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(CASE REPORT)

Right-Sided Infective Endocarditis: When Should It Be Suspected? *A Case Series*

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Abstract

There has been a notable increase in hospitalizations due to Right-Sided Infective Endocarditis (RSIE), affecting the endocardium and right chambers. With a rising incidence, ranging between 5-10%, and an approximate mortality rate of 6%, (RSIE) poses specific challenges in terms of diagnosis and treatment. Its atypical clinical presentation compared to left endocarditis and its therapeutic particularities underline the need for greater understanding. In this series of cases, we proposed to highlight crucial data to suspect and guide the diagnosis and timely treatment to avoid complications.

Keywords: Bacterial endocarditis; Tricuspid valve; *Staphylococcus aureus*; Bacteremia

1. Introduction

Infective endocarditis (IE) is a disease that poses a significant challenge to global public health. It primarily involves the endocardial surface and cardiac valves (prosthetic or native), as well as implanted cardiac devices. The estimated global annual incidence in 2019 was 13.8 cases per 100,000 inhabitants [1,2]. Regarding right-sided infective endocarditis (RSIE), its incidence is considerably lower than that of left-sided involvement, accounting for approximately 5–10% of all IE cases [3]. Several risk factors have been associated with RSIE, with intravenous drug use being the most prominent, representing up to 89% of cases [4]. Other relevant risk factors include the presence of cardiac implantable electronic devices [5,6], intravascular devices such as hemodialysis catheters, and underlying right-sided structural cardiac abnormalities. Additional comorbid conditions associated with RSIE include human immunodeficiency virus (HIV) infection and hepatitis C virus infection [7,8]. Reported mortality rates reach approximately 6%, and complications have been described in up to 61% of cases [9].

In this case series, we describe two patients with RSIE involving the native tricuspid valve and well-defined risk factors, including the use of intravascular devices—specifically hemodialysis catheters—and intravenous drug use. In both cases, comorbidities such as hepatitis C virus infection and pulmonary tuberculosis were identified, along with complications inherent to RSIE. Furthermore, we discuss various diagnostic and therapeutic approaches for multidisciplinary teams, as well as the management challenges encountered in these clinical scenarios.

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2. Case presentation

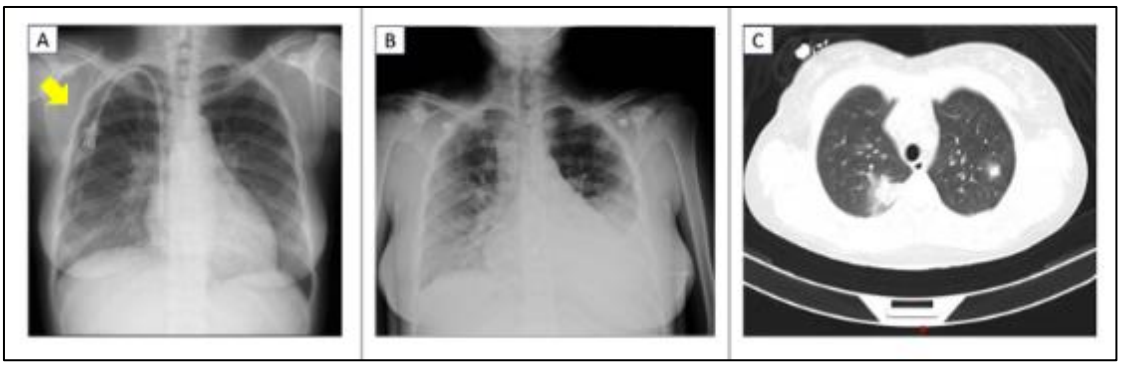
2.1. Case 1

A 34-year-old female with a medical history of stage 5 chronic kidney disease on intermittent hemodialysis presented with a 6-day history of fever, cough, and progressive dyspnea. She had previously received outpatient treatment with an oral macrolide without clinical improvement. On admission, vital signs revealed tachycardia (111 bpm), tachypnea (29 breaths/min), and normoxemia. Physical examination showed scleral icterus, grade II jugular venous distention at 45°, a positive hepatojugular reflux, grade II pretibial pitting edema, and serous discharge from a right subclavian tunneled catheter without local inflammatory signs.

