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# A Comprehensive Review on Endocarditis

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**ABSTRACT**

Risk analysis of endocardial prophylaxis in adolescents with high risk of morbidity and death. One of the most harmful and possibly fatal infections is infective endocarditis. Mitral-valve prolapse is the most prevalent risk factor for cardiovascular disease in industrialized nations. Infective Endocarditis is cause for 65,000 deaths annually. To lessen these potential causes of bacteremia, all at-risk children should receive the finest oral health education available. Endocarditis categorical prophylactics in adolescents, where endocarditis is linked to high rates of morbidity and death, prophylaxis is crucial..The risk groups for cardiac conditions are high, intermediate, and insignificant. For those who fall into the high- and moderate-risk categories, prophylaxis is advised; these conditions are mainly classified according to the likelihood of getting endocarditis and the severity of that infection. For instance, people who fall into the high-risk group are much more likely to experience a serious endocardial infection, which is frequently linked to high rates of morbidity and death. There is no higher risk of endocarditis in people with negligible risk than there is in the general community.

## INTRODUCTION

One of the most harmful and possibly fatal infections is infective endocarditis [1].An infection of the endocardium, which most commonly occur in the heart valves and also in ventricles, atria and large blood vessels of chest results in a condition known as infective endocarditis[2].Vegetation caused by infective endocarditis grows on the valves and produces toxins and enzymes that destroy tissue and result in perforations in the valve[3]

Via the significant contributions of William Osler towards the end of the 19th century, the clinicopathological signs of the infection of heart valves—first documented by the French physician Lazare Rivière more than 350 years ago—were better identified[4].

While being a rare condition, infectious endocarditis (IE) has a considerable negative impact[5]. It is an infection of the heart's endothelium[6]. It affects all heart valves, natural or artificial, as well as any intracardiac devices and, less frequently, any non-functioning embryonic remains that are located in the right atrium (RA)[7].

The clinical characteristics of this complicated illness have evolved over the last several decades. It is now primarily an infection of the elderly, and mitral-valve prolapse is the most prevalent risk factor for cardiovascular disease in industrialized nations. In comparison, rheumatic heart disease is a major risk factor in developing nations [8]. A point of infection within the cardiac characterizes infectious endocarditis, a condition that is dreaded in the field of cardiology. It is commonly acquired in a clinical setting, and more than half of cases now involve people who have no history of cardiac disease. Even with the best treatment, mortality after one year is close to 30%. Infective endocarditis presents significant difficulties. Its origin, clinical symptoms, and outcome are all diverse. Staphylococcus aureus, which has emerged as the primary pathogen in the industrialized world, causes an aggressive version of the illness, frequently in groups of patients who are fragile or elderly[9]. Infective endocarditis continues to be a serious and deadly disease despite recent advances in diagnosis and treatment. It is noteworthy that IE has evolved

into a condition with a more severe presentation than previously noted and is now frequently associated with S aureus infections in patients who have previously received medical care. More care must be taken to effectively treat all patients with S aureus bacteremia. Surgery is performed on almost 50% of IE patients, so highlighting surgical indications early may reduce mortality[10].An elevated chance of morbidity and death is associated with infectious

endocarditis (IE). Good patient outcomes depend on early identification, efficient therapy, and quick detection of complications. The more frequently encountered bacteria, such as streptococci, enterococci, staphylococci, and the HACEK pathogens (Hemophilus parainfluenzae, Hemophilus aphrophilus, Actinobacillus [Hemophilus] actinomycetemcomitans, Cardiobacterium hominis, Eikenella species, and Kingella species [11].A extended parentral course of therapy is necessary for infectious endocarditis, which affects patients[40].

## EPIDEMIOLOGY

The epidemiology of infectious endocarditis (IE) is based on youthful patients with rheumatic fever, have given way to elderly people with the several concurrent illness.[33]When compared to endocarditis, prosthetic valve endocarditis or infection on another cardiac device, infectious endocarditis is the most severe and potentially fatal compication of heart valve diseases[34].Depite advancements in diagnostic testing, antibiotic therpy and surgical treatment, infectious endocarditis still has a high morbidity and fatality rate [35].

