

Chapter 111

High Altitude Mountain Sickness

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INTRODUCTION

The landing of “Curiosity” on Mars is just one step in the quest of man to be on the top of everything which started with his advent of mountaineering. Till 18th century people living at low altitude were forbidden from going to high altitude and vice versa to prevent the onslaught of altitude-related problems. Though the earliest civilizations thrived by the river sides, the oldest human skeleton (9,000 years) has been found at 14,000 ft. Mountaineering and mountain exploration present two faces: (1) the romantic and (2) the scientific. In the mountains, one is exposed to the stress of high altitude, the stress of cold and the inherent dangers of topography. The origins of mountaineering and of respiratory physiology are interwoven and many important observations have been made by laymen as well as scientists working with the great climbers and the troops at high altitudes. Every year, hundreds of adventurous trekkers risk money and their lives to scale various peaks. India and Pakistan are spending billions of rupees and still losing hundreds of able bodied persons, without fighting, for want of proper knowledge to combat the problems of high altitude, an enigma.

EFFECTS OF ALTITUDE

Atmospheric pressure and temperature badly affect the mountaineers and troops at high altitude, besides terrain, wind, ultraviolet light, rain and snow. Air, a mixture of nitrogen (79%), oxygen (20.94%) and carbon dioxide (0.04%), exerts a pressure of 760 mm Hg at sea level. The atmospheric (barometric) pressure, falls by 24 mm Hg with each rise of 1,000 ft. The effect of each gas depends on its individual or partial pressure, that of nitrogen (pN_2) is 570 mm Hg, of oxygen (pO_2) is 152 mm Hg and of carbon dioxide (pCO_2) is 38 mm Hg at sea level. The relative percentage of oxygen and carbon dioxide in the air remains the same up to a height of 70,000 ft but their partial pressures decrease as we ascend. Hence, at a height of 21,000 ft with the barometric pressure at 334.8 mm Hg, the pO_2 will be 62 mm Hg, producing a state of hypoxia, the basic cause of all the altitude-related illnesses. A fall in both pO_2 and pCO_2 causes reactionary respiratory and cardiac stimulation resulting in tachycardia and tachypnea, the two major responses to high altitude, being further enhanced by the cold and winds. People at high altitudes have usually got conditioned (acclimatized) to these changes whereas a new comer has to get acclimatized either at ground level by strenuous exercise, by use of decompression chambers, or by gradual ascent; otherwise he is likely to suffer from altitude-related illnesses which may prove fatal (Figure 1).

Any type of disease can occur and get worsened at high altitude, defined as a height of 9,000 ft or more at which the rarefaction of air

produces physiological as well as pathological effects detrimental to life. This chapter confines itself to diseases due to the effect of high altitude only.

ACUTE MOUNTAIN SICKNESS

Its probable first account has been given by Plutarch in his comments on Alexander’s invasion of India: “Many then were the dangers—but the greatest harm came from severity of weathers”. Similarly, a Chinese record mentions of “Lesser Headache Mountains” and the “Greater Headache Mountains” where men’s bodies become feverish, they lose color and are attacked with headache, dizziness and vomiting. Being the most frequent type of altitude sickness, in young robust trekkers and troops (17–28 years) at 9,000–17,000 ft. It manifests 6–10 hours after a rapid ascent and generally subsides in 1–2 days, but may occasionally develop into the more serious condition-HACE (high altitude cerebral edema).

Symptoms

Headache, without dehydration at 8,000 ft or more is the primary symptom usually combined with one or more of the following:

- Lack of appetite, nausea, vomiting and diarrhea
- Fatigue or weakness
- Dizziness or lightheadedness
- Insomnia
- Pins and needle pricks



Figure 1: The author at Khardung – LA (19,800 ft)
(The highest motorable pass in the world)

Pulmonology

- Shortness of breath upon exertion
- Nose bleed
- Persistent rapid pulse
- Drowsiness
- General malaise
- Peripheral edema (swelling of hand, feet and face)
- Periodic breathing at night with spells of apnea.

