ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 11 | November - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

THE SILENT SIEGE: A CASE REPORT ON NATIVE VALVE MRSA ENDOCARDITIS AND THE CASCADE OF EMBOLIC STORMS

Internal Medicine

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ABSTRACT

Infective endocarditis (IE) is a serious infection impacting the heart's valves and inner lining. Despite advances in diagnostic modalities, antimicrobial therapies, and surgical treatments, IE remains a worldwide health challenge. Key risk factors include congenital heart anomalies and rheumatic heart disease, which is more common in regions like India. Mortality rates for native and prosthetic valve IE are comparable early on, but infections caused by gram-negative bacteria and fungi tend to have worse outcomes. Clinical diagnosis integrates symptoms, microbiology, and imaging methods per the modified Duke criteria. A middle aged woman with no known comorbidities prolonged episode of intermittent fever and progressive fatigue, complicated by exertional dyspnea. Examination revealed pallor, pedal edema, a new murmur, and papilledema. Initial investigations showed anemia, leukocytosis, and systemic inflammation, with negative tropical infection workup. Blood cultures via multiplex PCR grew methicillin-resistant Staphylococcus aureus. Echocardiography confirmed a large vegetation on the aortic valve. CT imaging revealed splenic infarcts, hepatosplenomegaly, pericardial effusion, and thrombus in the superior mesenteric artery. Bone marrow aspiration showed hemophagocytic lymphohistiocytosis, and tropical infections were ruled out. Antimicrobial therapy was tailored to culture sensitivity, with anticoagulation started after embolic complications. This case highlights the aggressive course and systemic embolic complications characteristic of native valve MRSA infective endocarditis. It emphasizes the necessity for prompt diagnosis, comprehensive monitoring, coordinated multidisciplinary care, and timely surgical intervention to optimize patient outcomes.

KEYWORDS

Infective endocarditis, MRSA (Methicillin-resistant Staphylococcus aureus), Aortic valve vegetation, Hemophagocytic lymphohistiocytosis

INTRODUCTION

Infective endocarditis (IE) is a disease characterized by infection of the heart's endocardial lining, most commonly affecting the heart valves but occasionally involving other inner heart structures. Congenital heart anomalies such as ventricular septal defects, bicuspid aortic valves, patent ductus arteriosus, and coarctation of the aorta elevate the risk for developing IE. The global occurrence of IE is estimated to be between 3 and 10 cases per 100,000 individuals annually [1] –[4]. Despite improvements in diagnostic methods, availability of new antibiotics, and advances in surgical procedures, the burden of IE remains substantial worldwide [5]. In India, rheumatic heart disease predominates as an underlying etiology, whereas in developed countries, congenital heart disorders are more frequently implicated [6] – [8]. Indian studies reveal that nearly 76% of IE cases manifest in patients younger than 40 years, with a mean age of 27.6 years [7]. Though less common in children, IE incidence is rising in the pediatric population. IE classification depends on the infection location, presence of cardiac devices or prosthetic valves, and potential modes of acquisition, including community transmission, hospitalization, or intravenous drug use. Patients with prosthetic heart valves or other implanted cardiac materials face a significantly elevated risk. Prosthetic valve endocarditis (PVE) is stratified as early if occurring within one year of valve replacement. Staphylococcus species, followed by Streptococcus, are the most common causative organisms for PVE.

Infective endocarditis (IE) is associated with a significant mortality risk. Around 20% of patients die during their hospitalization, and mortality rates can rise to between 25% and 30% within six months following the infection [9] – [11]. The early mortality rates are similar for both native valve endocarditis (NVE) and prosthetic valve endocarditis (PVE), as well as for infections involving either the mitral or aortic valves. However, individuals affected by IE due to gram-negative bacteria or fungal pathogens experience the highest mortality, with rates approaching 50% [10], [12] - [14].

