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Management of Infective Endocarditis in Infarction Stroke

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Abstract

Infective endocarditis (IE) is an infection of the endocardial surface of the heart often involving the heart valves characterized by the formation of vegetations composed of bacteria, fibrin, and platelets. The incidence of IE in high-income countries is estimated at 3–10 per 100,000 person-years, with regional variations; this rate remains relatively stable, but the complexity of cases and neurological complications remain a major concern. Two authors conducted a comprehensive literature search of PubMed, Scopus, NCBI, and Cochrane CENTRAL databases from 2024 to July 2025. Studies were reviewed based on incidence of infective endocarditis in infarction stroke. Infective endocarditis remains an important cause of ischemic stroke, primarily through septic emboli originating from vegetations on the heart valves. Key risk factors include the size and mobility of the vegetation, left valve involvement (mitral or aortic), and virulent pathogens such as Staphylococcus aureus. Clinical vigilance, timely antimicrobial therapy, and careful timing of surgery are key to successful management of ischemic stroke due to IE.

Introduction

Infective endocarditis (IE) is an infection of the endocardial surface of the heart often involving the heart valves characterized by the formation of vegetations composed of bacteria, fibrin, and platelets (Thiene, G., & Basso, 2006; Witten et al., 2025). These vegetations can release septic-laden fragments (septic emboli) into the systemic circulation, causing vascular occlusion in target organs, including cerebral arteries, leading to ischemic stroke (Li et al., 2024). According to Stoneham et al. (2021) and Vogkou et al. (2016) The etiology of IE involves bacteria (most commonly Staphylococcus aureus, streptococci, enterococci) or, less commonly, fungi; bacteria enter the bloodstream (bacteremia) from sources such as skin infections, catheter insertion, invasive procedures, or intravenous drug use.

On the damaged endocardial surface, microorganisms adhere and promote the formation of vegetations; large and/or mobile vegetations pose a risk of releasing septic emboli to the brain (Stawicki, 2018; Wahab et al., 2025; Kaushik et al., 2024). In addition to ischemic obstruction, the infectious component of vegetations can cause vascular inflammation, cerebral abscess, or mycotic aneurysm formation, increasing the risk of bleeding (Cabezon et al., 2024; Ielapi et al., 2021; Wilson et al., 2016). The incidence of IE in high-income countries is estimated at 3–10 per 100,000 person-years, with regional variations; this rate remains relatively stable, but the complexity of cases and neurological complications remain a major concern (Fan et al., 2023; Bray et al., 2018).

Recent studies and reviews report that approximately 20–40% of IE patients experience neurological complications, including ischemic stroke, during the course of their disease; with

routine brain imaging, asymptomatic ischemic lesions are more frequently detected. Furthermore, surveillance in several regions has shown an increase in IE cases associated with intravenous opioid (IV) drug use, contributing to the peak incidence of stroke in young populations in some countries (Satish et al., 2025; Imoisili, 2024; Salehi et al., 2019; Westover et al., 2007; Volkow & Blanco, 2023).

Ischemic stroke in IE is a clinical emergency because it is associated with higher morbidity and mortality than non-IE stroke; neurological complications also influence the timing of valve surgery, acute reperfusion strategies (contraindications to intravenous thrombolysis when IE is suspected), and the need for selective endovascular intervention (mechanical thrombectomy) in large vessel occlusions. The 2023 ESC guidelines emphasize the importance of a multidisciplinary approach (endocarditis team) and state that after TIA or non-hemorrhagic stroke, necessary cardiac surgery should not always be postponed, whereas cases with intracranial hemorrhage require delay and careful risk assessment. Therefore, early detection of IE in stroke patients (blood cultures, TTE/TEE, neuroimaging) and rapid coordination between specialists are crucial to reduce adverse outcomes (Delgado et al., 2023; Santos-Patarroyo et al., 2025; Basem et al., 2025; Babes et al., 2023) Therefore, researchers want to know more about the incidence of infective endocarditis in infarction stroke.

Methods

The methodology used in this study was narrative literature review, this allowed the investigators to synthesise, judge and place the current scientific evidence regarding the management of infective endocarditis in the context of ischemic stroke. To ensure academic rigour and reduce selection bias, two authors independently completed the review. The search of the literature has covered four major biomedical databases, including PubMed, Scopus, NCBI, and Cochrane CENTRAL, with the search being restricted to the period between 2024 and July 2025 to make sure that evidence represents the current clinical practice. A set of search terms that were relevant, such as infective endocarditis, ischaemia or infarction stroke, clinical management, antimicrobial therapy and valve surgery were refined in a series of iterations to ensure the search term maximised agreement with the objectives of the review.

