HIGH ALTITUDE ILLNESS: EXPERIENCE OF ONE YEAR

Azmat Hayat, *S. Karamat Hussain Bokhari, **M. Mazhar Hussain, ***Sohail Aziz, ***M.M.H. Nuri, ****Tassawar Hussain, ***Syed Afzal Ahmad

CMH Peshawar, *CMH Lahore, **Army Medical College Rawalpindi, ***AFIC/NIHD Rawalpindi, ****GHQ Medical Directorate Rawalpindi

ABSTRACT

Objectives of this study was to analyze symptoms of altitude illness commonly necessitating evacuation of patients from high altitude to the base hospitals in Siachin area by simple clinical screening. This was a prospective observational. Place and duration of study was Northern areas of Pakistan. This study was carried out at altitudes of 3633 meters (GOMA) and 2833 meters (Siksa) from Oct 2003 till Oct 2004. One hundred and thirty eight patients of altitude illness were studied, who were evacuated from altitude above 4000 meters. Out of 138 cases, 103 (74.6%) patients suffered from acute mountain sickness (AMS), while 21(15.2%) patients developed high altitude pulmonary edema (HAPE) and 14 (10.1%) patients developed high altitude cerebral edema (HACE), of which 3 patients were having concomitant HAPE. The most common symptom combination was headache and vomiting which was the presenting feature in 53(38.4%) patients, followed by headache, loss of appetite and insomnia, in 41(29.7) patients. Headache was the most common single symptom present in about 120(86.96%) patients that required evacuation. The next common symptom was vomiting that was found in 51(36.96%) patients while shortness of breath was present in 33(23.91%) patients. We conclude that headache is the most common presenting symptom in all the patients of AMS. Therefore, headache at high altitude should be taken seriously, if does not respond to common medication, then patient should be evacuated to a lower altitude. The incidence of AMS, HAPE, and HACE may be reduced by improving the physical fitness of mountaineers and observing the protocol of acclimatization.

Keywords: High altitude, AMS, HAPE, HACE)

INTRODUCTION

Mountains have fascinated and attracted mankind for centuries. By the end of last century most peaks in the world were scaled and early climbers mentioned experiencing some untoward symptoms, now commonly described, as mountain sickness. By the beginning of the 20th century, hypoxia was the known major cause of these symptoms, even though, many questions regarding precise mechanism of altitude illness remain unanswered. Despite obvious dangers inhereted in climbing, the altitude illness is experienced by nearly all people who spend significant time on mountains and continue to seek the remoteness and pleasure of high places. These individuals frequently experience acute illness soon after ascent. With longer stays at altitude, symptoms improve as the process of 'acclimatization' sets in. Three major syndromes; acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE), are now commonly accepted high altitude illnesses.

Acute mountain sickness is a syndrome of nonspecific symptoms which are subjective in severity amongst different people. The Lake Louise Consensus Group defined acute mountain sickness as the presence of

Correspondence: Maj Azmat Hayat, Medical Specialist, Combined Military Hospital, Peshawar

headache in an unacclimatized person who has recently arrived at an altitude above 2500 m plus the presence of one or more of the following: gastrointestinal symptoms (anorexia, nausea, or vomiting), insomnia, dizziness, and lassitude or fatigue [1]. The factors that affect the incidence and severity of AMS, include the rate of ascent, altitude attained (especially altitude of sleep), duration of exposure, and amount of exercise undertaken at altitude [2]. The most important but least understood variable is the underlying physiological susceptibility of the individual to develop AMS.

HAPE is a serious and potentially life-threatening manifestation of altitude illness. The early description of HAPE was given by Mosso, in 1898, who described a fatal case on Mont Blanc [3]. Fifteen years later, in 1913, Raven hill described different types of mountain sickness, which included HAPE in Andes [4]. These early reports ascribed HAPE as a cardiac disease which were largely ignored. However, with Houston's report [5] in 1960, it was finally recognized as an unusual form of pulmonary edema instead of heart failure. The first symptom of HAPE usually appears within 1-3 days of arrival at high altitude that is commonly aggravated by exercise and consists of cough, shortness of breath, chest tightness, and fatigue. HAPE is usually preceded by the symptoms of AMS in approximately half of the cases and the person often experiences diminishing effort tolerance to the point of being breathless at rest. The cough is initially dry that becomes wet and productive and tinged with blood. Auscultation of the chest reveals crackles. As the gas exchange worsens in lungs the patient becomes increasingly cyanosed.

