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CASE REPORT

High Altitude Pulmonary Edema in a Patient with Previous Pneumonectomy

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High altitude pulmonary edema (HAPE) is a life-threatening illness that can occur in individuals ascending to altitudes exceeding 2400 m. The risk factors are rapid ascent, physical exertion and a previous history of HAPE. This work presents a case study of a 74-year-old man who underwent left side pneumonectomy 40 years ago and subsequently experienced several instances of HAPE. The well-known risk factors for HAPE were excluded in this patient. We suspect that the post-pneumonectomy condition may be a risk factor for HAPE based on this case. [*J Formos Med Assoc* 2007;106(4):320–322]

Key Words: altitude sickness, pneumonectomy, pulmonary edema, risk factors

High altitude pulmonary edema (HAPE) is a life-threatening illness that can occur in people ascending to altitudes exceeding 2400 m. The incidence varies between 2% and 15% of exposed individuals. The risk factors are well known, and include rapid ascent, physical exertion and a previous history of HAPE. The symptoms of HAPE may be as minor as an insidious cough, or as severe as breathlessness that does not respond to rest and the production of frothy, frequently rusty, sputum. We hypothesize that accentuated hypoxemia and abnormal production of hypoxic pulmonary vasoconstriction, at a certain altitude, induce elevated transmural pressure and the release of cytokines and inflammatory mediator. Then, increasing hydrostatic pressure and altering alveolar capillary permeability are induced, and causes a form of noncardiogenic pulmonary edema known as HAPE.

Following pneumonectomy, lung volume and lung compliance decrease while airway resistance increases. The diffusing capacity for carbon monoxide when corrected to lung volume is generally normal, while the arterial oxygen saturation, PO_2 and PCO_2 at rest remain unchanged. The resting values of systolic pulmonary artery pressure, central venous pressure and pulmonary vascular resistance index following pneumonectomy remain roughly constant.

To our knowledge, the occurrence of HAPE following pneumonectomy has never been examined in the English and Chinese literature. This work is thus the first to present HAPE occurring following left side pneumonectomy.

Case Report

A 74-year-old man developed bronchiectasis following severe pneumonia in childhood. Left side pneumonectomy was performed 40 years prior to presentation due to repeated hemoptysis. The patient also suffered from hypertension that was well controlled with atenolol (50 mg/day) lasting 3 years. The patient had never gone to high-altitude

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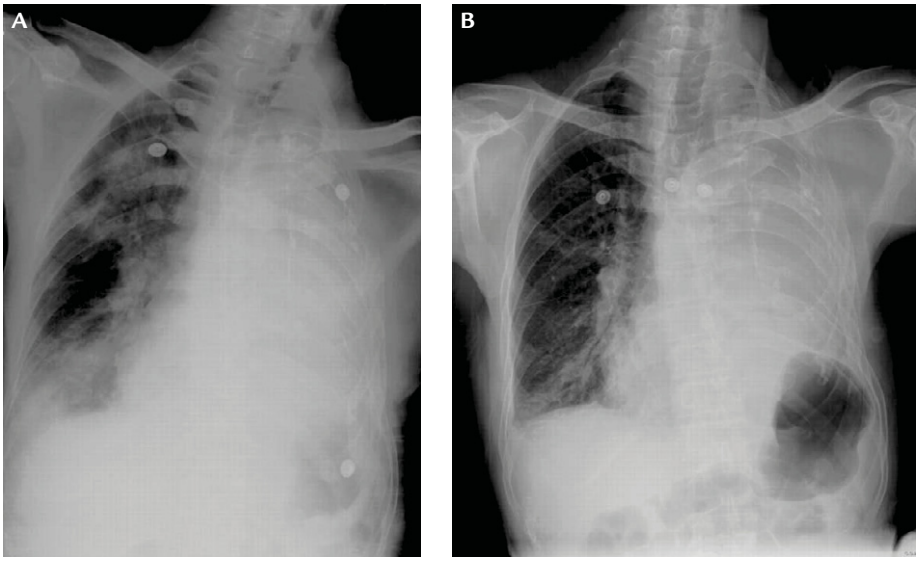

