A case of congestive heart failure

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A retired engineer first came under my care in August 1964 when he was 66 years-old, complaining of shortness of breath on exertion and swelling of his ankles. No records were available, but from the patient's account it was clear that he had been suffering from congestive cardiac failure intermittently for at least four years. He had been admitted to hospital for two weeks with this diagnosis in 1962.

When I first saw him there were no significant physical signs in the heart, lungs, or abdomen. The blood pressure was 150/90, and the pulse 74, and regular. A little ankle oedema was present, the haemoglobulin was 12·9 grams per 100 ml, his weight 13 stones clothed, which was about his average.

No precipitating cause for his decompensation was apparent, and a diagnosis of congestive heart failure secondary to ischaemic heart disease was proposed.

For the next 12 months, cardiac compensation was achieved with digoxin 0·25 mgm, b.d. with bendrofluazide 5 mg on alternate days. He lived a normal life for a man of his age in retirement.

In September 1965, his condition unaccountably deteriorated. He became breathless, even at rest, and he was cyanosed. His jugular venous pressure was raised. Both heart and liver were clinically enlarged. There were signs of gross fluid retention and albumen was present in his urine. Despite strict rest and increased diuretics, his condition became worse. No diuresis was achieved, however, with bendrofluazide, triamterene or with ' Mersalyl ', and in October 1965 he was admitted to hospital.

There his serious condition persisted. Radiograph of chest showed considerable cardiomegaly, there was gross oedema and ascites and there was a gallop rhythm. His electrocardiogram showed left bundle-branch-block. After a stormy passage, a profuse diuresis was finally achieved with ethacrynic acid.

After five weeks, the patient was well enough to return home, though still in a state described as " precarious ". Soon after doing so, he developed a common side-effect (gastroenteritis) to ethacrynic acid, which had to be stopped. Fortunately, by this time he had again become responsive to a combination of bendrofluazide and frusemide with potassium supplements. He slowly and steadily improved to such an extent in fact that by May 1966 there were no overt symptoms or signs of cardiac inadequacy. His weight was now 11 stone 12 lbs. His blood pressure was 130/80, and the electrolytes were normal, but the urea was 54 mgm/100 ml. For two years he led a quiet, but normal, life without setback.

In February 1968 he presented again with early signs of congestive failure. In view of previous difficulties in establishing diuresis, and after discussion with the consultant who had looked after him in hospital, early admission to a hospital bed was decided upon. Fortunately on this occasion his recovery was rapid and without complications.

On his return home, the patient again remained well for a further period of 18 months, although increasing doses of diuretics occasionally became necessary in order to maintain. adequate diuresis (frusemide 80 mg plus bendrofluazide 5 mg and six Slow-K tablets daily). He paid the inevitable penalty of a rise in serum urea, and also had attacks of gout, his uric acid rising to 12·4 mg/100 ml.

Throughout his illness the patient had displayed bouts of irritability and bad temper which coincided with the earliest signs of recurring heart failure. Sometimes he became confused and refused treatment, although he was an intelligent man quite capable of understanding his illness. Unfortunately, this led to further bouts of failure which required hospital treatment,

not so much because of the medical difficulties of his treatment as to ensure that it was being carried out.

In December 1969, for example, he was found in extremis following a syncopal attack. He was then deeply lethargic, constipated to the point of obstruction and again in severe congestive failure. His pulse was irregular at about 50/minute and a Stokes-Adams attack was suspected. The electrocardiogram showed atrio-ventricular dissociation, frequent ventricular extra-systoles, and a prominent U-wave, suggestive of potassium depletion. He was admitted to hospital as an emergency, and his serum potassium was found to be 2·2 meq/100 ml and urea 120 mg/100 ml. Subsequent enquiry revealed that he had ceased taking spironolactrone, a recent addition to his therapy, and had been taking full doses of frusemide without potassium supplements. Clearly his arrhythmia had been caused by digitalis toxicity.

Improvement after this episode was more protracted, and admission was again felt to be necessary in March 1970. On this occasion, when he left hospital, his blood urea had risen to 160 mg/100 ml. In his discharge letter, the consultant wrote " This is a situation which carries a very poor prognosis . . . but I hope he gets a few months of reasonable comfort."

