Chapter

Surgical Treatment of Infective Endocarditis

Sudeep Das De, Sanjeet Singh Avtaar Singh, Ahmed Al-Adhami and Nawwar Al-Attar

Abstract

Infective endocarditis carries a heavy disease burden with a high in-patient mortality. Surgery is the mainstay of treatment in 50% of patients diagnosed with infective endocarditis. Surgery for infective endocarditis can be challenging; a detailed understanding of surgical anatomy is essential and several fundamental principles need to be taken into consideration including optimal timing, radical debridement, decision to repair versus replace as well as the optimal choice for reconstruction. Outcomes of surgery depend on several factors including patient characteristics, the valve (s) involved, the virulence of the organism, and the extent of invasion of the infective process. Despite recent advances in treatment and improved outcomes, there remains areas for potential research including the ideal valve prosthesis/substitute and the optimal material for reconstruction. In this chapter, we will discuss the technical challenges and pitfalls in the surgical treatment of infective endocarditis, the predictors of outcome as well as novel strategies in treatment.

Keywords: endocarditis, prosthesis, surgery, repair, reconstruction

1. Introduction

The incidence of infective endocarditis is approximately 3–10 cases per 100, 000 per year and is more common in males and in the elderly. It is associated with a heavy disease burden with an in-hospital mortality ranging from 20 to 30% [1]. In native valve endocarditis (NVE), left sided heart valves are more commonly affected with right sided involvement in 5–10% of patients [2]. Surgery remains the mainstay of treatment in 50% of patients diagnosed with infective endocarditis.

2. Classification

Infective endocarditis can be classified into four groups: (1) Native valve endocarditis (NVE) (2) Prosthetic valve endocarditis (PVE) (3) Intravenous drug abuse (IVDA) infective endocarditis and (4) nosocomial infective endocarditis. The microbiology varies depending on the type of endocarditis. In community acquired IE, the most prevalent organism is Streptococcus viridians, whereas nosocomial

IE is more commonly caused by *Staphylococcus Aureus*. Native valve endocarditis of the tricuspid valve is predominantly seen in cases of IVDA with the main organism being Staphylococcus Aureus [2, 3]. Prosthetic valve endocarditis (PVE) cases that occur within the first year after surgery are considered early and cases that occur after 1 year are termed late. The offending organism in early PVE is commonly Staphylococcus Epidermis and Staphylococcus Aureus. Microbiology in late PVE is similar to native IE with Streptococcus viridians and Staphylococcus Aureus being the prevalent organism. Infective endocarditis can be further classified temporally with acute endocarditis being caused by more virulent organisms such as Staphylococcus Aureus and presenting with severe sepsis and rapid destruction of the valve and surrounding structures. Subacute endocarditis is caused by less virulent organisms such as viridians group Streptococcus and has a more indolent nature with a prolonged clinical course.

3. Pathophysiology of infective endocarditis

Infective endocarditis results from (1) Disruption of the valvular endocardial surface resulting in turbulent flow and (2) Adherence of blood bourne micro-organisms typically bacteria to the damaged endocardial surface. Endocardial damage occurs in degenerative calcific disease, rheumatic heart disease, congenital heart disease and from iatrogenic causes such as cardiac catheterization. Common causes of bacteraemia include intravenous drug abuse, long term indwelling catheters and invasive medical procedures. Complications of infective endocarditis can be from embolic phenomena including a stroke, kidney and splenic infarcts or due to direct invasion of surrounding structures resulting in problems such as paravalvular abscesses, conduction system pathology and fistulae.

