

Editorial

 Altitude-Related Illnesses[☆]


Enfermedades por exposición a la altura

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Recent years have seen an increase in tourism, work, and recreational activities at high altitude, defined as heights greater than 2000 meters above sea level (MASL). Barometric pressure decreases with altitude, as does oxygen pressure, due to the constant proportion of oxygen in atmospheric air; therefore, the higher the altitude, the lower the pressure of inspired oxygen. A series of physiological responses help individuals from lower altitudes tolerate and adapt to low oxygen conditions. However not everyone develops these adaptive responses, and some individuals may present acute mountain sickness (AMS), cerebral edema and/or high-altitude pulmonary edema.¹

But why do these phenomena occur? Hypoxia activates genes in the lungs that use various signaling pathways to produce proinflammatory cytokines and chemokines that promote the migration of transendothelial cells, exerting a potent proangiogenic effect. Local alveolar inflammation develops, releasing other molecules into the bloodstream and leading to systemic inflammation.² The inflamed tissue becomes severely hypoxic, when the diffusion distance between the capillaries and the metabolically active cells increases, in turn causing more inflammation.³ Vasodilation is observed in these tissues, but vasoconstriction develops in the lung: initially this improves the ventilation/perfusion ratio, but as it persists, it causes an increase in pulmonary pressure that may eventually lead to pulmonary edema and/or an increase in blood flow to the brain, causing vasogenic cerebral edema.⁴ The expression of these molecular and inflammatory processes – not yet fully understood – varies according to genetic, which determine the variability in the response to hypobaric hypoxia.⁵

An understanding of the physiopathology of acute high-altitude illness and a study of the factors associated with its development, such as speed of ascent, height of ascent, underlying diseases, and cold conditions, are necessary for diagnosis and appropriate management. There is no accurate way of diagnosing AMS, also known as “soroche”, because the signs and symptoms are atypical.⁶ The cardinal symptom is headache, an indication of incipient cerebral

edema. AMS is accompanied by sleep disorders, fatigue, dizziness, anorexia and nausea.⁷ In some cases, cough and dyspnea also present as signs of pulmonary edema. AMS generally develops between 4 and 24 h after ascending to a new altitude, and generally resolves within 2–3 days if the individual remains at the same altitude, with symptoms disappearing as adaptation progresses. Symptomatic treatment, such as acetaminophen and/or dimenhydrinate may be necessary in mild cases, while moderate cases may require acetazolamide, which stimulates respiration by causing metabolic acidosis, and/or steroids for their anti-inflammatory effect. In severe cases, these medications should be administered along with supplemental oxygen to correct hypoxia.⁸ If the AMS improves or resolves, the patient may remain at altitude. However, if high-altitude pulmonary or cerebral edema occur, treatment is required, and the patient must descend at least 500–1000 m, or preferably to sea level.

It might be assumed that altitude sickness occurs only in cases of acute exposure, but natives living at high altitudes and individuals born at lower levels but who have adapted to hypobaric hypoxia may lose this adaptation and develop chronic mountain sickness (CMS) or Monge’s disease, and subacute mountain sickness. For decades it was thought that chronically exposed individuals adapted to altitude by developing polycythemia. However, this response is observed only among natives of the Andes, but not among Tibetans and Ethiopians, the other 2 populations who having been living for centuries at high altitudes.⁹ Both ethnic groups are chronically adapted to altitude without developing polycythemia. In Andean natives the ventilatory response to hypobaric hypoxia is absent, so they persist hypoxic by not increasing their respiratory rate. This increases erythropoietin production and/or reduces production of its soluble receptor, causing polycythemia.¹⁰ CMS is diagnosed using the Qinghai score, with 5 or more points indicating presence of the disease. In this scale, excessive erythrocytosis (hemoglobin >21 g/dl in men and >19 g/dl in women) scores 3 points, while breathlessness and/or palpitations, sleep disturbances, cyanosis, dilation of veins, paresthesia, headache, and tinnitus are scored on a scale of 0–3.¹¹ This disease is more common among adult males who live at more than 4000 MASL. Overproduction of red blood cells in response to sustained hypoxia, usually associated with a lung problem, causes the symptoms of CMS. In contrast to CMS, subacute mountain sickness is a cardiovascular

[☆] Please cite this article as: Accinelli RA, López LM. Enfermedades por exposición a la altura. Arch Bronconeumol. 2018;54:115–116.

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disease that begins with pulmonary arteriolar vasoconstriction in response to hypoxia, leading to pulmonary hypertension, cor pulmonale, and, ultimately, heart failure. It occurs in healthy children born at a low altitudes who between the first and the six month of age are moved to live at high altitude (infantile form), and in young people from sea level areas who have resided for several months performing physical activity in extreme altitudes, being the first cases described in people residing between 5800 and 6700 MASL (adult form).¹²

Although carriers of all of these diseases should receive specific treatment for their symptoms, such as emergency phlebotomy for CMS, and oxygen, digitalis, and diuretics for subacute mountain sickness, the definitive treatment for both is descent and residence in lower altitudes.

In conclusion, physicians must understand the physiological changes that occur when a person is exposed to altitude for a few days or for longer periods, and must take into account the diagnosis and treatment of diseases associated with hypobaric hypoxia.

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