sciendo Internal Medicine 2025 vol. XXII No. 2 - www.srmi.ro 49
10.2478/inmed-2025-0329

INFECTIVE ENDOCARDITIS WITH S. GORDONII ON NATIVE MITRAL ANTERIOR VALVE

Florin Mitu[1,2,3,4], Ilinca-Christiana Comănescu[5], Radu-Ștefan Miftode[6], Maria Magdalena Leon[1,2*], Alexandra Maștaleru[1,2*], Miftode Egidia Gabriela [5,7]

[1.] Clinical Rehabilitation Hospital, 700661 Iasi, Romania

[2.] Department of Medical Specialties I, "Grigore T. Popa" University of Medicine and Pharmacy, 700115 Iasi, Romania

[3.] Academy of Medical Sciences, 030167, Bucharest, Romania

[4.] Academy of Romanian Scientists, 700050, Iasi, Romania

[5.] "Sf. Parascheva" Hospital of Infectious Diseases, Iasi, Romania

[6.] Department of Internal Medicine I (Cardiology), Faculty of Medicine, University of Medicine and Pharmacy "Gr. T. Popa", 700115 Iasi, Romania

[7.] University of Medicine and Pharmacy "Grigore T. Popa", 700115 Iași, România

Correspondence to: leon_mariamagdalena@yahoo.com ; alexandra.mastaleru@gmail.com

Abstract

We report the case of a 66-year-old male with a complex medical history, including grade 3 essential hypertension NYHA class II chronic heart failure with preserved LVEF and chronic ischemic heart disease. The patient was also diagnosed with ankylosing spondylitis, receiving biologic therapy with etanercept, type II diabetes mellitus and gout. The patient presented with a progressive aggravation of his chronic pathology three months anterior to hospitalization, characterized by significant physical asthenia, exertional dyspnea, anterior chest pain, tremor of the extremities, fever with chills and a syncopal episode. These symptoms prompted evaluation in the Cardiology Department of "St. Spiridon" Hospital, Iași, where the diagnosis of infective endocarditis (IE) involving the native anterior mitral valve was established.

The diagnosis was based on clinical presentation, laboratory investigations, echocardiographic findings, and the isolation of Streptococcus gordonii from blood cultures obtained during a febrile episode. Following diagnosis, the patient was referred to the "St. Parascheva" Infectious Diseases Clinical Hospital, Iași, for specialized antimicrobial therapy and further management. Notably, despite the patient's immunocompromised state — attributed to diabetes mellitus and ongoing immunosuppressive therapy with a tumor necrosis factor (TNF) inhibitor — clinical progression was favorable.

This was evidenced by resolution of fever, a marked reduction in inflammatory markers and nitrogen retention parameters, and a stable echocardiographic profile at discharge. This case underscores the importance of early diagnosis and appropriate antimicrobial therapy in

managing infective endocarditis, even in patients with multiple comorbidities and immunosuppressive conditions, thus highlighting the potential for non-surgical resolution in selected cases.

Keywords: *infective endocarditis, Streptococcus gordonii, immunosuppressed status*

Rezumat

Prezentăm cazul unui bărbat în vârstă de 66 de ani cu un istoric medical complex, ce include hipertensiune arterială esențială de gradul 3, insuficiență cardiacă cronică de clasa a II NYHA cu fracție de ejecție prezervată, cardiopatie ischemică cronică. Pacientula fost diagnosticat cu spondilită anchilozantă (fiind sub terapie biologica cu etanercept), diabet zaharat tip II și gută. Pacientul a manifestat o simptomatologie cu evoluție progresivă trei luni anterior internării, aceasta constând în astenie fizică marcată, dispnee la efort minim, durere toracică anterioară, tremor al extremităților, febră însoțită de frisoane și un episod de sincopal. Aceste simptome au determinat evaluarea în Secția de Cardiologie a Spitalului „Sf. Spiridon" din Iași, unde a fost stabilit diagnosticul de endocardită infecțioasă (EI) cu implicarea valvei mitrale anterioare native.