Diagnosing IE relies on a combination of clinical evaluation, microbiological testing, and imaging studies. The clinical presentation is frequently nonspecific, often including symptoms like fever, chills, fatigue, and weight loss. Fever remains the most prevalent symptom, observed in approximately 95% to 100% of cases, regardless of whether the infection involves native or prosthetic valves. Confirmation of IE diagnosis is based on the modified Duke criteria, which require the presence of either two major criteria, one major plus three minor criteria, or five minor clinical criteria. Modified Duke criteria for diagnosing infective endocarditis consist of Major Criteria that include Two positive blood cultures with typical microorganisms collected at least 12 h apart (or one positive culture for Coxiella burnetii) and Evidence of endocardial involvement (new murmur, echocardiographic evidence of cardiac mass, abscess, valve dehiscence). Minor Criteria comprise of Fever > 38°C, Vascular phenomenon (systemic emboli, Janeway lesions), Immunologic phenomenon (Osler nodes, Roth spots), Predisposition to infective endocarditis (previous infective endocarditis or intravenous drug abuse) and Microbiologic evidence that does not meet major criteria [15].

Keywords

Anticoagulation, Antimicrobial therapy, Aortic regurgitation, Aortic valve, Biofire, Blood cultures, Bone marrow aspiration, C-reactive protein, Embolic events, Erythrocyte sedimentation rate, Heart failure, Hemophagocytic lymphohistiocytosis, Methicillin-resistant Staphylococcus aureus, Modified Duke criteria, Multidisciplinary management, Multiplex PCR, Packed red blood cells, Papilledema, Pericardial effusion, Sinus tachycardia, Splenic infarct, Staphylococcus aureus, Superior mesenteric artery, Thrombus, Transthoracic echocardiography, Vegetation.

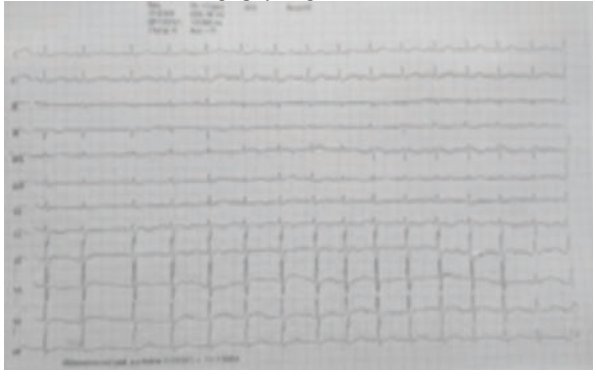

Figure 1: ECG showing tachycardia

Case Presentation

Presentation:

A 43-year-old woman, gravida 3, para 2, abortion 1, with menopause at 41 years of age and no prior history of significant comorbidities, presented to the hospital with complaints of intermittent fever persisting for forty days and progressively worsening easy fatigability over the same duration. Ten days prior to admission, she developed a non-productive cough and exertional dyspnea, escalating from New York Heart Association (NYHA) class I to class III within the brief period. Her medical and surgical history was unremarkable: there was no record or suggestion of previous cardiac surgery, valvular heart disease, similar episodes, dental or invasive procedures, nor a prior diagnosis of infective endocarditis. She also denied skin rashes, joint pain, chest pain, palpitations, bleeding, abdominal symptoms, altered sensorium or abnormal movements. There was no significant personal or family medical history, and for her initial symptoms she had only sought over-the-counter medications at a local clinic without formal medical evaluation.

Clinical Examination:

Upon arrival, the patient was alert but appeared clinically ill. She was found to be pale, with bilateral pitting pedal edema, and her vital signs showed blood pressure of 180/80 mm Hg, a regular high-volume pulse rate of 126 beats per minute, and a respiratory rate of 20 breaths per minute with oxygen saturation of 96% on room air. Jugular venous pressure was not raised, all peripheral pulses were palpable and symmetrical, and there were no classical peripheral stigmata of infective endocarditis observed. Cardiovascular examination revealed normal first and second heart sounds (S1, S2) and a grade I end-diastolic murmur; chest auscultation revealed bilateral wheezing but otherwise preserved vesicular breath sounds and symmetrical chest movements. Abdominal exam indicated a soft, non-tender liver palpable 1cm below the right costal margin, with audible bowel sounds and no further organomegaly or tenderness. Neurological examination showed the patient to be fully conscious (GCS E4V5M6) with normal higher mental functions, symmetrical Pupils reacting to light, and no signs of focal neurological deficit. Fundoscopic evaluation identified grade 1 papilledema bilaterally.

