



Healthcare-Associated Infective Endocarditis From Central-Line Cannulation And Prosthetic Valvular Replacement In A Resource-Limited Setting Tertiary Health Institution, Southwest, Nigeria: A Case Series

Article History:

Received: 04-01-2025
Accepted: 27-01-2025
Publication: 16-04-2025

Cite this article as:

Oguntola, B. O., Adesina, O. B., Fasaanu, A. N., & Irek, E. O. (2025). Healthcare-Associated Infective Endocarditis From Central-Line Cannulation And Prosthetic Valvular Replacement In A Resource-Limited Setting Tertiary Health Institution, Southwest, Nigeria: A Case Series. *Journal of Heart Valve Disease Innovation*, 30(1). 38-44. doi.org/10.36923/jhvd.v30i1.254

©2025 by author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution License 4.0 International License.

Corresponding Author(s):

Emmanuel Oladayo Irek

Department of Medical Microbiology and Parasitology, Afe Babalola University Multi-system Hospital, Ado-Ekiti, Ekiti, Nigeria. Email: dj1irek@yahoo.com

Busayo Onafowoke Oguntola¹, **Oluwaseyi Bisola Adesina²**, **Ayodeji Nelson Fasaanu³**, **Emmanuel Oladayo Irek⁴**

Abstract: Infective endocarditis (IE) is a rare but life-threatening cardiac condition. Healthcare-associated infective endocarditis (HAIE) is an emerging subtype that occurs more than 48 hours after hospital admission, often following invasive procedures. Its occurrence in patients with end-stage kidney disease (ESKD) is not uncommon. Prosthetic valve endocarditis may also be healthcare-associated (HA-PVE) when it develops within one year of valve replacement. Antimicrobial-resistant *Staphylococcus* species are increasingly implicated in both forms. Amidst a paucity of data on HAIE in Nigeria, we report a case series involving both native valve HAIE in ESKD patients and prosthetic valve HAIE (HA-PVE). These cases were identified over one year in a 150-bed private tertiary healthcare institution in Southwest Nigeria. Patients were diagnosed based on the modified Duke criteria. HAIE was attributed to central-line cannulation in ESKD patients, while HA-PVE occurred within a year of valve surgery. Methicillin-resistant *Staphylococcus* species were the causative pathogens. Elevated erythrocyte sedimentation rates and neutrophilia were consistent findings. This study emphasizes the importance of early and thorough cardiovascular evaluation in ESKD patients with central-line access to facilitate early IE detection. Hemogram and sepsis biomarkers may serve as adjuncts to blood culture in confirming the diagnosis. Glycopeptide antibiotics should be considered as first-line empirical therapy. Healthcare-associated IE involving native and prosthetic valves can present with diverse symptoms. The aforementioned diagnostic and management strategies are particularly relevant in resource-limited settings.

Keywords: Infective Endocarditis, Healthcare-Associated Infection, Methicillin-Resistant *Staphylococcus aureus*, Prosthetic Cardiac Valve Replacement

1. Introduction

Infective endocarditis (IE) is an infection of the endocardial surface and heart valves and is potentially life-threatening (Baddour et al., 2015; Cimmino et al., 2023). It is a rare condition in the general population, with an incidence of 1.5–11.6 cases per 100,000 people (Holland et al., 2016; Tleyjeh & Abdulhak, 2018). In-hospital mortality attributed to IE is about 20% both in Africa and globally (Cahill & Prendergast, 2016; Noubiap et al., 2022).

Historically, IE has been classified based on the progression of the cardiac lesion into acute, subacute, or chronic forms (Geller, 2013). The acute form is usually caused by organisms such as *Staphylococcus aureus*, *Streptococcus pyogenes*, and *Streptococcus pneumoniae*. This type is associated with mortality within a few days to less than six weeks and typically affects native valves (Cunha et al., 1996). The subacute and chronic forms progress more slowly and mostly affect prosthetic valves, with mortality occurring beyond six weeks (Al-Shura, 2014).

An emerging classification of IE is healthcare-associated (or hospital-acquired) IE (HAIE), which manifests more than 48 hours after hospital admission or within six months following hospital-based invasive procedures such as central-line insertion (Ben-Ami et al., 2004; Lomas et al., 2010). This is a particularly concerning form of IE due to its atypical and non-specific symptomatology in otherwise healthy individuals receiving healthcare. The incidence of HAIE is approximately 27 cases per 100,000 persons, accounting for about 16% of cases (Ben-Ami et al., 2004; Lomas et al., 2010).

