Summary

The case records of nineteen patients, with infective endocarditis, including four with recurrent episodes, were reviewed. The diagnosis required a high index of suspicion in most cases. Rheumatic and congenital valve abnormalities were the major risk factors and dental, periodontal disease and surgical instrumentation were the precipitating sources of infection.

Organisms were cultured from fifteen patients and included streptococci, staphlococci, coxiella burnetti and brucella abortus. Four patients had negative cultures in life. Five patients required valve surgery as part of their management. Early referral of all patients with endocarditis to cardiac centres is recommended.

Introduction

Infective endocarditis is uncommon and is often a difficult diagnosis to establish. Much has been written about the changing spectrum of the disease, of host-variables, of the nature and sensitivity of responsible organisms and the reasons for recurrent attacks (Wilson and Washington, 1977). With this in mind it was decided to review the case notes of patients identified by the medical records department as being treated for endocarditis in the cardiac department of the hospital between November 1973 and February 1983 and compare the "Irish experience" with series from abroad with regard to responsible organisms, patient risk factors, management requirements and subsequent morbidity and mortality. The diagnosis of endocarditis was established by the association of positive blood cultures with valvular lesions or by valve cultures at cardiac surgery or at autopsy.

Results

Nineteen patients were found to have infective endocarditis, seventeen were male and two were female. The age range was from fifteen to sixty-three years (mean 41.8 years). Ten patients were from an urban and nine from a rural background. The mean duration of symptoms prior to hospital admission was forty-three days (range 1-365 days). Two patients had symptoms for more than six months before diagnosis. Surprisingly two patients were identified as having endocarditis when admitted for unrelated problems. In the other fifteen, symptoms were only present for an average of fourteen days before diagnosis.

Six patients gave a definite previous history of rheumatic fever and four of these were known to have pre existing valve lesions. One had suffered three separate episodes of rheumatic fever. Five patients had congenital heart disease. One had a patent ductus arteriosus, one had Tetralogy of Fallot with a Blalock Shunt in situ and three were found to have bicuspid aortic valves. A further patient had severe mitral valve prolapse and yet another had mitral regurgitation of uncertain aetiology.

The probable source of infection was the teeth and gums in five patients, a fissure-in-ano in one, an anal stretch procedure in another, and open heart

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surgery for aortic valve replacement in another.

Thus thirteen patients had underlying cardiac disease, eight had an active source of infection or recent procedure likely to be associated with bacteraemia and in only two was no predisposing cardiac condition or source of infection evident. Drug abuse was not a factor in these patients.

Clinical Signs and Symptoms

There are no specific symptoms for endocarditis. Table I illustrates the variety of presenting complaints in our nineteen patients. Most of these raise the possibility of infection without indicating its location.

In four patients the underlying endocarditis was obscured by a dramatic neurological presentation; one patient presented with bacterial meningitis, one with grand mal seizures and two with hemiparesis, one due to a cerebral abscess and one to a sterile embolus. The asymptomatic patient was found to have endocarditis at the time of an elective aortic valve replacement. The infecting organism was brucella abortus.

On admission cardiac murmurs were heard in all nineteen patients. Murmurs arising from the aortic valve were present in seven, from the mitral valve in six and from both in four. The patient with the patent ductus arteriosus had a loud systolic pericardial friction rub and a prominent diastolic murmur at the left sternal edge. At first he was thought to have aortic endocarditis. The patient with the Blalock shunt had the usually associated murmur. Five patients were noted to have changing murmurs in the early stages of their illness.

Five patients were afebrile on admission and in five others the temperature was less than 37.5°C. Nine patients had or subsequently developed splinter haemorrhages. In five the spleen was palpable on admission and in one the spleen subsequently enlarged. Two patients had Roth spots and one had Janeway lesions. One patient had a pleural and pericardial effusion. Two patients had finger clubbing but one of these had congenital cyanotic heart disease.

Investigations

Microscopic haematuria was present in ten patients. Eight had a white cell count of greater than 11 x 10⁹/L on admission. The ESR (Westergreen Method) was measured in seventeen of the nineteen patients on admission and was less than 40 mm/hr in nine, between 40-48 mm/hr in four, and greater than 80 mm/hr in four. The haemoglobin level was less than 10 g/dL in two patients. In three of thirteen patients in whom the test was performed, the rheumatoid factor was weakly positive.

Nine patients had normal ECG recordings throughout their illness, three tracings showed first degree AV block, one showed atrial fibrillation and six showed left ventricular hypertrophy. One of the latter patients transiently developed atrial fibrillation after admission.

Chest x-rays were helpful in establishing the presence of pulmonary oedema, and in assessing cardiac size. Five patients had evidence of left ventricular failure, seven had left ventricular enlargement and two showed prosthetic