▪ Headache Medicine 2025, 16(4): 263-269 ▪ p-ISSN 2178-7468, e-ISSN 2763-6178

Headache Medicine

▪ DOI: 10.48208/HeadacheMed.2025.40

Review

High-altitude headache: a narrative review

Andrea Veronica Marengo[1,2], Malena Tejada[3], María Isabel Cusicanqui Giles[4,5], Katherine Natali Ramirez Andia[6]

[1]Department of Neurology, Hospital Perrupato, San Martín, Mendoza, Argentina.
[2]Chair of Neurology, Faculty of Medicine, National University of Cuyo, Mendoza, Argentina
[3]Critical Care Unit, Hospital Santa Caterina, Girona, Spain
[4]Department of Neurology, Hospital de Clínicas, La Paz, Bolivia
[5]Faculty Member, Universidad Mayor de San Andrés, La Paz, Bolivia
[6]Neurologist, University Hospital of Clínicas, La Paz, Bolivia

Andrea Veronica Marengo
dra.andreamarengo@gmail.com

Edited by:
Marcelo Moraes Valença

Purpose of review
This narrative review aims to summarize current evidence on the epidemiology, pathophysiological mechanisms, risk factors, differential diagnosis, and preventive and therapeutic strategies of High-altitude headache (HAH) to raise awareness among physicians.

Recent findings
HAH is the most common neurological symptom associated with acute mountain sickness (AMS) and has become a global health concern due to the increasing exposure to high altitudes through tourism, sports and work. Its pathophysiology is complex and multifactorial, involving hypobaric hypoxia, blood–brain barrier dysfunction, and trigeminovascular system activation.

Conclusions
The diagnostic criteria of the ICHD-3 and the Lake Louise Score are highlighted as essential clinical tools, especially regarding moment of evacuation. Knowledge gaps were identified in areas such as biomarkers, updated epidemiological data, diagnostic standardization and vulnerable populations. Improving understanding and management of HAH is critical as global exposure to high-altitude environments continues to rise.

Keywords:
High-altitude headache
Hypobaric hypoxia
Acute mountain sickness

Received: October 4, 2025
Revised: November 17, 2025
Accepted: December 22, 2025

Introduction

High-altitude headache (HAH) is the most frequent neurological manifestation of hypobaric hypoxia and the cardinal symptom of acute mountain sickness (AMS) (1). Its clinical relevance has increased steadily over recent decades due to the growing exposure of humans to high-altitude environments through tourism, occupational activities—particularly mining—and sports. In these settings, many individuals ascend rapidly without adequate acclimatization, significantly increasing the risk of altitude-related illness.

According to the *International Classification of Headache Disorders*, 3rd edition (ICHD-3), HAH is classified as a secondary headache attributed to disorders of homeostasis related to reduced oxygen availability. It typically develops within 6–24 hours after ascent above 2,500 m and usually resolves with descent (2). Epidemiological studies consistently show that the prevalence of high-altitude headache increases progressively with altitude and inadequate acclimatization (1).

The pathophysiology of HAH is complex and likely multifactorial. Hypobaric hypoxia is hypothesized to trigger cerebral vasodilation, disruption of the blood–brain barrier, and mild cerebral edema, as well as activation of the trigeminovascular system through mechanisms similar to those observed in migraine. Individual susceptibility factors—including a history of migraine, previous AMS, rapid ascent, obesity, and cardiopulmonary comorbidities—further increase the risk of developing HAH (3).

Despite its high prevalence and its potential progression to severe complications such as high-altitude cerebral edema, HAH remains underrecognized in clinical practice (4,5). Improving knowledge surrounding this condition is essential to optimize early recognition and management, particularly in the context of increasing global exposure to high-altitude environments.

The aim of this narrative review is to analyze the epidemiology, pathophysiology, risk factors, clinical features, differential diagnosis, and preventive and therapeutic strategies of high-altitude headache, integrating current evidence to support its early recognition and management in clinical practice.

