

Short reports

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Fulminant diphtheritic mitral valve endocarditis

This report of a case of fulminant mitral valve endocarditis due to a nontoxogenic strain of *Corynebacterium diphtheriae* serves to emphasize that any organism isolated should be fully identified and should be regarded as a possible pathogen.

Case report

An 11-year-old White boy complained of headache, generalized body pain, and left-sided chest pains for 2 days. He had been well beforehand. He had been fully immunized against diphtheria as an infant and had received boosters of toxoid at 5 and 10 years of age. There was no history of rheumatic fever or of the presence of a heart murmur. Two months before he had had two teeth extracted without antibiotic cover and a month later he had developed a septic sore, the result of a thorn in his hand. This had been removed and he was given penicillin by mouth for 5 days.

Examination at another hospital showed no abnormality except fever. X-ray examination of the chest showed a normal cardiac shadow and lung fields. His Hb was 14.0 g/dl and the total white cell count 9900/mm³ with 90% polymorphonuclear cells. A provisional diagnosis of pleurodynia was made and acetylsalicylic acid was prescribed. When he was seen by one of us 6 days after the onset of his illness he was toxic, ill, dyspnoeic, and his temperature was 39 °C. The right lower leg felt cold and a heart murmur of mitral regurgitation was heard. He was admitted to hospital.

On admission the patient's left ventricular impulse was forceful and a grade 4/6 apical pansystolic murmur was heard. The liver was 3 cm below the costal margin but the spleen was not palpable. No faucial, nasal, or cutaneous lesions were present. The right lower limb felt colder than the left but the dorsalis pedis pulses seemed equal. An electrocardiogram showed a sinus tachycardia with a rate of 140 min, an AQRS of +60°, low voltage complexes in the standard and the extremity leads, and a QRS width of 0.10 s, suggesting an intraventricular delay. No ST-T changes or evidence of chamber hypertrophy were present. X-ray examination of the chest showed a slight increase in heart size when compared with the films taken 4 days earlier, the cardiothoracic ratio being 0.52%. There was no evidence of enlargement of the cardiac chamber and the upper zone pulmonary veins were slightly distended.

Immediately after withdrawing blood for a single blood culture (from which no organisms were isolated) soluble penicillin 500 000 IU was given intravenously every hour. Digoxin, frusemide, and oxygen were also

given. Hb was now 11.1 g/dl, the white cell count 17 100 with 72% polymorphonuclear cells, and the erythrocyte sedimentation rate (Wintrobe) 30 mm/h. The patient remained ill and febrile, dyspnoea progressed in severity, the liver gradually increased in size, and increasing restlessness required sedation. Within 24 hours of admission splinter haemorrhages were seen in the finger-nail beds and petechiae were present in the conjunctivae. The right lower extremity remained colder than the left, suggesting an embolic occlusion of the major vessel. The spleen could still not be felt. Despite vigorous medical treatment urinary output dropped, and before emergency mitral valve replacement could be initiated intractable pulmonary oedema resulted in death 30 hours after admission to hospital.

Necropsy findings. At the request of the parents necropsy was limited to the heart. It weighed 170 g and was generally dilated. The cavity of the left ventricle was enlarged and the free margin and atrial surface of the mitral valve curtains were covered with pale, friable vegetations. These extended on to the posterior wall of the atrium (McCallum's area) and along the chordae tendinae to the papillary muscles. One large polypoidal vegetation projected into the ventricular cavity. The valve was not eroded. The chordae were not thickened and neither the mitral nor aortic valves showed evidence of previous rheumatic valvulitis. There were subendocardial petechial haemorrhages on the septum beneath the aortic valve. The right atrium and ventricle appeared normal apart from dilatation. No congenital cardiac lesions were present.

Histopathology. The atrial surface of the mitral valve was covered with large fibrinous vegetations showing masses of Gram-positive bacilli. The vegetations were spreading as a thin layer on to the ventricular surface. The valve tissue showed moderate polymorph infiltration and endothelial proliferation but no organization. In the myocardium there were scattered petechial haemorrhages and focal necroses of the Bracht-Wachter type. There was no indication of previous or active rheumatic carditis.

