Archives of Cardiovascular Disease 116 (2023) 159–166

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Review

Infective endocarditis after transcatheter pulmonary valve implantation in patients with congenital heart disease: Distinctive features

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HIGHLIGHTS

- IE is a feared complication of TPVI that affects valve durability and outcomes.
- IE following TPVI in CHD exhibits several distinctive features.
- Several risk factors are associated with IE.
- Patient and parent education on IE prevention should be provided.

ARTICLE INFO

Article history:
Received 23 January 2023
Received in revised form 25 January 2023
Accepted 27 January 2023
Available online 17 February 2023

Keywords:
Pulmonary valve
Infective endocarditis
Congenital heart disease
Infections
Interventional cardiology

ABSTRACT

The introduction of transcatheter pulmonary valve implantation (TPVI) has greatly benefited the management of right ventricular outflow tract dysfunction. Infective endocarditis (IE) is a feared complication of TPVI that affects valve durability and patient outcomes. Current recommendations provide only limited guidance on the management of IE after TPVI (TPVI-IE). This article, by a group of experts in congenital heart disease in children and adults, interventional cardiology, infectious diseases including IE, and microbiology, provides a comprehensive review of the current evidence on TPVI-IE, including its incidence, risk factors, causative organisms, diagnosis, and treatment. The incidence of TPVI-IE varies from 13–91/1000 person-years for Melody valves to 8–17/1000 person-years for SAPIEN valves. Risk factors include history of IE, DiGeorge syndrome, immunosuppression, male sex, high residual transpulmonary gradient and portal of bacteria entry. Staphylococci and streptococci are the most common culprits, whereas *Staphylococcus aureus* is associated with the most severe disease. In addition to the modified Duke criteria, a high residual gradient warrants a strong suspicion. Imaging studies are helpful for the diagnosis. Intravenous antibiotics guided by blood culture results are the mainstay of treatment. Invasive re-intervention may be required. TPVI-IE in patients with congenital heart disease exhibits several distinctive features. Whether specific valve types are associated with a higher risk of TPVI-IE requires further investigation. Patient and parent education regarding IE prevention may have a role to play and should be offered to all patients.

Abbreviations: CHD, congenital heart disease; IE, infective endocarditis; PET-CT, positron emission tomography-computed tomography; RVOT, right ventricular outflow tract; TAVI, transcatheter aortic valve implantation; TEE, transoesophageal echocardiography; TPVI, transcatheter pulmonary valve implantation; TTE, transthoracic echocardiography.

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https://doi.org/10.1016/j.acvd.2023.01.008
1875-2136/

1. Introduction

Congenital heart disease (CHD) is the most common congenital abnormality, affecting nearly 1% of neonates [1]. The therapeutic advances achieved in recent decades have greatly increased the life expectancy of patients with CHD [2]. A complication that continues to raise challenges is infective endocarditis (IE), whose incidence is increased up to 100-fold compared with the general population, due to the frequent need for intracardiac prosthesis implantation [3–6]. In CHD such as conotruncal defects, the right ventricular outflow tract (RVOT) is reconstructed using bovine jugular vein conduits, homografts, prosthetic valved conduits and/or bioprosthetic valves. Subsequently, pulmonary regurgitation, conduit degeneration or conduit mismatch due to growth may require repeated valve and/or conduit replacement. The introduction in 2000 of transcatheter pulmonary valve implantation (TPVI) proved to be a major advance associated with good short- and long-term outcomes [7]. Valves available for TPVI include the balloon-expandable Melody and SAPIEN valves [8–15] and the more recent self-expandable Venus, Pulsta and Harmony valves [16–18]. IE after TPVI (TPVI-IE) remains a challenging adverse event that affects valve durability and patient outcomes [13,18–25]. However, national and international recommendations provide only meagre guidance about the management of TPVI-IE in patients with CHD [26,27].

