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Faith over Physiology: A Case of Hyper-Acute HAPE in a Fasting Pilgrim at Gosaikunda Lake (4,380 m)

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Case Report

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Abstract

Background

High-altitude pulmonary edema (HAPE) is a life-threatening, non-cardiogenic pulmonary condition that typically develops 2–5 days after rapid ascent above 2,500 meters. While HAPE frequently coexists with acute mountain sickness (AMS), it can occur independently. High-altitude religious pilgrimages introduce unique physiological stressors—including ritual fasting, barefoot walking, severe cold exposure, heavy exertion, and rapid ascent driven by religious motivation—that are rarely encountered in standard recreational trekking populations.

Case presentation

A 62-year-old woman from Kathmandu developed sudden-onset dyspnea within hours of arriving at Gosaikunda Lake (4,380 m) during the annual Janai Purnima pilgrimage. Her ascent was completed in under 48 hours, compounded by ritual fasting and barefoot trekking over rugged terrain. She presented without typical AMS symptoms such as headache, nausea, or vomiting. On evaluation, her room-air oxygen saturation was 80%, which improved to 96% on 4 L/min oxygen therapy. She demonstrated a respiratory rate of 30 breaths/min and bilateral basal crepitations. Managed with supplemental oxygen, oral nifedipine, and acetazolamide, her symptoms resolved rapidly during assisted descent to Chandanbari (3,330 m).

Conclusions

This case highlights an atypical, rapid presentation of HAPE triggered by the unique intersection of altitude and pilgrimage-associated physiological stressors (fasting, cold exposure, barefoot exertion) without preceding neurological signs of AMS. Recognizing these unique risk factors in pilgrimage medicine is critical for early field recognition, prompt descent, and avoiding fatal outcomes during mass high-altitude religious gatherings.

Background

High-altitude pulmonary edema (HAPE) is a potentially life-threatening, non-cardiogenic pulmonary condition that typically occurs in unacclimatized individuals who ascend rapidly to altitudes above 2,500 meters [1, 2]. HAPE remains one of the most serious forms of high-altitude illness, with an incidence among Himalayan trekkers and climbers in the Alps around 4% depending on the rate of ascent, altitude reached, and individual susceptibility [2].

HAPE results from excessive hypoxic pulmonary vasoconstriction, leading to elevated microvascular pressures that cause mechanical stress and high-permeability, non-inflammatory leakage of fluid and red

blood cells into the alveoli. Regional uneven vasoconstriction and impaired endothelial and epithelial function further contribute to susceptibility by overwhelming the lung's capacity to maintain fluid balance [3, 4].

While HAPE often follows symptoms of acute mountain sickness (AMS) such as headache, fatigue, nausea, and dizziness [5], it can also present independently, especially in individuals with rapid ascent profiles or increased susceptibility [6]. HAPE is characterized by dry cough, progressive dyspnea with exertion, and decreased exercise tolerance, generally developing within 2 to 4 days after arrival at high altitude [4]. The absence of AMS symptoms can delay diagnosis, increasing the risk of progression to respiratory failure. Early recognition and prompt intervention—primarily descent and oxygen therapy—are critical to prevent morbidity and mortality.

While traditional risk factors such as rapid ascent and ambient cold exposure are well recognized, high-altitude religious pilgrimages in the Himalayas introduce a distinct clinical entity known as pilgrimage medicine. Every year, thousands of pilgrims ascend rapidly to sacred high-altitude destinations like Gosaikunda Lake (4,380 m). These journeys combine extreme altitude with unique non-altitude physiological stressors, including ritual fasting, barefoot trekking over freezing alpine terrain, extreme physical exhaustion, sleep deprivation, and intense emotional devotion that often drives pilgrims to ignore early warning signs of illness.

This case report describes a 62-year-old woman who developed early-onset HAPE at 4,380 meters in Gosaikunda, Nepal, without preceding symptoms of AMS. Her case is notable for the rapid onset of symptoms within hours of arrival at high altitude, absence of prior cardiopulmonary disease, complete resolution with descent and oxygen therapy, and the presence of compounding pilgrimage-specific risk factors.