HACE is an extreme form of mountain sickness. The signs and symptoms of HACE may progress rapidly (within 12 hours) from minimal manifestations to coma. Typically, this progression occurs slowly. Often the symptoms of HACE begin at night and occasionally result in loss of consciousness during sleep. Most cases of HACE occur after individuals have been at altitude for several days.

Due to the development of road links to the northern areas of Pakistan there had been an increase in number of people visiting high altitude. It provided the unique opportunity to study the effects of high altitude on health in people ascending to the extreme altitudes and effects of strenuous physical activity for prolonged period of time.

The aim of the study was to analyse symptoms of altitude illness commonly necessitating descent or evacuation of patients to low altitude by simple clinical screening.

MATERIAL AND METHODS

This study was carried out at altitudes of 2833 meters (Siksa) and 3633 meters (Goma) from October 2003 till October 2004. 138 patients were studied who were evacuated from various high altitudes greater than 4000 meters on account of high altitude illness. The patients were first examined by the doctor at the respective altitude and a detailed history alongwith complete clinical examination was recorded. The criteria for the referral of the patient to the base hospital was as follows:

1. The diagnosis and classification of AMS on the basis of Lake Louise Conesus scoring system. (1): In this system five symptoms (headache, gastrointestinal upset, fatigue, dizziness, and sleep disturbance) were recorded with the severity score 0 to 4. The cases of mild AMS (score less than 4) whose symptoms improved with initial treatment at the same altitude, were not evacuated to low altitude and hence not included in this study.

2. The diagnostic criteria for HAPE was the presence of at least 2 symptoms and 2 signs from the following list, in the patients who had the history of recent gain in altitude:

(a) Symptoms (1) Dyspnea at rest (2) Cough (3) Weakness or decreased exercise performance (4) Chest tightness or congestion.

(b) Signs (1) Rales or wheezing in at least one lung field, (2) Central cyanosis, (3) Tachypnea, (4) Tachycardia.

3. The Lake Louise definition states that HACE "can be considered as the 'end stage' of severe AMS. In the setting of a recent gain in altitude, the presence of a change in mental status and/or ataxia in a person with AMS, or presence of both disturbed mental status and ataxia in a person without AMS [6].

In the base hospital the patients underwent complete clinical examination that included:

- Pulse rate / min
- Arterial blood pressure, systolic/diastolic (mm Hg)
- Oxygen saturation by pulse oximeter (%)
- Complete systemic examination

The following laboratory investigations were done in all the patients:

- Blood complete picture.
- Urine routine examination
- Electrocardiogram
- X ray chest
- Blood urea and electrolytes.

The data was analyzed on the spss version 11.

RESULTS

The physical features of 138 patients who were evacuated from different high altitudes are given in (table-1). The patients were also categorized into three altitude illnesses (AMS, HAPE and HACE) as shown in (table-2). It reveals 103 (74.2%) cases of acute mountain sickness (AMS), 21(15.44%) cases of high altitude pulmonary edema (HAPE), while 14 (10.29%) cases were of high altitude cerebral edema (HACE). There were three cases who were suffering from both HACE and HAPE. The most common symptoms were; headache and vomiting which were the presenting feature in 53(38.4%) patients, followed by headache, loss of appetite, insomnia, in 41(29.7) patients. Headache was the most common symptom that led to the evacuation of 120(86.96%) patient while second common symptom was vomiting, found in 51(36.96%) patients while shortness of breath was present in 33(23.91%) patients (table-3).

Amongst 103 cases of AMS, the most common single symptom that led to evacuation (descent) was headache in these patients, while symptoms accompanied with headache were vomiting and loss of appetite.

There were 21(15.22%) patients of HAPE amongst this group, 11 patients had ECG changes suggestive of right sided strain pattern secondary to raised pulmonary artery pressure. These changes consisted of right bundle block pattern, with R wave in lead V1, P pulmonale and T wave inversion in anterior chest leads. The most common symptoms were; shortness of breath and cough which was present in more than half of the cases while the most common single symptom was shortness of breath in these patients. The group of patients with ECG changes had chest pain as well. The most common clinical findings were crepitations on auscultation of chest which were largely bilateral although some cases had unilateral crepitations (right midzone), loud second heart sound (pulmonary component) and decreased arterial oxygen saturation. The radiological finding was the presence of bilateral opacities in both lung fields in most of the patients of HAPE while a few had unilateral opacities (mid zone right sided) and only one patient had increased CTR with overt findings of right sided failure.