Initial laboratory studies demonstrated leukocytosis (15,500 cells/mm^3) with neutrophilia (85%), moderate thrombocytopenia (85,000 cells/mm^3), and grade 2 anemia according to the WHO classification (Hb: 9.87 g/dL). Renal function tests revealed significant impairment (creatinine: 14.3 mg/dL; blood urea nitrogen: 43 mg/dL). Serum electrolytes showed mild hyponatremia, with otherwise unremarkable findings (sodium 130 mEq/L, chloride 99 mEq/L, potassium 5.3 mEq/L). Hyperbilirubinemia was also noted (total bilirubin 1.8 mg/dL, direct bilirubin 1.4 mg/dL, indirect bilirubin 0.4 mg/dL), along with markedly elevated acute-phase reactants (C-reactive protein 31 mg/dL). Chest radiography revealed no parenchymal abnormalities and confirmed the presence of a right subclavian catheter (see Figure 1-A).

Based on these findings, a search for an infectious source was initiated, including aerobic and anaerobic blood cultures as well as catheter-drawn cultures. Endemic tropical infections relevant to the regional epidemiological context were ruled out, including dengue (negative NS1 antigen), malaria (negative thick blood smear), and leptospirosis (negative IgM and IgG). During observation, the patient developed worsening dyspnea requiring high-flow supplemental oxygen, persistent fever, and a Sequential Organ Failure Assessment (SOFA) score of 4. She was subsequently transferred to the intensive care unit with a diagnosis of sepsis, presumed secondary to bacteremia originating from an intravascular device infection; concomitant pneumonia could not be excluded.

Empirical antimicrobial therapy with a third-generation cephalosporin combined with a glycopeptide was initiated, and removal of the tunneled catheter was indicated.



(A) Chest radiograph showing no pleuroparenchymal abnormalities and a right subclavian catheter in situ. (B) Follow-up chest radiograph obtained after removal of the tunneled catheter, demonstrating bilateral interstitial and patchy alveolar infiltrates. (C) Non-contrast chest computed tomography revealing interstitial–alveolar opacities with a tendency toward consolidation in the posterior segment of the right upper lobe and the posterior basal segment of the left lower lobe, consistent with a pneumonic pattern.

Figure 1 Radiological course, case 1

Further evaluation included a transthoracic echocardiogram, which demonstrated dilated cardiomyopathy with depressed systolic function (LVEF 32%), segmental wall motion abnormalities with diffuse generalized hypokinesia, and mild mitral and tricuspid regurgitation. Non-contrast chest computed tomography revealed a right basal consolidative focus and a moderate left pleural effusion (see Figures 1-B and 1-C). Final blood culture results reported methicillin-resistant *Staphylococcus aureus* (MRSA), mecA gene positive with an MLSb phenotype (see Table 1).

The Infectious Diseases service was consulted and recommended transesophageal echocardiography due to a high risk of endocarditis, with a VIRSTA score of 6. Findings included an interatrial septum with a tunnel-type patent foramen ovale and a left-to-right shunt at rest. At the level of the tricuspid valve, a homogeneous, highly mobile, cylindrical mass measuring 15 × 2 mm was observed, suggestive of vegetation. Duke criteria were applied, fulfilling two major criteria

and establishing a definitive diagnosis of right-sided native valve (tricuspid) infective endocarditis secondary to bacteremia from an intravascular device infection.

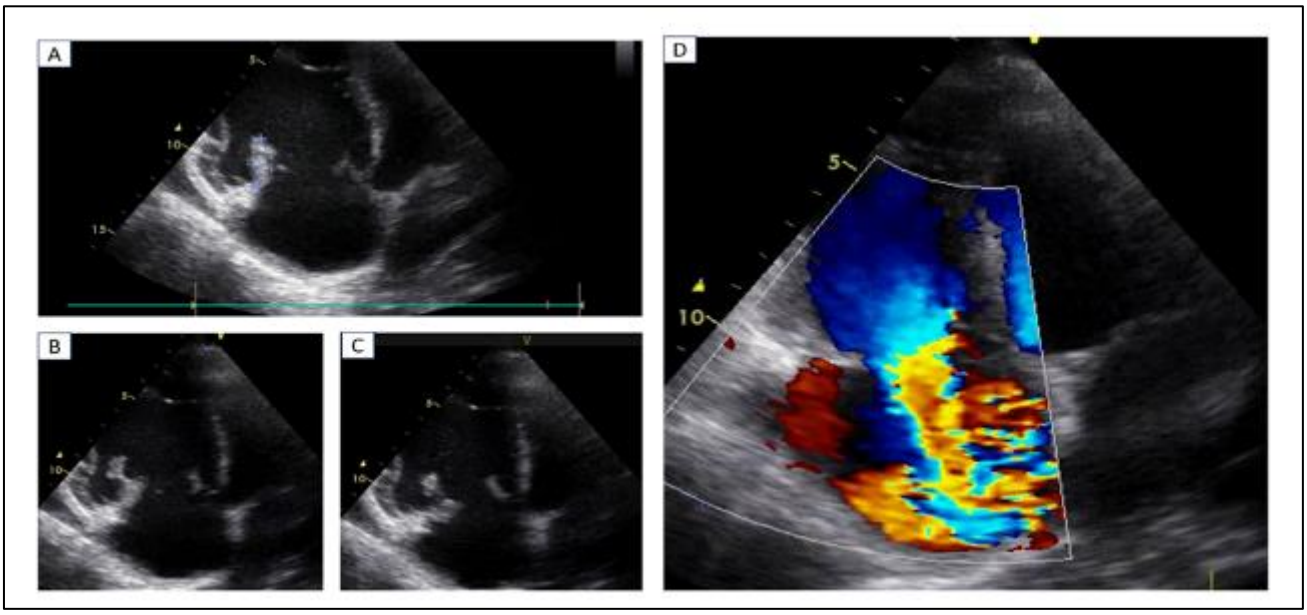
During hospitalization, the patient was evaluated by the cardiovascular surgery team, which determined that there were no indications for surgical management. Therefore, a 6-week course of targeted antimicrobial therapy based on culture and susceptibility results was completed successfully. Follow-up echocardiographic assessment at the end of therapy demonstrated a reduction in vegetation size and satisfactory clinical recovery.

2.2. Case 2

A 40-year-old male with a history of heroin use disorder on methadone maintenance therapy and pulmonary tuberculosis undergoing intensive-phase anti-tuberculosis treatment presented to the emergency department with a 7-day history of dull abdominal pain in the right upper quadrant and epigastrium, rated 7/10 on the numeric pain scale. Symptoms were associated with watery diarrhea without mucus or blood, four episodes of food-content emesis, and asthenia. In the 12 hours prior to admission, he developed dyspnea.

On initial medical evaluation, the patient appeared somnolent, tachycardic, and tachypneic, requiring supplemental oxygen via nasal cannula at 3 L/min. Physical examination revealed scleral icterus, dry oral mucosa, cavitated dental caries affecting eight teeth, bilateral crackles on lung auscultation, intercostal retractions, and right upper quadrant tenderness with a positive Murphy's sign, without signs of peritoneal irritation.

He was transferred to the intensive care unit with a diagnosis of sepsis of probable abdominal origin, a SOFA score of 7, and a high risk of respiratory failure. Admission laboratory studies showed leukocytosis (29,880 cells/mm^3) with left shift (88%), mild thrombocytopenia (134,100 cells/mm^3), and WHO grade III anemia (Hb: 6.95 g/dL). Renal function tests revealed elevated nitrogenous waste products (creatinine: 3.28 mg/dL; blood urea nitrogen: 62 mg/dL). Acute-phase reactants were markedly elevated (CRP: 15.59 mg/dL; ferritin >2000 ng/mL). Electrolyte analysis showed moderate hyponatremia (128.9 mEq/L). Hyperbilirubinemia was present (total bilirubin 4.8 mg/dL), predominantly direct (3.96 mg/dL). Serum amylase and lipase levels were within normal limits.



(A) A 22 × 8 mm vegetation located on the leaflet adjacent to the right ventricular free wall. (B, C, D) Disruption of valvular architecture due to rupture of the septal leaflet, along with vegetation on the leaflet adjacent to the right ventricular free wall, resulting in a large coaptation defect and severe tricuspid regurgitation.

Figure 2 Transthoracic echocardiogram, case 2

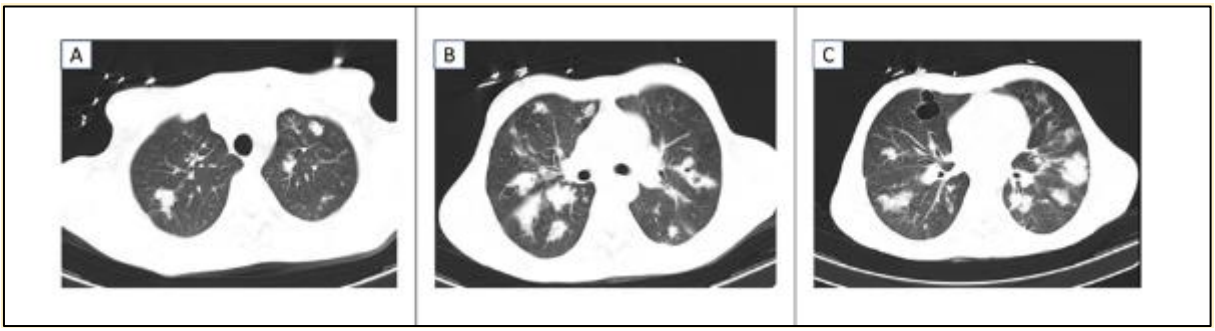
Given the suspicion of sepsis, empirical antimicrobial coverage with a ureidopenicillin and a glycopeptide was initiated. Blood cultures, urine culture, stool culture, and bronchial secretion cultures were obtained, and acquired immunosuppression was ruled out. Urine culture results were negative, HIV testing was negative, and SARS-CoV-2 antigen testing was negative. Peripheral blood cultures returned positive, with isolation of methicillin-sensitive *Staphylococcus aureus* (MSSA) (see Table 1), consistent with secondary bloodstream infection due to bacteremia.

An Infectious Diseases consultation was requested, and transthoracic echocardiography was performed. Antimicrobial therapy was subsequently de-escalated to oxacillin. Transthoracic echocardiographic findings demonstrated a tricuspid valve with annular dilation, thickened leaflets, and vegetations >20 mm involving the septal leaflet (15 × 5 mm) and the free-wall leaflet (21 × 5 mm), associated with severe tricuspid regurgitation. Duke criteria were applied, fulfilling two major criteria and one minor criterion, establishing a definitive diagnosis of right-sided native valve (tricuspid) infective endocarditis.

Additionally, evidence of embolic phenomena to the pulmonary parenchyma was identified on non-contrast chest computed tomography, which showed patchy alveolar opacities distributed along the pulmonary vasculature (see Figure 3), supporting an indication for cardiovascular surgical management. Embolic involvement of the central nervous system was ruled out by non-contrast cranial computed tomography, which was within normal limits.

