Acute mountain sickness

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Abstrak

Acute mountain sickness merupakan sindrom klinis yang berkaitan dengan ketinggian. Dikenal 2 bentuk, yakni HAPE (high altitude pulmonary edema) dan HACE (high altitude cerebral edema). Keadaan ini terjadi akibat pajanan mendadak terhadap ketinggian, biasanya lebih dari 8000 kaki. Meskipun biasanya kedua jenis kelainan ini sembuh dengan sendirinya, namun dalam keadaan tertentu dapat mengancam jiwa. Patofisiologi kelainan ini belum diketahui dengan pasti. Tindakan yang harus dilakukan untuk menolong pasien adalah menurunkan ketinggian, sedangkan tindakan lain masih kontroversial. Tirah baring, pemberian oksigen, serta penggunaan morfin, diuretik, steroid, atau nifedipin secara hati-hati banyak dianjurkan oleh para ahli. ***(Med J Indones 2001; 10: 115-20)***

Abstract

Acute mountain sickness, HAPE (high altitude pulmonary edema) and HACE (high altitude cerebral edema) are associated with acute exposure to altitudes greater than 8000 ft. Although usually self limiting, they can be life threatening. We are not yet clear about the pathophysiological processes in acute mountain sickness. Descent to lower elevation is the definitive treatment for altitude illness. There is no unanimity of opinion regarding other modes of therapy. Treatment consists of bed rest, oxygen inhalation and judicious use of morphine, diuretics, steroids and nifedipine as vasodilator therapy. ***(Med J Indones 2001; 10: 115-20)***

Keywords: *Acute mountain sickness, high altitude pulmonary edema, high altitude cerebral edema*

Some 568 Himalayan peaks are higher than 6500 meters and excluding Antarctica about 25% of world's land surface lies above 3050 meters. Mountains and high plateaus are home to over 300 million people[1] and at least half of this number live above 2400 meters. Acute mountain sickness should be considered as benign or malignant with the later comprising pulmonary, cerebral and mixed forms. Benign (mild) acute mountain sickness is common in low landers going to high altitude and presents no physical signs, although those affected show a characteristic facies manifesting their malaise. It requires little or no treatment. In contrast, the serious or malignant form is potentially life threatening and immediate descent to low altitude is mandatory. It occurs in two major types, one involving the lungs called "high altitude pulmonary edema" hape and other affecting brain called "high altitude cerebral edema" or hace. So benign acute mountain sickness, high altitude pulmonary edema and high altitude cerebral edema are separate but related disorders.[2]

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Subacute infantile mountain sickness is a recently recognized illness in infants, who were born at low altitude and subsequently taken to live at high altitude (3600 m), in Lhasa (Tibet). This condition which develops rapidly and is usually fatal within a few months, is characterized by congestive heart failure, with extreme medial hypertrophy of muscular pulmonary arterioles and the appearance of new muscle in pulmonary arterioles.[3]

Adult subacute mountain sickness is a newly described syndrome affecting previously healthy young men exposed to hypobaric hypoxia and a cold environment at very high altitude (5800-6700 m) for up to six months. This is characterised by slowly progressive congestive heart failure with generalized edema which resolves rapidly without any treatment, on transfer to sea level.[4]

HISTORY

Acute mountain sickness was known to the Chinese in ancient times as they traversed mountain passes between great headache and little headache mountains in the present day Afghanistan. According to Gilbert,[5] Too Kin (37 to 32 B.C.) a government official during reign of emperor Chung Ti described big headache and

little headache mountains, which were perhaps Ulugh Rabet Pass (4200 m) in China and Kilik pass (4830 m) in Karakoram along silk routes form China to present day Afghanistan. About A.D. 400, Fa Hsien,[6] a Chinese monk travelling in Kashmir, lost his companion who died after foaming from his mouth, while they were ascending a high mountain pass in Kashmir. The Jesuit priest Father Joseph Acosta, lived in Peru during sixteenth century, he described both this syndrome and deaths which occurred in the high Andes. Houstan[8] first described HAPE in modern english literature.

IMPORTANCE OF HIGH ALTITUDE STUDIES

The number of tourists visiting high mountain areas is increasing explosively and it seems undisputed that thousands more people will suffer from acute mountain sickness every year, with increasing mortality.

Introduction in recent years of trekking and "adventure holidays" in remote mountainous areas raises the likelihood of tourists being suddenly exposed to the hypoxia of high altitude after rapid transit from sea level. There is now a substantial tourist industry based on the Andean empire of Incas, centered in it's capital cuzco (3400 m) and the lost city of Incas, Majchu Picchu (2440 m). In view of increasing number of tourists and civilians venturing to mountainous areas due to their work, it is imperative that professionals dealing with health care be aware of this entity.

