

Acute Mountain Sickness

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ABSTRACT

Acute Mountain Sickness (AMS) is a condition that can affect people ascending to higher altitudes. The aim of this review is to provide a brief overview of AMS to introduce medical students to the highly complex and interesting scenario involving the treatment of a patient at altitude. AMS presents with a number of symptoms including headaches, nausea and disturbed sleep. As the condition progresses it can lead to serious complications including both pulmonary and cerebral oedema and ultimately death. At altitude it can be both dangerous and problematic to treat and evacuation is usually a patient's best option. As well as addressing the common presentation and treatment of AMS there are a number of ethical considerations created by such a unique environment.

Introduction

Tens of thousands of people are attracted to high altitude environments around the world every year either to experience a unique landscape or to revel in the satisfaction of a successful summit attempt. Doctors working in or around these locations will encounter altitude illness in a variety of forms. Indeed as mountaineering, skiing and rock climbing continue to be popular activities the prevalence of AMS remains at a high level. Maggiorini *et al* studying climbers in the Swiss Alps found that at 3650m 34% of the climbers presented with three or more symptoms and signs of AMS¹. Although the focus of this review is AMS, patients in a high altitude and/or mountain environment may require care for various other potentially serious illnesses including hypothermia, frostbite, dehydration, snow blindness and trauma.



High altitude is an environment lacking in oxygen. Although the concentration of oxygen remains constant well beyond the height of Mt. Everest, the partial pressure of oxygen decreases at a rate proportional to the drop in barometric pressure. The barometric pressure at the height of Mt. Everest, 8848m, is a third of that at sea level and consequently the air contains only a third as much oxygen². Too rapid an ascent is very dangerous and is the main cause behind altitude illness. A period of acclimatisation is required to develop physiological adaptations that help the body

to cope with a high altitude environment and give us the best chance of preventing altitude illness.

There are acclimatisation limits to the human body and even the best acclimatisers cannot endure the extreme hypoxia of high altitude for long. The loss of appetite at even a modest altitude, the increasing fatigue on ascent and the delirium resulting from lack of oxygenation of the brain tissue all have a debilitating effect on the body. The cumulative effect of oxygen debt means that permanent habitation at extreme altitude is impossible, and climbers refer to areas over 8,000m as the Death Zone for good reason.

The physiological adaptation to high altitude, or acclimatisation, takes place by a number of processes within the body. Although some of these adaptations take place at a biochemical level shortly after the initial exposure to altitude, a number of 'struggle' responses, including the hypoxic ventilatory response and an increase in cardiac output, are the most important during early acclimatisation. The hypoxic ventilatory response is an increase in depth and rate of inhalation in response to a decrease in the oxygen saturation of the blood. This is detected by the carotid body and the medulla effects these changes². The increased level of ventilation leads to an increase in blood pH, known as respiratory alkalosis and a reduction in the carbon dioxide content of the blood. The respiratory alkalosis slows the initial hyperventilation as the body does not tolerate the alkalosis. The respiratory alkalosis results in a compensatory increase in renal excretion of bicarbonate and the pH returns towards normal enabling ventilation to further increase. This process that maximises ventilation is referred to as ventilatory acclimatisation and takes between 4 and 7 days at a given altitude. There are a number of other physiological processes involved in acclimatisation including the production of erythropoietin and changes to the pulmonary and cerebral circulations but these are outwith the scope of this article.

Acute Mountain Sickness (AMS)

Although it is well known that AMS is caused by hypobaric hypoxia the exact mechanism that results in the illness is unclear although the main risk factor is a rapid rate of ascent. A popular theory is that AMS is a subclinical high altitude cerebral oedema^{2,10}. The brain increases in size in all people ascending to altitude. The increase in cerebral blood flow and resulting increase in blood volume and intracranial pressure could provide explanation for the symptoms of mild AMS. In severe AMS and high altitude cerebral oedema (HACO) there is a leaky barrier between the blood and the brain, a result of increased vascular permeability as a physiological response to the hypoxia². The increased endothelial permeability is secondary to chemical mediators in the blood and allows fluid to move between compartments and accumulate in areas where it should not be. Intracranial pressure increases and presents with progressively worsening neurological symptoms varying in severity from disorientation to loss of memory, hallucinations and finally coma⁶.



The pulmonary form of high altitude sickness (HAPO) arises from regional pressure differences of the vascular bed within the lungs. The areas of vasoconstriction resulting from a maladaptive response to the hypoxia produce areas of high pressure within the blood vessels⁸. The high pressure within the capillaries results in a vascular leak into the alveoli and leads to pulmonary oedema.

