Case report

Comorbid cerebral and pulmonary edema at 7010 m/23000 ft: an extreme altitude perspective

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Abstract:
High Altitude Cerebral Edema (HACE) and High Altitude Pulmonary Edema (HAPE) are two dreaded altitude emergencies which can independently lead to complications. Two cases of suspected comorbid HACE and HAPE were managed at 5800 m/19000 ft in Karakoram Himalayas. Altitude acclimatization, purported to prevent high altitude illness, may not be protective. Comorbid HACE and HAPE at extreme altitude may present atypically necessitating high index of suspicion and prompt clinical decision making in challenging situations. One man HAPE bag/PHC is an excellent temporary measure in cases of delayed descent/evacuation. Due attention to extreme altitude emergencies is required in view of increased recreational, scientific and military activities at extreme altitude.

Key words: High Altitude Cerebral Edema (HACE); High Altitude Pulmonary Edema (HAPE); extreme altitude

Introduction
High Altitude Cerebral Edema (HACE) and High Altitude Pulmonary Edema (HAPE) are two dreaded altitude emergencies. HACE presents as headache not relieved by acetaminophen, incoordination, ataxia, altered mentation, diplopia, seizures, coma and high mortality. HAPE presents as dyspnea at rest, hemoptysis, incapacitating fatigue, cyanosis, chest tightness or congestion, wheezing, crepitations mostly along with headache, fever, tachycardia and tachypnoea. Both HACE and HAPE can independently lead to complications and together they can form a lethal duo leading to death within a few hours. Extreme altitude, defined as altitude above 5500 m/18000 ft, challenges humans to extremes of hypoxia, cold, terrain, weather and isolation. The disease presentation may be altered and diagnosis, management, descent and evacuation become difficult. Altitude acclimatization is purported as preventive, though at extreme altitude, altitude emergencies may occur after many days of stay. Despite reports of comorbid HACE and HAPE, there is little scientific attention. Two cases of suspected comorbid HACE and HAPE managed at 5800 m/19000 ft in Karakoram Himalayas are discussed with special emphasis to management in low resource set up at extreme altitude.

Case 1
A healthy mountaineer of 36 years, acclimatized at 7010 m/23000 ft and doing routine work for 28 days, presented with sudden onset breathlessness at rest at 2200 h after attempting to climb higher that day. Preliminary examination revealed normal temperature, hypertension, tachycardia and tachypnoea. The patient was administered oral acetazolamide 250 mg, dexamethasone 2 mg. Immediate descent was assisted by a paramedic through support-carry on foot to 6700 m/22000 ft followed by descent to 6400 m/21000 ft by 2345 h. The patient was nursed in a portable hyperbaric chamber (PHC/HAPE Bag) for an hour. He was subsequently lowered over a steep (70°) crevasse-laden ice wall in a basket stretcher which was belayed from both ends over ice with support by another mountaineer. He was received in a drowsy state in supine position at the base of the ice wall by 0130 h and transported to a nearby tent at 5800 m/19000 ft by 0145 h. The patient was dyspneic, centrally cyanosed, drowsy and had a pulse of 142/min, respiratory rate (RR) of 44/min, blood pressure (BP) 156/92 mm Hg, oxygen saturation 69% and bilateral fine patchy crepitations. The patient was administered oral acetazolamide 250 mg, dexamethasone 2 mg. Immediate descent was assisted by a paramedic through support-carry on foot to 6700 m/22000 ft followed by descent to 6400 m/21000 ft by 2345 h. The patient was nursed in a portable hyperbaric chamber (PHC/HAPE Bag) for an hour. He was subsequently lowered over a steep (70°) crevasse-laden ice wall in a basket stretcher which was belayed from both ends over ice with support by another mountaineer. He was received in a drowsy state in supine position at the base of the ice wall by 0130 h and transported to a nearby tent at 5800 m/19000 ft by 0145 h. The patient was dyspneic, centrally cyanosed, drowsy and had a pulse of 142/min, respiratory rate (RR) of 44/min, blood pressure (BP) 156/100 mm Hg, oxygen saturation 69% and bilateral fine patchy crepitations. The patient was administered oral acetazolamide 250 mg 8 hourly, intramuscular dexamethasone 2 mg 12 hourly, oral warm fluids and nursed in PHC for repeated one hour sessions overnight. Oxygen was not available. On repeat examination in the morning, there was symptomatic improvement, cough was observed, pulse was 110/min, RR 40/min, BP 144/92 mm Hg, oxygen saturation 76%, cyanosis had

