CLINICAL DIMENSION

Infective Endocarditis: A Challenging Diagnosis

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The critical care nurse plays a key role in the management and care of the patient with Infective Endocarditis. The nurse's sensitive assessment of post-operative cardiac surgery patients helps to identify this complication in the early stages so that early antibiotic therapy can improve outcomes for patients with this complication. Likewise, the nurse's assessment which uncovers a history of recent dental techniques or drug abuse helps identify this complication in newly-admitted patients. By administering and evaluating the success of antibiotic therapy the nurse helps the patient combat the Infective Endocarditis.

Infective Endocarditis has a mortality rate of 15% to 40% percent despite antibiotic therapy.[1] There are three major reasons for this high mortality: failure to prevent the disease, late recognition once the disease is present, and ineffective antibiotic therapy.[2] The critical care nurse plays an important role in each of these factors. The nurse provides preventive measures, assesses for early signs of infection, and administers the antibiotic therapy. When a critically ill patient has Infective Endocarditis, the critical care nurse provides comprehensive nursing care to minimize the risks from life-threatening complications.

Pathophysiology

The basic lesion of Infective Endocarditis is a large, friable infected vegetation on the leaflets of a damaged heart valve, the endocardial lining of an abnormal surface of a heart chamber, or the endothelium of a blood vessel.[3] The vegetation consists of three layers: a thick inner layer composed of platelets, fibrin, white blood cells, and collagen; a middle layer composed mainly of bacteria; and an outer layer of fibrin and additional bacteria.[4]

Vegetations are single or multiple, several centimeters in size, and are usually found attached to the free edge of the valve surface.[5] The color of the vegetations varies from pink, red, yellow, or green in the early stages to gray after healing.[6] Clumps of vegetation are sometimes referred to as verrucae[7] (see Figure 1).

Lesion Sites

Most lesions occur where there is a preexisting congenital or acquired structural abnormality, but lesion sites are sometimes found on apparently normal heart surfaces.[8] Hemodynamic alterations influence where an infective vegetation localizes. Alterations in blood flow create areas of trauma on the endothelial surface to which platelets readily adhere. Due to the inherent stickiness of platelets, the platelets and fibrin adhere to these traumatized sites. Bacteria readily colonize in this platelet-fibrin mass with subsequent infection and vegetation.[9,10] Because of the avascular nature of the platelet-fibrin mass, bacteria proliferate and the host is unable to eliminate this infective process. In fact, vegetations of Infective Endocarditis actually protect the infecting organism from host defenses.[11]

The pathogens result in an inflammatory growth which causes destruction of the infected tissue. Vegetation interferes with normal alignment of the valve cusps resulting in incomplete closure or regurgitation. This may manifest as murmurs.[4] The pathogens injure the valve leaflet until they penetrate the valve's full thickness. In some patients this results in perforation of leaflets or secondary spread to the chordae tendinea or papillary muscles resulting in compromised function of the affected valve and subsequent signs of backward failure. Additionally, the pressure of turbulent blood flow through the affected valve may cause the verrucae to crumble, creating peripheral embolization.[6]

It has been shown that bacteria are deposited in areas where the velocity of blood flow is high with decreased lateral pressure. As a result, verrucae usually develop on regurgitant rather than stenotic valves and on the low pressure side of insufficient valves. Thus, the atrial side of the mitral valve and the ventricular side of the aortic valve become involved in the presence of valvular insufficiency.[11,12] The mitral valve is the most commonly affected one, followed by involvement of the aortic valve, although coexisting infection of the mitral and aortic valves has been reported as a frequent occurrence.[3] Gregoratos and Karliner reported that tricuspid valve endocarditis develops with a mean incidence of 2% and occurs primarily in parenteral drug abusers. Pulmonic valve endocarditis is an even more uncommon occurrence, as is endocarditis involving both sides of the heart which reportedly has a 1% incidence.[13] The presence of ventricular septal defects, coarctation of the aorta, patent ductus, or Tetralogy of Fallot may also alter blood flow resulting in endothelial destruction and infection at the damaged site. A higher incidence of Infective Endocarditis has also been reported in patients with asymmetric septal hypertrophy, Idiopathic Hypertrophic Subaortic Stenosis (IHSS), and mitral valve prolapse.[11]

