechanisms

All three components of Virchow's triad contribute to bioprosthetic thrombosis (Figure 5). Surface damage occurs through incomplete endothelialisation of the prosthesis, residual glutaraldehyde, leaflet asymmetry following crimping, and adsorption of plasma proteins [44,38]. Stasis is intrinsic to regions of low or recirculating flow, exemplified by the neo-sinus formed between the transcatheter valve and the native aortic root [39]. A hypercoagulable state, whether primary or related to malignancy, chronic kidney disease, atrial fibrillation, or recent surgery, completes the triad [41,44].

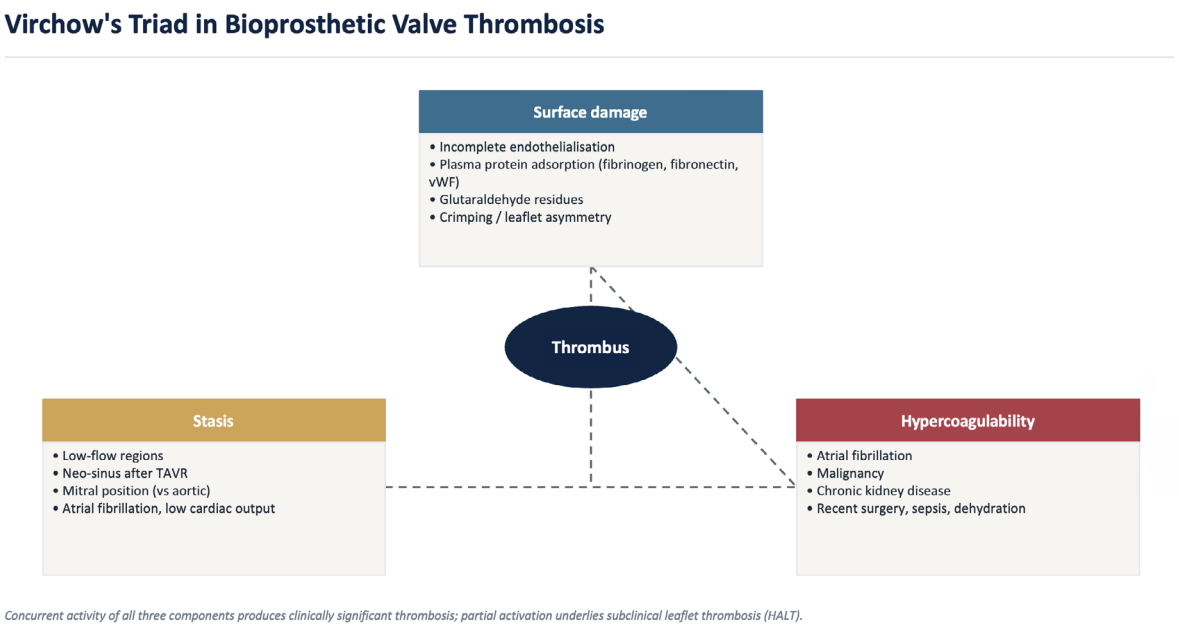

Figure 5: Schematic representation of Virchow's triad in bioprosthetic valve thrombosis. Surface damage (incomplete endothelialisation, plasma protein adsorption), stasis (low-flow regions, neo-sinus), and a hypercoagulable state (atrial fibrillation, malignancy, recent surgery) interact to produce thrombus formation. Concurrent activity of all three components produces clinically significant thrombosis; partial activation underlies subclinical leaflet thrombosis (HALT).

Hypoattenuated leaflet thickening and reduced leaflet motion

On contrast-enhanced cardiac CT, leaflet thrombus is visualised as a meniscus-shaped region of low attenuation along the aortic surface of the leaflet, designated HALT. When associated with restricted motion (RELM, or hypoattenuation affecting motion, HAM), haemodynamic consequence is more likely [11,45]. Distinguishing thrombus from chronic pannus is critical, as pannus does not respond to anticoagulation and typically requires surgical revision; a Hounsfield unit threshold of 145 has been proposed, with masses below this value favouring thrombus and those above pannus, although this threshold is a useful guide rather than an absolute discriminator (Figure 6) [46].

