SciMedCentral

Clinical Research in Pulmonology

Short Communication

Neurological Disorders Occurring at High Altitude on Mount Kilimanjaro, Tanzania Results From a Two Year Referral Centre Survey

Dekker Marieke Cornelia Johanna*, and Howlett William Patrick

Department of Medicine, Kilimanjaro Christian Medical Centre, Tanzania

***Corresponding author**
Marieke C.J. Dekker, consultant neurologist, Department of Medicine, Kilimanjaro Christian Medical Centre, P.O. Box 3010, Moshi Kilimanjaro, United Republic of Tanzania. Tel: 255 784 669 314

Submitted: 07 July 2023

Accepted: 22 September 2023

Published: 25 September 2023

ISSN: 2333-6625

Abstract

High-altitude Illnesses (HAI) in the climbing population on Mount Kilimanjaro form a small but steady patient population in our tertiary referral centre. The aim of this study was to document neurological disorders in climbers independent of HAI.

Methods: A two year retrospective sampling study was conducted of patients presenting at Kilimanjaro Christian Medical Centre (KCMC) with features suggestive of HAI. Clinical, demographic and altitude-related data were collected through patient record review. Five patients who did not qualify for HAI inclusion who showed neurological signs and symptoms are discussed in more detail.

Results: We identified 62 climbers with a diagnosis of HAI related disorders. A total of fifty-six (90%) had classical features of HAI, including acute mountain sickness (AMS) (n=8; 14%), high altitude pulmonary edema (HAPE) (n=30; 54%), HACE (n=7; 12%) and combined HAPE/HACE (n=11; 20%). A total of 6/62 (10%) were found to have non-HAI related disorders. These included five with neurological disorders, three of whom presented with new onset seizures, one with subarachnoid haemorrhage and one with fatal head injury as a result of a fall. One patient with a history of bronchiectasis presented with clinical features suggestive of HAPE but was found to have an exacerbation of bronchiectasis complicated by respiratory tract infection.

Conclusion: In a cohort of hospitalised climbers on Mount Kilimanjaro presenting with suspected HAI, a small series of non-HAI related, mostly neurological disorders were identified. Their onset was also likely to be related to the effects of high altitude.

INTRODUCTION

Mountains have always held a deep fascination for mankind but carry health risks and hazards. The main risks are those associated with climbing or ascending mountains to high altitude and these are collectively termed high altitude illness (HAI). Other hazards include trauma and medical or surgical coincidental illnesses arising during the ascent. With increasing global travel, mountain climbing or trekking has now become popular and feasible for people with no previous 50 mountaineering experience and thereby places a large number of people at risk of HAI and related disorders.

Mount Kilimanjaro (5895m) is the highest free-standing mountain in the world and it very aptly illustrates the health hazards of high altitude climbing or trekking because of the ease of ascent. Technically the climb is relatively simple, which may lead to uphill climbing rates exceeding 1,000 55 meters a day[1-2]. Annually between 45000 and 500003 tourists attempt to climb Mount Kilimanjaro, and ascent can be done by untrained individuals over a period of 5-8 days with varying success rates of reaching the top ranging from 25 to 85% with the higher rates of success the longer the duration of the climb2.

The term high altitude illness (HAI) includes acute mountain sickness (AMS, prevalence 40-90% 60 depending on setting4), high altitude cerebral edema (HACE, 0.5-1%4) and high altitude pulmonary edema (HAPE, ref range 0.2-7%4). While AMS is a common and relatively benign disorder, HACE and HAPE though relatively uncommon are serious and can lead to death. These altitude related disorders affect trekkers and mountain climbers typically at altitudes typically starting at 2,500 meters or greater4 occurring with increasing frequency and severity in association with increasing 65 altitude. The reduction in inspired oxygen partial pressure that occurs with increasing altitude is responsible for the reduced oxygen level in blood and the consequent HAI [5]. The main risk factor for developing acute HAI is the rate of ascent and sleeping elevation in meters per day [2].

When ill climbers are admitted to hospital in a critical condition this is typically with the severe end of the spectrum of HAPE (respiratory difficulties) and HACE (altered level of consciousness), 70 or uncommonly seizures. Due to the increased local awareness of the likelihood of HAI such patients may already have a working diagnosis of HACE, HAPE or a combination of both. Very occasionally they turn out to have a non-HAI related

disorder. Such neurological disorders may have either occurred on their own or may have been triggered by the physiological and climatological variables occurring at high altitude. Neurological disorders are the most common disorder and the rationale of this short patient series is to address the non-HAI related neurological disorders in a cohort of Kilimanjaro climbers [5], who attended or were admitted to the local referral hospital.

