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High altitude pulmonary edema in mountain climbers

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ABSTRACT

Every year thousands of ski, trekking or climbing fans travel to the mountains where they stay at the altitude of more than 2500–3000 m above sea level or climb mountain peaks, often exceeding 7000–8000 m. High mountain climbers are at a serious risk from the effects of adverse environmental conditions prevailing at higher elevations. They may experience health problems resulting from hypotension, hypoxia or exposure to low temperatures; the severity of those conditions is largely dependent on elevation, time of exposure as well as the rate of ascent and descent. A disease which poses a direct threat to the lives of mountain climbers is high altitude pulmonary edema (HAPE). It is a non-cardiogenic pulmonary edema which typically occurs in rapidly climbing unacclimatized lowlanders usually within 2–4 days of ascent above 2500–3000 m. It is the most common cause of death resulting from the exposure to high altitude. The risk of HAPE rises with increased altitude and faster ascent. HAPE incidence ranges from an estimated 0.01% to 15.5%. Climbers with a previous history of HAPE, who ascent rapidly above 4500 m have a 60% chance of illness recurrence. The aim of this article was to present the relevant details concerning epidemiology, pathophysiology, clinical symptoms, prevention, and treatment of high altitude pulmonary edema among climbers in the mountain environment.

1. Introduction

More than 140 million people worldwide, mainly from Asia, North, Central and South America live in regions lying 2400 m above sea level, i.e. in high-mountain areas (Bhagi et al., 2014). A substantial number of people live permanently in low-land areas but work at higher altitudes, e.g. in astronomical observatories (Hawaii, 4200 m) or in mines (The Andes, 4500 m) (Richalet, 1995). Hostilities along the Indian and Pakistani border have been conducted for many years at an altitude of 5000 m above sea level (Singh and Roy, 1969) and the international forces military operation in Afghanistan is often carried out at the elevation exceeding 3000 m above sea level (Korzeniewski, 2008). Every year thousands of ski, trekking or climbing fans travel to the mountains where they go skiing at an altitude of more than 2500–3000 m, or climb summits exceeding 7000–8000 m. All of these people are, either temporarily or permanently, exposed to environmental conditions characterized by low air pressure, low oxygen concentration, low ambient temperature and increased solar radiation. The lack of basic knowledge on the risk factors prevailing in high-mountain areas may have disastrous consequences which can be potentially health- or life-threatening. High mountain climbers are at a serious risk from the effects of adverse environmental conditions prevailing at higher elevations. The illnesses they experience may be classified into four categories: diseases resulting from hypotension and hypoxia, diseases resulting from the exposure to low temperatures, diseases associated with the effects of solar radiation, exacerbation of previously asymptomatic medical conditions. Illnesses associated with hypotension and hypoxia form a category of diseases which are particularly dangerous and potentially deadly for mountain climbers. They cover a wide range of medical conditions from mild to life-threatening ones (Korzeniewski, 2014). The severity of these illnesses is largely dependent on elevation (the higher the altitude, the lower the air pressure and oxygen concentration), time of exposure to high mountain conditions (hours, days, weeks) as well as the rate of ascent and descent. Additionally, the intensity of exertion, psychophysical condition of a climber, age as well as co-existing health problems contribute to the development of pathological

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changes. Acute mountain sickness (AMS), a condition associated with the effects of low air pressure and low oxygen concentration, is the most common health problem in mountain climbers. AMS presents as a collection of different symptoms (headache, nausea, vomiting, general malaise, fatigue) and typically affects unacclimatized lowlanders who have quickly changed elevation. It needs to be pointed out that high fitness level does not prevent the onset of AMS (Advisory Committe Statement, 2007). The symptoms usually appear 3–24 h after ascent above 1800 m in less than 24 h and subside 3–7 days following the arrival at higher altitudes. The symptoms may reappear (even after undergoing acclimatization) any time the climber quickly changes the elevation. 10–20% of people who have climbed to 1800–2400 m in less than 24 h develop the symptoms of AMS. The majority of climbers who ascended to a height of 3600–4300 m suffer mild symptoms of AMS, 50% experience moderate symptoms while 12–18% may develop a severe form of AMS. A rapid ascent to the altitude of over 5300 m produces serious pathological symptoms in the majority of climbers. The disease can affect both those who climbed from low to high altitude within a short period of time, as well as those who, being at high altitude, climbed even higher. Thus, the cause of AMS is not high altitude itself, but rather rapid changes in elevation within a short period of time (Korzeniewski, 2014). In the great majority of cases AMS is a minor affliction resolving in a few days. However in a small proportion of people (2–3%) going to high altitude there develops the potentially lethal condition of high altitude cerebral or pulmonary edema or a mixture of these two (Milledge, 2006).

High altitude pulmonary edema (HAPE) is yet another disease resulting from hypotension and hypoxia. HAPE is a life-threatening non-cardiogenic pulmonary edema which typically occurs in lowlanders who ascend rapidly to altitudes above 2500–3000 m (Paralikar, 2012). It is the most common cause of death resulting from the exposure to high altitude (Pennardt, 2013). High altitude pulmonary edema manifests in two forms. It can occur in mountaineers returning from a sojourn at a low altitude, also known as "reentry" HAPE, and in rapidly climbing unacclimatized lowlanders. It usually occurs within 2–4 days of ascent above 2500–3000 m (Hackett and Roach, 2001). The illness rarely occurs after more than 4 or 5 days at the same altitude, most likely because of remodeling and adaptation (West and Mathieu-Costello, 1999).