Investigations:

Initial investigations were remarkable for marked anemia (hemoglobin 6.3 g/dl), leukocytosis (white blood cell count: 16,000/cumm with 90% neutrophils), and evidence of systemic inflammation with elevated C-reactive protein (12 mg/dl) and erythrocyte sedimentation rate (40 mm in the first hour). Each of the common tropical infections endemic to her region was ruled out, and both chest radiography and abdominal ultrasonography were essentially normal. An ECG showed sinus tachycardia. (Figure 1) Bone marrow aspirate performed revealed a reactive, cellular marrow with two to three loci of hemophagocytic lymphohistiocytosis. Other relevant laboratory tests, including tuberculosis screening, returned negative.

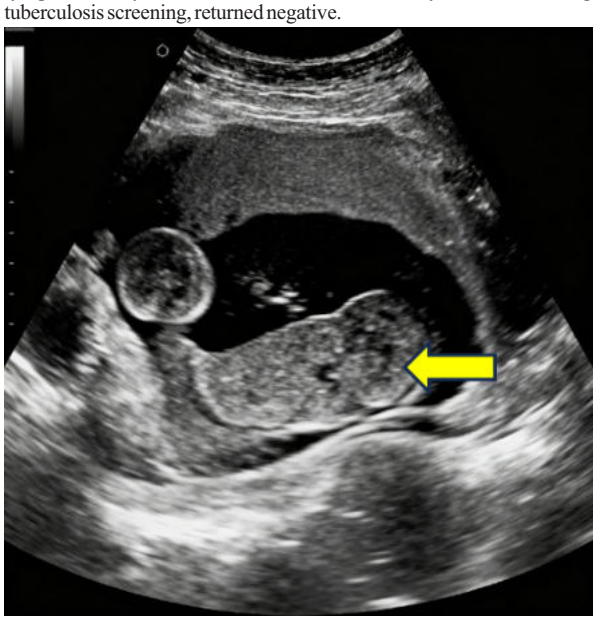

Figure 2: 2D Echo showing Large Vegetation 22x15mm over Aortic Valve

Diagnosis:

The clinical picture raised suspicion for infective endocarditis, especially given the presence of new-onset murmur and systemic signs. Blood cultures drawn upon admission, processed via Biofire (multiplex PCR), grew methicillin-resistant Staphylococcus aureus (MRSA), confirming the diagnosis microbiologically. Transthoracic echocardiography demonstrated a large vegetation measuring 22 x 15 mm attached to the aortic valve (Figure 2), and the diagnosis of native valve infective endocarditis was supported by modified Duke's criteria, with both major criteria being met. No other valves were involved on imaging. No diagnostic challenge was faced, for example, cultural or financial.

Complications:

Shortly into her hospital stay, the patient's clinical course was complicated by recurrent episodes of left ventricular failure, manifesting as acute shortness of breath and signs of decompensated heart failure, necessitating conservative management with diuretics and supportive care. Antimicrobial therapy was commenced with intravenous ceftriaxone and vancomycin, tailored to culture and sensitivity reports, along with transfusions of packed red blood cells for anemia.

Despite appropriate therapy, the patient developed sudden onset of pain in the left hypochondrium. An urgent bedside echocardiogram at this time did not reveal any new vegetations on the aortic valve, but further radiological assessment with contrast-enhanced CT (CECT) of the chest, abdomen, and pelvis demonstrated hepatosplenomegaly, multiple wedge-shaped hypodensities (representing splenic infarcts), and moderate pericardial effusion (16 mm in diameter). Notably, a round, enhancing lesion was seen lateral to the right psoas in the retroperitoneum, and “tree-in-bud” pattern nodules were detected in the lower lobe of the left lung, with lymphadenopathy.

She subsequently experienced acute abdominal pain and worsening clinical status. CT angiography revealed an acute thrombus in the superior mesenteric artery and multiple further splenic infarcts. An axial section of CT Abdomen shows thrombus in superior mesenteric artery. (Figure 3)