There were 14(10.14%) cases of HACE (table-2). Their common symptom was headache accompanied by vomiting and weakness of one side of body. Headache was the commonest single symptom, present in almost all the cases. The other common clinical findings were hemiplegia in 5 (35.7%) patients, ataxia in 2 patients and cranial nerve palsy in one patient.

There were 03 cases who developed moderate AMS at relatively lower altitude of 3633 meters and had to be admitted in the hospital. In addition, 2 cases had a history of prolong stay of 60 days at 6000 meters altitude who developed severe degree of AMS and had to be evacuated to the base hospital.

DISCUSSION

The incidence of AMS in present study was 3.36% as compared to previous studies which varied from 30% [7] to as high as 68% [8]. The low incidence of AMS in our study may be due to the reason that we selected only those cases who were evacuated on account of moderate to severe AMS and did not include overwhelming majority of soldiers suffering from mild AMS, because they came out of symptoms after initial few days.

Multiple factors contribute to the development of AMS, but the exact role each of such factors in the development of AMS is unknown. Sutton and Lassen suggested that hypoxia stimulates increased cerebral blood flow that results in vasogenic cerebral edema [9] whereas some studies have shown that individuals with lower vital capacity and weak hypoxic ventilatory response are more likely to experience altitude illness [10–12]. The other factors which have been considered contributory to pathophysiology of AMS include fluid retention [13], anti diuretic hormone and atrial natriuretic factor alongwith neurological changes producing secretion of hormones promoting fluid retention [14].

The incidence of HAPE in our study was 0.75% as compared to other studies which vary between 2.5 - 5% [15]. There were 11 patients who manifested ECG changes suggestive of right sided cardiac strain pattern [16,17]. Some of our cases of severe AMS also had ECG changes suggestive of right sided strain secondary to raised pulmonary artery pressure. [18,19,20]. The other findings in the cases of HAPE include; the absence of left ventricular failure and thrombosis in capillaries and arteries which are commonly found in many fatal cases of HAPE. The recent work on pathophysiology of HAPE advocate the over perfusion and, rupture of pulmonary capillaries, alongwith the inflammatory leukotrienes as the mechanism for the development of HAPE [21]. The over perfusion mechanism postulates that uneven hypoxic pulmonary vasoconstriction results in areas of decreased blood flow and areas of excessive perfusion. The overperfused areas in lungs are the proposed sites for the leakage of edema fluid. The nonhomogeneous vasoconstriction proposed by Hultgren [21] would transmit high pulmonary artery pressure to the pulmonary capillaries in overperfused areas. West et al have suggested that HAPE results due to the rupture of pulmonary capillaries subjected to high wall stresses produced by the high pressure in pulmonary vessels [22]. Bronchoalveolar lavage studies show that edema fluid in HAPE has a high protein concentration alongwith various inflammatory markers, such as complement C5a and leukotriene B4 [11].

HACE is the severe form of cerebral manifestation of altitude illness [23]. Its pathophysiology shares many etiologic factors with AMS but despite similarities, it is not yet clear as to why only a few persons with AMS develop HACE? Although persons with AMS do have mild neurological symptoms but a very few cases actually develop full blown HACE. The incidence of HACE in our study was 0.46% which was low as compared to other studies in which it varies between 1.8 - 31% [7,8]. The low incidence of HACE in our study may be attributed to the physical fitness of our volunteers and strictly observing graded ascent as compared to the international trekkers [24,25]. In our study there was no case of HAPE who later developed HACE, hitherto we 3 cases of HACE accompanied by HAPE [26]. The mean stay at high altitude for this group of patients was longer than the mean stay of the whole group (18.90 + 7.84 vs. 10.20 + 1.62 days). This finding supports the view that longer stay at high altitude results in polycythemia and predispose the development of HACE. The mean altitude from which this group of patients were evacuated, was higher than for the AMS group of patients (5907.93 + 174.65 vs. 5621.49 + 81.91 meters) (table-2). It supports the hypothesis that at extreme altitude there occurs severe hypoxic insult that can lead to the development of complicated form of mountain sickness. MRI of patients with HACE reveals edema of the white matter, especially in the corpus callosum [27]. The current pathophysiologic mechanisms of HACE under investigation includes, cytotoxic cellular edema of brain due to hypoxia [28], vasogenic edema resulting due to the increased cerebral blood flow and leakage of fluid into the brain [29] alongwith the role of cerebral angiogenesis, osmotic swelling, ischemia [30] and tight fit hypothesis [10].