MATERIALS AND METHODS

We reviewed the medical records of climbers presenting to KCMC for a two year period (from January 2016 to December 2017). An overview of HAI related morbidity and mortality during this time period has been recently published [6]. Ethical clearance for this study was received from the Kilimanjaro Christian Medical University College. We obtained hospital files of those attending the emergency department (ED) or admitted to the medical unit and medical intensive care unit of KCMC with a diagnosis of high altitude related disorders. We recorded demographic, clinical, and altitude related data, and reviewed laboratory results, X-rays, and head computed tomography (CT) images. An magnetic resonance imaging (MRI) scanner was unavailable during the period of our study. For the diagnosis of AMS [7,8]. we used the recent Lake Louise 2018 criteria including headache with a score of 3 or greater being diagnostic [8]. HAPE and HACE were defined using the 1992 Lake Louise criteria [7]. Progression from AMS to HACE is there when there is confusion or altered level of consciousness or ataxia. Without preceding AMS both symptoms are required to diagnose HACE. HAPE is constituted by any combination of at least two signs (crackling or wheezing on auscultation, central cyanosis and increased respiratory or heart rate) and at least two symptoms (shortness of breath at rest, cough, impaired exercise tolerance and chest tightness) [7]. The following diagnostic criteria were used to define the stages of HAI. AMS: The 2018 Lake Louise Acute Mountain Sickness Score8: An AMS total score of 3 or greater from the following 4 related symptoms was diagnostic of AMS. Headache (0-3), Gastrointestinal (0-3), Fatigue/weakness (O-3), Dizziness/light headedness (0-3) [8]. HACE: The presence of a change in mental status and/or ataxia in a person with AMS, or the presence of both mental status change and ataxia in a person without AMS.7 HAPE: The presence of the following: Symptoms: at least two of: dyspnoea at rest, cough, weakness or decreased exercise performance, chest tightness or congestion. Signs: at least two of: rales or wheezing in at least one lung field, central cyanosis, tachypnoea, tachycardia [7]. The neurological disorders were diagnosed according to following criteria. Seizure was defined as a a paroxysmal event characterized by stereotypical signs and symptoms resulting from abnormal electrical activity. The two authors independently reviewed the clinical diagnosis and additional investigations in each case.

RESULTS

In total 62 patients with HAI were analysed, 47 inpatients and 15 outpatients of whom 56 presented with HAI, which included AMS (n=8; 14%), HAPE (n=30; 54%), HACE (n=7; 12%), and combined features of HAPE as well as HACE (HAPE/HACE) (n=11; 20%). A comprehensive overview of the series of HAI patients was published elsewhere.6 One patient with known bipolar disease who presented with psychotic behaviour had cerebral oedema on neuroimaging suggesting HACE and was included in the HAI series.

Six patients (11%) had a disorder which did not correspond to HAI. One of the six had a HAPE mimic which was a clinical exacerbation of pre-existent bronchiectasis and recurring bronchitis without evidence of HAPE either on auscultation or on chest X-ray. The five remaining patients with non-HAI disorders all had neurological presentations [Table 1].

Three patients had first ever, new onset generalised tonic clonic seizures which occurred while climbing. The likely causes were hyponatremia in two patients and alcohol intake in one patient. One patient with hyponatremia had the recommended fluid intake for altitude climbing but had persistent hyponatremia during hospital admission. A chest X ray to rule out HAPE and lung malignancy was normal. The other patient with hyponatremia had intentional polydipsia, ingesting up to 6 litres of fluid per day upon the instruction to drink plenty of fluids. Fluid restriction caused a rapid normalisation of sodium levels. The patient also presented with a severe backache upon arriving in hospital and was discovered to have an L1 vertebral compression fracture incurred as a result of the seizure and presumed fall. All patients with a history of seizure had typical postictal symptoms which resolved fully within 24 hours.

A history of thunderclap headache was present in one patient who was found on CT-angiography examination of the head to have reversible cerebral vasoconstriction syndrome (RCVS) with minimal subarachnoid haemorrhage. There were no abnormalities on neurological examination and the patient made a full recovery. Repeat CT-angiography on day [3], of symptoms showed normalization of vessel calibre and reduction of blood in the subarachnoid space. The sixth patient had traumatic brain injury as a result of an accidental fall shortly after summiting, not preceded by HAI symptoms. He died shortly after hospital admission.