Healthcare-associated prosthetic valve endocarditis (HA-PVE) occurs within the first year following valve replacement, with an incidence ranging from 13% to 17% (Mkoko et al., 2022). Both HAIE and HA-PVE are increasingly associated with *Staphylococcus aureus* or other staphylococcal species as causative agents in culture

¹Cardiology Unit, Department of Internal Medicine, Afe Babalola University Multi-System Hospital/Afe Babalola University, Nigeria

²Department of Internal Medicine, Afe Babalola University Multi-System Hospital/Afe Babalola University, Nigeria

³Nephrology unit, Department of Internal Medicine, Afe Babalola University Multi-System Hospital/Afe Babalola University, Nigeria

⁴Department of Medical Microbiology and Parasitology, Afe Babalola University Multi-system Hospital, Nigeria

positive cases (Benito et al., 2009; Kouijzer et al., 2022). The emergence of methicillin-resistant staphylococcal species further complicates the management of HAIE and HA-PVE. These conditions are particularly concerning in patients with underlying renal disease, in whom both morbidity and mortality are elevated.

Amidst scattered reports of IE cases in Nigeria (Ige et al., 2017; Uguru et al., 2016) and a general scarcity of data from Africa (Pecoraro & Doubell, 2020), we report cases of HAIE and HA-PVE among patients presenting between January and September 2024 at a private tertiary health institution. We also describe approaches for early detection and effective management.

2. Ethical Considerations

Informed consent: Written informed consent was obtained from the patients or their next of kin (in the case of demise) after adequate explanation was provided and confidentiality was assured.

Data protection: The patients' identifying information was stored on a password-protected computer, accessible only to the authors and the managing team.

3. Case Presentations

Case 1 – Mr. O.S., a 44-year-old male with end-stage kidney disease (ESKD) secondary to chronic glomerulonephritis diagnosed three years ago, was admitted to the facility due to a fever (38.5°C) of seven days' duration and generalized body weakness lasting two weeks. He had been receiving haemodialysis via a tunneled right internal jugular catheter (IJC), which was inserted three weeks prior to admission due to worsening renal parameters: creatinine – 1031.8 µmol/L (reference range: 59–112), urea – 29 mmol/L (2.9–7.5), potassium – 4.8 mmol/L (3.5–5.0).

There were no clinical signs suggestive of localized infection, and the malaria rapid test (histidine-rich protein-2) was negative. He was initially managed for central line-associated bloodstream infection (CLABSI) and uremic encephalopathy in the context of ESKD.

On admission, his Glasgow Coma Score was 14/15. He had a left-sided cranial nerve VII upper motor neuron lesion and asymmetrical limb weakness: power was 3/5 in the left upper and lower limbs, while the right upper and lower limbs had at least 4/5 strength. Cranial magnetic resonance imaging (MRI) revealed hyperintense lesions in the left parietal area and multiple frontal lesions, indicating acute septic infarcts in the left precentral gyrus, right periventricular white matter, right subependymal region, and right cuneus.

Laboratory results showed a total white blood cell count of 30,900 cells/µL (4,000–11,000), neutrophil count of 89.2% (50–70%), haemoglobin concentration of 8.0 g/dL (13.5–17.5), and an erythrocyte sedimentation rate (ESR) of 140 mm/hr (<15 mm/hr). He was empirically started on intravenous (IV) vancomycin 300 mg (renal-adjusted dose) every 8 hours and IV gentamicin 80 mg every 12 hours for 72 hours while awaiting blood culture results.

Cardiovascular examination revealed a pulse rate of 96 beats per minute, blood pressure of 139/73 mmHg, and an apex beat located at the 6th left intercostal space along the anterior axillary line. Heart sounds S1 and S2 were audible, with a diastolic murmur heard at the left sternal border. Transthoracic echocardiography showed vegetations on the mitral, aortic, and tricuspid valves, with moderate to severe aortic regurgitation. The patient had a normal left ventricular (LV) systolic function (Teicholz EF of 58%) and Grade I LV diastolic dysfunction. The right ventricle (RV) was of normal size with preserved systolic function.

Blood cultures processed using a semi-automated system revealed methicillin-resistant *Staphylococcus aureus* (MRSA), sensitive to aminoglycosides, tigecycline, clindamycin, azithromycin, and linezolid. A final diagnosis of HAIE secondary to CLABSI, intracerebral septic infarcts with a facial nerve upper motor neuron lesion, and ESKD was made. The antibiotic regimen was extended for three weeks, and the central line was changed. A repeat blood culture at day 21 showed no bacterial growth. The patient was discharged after six weeks of admission to continue maintenance dialysis as an outpatient. However, he re-presented acutely to a local hospital less than two weeks later and subsequently died. Details surrounding his demise could not be fully ascertained.

