Cryptic Septic Endocarditis and Systemic Thromboembolism Triggered by Oral Microflora

Sriharsha Kanuri[1]*, Samprith Ala[1], Stephen Fletcher[1], Gagandeep Grewal[1]

[1]Merit Health Wesley Health Center, 5001 Hardy St, Hattiesburg, MS 39402, USA

*Corresponding author: Sriharsha Kanuri · harsha9009@gmail.com

Received: 14 September 2025 · Peer reviewed, accepted and published: 26 September 2025

Swiss Journal of Radiology and Nuclear Medicine · www.sjoranm.com · SJORANM GmbH · Rosenweg 3 · CH-6340 Baar · Switzerland

Abstract

Streptococcus anginosus is part of the commensal flora and can be associated with invasive infection. We describe an adult man with no recognized predisposing cardiac disease who presented with fever, nausea, vomiting, memory impairment and respiratory symptoms. Echocardiography demonstrated a vegetation on the native aortic valve with severe aortic regurgitation, and abdominal CT showed splenic embolic infarctions. Blood cultures were reported as positive, with S. anginosus considered the most likely organism. Treatment included antibiotics followed by prosthetic aortic valve replacement. This case illustrates the need to consider infective endocarditis despite nonspecific symptoms and the absence of recognized predisposing conditions.

Keywords: Streptococcus anginosus; Gram-positive cocci; sepsis; vegetations; thromboembolism; splenic infarction; aortic regurgitation

Introduction

Streptococcus anginosus is a gram-positive streptococcus that usually resides in the mucous membranes of the oral cavity, with eventual migration to the nasopharynx, gastrointestinal tract and genitourinary tract [1]. The risk of infection with S. anginosus amplifies in the presence of risk factors such as cancer, hematological malignancies, peritonitis, gingivitis, type II diabetes mellitus, chronic kidney disease, chronic respiratory disease, heart failure, dementia, viral hepatitis, peptic ulcer disease, smoking, alcohol, drugs, CNS disorders and myocardial infarction [1]. Previous reports suggest involvement in brain abscess, pleural empyema, pneumonia, pericarditis and endocarditis [1].

This case describes native aortic valve endocarditis with splenic embolic infarctions in an adult man with no recognized predisposing cardiac disease. The reported culture findings were considered most consistent with S. anginosus. The case illustrates that this presentation can occur even when no predisposing condition is recognized.

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Clinical Case

An adult man presented to the emergency department with fever, nausea and vomiting, fatigue, and difficulty forming new memories. His wife was at the bedside helping him provide the history. For the past 3 to 4 weeks he had an upper respiratory infection with a productive cough. Two weeks earlier he had started vomiting, with decreased appetite and vomiting every time he tried to eat. His wife stated that at approximately 8:30 that morning he had become confused, with repetitive speech and repeated questions.

He and his wife were at an oil-change mechanic shop. He spent much of his time outdoors in the heat and reported dehydration. One night that week he had awakened with severe left calf cramping. After fevers began the previous week, he attended an urgent care clinic and received a Z-Pak. He declined COVID testing. Influenza testing on 23 June 2025 was negative.

He and his wife had traveled to Indiana the previous month. They drove but took frequent breaks to assist her special-needs son, who had developed disabilities, hearing loss and quadriplegia after pneumococcal meningitis at 8 months of age. They had a cat, but the patient did not change the litter box. He had no contact with cattle, farm animals or rodents, and recalled no tick bites or recent mosquito bites. The mechanic shop was well ventilated. His home used city water rather than well water. He had no other sick contacts or contributory family history.

He denied a personal or family history of autoimmune disease, immunocompromised states, cardiac disease or clotting disorders. He was a nondrinker, nonsmoker and non-IV drug user and denied trauma. Chest radiography showed bilateral pleural effusions and bibasilar pulmonary disease (Figure 1). Abdominal radiography was unremarkable (Figure 2).