Methods

A narrative review was conducted in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) guidelines. A comprehensive literature search was performed primarily in PubMed/MEDLINE and complemented by Google Scholar, including publications from January 2000 to December 2024. MeSH terms and free-text keywords related to high-altitude headache, acute mountain sickness, hypobaric hypoxia, prevention, and treatment were used.

The search yielded approximately 420 records. Titles and abstracts were screened to identify articles relevant to the scope of this review, and selected publications were evaluated in full text. A total of 39 studies considered most relevant to the objectives of the review were included in the narrative synthesis.

Eligible publications comprised original research articles, observational studies, clinical trials, systematic reviews, meta-analyses, and consensus or guideline documents. Non-English publications, case reports, editorials, letters, and conference abstracts were excluded. Study selection and qualitative appraisal were performed by two reviewers, focusing on study design, consistency of diagnostic criteria, methodological quality, and clinical relevance. The findings were synthesized using a descriptive narrative approach with emphasis on clinically meaningful aspects of HAH.

Pathophysiology

Hypobaric hypoxia at high altitude

Although the fraction of oxygen in the air remains constant at higher altitudes (around 21%), the concomitant reduction of barometric pressure leads to a decline in partial pressure of inspired oxygen (pPO_2), thus into reduced alveolar pPO_2, and, consequently, a diminished arterial oxygen tension. This is called hypobaric hypoxia (6,7).

Neurovascular Responses and Blood–Brain Barrier Disruption

Although the precise pathophysiological basis of high-altitude headache is only partially known, hypobaric hypoxia is believed to trigger complex neurohumoral and hemodynamic responses, including microvascular vasodilation, with increased hydrostatic capillary pressure and liberation of chemical mediators such as nitric oxide, bradichinine and vascular endothelial growth factor (VEGF), with consequential dysfunction of Brain blood barrier (8). The enhanced permeability along with vasodilation and augmented production of cerebrospinal fluid can lead to increased intracranial pressure and cerebral edema. The severity of headache symptoms correlates with the degree of arterial dilation and flow in cerebral arteries, particularly the internal carotid and vertebral arteries (9).

Trigeminovascular System Activation and Headache

The vascular changes activate the trigeminovascular

system, similar to what occurs on migraine, and plays a critical role in headache pathogenesis at altitude (10). The release of calcitonin gene-related peptide (CGRP) induces further vasodilation and vascular protein leakage, exacerbating nociceptive signaling. Additionally, compromised cerebral venous outflow, whether due to anatomical variants or hypoxia-induced venous distension, may intensify intracranial hypertension and headache severity (9). Individuals with reduced cerebrospinal fluid volume relative to brain parenchymal volume exhibit diminished compensatory capacity to cerebral edema, predisposing them to more pronounced neurological symptoms (8).

Acclimatization: the body response to high altitude

Hypobaric hypoxia elicits a complex physiological response primarily mediated by activation of the sympathetic nervous system. This process is initiated via chemosensory input from the carotid bodies and subsequent integration within the brainstem respiratory centers, culminating in an increase in respiratory rate, tidal volume, and heart rate(9). These responses enhance cardiac output to optimize systemic oxygen delivery. The hyperventilatory response induces an acute respiratory alkalosis, which, after 24–48 hours, is partially compensated by renal bicarbonate excretion. This adaptive process is frequently accompanied by reduction in plasmatic levels of calcium and phosphate (11).

Following acute exposure to high altitude, EPAS1 gene expression is rapidly modulated via hypoxia-inducible factor 1 (HIF-1), a heterodimer critical for cellular adaptation to hypoxic stress through the regulation of homeostatic pathways. Activation of the HIF-1 alpha isoform promotes erythropoietin synthesis, enhancing erythropoiesis and serving as a central mechanism in physiological acclimatization. In addition, HIF-1 contributes to ventilatory regulation by influencing respiratory neuronal circuits within the brainstem, which augments the ventilatory response and improves tissue oxygenation during hypoxia (12,13). The sum of these responses, occurring over several days to weeks, constitute acclimatization (14).

