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ABACTERIAL FORM OF ENDOCARDITIS WITH NECROSIS OF THE EARS

BY

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The case here reported, in which symmetrical necrosis affected the ears of a patient with the clinical features of subacute bacterial endocarditis, will be found of interest. Repeatedly negative blood cultures, failure to respond to penicillin, myocardial infarction, and death in uraemia were other unusual features.

Case Report

A labourer aged 30 was admitted to hospital on February 27, 1947. He had had rheumatic fever at the age of 6 and schizophrenia at 25. In August, 1946, he had been admitted to St. Mary Abbots Hospital complaining of headaches, rigors, and pain and bleeding of both pinnae. He had an irregular fever and showed signs of mitral stenosis and aortic incompetence. Seven blood cultures were sterile. The haemoglobin was 83%; white blood cells varied from 5,000 to 8,000 per c.mm. (neutrophils 31–60%, lymphocytes 29–40%, monocytes 5–27%). The urine contained red blood cells and occasional hyaline and granular casts. He was treated with penicillin, 80,000 units three-hourly for seven weeks—a total of 31,000,000 units. Some lowering of the temperature occurred, but there was little improvement in the general condition, and he discharged himself from hospital on October 30, 1946.

On admission to the Central Middlesex Hospital he complained of malaise, anorexia, and rigors. Ten days previously he had developed a sudden pain in his left arm which spread to the left of the sternum and lasted two days.

Examination revealed clubbing of nails, healthy teeth, a small haemorrhage in the right fundus, and regular pulse. Blood pressure 150/60. The heart was not enlarged. There were signs of mitral stenosis and aortic incompetence. The liver and spleen were enlarged to three fingerbreadths below the costal margin, and the spleen was tender. On the left pinna a tender blue area with incipient necrosis was present.

Investigations.—Eight blood cultures taken between February 28 and April 3 were sterile. Aerobic and anaerobic cultures were maintained for 14 and in some cases 21 days. The urine contained albumin and many red blood cells. Blood urea rose from 66 to 274 mg. per 100 ml. Plasma proteins: albumin 3 g., globulin and fibrinogen 4.5 g. per 100 ml. Takata-Ara, + + +. Thymol turbidity test, 15 units. Serum colloidal gold reaction, + + + + +. Haemoglobin fell from 56% to 40%, and the white blood cells varied from 5,000 to 16,000 per c.mm. The sedimentation rate (Westergren) rose from 98 to 124 mm. per hour. The Wassermann reaction was negative. The urea clearance was 12% of normal. Serial electrocardiograms showed the characteristic appearances of a recent anterior cardiac infarction (Fig. 1). Biopsy from the left pinna on March 27 showed many greatly dilated capillaries, some of which contained small projections of endothelium-covered fibrinoid material. Full pathological details have been published by Pagel (1949).

A diagnosis of subacute infective endocarditis was made. The cardiac infarction was considered embolic in origin. The patient had an irregular fever up to 101° F. (38.3° C.) for the first six weeks. After the eight sterile blood cultures penicillin was started, 200,000 units being given every three hours for six weeks—a total of 70,400,000 units. Although his temperature became lower his general condition deteriorated. On April 13 he awoke with pain in the right pinna, which was found to be reddened and swollen, with mottled blue areas, and a few days later necrosis appeared. Meanwhile separation of the necrotic portion of the left pinna occurred (Fig. 2). He died in uraemia on June 10, 1947.

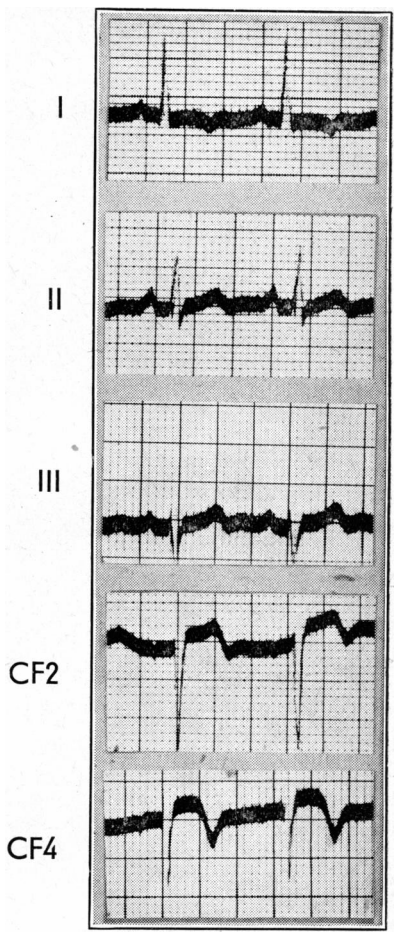

FIG. 1.—Electrocardiogram taken on March 4, 1947, showing changes of recent anterior cardiac infarction.

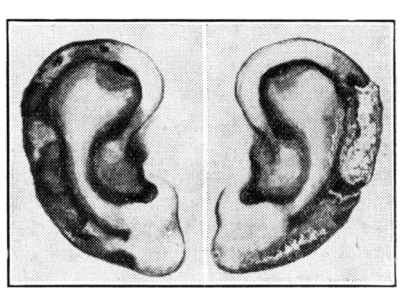

FIG. 2.—Necrosis of pinnae, with impending separation of the left pinna, April 28, 1947.

Post-mortem Examination.—The chief abnormal findings were as follows. *Heart*:—Weight, 460 g. (normal 300 g.). The aortic valve was bicuspid and showed a number of yellowish-red vegetations up to 0.5 cm. long, the largest of which lay close to the orifice of the left coronary artery. There was marked narrowing of the anterior descending branch of the left coronary artery 3 cm. from its origin. A healed infarct was present at the apex of the left ventricle. *Kidneys*:—right, 230 g.; left, 240 g. (normal 125–170 g.); flea-bitten appearance; scar of old infarct on left. *Liver*:—1,930 g. (normal 1,400–1,600 g.); well-marked nutmeg appearance. *Spleen*:—450 g. (normal 150–200 g.). *Brain*:—recent infarct in right parietal cortex and small cyst in right occipital lobe.

Histology.—*Aortic valve*:—Extensive red and white thrombotic vegetations. No bacteria seen in Gram preparation. *Myocardium*:—Fresh changes consisting of small focal areas of interstitial infiltration with polymorphs and mononuclear cells, presumably embolic in origin. Old changes presumed to be rheumatic, consisting of scars and small areas of perivascular fibrosis and calcification surrounded by a few multinucleated giant cells and lymphocytes. *Kidney*:—Most of the glomeruli showed patchy and chronic changes. Many of the tufts were partially replaced by connective tissue. Proliferative and endothelial changes were slight, and no crescents were seen. Interstitial fibrosis and patchy tubular atrophy were present. It

appeared to be a chronic process in which parts of the glomerular tufts had previously been the site of multiple embolic foci but had become fibrosed. Conclusion: chronic embolic nephritis.

Discussion

The most striking feature of this case of endocarditis was the necrosis which affected the helix of each ear. Tender blue swellings first appeared, necrosis slowly developed, and separation of the dead tissue occurred later. The left ear, which had been affected on his first visit to hospital, appeared normal on his second visit, but later relapsed. This slowly developing and remitting process, which affected each ear in turn, was most unlike an embolic process, and the non-embolic nature of the condition was favoured by the histological appearances, which are discussed in detail by Pagel (1949). No similar cases of "acronecrosis" in subacute bacterial endocarditis have been found in the literature.

Cardiac infarction is a rare occurrence in this condition. Hamman (1941), reviewing 40 recorded cases of coronary embolism, found that bacterial endocarditis was the source of the embolism in 19 out of 38 cases in which the aetiology was stated. In most of the cases the diagnosis was only made at necropsy following sudden death. In the present case the characteristic electrocardiographic changes confirmed the diagnosis three months before death.

There were several other atypical features. (1) Progressive renal failure with death in uraemia is rare in subacute bacterial endocarditis. Fishburg (1939) states that the course of the disease is hardly ever modified by multiple glomerular embolization. Although glomerular nephritis may occur and cause uraemia, there was no histological evidence of this in the present case. The picture was one of chronic embolic nephritis. (2) The repeatedly sterile blood cultures. Seven were taken before his first course of penicillin and a further eight before the second course, but all fifteen were sterile. Although the inability to grow an organism cannot exclude bacterial endocarditis, it is unusual to get fifteen sterile cultures at a time when fever and haematuria indicate definite activity of the disease. Moreover, no organisms could be found in the vegetations at necropsy. (3) The signs of liver damage as shown by the liver function tests. (4) The failure to respond to penicillin. Although the temperature became lower, no clinical improvement occurred. Since the patient was given 1,600,000 units daily for six weeks and renal failure was present, a high blood level of the drug was probably reached.

Several authors have described a form of endocarditis in which persistently negative blood cultures occur although the clinical features resemble the bacterial form. Baehr (1931) found that 57 out of 161 cases of subacute bacterial endocarditis followed to necropsy were free from bacteria. Blood cultures were sterile and the vegetations contained no bacteria. Several Spanish authors have recently described a number of cases of "abacterial endocarditis" (de Bes *et al.*, 1946; Jimenez Diaz, 1946). They believe that bacterial and abacterial endocarditis are separate entities. Bacterial endocarditis is characterized by persistently positive blood cultures, a short clinical course without remissions, and a rapid response to penicillin. In abacterial endocarditis, on the other hand, the blood cultures are persistently negative, the course is more prolonged, with remissions, and there is a complete failure to respond to penicillin. Other features of this type are a raised plasma globulin, positive empirical liver-function tests, a higher sedimentation rate than is usually found in the bacterial form, and a greater enlargement of the liver and spleen. A renal course with albuminuria, haematuria, urinary casts, and renal failure may occur, and the outcome is always fatal. Of 67 cases described by Landazuri and his colleagues (1946), 25 were abacterial and the remainder bacterial. Camelin *et al.* (1947) and Donzelot *et al.* (1947) have described similar cases from France. Such a high percentage of cases with negative blood cultures contrasts with the experience of Bramwell (1948), who recovered an organism in 50 out of 52 cases.

In bacterial endocarditis the various pathological changes are attributed to bacterial emboli arising from the heart valves, but in abacterial cases such an explanation is clearly not possible. It has been suggested that a heightened immunity of the host accounts for the absence of bacteriaemia, and possibly for the visceral lesions as well. A retention of the bacteria in the tissues has also been put forward as an explanation. Whatever the real cause of this variety, it is important that it should be recognized, as there seems to be no doubt that it is a true clinical entity.

Summary

A case of the abacterial form of endocarditis is described in which the following features occurred: symmetrical necrosis of the ears, cardiac infarction, liver damage, failure to respond to penicillin, and death in uraemia as a result of chronic "embolic" nephritis.

A review of the literature suggests that the abacterial form of endocarditis is a clinical entity.

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The American Society for the Prevention of Blindness has announced the results of a study of eye accidents in the schools in Louisville, Kentucky. The society estimates that American children suffer yearly 66,000 eye injuries, of which 750 are so serious that the patient loses the sight of an eye. Among Louisville children, an average of 117 eye accidents occurred annually. Boys suffered 75% of the injuries, though the number of boys and girls were approximately equal, and most of the accidents occurred in sports and play. To cut down the high accident rate among children four recommendations are made: (1) Prohibition of fireworks and weapons for children; these include air-rifles, bows and arrows, and sling shots. (2) Better supervision of play, to lessen the chances of children improvising unsafe games and using dangerous makeshift playthings. (3) Education of the children on hazards to the eye. (4) Incentives to make children more safety conscious, such as poster and essay contests and clubs. The main immediate causes of eye injuries, according to the report, are blows; accidental injuries with sharp objects such as pointed sticks, pens, and pencils; falls; foreign bodies; and fireworks. Air-guns and such toys accounted for 17% of the serious cases, and the society considers there is no longer an excuse for permitting children to play with them.