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View point Article

High altitude sickness: Physiological basis, Prevention and Treatment

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Abstract

High altitude sickness is a clinical condition that occur in non-acclimatized individuals after rapid ascent to high altitude (>2500 meter). The clinical manifestations of high altitude sickness usually appear within a few hours to two days of ascent and typically consists of headache accompanied by loss of appetite, nausea, vomiting, disturbed sleep, fatigue, and dizziness. Major risk factors include rate of ascent, maximum altitude achieved and sleeping altitude. The initial body response to reduced partial pressure of oxygen at high altitude is the increase ventilation. This is brought about by hypoxic stimulation of the peripheral chemoreceptors which sense the low PaO_2 in the arterial blood. As ventilation increases, CO_2 is washed out and $PaCO_2$ drops and blood becomes alkaline. Slow ascent is the key to prevent high altitude sickness and rapid descend is the most effective treatment measure. Oxygen therapy and acetazolamide (carbonic anhydrase inhibitors) might be effective for both prevention and treatment. The aim of this article is to discuss clinical manifestations, prevention, and treatment options on the basis of body responses to hypoxia at high altitude.

Keywords: Clinical manifestations; High altitude sickness; Hypoxia.

Declaration: There is no conflict of interest.

Introduction

High Altitude Sickness results from exposure to acute hypoxia when ascending to a higher altitude.[1] High altitude is generally considered more than 2500 meter height from the sea level. As a consequence of the increased altitude, the barometric pressure falls and the partial pressure of inspired oxygen decreases leading to tissue hypoxia. [2] The barometric pressure at sea level is 760 mm Hg, and it falls to half of this value at an altitude of 5,800 m on a typical mountain in the Himalayas or the Andes. The partial pressure of oxygen at the altitude of Everest Base Camp (5,300 meters) is about one-half of the sea level value. The summit of Mount Everest is the highest point above sea level on the earth's surface (8,850 meters), and has a partial pressure of oxygen about one-third of the sea-level value.[4]

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A subject on the summit of Mt. Everest tolerates close to six times more hypoxia than a sea-level resident. [1] The Classical physiological responses to high altitude hypoxia include hyperventilation, polycythemia, hypoxic pulmonary vasoconstriction, changes in oxygen affinity of hemoglobin, increases in oxidative enzymes, and increased concentration of capillaries in peripheral muscle.[3]

Acclimatization refers to the physiological changes that occur in the body in response to the hypoxia of high altitude, and it is often used as an example of how the humans can successfully adapt to a hostile environment.[3] A primary challenge for vertebrate life at high altitude is that the reduced partial pressure of O_2 that limits rates of aerobic metabolism, thereby compromising physiological performance capacities that affect survival and reproduction. Physiological adaptations that contribute to hypoxia tolerance could represent environmentally induced changes and genetically based changes.[5]

Clinical Manifestations of High Altitude Sickness

Common clinical manifestations of high altitude sickness include headache, anorexia, nausea, malaise, lack of energy, sleeplessness and occasionally vomiting. The symptoms often occur within few hours of ascent and become worst on the following two days. Mild to moderate form of high altitude sickness is usually self-limiting, and after 2 or 3 days the symptoms disappear.[3] Severe and potentially life threatening manifestations of high altitude sickness are High Altitude Pulmonary Edema (HAPE) and High Altitude Cerebral Edema (HACE).

Rate of ascent is an important factor determining the incidence of High Altitude Sickness. The incidence of the sickness in trekkers in Nepal who ascend to 3,800 m over 10 days is much smaller than for those who fly in.[3] Nocturnal Cheyne-Stokes breathing is a common experience when sleeping at high altitude. It results from the fluctuations of PaO_2 and $PaCO_2$ that are exaggerated during sleep, causing alternating periods of apnea and hyperpnoea.[2]

Some physiological differences have been observed between the high altitude dwellers of the Andes and sea level residents. The lung volume is increased in all dimensions at high altitude dwellers, the largest expansion being in functional residual capacity and residual volume. The significance of these differences in terms of adaptation is not known.[2] The breathing rate is increased at high altitude dwellers without a change in tidal volume. The consequent increase in alveolar ventilation reduces the oxygen pressure gradient from ambient air to alveolar air by 16 mm as compared to sea level and reduces alveolar pCO_2 to 30 mm.[3]

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Arterial blood of high altitude dwellers transports more oxygen than at sea level because of an increase in hemoglobin concentration.[5] The greatest protection against tissue hypoxia at high altitudes comes from the shape of the oxygen dissociation curve rather than from major changes in the respiratory system.[5] The curve is shifted towards the right side so that more oxygen is released from hemoglobin to the tissues.

