High Altitude Pulmonary Oedema (HAPE) in an Indian Pilgrim

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Abstract
Increasing number of Hindu pilgrims visit the Himalayas where some of them suffer from high altitude illness including the life threatening forms, high altitude pulmonary oedema (HAPE) and high altitude cerebral oedema. Compared to tourists and trekkers, pilgrims are usually ignorant about altitude illness. This is a case of a pilgrim who suffered from HAPE on his trip to Kailash-Mansarovar and is brought to a tertiary level hospital in Kathmandu. This report emphasises on how to treat a patient with HAPE, a disease which is increasingly being seen in the high altitude pilgrim population.

Introduction
High altitude pulmonary oedema (HAPE), a life-threatening form of altitude illness, is a non-cardiogenic pulmonary oedema that occurs in susceptible persons after 2 to 4 days of ascent to altitude over 2500 meters. Many people visit the Himalayas and are thus exposed to altitude illness, including the severest forms i.e. HAPE and high altitude cerebral oedema. The persons at risk include not only trekkers, climbers and porters, but also pilgrims. The present case is of an Indian pilgrim who went to Kailash-Manasarovar (4500 m) in Tibet, developed HAPE and was treated at a university hospital in Kathmandu.

Case
A 37 year-old previously healthy man fell ill when he was on his pilgrimage to Lake Mansarovar in Mount Kailash region, Tibet. He had never before been to altitude over 2000 m. His illness started with symptoms of cough and cold, for which he took some anti-cold medications. As he ascended further, he began to experience increased tiredness compared to his fellow pilgrims. He began to have difficulty breathing along with cough, first dry and later with frothy discharge. When he was brought to Kathmandu (1300 m), he had been sick for 3 days. At the hospital, he had a respiratory rate of 32 per minute and a heart rate of 96. He was afebrile and acyanotic. On auscultation, coarse crackles were heard bilaterally at the basal regions. Oxygen saturation (SpO₂) was 82%.

He was given oxygen at 4 liters/minute and nebulised with salbutamol. 20 mg of furesomide, 100 mg of hydrocortisone and 1 g of ceftriaxone were also given intravenously with the provisional diagnosis of HAPE.

His white cell count was 11,000/mm³ with 75% neutrophils. Electrolytes, including urea and creatinine were normal. ECG was normal as well. Chest X-ray (Figure 1) showed heterogeneous opacity in middle and lower zones of both the lungs, which was suggestive of pulmonary oedema. Echocardiogram revealed a pulmonary artery pressure (PAP) of 50 mmHg with no other abnormalities.
Overnight, he continued to receive oxygen at 3 litres/minute with regular monitoring of SpO₂. He was nebulised with salbutamol and ipratropium. He was administered 1 g of iv ceftriaxone twice daily, 500mg of oral levofloxacin once daily, 40 mg of oral pantoprazole once daily and 20 mg of delayed release nifedipine twice daily.

The next day, he had improved tremendously. Respiratory and pulse rates had returned to baseline. SpO₂ was 97% on room air. He was discharged with oral medications: levofloxacin 500 mg once daily for 5 days, cefixime 200 mg twice daily for 5 days, and delayed release nifedipine 20 mg twice daily for 5 days. He was advised to follow up 3 days later. On follow-up chest X-ray (Figure 2) showed dramatic changes. The infiltrates had almost cleared. On repeat echocardiography, PAP was 25 mmHg.

**Discussion**

A rapid rate of ascent, a prior history of HAPE, existing respiratory tract infections, and cold environmental temperatures put a person at risk of getting HAPE, which is a non-cardiogenic pulmonary oedema due to hypoxia of high altitude-induced pulmonary hypertension. Males are at more risk than females and it can occur even at moderate altitudes in persons with abnormalities of the pulmonary circulation leading to pulmonary hypertension.²

The first symptom of HAPE is a reduction in exercise tolerance greater than expected at the given altitude. A dry persistent cough may later progress to production of blood-tinged sputum. Fever is sometimes present. Initially, the only physical findings could be slightly exaggerated tachycardia and tachypnoea with exercise, which progress to tachypnoea and tachycardia even at rest. Crackles may be heard on auscultation, but are not necessary for diagnosis.²

Since HAPE accounts for most deaths from altitude illness,³ early diagnosis and treatment are important. While diagnoses in the field have to be relied on history and clinical evidence, some investigations would be helpful in the hospital settings. Pulse oximetry is both easy and useful and can be used both in the field and in hospital. Chest X-ray shows patchy infiltrates, in middle and lower zones, which may be confined to one lung in mild cases and in both lungs in more severe ones.⁴ ECG is useful to rule out cardiac causes; findings in HAPE may be right ventricular strain or even hypertrophy.² Peripheral white cell count may be raised which may sometimes give an impression of chest infection. Other tests like, arterial blood gases and pulmonary function tests are not necessary for diagnosis and treatment.

There is no consensus among physicians worldwide, on how to best treat HAPE. Wilderness Medical Society has tried to minimise this confusion by devising an evidence-based guideline with the help of its expert panel.⁵ The suggested approach is descent, oxygen when available, and nifedipine as an adjunctive therapy. Hyperbaric bag (Gamow bag®, for example), can also be used when descent is not possible. It is a portable nylon chamber which can be inflated with the patient inside. With inflation, the barometric pressure inside the bag increases and more oxygen is available to the patient. It is very effective but it only «buys time» and is just a temporising measure.

The current guideline refutes the role of diuretics like furosemide since many patients present with intravascular volume depletion. Although there is some role of beta-agonists, steroids have no role in the treatment of HAPE.

A history of high altitude travel, suggestive
symptoms, chest findings, low SpO₂ and X-ray findings with increased PAP have led to the diagnosis of HAPE in this patient. Absence of fever and normal leucocyte count further point towards this diagnosis, instead of pneumonia. The PAP returned to normal in 3 days; and most importantly there was rapid improvement in chest X-ray, which is typical of HAPE, and is not detected with pneumonic consolidations which is the closest differential diagnosis here.

Prompt descent to Kathmandu (1300 m) from 4500 m probably saved the patient’s life as it is the mainstay of therapy. Supplemental oxygen to bring the saturation to over 90% also helped him. However, since the diagnosis had already been made, there are no reasons for administering hydrocortisone and furesomide. Initial antibiotic use could have been reasonable, but there is no rationale for continuing it since infection had been precisely ruled out. Administration of delayed release nifedipine is as per the guideline, though its dose should have been 60 mg per day in divided doses. He was discharged with nifedipine for 5 more days, which was unnecessary.

Compared to trekkers and climbers, pilgrims in the Himalayas know less about prevention and treatment of altitude illness. When a climber or a trekker gets sick and dies, everybody is alerted. But a pilgrim dies quietly in the mountains. Because pilgrimages are done in large numbers, sometimes the number of fatalities may be high. Hence, prevention and treatment of altitude illness, including HAPE should be emphasised in the pilgrim population.

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