Case 2 – Mrs. O.D.O., a 55-year-old woman with ESKD secondary to hypertensive nephropathy and chronic atrial fibrillation (both diagnosed two months before admission), presented to the facility with a high-grade fever of one day's duration and left hip pain for one week. There was no history of trauma. She had been on maintenance haemodialysis via IJC for two months, with her last dialysis session occurring six days before presentation. She was taking bisoprolol 2.5 mg daily for chronic atrial fibrillation.

On examination, she appeared acutely ill, mildly pale, and febrile (39.0°C). Her pulse was 220 beats per minute (normal: 70–80), irregular, and of small volume. Her blood pressure was 81/44 mmHg (normal: at least 90/60 mmHg). Transthoracic echocardiography revealed impaired LV systolic function (Simpson's biplane EF of 42.3%), a large vegetation (1.41 × 0.64 cm) on the posterior tricuspid valve leaflet, and mild to moderate tricuspid regurgitation. The RV was normal in size but had impaired systolic function.

Malaria rapid testing (histidine-rich protein-2) was negative. Blood culture yielded MRSA, susceptible to tigecycline, clindamycin, azithromycin, and linezolid. Laboratory results included: total white blood cell count – 6,600 cells/µL, neutrophils – 92.3%, haemoglobin – 9 g/dL (11–15), serum potassium – 5.6 mmol/L, urea – 25.3

mg/dL, creatinine – 1330 μ mol/L, calcium – 2.04 mmol/L (2.05–2.55), uric acid – 0.37 mmol/L (0.15–0.35), phosphorus – 1.88 mmol/L (0.74–1.52), and ESR – 100 mm/hr (<15 mm/hr).

She was initially commenced on IV vancomycin 300 mg every 8 hours and IV gentamicin 80 mg every 12 hours for 72 hours. However, IV gentamicin was discontinued due to antibiotic resistance, and IV vancomycin was continued for four weeks. She remained on haemodialysis and was managed for septic shock secondary to HAIE from CLABSI on the background of ESKD. Following serial dialysis sessions and antibiotic therapy, blood parameters normalized, and systemic inflammatory signs resolved within one week of treatment initiation. Blood cultures were negative after two weeks.

Despite initial clinical improvement, her renal function deteriorated significantly over time. Her condition regressed clinically, and she eventually died on the 43rd day of admission.

Case 3- Mr. O.D., a 44-year-old man being managed for ESKD secondary to chronic glomerulonephritis, was admitted with a one-day history of fever. He had a right internal jugular catheter (IJC) inserted five months earlier for maintenance dialysis at another tertiary hospital.

On presentation to the facility, he was in respiratory distress (respiratory rate: 40 cycles per minute), febrile (38.3°C), pale, and mildly dehydrated. He had a bounding, rapid pulse of 119 beats per minute and a blood pressure of 186/93 mmHg (which was subsequently corrected). An initial clinical assessment of CLABSI and ESKD was made after ruling out other organic causes of infection and malaria.

He was commenced on IV vancomycin 300 mg every 8 hours and IV amikacin 200 mg every 12 hours for 72 hours. Blood culture isolated methicillin-resistant coagulase-negative *Staphylococcus* (MRCoNS), susceptible to tigecycline, clindamycin, and linezolid. Amikacin was subsequently discontinued based on the antibiotic resistance profile.

Laboratory findings included: total white cell count – 15,700 cells/ μ L, neutrophil count – 86.3%, haemoglobin concentration – 7.6 g/dL, potassium – 4.6 mmol/L, urea – 21.2 mmol/L, creatinine – 608 μ mol/L, ESR – 33 mm/hr.

Nine days into admission, the patient developed a murmur heard over the aortic region (upper left parasternal area), along with a third heart sound (S3). Transthoracic echocardiography revealed a dilated left ventricle (LV) with normal systolic function (Simpson's biplane EF of 58.4%), concentric LV hypertrophy, bi-atrial dilatation, grade III LV diastolic dysfunction, and multiple vegetations on the aortic valve cusps with severe aortic regurgitation.

An electrocardiogram (ECG) showed sinus tachycardia with ventricular premature complexes, left axis deviation, left atrial abnormalities, a short PR interval, left ventricular hypertrophy, and anteroseptal T-wave inversion. Auscultation revealed bibasal fine crepitations in the lungs. Ascites and bilateral pedal oedema were also present.

The final diagnosis was ESKD with HAIE secondary to CLABSI, complicated by severe aortic regurgitation. IV vancomycin 300 mg every 8 hours was continued for two weeks, initially leading to clinical improvement. However, in the third week, the patient's condition deteriorated despite supportive interventions. Repeat echocardiography revealed worsening cardiac function, concentric LV hypertrophy, bi-atrial dilatation, grade III LV diastolic dysfunction, and perforation of the aortic right coronary cusp with worsening aortic regurgitation. Multiple vegetations were again noted on the aortic valve cusps.

