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Chapter

Right-Sided Infective Endocarditis

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Abstract

Infective endocarditis (IE) at the right side represents the 5–10% of IE cases. It is more frequent in people with intravenous drug addiction (IVDA); however, there is another population susceptible to this infection; hemodialytic patients, intracardiac devices, and congenital heart diseases are included inside this group. Right-sided infective endocarditis (RSIE) has lower mortality than the left-sided infective endocarditis (LSIE). Common symptoms secondary to right-sided endocarditis are the respiratory symptoms characterized by a cough, hemoptysis, persistent fever, dyspnea, and chest pain. Echocardiography and blood cultures are the first tools to perform the diagnosis. The tricuspid valve is the main anatomical structure affected. Medical treatment with antibiotic therapy resolves the infection majority of the time; the surgical treatment is indicated in some cases, such as right-heart failure due to severe tricuspid valve regurgitation; inability to eliminate bacteremia or organism; resistance to culture-directed antibiotic treatment, within 7 days; and tricuspid valve vegetations >20 mm. RSIE implies a better prognosis than LSIE. Concomitant left-sided IE carries a worse prognosis than right-sided infection alone, due predominantly to its greater likelihood for invasion and abscess formation.

Keywords: infective endocarditis, right-sided infective endocarditis, tricuspid valve, intravenous drug addiction, echocardiography, antibiotic, surgery, hemodialysis, intensive care unit, pulmonary valve

1. Introduction

Infective endocarditis (IE) at the right side of the heart is quite rare; it represents the 5–10% of IE cases. It is seen most frequently in people with intravenous drug addiction; nevertheless, other portions of the population are in high risk of developing this disease such as patients with indwelling catheters, cardiac devices, congenital cardiac pathologies, and immunocompromised diseases [1–3].

The evolution of right-heart IE is much better than the left-side IE with a lower rate of mortality (3–30%) [3]. This pathology is more frequent in people between 20 and 61 years, with a mean age of 38 ± 15 years [4].

Staphylococcus aureus is the predominant organism (60–90% of cases) with the methicillin-resistant strains becoming more prevalent lately [3, 5]. The tricuspid valve is by far the most effective structure (90%) in right-side infective endocarditis (RSIE) [5].

2. Diagnosis

2.1 Clinic manifestations

Common symptoms secondary to right-sided endocarditis are the respiratory symptoms characterized by a cough, hemoptysis, persistent fever, dyspnea, and chest pain [4].

In exceptional circumstances, right-heart failure can arise, generated by the increase in pulmonary pressure, severe tricuspid valve regurgitation, or obstruction of pulmonary circulation through multiple pulmonary emboli [4, 6].

The diagnosis of RSIE is often delayed because the signs and symptoms are relatively different concerning the LSIE clinical setting; the Duke's modified criteria do not have value in the RSIE. The low incidence of RSIE also plays an essential factor in the underdiagnosis of this disease.

There are reports in which the 76% of the patients had gotten an antibacterial treatment before the endocarditis's diagnosis because they developed some signs and symptoms that were misunderstood as a febrile syndrome or pneumonia [4].

An acute beginning of the disease is seen at the majority of the patients; only a few cases have been reported with chronic symptoms (more of 2 months) [4].

It is frequent that right-side vegetations dislodge microemboli to the pulmonary region. The pulmonary embolism (PE) can induce pulmonary infarction, abscesses, pneumothoraxes, and purulent pulmonary effusions.

Persistent fever associated with pulmonary events, anemia, and microscopic hematuria, the so-called "tricuspid syndrome," is the sign of clinical alert for tricuspid valve IE [3, 4, 7].

Revilla et al. found 24% of their patients with this syndrome, and the other 65% had at least two of the three signs [4].

2.2 Complementary exams

2.2.1 Laboratory

Nowadays it is routinary to order blood tests for any patient admitted at the hospital, and it is reasonably used if the suspicion of infection is thought. Some findings such as high titers of white blood cells, procalcitonin, and C-reactive protein can support the infection diagnosis; nevertheless, these variables are not used as criteria to diagnose infective endocarditis [5, 8].

The anemia has been described as part of the tricuspid syndrome, so the values of hemoglobin and hematocrit below the normal range can be found in the blood test, which probably will reveal a normocytic, normochromic anemia patron [3, 4, 7].

The urine test can show microhematuria which also is part of the tricuspid syndrome.

2.2.2 Cultures

Right-sided endocarditis in IVDA is commonly caused by *S. aureus* and *Pseudomonas aeruginosa*, and other Gram-negative organisms, fungi, streptococci, and enterococci have also been found [4, 6].

In the majority of patients, the microorganism can be identified through blood cultures if they are adequately collected. The 2015 ESC endocarditis guidelines recommend a technique of recollection minutely sterile of at least three sets of samples with an interval of 30 minutes; each sample must contain 10 ml of blood

and should be incubated in both aerobic and anaerobic atmospheres. Another crucial aspect is the recollection of samples from a peripheral vein instead of central venous catheter due to the risk of contamination and wrong interpretation [5].

Occasionally, the blood cultures can be negative by different reasons, especially if an antimicrobial therapy was established before the acquisition of the samples. The blood cultures usually become negatives after 48 hours from the beginning of antibiotics [4].

2.2.3 Image

Currently, the diagnosis of IE requires the finding of an infective process inside the heart, reason why the imaging techniques are valuable to diagnose or discard IE. The echocardiography is the most important and more used tool to diagnose, manage, and monitor patients with IE [5].

However, other imaging methods have been developed in the last decades, allowing us to back the diagnosis of IE when the echography is not entirely clear in some cases (**Table 1**).

