Cryptic Septic Endocarditis and Systemic Thromboembolism Triggered by Oral Microflora

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Swiss Journal of Radiology and Nuclear Medicine - www.sjoranm.com - Rosenweg 3 in CH-6340 Baar, Switzerland

Abstract

This unique clinical case underscores the pathogenic nature of oral commensal microbial flora such as Streptococcus Anginosis in provoking infective endocarditis of physiologically normal native heart valves despite the absence of priming systemic risk factors. Understanding the triggering events for this pathological transformation of Stroptococcus Anginosus in normal patients is very much necessary. This will allow the clinicians to anticipate this transpiration in the clinical settings and execute appropriate therapeutic measures to minimize the morbidity and mortality.

Keywords: *Streptococcus Anginosis, Gram-Positive-Cocci, Sepsis, Vegetations, Thromboembolism and Splenic Infarction, Aortic Regurgitation*

[*]Corresponding author: Sriharsha Kanuri - received: 14.09.2025 - peer reviewed, accepted and published: 30.09.2025

Introduction

Streptococcus Anginosus is a gram-positive streptococci that is usually resides in the mucous membranes of the oral cavity, with eventual migration to nasopharynx, gastrointestinal tract and genitourinary tract [1]. The risk of infection with S. Anginosus amplifies under 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 that, they are involved in causing brain abscess, pleural empyema, pneumonia, pericarditis, and endocarditis [1].

We present a clinical case where a young patient with no predisposing factors presented with vague clinical symptoms had aortic valve endocarditis and splenic infarction secondary to infection with Streptococcus Anginosus. This is a rare clinical presentation due to the fact that Streptococcus Anginosus escaped into the blood stream from the gastrointestinal tract and infected the physiologically normal aortic valve in a young male with otherwise no risk factors, thence initiating cascading chain of events that ultimately culminated into infective endocarditis, sepsis, thromboembolism and splenic infarction.

Clinical Case

36-year-old male who presents to the ER with fever, nausea with vomiting, fatigue, and difficulty forming new memories. Patient's wife is at bedside helping him provide history. Patient states for the past 3 to 4 weeks he has had an upper respiratory infection with cough. Cough has been productive. Two weeks ago he started vomiting. Since then he has had decreased appetite, vomits everytime he tries to eat. His wife states this

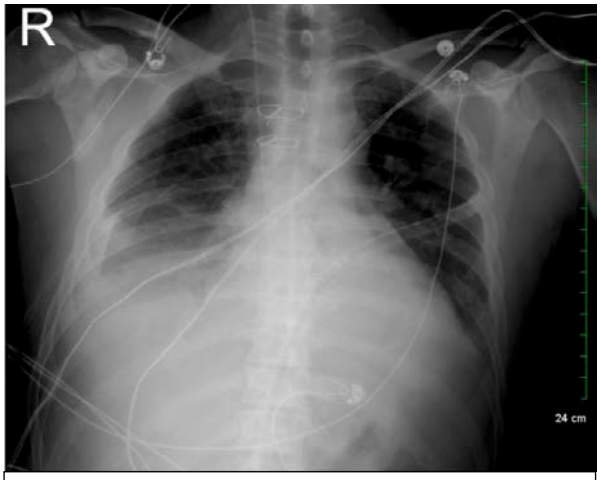


Figure 1: Chest X ray: *Bilateral pleural effusions with bibasilar airspace disease. No pneumothorax is seen. Heart size is stable. Right IJ catheter is stable. Left chest tube is stable. Sternal wires are noted.*

morning around 8.30 he started becoming con-fused with repetitive speech and asking the same questions over and over again. He and his wife were on an oil change mechanic shop. He was out in the heat most days. States he has been very dehydrated. Woke up one night this week with severe left calf cramping. After fevers started coming on the last week to the urgent care clinic received a Z-Pak. States that they tried to test him for COVID but he would not let him. BP here