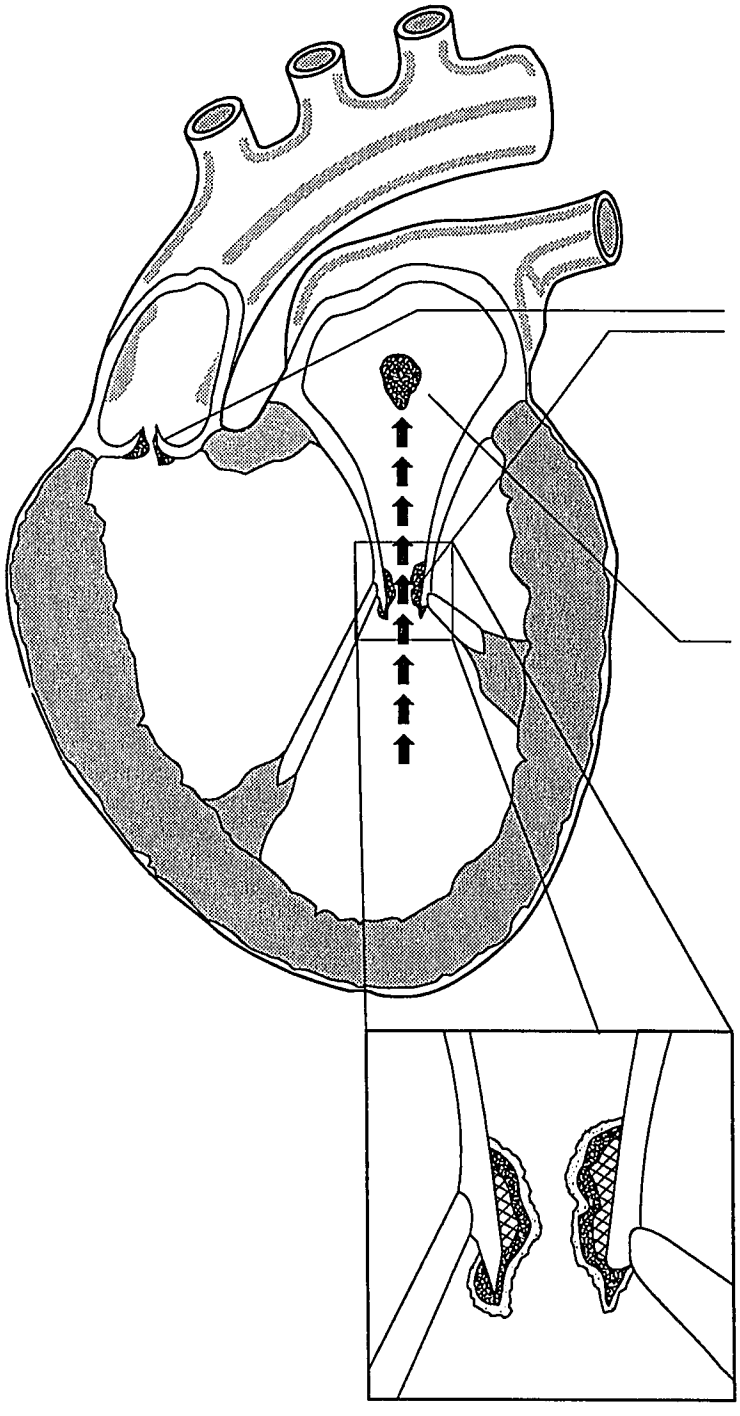
The infection also effects prosthetic valves although the pathology differs between native and prosthetic valve endocarditis. With native valve endocarditis, the infected vegetation is basically situated on the valve leaflet. Occasionally infection destroys the leaflet and extends into the valve annulus. Infection of prosthetic valves develops at the interface between the sewing ring of the prosthesis and the valve annulus and extends into the annular tissue. As a result of this invasive infection, annular or valve ring abscesses develop. Infection causes necrosis of annular tissue which allows sutures anchoring the prosthesis to pull free resulting in partial dehiscence of the valve from the annulus with subsequent leakage.[14]

Identifying Patients at Risk

The critical care nurse identifies patients at risk for Infective Endocarditis. The nurse observes patients for the presence of risk factors that signal a good chance of the complication. Watch for risk factors such as:

- Presence of any intracardiac prosthetic materials
- Recent exposure to dental, upper respiratory, gastrointestinal, or genitourinary surgeries, procedures, or invasive techniques.
- Current history of intravenous drug abuse[7,10]

The critical care nurse assesses for drug use, because the parenteral drug abuser is at high



Verrucae usually develop on regurgitant rather than stenotic valves and on the low pressure side of insufficient valves. As a result, the atrial side of the mitral valve and the ventricular side of the aortic valve become involved in the presence of valvular insufficiency.

Alterations in blood flow create areas of trauma on the endothelial surface to which platelets readily adhere.

Vegetation consists of three layers: a thick inner layer composed of platelets, fibrin, WBC's, and collagen; a middle layer composed mainly of bacteria; and an outer layer of fibrin and additional bacteria.

Figure 1: Diagram showing the most common location of verrucae on the cardiac valves Infective Endocarditis, Close-up box shows the three layers in the verrucae.