A repeat blood culture in the third week showed no bacterial growth. The patient died on the 29th day of admission.

Case 4 – Mr. O.A., a 22-year-old male university graduand with a prosthetic mitral valve (mechanical) replacement and left atrial appendage ligation following primary mitral valve regurgitation due to rheumatic valvular disease (performed one year prior at a private health facility), presented with intermittent fever, chills, and rigors of over two weeks' duration.

He had been self-medicating with tablets ciprofloxacin 500 mg twice daily and amoxicillin 500 mg three times daily (obtained over the counter), without clinical improvement. He had been on oral warfarin (5 mg/2.5 mg on alternate days) since valve replacement. On admission, he was intermittently febrile (37.8–38.2°C). Malaria rapid test (histidine-rich protein-2) was negative.

Transthoracic echocardiography revealed normal LV systolic function (Simpson's biplane EF of 58.8%), a functioning On-X bileaflet mitral mechanical valve in situ, with no vegetations or significant paravalvular leaks.

Haematological investigations showed: total white cell count – 8,100 cells/ μ L, neutrophils – 63.8%, haemoglobin – 11.2 g/dL, ESR – 150 mm/hr, C-reactive protein (CRP) – 89.7 mg/L (reference: 1–10 mg/L). Blood cultures (three sets) yielded MRSA, susceptible to aminoglycosides, tigecycline, clindamycin, azithromycin, ciprofloxacin, rifampicin, and linezolid.

He was commenced on oral rifampicin 600 mg daily for six weeks, IV vancomycin 500 mg every 8 hours, and IV amikacin 250 mg every 12 hours for 72 hours. However, he developed an idiopathic skin reaction to vanco-

mycin and was switched to oral linezolid 600 mg twice daily for six weeks. IV amikacin was discontinued after one week, as the patient was clinically stable for outpatient care.

A follow-up sepsis biomarker evaluation after two weeks showed improvement (CRP: 76.5 mg/L), and repeat blood cultures were negative.

4. Methods

HAIE patients were defined as those with bloodstream infections following central line cannulation for haemodialysis in the setting of ESKD, and who developed a cardiac murmur upon cardiovascular evaluation. The assessment of infective endocarditis was based on the modified Duke criteria (Fowler et al., 2023; Li et al., 2000), requiring echocardiographic evidence of vegetation and positive blood cultures.

HA-PVE was diagnosed based on the time frame of presentation (within one year of valve replacement for early-onset cases), along with symptoms and signs consistent with infective endocarditis. This assessment was also guided by the modified Duke criteria (Fowler et al., 2023; Katch et al., 2017; Li et al., 2000).

Treatment was tailored based on the antibiogram of the isolated organism from blood cultures, in accordance with established infective endocarditis treatment guidelines (Baddour et al., 2015).

5. Discussion

Healthcare-associated infective endocarditis (HAIE) is an emerging form of IE that arises from healthcare-related procedures. It often follows a complicated clinical course due to its non-specific presentation. Patients with end-stage kidney disease (ESKD) are particularly prone to IE due to the increased incidence of degenerative heart valve disease, immune system impairment, malnutrition, and associated comorbidities (Tarng & Huang, 1998). Similarly, cardiac valve replacement—especially mechanical prostheses—predisposes patients to IE, primarily through direct microbial contamination of the prosthetic valve (Calderwood et al., 1985).

In the cases reported here, *Staphylococcus* species—namely *Staphylococcus aureus* and coagulase-negative *Staphylococcus*—were the causative pathogens. All were methicillin-resistant, which significantly complicated treatment. The isolation of these organisms was not unexpected, as they are globally predominant in HAIE (Cunha et al., 1996; Holland et al., 2016; Miguel-Lopez-Dupla & Sebastian Hernandez, 2006; Tleyjeh & Abdulhak, 2018), particularly in culture-positive cases. This contrasts with culture-negative IE, which presents a therapeutic dilemma (McHugh & Saleh, 2023). In Africa, *Staphylococcus* and *Streptococcus* species are the most common pathogens in culture-positive IE (Djibril Marie et al., 2017; Noubiap et al., 2022), though recent trends show increasing detection of MRSA and coagulase-negative staphylococci (Santos et al., 2024). Notably, the incidence of MRSA remains relatively low in temperate regions (Halavaara et al., 2023). Its higher occurrence in tropical settings may be due to inadequate infection prevention and control (IPC) measures, which facilitate the emergence of antimicrobial-resistant organisms such as MRSA in hospital environments (O'Neill, 2016; Varma et al., 2018).