Risk factors

Risk factors for HAH include individual susceptibility, permanent residence below 900 m, obesity, pre-existing cardiopulmonary disease: persistence of a patent foramen ovale, congenital pulmonary abnormalities, Holmes-Adie syndrome, Down syndrome, prior history of AMS, personal history of migraine, strenuous physical activity, low oxygen saturation, and insufficient fluid intake (15–19).

Clinical Features

High-altitude headache typically develops within 6–24 hours after arrival at altitude and is commonly described as a dull, throbbing, bilateral headache, frequently involving the occipital or frontal regions (1,12,20).

High-altitude headache frequently occurs in association with AMS (1). Acute mountain sickness is characterized by the presence of headache accompanied by gastrointestinal symptoms (nausea, vomiting, anorexia), dizziness, fatigue or weakness, and sleep disturbances, particularly insomnia (4,6). These symptoms usually emerge within 24 hours after ascent to altitudes above 2,500 m and may vary in severity depending on the rate of ascent and the degree of acclimatization (21–23).

Diagnosis

High-altitude headache is one of the cardinal symptoms of AMS and is recognized by the ICHD-3 as a specific diagnostic entity related to exposure to high altitudes (2). Diagnosis is based on both the clinical characteristics of the headache and its temporal association with ascent above 2,500 meters. Table 1 summarizes the diagnostic criteria proposed by the ICHD-3 for high-altitude headache.

Table 1. Diagnostic criteria for high-altitude headache (HAH) according to the International Classification of Headache Disorders, 3rd edition beta (ICHD-3β)

Diagnostic criteria for High Altitude Headache according to ICHD-3β
A- Headache fulfilling criterion C
B- Ascent to an altitude above 2,500 m
C.Causation is demonstrated by at least two of the following:
1.Headache develops in temporal relation to continued ascent.
2. One or both of the following:
a) Headache has significantly worsened in parallel with further ascent.
b) Headache resolves within 24 hours after descending to an altitude below 2,500 m.
3. Headache has at least two of the following three characteristics:
a) Bilateral location.
b) Mild to moderate intensity.
c) Aggravated by exertion, movement, straining, coughing, and/or bending forward.
D. Not better accounted for by another ICHD-3β diagnosis.

High-altitude headache (HAH) is diagnosed when these criteria are fulfilled after ascent above 2,500 m and no alternative ICHD-3β diagnosis better explains the symptoms (Olesen et al.(2); ICHD-3β).

In addition, the Lake Louise Score, revised in 2018, is widely used to assess both the presence and severity of AMS (24). It includes headache as a mandatory symptom, along with fatigue, dizziness, and gastrointestinal disturbances. The total score allows for classification of AMS severity, thereby guiding clinical decision-making and therapeutic management (23). Table 2 shows the Lake Louise Scoring System for AMS diagnosis.

Table 2: Lake Louise Scoring System for AMS diagnosis

Lake Louise Scoring System (2018)
Headache
0 — None
1 — Mild headache
2 — Moderate headache
3 — Severe, incapacitating headache
Gastrointestinal Symptoms
0 — Good apetite
1 — Loss of appetite or nausea
2 — Moderate nausea or vomiting
3 — Severe, incapacitating nausea and vomiting
Fatigue and/or Weakness
0 — No fatigue or weakness
1 — Mild fatigue/weakness
2 — Moderate fatigue/weakness
3 — Severe, incapacitating fatigue/weakness
Dizziness/Lightheadedness
0 — No dizziness or lightheadedness
1 — Mild dizziness/lightheadedness
2 — Moderate dizziness/lightheadedness
3 — Severe, incapacitating dizziness/lightheadedness
Functional Clinical Assessment of AMS
Overall, if you experienced AMS symptoms, how did they affect your activities?
0 — Not at all
1 — Symptoms present, but did not require changing activity or itinerary
2 — Symptoms forced me to stop ascending or to descend on my own
3 — I had to be evacuated to a lower altitude

A diagnosis of AMS is established when the total Lake Louise Score is ≥3, provided that headache is present as a mandatory symptom. This scale is widely used by mountaineers and can be self-administered (Roach et al.(24)).

