CASE REPORT

High-Altitude Cerebral Edema With Absence of Headache

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Headache is the cardinal symptom of acute mountain sickness (AMS). The headache normally worsens, with increased cerebral affection and the development of high-altitude cerebral edema (HACE). A Norwegian expedition aimed to climb Baruntse (7129 m) in Nepal in 2003. At 5400 m a 35-year-old man felt exhausted. The next day he aborted his attempt at further climbing as a result of extreme fatigue. Over the next 24 hours he developed cough, dyspnea, and severe hypoxia before progressing to ataxia and blurred vision. At no point did he experience headache or nausea. The patient was evacuated by helicopter. He improved immediately after descent and recovered completely within a week. The speed of progression from AMS to HACE varies. Abrupt onset of HACE is occasionally reported. High-altitude pulmonary edema (HAPE) may induce severe hypoxia that can lead to rapid development of HACE. High-altitude cerebral edema in the setting of HAPE was the most likely diagnosis despite the unusual lack of headache. Rapid onset of HAPE with subsequent severe desaturation should raise awareness of the development of HACE, even in the absence of headache.

Key words: HACE, HAPE, headache

Introduction

Acute mountain sickness (AMS) is the collective term for a syndrome of nonspecific symptoms that can affect unacclimatized travellers shortly after ascent to altitudes greater than 2500 m. Acute mountain sickness is defined as the presence of headache and one or more of the following symptoms: nausea, vomiting, anorexia, fatigue, dizziness, or sleep disturbances. Headache is the cardinal symptom.¹ In extreme cases, AMS may progress to high-altitude cerebral edema (HACE), an acute encephalopathy characterized by ataxia and depressed level of consciousness.² Brain edema formation diminishes when inspired and alveolar PO₂ is increased by supplemental oxygen, descent, or hyperbaric treatment.³ Many conditions can mimic HACE; however, the absence of headache indicates another etiology.⁴ High-altitude pulmonary edema (HAPE) is characterized initially by fatigue, dyspnea, and cough. HAPE progresses to a life-threatening condition if untreated. This case report describes a climber with severe HAPE and concomitant HACE without headache.

Case report

The Norwegian Baruntse Expedition started from Lukla, Nepal (2800 m) on May 5, 2003; the team had the goal of climbing Baruntse (7129 m). The group consisted of 5 young and healthy men with climbing and high-altitude experience, although the patient in question had never been above 3500 m. The plan for acclimatization was to stay in Lukla for 1 night before increasing the sleeping altitude by 300 m every night, allowing 2 extra resting days for every 1000-m altitude gain. The team followed this plan but stayed a total of 6 additional nights at various altitudes, as all 5 climbers in turn developed diarrhea.

On the morning of the 16th day of the expedition, at an elevation of 5400 m, the patient, a 35-year-old man, felt tired. Despite feeling unwell, he climbed 400 m of easy rock and ice and moved to another camp at 5400 m to spend the night. The next day he was not improved and still felt exhausted. He therefore descended to 5300 m. Upon examination he had slightly cyanotic lips and dyspnea at rest. Auscultation of the chest revealed fine crepitations bilaterally. His blood pressure was 145/90 mm Hg and his respiratory rate was 25 breaths per mi-

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nute. He was drowsy and was not able to drink or eat properly. He had no headache or nausea. He was placed in a sleeping bag in a tent, which warmed him and allowed for good peripheral circulation. Oxygen saturation measured at a fingertip with a PulsiQuant 8040 pulse oximeter (GTS, Hong Kong, China) showed 46% while the patient was breathing ambient air. The accuracy of the PulsiQuant pulse oximeter between a SpO_2 value of 50% to 70% is $\pm 3\%$.