Although genetic typing was not performed, the phenotypic similarities of the methicillin-resistant *Staphylococcus* species suggest possible relatedness, especially given the clustering of cases within a short time frame. Serial blood cultures taken from both peripheral veins and central lines confirmed the presence of these pathogens as the causative agents in CLABSI-associated IE. The mortality observed occurred within 6–8 weeks of diagnosing HAIE, likely due to the virulence and resistance profile of the pathogens, as well as the presence of significant comorbidities.

Central line-associated bloodstream infection was the identified source of HAIE, as none of the patients had a prior history of cardiovascular infection on previous cardiac evaluations. Central line insertions are a major source of HAIE globally, among other healthcare procedures (Ruduan et al., 2024; Tarng & Huang, 1998). In our series, the time between internal jugular catheter (IJC) placement and IE onset ranged from 3 to 24 weeks. Apart from the direct anatomical proximity of the IJC to the heart, leading to bacteraemia and subsequent vegetation, the possibility of pre-existing non-bacterial thrombotic endocarditis (NBTE) due to uraemia in ESKD may have predisposed these patients to vegetation (Asopa et al., 2007).

The IJC was inserted on the right side, which drains into the right heart. Therefore, right-sided vegetations were expected (Kale & Raghavan, 2013). However, in these cases, vegetations were found across multiple valves, particularly on the aortic valve, with accompanying regurgitation. This may be attributed to the virulence of the implicated *Staphylococcus* species (Petti & Fowler, 2002), differing from findings in Senegal, where IE predominantly resulted in mitral regurgitation (Djibril Marie et al., 2017)—a pattern consistent with native valve IE without central line use.

The most common presenting symptom among HAIE patients was fever. Fever, cardiac murmurs, and neurological septic emboli are hallmark features of IE (Djibril Marie et al., 2017; Mkoiko et al., 2022). Musculoskeletal symptoms, such as hip pain, were also observed in one patient. While bone pain is an unspecific symptom, it is not uncommon in IE (Churchill et al., 1977). In this case, renal osteodystrophy was considered a differential diagnosis, given the underlying chronic kidney disease, but imaging and electrolyte assessments excluded it. Relative neutrophilia and elevated ESR were found in all HAIE patients—ESR being a non-specific marker of

inflammation (Gouriet et al., 2006; Hogevis et al., 1997). C-reactive protein (CRP), which is more specific, was only available for the patient with HA-PVE and was used to monitor treatment response.

The HA-PVE case in this series followed mitral valve replacement for rheumatic heart disease (RHD), which remains the leading cause of valve replacement in adults (Levin et al., 2014) and the most frequent cause of IE in African adults (Noubiap et al., 2022). HA-PVE is often caused by *Staphylococcus aureus* or coagulase-negative staphylococci, as seen in our patient (Kouijzer et al., 2022). Although no vegetations were detected on transthoracic echocardiography, serial blood cultures confirmed bloodstream infection—a known precursor to prosthetic valve vegetation. While vegetation-negative IE is rare, the modified Duke criteria allow for a “possible IE” diagnosis in the presence of positive blood cultures (Katch et al., 2017). A transoesophageal echocardiogram (TOE), which offers better sensitivity, was unavailable and could have revealed vegetations missed by transthoracic echocardiography (Daniel et al., 1993).

The use of glycopeptides such as vancomycin was appropriate, given the isolation of methicillin-resistant *Staphylococcus* species. Despite appropriate treatment, the HAIE patients in this series died. The recorded mortality rate was higher than the global average for IE (Cahill & Prendergast, 2016; Noubiap et al., 2022). Contributing factors likely included co-existing comorbidities, healthcare-associated infections, and the antimicrobial resistance profile of the pathogens (O’Neill, 2016; Varma et al., 2018). In contrast, the patient with HA-PVE improved significantly, likely due to early presentation and timely initiation of therapy. Early diagnosis is crucial in reducing morbidity and mortality from IE (Noubiap et al., 2022).

Preventive strategies in our setting should include implementation of a CLABSI prevention bundle (Gupta et al., 2021; Maharaj & Parrish, 2012) as part of a broader IPC framework (O’Neill, 2016), especially in patients with ESKD. Maintaining good oral hygiene and using prophylactic antimicrobials judiciously during procedures such as dental extractions can also reduce the risk of IE (Maharaj & Parrish, 2012). Since RHD is the most common cause of valve replacement in adults (Maharaj & Parrish, 2012; Mkoko et al., 2022), its prevention through early diagnosis and treatment of rheumatic fever is essential in reducing the risk of prosthetic valve endocarditis (PVE). Lastly, robust IPC practices are essential in preventing HA-PVE, particularly in early-onset cases.