Case presentation

A 62-year-old female from Thankot, Kathmandu, presented to the temporary Himalayan Rescue Association health camp at Gosaikunda with acute-onset shortness of breath at rest, which began around 07:00 AM and persisted for two hours, prompting her visit to the camp at 09:00 AM. She reported being in her usual state of health before this episode and denied any history of cough, sputum production, fever, chest pain, orthopnea, or pedal swelling suggestive of cardiac disease. She also denied headache, nausea, vomiting, or other symptoms of acute mountain sickness. Her past medical history was unremarkable, with no known diagnosis of chronic obstructive pulmonary disease, asthma, or cardiovascular disease, and she had not used acetazolamide or any other prophylactic medication before ascent.

On examination, the patient was fully conscious and oriented, with a Glasgow Coma Scale score of 15/15. Her oxygen saturation on room air was 80% on pulse oximetry at rest, which improved to 96% with 4 liters of oxygen via mask. Her blood pressure was 110/80 mmHg, axillary temperature was 99.6°F (37.6°C), and she had a respiratory rate of 30 breaths per minute with labored breathing. Chest

auscultation revealed occasional bilateral basal crepitations. The heel-to-toe test showed no signs of ataxia; the neurological assessment, including tandem walk and finger-to-nose testing, was normal. There was no peripheral edema or jugular venous distension. Cardiovascular examination was unremarkable, with no murmurs on auscultation.

Her ascent profile revealed that she began her pilgrimage from Kathmandu (1,300 m) and traveled by bus to Dhunche (1,950 m).(Fig. 1) On the first day, she trekked from Dhunche to Dhimsa (3,050 m). On the second day, she ascended directly from Dhimsa to Gosaikunda (4,380 m) by evening, gaining a significant altitude in a short time. She spent the night at a hotel beside Gosaikunda Lake. On the following morning, she developed sudden breathlessness at rest, which progressively worsened. Furthermore, her ascent was heavily influenced by traditional pilgrimage practices during the Janai Purnima festival. She underwent ritual fasting with near-total restriction of food and fluid intake over 24 hours, walked barefoot over cold, wet, and rocky alpine trails during the final stage of the climb, and experienced prolonged cold exposure and severe physical exertion without adequate rest.

Given the recent rapid ascent, acute-onset dyspnea, hypoxemia, and bilateral basal crepitus in the absence of prior respiratory or cardiac disease, a diagnosis of High-Altitude Pulmonary Edema (HAPE) was made. The patient was immediately managed with supplemental oxygen, oral acetazolamide 250 mg, and nifedipine 20 mg, and was advised to descend promptly. The Himalayan Rescue Association team at Gosaikunda, in coordination with the Nepal Army, assisted her descent with supplemental oxygen. During the descent, her symptoms gradually improved, and by the time she reached Chandanbari (3,330 m), she was able to walk without supplemental oxygen and reported complete resolution of her shortness of breath.

Discussion

This 62-year-old woman developed acute shortness of breath at rest within hours of arriving at Gosaikunda (4,380 m), despite having no prior cardiopulmonary disease and no preceding symptoms of acute mountain sickness (AMS). Her case is notable for the early onset of high-altitude pulmonary edema (HAPE) without the typical prodromal features of AMS, such as headache, nausea, or vomiting. The absence of these warning signs may have delayed recognition and intervention, increasing the risk of deterioration had she not sought care promptly.

High-altitude pulmonary edema (HAPE) results from exaggerated hypoxic pulmonary vasoconstriction, leading to elevated pulmonary artery pressures and mechanical stress failure of the alveolar-capillary barrier, causing non-inflammatory, protein-rich fluid leakage into the alveoli [7]. Uneven regional vasoconstriction contributes to localized overperfusion and capillary stress, while impaired nitric oxide signaling and increased endothelin levels further exacerbate vascular pressure and permeability [3, 4]. Recent evidence also shows that patients with HAPE exhibit elevated levels of cell-free DNA (cfDNA), reduced cfDNA integrity, and increased mitochondrial cfDNA, which correlate with hypoxemia and

disease severity, suggesting a potential role for cfDNA as a biomarker of hypoxia-induced pulmonary injury [8].

