ACUTE BACTERIAL ENDOCARDITIS WITH A POSITIVE BLOOD CULTURE IN A CHILD AGED ONE YEAR

BY

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Cases of acute bacterial endocarditis in infancy are reported from time to time. Sansby and Larson (1930) give a review of the literature and report a case in an infant of five weeks old. MacGregor and McKendry (1944) report a case of foetal endocarditis, the infant being cyanotic through its fifteen days of life. The valve cusps showed nodular thickenings and other signs of inflammatory changes. Dean (1912) reports a case due to the pneumococcus in a child of three years in which both mitral and tricuspid valves were affected, and he comments on the fact that the valves on the right side of the heart were involved. The pneumococcus was found in the cerebrospinal fluid before death, in the heart blood before the heart was opened, and in films made from the surface of the brain. There was no pneumonia.

Endocarditis in infancy is most likely to be caused by the streptococcus, especially if a massive dose results from an acute throat infection. Endocarditis resulting from chronic tonsillitis and repeated infections, such as those met with in rheumatism, is not usually seen in infancy though it is at a later age. Holt and McIntosh report that in a thousand autopsies they found no case of endocarditis under three years old.

Brandes (1933) reports a case of gonococcal endocarditis of the tricuspid valve in an infant of ten days suffering from gonorrheal conjunctivitis. The child had a patent ductus arteriosus which may have contributed to the endocarditis. Kehr and Adelman (1942) report a case due to a haemolytic streptococcus, and Sanders (1933-4) a case associated with pyodermic infection. Wolff (1940) records two cases in infants diagnosed during life and treated as out patients and cured after severe illnesses: one of them had a purulent pericarditis. Both cases were staphylococcal in origin, from furuncles at the back of the head. These cases were treated by blood intravenously, and intramuscularly by small doses of prontosil and vitamin C. Recovery was apparently complete.

Clinical Aspects

In Sansby and Larson’s (1930) case there was a sudden onset with fever, rapid loss of weight, frequent stools, and a dulled sensorium suggesting a toxicosis. Treatment had no effect. No murmur was detectable in the heart and only an autopsy explained the rapid progress of the illness.

Case History

Our case showed the following history and clinical features and pathological findings.

S. H., a female, aged one year and one month, admitted on April 25, 1946, died on May 8, 1946. Delivery had been normal. The history was uneventful until the age of three months, when she had attacks of lethargy. In these she was ill and lifeless and feverish for about ten days. The first attack was diagnosed as bronchitis, the second as gastro-enteritis. With these attacks the ears discharged. In September, 1945, she was admitted to Booth Hall Hospital with severe gastro-enteritis and dehydration, and she had a transfusion. Dr. Patterson, Medical Superintendent, reports that there were no cardiac murmurs when she was discharged.

On examination on April 25, 1946, there was a mucoid discharge from the right ear, a loud systolic murmur all over the precordium, and tachycardia; a diagnosis of endocarditis was made. The murmur did not suggest pericarditis. Dullness was present over the left side of the chest below the apex, with harsh breathing in the left axilla; the abdomen was a little tumid and the spleen palpable, but not readily so.

The child improved at first and tried to play in her cot, but she was still pale and the murmur had not diminished.

On April 25 a radiograph of the heart shadow showed only slight general enlargement, globular and suggestive of pericarditis. Some hilar thickening was present. A blood count showed 54 per cent. haemoglobin, 3,500,000 red cells and 6,800 white cells per ml. of blood, polymorphs 57 per cent. A blood culture showed a profuse growth of pneumococci. Sulphaguanidine and penicillin, 10,000 units three-hourly, were given, and later
sulphamezathine. An oxygen tent was used. On May 2, the heart sounds were free from murmur and blood culture negative, but the blood count showed 45 per cent. haemoglobin and 4,230,000 red cells per ml. of blood.