The fourth most common reason for a life-threatening infectious disease conditions is infected endocarditis [3]. In worldwide infective endocarditis is cause for 65,000 deaths annually [12]. I5-10 % of all infective endocarditis are right-sided infective endocarditis and moreover 90% of right-sided infective endocarditis occur in tricuspid valve[13]. It is noted that over half of the IE case occur in people without a confirmed structural heart disease. The highest rate is seen in people aged 70 to 80 of age and occurs more commonly in men [2]. Prosthetic valve endocarditis is more common in patients with prosthesis [14].

With a mortality rate of up to 30% at 30 days, it has an annual incidence of 3–10/100,000 people. Because of increased use of intravenous lines and intracardiac devices, the epidemiology of IE has gradually evolved over time, with healthcare-associated IE currently accounting for 25–30% of modern cohorts[6]. Mortality rates are between 20% and 40%[14]. With an annual incidence of 1.5 to 15 cases per 100,000 people, it is regarded as a rare condition. The United States has the greatest rates, whilst Denmark has a lower incidence[4].

Even though real demographic traits, including both instances and the total population at risk, are challenging to acquire, estimating the frequency of infective endocarditis (IE) is problematic. Several well-designed epidemiologic studies over the last decade have given both statistics on the incidence of endocarditis and insight into groups at risk[27].

## TYPES

Based on pathogenic microorganisms infective endocarditis is classified into 2 types : subacute and acute infective endocarditis [15].

The causative agent for subacute endocarditis are Streptococcus mutans, Streptococcus viridians and HACEK group (Haemophilus spp, Aggregatibacter spp, Cardiobacterium hominis, Eikenella corrodens, Kingella spp) and it is presented with low-grade fever and mild heart failure symptoms [15]. Tricuspid valve infective endocarditis (TVIE) is most commonly caused by Staphylococcus aureus and the second most common pathogens that cause TVIE were Streptococci and Enterococci. The right-side infective endocarditis(RSIE) was also caused by fungi and gram-negative bacilli [13].

## CAUSES

The three bacterial species such as staphylococcus spp., streptococcus spp., and enterococcus spp. are the most frequent causes of infective endocarditis. Yet, clinicians must increasingly take drug – resistant infections, atypical bacteria, and fungi into account in certain patient populations[36].

According to the majority of research, Staphylococcus aureus now accounts for about 26.6% of all cases of IE, followed by enterococci, various streptococci, and the viridans group streptococci. Together, these microorganisms are responsible for 80–90% of all endocarditis cases [6]. The two most clinically significant species are Enterococcus faecalis and Enterococcus faecium; E. faecalis accounts for roughly 97% of all IE infections and primarily affects the elderly and people with comorbidities [22]. Gram-negative bacilli can cause cases in 2-5% of instances (both aerobic Gram-negative bacilli or by the known HACEK group: Haemophilus spp., Aggregatibacter actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae)[4].

## VARIOUS ETIOLOGICAL BACTERIA ASSOCIATED WITH INFECTIVE ENDOCARDITIS [IE]

In the presence of persistent bacteremia with gram-positive cocci, the causative description of infective endocarditis is simple to make. However, the blood culture may contain a bacterium hardly ever associated with endocarditis, such as Lactobacillus spp., Klebsiella spp., or nontoxigenic Corynebacterium, Salmonella, Gemella, Campylobacter, Aeromonas, Yersinia,

Nocardia, Pasteurella, Listeria, or Erysipelothrix spp., that needs additional examination to establish the relationship with endocarditis, or the blood culture may be uninformative despite a supportive clinical evaluation. In the latter scenario, either picky extracellular or internal bacteria are the causative agents. Abiotrophia, HACEK group bacteria, Clostridium, Brucella, Legionella, Mycobacterium, and Bartonella spp. are examples of fastidious extracellular bacteria that require supplemental media, extended growing times, and specific culture conditions. Bacteria that are intracellular, like Coxiella burnetii, cannot be regularly separated [28]. A known but uncommon source of infective endocarditis, accounting for about 3% of cases, is the HACEK group of fastidious gram-negative microbes. Fever, splenomegaly, a novel or shifting murmur, and microvascular phenomena were the most typical initial symptoms. The majority of patients had symptoms from two weeks to six months before their diagnoses [32].

**PATHOPHYSIOLOGY**

The pathophysiology of IE is complicated, and it is still unknown why some bacteria can cling, last, and harm the cardiac valves in such a setting that would seem to be unfavorable [18].