Cardiovascular surgery initially deferred surgical intervention due to the patient's clinical condition, particularly moderate malnutrition, recommending completion of a 4–6-week course of antimicrobial therapy. A follow-up inpatient transthoracic echocardiogram performed 14 days after initiation of antibiotic therapy demonstrated a reduction in vegetation size on the septal leaflet (9 × 3 mm) and the free-wall leaflet (9 × 6 mm). The patient showed progressive clinical improvement, including successful weaning from supplemental oxygen and stabilization of vital signs, and completed the antimicrobial regimen.

A multidisciplinary medical board subsequently evaluated the case to determine surgical timing. It was concluded that valvuloplasty should be deferred until improvement of nutritional status in the outpatient setting. After completion of therapy and performance of follow-up transthoracic echocardiography and control fiberoptic bronchoscopy, the patient was discharged with outpatient follow-up by Cardiology and Cardiovascular Surgery.



(A, B, C) Non-contrast chest computed tomography demonstrating airspace opacities with air bronchograms, accompanied by multiple small bilateral pulmonary nodules, some of which are cavitated, findings suggestive of septic emboli.

Figure 3 Pulmonary emboli

Table 1 Antibiogram report for cases 1 and 2.

Case	**Case 1**		**Case 2**	
Sample	**Blood cultures X2**	**Catheter-drawn culture**	**Blood cultures X2**	
Organism	*Staphylococcus aureus*		*Staphylococcus aureus*	
Resistance markers	*MRSA mecA gene and inducible MLSb phenotype*		Methicillin-sensitive	
Antibiogram	**MIC (µg/mL)**	**Resistance profile**	**MIC (µg/mL)**	**Resistance profile**
Oxacillin	>2	R	<= 0,5	S
Clindamycin		I	<= 0,5	S
Erythromycin	>4	R	<= 0,25	S
Gentamicin	<= 2	S	<= 2	S
Vancomycin	1	S	1	S
TMP/SMX	<= 0,5/9,5	S		

Ciprofloxacin	<= 0,5	S	>2	R
Penicilin	>1	R	0,25	R
Daptomycin	<= 1	S	<= 1	S
Linezolid	<= 1	S		
Minocycline	<= 1	S	<= 1	S
Ceftaroline	<= 0,5	S	<= 0,5	S

MRSA: Methicillin-resistant *Staphylococcus aureus*; *MLSb: Macrolides, lincosamides, and streptogramin B; *TMP/SMX: Trimethoprim/sulfamethoxazole; *MIC: Minimum inhibitory concentration; *S: Susceptible; *R: Resistant

3. Discussion

Right-sided endocarditis was first described in 1950 by Dr. Hugh Husse [10]. Despite the limited availability of academic data on right-sided infective endocarditis (RSIE), a concerning increase in its incidence has been observed, largely attributed to the rising prevalence of risk factors such as the use of vascular devices, intravenous drug use, and intracardiac devices [1,10]. Several reasons have been proposed to explain why right-sided endocarditis is less common than left-sided disease, including the lower prevalence of valvular pathology involving the tricuspid and pulmonary valves, the relative rarity of congenital malformations at this level, specific vascular and endothelial properties, and significantly lower pressure gradients, flow velocities, and wall tension across right-sided cardiac valves and chambers, as well as the lower oxygen content of venous blood [2].

The tricuspid valve is the most commonly affected structure, followed by the pulmonary valve [12]. Both are infrequently reported in the literature, particularly in cases involving native valves, which account for only approximately 10% of reported cases [3,6]. RSIE is associated with intravenous drug use in up to 90% of cases, while the presence of a hemodialysis catheter confers an approximately 18-fold higher risk compared with the general population [6,13]. Furthermore, underlying right-sided structural cardiac abnormalities also play a significant role [7].

In our clinical cases, a relevant risk factor was identified in patient 1, who had a tunneled hemodialysis catheter and a patent foramen ovale. In patient 2, the main risk factors included intravenous drug use and the presence of cavitated dental caries. These findings underscore the importance of considering these specific risk factors when evaluating and managing suspected right-sided infective endocarditis.