Risk Factors

- *Home elevation (altitude of residence) and maximum sleeping altitude (while ascending)* are preventive factors whereas the risk is increased in those living at sea level
- *Rate of ascent* directly affects the onset, symptom severity and prevalence, being slower in the ascent to mount Everest region in Nepal as compared to the mount Kilimanjaro where the rate of ascent is more rapid
- *Latitude* affects oxygen availability, hemoglobin saturation and the risk of development. The Earth bulges at the equator and is punched at the poles, PO₂ being higher at the equator than at the poles, at an equivalent altitude, climbers will be less hypoxic on Kilimanjaro (3's) or even on Everest (23'N) than on Denali (63'N) (Central Alaska)
- *Gender and age* are immaterial. However, women and younger individuals suffer more
- *Intensity of exercise* at high altitude increases the risk due to widened alveolar arterial pressure difference which reduces hemoglobin saturation. Hence, rest on the first day is the rule
- *Arterial oxyhemoglobin saturation* when lower than that expected for a given altitude worsens the condition because of diffusion impairment or venous admixture
- *Prior acute mountain sickness (AMS)* is an important predictor of risk while a successful recent or extreme altitude exposure lowers it
- *Genetic adaptations:* Humans have lived and worked at high altitude for ages; the Sherpas and Tibetans in the Himalayas and the Quiche and Andean in the Andes, the former respond to their hypoxic environment with a high ventilatory response and the latter with a high hemoglobin concentration, although pointing to a genetic component, is not substantiated. Hypoxia downregulates protein synthesis but the up-regulated cellular functions include the release of erythropoietin resulting in an increased hematocrit and O₂ carrying capacity and a polymorphism in the angiotensin converting enzyme gene, in elite mountaineers and in endurance athletes
- *Pre-existing diseases* of lungs are high risk, while severe anemia, sickle cell disease and overt heart failure are strict contraindications for ascent, but asymptomatic ischemic heart disease (IHD) patients may pursue slowly.

Pathophysiology

Mild to moderate AMS presents with relative hypoventilation, fluid retention and redistribution and increased sympathetic drive, without a rise in intracranial pressure. Exacerbation by exercise after immediate altitude exposure without any signs of pulmonary gaseous exchange abnormalities and only small difference in fluid balance, suggests a role for vasogenic cerebral edema, elevated cerebral blood volume (CBV) brain swelling, fluid retention/redistribution and intracranial hypertension in severe AMS and HACE. Hypoxemia stimulates cellular and molecular responses that may alter endothelial permeability or provide cellular protection against oxygen derived free radical damage to the endothelium, Hypoxemia is also implicated in up-regulation of inducible nitric oxide synthase and nitric oxide has been implicated in the pathophysiology of headache and blood brain barrier (BBB) permeability. Through

peripheral chemoreceptor activation, hypoxemia can elevate circulating arginine vasopressin levels which in turn causes antidiuresis and increased extracellular water level. These changes cause elevated intracranial pressures in those with a cerebrospinal capacitance that cannot buffer the swelling brain which ultimately determines who develops AMS. The trigeminovascular system is activated at high altitude by both chemical and mechanical stimuli resulting in headache seen in AMS.

Laboratory Evaluation

Individuals with the lower initial partial pressure of end-tidal pCO₂ and corresponding high oxygen saturation level tend to have a lower incidence of AMS than those with high end-tidal pCO₂ and low oxygen saturation levels.

The Lake Louise assessment system of AMS is based on a self-report questionnaire as well as a quick clinical assessment.

Prevention

Ascending slowly and avoiding strenuous activity such as skiing, hiking, etc. in the first 24 hours reduces the symptoms. Alcohol causes dehydration, exacerbates AMS, hence be avoided.

Altitude Acclimatization

Altitude acclimatization is the process of adjusting to decreasing oxygen levels at higher elevation, most climbers and high altitude trekkers take the "climb-high, sleep-low" approach. A typical acclimatization regimen might be to stay a few days at a base camp, climb to the higher camp (slowly) and then return to base camp. A subsequent climb to the higher camp then includes an overnight stay. This process is repeated a few times, extending the time spent at higher altitude to let the body adjust to the oxygen level there, resulting, in the production of additional red blood cells. The general rule of the thumb is to not ascend more than 1,000 ft per day to sleep, i.e. climb up to 13,000 ft from 10,000 ft and descend back to 11,000 ft to sleep. Simulated altitude equipment; the hyperbaric chambers being used to reduce the total time required on the mountain itself has not proved to be as useful. Prophylactic use of acetazolamide (125 mg BD) has proven to be efficacious, while the role of magnesium and leukotriene receptor blockers is still experimental.

Medical Treatment

Acute mountain sickness is usually self-limiting. Complete bed rest with plenty of fluids and oxygen enrichment can counteract its hypoxia related effects. At 11,155 ft raising the oxygen concentration level by 5% provides an effective altitude of 10,000 ft more tolerable for surface dwellers. Acetazolamide may help in a rapid ascent to sleeping altitude above 9,000 ft, in a dosage of 125–250 mg twice daily, starting from 24 hours before ascending until a few days at the highest altitude or on descending. For those with allergy to sulfa or its toxic side effects, dexamethasone 4 mg stat followed by 4 mg/12 hourly may suffice.

For more serious cases, use of hyperbaric (decompression) chambers (**Figure 2**), produce excellent results, which if not available, or where rapid descent is impractical, a Gamow bag (**Figure 3**), a plastic portable hyperbaric chamber inflated with a foot pump is generally used both to treat at high altitude and as an aid for descent.

Sumatriptan (50 mg PO within 1 hour of ascent) has preventive effect. Antioxidants have not been found to be effective. The use of phosphodiesterase-5 (PDE-5) inhibitors like sildenafil and tadalafil has been limited by the possibility of their worsening the headache of AMS. Myo-inositol triphosphate (ITPP) which increases the amount of oxygen released by hemoglobin has proved useful. The locals have been using their indigenous medications like: coca leaves



Figure 2: The author in a hyperbaric chamber (HARC, Leh)



Figure 3: The author in a Gamow bag (HARC, Leh)

by Aymaras of the Altiplano, the extract of tissue of *Radix rhodiola* by the Chinese and the Tibetans (the latter also use uncooked fish lying by the side of lake Manasarovar) and *Ginkgo biloba* by the Nepalese for relief.

HIGH ALTITUDE CEREBRAL EDEMA

It is a likely continuum of AMS. Individuals with high Lake Louise scores should be carefully monitored for the signs of ataxia, confusion, hallucinations and coma. The exact processes are unknown and the pathophysiology is the same as mentioned in severe AMS (vide supra). Magnetic resonance imaging (MRI) studies of patients showed the majority had intense T2 signals in white matter areas, particularly the splenium of the corpus callosum, without gray matter involvement, with predilection for reversibility and positive response to steroids, proving its vasogenic origin. The usual cause of death is brain herniation.

Treatment

All the measures taken for AMS may be used in a greater degree. The use of decompression chambers has significantly reduced mortality. If not available, a Gamow bag is a good substitute and useful for descent as well. Normal oxygen with steroids in the doses of 8 mg dexamethasone stat with 4–8 mg/4 hourly along with acetazolamide (250–500 mg BD) has proven efficacious. Rapid descent is the infallible cure.

HIGH ALTITUDE PULMONARY EDEMA

A potentially fatal consequence of rapid ascent occurring 2–4 days after arrival at altitudes greater than 10,000 ft (occurs at lower altitude as well, with 47 cases reported in 7 years at 8,400 ft). Early diagnosis is difficult since many of the symptoms (shortness of breath, tachypnea, tachycardia, reduced arterial saturation, fatigue and cough) are often present in unaffected climbers, particularly in cold, dry or dusty environments. It is characterized by incapacitating fatigue, dyspnea at rest, or orthopnea and a dry nonproductive cough progressing to a productive cough with pink frothy sputum due to hemoptysis with central cyanosis and a few rales with ronchi in the right middle lobe to begin with, which may extend to the whole of the lung. At times the lungs may be clear. A noncardiogenic type of edema without S3 gallop characterized by high content of proteins and cells along with complement factors C5a and LTB4 in bronchoalveolar lavage, with neutrophil chemotaxis may be present. Presence of fever does not imply infection and many people have died being misdiagnosed as pneumonia.

The exact pathogenesis though still evasive, it is considered to be due to an enhanced pulmonary reactivity to hypoxia resulting in an exaggerated increase in pulmonary artery pressure. The basic defect is a hydrostatic-induced permeability leak with mild alveolar hemorrhage. This is due either to nonhomogeneous hypoxic pulmonary vasoconstriction resulting in high pressure in the pulmonary capillaries supplied by dilated arterioles causing damage to the capillary walls leading to a leak of high protein edema fluid with erythrocytes; or an increase in pulmonary capillary pressure due to hypoxic pulmonary venous constriction.

Treatment

Descent or simulated descent in decompression chambers provides excellent results. Oxygen, rest and descent in a Gamow bag are the alternative measures. Diuretics like frusemide do not help much. Nifedipine 10 mg orally followed by 30 mg extended release formulation every 12–24 hours and phosphodiesterase-5 inhibitors such as sildenafil and tadalafil (10 mg BD) have been used both for prophylaxis and treatment. Dexamethasone (8 mg BD) has given better results along with Salmeterol inhalation (125 µg BD). Use of minoxidil for its K⁺ channel activation to relieve pulmonary hypertension is experimental.