Distinguishing Thrombus, Pannus and Vegetation

Feature	Thrombus	Pannus	Vegetation
Time course	Days–months	Months–years	Days–weeks
Echo appearance	Iso/hypoechogenic mass on leaflet surface	Hyperechoic, fixed, near ring/cusp base	Mobile mass independent of leaflet motion
Leaflet motion	Reduced (HAM)	Reduced or normal	Normal or restricted by mass
CT attenuation (HU)	<145 (often <90)	≥145	Variable, usually low
Calcification	Absent	May be present	Absent
Clinical context	AF, low flow, suboptimal anticoagulation	Late presentation, slow gradient rise	Fever, positive blood cultures, embolism
Initial therapy	Vitamin K antagonist	Surgical revision	Targeted antibiotics ± surgery

HAM = hypoattenuation affecting motion; HU = Hounsfield units; AF = atrial fibrillation. Adapted from contemporary multimodality imaging recommendations.

Figure 6: Distinguishing features of thrombus, pannus, and vegetation on cardiac computed tomography and echocardiography. Hounsfield unit measurement on CT (threshold of 145) separates thrombus (lower attenuation) from pannus (higher attenuation), while echocardiographic features and clinical context guide the differentiation from infective vegetation. The HU threshold is a useful guide rather than an absolute discriminator; clinical correlation is essential.

Clinical course and management

Up to half of subclinical thrombus episodes resolve spontaneously over weeks to months without specific intervention [43]. Initiation of vitamin K antagonist therapy generally reverses haemodynamic abnormalities in clinically significant cases, although recurrence after withdrawal is well described [11]. Chronic, organised thrombus may evolve into fibrosis and calcification, producing irreversible structural valve damage and converting what began as a thrombotic process into established SVD [18,43]. The GALILEO and ATLANTIS trials demonstrated that routine direct oral anticoagulation after TAVR does not improve clinical outcomes and may increase bleeding, supporting a targeted rather than universal approach to anticoagulation [47,48].

Prosthetic Valve Endocarditis

Epidemiology of early PVE

Prosthetic valve endocarditis is a major cause of EBVF, accounting for up to one-third of early reinterventions in some series [12,13]. The cumulative incidence at one year is approximately 1 to 3 per cent for both surgical and transcatheter aortic prostheses, and appears comparable between mechanical and biological valves [12,13]. Early PVE (within 12 months) is most commonly attributable to perioperative contamination, with Staphylococcus aureus, coagulase-negative staphylococci, and Enterococcus species predominant [12,49]. Late PVE is more often community-acquired and caused by oral streptococci or enterococci. Following TAVR, an additional risk derives from the use of non-sterile environments such as catheterisation laboratories [13,50].

Pathological patterns

In early PVE, the infective process typically involves the suture line and sewing ring, producing perivalvular abscess, pseudoaneurysm, fistula and dehiscence [12]. Late PVE more frequently affects the leaflets themselves. Following TAVR, vegetations may form on prosthetic leaflets, the metallic stent frame, or by extension the anterior mitral leaflet; perivalvular complications occur in approximately 20 per cent of TAVR-PVE cases [13,50].

Diagnostic challenges

The modified Duke criteria perform less well in the prosthetic valve setting, with sensitivity in early PVE reduced by acoustic shadowing, small vegetations, and negative blood cultures in patients receiving empirical antibiotics [51,52]. Transthoracic echocardiography has a sensitivity of approximately 70 per cent for vegetations and only 50 per cent for abscesses; transoesophageal

echocardiography improves sensitivity to nearly 90 per cent [53]. Cardiac computed tomography offers excellent characterisation of perivalvular complications [54].

^{18}F-fluorodeoxyglucose positron emission tomography combined with CT has substantially improved diagnostic accuracy in PVE. The 2023 European Society of Cardiology guidelines have explicitly incorporated ^{18}F-FDG PET/CT as a major Duke criterion for prosthetic valve endocarditis occurring more than three months after implantation, recognising that abnormal prosthetic or periprosthetic uptake can support the diagnosis of PVE [52,55]. However, interpretive caution is required in the early postoperative period, when sterile inflammation may produce false-positive uptake. White-cell labelled scintigraphy (WBC SPECT/CT) provides a complementary modality with higher specificity, and is particularly useful when FDG-PET is inconclusive or in the early post-operative window [55,56].

Outcomes

Despite advances in diagnosis and management, PVE remains associated with high mortality. In-hospital mortality ranges between 15 and 25 per cent, and one-year mortality approaches 40 per cent in some series [12,57]. Surgery, when indicated by the presence of severe regurgitation, perivalvular complications, large vegetations, or persistent infection, should not be delayed in suitable candidates [52,57].

Non-structural Causes of Early Failure

Patient–prosthesis mismatch

Patient–prosthesis mismatch occurs when the effective orifice area of the implanted prosthesis is too small relative to body surface area, producing residual high transvalvular gradients despite normal valve function [14]. Severe PPM (indexed effective orifice area less than 0.65 cm^2/m^2 in the aortic position) is associated with persistent left ventricular hypertrophy, impaired regression of pulmonary hypertension, and increased early and late mortality [14,15]. Strategies to mitigate PPM include preoperative computed tomography sizing, root enlargement procedures, and selection of supra-annular or stentless prostheses in patients with small annuli [14,58].

