SHORT REPORTS

Fibrinous Peritonitis: A Complication of Practolol Therapy

Practolol (Eraldin) is widely used for treating angina pectoris and cardiac arrhythmias, and is prescribed for other cardiovascular disorders such as hypertension. But patients have been reported having ocular lesions[1] and others have developed cutaneous reactions, most resembling psoriasis.[2 4] We report here a new complication.

Case Report

Mrs. A. B., aged 56, developed angina pectoris in 1970 and in 1972 was put on practolol 100 mg three times a day in addition to oxazepam 15 mg three times a day and digoxin 0·25 mg twice a day which she had been having since 1971. She developed a rash on her hands, which spread rapidly, and a dry lumpy sensation in her eyes, with reduced secretion of tears.

In March 1974 she first noticed abdominal discomfort and one month later her general practitioner found a cystic swelling arising from the pelvis. At a gynaecology department, 14 days later, the swelling had vanished. During the summer she suffered from abdominal pain, distension, pyrexia, and sweating, and was admitted to hospital in August. She had a lower abdominal swelling. Routine laboratory and radiological investigations gave normal results.

Mrs. A. B. went home after the practolol had been reduced to 100 mg twice a day. But after two attacks of pain, nausea, and vomiting she was readmitted to hospital for an emergency laparotomy. There were soft masses in the epigastrium and the pelvis. The abdomen was full of fibrinous adhesions, which in places formed cocoons around several loops of bowel. These cocoons were filled with clear serous fluid and were the masses felt on abdominal examination. The obstruction was due to kinking of the small intestine, which was dissected out by blunt and sharp dissection. Practolol-induced fibrinous adhesions were diagnosed at operation and the treatment stopped. The rash faded and her postoperative course was satisfactory until the fourth day, when she complained of severe chest pain, collapsed and died.

Necropsy showed a bluish purple lichenoid rash on the back of both thighs. There were fine tacky adhesions between visceral and parietal pericardium, visceral and parietal pleura, and in the peritoneum. Apart from severe coronary arteriosclerosis with a fresh thrombosis at a stenosis in the first part of the left coronary artery and oedema of the lungs, all the major organs of the body looked normal. The skin presented striking changes of the type described by Felix *et al.*[4]

Discussion

This patient had the typical lichenoid skin lesions and polyserositis associated with practolol medication.[4] The eye symptoms described by Wright[1] were also present but there was no evidence of corneal damage. She had developed intestinal obstruction due to fibrinous peritoneal adhesions—a new feature of the reaction to practolol. A personal communication from the Committee for Drug Safety confirms that there have been three other similar unreported cases of intestinal obstruction in Great Britain all of which were associated with practolol medication.

The appearance of the abdominal cavity at operation is unique and bizarre with soft but well-organized fibrin cocoons enveloping the abdominal viscera. The adhesions were separated by a combination of blunt and scissor dissection to relieve the obstruction. The preoperative diagnosis was difficult owing to the transient nature of the symptoms and signs and the misleadingly normal results of laboratory and radiological investigations. Nevertheless, an awareness that practolol may cause symptoms resembling subacute intestinal obstruction and the finding of an evanescent abdominal mass may help to alert doctors, as might the presence of splenomegaly, or other side effects such as ocular symptoms, rash, arthropathy, or pleural and pericardial effusion.

We urge that subacute small intestinal obstruction in patients undergoing treatment with practolol should be treated by immediate withdrawal of the drug as the condition may be reversible. Why patients on practolol should develop a polyserositis with fibrinous peritoneal adhesions and intestinal obstruction is speculative at present,[4 5] but the multiplicity of side effects of this useful drug must call into question the value of its continued and widespread use.

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[2] Wiseman, R. A., *Postgraduate Medical Journal*, 1971, **47**, 68.
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Practolol and the Nephrotic Syndrome

Many forms of medication may damage the kidneys. Some drugs (trimethadione, penicillamine, and gold) may cause the nephrotic syndrome. Other drugs—for example, hydralazine, procainamide, and practolol[1]—may induce a syndrome similar to systemic lupus erythematosus (S.L.E.). In addition practolol has recently been reported as causing a characteristic psoriasiform rash[2] and eye lesions.[3]

Case History

A 65-year-old retired lorry driver was admitted to hospital in August 1974. He had had vitiligo for 25 years, a paroxysmal cardiac dysrhythmia, and chest pain suggesting angina pectoris. In addition to lanatoside C he had been treated with practolol 400 mg per day for six months and 600 mg per day for two and a half years.

Before admission he had developed blurring of vision, but no dryness of his eyes, and circular discrete skin lesions around his ankles, which later appeared on his arms and trunk. He had also experienced discomfort in his left shoulder and right hip, and his feet had begun to swell. Three weeks before admission his weight had increased by 12 kg and he had noticed some swelling of his wrists and around his eyes. On examination he had diffuse vitiligo, periorbital, limb, and sacral oedema. He was initially mildly hypertensive but soon became normotensive. He was apyrexial and had no joint lesions.

Investigation showed a haemoglobin level of 14·9 g/dl, serum albumin 22 g/l, cholesterol 18·9 mmol/l (730 mg/100 ml), and blood urea 15·8 mmol/l (95 mg/100 ml). Urine microscopy showed a few granular casts and red and white cells. Urine protein was 25·5 g per 24 hours but no Bence-Jones protein was present. Chest radiography showed a pleural effusion at the left base.

On light microscopy a renal biopsy showed no outstanding features. The glomeruli showed no obvious thickening of the basement membrane with either periodic-acid Schiff or silver stain, and no cellular proliferation. Some mild interstitial round cell infiltration was apparent. The tubules and blood vessels were unremarkable. A skin biopsy showed hyperkeratosis, foci of parakeratosis, and marked epidermal hyperplasia. There was some mild perivascular round cell infiltration.

Practolol was discontinued. His eye symptoms, the skin lesions, and the nephrotic syndrome resolved over several weeks. Seven weeks later his blood urea was 5·6 mmol/l (34 mg/100 ml), serum albumin 37 g/l, and the urine protein 1·0 g per 24 hours. The antinuclear factor remained positive in low titre. His paroxysmal cardiac dysrhythmia was controlled with propranolol.

Discussion

The clinical manifestations in this case, including the nephrotic syndrome, were probably induced by practolol. The duration of treatment before the occurrence of symptoms, the clinical appearance of the skin lesions, the eye symptoms, and the improvement of the clinical and laboratory features after discontinuation of the drug supported this. The clinical features also suggested the diagnosis of an S.L.E. syndrome. But renal involvement in drug-induced S.L.E. is rare.[4] In this case the nephrotic syndrome was severe; the antinuclear factor titre was low and the serum complement level high. The renal findings were not very abnormal on light microscopy, nor was the glomerular basement membrane perceptively altered. These features were compatible with minimal change glomerulonephritis and with the microscope appearance of a drug-induced nephrotic syndrome.[5]

In this case it seems unlikely that the nephrotic syndrome was part of an S.L.E. syndrome or a coincidental feature of the clinical