CHRONIC MOUNTAIN SICKNESS

Chronic mountain sickness also called “chronic seroche” and “Monge’s disease” represents a clinical picture, seen in South-America, Colorado and Han Chinese in Tibet when individuals normally acclimatized to and residing at high altitude lose, their ability to adapt to the low oxygen tension at which they have been living for a long period without symptoms, in the absence of any pulmonary disease like pulmonary fibrosis, silicosis, kyphoscoliosis or pulmonary emphysema and a normal thoracic cage. It appears to be a particular variety of alveolar hypoventilation probably due to a decreased sensitivity of the respiratory center to carbon dioxide, or an irreversible insensitivity of the peripheral chemoreceptors to hypoxic stimuli, resulting in excessive polycythemia and higher degree of pulmonary hypertension and needs to be distinguished from *high altitude pulmonary hypertension*, a subacute disease of long-term high altitude residents and afflicting the Indian troops living for prolonged periods at high altitudes in the absence of polycythemia.

Both are characterized by decreasing exercise tolerance, a gradually increasing fatigue with headache, paresthesias and a feeling of somnolence and neurological and psychic disturbances. Cyanosis and clubbing of the fingers are common leading to signs of congestive cardiac failure.

Treatment

Descent to sea level. Some palliation may be obtained by venesection and acetazolamide. Use of oxygen increases arterial oxygen saturation with improvement in neuropsychiatric symptoms and a fall in pulmonary artery pressure without any changes in polycythemia.

THROMBOSIS

High altitude increases the tendency to thrombosis and thrombophlebitis especially in the legs. The increase in hemoglobin and red cell count leads to an increase in blood viscosity. There is also a fall in arterial pressure. Vasoconstriction due to hypoxia and cold, extreme dehydration due to increased respiration and curtailment of physical activity, especially when there are storms, usually occur over 23,000 ft. All these may contribute to thrombosis. Adequate hydration and exercise are obviously important in prevention. In severe cases, oxygen should be given and the patient immediately evacuated to a lower level. Anticoagulants should not be started until adequate control is available.

COLD INJURY

There are two distinct types of cold climate: wet cold and dry cold. The wet cold climate covers large areas of the world's so called temperate zone, where the range of air temperature varies between 10°C and -5°C. Wind, rain, sleet and mud are not uncommon. Hypothermia and immersion injury occur under these conditions. In dry cold regions, the temperature rarely rises above freezing point; so snow, ice and wind contribute to cold stress. Hypothermia, frostbite and immersion injury can occur. The wind chill effect is an important factor as the cooling effect of wind and cold on exposed tissues is more dangerous than that of cold alone. Cold injury is more in the mountains as the temperature drops about 1°C for every 180 meter of ascent and as a still day is rare, the wind chill factor has always to be taken into account. Mountaineers and troops at high altitude suffer frostbite and other injuries despite being fully clothed, physically fit and ascending normally because hypoxia leads to loss of concentration and forgetfulness. So, vital items of equipment such as headgear and gloves may be lost or not worn. Increased heat loss occurs due to tachypnea. Loss of appetite and weight especially loss of fat, causes diminished body insulation. Other factors reducing blood supply to the periphery (thrombosis-supra) result in impairment of tissue nutrition and thereby necrosis. Proper clothing with amenities for weather forecast and informing the accompanying paramedics of the slightest change or injury is very helpful. However, immediate descent is the final cure.

Psychological Changes

The mountaineer (climbers and troops) through a long and patient study of his craft and as a result of a variety of experiences gained in different kinds of mountain country can learn to move safely in situations, but for his skill would be unacceptably dangerous. Hence, develops an entirely different personality. He appears to be more intelligent with abstract thinking, but socially reserved and detached compared to other people and tends to be withdrawn and cautious. He prefers one or two close friends to large groups. Being self-sufficient, he likes to make his own decisions is highly competitive,

aggressive and resourceful in his chosen sphere of activity. Two main types of personalities are seen:

1. The schizoid-psychasthenic type is the commonest type, characterized by features such as being secretive, reserved and avoiding contact with other people. He is emotionally sensitive but has difficulty in revealing this. A feeling of inferiority with a high level of aspiration may be noted. He is extremely active and self-reliant but tends to be eccentric with unconventional behavior. They have a marked difficulty in submitting to social and collective discipline, resulting in breakdown in familial, social and occupational circles.
2. The astheno-neurotic type is characterized by timidity, shyness and many neurotic symptoms. They try to be conspicuous among their contemporaries and have high ambitions and aspirations. For many childhood illness and other factors in early life have led to a feeling of inferiority in personal worth and physical efficiency, thus avoiding their peers and in desire to test themselves seek difficulties and danger. They often tread to routes and places which prove suicidal.

This chapter is dedicated to the valor of our soldiers who guard the inaccessible terrains of our frontiers and who are called for any type of calamity and to their savior and guides like Brig. Hemashree (AMC), then Col. Commandant High Altitude Research Center (HARC) Leh and Dr Muzzafar Ahmed, Member Disaster Management Committee of India, then Director Health Services Kashmir.

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