Figure 1 is part of the DCCN STATPack™. From Snelson, C.; Cline, B.A.; Luby, C., Infective Endocarditis: A Challenging Diagnosis. Dimensions of Critical Care Nursing (DCCN), January/February 1993.

risk for Infective Endocarditis. Durack reports that the bacteria can be introduced directly by injection of contaminated materials or indirectly when the user injects intravenously or "skin pops" subcutaneously and becomes contaminated with his own skin flora. This route of administration has been associated with a high frequency of tricuspid valve involvement.[15]

The epidemiology of Infective Endocarditis in the parenteral drug abuser has also shown a relationship to geographic location. The causative agent in San Francisco has frequently been Serratia marcescens; Pseudomonas aeruginosa has been found in Detroit and Chicago; in Cleveland, enterococcal infections have been reported because toilet water is often used as the medium for injection.[11]

Also collect other important information from the patient's history which help the physician diagnose the problem or suggest the best antibiotic therapy. Ask about:

- Duration of presenting symptoms
- Drug allergies and type of allergic response
- Recent treatment for an infection with antibiotics
- Pre-existing renal disease

Assisting with the Medical Diagnosis

Since this disease is difficult to identify prior to the acute phase, the nurse's assessment and documentation of symptoms is critical to the physician's ability to rule out other conditions and make an accurate diagnosis of Infective Endocarditis. The physician makes the definitive diagnosis of Infective Endocarditis by examining the valves in surgery or by positive gram stain of a valvular vegetation. Since this is almost impossible to obtain in the ambulatory patient, most diagnoses of Infective Endocarditis are speculatively based on symptoms recorded through the detailed nursing history.

Recognizing the Infectious Agent

Once the diagnosis of Infective Endocarditis is suspected, the critical care nurse helps identify the infecting pathogen. This is critical in order to determine the most specific antimicrobial regimen for the patient. Correct etiologic diagnosis requires the isolation of the pathogen from the blood stream. The critical care nurse ensures prompt and proper obtainment of a blood culture specimen. Nearly all blood cultures from a given patient with endocarditis are positive. In the absence of recent antibiotic treatment, 5% or fewer patients with endocarditis have a persistently negative blood culture.[1] Antimicrobial susceptibility of the infecting organism is then determined once the blood culture is found to be positive.

The nurse reviews the blood culture results to recognize the infectious agent and prepare for antibiotic orders. Nearly all bacteria isolated from humans have been known to cause Infective Endocarditis, however gram positive cocci account for 80% of the active cases. In particular, Viridans streptococci, enterococci, Streptococci bovis, and staphylococci are the most common organisms. The prevalence of these causative organisms is thought to be related to their increased ability to adhere to platelet-fibrin thrombi, damaged valves, or normal valvular endothelium.[11]

Viridans strep is a common bacteria of the oral cavity. This organism may be introduced into the systemic circulation after vigorous tooth brushing, using a high pressure oral water cleaning device, completing dental work, or following upper respiratory, gastrointestinal, or genitourinary invasive procedures.[10]

Endocarditis occurs two to three times more frequently after prosthetic valve replacement than with any other type of heart surgery. Karchmer reported patterns of organisms causing prosthetic valve endocarditis. S. aureus was identified as a common cause of nosocomial infections after cardiac surgery, and S. epidermidis as a frequent contaminant of the cardiac bypass system and intravenous lines.[14]

Prosthetic valve endocarditis is classified by the type of early symptoms. Early-onset endocarditis occurs when infection develops in the perioperative value-replacement period. Prolonged cardiopulmonary bypass, intracardiac foreign bodies, and invasive procedures during the immediate postoperative period predispose these patients to Infective Endocarditis.[10] Late-onset endocarditis develops two months after surgery due to another initiating event.

Candida albicans, Aspergillus, and Histoplasma cause the majority of the cases of mycotic

or fungal Infective Endocarditis. Critically ill patients at risk for the development of fungal endocarditis are those with total parenteral nutrition (TPN) and central intravenous lines especially with concurrent immunosuppression, parenteral drug abusers, and patients who have had cardiac surgery.[10,13]

The critical care nurse assesses the patient for symptoms which may help identify the type of infectious organism. Most patients have the usual symptoms of infection: fever, chills, weakness, dyspnea, sweats, malaise, cough, splenomegaly, and hematuria. Rahimtoola describes the classic syndrome of fever, murmur, splenomegaly, and anemia.[2] A patient who is infected with staphylococcus often has an acute fulminant process with marked temperature elevations and shaking chills. Streptococcal infections usually produce a less acute process with moderate temperature elevations, chills, weakness, splenomegaly, anorexia, and night sweats.[10]

Echocardiography also has prognostic value for the identification of large mobile verrucae. These are more likely to embolize leading to serious systemic complications. The presence of a large vegetation may determine if a patient receives surgical resection as well as antibiotic therapy. The additional use of Doppler ultrasound can determine the competency of the valves involved. If the valves show significant regurgitation, then there is a greater likelihood of surgical replacement of the valve.[16]

Vascular changes often accompany Infective Endocarditis. Physical assessment of the patient reveals splinter hemorrhages and petechiae of the skin, buccal, pharyngeal or conjunctival mucosa.[4] Splinter hemorrhages are found in the distal third of the nailbed and appear as black or brown lines under the nail.[7]

Less common vascular changes include Janeway lesions, Osler nodes, and Roth spots. Janeway lesions are painful papular lesions of the dorsum surface of the hands and feet. Osler nodes are painful subcutaneous nodes on the pads of the fingers and toes. Round or oval retinal hemorrhages with pale or white centers are known as Roth's spots.[7]

Assessing for Cardiac Complications

The critical care nurse is responsible for a comprehensive cardiac assessment. Assess heart sounds for the presence of S3, S4, or a summation gallop. These findings in conjunction with a new murmur may indicate heart failure. Localization of sounds helps identify right or left heart pathology. Valvular dysfunction with left heart failure is the most dangerous complication of Infective Endocarditis.[17] A patient with heart failure requires intense monitoring in a critical care environment. He or she may need a balloon tip flow-directional pulmonary artery catheter to monitor pulmonary wedge pressures and cardiac output. Depending upon the extent of the left-sided failure, the patient may need oxygen, vasodilator therapy, bedrest, and fluid restriction.[18]

Right-sided Infective Endocarditis is suspected when the patient has no murmur, but shows symptoms of systemic infection and venous congestion. The critical care nurse assesses for right-sided failure by examining the patient for venous congestion including jugular venous distention at a 45 degree angle, hepatomegaly, a positive hepatojugular reflux, and peripheral edema.

The cardiac assessment includes monitoring of the patient's electrocardiogram. The electrocardiogram is carefully assessed for conduction abnormalities, ventricular hypertrophy, axis deviation, and ST-T wave abnormalities secondary to the embolization of vegetation into a coronary artery.[2]

Assessing Neurological Status

The critical care nurse assesses the neurological status of the patient with Infective Endocarditis because neurological complications occur in 30% to 40% of the cases. The most common neurological complication is cerebral embolism caused by pieces of verrucae from the left side of the heart which travel to the brain causing paralysis, sudden blindness, aphasia, ataxia, and cortical sensory loss.[2] Indeed, the sudden development of hemiplegia in a critically ill young adult is suspicious for Infective Endocarditis.[15]

Monitor the patient with neurological involvement for accelerating loss of neurological function. Careful documentation of neurological status is maintained in order to assess these trends. The patient may need anticoagulation, cardiac monitoring, and supportive nursing care.

This supportive care includes proper positioning, nutritional support, and the maintenance of a safe environment.[18]

Determining Musculoskeletal Involvement

The critical care nurse completes a strength analysis of the patient since musculoskeletal symptoms are now recognized as a more common occurrence in Infective Endocarditis. Arthralgia and arthritis of the proximal joints and lower extremities are a common complaint. Lumbar back pain and myalgias occur in 45% of the reported cases of Infective Endocarditis. These musculoskeletal symptoms usually resolve with antibiotic therapy.[2]

Assessing Renal Complications

Continually assess for renal complications because glomerulonephritis is one of the most common complications of Infective Endocarditis. It is caused by the deposition of immune complexes in the renal basement membrane.[17] Although glomerulonephritis is a common complication, fortunately, it is usually not severe enough to cause renal failure. The critical care nurse assesses hourly intake and output; urine quality; blood urea nitrogen, potassium, sodium, and creatinine levels; and the presence of costovertebral angle (CVA) tenderness.[18]

While the glomerulonephritis may not cause kidney failure, the antibiotic therapy may. The risk of renal failure may preclude the use of aminoglycoside antibiotics due to their nephrotoxic effects.[1] If renal failure does develop, the patient needs dialysis to eliminate toxic wastes from the body.

Monitoring Antibiotic Therapy

The critical care nurse monitors the results of antibiotic therapy and prevents or minimizes the risk of drug side effects. The type of antibiotic prescribed varies with the type of organism causing Infective Endocarditis.

Specific recommendations for the selection of antibiotic treatment of Infective Endocarditis based on isolation of the infecting microorganism are the consensus of an ad hoc writing group of the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the American Heart Association's Council of Cardiovascular Disease in the Young.[1] Since vegetation protects the infecting organism from host defenses, the antibiotic must exert a bactericidal rather than a bacteriostatic effect on the microorganism.[1,2,14] Parenteral antibiotic therapy with the appropriately sensitive antimicrobial agent is necessary in order to obtain high and predictable serum antibiotic levels to achieve this effect.

The nurse assesses for positive effects of the antibiotic. If improvement in the patient's clinical condition has not occurred within 48 hours after initiating antibiotic therapy, then the critical care nurse collaborates with the physician to re-evaluate antibiotic therapy.