Right-sided endocarditis is more common in younger patients [9]. Regarding clinical presentation, fever is the most frequent symptom, occurring in approximately 90% of cases, followed by respiratory symptoms such as dyspnea, pleuritic chest pain, cough, and hemoptysis, which are present in up to 53% of patients [14,15]. On physical examination, cardiac murmurs are not commonly detected, and systemic embolic or immunologic phenomena are generally absent unless paradoxical embolism occurs in the presence of a patent foramen ovale [16,17]. Other clinical findings are related to common complications, particularly septic pulmonary embolism, which occurs in up to 61% of cases [2,9] (see Figure 4).

Right-sided endocarditis requires a high index of suspicion. Key features that should prompt consideration include persistent fever accompanied by respiratory symptoms in patients with the aforementioned risk factors. Initial evaluation should include blood cultures (aerobic, anaerobic, and from intravascular devices) and first-line imaging with echocardiography [1]. Reported sensitivity for vegetation detection varies according to the type of echocardiographic modality used. For native valves, transthoracic echocardiography has a sensitivity of approximately 70%, whereas in prosthetic valves it is around 50%. In contrast, transesophageal echocardiography demonstrates higher sensitivity, reaching 96% for native valves and 92% for prosthetic valves [18]. Additional complementary studies, such as chest radiography and electrocardiography, are also important for evaluating potential complications [6].

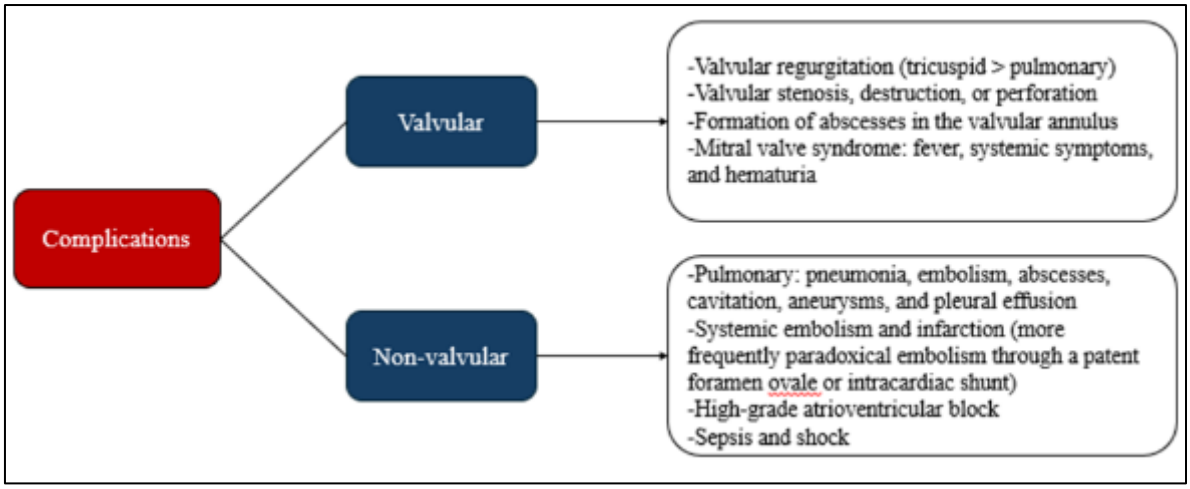


Figure 4 Valvular and non-valvular complications of right-sided endocarditis

Regarding microbiological isolation, the principal pathogen is *Staphylococcus aureus* (60–90% of cases), followed by coagulase-negative Staphylococcus, Streptococcus pneumoniae, Pseudomonas aeruginosa, Gram-negative bacilli, and less frequently, fungal infections [2,6]. Methicillin-resistant strains (MRSA) are of particular concern due to their more aggressive clinical course. More recently, vancomycin-resistant *Staphylococcus aureus* (VRSA) and vancomycin-intermediate *Staphylococcus aureus* (VISA) strains have also been reported, increasing the risk of therapeutic failure and prompting the need for alternative treatment strategies [6].

