Left ventricular failure with labetalol

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Summary

A patient treated for hypertension with labetalol developed left ventricular failure. When the drug was withdrawn and the BP controlled with 2 other agents, the signs of heart failure regressed. The ill effects in this case could have been because the β -blocking effects of labetalol are 4 to 6 times greater than the α -blocking effects. Caution should be exercised when prescribing this drug for patients with heart failure or with previous symptoms.

Case report

A Caucasian mortuary attendant had a myocardial infarction when 38 years old. He made a good recovery apart from post-infarction angina pectoris. Three years later and for 4 years thereafter he had a series of minor surgical procedures at the end of which time his BP was 140/90 mmHg.

One year later he was investigated for exercise pain in the left arm. Examination showed a blood pressure of 130/80 mmHg, in his right arm. The left radial pulse was absent. An aortogram revealed left subclavian artery stenosis with retrograde filling via the left vertebral artery. A left carotid-subclavian bypass operation was performed, and at discharge his BP was 120/70 mmHg.

Two years and 2 months later he presented again with chest pain. His blood pressure was 190/110 mmHg and there was a fourth heart sound, grade II retinopathy and a soft apical systolic murmur. Investigations of his hypertension suggested right renal artery stenosis, with increased secretion of renin from the affected side, and suppression on the contralateral. A right ilio-renal Dacron bypass graft was inserted.

The arterial pressure did not fall after the operation. He was re-admitted one month after discharge with further chest pain, which was shown to be due to a fresh inferior myocardial infarction. Thereafter he was treated with propranolol, methyl dopa and chlorthalidone, with variable control of his blood pressure.

Eighteen months later he was re-admitted because of palpitations, dyspnoea and nocturia. His blood pressure had risen to 210/140 mmHg but there was no change in the fundal appearances. X-rays showed

features of left ventricular failure, but there were no fresh ECG changes, and no alteration in serum LDH or AST to suggest a further myocardial infarction. His renal function had deteriorated, the serum creatinine having risen to 159 $\mu\text{mol/l}$ from 96 $\mu\text{mol/l}$ since his previous admission. He was treated with frusemide and spironolactone, and was given labetalol 200 mg thrice daily which was increased to 400 mg thrice daily one week later. Although the BP was reduced to 120/90 mmHg his condition deteriorated despite large doses of frusemide (1 g daily) and spironolactone. He became faint on standing although there was only a 5-mmHg orthostatic fall in BP. Chest X-ray showed increasing evidence of left ventricular failure and there was a small rise in the serum creatinine from 159 $\mu\text{mol/l}$ to 217 $\mu\text{mol/l}$. Labetalol was therefore withdrawn and he was treated with a combination of prazosin (6 mg daily) and clonidine (0.6 mg daily, later increased to 0.9 mg daily). Without any alteration in diuretic therapy his heart failure improved, the dose of frusemide was subsequently reduced and the spironolactone stopped.

When last seen his blood pressure was 150/100 mmHg. Both clinically and radiologically there was no evidence of heart failure.

Discussion

Labetalol has α -blocking properties as well as being a β -blocker (Richards *et al.*, 1974). It has been credited with the advantage that it lowers arterial pressure without reflex tachycardia and without any reduction in cardiac output (Edwards and Raftery, 1976). It causes no deterioration in calculated indices of left ventricular function, except at extreme levels of exercise. The α -blocking activity of the drug gives it the theoretical advantage over those which are only β -blockers, particularly in patients with symptoms of and a previous history of heart failure.

In the case reported here labetalol seems to have provoked heart failure. When withdrawn, and the BP controlled with 2 other agents, the radiological and clinical signs of left ventricular failure regressed. It may be speculated that there was a temporary impairment of renal function, although the rise in serum creatinine was very small and he did not develop

dependent oedema. When another drug with only vasodilating properties, prazosin, was used, the heart failure resolved. It is possible that the provocation of heart failure in this case was due to the β -blocking effects of labetalol being 4 to 6 times greater than the α -blocking effects (Brittain and Levy, 1976).

A recent report that intravenous administration of labetalol produced hypertension (Crofton and Gabriel, 1977) and the present report might suggest that in some patients there is a marked β -blocking effect on the myocardium and arterioles. Although the clinical trials phase of labetalol did not reveal any patient who developed left ventricular failure, this case report suggests that caution should be exercised in the use of the drug in patients with

heart failure or with previous symptoms, as is recommended in treatment with β -blockers.

References

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