Administering Antibiotics for Viridans Streptococcal Endocarditis

The critical care nurse monitors and reduces the incidence of side effects for antibiotics administered to combat Viridans streptococcal infections. Viridans streptococci are the most common etiologic agents in subacute endocarditis of native heart valves and congenital cardiac abnormalities. Viridans streptococci include Streptococcus sanguis, salivarius, and mutans. Parenteral penicillin for four weeks without an aminoglycoside can be expected to achieve bacteriologic cure in up to 99% of the adult patients for highly penicillin susceptible Viridans streptococci.[1] The addition of streptomycin or gentamicin exerts an *in vitro* synergistic killing of Viridans streptococci. A two week regimen of parenteral combination therapy with penicillin and streptomycin is appropriate for uncomplicated cases of highly penicillin susceptible Viridans streptococci occurring in patients at low risk for aminoglycoside toxicity.[1,2,9,14] The two week regimen is not recommended for patients with complications such as shock, extracardiac foci of infection, or intracardiac abscess.[1]

The critical care nurse has a responsibility to differentiate between patients with complicated versus uncomplicated cases of Viridans streptococci endocarditis and to assess for the development of toxic reactions secondary to aminoglycoside therapy. Prolonged aminoglycoside therapy can be complicated by nephrotoxicity and ototoxicity. Streptomycin is more commonly associated with ototoxicity and desirable therapeutic peak serum streptomycin levels are considered to be 20 to 30 micrograms/mL.[1] Trough levels should be less than 5 micro-grams/mL.[19]

Currently, gentamicin is most widely used in clinical practice even though it is associated with nephrotoxicity. This medication can be administered intramuscularly or intravenously. Gentamicin serum levels are readily available; desirable therapeutic levels should be maintained at 3 micrograms/mL.[1]

The critical care nurse assumes the responsibility for obtaining accurately timed serum peak and trough aminoglycoside level specimens, administering accurate and timely doses of the antibiotics and monitoring the patient for symptoms related to aminoglycoside toxicity.

These recommendations also apply to Infective Endocarditis from Streptococcus bovis.[2,9] Streptococcus bovis is a non-enterococcal group D streptococcus. Streptococcus bovis is frequently associated with pathologic conditions of the gastrointestinal tract, especially carcinoma of the colon.[1,14]

Vancomycin is recommended as an effective alternative and the drug of choice in patients with immediate-type hypersensitivity to penicillin.[1] The critical care nurse assesses patients for complications such as thrombophlebitis, ototoxic and occasionally nephrotoxic reactions related to prolonged intravenous use of this drug.

Cephalosporins should not be used for the treatment of streptococcal endocarditis in patients with a history of immediate-type hypersensitivity reaction to penicillin, but can be used in patients with other manifestations of penicillin allergy.[1,14] When the choice is made to use cephalosporin therapy, the critical care nurse is alert for the development of allergic reaction and is prepared to initiate immediate treatment if the reaction is serious. (See Table 1 for guidelines on the antibiotics used for Infective Endocarditis.)

Administering Antibiotics for Enterococcal Endocarditis

Enterococci are streptococci that normally inhabit the gastrointestinal tract and include Streptococcus faecalis, faecium, and durans. Treatment of enterococcal endocarditis is complicated because these organisms are more resistant to penicillin G and expanded-spectrum penicillins as well as uniformly resistant to cephalosporins and aminoglycoside antibiotics.[1] The combination of penicillin, ampicillin, or vancomycin plus an aminoglycoside antibiotic has been found to exert a synergistic bactericidal effect on these organisms in vitro. Currently there is not a high-level resistance to the aminoglycoside gentamicin and the enterococci are killed by the combination of penicillin and gentamicin. Therefore, it is the preferred therapy for enterococcal endocarditis.[1,14]

It is important for the critical care nurse to establish duration of symptoms prior to initiation of antibiotic therapy in patients with enterococcal Infective Endocarditis. Patients with symptoms of infection greater than three months as well as those with prosthetic valve enterococcal endocarditis should receive six weeks of antibiotic therapy. The overall cure rate is approximately 75 percent for enterococcal endocarditis.[1]

Administering Antibiotics for Staphylococcal Endocarditis

Infective Endocarditis may be caused by staphylococci that are coagulase positive such as staphylococcus aureus or coagulase negative like staphylococcus epidermidis. These staphylococci infect either native or prosthetic valves, however most cases of coagulase negative staphylococcal endocarditis occur in patients with valvular prostheses. Re-assess the patient with this type of Infective Endocarditis for a history of prosthetic valve surgery. Also, check again on intravenous drug use because intravenous drug abuse is often caused by coagulase positive Staphylococcus aureus. S. aureus endocarditis in intravenous drug abusers with tricuspid valve involvement have a cure rate as high as 85%.[1] Endocarditis secondary to Staphylococcus aureus in non-addicts primarily involves valves on the left side of the heart and is associated with a lower mortality of 25% to 40%. Plan for the management of either right or left sided heart failure based upon knowledge of the etiology of the staphylococcus endocarditis.