4. Diagnosis, initial treatment and indications for surgery

The diagnosis of infective endocarditis is made with the modified Duke criteria [4]. A high index of suspicion is needed in cases where cultures are negative. This can occur in up to 2–7% of cases [5, 6] and is most commonly caused by premature administration of antimicrobial therapy prior to taking blood cultures, and infection with fastidious bacteria or fungi. Once blood cultures have been taken, the first line of treatment is aggressive broad spectrum antibiotics administered empirically, followed by surveillance blood cultures and serial transthoracic echocardiograms. In the recently published Partial Oral Treatment of Endocarditis (POET) trial [7], it was demonstrated that in patients with left-sided infective endocarditis from certain specified organisms, partial oral antibiotic treatment after initial intravenous treatment was non-inferior to treatment with only intravenous antibiotics. This study however has several limitations with regards to the generalisability of the findings to the general population. Firstly, only patients with left sided endocarditis caused by strep species, *E. faecalis*, *S. aureus* or coag-neg staph were included. These organisms represent 70–75% of all cases of infective endocarditis. Patients with Methicillin resistant Staph aureus (MRSA) endocarditis were not included and there were very few intravenous drug users in the study. Highly compliant patients were selected and in the outpatient oral antibiotic therapy group, patients were followed up 2 to 3 times per week and this may not reflect real world clinical practice. From a surgical

perspective, there is a risk that in patients on oral antibiotic therapy being followed up in the community, the sequelae of the disease process which may necessitate surgery may be missed, leading to a higher morbidity and mortality. An inpatient setting may allow more active surveillance of the patients with serial transthoracic echocardiograms and blood tests.

The indications for surgery are in line with the American College of Cardiology (ACC)/American Heart Association (AHA) or European Society of Cardiology (ESC) guidelines [8, 9]. In general, surgery is indicated when there is heart failure, worsening sepsis despite optimal antimicrobial therapy, a high embolic risk associated with large, mobile vegetations, perivalvular abscess, and virulent causative organisms such as *S. Aureus* and fungal endocarditis. Prosthetic valve endocarditis usually requires surgical treatment.

5. Pre-operative investigations

The first line of investigation in infective endocarditis is transthoracic echocardiogram (TTE) which has a sensitivity of approximately 25% in cases where the vegetation size is less than 5 mm and 70% where the vegetation size is 6-10 mm. Trans-oesophageal echocardiography (TOE) has a sensitivity and specificity of 95 and 90%, respectively [10]. TOE is the preferred investigation in cases of prosthetic valve endocarditis and where intracardiac complications such as abscesses and fistulae are suspected. At our institution, we perform a Positron Emission Tomography/Computed Tomography (PET-CT) when there is diagnostic difficulty in cases of prosthetic valve endocarditis. When there is suspected embolic phenomena in the visceral organs, a CT Abdomen/Pelvis should be performed. When there is evidence of neurological complications, a CT and/or MRI brain is needed to detect embolic infarcts or less often a haemorrhage. Haemorrhages are associated with a higher likelihood of mycotic aneurysms and further evaluation is needed if this is suspected. In non-emergency cases, patients above 40 years of age with cardiovascular risk factors should have coronary angiography to exclude coronary artery disease. If there is a large aortic valve vegetation, however this should be avoided as there is a risk of dislodging the debris. An alternative is CT Coronary angiography; however both these investigations are associated with contrast related renal toxicity and the risks have to be evaluated.

6. Surgical principles

In general, patients undergoing urgent or emergency surgery for IE tend to be unwell, septic, coagulopathic and fluid overloaded. Pre-operatively it is essential to have blood products including platelets, fresh frozen plasma, cryoprecipitate and anti-fibrinolytic agents (especially in reoperations) available. In addition, intra-operative TOE is important in all cases of IE. Fluid overload can be addressed intraoperatively by filtration on cardiopulmonary bypass. Good exposure of the operative field is needed and we recommend a full median sternotomy in all cases. Due to many patients being in heart failure and the potential for operations to be complex and lengthy, careful attention needs to be given to myocardial protection. In addition to routine antegrade cardioplegia, in cases of severe aortic regurgitation and large aortic valve vegetations which may obstruct the coronary ostia, we administer retrograde cardioplegia.

There are also several specific fundamental principles in infective endocarditis surgery, which include: (1) Optimal timing, (2) Radical debridement, (3) Repair versus replacement strategy, (4) Optimal choice of prosthesis/material for reconstruction and 5) Avoidance of contamination of the surgical field.

