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Case Report

Factors Affecting Infectious Endocarditis, Clinical Manifestation and Treatment Modalities – A Nurse's perspective and Overview

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Abstract: Infectious endocarditis virulent microorganisms colonize the endocardium heart valve. It is a rare disorder, and if it is not adequately recognized and treated, it can cause severe and quick morbidity and mortality; as per incidence in the US, approximately 10,000 to 15,000 are affected by infectious endocarditis. Nurses are critical in the prevention, early detection, early diagnosis work-up, and prompt, successful care of this deadly disease and its complications. This article provides a review and updates on this striking yet ambiguous disease. Nurses will be effectively able to plan, incorporate, and evaluate the care required by this distinctive and demanding patient population if existing understanding is transferred into practice. Monitoring vitals like pulse, blood pressure, and temperature and alerting the physician significantly decreases morbidity. Administration of appropriate antibiotics at the proper doses and timing also matters. They also need to monitor the patient's renal status, including blood urea nitrogen levels, creatinine clearance levels, and urine output, especially a sudden appearance of blood in the urine. A Simple look at the nail beds for cyanosis is not a complete replacement for pulse oximetry but remains valid. As far as taking specimens for investigations, precise knowledge is needed. Using vacuum-extracted blood collection systems when we contemplate culture specimens is preferable. A complete education about possible infection sources and cleanliness, like decayed teeth, should be explained to patients. All plastics inside, like intravenous access and urinary catheter, should be treated with complete asepsis. As the disease is sudden, a calm, soothing approach towards patients and attendees goes a long way in getting better outcomes for these patients.

Keywords: Cardiac, Infective Endocarditis, Nurse, Asepsis, Hygiene

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1. INTRODUCTION

Through the significant contributions of William Osler towards the end of the 19th century, the clinicopathological signs of the infection of heart valves—first documented by the French physician Lazare Rivière more than 350 years ago—were better identified [1]. An infection of the heart's endothelium is known as infectious endocarditis (IE), also called bacterial endocarditis. With a mortality rate of up to 30% at 30 days, it has an annual incidence of 3–10/100,000 people. Currently, up to 40–50% of affected patients require valve surgery at some point during the clinical course, with overall mortality remaining around 25% per year affected infective endocarditis.[2] The colonization of the heart valve endocardium by aggressive microorganisms is called infectious endocarditis. It is an uncommon illness that, if improperly recognized and managed, can cause severe and quick morbidity and fatality. The interprofessional team's role in treating patients with infected endocarditis is highlighted in this activity's assessment of the condition's evaluation and management.[3] Given that infective endocarditis (IE) is a rare clinical syndrome with medical doctors and low-volume centers lacking the necessary experience in its management, the principle of forming an Endocarditis Team would be beneficial in optimizing patient outcomes with IE. Because it allows for timely diagnosis and appropriate comprehensive management, a multidisciplinary approach could significantly decrease the hitherto alarmingly high mortality and morbidity among patients with IE. Decisions made by the Endocarditis Team must adhere to a standard protocol based on the current clinical guidelines for treating IE. Following hospital discharge, follow-up, and outpatient clinics are critical due to the likelihood of residual infection and the risk of developing recurrent endocarditis or heart failure, especially in the first two years. Education of patients and healthcare providers [4] is essential for successfully implementing the Endocarditis Team concept. Endocarditis is a grave, life-threatening condition best handled by a multidisciplinary team that includes a cardiologist, cardiac surgeon, infectious disease expert, internist or a medical officer, pharmacist, and intensive care unit nurses. Endocarditis has a high morbidity, and while treatment is effective, the key today is to try to avoid it in the first place, for which a paramedical team has a significant role to play

1.1 Classification

1.1.1 Ie comes in two types

Acute IE — develops abruptly and might become life-threatening in a matter of days
The development of sub-acute or chronic IE (also known as sub-acute bacterial endocarditis) takes weeks to months.[4]