Figure. Chest radiographs of high altitude pulmonary edema: (A) before treatment; (B) 3 days following the use of diuretics.

areas before his pneumonectomy and at that time had no history of altitude sickness or HAPE. The patient's history of altitude sickness and HAPE began 15 years ago when he developed dyspnea on approaching 3500 m during a 2-day climb to 4000 m. The second occasion was 4 years ago when he developed dyspnea while taking a cable car up to 3000 m. The symptoms were relieved rapidly on his return to 2000 m. HAPE was suspected clinically on both of these occasions. On the third occasion, the patient had already spent more than 24 hours at 2400 m, but developed dyspnea on taking a cable car to 3300 m the following day. The patient was hospitalized on this last occasion. Chest radiograph demonstrated infiltration of the right upper and lower lobes with alveolar character (Figure A). No fever or leukocytosis was found on admission. The electrocardiogram showed mild tachycardia without ischemic sign. The sputum smear and culture showed normal flora. The infiltration improved rapidly with the use of diuretics (Figure B). HAPE was diagnosed both clinically and based on chest radiographic changes.

Discussion

HAPE accounts for the majority of deaths from altitude sickness. HAPE is totally preventable provided appropriate precautions are taken, particularly for high-risk groups. Currently, certain risk factors for HAPE are well known, including rapid ascent, strenuous exercise and previous history of HAPE.[1–3] Although four cases of congenital absence of the right pulmonary artery developing HAPE have been described previously, the previous literature has never described the occurrence of HAPE following pneumonectomy.[4] This work describes a case of HAPE following left side pneumonectomy, and emphasizes that the post pneumonectomy condition is a possible risk factor for HAPE.

How rapid the ascending rate needs to be to induce pulmonary edema is still not clearly defined. Although the patient ascended 1000 m on the following day, no other members who were with him developed the symptoms of HAPE on the third occasion. Also, on the previous occasions, no companion developed the symptoms of HAPE. The patient denied HAPE development before pneumonectomy. The patient also did not have strenuous exercise on these three occasions.

Several studies have found evidence that a risk of pulmonary edema exists immediately post-pneumonectomy. Zeldin et al demonstrated that blood flow to the remaining lung increased 5- to 6-fold due to both volume loading and pneumonectomy.[5] Cagle and Thurlbeck also suggested

that the compensatory growth of the contralateral lung, including the increase in pulmonary blood volume or edema, achieves a lung weight similar to the previous two lung weights.[6] Caras reported that once the unused pulmonary vascular reserve is depleted following lung resection, the increased blood flow to the remaining lung would increase pulmonary arterial pressure given unchanged pulmonary vascular resistance.[7] Immediately post-surgery, the increased permeability of the non-operated lung, as a "leaky lung", was also demonstrated by Waller et al.[8]

In contrast to our inference, Lee et al reported that pneumonectomy does not seriously increase susceptibility to hydrostatic pulmonary edema resulting from hemodynamic challenge.[9] Furthermore, another investigation demonstrated no contribution from either vascular congestion or increased extravascular protein and water to the observed weight gain of the remaining lung.[10] Moreover, overperfusion, with or without alveolar hypoxia, was not found to increase lung endothelial barrier protein permeability.[11] These studies thus suggest that following pneumonectomy, the remaining lung is not susceptible to hydrostatic or high permeability pulmonary edema.

This work reports the first case of HAPE in a patient with previous left side pneumonectomy. The post-pneumonectomy condition is considered to be a risk factor for HAPE. However, as just one case has been described here, more post-pneumonectomy cases should be collected to obtain statistics to support the conclusions presented here. If the risk factor is recognized, post-pneumonectomy patients planning travel to high altitude areas should be educated and given special precaution. Moreover, further study should be conducted to examine the etiology of susceptibility to HAPE in patients who have undergone pneumonectomy.

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