A case report of high altitude sickness with features of acute mountain sickness (AMS), high altitude cerebral edema (HACE) and high altitude pulmonary edema (HAPE)

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Abstract

High altitude sickness comprises of acute mountain sickness (AMS) high altitude cereberal edema (HACE) and high altitude pulmonary edema (HAPE) its incidence is 0.1- 4%. It occurs at an altitude of above 2500meters with rapid accent without acclimitisation. We present a case report of a 22 year old male who had gone for pilgrimage at high altitude with rapid accent to a altitude of more than 3590m developed high altitude sickness with features of HAPE and HACE .He was treated with steroids and invasive mechanical ventilation ventilation and supportive care, responded well and was discharged after full recovery without any sequelea.

Key words High altitude pulmonary edema, High altitude cereberal edema, invasive mechanical ventillation. acclimitization

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Introduction

Two forms of high altitude illness can be distinguished a cerebral form called acute mountain sickness (AMS) and a pulmonary form called high altitude pulmonary oedema (HAPE). Altitude, the rate of ascent, and individual susceptibility in particular are the major determinants of AMS and HAPE in mountaineers and trekkers. Among trekkers in the Himalayas and mountaineers in the Alps ascending at a rate of >600 m/day prevalence of AMS at altitudes between 4000 m and 5600 m is 30–60% [1–7]. In contrast to AMS, HAPE is less frequent. The estimated incidence of HAPE in visitors to ski resorts in the Rocky Mountains of Colorado is 0.01–0.1% [8]. In a general alpine mountaineering population, the prevalence of HAPE is <0.2% [9]. The HAPE incidence among trekkers in the Himalayas and climbers in the Alps ascending at a rate of >600 m/day is around 4% In an unselected population of Indian soldiers, airlift to an altitude of 5500 meters was associated with a HAPE incidence of up to 15% [10].

Case report

A 22 year old male from Calcutta india about 1.5-9meters above sea level had arrived for pilgrimage (Amarnat shrine pilgrimage) located at an altitude of 38884m (amaranth cave). Patient had climbed to an altitude of 3574m (sheshnag) in single day after arrival at base camp (pahalgam altitude 2740m). On night of arrival at this altitude he complained of tightness in chest, breathlessness, tiredness exhaustion, altered sensorium in form of irrelevant talking improper behaviour like refusal to feed and poor response to verbal commands. He was managed at camp site health care facility with oxygen and iv fluids, but he continued to deteriorate, next day he was shifted to base camp and then to our hospital for further management. There was also h/o episode of seizure at base camp. On examining the patient at arrival in our facility his GCS was 10 /15 pulse 108 bpm Bp 130/80 RR 35, Tem. 101, Spo_2 80%. Systemic examination was unremarkable apart from low GCS.

His labs where Hb 13.6gm/dl TLC 20,100, Platlets 74 thousand, urea 33mg/dl, creat. 0.87mg/dl, Na 146meq K 4.1meq Glu. 113. ABG Ph 7.40 Pco_2 39 Po_2 50 $So_2$80% Hco_3 25. Ecg showed sinus Tachycardia, X ray diffuse bilateral infillitrates. NCCT head was equivocal, CSF wnl and ECHO wnl. Base line cultures where sent. Based on history clinical examination and labs a preliminary diagnosis of High altitude sickness with HACE and HAPE was made with differential diagnosis of pneumonia with septic encephalopathy.

Patient was started on dexamethasone and broad spectrum antibiotics a cvp line and an arterial line was placed for invasive blood pressure moinitering fluid management. On second day of admission patients GCS deterioted to 6/15 and hypoxia also increased and patient was n respiratory distress. Patient was intubated and invasive mechanical ventilation started with routine care. During course of admission in ICU patients ABG and hypoxia improved, serial chest radiograph showed clearance of infilitrates and GCS also showed improvement. Patient remained on ventilation for two

days and was extubated on 4thh day of admission. He was discharged to ward after 6th day of admission with no sequeale.

Discussion

HAPE presents within 2–5 days after arrival at high altitude[10–12]. It is rarely observed below altitudes of 2500–3000 m and after 1 week of acclimatization at a particular altitude. Our patient came from low altitude 1.5-9m above sea level to a altitude of 3500m without any acclimatization and prophylytic medication use hence was a high risk for high altitude sickness.

Early symptoms of HAPE include exertional dyspnoea, cough, and suddenly reduced exercise performance. As pulmonary oedema progresses, orthopnoea, breathlessness at rest, and gurgling in the chest develop, cough worsens, and pink frothy sputum reveals overt pulmonary oedema [10–12]. There may becyanosis, tachypnoea, tachycardia, and frequently body temperature >37.5 °C[13]. HACE presents as disturbances of consciousness that may progress to deep coma, psychiatric changes of varying degree, confusion, and ataxia of gait. Our patient had similar presentation, furthermore his x ray finding, along with clinical picture and labs improved within a day of invasive ventilation and starting on steroid. Which ruled out any infective process involving lungs his bed side ECHO was unremarkable.

HAPE is a non-cardiogenic pulmonary edema characterized by patchy pulmonary vasoconstriction that leads to over perfusion in some areas, hence increased pulmonary capillary pressure (>18 mmHg) and capillary "stress" failure. There is also role endothelial dysfunction due to hypoxia causing impaired release of nitric oxide (an endothelium-derived vasodilator) and at same time increased release endothelin-1 (a potent vasoconstrictor).

Impaired cerebral autoregulation in the presence of hypoxic cerebral vasodilatation and altered permeability of the blood-brain barrier due to hypoxia-induced chemicals may all contribute to brain edema in case of HACE[14]. Preventive measure include slow ascent limiting the average daily ascent rate above altitude 2000 m to less than 350–400 m/day[15]. Which was not followed in this patient. Nifedipine 20 mg every 8 h starting 24 h before ascent or 10 mg tadalafil bid (phosphodiesterase-5 inhibitor) continued until descent has been found to decrease the incidence of HAPE from 63% to 10%.)[16]. If AMS is present despite pulmonary vasodilator prophylaxis, additional acetazolamide is recommended [17,18], dexamethasone may also have role in prevention[19,20].

Descent to lower altitude, use of supplementary oxygen (aimed at bringing oxygen saturation to >90%) minimum exertion and warm environment are enough for treating in mild cases of HAPE. In AMS / HACE additionally use of acetazolamide treat most cases. For severe cases HACE dexamethasone (8 mg orally or parenterally) is highly effective. When descent is not possible a portable hyperbaric chamber is effective and buys time [14].

Conclusion

We received patient when disease process was established. Patient was from low altitude region had neither received any acclimatization nor any medical prophylaxis before embarking on high altitude journey. Thus availability of prophylactic measures and education of high altitude sickness can go long way in prevention of high altitude sickness in high risk group particularly in case of people going for pilgrimage in large groups.

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