Hydrallazine alone in acute left ventricular failure

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Summary

A patient presented with severe acute left ventricular failure and was treated with hydrallazine and oxygen alone. He made a rapid and full recovery as judged by clinical, radiological and blood gas evidence.

Introduction

The potent loop diuretics have an unrivalled place in the management of acute left ventricular failure (LVF). Few situations can be envisaged in which one of these agents cannot be given, although the therapeutic response may in many cases be poor (e.g. in renal failure). There seems little need for alternative agents to fulfil the role of loop diuretics, although the vasodilators may be such agents. Vasodilator therapy is most frequently used in end-stage chronic heart failure, and hence the therapeutic response is often disappointing.

The case is now reported of a patient in acute LVF who was given, partly by accident, only a vasodilator, and who subsequently made a good recovery.

Case report

A 58-year-old lorry driver had been in good health until 2 months previously when he had begun to notice slight effort dyspnoea. On the day of admission he awoke at 3.00 a.m. in a state of severe breathlessness, and was brought to hospital. There was no chest pain. His BP had never been recorded before this admission, and the only other significant factor was a strong family history of ischaemic heart disease.

On examination he was cyanosed and severely dyspnoeic, with cold clammy extremities, BP 215/130 mmHg, pulse 120/min regular. The jugular venous pressure was not easily seen and there were harsh crackles over both lung fields. A gallop rhythm was heard at the left sternal edge. There was no peripheral oedema.

ECG showed sinus tachycardia with left ventricular hypertrophy and strain.

No arrhythmias were seen during 48 hr of cardiac monitoring, and no new ECG changes appeared suggesting myocardial infarction. Arterial blood

gas analysis taken on air showed hypoxia (Pa_{O_2} : 7.42 kPa), and chest X-ray showed left ventricular failure, with borderline cardiomegaly and a retrosternal thyroid (later confirmed on thyroid scan (Fig. 1 (a))).

Fig. 1. Radiographs taken (a) at presentation, and (b) 5 hr later with the same exposure, by the same machine.

Having made an initial diagnosis of acute LVF of uncertain cause, it was decided to treat the patient with i.v. frusemide and a single i.m. injection of hydralazine—in view of the elevated BP in the presence of heart failure.

The patient was given controlled oxygen therapy, and 20 mg of hydralazine, and in error the frusemide was omitted. No diamorphine was given. Nevertheless, he made a good recovery from this point onwards, as judged clinically after one hour, by a rise in Pa_{O_2} after 6 hr to a value of 17.9 kPa on 28% oxygen, and by radiological improvement over 5 hr (Fig. 1(b)). Complete resolution of all changes was seen on films taken after 5 days. Only 100 ml of urine was passed during the first 4 hr of his admission, but one litre was passed over the next 16 hr, and 1635 ml over the subsequent 24 hr, attaining a negative fluid balance.

The improvement persisted, and it became apparent that he was mildly hypertensive (average BP 150/100 mmHg). He was later started on a thiazide diuretic for this.

Discussion

This patient made a good response to vasodilator therapy in acute LVF, and at no point required a loop diuretic. Hydralazine worked because of its

ability to reduce cardiac afterload (Opie, 1980; Mason 1978), and because of its positive inotropic effect (Khatri *et al.*, 1977). Hydralazine and other vasodilators now seem to be well established in the management of the chronically failing heart, (Chatterjee *et al.*, 1976; Fitchett *et al.*, 1979), and also as an adjunct to other therapy in acute heart failure (Chatterjee and Parmley, 1977). The authors have now described the use of hydralazine in isolation in the management of this condition.

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