1.1.2 Factors causing affecting ie

Risk factors and the environment of bacterial infection, such as healthcare vs. community, are indicators of the underlying infectious etiology. Early prosthetic valve endocarditis can result in medical cases. Coagulation-negative staphylococci, such as S. aureus, are less dangerous. Epidermidis are commonly found in newly implanted prosthetic valves or indwelling vascular devices. Enterococcal infection is common in both nosocomial and non-nosocomial infections, accounting for 15-18% of cases in each.[5]

1.1.3 Mortality rates and trends

Within the first 30 days, the mortality rate for infective endocarditis is approximately 20%, though exact rates vary between population-based studies. A study of nearly 350 cases of endocarditis in Virginia discovered mortality rates of 20.7% at 30 days, 26.2% at 90 days, and 29.2% at 180 days after admission. At 6.7 years of follow-up, one study of adult congenital heart disease patients found a mortality rate of 19.4%. Another survey of endocarditis in the United States over several decades discovered that in-hospital mortality fell after the 1960s (when it was around 30%) and then stabilized at about 20%. An international cohort study found that longer follow-up after endocarditis admission resulted in significantly higher mortality rates[5,6]. Immunosuppressants, intravenous drug use, poor dental health, degenerative valve disease, and rheumatic heart disease frequently lead to community-acquired illnesses. Viridans group streptococci account for roughly 20% of community-acquired infections, despite being relatively infrequent in healthcare-related infections.[6]

1.2 Clinical manifestations

1.2.1 Cardiac manifestation

On the atrial wall of atrioventricular valves, the typical vegetation in the heart is often in the line of leaflet closure. The infection may result in the rupture of the chordate tendineae, interventricular septum, or papillary muscle, as well as the perforation of a valve leaflet. Particularly with S. aurous, valve ring abscesses with fistula development in the heart or pericardial sac may occur.[7]

1.2.2 Renal manifestations

IE may encounter an immune complex glomerulonephritis, an abscess caused by direct seeding by an embolus, or an infarction caused by emboli in the kidney.

1.2.3 Neurological manifestations

IE most frequently arises from cerebral emboli. These are clinically apparent in approximately 20–30% of patients. The incidence of stroke in IE is 4.82 cases per 1,000 patient days in the first week of IE and drops rapidly after starting antibiotics [8].

1.2.4 Spleen manifestations

Splenic infarcts can also be clinically hidden, but they are typically discovered during autopsies of people who died as a consequence of IE86. Splenic abscesses are characterized by discomfort, fever, and leukocytosis. Splenomegaly affects approximately 10% of contemporary IE patients in the civilized countries.[9]

1.2.5 Pulmonary manifestations

Septic pulmonary emboli can develop due to thromboembolic showering, in which microscopic emboli ("showers")clog and lodge inside small blood channels. This may occur with or without an infarction. This is a standard tricuspid valve IE complication.

Pneumonia, pleural effusions, or empyema are common complications of septic pulmonary emboli.[10]

1.2.6 Skin manifestations

Among the skin conditions in IE are Petechiae, cutaneous infarcts, Osler's nodes, and Janeway lesions. The dermal vessels are encircled by a diffuse perivascular infiltration made mostly of neutrophils and monocytes. The lesions may contain immune complexes. Bacteria, neutrophils, necrosis, and subcutaneous hemorrhage are present in Janeway lesions brought on by septic emboli.[11]

1.2.7 Ocular manifestations

Roth's spots in the eyes are possible in IE patients. In addition, S. aureus IE is particularly prone to endophthalmitis. For instance, 10 out of 23 (43 percent) individuals with IE also had an ocular infection in a prospective cohort of patients with S. aureus bacteremia [12].

Table 1 Modified duke criteria for the diagnosis of, i.e. (clinical criteria) [13]

Major criteria	Minor criteria
Persistently positive blood cultures for any organism (at least two positive cultures of samples drawn >12 h apart)	Fever ≥38.0°C
Single positive blood culture for Coxiella burnetii or anti-phase I IgG antibody titer of >1:800	Vascular phenomena: arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions
Echocardiogram positive for IE, including vegetation, abscess, new partial dehiscence of a prosthetic valve, or new valve regurgitation.	Microbiological evidence that does not meet significant criteria
	Positive blood culture not meeting significant criteria.
	Immunological phenomena (Osler nodes, Roth spots, rheumatoid factor, or glomerulonephritis)

The modified DUKE criteria for the diagnosis of IE by means of clinical criteria are tabled above.

