PROGNOSIS OF 'NUTRITIONAL' HEART DISEASE IN THE BANTU

REPORT OF A CASE OF CHRONIC REVERSIBLE HEART FAILURE

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The course of 'nutritional' heart disease described by Gillanders[1] is characterized by frequent relapses leading ultimately to death. That this apparently inevitable progression may not necessarily be the fate of all these patients, can be suspected from the discrepancy between the incidence in the wards and the necropsy room. Clinically, I recorded[2,3] 'nutritional' heart disease ('idiopathic cardiac hypertrophy', 'cryptogenic heart disease,' or 'African myocardopathy') as being the commonest cause of heart disease in the Johannesburg Bantu population; it was responsible for 37·5% of 275 cases of heart disease admitted to my unit at Baragwanath Hospital in 1957. Surprisingly, Higginson *et al.*[4] found it ranked only fourth in the causes of death from heart failure, forming 14·9% of 537 cases. It might be submitted that the diagnosis was inaccurate; however clinico-pathological correlation in individual cases does not support this contention. A more likely explanation is that a number of patients recovered and naturally enough ceased to attend the out-patient follow-up clinic. A chance encounter with an ex-patient afforded me the first proof that this form of heart failure was reversible. Seftel[5] has recorded reversal of a similar condition associated with pregnancy, but these cases had suffered only a single attack of congestive failure; those that relapsed remained in failure or died.

CASE REPORT

First Admission

D.W., a Xhosa male aged 43, was admitted to Baragwanath Hospital in October 1953 in congestive heart failure. He had been born in Queenstown, Cape Province, but had lived in Johannesburg since 1930. A month previously, troublesome palpitations on exertion and a cough productive of a small amount of colourless sputum had started. He had never noticed blood-staining of the sputum. For 2 weeks before admission he had become breathless on walking a few yards and was aware of swelling of the ankles and pain in the right hypochondrium. He slept propped up on 3 pillows but had not suffered from paroxysmal nocturnal dyspnoea. Systematic enquiry revealed that his diet was adequate and regularly included meat. He had been in hospital previously for a minor injury only. There was no history of joint pains.

On examination the jugular venous pressure was elevated to the angle of the jaw, when the patient sat up at 45°. The heart size was clinically indeterminate, the second sound at the pulmonary area was louder than that at the aortic, a rough systolic murmur was audible at the left border of the sternum, and a gallop rhythm was heard towards the (presumed) apex of the heart. There was no right-ventricular lift. Crepitations were heard at the base of each lung and there were signs of bilateral pleural effusion; ascites and oedema were demonstrable. The blood pressure in the right arm was 116/100 mm.Hg and in the left, 120/96 mm.Hg. The liver was tender and the edge palpable 4 fingerbreadths below the costal margin in the nipple line. The pulse was regular and of small amplitude, and the rate was 110 per minute. Fluoroscopic examination on the sixth hospital day showed that the cardiac pulsations were not active.

The patient was treated with bed-rest, low-sodium diet, mersalyl injections and digitalis. The gallop rhythm persisted for 4 days and the patient rapidly lost the signs of congestive failure and was discharged to the out-patient clinic. The urine had contained a trace of albumin, but no sugar was found in it on admission. The standard Eagle test for syphilis was positive, serum albumin was 2·9 g. and the globulin 4·5 g. per 100 ml.

Second Admission

The patient was readmitted in August 1954, again in con-

gestive heart failure. Two weeks previously the breathlessness on exertion and swelling of the ankles had recurred. He started to cough 9 days before attending the hospital; this was followed after an interval of 4 days by pain of a pleuritic character in the chest, and 3 days later he noticed blood in the sputum. He again claimed that he took alcohol (Kaffir beer) in moderation and that his diet was adequate. Certainly, he appeared well nourished. The blood pressure was 140/110 mm.Hg and the pulse rate was 100 per minute, the rhythm regular, and the amplitude small. The skin of the extremities was cool. The heart signs were much as before with the addition of a definite parasternal lift. Oedema was gross and hydrarthrosis of both knees was noted, the sputum was slightly blood-stained, and the spleen was palpably enlarged — this was attributed to venous congestion. A right basal pulmonary infarct was diagnosed. The electrocardiograph showed a normal sinus rhythm — the S wave in V2 was 26 mm. deep and the R wave 13 mm. in V5; the T wave was biphasic in V5 and flattened in V6. Haemoglobin was 18·6 g. per 100 ml. Fluoroscopy again showed a generally-enlarged heart with poor pulsation. The patient responded to the same measures as were used previously, and was discharged to the out-patient clinic on the eighth day.

Third and Fourth Admissions

Unfortunately he went to a country district, ceased taking digitalis, and returned in May 1955 in congestive failure. The oedema was so gross that blisters had developed on the legs and ulceration was present on the medial aspect of the lower third of the left leg. Blood pressure was 110/80 mm.Hg. He weighed 146 pounds on admission. A parasternal heaving impulse was again present and a high-pitched systolic murmur was noted towards the apex, and the pulse was regular at 100 beats per minute. The heart was grossly enlarged and the lungs congested on radiological examination. Digoxin, ammonium chloride and mersalyl were given. In 14 days the patient was no longer in failure and his weight was reduced to 121 pounds.