On May 8 the child became worse, and developed dyspnoea, extreme pallor, bronchial breathing, and râles suggesting terminal pneumonia. She died at 10 p.m. The temperature throughout showed only occasional rises up to 106° F. The pulse was very rapid, between 130 and 170 per minute. The respirations were slightly raised.

Post-mortem findings. There was slight wasting of the subcutaneous tissues. The heart (80 g.) was increased in weight for the age by about one-third. This was mainly due to hypertrophy of the left ventricle (see illustration, Plate VII). A large vegetation was present on the anterior curtain of the mitral valve (1 cm. broad by 0·7 cm. deep) and extending on to the chordae; a much smaller vegetation was present on the posterior curtain. There was no thickening of the cusps. Microscopically the large vegetation was found to consist mostly of thrombus which in some places was undergoing organization, whilst in other places there were groups of organisms surrounded by polymorphic leucocytes. The organisms were in pairs, gram positive, and lanceolate ovoid in shape. Polymorphs were also present in the valve cusps at the base of the vegetation, but the remainder of the valve appeared normal. The other valves were normal. Between the lobes of the lungs were fine adhesions. Above the bronchi at the hilum of each lung was a mass of glands (2 to 2·5 c.mm. in diameter). The outer surfaces of the lungs were dark red, but there were a few lighter pink patches which tended to be raised. The cut surface showed a similar appearance, i.e. mainly dark red with smaller pink patches scattered throughout. The lungs were indurated throughout the greater part. Microscopically, many of the alveoli were filled with macrophage cells (large cells with pale nuclei and abundant slightly eosinophilic cytoplasm); some also contained a little fibrin and other polymorphs. Relatively few alveoli were aerated. Some of the alveolar walls tended to be thick due to the large numbers of mononuclear cells in them; others were congested. A moderate number of bronchioles contained polymorphs and some macrophage cells, and were surrounded by a loose zone of lymphocytes which extended into the inter-lobula septa. The appearances were those of early resolution. Both mastoids were healthy; a little greenish mucoid material was present in both middle ears. The brain was slightly congested. The mesenteric lymph nodes were slightly enlarged. The spleen (40 g.) was nearly twice its normal weight; the Malpighian bodies were well defined. The other organs showed no significant abnormality.

Comment

The appearances were those of bacterial endocarditis of the mitral valve and resolving bronchopneumonia with marked macrophage reaction. The immediate cause of the left ventricular hypertrophy was not obvious.

REFERENCES


(For illustrations to this article see Plate VI, p. 258.)

REVIEWS


The handicaps to which the title refers are those produced by cerebral palsy. Mrs. Collis studied them under W. M. Phelps at the Children's Rehabilitation Institute at Cockeysville, Maryland, in 1941. She has been chief therapist at the L.C.C.'s Cerebral Palsy Unit since its inception in 1943. In her conception of the disease and in the physiotherapy she employs she follows Phelps's teaching, but unlike him she makes little or no use of splints or drugs.

Treatment is based on certain theories of muscular action:

From the point of view of movement cerebral palsies may be divided into five types, of which two are common: the spastic and the athetoid.

Volitional movement is initiated by the cortex, which controls contraction and relaxation of the muscle acting as prime mover. Contraction and relaxations of synergists, antagonists, fixators, etc., are laid down as an invariable action pattern in subcortical centres, ready to be activated by the cortical impulse.

In spastic parasesis the cortex is involved and many prime movers, particularly those put in tension
h.—Radiograph of B half an hour after barium meal, showing biloculation of the stomach and gross dilatation.
j.—Radiograph of B showing distortion of pyloric outlet and duodenal bulb.
k.—Radiograph of B showing marked redundancy and dilatation of colon as far as the splenic flexure.

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(1)—Heart, showing hypertrophy of left ventricle, and vegetation on anterior curtain of mitral valve.
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Arch Dis Child 1947 22: 253-258
doi: 10.1136/adc.22.112.253