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reduced but crepitations were present. Altered mental state, staggering gait on tandem gait test and incoordination were observed. Comorbid HACE and HAPE were diagnosed as per Lake Louis consensus criteria.\textsuperscript{2} The patient was continued on pharmacotherapy, oral feeds and PHC nursing without oxygen for more than 30 hours with gradual improvement while awaiting opportune weather till evacuation by helicopter the next day. The patient had improved mentation, pulse 100/min, RR 32/min, BP 140/88 mm Hg, oxygen saturation 78% though was ataxic at the time of evacuation.

**Case 2**

A healthy acclimatized paramedic of 28 years, acclimatized at 7010 m/23000 ft and doing routine work for 26 days, who had helped the above patient to descend from 7010 m/23000 ft to 6700 m/22000 ft late in the night, presented with breathlessness at rest at 0200 h. Preliminary examination revealed normal temperature, hypertension, tachycardia and tachypnoea. Immediate management and descent were similar to that offered to the above patient. The drowsy patient in supine position was brought to 5800 m/19000 ft by 0500 h. The patient was dyspneic, centrally cyanosed and had a pulse of 148/min, RR 40/min, BP 152/98 mm Hg, oxygen saturation 70%, bilateral fine patchy crepitations and altered mental status. The patient was managed by the same management protocol as the first patient. After 4 hours, there was symptomatic improvement, pulse was 106/min, RR 30/min, BP 142/94 mm Hg, oxygen saturation 78%. Cyanosis reduced but crepitations, altered mental status and abnormal tandem gait test were present. Comorbid HACE and HAPE was diagnosed as per Lake Louis consensus criteria.\textsuperscript{2} Continued on pharmacotherapy, oral feeds and PHC nursing without oxygen for more than 27 hours, the patient showed gradual improvement, improved mentation, ataxia, mild cough, pulse 102/min, RR 32/min, BP 140/90 mm Hg, oxygen saturation 80% by next morning when he was air evacuated on improvement of weather conditions.

**Discussion**

Considering the lethality of high altitude emergencies, a high index of suspicion, prompt diagnosis, management and evacuation are required at extreme altitude. Extreme altitude illnesses are likely to have an atypical, muted or overlapping presentation obscuring the diagnostic paradigm.\textsuperscript{6} In the backdrop of tent infrastructure and limited resources, a diagnosis of exclusion was based on clinical presentation and immediate treatment was started. Based on the clinical features, acute high altitude emergencies (HACE, HAPE), thromboembolic phenomena (cerebral venous thrombosis/CVT, pulmonary thromboembolism/PTE), cardiorespiratory diseases (bronchitis, myocardial infarction) and general illness (fatigue, dehydration, hypoglycemia) were considered. The diagnosis of high altitude emergencies was kept first as they have a higher incidence and most of the clinical features were concordant. HACE was diagnosed on the basis of altered mental status and ataxia whereas HAPE was diagnosed on the basis of dyspnoea at rest, weakness, tachypnoea, tachycardia, central cyanosis and crepitations.\textsuperscript{2} Thromboembolic phenomena were excluded as there was no history of prolonged immobilization, swelling of extremities, progressive breathlessness over many days or calf tenderness. Clinical diagnosis without diagnostic modalities has limitations in differentiating HACE from CVT and HAPE from PTE or pulmonary microemboli. Bronchitis, asthma or myocardial infarction may present as HAPE and were excluded based on past history and clinical presentation and thereafter confirmed by response to treatment. General illness such as fatigue, dehydration and hypoglycemia, which may have been present before onset or may have set in during patient’s movement over 3 hours in temperature around -30°C, may precipitate a moderate hypoxic insult to be presented as HACE and could not be completely excluded. The Lake Louis consensus criteria provide a clinical approach to high altitude illness, albeit they may have limited applicability for other similarly presenting illness occurring in a setting of recent gain in altitude.\textsuperscript{2} Comorbid HACE and HAPE may be diagnostically and therapeutically challenging. HACE may be missed in entirety as HAPE induced hypoxic depression may cause obtunded mentation and ataxia as seen in HACE.\textsuperscript{6} HAPE induced hypoxia is also known to accelerate the development of HACE. HACE doesn’t affect finger-nose test for ataxia. These two patients presented with symptoms of suspected HAPE, possibly with covert comorbid HACE, which was diagnosed the next day when they were able to stand. Headache was not seen in both cases.\textsuperscript{6} Hypertension may have been related to other stressors. Altered vitals may contribute to the diagnosis and monitoring of high altitude illness as observed in these patients. Treatment is difficult due to delay in descent/evacuation, unavailability of...
oxygen and infrastructural limitations. Astute prudence need to be exercised at extreme altitude where an over-diagnosis and to err on the safer side is likely to be beneficial.