He again failed to renew his supply of digoxin and returned late in July 1955 in congestive heart failure. The blood pressure was 124/96 mm.Hg, the pulse amplitude was small and the rate was 84 per minute, he was not dyspnoeic at rest, and the jugular venous pulsation reached the angle of the jaw. The parasternal heave, apical systolic murmur and gallop rhythm were prominent features. On the sixth hospital day the patient coughed blood. The gallop rhythm had disappeared by the twelfth hospital day and the parasternal heave was much less obvious. The heart was enlarged on radiological examination. An electrocardiograph showed that the S wave in V3 measured 29 mm. and the R wave in V5, 20 mm.

Fifth and Sixth Admissions

Late in October 1955 the patient again presented himself because of congestive heart failure. His blood pressure was 100/60 mm.Hg and the pulse rate was 88 per minute. The apical impulse was noted to be in the sixth interspace without the mid-clavicular line, and a marked parasternal heave was present. By the sixth hospital day both the systolic murmur and the praecordial heave had disappeared. The radiological picture had not changed. The S wave in V3 was 26 mm. and the R wave in V5, 24 mm. Treatment was as before and the patient was discharged on the eighth hospital day.

The sixth admission took place in April 1956 because of breathlessness, hepatic pain, and cough, starting 2 - 4 days

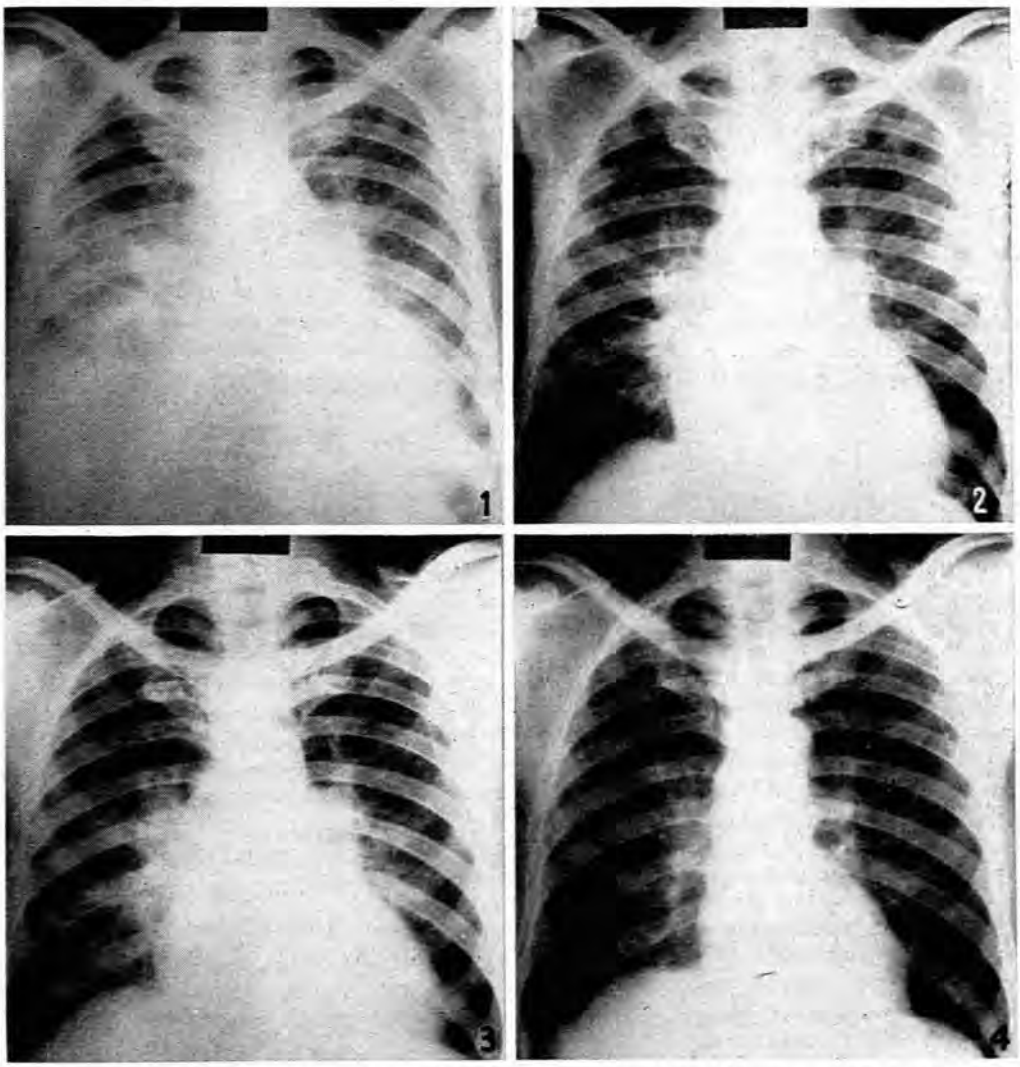

Fig. 1. 4 May 1955. Two days after third admission in congestive failure — cardiomegaly and pulmonary congestion.

