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SHORT CASES IN CARDIOLOGY

Pulmonary oedema and pleural effusion in two patients with primary pulmonary hypertension treated with calcium channel blockers

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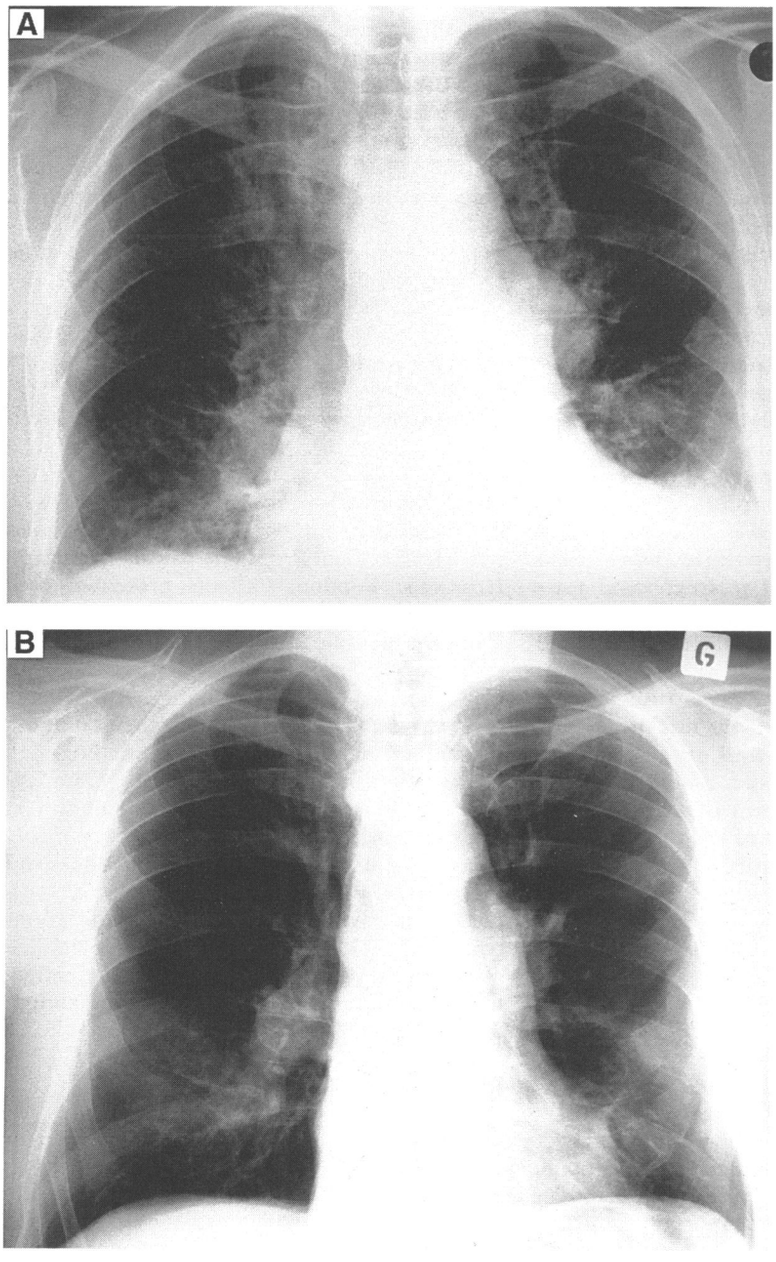
Accepted for publication
6 November 1995



(A) Chest x ray on admission, showing pulmonary oedema and bilateral pleural effusion. (B) Chest x ray after nifedipine treatment was stopped.

A 63 year old man who had had primary pulmonary hypertension (PPH) for 2 years was admitted in our department with right heart failure. This patient fulfilled all the diagnostic criteria for PPH.[1] Before treatment with calcium channel blockers was started his haemodynamic acute response to vasodilators was determined. Nifedipine (60 mg/day) was started. On admission to our department, this patient had severe dyspnoea at rest and was cyanosed. He showed marked signs of right heart failure, associated with signs of pulmonary oedema and pleural effusion which were confirmed by the chest radiography (fig 1A). Despite oxygen therapy and an intravenous infusion of frusemide, his haemodynamic and respiratory condition did not improve. A few hours later we decided to stop nifedipine. Subsequently, we saw a rapid decrease of dyspnoea, pulmonary oedema, and pleural effusion (fig 1B). This patient died 6 months later from a recurrence of right heart failure. Necropsy showed micro-thrombolic pulmonary arteriopathy. A 64 year old woman with PPH who was treated in our department with nifedipine (120 mg/day) has had the same medical history.

Because the clinical improvement occurred only after the wash-out time of nifedipine,[2] we concluded that nifedipine was the cause of this life-threatening state. The mechanism of these adverse effects seems to be either a negative cardiac inotropic effect or a modification of capillary blood flow, like that described in peripheral oedema caused by the dihydropyridines[3] or a combination of both mechanisms. A modification of capillary blood flow may increase pulmonary capillary hydrostatic pressure and may cause fluid to cross from blood to tissue.

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