Paravalvular regurgitation

Paravalvular leak after surgical valve replacement most often reflects suture dehiscence in the context of friable annular tissue. After TAVR, paravalvular regurgitation is generally a consequence of valve undersizing or calcium-induced incomplete sealing, although its incidence has fallen substantially with newer-generation devices [17,40]. Even mild to moderate paravalvular regurgitation has been linked to reduced survival and accelerated structural leaflet degeneration [17,40].

Procedural and surgical factors

Technical factors operative within the perioperative period include valve malposition, embolisation, inappropriate sizing, and surgical injury to adjacent structures. Cardiac CT prior to redo or transcatheter intervention is essential to evaluate sternal proximity, vascular access, and the risk of coronary obstruction during valve-in-valve procedures, which is estimated at 2.3 per cent compared with 0.7 per cent for de novo TAVR [59,60]. A virtual transcatheter heart valve to coronary ostium distance of less than 4 mm identifies high-risk anatomy [59].

Very early failure within 30 days

A subset of EBVF presents within the first thirty postoperative days and is almost always attributable to identifiable technical or procedural factors rather than to disease processes intrinsic to the prosthesis. Suture-related complications dominate this very early window in surgical practice. Prosthesis malposition encompasses both gross misalignment of a surgical valve and suboptimal deployment of a transcatheter device [40]. Acute valve thrombosis presenting within days of implantation is uncommon but potentially catastrophic [41,43]. Iatrogenic injury to surrounding structures may not be recognised until the patient fails to wean from cardiopulmonary bypass [60].

Diagnostic Approach to Suspected Early Failure

The investigation of suspected EBVF begins with transthoracic echocardiography, which should be compared with a baseline study performed prior to discharge or at six weeks (Figure 7) [61]. New or progressive elevation of transvalvular gradients, new regurgitation, leaflet thickening, restricted leaflet motion, paravalvular jets, vegetation-like masses, and evidence of valve rocking should prompt further evaluation. Transoesophageal echocardiography is essential whenever transthoracic imaging is suggestive but not definitive, especially in suspected endocarditis or mitral prosthesis dysfunction [53,61].

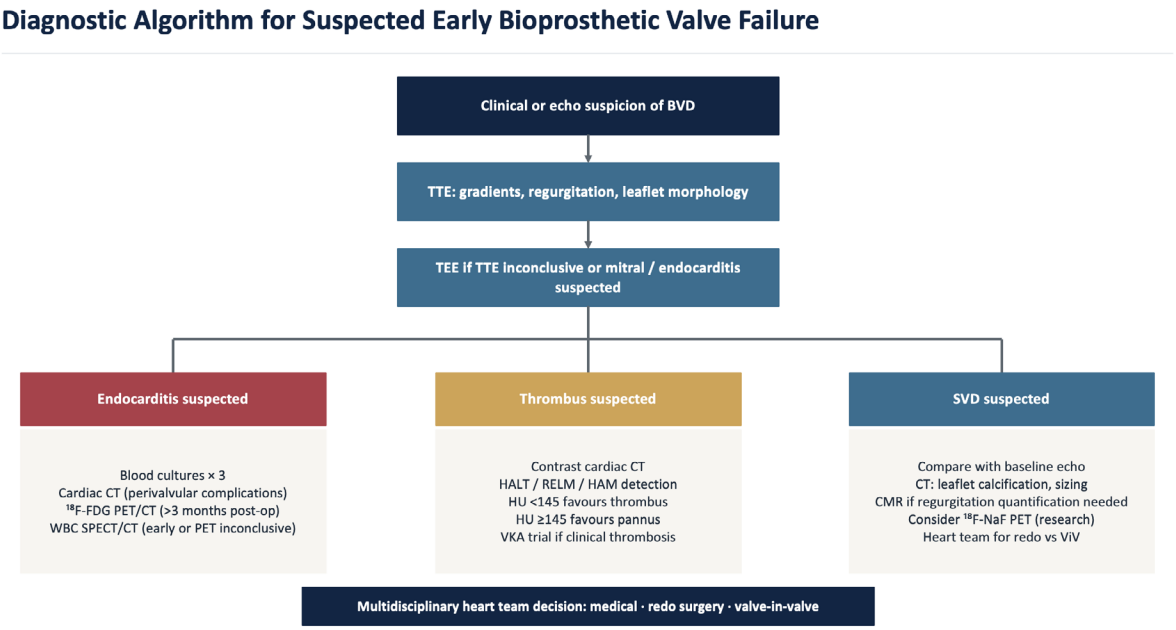

Figure 7: Suggested diagnostic algorithm for suspected early bioprosthetic valve failure. Transthoracic echocardiography is the first-line investigation, with escalation to transoesophageal echocardiography, cardiac CT, and nuclear imaging (^{18}F-FDG PET/CT, WBC SPECT/CT) based on the clinical question and initial findings. This figure should be considered the central framework of the manuscript, integrating the temporal-mechanistic approach with the multimodality imaging algorithm.