6. Limitations

The interventions described in this case series cannot be generalized to the broader population, as individualization of treatment is essential. Furthermore, the mortality reported may not be solely attributable to cardiac complications, as the patients presented with significant comorbidities.

7. Conclusions

This case series highlights the potential for infective endocarditis to develop following central line insertion in our setting. Patients with central lines should undergo regular cardiovascular evaluation and screening with complete blood counts, ESR, and sepsis biomarkers to identify early warning signs. This report aims to raise awareness, encourage preventive measures, and promote early management of IE, especially in patients undergoing central line cannulation.

Additionally, in ESKD patients, prompt management of uraemia is vital to prevent NBTE, which predisposes to cardiac vegetation. Fever in patients with prosthetic heart valves should be investigated thoroughly, and any bloodstream infection should be promptly treated to prevent the progression to overt infective endocarditis.

Acknowledgement statement: The authors wish to thank God for the insights into good clinical management. We appreciate the renal, medical microbiology, and cardiology units of ABUAD MSH for their intellectual input.

Conflicts of interest: The authors declare that they have no financial or personal relationships which may have inappropriately influenced them in the development of this article.

Author contribution statements: E.O.I. and B.O.O. developed the concept of the manuscript. All the authors gathered the information from the database and reviewed the manuscript. All authors are in consonance with the final draft of the manuscript.

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data Availability Statement: Please contact the corresponding author for any additional information on data used in this article.

Disclaimer: The views and opinions expressed in this article are those of the authors and do not reflect the journal editor’s official policy or position.

References

- Asopa, S., Patel, A., Khan, O. A., Sharma, R., & Ohri, S. K. (2007). Non-bacterial thrombotic endocarditis. *European Journal of Cardio-Thoracic Surgery*, 32(5), 696–701. <https://doi.org/10.1016/j.ejcts.2007.07.029>