The patient's presentation—acute dyspnea at rest, hypoxemia (SpO_2 80% on room air), tachypnea, and bilateral basal crepitations—aligns with classical HAPE features [5, 6]. However, the absence of headache, nausea, or vomiting challenges the conventional diagnostic sequence where AMS precedes or accompanies HAPE [4]. This supports growing evidence that HAPE and AMS are pathophysiologically distinct conditions: AMS is primarily a neurovascular reaction to hypoxia—characterized by cerebral vasodilation, increases in intracranial pressure, and possibly fluid shifts in brain tissue [9]. In contrast, HAPE is driven by exaggerated hypoxic pulmonary vasoconstriction, elevated pulmonary artery and capillary pressures, patchy over-perfusion, and capillary stress-failure in the lung [10]. The absence of AMS symptoms may reflect individual variability in cerebral versus pulmonary responses to hypoxia, suggesting that some individuals may be selectively vulnerable to pulmonary complications without experiencing neurological symptoms. Clinically, this distinction is critical, as reliance on AMS symptoms alone may delay recognition of HAPE, particularly in older adults or those with atypical presentations.

Another unique aspect of this case is the rapid onset of HAPE symptoms—within hours of arrival at 4,380 meters—which deviates from the typical 2–4 day latency period observed in most cases [1, 5, 11]. Such early presentation is rare and is strongly driven by the compounding physiological factors inherent to pilgrimage medicine. Millions of pilgrims undertake rapid, arduous ascents to Himalayan shrines, often under severe physical and environmental stress [13]. In this patient, ritual fasting induced dehydration and caloric deficits, compromising vascular regulation and metabolic resilience. Barefoot walking on freezing alpine ground exposed her to continuous severe cold stress, which stimulates systemic sympathetic drive and accentuates hypoxic pulmonary vasoconstriction [3, 7]. Heavy physical exertion on steep inclines without prior acclimatization or footwear sharply raised oxygen uptake and cardiac output, forcing high blood flow through constricted pulmonary microvessels and triggering stress failure. Together with her rapid ascent profile and potential age-related reductions in ventilatory and endothelial reserves, these factors combined synergistically to accelerate pulmonary edema formation [3, 6, 7].

In this case, the patient's symptoms resolved completely with descent and supplemental oxygen alone, without the need for advanced interventions. She showed marked improvement upon reaching Chandanbari (3,330 m), consistent with the typical reversibility of HAPE when recognized and treated early. This reinforces the principle that descent remains the cornerstone of HAPE management, with supplemental oxygen and pharmacologic agents like nifedipine serving as adjuncts [12, 13]. The clinical context of rapid ascent, hypoxemia, bilateral crepitations, and dramatic response to descent strongly supported a diagnosis of HAPE.

Limitations in this case include the lack of imaging, such as a chest X-ray or ultrasound to confirm pulmonary edema, no echocardiography done to rule out structural heart anomalies which have been associated with increased HAPE susceptibility, and absence of biomarkers (e.g., BNP, CRP) to rule out

cardiac or infectious causes. However, the rapid clinical improvement with descent and oxygen therapy strongly supports the diagnosis of HAPE. Another limitation is the absence of prior altitude exposure history, which could help assess individual susceptibility.

Future directions should focus on developing point-of-care diagnostic tools for HAPE in remote high-altitude settings, integrating portable ultrasound and pulse oximetry with clinical scoring systems. Genetic screening and endothelial function testing may also help identify high-risk individuals before ascent. Public health initiatives must specifically target high-altitude mass pilgrimages, educating pilgrims on the severe physiological risks of combining rapid unacclimatized ascent with ritual fasting, barefoot trekking, and cold exposure.

Conclusions

This case highlights an atypical presentation of high-altitude pulmonary edema (HAPE) characterized by early onset and absence of preceding acute mountain sickness (AMS) symptoms in a previously healthy woman. The rapid ascent without acclimatization, combined with pilgrimage-specific physiological stressors (ritual fasting, barefoot trekking, severe cold exposure, and heavy physical exertion) and age-related physiological vulnerability, likely contributed to the development of HAPE within hours of reaching 4,380 meters. This case underscores the critical importance of early recognition and prompt descent in managing high-altitude pulmonary edema, especially when symptoms arise in the absence of acute mountain sickness. Many individuals undertaking high-altitude travel invest considerable effort and may be reluctant to descend, even when symptoms worsen. However, continuing ascent—especially by alternative transport—can be catastrophic. While acetazolamide is effective for AMS prophylaxis, it has no established role in preventing or treating HAPE. Oxygen therapy and pharmacologic agents like nifedipine may support recovery, but descent remains the cornerstone of treatment.