Despite this ominous prognosis, he again showed a phenomenal capacity for recovery. Except for one slight setback in April 1971, he improved steadily and indeed worked hard for one period of six months looking after a sick wife who was bedridden with a terminal illness with cancer of the colon.

Not long after his wife's death, he went on holiday on a Mediterranean cruise, and returned looking fit and well. At this time (January, 1972) his weight had returned to 11st. 8½lb. and even his blood urea had returned to 66 mg/100 ml.

Encouraged by the happy outcome of this trip, he set off on a similar one in May 1973. On his return from this, however, he again had symptoms and signs of decompensation. He had gained a stone in weight and was short of breath on slight exertion. He was cyanosed, his venous pressure raised, and his liver was much enlarged. One interesting difference in this episode from previous ones was an evident precipitating cause—while abroad he had caught an upper respiratory infection requiring antibiotic treatment, and there were still signs of chest infection on his return. Intensive treatment restored the position, and at the time of writing he is again feeling well and enjoying a reasonable quality of life for a man of 76 years of age. He remains, however, rather overweight and enlargement of the liver persists. His blood urea has not fallen below 100 mg/100 ml.

Discussion

The pathogenesis of cardiac oedema has been well described by Jan Brod (1972). It can be summarised by saying simply that when the heart ceases to meet the oxygen requirements of the body, a chain reaction is set off which leads to salt and water retention. The ability of the physician to intervene in this reaction largely decides the prognosis for the patient. Even as recently as 1960 Wood suggested that " When it (congestive heart failure) occurs in the natural course of incurable heart disease, few patients survive more than a year or two." The introduction of new and potent diuretics with different modes of action, however, continues to improve the prognosis.

The case history summarised above is no doubt exceptional. To recover from seven episodes of heart failure requiring admission to hospital is in itself remarkable. For the patient still to be enjoying a good quality of life after 12 years is even more so. This in itself is perhaps sufficient reason for recording the case. In addition, however, it serves to illustrate some possible trends and lessons.

(1) This case history illustrates well to what extent the prognosis in heart failure has been improved by the introduction of potent diuretic agents. This is especially true when there are no important correctable structural factors such as valvular defects or substantial muscle loss from infarction. Although this case is exceptional, all general practitioners will no doubt be able to recall similar if less dramatic examples from experience.

(2) The need for careful control, particularly of electrolyte balance, is illustrated. This man became acutely ill on one occasion as a result of his confusion over the use of potassium supplements and the consequent digoxin toxicity.

(3) It is often difficult to ensure adequate rest at home and this is vital in cardiac insufficiency. This case history illustrates how this can sometimes be achieved only by admission to hospital.

(4) It is interesting to record that this man's life was probably saved by the introduction of ethacrynic acid. This substance became available at a time when all other attempts at producing diuresis had failed.

(5) It is worth noting how valuable it is to have a number of different substances with diuretic properties. There were several occasions in this man's history when diuresis could only be achieved by the concurrent use of two or more drugs in full doses.

(6) At a time when the scientific evaluation of heart failure is so much to the fore, it is worth remembering that occasionally the first symptom of impending failure is a change of mood. This patient illustrated this well—irritability or bad temper proved a helpful warning of impending trouble.

(7) Similarly, when the growth of specialism is thought (by some) to have removed serious illness and its scientific management from the field of general practice, it is worth recording an instance of the reverse. This case illustrates well how a general practitioner can, and indeed must, play a vital role in the long-term scientific management of serious illness. He holds a key position in this management. To be successful he must pursue careful and continuous clinical observation, supported by the X-ray and pathological services of the hospital and by electrocardiography. With this kind of support, and with appropriate training, he can manage most cases without other assistance. In cases with unusual features, such as the one here described, he may however, need specialist advice.

(8) Finally, the case illustrates the good results that can come from well controlled management of heart failure. The hospital, of course, plays a vital role, but with our current limited resources, it might well be that greater benefits can be brought to patients with cardiac failure by the diversion of more resources to general practice.

REFERENCES

Brod, J. (1972). *British Medical Journal*, **1**, 222–228.
Wood, P. (1960). *Diseases of the heart and circulation.* London: Eyre and Spottiswood.

ADDENDUM

This patient died in February 1975, aged 77. This was in his ninth episode of congestive failure requiring hospital admission—15 years after his first such admission.