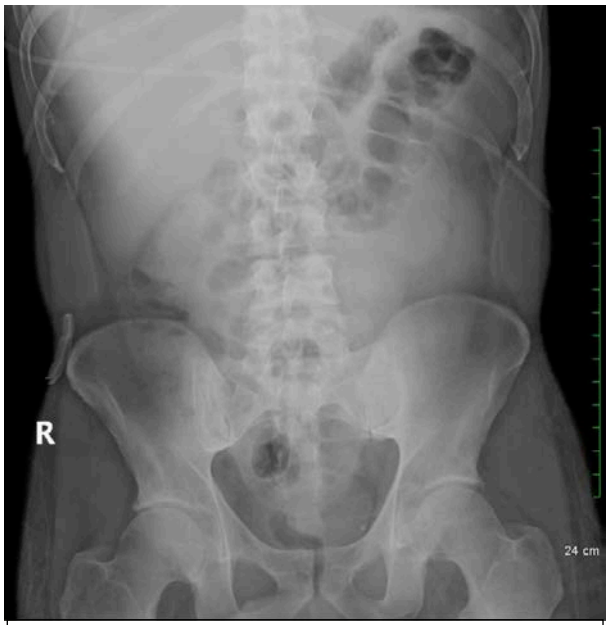


Figure 2: Abdominal X-ray: *No acute abnormality.*

today on 6/23/2025 and was tested for flu and negative. He had his wife traveled to Indiana last month, they drove but took frequent breaks every couple hours to help with her special needs son developed an electrical disabilities, hearing loss, quadri-plegia after contracting strep pneumonia

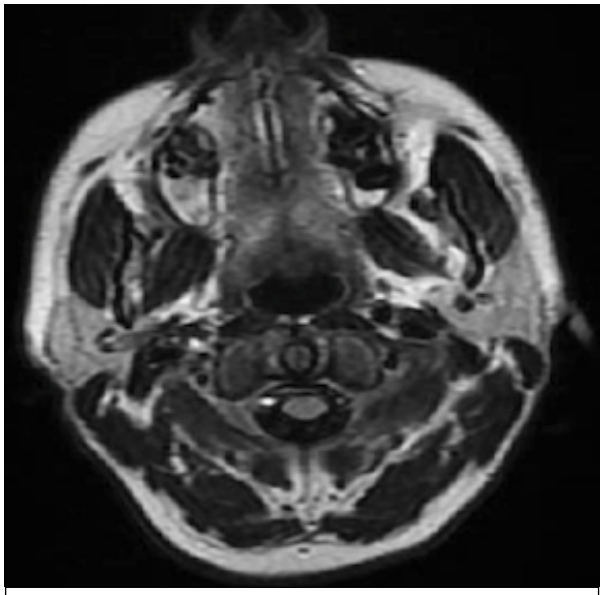


Figure 3: MRI of the Brain: *There is no restricted diffusion to suggest acute ischemia. Ventricles size and contour appropriate. There is no mass, midline shift. No hemor-rhage is seen. There is no focal parenchymal signal ab-normality identified. After the injection of contrast there is no abnormal enhancement. Normal MRI brain without and with contrast.*

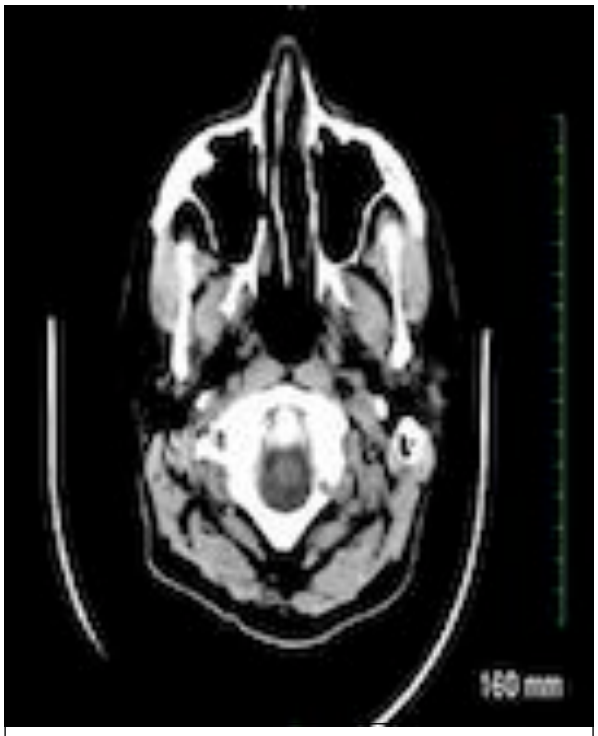


Figure 4: CT-scan of the Brain: *No acute intracranial hemorrhage, herniation or hydrocephalus.*

meningitis when he was 8 months old. Have a cat but patient does not change the litter box. Not around cattle or farm animals or rodents. He does not recall any tick bites. No recent mosquito bites. His mechanic shop is well ventilated. Home water is city water, not well. He has no other sick contacts. No contributory family history. Denies any