The aim of this review was to describe the features of TPVI-IE in patients with CHD. We evaluated the incidence, risk factors, diagnosis, microbiological characteristics and outcomes of this form of IE and looked for differences with IE complicating other conditions.

2. Methods

A group of experts in congenital heart disease in children and adults, interventional cardiology, infectious diseases including IE, and microbiology examined the data concerning IE after TPVI, transcatheter aortic valve implantation (TAVI), surgical prosthetic valve replacement and on cardiac implantable electronic devices and complex CHD, retrieved by searching PubMed, and provide a comprehensive review of current evidence. Only articles in English published after 2000, year of the first publication of TPVI, were considered. Keywords included pulmonary valve, infective endocarditis, congenital heart disease, Melody valve, Sapien valve, transcatheter pulmonary valve implantation and percutaneous pulmonary valve implantation.

3. Incidence of TPVI-IE

Table 1 reports the incidence of TPVI-IE and, for comparison, of IE in various other cardiac conditions. Although exceedingly rare with the native pulmonary valves, the estimated incidence for transcatheter-implanted prosthetic pulmonary valves is 16–27/1000 person-years [8,20,24,28,29]. There are significant differences according to patient features and type of valve and materials used (e.g. from 13–91/1000 person-years for Melody valves and 8–17/1000 person-years for SAPIEN valves [8,9,13,20,23,24,28]). Data for Venus valves remain limited but the 1-year incidence has ranged from 0 – 73/1000 person-years [16,18]. IE on surgically implanted homografts and Contegra® bovine-jugular-vein conduits has an estimated incidence of 2–2.7 and 10–11.2/1000 person-years, respectively [30,31].

4. Risk factors for TPVI-IE

The Graphical Abstract and Table 2 list the risk factors for TPVI-IE.

Table 1
Incidence of infective endocarditis after transcatheter pulmonary valve implantation and in various cardiac conditions [3,13,20,23,24,26–32,47,58,90,92,94,99].

Patient characteristics	Incidence of IE per 1000 patient-years
Congenital heart diseases	0.4–0.7
Complex congenital heart disease	1.5
Congenital heart diseases with intracardiac devices	1.3
Transcatheter prosthetic pulmonary valves	16–27
Melody valves	13–91
Sapien valves	8–17
Venus valves	0–73 *(limited data)*
Surgical prosthetic pulmonary valves	
Contegra	10–11
Homograft	2–3
Bioprosthetic valves	3–15
General population	0.03–0.08
Surgical prosthetic valves	3–12
Cardiac implantable electronic devices	2–5
Transcatheter aortic valve implantation	7–30

Table 2
Factors that influence the risk of infective endocarditis after transcatheter pulmonary valve implantation.

Confirmed risk factors	
History of infection	History of infective endocarditis
Genetic syndrome	DiGeorge syndrome (22q11.2 deletion)
Comorbidities	Human immunodeficiency virus, chronic neutropenia, immunosuppressive therapy
Sex	Male
Postprocedural factors	Increased residual gradient
Portal of bacteria entry	cutaneous or oral infections, dental procedures, oral trauma, intravenous therapy or drug abuse
Controversial risk factors	
Type of prosthesis	Bovine jugular vein valves (Contegra and Melody)
Role of valve thrombosis	Discontinuation of antithrombotic therapy
Protective factors	
Dental care	Oral hygiene measures, prophylactic antibiotic therapy
Educational programme	Skin hygiene, alert card, knowledge of risk factors and symptoms of infective endocarditis, knowledge of appropriate response to symptoms

4.1. Previous IE

A history of IE is a strong and independent risk factor for subsequent IE, as observed for all cases of IE [24,32–34]. In patients with prosthetic valves, a history of IE may increase the risk 400-fold [35]. Individual characteristics of immune responses, frequent healthcare use and presence of an implanted cardiac device may affect the risk of recurrent IE [36].

4.2. Comorbidities and genetic syndromes

Cyanotic CHD is associated with a higher risk of IE [37,38]. However, because TPVI is nearly always performed after surgical repair to relieve the cyanosis, cyanotic CHD is not a risk factor for TPVI-IE.

CHD is common in several genetic syndromes including trisomy 21 and DiGeorge syndrome. Trisomy 21 is not among the known risk factors for IE [39]. One feature of DiGeorge syndrome (22q11.2 deletion) is thymic hypoplasia, with immunodeficiency in up to 75% of patients [40]. Conotruncal defects are common and TPVI is therefore often required. DiGeorge syndrome is associated with a higher risk of TPVI-IE [41]. Additionally, the degree of learning disability associated with such genetic syndromes may decrease compliance with optimal dental hygiene and care as well as other preventive measures for IE. Immunodeficiencies increase the risk of IE, notably

after TPVI [23,24,40,42]. Causes of immunosuppression in CHD include DiGeorge syndrome, human immunodeficiency virus infection, immunosuppressive therapy and chronic neutropenia [40]. Genetic causes may be underdiagnosed [40]. Comorbidities associated with relative immune-system compromise (renal failure, diabetes mellitus, corticosteroid treatment or haematoma formation) are uncommon in young patients with CHD [40,43–45].

4.3. Male sex

About two-thirds of TPVI-IE cases were in males [20,23,33]. One possible explanation is the higher frequency of CHD in males. However, in other cardiac conditions IE is also more common in males, suggesting a protective effect of oestrogens [46].

4.4. Age

Assessment of relationships between age and IE risk are biased by several factors. Although CHD often requires invasive cardiac procedures in childhood, TPVI is performed percutaneously through large sheaths and is therefore primarily used for adults and older children weighing more than 20 kg. Most self-expandable valves and the SAPIEN valve are larger than the Melody valve, explaining the younger age at TPVI-IE with the Melody valve. Most studies on TPVI-IE included patients older than 15 years [24,47], and a median age of 18 years has been reported in patients with TPVI-IE [23,48]. Nonetheless, the risk of TPVI-IE may be higher before 12 years of age [24,33]. Variable adherence to hygiene measures in adolescence may increase the risk of having dental or cutaneous portals of bacteria entry and, therefore, of developing IE [24,48–50].

4.5. Type of TPVI material and other implants

The risk of IE is higher with valved than non-valved RVOT conduits [34] and perhaps with bovine-jugular-vein conduits compared with other materials [19,30,51–55].

The incidence of IE after TPVI is similar overall to what is reported after surgical valve and conduit implantation [28,51,56]. Whether the type of transcatheter pulmonary valve influences the risk of IE is controversial, as studies of Melody and SAPIEN valves have produced conflicting results [13,21,23,24,40,42,57]. Despite the shorter follow-up with the pulmonary SAPIEN valves, cases of IE have been documented [25]. The smaller diameters of Melody valves compared with SAPIEN valves has been suggested as a confounding factor explaining these discrepancies. Few data are available for the more recent self-expandable valves [18].

4.6. TPVI-related factors

A high residual transpulmonary gradient is a significant risk factor for IE [24,58,59]. Achieving a low RVOT gradient is therefore a key goal of TPVI [13,41,47,48,60]. Bacterial inoculation during TPVI seems exceedingly rare. Balloon post-dilation may induce valve damage, facilitating bacterial adhesion [61,62].

4.7. Portal of bacteria entry

An infectious episode, atopic dermatitis, skin wounds, nail biting, tattooing and body piercing may increase the risk of IE, notably via *Staphylococcus aureus* bacteraemia IE [33,48,50,63,64]. Dental procedures and poor dental hygiene, and smoking are also risk factors, and prophylactic antibiotic therapy is indicated in patients after TPVI when dental care or other invasive procedures are required [26,27,41,48,65,66]. Intravenous line placement and surgery at any site carry a risk of nosocomial staphylococcal infection [67,68]. Other portals of bacteria entry such as those in the gastrointestinal tract are less common in young patients undergoing TPVI for CHD. Patient education about bacteraemia prevention, notably via good dental and skin care, good diet and smoking avoidance, should be provided.

4.8. Thrombosis

Thrombus formation after TPVI may promote bacterial adhesion to the prosthetic material, and IE may worsen thrombosis [58]. Antithrombotic therapy is usually given after TPVI, although for highly variable durations. Aspirin discontinuation may be associated with an increased risk of IE [41]. Early discontinuation of aspirin may increase the risk of thrombosis [58,69].

5. Bacterial adhesion and biofilm formation

Bacteria can adhere to prosthetic valves and particularly after TPVI via several mechanisms [61,70,71], although further studies are needed to clarify inconsistencies in study results [72]. Bacteria may coat the prosthetic material with a polysaccharide matrix, or biofilm, which limits antibiotic penetration and boosts pathogen virulence. *S. aureus* may be particularly prone towards producing biofilm formation [73]. Bacteria within biofilms may enter a state of latency characterized by decreased antibiotic susceptibility and may express biofilm-specific antibiotic-resistance genes. Current guidelines for IE antibiotic therapy specify which drugs penetrate best within biofilm [74].

6. Diagnosing infective endocarditis

IE is diagnosed based on the modified Duke criteria established in the general population and described elsewhere [26]. No criteria specific for patients with TPVI for CHD exist.

6.1. Cardiac imaging

Transthoracic echocardiography (TTE) and transoesophageal echocardiography (TEE) are the first-line imaging modalities. However, as in other cases of prosthetic valve IE, particularly in transcatheter-implanted prostheses, TTE and TEE findings are more frequently inconclusive than for IE on native valves. Positron emission tomography-computed tomography (PET-CT) can improve the diagnosis of IE and is recommended when IE is considered as possible or excluded despite clinical suspicion [26]. Intracardiac echocardiography is occasionally used to visualize vegetations [20,47,57,75]. The estimated sensitivity and specificity of echocardiography for diagnosing IE are 72% and 74%, respectively [12,28,33,75]. However, achieving a definitive diagnosis of TPVI-IE by TTE and TEE is challenging, and vegetations are difficult to visualize on prosthetic valves [48]. The type of CHD and anterior position of the pulmonary valve may also raise challenges with TTE/TEE, and the vegetations may be atypical [48]. With valved conduits, IE may produce acute RVOT obstruction rather than mobile vegetations. A rapid increase in the transpulmonary gradient, although not a Duke criterion, is more common than new valve regurgitation in TPVI-IE and, when combined with signs of infection, strongly suggests IE [12,31,41]. These specificities of IE after TPVI may diminish the ability of echocardiography to make a definite diagnosis [12,28,33,57,75]. The European Society of Cardiology recommends repeated echocardiography in patients with a strong suspicion of IE but negative imaging [26]. However, even repeated TTE/TEE may have less than 50% sensitivity in patients with prosthetic valve IE, supporting the use of other imaging techniques such as injected computed tomography scan, magnetic resonance imaging and PET-CT [20,26,57,76]. The latter is particularly useful [5,26]

Table 3
Microbiological epidemiology of infective endocarditis after transcatheter pulmonary valve implantation.

Staphylococci (33–54%)		
	Staphylococcus aureus	63–72%
	Coagulase–negative staphylococci	28–37%
Streptococci (26–50%)		
	Viridans-group streptococci	81%
	Other streptococci	19%
	Enterococcus faecalis	Exceptional
HACCEK (*Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Capnocytophaga*, *Eikenella*, *Kingella*) bacteria) (3–13%)		
Other microorganisms (13–20%)		
Oral flora	*Abiotrophia defectiva* *Rothia dentocariosa* *Aerococcus viridans*	
Cutaneous flora	*Corynebacteria* spp. *Cutibacterium acnes*	
Other	*Escherichia coli* *Klebsiella* spp. *Acinetobacter* spp. *Bartonella henselae* *Coxiella burnetti*	Case reports
Fungi	*Aspergillus fumigatus* *Candida albicans*	
Negative blood cultures (1–11%)		

in case of multiple abscesses or pseudoaneurysms [77,78] and can be considered at an early stage [79]. However, Dacron material may impair PET-CT performance [77,78]. Magnetic resonance imaging may also improve IE diagnosis by showing vegetations, valve obstruction, cardiac abscesses, the myocardial inflammatory process and mycotic aneurysm [76].

6.2. Microbiological tests

Positivity of at least two blood cultures is a major Duke criterion [26]. Four to six 10-mL bottles should be collected to increase sensitivity, as soon as IE is suspected, without waiting for a fever peak [26]. When a single culture is positive, three minor Duke criteria are required to diagnose IE [26,48]. Among patients with CHD and intracardiac prosthetic material, nearly 11% of IE cases were blood-culture negative [34]. Antibiotic initiation before sampling, slow-growing bacteria and prosthetic instead of native valves are associated with higher proportions of culture-negative cases [80]. Cultures should be kept for at least 10 days to ensure that slow-growing organisms are detected. When cultures are negative, polymerase chain reaction testing for 16S RNA can be performed on blood samples or tissue samples collected during surgery, and serological and histological tests can also help [48,81].

7. Causative organisms

TPVI-IE is caused chiefly by Gram-positive bacteria, with staphylococci and streptococci accounting for over two-thirds of cases [24]. Table 3 lists the main culprits.

7.1. Staphylococci

S. aureus causes over one-third of TPVI-IE cases and is associated with a high risk of embolization, septic shock, respiratory distress, renal failure, skin necrosis and death. Obstruction of the pulmonary valve or conduit may occur, causing right ventricular dysfunction and/or septic shock [48].

Coagulase-negative staphylococci can also cause IE. Species found in the skin flora (e.g. *S. epidermidis*, *S. lugdunensis*, and *S. capitis*) may enter the bloodstream through skin lesions [82,83].

7.2. Streptococci

Streptococci, notably viridans species, are commensals that can enter the bloodstream through nasopharyngeal and oral portals due to, for instance, poor dental hygiene or dental procedures [47,84,85]. These organisms cause up to 50% of TPVI-IE cases. Streptococci are more often community-acquired compared with staphylococci and are more common in late IE [41,47,48,84].

7.3. Enterococci

Enterococcal IE is rarely reported in patients with CHD. Cases have chiefly occurred in older patients after TAVI or CIED implantation. The main source of these bacteria is the gastrointestinal flora [86].

7.4. HACCEK (Haemophilus, Actinobacillus, Cardiobacterium, Capnocytophaga, Eikenella, Kingella) bacteria

HACCEK bacteria are nasal and pharyngeal commensals and can also be found in the gastrointestinal tract. They can adhere to cardiac prostheses. Cases of IE have occurred chiefly in patients with CHD and prosthetic valves [87,88].

7.5. Other bacteria

Less common causative pathogens include Gram-negative bacteria (e.g. Enterobacteriaceae and *Acinetobacter* sp.), Gram-positive bacilli (e.g. *Corynebacteria* sp., *Abiotrophia* sp., *Cutibacterium* sp. and *Rothia* sp.), Gram-positive cocci (e.g. *Aerococcus* sp.) and fastidious bacteria (e.g. *Bartonella* sp. and *Coxiella* sp. In immunocompromised patients, IE may be due to fungi, which carry a risk of severe complications [19,89]. Some of these uncommon bacteria may cause acute-onset IE with septic complications, reintervention or death [41,90].