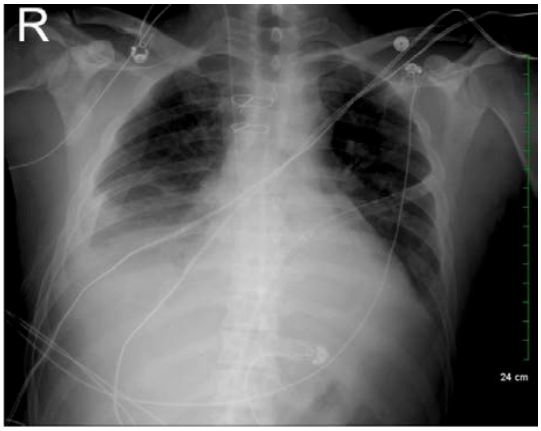


Figure 1. Chest radiograph: bilateral pleural effusions with bibasilar airspace disease. No pneumothorax. Stable heart size, right IJ catheter and left chest tube; sternal wires are present.

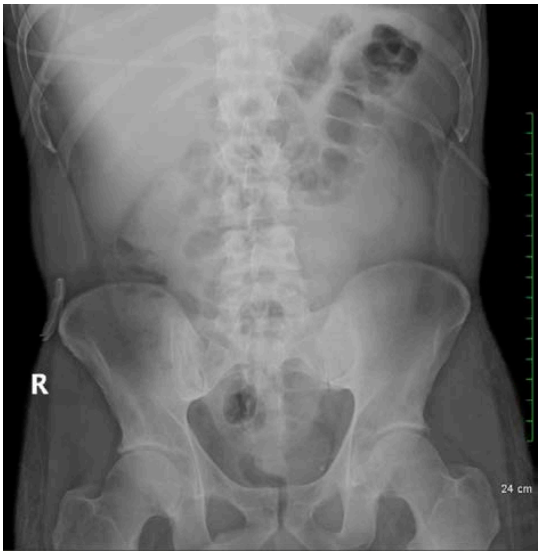


Figure 2. Abdominal radiograph: no acute abnormality.

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Imaging and Clinical Course

CT of the head and MRI of the brain were unremarkable (Figures 3 and 4). Liver ultrasonography reportedly showed mild diffuse coarse hepatic echotexture and a 2.2 cm nonvascular echogenic focus in the posterior right hepatic lobe, potentially a hemangioma. It likely corresponded to an approximately 2 cm partially low-density lesion with peripheral nodular enhancement on single-phase CT, limited in characterization but suggestive of hemangioma. The gallbladder was partially distended with borderline wall thickening, without sonographic findings of acute cholecystitis.

Carotid ultrasonography showed no significant abnormality or narrowing (Figure 6). Contrast-enhanced CT of the abdomen and pelvis showed multiple acute or subacute splenic infarcts with mild splenomegaly and a mild left pleural effusion (Figure 7). Echocardiography showed an estimated EF of 64%, mild left atrial dilatation, a structurally normal mitral valve, moderate mitral regurgitation, severely elevated pulmonary artery pressure of 90 mmHg and a dilated IVC. TEE showed severe aortic regurgitation with a 1 cm vegetation on the right coronary cusp of a tricuspid aortic valve, moderate mitral regurgitation and mild tricuspid regurgitation (Video 1). Echocardiographic Doppler images from the published image set are shown in Figure 5.

While culture results were pending, treatment was started with vancomycin, cefepime, ampicillin and gentamicin. Blood cultures were reported as positive, with Streptococcus anginosus considered the most likely organism. He was treated with intravenous penicillin G potassium. During follow-up he developed a migraine-like headache, and neurology was consulted. The subsequent course included aortic valve replacement with a prosthetic heart valve.

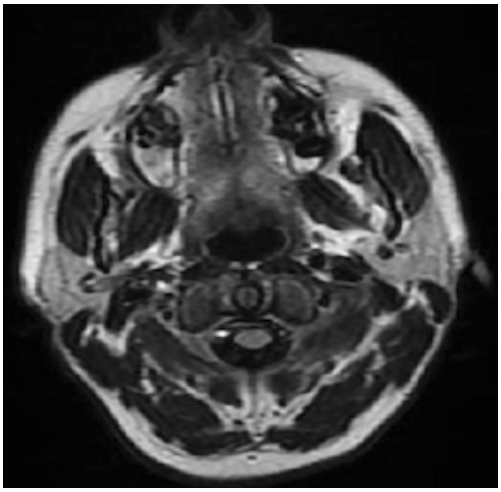


Figure 3. Brain MRI: no restricted diffusion, focal parenchymal signal abnormality, hemorrhage, mass or abnormal enhancement. Ventricular size and contour are appropriate.

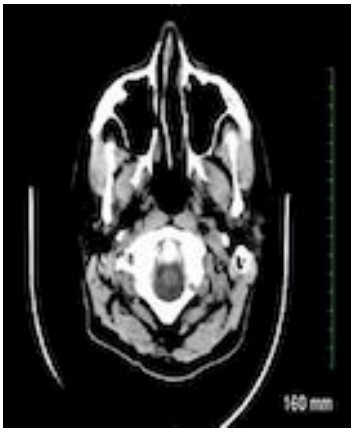


Figure 4. Brain CT: no acute intracranial hemorrhage, herniation or hydrocephalus.

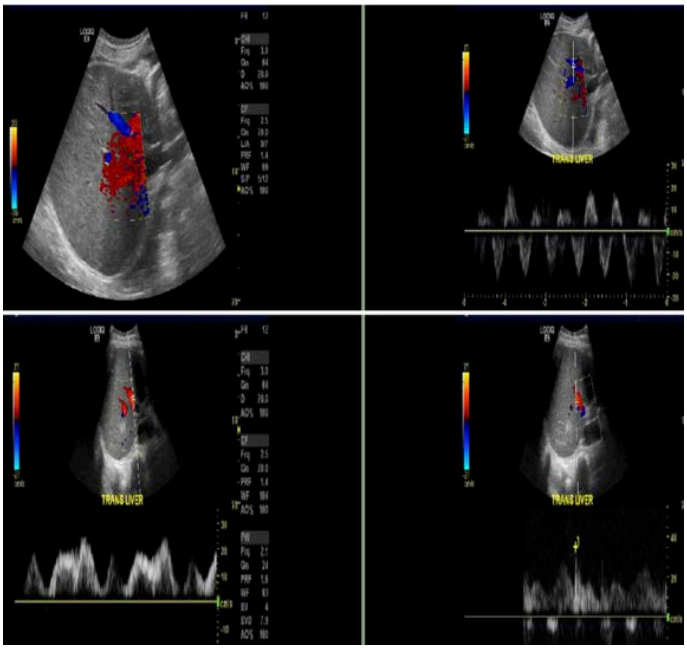


Figure 5. Echocardiographic Doppler images from the published image set.

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Carotid Ultrasound

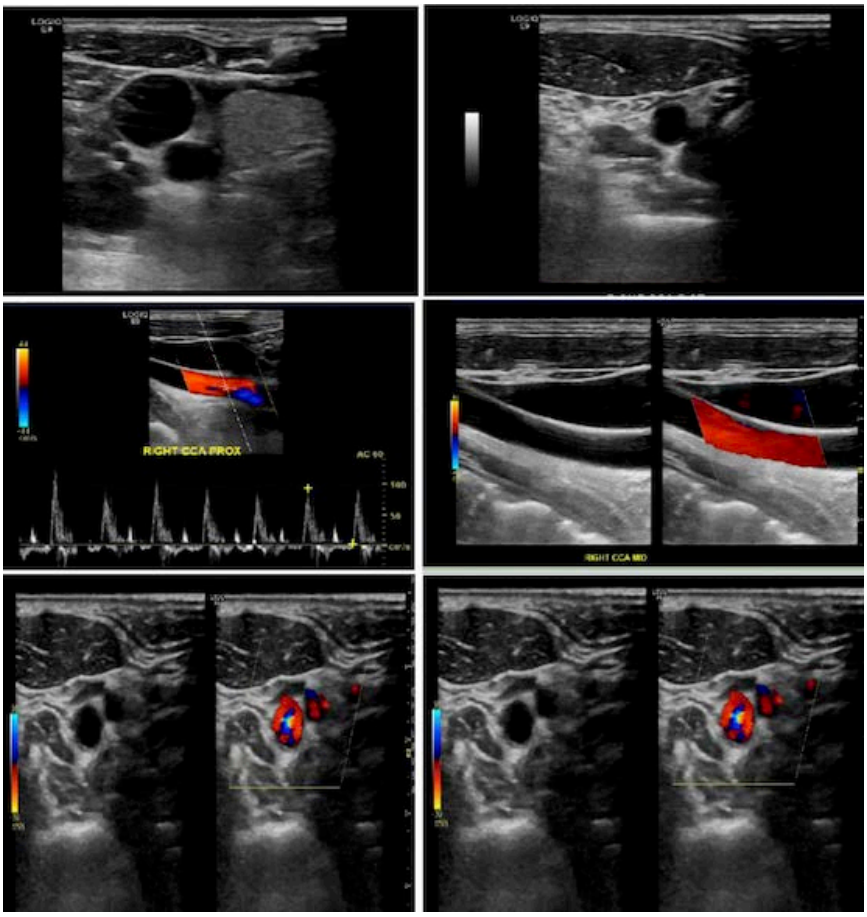


Figure 6. Carotid ultrasonography: no significant plaquing or stenosis. ICA stenosis is less than 50% bilaterally.

Discussion

We present a case of an adult patient with unsuspected splenic infarction secondary to aortic valve endocarditis associated with a suspected oral mucosal commensal. No recognized risk factors such as immunosuppression, valve disease, heart disease or IV drug use were reported.

Some risk factors implicated in the genesis of endocarditis include immunosuppression, intravenous drug use, poor dentition, degenerative valve disease and rheumatoid valve disease [2]. In hospitalized patients, recent vascular catheterization, prosthetic valve replacement and hemodialysis heighten the proclivity towards valvular endocarditis [2,3]. Our patient developed infective endocarditis without the above-mentioned recognized risk factors.

Among microbial organisms involved in bacterial endocarditis, gram-positive and gram-negative organisms account for 80% and 20% of cases, respectively [3]. Of gram-positive organisms, 40-45% are staphylococcal species (Staphylococcus aureus and coagulase-negative staphylococci), while 35-40% are streptococcal species (oral streptococci, Streptococci gallolyticus and enterococci). Gram-negative species account for roughly 20% of cases; 20% of these are attributed to Haemophilus, Aggregatibacter, Cardiobacterium and Eikenella corrodens, while Candida albicans is implicated in 2% of cases [3].

In this patient, the reported blood culture findings were considered most consistent with S. anginosus. This organism is part of the commensal flora and can be associated with invasive infection. Invasive systemic infections have been reported in the setting of immunosuppression, cancer and chronic disease [1].

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Abdominal CT

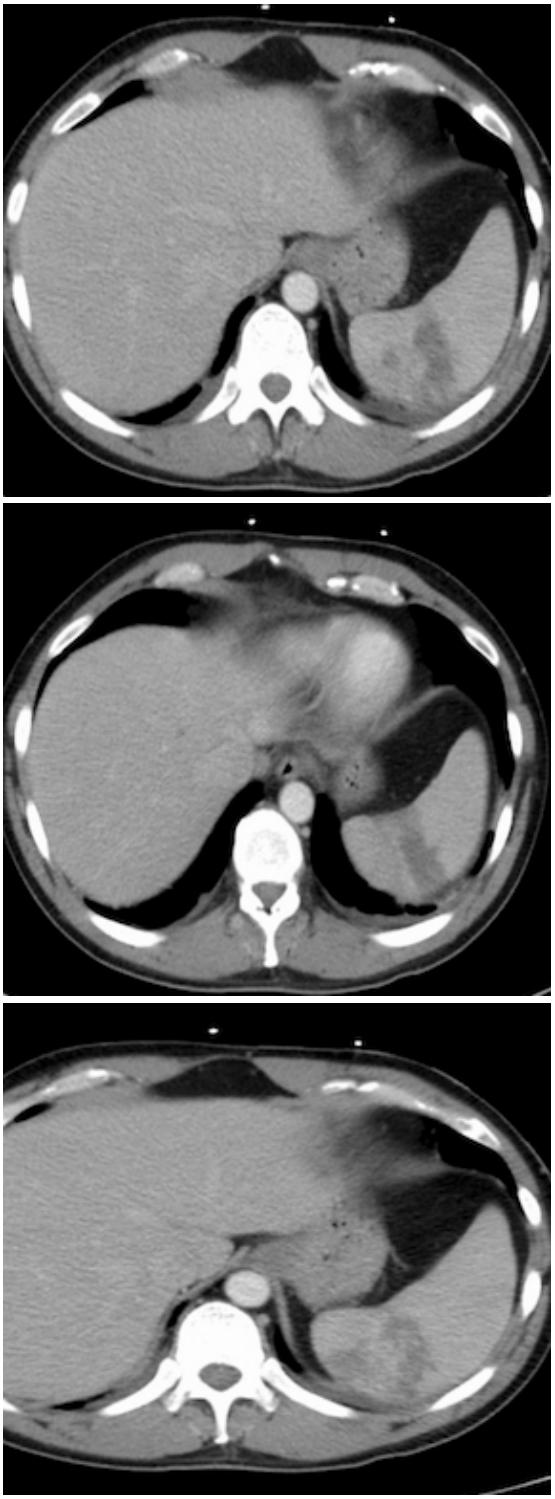


Figure 7. Abdominal CT: multiple splenic infarcts.

Our patient developed a thick bacterial vegetation on the aortic valve. In previous studies, aortic valve involvement predominated (47%-51%) compared with mitral valve involvement (30%-31%) in bacterial endocarditis [3,4]. Bacteria in the bloodstream do not usually adhere to the valvular surface. Exposure of the valvular subendothelial surface can initiate a cascade facilitating bacterial attachment [5,6]. Deposition of fibrin-platelet aggregates provides a stable scaffold for adherence [5,6].

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Adherence of bacterial colonies to the fibrin-platelet framework is further stabilized by plasma membrane components such as dextran [6]. The number of colony-forming units (CFU) in infected heart valves has been estimated at 10^9-10^{11} CFU per gram of valve tissue [7]. Activation of coagulation and additional fibrin-platelet deposition can surround the bacterial colonies [6], conferring resistance to host immune responses, including bacteria-specific antibodies [8]. The adaptive humoral immune response is triggered by bacterial colonization. Nevertheless, immune complexes, complement activation and neutrophil-mediated inflammation may not eradicate the bacterial colonization [7,9]. Instead, secondary immune phenomena such as glomerulonephritis, vasculitis and hypergammaglobulinemia can contribute to additional tissue damage [7,10].

Abdominal and pelvic CT in our patient revealed multiple splenic infarctions with bilateral pleural effusions and basal pulmonary pathology. Reports indicate that vegetations larger than 10 mm are more likely to be associated with systemic and pulmonary embolism [11]. In left-sided endocarditis, embolism to systemic organs such as the liver, kidney and spleen has been reported in 40% of cases [12]. The spleen is a common site of systemic embolism [13]. Splenic emboli can cause infarction, abscess, hemorrhage and rupture, related to the limited collateral supply and slow blood flow in the red pulp [13]. Symptoms of splenic infarction include left upper quadrant pain, tenderness, friction rub, nausea, vomiting and hiccups [13,14]. Endocarditis can present with fever, myalgia, chills, dyspnea, back pain, arthralgia, weight loss, stroke, heart failure, systemic embolization, septic shock and immunological manifestations, including hemorrhages, Osler nodes, Roth spots and Janeway lesions [3]. Our patient also presented with nausea, vomiting, memory impairment, headache and back pain.

Early CT or MRI of the abdomen can aid the diagnosis of infarcts and abscesses in systemic organs [13]. Three sets of blood cultures at 30-minute intervals before broad-spectrum antibiotics have been described for confirmation and organism identification [13,14]. Initial transthoracic echocardiography (TTE) assesses chamber and vegetation morphology (reported sensitivity 61% and specificity 94%) [14]. If TTE is nonconfirmatory, transesophageal echocardiography (TEE) provides further assessment of disease morphology and complications such as valvular regurgitation and perivalvular lesions (reported sensitivity 75% and specificity 90%) [3,15].

In this case, the diagnosis was based on the combination of the echocardiographic vegetation with severe aortic regurgitation, systemic symptoms, reported positive blood cultures and splenic embolic infarctions.

Treatment approaches described for infective endocarditis with splenic infarction include antibiotics, splenectomy and valve replacement [13]. Empiric antibiotic combinations pending culture results include ampicillin, gentamicin and flucloxacillin, or vancomycin and gentamicin in penicillin allergy [12]. Antibiotics should be tailored to culture results, with reported treatment durations of 4-6 weeks for native valve endocarditis [12]. According to the cited American College of Cardiology guidance, vegetations larger than 10 mm, systemic embolism and heart failure may necessitate valve replacement [16]. The patient underwent aortic valve replacement with a prosthetic heart valve.

Video 1. Transesophageal echocardiography demonstrating an approximately 1 cm vegetation on the right coronary cusp of the tricuspid aortic valve with severe aortic regurgitation. Article and supplementary material · Original published video

Summary

This case describes native aortic valve endocarditis with splenic embolic infarctions in an adult man with no recognized predisposing cardiac disease. The reported culture findings were considered most consistent with S. anginosus. The case illustrates that this presentation can occur even when no predisposing condition is recognized.

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16

Correspondence

Sriharsha Kanuri · harsha9009@gmail.com
Dept. of Internal Medicine, Merit Health Wesley Health Center, 5001 Hardy St, Hattiesburg, MS 39402, USA



Declarations

Consent for publication: The authors clarify that written informed consent was obtained and the anonymity of the patient was ensured. This study submitted to Swiss J. Rad. Nucl. Med. has been conducted in accordance with the Declaration of Helsinki and according to requirements of all applicable local and international standards.

Competing interests: No competing interests.

Funding: No funding resources.

Ethical approval: Not applicable.

Consent for publication: Informed consent taken.

Availability of data and materials: Not applicable.

Acknowledgements: Not applicable.

Authors' Contributions

Conceptualization: S.H.K. & S.A.; Methodology: S.H.K. & S.A.; Software: N.G.; Validation: N.A.; Formal analysis: N.A.; Investigation: N.A.; Resources: N.A.; Data curation: N.A.

Writing - original draft preparation: S.H.K. & S.A.; Writing - review and editing: S.H.K. & S.A.; Visualization: S.H.K.; Supervision: G.G. & S.F.; Project administration: G.G. & S.F.

Disclaimer/Publisher's Note

The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of Swiss J. Radiol. Nucl. Med. and/or the editor(s). Swiss J. Radiol. Nucl. Med. and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

License Policy

This work is licensed under a Creative Commons Attribution 4.0 International License. This license requires that reusers give credit to the creator. It allows reusers to distribute, remix, adapt, and build upon the material in any medium or format, even for commercial purposes.