Multimodality imaging extends the diagnostic reach. Cardiac computed tomography is particularly valuable for distinguishing thrombus from pannus (Figure 6), identifying HALT, characterising perivalvular complications, and planning reintervention [11,45,46,54]. Cardiac magnetic resonance, while limited by metallic artefact, provides accurate quantification of regurgitation severity and ventricular remodelling [62]. Nuclear imaging, particularly ^{18}F-FDG PET/CT and WBC SPECT/CT, has become indispensable in the diagnostic algorithm for prosthetic endocarditis [52,55,56]. The 2023 ESC guidelines have explicitly incorporated FDG-PET/CT into the major Duke criteria for late prosthetic valve endocarditis, recognising that abnormal prosthetic or periprosthetic uptake can support the diagnosis, while cautioning that postoperative inflammation in the early weeks after surgery requires interpretive caution [52].

Time from implant	Most likely causes	First escalation test
≤30 days	Malposition, PVL, dehiscence, acute thrombosis	TEE ± CT
30 days–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.		

Table 2: Temporal-mechanistic guide to investigation of suspected EBVF.

Management Considerations

Management of EBVF is determined by the underlying mechanism. Subclinical thrombus without haemodynamic consequence may be observed, while clinically significant thrombosis warrants vitamin K antagonist therapy with serial imaging [11,43]. Endocarditis demands prolonged targeted antibiotic therapy guided by blood cultures, with surgical intervention indicated for severe regurgitation, perivalvular extension, large vegetations, uncontrolled infection, or recurrent embolism [52,57]. Established structural valve degeneration, when symptomatic and severe, requires either redo surgery or transcatheter valve-in-valve replacement, with the choice influenced by

surgical risk, anatomy, and the risk of coronary obstruction or left ventricular outflow tract obstruction [58,59,60].

Redo surgical aortic valve replacement carries reported in-hospital mortality between 5 and 13 per cent, while transcatheter valve-in-valve achieves procedural success in approximately 95 per cent of selected high-risk patients with one-year mortality of 15 to 20 per cent for aortic and mitral procedures respectively [58,60,63]. Recent registry data have reinforced the concept that redo-SAVR and ViV-TAVR serve complementary roles within a lifetime management framework, with patient selection guided by risk profile and anatomical suitability rather than a competing treatment paradigm [63].

Heart team decision framework

The choice between redo surgery, transcatheter valve-in-valve, and continued medical therapy in EBVF is rarely straightforward, and benefits substantially from structured heart team review. Six considerations dominate the discussion: the underlying mechanism, patient surgical risk, anatomical suitability for ViV (confirmed by gated cardiac CT with attention to coronary ostial height and sinus dimensions) [59], the prosthesis label size of the original valve, the patient's life expectancy weighed against durability of a second-tier intervention, and mitral-specific screening for left ventricular outflow tract obstruction [58].

Factor	Favours redo surgery	Favours valve-in-valve
Patient age and life expectancy	Younger (<65 years), good longevity expected	Older (>75 years), limited life expectancy
Surgical risk	Low to intermediate (STS <4%)	High or prohibitive (STS >8%)
Original valve size	Small (≤21 mm) with risk of severe ViV PPM	Adequate (≥23 mm aortic)
Coronary ostial height (CT)	Low (<10 mm) — high obstruction risk for ViV	Adequate (>10 mm)
Mechanism of failure	Endocarditis with paravalvular extension; pannus; severe PVL	Pure SVD with central regurgitation or stenosis
Concomitant cardiac disease	Multiple lesions requiring surgery (e.g. CABG, mitral)	Isolated valve dysfunction
Prior sternotomy	Acceptable surgical access; minimal patent grafts at risk	Hostile re-entry; patent grafts crossing midline
Mitral position	Adequate neo-LVOT predicted; small or hostile annulus	Adequate predicted neo-LVOT (≥1.7 cm^2)
CABG, coronary artery bypass grafting; CT, computed tomography; LVOT, left ventricular outflow tract; PPM, patient–prosthesis mismatch; PVL, paravalvular leak; STS, Society of Thoracic Surgeons risk score; SVD, structural valve degeneration; ViV, valve-in-valve.		