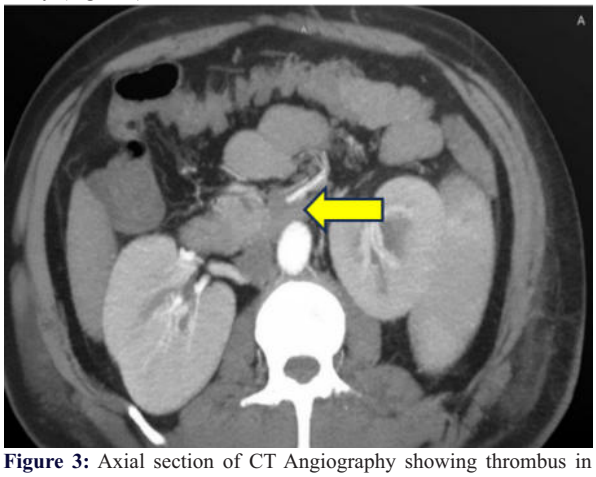

Figure 3: Axial section of CT Angiography showing thrombus in Superior Mesenteric Artery (Contrast taken up by Aorta but Superior Mesenteric Artery which lies just anterior to the aorta should have also shown the contrast, but is not seen due to presence of thrombus)

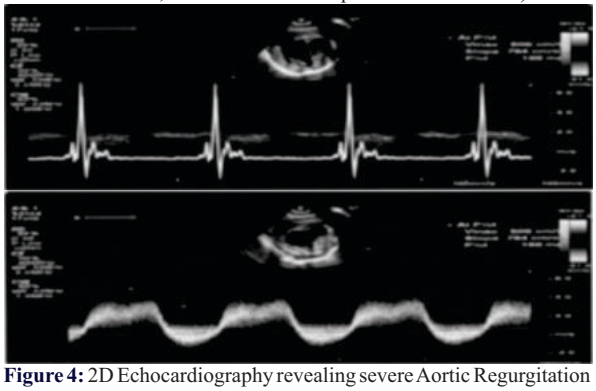

Figure 4: 2D Echocardiography revealing severe Aortic Regurgitation

After multidisciplinary consultation, anticoagulation therapy was initiated alongside the ongoing antibiotics. The course of her condition was further complicated by the development of moderate-to-severe aortic regurgitation, as detected clinically by a new mid-diastolic murmur and quantitatively confirmed by echocardiography (Figure 4); this ultimately necessitated surgical aortic valve replacement. Post surgical repair, patient recovered over a period of 02-03 months, with gradual return into her routine life, expressing satisfaction to treatment provided to her over her entire hospital stay.

DISCUSSION

Infective endocarditis (IE) is a serious condition that leads to significant morbidity and mortality, especially in patients with pre-existing cardiovascular disease or those who have undergone cardiac surgery. Key factors that contribute to an increased risk of death include the onset of heart failure, the presence of intracardiac abscesses, embolic complications, large mobile vegetations on heart valves, hemodynamic instability, changes in mental status, immunosuppressed states, and advanced age [11] – [14]. Two major challenges that clinicians face when managing IE are accurately identifying the responsible pathogen and deciding the optimal timing for surgical intervention. Laboratory diagnosis heavily depends on inflammatory markers and blood culture results, while echocardiography is indispensable for visualizing vegetations and abscesses, assessing the severity of valve regurgitation, monitoring heart failure progression, and evaluating overall cardiac function over time.

Predisposing factors contributing to embolism have been widely described in published studies. The EURO-ENDO investigation identified several associated risks, including advanced age, prior episodes of pulmonary embolism, history of heart failure, use of statins, prior treatment with Vitamin K antagonists (VKA), infective endocarditis (IE) involving the aortic or tricuspid valves, device-related IE, pulmonary endocarditis, endocarditis linked to pacemakers or intracardiac defibrillators, the presence and size of vegetations, positive blood cultures, and infection with Staphylococcus aureus. [16].

Mesenteric ischemia, although uncommon, represents a critical surgical emergency with high morbidity and mortality if not managed urgently and appropriately. Arterial embolism accounts for 40 to 50% of acute mesenteric ischemia cases, with the heart being the primary source of emboli affecting the mesenteric arteries. Literature documenting mesenteric ischemia secondary to infective endocarditis is sparse, with only limited case reports available. Additionally, the use of anticoagulation therapy in cases of septic emboli leading to mesenteric ischemia is not well-studied and requires further investigation. The bowel accounts for a substantial proportion of metabolic activity, receiving 20–25% of cardiac output. At rest, the small intestine alone holds around one-third of the body's total blood volume, positioning it as the circulatory system's principal vascular reservoir. Notably, the small intestine is highly susceptible to ischemia, as 70–85% of its blood flow is directed to the mucosa, with the remaining supply allocated to the serosal and muscularis layers. [17,18]. The mucosa, due to its high metabolic needs, is particularly vulnerable to ischemic damage, with injury becoming apparent after only 20–60 minutes of reduced perfusion. If blood flow is not restored, this can progress to transmural bowel infarction within 8–12 hours. The degree and potential reversibility of intestinal injury depend on factors such as the site and extent of vascular occlusion, the number of affected vessels, the presence of collateral circulation, and the duration of ischemia. While collateral vessels may offer protection for up to 12 hours even when 75% of normal bowel perfusion is diminished, severe injury can occur after just 4–6 hours in cases of total perfusion loss [19].

