CASE REPORTS

HIGH ALTITUDE CEREBRAL AND PULMONARY EDEMA IN AN ACCLIMATIZED SOLDIER WITH NO CLASSIC SYMPTOMS – A CASE REPORT

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ABSTRACT

Acute mountain sickness (AMS) and high altitude cerebral edema (HACE) are considered to represent two points along a single spectrum of disease, with the same underlying pathophysiology. Onset of AMS is usually delayed for 6 to 12 hours following arrival at high altitude, but can occur as rapidly as one to two hours or as late as 24 hours. HACE generally occurs in individuals with AMS and/or high altitude pulmonary edema (HAPE) at elevations over 3000 to 3500 m (9,500 ft.). The hallmarks of HACE are encephalopathic symptoms and signs, including ataxic gait, severe lassitude, and progressive decline of mental function and consciousness (irritability, confusion, impaired mentation, drowsiness, stupor, and finally coma). The onset of encephalopathy and ataxia signifies the transition from AMS to HACE and occurs unpredictably, requiring as long as three days or as little as 12 hours. The patient, a well acclimatized young and fit serving soldier remained asymptomatic for over a month after he climbed gradually to a height of around 15,000 ft from a height of 8,000 ft as per acclimatization protocol. He did not develop classic encephalopathic signs/symptoms of HACE or HAPE as mentioned above. After spending two weeks on the post, he just once recalled the names of his relatives loudly and showed slightly withdrawn behavior from his daily routine. His vital signs and clinical examination was normal and in a very short course of a few hours he developed cardiac arrest. An autopsy performed later on, revealed edema on gross and microscopic examination of the brain and both lung tissues, characteristic of both HACE and HAPE respectively.

Keywords: Acute mountain illness, High altitude cerebral edema, High altitude pulmonary edema.

INTRODUCTION

Anyone who travels to high altitude is at risk of developing high altitude illness. Acute mountain sickness (AMS) and high altitude cerebral edema (HACE) represent a continuum of one form of such illness. AMS and HACE are generally considered to represent two points along a single spectrum of disease, the concept that AMS/HACE represents a continuum, and that AMS can progress to fatal HACE, fits with clinical experience and is helpful for management. Cerebral edema is consistently found in neuroimaging and at autopsy in patients with severe AMS or HACE[1-3]. MRI studies reveal reversible vasogenic brain edema, with characteristic T2 signal increase in the splenium of the corpus callosum and subcortical white matter. These findings indicate increased blood-brain barrier (BBB) permeability. Increased cerebral blood flow and the loss of autoregulation of intracranial pressure may contribute to such an increase. Chemical factors (e.g. vascular endothelial growth factor, nitric oxide, cytokines) may also play a role by altering endothelial permeability[4,5].

HAPE/HACE in well acclimatized individuals is not very well reported in the literature. As HACE is generally taken as a continuum rather than an individual entity, thus HAPE/HACE without any signs of AMS and in well acclimatized individuals is a rarity.

CASE REPORT

A 30 year old well built soldier, previously fit and healthy was posted to the Northern areas.

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Received: 18 May 2016; revised received: 29 Sep 2016 accepted: 30 Sep 2016

He reached 8,000 feet height on 9th December 2015, where he spent almost 9 days. His medical checkup was done before further ascent. He was found to be medically fit with normal vital signs and basic investigations including ECG and basic labs. On 17th Dec 2016, he was sent to a height of 10,500 ft. He remained asymptomatic and did not show any signs of acute mountain sickness. At this height, he stayed for almost 14 days. During this time, his medical examination was done again and he was found to be fit in every regard. On 31st December, he moved to a height of 14,000 ft., where again he did not show any signs of acute mountain sickness. He stayed at this height for about 2 days, then he climbed up to a height of 15,000 ft. on 2nd Jan 2016. On reaching his final destination of duty, he was asymptomatic with normal vital signs and clinical examination. The soldier remained well on the post. He took part in routine activities. He performed his duties up to the mark and engaged himself in leisure activities. He was social and well conversant. While on the post, his medical examination including vital signs was done on daily basis by a paramedic employed on the post as a routine protocol being followed on the forward posts. On 16th January at 0930 hours, after he got up from sleep, he recalled the names of his wife and mother loudly, which was a little unusual for him as he never did such a thing before. The medic on the post took his vital signs which were normal and clinical examination done by the paramedic at the post was also normal and later the Medical Officer at the base, himself, spoke to him. He was fully conscious and oriented. He took his breakfast at 1000 hours. He was a little withdrawn as he did not play board games (Ludo) with them. At 1300 hours, he told his post fellows that he was a little tired and needed some rest. He went to his room and slept for a while. At 1530 hours, his post fellows went inside the room to check him, they found him unresponsive. Immediate cardiopulmonary resuscitation (CPR) was started by the paramedic staff posted there and it was continued for about an hour; however, he could not be revived.

An autopsy performed later showed edematous lungs and brains on gross examination and microscopic examination confirmed the presence of edema in the brain and lungs characteristic of the pattern found in both HACE and HAPE.

DISCUSSION

Acute mountain sickness (AMS) is by far the most common high altitude illness (HAI). As with all HAI, the risk of AMS depends upon individual susceptibility, the elevation reached, and the rate of ascent. Thus, while AMS is uncommon below 2000 m (6500 ft.), it is quite common (approximately 25 percent incidence) at elevations between 2000 and 3000 m. AMS occurs in both genders and at all ages, although the incidence decreases among those 50 to 60 years or older[6-8]. Neither youth nor physical fitness confers protection against AMS. Obesity, heavy exertion upon arrival at altitude, and residence at low altitude prior to ascent all appear to increase the risk[9].

The incidence of HACE is reported to be 0.1 to 2 percent at elevations in excess of 3000 to 4000 m (9800 to 13,000 ft.), although HACE has been reported at altitudes as low as 2100m. HACE is often complicated by concomitant HAPE. In fact, pure cerebral edema without pulmonary edema appears to be uncommon[6].

Hallmarks of HACE are encephalopathic symptoms and signs, including ataxic gait severe lassitude, and progressive decline of mental function and consciousness (irritability, confusion, impaired mentation, drowsiness, stupor, and finally coma). Signs of abnormal coordination, such as impaired performance of finger-to-nose and heel-to-shin testing, may be present. Focal neurologic findings, such as hemiparesis, slurred speech, or a discrete visual deficit, may rarely develop but are not typical and should raise concern for an alternative diagnosis, such as ischemic stroke, intracranial hemorrhage, or hypoglycemia. The onset of general neurological signs (encephalopathic signs

as mentioned above) signifies the transition from AMS to HACE. This transition can occur unpredictably and may require as long as three days or as little as 12 hours. HACE develops faster in patients with HAPE, most likely as a result of severe hypoxemia[6]. Patients with HAPE manifest pulmonary findings, such as crackles. Early symptoms of HACE may be missed or mistaken for exhaustion. Lethargy and irritability may manifest initially as diminished climbing performance, lack of participation in group activities, and the desire to be left alone. Even ataxia, the earliest physical sign of HACE, may be missed if the patient is lying in a tent, insisting that he is well and simply wants to be left alone. Dexamethasone is generally well tolerated and used in the management of both HAPE / HACE[10].

A working diagnosis of HACE can be made and treatment started in any patient with a history of recent ascent (especially above 3500m) and signs of encephalopathy. A high index of suspicion should always be kept in mind as this disease is rapidly fatal as happened in our patient. After descent, the clinician can reconsider the differential diagnosis[4].

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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