Table 3: Heart team factors favouring redo surgery versus transcatheter valve-in-valve in early bioprosthetic valve failure.

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) [26,27]. Tissue engineering aimed at decellularisation, removal of α-gal antigens using GTKO porcine donors, and improved fixation chemistries may diminish the immune contribution to structural failure [29,30]. 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 [33,34,11]. Refinements in transcatheter valve design, including reduced-profile delivery systems, improved sealing skirts, and durable polymer leaflets, hold promise for both primary and valve-in-valve procedures [4,17,28].

Conclusion

Early bioprosthetic valve failure is multifactorial, encompassing accelerated structural degeneration, thrombosis, endocarditis, and non-structural mechanisms including patient–prosthesis mismatch and procedural complications. The clinical phenotype is often non-specific, and the timely recognition of the underlying mechanism depends on systematic multimodality imaging (Figure 7) and a high index of suspicion. As bioprosthetic valves are increasingly implanted in younger and lower-risk patients, the early identification, characterisation, and treatment of EBVF will assume growing clinical importance.

Three principles deserve emphasis. First, the temporal phase of failure provides important diagnostic guidance (Figure 2, Table 3): very early failure within 30 days is almost always procedural, infective and thrombotic mechanisms dominate the first year, and accelerated structural degeneration becomes prominent thereafter. Second, no single imaging modality is sufficient. Transthoracic and transoesophageal echocardiography, cardiac CT (Figure 6), cardiac magnetic resonance, and nuclear imaging each contribute distinct information, and the diagnostic algorithm must remain adaptive to the clinical question. Third, treatment decisions in EBVF are not simply technical but profoundly strategic (Table 2), with implications extending beyond the immediate intervention to the patient's lifetime trajectory of valve replacement; these decisions are best made within a structured heart team framework that integrates surgical, interventional, imaging, and infectious disease expertise.

Acknowledgements

The authors acknowledge the multidisciplinary heart team at the Wessex Cardiac Centre for the clinical experience that has informed this review.

Author contributions

SKO conceived the review and drafted the manuscript. SA contributed to manuscript drafting, critical revision for important intellectual content, and final approval. All authors agree to be accountable for all aspects of the work.

Conflicts of interest: none declared.

Funding: none.

What This Review Adds

What is already known	What this review adds
Late SVD is common after bioprosthetic replacement	EBVF is a distinct syndrome with different mechanisms from late SVD
Thrombosis, endocarditis and SVD are recognised causes of valve dysfunction	Time-from-implantation can help prioritise likely aetiology and guide investigation
Imaging for prosthetic valve assessment is multimodal	This review links imaging findings to management decisions within a temporal-mechanistic framework

References

1. Brown JM, O'Brien SM, Wu C, Sikora JAH, Griffith BP, et al. (2009) Isolated aortic valve replacement in North America comprising 108,687 patients in 10 years: changes in risks, valve types, and outcomes in the Society of Thoracic Surgeons National Database. J Thorac Cardiovasc Surg.137:82–90.
2. Goldstone AB, Chiu P, Baiocchi M, Lingala B, Patrick WL, et al. (2017) Mechanical or biologic prostheses for aortic-valve and mitral-valve replacement. N Engl J Med. 377:1847–1857.
3. Rodriguez-Gabella T, Voisine P, Puri R, Pibarot P, Rodés-Cabau J (2017) Aortic bioprosthetic valve durability: incidence, mechanisms, predictors, and management of surgical and transcatheter valve degeneration. J Am Coll Cardiol. 70:1013–1028.
4. Mack MJ, Leon MB, Thourani VH, Pibarot P, Hahn RT, et al. (2023) Transcatheter aortic-valve replacement in low-risk patients at five years. N Engl J Med. 389:1949–1960.
5. Kostyunin AE, Yuzhalin AE, Rezvova MA, Ovcharenko EA, Glushkova TV, et al. (2020) Degeneration of bioprosthetic heart valves: update 2020. J Am Heart Assoc. 9:e018506.
6. Persson M, Glaser N, Nilsson J, Friberg Ö, Franco-Cereceda A, et al. (2022) Comparison of long-term performance of bioprosthetic aortic valves in Sweden from 2003 to 2018. JAMA Netw Open. 5:e220962.
7. Belluschi I, Buzzatti N, Castiglioni A, De Bonis M, Maisano F, et al. (2021) Aortic and mitral bioprosthetic valve dysfunction: surgical or percutaneous solutions? Eur Heart J Suppl. 23:E6–E12.
8. Sá MPBO, Van den Eynde J, Simonato M, Cavalcanti LRP, Doulamis IP, et al. (2021) Valve-in-valve transcatheter aortic valve replacement versus redo surgical aortic valve replacement: an updated meta-analysis. JACC Cardiovasc Interv.14:211–20.
9. Thyregod HGH, Jørgensen TH, Ihlemann N, Steinbrüchel DA, Nissen H, et al. (2024) Transcatheter or surgical aortic valve implantation: 10-year outcomes of the NOTION trial. Eur Heart J. 45:1116–1124.
10. Alaour B, Tomii D, Nakase M, Heg D, Stortecky S, et al. (2025) Hemodynamic valve deterioration after transcatheter aortic valve replacement: incidence, predictors, and clinical outcomes. JACC Cardiovasc Interv. 18:72–85.
11. Makkar RR, Blanke P, Leipsic J, Thourani V, Chakravarty T, et al. (2020) Subclinical leaflet thrombosis in transcatheter and surgical