Differential diagnosis of high-altitude headache

The differentiation between HAH and other primary or secondary headache disorders is essential, as overlapping clinical features may lead to misdiagnosis.

Migraine represents the most important differential diagnosis. Broessner et al. suggested that low barometric pressure alone does not trigger migraine (18); however, a recognized association exists between low barometric pressure combined with high altitude and migraine onset (25–27). Unlike high-altitude headache, migraine typically occurs in individuals with a previous history of attacks, may be preceded by aura, and is often accompanied by photophobia, phonophobia, nausea, and disabling pulsatile pain (27).

Tension-type headache is characterized by bilateral, pressing pain (28). In contrast to high-altitude headache, it is not exacerbated by physical exertion and is not associated with symptoms of acute mountain sickness, as defined by the ICHD-3 (2).

Primary exertional headache usually presents as bilateral, pulsatile pain triggered by intense physical activity; however, it is not directly related to hypobaric hypoxia or exposure to high altitude (29,30).

Finally, other secondary causes of headache, such as sinusitis, dehydration, carbon monoxide exposure, or intracranial pathology, should be carefully excluded, particularly when headache characteristics are atypical or disproportionately severe, in accordance with the diagnostic framework of the ICHD-3 (2).

In summary, although HAH shares features with migraine, tension-type headache, and primary exertional headache, its defining diagnostic characteristic is the close temporal relationship with ascent above 2,500 m and resolution within 24 hours after descent. Careful clinical evaluation, assessment of migraine history, and identification of associated systemic symptoms are critical for accurate diagnosis.

Prevention and treatment of altitude headache

The prevention of altitude headache is crucial to avoid systemic complications (22,31).

1. Preventive measures during ascent (32,33).

a. Non-pharmacological:

- Gradual ascent: Do not exceed 500 m/day above 3,500 m; avoid long ascents with large elevation gains.
- Sleep at lower altitude when possible ("climb high, sleep low").
- Rest days after every 1,000 m of ascent.

- Adequate hydration: 3–4 L/day, avoiding dehydration.
- Carbohydrate-rich nutrition (70–75% of daily caloric intake).
- Avoid intense physical exertion during the first days.
- Adequate rest and sleep to improve tolerance.
- Avoid alcohol and tobacco.
- Avoid barbiturates and hypnotics. However, in cases of high-altitude insomnia, zolpidem or other short-acting sleep inducers may be considered, given their relative safety and effectiveness in this setting (32).

Drug prophylaxis

Acetazolamide is the first-line pharmacological option for the prevention of high-altitude headache. The recommended dose is 125 mg every 12 hours, initiated 24 hours before ascent and continued during altitude exposure. Its efficacy in reducing the incidence of HAH and AMS has been consistently demonstrated (31–34).

Nonsteroidal anti-inflammatory drugs have also been evaluated for prophylactic use, particularly during rapid ascents or when adequate acclimatization is not feasible (22).

- Ibuprofen: 600 mg every 8–12 hours.
- Naproxen: 500 mg every 12 hours. Prophylactic administration of these agents has shown effectiveness in reducing the occurrence of high-altitude headache.

Other medications have been studied with more limited evidence. Sumatriptan was evaluated in a clinical trial in which a single oral dose of 50 mg administered within the first hour after ascent was associated with a reduced incidence of headache and acute mountain sickness.

Dexamethasone is most commonly used for the prevention or treatment of severe altitude-related illnesses, such as high-altitude cerebral edema (22,35). It may be considered when acetazolamide is contraindicated or unavailable; however, its use should be restricted to specific circumstances, such as high-altitude workers or rescue personnel who must ascend rapidly and cannot undergo gradual acclimatization (6,9). Table 3 summarizes recommended drug dosages and their preventive effects.