1.3 Diagnostic Evaluation

1.3.1 History and Physical examination

Patients keep mentioning the slow onset of fevers, chills, malaise, and fatigue, which usually requires medical evaluation within the first month. More than 95% of patients in a large, multinational study had a fever, commonly defined as a temperature greater than 38.0 degrees. Predisposing conditions and risk factors are frequently revealed by history, which aids in diagnosis. Recent or previous indwelling catheterization, intravenous drug use, recent pacemaker placement, or background of prosthetic valves all point to a risk of endocardial injury.[14]

1.3.2 Blood culture

IE must be made based on positive blood cultures, which also offer organisms for identification and susceptibility testing. The right way to collect samples is to use an aseptic, no-touch technique to collect three blood samples (10 mL each in anaerobic and aerobic bottles) from different access sites, spaced at least an hour apart. Using vacuum-extracted blood collection systems is preferable when contemplating culture specimens. However, persistent bacteremia in numerous culture bottles of a typical organism is hugely suggestive. Isolated positive blood cultures are equivocal for IE.[15]

1.3.3 Echocardiography

The primary imaging technique for diagnosing endocarditis is transthoracic echocardiography (TTE), which has varying sensitivity rates for valvular and paravalvular abnormalities like vegetations (sensitivity around 65 percent), new regurgitation or dehiscence of prosthetic valve, perforations, abscesses, and fistulae. In the case of valvular or paravalvular complications, prosthetic valve endocarditis (PVE), and endocarditis linked to CIEDs, as well as when TTE is negative, transesophageal echocardiography (TOE) offers better detection and characterization of local abnormalities (sensitivity for intracardiac vegetations of 95 %).[16] The risk factors can be classified as follows

1.4 Cardiac risk factors

Bicuspid aortic valve Mitral valve prolapse Congenital heart disease Rheumatic valve disease Previous episode of infective endocarditis, heart failure

1.5 Vascular risk factors

Chronic hemodialysis Intravenous drug uses Indwelling line for venous access Immunocompromised state Human immunodeficiency virus infection.

1.6 Device-related risks

Patients with an implanted cardiac device or prosthetic valve. Periprocedural complications Signs/symptoms of infection in the periprocedural period. Hematoma formation after device placement Need for revision of device placement

1.7 Drugs and another disease

1.7.1 Chronic anticoagulation

Corticosteroid, Hypertension, Poorly controlled diabetes Coronary artery disease. Chronic obstructive lung disease increasing age, advanced cancer. See table 2 which describes the risk factors from a different perspective :

Table 2 shows the risk factors.

Risk Factor	Description
Previous endocarditis	A history of infective endocarditis increases the risk of recurrent infection.
Heart valve abnormalities	Congenital heart valve abnormalities or prosthetic heart valves increase the risk of endocarditis.
Heart disease	Conditions such as congenital heart disease, rheumatic heart disease, and cardiomyopathies increase the risk of endocarditis.
Intravenous drug use	Sharing contaminated needles and injection equipment can introduce bacteria into the bloodstream, leading to endocarditis.
Certain medical procedures	Procedures such as dialysis, dental procedures, and specific surgical procedures can increase the risk of endocarditis.
Poor dental hygiene	Poor oral hygiene and gum disease can lead to the introduction of oral bacteria into the bloodstream, increasing the risk of endocarditis.
Compromised immune system	Individuals with compromised immune systems, such as those undergoing chemotherapy or with HIV/AIDS, are at an increased risk of endocarditis.