The two most important risk factors for AMS are a high rate of ascent and a high sleeping altitude. Deficient or excessive physiological adaptations to altitude also predispose the patient to illness. These include excessive pulmonary vasoconstriction or too dramatic an increase in cerebral blood flow. It has been well documented⁷ that age, sex, previous exposure and physical fitness have no significant effect on the likelihood of an individual to develop AMS. However, previous long-term exposure, such as living at an intermediate altitude, bestows the individual with a greater ability to acclimatise to more extreme altitudes.

Clinical presentation of AMS

Headaches are the most common feature of AMS. These may or may not present with:

- gastrointestinal upset including nausea, vomiting and loss of appetite
- fatigue
- dizziness
- insomnia

Fluid retention is a classical sign of AMS, as opposed to the usual diuresis associated with acclimatisation, and may present with oliguria as well as peripheral and facial oedema². As the AMS worsens the patient will suffer from increasingly severe headaches⁶. Severe AMS is an increase in the severity of the above symptoms along with continuing decline in mental status. This commonly presents as an inability to conduct coordinated movements and loss of mental function¹⁰. Cerebral oedema leads to a further decrease in mental function (commonly ataxia, stupor and third and sixth cranial nerve palsies resulting from compression of brain structures because of raised intracranial pressure²) and if left untreated can result in coma. Comatose patients require additional care such as airway management and bladder drainage. Pulmonary oedema commonly presents with several of the following symptoms and signs:

- Shortness of breath at rest
- Productive cough
- Decreased exercise tolerance
- Chest tightness
- Crackles or wheezing
- Central cyanosis
- Tachypnoea
- Tachycardia

Pulse oximetry provides a useful diagnostic tool for HAPO though the normal ranges are highly dependent on altitude and are much lower than the normal range at sea

level. However oxygen saturations significantly lower than 75% at altitudes above 5500m are usually diagnostic of HAPO¹⁰.

Management of AMS

Prevention is always better than cure in AMS and risk factors must be carefully managed. One of the worst things any climber can do is ascend too rapidly without allowing their body time to acclimatise.

The only real cure for AMS is descent or acclimatisation. Mild AMS is usually self-resolving and the patient should recover given an extra 12 to 36 hours in which to acclimatise whilst ascent is halted². Acetazolamide is known to be effective in speeding acclimatisation and studies have shown it to be useful prophylactically¹¹⁻¹². The drug acts by stimulating ventilatory acclimatisation. A bicarbonate diuresis is induced thereby acidifying the blood and stimulating ventilation. As the patient acclimatises the symptoms will resolve. Peripheral paraesthesia and altered taste are common though benign side effects¹² but resolve when the medication is stopped.

In moderate to severe AMS immediate descent is required along with low flow oxygen if available. Acetazolamide and/or dexamethasone should be given. Dexamethasone is a glucocorticoid, commonly used to combat inflammation¹³ and is successful in treating the vasogenic oedema responsible for severe AMS and HACO. The drug acts to stabilise the blood brain barrier and reducing the leakiness of the vessels¹⁴. The use of acetazolamide to accelerate acclimatisation and a brief course of dexamethasone to treat AMS can be a successful combination². The treatment of HACO is the same as for severe AMS: oxygen, descent and dexamethasone¹⁰. HACO is a life threatening condition and can result in death within hours if the patient does not descend. A rapid descent can be life saving and delay can result in a slightly confused, ataxic patient becoming comatose and unable to walk at all.

As acetazolamide acts by accelerating acclimatisation there is no risk of masking serious symptoms and as such patients can continue to ascend whilst on the drug after ensuring they are symptom free. It does not prevent worsening illness if the patient continues to ascend while symptoms are present¹⁰. However dexamethasone treats the pathology and so patients should not continue to climb until they are symptom free off the steroid. Dexamethasone should never be taken during ascent, it does not promote acclimatisation and rebound symptoms can occur when it is discontinued².

In a situation where descent is impossible a hyperbaric bag can be used to simulate descent. This is a portable person sized bag that is pressurised using a foot pump. It simulates the conditions of lower altitude in order to buy time until descent or treatment becomes available¹⁶. A combination of simulated descent and dexamethasone is the treatment of choice in a situation where the patient cannot be transported to a lower altitude¹⁵. Immediate descent and oxygen is the optimal treatment in pulmonary oedema at altitude². As for AMS and HACO the hyperbaric bag can be a



A hyperbaric bag into which the patient is placed in order to simulate descent.¹³

useful addition when immediate descent is not possible.