Table 3. Offers a summarised version of drug dosage and its effect.

Drug	Dosage	Indication	Efficacy	Side effects
Acetazolamide	125 mg every 12 hours	Start 24 hours before and continue during ascent	Reduces the risk of altitude headache and acute mountain sickness	Fluid and electrolyte imbalance, polyuria, dysgeusia, paresthesia, allergy
Ibuprofen	600 mg TID or as tolerated.	Start before ascent and continue the first few days of exposure to altitude.	There are many studies (e.g. HEAT) that recommend its efficacy in preventing altitude headaches and mild to moderate mountain sickness.	Renal toxicity, gastrointestinl toxicity, allergies
ASA	320 mg before ascent, then 3 doses every 4 hours	Start one hour before arrival at altitude, continue in the first 24 hours according to the schedule	Decreases the occurrence of headache compared to placebo in the HEAT study	Contraindicated in coagulopathy, risk of hemorrhage and gastrointestinal toxicity

Dosages and indications are based on available evidence from randomized controlled trials and consensus guidelines (Davis et al.(32); Angelini et al.(31), Toussaint et al.(34), Zelmanovich et al.(35).

Acute mountain sickness assessment and management

As discussed above, the diagnosis of AMS using validated tools such as the Lake Louise Scoring System allows early identification of affected individuals and provides guidance for clinical decision-making. A score of ≥3, particularly when associated with headache, fatigue, gastrointestinal symptoms, and dizziness, strongly suggests AMS.

Alarm signs requiring urgent evacuation or immediate descent include resting oxygen saturation below 85%, progressive dyspnea, ataxia, or altered level of consciousness, which may indicate progression to high-altitude cerebral edema or high-altitude pulmonary edema. Immediate descent of 500–1,000 m remains the cornerstone of management (22).

Supplemental oxygen or portable hyperbaric chambers may support descent but are not always available in high-altitude settings. Pharmacological measures, such as acetazolamide or dexamethasone, are more widely accessible and are particularly useful when descent is delayed (12,35).

Conclusion

High-altitude headache is a highly prevalent yet underrecognized condition and represents the primary symptom of AM. Its pathophysiology is complex and multifactorial, involving hypobaric hypoxia, cerebral vasodilation, and activation of the trigeminovascular system, with individual susceptibility playing a central role. High-altitude headache may affect up to 80% of individuals ascending above 3,000 m, particularly during rapid ascent and in the absence of adequate acclimatization.

Although often self-limiting, HAH may progress to severe altitude-related complications, such as high-altitude cerebral edema, if not promptly recognized and appropriately managed. Early identification, gradual ascent, and the implementation of effective preventive strategies are therefore essential.

The use of validated diagnostic tools, including the ICHD-3 and the Lake Louise Scoring System, is critical for accurate diagnosis, clinical monitoring, and risk stratification.

Further research on HAH is needed, particularly to clarify its underlying pathophysiological mechanisms, identify reliable biomarkers, standardize diagnostic criteria, and update epidemiological data. Additional studies are also required to evaluate genetic susceptibility, comorbid conditions, treatment efficacy, and the impact of high-altitude exposure on vulnerable populations, such as children, older adults, and individuals exposed to strenuous physical exercise.

Greater awareness and improved preventive approaches are necessary to reduce the global burden of HAH, especially as recreational, occupational, and tourism-related exposure to high-altitude environments continues to increase worldwide.