8. Complications

Complications of TPVI-IE include embolism, abscesses, acute severe valve regurgitation, valve stenosis, arrhythmias and renal dysfunction [48]. After Melody-valve TPVI, severe RVOT obstruction may lead to right ventricular dysfunction and shock, with a peak-to-peak gradient above 60 mmHg in one-third of cases and reintervention in nearly two-thirds of cases. Severe pulmonary-valve stenosis with acute gradient elevation over 30 mmHg has also been described [12,28,48,75]. The high cardiac output related to sepsis may mask valve failure [48,91]. Embolization of cardiac vegetation may result in septic pulmonary emboli, mycotic pulmonary artery aneurysms and pulmonary abscesses [20,25,57]. *S. aureus* is the cause in up to half of patients with such events [26]. Perivalvular abscess, pseudoaneurysm and/or fistula formation are also among the complications of TPVI-IE [26]. Spondylodiscitis and vertebral osteomyelitis seem rare in patients with TPVI-IE.

9. Treatment

Intravenous antibiotic therapy is the main treatment of TPVI-IE in every case, and must be given for at least 6 weeks after reversion of blood-culture negativity or surgical valve explantation [28]. International guidelines for the pharmacological treatment of prosthetic valve endocarditis should be followed [26,92].

Urgent transcatheter valve dilatation or stent insertion can restore haemodynamic stability by rapidly decreasing the RVOT gradient and can obviate the need for surgery [23,41,93]. Surgery is usually required in patients with severe obstruction, uncontrolled infection or complications [24,41]. IE is the main reason for reintervention on the pulmonary valve [13,93]. Redo TPVI may be considered at a distance from the end of the treatment of TPVI-IE. Further studies are needed to determine the optimal strategy for replacing the prosthetic pulmonary valve.

10. Patient outcomes

IE-associated mortality in patients with CHD ranges from 1.9 – 16% [4,20,34,41,94,95]. These lower rates compared with other IE patient cohorts may relate to the younger age of CHD patients or its right-sided location [48,75]. In TPVI-IE, abscess formation, septic shock and severe right ventricular dysfunction are associated with higher mortality rates [24,25,94]. Furthermore, mortality is higher with *S. aureus* than streptococci, notably in the presence of valve obstruction [96]. *S. aureus* IE necessities cardiac surgery in the majority of cases [97].

TPVI-IE-related reintervention rates were reported to be 4.8% at 5 years and 10.3% at 8 years [24]. Both surgery and repeat TPVI had high mortality rates, at 8.7% and 13%, respectively [53,98].

Recurrent IE on Melody valves has occurred chiefly within the first year after the initial episode, suggesting that some cases might constitute relapses [48].