The first level of screening was done by a methodical review of the titles and abstracts of the retrieved records. Manuscripts which were not related to stroke management or covered infective endocarditis solely on a pathophysiological basis and did not give therapeutic implications were also excluded. The entire text of the chosen articles was then carefully reviewed to ensure that all of the articles included a meaningful discourse on diagnosis, treatment interventions, when cardiac surgery is administered, administration of antibiotics, and reperfusion in patients with infective endocarditis. This strict inclusion was necessary to make sure that the literature studied was thorough but narrowed down to the clinical question under consideration.

Information was extracted through a thorough close reading of every publication so that the investigators were able to identify conceptual connections between the pathogenesis of cardiac vegetations and cerebrovascular embolic events. Primary results were then grouped into thematic sectors which included diagnostic work-up, antimicrobial guidelines, surgical decision-making, the possibility of mechanical thrombectomy, and the risk of thrombolytic therapy. During the process, cross-articles comparison was undertaken by the authors and areas of agreement were observed with some clinical mysteries still persisting to elicit debate in the field of cardiology and stroke. The comparative analysis helped to make the shift between summarisation and synthesis, which critically incorporates the evidence.

The current research is therefore more than a mere compilation of previous information, as it hopes to create a sensible scholarly discourse, a detailed representation of how the changing guidelines recommendations can be used to guide clinical decision making in the real world. The following results and discussion sections can be viewed as the final phase of the interpretative engagement with the chosen literature, which is logically structured to make the steps of the methodology meet the insights that were revealed.

Results and Discussion

General Description

Infectious endocarditis is an important cause of ischemic stroke in patients with cardiac infection; large reviews report that approximately 20–40% of patients with IE develop a clinical ischemic stroke during their disease, and a higher proportion will have asymptomatic ischemic lesions on routine brain imaging. Because of this significant number, clinical suspicion of IE should be raised in stroke patients with signs of systemic infection (fever, leukocytosis) or new cardiac findings such as murmurs (Sanguettoli et al., 2024).

Pathophysiology and Risk Factors

IE occurs due to the attachment of microorganisms from the blood circulation to the endocardial surface, then multiplying, especially on damaged valves. These attached bacteria form colonies, which are then drawn from the blood for nutrients. The presence of these bacterial colonies facilitates thrombosis, an event facilitated by thromboplastin, which is produced by leukocytes reacting with fibrin. The new fibrin network surrounds the bacterial colonies and causes vegetation to grow. The endocardial areas frequently affected are the mitral and aortic valves. Vegetation also occurs in areas with jet lesions, causing the endothelium to become rough and fibrosis to occur.

Turbulence also occurs, affecting the endothelium. Vegetation can be small to large, white to brown in color. Colonies of microorganisms are mixed with platelet fibrin, surrounding which an inflammatory reaction occurs (Braï et al., 2023). Stroke in IE is usually caused by septic emboli from vegetations on the left heart valve (aortic or mitral) that break loose and obstruct a cerebral vessel. The risk of embolism is influenced primarily by the characteristics of the vegetation size (larger vegetations) and mobility (more mobile vegetations) are strong predictors of embolic events and by the type of pathogen (Staphylococcus aureus tends to be more emboligenic). Several studies have shown that each 1 mm increase in vegetation size significantly increases the embolic risk, so that a size greater than 10 mm is often used as a criterion for early surgery to prevent recurrent embolism (Baddour et al., 2023; Delgado et al., 2023).

Clinical Presentation and Recommended Investigations

Clinical manifestations will appear two weeks after the precipitating factor occurs. Onset is usually gradual, with a mild fever. This occurs when the pathogenicity of the bacteria is low. If the bacteria are highly pathogenic, the onset is acute, with a high fever. Patients with IE generally experience the following manifestations (Braï et al., 2023): (1) Congestive heart failure, which accounts for 50-70% of cases, is caused by heart valve damage. Infection can spread to other parts of the heart and cause myocarditis, myocardial abscess, arrhythmias, and purulent pericarditis. Examination reveals a rapid pulse due to fever or decreased cardiac function. Irregularities in the rhythm indicate an abscess near the conduction system. The presence of a triad of fever, anemia, and a heart murmur suggests endocarditis. This murmur is due to mitral or aortic valve regurgitation, most commonly due to mitral or aortic insufficiency; (2) Embolism occurs in 30-40% of cases, resulting from the detachment of vegetation from the heart.

Depending on the size and number of emboli, they can affect large blood vessels or cause diffuse micro-emboli to the central nervous system or other organs. Embolism to the nervous system occurs in 50%, to abdominal organs in 40%, and to other organs in 10%, including Janeway lesions (Lee & Siddiqui, 2025); (3) Bacteremia, causing general symptoms such as fever, sweating, loss of appetite, anemia, splenomegaly, and distant metastasis. Other suspicious symptoms include general weakness, headache, back pain, and arthralgia (Chidurala & Bheemarasetti, 2022); (4) Immunological reactions cause hypergammaglobulinemia, immune complex formation, and rheumatoid factor (+). The longer the endocarditis infection lasts, the higher the immune complex levels become, leading to petechiae.

Physical examination of endocarditis patients will reveal signs of infection, including fever, sweating, shivering, weakness, and weight loss. Moderate to severe anemia may cause the patient to appear pale. Vascular phenomena will be prominent, with pethechiae appearing in the blood vessels of the skin and mucous membranes, or due to capillary fragility, which can also be detected with the Teyrniquet test. If the germs are pathogenic, DIC may also occur. Splinter hemorrhage appears as thin, reddish lines under the nails but does not reach the tip of the nail. This is due to micro-emboli in the capillary blood vessels under the nail. Osler's nodes are soft, painful, reddish nodules usually found on the fingers. This is due to the inflammatory process around the distal area of the embolized artery.

Therefore, in the acute phase, the causative germs can be found in this area. It is also suspected to be immunological, especially in sub-acute cases (Braï et al., 2023); (6) Janeway lesions are painless red spots that appear pale when pressed and are less than 5 mm in size. They are usually located on the palms of the hands or soles of the feet. Clubbing of the fingers occurs in 10% of embolism cases. Decreased or absent arterial pulses in the extremities indicate obstruction of a large artery by vegetation fragments. (5) Other symptoms that may arise include splenic, renal, and intestinal infarction, characterized by pain, such as acute abdominal pain and peritonitis. Myocardial infarction can occur due to blockage of the coronary arteries, which can lead to heart failure and death. Neurological complications will further worsen the condition of patients with endocarditis. Other possible clinical manifestations include arthritis, glomerulonephritis, tenosynovitis, clubbing, symptoms of uremia, proteinuria, and hematuria.

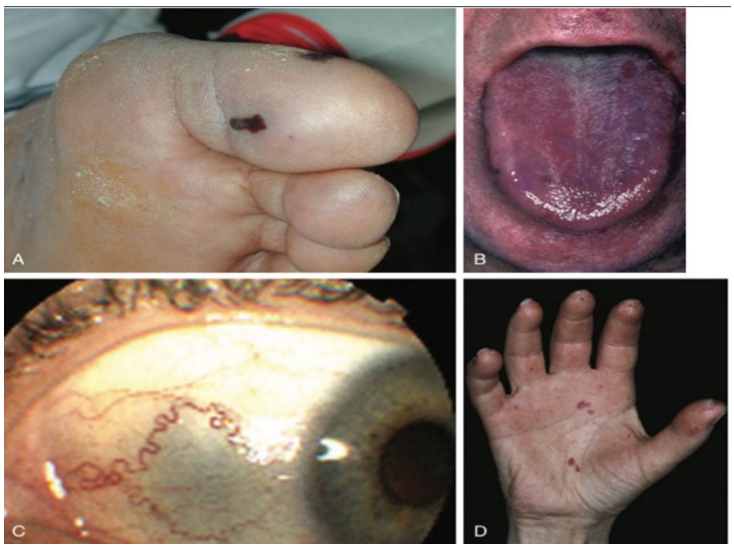
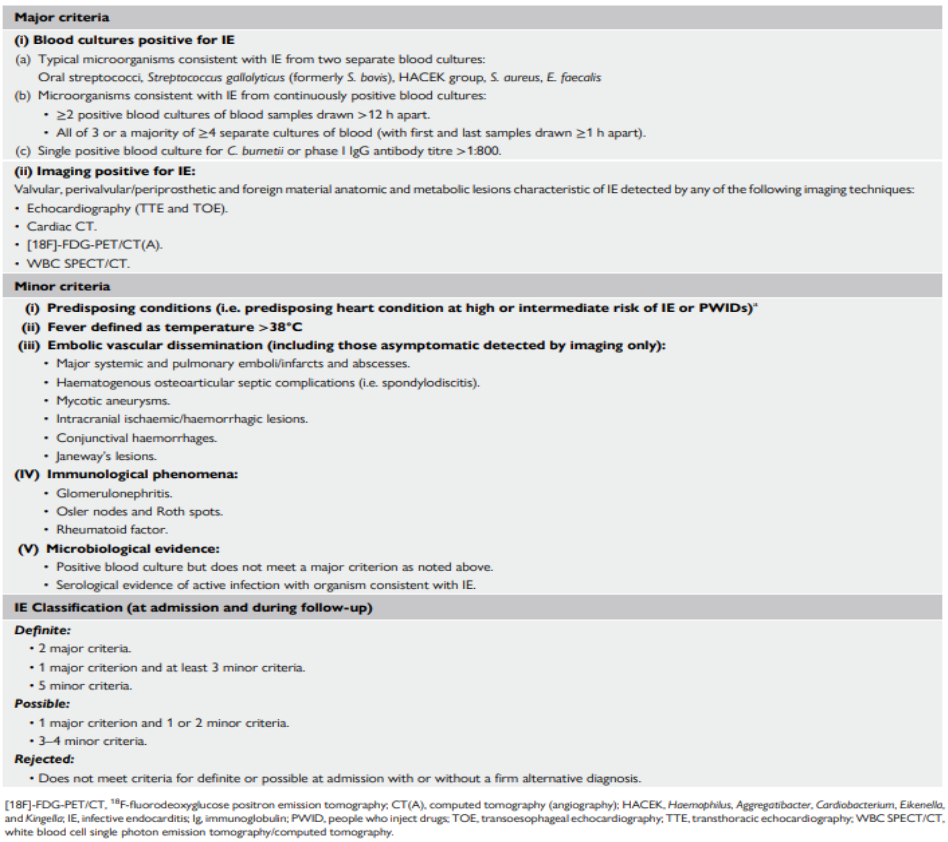


Figure 1. Clinical manifestasion of IE: A. Osler nodes; B. Janeway lesion; C. Roth spots; D. Subungual hemorrhages (Lipner, 2025)

Patients with IE and stroke may present with neurological features typical of ischemic stroke (hemiparesis, aphasia, visual disturbances, etc.) and often present with signs of infection (fever, night sweats) or cardiac symptoms (dyspnea, new murmur). To establish the IE–stroke relationship, a comprehensive workup should be performed: repeated blood draws for culture

(at least 3 draws before antibiotics if possible), initial transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) if TTE is inconclusive or suspicion of vegetation is high, and neuroimaging (head CT/CT-Angiography to detect large occlusions; brain MRI if available to assess ischemic/asymptomatic burden). The 2023 ESC guidelines emphasize immediate cardiac screening in stroke patients with signs of infection, as therapeutic decisions (antibiotics and surgery) depend on these findings, particularly on the major and minor findings presented in the table below.



Major criteria

(i) Blood cultures positive for IE

(a) Typical microorganisms consistent with IE from two separate blood cultures:
Oral streptococci, *Streptococcus gallolyticus* (formerly *S. bovis*), HACEK group, *S. aureus*, *E. faecalis*

(b) Microorganisms consistent with IE from continuously positive blood cultures:
- ≥2 positive blood cultures of blood samples drawn >12 h apart.
- All of 3 or a majority of ≥4 separate cultures of blood (with first and last samples drawn ≥1 h apart).

(c) Single positive blood culture for *C. burnetii* or phase I IgG antibody titre >1:800.

(ii) Imaging positive for IE:

Valvular, perivalvular/periprosthetic and foreign material anatomic and metabolic lesions characteristic of IE detected by any of the following imaging techniques:
- Echocardiography (TTE and TOE).
- Cardiac CT.
- [18F]-FDG-PET/CT(A).
- WBC SPECT/CT.

Minor criteria

(i) Predisposing conditions (i.e. predisposing heart condition at high or intermediate risk of IE or PWIDs)*

(ii) Fever defined as temperature >38°C

(iii) Embolic vascular dissemination (including those asymptomatic detected by imaging only):
- Major systemic and pulmonary emboli/infarcts and abscesses.
- Haematogenous osteoarticular septic complications (i.e. spondylodiscitis).
- Mycotic aneurysms.
- Intracranial ischaemic/haemorrhagic lesions.
- Conjunctival haemorrhages.
- Janeway's lesions.

(IV) Immunological phenomena:
- Glomerulonephritis.
- Osler nodes and Roth spots.
- Rheumatoid factor.

(V) Microbiological evidence:
- Positive blood culture but does not meet a major criterion as noted above.
- Serological evidence of active infection with organism consistent with IE.

IE Classification (at admission and during follow-up)

Definite:
- 2 major criteria.
- 1 major criterion and at least 3 minor criteria.
- 5 minor criteria.

Possible:
- 1 major criterion and 1 or 2 minor criteria.
- 3–4 minor criteria.