2.2.3.1 Radiography

It can be quite normal or shows a variety of findings, such as cardiomegaly, pulmonary septic emboli, or pleural effusion [4].

2.2.3.2 Echocardiography

The benefits that the echocardiography brought to the cardiology area are well-known, and they can help us to detect anomalies related to IE. It is the gold standard imaging test for IE, becoming one of the first steps that we must do if IE is suspected [3, 9].

The same as the LSIE, the transthoracic echocardiography (TTE) is the first modality recommended to perform if RSIE is suspected. The sensitivity of TTE to detect vegetations is roughly 75% and its specificity over 90%. When the hunch of IE is high, but the TTE is negative, the transesophageal echocardiography (TOE) must be used because its sensitivity is higher than TTE, approximately 96%. Some experts indeed recommend TOE even if the TTE is positive for IE; nevertheless, it does not apply for RSIE in which an explicit finding of IE is enough for the diagnosis [5, 9].

Chest radiography	Echocardiography	Computed Tomography	Nuclear imaging
Cardiomegaly	Vegetations	Abscess	Cardiac enhancement
Pulmonary septic emboli	Abscess	Pseudoaneurysm	Septic emboli
Pleural effusion	Prosthetic dehiscence	Fistula	

Table 1.
Imaging technique findings in the right-sided infective endocarditis.

The 2015 ESC guidelines also suggest the use of TOE when the suspicion of IE is present in patients with a prosthetic heart valve and intracardiac device [5].

There are some "typical lesions" of IE that we can detect in the echocardiography, such as vegetations, abscess, pseudoaneurysm, valve aneurysm, perforation, fistula, and dehiscence of the prosthetic valve, being the vegetation of the landmark lesion of this disease (**Figure 1**) [5, 9].

Occasionally, parts of the vegetations can be visualized floating in the right ventricle or entrapped in the subvalvular apparatus. TTE usually allows assessment of tricuspid valve involvement because of the valve's anterior location and large natural vegetations. TOE imaging is more sensitive to detect vegetations than TTE imaging, especially in the case of abscesses, and associated left-sided involvement [6].

2.2.3.3 Computed tomography (CT)

Cardiac computed tomography (CCT) can improve the diagnosis of IE when abscesses and pseudoaneurysm are present, due to its higher sensitivity (approximately 81%) in comparison with TTE and TOE (roughly 63%). The combination of echocardiography and CCT to diagnose abscess/pseudoaneurysm reaches 100% sensitivity. In pulmonary/right-sided endocarditis, CT may reveal concomitant pulmonary disease, including abscesses and infarcts [5, 10].

2.2.3.4 Magnetic resonance (MR)

The use of MR in the IE setting is focused on the diagnosis of cerebrovascular events related to IE. This imaging modality has better sensitivity than CT to detect brain hemorrhage and infectious intracranial aneurysms (IIAs) [5, 11].

2.2.3.5 Nuclear imaging

The incorporation of positron-emission tomography (PET) in the modified Duke's criteria is addressed to enhance the IE diagnosis in some situations where the
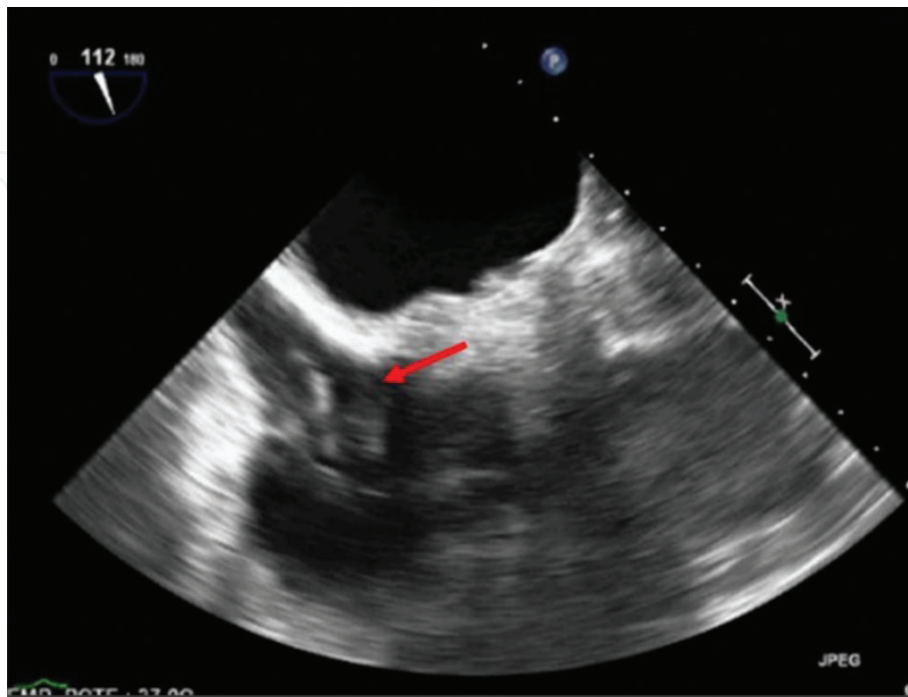

Figure 1.
Transesophageal echocardiogram: a hyperechoic lesion (red arrow) is seen at level of pulmonary valve, prolapsing to right ventricle outflow tract.

clinical suspicion is not always confirmed with the echocardiography. This imaging technique is especially valuable in the diagnosis of prosthetic valve infective endocarditis (PVIE) [5, 12].

There are also reports where the PET helped to determine the extension of pacemaker or defibrillator infection, consequently improving the adequate surgical intervention [13].

Peripheral embolic and metastatic infectious events can also be detected with this technique; nevertheless, their specificity is lower in brain septic emboli [5].