6.1 Optimal timing

Optimal timing of surgery for infective endocarditis remains a challenging decision for cardiac surgeons. The benefits of delaying surgery to allow adequate antibiotic therapy and time for optimising the patients' needs to be balanced with the risks of further haemodynamic deterioration and septic emboli during the waiting period. In general, once any of the indication for surgery outlined above are presented, early surgery is recommended [11]. An exception to this is if there any neurological complications. Ischaemic embolic events are more common in haemorrhagic strokes, with north associated with a high morbidity and mortality [12, 13]. In ischaemic strokes, there is a risk of haemorrhagic conversion with systemic heparinization and cardiopulmonary bypass. It is recommended that in cases of ischaemic stroke and haemorrhagic stroke, surgery should be delayed for 2 weeks and 4 weeks respectively. Ultimately, clinical judgement should be exercised in each case for the optimal timing of surgery.

6.2 Radical debridement

It is imperative to ensure radical debridement of all infected and necrotic tissue prior to reconstruction to minimise the risk of recurrence. A thorough knowledge of surgical anatomy, especially with regards to the aortic root, the left ventricular outflow tract, intra-ventricular septum and the aorto-mitral continuity is needed to perform a safe and adequate debridement followed by reconstruction.

6.3 Repair versus replacement strategy

In clinical practice, most patients with infective endocarditis undergo valve replacement. A repair strategy is recommended if possible after the primary goal of radical debridement is achieved and there is adequate tissue remaining. This is usually more commonly the case in mitral valve endocarditis. A repair strategy avoids the need for long-term anticoagulation when compared to mechanical valves, limits the amount of prosthetic material and hence recurrence and it is also well established in the mitral position that repair offers better long term survival compared to replacement [14].

6.4 Choice of prosthesis/material for reconstruction

The choice of prosthesis should be in line with current guidelines depending on the patients age, comorbidities, compliance and presence of any contra-indications to anticoagulation. There is no evidence the suggest any difference in outcomes between biological and mechanical valve prostheses in the setting of active infective endocarditis [15, 16]. If there is limited valve leaflet/annular destruction, autologous pericardial patches can be used. In cases of more extensive destruction, a bovine pericardial patch can be used. If there is significant aortic root destruction, an aortic homograft can be used for reconstruction. We will discuss these options in further detail in the following sections.

6.5 Avoidance of contamination of the surgical field

Once debridement has been completed, it is important to ensure that there is minimal further contamination of the surgical field prior to reconstruction. Instruments used for debridement, drapes, suction and surgical gloves should be changed before proceeding.

7. Native valve endocarditis

7.1 Aortic valve

In native aortic valve endocarditis, direct local complications include destruction of the aortic annulus, formation of annular abscesses, conduction tissue pathology, and fistulae. Intra-operatively, we avoid manipulation of the heart prior to applying the aortic cross clamp and arresting the heart to avoid the risk of the aortic vegetations dislodging. A transverse autotomy is performed to expose the aortic valve. If there is annular destruction towards the aorto-mitral continuity an oblique aortotomy towards the middle of the non-coronary cusp can be performed.

If there is a small area of leaflet perforation, the valve can be repaired using autologous pericardium. In most cases the valve is excised and if the annulus is involved, complete debridement of the infected tissue is needed. The defect is then reconstructed prior to implantation of the prosthetic valve. If the defect is small, autologous pericardium is used and for larger defects bovine pericardium is an alternative. If there is significant destruction of the annulus with discontinuity of the ventriculo-aortic junction, an reconstruction with an aortic homograft is the treatment of choice [17, 18]. The size of the the homograft is usually 2-3 mm less than the diameter of the native annulus. In cases where the aorto-mitral continuity is involved, the anterior leaflet of the mitral valve from the homograft can be used for reconstruction of the disruption. The use of freestyle aortic root replacements [19] and the Ross procedure [20] has also been reported in younger patients in extensive infective endocarditis of the aortic root.

7.2 Mitral valve

In native mitral valve endocarditis, the most common site of vegetations is on the leaflets near the annulus on the atrial side. They can however involve any part of the mitral valve apparatus. In severe cases, there is destruction of the atrioventricular junction with abscess formation. In our standard practice, we perform a median sternotomy, institute bi-caval cannulation and approach the mitral valve either via Sondergaard's groove or a trans-septal approach. If there is limited involvement of the leaflet tissue, repair can be attempted after debridement. Perforations of the anterior and posterior leaflets can be repaired using an autologous pericardial patch. A frequently involved region is the P2 region of the posterior leaflet. Standard principles of mitral valve repair apply when approaching repair in the setting of infective endocarditis. A triangular resection followed by closure is performed or if a wider region of P2 is involved a quadrangular resection followed by a slideplasty of the remaining tissue. An annuloplasty ring is then secured. Whenever possible we try to avoid added prosthetic material such as neochordae when attempting repair. More often, there is limited native tissue post debridement and we proceed to mitral valve

replacement. In more severe cases where there is annular destruction, the annulus has to be reconstructed. There are two approaches to annular reconstruction described by Carpentier and David.