1.8 Length of Antibiotic Therapy

The possibility of a microbiological recurrence is real even in infections brought on by susceptible microorganisms in endocarditis. Long-term therapies are frequently advised for this reason; typically, four weeks for native valve endocarditis and six weeks for PVE. [17]

1.9 Prophylaxis

Antibiotic prophylaxis for dental treatment is presently recommended for patients with a prosthetic valve, a transplanted heart with valvular dysfunction, a history of endocarditis, or congenital heart disease that is (a) unrepaired, (b) in the first six months after prosthetic material repair, or (c) with residual shunt defects. Antibiotic prophylaxis for other invasive surgery is not recommended for cardiac patients unless they are linked to a preexisting infection [17](e.g., ous somewhere and abscess drainage). Infective endocarditis (IE) includes cardiac, neurologic, musculoskeletal, renal, metastatic, and pulmonary complications and systemic infection challenges. More than one complication can occur at the same time, according to a national cohort study. A lengthier follow-up time after admission for endocarditis was also associated with significantly higher mortality rates.

1.10 Surgical treatment

Acute heart failure, severe infection with local consequences, and recurrent arterial embolization all call for early surgical intervention, including valve repair instead of replacement. Heart failure symptoms caused by acute valvular impairment often demand surgical intervention within 24 hours. In the event of an accompanying atrioventricular block, paravalvular abscess, or the presence of damaging infiltrative lesions, also advises early surgical therapy before the end of the initial antibiotic course. [18, 19]

1.11 Nursing interventions

The patient should be given a serene, quiet environment, and his vital signs should be recorded every two to four hours. Prepare him to receive medication through a peripherally implanted central catheter or a new I.V. line. (See figure 1) Existing venous access lines need to be removed since they might be contaminated.[20] Education and application of tepid sponging at wanton times may decrease the fever and its associated harmful effects on the heart.

1.12 Debubbling techniques

When the staff nurse tries to inject through the port of the intravenous system, the air bubble should not go in. The syringe barrel must always be kept vertically up to prevent air injection, as these patients may have cardiac shunts. In addition, the user should be strictly aseptic when giving injections.

1.13 Monitoring

Additionally, they may need to check the patient's SpO2 often and assess for signs and symptoms of heart failure, such as dyspnea, orthopnea, and crackles, and keep an eye out for any worsening or recurrence of his fever. Additionally, conducting multisystem evaluations to spot systemic embolization that could develop in the kidneys, spleen, intestines, extremities, or brain. For example, any hematuria should be immediately informed.

1.14 Hygiene in health

They may need to teach them the value of consulting with their primary care physician before receiving prophylactic antibiotics before dental work or operations involving the respiratory, genitourinary, or gastrointestinal tracts before discharging him. Additionally, instructions for him to let his dentist and all other

medical professionals know about his situation should be proper. Finally, malnutrition is associated with poor outcomes in any sick patient. Hence an eye on his nutritional intake rather than a simple intake output chart will throw light on more important things for better results.

A -showing blood in the cannula site, B showing sterile prep before the procedure C showing wearing sterile gloves. D showing usage of disposable catheters.

Fig 1 shows the different small techniques to decrease t he infective endocarditis incidence.

1.15 Future directions

Considering the consistently increased prevalence, complication rates, and mortality rates of infective endocarditis, developing improved diagnostic tools and treatment modalities remains critical. In addition, with the shifting epidemiology of these infections, clinicians must recognize the identification of infective endocarditis soon in a patient's illness journey to improve outcomes.

2. CONCLUSION

Since infectious endocarditis is a complicated condition, experts from various fields are needed to help treat it. The importance of a multidisciplinary approach in IE must be emphasized in both IE research and illness care. From the nursing perspective, a simple look at the nail beds for cyanosis is not a complete replacement for pulse oximetry but remains useful. A comprehensive education about possible infection sources and cleanliness, like decayed teeth, should be explained to patients. All plastics inside, like intravenous access and urinary catheters, should be dealt with complete asepsis. As the disease is sudden, a calm, soothing approach towards patients and attendees goes a long way in getting better outcomes for these patients.

3. AUTHOR CONTRIBUTION STATEMENT

Malarmathi. M, Rajalakshmi, Dhivya.N, and Meena.M conceptualized and gathered the data with regard to this work and analysed these data and necessary inputs were given towards the designing of the manuscript. All authors discussed the methodology and results and contributed to the final manuscript.

4. CONFLICT OF INTEREST

Conflict of interest declared none.

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