Diagnosticul s-a bazat pe examenul clinic, investigațiile de laborator, rezultatele ecocardiografice și izolarea Streptococcus gordonii din hemoculturile obținute în timpul unui episod febril. În urma diagnosticului, pacientul a fost trimis la Spitalul Clinic de Boli Infecțioase „Sfânta Parascheva", Iași, pentru terapie antimicrobiană de specialitate și management ulterior. În special, în ciuda stării imunocompromise a pacientului - atribuită diabetului zaharat și terapiei imunosupresoare continue cu un inhibitor al factorului de necroză tumorală (TNF) - evoluția clinică a fost favorabilă.

Acest lucru a fost evidențiat prin rezoluția febrei, o reducere marcată a markerilor inflamatori și a parametrilor de retenție azotată, precum și un profil ecocardiografic staționar la externare. Acest caz subliniază importanța diagnosticului precoce și a terapiei antimicrobiene adecvate în gestionarea endocarditei infecțioase, chiar și la pacienții cu comorbidități multiple și condiții imunosupresoare, și evidențiază potențialul de rezolvare nechirurgicală în cazuri selecționate.

Cuvinte cheie: *endocardită infecțioasă, Streptococcus gordonii, status imunodeprimat*

Introduction

Infective endocarditis (IE) is an infection of the heart valves, endocardial surface, or cardiac devices, with persistent incidence and mortality over the past 30 years despite advancements in diagnosis and treatment (1). The epidemiology has shifted toward older, chronically ill patients and more health care–associated infections, with *Staphylococcus* species becoming more prevalent than penicillin-sensitive *Streptococcus*.

IE now presents with nonspecific symptoms, making early diagnosis — especially in emergency settings — dependent on high clinical suspicion. Fever of unknown origin, new heart murmurs, or unexplained embolic events should prompt immediate blood cultures and echocardiography. Although IV antibiotics are the mainstay of treatment, nearly half of patients eventually require surgery. Management benefits from early involvement of cardiology and infectious disease specialists, while antibiotic guidelines remain largely based on expert consensus due to limited clinical trial data[2-5].

IE remains a rare condition, with an annual incidence of 3 to 10 cases per 100,000 people, stable over the past two decades but varying by region. In low-income countries, rheumatic heart disease continues to be the main risk factor, while in developed countries, cases are now more often linked to injection drug use, degenerative or congenital valve disease, prosthetic valves, and cardiac devices.

The average age of IE patients has risen significantly, with most now over 70 and commonly presenting with comorbidities such as diabetes, cancer, COPD, and liver disease. Although overall incidence is unchanged, *Staphylococcus aureus* has become a leading cause, responsible for about 25% of cases in high-income countries, and IE remains more common in men[2,3,6].

Clinical signs and symptoms

Despite being recognized for over a century, IE remains challenging to diagnose due to its highly variable and nonspecific presentation. Clinical manifestations can range from subtle, flu-like symptoms in stable patients to rapid-onset sepsis with multiorgan failure, depending on host factors, vegetation location, and pathogen virulence.

Common symptoms like fatigue, weight loss, dyspnea, back pain, or neurological signs often mimic other conditions, leading to frequent misdiagnosis or delayed recognition.

Regarding the most frequently signs in IE, more than half of the patients suffer from heart murmurs, congestive heart failure, septic pulmonary emboli, fever or sepsis of unclear origin, splinter hemorrhages, Roth spots, glomerulonephritis, cerebral complications or peripheral emboli and abscesses. Regarding the symptoms, some of the most frequently met in patients with IE are fever with chills, malaise, poor appetite, dyspnea, weight loss [3,7].

Clinical case presentation

We present the case of a 66-year-old male patient from a rural area with significant cardiovascular pathology, including essential arterial hypertension grade 3, stage 2, chronic heart failure NYHA class II with preserved LVEF, chronic ischaemic heart disease, moderate-severe mitral insufficiency, moderate tricuspid insufficiency, and mild aortic insufficiency. Additionally, the patient has ankylosing spondylitis under biologic therapy, type II diabetes mellitus, and gout. The heredo-colateral history of the patient is without pathologic significance.

The patient has reported symptoms for approximately three months prior to hospitalization, including significant physical weakness, dyspnea upon minimal exertion,

anterior chest pain, extremity tremors, fever, chills, and a syncopal episode. Consequently, he was transported by the County Ambulance Service to the Cardiology Department of "St. Spiridon" Clinical Hospital in Iasi, where he was admitted from September 3 to September 11, 2024.