Ischemia induces a robust inflammatory response characterized by increased vascular permeability and structural compromise of the intestinal wall. Grossly, the ischemic bowel demonstrates a dusky discoloration with dark-red to brown hues, often accompanied by hemorrhage and thickening with edema involving the submucosal and muscularis propria layers in advanced injury. A hallmark feature is the clear demarcation between infarcted and viable tissue. Histopathologically, early ischemic injury exhibits mucin depletion, hemorrhagic mucosal necrosis, congestion and hyalinization of the lamina propria, and crypt atrophy. Extension into the submucosa and muscularis propria indicates severe ischemia and manifests as edema and hemorrhage. Transmural infarction predisposes to perforation and secondary peritonitis, complicating the clinical course. Acute neutrophilic infiltration is typically limited but becomes apparent upon reperfusion, mediating further tissue injury via oxidative stress mechanisms. This ischemia-reperfusion injury exacerbates mucosal and cellular damage through neutrophil-driven inflammation and reactive oxygen species generation, ultimately intensifying cell death and impairing tissue recovery. These pathological insights underscore the critical temporal window for intervention to mitigate irreversible bowel damage and systemic complications [20] , [21].

The treatment of infective endocarditis (IE) necessitates a coordinated effort among a team of specialists, including cardiologists, cardiothoracic surgeons, intensivists, and infectious disease experts. Empirical antibiotic treatment should begin promptly after blood cultures are drawn; however, if the patient's clinical condition is stable, it is acceptable to wait for culture results before initiating therapy. The majority of patients respond favorably to antibiotics alone, but healthcare providers often face the difficult decision of whether valve replacement surgery is indicated.

This case illustrates the aggressive nature and rapid development of serious complications associated with native valve MRSA infective endocarditis, such as recurrent heart failure, widespread embolic events including splenic infarctions and mesenteric ischemia, as well as extensive damage to the heart valves necessitating surgical repair. The successful outcome was reliant on a multidisciplinary treatment strategy involving early detection of the responsible pathogen, administration of targeted antibiotics, close surveillance for disease complications by imaging modalities, and timely valve replacement surgery. It highlights the critical need for individualized treatment plans guided by established clinical protocols and risk assessment, especially when managing complex cases with high-risk pathogens and embolic risks. Seamless collaboration between cardiology, infectious disease, surgical, and intensive care experts remains essential to enhancing prognosis in these challenging patient scenarios.

CONCLUSION

This case demonstrates the aggressive progression and serious complications linked to native valve infective endocarditis caused by methicillin-resistant Staphylococcus aureus (MRSA). The presence of a large, highly embolic vegetation measuring 22×15 mm led to multiple systemic emboli, including splenic infarctions and thrombosis of the superior mesenteric artery. Initiation of focused antimicrobial treatment combined with anticoagulation therapy after thorough multidisciplinary consultation was appropriate given the embolic septic complications. However, sustained vegetation larger than 10 mm with ongoing embolic events, as observed here, strongly indicates the need for early surgical intervention. Such timely surgery potentially could have prevented the advancement to severe aortic valve regurgitation and recurrent heart failure. This highlights the critical importance of prompt surgical decision-making in managing high-risk infective endocarditis cases to improve clinical prognosis, consistent with recommended guidelines endorsing valve replacement when large, mobile vegetations and embolic risks are present.