Rejected:
- Does not meet criteria for definite or possible at admission with or without a firm alternative diagnosis.

[18F]-FDG-PET/CT, ^{18}F-fluorodeoxyglucose positron emission tomography; CT(A), computed tomography (angiography); HACEK, *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, and *Kingella*; IE, infective endocarditis; Ig, immunoglobulin; PWID, people who inject drugs; TOE, transoesophageal echocardiography; TTE, transthoracic echocardiography; WBC SPECT/CT, white blood cell single photon emission tomography/computed tomography.

Figure 2. Cardiology Modified Diagnostic Criteria of IE (Delgado et al., 2023)

Management for Suspect IE and Definite IE

A microbiologist should be consulted if there is clinical concern about an infection of the inner A microbiologist should be consulted if there is clinical concern about an infection of the inner lining of your heart, but blood tests after 48 hours show no germs. The recommended next step is to use a blood culture diagnostic kit that includes the suspected microorganism. If the results are negative, the next step is to perform systematic serological testing. Serological testing should be performed considering the patient's clinical condition, such as those with severe immunodeficiency, local epidemiology, and the specificity of the test used (Delgado et al., 2023). This layered diagnostic approach is essential because culture-negative endocarditis often results from fastidious organisms or prior antibiotic exposure, which may suppress microbial growth and lead to false-negative standard cultures. In such cases, specialist input becomes crucial to determine which pathogens are most likely based on clinical manifestations, exposure history, comorbidities, and risk factors, thereby guiding targeted laboratory investigations.

Furthermore, the use of diagnostic kits that include suspected microorganisms allows clinicians to broaden the detection window for rare or atypical pathogens that may not be captured by routine cultures. When serological testing is carried out, it aims to identify immune responses specific to organisms such as Coxiella burnetii, Bartonella spp., or Brucella spp., which are recognized causes of culture-negative endocarditis. The interpretation of serology must also

consider antibody levels, potential cross-reactions, and the timing of infection, since patients with immunodeficiency may fail to mount adequate antibody responses. For this reason, clinical judgment, microbiology expertise, and epidemiological context work together as central components in making an accurate diagnosis when conventional laboratory tests remain inconclusive.

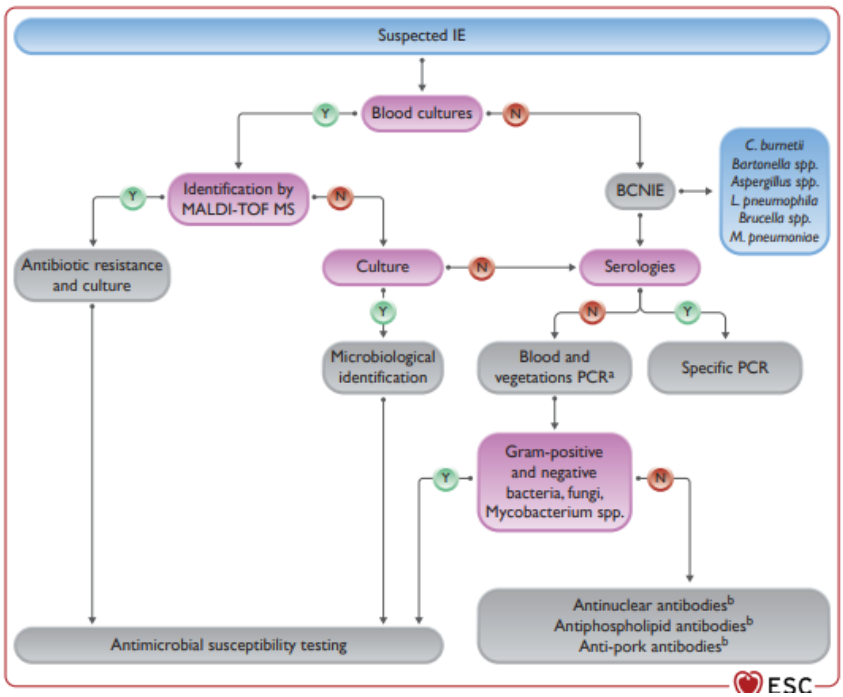


Figure 3. Flowchart Treatment for Suspect IE (Delgado et al., 2023)