To define the therapeutic strategy for right-sided valvular endocarditis, it is essential to consider prompt initiation of empirical antibiotic therapy followed by targeted therapy once blood culture results are available. All permanent intravascular devices should be removed, and patients should be individually assessed to determine whether they would benefit from surgical management [1].

Empirical antibiotic therapy depends on microbiological suspicion, prior use of intravascular devices, the valve involved, and a history of intravenous drug use [19,20]. Immediate initiation of empirical antibiotic therapy after obtaining blood cultures should take into account the presence of hemodynamic instability. In clinically stable patients, antimicrobial therapy may be initiated once culture results are obtained. Empirical treatment for MRSA consists of vancomycin; however, daptomycin at doses of 8–10 mg/kg intravenously once daily represents an alternative option [6,20]. Once microbiological results are available, it is essential to determine whether the infection is due to methicillin-sensitive *Staphylococcus aureus* (MSSA); if so, antibiotic therapy should be de-escalated to beta-lactam agents such as oxacillin or cefazolin [1,21]. Another key objective in pharmacological management is early microbiological response to treatment; therefore, obtaining blood cultures every 48 hours from initiation of therapy until clearance of bacteremia is of critical importance [1,6].

In Case 1, antibiotic therapy with a third-generation cephalosporin and a glycopeptide was initiated immediately after blood cultures were obtained, considering the need for supplemental oxygen, clinical signs such as tachypnea and tachycardia, and the high risk of MRSA infection. During hospitalization, cultures returned positive for MRSA, and vancomycin therapy was continued for 4–6 weeks. In Case 2, following positive blood culture results for MSSA and transthoracic echocardiographic findings consistent with tricuspid valve endocarditis, the initial empirical antibiotic regimen was discontinued and targeted therapy with oxacillin was initiated for 4 weeks.

4. Conclusion

The increasing incidence of right-sided infective endocarditis due to the growing use of intravenous drugs and vascular catheters, combined with low clinical suspicion resulting from its often atypical presentation and the ongoing challenge of antimicrobial resistance, underscores the need for clinicians to maintain a lower threshold of suspicion for RSIE—particularly in patients presenting with fever of unknown origin and/or subsequent development of respiratory symptoms. It is also important to recognize that an initial transthoracic echocardiogram that does not detect vegetations does not exclude the diagnosis of endocarditis. Therefore, familiarity with available risk scores for specific pathogens is essential to estimate risk and determine the need for repeat imaging. Identification of patients with risk factors, implementation of diagnostic algorithms, knowledge of local epidemiology, and strengthening of interdisciplinary

teams within healthcare institutions are proposed as key strategies for timely and effective management of this condition.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Delgado V, Ajmone Marsan N, De Waha S, Bonaros N, Brida M, Burri H, et al. 2023 ESC Guidelines for the management of endocarditis. European Heart Journal. 14 de octubre de 2023;44(39):3948-4042.

[2] Shmueli H, Thomas F, Flint N, Setia G, Janjic A, Siegel RJ. Right-Sided Infective Endocarditis 2020: Challenges and Updates in Diagnosis and Treatment. JAHA. 4 de agosto de 2020;9(15):e017293.

[3] Akinosoglou K, Apostolakis E, Marangos M, Pasvol G. Native valve right sided infective endocarditis. European Journal of Internal Medicine. septiembre de 2013;24(6):510-9.

[4] Hubers SA, DeSimone DC, Gersh BJ, Anavekar NS. Infective Endocarditis: A Contemporary Review. Mayo Clinic Proceedings. mayo de 2020;95(5):982-97.

[5] Blomström-Lundqvist C, Traykov V, Erba PA, Burri H, Nielsen JC, Bongiorni MG, et al. European Heart Rhythm Association (EHRA) international consensus document on how to prevent, diagnose, and treat cardiac implantable electronic device infections—endorsed by the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), the Latin American Heart Rhythm Society (LAHRS), International Society for Cardiovascular Infectious Diseases (ISCVID), and the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal. 1 de junio de 2020;41(21):2012-32.

