Reversal of hypoxia in a patient with chronic obstructive pulmonary disease, pulmonary hypertension, and oedema during treatment with long term domiciliary oxygen

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Abstract

A case of chronic obstructive pulmonary disease with pulmonary hypertension and oedema is presented, with reversal of hypoxaemia over 18 months during treatment with long term oxygen therapy.

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The prognosis of patients with chronic obstructive pulmonary disease (COPD) complicated by hypoxaemia, hypercapnia, pulmonary arterial hypertension, and oedema is grave with one third of such patients dying within three years.^{1,2} Only long term oxygen therapy (LTOT) improves survival in such patients^{3,4} and may halt the progression of pulmonary hypertension.^{5,6}

Case report

A 63 year old man, an ex-smoker with long-standing COPD complicated by respiratory failure, was transferred to our unit from another hospital where, despite treatment with diuretics and oxygen, his oedema had not improved.

On examination he was dyspnoeic at rest with pitting oedema of both legs and elevated jugular venous pressure. His heart rate was 100 beats/min, regular, and his blood pressure was 140/90 mm Hg; no murmurs were audible. His chest was hyperinflated with bilateral basal crepitations and widespread rhonchi. Abdominal examination revealed a markedly enlarged liver.

The haemoglobin concentration and white blood count were normal. Blood urea was elevated (21 mmol/l) with normal electrolytes. Levels of alkaline phosphatase at 552 units/l and γ -glutamyl transferase at 395 units/l were both increased on admission and returned to normal before discharge, but albumin was normal. Chest radiographic examination showed cardiomegaly, prominent pulmonary arteries, and lower zone patchy shadowing (fig). Routine 12 lead elec-

trocardiography showed sinus tachycardia 110/min, right axis deviation, P pulmonale, and signs of right ventricular hypertrophy. Sputum culture on one occasion grew *Pseudomonas aeruginosa*. His arterial blood gas tensions on admission while breathing oxygen, 2 l/min via nasal prongs, were: arterial oxygen tension (PaO_2) 5.9 kPa, arterial carbon dioxide tension (PaCO_2) 11.0 kPa, $[\text{H}^+]$ 45.6 nmol/l, and HCO_3^- 43.9 mmol/l. Initial treatment with intravenous frusemide (100 mg twice daily) and, additionally, metolazone (5 mg) did not produce a significant diuresis but further impaired renal function (peak blood urea 34.0 mmol/l). In view of this he was commenced on an infusion of dopamine (2 $\mu\text{g/kg/min}$) via a Swan-Ganz catheter. At this time his pulmonary arterial pressure was 28 mm Hg, pulmonary wedge pressure 5 mm Hg, and cardiac output 6.1 l/min. Right ventricular ejection fraction was 0.36 (normal range >0.45).⁷ Treatment with dopamine produced a significant diuresis and his condition improved. He remained stable and oedema free thereafter on frusemide 120 mg/day.

Before discharge his forced expiratory volume in one second (FEV_1) was 0.6 litres and forced vital capacity (FVC) 2.0 litres without evidence of significant reversibility to nebulised bronchodilators. Arterial blood gas tensions while breathing air were: PaO_2 5.6 kPa, PaCO_2 7.9 kPa, $[\text{H}^+]$ 34.6 nmol/l; while breathing oxygen, 2 l/min by nasal prongs, the values were: PaO_2 8.0 kPa, PaCO_2 7.5 kPa, $[\text{H}^+]$ 36.9 nmol/l. On discharge LTOT was prescribed at a flow rate of 2.0 l/min via nasal prongs delivered from an oxygen concentrator. He was reviewed every three months and remained stable with PaO_2 while breathing air of 6.5-7.4 kPa and no clinical evidence of fluid retention nor change in

Posteroanterior chest radiograph showing cardiomegaly, prominent pulmonary arteries, and bilateral lower zone patchy shadowing.

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FEV₁ over the first 12 months. After 18 months, however, his Pao₂ was found to be 8.6 and 9.4 kPa on two occasions six weeks apart without any change in his treatment. There was also no evidence of any improvement in his pulmonary hypertension as assessed non-invasively by measuring the width of the right basal pulmonary artery on chest radiography, nor did his right ventricular ejection fraction show any change (0.38). His Pao₂ value was therefore no longer within the Department of Health criterion for LTOT (Pao₂ <7.3 kPa breathing air). Oxygen saturation (SaO₂) was measured at home over a 24 hour period with a pulse oximeter while breathing oxygen. He spent 95% of the time from 09.00–21.00 hours and 93% of the time from 21.00–09.00 hours with an SaO₂ value above 90% and did not desaturate while on oxygen below 80%. While breathing air his SaO₂ was above 90% for 60% and 40% of the day and night respectively, and again he did not desaturate below 80%.

Discussion

The development of the first episode of peripheral oedema in patients with COPD is often associated with an infection of the lower respiratory tract. Successful treatment with antibiotics and symptomatic therapy with controlled oxygen and diuretics is usually sufficient to reverse hypoxia and restore clinical stability.⁸ In our patient such management was not sufficient and he continued to deteriorate and required treatment with dopamine. Among patients with COPD, respiratory failure, and oedema who fail to improve during therapy are those with pulmonary embolism. Our patient did not, however, have symptoms or signs of deep venous thrombosis. A ventilation-perfusion scan would have been difficult to interpret because of his severe COPD and, furthermore, the clinical course did not suggest such a diagnosis.

Weitzenblum *et al*¹ have shown that Pao₂ values in patients with COPD can improve spontaneously up to three months after an acute exacerbation. Our patient was still significantly hypoxic (Pao₂ 6.4–7.4 kPa while breathing air), however, when seen at the clinic 12 months after discharge. His remarkable improvement was observed first after 18 months of treatment with LTOT when his Pao₂ while breathing air was 8.6 and 9.4 kPa on two occasions and SaO₂ was more than 90% for more than 50% of a 24 hour period. A possible explanation for the improvement is

an improvement in airflow limitation, either spontaneously or in response to treatment; however, no changes in FEV₁ or in treatment occurred over the follow up period. Improvement in pulmonary haemodynamics or right ventricular function would be unlikely to occur de novo after 18 months treatment with LTOT since this treatment has been shown to prevent worsening but not to improve pulmonary arterial pressure.^{3–5} The lack of change in heart size or right ventricular ejection fraction does not suggest any major change in cardiac function. Furthermore, the width of the right basal pulmonary artery, as a non-invasive measure of pulmonary arterial pressure,⁹ did not change with treatment. Spontaneous improvement in occult lower respiratory infection or pulmonary thromboembolism 18 months after the acute exacerbation of COPD cannot be excluded, but also seems unlikely. The explanation for the improvement in Pao₂ therefore remains unclear.

Improvement in Pao₂ 18 months after discharge is unusual in patients treated with LTOT.¹⁰ This patient reinforces the point that reassessment is necessary to determine the continued need for oxygen therapy.

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