Declarations

Ethics approval and consent to participate

No ethical approval was needed from the institutional review committee for this single case report. Written informed consent was obtained from the patient to participate in this study.

Consent for publication

Informed written consent was obtained from the patient including clinical details and clinical images for publication.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Govinda Bhandari initiated the research and wrote the manuscript, and Padam Raj Joshi reviewed and edited the manuscript.

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Figures

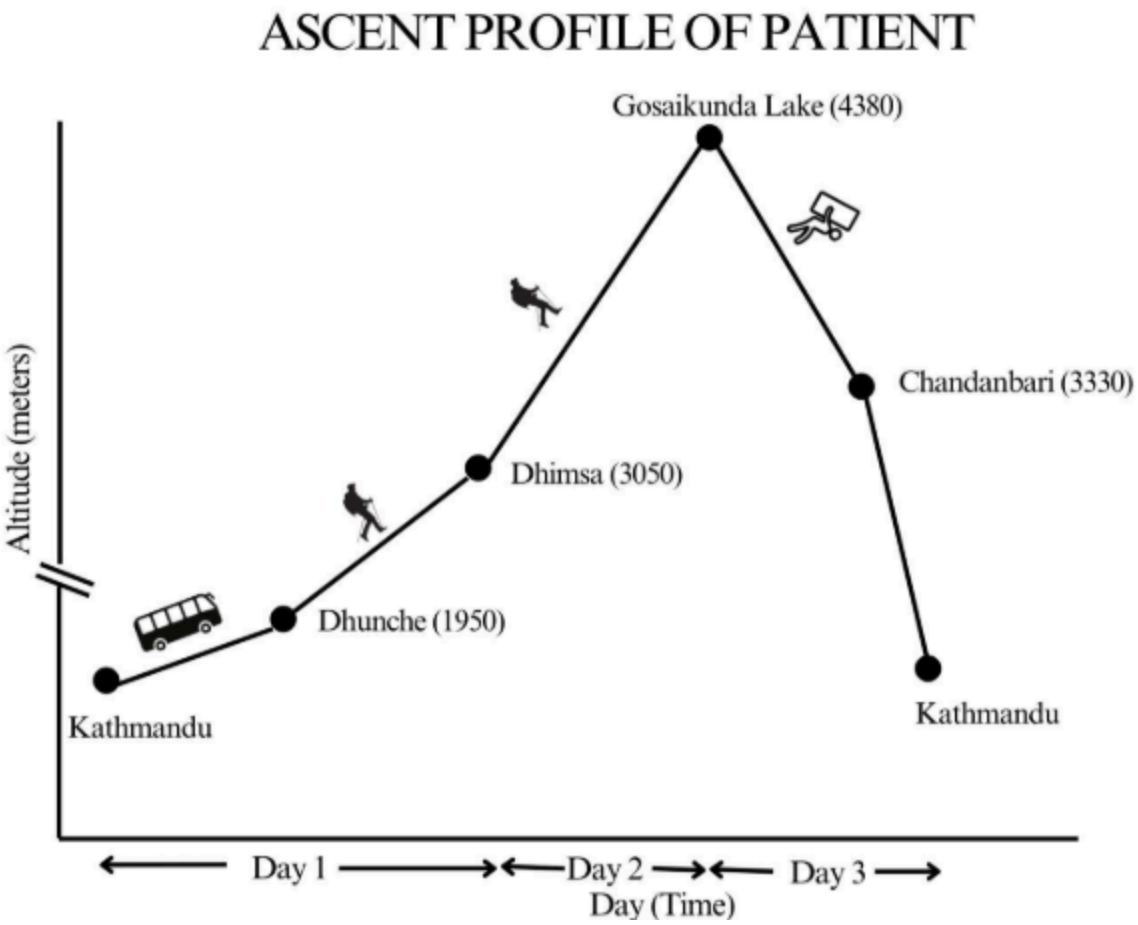


Figure 1

Ascent profile of patient from Kathmandu to Gosaikunda Lake