bioprosthetic valves: PARTNER 3 cardiac CT substudy. J Am Coll Cardiol. 75:3003–3015.

12. Anantha-Narayanan M, Reddy YNV, Sundaram V, Murad MH, Erwin PJ, et al. (2020) Endocarditis risk with bioprosthetic and mechanical valves: systematic review and meta-analysis. Heart.106:1413–1419.
13. Moriyama N, Laakso T, Biancari F, Raivio P, Jalava MP, et al. (2019) Prosthetic valve endocarditis after transcatheter or surgical aortic valve replacement with a bioprosthesis: results from the FinnValve registry. EuroIntervention.15:e500–e507.
14. Pibarot P, Dumesnil JG (2006) Prosthesis-patient mismatch: definition, clinical impact, and prevention. Heart. 92:1022–1029.
15. Head SJ, Mokhles MM, Osnabrugge RLJ, Pibarot P, Mack MJ, et al. (2012) The impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: a systematic review and meta-analysis of 34 observational studies comprising 27 186 patients with 133 141 patient-years. Eur Heart J. 33:1518–1529.
16. Pibarot P, Herrmann HC, Wu C, Hahn RT, Otto CM, et al. (2022) Standardized definitions for bioprosthetic valve dysfunction following aortic or mitral valve replacement: JACC state-of-the-art review. J Am Coll Cardiol. 80:545–561.
17. Fazzari F, Baggiano A, Fusini L, Ghulam Ali S, Gripari P, et al. (2023) Early biological valve failure: structural valve degeneration, thrombosis, or endocarditis? J Clin Med.12:5740.
18. Capodanno D, Petronio AS, Prendergast B, Eltchaninoff H, Vahanian A, et al. (2017) Standardized definitions of structural deterioration and valve failure in assessing long-term durability of transcatheter and surgical aortic bioprosthetic valves: a consensus statement from the European Association of Percutaneous Cardiovascular Interventions (EAPCI) the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Eur J Cardiothorac Surg. 52:408–417.
19. Généreux P, Piazza N, Alu MC, Nazif T, Hahn RT, et al. (2021) Valve Academic Research Consortium 3: updated endpoint definitions for aortic valve clinical research. Eur Heart J. 42:1825-1857.
20. Salaun E, Clavel MA, Rodés-Cabau J, Pibarot P (2018) Bioprosthetic aortic valve durability in the era of transcatheter aortic valve implantation. Heart.104:1323–1332.
21. Dvir D, Bourguignon T, Otto CM, Hahn RT, Rosenhek R, et al. (2018) Standardized definition of structural valve degeneration for surgical and transcatheter bioprosthetic aortic valves. Circulation 137:388–399.
22. Bourguignon T, Bouquiaux-Stablo AL, Candolfi P, Mirza A, Loardi C, et al. (2015) Very long-term outcomes of the Carpentier-Edwards Perimount valve in aortic position. Ann Thorac Surg. 99: 831–837.
23. Mahjoub H, Mathieu P, Larose E, Dahou A, Sénéchal M, et al. (2015) Determinants of aortic bioprosthetic valve calcification assessed by multidetector CT. Heart. 101:472–477.
24. Schoen FJ, Levy RJ (2005) Calcification of tissue heart valve substitutes: progress toward understanding and prevention. Ann Thorac Surg. 79:1072–1080.
25. Nair V, Law KB, Li AY, Phillips KRB, David TE, et al. (2012) Characterizing the inflammatory reaction in explanted Medtronic Freestyle stentless porcine aortic bioprosthesis over a 6-year period. Cardiovasc Pathol. 21:158–168.
26. Bartus K, Litwinowicz R, Kuśmierczyk M, Bilewska A, Bochenek M, et al. (2018) Primary safety and effectiveness feasibility study after surgical aortic valve replacement with a new generation bioprosthesis: one-year outcomes. Kardiol Pol. 76:618–624.
27. Beaver T, Bavaria JE, Griffith B, Svensson LG, Pibarot P, et al. (2024) Seven-year outcomes following aortic valve replacement with a novel tissue bioprosthesis. J Thorac Cardiovasc Surg.168:781–791.