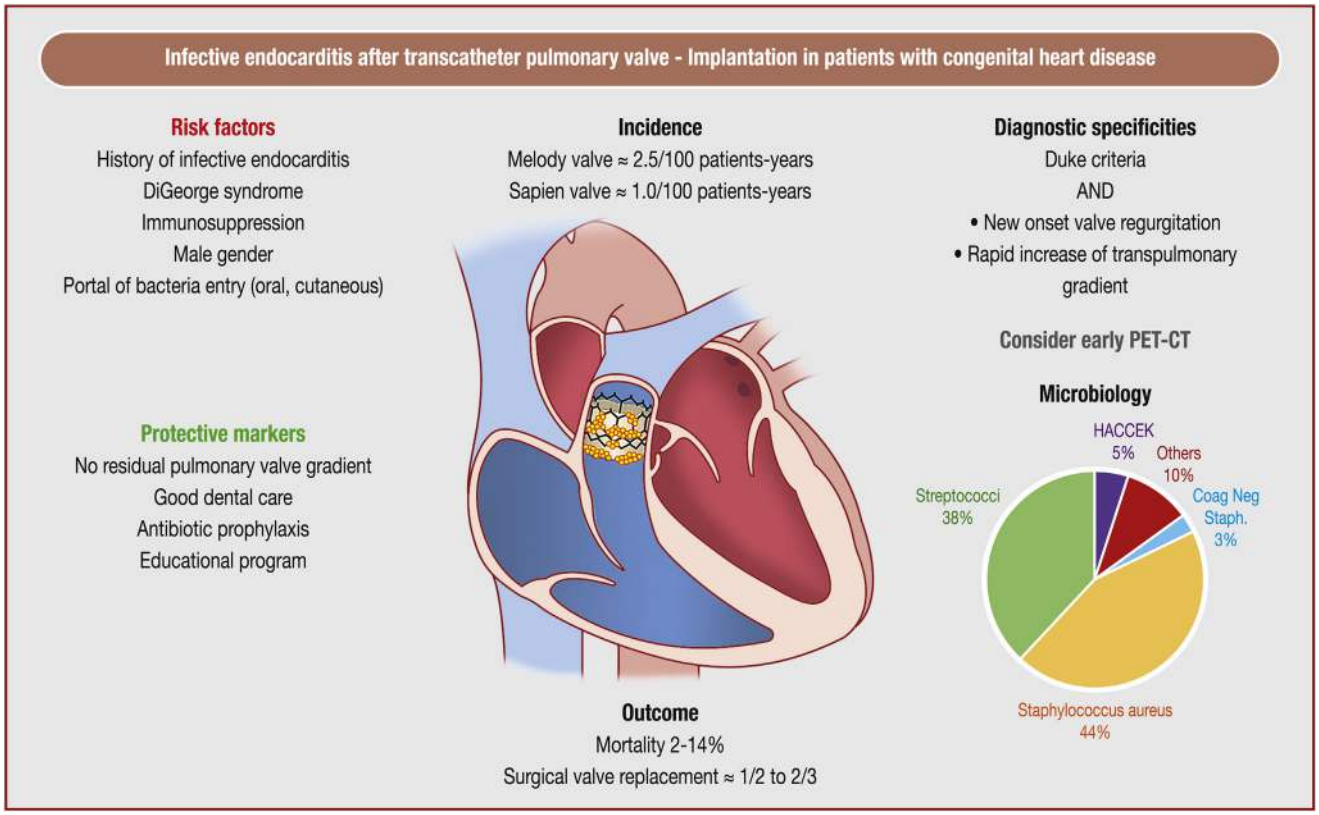
11. Conclusion and future directions

IE after TPVI is a severe event that adversely affects valve durability and patient outcomes. Genetic syndromes, immunodeficiency, previous IE and a high transpulmonary pressure gradient are major risk factors that require preventive measures including a patient/family educational programme [48,66]. Whether some valve types carry a higher risk of IE than others remains unclear, and further studies to clarify this point are urgently needed.

The performance of the modified Duke criteria in diagnosing TPVI-IE is limited by the difficulty of assessing prosthetic-valve involvement. A rapid increase in the transpulmonary gradient may be considered as a major criterion for IE in patients with CHD and TPVI, and the use of multimodality imaging should be widely considered. Moreover, negative blood-cultures in about 11% of cases warrant additional investigations of serological, molecular and histopathological tests.

Detailed data on pathogenicity and adhesion properties of bacteria responsible for TPVI-IE may provide useful information for improving treatment strategies.

Finally, the creation of a multidisciplinary IE team with expertise in CHD, imaging, infectious diseases and microbiology would likely improve the management of IE in this setting and, therefore, patient outcomes [26,48]. In addition, specific TPVI-IE guidelines should be proposed and implemented to minimize this devastating complication and safeguard better outcomes (Central Illustration).



Central Illustration. Infective endocarditis after transcatheter pulmonary valve implantation in patients with congenital heart disease: Incidence, risk factors, protective markers, diagnostic characteristics, microbiology and clinical outcomes. HACCEK, *Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Capnocytophaga*, *Eikenella*, *Kingella*.

Sources of funding

This study was granted by Marie-Lannelongue hospital.

Acknowledgments

The authors would like to thank Antoinette Wolfe, Emmanuelle Fournier, Florence Lecerf and Sarah Cohen for their contribution to this work.

Disclosure of interest

S.H. has received proctoring and consultant fees from Abbott outside the submitted work. A.F. has received proctoring and consultant fees from Abbott, Occlutech, and Medtronic, outside the submitted work. The other authors declare that they have no competing interest.

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