In technique described by Carpentier [21], figure of eight sutures are applied directly to approximate the separation of the atrioventricular groove. Valve sutures with a large needle are then placed around this suture line. This technique is not commonly used and is reserved only in cases of very narrow atrioventricular defects. In the more commonly used technique described by David [22], a semi-circular pericardial patch is fashioned with one end secured to the endocardium of the ventricle and the other end to the left atrium. The patch should be larger than the defect size to avoid any tension. The mitral valve prosthesis is then secured with pledgeted sutures with part of it anchored onto the patch.

7.3 Tricuspid valve

Our approach to the tricuspid valve is via median sternotomy and bicaval cannulation with snaring of the cavae. Most surgeons at our institution arrest the heart to perform the operation. The advantage of this is a bloodless field as well as the aorta being collapsed and the aortic valve leaflets less prone to injury during the tricuspid valve procedure. In very sick patients however, the procedure can be done on a beating heart, with the additional advantage of observing any conduction defects during the operation.

In native tricuspid valve endocarditis there are three options for treatment: (1) Valvectomy, (2) Repair/Reconstruction and (3) Replacement.

When there is severe involvement of the leaflets, complete excision of the tricuspid valve can be performed and a second stage procedure can be done following aggressive antiobiotic therapy and treating the drug dependence of the patient. This can only be done if the pulmonary pressures are not high [23]. In practice however this is seldom done, and 20% of patient will develop right heart failure [24, 25]. When there is limited infection, there are several repair/reconstructive options. These include the use of pericardial patches, excision of the posterior leaflet and biscuspidization of the tricuspid valve, slideplasty and the use of neochordae [26, 27]. The reconstruction can be reinforced with an annuloplasty ring.

Several studies have shown no difference between biological and mechanical valves in the tricuspid position [28, 29]. Biological valves in the tricuspid position have also demonstrated longer durability compared those in the mitral position [30]. In addition, mechanical valves in the tricuspid position require higher INR values. For these reasons, in our clinical practice we use a mitral bioprosthesis for tricuspid valve replacement (TVR). Following TVR, we secure permanent epicardial pacing leads.

8. Prosthetic valve endocarditis

Prosthetic valve endocarditis (PVE) is more common in the aortic than in the mitral position due to more mitral valve repair cases and less prosthetic material. PVE is classified as early if it occurs within 1 year post-operatively and late if it occurs after that. The incidence of early PVE is 1% per year [31]. Early PVE is associated with intra-operative contamination. Risk factors include native valve endocarditis, longer cardiopulmonary bypass times, and long term indwelling lines and

catheters. The incidence of late PVE is 0.5 to 1% per year [32, 33]. Late PVE is usually attributed to hospital-acquired infections and is seen in patients with long-term comorbidities who require frequent admissions for procedures such as haemodialysis and also in patients who are immunosuppressed. In earlyPVE, the interface between the sewing ring and annulus is usually involved resulting in valve dehiscence and a para-valvular leak. Compared to native valve endocarditis (NVE), PVE more commonly results in abscess formation involving the intraventricular septum causing conduction blocks, as well as development pseudoaneurysms. The treatment of PVE is more aggressive and surgery is usually the definitive treatment for PVE. In severe cases, PVE may extend into intervalvlular fibrosa and require replacement of both aortic and mitral valves. This is less common in native valve endocarditis. In these cases, an extended transseptal approach can be performed for improved exposure to both valves. A bovine pericardial patch can be used to reconstruct the intervalvular fibrosa. The mitral valve is secured to the annulus posteriorly, medially and laterally. The superior part of the mitral sewing ring is secured to the patch which can also be used to cover the left atrium. Once the mitral valve prosthesis is secured, the aortic valve prosthesis is then secured partly to the healthy annulus and to the patch. Attention must be given to the angle between the aortic and mitral valve prostheses to be similar to the normal aorto-mitral angle. As mentioned previously, an aortic homograft can also be used. The aorto-mitral curtain of the homograft can be used to reconstruct the native anterior mitral valve leaflet. If there is extensive damage to the native mitral valve, the mitral valve prosthesis can be secured to the intervalvular fibrosa of the homograft [17, 18].

