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Infective Endocarditis

Infective endocarditis includes acute and sub-acute bacterial endocarditis, as well as nonbacterial endocarditis caused by viruses, fungi, and other microbiologic agents. Diagnosis may be difficult when delayed; and special risk groups have emerged, including intravenous drug users, survivors of cardiac surgery, patients taking immunosuppressant medications, and patients who require chronic intravascular catheters. Many patients get endocarditis on what was thought to be a previously healthy native valve. Furthermore, endocarditis from oral flora may occur without a preceding dental procedure.

ETIOLOGY

Viridans-type streptococci (α-hemolytic streptococci) and *Staphylococcus aureus* are the leading causative agents for endocarditis in pediatric patients. In ≈6% of cases, blood cultures are negative for any organisms. No relationship exists between the infecting organism and the type of congenital defect, the duration of illness, or the age of the child.

1-Staphylococcal endocarditis is more common in patients with no underlying heart disease.

2- Viridans group streptococcal infection is more common after dental procedures.

3-Group D enterococci are seen more often after lower bowel or genitourinary manipulation.

4- Pseudomonas aeruginosa or Serratia marcescens is seen more frequently in intravenous drug users.

5-Fungal organisms are encountered after open heart surgery.

6- Coagulase-negative staphylococci are common in the presence of an indwelling central venous catheter.

EPIDEMIOLOGY

In developed countries, congenital heart disease is the overwhelming predisposing factor. Endocarditis is rare in infancy; in this age group, it usually follows open heart surgery or is associated with a central venous line.

Patients with congenital heart lesions in which blood is ejected at high velocity through a hole or stenotic orifice are most susceptible to endocarditis. Vegetations usually form at the site of the endocardial or intimal erosion that results from the turbulent flow. Children with ventricular septal defects (VSDs), left-sided valvular disease such as aortic stenosis, tetralogy of Fallot, and systemic-pulmonary arterial communications (patent ductus arteriosus or Blalock-Taussig shunts) are at highest risk. Surgical correction of congenital heart disease may reduce but does not eliminate the risk of endocarditis, with the exception of repair of a simple atrial septal defect or patent ductus arteriosus. Children who have undergone valve replacement or valved conduit repair are also at high risk.

In ≈30% of patients with infective endocarditis, a predisposing factor is recognized. A surgical or dental procedure can be implicated in ≈65% of cases in which the potential source of bacteremia is identified. Poor dental hygiene in children with cyanotic heart disease results in a greater risk for endocarditis. Primary bacteremia with *Staphylococcus aureus* is another risk for endocarditis (10% risk).The occurrence of endocarditis directly after heart surgery is relatively low

CLINICAL MANIFESTATIONS

Early manifestations are usually mild, especially when viridans group streptococci are the infecting organisms. Prolonged fever without other manifestations (except, occasionally, weight loss) that persists for as long as several months may be the only symptom. **New or changing heart murmurs** are common, particularly with associated heart failure. Splenomegaly and petechiae are relatively common. Serious neurologic complications such as embolic strokes, cerebral abscesses, mycotic aneurysms, and hemorrhage are most often associated with staphylococcal disease and may be late manifestations. Myocardial abscesses may occur with staphylococcal disease and may damage the cardiac conducting system, causing heart block, or may rupture into the pericardium and produce purulent pericarditis. Such manifestations include **Osler nodes** (tender, pea-sized intradermal nodules in the pads of the fingers and toes), **Janeway lesions** (painless small erythematous or hemorrhagic lesions on the palms and soles), and **splinter hemorrhages** (linear lesions beneath the nails). These lesions may represent vasculitis produced by circulating antigen-antibody complexes.

DIAGNOSIS

The critical information for appropriate treatment of infective endocarditis is obtained from blood cultures. All other laboratory data are secondary in importance .Three to five separate blood collections should be obtained after careful preparation of the phlebotomy site. Contamination presents a special problem inasmuch as bacteria found on the skin may themselves cause infective endocarditis. The timing of collections is not important

because bacteremia can be expected to be relatively constant. In 90% of cases of endocarditis, the causative agent is recovered from the 1st two blood cultures. The laboratory should be notified that endocarditis is suspected so that, if necessary, the blood can be cultured on enriched media for longer than usual (>7 days) to detect nutritionally deficient and fastidious bacteria or fungi. Antimicrobial pretreatment of the patient reduces the yield of blood cultures to 50–60%. Other specimens that may be cultured include scrapings from cutaneous lesions, urine, synovial fluid, abscesses, and, in the presence of manifestations of meningitis, cerebrospinal fluid. Serologic diagnosis is necessary in patients with unusual or fastidious microorganisms .