DISCUSSION

HAPE and HACE are the most serious and life threatening medical disorders encountered in high altitude climbers, affecting >1% of climbers, depending on altitude.4,9 Central nervous system involvement is the main symptom complex involved in the various mechanisms of hypoxic organ dysfunction. HACE encephalopathy and HAPE-induced hypoxic encephalopathy are therefore correctly the first diagnosis considered in climbers presenting with neurological disorders [10]. However not all neurological disorders that present in high altitude climbers are HAI related. Neurological disorders that occur at altitude, independently of HAI11 need to be correctly identified as their management differs from HAI which typically involves oxygen and descent. This category includes a wide range of neurological disorders, including strokes [12-15], cerebral venous

Table 1: Characteristics of non-HAI neurological conditions in tourist climbers from Mount Kilimanjaro

Age	Altitude of onset (m)	Clinical features	Typical HACE symptoms (headache, nausea vomiting, confusion, ataxia)	CT scan/laboratory results	Outcome
55	3600	First ever generalised seizure with polydipsia of 6l/day, new backache on arrival in hospital.	Mild headache	Normal CT brain, vertebral fracture L1 on thoracolumbar CT	Discharged after 72h. Underwent spinal stabilisation and decompression of L1
58	3500	First ever generalised seizure with persistent hyponatremia	-	Normal CT brain results, Na 113 (111-116) mmol/l	Clinical recovery after 24h. Medical evacuation to Kenya after 48h
23	5600	First ever generalised seizure with alcohol intake on mountain and history of alcohol use	-	Normal CT brain	Discharged within 24h, on own request
52	4700	Two episodes of thunderclap headache on 4700m and upon return to 1800m, no loss of consciousness	No HACE symptoms prior to sudden headache	Minimal subarachnoidal haemorrhage and calibre changes on CT angiography. of brain	Medical evacuation to Kenya after 24h. At 72 h good clinical recovery and normalisation vessel calibre on CT brain
18	5700	Severe traumatic brain injury after fall on rock with Glasgow Coma Score of 3.	No HACE symptoms prior to fall	CT brain: extensive haemorrhagic contusion and skull fracture right frontal	Clinical brain death on admission

thrombosis16 seizures [17-21], visual disturbances [22], and headaches [23]. Very rarely an underlying neurological disorder unrelated to altitude may present for the first time in climbers at altitude [24,25].

The distribution of HAI subtypes and risk factors reported on Mount Kilimanjaro is largely comparable with that reported in other high altitude climber populations at similar altitudes.6 The number of cases of HAI mimics in this study 6/62 (10%) was relatively small compared to the total number of cases of HAI 56/62 (90%). Four of the six cases of HAI mimics presented with new onset neurological disorders, three of which were generalised seizures, two were attributed to hyponatremia, one of which was caused by polydipsia. One case was attributed to alcohol consumption during climbing. Seizures can occur in people with well-controlled epilepsy and in people with no history of epilepsy. Possible mechanisms at altitude include hypoxia, respiratory alkalosis, hypocapnia and lack of sleep. Hyponatremia causing new onset seizures has been previously reported in climbers [26]. One climber presented with thunderclap headache with reversible cerebral vasoconstriction syndrome (RCVS) and minimal subarachnoid haemorrhage. RCVS has also been described at high altitude [27].

Contributory reasons for the low numbers of HAI mimics reported in this study include a high level of awareness among mountaineering guides and porters for the classic forms of HAI and the existence of only one regional referral hospital for the more serious cases of HAI in the catchment area of Mount Kilimanjaro.6 Climbers presenting with mild and transient neurological signs and symptoms of AMS typically improve on descent and are not referred to hospital. Our small sample therefore was not representative of some of the commonly seen neurological conditions associated with high altitude4 including high altitude headaches and high altitude retinopathy.