Table-1: Basic parameters of the study group

Parameters	Range	Mean	SEM(±)
Age(years)	18 – 48	28.32	0.58
Evacuating altitude(meters)	4000–7000	5624.92	70.39
Systolic blood pressure(mm Hg)	90 – 140	114.90	0.81
Diastolic blood pressure(mm Hg)	70 – 100	76.31	0.96
Oxygen saturation of blood %	86% – 92%	89.21%	0.90

Table-2: Frequency of high altitude illness in patients requiring evacuation to low altitude with duration of stay at high altitude.

High Altitude Diseases	No of Patients	Mean age	Mean evacuating altitude	Mean period of stay at HA	Most common symptom
AMS	103	28.37 ± 0.58 yrs	5621.49 ± 81.91 meters	10.49 ± 2.20 days	Headache
HAPE	21(15.54%)	29.58 ± 1.32 yrs	5694.95 ± 168.81 meters	13.33 ± 4.88 days	Shortness of breath
HACE	14(12.72%)	27.00 ± 1.31 yrs	5907.93 ± 174.65 meters	18.90 ± 7.84 days	Headache and Vomiting

Table-3: Symptoms of patients evacuated to base hospital.

Presenting complaints	Frequency	Percent
Chest pain and hemoptysis	1	0.7
Shortness of breath and cough	9	6.5
Headache, loss of appetite and insomnia	41	29.7
Headache, shortness of breath	4	2.9
Headache, chest pain and shortness of breath	12	8.7
Headache and vomiting	53	38.4
Headache, vomiting and weakness one half body	5	3.6
Headache and diplopia	1	0.7
Shortness of breath, cough and chest pain	6	4.3
Shortness of breath, swollen feet and pain abdomen	1	0.7
Shortness of breath, hemoptysis and dizziness	1	0.7
Headache and dizziness	4	2.9
Total	**138**	

Al though the incidence of high altitude illnesses has decreased, due to strict adherence to the standard protocol of ascent, but a lot of morbidity and mortality is still associated with these illnesses. Which is a source of apprehension in both the patients as well as in doctors. Moreover, these are major causes of human and financial loss and limitation to the operational capabilities of the army. The acclimatization protocol is excellent, effective and time tested, but there is a need to improve awareness about high altitude diseases both in newly engaged troops and in junior doctors to further reduce the number of these cases.

CONCLUSION

The present study concludes that headache is the most common symptom of AMS and it should be taken seriously if does not respond to common medication, or is accompanied by shortness of health and/or vomiting.