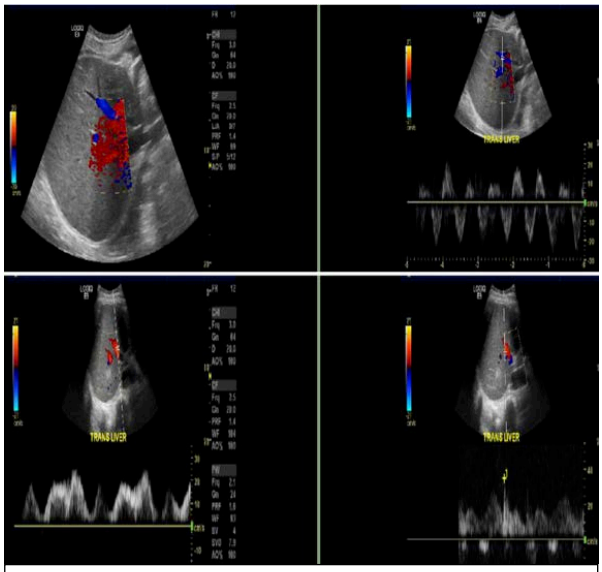


Figure 5: Ultrasonography liver: *Mild diffuse coarse hepatic echotexture. 2.2 cm nonvascular echogenic focus posterior right hepatic lobe, potentially hemangioma likely reflecting roughly 2 cm partially low-density lesion with peripheral nodular enhancement posterior right hepatic lobe on CT limited in characterization with single-phase imaging but suggestive of hemangioma. Partially distended gallbladder with borderline gallbladder wall thickening. No sonographic findings to suggest acute cholecystitis.*

personal or family history of autoimmune disease or immunocompromised states. No cardiac issues. No personal or family history of clotting disorders. He is a nondrinker, nonsmoker, non-IV drug user. Denies any trauma. Chest X-ray showed bilateral pleural effusions and bibasilar pulmonary disease (Figure 1). Abdominal X-ray is unremarkable (Figure 2). CT head and MRI brain was obtained and unremarkable (Figure 3 & 4). Ultrasonography liver showed hemangioma and gall bladder distension (Figure 5). Ultrasonography carotid showed no significant abnormality or narrowing (Figure 6). CT abdomen pelvis with contrast (Figure 7) showed multiple acute or sub-acute splenic infarcts with mild splenomegaly and mild left pleural effusion. Echocardiogram performed today revealed EF estimated 64%, LA mildly dilated, structurally normal mitral valve, moderate mitral regurgitation, severely elevated PA pressure 90 mmHg and dilated IVC. TEE (Video 1) performed showing severe aortic regurgitation with vegetation 1 cm on the right coronary cusp, tricuspid aortic valve moderate mitral regurgitation, and mild TR.

While awaiting the culture results, treatment was started with Vancomycin, Cefepime, Ampicillin and Gentamycin. Blood cultures are now showing gram-positive cocci in chains and rods, most likely Streptococcus Anginosus. He is currently treated with Penicillin G potassium 3 million units 100 ml/hr, IV piggyback Q4H 07. During follow up, he developed migraine like headache and neurology is consulted. He was ultimately planned for Aortic valve replacement with prosthetic heart valve.

Discussion

We present a unique case of young patient who presented with unsuspected splenic infarction which is secondary to aortic valve endocarditis provoked by oral mucosal commensal. Surprisingly, he does not present any risk factors such as immunosuppression, valve disease, heart disease or IV drug abuse.

Some of the risk factors that are incriminated in the genesis of endocarditis include immunosuppression, intravenous drug abuse, poor dentition, degenerative valve disease and rheumatoid valve disease [2]. In the hospitalized patients, recent vascular catheterization, prosthetic valve replacement, and hemodialysis, will heighten the proclivity towards developing valvular endocarditis [2, 3]. Surprisingly, our young patient developed infective endocarditis in the absence of any of the above-mentioned risk factors.