Endothelial injury and inflammation→ deposition of sterile fibrin-platelet aggregates in the damaged site (Non- Bacterial Thrombotic Endocarditis)→ bacterial colonization in the lesions→ maturation and growth of the Fibrin-Platelet aggregates→clumps of bacteria at high inoculums (VEGETATION)[4]→ progression of valvular damage and vegetation maturation→ severe valvular damage and disseminated[18]. The vegetation represents the pathological hallmark of infective endocarditis and determines its main clinical manifestations, namely:

1) When this "complete bacterial lesion" grows, a high inoculum of bacteremia is produced, which has the potential to develop distant septic metastases.

2) Valvular damage from the invasion of the structures to which this vegetation is tethered may have a deleterious impact on the patient's hemodynamics.

3) This friable mass has the potential to separate into pieces that result in distant embolisms, which can negatively impact the function and prognosis of the people who are impacted[4]

For instance, humoral innate immune responses are particularly effective against Gram-negative bacteria. Gram-positive bacteria, especially some strains of streptococci and S. aureus, have special molecules on their surfaces called adhesins, such as "secretable expanded repertoire adhesive molecules" and "microbial surface components recognizing adhesive matrix molecules" (MSCRAMMs) (SERAMs). These adhesins identify integrins, particular ligands that are present on the damaged or swollen endothelium surface[4].

It has been suggested that coagulation is "at the heart" of IE. The complement system is activated by the coagulation system in a number of different ways. The cleavage product that results from the coagulation cascade, thrombin, connects clotting and inflammation and plays a crucial role in hemostasis. Immunothrombosis, a term used to describe the close relationship between coagulation and the innate immune system, is kept in a delicate balance in healthy individuals. A variety of illnesses, particularly IE, can develop when this balance is upset[18].

## RISK FACTORS

In comparison to earlier decades, patients with IE are older, have more comorbidities such as diabetes mellitus, chronic kidney failure, chronic obstructive pulmonary disease or have cardiac implantable electronic devices [12]. A study conducted by Regueiro et al. concluded that male gender, patients with moderate to severe paravalvular leakage have an increased risk of TAVI-IE [14]. Congenital heart disease or valvular disease associated with rheumatic fever are the risk factor for subacute infective endocarditis and moreover, intravenous drug use, diabetes, malignancies are the risk factor for acute infective endocarditis. Procedures like valve replacement, pacemakers and using implantable defibrillators and long-term intravascular catheters were also the risk factor for infective endocarditis[15]. Moreover, age-related degenerative valvular disease, increased need for cardiac devices and invasive procedures including hemodialysis for end-stage renal were the risk factor for IE [1].

## CLINICAL PRESENTATION

The clinical presentation of IE varies greatly and can take the form of an acute, subacute, or chronic illness depending on the underlying cardiac problems, pre-existing comorbidities, and the causative microorganisms. Up to 90% of patients arrive with fevers, night sweats, exhaustion, loss of appetite, and weight, with 25% arriving with signs of embolic phenomenon[6].

Endocarditis can manifest as a mild or acute condition[36].A new onset heart murmur, which can be present in upto 75% of patients, is a defining symptoms of acute infective endocarditis [36].When patients have nonspecific symptoms spanning weeks to months including tiredness, dyspnea, low grade fever, malaise, chills, sweats, back pain, arthralgias, weight loss or subacute infective endocaditis can be considerably more challenging to diagnose[36].

**DIAGNOSIS**

Imaging, microbiological results and clinical presentation are used for the diagnosis of infective endocrditis[36].The modified Duke Criteria are used to diagnose IE, however their diagnostic efficacy is restricted in patients with prosthetic valve or cardiac implanted electronic device(CIED) endocarditis, as well as in the early stage of the illness[37].

Due to its speed, accessibility and affordability compared to other imaging procedures, electrocardiography continues to be the most routinely used examination for the diagnosis of infective endocarditis[33].

Transthoracic echocardiography (TTE) is frequently the initial test of choice [33]. Its sensitivity for identifying vegetation on natural valves is only around 70%, while for identifying vegetation on artificial valves, is approximately 50% [36].

Transesophageal echocardiography is the recommended first imaging test for individuals with prosthetic valves who have no contraindications[33].Transesophageal echocardiogram (TOE) is more sensitive and specific, with a range of about 80 – 95 %, as opposed to transthoracic echocardiography (TEE) which has a high sensitivity that varies from 50 to 80% [39].