The majority of staphylococcus endocarditis acquired in the community and hospital are highly resistant to penicillin G. For this reason, the drugs of choice are usually methicillin, nafcillin, and oxacillin.[1,2,9,14] Methicillin is seldom used due to concern for the increased incidence of interstitial nephritis.[1]

Treatment for staphylococcal endocarditis of native valves or without the presence of intracardiac prosthetic materials differs

Table 1: Antibiotics for Various Types of Endocarditis*

Allergy	*Possible Antibiotic*	*Pharmacodynamics and Nursing Considerations*
Type I: Viridans Streptococci and Streptococcus Bovis Infection		
Penicillin Susceptible, Penicillin G Resistant, No penicillin allergy	Aqueous crystalline penicillin G IV (4 weeks)	•Preferred in patients older than 65 or with impairment of 8th cranial nerve or renal function
	Aqueous crystalline penicillin G IV (2-4 weeks) or Procaine penicillin G with Streptomycin, IM (2 weeks) or Procaine penicillin G IV with Gentamicin IM/IV (2 weeks)	•Procaine penicillin not recommended for children •Observe for risk of ototoxicity and nephrotoxicity •Maintain good hydration
Penicillin Susceptible, Penicillin G Resistant, Patient, with Penicillin Allergy	Cephalothin or Cefazolin, IV (4 weeks); with Streptomycin, IM (2 weeks) or with Gentamicin, IM/IV (2 weeks)	•Dosage reduced in patients with moderate to severe renal dysfunction •Possible cross allergenicity with penicillin •Increased incidence of thrombophlebitis with continuous infusion •Dosing on a mg/kg basis will produce higher serum levels in obese than lean patients
	Vancomycin, IV (4 weeks)	•Obtain peak serum levels one hour after infusion •Dosing on a mg/kg basis will produce higher serum levels in obese than lean patients •Ototoxic and nephrotoxic with elderly at risk for ototoxic effects •Thrombophlebitis at injection site •Extravasation with necrosis and sloughing due to infiltration •Rapid injection may cause "red man" syndrome with hypotension and cardiac arrest
Type II: Enterococci Infection		
Patient Has No Penicillin Allergy	Aqueous crystalline penicillin G, IV (4-6 weeks) with Gentamicin, IM/IV (4-6 weeks) or Streptomycin, IM (4-6 weeks)	•Dosages should be adjusted to obtain acceptable peak levels •Check to confirm no penicillin allergy
	Ampicillin, IV (4-6 weeks) with Gentamicin, IM/IV (4-6 weeks) or Streptomycin, IM (4-6 weeks)	•Evaluate to confirm no allergy to penicillins •Assess for fever or chills •Assess for rash, the most common allergic reaction •Monitor renal function *(continued on next page)*

Table 1 (Continued)		
Penicillin Allergic Patients	Vancomycin, IV (4-6 weeks) with Gentamicin, IM/IV (4-6 weeks) or Streptomycin, IM (4-6 weeks)	•Desensitization to penicillin should be considered •Cephalosporins are not satisfactory alternatives •Choice depends on resistance level of infecting strain
Type III: Staphylococcus Infection		
Methicillin Susceptible, No penicillin Allergy, and No Cardiac Prosthetic Material	Nafcillin IV (4-6 weeks) or Oxacillin with optional Gentamicin, IM/IV (3-5 days)	•Preferred over methicillin due to increased incidence of interstitial nephritis •Cross allergenicity with Cephalosporins •Rotate IV sites to avoid venous irritation •Causes acute interstitial nephritis preceded by fever and rash
Penicillin Allergy Patient, No Cardiac Prosthetic Material	Cephalothin, IV (4-6 weeks) or Cefazolin with optional Gentamicin, IM/IV (3-5 days)	•Avoid IM route as cephalothin causes pain IM •Check renal function
	Vancomycin, IV (4-6 weeks)	•Check site daily for phlebitis and irritation. •Monitor renal function •Watch for hearing loss
Penicillin Allergy, Methicillin Resistant, No Cardiac Prosthetic Material	Vancomycin, IV (4-6 weeks)	•Same as above
Methicillin Susceptible, With Cardiac Prosthetic Materials	Nafcillin, IV (>6 weeks) or Oxacillin with Rifampin, po (>6 weeks) and Gentamicin, IM/IV (2 weeks)	•Monitor renal, hepatic, and hematopoietic functions •Rapid injection may cause thrombophlebitis •Contains 3.1mEq. of Na/gm.; should be considered in congestive heart failure or limited sodium intake patient •Inhibited by aminoglycosides •Penicillin-allergic patients use vancomycin or first generation cephalosporins with coagulase negative staphylococcal endocarditis
Methicillin Resistant, with Cardiac Prosthetic Materials	Vancomycin, IV (>6 weeks) with Rifampin, po (>6 weeks) and Gentamicin, IM/IV (2 weeks)	•Check site daily for phlebitis and irritation. •Monitor renal function •Assess for hearing loss