This paper reports a death resulting from an accidental fall occurring at high altitude. Trauma has previously been reported as a cause of death in climbers on Mount Kilimanjaro28 but is considered to be relatively uncommon as technically it is not a difficult mountain to ascend. One of the six patients had a HAPE mimic with an exacerbation of pre-existent bronchiectasis and recurring bronchitis without evidence of HAPE which resolved with treatment. Pneumonia has been reported as a cause of death in climbers on Mount Kilimanjaro.[28]

Although a degree of co-occurrence of AMS or HACE with these disorders cannot be excluded most patients had onset of symptoms at a lower altitude than would be expected in climbers presenting with HAPE and HACE. This was particularly the case with seizure patients with onsets at altitudes of 3500 and 3600 m [Table 1]. One case of seizure which occurred at 5600m was considered to be alcohol induced. In contrast in the same series, the altitude of onset of HACE was at 4300±570 m (3500-4700) and of HAPE/HACE at 5000±430 m (4600-5700).

The likelihood of neurological disorders independent of HAI patients increases with the growing numbers of tourist climbers.3,6 This may be particularly the case on Mount Kilimanjaro where in any one year 40000-50000 climbers attempt the climb, usually with over 70% success rate depending on the route taken and days spent.3,6 In 40 years of clinical practice at the main referral hospital at the foot of Mount Kilimanjaro we have encountered other neurological disorders in climbers including stroke, cerebral venous sinus thrombosis, a first manifestation of manic psychosis and recently a cerebral malaria with renal failure, possibly precipitated by high altitude hypoxia in an expatriate tourist climber coming from a malaria endemic area. The easy accessibility of Mount Kilimanjaro to unusual climber categories, frequently with disabilities adds to the variety of neurological conditions encountered among climbers. Medical queries arising in climbers from outside the hospital continue to bring neurological disorders to our attention. Typical examples are exacerbations in migraines, and pre-existing seizure disorders worsened by hypoxia, climatological factors and sleep deprivation. Advice and queries and about the use of medications whilst climbing is frequently sought, for example the

use of a baclofen pump in cerebral palsy and immunomodulatory drugs in multiple sclerosis. With the increasing age of climbers, recent additions to this are a worsening of Parkinson's disease due to interrupted medication; obstructive sleep apnoea due to interrupted use of continuous positive pressure airway machines and the continuing use of cholinesterase inhibitors for climbers with dementia, leading to dizziness and falls. Guidelines and recommendations for travellers to altitude with pre-existing neurological and psychiatric disorders have been recently published [29,30].

CONCLUSIONS

The paper reports a small number of medical HACE mimics including new onset seizures and a reversible vascular event, all of which had a favourable outcome. Hopefully this will lead to an increasing sense of awareness of the importance of coincident neurological disorders amongst clinicians working in similar high altitude regions of the world.

ACKNOWLEDGEMENTS

We acknowledge Dr A. Sadiq, head of the Department of Radiology at KCMC, for x-ray review. Our sincere gratitude to Mr. S. Mtuy (SENE) and Mr. D. Brice-Bennett (Marangu Hotel) for additional information about geographical landmarks on Mount Kilimanjaro.