Based on clinical, radiological, and laboratory criteria, IE can be diagnosed[7].

Depending on the situation or the level of suspicion, the initial investigation, in this case, may comprise either an adequate blood culture, an echocardiogram, or both[23].

Investigation and consideration criteria for potential infective endocarditis

a) A feverish illness and a new valve regurgitation murmur.

b) A febrile illness, an at-risk heart lesion that already exists, and no clinically evident site of infection.

c) A febrile illness accompanied by any of the following:

- Predisposition and recent intervention with accompanying bacteraemia

- Congestive heart failure symptoms

- A new conduction disruption

- Embolic event, Roth spots, splinter hemorrhages, Janeway lesions, and Osler's nodes are examples of vascular or immunological phenomena.

- A recent stroke

- Unknown causes of peripheral abscesses (renal, splenic, cerebral, and spinal).A protracted history of sweats, weight loss, anorexia or malaise and an at-risk cardiac lesion.

d) Any newly reported unexplained embolic event (e.g. cerebral or limb ischemia)

e) Consistently positive blood cultures without apparent cause

f) Bloodstream infection is caused by an intravascular catheter with continuously positive blood cultures 72 hours after catheter removal[23].

To assist in diagnosing IE, the modified Duke criteria can be employed[6].

Definite infective endocarditis = two major, or one major and three minor, or five minor

Possible infective endocarditis = one major and one minor, or three minor.

➔ Major criteria:

- blood cultures:

1) Typical microorganisms consistent with IE from two separate blood cultures:

a) Viridans group streptococci, Streptococcus bovis group, HACEK group, Staphylococcus aureus;

b) Community-acquired enterococci with the absence of a primary focus;

2) Microorganisms consistent with IE from persistently positive blood cultures:

a) ≥2 positive blood cultures drawn >12 hours apart;

b) all of three or a majority of ≥4 separate blood cultures (first and last samples ≥1 hour apart);

c) single positive blood culture for Coxiella burnetii or phase IgG antibody titer>1:800

3) Imaging:

a) echocardiogram positive for IE:

- vegetation
- abscess, pseudoaneurysm or intracardiac fistula
- valvular perforation or aneurysm
- new partial dehiscence of prosthetic valve

b) abnormal activity around the site of a prosthetic valve detected by PET/CT assuming >3 months after surgery or radio-labeled leucocyte-SPECT/CT

c) definite paravalvular lesions by cardiac CT

➔ Minor criteria:

- Predisposing heart condition or intravenous drug use.
- Fever >38°C.
- Vascular phenomena (including those detected by imaging alone): arterial emboli, splenic infarction, mycotic aneurysms, intracranial hemorrhage and Janeway lesions.
- Immunological phenomena: glomerulonephritis, Osler's nodes, Roth's spots and rheumatoid factor.
- Microbiological evidence: positive blood cultures not meeting the major criteria above or serological evidence of infection with organism consistent with IE[6].

Clinical suspicion, microbiological correlation, and additional imaging with whole-body computed tomography (CT), cerebral magnetic resonance imaging (MRI), or increasingly 18F-labelled fluoro-2-deoxyglucose positron emission tomography (18F-FDG-PET)/CT may be necessary in cases of prosthetic valve endocarditis or implantable electronic device infections[6].

➔ Microbiological Diagnosis:

Positive blood cultures for diagnosing, identifying, and testing for susceptibility to an organism. The right way to collect samples is to use an aseptic, no-touch technique to collect three blood samples (10 ml each in anaerobic and aerobic bottles) from different access sites, spaced at least an hour apart. Blood cultures with isolated positive results are inconclusive for IE. If blood cultures show no development, there is still a high clinical suspicion of IE. It is important to rule out other potential causes of culture-negative endocarditis, including those caused by malignancy, hypercoagulable states, systemic lupus erythematosus (Liebman-Sacks endocarditis), trauma, Bartonella species, Coxiella burnetii, Tropheryma whipplei, and some fungi (especially Aspergillus species)[6]

➔ Cardiac imaging:

Only if there is a strong clinical suspicion of IE and ideally when the findings of blood cultures are obtained, should transthoracic echocardiography (TTE) be requested. Echocardiographic vegetations are septic thrombi and need to be confirmed by microbiology. They typically reside on the cardiac valves' upstream surfaces and can result in regional or systemic problems. TTE is around 70% sensitive to identifying vegetation on native valves. Those with prosthetic valves see a 50% reduction in this, whereas patients with implanted electronic devices get a lesser reduction. In cases where TTE is inconclusive but the microbiological is clinically indicative of IE, a second TTE may be necessary after a gap of 5-7 days.