Ethical considerations

Denial of symptoms is extremely common among climbers who have invested a large amount of money and time in their pursuit. The possibility of not succeeding is always hard to accept on a once in a lifetime trip to climb any of the big mountains. This can result in other climber's lives being put at risk and it is the doctor's responsibility to detect symptoms early and prevent severe illness if possible. However a balance between patient-doctor confidentiality and group safety must be sought. Certainly the climber in question should be consulted before anyone else is notified.

The high altitude environment is not easily accessible to other doctors or medical supplies and many mountain teams are without a doctor. This presents issues in terms of medical resource allocation. A doctor should never refuse to treat someone, but resources must also be used in the most efficient way.

There are a number of ethical pressures exerted upon climbers by the mountaineering community itself. Although the use of bottled oxygen is commonplace at high altitude a core group of climbers would possibly view bottled oxygen as 'cheating' and would most definitely think of prophylactic acetazolamide in the same manner. A climber using prophylactic medication would definitely be viewed negatively alongside one who was not.

Conclusion

Acute mountain sickness can be life threatening if not managed carefully. However by ensuring climbers are aware of the risks and consequences and providing treatment when necessary, dangers can be mitigated and climbers protected. Even with the implementation of these measures the high altitude environment is extremely dangerous. The hypoxia, low temperatures and dangerous surroundings all endanger the lives of mountaineers. However climbers continue to expose themselves to these risks. Whether this be for personal achievement, respect or for a host of other reasons known only to the individual, people will always strive to perform at and beyond the limit of their capabilities and doctors will continue to have a place in the provision of care at altitude.

References

1. Maggiorini M, Buhler B, Walter M, Oelz O. Prevalence of acute mountain sickness in the Swiss Alps. *BMJ*. 1990; 301: 853-5
2. Tintinalli JE, Kelen GD, Stapczynski JS. *Emergency Medicine: A Comprehensive Study Guide* 6th Edition. United States of America: The McGraw-Hill Companies; 2004. p. 1263-1271.
3. The Travel Doctor. Altitude or Mountain Sickness. c2001 [updated 2011; cited 2011 Feb 23]. Available from: <http://www.traveldoctor.co.uk/altitude.htm>
4. Milledge JS. Acute mountain sickness. *Thorax*. 1983; 38: 641-645
5. Ward MP, Milledge JS, West JB. *High Altitude Medicine and Physiology* 3rd Edition. London: Arnold; 2000.
6. Thomas E. Dietz, MD. AMS Worksheet. c2000 [updated 2000 May 8; cited 2011 Feb 28]. Available from: <http://www.high-altitude-medicine.com/AMS-worksheet.html>

7. Thomas E. Dietz, MD. The High Altitude Medicine Guide: Altitude Illness Clinical Guide For Physicians . c2000 [updated 2000 May 8; cited 2011 Feb 28]. Available from: <http://www.high-altitude-medicine.com/AMS-medical.html#AMS>
8. Greene MK, Kerr AM, McIntosh IB, Prescott RJ. Acetazolamide in prevention of acute mountain sickness: a double-blind controlled cross-over study. *British Medical Journal*.1981; 283: 811-813.
9. Frank J. Current Prevention and Management of Acute Mountain Sickness. *The Yale journal of biology and medicine*. 1992; 65: 337-341.
10. Selma TP, Beizer JL, Higbee MD eds. *Geriatric Dosage Handbook: Including Clinical Recommendations and Monitoring Guidelines [Dexamethosone]*. 12th ed. Hudson, Ohio: Lexi-Comp; 2007
11. Khan E. Understanding the use of corticosteroids in managing cerebral oedema. *British Journal of Neuroscience Nursing*. 2008; 4 (4): 156-162.
12. Keller HR, Maggiorini M, Bartsch P, Oelz O. *Simulated descent v dexamethasone in treatment of acute mountain sickness: a randomized trial*. *BMJ*. 1995; 310: 1232-1235.
- Image 13. Wikipedia. Gamow Bag. c2003 [updated 2010 Feb 4; cited 2011 Mar 29]. Available from: http://en.wikipedia.org/wiki/Gamow_bag
- Image 14. Alexandre Buisse. UKclimbing.com [updated 2010 Feb 4; cited 2011 Mar 31]. Available from: <http://www.ukclimbing.com/articles/page.php?id=2413>
- Image 15. *Everest ER: Himalayan rescue association*. [cited 2011 Mar 31]. Available from: <http://www.everester.org/AboutUs.aspx>
16. *Climbing high: The climbing Guide* Available from: <http://www.climbing-high.com/the-gamow-bag.htm>