Out of the microbial organisms involved in the inception of bacterial endocarditis, gram-positive and gram negative account for 80% and 20% cases [3]. Out of gram positive species, 40-45% are provoked by staphylococcus species [Staphylococcus aureus & coagulase negative staphylococcus] while 35-40% are elicited by streptococcus species [Oral streptococci, Streptococci Gallo-lyticus and Enterococci]. Gram negative species account roughly for 20% of the cases. 20% of them are incited by Haemophilus aggregatibacter, Cardiobacterium and Eikenella corrrodens, while Candida albicans are incriminated in 2% cases [3]. In our patient, blood culture revealed Streptococcus Anginosus (gram-positive cocci & rods). This microorganism usually colonizes the mucous membrane of the oral cavity and is non-pathogenic. Invasive systemic infections are reported and transpires in the setting of immunosuppression, cancer, and chronic diseases [1].

Our patient has developed a thick bacterial vegetation on the aortic valve. In previous studies, it was determined that aortic valve

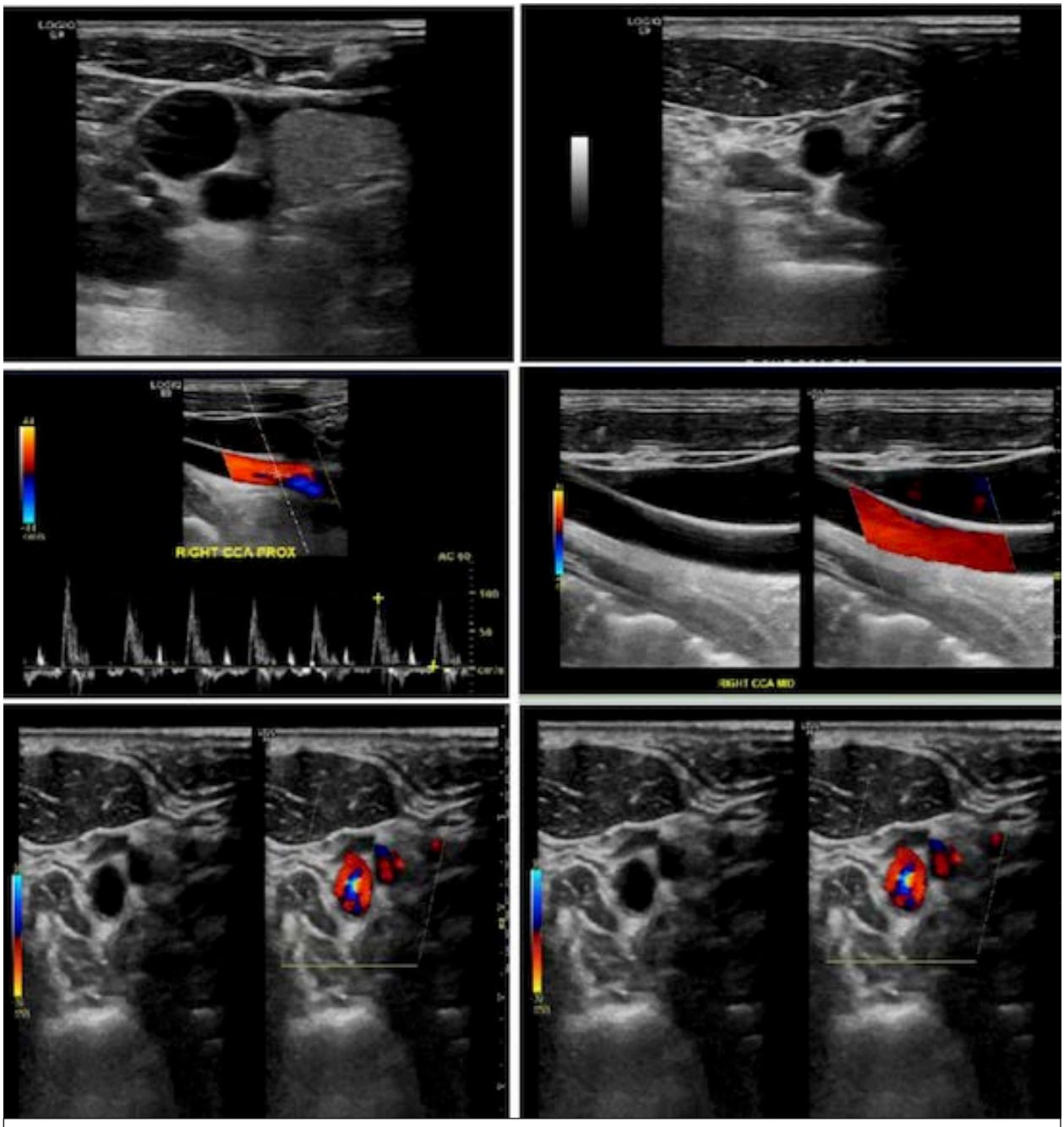


Figure 6: Carotid USG: *No significant plaquing or stenosis is seen. ICA stenosis is less than 50% bilaterally.*

involvement is predominant (47%-51%) as compared to mitral valve (30%-31%) for bacterial endocarditis [3, 4]. Bacteria floating around in the blood do not usually clasp to the valvular surface. However, mutilation and laying bare of valvular sub-endothelial surface is a turning point that will have a cascading effect by greasing the wheels for future sinister events [5, 6]. As deposition of fibrin-platelet aggregates on the valvular surface materializes, a viable and stable scaffolding for bacterial clinging is brought into existence [5, 6]. This adherence of the bacterial colonies to the fibrin-platelet framework is further ratified and stabilized by their plasma membrane components such as dextran [6]. The number of colony forming units (CFU) of bacteria in the heart valves is estimated to be 109–1011 CFU per gram of heart valve tissue [7] Presumably, the protracted asylum of the bacterial colonies on the valvular surface is shrewdly executed by the activation of the clotting mechanism, thus triggering the deposition of more fibrin-platelet on the bacterial colonies [6]. As these bacterial colonies are wrapped around by fibrin-platelet meshwork, they become resistant to host immune responses including

bacteria-specific antibodies [8]. As expected, adaptive humoral immune response is triggered as a counterattack to bacterial colonization of valvular tissue. Nevertheless, tissue defenses including immune complexes, complement activation and neutrophil mediated inflammation, are not adept enough in eradicating the bacterial colonization [7, 9].