The initial clinical examination upon admission to the Cardiology ward indicated a mildly affected general condition, afebrile, hemodynamically and respiratory stable, with a blood pressure of 145/85 mmHg, heart rate of 100 bpm, and a grade III/VI systolic murmur at the mitral focal point, normal skin coloration, and absence of oedema.

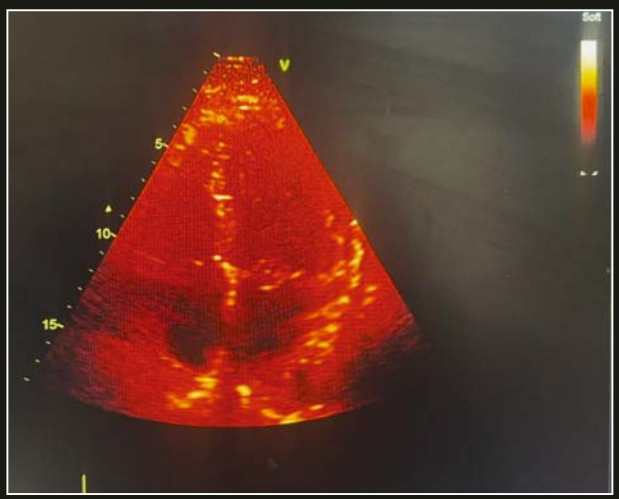
The biological examination on admission revealed mild thrombocytopenia, hyperglycemia (118mg/dl), NTproBNP = 958pg/ml, mild inflammatory syndrome (CRP=2mg/dl).

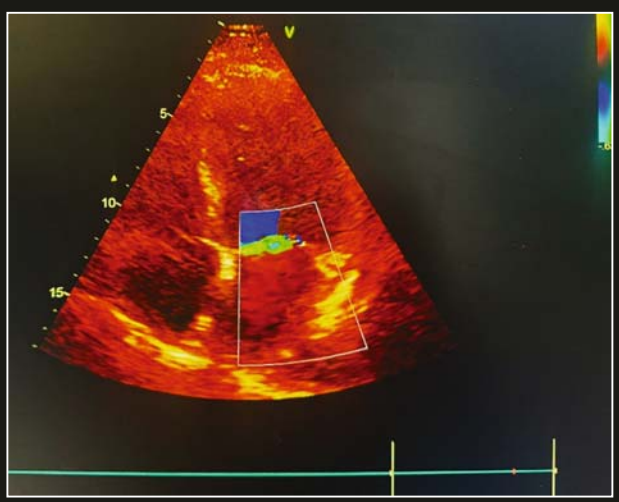
Imaging investigations performed in the Cardiology Department of "St. Spiridon" Clinical Hospital

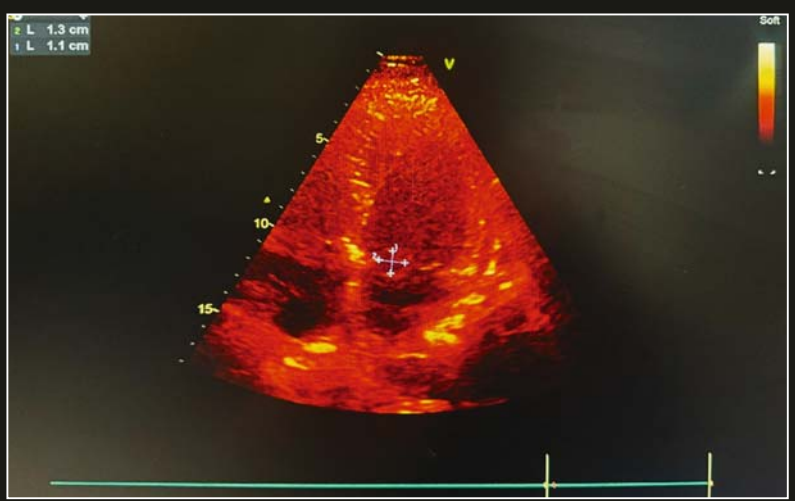
- **EKG:** sinusal tachycardia, 100 bpm, intermediate QRS axis, without ST-T segment changes.
- **Echocardiography**: undilated LV, unhypertrophied, without segmental kinetic changes, with normal systolic function, LVEF = 60% , LA = 48/52mm, RA = 42/36mm, E/A wave >2, tricuspid Ao valve with preserved opening, Vmax Ao=1.5m/s. An addition image at the level of the atrial face of the AMV that has 11/8 mm, with aneurysm appearance (Figures 1-3).
- **Transesophagean echocardiography**: P2, P3 prolapse. Isoechoic adductor image, with its own mobility, diameter = 5 mm, suggestive for vegetation at P2 level, generating moderate-severe MR, PISA radius = 10 mm, vena contracta = 5 mm, with eccentric jet. Tricuspid Ao valve with no additive images. No images of abscess at mitro-aortic annulus. Free LA appendage.

