

Pleural fibrosis after practolol therapy

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The principal manifestations of a practolol reaction involve the skin, mucous, and serous membranes (Nicholls, 1976). Under the heading oculomucocutaneous syndrome are described rashes, eye involvement, impairment of hearing, and dryness of the mouth and nasal passages. The peritoneum, pleura, pericardium, and joints may be affected by polyserositis with effusions into serous cavities and fibrosis of serous membranes.

We describe two patients who developed pleural fibrosis after treatment with practolol.

Case reports

CASE 1

In 1973 a 56-year-old process worker started taking practolol 300 mg a day for angina. After

17 months the drug was stopped because of a skin reaction. In 1975 he underwent two operations for intestinal obstruction due to fibrinous peritonitis. Progressive exertional breathlessness then developed until, by January 1976, he was dyspnoeic walking on level ground. Chest radiography (fig 1) showed bilateral effusions with considerable pleural thickening, particularly on the right. Fifty ml of straw-coloured fluid was aspirated, and a portion of fibrin, similar to that removed at laparotomy, was obtained by pleural biopsy. Culture for tubercle bacilli and examination for antinuclear factor were negative. Treatment with prednisone was started but was discontinued after six months as there was no symptomatic or radiological improvement. Pulmonary function tests showed a restrictive defect with some airways obstruction,

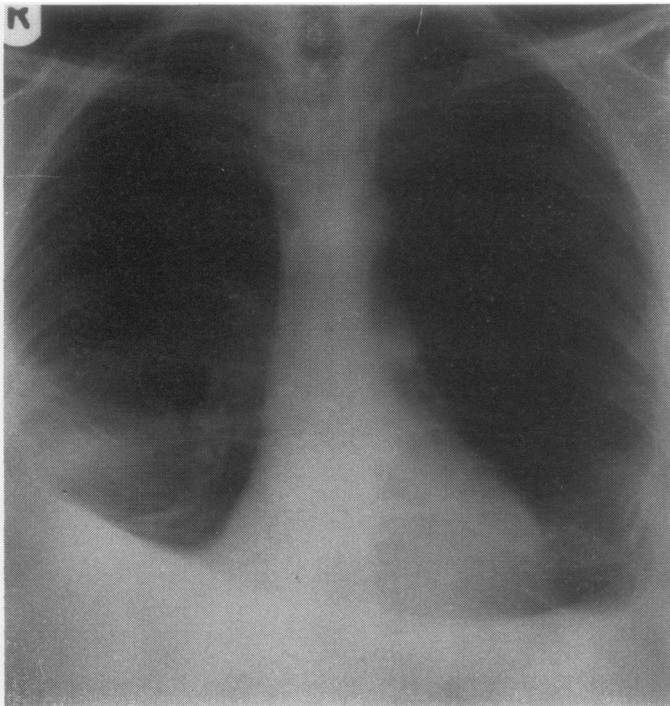


Fig 1 Case 1. Chest radiograph showing bilateral pleural fibrosis.

and regional lung function studies with xenon-133 showed reduced ventilation and perfusion, particularly at the lung bases. At thoracotomy the pleura over the right lower chest and diaphragm was grossly thickened, up to 2 cm in places, while the pleura over the upper chest was 3–5 mm thick. Fine adhesions to the lung were separated, and the pleural peel was removed from the lower chest posteriorly and laterally and from the diaphragm. No pericardial thickening was present. Histologically the material removed was acellular hyaline fibrous tissue. The postoperative course was complicated by empyema, which eventually required surgical drainage. Considerable pleural thickening remains at the right lung base, and although the patient felt an initial symptomatic improvement he is still very breathless.

CASE 2

A retired bank manager presented with effort dyspnoea early in 1977. He had taken practolol for 12 months after a myocardial infarct in 1973. In 1974 the drug was changed to sotalol because of skin and eye side effects. The chest radiograph (fig 2) was similar in appearance to that of the

first patient, and pulmonary function tests showed a restrictive defect. Treatment with steroids was not given because of the lack of response in the previous case. When last seen the patient remained breathless, although some improvement in his symptoms had accompanied reduction in weight; the appearance of the chest radiograph was unchanged.

Discussion

The most striking feature of these two patients was the severe bilateral pleurisy that developed months after treatment with practolol had been discontinued. It was anticipated that the first patient would be improved by pleurectomy, but unfortunately the benefits of this procedure were reduced by the postoperative empyema. Pleurectomy is, however, technically feasible and should be considered in other patients in whom the major problem is pleural restriction.

The diagnosis of pulmonary reactions to practolol is facilitated by a previous history of a well-recognised side effect such as plastic peritonitis. Marshall *et al* (1977) described six patients

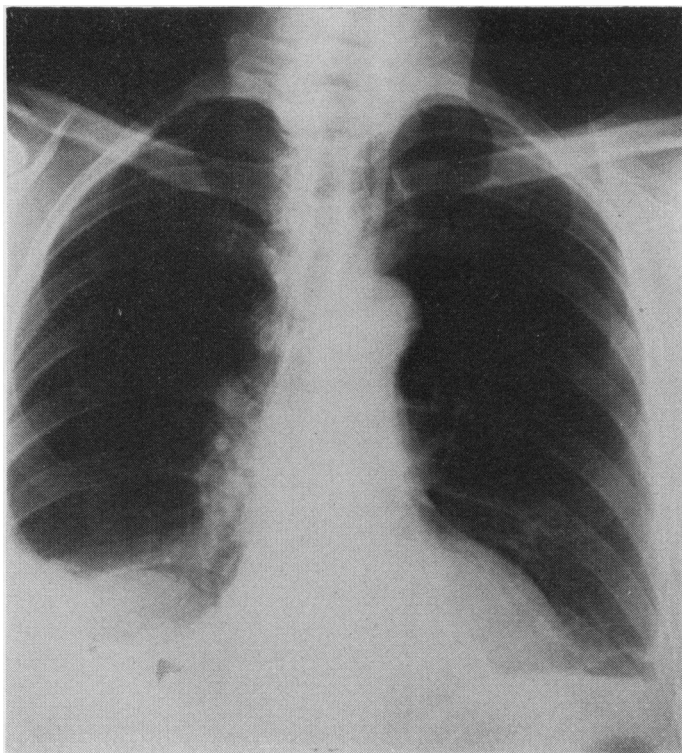


Fig 2 Case 2. Chest radiograph showing bilateral pleural fibrosis.

in this category who developed pleural disease while being followed up after their abdominal operations. However, there may be difficulties in diagnosis when such a history is not forthcoming. The possibility of a drug reaction should be considered when any patient with unexplained pulmonary disease gives a history of previous treatment with practolol.

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References

- Nicholls, J T (1976). Adverse effects of practolol. *Annals of Clinical Research*, **8**, 229-231.
- Marshall, A J, Eltringham, W K, Barritt, D W, Davies, J D, Griffiths, D A, Jackson, L K, Laszlo, G, and Read, A E (1977). Respiratory disease associated with practolol therapy. *Lancet*, **2**, 1254-1257.

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