A correct interpretation of PET must be taken in some conditions which can make us misinterpret the findings, for instance, a recent cardiac surgery usually shows enhancement at the mediastinal area due to the inflammatory response. Some conditions can show similar patterns to that of IE, such as an active thrombus, soft atherosclerotic plaques, vasculitis, primary cardiac tumors, cardiac metastasis from a non-cardiac tumor, postsurgical inflammation, and foreign body reactions [5].

3. Treatment

3.1 Medical treatment

The same fundamental aspects about the antibiotic therapy in IE is applied to the right-sided endocarditis, making emphasis in the early and proper setting of the cultures, the prompt and adequate starting of empirical antimicrobial therapy (if the suspicious of IE is higher), and the administration of a culture-antibiogram sensible antibiotic.

One aspect that changed in the antimicrobial treatment of RSIE in comparison with LSIE is the duration of the therapy when the implicated bacteria is the methicillin-sensible *Staphylococcus aureus*, due to the 2015 European Society of Cardiology guidelines for the management of infective endocarditis recommending a short treatment of 2 weeks in this scenario. This approach is attributed to the less aggressive evolution of RSIE with these bacteria [5].

The prophylactic treatment in the patient with high suspicion of RSIE should cover *Staphylococcus aureus*, streptococci, and enterococci and should include penicillinase-resistant penicillins or vancomycin, depending on the local prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) [6].

3.2 Surgery treatment

In RSIE, the medical treatment usually resolves the disease; nevertheless, the surgery for right-sided infective endocarditis is recommended in the following situations: (1) right-heart failure due to severe tricuspid valve regurgitation, (2) inability to eliminate bacteremia or organisms resistant to culture-directed antibiotic treatment, within 7 days, and (3) tricuspid valve vegetations >20 mm [1–3, 5].

During the surgery, most of the infected tissue must be removed; if it is possible, we should try to repair the native valve but guarantee the adequate functioning of the valve. When a valve-sparing is impossible, the implantation of a prosthetic valve is necessary, always trying to use the less foreign material to diminish the risk of IE recurrence [14].

Sometimes the endocardial destruction is highly extensive that compromises the valve repairing as well as the valve prosthesis replacement; this scenario is hideous and requires the reconstruction of the annular structure using endocardium patch or other materials.

Another potential complication of IE can be the formation of ventricular septal defect due to the infection's aggressiveness which can show communication between the right ventricle and left ventricle through the membranous septum. This anatomical defect also can be figured out with a pericardium patch [15].

Some surgeons can feel uncomfortable with the idea of setting up a prosthetic valve in tricuspid position due to being afraid of high gradients through the valve and the potential thrombosis of the prosthesis. However, large prostheses (>30 mm) guarantee low transvalvular gradients, and the incidence of thrombosis is small if the patient has an adequate anticoagulation control (biological and mechanic prostheses are anticoagulated). Moreover, bioprosthesis degeneration develops more slowly owing to the low-pressure conditions in the right ventricle [6].

In 1991, Arbulu et al. published a paper showing their experience in tricuspid valvulectomy without replacement, generally indicated for IVDA, to avoid the potential IE recurrence; nevertheless, about 25% of patients cannot tolerate tricuspid regurgitation and require a second operation for tricuspid valve replacement [14, 16].

4. Prognosis

RSIE implies a better prognosis than LSIE; the previous study revealed the mortality of right-sided IE is 12% in-hospital patients and 0–7.3% for surgical patients. However, these percentages increase at least twice in patients with intensive care unit (ICU) admission; actually, this issue will be described forward [3, 9].

Concomitant left-sided IE carries a worse prognosis than right-sided infection alone, due predominantly to its greater likelihood for invasion and abscess formation [7].

5. Prevention

The high increase of bacterial resistance throughout the last decades has produced a change in the IE guidelines from 2002. The same criteria for LSIE are applied to RSIE regarding the antimicrobial prophylaxis, being reserved only in patients with a high risk of endocarditis, particularly those with PVIE [5].

Nevertheless, there are some aspects that the last IE guidelines do not approach which are very relevant that need to be highlighted. One of the most critical issues is the quite strict aseptic measurements that healthcare professionals must take during routine procedures, especially invasive maneuvers in high-risk patients such as immunocompromised, hemodialytic (HD), cyanotic congenital heart disease (CHD) patients, etc.

The change in some hospital policies can diminish the incidence of bacteremia and IE, such as have been shown in some publications [17].

6. RSIE in intensive care units (ICU)

There are few publications about the characteristics of RSIE in ICU. It is noteworthy that patients with IE admitted in ICU have a higher rate of morbidity and mortality than non-ICU patients. The only study describing the outcome of IDUs with RSIE needing ICU admission reported a mortality of 26% [2].

Some factors have been associated with a worse prognosis: acute respiratory failure requiring mechanical ventilation, shock, Simplified Acute Physiology Score (SAPS II) ≥ 20, and Sequential Organ Failure Assessment (SOFA) ≥ 3 [2, 5].

Other elements that play an essential role at the 30-day survival are age <45 years, Charlson score < 3, endocarditis diagnosed before ICU admission, aminoglycoside use, the presence of septic pulmonary embolism, and a single surgical indication for patients needing a surgical procedure [2].

Reasons for admission to the ICU were a congestive cardiac failure (64%), septic shock (21%), neurological deterioration (15%), and cardiopulmonary resuscitation (9%). Younger patients have a better prognosis because they usually present a minimal dysfunction of the right-sided valve, low risk of pulmonary embolism, and reasonable response to appropriate antibiotic therapy [2].

Opposite to the last IE guidelines, which no longer recommend the aminoglycosides in the treatment of native valve staphylococcal endocarditis, Georges et al. found a better survival in their patients treated with a combination of penicillins or vancomycin with gentamicin [2].

7. Risk factors

It is imperative to describe this pathology in the people with susceptible risk factors (**Table 2**).

7.1 RSIE in people with intravenous drug addiction (IVDA)

The majority of cases of RSIE reports in the literature are in drug abusers. This kind of populations of RSIE represents the 32–86% of all IE [2, 3].

There are multiple explanations about the preference of infection in the right side of the heart at this group of the population, being the leading causes of the poor hygiene with unsafe injection practices and the affected immunology well-being. The higher bacterial load and the variety of effects of injected substances over the endocardium also play an essential role in the physiopathology of the infection [7].

Intravenous drugs abuser's	Indwelling catheters	Intracardiac devices	Congenital heart diseases
32-86% cases of RSIE	HD patients in risk of bacteremia and IE	Worse prognosis and mortality (11-36%)	IE more often is adults than pediatrics
Reinfection 28%, reoperation 20%	Incidence of 8% of all IE	Removal of the infected device is mandatory	VSD is the main anomaly in RSIE
HIV association doesn't increase mortality	AVF diminish risk of IE		Stretococci and Staphylococci are the most frequent bacterias
Survival is equal in comparison with not drug abusers	Noncuffed catheters increase risk of IE		

AVF: arteriovenous fistula, HD: hemodialytic, HIV: human immunodeficiency virus, IE: infective endocarditis, RSIE: right-sided infected endocarditis, VSD: ventricular septal defect.

Table 2.
Characteristics of principal risk factors in the right-sided infective endocarditis.

The incidence of reinfections and reoperations is about 28 and 20%, respectively; however, the survival described in some papers is almost equal between drug abusers and not drug abusers, in which results are very striking [7].

Sometimes IVDA also presents human immunodeficiency virus (HIV) which can aggravate the predisposition to IE if this disease is not well-controlled. The death rates in this subgroup of patients are about 5–10% [1]. The HIV affects both humoral and cellular immunities which provoked a predisposition for recurrent episodes of bacteremia that cause valve damage, fibrin deposition, thrombus formation, and adherence by bacteria in the endocardium; it is common to find abscess developments and large vegetations, which are indications for early surgical treatment [18].

The choice of empiric antimicrobial therapy depends on the suspected microorganism and type of drug and solvent used by the addict and the location of infection.

As previously was described, the empirical antimicrobial therapy must cover *S. aureus*; the combination of penicillinase-resistant penicillins or vancomycin or daptomycin with gentamicin is recommended [5].

The 2015 ESC IE guidelines recommend an antipseudomonal therapy in patients with pentazocine addiction if IE is suspected; nevertheless, there are few and relatively old studies about this issue [5, 19, 20].

If an IVDA uses brown heroin dissolved in lemon juice, *Candida* spp. (not *Candida albicans*) should be considered and antifungal treatment added [5].

7.2 RSIE in people with no IVDA

Although the majority of IE at the right side of the heart is fairly reported in IVDA, there is an increasing incidence in another type of patients, mainly highlighting the people with indwelling catheters and cardiac devices. The 5–10% of RSIE occur in nonaddicted patients [3].

7.2.1 Indwelling catheters

It is estimated that more than 3 million people worldwide require dialysis for end-stage renal disease, and this number is expected to rise sharply because of the aging of the population and an increasing prevalence of diabetes and cardiovascular comorbidities paralleled by a decline in cardiovascular mortality, particularly in very elderly patients (>80 years). For instance, in the United States, this augmentation is about 3.2% per year [21, 22].

Hemodialysis patients are at increased risk for bacteremia, including an estimated 37,000 central line-associated bloodstream infections related to outpatient hemodialysis in the United States in 2008. The elevated incidence of bacteremia increases the risk for infective endocarditis [22, 23].

The average duration on HD before the diagnosis of IE was 30 months (range, 4–66 months). IE is one of the most important causes of increased mortality and morbidity among hemodialysis patients [24].

The *European Heart Journal* states that more than two-thirds of patients undergoing hemodialysis suffer from some infection and that one-third of these patients experience IE [24].

IE occurs 18 times more frequently in chronic HD patients than in the general population [25, 26].

The use of temporal or permanent central catheters, the constant puncture of arteriovenous fistulas, the long and frequent hospitalizations that some of these patients have to suffer during their disease, the various surgical procedures related

with the creation of fistulas, and the underlying alteration of their defenses become susceptible to this population to develop IE.

The IE in HD patients is calculated about at 8% of all observed IE cases regarding the largest international cohort collected to date [27].

The incidence of IE usually increases with the time after the initiation of hemodialysis; however, some studies found a rise of this incidence in the first 5 months after the initiation of hemodialysis [26, 28]. This contradictory results can be probably due to the aseptic technique during the manipulation of the catheter and arteriovenous fistulas of these patients.

Patients in HD also present an increase in the incidence of endocarditis after aortic valve replacement surgery, affecting at the same time the short-term and long-term survival [22].

Most of the studies show that central catheters are a risk factor for bacteremia and endocarditis [6, 7, 10]; nevertheless, Farrington et al. did not find an increase of endocarditis in patients with central catheters in comparison with patients with arteriovenous fistulas [22].

Besides, the rates of IE are more significant in patients with non-cuffed catheters than cuffed catheters; the vascular grafts have more incidence of IE than AV fistulas. Furthermore, peritoneal dialysis has then lesser rates of IE due to the lack of contact of the line with luminal vessels [29].

The morbidity and mortality are higher than the general population; in the 20% of hemodialysis-related IE, the tricuspid valve is the principal place affected at the right side of the heart.

The pathogenesis of IE in HD patient can be attributed to recurrent episodes of bacteremia, the immunological compromise of hemodialytic patients and heart valvular deterioration-calcification frequently founded in this patients.

It can sound logical that the majority of cases of IE in HD patients should happen on the right cavities, similar to what occurs in IVDA; however, the left-side heart (90%) is the more frequent infected place in HD patients, the mitral being the main valve affected. The affectation of the right cavities is roughly 10%. Nevertheless, some papers report an incidence of RSIE in HD patients of between 0 and 50% [30, 31].

Between the multiple explanations of pathogenesis RSIE in HD patients, the high turbulent flow throughout the valves can provoke a deterioration at these structures, becoming more susceptible to bacterial implantation. Nonetheless, the low pressures at the right cavities might not present the same effect in their valves. One possible cause can be the associated pulmonary hypertension that some patients express, due to multiple factors, such as an increased cardiac output (hypervolemic condition and arteriovenous fistula), an increased pulmonary vascular resistance (uremic endothelial dysfunction and pulmonary artery calcifications), and elevated pulmonary capillary wedge pressure caused by heart failure or mitral valve disease [17].

7.2.1.1 Prevention

Patients in HD have an increased risk of developing IE due to all the reasons described before, so to take some measurements sounds logical to diminish the incidence of bacteremia which can result in an IE.

In some hospitals, their politics have been changed regarding the hemodialysis procedure with the intention to ameliorate the arteriovenous life expectancy and decrease the local and systemic infections. For instance, Oun HA et al. have published a lowering in the bacteremia and IE at his hospital adopting new strategies, such as changing the lock solution to taurolidine, cleaning the puncture site with chlorhexidine 2%, and using the buttonhole technique instead of the rope ladder technique.

Nonetheless, it is important to mention that the buttonhole technique had a modest but not significative rising of bacteremia following the move to buttonhole [26].

The arteriovenous fistula (AVF) must always be the best option to perform HD due to their low rates of bacteremia and IE, so, it is imperative to develop an adequate surgical technique and improve the care of the fistula. Whenever it is possible, the fistula must be carried out at the distal part of the arms, trying to preserve the proximal areas to future AVF if the distal fistula fails at some point. If the HD needs a temporary or permanent catheter, the cuffed ones always are preferable to non-cuffed catheters, because the former cause fewer rates of IE [29].

The patient and healthcare personnel must be informed and trained regarding the proper care of the AVF and catheters to lower the probability of bacteremia and IE. The cleaning of the surgical area is paramount as well as the correct AVF puncture.

7.2.2 Intracardiac devices (ICD)

Nowadays ICD are widely used worldwide; their implementation in the cardiology area has improved the quality of life of many people and increased the survival; nonetheless, they have side defects, the endocarditis being one of the most severe complications.

The IE on a cardiac device is increased in the last 10 years in the first-world countries, even becoming the most common cause of IE in some regions. This phenomenon is caused mainly by the rise in the longevity in these countries which results in a growing number of intracardiac devices implanted (pacemakers, cardiac defibrillator, cardiac resynchronizer, or ventricle assist device) [32].

This IE is associated with a worse prognosis and high mortality (11–36%) [32–34]. The pacemaker generator or lead change is the higher factor of risk for IE on the cardiac device. The tricuspid valve is the most common site of RSIE associated with this kind of devices [7, 35].

The removal of the infected device is mandatory in the treatment of intracardiac device infective endocarditis (ICDIE) because it decreases the hospital mortality [32]. Patients with device-related infection and intracardiac vegetations higher or equal at 1 cm have historically undergone surgery for device removal due to the potential risk for septic embolization [34].

7.2.3 Congenital heart disease (CHD)

The risk of IE in patients with adult congenital heart disease (ACHD) is substantially higher (15–140 times) than in the general population. The RSIE in CHD is more often in adults than pediatric patients [5, 36].

The ventricular septal defect (VSD) is the most frequent anomaly in right-sided IE with an incidence of 0.2–2% of all IE [37].

The risk of IE can occur either in repaired or not repaired VSD, with a higher increase in the last one [38].

A recent paper from Tutarel et al. found an incidence of 15.9% of IE in patients with VSD; the 50% of these cases were associated with infections of either the tricuspid valve or the right ventricular outflow tract [36].

The 2015 ESC IE guidelines describe that the distribution of causative organisms does not differ from the pattern found in acquired heart disease, with streptococci and staphylococci being the most common strains. Another study found the streptococci responsible for 50% of congenital heart disease infective endocarditis (CHDIE) and the staphylococci with a 31% incidence [5, 36].

The pulmonary valve is affected in almost 32% of patients from which over an 84% are prosthetic and near 16% native valve [36].

8. Locations

Unlike the left-sided IE mainly occurring on the aorta or mitral valve, right-sided IE could involve the tricuspid valve (82%), pulmonary valve, eustachian valve, interventricular septum, right ventricular free wall, or CS [4, 9].

8.1 Tricuspid valve (TV)

The vast majority of RSIE cases involve the TV (approximately 90%). The high risk of vegetations on the TV is septic PE resulting in various pulmonary complications such as pneumonia and pulmonary abscess.

Uncomplicated tricuspid valve endocarditis can be successfully treated medically in 80% of patients and in the remaining 20% with very large vegetations and expectably poor antibiotic penetration [6].

The infection of the native tricuspid valve in nonaddicted adults occurs in younger patients (under 50 years). In the majority of cases (70%), there are underlying medical conditions such as alcoholism, abortion, colon disease, immunodeficiency, permanent catheters, septic processes in the oral cavity, skin, or genitals, etc. The 25% of cases require valve replacement or surgery [3] (**Figure 2**).

8.2 Pulmonary valve (PV)

RSIE in PV happens in less than 10% of the patients [7]. Most of the cases of pulmonary valve infective endocarditis (PVIE) are provoked by prosthetic material present at this place due to previous surgeries or interventional procedures focused on figuring a congenital disease out.

Bovine jugular grafts are associated with a significantly higher risk of late endocarditis compared with homografts [39].

However, Robichaud et al. did not find an increased risk of PVIE regarding the type of valve, including bovine jugular vein grafts [40].

The rate of IE in transcatheter pulmonary valve implantation is higher than surgical homograft implantation [41].
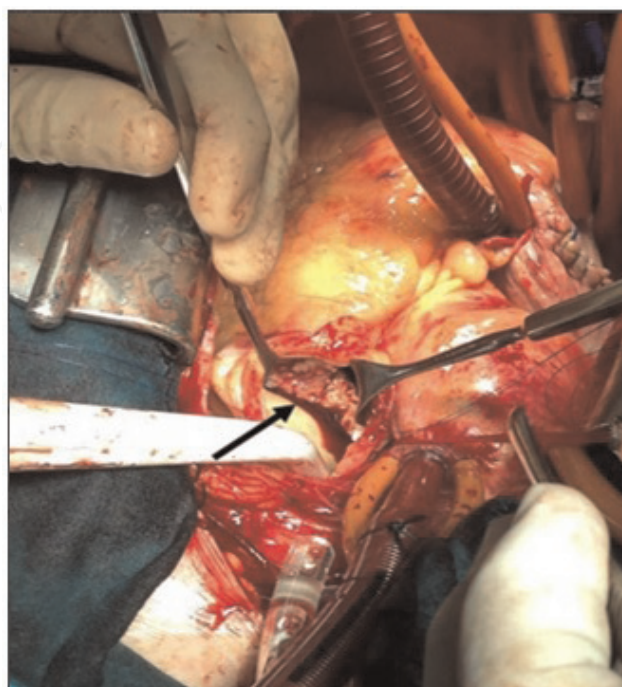

Figure 2.
Pulmonary native endocarditis: a giant mass anchored to the posterior leaflet of pulmonary valve [42].

8.3 Other sites

Uniquely few case reports have been published about RSIE in other locations different to tricuspid and pulmonary valves.

8.3.1 Eustachian valve

Reports of eustachian valve infective endocarditis (EVIE) are approximately 29 cases [43]. An incidence of 3.3% in patients with right-sided endocarditis has been reported [44].

Eustachian valve is a rudimentary structure in adults and, during fetal life, directs oxygenated blood from the inferior vena cava through the foramen ovale and into the left atrium [43, 45].

IVDA is the main high-risk population to develop an EVIE (over 50% of cases). *Staphylococcus aureus* is the most common bacteria implicated in this process [43]. TOE is necessary to identify the vegetation at eustachian valve because this structure is not accessible with TTE [45].

8.3.2 Coronary sinus

There are only eight reported cases of IE in the coronary sinus (CS). The clinical manifestations, the complementary test, the responsible bacteria, and antibiotic treatment are very similar to the other RSIE locations. The CSIE has some features; the CS is always dilated and generally the only affected valve; the vegetation is usually mobile and has a tubule shape with a length of >10 mm [9, 46].

9. Conclusions

RSIE is a pathology scarcely studied because there are few articles released about it. One of the significant reasons about the RSIE little information is the low incidence of this disease; nevertheless, the rates of frequency of this infection are rising nowadays due to the steady increase of HD patients and implanted ICD.

- RSIE clinic criteria are necessary to establish to help in the diagnosis of the disease, such as modified Duke criteria.

- Healthcare personnel must be aware of this illness, keeping their suspicion in high-risk patients and performing the proper complementary test to confirm or discard this infection.

- Hospital policies should be continuously updated to diminish the incidence of RSIE, an adequate epidemiologic analysis about RSIE cases, the population in potential risk to acquire the infection, and the most frequent bugs implicated in this one.

Conflict of interest

None.

Nomenclature

ACHD	adult congenital heart disease
AVF	arteriovenous fistula
CHD	congenital heart disease
CHDIE	congenital heart disease infective endocarditis
CS	coronary sinus
CT	computed tomography
EVIE	eustachian valve infective endocarditis
HD	hemodialytic
HIV	human immunodeficiency virus
ICD	intracardiac devices
ICU	intensive care unit
IE	infective endocarditis
IIAs	infectious intracranial aneurysms
IVDA	intravenous drugs addiction
LSIE	left-side infective endocarditis
MRSA	methicillin-resistant *Staphylococcus aureus*
MR	magnetic resonance
PET	positron-emission tomography
PE	pulmonary embolism
PVIE	prosthetic valve infective endocarditis
PV	pulmonary valve
RSIE	right-side infective endocarditis
SAPS	Simplified Acute Physiology Score
SOFA	sequential organ failure assessment
TTE	transthoracic echocardiography
TOE	transesophageal echocardiography
TV	tricuspid valve
VSD	ventricle septal defect

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References

[1] Horimoto K, Kubo T, Matsusaka H, Baba H, Umesue M. Right-sided infective endocarditis with a ruptured sinus of Valsalva and multiple septic pulmonary emboli in a patient with atopic dermatitis. Internal Medicine. 2015;**54**(7):797-800

[2] Georges H, Leroy O, Airapetian N, Lamblin N, Zogheib E, Devos P, et al. Outcome and prognostic factors of patients with right-sided infective endocarditis requiring intensive care unit admission. BMC Infectious Diseases. 2018;**18**(1):85

[3] Varona J, Guerra J. Tricuspid valve endocarditis in a nonaddicted patient without predisposing myocardiopathy. Revista Española de Cardiología. 2004;**57**:993-996

[4] Revilla A, López J, Villacorta E, Gómez I, Sevilla T, del Pozo MA, et al. Endocarditis derecha aislada en pacientes no adictos a drogas por vía parenteral. Revista Española de Cardiología. 2008;**61**(12):1253-1259

[5] Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zott F. 2015 ESC Guidelines for the management of infective endocarditis: The task force for the management of infective endocarditis of the European Society of Cardiology (ESC) Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). European Heart Journal. 2015;**36**(44):3075-3128

[6] Weymann A, Schmack B, Rosendal C, Rauch H, Karck M, Tochtermann U, et al. Tricuspid valve endocarditis with septic pulmonary emboli in a drug addict. British Heart Journal. 1990;**63**(6):368-369

[7] Hussain ST, Witten J, Shrestha NK, Blackstone EH, Pettersson GB. Tricuspid valve endocarditis. The Annals of Cardiothoracic Surgery. 2017;**6**(3):255-261

[8] Póvoa P, Salluh JI. Biomarker-guided antibiotic therapy in adult critically ill patients: A critical review. Annals of Intensive Care. 2012;**2**(1):32

[9] Song G, Zhang J, Zhang X, Yang H, Huang W, Du M, et al. Right-sided infective endocarditis with coronary sinus vegetation. BMC Cardiovascular Disorders. 2018;**18**(1):111

[10] Hryniewiecki T, Zatorska K, Abramczuk E, Zakrzewski D, Szymański P, Kuśmierczyk M, et al. The usefulness of cardiac CT in the diagnosis of perivalvular complications in patients with infective endocarditis. European Radiology. 2019. DOI: 10.1007/s00330-018-5965-2

[11] Migdady I, Rice CJ, Hassett C, Zhang LQ, Wisco D, Uchino K, et al. MRI presentation of infectious intracranial aneurysms in infective endocarditis. Neurocritical Care. 2018. DOI: 10.1007/s12028-018-0654-1

[12] Álvarez Velasco R, Salvat Dávila C, Vigil Díaz C. Usefulness of PET scan in the diagnosis of infective endocarditis. Medicina Clínica (Barcelona). 2018. DOI: 10.1016/j.medcli.2018.07.017

[13] Diemberger I, Bonfiglioli R, Martignani C, Graziosi M, Biffi M, Lorenzetti S, et al. Contribution of PET imaging to mortality risk stratification in candidates to lead extraction for pacemaker or defibrillator infection: A prospective single center study. European Journal of Nuclear Medicine and Molecular Imaging. 2019;**46**(1):194-205

[14] Carozza A, Renzulli A, De Feo M, Ismeno G, Della Corte A, Dialetto G, et al. Tricuspid repair for infective

endocarditis: Clinical and echocardiographic results. Annals of Thoracic and Cardiovascular Surgery. 2012;**18**(5):481-484

[15] Durden RE, Turek JW, Reinking BE, Bansal M. Acquired ventricular septal defect due to infective endocarditis. Annals of Pediatric Cardiology. 2018;**11**(1):100-102

[16] Arbulu A, Holmes RJ, Asfaw I. Tricuspid valvulectomy without replacement. Twenty years' experience. The Journal of Thoracic and Cardiovascular Surgery. 1991;**102**(6):917-922

[17] Zea-Vera R, Sanchez M, Castañeda E, Soto-Arquiñigo L. Surgical management of multiple valve endocarditis associated with dialysis catheter. Case Reports in Infectious Diseases. 2016;**2016**:4072056

[18] Cicalini S, Forcina G, De Rosa FG. Infective endocarditis in patients with human immunodeficiency virus infection. Journal of Infection. 2001;**42**:267-271

[19] Shekar R, Rice TW, Zierdt CH, Kallick CA. Outbreak of endocarditis caused by *Pseudomonas aeruginosa* serotype O11 among pentazocine and tripelennamine abusers in Chicago. The Journal of Infectious Diseases. 1985;**151**(2):203-208

[20] Wieland M, Lederman MM, Kline-King C, Keys TF, Lerner PI, Bass SN, et al. Left-sided endocarditis due to *Pseudomonas aeruginosa*. A report of 10 cases and review of the literature. Medicine (Baltimore). 1986;**65**(3):180-189

[21] Viecelli AK, Lok CE. Hemodialysis vascular access in the elderly-getting it right. Kidney International. 2019;**95**(1):38-49

[22] Farrington DK, Kilgo PD, Thourani VH, Jacob JT, Steinberg JP. High risk of prosthetic valve endocarditis and death after valve replacement operations in dialysis patients. The Annals of Thoracic Surgery. 2016;**101**(6):2217-2223

[23] Ishida K, Brown MG, Weiner M, Kobrin S, Kasner SE, Messé SR. Endocarditis is a common stroke mechanism in hemodialysis patients. Stroke. 2014;**45**:1164-1166

[24] Habib Khan Y, Sarriff A, Hayat Khan A, Azreen Syazril A, Mallhi TH. Infective endocarditis and chronic kidney disease: How to deal with complications. The Malaysian Journal of Medical Sciences. 2015;**22**(4):73-75

[25] Omoto T, Aoki A, Maruta K, Masuda T. Surgical outcome in hemodialysis patients with active-phase infective endocarditis. Annals of Thoracic and Cardiovascular Surgery. 2016;**22**(3):181-185

