INFECTIVE ENDOCARDITIS

is due to microbial infection of a heart valve (native or prosthetic), the lining of a cardiac chamber or blood vessel, or a congenital anomaly (e.g. septal defect),as in areas of endocardial damage caused by a high-pressure jet of blood, such as ventricular septal defect, mitral regurgitation and aortic regurgitatio -

in. The underlying condition : rheumatic heart, congenital heart disease in and some other cardiac abnormality (e.g. calcified atrial valve, floppy mitral valve) ,The remainder (32%) were not thought to have a pre-existing cardiac abnormality.

The causative organism is usually a bacterium. But: staphylococcal endocarditis of the tricuspid valve is a common complication of intravenous drug misusers. In contrast, the risk of endocarditis at the site of many haemodynamically important low-pressure lesions (e.g. a large atrial septal defect) is negligible. The avascular valve tissue and presence of fibrin aggregates help to protect proliferating organisms from host defence mechanisms, forming vegetations, composed of organisms, fibrin and platelets grow and may become large enough to cause obstruction; they may also break away as emboli. Adjacent tissues are destroyed and abscesses may form; valve regurgitation may develop or increase if the affected valve is damaged by tissue distortion, cusp perforation or disruption of chordae. Extracardiac manifestations such as vasculitis and skin lesions are due to emboli or immune complex deposition.

The *viridans* group of are commensals in the upper respiratory tract that may enter the blood stream on chewing or teeth-brushing, or at the time of dental treatment, and are common causes of subacute endocarditis .Other organisms, including *Enterococcus faecalis*, *E. faecium* and *Strep. bovis*, may enter the blood from the bowel or urinary tract.

Staph. aureus is a common cause of acute endocarditis, originating from skin infections, abscesses or vascular access sites (e.g. intravenous and central lines), or from intravenous drug misuse. It is a highly virulent and invasive organism, usually producing large vegetations, rapid valve destruction and abscess formation. Post-operative endocarditis after cardiac surgery may affect native or prosthetic heart valves or other prosthetic materials. The most common organism is a coagulase-negative staphylococcus *(Staph. epidermidis)*, which is a normal skin commensal. There is frequently a history of post-operative wound infection with the same organism.

In Q fever endocarditis (rare) due to *Coxiella burnetii*, the patient often has a history of contact with farm animals. The aortic valve is usually affected and there may be hepatic complications and purpura.

Brucella endocarditis (rare) is associated with a history of contact with goats or cattle and often affects the aortic valve.

Yeasts and fungi endocarditis (rare)*(Candida*, *Aspergillus)* may attack previously normal or prosthetic valves, particularly in immuno-compromised patients or those with indwelling intravenous lines. Abscesses and emboli are common(complicating fungal endocarditis), therapy is difficult (surgery is often required) and the mortality is high.

Common causative organisms, in elderlies: often enterococci (from the urinary tract) and *Streptococcus bovis* (from a colonic source).

DIAGNOSIS OF INFECTIVE TIS (MODIFIED DUKE CRITERIA)

Major criteria

- Positive blood culture :
  - Typical organism from two cultures
  - Persistent positive blood cultures taken > 12 hours apart
  - Three or more positive cultures taken over more than 1 hour
- Endocardial involvement
  - Positive echocardiographic findings of vegetations
  - New valvular regurgitation

Minor criteria

- Predisposing valvular or cardiac abnormality
- Intravenous drug misuse
- Pyrexia ≥38°C , prolonged course of fever – not responding to emperic AB. Tratement – no apparent focus of infection.
- Embolic phenomenon (cerebral emboli/ Roth spots in retina/ petechial skin spots/petechial spots on oral or subconjuctival mucus membranes / nail fold infarcts
- Vasculitic phenomenon (hematuria : glomerulonephritis) / cerebral infarct.)
- Blood cultures suggestive-organism grown but not achieving major criteria
- Suggestive echocardiographic findings (abnormal valve cusps / shunting)

- **Definite endocarditis:** two major, or one major and three minor, or five minor
- **Possible endocarditis:** one major and one minor, or three minor

Three sets of blood cultures should be taken prior to commencing therapy, and these need not wait for episodes of pyrexia. Aseptic technique is essential and the risk of contaminants should be minimized by sampling from different venepuncture sites. An in-dwelling line should not be used to take cultures. Aerobic and anaerobic cultures are required.

Echocardiography is the key investigation for detecting and following the progress of vegetations, for assessing valve damage and for detecting abscess formation.

Transoesophageal echo is valuable for identifying abscess formation and investigating patients with prosthetic heart valves. Failure to detect vegetations

does not exclude the diagnosis and should not delay treatment.

Elevation of the ESR, a normocytic, normochromic anaemia and leucocytosis are common. Microscopic haematuria is usually present.

ECG may show the development of atrioventricular block (due to abscess formation).

Management

Any source of infection should be removed as soon as possible; for example, a tooth with an apical abscess should be extracted.

Empirical treatment depends on the mode of presentation, the suspected organism, and whether the patient has a prosthetic valve and/or penicillin allergy.

If the presentation is acute, flucloxacillin and gentamicin are recommended, and for a subacute or indolent presentation, benzyl penicillin and gentamicin.

In those with either penicillin allergy, a prosthetic valve or suspected meticillin-resistant *Staph. aureus* (MRSA) infection, triple therapy with vancomycin, gentamicin and oral rifampicin should be considered.

By culture and sensitivity,determination of the minimum inhibitory concentration (MIC) is essential to guide antibiotic therapy.

A 2-week treatment regimen may be sufficient for fully sensitive strains of *Strep. viridans* and *Strep Bovis*

(2 weeks : combination therapy)- followed by other 2 weeks with benzyl penicillin alone)- if sensitive , IF not: 4 by 4.;Enterococci: 4 weeks, for native valves / 6 weeks ,, for prosthetic valves.

Cardiac surgery (débridement of infected material and valve replacement) for those with *Staph. aureus* and fungal infections ; antimicrobial therapy must be started before surgery.

INDICATIONS FOR CARDIAC SURGERY IN INFECTIVE ENDOCARDITIS

- Heart failure due to valve damage
- Failure of antibiotic therapy (persistent or uncontrolled infection)
- Large vegetations on left-sided heart valves with evidence or'high risk' of systemic emboli
- Abscess formation

Patients with prosthetic valve endocarditis or fungal endocarditis often require cardiac surgery.

ANTIBIOTIC PROPHYLAXIS AGAINST ENDOCARDITIS

Procedure	**Antibiotic regimen**
Dental or upper respiratory tract procedures under general anaesthetic	Amoxicillin 1 g i.v. at induction *plus* amoxicillin 0.5 g orally 6 hrs later
If allergic to or received penicillin in last month	Vancomycin 1 g i.v. infusion over at least 100 mins *plus* gentamicin 120 mg i.v. at induction
Special-risk patients, i.e. prosthetic valve or previous endocarditis Genitourinary procedures	Amoxicillin 1 g i.v. *plus* gentamicin 120 mg i.v. at induction *plus* amoxicillin 0.5 g orally 6 hrs later
If allergic to or received penicillin in last month	Vancomycin 1 g i.v. infusion over at least 100 mins *plus* gentamicin 120 mg i.v. at induction

Obstetric and gynaecological procedures or gastrointestinal surgery/instrumentation-treat only special-risk patients.