28. Sellers SL, Turner CT, Sathananthan J, Cartlidge TRG, Sathananthan G, et al. Transcatheter aortic heart valves: histological analysis providing insight into structural valve degeneration. JACC Cardiovasc Imaging.12:135–145.
29. Senage T, Paul A, Le Tourneau T, Fellah-Hebia I, Vadori M, et al. (2022) The role of antibody responses against glycans in bioprosthetic heart valve calcification and deterioration. Nat Med. 28:283–294.
30. Manji RA, Lee W, Cooper DKC (2015) Xenograft bioprosthetic heart valves: past, present and future. Int J Surg. 23:280–284.
31. Côté N, Pibarot P, Clavel MA (2017) Incidence, risk factors, clinical impact, and management of bioprosthesis structural valve degeneration. Curr Opin Cardiol. 32:123–129.
32. Pibarot P, Ternacle J, Jaber WA, Salaun E, Dahou A, et al. (2020) Structural deterioration of transcatheter versus surgical aortic valve bioprostheses in the PARTNER-2 trial. J Am Coll Cardiol. 76:1830–1843.
33. Cartlidge TRG, Doris MK, Sellers SL, Pawade TA, White AC, et al. (2019) Detection and prediction of bioprosthetic aortic valve degeneration. J Am Coll Cardiol. 73:1107–1119.
34. Kwiecinski J, Tzolos E, Cartlidge TRG, Fletcher A, Doris MK, et al. (2021) Native aortic valve disease progression and bioprosthetic valve degeneration in patients with transcatheter aortic valve implantation. Circulation.144:1396–1408.
35. Werner P, Gritsch J, Scherzer S, Gross C, Russo M, et al. (2021) Structural valve deterioration after aortic valve replacement with the Trifecta valve. Interact Cardiovasc Thorac Surg. 32:39–46.
36. Hirji SA, Ramirez-Del Val F, Kolkailah AA, Ejiofor JI, McGurk S, et al. (2022) Outcomes of surgical aortic valve replacement with a Trifecta bioprosthesis at five years. J Thorac Cardiovasc Surg.164:1738–1747.
37. United States Food and Drug Administration (2023) Abbott Trifecta valves: potential risk of early structural valve deterioration — letter to health care providers. February 27, 2023. Updated July 31, 2023.
38. Sellers SL, Sathananthan J, Bouchareb R, Mostaco-Guidolin LB, Lau KPL, et al. (2022) Impact of over-expansion on SAPIEN 3 leaflet integrity. Cardiovasc Pathol. 57:107397.
39. Hatoum H, Yousefi A, Lilly S, Maureira P, Crestanello J, et al. (2018) An in vitro evaluation of turbulence after transcatheter aortic valve implantation. J Thorac Cardiovasc Surg. 156:1837–1848.
40. Pibarot P, Hahn RT, Weissman NJ, Arsenault M, Beaudoin J, et al. (2017) Association of paravalvular regurgitation with 1-year outcomes after transcatheter aortic valve replacement with the SAPIEN 3 valve. JAMA Cardiol. 2:1208–1216.
41. Dangas GD, Weitz JI, Giustino G, Makkar R, Mehran R (2016) Prosthetic heart valve thrombosis. J Am Coll Cardiol. 68:2670–2689.
42. Chakravarty T, Søndergaard L, Friedman J, De Backer O, Berman D, et al. (2017) Subclinical leaflet thrombosis in surgical and transcatheter bioprosthetic aortic valves: an observational study. Lancet 389:2383–2392.
43. Shahinian JH, Chan V, Pislaru SV (2022) Chronic thrombosis of bioprostheses: diagnosis and management. Prog Cardiovasc Dis.72:15–20.

44. Bonnefous L, Kharoubi M, Bézard M, Oghina S, Galat A, et al. (2021) Predictive factors of leaflet thrombosis after TAVR. JACC Cardiovasc Imaging.14:1525–1527.

45. Jilaihawi H, Asch FM, Manasse E, Ruiz CE, Jelnin V, et al. (2017) Systematic CT methodology for the evaluation of subclinical leaflet thrombosis. JACC Cardiovasc Imaging.10:461–470.

46. Gündüz S, Özkan M, Kalçik M, Gürsoy OM, Astarcioglu MA, et al. (2015) Sixty-four-section cardiac computed tomography in mechanical prosthetic heart valve dysfunction: thrombus or pannus. Circ Cardiovasc Imaging. 8: e003246.

47. Dangas GD, Tijssen JGP, Wöhrle J, Søndergaard L, Gilard M, et al. (2020) A controlled trial of rivaroxaban after transcatheter aortic-valve replacement. N Engl J Med. 382:120–129.

48. Collet JP, Van Belle E, Thiele H, Berti S, Lhermusier T, et al. (2022) Apixaban vs. standard of care after transcatheter aortic valve implantation: the ATLANTIS trial. Eur Heart J. 43:2783–2797.

49. Wang A, Athan E, Pappas PA, Fowler VG, Olaison L, et al. (2007) Contemporary clinical profile and outcome of prosthetic valve endocarditis. JAMA. 297:1354–1361.

50. Alexis SL, Malik AH, George I, Hahn RT, Khalique OK, et al. (2020) Infective endocarditis after surgical and transcatheter aortic valve replacement: a state of the art review. J Am Heart Assoc. 9:e017347.

51. Erba PA, Pizzi MN, Roque A, Salaun E, Lancellotti P, et al. (2019) Multimodality imaging in infective endocarditis: an imaging team within the endocarditis team. Circulation.140:1753–1765.

52. Delgado V, Ajmone Marsan N, de Waha S, Bonaros N, Brida M, et al. (2023) 2023 ESC guidelines for the management of endocarditis. Eur Heart J. 44:3948–4042.

53. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, et al. (2015) 2015 ESC guidelines for the management of infective endocarditis. Eur Heart J. 36:3075–3123.

54. Saeedan MB, Wang TKM, Cremer P, Wahadat AR, Budde RPJ, et al. (2021) Role of cardiac CT in infective endocarditis: current evidence, opportunities, and challenges. Radiol Cardiothorac Imaging. 3:e200378.

55. Pizzi MN, Roque A, Fernández-Hidalgo N, Cuéllar-Calabria H, Ferreira-González I, et al. (2015) Improving the diagnosis of infective endocarditis in prosthetic valves and intracardiac devices with 18F-FDG PET/CT angiography. Circulation.132:1113–1126.

56. Rouzet F, Chequer R, Benali K, Lepage L, Ghodbane W, et al. (2014) Respective performance of 18F-FDG PET and radiolabeled leukocyte scintigraphy for the diagnosis of prosthetic valve endocarditis. J Nucl Med. 55:1980–1985.

57. Habib G, Erba PA, Iung B, Donal E, Cosyns B, et al. (2019) Clinical presentation, aetiology and outcome of infective endocarditis. Results of the ESC-EORP EURO-ENDO (European infective endocarditis) registry: a prospective cohort study. Eur Heart J. 40:3222–3232.

58. Tarantini G, Sathananthan J, Fabris T, Landes U, Bapat VN, et al. (2022) Transcatheter aortic valve replacement in failed transcatheter bioprosthetic valves. JACC Cardiovasc Interv. 15:1777–1793.

59. Blanke P, Weir-McCall JR, Achenbach S, Delgado V, Hausleiter J, et al. (2019) Computed tomography imaging in the context of transcatheter aortic valve implantation: an expert consensus document of the Society of Cardiovascular Computed Tomography. J Cardiovasc Comput Tomogr. 13:1-20.

60. Ribeiro HB, Rodés-Cabau J, Blanke P, Leipsic J, Kwan Park J, et al. (2018) Incidence, predictors, and clinical outcomes of coronary obstruction following transcatheter aortic valve replacement for degenerative bioprosthetic surgical valves: insights from the VIVID registry. Eur Heart J. 39:687–695.

61. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, et al. (2022) 2021 ESC/EACTS guidelines for the management of valvular heart disease. Eur Heart J. 43:561–632.

62. Bohbot Y, Renard C, Manrique A, Levy F, Maréchaux S, et al. (2020) Usefulness of cardiac magnetic resonance imaging in aortic stenosis. Circ Cardiovasc Imaging. 13:e010356.

63. Hirji SA, Percy ED, Zogg CK, Malarczyk A, Harloff MT, et al. (2020 Comparison of in-hospital outcomes and readmissions for valve-in-valve transcatheter aortic valve replacement vs. reoperative surgical aortic valve replacement: a contemporary assessment of real-world outcomes. Eur Heart J. 41:2747–2755.