[26] Oun HA, Price AJ, Traynor JP. Infective endocarditis in patients on haemodialysis–Possible strategies for prevention. Scottish Medical Journal. 2016;**61**(2):97-102

[27] Murdoch DR, Corey GR, Hoen B, International collaboration on endocarditis-prospective (ICE-PCS) Investigators, et al. Clinical presentation, etiology and outcome of infective endocarditis in the 21st century: The International Collaboration on endocarditis-prospective cohort study. Archives of Internal Medicine. 2009;**169**(5):463-473

[28] Chaudry MS, Carlson N, Gislason GH, Kamper AL, Rix M, Fowler VG Jr, et al. Risk of infective endocarditis in patients with end stage renal disease. Clinical Journal of the American Society of Nephrology. 2017;**12**(11):1814-1822

[29] Bentata Y. Physiopathological approach to infective endocarditis in chronic hemodialysis patients: Left heart

versus right heart involvement. Renal Failure. 2017;**39**(1):432-439

[30] Krcmery V, Kisac P. Bacterial endocarditis in haemodialysis patients. Is frequent heparinisation protecting embolisation? European Journal of Clinical Microbiology & Infectious Diseases. 2011;**30**:1627-1628

[31] Bentata Y, Haddiya I, Ismailli N, et al. Infective endocarditis in chronic hemodialysis: A transition from left heart to right heart? Saudi Journal of Kidney Diseases and Transplantation. 2016;**27**:1200-1206

[32] Ortiz-Bautista C, López J, García-Granja PE, Vilacosta I, Sevilla T, Sarriá C, et al. Endocarditis infecciosa derecha en portadores de dispositivos cardiacos: Perfil clínico y pronóstico. Medicina Clínica. 2017;**149**:477-482. DOI: 10.1016/j.medcli.2017.03.055

[33] Chu VH. Clinical characteristics and outcome of infective endocarditis involving implantable cardiac devices. Journal of the American Medical Association. 2012;**307**:1727. DOI: 10.1001/jama.2012.497

[34] Grammes JA, Schulze CM, Al-Bataineh M, Yesenosky GA, Saari CS, Vrabel MJ, et al. Percutaneous pacemaker and implantable cardioverter-defibrillator lead extraction in 100 patients with intracardiac vegetations defined by transesophageal echocardiogram. Journal of the American College of Cardiology. 2010;**55**:886-894. DOI: 10.1016/j.jacc.2009.11.03

[35] Xu Z, Shi Q, Mei J, Tan Y. Bioprosthetic tricuspid valve replacement for tricuspid valve endocarditis secondary to infected endocardial pacemaker leads. Journal of Cardiac Surgery. 2017;**32**:5-8. DOI: 10.1111/jocs.12878

[36] Tutarel O, Alonso-Gonzalez R, Montanaro C, Schiff R, Uribarri A, Kempny A, et al. Infective endocarditis in adults with congenital heart disease remains a lethal disease. Heart. 2018; **104**(2):161-165

[37] Lee M-R, Chang S-A, Choi S-H, Lee G-Y, Kim E-K, Peck K-R, et al. Clinical features of right-sided infective endocarditis occurring in non-drug users. Journal of Korean Medical Science. 2014;**29**:776-781

[38] Lee PT, Uy FM, Foo JS, Tan JL. Increased incidence of infective endocarditis in patients with ventricular septal defect. Congenital Heart Disease. 2018;**13**(6):1005-1011

[39] Mery CM, Guzman-Pruneda FA, De Leon LE, Zhang W, Terwelp MD, Bocchini CE, et al. Risk factors for development of endocarditis and reintervention in patients undergoing right ventricle to pulmonary artery valved conduit placement. The Journal of Thoracic and Cardiovascular Surgery. 2016;**151**:432-439. 441.e1-2

[40] Robichaud B, Hill G, Cohen S, Woods R, Earing M, Frommelt P, et al. Bioprosthetic pulmonary valve endocarditis: Incidence, risk factors, and clinical outcomes. Congenital Heart Disease. 2018;**13**(5):734-739

[41] Van Dijck I, Budts W, Cools B, Eyskens B, Boshoff DE, Heying R, et al. Infective endocarditis of a transcatheter pulmonary valve in comparison with surgical implants. Heart. 2015;**101**: 788-793

[42] Narvaez A, Granda A, Sierra J, Adrio B, Reija L. Aortic and pulmonary endocarditis with hemorrhagic stroke complication: Which is the ideal surgical time? Cirugia Cardiovascular. 2016;**23**: 255-258

[43] Kottam A, Kaur R, Bhandare D, Zmily H, Bheemreddy S, Brar H, et al. Actinomycotic endocarditis of the eustachian valve: A rare case and a review of the literature. Texas Heart Institute Journal. 2015;**42**(1):44-49. DOI: 10.14503/THIJ-13-3517

[44] San Román JA, Vilacosta I, Sarriá C, Garcimartín I, Rollán MJ, Fernández-Avilés F. Eustachian valve endocarditis: Is it worth searching for? American Heart Journal. 2001;**142**(6):1037-1040

[45] Mordi I, Tzemos N. Think twice, look twice: Eustachian valve endocarditis due to *Escherichia coli*. Journal of Cardiology Cases. 2012;**7**(2): e51-e52. DOI: 10.1016/j.jccase. 2012.10.003

[46] Machado MN, Nakazone MA, Takakura IT, Silva CM, Maia LN. Spontaneous bacterial pericarditis and coronary sinus endocarditis caused by oxacillin-susceptible *Staphylococcus aureus*. Case Reports in Medicine. 2010; **2010**:984562. DOI: 10.1155/2010/984562