Improved gas exchange after pneumonectomy

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these patients is helpful for assessing the mechanism and severity of impairment of their gas exchange.


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Spontaneous improvement in a patient with the hepatopulmonary syndrome assessed by serial exercise tests

K B Saunders, S S D Fernando, H R Dalton, A Joseph

Abstract

A 37 year old patient with chronic active hepatitis progressing to cirrhosis presented with increasing breathlessness and was found to be hypoxic with finger clubbing. A progressive exercise study with measurement of oxygen saturation (SaO₂) showed abnormally high ventilation and desaturation to 81% at 100 W. Serial studies over nearly two years showed, first, deterioration, then improvement with lower ventilation and higher saturation levels at all work loads. This could not be correlated with any change in treatment with azathiprine, prednisolone, or propranolol.

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About one third of patients with hepatic cirrhosis are found to have arterial hypoxaemia without any of the usual cardiorespiratory causes. A few have obvious cyanosis with clubbing. The hypoxia is due either to right to left shunts through large arteriovenous connections within the lungs, or to peripheral vaso-dilatation of the fine branches of the pulmonary artery at both precapillary and capillary levels, leading to incomplete diffusion equilibration between alveolar and end capillary blood. The pathophysiology has recently been fully reviewed.³

Case report

The patient presented at the age of 27 with nose bleeds, jaundice, anaemia, and swelling of legs and abdomen. Chronic active hepatitis was diagnosed by liver biopsy and, over subsequent years, further biopsies showed progression to cirrhosis despite treatment with prednisolone and azathioprine which had been prescribed for about five years, in varying dosage, before the present study. The cause of the chronic active hepatitis was never established. An autoantibody screen, including antibodies to smooth muscle and mitochondria, was negative. Hepatitis B serology was negative and hepatitis C serology was not available at that time. The concentration of plasma α₁-antitrypsin was not directly measured, but the level of α₁-globulin was increased.

Ten years after his first attendance he complained of breathlessness and was referred for respiratory assessment. He was clubbed but not cyanosed at rest. He said he was unusually short of breath on exertion, but there was no serious limit to his exercise tolerance and throughout the period of the study he worked full time as a welder on a British Rail track gang, and could play 36 holes of golf in a day without discomfort. During the period of study standard liver function tests were abnormal, and varied without any systematic trend with ranges as follows: bilirubin 55–90 μmol/l, albumin 30–32 g/l, alkaline phosphatase 191–344 IU/l, gamma-GT 93–134 IU/l, and alanine transaminase 28–87 IU/l. He continued to drink alcohol, but it was difficult to assess in what quantity.

Blood gas measurements were obtained sitting (Pao₂ 9.2 kPa, Paco₂ 4.3 kPa) and lying (Pao₂ 9.3 kPa, Paco₂ 3.8 kPa). Pure oxygen was given from a balloon reservoir with careful exclusion of leaks from the breathing circuit whereupon Pao₂ rose to 60 kPa. A pulmonary perfusion scan with technetium-99 m labelled albumin microspheres, analysed according to the method of Chlivers et al,¹ showed that 42% of the tracer passed through the lungs. The chest radiograph was normal. Lung volumes, both static and dynamic, were normal, but transfer factor for carbon monoxide (TLCO) and transfer coefficient (Kco) were
low (6·2 mmol/min/kPa and 0·85 mmol/min/kPa/1 respectively), predicted normal values being 10·6 and 1·7.

A progressive exercise study was done with two minute increments measuring pulse rate, ventilation breath-by-breath, and \( \text{SaO}_2 \) by pulse oximetry (Biox). The study was ended by the supervisor when saturation fell to 80% at 100 W (fig 1), although the patient was comfortable and had not contemplated stopping. Pulse rates were normal, but ventilation approached double normal values at higher loads.

A second study performed 35 weeks later had to be ended when saturation fell to 80% at only 60 W. We anticipated that the patient would continue to deteriorate and might require liver transplantation, and decided to assess his problem by serial exercise studies. Results taken from these studies at the single work load of 60 W are shown in fig 2. Results at other work loads were concordant; 60 W was the highest work load which was completed on every attendance with saturation more than 80%.

At the same time propranolol 40 mg three times daily was prescribed both as a prophylactic against bleeding from varices, and because it might improve oxygenation by slowing flow through dilated pulmonary vasculature. A third study (fig 2) after two weeks of treatment with propranolol showed the expected fall in pulse rate, some decrease in ventilation, but no change in saturation. A fourth study was done a week after stopping propranolol and the drug was then again prescribed between weeks 36 and 56. Pulse rate again slowed as expected but, to our surprise, ventilation fell at all exercise levels from 70/1/min to 45/1/min at 60 W, and saturation levels improved from 84% to 92% at 60 W.

We then stopped treatment with propranolol. Pulse rate rose, but to lower levels than originally, and the improvements in ventilation and saturation were maintained or enhanced over a period of six months. Finally, the patient could exercise to 100 W with \( \text{SaO}_2 \) falling only to 93%.

At this stage the perfusion scan was repeated, showing that the percentage of tracer passing through the lungs, previously 42%, was now less than 2%. \( \text{PaO}_2 \) was 10·3 kPa and \( \text{PaCO}_2 \) 4·8 kPa at rest. \( \text{TLCO} \) and \( \text{KCO} \) were still low, however, and hardly changed (64/2 and 0·93 respectively); the patient was still clubbed.

Discussion

Our patient had the typical signs and investigative findings of the hepatopulmonary syndrome, with the exception of platypnoea. It is likely that in such patients some powerful circulating pulmonary vasodilator interferes with hypoxic vasoconstriction and produces functional and, after a time, structural vasodilatation of the pulmonary microvasculature. Hypoxia is caused by anatomical shunting within the lung, through large arteriovenous connections to areas with high local flow relat-
Late asthmatic response to inhaled glacial acetic acid

S Kivity, E Fireman, Y Lerman

Abstract
A patient with bronchial asthma who developed a late asthmatic response to inhalation challenge with glacial acetic acid is presented. This is believed to be the first description of a reaction to this allergen in an asthmatic patient.

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Exposure to glacial acetic acid has so far been reported to induce either reactive airway disease, as described following accidental exposure,1 or interstitial pneumonitis.2 We report a patient with suspected asthma to glacial acetic acid who developed a late reaction to inhalation challenge with glacial acetic acid carried out in a special environmental chamber.

Case report
The patient was a 58 year old man who had developed asthma over the previous two years. He reported having asthma during childhood, but had been symptom-free from the age of 11 to 56 years. Over the previous two years, however, he had been exposed to glacial acetic acid in the canning factory where he worked. Glacial acetic acid was used in the pickling of cucumbers and the fumes were released into the surrounding air. Exposure to glacial acetic acid fumes occurred during the 10 minute process of heating the mixture when the patient stood in the vicinity unmasked. A few months after the initial exposure he began having asthmatic attacks which were relieved with salbutamol. Symptoms occurred initially only on working days, but soon became more frequent, occurring also over weekends and on holidays. As the symptoms worsened he was given daily treatment with high dose beclamide (800 µg/day), supplemented with salbutamol as required.

Exposure to a small amount of glacial acetic acid was on a daily basis, and occasionally to increased amounts, with subsequent worsening of symptoms. Shortness of breath occurred a few hours after exposure to glacial acetic acid and continued also at home. Occasional exposure to cigarette smoke, cold air, or other irri-