The index of suspicion should be high when evaluating infection in a child with an underlying contributing factor. Two-dimensional echocardiography can identify the size, shape, location, and mobility of the lesion; when combined with Doppler studies, the presence of valve dysfunction (regurgitation, obstruction) can be determined and its effect on left ventricular performance quantified. Echocardiography may also be helpful in predicting embolic complications, given that lesions >1 cm and fungating masses are at greatest risk for embolization. The absence of vegetations does not exclude endocarditis.

The **Duke criteria** help in the diagnosis of endocarditis:-

Major criteria include (1) positive blood cultures (two separate cultures for a usual pathogen, two or more for less typical pathogens) and (2) evidence of endocarditis on echocardiography (intracardiac mass on a valve or other site, regurgitant flow near a prosthesis, abscess or new valve regurgitant flow). **Minor criteria** include predisposing conditions, fever, embolic-vascular signs, immune complex phenomena (glomerulonephritis, arthritis, rheumatoid factor, Osler nodes, Roth spots), a single positive blood culture or serologic evidence of infection, and echocardiographic signs not meeting the major criteria. Two major criteria, one major and three minor, or five minor criteria suggest definite endocarditis. The presence of newly diagnosed clubbing, splenomegaly, splinter hemorrhages, and petechiae; a high erythrocyte sedimentation rate; a high C-reactive protein level; and microscopic hematuria.

PROGNOSIS AND COMPLICATIONS

In the pre-antibiotic era, infective endocarditis was a fatal disease. Despite the use of antibiotic agents, mortality remains at 20–25%. Serious morbidity occurs in 50–60%, the most common is heart failure caused by vegetations involving the aortic or mitral valve. Myocardial abscesses and toxic myocarditis may also lead to heart failure. Pulmonary emboli may occur in children with VSD or the tetralogy of Fallot. Other complications include mycotic aneurysms, rupture of a sinus of Valsalva, obstruction of a valve secondary to large vegetations, acquired VSD, and heart block as a result of involvement

(abscess) of the conduction system. Additional complications include meningitis, osteomyelitis, arthritis, renal abscess, and immune complex–mediated glomerulonephritis.

TREATMENT

Antibiotic therapy should be instituted immediately once a definitive diagnosis is made. Several weeks are required for a vegetation to organize completely; A total of 4–6 wk of treatment is recommended. Depending on the clinical and laboratory responses, antibiotic therapy may require modification and, in some instances, more prolonged treatment is required. With highly sensitive viridans group streptococcal infections, shortened regimens that include oral penicillin for some portion have been recommended.

Digitalis, salt restriction, and diuretic therapy are used for the treatment of heart failure. Surgical intervention for infective endocarditis is indicated for severe aortic or mitral valve involvement with intractable heart failure. Other surgical indications include failure to sterilize the blood despite adequate antibiotic levels, myocardial abscess, recurrent emboli, new heart block, and increasing size of vegetations while receiving therapy. Although antibiotic therapy should be administered for as long as possible before surgical intervention, active infection is not a contraindication if the patient is critically ill as a result of severe hemodynamic deterioration from infective endocarditis. Removal of vegetations and, in some instances, valve replacement may be lifesaving, and sustained antibiotic administration will most often prevent reinfection. Replacement of infected prosthetic valves carries a higher risk.

Fungal endocarditis is difficult to manage and has a poorer prognosis. It has been encountered after cardiac surgery, in severely debilitated or immunosuppressed patients, and in patients on prolonged courses of antibiotics. The drugs of choice are amphotericin B (liposomal or standard preparation) and 5-fluorocytosine. Surgery to excise infected tissue is occasionally attempted, though often with limited success.

PREVENTION

Antimicrobial prophylaxis before various procedures and other forms of dental manipulation may reduce the incidence of infective endocarditis in susceptible patients . Proper general dental care and oral hygiene are most important in decreasing the risk of infective endocarditis in susceptible individuals.