CASE REPORTS

CORONARY EMBOLISM WITH INFARCTION IN BACTERIAL ENDOCARDITIS

BY

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One of the classical symptoms of subacute bacterial endocarditis is embolic occlusion of peripheral arterioles and, similarly, coronary embolism is becoming increasingly recognized as more common. A clinically recognizable attack of myocardial infarction due to a large coronary embolism is, however, an unusual complication.

Coronary embolism due to subacute bacterial endocarditis was first described by Virchow in 1856: Saphir (1935) reported finding coronary emboli microscopically in 18 of 35 cases of bacterial endocarditis, and de Nevasquez (1939) reported similar findings in 16 of his 20 cases. Garvin and Work (1939) described three cases of coronary embolism that they encountered in 12,300 consecutive necropsies, in each case the source of the emboli being an ulcerative endocarditis of either the aortic or the mitral valves: they added a fourth case, of sudden death of a man, where the necropsy revealed emboli, composed of material identical with that on the heart valves, present in branches of the left coronary artery. In none of these cases was the diagnosis made clinically. Saphir *et al.* (1950) similarly found 17 cases with embolic material in the small coronary divisions, and a further 19 with ischæmic necrosis in the myocardium, in their 76 cases of bacterial endocarditis.

Perry (1952) studied the hearts of 52 cases of subacute bacterial endocarditis that were in the pathological collection of the Mayo Clinic, and concluded that miliary infarcts were the commonest lesion encountered, being found on microscopical examination in 47 (90%) of the cases. Brunson (1953) found that 125 of the 316 reported cases coming to necropsy showed evidence of coronary embolism due to the endocarditis: he described 9 cases of subacute bacterial endocarditis that he had studied, and as 7 of these had coronary emboli, he believed that this was relatively common if looked for.

In contrast with the microscopic miliary infarcts and the post-mortem findings of scarring and necrosis of the myocardium, clinical evidence of coronary infarction is much less common. Cates and Christie (1951) in their review of the 442 patients with subacute bacterial endocarditis, treated in fourteen centres appointed by the Medical Research Council, found that coronary embolism was diagnosed during life in only 8, of whom 6 died, and post-mortem in another 8.

Walker (1952), described a case in which death was due to the occlusion of the right coronary artery by large friable vegetations, which had almost completely replaced the aortic valves. Ravera (1952), made a cardiographic study of a fatal case in which there was an extensive coronary infarct, the bacterial endocarditis being revealed by the acute episode of the coronary occlusion, as in our case. Altana and Morrati (1954) reported a case, confirmed cardiographically, of myocardial infarction due to an embolism in a man, aged 37, with bacterial endocarditis: intensive and prolonged treatment with penicillin and heparin resulted in clinical recovery from both the infarction and the endocarditis. Nalimov (1957) reported that 9 of 125 cases of subacute bacterial endocarditis were complicated by myocardial infarction, and in 7 this was confirmed by autopsy: the cause was embolism, coronary endovasculitis, or a lesion of the aortic valve such as to block a coronary artery

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at its origin. The occurrence of subacute bacterial endocarditis in a case of coronary thrombosis has also been described by various authors (Joffe, 1955 and Kedra, 1957). This possibility must be borne in mind, if, as in this case, the diagnosis of endocarditis is not made until after the coronary infarction has taken place.

Case Report

A woman, aged 23 years, was first seen by me in 1957, at the start of her second pregnancy. She gave a history of rheumatism at the age of 12 years. She was completely free from any symptoms and led a full active life. Examination revealed a loud harsh systolic murmur, maximal at the apex but audible over the whole cardiac area, and a soft diastolic murmur audible at the apex. The diagnosis made was mitral regurgitation due to rheumatic heart disease, with complete compensation. The pregnancy and confinement were normal, and, against my advice, she even took a part time job in addition to her increased household duties.

In September, 1959, the patient came to see me, complaining that she had had swellings of the joints for the previous fortnight. She felt tired, and sweated a lot. Examination revealed a temperature of 99·6° F. and pulse rate of 120 a minute. The heart sounds were unchanged. There was swelling of the left ankle. A diagnosis of rheumatic fever was made, and the patient was sent home for complete bed rest and given soluble aspirin, 15 grains 6 hourly, and oral penicillin V, 250 mg. 6 hourly. With this regime she improved, until four days later she had a severe attack of acute retrosternal pain radiating down the left arm, and an aching in her lower jaw. When seen shortly afterwards, she was shocked and in pain as described. A diagnosis of coronary thrombosis was made, and the patient was given morphia and admitted to the Harwich and District Hospital.

An electrocardiogram (Fig. 1) confirmed the diagnosis of coronary occlusion, indicating a posterior

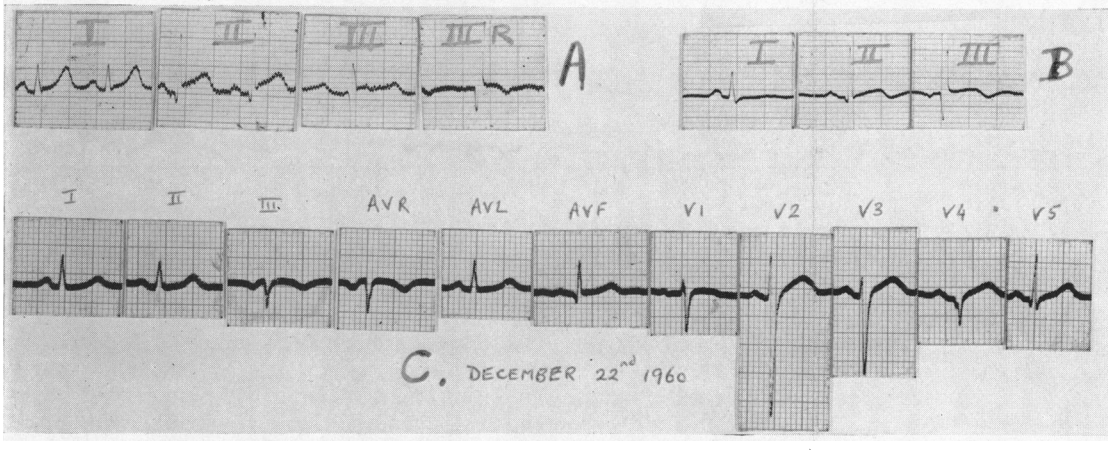

FIG. 1.—Serial electrocardiograms. (A) Showing the high take off in leads II and III and inverted T wave in lead III (5/10/59). (B). Still showing similar changes in lead III but with the T wave flat in lead I (26/10/59). (C). Normal except for V4, which shows a Q wave and no R wave, residual evidence of an anterior infarct (22/12/60).

infarction. At this stage some form of collagen disease seemed the most likely pathological process that would link up the findings, but no lupus erythematosis cells were found and the serum proteins and electrophoretic pattern were normal. A catheter specimen of urine showed a trace of albumen, but no red cells. The hæmoglobin was 78 per cent, the W.B.C. 10,000, and neutrophills 65 per cent. The E.S.R. was 120 mm. fall in one hour. Serum transaminase was 42 Sigma Frankel units/ml. Treatment with oral penicillin V, 250 mg. q.q.h., and aspirin, 15 grains q.q.h., was continued. The patient's condition improved. After ten days her temperature became normal, and her E.S.R. fell from 120 to 30–40 mm. an hour.

After five weeks, the patient having recovered from her coronary occlusion, the oral penicillin was stopped, to see if it was masking a temperature, as subacute bacterial endocarditis was suspected. Seven days later she became pyrexial (see Fig. 2). A blood culture was taken, and this gave a growth of *Streptococcus*

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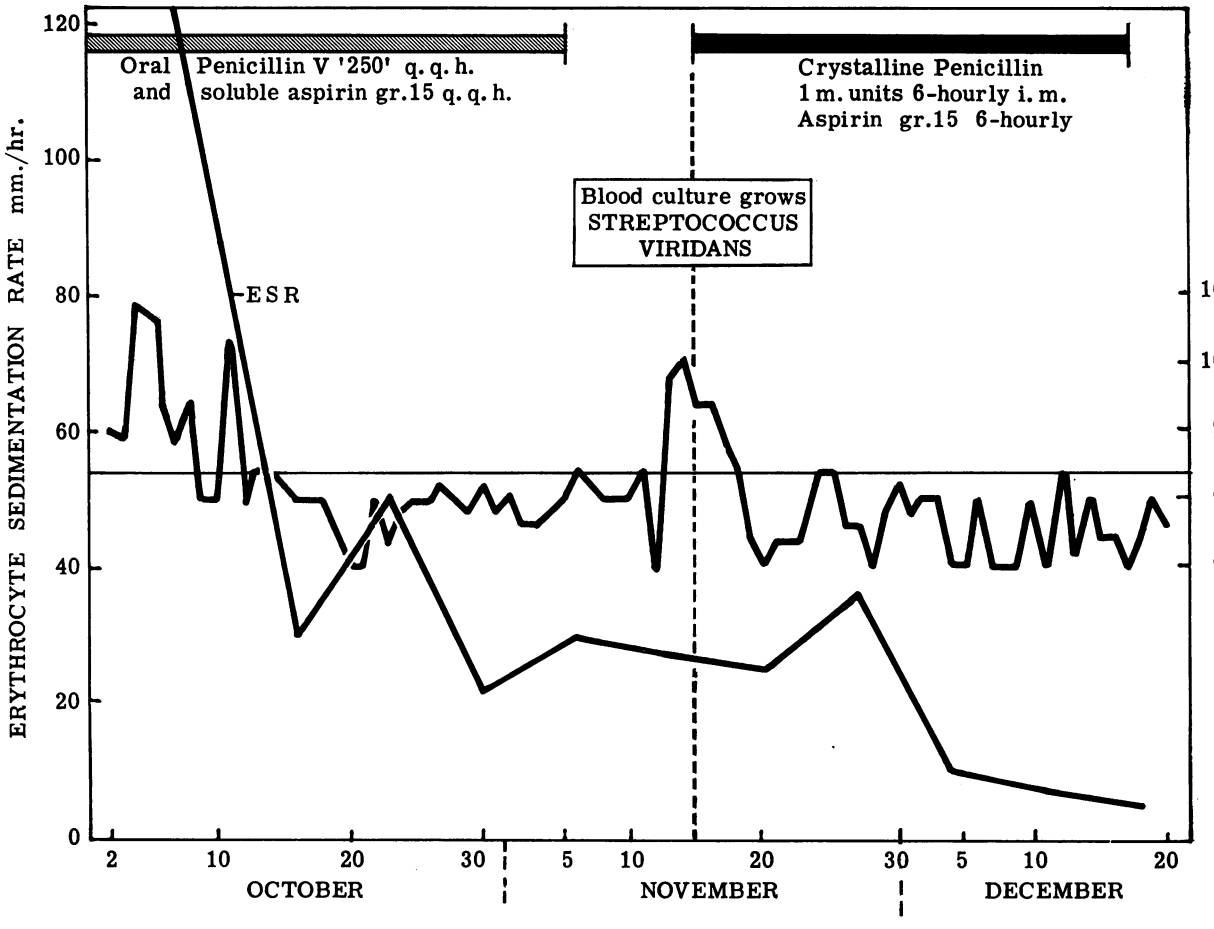

FIG. 2.—Chart, showing the temperature and sedimentation rate (E.S.R.) in relation to the treatment adopted.

viridans, sensitive to penicillin. At this stage it was noted that the soft tip of the spleen could just be felt. Now that the diagnosis of subacute bacterial endocarditis had been made, crystalline penicillin 1,000,000 units 6 hourly, with prednisone and soluble aspirin were given. Thereafter the patient improved greatly. She looked and felt much better, and, in a further month, her E.S.R. had fallen to 7 mm. an hour. At this stage her penicillin was stopped and a gradual reduction in the steroid and salicylate therapy was made. The cardiogram had improved and the blood culture was negative.

Screening of the heart and barium swallow on December 17th showed "increased amplitude of pulsation on the inferior aspect of the left ventricle in support of an area of infarction here." On discharge from hospital in December, the patient was well, and the heart sounds were unchanged from the time of admission. The electrocardiogram had become nearly normal.

She continued to be well and led a fairly active life until in early April, 1960, she got a recurrence of her symptoms of aching in the jaw, sweating, tachycardia, and joint pains. She was readmitted to this hospital and penicillin, 500,000 units i.m., 6 hourly, was recommenced. Blood culture (not taken, unfortunately, until the next day) was sterile. After ten days the patient was considerably worse. Her temperature ranged up to 101° F., her pulse rate was about 120, and the E.S.R. rose from 45 to 100 mm. an hour. The soft tip of a spleen could be felt. The penicillin was increased to 1,000,000 units 6 hourly, and thereafter, the patient made an uneventful recovery. Despite the negative blood culture, it seems highly likely that this second illness was a second attack of bacterial endocarditis.

The patient's intramuscular penicillin was continued for seven weeks, 1,000,000 units 6 hourly for the

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first five weeks and twice daily for the last two weeks, since when, the patient remained on oral penicillin V, 250 mg. bd. When last seen, the patient was well. A recent cardiogram (22/12/60) shows no evidence of a posterior infarct now, though V4 shows a Q and no R wave, residual evidence of an anterior infarct (Fig. 1C).

Discussion

One point of interest in this case is the fact that it is rare to get coronary occlusion in a woman under 40 years of age (Lewis, 1946). This statement refers, however, to coronary occlusion following atheroma, and as coronary embolism and coronary thrombosis are clinically indistinguishable, the signs of bacterial endocarditis must be searched for in all cases of coronary infarction in young people. Furthermore, the diagnosis must be made as early as possible, as the prognosis worsens if treatment is delayed (Cates and Christie, 1951).

It is not surprising to find that the additional complication of a large coronary infarct is so often lethal to a patient already suffering from bacterial endocarditis. Should the patient recover from both these conditions, the effect of the infarction might be expected to be less than the infarction following coronary thrombosis, the remaining vessels being healthier and supplying a better circulation. The return of the cardiogram to so near normal so quickly in our case might support this.

It is interesting to see how the oral penicillin, which was given initially as a prophylactic measure for "rheumatic fever" brought and kept the temperature down to normal (Fig. 2). Despite its use, the organism remained sensitive to high doses of penicillin. The second attack so soon after the first would seem to indicate that the penicillin had not been given for a sufficiently long time, though Barritt and Gillespie (1960) suggest that each attack is a fresh infection.

They also point out the importance of taking blood cultures before chemotherapy is instituted. A positive blood culture was obtained a month after the onset of the first attack, by discontinuing the oral penicillin for five days, during which time pyrexia developed, and a blood culture was taken. Intramuscular penicillin was then instituted immediately. From a diagnostic point of view it would have been more satisfactory and informative to have taken the blood culture on admission. At this stage, however, the patient was battling with a fairly severe attack of coronary infarction, and it did not seem justifiable to withhold any drug that might be helping her. On the other hand, as oral penicillin was effectively keeping the temperature normal, there seemed no point in clouding the issue further by giving the large doses of intramuscular penicillin necessary to eradicate the as yet unproven attack of subacute bacterial endocarditis. Furthermore, coronary infarction being such an unusual complication, the underlying subacute bacterial endocarditis was not initially suspected.

Summary

A case of coronary infarction occurring in a young married woman, with known rheumatic disease is described. Blood cultures grew *Streptococcus viridans*, which confirmed the diagnosis of subacute bacterial endocarditis. The infarction is thought to have been due to an embolism composed of vegetations from the diseased heart. The patient made a good recovery after a relapse. Other reported cases are discussed, but one similar to this is uncommon.

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