Given the patient's immunosuppressed condition (diabetic, undergoing biologic treatment), the presence of symptoms (fever, chills), clinical findings (systolic murmur grade III/VI at the mitral focal point), and echocardiographic results, there is a heightened suspicion of infective endocarditis.

Three sets of blood cultures are obtained during the febrile episode, and empiric antibiotic therapy is initiated with Ceftriaxone at 4g/day and Gentamicin at 240mg/day for three days, followed by a reduction of the Gentamicin dosage to 160mg/day based on Creatinine Clearance levels. Considering the identification of an aneurysm-like image on the atrial side of the AMV, anticoagulant therapy with Enoxaparin (Clexane) at a dosage of 0.4 ml/day is administered alongside the forementioned antibiotic therapy.







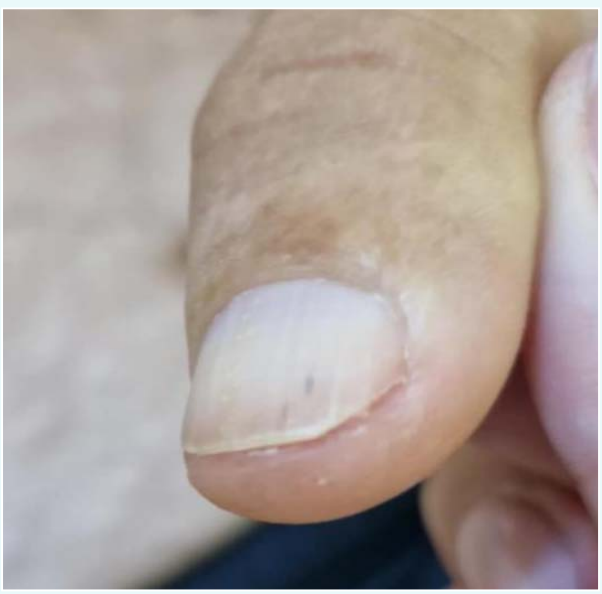
Figures 1-3.
Echocardiography images

Subsequently, blood cultures revealed the presence of Streptococcus gordonii, prompting the transfer of the patient, in the ninth day after admission to the Clinical Hospital of Infectious Diseases "St. Parascheva" Iasi.

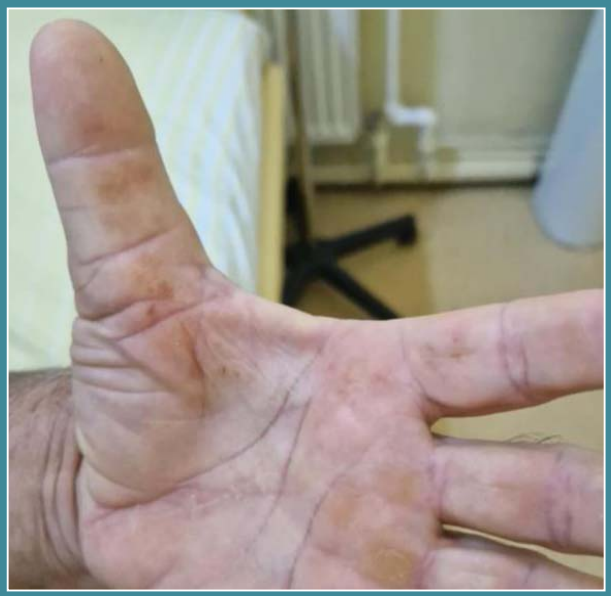
The clinical examination indicated a generally stable condition; the patient was afebrile, conscious, and cooperative, with no present oedema.