[6] Chahoud J, Sharif Yakan A, Saad H, Kanj SS. Right-Sided Infective Endocarditis and Pulmonary Infiltrates: An Update. Cardiology in Review. septiembre de 2016;24(5):230-7.

[7] Rushani D, Kaufman JS, Ionescu-Ittu R, Mackie AS, Pilote L, Therrien J, et al. Infective Endocarditis in Children With Congenital Heart Disease: Cumulative Incidence and Predictors. Circulation. 24 de septiembre de 2013;128(13):1412-9.

[8] Baniya A, Duwadee P, K.C. S, Shahi M, Khan A, Rijal Y, et al. Infective endocarditis of tricuspid valve following septic abortion: a case report. Annals of Medicine & Surgery. diciembre de 2023;85(12):6262-5.

[9] Lassen H, Nielsen SL, Gill SUA, Johansen IS. The epidemiology of infective endocarditis with focus on non-device related right-sided infective endocarditis: A retrospective register-based study in the region of Southern Denmark. International Journal of Infectious Diseases. junio de 2020;95:224-30.

[10] Hussey HH. The Pathogenesis of Septic Pulmonary Embolism. Medical Clinics of North America. 1950;34(6):1751-60.

[11] Goud A, Abdelqader A, Dahagam C, Padmanabhan S. Isolated pulmonic valve endocarditis presenting as neck pain. Journal of Community Hospital Internal Medicine Perspectives. enero de 2015;5(6):29647.

[12] Hussain ST, Witten J, Shrestha NK, Blackstone EH, Pettersson GB. Tricuspid valve endocarditis. Ann Cardiothorac Surg. mayo de 2017;6(3):255-61.

[13] Pericàs JM, Llopis J, Jiménez-Exposito MJ, Kourany WM, Almirante B, Carosi G, et al. Infective Endocarditis in Patients on Chronic Hemodialysis. Journal of the American College of Cardiology. abril de 2021;77(13):1629-40.

[14] 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. European Heart Journal. 14 de octubre de 2019;40(39):3222-32.

[15] Yuan S-M. Right-sided infective endocarditis: recent epidemiologic changes. Int J Clin Exp Med. 2014;7(1):199–218.

[16] Pais JP, Sousa M, Mota R, Cambão AR, Nascimento A. Right-Side Endocarditis: A Typical Presentation in an Atypical Patient. Cureus . 19 de octubre de 2021 [citado 26 de diciembre de 2023]; Disponible en: https://www.cureus.com/articles/74144-right-side-endocarditis-a-typical-presentation-in-an-atypical-patient

[17] Lee MR, Chang SA, Choi SH, Lee GY, Kim EK, Peck KR, et al. Clinical Features of Right-Sided Infective Endocarditis Occurring in Non-Drug Users. J Korean Med Sci. 2014;29(6):776.

[18] Dingen H, Urheim S, Jordal S, Saeed S. Echocardiographic assessment in infective endocarditis: Transthoracic or transesophageal echocardiography, or both? Current Problems in Cardiology. enero de 2024;49(1):102151.

[19] Cuervo G, Escrihuela-Vidal F, Gudiol C, Carratalà J. Current Challenges in the Management of Infective Endocarditis. Front Med. 22 de febrero de 2021;8:641243.

[20] Comeaux S, Jamison K, Voeltz M. Contemporary Features and Management of Endocarditis. Diagnostics. 28 de septiembre de 2023;13(19):3086.

[21] Baddour LM, Wilson WR, Bayer AS, Fowler VG, Tleyjeh IM, Rybak MJ, et al. Infective Endocarditis in Adults: Diagnosis, Antimicrobial Therapy, and Management of Complications: A Scientific Statement for Healthcare Professionals From the American Heart Association. Circulation. 13 de octubre de 2015;132(15):1435-86.