Abbreviations:

1. IE: Infective Endocarditis
2. NVE: Native Valve Endocarditis
3. PVE: Prosthetic Valve Endocarditis
4. MRSA: Methicillin-Resistant Staphylococcus aureus
5. NYHA: New York Heart Association (classification)
6. PCR: Polymerase Chain Reaction
7. CT: Computed Tomography
8. CECT: Contrast-Enhanced Computed Tomography
9. ECG: Electrocardiogram
10. CRP: C-Reactive Protein
11. ESR: Erythrocyte Sedimentation Rate
12. GCS: Glasgow Coma Scale
13. CNS: Central Nervous System
14. EE: Embolic Events
15. TF: Tissue Factor
16. P/A: Per Abdomen (clinical examination)
17. PRBC: Packed Red Blood Cells
18. CVS: Cardiovascular System
19. HMF: Higher Mental Functions

DECLARATIONS:

1. Funding (information that explains whether and by whom the

research was supported)
No Funding received/ no financial support or sponsorship
2. Conflicts of interest/Competing interests (include appropriate disclosures)
Nil
3. Ethics approval (include appropriate approvals or waivers)
Not applicable
4. Consent to participate (include appropriate statements)
The authors confirm that they have secured patient consent form. The patient has provided her consent for the images and other clinical details to be published in the journal. She understands that her name and initials will not be disclosed, and efforts will be made to maintain her anonymity, although complete anonymity cannot be guaranteed.
5. Written Consent for publication (include appropriate statements)
Written informed consent obtained from patient, ensuring anonymity would be maintained.
6. Availability of data and material (data transparency)
The data supporting the findings of this case report are available within the article. Relevant clinical details, diagnostic investigation results, imaging findings, and management information have been presented comprehensively to support the conclusions, ensuring transparency and reproducibility.
7. Code availability (software application or custom code)
No custom software or code was used in the preparation or analysis of the clinical data in this case report.
8. Authors' contributions
Dr. Debanjan Saha led the conception, design, and execution of the case report, including data collection, clinical analysis, and manuscript drafting. Dr. Vikalp Jain assisted with data collection and contributed to the literature review and manuscript revision. Dr. Yegade Walmik Shrihari provided expert guidance, supervised the clinical interpretation, and critically reviewed the manuscript. All authors reviewed and approved the final version of the manuscript.