- Baddour, L. M., Wilson, W. R., Bayer, A. S., Fowler, V. G., Tleyjeh, I. M., Rybak, M. J., Barsic, B., Lockhart, P. B., Gewitz, M. H., Levison, M. E., Bolger, A. F., Steckelberg, J. M., Baltimore, R. S., Fink, A. M., Gara, P. O., Taubert, K. A., & Heart, A. (2015). *Infective Endocarditis in Adults: Diagnosis, Antimicrobial Therapy, and Management of Complications*. Infectious Diseases Society of America Larry. <https://doi.org/10.1161/CIR.0000000000000296>
- Cahill, T., & Prendergast, B. (2016). Infective endocarditis. *Lancet*, 387, 882–93. Calderwood, S. B., Swinski, L. A., Waternaux, C. M., Karchmer, A. W., & Buckley, M. J. (1985). Risk factors for the development of prosthetic valve endocarditis. *Circulation*, 72(1), 31–37. <https://doi.org/10.1161/01.CIR.72.1.31>
- Churchill, M. A., Geraci, J. E., & Hunder, G. G. (1977). Musculoskeletal manifestations of bacterial endocarditis. *Annals of Internal Medicine*, 87(6), 754–759. <https://doi.org/10.7326/0003-4819-87-6-754>
- Cunha, B. A., Gill, M. V., & Lazar, J. M. (1996). ACUTE INFECTIVE ENDOCARDITIS: Diagnostic and Therapeutic Approach. *Infectious Disease Clinics of North America*, 10(4), 811–834. [https://doi.org/10.1016/S0891-5520\(05\)70328-7](https://doi.org/10.1016/S0891-5520(05)70328-7)
- Daniel, W. G., Mügge, A., Grote, J., Hausmann, D., Nikutta, P., Laas, J., Lichtlen, P. R., & Martin, R. P. (1993). 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*, 71(2), 210–215. [https://doi.org/10.1016/0002-9149\(93\)90740-4](https://doi.org/10.1016/0002-9149(93)90740-4)
- Djibril Marie, B., Mboup, M. C., Zeba, N., Dia, K., Fall, A. N., Fall, F., Fall, P. D., & Gning, S. B. (2017). Infective endocarditis in Principal Hospital of Dakar: a retrospective study of 42 cases over 10 years. *The Pan African Medical Journal*, 26, 40. <https://doi.org/10.11604/pamj.2017.26.40.10020>
- Fowler, V. G., Durack, D. T., Selton-Suty, C., Athan, E., Bayer, A. S., Chamis, A. L., Dahl, A., Dibernardo, L., Durante-Mangoni, E., Duval, X., Fortes, C. Q., Fosbol, E., Hannan, M. M., Hasse, B., Hoen, B., Karchmer, A. W., Mestres, C. A., Petti, C. A., Pizzi, M. N., ... Miro, J. M. (2023). The 2023 Duke-International Society for Cardiovascular Infectious Diseases Criteria for Infective Endocarditis: Updating the Modified Duke Criteria. *Clinical Infectious Diseases*, 77(4), 518–526. <https://doi.org/10.1093/cid/ciad271>
- Gouriet, F., Bothelo-Nevers, E., Coulibaly, B., Raoult, D., & Casalta, J. P. (2006). Evaluation of sedimentation rate, rheumatoid factor, C-reactive protein, and tumor necrosis factor for the diagnosis of infective endocarditis [1]. *Clinical and Vaccine Immunology*, 13(2), 301. <https://doi.org/10.1128/CVI.13.2.301.2006>
- Gupta, P., Thomas, M., Patel, A., George, R., Mathews, L., Alex, S., John, S., Simbulan, C., Garcia, M. L., Al-Balushi, S., & El Hassan, M. (2021). Bundle approach used to achieve zero central line-associated bloodstream infections in an adult coronary intensive care unit. *BMJ Open Quality*, 10(1). <https://doi.org/10.1136/bmjopen-2020-001200>
- Halavaara, M., Huotari, K., Anttila, V. J., & Järvinen, A. (2023). Healthcare-associated infective endocarditis: source of infection and burden of previous healthcare exposure. *Antimicrobial Stewardship and Healthcare Epidemiology*, 3(1), 1–6. <https://doi.org/10.1017/ash.2023.419>
- Hogevik, H., Olaison, L., Andersson, R., & Alestig, K. (1997). C-reactive protein is more sensitive than erythrocyte sedimentation rate for diagnosis of infective endocarditis. *Infection*, 25(2), 82–85. doi: 10.1007/BF02113580. PMID: 9108181. Holland, T. L., Baddour, L. M., Bayer, A. S., Hoen, B., Miro, J. M., & Fowler, V. G. (2016). Infective endocarditis. *Nature Reviews. Disease Primers*, 2, 16059. <https://doi.org/10.1038/NRDP.2016.59>
- Kale, S. B., & Raghavan, J. (2013). Tricuspid valve endocarditis following central venous cannulation: The increasing problem of catheter related infection. *Indian Journal of Anaesthesia*, 57(4), 390–393. <https://doi.org/10.4103/0019-5049.118564>
- Katch, T., Cooper, H., Yandrapalli, S., Khera, S., Fallon, J., Spielvogel, D., Aronow, W., & Panza, J. (2017). Prosthetic Aortic Valve Endocarditis Without Evidence of Vegetation - PubMed. *Journal of Heart Valve Disease*, 26(3), 365–367. <https://pubmed.ncbi.nlm.nih.gov/29092126/>
- Kouijzer, J. J. P., Noordermeer, D. J., van Leeuwen, W. J., Verkaik, N. J., & Lattwein, K. R. (2022). Native valve, prosthetic valve, and cardiac device-related infective endocarditis: A review and update on current innovative diagnostic and therapeutic strategies. *Frontiers in Cell and Developmental Biology*, 10, 995508. <https://doi.org/10.3389/FCELL.2022.995508>
- Levin, B. R., Sulong, M. A., Mohd Safari, S. N., Jaffar, N., Ramli, M. F., & Ali, R. M. (2014). Epidemiology, Clinical Profile and Cardiac Remodeling of Severe Rheumatic Heart Disease in Malaysia. *Journal of Cardiac Failure*, 20(8), S100. <https://doi.org/10.1016/J.CARDFAIL.2014.06.281>
- Li, J. S., Sexton, D. J., Mick, N., Nettles, R., Fowler, V. G., Ryan, T., Bashore, T., & Corey, G. R. (2000). Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America*, 30(4), 633–638. <https://doi.org/10.1086/313753>
- Maharaj, B., & Parrish, A. (2012). Prevention of infective endocarditis in developing countries. *Cardiovascular Journal of Africa*, 23(6), 303–305. <https://doi.org/10.5830/CVJA-2012-004>