During the next 2 hours the patient developed ataxia and blurred vision. Ophthalmoscopy was not done. His oxygen saturation steadily declined during the next 25 hours. Since HACE was suspected, the patient received 8 mg of dexamethasone intravenously, followed by 4 mg every 6 hours, to a total of 20 mg with no clinical improvement. He was already taking acetazolamide (500 mg) daily. Although HAPE was also suspected, he received no specific HAPE medication, as the team did not carry these medicines.

The expedition had a limited supply of oxygen (700 L) and a Gamow portable hyperbaric bag (Chinook Medical Gear, Durango, CO). Initially the patient received $10 \text{ L}\cdot\text{min}^{-1}$ of oxygen via face mask, and his saturation increased to 60% to 70%. He was then placed into the Gamow bag with a fresh air flow of $30 \text{ L}\cdot\text{min}^{-1}$. The pressure inside the bag was 13 kPa, and he had a SpO_2 value of 70% to 80% during compression without supplemental oxygen. Because of his severe coughing, the patient could tolerate only 3 sessions of 45 minutes in the Gamow bag before the treatment had to be terminated. Once outside the Gamow bag, the patient immediately desaturated. In order to conserve oxygen, treatment was continued with periods of oxygen ($2 \text{ L}\cdot\text{min}^{-1}$ via nasal cannula) for 45 minutes, followed by 15 minutes of breathing ambient air every hour for the next 25 hours. The lowest recorded SpO_2 value (off oxygen) was 28% despite apparently good peripheral circulation. At the same time, another climber, without symptoms, had a saturation of 86%, as expected for this altitude.

Evacuating the victim by land would have demanded climbing higher or several days of walking at the same altitude. A military rescue helicopter was therefore requested via satellite telephone in the morning of symptom onset. Ongoing military conflict in the area prevented evacuation until 25 hours later. On the second day, the patient was flown to Kathmandu (1300 m). His oxygen saturation on arrival was 85% without supplemental oxygen. His ataxia and vision disturbances persisted for 2 days. After a week at low altitude, the patient completely recovered clinically. No radiographic studies were performed.

Discussion

Our climber developed fatigue, dry cough, dyspnea, anorexia, mild cyanosis progressing to severe desaturation, a depressed level of consciousness, ataxia, and blurred vision within 24 hours. Despite a recent gain in altitude before onset of symptoms, his gain in sleeping altitude throughout the climb had not exceeded 300 m per night. He experienced no headache. Hyperbaric treatment, medications, and supplemental oxygen provided only temporarily relief. Emergency descent by helicopter, however, demonstrated immediate improvement, with complete recovery within a week.

Most commonly, HACE develops as a progression from AMS over a period of 24 to 36 hours, although abrupt onset of HACE is occasionally reported, especially at extreme altitude.^{6,7} Prodromal symptoms of AMS may be minor or absent, resulting in a diagnostic delay.⁷ Hultgren⁸ showed that the more complicated the case of HACE was, the less likely it was that headache would occur. High-altitude cerebral edema may occur independently of HAPE or concomitantly as HAPE develops.⁶ High-altitude cerebral edema is much more likely in patients with HAPE as a result of their more severe hypoxia. Thirteen percent of 52 patients with HAPE in the French Alps had stupor or coma,⁹ and 14% of 150 patients with HAPE in the Colorado Rockies developed HACE.¹⁰

In retrospect, we believe that the extreme desaturation due to HAPE masked the early-stage symptoms of AMS, including headache, progressing directly toward a more severe cerebral affection. High-altitude cerebral edema in the setting of HAPE was therefore the most likely diagnosis.

Differential diagnoses included acute psychosis, central nervous system infection, brain tumor, metabolic disorders, progressive/disseminated lower airway infection, and hypothermia,¹ none of which were likely in this climber.

Conclusion

Severe altitude illness may develop despite adherence to a conservative acclimatization regime. Availability of supplemental oxygen and portable hyperbaric chambers should not delay the decision to organize a rapid evacuation of affected patients to lower altitude. Rapid onset of HAPE with subsequent severe desaturation should raise awareness of the development of HACE, even in the absence of headache.

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