References

1. Carod-Artal FJ. High-altitude headache and acute mountain sickness. Neurología 2014;29:533–40. Doi:10.1016/j.nrl.2012.04.015.
2. Olesen J, et al. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018;38:1–211. Doi:10.1177/0333102417738202.
3. Burtscher J, Hüfner K, Kopp M, Schipplick F, Schobersberger W, Gatterer H. High altitude adaptation, common high-altitude disorders and the effects of high altitude on mental health. Sports Psychiatry 2024;3:197–208. Doi:10.1024/2674-0052/a000095.
4. Dietz TE, Hackett PH. High-Altitude Medicine. Travel Medicine, Elsevier; 2019, p. 387–400. Doi:10.1016/B978-0-323-54696-6.00042-2.
5. Łagowski W, Grodzka O, Domitrz I. Atypical neurological symptoms at high altitude: a systematic literature review. Travel Med Infect Dis 2025;66:102867. Doi:10.1016/j.tmaid.2025.102867.
6. Richalet J, Herry J. Médecine de montagne: Alpinisme et sports de montagne. France: Elsevier Masson; 2017.
7. Marengo A, Tejada M, Zirena IH, Molina S. Neurological Manifestations Associated with Exercise at Altitude. Curr Neurol Neurosci Rep 2025;25:29. Doi:10.1007/s11910-025-01418-6.
8. Marmura MJ, Hernandez PB. High-Altitude Headache. Curr Pain Headache Rep 2015;19:9. Doi:10.1007/s11916-015-0483-2.
9. Luks AM, Swenson ER, Bärtsch P. Acute high-altitude sickness. European Respiratory Review 2017;26:160096. Doi:10.1183/16000617.0096-2016.
10. Maini K, Schuster NM. Headache and Barometric Pressure: a Narrative Review. Curr Pain Headache Rep 2019;23:87. Doi:10.1007/s11916-019-0826-5.
11. West J, Schoene R, Luks A, Milledge J. High Altitude Medicine and Physiology 5E. 5th ed. London: CRC Press; 2012. Doi:10.1201/b13633.
12. Angelini C. Neurologic and Metabolic Challenges at High Altitudes. SVOA Neurology 2024;5:78–86. Doi:10.58624/SVOANE.2024.05.0132.
13. Aboouf MA, Thiersch M, Soliz J, Gassmann M, Schneider Gasser EM. The Brain at High Altitude: From Molecular Signaling to Cognitive Performance. Int J Mol Sci 2023;24:10179. Doi:10.3390/ijms241210179.
14. Berger MM, Luks AM. High Altitude. Semin Respir Crit Care Med 2023;44:681–95. Doi:10.1055/s-0043-1770063.
15. Burtscher M, Mairer K, Wille M, Broessner G. Risk factors for high-altitude headache in mountaineers. Cephalalgia 2011;31:706–11. Doi:10.1177/0333102410394678.
16. Richalet J-P, Larmignat P, Poitrine E, Letournel M, Canouï-Poitrine F. Physiological Risk Factors for Severe High-Altitude Illness. Am J Respir Crit Care Med 2012;185:192–8. Doi:10.1164/rccm.201108-1396OC.
17. Bian S-Z, Zhang J-H, Gao X-B, Li M, Yu J, Liu X, et