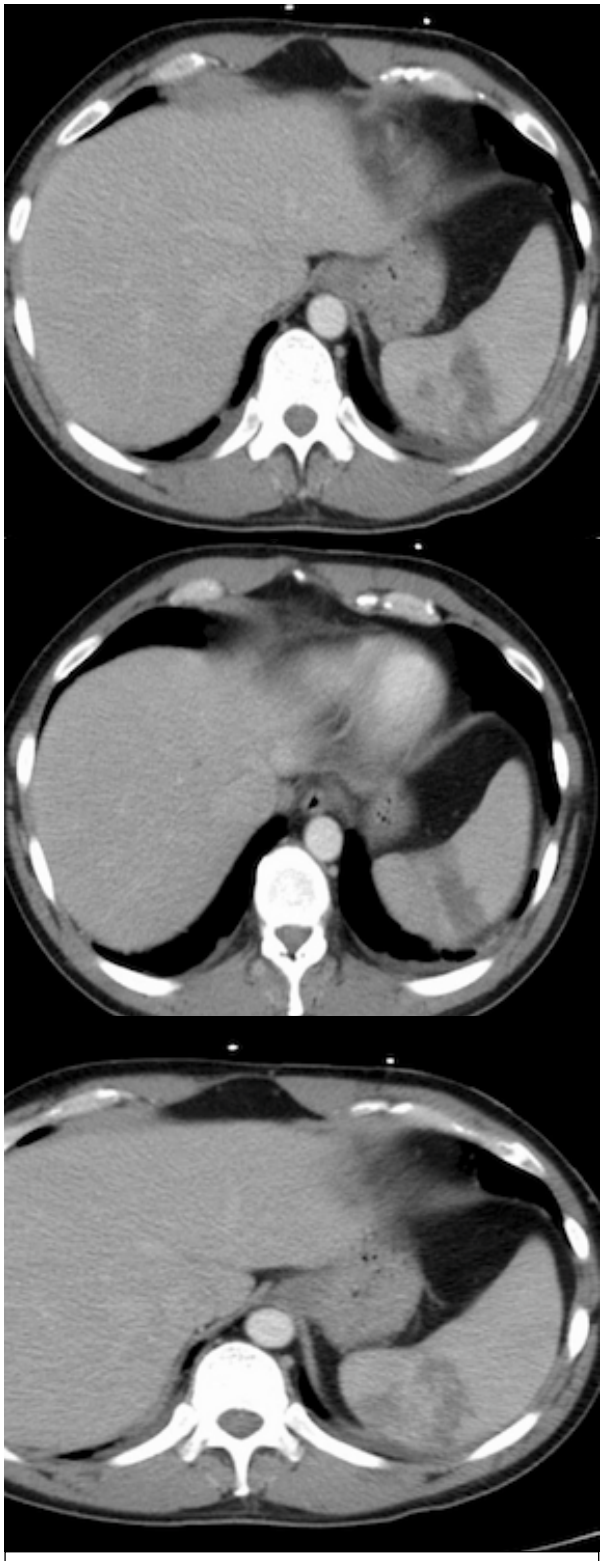