When a non-diagnostic TTE and high clinical suspicion of endocarditis are present, when prosthetic or device-related endocarditis is suspected, when S. aureus bacteremia is present, and when IE-related complications have occurred, transoesophageal echocardiography (TOE) is used to confirm the diagnosis of IE (heart block, new murmur, persistent fever, embolism and intracardiac abscess)[6].

➔ Additional imaging:

TTE and TOE may show to be inconclusive for patients with suspected prosthetic heart valve endocarditis (PVE) or cardiac implanted electronic device (CIED) endocarditis because of the presence of an artifact. In certain situations, supplementary tests such as 18F-FDG-PET/CT or radiolabelled leucocyte single-photon emission computed tomography-CT (SPECT-CT)

may be done to see if the prosthetic heart valve is inflamed or infected, which would support a diagnosis of IE[6].

➔ Vegetation size: In comparison to two-dimensional transesophageal echocardiography, real-time three-dimensional transesophageal echocardiography (RT3DTEE) has improved vegetation characterization (2DTEE). The vegetations were larger and covered a larger area when measured using RT3DTEE as opposed to 2DTEE[24].

➔ Accepted biomarkers:

- A high CRP level (CRP > 40 mg/L) is a reliable indicator.

Mean platelet volume (MPV), which is linked to platelet activation and function, has drawn more attention as a result of its ability to predict the development of embolism problems in IE patients. Patients with EE had higher MPV levels than people without EE, according to research.

- Anti-2-glycoprotein I (2GPI) antibodies have been shown to boost platelet activation, which raises the risk of thrombosis and the development of vegetation in IE patients.

- Because of the accelerated fibrin turnover, a high D-Dimer level may be a useful indicator of embolism.

- The presence of microbial infection and inflammation causes troponin I to be often increased in IE.

- MMPs can disrupt the tenuous connection between plants and the diseased cardiac tissues to which they are attached, meaning that elevated blood levels of MMPs may be used to predict the development of EE[24].

Duke's criteria are used to diagnose infective endocarditis which consists of pathological criteria to detect microorganisms and to confirm the presence of endocarditis and clinical criteria which require blood cultures, echocardiographic findings and clinical symptoms[15,13]. Based on the guidelines diagnosis of infective endocarditis should be done only after obtaining more number of positive culture reports. Even though it has been reported that the introduction of echocardiography does not reduce the undetected rate of diagnosis, transesophageal echocardiography is used for diagnosis in suspected infective endocarditis cases[15]. Metagenomic next-generation sequencing (mNGS) will be used as a diagnostic tool

in the future to sequence and identify nucleic acids from a mixed population of microorganisms [1].

## DIAGNOSIS OF INFECTIVE ENDOCARDITIS ASSOCIATED WITH CAUSATIVE AGENT

C. burnetti and Bartonella spp. are the two most frequent causative factors of culture-negative endocarditis. Serological testing is typically used to diagnose them. Whipple's bacillus endocarditis was identified through a comprehensive pathologic evaluation of removed cardiac valves using periodic acid–Schiff (PAS) staining and molecular techniques. When an etiologic diagnosis cannot be determined through standard laboratory testing, a pathologic inspection of the valve using specialized stainings, such as Warthin-Starry, Gimenez, and PAS, and broad-spectrum PCR should be carried out consistently[28].

## CHALLENGES IN INFECTIVE ENDOCARDITIS

One of the main problems with the illness is getting a quick and accurate diagnosis in instances of probable IE. Delayed identification and therapy initiation result in problems and poorer clinical results. Acute sepsis, a sluggish low-grade fever sickness, a cardiac failure syndrome, or a stroke are just a few of the clinical presentations that are infamously varied. Furthermore, patients with prosthetic valve endocarditis (PVE) or cardiac device infection (CDI) have a reduced sensitivity for the modified Duke criteria, which were initially developed for research reasons and recommended by AHA recommendations for the assessment of patients with suspected IE (59, 60). Due to ambiguous or inconclusive results on echocardiograms or blood cultures, up to 30% of patients with later-proven IE are classified as "possible"[9].

## MANAGEMENT

The current 2015 European Cardiology Society guidelines suggest that for the management of infective endocarditis, antibiotics are indicated as empirical therapy for multiple weeks and in targeted population for 6-8 weeks. For proper selection of antibiotics, identification of particular causative agent that had caused IE is very essential. The primary aim of the treatment is to eradicate the pathogenic organism. The presence of lesions in the vegetation with more bacteria and disturbance in antibiotic penetration due to bioflim formation affects the treatment of IE [2].

Antibiotic therapy along with cardiac surgery is the standard treatment for IE. Clinical practice guideline suggests that in certain group of patients, the administration of only 2 weeks of antibiotic treatment is recommended, moreover reports of prospective studies and clinical trial had shown the safety and efficacy of this treatment in left-sided native valve IE caused by penicillins sensitive streptococci and in right-sided IE associated with the use of parenteral drugs caused by methicillin sensitive S. aureus [12]. Initial therapy for the management of S.aureus infection is rifampin [3].

The role of multidisciplinary has become very important in the 21st century for the management of left sided endocarditis which is an infection of the entire mitral or aortic valve , as well as the structure that are connected to them anatomically [16].They had become the standard of care for the diagnosis and treatment of endocarditis since their initial publication in 2009 demonstrating the mortality benefits associated with endocarditis[17].Oxacillin is a better antibiotic for Methicillin Sensitive Staphylococcus Aureus (MSSA) than vancomycin once the organism is identified which indicates the need for de-escalation of antibiotics [18].

If the vegetation is greater than 20mm surgery will be recommended according to European Society of Cardiology [18].For various groups of patients who affected with LSE surgery is considered as most effective treatment. Samura et al. conducted a study in 152 patients to evaluate the impact of emergency surgery in patients with left-sided native valve IE complicated with acute cerebral infarction and they concluded that there was a high rate of death in patients who underwent delayed surgery compared to those who received early valve operation [16].

1) Antibiotic treatment:

The sulfonamides, which were made accessible in the middle of the 1930s, were the first truly effective antibacterial agents. Early trials with oral sulfonamides in the treatment of IE were unsatisfactory [26]. It is crucial to choose the right bactericidal regimen and administer it for the right amount of time if you want to successfully treat this illness.

a) Aminoglycosides:

Gentamicin dosage needs to be based on actual body weight. Serum gentamicin levels should be monitored frequently when used to treat Gram-positive endocarditis to ensure pre-dose (trough) levels stay below 1 mg/L and post-dose levels stay between 3 and 5 mg/L. Patients

with compromised renal function should have their doses modified daily to account for measured or estimated creatinine clearance. Use regional protocols to track and modify dose regimens if "once-daily" gentamicin dosing regimens (such as the Hartford regimen) are employed to treat IE brought on by Enterobacteriaceae or Pseudomonas aeruginosa[23].

b) Glycopeptides:

- Vancomycin dosage and level monitoring should be done in accordance with regional protocols. Its levels should be tracked, and the dosage should be changed as necessary, to keep the serum pre-dose level between 15 and 20 mg/L.

- Teicoplanin should be started at a high dose (10 mg/kg body weight every 12 hours, then 10 mg/kg daily), with the interval between doses being modified based on renal function. To guarantee values of 20 mg/L, teicoplanin serum trough levels must be monitored at least once a week. When gentamicin combination therapy is essential, teicoplanin—which is less nephrotoxic than vancomycin—should be taken into consideration for sensitive isolates (apart from staphylococci)[23].

c) Beta-Lactams:

Both ampicillin and amoxicillin are acceptable antibiotics since they are regarded as microbiologically identical. For susceptible isolates, benzylpenicillin may be substituted with amoxicillin, which has a wider spectrum and a higher risk of causing Clostridium difficile infection. Penicillin should be administered six times daily due to the fact that due to its short serum half-life, and the time-dependent killing of streptococci by penicillin[23]**.** The inability of -lactams to kill bacteria and the propensity of E. faecalis to develop biofilm at a higher rate than E. faecium (87%-95% vs 16%-29%, respectively) make treating E. faecalis infections difficult and may be a factor in the steady mortality rates[22]**.**

Endocarditis complications and surgical indications: Up to 50% of patients will need surgery for IE. The following are the main signs of this:

➢ Heart failure

- cardiogenic shock due to progressively developing natural or artificial valve blockage, regurgitation, or fistula - urgent surgery (within 24 hours).

- significant valve dysfunction, heart failure symptoms, and a subpar hemodynamic response - immediate surgery (7 days).

➢ Failure to control infection – urgent surgery

- local continuing infection, such as an aortic root abscess, aneurysm, or growing vegetation.

- a difficult-to-treat bacterial infection (fungi or multiresistant organism, staphylococci or non-HACEK Gram-negative bacilli on a prosthetic valve).

- the continued presence of positive blood cultures in spite of the use of effective antibiotics or insufficient management of metastatic septic foci.

➢ Prevention of septic emboli – urgent surgery

- Vegetation >10 mm with an embolic event while on appropriate antibiotic therapy

- Vegetation >30 mm

- Vegetation >10 mm and severe native or prosthetic valve disease and the patient is at low operative risk[6].

➢ For new embolic events, vegetation size and movement are crucial echocardiographic indicators [38].

**FOLLOW UP:**

Within the first 12 months following endocarditis treatment, the majority of problems happen. Based on the clinical circumstances, vigilant follow-up should be done ideally at 1, 3, and 6 months[6].

In both native valve endocarditis (NVE) and prosthetic valve endocarditis, the main reasons for mortality were heart failure, annular and myocardial infections, heart block, and coronary embolism, which are most commonly seen with staphylococcal and fungus endocarditis (PVE). In NVE, surgery substantially increased survival in all patients with staphylococcal endocarditis and in patients with mild or severe heart failure. Surgery had a substantial impact on mortality in PVE patients with intermediate or severe heart failure as well as in the total cohort of late PVE patients.[29].

## SURGICAL MANAGEMENT FOR ENDOCARDITIS

### Surgical procedures

Control of infection and restoration of cardiac morphology are the 2 main goals of the surgery. The type of prosthesis used (mechanical versus biological) or the mode of surgery (replacement versus repair) have no bearing on operative mortality, though repair techniques, when appropriate, offer long-term benefits, such as a decreased risk of late complications (particularly recurrent IE) and the avoidance of the need for lifelong anticoagulation. The use of homografts in aortic IE remains debatable due to a greater risk of late complications, but they do give a decreased risk of recurring infection. In the most severe situations with repeated prosthetic valve endocarditis, cardiac transplantation may be contemplated.[31].

### Preoperative Guidance - Prior to Surgery: The Considerations

Transthoracic and transesophageal ultrasound are now ubiquitous, and it is well-known how useful they are for assessing IE complications. Transesophageal imaging is advised for almost all patients whose operation is being considered due to its better sensitivity and specificity.

Cardiac catheterization is not obligated for structural definition or diagnosis. Men over the age of 40, postmenopausal women, people with a history of ischemic heart disease, and those with a poor risk factor profile should all be given coronary angiography consideration. However, care must be taken if there are large aortic vegetations because they could become dislodged by catheter manipulation. If accessible, alternative noninvasive methods like MRI or multislice CT may be used.[31].

## TO DODGE ENDOCARDITIS

Although preferable, using antimicrobial drugs to prevent endocardial infection is not always feasible. Many possible causes of bacteremia are not immediately apparent, and some bacteremias happen accidentally (such as those linked to food consumption or oral hygiene practices) and cannot rationally be avoided. Organisms that may have their origins in the oral region are a major factor in many instances of native valve endocarditis. To lessen these potential causes of bacteremia, all at-risk children should receive the finest oral health education available [30].

## ENDOCARDITIS CATEGORICAL PROPHYLACTICS

In adolescents, where endocarditis is linked to high rates of morbidity and death, prophylaxis is crucial. The risk groups for cardiac conditions are high, intermediate, and insignificant. For those who fall into the high- and moderate-risk categories, prophylaxis is advised; these conditions are mainly classified according to the likelihood of getting endocarditis and the severity of that infection. For instance, people who fall into the high-risk group are much more likely to experience a serious endocardial infection, which is frequently linked to high rates of morbidity and death. There is no higher risk of endocarditis in people with negligible risk than there is in the general community[30].

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