Rapid Communication

Delayed-onset high-altitude pulmonary edema

Sanjay Singhal, Srinivasa Alasinga Bhattachar1, Vivek Paliwal2, Kamal Pathak2

Departments of Pulmonary Medicine, 1Radiology, 153-General Hospital, 1High Altitude Medical Research Centre, Leh (Jammu and Kashmir), India

CASE REPORT 1

A 28-year-old male with no co-morbid illness was admitted to our hospital (located at a height of ~11,500 ft) with complaints of breathlessness and cough associated with pinkish sputum. He denied history of fever and chest pain. He was inducted to high-altitude area (height ~11,500 ft) 4 months back. General examination revealed the following: temperature 98°F, blood pressure 106/82 mm Hg, pulse rate 106/min, respiratory rate 24/min, and oxygen saturation 64% at room air and 96% on oxygen inhalation. Chest auscultation revealed bilateral diffuse crepts. Arterial blood gas analysis on room air suggested severe type-I respiratory failure (pH: 7.443, PaO₂: 28.1, PaCO₂: 33.0). Electrocardiogram showed no significant abnormality. Chest radiograph showed bilateral diffuse confluent alveolar opacities [Figure 1]. Hematological investigation revealed hemoglobin of 13.4 g/dl and total leukocyte count of 15,400/cm³ with neutrophil predominance (81%). Based on clinical and radiological parameters, he was diagnosed to have high-altitude pulmonary edema (HAPE). Detailed history disclosed that he had features of upper respiratory tract infection 1 week back and over-exerted on the day of admission, which could be the risk factors for HAPE in this individual after prolonged stay in a high-altitude area. He was managed with bed rest and high-flow oxygen inhalation to which he responded well clinically as well as radiologically as was evident in repeat chest radiograph done after 36 h [Figure 2].

CASE REPORT 2

A 39-year-old male with no co-morbid illness was admitted to our hospital on the evening of 15 January 2014 with severe breathlessness and cough. General examination revealed the following: temperature 98°F, blood pressure 146/90 mm Hg, pulse rate 136/min, respiratory rate 36/min, and oxygen saturation 53% at room air and 70% on high-flow oxygen. Chest auscultation revealed bilateral diffuse crepts. On nervous system examination, ataxic gait was present. Systemic evaluation was otherwise unremarkable. He was inducted to high-altitude area (height ~11,500 ft) on 28 November 2013. Based on clinical findings, he was suspected to have severe HAPE with associated high-altitude cerebral edema (HACE). Detailed evaluation regarding the risk factors for HAPE after prolonged stay in high-altitude area could not be undertaken in view of severe respiratory distress.

Immediate descent by air was not possible as it was already overnight and movement by road was blocked by snow. He was started on dexamethasone, nifedipine, and high-flow oxygen. In view of life-threatening hypoxemia and impossible immediate descent, he was started on therapy with hyperbaric chamber. He showed some response inside the hyperbaric chamber, but when he was taken out of the chamber, he had cardiorespiratory arrest and could not be revived. Autopsy findings showed parenchyma of both lungs to be edematous and frothy discharge on cut section consistent with bilateral pulmonary edema, normal brain and other viscera, suggesting HAPE as the cause of death.

DISCUSSION

HAPE is a potentially fatal medical condition of high altitude and usually occurs within 2-4 days of ascent to altitude above 8000 ft. HAPE after more than 4 days at the same altitude has not been reported till now.

Management of HAPE is predominantly descent; other modalities like supplemental oxygen aimed at maintaining oxygen saturation above 90% and use of portable hyperbaric chamber are options to be exercised when
descent is not feasible due to weather or logistic limitations. Nifedipine (a pulmonary vasodilator) at a dose of 60 mg daily in divided doses is to be administered as an adjunct to oxygen or descent.\(^1\) Continuous positive airway pressure (CPAP) is recommended as an adjunct to oxygen in a hospital setting.\(^1\) Beta-agonists and phosphodiesterases have not been assessed for effectiveness in systematic studies, though reports mention these agents to be of use in HAPE.\(^1\) Diuretics have no role in HAPE as patients are already likely to have volume depletion.\(^1\)

The use of hyperbaric chamber is limited to situations where immediate descent is not possible. There are various other indications of hyperbaric oxygen therapy,\(^2\) but in a high-altitude setting, it is of use in acute mountain sickness (AMS), HAPE, and HACE in situations where descent is not feasible. The optimum duration of management of AMS, HAPE, and HACE using portable hyperbaric chamber at high altitude according to a field study is 2, 4, and 6 h, respectively, at an altitude of 13,920 ft.\(^3\) It is presently not recommended for management of cases of frost bite due to insufficient data.\(^4\) The only absolute contraindication for hyperbaric oxygen therapy is untreated tension pneumothorax, whereas relative contraindications are impaired pressure equalization and cardiac disease.\(^2\)

A study has demonstrated that exercise in hypoxic conditions manifests changes in pulmonary capillary permeability, similar to HAPE.\(^5\) This phenomenon of sub-clinical HAPE associated with physical exertion at high altitude has been confirmed using chest sonography in recreational climbers.\(^6\) It is likely that in case 1, similar process of exertion-induced pulmonary edema could have occurred. The inflammation due to upper respiratory tract infection could have resulted in priming of pulmonary endothelium, manifesting as HAPE in response to exertion.\(^7\)

In case 2, the findings of normal brain on autopsy suggest the neurological manifestation is most likely due to hypoxic encephalopathy rather than HACE. Distinction between both conditions is difficult.\(^1\) Good response to portable hyperbaric chamber has been described in a study on pulmonary edema at extreme altitude.\(^8\) Patient-related factors like severity of condition, time elapsed since the occurrence of symptoms, and general health status could to be attributed to the difference in response to therapy using hyperbaric chamber. The study\(^8\) emphasizes on occurrence of HAPE after prolonged stay at 18,000 ft. A similar occurrence in acclimatized individuals is likely to occur after prolonged stay at 11,500 ft in susceptible individuals, especially with risk factors like upper respiratory tract infection and unaccustomed physical exertion. A case of HAPE in a Ladakhi native highlander was reported in association with unaccustomed physical exertion.\(^9\) This further strengthens the possibility of unaccustomed physical exertion emerging as a strong predisposing factor for HAPE even after acclimatization at high altitude.

HAPE is a potentially fatal form of acute altitude illness; high suspicion should arise for early diagnosis of HAPE even after acclimatization, to prevent mortality.

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Figure 1: Chest radiograph showing bilateral diffuse confluent alveolar opacities suggesting high-altitude pulmonary edema

Figure 2: Chest radiograph showing significant resolution after oxygen therapy
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