9. Results

The results of surgery for infective endocarditis depend on several factors including patient charactersitics, the valve(s) involved, the virulence of the organism, the extent of invasion of the infective process. Generally, prosthetic valve endocarditis has a worse prognosis than native valve endocarditis [34, 35] and nosocomial infections are also associated with a poorer outcome compared to community-acquired infections [36]. In cases of isolated simple native aortic valve endocarditis, the operative mortality is less than 10% whereas more complex cases and prosthetic valve endocarditis is associated with a higher mortality [37–39]. Recent advances in operative techniques have however yielded excellent results with comparable outcomes in native versus prosthetic valve endocarditis as well as simple endocarditis compared to more invasive disease [40].

Overall, the results for mitral valve endocarditis tends to be worse compared to aortic valve endocarditis, and this is especially the case in more invasive disease. This is likely explained by the fact that it is more difficult to debride and drain the the atrioventricular groove [22]. In addition, there is currently no equivalent to the aortic homograft in the mitral position as a viable reconstructive option [41].

Mitral valve repair has shown excellent results when compared to mitral valve replacement in IE with lower in-hospital mortality as well as better overall and infection free survival [42]. This can be attributed to the fact that patients in whom mitral valve repair is feasible tend to have less invasive disease and are generally systemically better, there is less prosthetic material used, and left ventricular function is preserved with mitral valve repair.

Native tricuspid valve repair and replacement have excellent results, with repair recommended whenever feasible [28, 43]. The outcome is also dependent on patient rehabilitation and avoidance of drug dependence.

Concomitant aortic and mitral valve endocarditis is associated with a worse prognosis when compared to single valve endocarditis [40]. Similarly, concomitant right and left sided endocarditis has a worse outcome than isolated right sided endocarditis [44].

Overall the long term survival following surgery for endocarditis is reported as between 50 and 60% at 15 years. There is also no difference in survival or recurrence rates between bioprosthetic and mechanical valves [16, 34].

10. Further development

Despite the recent advances in the treatment of infective endocarditis, there remains challenges including optimal penetration of antibiotic therapy, and the ideal material or prosthesis for reconstruction. Several novel approaches have been described in pre-clinical models to tackle the problem of biofilm formation in infective endocarditis. These include non-antibiotic strategies such as the administration of anti-thrombotic agents, hyperbaric oxygen therapy, and agents which potentially disrupt the gene regulation of bacteria during biofilm formation [45]. The use of novel extra-cellular matrix patches for mitral valve as well as tricuspid valve reconstruction has also been reported [46, 47]. Allograft mitral valve replacement has also been reported in severe aortic and mitral valve endocarditis as well as in isolated tricuspid valve endocarditis [41, 48, 49]. Another area of ongoing research is the development of bioengineered valves as a viable prosthesis in the setting of infective endocarditis with the potential of avoiding biofilm formation and recurrence of infection and long term durability [50].

11. Conclusions

The surgical treatment of infective endocarditis can be challenging. A thorough understanding of surgical anatomy is essential and several fundamental principles should be taken into consideration including optimal timing, radical debridement, decision to repair versus replace as well as the optimal choice for reconstruction. The results for infective endocarditis have improved with reports of similar outcomes between simple and more invasive endocarditis. There are potential areas for further research including developing the ideal prosthesis/substitute as well as the optimal material for reconstruction.

Conflict of interest

The authors declare no conflict of interest.