High-altitude pulmonary edema (HAPE) is a potentially lethal form of high-altitude sickness. It is most often seen in people who ascend rapidly. A typical presentation of HAPE is persistent dry cough in an individual who has been at high altitude for 1 or 2 days. Over the course of a few hours, the patient becomes increasingly breathless. This is more noticeable at night when the patient is lying down. Initially the cough is dry but as it progresses it produces frothy white sputum. In severe cases, this becomes pink because of the presence of blood. On examination, crackles are heard particularly at bases of the lungs. Cyanosis, tachycardia and fever may develop. Chest radiograph and USG would show obvious changes of pulmonary edema. Patients who do not descend or get proper treatment may rapidly deteriorate and die. Pathophysiology is believed that the hypoxic pulmonary vasoconstriction is uneven and patchy in response to hypoxia. Non homogeneous vasoconstriction allows high pulmonary artery pressures to be transmitted to pulmonary capillaries in over perfused vessels leading to oedema. [4] Hypoxia inducible factor and nitric oxide synthase appear to have important roles in the activation of vascular endothelial growth factor aggravating the process of vasodilatation and capillary leakage. HAPE appears to be a direct pressure effect as there is no evidence of inflammatory mediators in early bronchoalveolar lavage.[4]

High-altitude cerebral edema (HACE) is uncommon but potentially lethal. It often begins as a severe form of high altitude sickness. HACE is usually characterized by unsteady gait, increased vomiting and gradual loss of consciousness. The patient develops headache that may not respond to analgesics. Symptoms might progress to ataxia, confusion, altered consciousness, coma, and sometimes death. On examination there is papilledema, extensor plantar responses, and occasional focal neurologic signs affecting cranial nerves. The pathophysiology of HACE is local vasodilatation due to hypoxia which leads to increased blood flow into the capillaries. Due to increased capillary pressure, fluid leaks into the cerebral tissues leading to cerebral oedema.[3]

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Treatment and prevention of High Altitude Sickness

Slow ascent is the most important measure to prevent high altitude sickness. A suggested strategy is 300 m a day above 3000 m with a rest day every 1000 m. Most important treatment for severe AMS or HACE or HAPE is descent to low altitude. Mild manifestations of high altitude sickness may be managed by resting at the same altitude.[4] Individuals suffering from headache and only moderate AMS (Lake Louise score between 6 and 9) can stay at altitude in general. Appropriate fluid intake, NSAIDs like paracetamol and antiemetics might be recommended. It is strictly advised against ascending to higher altitudes before symptoms have disappeared. If symptoms persist or become even aggravated, descent is mandatory. In severe AMS, descent should be performed passively (if possible), administering low-flow oxygen (2–4 L/min; if available) or using a hyperbaric bag (if available). Pharmacological treatment of AMS includes acetazolamide (a carbonic anhydrase inhibitor) and/or dexamethasone, particularly in more severe AMS. [6] Nifedipine might be good treatment option for HAPE. Mortality is also high for HAPE (about 50%) without option for descent or appropriate treatment. [6]

People with pre-existing cardiorespiratory diseases such as coronary heart disease, pulmonary hypertension, chronic pulmonary disease or obstructive sleep apnea should be assessed specifically by an expert before undergoing travel to high altitude. Physical preparation is essential to optimize aerobic capacity and endurance performance in the setting of high-altitude climbing because at high altitude VO2max declines significantly due to decreased oxygen level. Specific exercise is also needed to achieve adaptation of the musculoskeletal system especially when planning strenuous trekking or mountaineering trip. Thus, the amount, intensity and type of physical exercise depend on the kind of travel planned and the individual health status and fitness level. So, endurance training combined with core strengthening is recommended for high altitude climbers. Slow and graded ascent is the most common strategy used to prevent high altitude illness. It is commonly recommended not to exceed an ascent rate of 500 m per day to sleep at altitudes above 2,500 m.[6]

Hypoxia inducible factors (HIFs) functions as a major determining factor for the response of the body to oxygen deprivation. Genetic change is another exciting area of research as there is recent discovery of genetic changes in Tibetans.

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Important recent advances have been made on mitigation of the effects of the hypoxic environment. Oxygen enrichment of room air is very powerful mitigating factor for high altitude sickness. Every 1% increase in oxygen concentration reduces the equivalent altitude by about 300 m. This procedure is used in numerous facilities at high altitude and in a Chinese train to Lhasa. [3]

Conclusion

Effects of High Altitude might range from mild symptoms of high altitude sickness to severe form such as high altitude pulmonary oedema (HAPE) and high altitude cerebral oedema (HACE). Exposure to hypoxia in low landers can set a series of compensatory mechanism in the human body to increase altitude tolerance called acclimatization. As barometric pressure falls at higher altitude, partial pressure of individual gases also fall which leads to hypoxia. Incidence and severity of high altitude sickness is related to speed of ascent, maximum altitude achieved and duration spent at altitude. Main treatment measure for severe form of high altitude sickness is descent to low altitude as rapidly as possible. Oxygen, fluids and acetazolamide are also beneficial. Dexamethasone can be used in severe cases of altitude sickness. Altitude acclimatization is the important measure of prevention from high altitude sickness.

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