al. Risk factors for high-altitude headache upon acute high-altitude exposure at 3700 m in young Chinese men: a cohort study. J Headache Pain 2013;14:35. Doi:10.1186/1129-2377-14-35.
18. Broessner G, Rohregger J, Wille M, Lackner P, Ndayisaba J-P, Burtscher M. Hypoxia triggers high-altitude headache with migraine features: A prospective trial. Cephalalgia 2016;36:765–71. Doi:10.1177/0333102415610876.
19. Queiroz LP, Rapoport AM. High-altitude headache. Curr Pain Headache Rep 2007;11:293–6. Doi:10.1007/s11916-007-0206-4.
20. Schneider M, Bärtsch P. Characteristics of Headache and Relationship to Acute Mountain Sickness at 4559 Meters. High Alt Med Biol 2018;19:321–8. Doi:10.1089/ham.2018.0025.
21. Bärtsch P, Swenson ER. Acute High-Altitude Illnesses. New England Journal of Medicine 2013;368:2294–302. Doi:10.1056/NEJMcp1214870.
22. Hofmeyr R, Tölken G, De Decker R. Acute high-altitude illness. South African Medical Journal 2017;107:556. Doi:10.7196/SAMJ.2017.v107i7.12612.
23. Paul S, Gangwar A, Bhargava K, Khurana P, Ahmad Y. Diagnosis and prophylaxis for high-altitude acclimatization: Adherence to molecular rationale to evade high-altitude illnesses. Life Sci 2018;203:171–6. Doi:10.1016/j.lfs.2018.04.040.
24. Roach RC, Hackett PH, Oelz O, Bärtsch P, Luks AM, MacInnis MJ, et al. The 2018 Lake Louise Acute Mountain Sickness Score. High Alt Med Biol 2018;19:4–6. Doi:10.1089/ham.2017.0164.
25. Davis C, Reno E, Maa E, Roach R. History of Migraine Predicts Headache at High Altitude. High Alt Med Biol 2016;17:300–4. Doi:10.1089/ham.2016.0043.
26. Okuma H, Okuma Y, Kitagawa Y. Examination of fluctuations in atmospheric pressure related to migraine. Springerplus 2015;4:790. Doi:10.1186/s40064-015-1592-4.
27. Karle FJ, Auerbach PS. Migraine Headache Confounding the Diagnosis of Acute Mountain Sickness. Wilderness Environ Med 2014;25:60–8. Doi:10.1016/j.wem.2013.10.006.
28. Ashina S, Mitsikostas DD, Lee MJ, Yamani N, Wang S-J, Messina R, et al. Tension-type headache. Nat Rev Dis Primers 2021;7:24. Doi:10.1038/s41572-021-00257-2.
29. Poussel M, Laroppe J, Hurdiel R, Girard J, Poletti L, Thil C, et al. Sleep Management Strategy and Performance in an Extreme Mountain Ultra-marathon. Research in Sports Medicine 2015;23:330–6. Doi:10.1080/15438627.2015.1040916.
30. Sandoe CH, Kingston W. Exercise Headache: a Review. Curr Neurol Neurosci Rep 2018;18:28. Doi:10.1007/s11910-018-0840-8.
31. Angelini C, Giardini G, Falla M. Travel to altitude with neurological disorders — recommendation of the UIAA Medical Commission. Health Promotion & Physical Activity 2021;15:29–39. Doi:10.5604/01.3001.0015.0506.
32. Davis C, Hackett P. Advances in the Prevention and Treatment of High Altitude Illness. Emergency Medicine Clinics 2017;35. Doi:10.1016/j.emc.2017.01.002.
33. Falla M, Giardini G, Angelini C. Recommendations for traveling to altitude with neurological disorders. J Cent Nerv Syst Dis 2021;13. Doi:10.1177/11795735211053448.
34. Toussaint CM, Kenefick RW, Petrassi FA, Muza SR, Charkoudian N. Altitude, Acute Mountain Sickness, and Acetazolamide: Recommendations for Rapid Ascent. High Alt Med Biol 2021;22:5–13. Doi:10.1089/ham.2019.0123.
35. Zelmanovich R, Pierre K, Felisma P, Cole D, Goldman M, Lucke-Wold B. High Altitude Cerebral Edema: Improving Treatment Options. Biologics 2022;2:81–91. Doi:10.3390/biologics2010007.

Andrea Veronica Marengo
https://orcid.org/0000-0002-6080-5738
Malena Tejada
https://orcid.org/0009-0006-4718-6190
María Isabel Cusicanqui Giles
https://orcid.org/0009-0008-0836-8463
Katherine Natali Ramirez Andia

Ethics statement: This article does not involve studies with human participants or animals conducted by any of the authors.
Authors' contributions: All authors contributed to the literature review. AM, MT: drafted the initial version of the manuscript. MT: contributed to manuscript revision and translation. RAK: wrote the treatment section. CGMI: wrote the pathophysiology section. AM: designed the tables. All authors reviewed and approved the final manuscript.
Conflicts of interest: The authors declare no conflicts of interest, including financial or non-financial interests.
Funding: The authors received no financial support for the research, authorship, and/or publication of this article.