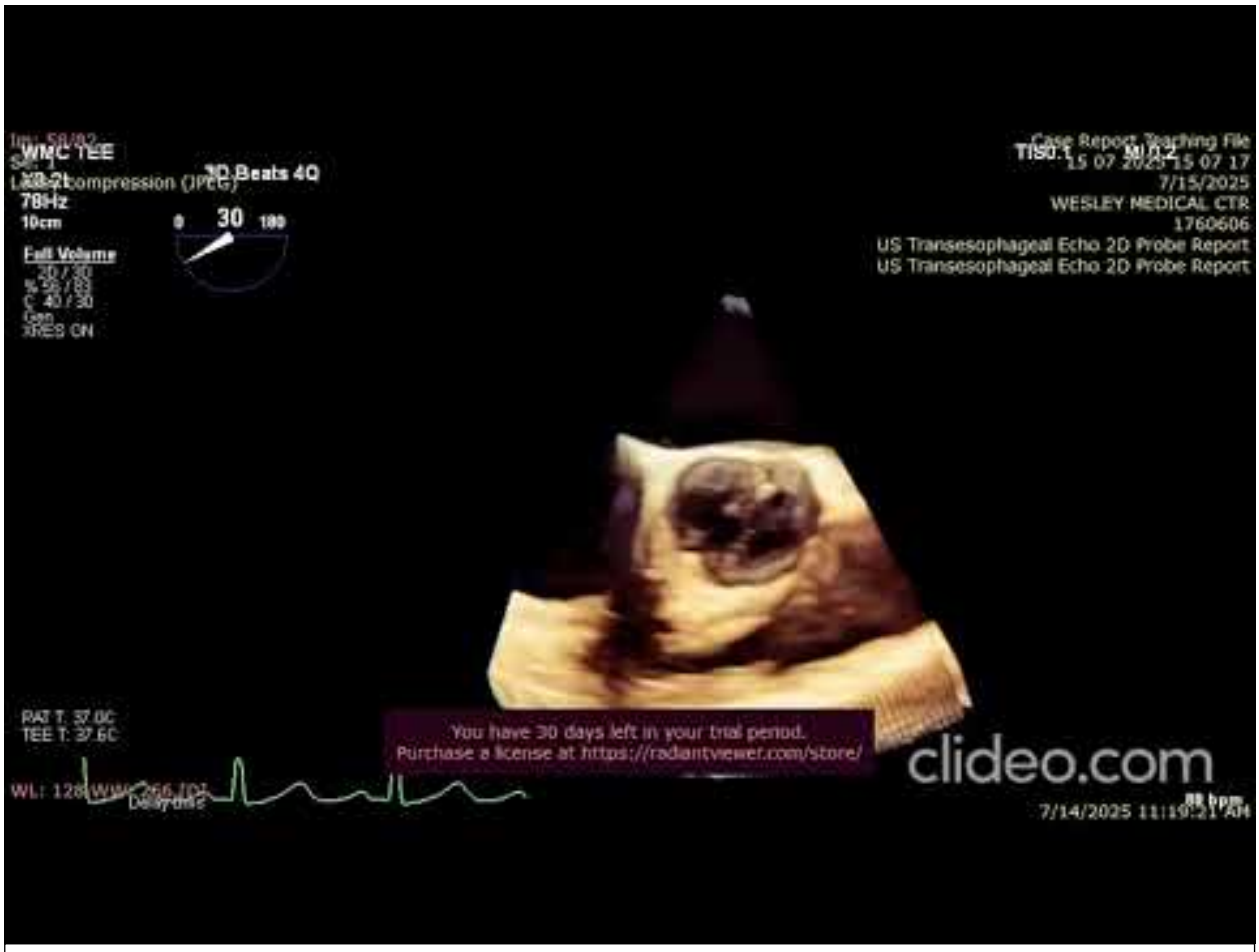