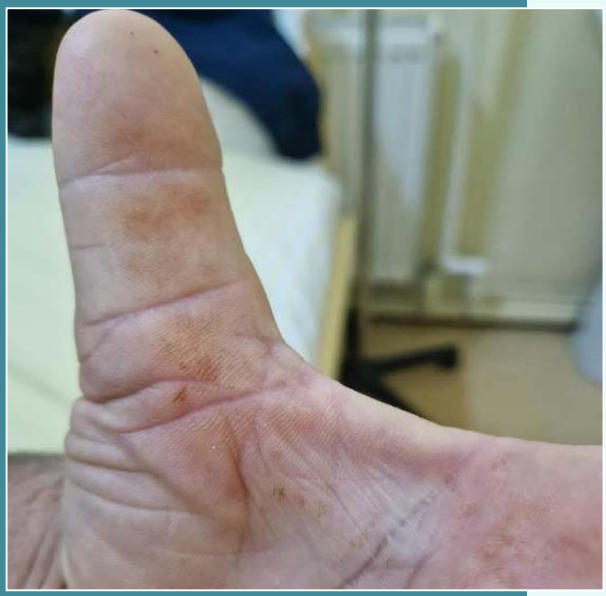
There was evidence of partial edentulism with root debris, Janeway lesions located in the left palmar region, and splinter haemorrhages in the nail beds of both upper limbs (Figures 4-7). Cardiac auscultation revealed rhythmic heart sounds and a grade III/VI systolic murmur in the mitral area. Pulmonary assessment showed physiological findings, with blood pressure recorded at 117/67 mmHg, heart rate at 60 bpm, and oxygen saturation at 96%. The abdomen was painless, and there were no signs of meningeal irritation or abnormalities in urinary and digestive function.The paraclinical examinations after admission to the Infectious Diseases Ward showed the following changes: mild normochromic, normocytic anemia, thrombocytopenia, inflammatory syndrome, mild hypokalemia, nitrogen retention syndrome, hypocholesterolemia, urinary sediment: proteinuria, minimal hematuria, glycosuria, negative urine culture (Table 1).

The differential diagnosis in this particular case was made with:



Figures 4 and 5. *Splinter nail hemorrhages*





Figures 6 and 7. *Janeway lesions – left palmar region*

Table 1.
Paraclinical test results

	Blood test	Value
12.09.2024	Leucocytes/mm^3	6 150
	Erithrocytes/mm^3	4 050 000
	Hemoglobin g/dL	12,9
	Hematocrit %	37,7
	Platelets/mm^3	116 000
	Lymphocytes %	29,1
	Neutrophils %	56,7
	INR	1,18
	PI %	77
	aPTT sec	24,2
	Fibrinogen g/L	5
	CRP mg/dL	6
	ESR mm/h	90
	eGFR ml/min	46,69
	Sodium mmol/L	136,8
	K mmol/L	3,54
	Chloride mmol/L	98,4
	Glucose mg/dL	84
	BUN mg/dL	57
	Creatinine mg/dL	1,53
	Total cholesterol mg/dL	117
	Urinary sediment	Glucosuria Proteinuria Hematuria
	Cantitative uroculture	<1000 UFC/ml

Internal Medicine

Clinical Cases

- SARS COV-2 infection, considering that the patient presented with specific symptoms, but the rapid SARS COV-2 Ag test was negative;
- lobar/interstitial pneumonia by the presence of fever, chills and chest pain, but clinically: absence of cough, and pulmonary auscultation shows physiologic MV, while thoracic xray does not show pathological changes;
- Cardiac myxoma, if we consider fever and syncopal episode, but paraclinically - echocardiography excludes the presence of a tumor.

The positive diagnosis of native anterior mitral valve endocarditis with Streptococcus Gordonii is supported by clinical examination (cardiac stetacustic - systolic murmur grade III/VI in the mitral valve), paraclinical findings (positive blood cultures; inflammatory biological syndrome - VSH, CRP; nitrogen retention syndrome) and imaging (Echocardiography - vegetation at P2 level). Thus, the major Duke diagnostic criteria for infective endocarditis (positive blood cultures, suggestive echocardiographic examination - vegetation) and the minor ones (fever, Janeway lesions) are met. Secondary diagnoses: Essential hypertension grade 3, stage 2, with very high additional risk. NYHA class II chronic heart failure with preserved LVEF. Chronic ischemic heart disease. Moderate-severe mitral insufficiency. Moderate tricuspid insufficiency. Mild aortic insufficiency. Type 2 diabetes mellitus treated with oral antidiabetics. Dyslipidemia. Ankylosing spondylitis on biologic therapy. Gout. Normochromic normocytic anemia. Thrombocytopenia. Hypokalemia. Nitrogen retention syndrome. Prophylactic anticoagulation.