REFERENCES

1. Pant, S., Patel, N.J., Deshmukh, A., Golwala, H., Patel, N., Badheka, A., Hirsch, G.A. & Mehta, J.L. (2015) Trends in infective endocarditis incidence, microbiology, and valve replacement in the United States from 2000 to 2011. Journal of the American College of Cardiology, 65, 2070–2076. DOI: 10.1016/j.jacc.2015.03.518, PubMed: 25975469
2. Bor, D.H., Woolhandler, S., Nardin, R., Brusch, J. & Himmelstein, D.U. (2013) Infective endocarditis in the U.S., 1998–2009: A nationwide study. PLOS One, 8, e60033. DOI: 10.1371/journal.pone.0060033, PubMed: 23527296.
3. Federspiel, J.J., Stearns, S.C., Peppercorn, A.F., Chu, V.H. & Fowler, V.G., Jr (2012) Increasing US rates of endocarditis with Staphylococcus aureus: 1999–2008. Archives of Internal Medicine, 172, 363–365. DOI: 10.1001/archinternmed.2011.1027, PubMed: 22371926.
4. Dayer, M.J., Jones, S., Prendergast, B., Baddour, L.M., Lockhart, P.B. & Thornhill, M.H. (2015) Incidence of infective endocarditis in England, 2000–13: A secular trend, interrupted time-series analysis. Lancet, 385, 1219–1228. DOI: 10.1016/S0140-6736(14)62007-9, PubMed: 25467569.
5. Cabell, C.H., Jollis, J.G., Peterson, G.E., Corey, G.R., Anderson, D.J., Sexton, D.J., Woods, C.W., Reller, L.B., Ryan, T. & Fowler, V.G. (2002) Changing patient characteristics and the effect on mortality in endocarditis. Archives of Internal Medicine, 162, 90–94. DOI: 10.1001/archinte.162.1.90, PubMed: 11784225.
6. Math, R.S., Sharma, G., Kothari, S.S., Kalaivani, M., Saxena, A., Kumar, A.S. & Bahl, V.K. (2011) Prospective study of infective endocarditis from a developing country. *American Heart Journal*, 162, 633–638. DOI: 10.1016/j.ahj.2011.07.014, PubMed: 21982654..
7. Garg, N., Kandpal, B., Garg, N., Tewari, S., Kapoor, A., Goel, P. & Sinha, N. (2005) Characteristics of infective endocarditis in a developing country–clinical profile and outcome in 192 Indian patients, 1992–2001. International Journal of Cardiology, 98, 253–260. DOI: 10.1016/j.ijcard.2003.10.043, PubMed: 15686775.
8. Choudhury, R., Grover, A., Varma, J., Khattri, H.N., Anand, I.S., Bidwai, P.S., Wahi, P.L. & Sapru, R.P. (1992) Active infective endocarditis observed in an Indian hospital 1981–1991. American Journal of Cardiology, 70, 1453–1458. DOI: 10.1016/0002-9149(92)90299-e, PubMed: 1442618.
9. Leone, S., Ravasio, V., Durante-Mangoni, E., Crapis, M., Carosi, G., Scotton, P.G., Barzaghi, N., Falcone, M., Chinello, P., Pasticci, M.B., Grossi, P., Utili, R., Viale, P., Rizzi, M. & Suter, F. (2012) Epidemiology, characteristics, and outcome of infective endocarditis in Italy: The Italian Study on Endocarditis. Infection, 40, 527–535. DOI: 10.1007/s15010-012-0285-y, PubMed: 22711599.
10. Nadji, G., Rusinaru, D., Rémadi, J.P., Jeu, A., Sorel, C. & Tribouilloy, C. (2009) Heart failure in left-sided native valve infective endocarditis: Characteristics, prognosis, and results of surgical treatment. European Journal of Heart Failure, 11, 668–675. DOI: 10.1093/eurjhf/hfp077, PubMed: 19553397.
11. Olmos, C., Vilacosta, I., Fernández, C., López, J., Sarriá, C., Ferrera, C., Revilla, A., Silva, J., Vivas, D., González, I. & San Román, J.A. (2013) Contemporary epidemiology and prognosis of septic shock in infective endocarditis. European Heart Journal, 34, 1999–2006. DOI: 10.1093/eurheartj/ehs336, PubMed: 23060453.
12. Tattevin, P., Revest, M., Lefort, A., Michelet, C. & Lortholary, O. (2014) Fungal endocarditis: Current challenges. International Journal of Antimicrobial Agents, 44, 290–294. DOI: 10.1016/j.ijantimicag.2014.07.003, PubMed: 25178919.
13. Smego, R.A., Jr & Ahmad, H. (2011) The role of fluconazole in the treatment of Candida endocarditis: A meta-analysis. Medicine (Baltimore), 90, 237–249. DOI: 10.1097/MD.0b013e3182259d38, PubMed: 21694646.
14. Thuny, F., Beurtheret, S., Mancini, J., Gariboldi, V., Casalta, J.P., Riberi, A., Giorgi, R., Gouriet, F., Tafanelli, L., Avierinos, J.F., Renard, S., Collart, F., Raoult, D. & Habib, G. (2011) The timing of surgery influences mortality and morbidity in adults with severe complicated infective endocarditis: A propensity analysis. European Heart Journal, 32, 2027–2033. DOI: 10.1093/eurheartj/ehp089, PubMed: 19329497.
15. 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, 30, 633–638. DOI: 10.1086/313753, PubMed: 10770721.
16. Habib G, Erba PA, Iung B, Donal E, Cosyns B, Laroche C, et al. Clinical presentation, aetiology and outcome of infective endocarditis. Results of the ESC-EORP EURO-ENDO (European infective endocarditis) registry: a prospective cohort study. Eur Heart J. 2019;40:3222–32.
17. Theodore S, Xia T, Saillant N. Intestinal Ischemia: Etiology and Foundational Concepts. **NEJM Evid** 2024;3(3):EVIDra2300266.
18. Gore RM, Yaghmai V, Thakrar KH, et al. Imaging in Intestinal Ischemic Disorders. **Radiol Clin North Am** 2008;46(5):845–875, v.
19. Kanasaki S, Furukawa A, Fumoto K, et al. Acute Mesenteric Ischemia: Multidetector CT Findings and Endovascular Management.
20. Garzelli L, Ben Abdallah I, Nuzzo A, et al. Insights into acute mesenteric ischaemia: an up-to-date, evidence-based review from a mesenteric stroke centre unit. **Br J Radiol** 2023;96(1151):20230232.
21. Mallick IH, Yang W, Winslet MC, Seifalian AM. Ischemia-reperfusion injury of the intestine and protective strategies against injury. **Dig Dis Sci** 2004;49(9):1359–1377.