- McHugh, J., & Saleh, O. A. (2023). Updates in Culture-Negative Endocarditis. *Pathogens*, 12(8), 1027. <https://doi.org/10.3390/PATHOGENS12081027>
- Miguel-Lopez-Dupla, & Sebastian Hernandez. (2006). Clinical Characteristics and Outcome of Infective Endocarditis in Individuals of the General Population Managed at a Teaching Hospital Without Cardiac Surgery Facilities. Study of 120 Cases. *Revista Espa de Cardiologia*, 59(11), 1131–1139.
- Mkoko, P., Cupido, B. J., Hitzeroth, J., Chin, A., & Ntsekhe, M. (2022). Profile, presentation and outcomes of prosthetic valve endocarditis in a South African tertiary hospital: Insights from the Groote Schuur Hospital Infective Endocarditis Registry. *South African Medical Journal*, 112(4), 288–294. <https://doi.org/10.7196/SAMJ.2022.v112i4.16146>
- Noubiap, J. J., Nkeck, J. R., Kwondom, B. S., & Nyaga, U. F. (2022). Epidemiology of infective endocarditis in Africa: a systematic review and meta-analysis. *The Lancet Global Health*, 10(1), e77–e86. [https://doi.org/10.1016/S2214-109X\(21\)00400-9](https://doi.org/10.1016/S2214-109X(21)00400-9)
- O'Neill, J. (2016). *Tackling drug-resistant infections globally: final report and recommendations*. The review on antimicrobial resistance (p. 84). Wellcometrust. <https://doi.org/https://doi.org/10.1016/j.jpha.2015.11.005>
- Pecoraro, A. J., & Doubell, A. F. (2020). Infective endocarditis in South Africa. *Cardiovascular Diagnosis and Therapy*, 10(2), 252–261. <https://doi.org/10.21037/CDT.2019.06.03>
- Petti, C. A., & Fowler, V. G. (2002). Staphylococcus aureus bacteremia and endocarditis. *Infectious Disease Clinics of North America*, 16(2), 413–435. [https://doi.org/10.1016/S0891-5520\(01\)00003-4](https://doi.org/10.1016/S0891-5520(01)00003-4)
- Ruduan, M. G. M., Aisyah, H. S., Haniff, W. I. W. Y., & Zurkurnai, Y. (2024). Left sided infective endocarditis in end stage kidney disease patient on right temporary internal jugular catheter. *International Journal of Cardiology*, 397(Supplement, 2024), 131738. <https://doi.org/10.1016/j.ijcard.2024.131738>
- Santos, D. A. M., Siciliano, R. F., Besen, B. A. M. P., Strabelli, T. M. V., Sambo, C. T., Milczwski, V. de M., Goldemberg, F., Tarasoutchi, F., Vieira, M. L. C., Paixão, M. R., Gualandro, D. M., Accorsi, T. A. D., Pomerantzeff, P. M. A., & Mansur, A. J. (2024). Changing trends in clinical characteristics and in-hospital mortality of patients with infective endocarditis over four decades. *Journal of Infection and Public Health*, 17(4), 712–718. <https://doi.org/10.1016/j.jiph.2024.02.017>
- Tarng, D. C., & Huang, T. P. (1998). Internal jugular vein haemodialysis catheter-induced right atrium endocarditis - Case report and review of the literature. *Scandinavian Journal of Urology and Nephrology*, 32(6), 411–414. <https://doi.org/10.1080/003655998750015214>
- Tleyjeh, I. M., & Abdulhak, A. A. Bin. (2018). Epidemiology and global burden of infective endocarditis. In A. J. Camm, T. F. Lüscher, G. Maurer, & P. W. Serruys (Eds.), *The ESC Textbook of Cardiovascular Medicine* (p. 0). Oxford University Press. <https://doi.org/10.1093/med/9780198784906.003.0067>
- Varma, J. K., Oppong-Otoo, J., Ondo, P., Perovic, O., Park, B. J., Laxminarayan, R., Peeling, R. W., Schultz, C., Li, H., Ihekweazu, C., Sall, A. A., Jaw, B., & Nkengasong, J. N. (2018). Africa Centres for Disease Control and Prevention's framework for antimicrobial resistance control in Africa. *African Journal of Laboratory Medicine*, 7(2). <https://doi.org/10.4102/ajlm.v7i2.830>

About the Author (s)



Dr. Emmanuel Oladayo Irek is a Consultant Medical Microbiologist in a Private tertiary hospital in Nigeria. He is an infection prevention expert and well tutored in antimicrobial resistance with extensive experience in laboratory management. His speciality includes healthcare-associated infections and antibiotic stewardship.



Dr. Adesina O.B. is currently serving as a medical officer at Afe Babalola Multi-System Hospital, a private tertiary health center in Nigeria. She oversees the procedural coordination for prospective renal transplant recipients and donors. She has a future professional interest in pediatrics.



Dr. Busayo O. Oguntola is a Consultant Cardiologist and a researcher at a private University and a Tertiary Hospital in Nigeria. She has special interests in the prevention of cardiovascular diseases such as valvular heart diseases, coronary heart disease, heart failure, and infective heart diseases.

Dr. Fasaanu A.N. is a Consultant Nephrologist in a Private tertiary hospital in Nigeria. He oversees renal transplantation in the facility.