Treatment was performed according to the ESC Guidelines for the Management of Infective Endocarditis - European Society of Cardiology (ESC) Working Group for the Management of Infective Endocarditis Agreed by: the European Association of Nuclear Medicine (EANM) and the European Association of CardioThoracic Surgery (EACTS), including Endocarditis Team Members (both core members and adjunctive specialists)[8], as it follows:

A.
Hospitalization, hygienic-dietary treatment, bed rest, diet according to the recommendations of the attending diabetologist.

B.
Continue etiologic treatment with Gentamycin 160 mg/day, adjusted according to Creatinine Clearance, for a duration of up to 14 days. De-escalate antibiotherapy from Ceftriaxone (administered for 9 days)

to Ampicillin 9 g/day for 20 days, resulting in a total treatment duration of 4 weeks, following the identification of S. gordonii. Additionally, provide symptomatic treatment and hydro-electrolyte rebalancing.

C.

Pathogenic treatment - continue cardiologist's recommendations: Carvedilol 6,25mg 1/2cp D + 1/2cp S, Lercanidipine 10mg 1cp/day, Perindopril 5mg 1cp/day, Dapagliflozin 10mg 1cp/day, Furosemide 40mg 1cp/day, Enoxaparin (Clexane) 0,4ml/day, Clopidogrel 75mg 1cp/day, Rosuvastatin 40mg 1cp/day.

Complications: During hospitalization, the patient exhibits macroscopic hematuria and significant thrombocytopenia(93,000/mm^3). Consequently, the decision is made to discontinue Clopidogrel and to maintain anticoagulant therapy with Clexane at a prophylactic dose of 0.4 ml/day, with instructions to continuously monitor the hemoleukogram and careful clinical observation for additional specific signs and symptoms.

The patient's evolution was clinically favorable after 4 weeks of treatment, and remained stable from an imaging perspective (control echocardiography at discharge showed no change in the additive image compared to the previous evaluation), with no complaints, absence of fever, and remission of the biological inflammatory syndrome.

The discharge recommendations included avoiding cold, damp environments, crowded places, and contact with ill individuals; managing underlying conditions; adhering to the cardiologist's directives, specifically maintaining the treatment regimen with the note that anticoagulant therapy (Clexane) will cease post-discharge; scheduling a follow-up cardiology consultation in approximately 14 days; returning to the Infectious Diseases department following the cardiology appointment; and receiving pneumococcal and seasonal influenza vaccinations.

The case is notable in that the patient, who is immunosuppressed (a diabetic with ankylosing spondylitis receiving biological treatment with Etanercept), presented with insidious onset of endocarditis. However, the disease course was favorable due to timely suspicion of the diagnosis and prompt initiation of antibiotic therapy, thereby eliminating the necessity for valve prosthesis.

Discussions

The Streptococcus viridans group (VGS) consists of low virulence bacteria most commonly found in the oral cavity, upper respiratory tract and gastrointestinal tract[9]. The group is classified into 6 major subgroups: the S. mutans group, the S. mitis group, the S. anginosus group, the S. salivarius group, the S. bovis group, the S. sanguinis group[9, 10]. The S. sanguinis group includes the bacteria: S. sanguinis, S. parasanguinis and S. gordonii and rarely causes invasive infections, especially infectious endocarditis[10].

S. gordonii is a rare cause of infective Endocarditis, not often reported in the literature. S. gordonii are Gram-positive cocci, which play an important role in alkalizing the oral cavity and producing a protective biofilm[10]. Once in the bloodstream, S. gordonii appears to have the pathogenic virulence factors for the development of infective endocarditis. The cell wall of S. gordonii contains a serine-rich glycoprotein,

GspB, which mediates binding to human platelets. After adhesion to platelets, the newly formed complex attaches to the fibronectin-rich extracellular matrix of heart valves and subsequently forms valve vegetations[11].