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Author details

Sudeep Das De[1*], Sanjeet Singh Avtaar Singh[1,2,3], Ahmed Al-Adhami[1,2] and Nawwar Al-Attar[1,2]

1 Department of Cardiothoracic Surgery, Golden Jubilee National Hospital, Glasgow, UK

2 Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, UK

3 Department of Cardiothoracic Surgery, Royal Infirmary of Edinburgh, Edinburgh, UK

*Address all correspondence to: sdasde@gmail.com

References

[1] Prendergast BD. The changing face of infective endocarditis. Heart. 2006;**92**(7):879-885

[2] Moss R, Munt B. Injection drug use and right sided endocarditis. Heart. 2003;**89**(5):577-581

[3] Fowler VG Jr, Miro JM, Hoen B, Cabell CH, Abrutyn E, Rubinstein E, et al. Staphylococcus aureus endocarditis: A consequence of medical progress. Journal of the American Medical Association. 2005;**293**(24):3012-3021

[4] Durack DT, Lukes AS, Bright DK. New criteria for diagnosis of infective endocarditis: Utilization of specific echocardiographic findings. Duke Endocarditis Service. American Journal of Medicine. 1994;**96**(3):200-209

[5] Lamas CC, Eykyn SJ. Blood culture negative endocarditis: Analysis of 63 cases presenting over 25 years. Heart. 2003;**89**(3):258-262

[6] Werner M, Andersson R, Olaison L, Hogevik H. A clinical study of culture-negative endocarditis. Medicine (Baltimore). 2003;**82**(4):263-273

[7] Iversen K, Ihlemann N, Gill SU, Madsen T, Elming H, Jensen KT, et al. Partial Oral versus intravenous antibiotic treatment of endocarditis. The New England Journal of Medicine. 2019;**380**(5):415-424

[8] Bonow RO, Carabello BA, Kanu C, de Leon AC Jr, Faxon DP, Freed MD, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (writing committee to revise the 1998 guidelines for the Management of Patients with Valvular Heart Disease): Developed in collaboration with the Society of Cardiovascular Anesthesiologists: Endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. Circulation 2006;**114**(5):e84-231

[9] Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zotti F, et al. 2015 ESC guidelines for the management of infective endocarditis: The task force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). European Heart Journal. 2015;**36**(44):3075-3128

[10] Daniel WG, Mügge A, Grote J, Hausmann D, Nikutta P, Laas J, et al. Comparison of transthoracic and transesophageal echocardiography for detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic positions. The American Journal of Cardiology. 1993;**71**(2):210-215

[11] Kang DH, Lee S, Kim YJ, Kim SH, Kim DH, Yun SC, et al. Long-term results of early surgery versus conventional treatment for infective endocarditis trial. Korean Circulation Journal. 2016;**46**(6):846-850

[12] Gillinov AM, Shah RV, Curtis WE, Stuart RS, Cameron DE, Baumgartner WA, et al. Valve replacement in patients with endocarditis and acute neurologic deficit. The Annals of Thoracic Surgery. 1996;**61**(4):1125-1129 discussion 30

[13] Matsushita K, Kuriyama Y, Sawada T, Yamaguchi T, Nagata S, Kawazoe K, et al. Hemorrhagic and ischemic cerebrovascular complications of active infective endocarditis of native valve. European Neurology. 1993;**33**(3):267-274

[14] Enriquez-Sarano M, Schaff HV, Orszulak TA, Tajik AJ, Bailey KR, Frye RL. Valve repair improves the outcome of surgery for mitral regurgitation. A multivariate analysis. Circulation. 1995;**91**(4):1022-1028

[15] Byrne JG, Rezai K, Sanchez JA, Bernstein RA, Okum E, Leacche M, et al. Surgical management of endocarditis: The society of thoracic surgeons clinical practice guideline. The Annals of Thoracic Surgery. 2011;**91**(6):2012-2019

[16] Moon MR, Miller DC, Moore KA, Oyer PE, Mitchell RS, Robbins RC, et al. Treatment of endocarditis with valve replacement: The question of tissue versus mechanical prosthesis. The Annals of Thoracic Surgery. 2001;**71**(4):1164-1171

[17] Glazier JJ, Verwilghen J, Donaldson RM, Ross DN. Treatment of complicated prosthetic aortic valve endocarditis with annular abscess formation by homograft aortic root replacement. Journal of the American College of Cardiology. 1991;**17**(5):1177-1182

[18] Yankah AC, Pasic M, Klose H, Siniawski H, Weng Y, Hetzer R. Homograft reconstruction of the aortic root for endocarditis with periannular abscess: A 17-year study. European Journal of Cardio-Thoracic Surgery. 2005;**28**(1):69-75

[19] Heinz A, Dumfarth J, Ruttmann-Ulmer E, Grimm M, Müller LC. Freestyle root replacement for complex destructive aortic valve endocarditis. The Journal of Thoracic and Cardiovascular Surgery. 2014;**147**(4):1265-1270

[20] Pettersson G, Tingleff J, Joyce FS. Treatment of aortic valve endocarditis with the Ross operation. European Journal of Cardio-Thoracic Surgery. 1998;**13**(6):678-684

[21] Carpentier AF, Pellerin M, Fuzellier JF, Relland JY. Extensive calcification of the mitral valve anulus: Pathology and surgical management. The Journal of Thoracic and Cardiovascular Surgery. 1996;**111**(4):718-729 discussion 29-30

[22] David TE, Feindel CM, Armstrong S, Sun Z. Reconstruction of the mitral anulus. A ten-year experience. The Journal of Thoracic and Cardiovascular Surgery. 1995;**110**(5):1323-1332

[23] Arbulu A, Holmes RJ, Asfaw I. Tricuspid valvulectomy without replacement. Twenty years' experience. The Journal of Thoracic and Cardiovascular Surgery. 1991;**102**(6):917-922

[24] Arbulu A, Holmes RJ, Asfaw I. Surgical treatment of intractable right-sided infective endocarditis in drug addicts: 25 years experience. The Journal of Heart Valve Disease. 1993;**2**(2):129-137 discussion 38-9

[25] Robin E, Thomas NW, Arbulu A, Ganguly SN, Magnisalis K. Hemodynamic consequences of total removal of the tricuspid valve without prosthetic replacement. The American Journal of Cardiology. 1975;**35**(4):481-486

[26] Bortolotti U, Tursi V, Fasoli G, Milano A, Frigato N, Casarotto D. Tricuspid valve endocarditis: Repair with the use of artificial chordae.

The Journal of Heart Valve Disease. 1993;**2**(5):567-570

[27] Lange R, De Simone R, Bauernschmitt R, Tanzeem A, Schmidt C, Hagl S. Tricuspid valve reconstruction, a treatment option in acute endocarditis. European Journal of Cardio-Thoracic Surgery. 1996;**10**(5):320-326

[28] Carrier M, Hébert Y, Pellerin M, Bouchard D, Perrault LP, Cartier R, et al. Tricuspid valve replacement: An analysis of 25 years of experience at a single center. The Annals of Thoracic Surgery. 2003;**75**(1):47-50

[29] Ratnatunga CP, Edwards MB, Dore CJ, Taylor KM. Tricuspid valve replacement: UK heart valve registry mid-term results comparing mechanical and biological prostheses. The Annals of Thoracic Surgery. 1998;**66**(6):1940-1947

[30] Cohen SR, Silver MA, McIntosh CL, Roberts WC. Comparison of late (62 to 140 months) degenerative changes in simultaneously implanted and explanted porcine (Hancock) bioprostheses in the tricuspid and mitral valve positions in six patients. The American Journal of Cardiology. 1984;**53**(11):1599-1602

[31] Agnihotri AK, McGiffin DC, Galbraith AJ, O'Brien MF. The prevalence of infective endocarditis after aortic valve replacement. The Journal of Thoracic and Cardiovascular Surgery. 1995;**110**(6):1708-1720 discussion 20-4

[32] Fang G, Keys TF, Gentry LO, Harris AA, Rivera N, Getz K, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia. A prospective, multicenter study. Annals of Internal Medicine. 1993;**119**(7 Pt 1): 560-567

[33] Grover FL, Cohen DJ, Oprian C, Henderson WG, Sethi G, Hammermeister KE. Determinants of the occurrence of and survival from prosthetic valve endocarditis. Experience of the veterans affairs cooperative study on Valvular heart disease. The Journal of Thoracic and Cardiovascular Surgery. 1994;**108**(2):207-214

[34] David TE, Gavra G, Feindel CM, Regesta T, Armstrong S, Maganti MD. Surgical treatment of active infective endocarditis: A continued challenge. The Journal of Thoracic and Cardiovascular Surgery. 2007;**133**(1):144-149