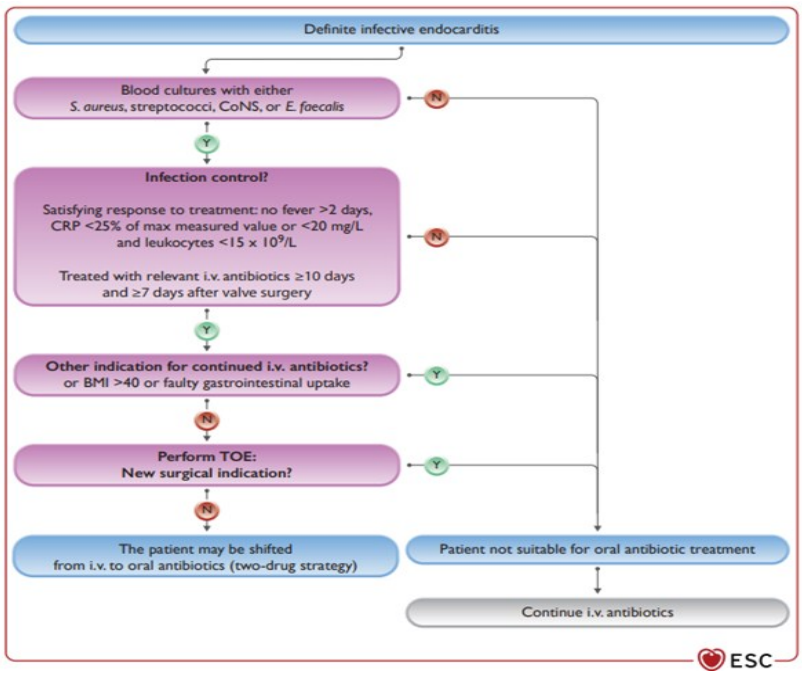


Figure 4. Flowchart Treatment for Definite IE

Stability criteria are very important in planning the patient's care, especially for those undergoing treatment with TOE. These criteria include blood tests, clinical signs, and the TOE itself. OPAT has proven to be a safe option for patients with IE who are stable and can be treated at home. Knowledge of the disease and how to identify infection signs is necessary for the patient, and preferably a caregiver. This involves monitoring their temperature daily and

looking for other indications that the condition is deteriorating or may be experiencing complications. Regular follow-up after discharge is also necessary, including daily checks by a nurse and weekly visits from the responsible doctor. Patients who are enrolled in the OCT program for intravenous catheters must undergo medical examinations and take responsibility for their catheter. Medical professionals must provide additional supervision to patients who are unable to monitor their own condition or lack nearby caregivers. In these cases, home treatment should be evaluated carefully before being recommended ((Delgado et al., 2023; Thapa et al., 2025).

Antibiotic Treatment and the Role of Cardiac Surgery

Management of IE requires antibiotic therapy as soon as possible after blood culture collection (or immediately if cultures are not possible), with an empirical regimen subsequently adjusted based on culture and sensitivity results. Indications for early valve surgery include heart failure due to valve regurgitation, uncontrolled infection (abscess, fistula), and prevention of recurrent embolism especially if large/mobile vegetations or recurrent embolism occur despite on-antibiotic therapy (Gonçalves et al., 2025). The 2023 ESC guidelines detail when emergency (≤24 hours) or urgent (several days) surgery is recommended, but the timing of surgery after the stroke event should be personalized: if the stroke is ischemic without hemorrhage, early cardiac surgery can often be considered; if there is intracranial hemorrhage or large hemorrhagic transformation, delaying surgery is usually necessary to reduce the risk of perioperative bleeding. This decision should be made by a team involving cardiologists, cardiac surgeons, stroke neurologists, and infectious disease specialists (Imazio, 2024).

Treatment of infective endocarditis should begin quickly. Three blood culture samples should be taken every 30 minutes before starting antibiotics. Antibiotics should be given to both native and late prosthetic valve endocarditis, as well as other pathogens like staphylococci, streptococci, and enterococcus. When a patient has been prescribed different antibiotics, the first one should be used. Coagulase-negative staphylococci (CoNS) need to be covered in prosthetic valve endocarditis but not in native valve endocarditis. In cases of early prosthetic valve endocarditis or healthcare-associated infective endovasculoma, the antibiotic regimen should also include methicillin-resistant staphylococci, enterococci, and non-HACEK Gram-negative bacteria. Once a specific germ has been identified, usually within 24 hours, the antibiotic therapy should be adjusted based on the antimicrobial susceptibility test. It is important to switch from the initial empirical treatment to a targeted therapy once the organism is identified within 24 to 48 hours (Delgado et al., 2023).