Bacteriology. Many Gram-positive bacilli were seen on direct smears from the cardiac vegetations and Albert's stain showed them to have metachromatic granules. The vegetations were cultured on blood agar and thioglycollate medium. After 24 hours' incubation at 37 °C in 5% carbon dioxide there was a heavy growth of colonies 1–2 mm in diameter. These were inoculated on to tellurite medium and biochemical tests were done. Glucose and maltose were fermented but not lactose, sucrose, or salicin. Catalase and nitrate reduction were positive and motility, gelatin liquefaction, starch hydro-

lysis, and urease were negative. On tellurite medium the colonies were medium in size, convex, rough, and dark grey. The toxigenicity test using an Elek plate was negative. The organism was identified as a nontoxicogenic *Corynebacterium diphtheriae*, gravis type. Dr. R. E. Weaver, Communicable Disease Centre, Atlanta, U.S.A., confirmed the identification. Sensitivity test results in minimum inhibitory concentrations ($\mu\text{g/ml}$) were as follows: gentamicin 0.01, penicillin 1.0, tetracycline 0.5, cephalothin 0.5, chloramphenicol 1.0, erythromycin 0.01, clindamycin 0.06, and lincomycin 0.25.

Discussion

Corynebacterial endocarditis is rare. In most reports the organisms are described as a 'diphtheroid' without detailed identification (Merzbach *et al.*, 1965; Reid and Greenwood, 1967; Davis *et al.*, 1963; Dismukes *et al.*, 1973; Manhas *et al.*, 1972; Stein, Harken, and Dexter, 1966). In most cases the organisms have been isolated from blood cultures in living patients, but in many of these cases other organisms were isolated in addition to the corynebacteria (Reid and Greenwood, 1967; Stein *et al.*, 1966). 'Diphtheroids' have also been isolated from blood cultures after cardiopulmonary bypass surgery for the repair of cardiac valvular defects (Davis *et al.*, 1963) and from infected prosthetic valves (Dismukes *et al.*, 1973; Manhas *et al.*, 1972; Stein *et al.*, 1966). The latter may present either early or late in the postoperative period.

Pike (1951) reported a case of endocarditis due to toxogenic *C. diphtheriae* and referred to earlier reports of cases in which nontoxicogenic organisms were isolated. Facial, nasal, or cutaneous diphtheritic lesions may or may not be present in such cases. Corynebacterial species are widely distributed, being found in the soil and atmosphere and as contaminants in blood cultures. Certain strains, including *C. hofmanni*, *C. xerosis*, and nontoxicogenic *C. diphtheriae*, are saprophytes in man. They are therefore potential causes of endocarditis.

Bacterial endocarditis in most cases affects valves deformed by acquired or congenital heart disease. Our patient had no congenital heart lesion and the macroscopical and microscopical appearances of the chordae tendinae and myocardium seemed to exclude antecedent rheumatic carditis. Normal valves may occasionally be the seat of endocarditis, particularly when caused by virulent organisms. The implication of our case is that any organism isolated from a patient with a clinical diagnosis of infective endocarditis should be regarded as the possible pathogen and not dismissed as a contaminant. Organisms should be identified fully and

not reported in vague terms such as 'diphtheroid', so that a more accurate idea of the source and nature of the organism may be obtained.

Summary

Fulminant endocarditis affecting the mitral valve in an 11-year-old boy was caused by a nontoxicogenic strain of *Corynebacterium diphtheriae*.

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Peritoneal dialysis and exchange transfusion in a neonate with argininosuccinic aciduria

Peritoneal dialysis has been used in conditions such as renal, cardiac, and hepatic failure. Although it has been used in the management of coma in urea cycle deficiency states (Siegel and Brown, 1973), its effectiveness in controlling ammonia intoxication has been questioned (Siegel and Brown, 1973; Saudubray *et al.*, 1973). In a patient with