[35] Manne MB, Shrestha NK, Lytle BW, Nowicki ER, Blackstone E, Gordon SM, et al. Outcomes after surgical treatment of native and prosthetic valve infective endocarditis. The Annals of Thoracic Surgery. 2012;**93**(2):489-493

[36] Hoen B, Alla F, Selton-Suty C, Béguinot I, Bouvet A, Briançon S, et al. Changing profile of infective endocarditis: Results of a 1-year survey in France. Journal of the American Medical Association. 2002;**288**(1):75-81

[37] Alexiou C, Langley SM, Stafford H, Lowes JA, Livesey SA, Monro JL. Surgery for active culture-positive endocarditis: Determinants of early and late outcome. The Annals of Thoracic Surgery. 2000;**69**(5):1448-1454

[38] d'Udekem Y, David TE, Feindel CM, Armstrong S, Sun Z. Long-term results of operation for paravalvular abscess. The Annals of Thoracic Surgery. 1996;**62**(1):48-53

[39] d'Udekem Y, David TE, Feindel CM, Armstrong S, Sun Z. Long-term results of surgery for active infective endocarditis. European Journal of Cardio-Thoracic Surgery. 1997;**11**(1):46-52

[40] Hussain ST, Shrestha NK, Gordon SM, Houghtaling PL,

Blackstone EH, Pettersson GB. Residual patient, anatomic, and surgical obstacles in treating active left-sided infective endocarditis. The Journal of Thoracic and Cardiovascular Surgery. 2014;**148**(3):981-8.e4

[41] Navia JL, Al-Ruzzeh S, Gordon S, Fraser T, Agüero O, Rodríguez L. The incorporated aortomitral homograft: A new surgical option for double valve endocarditis. The Journal of Thoracic and Cardiovascular Surgery. 2010;**139**(4):1077-1081

[42] Muehrcke DD, Cosgrove DM 3rd, Lytle BW, Taylor PC, Burgar AM, Durnwald CP, et al. Is there an advantage to repairing infected mitral valves? The Annals of Thoracic Surgery. 1997;**63**(6):1718-1724

[43] Gottardi R, Bialy J, Devyatko E, Tschernich H, Czerny M, Wolner E, et al. Midterm follow-up of tricuspid valve reconstruction due to active infective endocarditis. The Annals of Thoracic Surgery. 2007;**84**(6):1943-1948

[44] Musci M, Siniawski H, Pasic M, Grauhan O, Weng Y, Meyer R, et al. Surgical treatment of right-sided active infective endocarditis with or without involvement of the left heart: 20-year single center experience. European Journal of Cardio-Thoracic Surgery. 2007;**32**(1):118-125

[45] Lerche CJ, Schwartz F, Theut M, Fosbøl EL, Iversen K, Bundgaard H, et al. Anti-biofilm approach in infective endocarditis exposes new treatment strategies for improved outcome. Frontiers in Cell and Development Biology. 2021;**9**:643335

[46] Arbona MA, David TE, David CM, Rao V. Results of mitral valve reconstruction using substitute extracellular matrix. JTCVS Tech. 2022;**16**:43-48

[47] Gerdisch MW, Boyd WD, Harlan JL, Richardson JB Jr, Flack JE 3rd, Palafox BA, et al. Early experience treating tricuspid valve endocarditis with a novel extracellular matrix cylinder reconstruction. The Journal of Thoracic and Cardiovascular Surgery. 2014;**148**(6):3042-3048

[48] Couetil JP, Argyriadis PG, Shafy A, Cohen A, Berrebi AJ, Loulmet DF, et al. Partial replacement of the tricuspid valve by mitral homografts in acute endocarditis. The Annals of Thoracic Surgery. 2002;**73**(6):1808-1812

[49] Mestres CA, Castellá M, Moreno A, Paré JC, del Rio A, Azqueta M, et al. Cryopreserved mitral homograft in the tricuspid position for infective endocarditis: A valve that can be repaired in the long-term (13 years). The Journal of Heart Valve Disease. 2006;**15**(3):389-391

[50] Bonetti A, Marchini M, Ortolani F. Ectopic mineralization in heart valves: New insights from in vivo and in vitro procalcific models and promising perspectives on noncalcifiable bioengineered valves. Journal of Thoracic Disease. 2019;**11**(5):2126-2143