**Adapted from Bisno AL, et. al (1989) Antimicrobial treatment of Infective Endocarditis due to viridans streptococci, enterococci, and staphylococci. JAMA, 261 (10), 1471-1477. Copyright 1989, American Medical Association. Reproduced with permission.*

Table 1 is part of the DCCN STATPack™. From Snelson, C.; Cline, B.A.; Luby, C., Infective Endocarditis: A Challenging Diagnosis. Dimensions of Critical Care Nursing (DCCN), January/February 1993. ©1993 Hall Johnson Communications, Inc. This material is developed for the exclusive use of DCCN subscribers to teach in-house courses according to the *STATPack™ Instructions* (Page 17). If you are not a current subscriber, contact the publisher for written permission to copy, present, or adapt this material.

from that of prosthetic valve staphylococcal endocarditis. Therefore, each of these needs to be discussed separately.

The treatment of choice for staphylococcal endocarditis on native cardiac valves is parenteral nafcillin for four to six weeks.[9,14] The addition of gentamicin is recommended to minimize damage to heart valves, but limited to the first five days to avoid nephrotoxic reactions associated with prolonged aminoglycoside use.[1]

A high percentage of coagulase negative staphylococcal endocarditis without prosthetic materials is methicillin resistant.[1] The critical care nurse needs to be cautious with the interpretation of antimicrobial susceptibility testing because some systems fail to detect the methicillin resistance. Methicillin resistant S. aureus is also resistant to cephalosporins and this is not always reflected by susceptibility testing. Endocarditis due to methicillin resistant S. aureus must be treated with parenteral vancomycin.[1,2]

Staphylococcal endocarditis with intracardiac prosthetic materials can be either coagulase negative or positive in origin. Staphylococcal endocarditis that is coagulase negative and occurring within one year of surgery is also usually methicillin resistant. Optimal antibiotic therapy is provided by vancomycin combined with rifampin for six weeks and gentamicin, limited to the first two weeks. Endocarditis of this nature, particularly involving the aortic valve is often complicated by perivalvular and myocardial abscesses as well as valvular dysfunction[1]. The critical care nurse directs assessments towards the rapid identification of symptoms related to these complications and prepares the patient and family for the potential of valvular replacement surgery. Surgery is often required and life saving in this group of patients.

Prosthetic valve endocarditis caused by coagulase positive S. aureus is associated with a high mortality. Penicillinase-resistant nafcillin is the primary antibiotic of choice. Methicillin resistant regimens consist of vancomycin with the combined use of gentamicin for the first two weeks.[1]

Administering Antibiotics for Mycotic Endocarditis

Mycotic endocarditis from Candida albicans is frequently reported in narcotic addicts who "skin pop" or inject intravenously. It may be seen in patients following cardiac surgery due to contamination of surgical fields or the use of intravenous lines.[20] Antibiotic therapy is extremely limited and consists of Amphotericin B alone[9], or in combination with 5-Fluorocytosine.[2] There are a few cases reported of successful treatment of C. albicans endocarditis with fluconazole, however clinical trials are limited. Fluconazole is generally not recommended because it is effective against C. albicans and ineffective against aspergillus endocarditis.[19] Often surgical treatment with prosthetic valve replacement is indicated with mycotic endocarditis due to the limited drug therapy. Mycotic endocarditis yields a guarded prognosis secondary to toxicity of pharmacologic treatment and limited overall effectiveness.[2]

When caring for the mycotic endocarditis patient the critical care nurse does a continuous assessment for the toxic effects of antibiotic therapy and monitors the patient for therapeutic effectiveness of the drug. The critical care nurse prepares both the patient and family for the possibility of prosthetic valve replacement if antibiotic therapy is not effective. The combination of medical and surgical therapies reduces the mortality associated with mycotic endocarditis if the treatment is initiated within several days after diagnosis.[2]

Recognizing Crisis Level Symptoms

Sometimes antibiotic therapy is not successful. The critical care nurse is usually the first person to determine that the desired results are not occurring. In this case the nurse reassesses the patient and informs the physician about the patient's acute condition which may require immediate cardiac surgery.