REFERENCES

1. Roach RC, Bärtsch P, Oelz O, Hackett PH. Lake Louise AMS Scoring Consensus Committee. The Lake Louise acute mountain sickness scoring system. In: Sutton JR, Houston CS, Coates G, eds. Hypoxia and molecular medicine. Burlington, Vt.: **Charles S Houston 1993: 272-4.**
2. Peter H. Hackett and Robert C. Roach. High-Altitude Illness. **N Engl J Med 2001; 345: 107-114.**
3. Mosso. Life of Man on the High Alps. London, England: **T Fisher Unwin; 1898.**
4. Raven hill TH. Some experiences of mountain sickness in Andes. **J Trop Med Hygiene 1913; 16: 313-320.**
5. Houston CS: Acute pulmonary edema of high altitude. **N Engl J Med 1960; 263:478-80.**
6. Anonymous: The Lake Louise Consensus on the Definition and Quantification of Altitude Illness. In: Sutton JR, Coates G, Houston CS, eds. Hypoxia and Mountain Medicine. Burlington, Vt: **Queen City Printers 1992; 327-30.**
7. Basnyat B, Lemaster J, Litch JA. Everest or bust: a cross sectional, epidemiological study of acute mountain sickness at 4243 meters in the Himalayas. **Aviat Space Environ Med 1999; 70(9): 867-73.**
8. Basnyat B, Subedi D, Sleggs J, Lemaster J, Bhasyal G, Aryal B, Subedi N. Disoriented and ataxic pilgrims: an epidemiological study of acute mountain sickness and high altitude cerebral edema at a sacred lake at 4300m in the Nepal Himalayas. **Wilderness and Environmental Med 2000; 11: 89-93.**
9. Sutton JR, Lassen N. Pathophysiology of acute mountain sickness and high altitude pulmonary edema: an hypothesis. **Bull Physiopathol Respir (Nancy) 1979; 15(5): 1045-52.**
10. King AB, Robinson SM. Ventilation response to hypoxia and acute mountain sickness. **Aerosp Med 1972; 43(4): 419-21.**
11. Schoene RB, Swenson ER and Pizzo CJ: The lung at high altitude: bronchoalveolar lavage in acute mountain sickness and pulmonary edema. **J Appl Physiol 1988; 64(6): 2605-13.**
12. Moore LG, Harrison GL, McCullough RE. Low acute hypoxic ventilatory response and hypoxic depression in acute altitude sickness. **J Appl Physiol 1986; 60(4): 1407-12.**
13. Hackett PH, Rennie D, Hofmeister SE. Fluid retention and relative hypoventilation in acute mountain sickness. **Respiration 1982; 43(5): 321-90.**
14. Bartsch P, Shaw S, Franciolli M. Atrial natriuretic peptide in acute mountain sickness. **J Appl Physiol 1988; 65(5): 1929-37.**
15. Hackett PH, Rennie ID, Levine HD. The incidence, importance, and prophylaxis of acute mountain sickness. **Lancet 1976; 2: 1149-54.**
16. Oelz O, Maggiorini M, Ritter M, Waber U, Jenni R, Vock P, Bartsch P. Nifedipine for high altitude pulmonary oedema. **Lancet 1989; 2:1241-4.**

17. Grissom CK, Elstad MR. The pathophysiology of high altitude pulmonary edema. **Wilderness Environ Med 1999; 10: 88-92.**

18. Grover RF, Vogel JH, Voigt GC. Reversal of high altitude pulmonary hypertension. **Am J Cardiol 1966; 18(6): 928-32.**

19. Groves BM, Reeves JT and Sutton JR. Operation Everest II: elevated high-altitude pulmonary resistance unresponsive to oxygen. **J Appl Physiol 1987; 63(2): 521-30.**

20. Hackett PH, Creagh CE, Grover RF. High-altitude pulmonary edema in persons without right pulmonary artery. **N Engl J Med 1980; 302(19): 1070-3.**

21. Hultgren HN. High-altitude pulmonary edema: current concepts. **Annu Rev Med 1996; 47: 267-84.**

22. West JB, Colice GL, Lee YJ. Pathogenesis of high-altitude pulmonary edema: direct evidence of stress failure of pulmonary capillaries. **Eur Respir J 1995; 8(4): 523-9.**

23. Hackett PH, Roach RC. High-altitude medicine. Wilderness Medicine, (Auerbach PA, ed.). **St Louis, MO: Mosby; 1995: 1-37.**

24. Singh I, Khanna PK, Srivastava MC. Acute mountain sickness. **N Engl J Med 1969; 280(4): 175-84.**

25. Houston CS, Dickinson J. Cerebral form of high-altitude illness. **Lancet 1975; 2: 758-61.**

26. Hackett PH. The cerebral etiology of high-altitude cerebral edema and acute mountain sickness. **Wilderness Environ Med 1999; 10: 97-109.**

27. Hackett PH, Yarnell PR and Hill R. High-altitude cerebral edema evaluated with magnetic resonance imaging: clinical correlation and pathophysiology. **J Am Med Assoc 1998; 280(22): 1920-5.**

28. Hansen JE, Evans WO. A hypothesis regarding the pathophysiology of acute mountain sickness. **Arch Environ Health 1970; 21(5): 666-9.**

29. Lassen NA, Harper AM. High-altitude cerebral edema. **Lancet 1975; 2; 7945- 1154.**

30. Severinghaus JW. Hypothetical roles of angiogenesis, osmotic swelling, and ischemia in high-altitude cerebral edema. **J Appl Physiol 1995; 79(2): 375-9.**