Infective endocarditis

History
Lazerous Riverius recorded the first case of what is now known as infective endocarditis in 1723. He described a French magistrate with an irregular pulse, oedema, and congestion, who at autopsy had fleshy masses 'the size of hazelnuts' obstructing the aortic ostia[2]

Epidemiology
Incidence Unknown but at least 25/1000000 population
200 deaths/year-about 20% die
Increased risk >50years. The risk of acquiring IE is higher among patients with valvular heart disease (e.g., rheumatic valves, bicuspid aortic valves, myxomatous degeneration, etc.), congenital heart disease (e.g., coarctation, patent ductus arteriosus, ventricular septal defect, etc.), prosthetic cardiac valves, and among intravenous drug abusers [1]

Pathogenesis
Endothelial damage to valves---thrombotic vegetation----binding of blood borne microbes to thrombus.
In endocarditis the vegetations are found predominately on the left side of the heart (95 per cent). In a large autopsy series of more than 1000 cases reported over 50 years ago the mitral valve was involved in 86 per cent, the aortic in 55 per cent, the tricuspid in 20 per cent, and the pulmonary valve in only 1 per cent[2]
However, IVDA have a different valve involvement. The tricuspid valve is the most frequently affected (60% to 70%), follo wed by the mitral and aortic valves (20% to 30%); pulmonic valve infection is rare (< 1%). More than one valve is infected in 5% to 10% of cases. HIV-positive IVDAs have a higher ratio of right-sided IE and S. aureus IE than HIV-negative IVDAs.[3]
Symptoms caused by valve damage, heart failure, sepsis, septic emboli and circulating immune complexes

Clinical
Night sweats, anorexia, and weight loss were followed by the development of splinter haemorrhages and Osler nodes, Janeway lesions, vasculitic rash, nephritis, finger clubbing, and splenomegaly. The mean age has risen from under 40 years before 1940 to between 60 and 70 years today.
pyrexia, rigors, malaise, anorexia, headache, confusion, arthralgia, and anaemia murmurs
Emboli may lodge in any part of the circulation and present as a cerebrovascular accident-30% present with neurological problems, arterial occlusion of a limb, myocardial infarction, sudden unilateral blindness, or infarction of the spleen or a kidney. In right-heart endocarditis, recurrent septic pulmonary emboli may be misinterpreted as 'pneumonia'[2]

Diagnosis
Clinical-Duke Criteria

Major criteria	Minor criteria
1. Positive blood culture for infective carditis	1. Predisposition: predisposing heart condition or intravenous drug use
A. Typical micro-organism consistent with infective carditis from two separate blood cultures as noted below:	2. Fever: temperature > 38°C
(i) viridans streptococcia,*Streptococcus bovis*,	3. Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial haemorrhages, and Janeway's lesions
HACEK group or	
(ii) community-acquired *Staphylococcus aureus* orenterococci, in the absence of a primary focus or	4. Immunological phenomena: glomerulonephritis, Osler's nodes, Roth spots, and rheumatoid factor
B. Micro-organisms consistent with infective carditis from persistently positive blood cultures defined as: (i) at least two positive cultures of blood samples drawn > 12 h apart or	5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted in column oneb or serological evidence of active infection with organism consistent with infective carditis
(ii) all three of these or a majority of four or more separate cultures of blood (with first and last sample drawn at least 1 h apart).	6. Echocardiographic findings: consistent with infective carditis but do not meet a major criterion as noted in column one
2. Evidence of endocardial involvement	
A. Positive echocardiogram for infective carditis as defined as: (i) oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomical explanation or	
(ii) abscess or,	
(iii) new partial dehiscence of prosthetic valve.	
B. New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)	

Blood Cultures x3-any bugs
Viridans Strep species
Stapylococcus (esp common IVDA + Clamydia)
CRP and ESR up
Dipstick urine-proteiuria and haematuria
Serology (*Coxiella burnetii* (Q fever), bartonella, and chlamydia endocarditis)
Echocardioography-TOE, vegetations over 1-2mm. Vegetations, which in general tend to be larger on the right side, can be demonstrated in 80 to 100 per cent of cases[2]

Treatment
Antibiotics-local, after positive cultures if possible. If acute-emperic eg vancomycin/gentamycin OR benzyl pen/gent
?fluclox if IVDA

Surgery – valve excision/replacement/repair/repair of complications eg fistula
Surgery will be required in about 30 per cent of cases during the acute phase (first 4 months) of endocarditis and 20 to 40 per cent of cases thereafter[2]

References

1. Tak T. Reed KD. Haselby RC. McCauley CS Jr. Shukla SK. 'An update on the epidemiology, pathogenesis and management of infective endocarditis with emphasis on Staphylococcus aureus'. [Review] [51 refs] Source WMJ. 101(7):24-33, 2002.
2. Oxford Textbook of Medicine (Online via doctors.org)
3. Miro JM. del Rio A. Mestres CA. 'Infective endocarditis in intravenous drug abusers and HIV-1 infected patients'. [Review] [123 refs] Source Infectious Disease Clinics of North America. 16(2):273-95, vii-viii, 2002 Jun.
3.