Figure 7: CT-scan Abdomen: *Multiple splenic infarcts.*

Instead, they will rather assist in eliciting secondary immune phenomenon (glomerulonephritis, vasculitis, hypergammaglobunimia) widely reported in these patients and further provoke adjuvant tissue damage [7, 10].

CT scan abdomen/pelvis in our patient revealed, multiple splenic infarctions with bilateral sided pleural effusion and basal pulmonary pathology. Reports indicate that, bacterial vegetations greater than 10 mm size were more likely associated with systemic and pulmonary embolism [11]. In patients with left-sided endocarditis, embolism to systemic organs such as liver, kidney and spleen can be expected in 40% cases [12]. Spleen is the most common organ for predilection of systemic embolism in the bacterial endocarditis [13].The bacterial emboli that migrate to spleen are more likely provoke complications such as infarction, abscess, hemorrhage and rupture due to absence of collateral blood supply and slow-moving blood supply within red-pulp [13]. Typical symptoms that occur with 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 sequelae (Sphincter hemorrhages, conjunctival hemorrhages, Osler nodes, Roth spots and Janeway lesions) [3].
However, our patient presented also with most unusual symptoms and signs such as nausea, vomiting, global amnesia, headache and back pain.
Early diagnosis with CT scan and MRI abdomen is usually helpful, both of which have high sensitivity and specificity for diagnosis of infarcts / abscess in the systemic organs [13]. At the same,-three sets of blood cultures at 30 minutes intervals to confirming the diagnosis of endocarditis is deemed necessary before institution of broad spectrum antibiotics for coverage of most probable microorganisms involved [13, 14].

Initially, a transthoracic echocardiography (TTE) is performed to ascertain the heart chambers and vegetation morphology (Sensitivity 61% and Specificity 94%) [14]. If the TTE is non-confirmatory, then trans-oesophageal echocardiography (TEE) is the next



Video-Caption Transesophageal echocardiography (TEE):
1. The type of imaging 2D/3D
2. The acquisition window (e.g. parasternal, apical, etc.),
3. The parameters used: Color Doppler
4. The orientation / display: LAX/SAX
5. Pathology: Thick aortic valve vegetations consisting of Streptococcus Anginosus that ultimately lead to systemic embolism to the spleen.

step, which is more reliable (Sensitivity 75% and Specificity 90%) to investigate the disease morphology [Mobile, oscillating & irregular shape] and complications [Valvular Regurgitation & peri-valvular lesions] [3, 15].

Duke's clinical diagnosis of endocarditis has one of major criteria (Positive echocardiography, Positive blood culture and New valvular regurgitation) and one minor criteria (predisposing cardiac condition, high temperature, vascular phenomenon, and immunological sequelae) [3].
The cornerstone of therapy of patient with infective endocarditis with splenic infarct is a combination of antibiotics, splenectomy and valve replacement surgery [13]. Empiric antibiotic therapy pending blood culture results include Ampicillin, Gentamycin and Flucloxacillin. Or Vancomycin and Gentamycin (Pencillin Allergy) [12]. The antibiotics should be appropriately tailored based on the culture results and administered for 4-6 weeks in native valve endocarditis [12]. According to American College of Cardiology, vegetations size > 10mm, systemic embolism and heart failure might necessitate valve replacement surgery [16]. Our patient had a valve replacement surgery with a prosthetic heart valve.

Summary

This unique clinical case underscores the pathogenic nature of oral commensal microbial flora such as Streptococcus Anginosis in provoking infective endocarditis of physiologically normal native heart valves despite the absence of priming systemic risk factors. Understanding the triggering events for this pathological transformation of Stroptococcus Anginosus in normal patients is very much necessary. This will allow the clinicians to anticipate this transpiration in the clinical

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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 & Editing: S.H.K.& S.A.;
- Visualization: S.H.K.;
- Supervision: GG & S.F..;
- Project Administration: GG .& S.F.

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