The exposed patient is known to have dental problems, with partial edentulism, so the presence of root debris can be considered the main risk factor and the main entry site of Streptococcus gordonii. In this patient, infective endocarditis had an insidious onset with renal involvement. Although the patient has a personal pathologic history of gout and diabetes mellitus, it is conceivable that the renal involvement occurs in this context, but during hospitalization, although glycemic values were constantly elevated, uric acid values were within normal limits.

Several prognostic factors have been identified in this case. Favorable indicators include an early diagnosis and initiation of treatment, as well as the isolation of *Streptococcus gordonii*, which is sensitive to beta-lactam antibiotics.

However, the patient's prognosis is significantly influenced by a series of unfavorable factors: an immunocompromised status due to underlying diabetes and ongoing biological therapy, the presence of severe azotemia indicating advanced renal dysfunction, thrombocytopenia accompanied by macroscopic hematuria, and an echocardiographic finding of an atrial wall mass at the level of the mitral valve annulus (VMA) with an aneurysmal appearance.

Conclusions

This case highlights the complexity of managing infective endocarditis in immunocompromised patients, particularly those with multiple comorbidities and atypical echocardiographic findings. Despite the presence of favorable prognostic factors — such as early diagnosis and a beta-lactam-sensitive *Streptococcus gordonii* isolate — the patient's clinical course was complicated by severe renal impairment, hematologic abnormalities, and an unusual intracardiac mass suggestive of an aneurysmal lesion.

The case underscores the importance of individualized, multidisciplinary care and contributes to the growing body of evidence emphasizing the prognostic impact of host immune status and imaging findings in infective endocarditis. Documenting such cases is essential for refining diagnostic criteria, guiding therapeutic decisions, and improving outcomes in high-risk patient populations.

Bibliography

1. Egidia Gabriela Miftode. Endocarditele infecțioase. "Boli infecțioase" – Editura "Gr. T. Popa" U.M.F. Iași 2021

2. Li M, Kim JB, Sastry BKS, Chen M. Infective endocarditis. Lancet. 2024 Jul 27;404(10450):377-392. doi: 10.1016/S0140-6736(24)01098-5.

3. Mulliken JS, Bainbridge ED. Infective Endocarditis. Med Clin North Am. 2025 May;109(3):667-681. doi: 10.1016/j.mcna.2024.12.009.

4. Chambers HF, Bayer AS. Native-Valve Infective Endocarditis. N Engl J Med. 2020 Aug 6;383(6):567-576. doi: 10.1056/NEJMcp2000400.

5. López J, Olmos C, Fernández-Hidalgo N. New developments in infective endocarditis. Rev Esp Cardiol (Engl Ed). 2024 Sep;77(9):779-787. doi: 10.1016/j.rec. 2024.03.016.

6. Lisi M, Flamigni F, Russo M et al. Incidence and mortality of infective endocarditis in the last decade: a single center study. J Cardiovasc Med (Hagerstown). 2023 Feb 1;24(2):105-112. doi: 10.2459/JCM. 000000 0000001410.

7. Abdelgawad H, Azab S, Abdel-Hay MA, Almaghraby A. Clinical features and outcomes of infective endocarditis: a single-centre experience. Cardiovasc J Afr. 2023 May-Jun 23;34(2):82-88. doi: 10.5830/CVJA-2022-027.

8. Delgado V, Ajmone Marsan N, de Waha S et al; ESC Scientific Document Group. 2023 ESC Guidelines for the management of endocarditis. Eur Heart J. 2023 Oct 14;44(39):3948-4042. doi: 10.1093/eurheartj/ehad193.

9. Dadon Z, Cohen A, Szterenlicht YM et al. Spondylodiskitis and endocarditis due to Streptococcus gordonii. Ann Clin Microbiol Antimicrob. 2017 Oct 4;16(1):68. doi: 10.1186/s12941-017-0243-8.

10. Abranches J, Zeng L, Kajfasz JK et al. Biology of Oral Streptococci. Microbiol Spectr. 2018 Oct;6(5): 10.1128/microbiolspec.gpp3-0042-2018. doi: 10.1128/microbiolspec.GPP3-0042-2018.

11. Xiong YQ, Bensing BA, Bayer AS, Chambers HF, Sullam PM. Role of the serine-rich surface glycoprotein GspB of Streptococcus gordonii in the pathogenesis of infective endocarditis. Microb Pathog. 2008 Oct;45(4):297-301. doi: 10.1016/j.micpath. 2008.06.004.