The major cause of death among patients with Infective Endocarditis is congestive heart failure secondary to valvular dysfunction that is refractory to traditional medical management. Surgical intervention may be lifesaving in certain endocarditis patients. Indications for surgical intervention include:

- Valvular dysfunction with congestive heart failure
- Congestive heart failure not controlled by medical therapy
- Infection uncontrolled by antibiotic therapy

- Relapse of prosthetic valve endocarditis after appropriate antibiotic therapy
- Perivalvular/myocardial abscesses
- Coagulase negative staphylococcal endocarditis less than one year after cardiac valve replacement surgery
- Recurrent systemic emboli
- Acquired heart block/intraventricular conduction defects
- Pericarditis
- Mycotic aneurysm of a coronary artery or Sinus of Valsalva
- Atrial and ventricular septa rupture[21]

Evaluate the patient for the development of symptoms indicating progression of these clinical indications. Progression of the symptoms to the following level is significant and should be reported immediately:

- Pulmonary crackles
- New onset of S3 or S4
- New onset cardiac murmur
- Chest pain
- Jugular venous distention (JVD)
- Positive hepatojugular reflex (HJR)
- Sustained tachycardia
- Sustained hyperthermia, tachypnea, and dyspnea

Aggressive surgical therapy used for patients with these severe complications has reduced the mortality of congestive heart failure complications from 66% to 9 to 14%.[2]

Preparing for Emergency Surgery

If surgical therapy is indicated then preoperative teaching and preparation of the patient and family becomes the responsibility of the critical care nurse. Valvular replacement surgery of the infected valve is the recommended surgical procedure. During surgery the valvular lesion is corrected with prosthetic valve replacement, the infected tissue is excised and any abscesses are unroofed.[2]

The patient and family need to be instructed regarding the type of surgical incision. They should understand that post-operatively the sternum will be held together with stainless steel wires to promote healing by stabilizing and immobilizing the sternum. Describing the major aspects of the surgical procedure that will guide the patient's post-operative nursing management is a primary responsibility of the critical care nurse. If desired, the patient and family can be shown and be allowed to touch a prosthetic valve with the use of a heart model or anatomic pictures. The critical care nurse can then explain how this surgical procedure will correct hemodynamic abnormalities and symptoms.

The family needs to be reassured that they will be kept up-to-date during surgery regarding the patient's progress. they also need to be aware of how the patient will look after surgery.

Providing Patient Teaching

Initiate a teaching program for both the patient and family once acute management has been initiated and the patient stabilized. A teaching program for the patient with Infective Endocarditis should address the following key areas of information:

- Nature of the infection and effects on the heart
- Identification of predisposing factors and their relationship to future susceptibility
- Signs and symptoms of the disease with specific indications regarding the notification of a health care provider
- Current and prophylactic antibiotic regimens and side effects
- Self-care instructions that include oral hygiene guidelines, suggestions for avoiding upper respiratory infections, identification of situations that require prophylactic antibiotic therapy
- Necessary post discharge follow-up care

The American Heart Association Committee Report on the Prevention of Bacterial Endocarditis states that the maintenance of oral hygiene is a key factor in reducing potential sources of bacterial seeding.[22] The critical care nurse teaches the patients the following information regarding oral hygiene:

- Request prophylactic antibiotic therapy with all dental procedures including routine professional cleaning.
- Avoid pressurized oral water cleaning devices and/or dental flossing which causes gingival bleeding and thus introduces bacteria into the blood stream.
- Have dentures fitted properly and promptly, and seek treatment for ulcers produced by ill fitting dentures
- Adhere to daily oral hygiene practices[22]

The critical care nurse instructs the patient and family that

surgical and diagnostic procedures involving the genitourinary and gastrointestinal tract are high risk procedures for the introduction of bacteria. In particular, the patient with a prosthetic valve needs to be instructed on the importance of initiating antibiotic therapy prior to these types of surgical or diagnostic procedures.[22]

The critical care nurse provides patients and family members with information regarding specific symptoms that suggest infection and the need to seek prompt medical treatment for these symptoms. Emphasize the importance of reporting fever, chills, weakness, malaise, fatigue, and weight loss, especially within four weeks after completing antibiotic therapy.[1] Inform patients where to get an emergency identification card which will inform health-care professionals that the patient has had Infective Endocarditis so proper prophylactic treatment can be started if the patient suffers from trauma or infectious disease.

Conclusion

Comprehensive care provided by the critical care nurse to the Infective Endocarditis patient contributes significantly toward a successful patient outcome. Essential elements of critical care nursing practice for facilitating successful recovery and reduction of mortality from Infective Endocarditis include: early identification in the hospitalized population of predisposing risk factors, prompt recognition of signs and symptoms, expert nursing management of the patient at risk for or diagnosed with Infective Endocarditis, and teaching about future preventive techniques.

Clinical Research Questions

There are still many clinical research questions still needing investigation regarding the nursing care of Infective Endocarditis patients. These include:

- What socioeconomic factors increase the risk of developing Infective Endocarditis?
- What is the relationship between early identification of the signs and symptoms of Infective Endocarditis and the incidence of valvular dysfunction?
- How is patient recovery affected by timely administration of prescribed antibiotics?
- How does a formal patient teaching program affect the reoccurrence of Infective Endocarditis?

Key Words

Endocarditis, sepsis, cardiovascular disease.

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Acknowledgement

The authors would like to thank Darrell Hulisz, BS, Pharm.D., Pharmacotherpy Specialist, Akron General Medical Center, Akron, OH; Assistant Professor, Ohio Northern University for assistance on this manuscript.

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