SJORANM-LinkedIn: Check out our journal's LinkedIn profile with over 9,500 registered followers from the Radiologic & Nuclear Medicine Imaging field: LinkedIn

References

1. Pilarczyk-Zurek, M., et al. (2022). "The Clinical View on Streptococcus anginosus Group - Opportunistic Pathogens Coming Out of Hiding." Front Microbiol 13: 956677. https://doi.org/10.3389/fmicb.2022.956677
2. Yallowitz AW, D. L. (2025). "Infectious Endocarditis. "In: StatPearls [Internet]. https://www.ncbi.nlm.nih.gov/books/NBK557641/
3. Nappi, F. (2024). "Native Infective Endocarditis: A State-of-the-Art-Review." Microorganisms 12(7). https://doi.org/10.3390/microorganisms12071481
4. Bezak, B., et al. (2020). "Valve-related factors and incidence of prosthetic valve endocarditis." Kardiochir Torakochirurgia Pol 17(4): 178-182. https://doi.org/10.5114/kitp.2020.102341
5. Cheung, A. L., et al. (1991). "Fibrinogen acts as a bridging molecule in the adherence of Staphylococcus aureus to cultured human endothelial cells." J Clin Invest 87(6): 2236-2245. https://doi.org/10.1172/JCI115259
6. Sullam, P. M., et al. (1985). "Pathogenesis of endocarditis." Am J Med 78(6b): 110-115. https://doi.org/10.1016/0002-9343(85)90373-0
7. Lerche, C. J., et al. (2021). "Anti-biofilm Approach in Infective Endocarditis Exposes New Treatment Strategies for Improved Outcome." Frontiers in Cell and Developmental Biology Volume 9 - 2021. https://doi.org/10.3389/fcell.2021.643335
8. McCormick, J. K., et al. (2002). "Formation of Vegetations during Infective Endocarditis Excludes Binding of Bacterial-Specific Host Antibodies to Enterococcus faecalis." The Journal of Infectious Diseases 185(7): 994-997. https://doi.org/10.1086/339604
9. Kjerulf, A., et al. (1993). "Crossed immunoelectrophoresis used for bacteriological diagnosis in patients with endocarditis." Apmis 101(10): 746-752. https://doi.org/10.1111/j.1699-0463.1993.tb00175.x
10. Laxdal, T., et al. (1968). "Opsonic, agglutinating, and complement-fixing antibodies in patients with subacute bacterial endocarditis." The Journal of Laboratory and Clinical Medicine 71(4): 638-653. https://pubmed.ncbi.nlm.nih.gov/5651364/
11. Okan, T., et al. (2025). "Bilateral Multivalvular Infective Endocarditis Presenting as a Splenic Infarction and Acute Ischemic Stroke in a Young Immunocompetent Woman." Cureus 17(1): e77942. https://doi.org/10.7759/cureus.77942
12. Bayer, A. S., et al. (1998). "Diagnosis and management of infective endocarditis and its complications." Circulation 98(25): 2936-2948. https://doi.org/10.1161/01.CIR.98.25.2936
13. Alnasser, S. A., et al. (2019). "Successful Conservative Management of a Large Splenic Abscess Secondary to Infective Endocarditis." Ann Thorac Surg 107(4): e235-e237. https://doi.org/10.1016/j.athoracsur.2018.08.065
14. Baroudi, M. M., et al. (2023). "Subacute infective endocarditis presenting with an isolated splenic infarction." IDCases 32: e01752. https://doi.org/10.1016/j.idcr.2023.e01752
15. De Castro, S., et al. (2000). "Diagnostic Accuracy of Transthoracic and Multiplane Transesophageal Echocardiography for Valvular Perforation in Acute Infective Endocarditis: Correlation with Anatomic Findings." Clinical Infectious Diseases 30(5): 825-826. https://doi.org/10.1086/313762
16. Nishimura, R. A., et al. (2017). "2017 AHA/ACC Focused Update of the 2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines." Circulation 135(25): e1159-e1195. https://doi.org/10.1161/CIR.0000000000000503

Open Access · CC BY 4.0 · © 2025 The Author(s) · Published by SJORANM GmbH, Switzerland
ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 23(1):14-21 · DOI 10.59667/sjoranm.v23i1.16