Descent, involving lowering the altitude of the patient in the general area; and evacuation, involving transportation of the patient away from the area to institutionalized care, are life saving and form the mainstay of treatment. Ideal descent and evacuation should be rapid, feasible to conduct, involve fewer people and lead to maximum lowering of altitude for the most favourable outcome. Delays in descent due to limitations of night, weather, terrain, human help, communication, health, mountaineering and aviation capability may occur. Air evacuation is the fastest and most convenient mode at extreme altitude. Descent on foot at extreme altitude may be tediously protracted requiring expert mountaineering techniques, rotation of helping mountaineers/porters, enroute patient monitoring and may accelerate the progression of disease through ongoing exertion, cold exposure and hypoxia; as experienced in these cases. Human help is difficult to find and the support team is also rendered prone to accidents, high altitude emergencies and cold injuries may occur in the entire group in prolonged descent. The immediate descent of first patient on foot, carried out as the only available modality, proved beneficial although came at the cost of adverse health of the assisting paramedic. High altitude emergencies improve substantially on descent restoring near normal physiology in a breathless patient often leading to evasion of the hospital set up. Oxygen therapy at extreme altitude is limited by availability, warming and humidification. One man PHC forms an excellent substitute as a temporary measure till descent/evacuation.

While Acetazolamide and Dexamethasone help treat HACE and HAPE, Nifedipine forms the first choice in HAPE. Nifedipine was kept as a reserve due to the risk of hypotension which couldn’t have been monitored enroute. Patient improvement under pharmacologic, hyperbaric and descent strategies reaches a plateau and may not reach the optimal healthy state, requiring institutionalization. In the absence of clinical monitoring resources, uncertainty prevails and holding fort till final evacuation to institutionalized care remains a challenge. Comorbid HACE and HAPE may have good prognosis if immediate management is done. Cerebral changes in HACE and thromboembolic phenomena may be persistent and irreversible.

Healthcare delivery at extreme altitude is limited by unique problems which also effect experience and research. While acclimatization is purported to help survive extreme hypoxia, no successful acclimatization schedule has been documented beyond 5500 m/18000 ft, which is the cut off for extreme altitude. Complete acclimatization is not achieved even after weeks to months at extreme altitude due to inadequate physiologic compensation.

Extreme cold (up to -50°C) is combated by specialized cold weather clothing. Nutrition is compromised by inadequate availability of food and water, loss of appetite and thirst. Terrain restrictions (ice wall, steep slopes, crevasses, avalanches) make activities dangerous. Communication and evacuation systems are available at a premium. Medical facilities at extreme altitude are restricted to tent infrastructure without adequate staff, medicines and clinical equipment. The author was the only doctor at 19000 feet and no medical assistance was available when the cases occurred.

Altitude acclimatization, avoiding undue exertion, slow and graded ascent may help avoid altitude illness while maintaining activity and hydration may help avoid thromboembolic phenomena. Suspected comorbid HACE and HAPE in these two cases indicates that acclimatization may not protect against high altitude illness at extreme altitude. Reported cases of comorbid HACE and HAPE may represent the tip of an iceberg as these conditions present themselves at forbidden locations, improve on descent and evade the hospital set up thereby remaining under-reported to medical fraternity. Due attention to altitude illness is required in view of increased recreational, scientific and military activities at extreme altitude. Diagnostic difficulties will always arise at extreme altitude due to nonspecific and/or overlapping presentation necessitating high index of suspicion, prompt clinical decision making and to err on the safer side in challenging situations. One man HAPE bag/PHC is an excellent temporary measure in cases of delayed descent/evacuation.
References:


