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Case Report

Life Threatening High-Altitude Cerebral Edema and High-Altitude Pulmonary Edema in A Trekking Guide on the Kathmandu–Mansarovar Route

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Abstract

High-altitude illness remains a critical risk for travelers to mountainous regions, particularly when rapid ascent occurs without adequate acclimatization. We describe a 52-year-old trekking guide who developed simultaneous high-altitude cerebral edema (HACE) and high-altitude pulmonary edema (HAPE) while leading a group to Mansarovar via the Kathmandu–Rasuwagadhi–Kerung overland route. Despite extensive experience at altitude, he ascended from 1,400 m to over 4,600 m within two days without prophylaxis. He presented with profound hypoxemia, dizziness, ataxia, and dry cough. Descent was delayed due to logistical barriers, and he required two days of travel before reaching a tertiary hospital. Imaging confirmed pulmonary edema, and persistent neurological deficits were consistent with HACE. Treatment with supplemental oxygen and dexamethasone led to complete recovery. This case highlights that vehicular travel does not mitigate altitude risk and emphasizes the importance of gradual ascent, prophylactic measures, and timely intervention in preventing life-threatening outcomes.

Keywords: *Altitude, Cerebral edema, High altitude pulmonary edema*

Introduction

High-altitude illness remains a major challenge for trekkers and guides in the Himalayas, especially those with road access where rapid ascent often exceeds recommended safety margins [1]. The overland route from Kathmandu (1,400 m) to Mansarovar (4590m) via Rasuwagadhi-Kerung, opened in 2017, allows a gain of nearly 3,000 m within 48 hours, posing significant risks [2]. Despite awareness, experienced travelers may underestimate dangers, particularly when using vehicles rather than trekking on foot.

This case describes a 52-year-old trekking guide who developed severe HACE and HAPE after rapid ascent without prophylaxis, highlighting the critical importance of acclimatization and preventive measures even for seasoned professionals.

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Case Report

A 52-year-old male, non-smoker, trekking guide by profession with no known comorbidities, presented to the emergency department of a tertiary hospital in Kathmandu with shortness of breath and dizziness for three days. He was an experienced high-altitude guide who had embarked on an overland pilgrimage to Mansarovar from Kathmandu with 20 tourists and three other guides via the Rasuwagadhi–Kerung highway, a route opened for international travel in 2017. The rapid ascent profile of this route poses a significant risk for high-altitude illness.

On the first day, the team ascended from Kathmandu (1400 m) to Myalam (4350 m), gaining nearly 3000 m within a few hours. His companions took acetazolamide prophylaxis, but he declined due to prior tolerance at altitude. That night, he developed progressive shortness of breath, headache aggravated by coughing and straining, anorexia, and insomnia, which he attributed to dehydration and fatigue. Despite persistent symptoms, he continued the next day toward Saga (4640 m), a 10-hour drive through the Tibetan plateau. On arrival, he was unable to stand unaided, walked with an ataxic gait, and experienced worsening dyspnea with dry cough. His oxygen saturation was 35% on room air. As night had fallen, descent was deferred; he was kept on supplemental oxygen overnight. The following morning, he was transported by ambulance to Kathmandu with oxygen support, arriving at Tribhuvan University Teaching Hospital (TUTH) after two days of descent. At TUTH (1400 m), his consciousness had improved and oxygen saturation was 91% on room air. Examination revealed bilateral basal crepitations, and chest radiograph showed pulmonary edema. (Figure 1)

Figure 1: CXR showing Pulmonary Edema at presentation

Tandem gait testing demonstrated ataxia and dizziness. A working diagnosis of HACE with HAPE was made. He was admitted for observation with supplemental oxygen. By the second day, he maintained saturation without oxygen, and chest X-ray normalized by day three (Figure 2). However, dizziness and gait instability persisted despite a normal CT head. Administration of intravenous dexamethasone 8 mg stat led to rapid improvement. He was discharged the next day in stable condition.

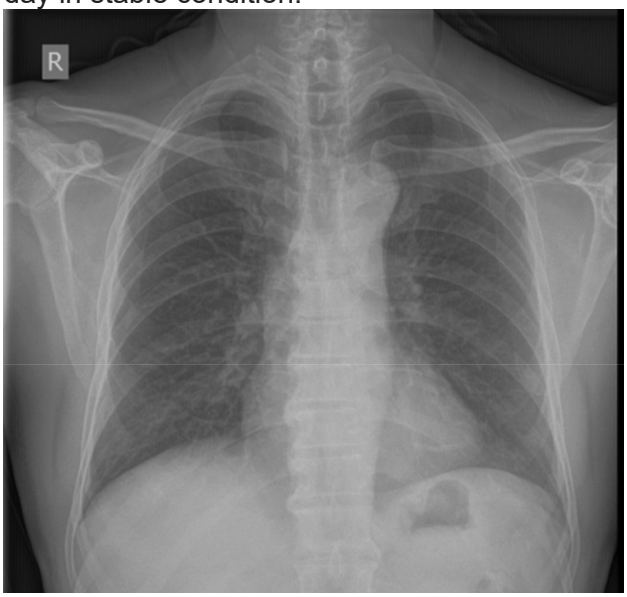

Figure 2: Normal CXR two days later

Discussion

This case underscores the growing risk of severe high-altitude illness (HAI) as overland routes to high-altitude destinations become more accessible [1]. Traditionally, trekking in the Himalayas involves gradual ascent, providing time for acclimatization. However, establishment of road access to high altitudes sees spike of high-altitude illness in those areas. This was seen in a study done in fall 2016, where more than half of patients with high altitude illness travelled to Manang from Besisahar by road [3]. Mount Kailash Mansarovar (4590m) is one of the high-altitude pilgrims with high incidence of altitude illness where the only route from Nepal was to travel by helicopter previously [4]. However, the opening of Kathmandu–Mansarovar route via Rasuwagadhi-Kerung in 2017 has now allowed rapid gain of elevation from 1,400 m to over 4,300 m on the first day, an increase of nearly 3,000 m [5]. This ascent profile far exceeds recommended safety margins.

The Wilderness Medical Society (WMS) guide-

lines emphasize that once above 2,500 m, daily sleeping altitude should increase by no more than 300–500 m, with an additional acclimatization day for every 1,000 m gained [6]. Skipping these measures predispose even healthy individuals to acute mountain sickness (AMS) and its severe complications. These guidelines updated periodically provide evidence-based recommendations for clinicians on best practices. A 6-day ascent resulted in lower incidence of AMS incidence (52.6%) compared to shorter routes on Mount Kilimanjaro, highlighting the importance of slow ascents. Slower ascent profiles and acetazolamide use can reduce AMS risk [7]. In this case, the absence of acclimatization was critical.

The patient, an experienced guide, perceived his risk to be low because of vehicular travel rather than trekking on foot. This misconception that traveling by vehicle negates the physiological stress of hypobaric hypoxia is common and dangerous. Hypoxia is primarily a function of barometric pressure and inspires oxygen fraction, not physical exertion [8]. Road travel can even exacerbate the risk by exposing individuals to higher altitudes more quickly, reducing the time available for ventilatory and hematological adaptation. This poses significant physiological challenges, particularly for those with preexisting medical conditions [9]. Risk factors include short trip duration, and travel agency-organized trips [10]. Guides and tourists alike should be advised that vehicles do not mitigate altitude risk; the ascent profile is the key determinant of illness.

Another significant factor was the non-use of prophylactic acetazolamide. Acetazolamide is well established to enhance acclimatization by increasing ventilation and reducing periodic breathing. A systematic review and meta-analysis revealed a 48% relative risk reduction in AMS incidence with acetazolamide prophylaxis [10]. While the patient's team used prophylaxis, his refusal, based on prior tolerance of high-altitude exposure, highlights an educational gap. In hindsight, prophylaxis and scheduled acclimatization halts particularly two nights at Kerung (2,800–3,000 m) would likely have prevented symptom progression.

Conclusion

This is one of the first case reports of altitude sickness on Kathmandu-Mansarovar journey by road. This case is notable because it illustrates the simultaneous occurrence of HACE and HAPE, a rare but life-threatening combination.

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Conflict of interest: None

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