Fig. 2. 29 July 1955. Two days after fourth admission in congestive failure.

Fig. 3. 5 March 1957. On maintenance therapy as an out-patient.

Fig. 4. 9 December 1959. Clinically and radiologically normal.

previously. He was in congestive heart failure of the low output type, the hands were cool, the blood pressure was 122/104 mm.Hg, and the heart rate was 116 per minute. Fluoroscopy on this occasion showed fair cardiac pulsation. The haemoglobin was 16·2 g. per 100 ml. His weight was 159 pounds on admission and 24 days later was 131 pounds. The S wave in V3 measured 33 mm. and the R wave in V6, 19 mm. The T wave was inverted in V5 and 6.

Further Course

In January 1957 he was admitted to a surgical ward for treatment of cellulitis of a leg following dog-bite. He was stated to be in congestive failure, his blood pressure was 100/70 mm.Hg, and a gallop rhythm and systolic murmur were noted. He has not been admitted again. When seen at the out-patient clinic in September 1957, he had not attended the out-patient department for 6 months. The jugular venous pressure was only slightly raised, the liver edge was 2 fingerbreadths below the costal margin, slight ankle oedema was present, and a loud systolic murmur was heard. Digoxin and mersalyl were restarted. Nineteen days later oedema was absent. Maintenance treatment was continued until the end of March 1958.

In May 1958 he was seen because of pellagra and was treated accordingly; there were no signs of heart failure but the liver was palpable. Pellagrous skin changes were present when next he was seen in August 1958. His next visit was in May 1959 when he was free from signs of failure.

In December 1959 he, somewhat belatedly, applied for an invalidity pension. Careful history-taking and examination failed to discover any evidence for heart failure, the blood pressure was 110/78 mm.Hg, and the liver edge was palpable one fingerbreadth below the costal margin, but the spleen was not felt. Radiological examination revealed a heart of normal size. An electrocardiograph showed the S wave to be 24 mm. in V2 and the R wave, 25 mm. in V6.

DISCUSSION

The course of the illness fell naturally into 3 periods, the first being the 2½ years from the onset of the heart failure, during which time there were 6 admissions to hospital. The interval between admissions varied from 2 to 10 months. There was subsequently a period of less severe failure during which the patient was admitted only once, and then to a surgical ward. He was seen infrequently in the out-patient clinic and received treatment sporadically for 2 years. Finally, there have been no signs of failure since May 1958. Indeed, in December 1959 there was radiological evidence of a normal-sized heart, in striking contrast to the films of May and July 1955 and March 1957 (Figs. 1 - 4).

This long period of freedom from heart-failure and the complete reversal of the cardiomegaly was outside our former experience of this type of heart disease, as was the disappearance of the heart murmur. Gillanders[1] claimed that his cases responded to ward diet alone and he showed examples of reduction in heart size. He emphasized, however, that they relapsed on returning home.

The period between successive admissions in his group was 2 weeks - 4 months. He stated that when the disease had been long established, enlargement of the heart and congestive failure became irreversible. There was no evidence in the present case that the patient's diet or living conditions improved over the years; in fact, he was unemployed throughout, and early on in the period of freedom from heart failure was treated for pellagra. There was thus a mysterious reversal of the chronic heart failure.

Acute reversible heart-failure was recognized in this population and described by Grusin,[6] but it forms only a small proportion of cases of heart disease. Furthermore this group of patients characteristically had actively-contracting hearts on fluoroscopy, with only 1 exception, and had warm extremities; only 1 of the series relapsed within 6 - 15 months and none developed an enlarged heart. Thus neither Seftel's[5] nor Grusin's[6] groups were strictly comparable to the present case.

There was never any suspicion that this illness differed from the usual run of idiopathic failures seen so commonly in this hospital. There had been no evidence of anaemia, thyrotoxicosis, pericarditis with effusion, beri-beri or scurvy, to explain the remarkable reversion to a normal circulatory state. Alcoholic cardiomyopathy had seriously to be considered, but seemed unlikely, since the amount of alcohol consumed did not appear to be excessive at any time. It is our experience that the Bantu are usually frank in answering questions about the amount and type of alcoholic drink consumed. This man more than satisfied Gillanders' criteria,[1] having had repeated attacks of congestive heart failure of a low output type with gross oedema, gallop rhythm, apical systolic murmur and pulmonary embolism. In 10 years' experience of this disease such a remarkable resolution of the clinical and radiological state had not been seen; it was noteworthy that the electrocardiograph remained unchanged. It might be argued that the present state is merely one of prolonged remission, but this in itself would be surprising in view of the previous relapsing course.

In an attempt to study the natural history of the disease, letters were sent to 35 individuals in whom the diagnosis of idiopathic heart failure had been made in 1953; not one reported for examination. The need for a thorough follow-up study of these patients, by means of personal visits to their homes, is obvious. In fact we know little more about the disease now than was known in 1951.

SUMMARY

A case of chronic heart failure, of the type described by Gillanders,[1] is reported in which reversion to clinical and radiological normality occurred. Present views on the prognosis of this disease need to be revised in the light of this finding.

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