The aim of this article was to present the relevant details concerning epidemiology, pathophysiology, clinical symptoms, prevention, and treatment of HAPE in mountain climbers.

2. Epidemiology

The prevalence of high altitude pulmonary edema depends on the degree of mountaineers' susceptibility, the rate of ascent, and the final altitude (Bärtsch et al., 2005). In 1970s, the incidence of HAPE was reported in 3% of adults trekking in Peru at 3782 m (Hultgren and Marticorena, 1978). Sophocles (1986) estimated the incidence of HAPE in visitors to ski resorts in the Rocky Mountains, Colorado (2500 m) to be 0.01–0.1%. His study also reported that men are more susceptible to HAPE as compared with women. A lower incidence of pulmonary edema in women was also reported in the study of Hultgren et al. (1996), where 84% of the HAPE patients at an altitude 2500 m were men as compared with women. In the same study, the average arterial oxygen saturation in patients with HAPE was 74% as compared with a normal average oxygen saturation of 92% at such altitude, showing the occurrence of hypoxemia in HAPE patients.

At an altitude of 4500 m, the prevalence of HAPE may vary, depending on the rate of ascent, between 0.2 and 6% in an unselected population (Bärtsch et al., 2002) and at 5500 m between 2 and 15% (Hackett et al., 1976). The reported incidence of HAPE ranges from an estimated 0.01% of skiers traveling from low altitude to 2500 m, to 15.5% of Indian soldiers rapidly climbing to altitudes of 5940 m (Hackett and Roach, 2012). According to Hochstrasser et al. (1986), the prevalence of HAPE in a general mountaineering population in Alps is <0.2% (Hochstrasser et al., 1986). Among trekkers in the Himalayas (Maggiorini et al., 1990) and climbers in the Alps (Hackett and Rennie, 1979) ascending at a rate >600 m/day HAPE incidence is around 4%. The risk of HAPE rises with increased altitude and faster ascent. The incidence among persons with an unknown history of high-altitude pulmonary edema is 0.2% if they ascend to 4500 m in 4 days and 2% if they ascend to 5500 m in 7 days. The incidence increases to 6% and 15%, respectively, when these altitudes are reached within 1–2 days (Bärtsch and Swenson, 2013).

Climbers with a previous history of HAPE, who ascend rapidly above 4500 m, have a 60% chance of HAPE recurrence (Bärtsch et al., 2002). Susceptible individuals can avoid HAPE if they ascend slowly with an average gain of altitude not exceeding 300–350 m/day above an altitude of 2500 m (Bärtsch et al., 2003). When an altitude of 4559 m is reached within 22 h, the HAPE incidence increases to 7% in mountaineers without, and to 62% in those with a history of radiographically documented pulmonary edema (Bärtsch et al., 2002). Susceptibility to HAPE may increase in mountaineers with unrecognized underlying illness; some individuals may have developed diastolic heart failure rather than HAPE, which was due to hypertensive heart disease (Bärtsch et al., 2003).

The HAPE mortality rates in previous years varied between 4% (Menon, 1965) and 11% (Lobenhoffer et al., 1982), depending on the rate of descent or oxygen treatment. HAPE had a reported mortality of 44% if untreated, compared with 6% among those who receive supplementary oxygen, descend to a lower altitude, or both (Lobenhoffer et al., 1982). Nowadays, largely due to a spectacular increase in the number of skiing, trekking and climbing tours, HAPE mortality rates in places where there is limited access to medical care can even reach 50% (Bärtsch and Swenson, 2013).

3. Pathophysiology

There are two typical settings for high altitude pulmonary edema. The first setting involves high-altitude inhabitants returning from sojourns at low altitude, while the second includes unacclimatized lowlanders rapidly ascending to high altitudes. The two forms most likely share the same pathophysiology (Bärtsch et al., 2003). HAPE is associated with pulmonary hypertension and elevated capillary pressure (Maggiorini et al., 2001).

3.1. Hypoxic pulmonary vasoconstriction

Mechanisms of high-altitude pulmonary edema include uneven hypoxic pulmonary vasoconstriction (HPV) that exposes pulmonary capillaries to high pressure, damaging their walls and leading to a high-permeability form of edema (Bhagi et al., 2014). The classical concept of a characteristic increase in HPV is proposed as an important pathogenic factor in the development of HAPE (Hackett et al., 1992; Scherrer et al., 1996). Alveolar hypoxia leads to an adaptive vasomotor response in the form of HPV, which is non-homogenous in nature (Bhagi et al., 2014). The pulmonary capillary pressure increases as a result of HPV, which occurs mainly in smaller pulmonary arteries (Archer and Michelakis, 2002). Recent studies strongly suggest non-uniform regional hypoxic arteriolar vasoconstriction as an explanation for how HPV occurring predominantly at the arteriolar level causes leakage. In areas of high blood flow due to lesser HPV, edema develops due to pressures that exceed the dynamic and structural capacity of the alveolar capillary barrier to maintain normal fluid balance (Swenson and Bärtsch,