REFERENCES

1. Luks AM, McIntosh SE, Grissom CK, Auerbach PS, Rodway GW, Schoene RB, et al. Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness: 2014 update. Wilderness Environ Med. 2014; 25: S4-S14.
2. Lawrence JS, Reid SA. Risk Determinants of Acute Mountain Sickness and Summit Success on a 6-Day Ascent of Mount Kilimanjaro (5895 m). Wilderness Environ Med. 2016; 27: 78-84.
3. 3.Croughs M, Nyakunga GB, Sakita FM, Kilonzo K, Mmbaga BT, Soentjens P, et al. Incidence and predictors of severe altitude illness symptoms in Mt. Kilimanjaro hikers: a prospective cohort study. J Travel Med. 2022; 29; 1-8.
4. Luks AM, Swenson ER, Bärtsch P. Acute high-altitude sickness. Eur Respir Rev. 2017; 26: 160096.
5. Burtscher M, Hefti U, Hefti JP.High-altitude illnesses: Old stories and new insights into the pathophysiology, treatment and prevention. Sports Med Health Sci. 2021; 3: 59-69.
6. Dekker MCJ, Mremi A, Sakita F, Mvungi M, Nyakunda G, Kilonzo K, et al. Altitude Related Disorders on Mount Kilimanjaro, Tanzania. Two Year Survey in a Local Referral Center. Wilderness Environm Med. 2021; 32: 36-40.
7. Hackett PH, Oelz O. The Lake Louise consensus on definition and quantification of altitude illness. 1992; 327-330.
8. Roach RC, Hackett PH, Oelz O, Bartsch P, Luks AM, MacInnis MJ, et al. The 2018 Lake Louise Acute Mountain Sickness Score. High Alt Med Biol. 2018; 19: 4-6.
9. Smedley T, Grocott MP.Acute high-altitude illness: a clinically orientated review. Br J Pain. 2013; 7: 85-94.
10. Wilson MH, Newman S, Imray CH. The cerebral effects of ascent to high altitudes. Lancet Neurol. 2009; 8: 175-191.
11. Basnyat B, Wu T, Gertsch JH. Neurological conditions at altitude that fall outside the usual definition of altitude sickness. High Alt Med Biol. 2004; 5: 171-179.
12. Basnyat B. Seizure and hemiparesis at high-altitude outside the setting of acute mountain sickness. Wilderness Environ Med. 1997; 8: 221-222.
13. Chandler HC, Mellor A. Sudden onset hemiplegia at high altitude. J R Army Med Corps. 2016; 162: 470-472.
14. Allado E, Chenuel B, Vauthier JC, Hily O, Richard S, Poussel M, et al. Transient Central Facial Palsy at High Altitude: A Case Report. High Alt Med Biol. 2021.
15. Sawicka M, Szymczak RK. Paradoxical embolism to the central nervous system in a young Polish woman on a trek in the Himalayas. J Travel Med. 2022; 29: taab110.
16. Khattar NK, Sumardi F, Zemmar A, Liang Q, Li H, Xing Y, et al. Cerebral Venous Thrombosis at High Altitude: A Retrospective Cohort of Twenty-one Consecutive Patients. Cureus. 2019; 11: e4940.
17. Basnyat B. Fatal grand mal seizure in a Dutch trekker. J Travel Med. 1998; 5: 221-222.
18. Daleau P, Morgado DC, Iriarte CA, Desbiens R. New epilepsy seizure at high altitude without signs of acute mountain sickness or high altitude cerebral edema. High Alt Med Biol. 2006; 7: 81-83.
19. Hennis PJ, Gilbert-Kawai E, Grocott MP. Isolated generalized tonic-clonic seizure at high altitude in a young male trekker with a positive family history of seizure. High Alt Med Biol. 2014; 15: 93-94.
20. Srinath R, Ahmad F, Asturkar V, Mathur A, Sondhi V, Nanda SK, et al. New-onset seizure at high altitude among healthy males. Seizure. 2022; 97: 82-87.
21. Burtscher M. Seizures during prolonged high-altitude exposure. Seizure. 2022; 99: 176-177.
22. Mashkovskiy E, Szawarski P, Ryzhkov P, Goslar T, Mrak I. (2016) Fulminant high altitude blindness. J Travel Med. 2016; 23: taw057.
23. Karle FJ, Auerbach PS. Migraine headache confounding the diagnosis of acute mountain sickness. Wilderness Environ Med. 2014; 25:60-68.
24. Speidel V, Purrucker JC, Klobučníková K. Manifestation of Intracranial Lesions at High Altitude: Case Report and Review of the Literature. High Alt Med Biol. 2021; 22: 87-89.
25. Szymczak RK, Sawicka M, Pyrzowski J. Climber exhibits first clinical manifestation of spinocerebellar ataxia on Karakoram expedition. J Travel Med. 2022; 29: taac020.
26. Spano SJ, Reagle Z, Evans T. Symptomatic hypotonic hyponatremia presenting at high altitude. Wilderness Environ. Med. 2014; 25: 69-74.
27. Tienviboon C, Punyagupta S, Pongtarakulpanit A, Prichanond S. Reversible cerebral vasoconstriction syndrome with increased intracranial pressure, probably related to altitude changes and windy winter travelling. J Med Assoc Thai. 2011; 94: 622-628.
28. Hauser M, Mueller A, Swai B, Moshi E, Ole Nguyaine S. Deaths due to high altitude illness among tourists climbing Mt. Kilimanjaro. Proceedings of the 2004 South African Travel Medicine Society. February 9-11, 2004; Cape Town, South Africa.
29. Falla M, Giardini G, Angelini C. Recommendations for traveling to altitude with neurological disorders. J Cent Nerv Syst Dis. 2021; 13: 11795735211053448.
30. Hüfner K, Sperner-Unterweger B, Brugger H. Going to Altitude with a Preexisting Psychiatric Condition. High Alt Med Biol. 2019; 20: 207-214.