Recommendations		Class[b]	Level[c]
In patients with community-acquired NVE or late PVE (≥12 months post-surgery), ampicillin in combination with ceftriaxone or with (flu)cloxacillin and gentamicin should be considered using the following doses:[255]		IIa	C
Adult antibiotic dosage and route			
Ampicillin	12 g/day i.v. in 4–6 doses		
Ceftriaxone	4 g/day i.v. or i.m. in 2 doses		
(Flu)cloxacillin	12 g/day i.v. in 4–6 doses		
Gentamicin[d]	3 mg/kg/day i.v. or i.m. in 1 dose		
Paediatric antibiotic dosage and route			
Ampicillin	200–300 mg/kg/day i.v. in 4–6 doses, up to maximum of 12 g/day		
Ceftriaxone	100 mg/kg i.v. or i.m. in 1 dose		
(Flu)cloxacillin	200–300 mg/kg/day i.v. in 4–6 equally divided doses		
Gentamicin[d]	3 mg/kg/day i.v. or i.m. in 3 equally divided doses		
In patients with early PVE (<12 months post-surgery) or nosocomial and non-nosocomial healthcare-associated IE, vancomycin or daptomycin combined with gentamicin and rifampin may be considered using the following doses:[395]		IIb	C
Adult antibiotic dosage and route			
Vancomycin[e]	30 mg/kg/day i.v. in 2 doses		
Daptomycin	10 mg/kg/day i.v. in 1 dose		
Gentamicin[d]	3 mg/kg/day i.v. or i.m. in 1 dose		
Rifampin	900–1200 mg i.v. or orally in 2 or 3 doses		
Paediatric antibiotic dosage and route			
Vancomycin[e]	40 mg/kg/day i.v. in 2–3 equally divided doses		
Gentamicin[d]	3 mg/kg/day i.v. or i.m. in 3 equally divided doses		
Rifampin	20 mg/kg/day i.v. or orally in 3 equally divided doses up to maximum of 900 mg/day		

Allergy to beta-lactams			
In patients with community-acquired NVE or late PVE (≥12 months post-surgery) who are allergic to penicillin, cefazolin, or vancomycin in combination with gentamicin may be considered using the following doses:		IIb	C
Adult antibiotic dosage and route			
Cefazolin	6 g/day i.v. in 3 doses		
Vancomycin[e]	30 mg/kg/day i.v. in 2 doses		
Gentamicin[d]	3 mg/kg/day i.v. or i.m. in 1 dose		

Figure 5. Recommendation for Antibiotics Regimen For IE (Delgado et al., 2023)

Indication for Surgery IE

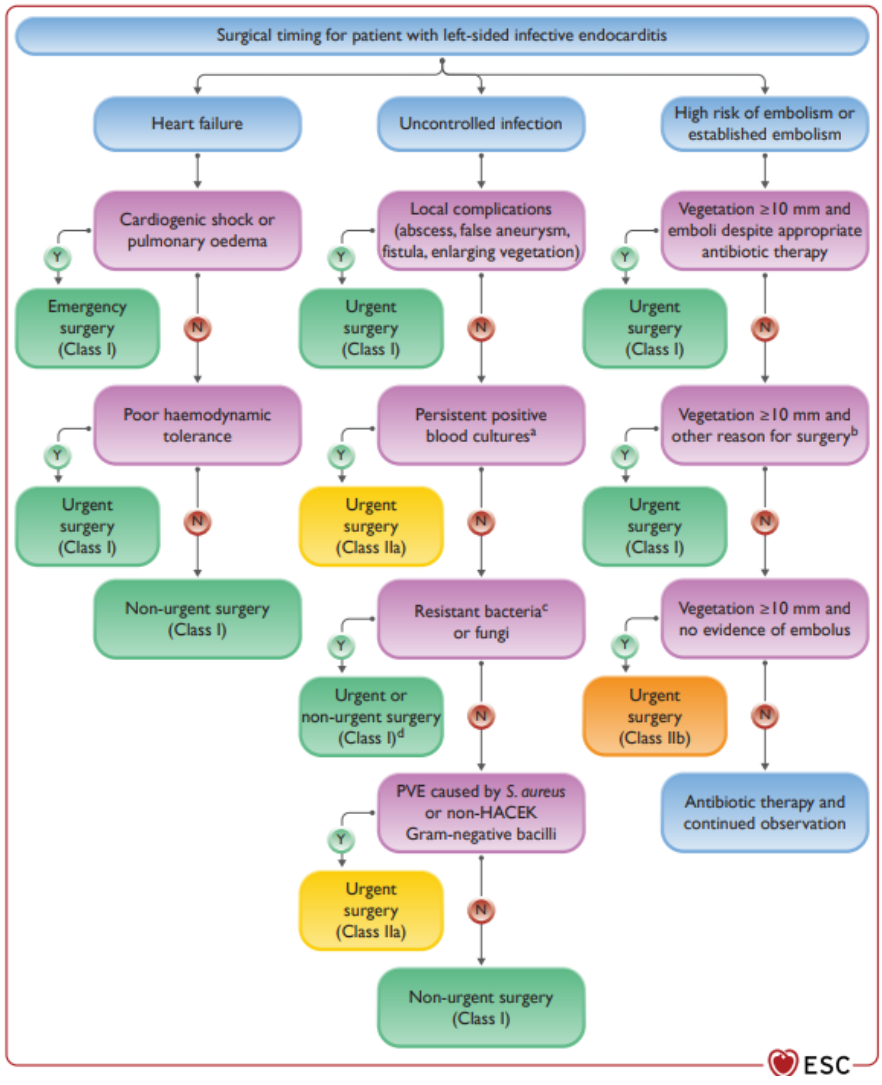


Figure 6. Surgical timing for IE (Delgado et al., 2023)

Surgery is the most suitable approach to manage the serious risks and complications of infectious endocarditis. Even though these patients are at risk of death, research indicates that surgery can raise the chances of survival by up to 20% in the first year. In cases of acute infective endocarditis, surgery is necessary for three reasons: heart failure, an infection that

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cannot be managed by medication, and the prevention of infection from spreading to critical areas like the brain and central nervous system. Many surgeries for infective endocarditis are done quickly. The Task Force has set a guideline that urgent surgery should happen within 3 to 5 days, but once the need for it is clear, delays should be avoided. Even with prolonged use of antibiotics, some patients require emergency surgery within 24 hours. However, there are exception cases. Another group of cases can be handled without rushing, meaning the surgery is scheduled during the same hospital stay. When antibiotics are used to completely eradicate the infection, the reasons and timing for treating any remaining valve issues are in line with standard valve treatment guidelines (Delgado et al., 2023).

Acute Stroke Management: Thrombolysis and Mechanical Thrombectomy (EVT)

The initial steps that need to be taken are patient stabilization and rapid assessment. Patients with AIS and suspected/confirmed IE should be immediately stabilized according to stroke protocols (ABC, glucose, head CT/cerebral angiography if available), but with the awareness that septic etiology/cardiac embolization increases the risk of hemorrhagic complications. Promptly initiate empiric antibiotics according to IE guidelines until culture results are available, as infection control reduces the risk of further embolization and influences the timing of cardiac intervention. Intravenous thrombolysis (IV tPA) is generally recommended to be avoided in patients with stroke caused by IE due to a higher risk of intracranial hemorrhage (bleeding into an infected ischemic area, ruptured mycotic aneurysm) and case series evidence suggesting adverse outcomes (Rodríguez-Montolio et al., 2024; Ashkanani et al., 2018). Conversely, mechanical thrombectomy (EVT) for large-vessel occlusion is beginning to be considered as a recanalization option in IE patients with large-vessel occlusion (LVO). Recent case series and meta-analyses suggest that EVT can achieve high technical recanalization rates, but late functional outcomes are less favorable and the risk of mortality/bleeding tends to be higher than in non-IE patients; therefore, EVT should be considered selectively after case-by-case discussion within a multidisciplinary team and a detailed risk–benefit assessment. Recent meta-analyses (2024–2025) report reasonably high reperfusion rates but warn of worse clinical outcomes overall (Thapa et al., 2025; Gonçalves et al., 2025).

Prognosis

Stroke in the context of IE is associated with higher morbidity and mortality than non-IE stroke; factors that worsen outcomes include vegetation size, infection with virulent organisms, heart failure, and delay in antibiotic therapy or surgery when indicated. Therefore, early detection of IE, blood culture collection, prompt cardiac imaging, and rapid decisions regarding antimicrobial therapy and surgery are key to improving outcomes. Clear communication to patients/families regarding why conventional thrombolysis may be unsafe, as well as the potential need for invasive intervention (EVT/valve surgery), is essential for sensitive and humane shared decision-making (Kremer et al., 2023).

Conclusion

Infective endocarditis remains an important cause of ischemic stroke, primarily through septic emboli originating from vegetations on the heart valves. In the past five years, evidence has shown that approximately one-fifth to one-third of IE patients will experience neurologic complications, with ischemic stroke being the most common manifestation. Key risk factors include the size and mobility of the vegetation, left valve involvement (mitral or aortic), and virulent pathogens such as Staphylococcus aureus. Clinical vigilance, timely antimicrobial therapy, careful timing of surgery, and mechanical thrombectomy are key to successful management of stroke infarc due to IE.

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