MILITARY OPERATIONS

From accounts of battle of Jumin (4400 m) in 1824 it would appear that some of the Spanish troops rapidly brought up from the coast to fight the native highlanders frothed at the mouth, almost certainly due to HAPE and died. These early military lessons in the powerful influence of high altitude in the conduct of battles were lost on the Indian Government in 1962 during Chinese incursions of its Himalayan borders. Large number of Indian soldiers were flown to the airstrip at Leh (3500 m) in Ladakh and provided the world medical literature with largest series of cases of acute mountain sickness ever recorded, with much serious illness and many fatalities.[9]

PREDISPOSING FACTORS

The altitude at which the risk of developing edema of the lung begins has been reported as about 3350 m in the Himalayas,[9] 3660 m in the Andes and some what lower (2590 m) in the Rockies. The condition commonly afflict the unacclimatized subject exposed to diminished barometric pressure who engages too quickly in strenuous physical exercision arrival. Previous acclimatization will not necessarily be against the development of the condition[9] and ascent for as little as 300 m may induce it. It also occurs on reentry to high altitude by those already living at such elevations after a stay at lower altitude.

INCIDENCE

Hackett et al[10] found overall incidence of acute mountain sickness 53% in men and 51% in women. Of the thousands of climbers making rapid ascent to the summit of Mt Rainier (4392 m) every year, one half to three quarter suffer from acute mountain sickness.[11] Incidence of malignant forms of acute mountain sickness was 4.3% in a series by Hackett et al[10] and 8.3% in large series of Indian Soldiers reported by Singh et al.[12] All ages are susceptible to the condition. Physical fitness offers no protection against the development of acute mountain sickness.

PATHOPHYSIOLOGY

Acute mountains sickness is not of immediate onset and is not directly related to hypobaric hypoxia. It takes several hours to develop and the nature of the mechanism of its development while initiated by hypoxia is still not clear. The normal response to altitude seems to be a mild diuresis, where as subjects destined to get AMS have an antidiuresis. Some symptoms develop in man immediately on his ascent to the mountains, they are the direct consequences of hypobaric hypoxia and may be regarded as the physiological components of early acclimatization. These adjustments are normal and harmless, but they give rise to unusual bodily sensations that may disturb the timid. Current thinking favours the hypothesis that hypoxia causes some alteration of fluid or electrolyte homeostasis with either water retention and/or shift of water from intracellular to extracellular compartments.[13,14]

This increase in extracellular fluid in turn results in the dependent and periorbital edema often seen in patients with acute mountain sickness.[15] It also causes mild cerebral edema, resulting in the symptoms of AMS. More severe cerebral edema causes full blown malignant condition of acute cerebral edema (HACE)

and pulmonary edema causes the pulmonary from (HAPE). Evidence of fluid retention is provided by the clinical observation of lower urine output in soldiers with AMS than in soldiers free of symptoms.[12]

Genesis of HAPE is multifactorial. At present it is not possible to be dogmatic about which mechanism is most important in the initiation and development of HAPE. Hultgren[16] suggested that edema is caused by a very powerful, but uneven, vasoconstriction, so that there is reduced blood flow in some parts of the lung and torrential blood flow in other parts. It has recently been proposed that HAPE is caused by damage to the walls of pulmonary capillaries as a result of very high wall stresses associated with increased capillary transmural pressure.[17] Schoene et al[18] and Hackett et al[19] by analysing branchoalevelolar lavage and pulmonary edema fluid respectively have shown conclusively that in HAPE the edema is of high protein permeability type rather than hemodynamic.

CLINICAL FEATURES

Few can ascend high mountains without suffering a mild headache and fuzziness in the head. Cause of headache is unknown. An increase in cerebral blood does not play a primary role and during attacks there is no elevation of systemic blood pressure. Other common symptoms are insomnia, anorexia, nausea, dizziness, reduced urine, vomiting and lassitude. Much of our knowledge of the symptomatology of benign acute mountain sickness comes from a study of 840 Indian troops[12] and of 146 trekkers with this condition in Himalayas[10]. Menon[20] presented largest series of 101 cases of HAPE managed by a single physician. Breathlessness was present in 84 cases, followed by chest pain in 66 cases, other common symptoms were Headache, nocturnal dyspnoea, dry cough, hemoptysis, nausea, insomnia and dizziness. There are initial symptoms of dry cough associated with breathlessness, palpitation, precardial discomfort, headache, nausea and vomiting. Pronounced weakness and fatigue are common early symptoms. The patient becomes extremely breathless and begins to cough up foamy pink sputum.

Headache is such a common feature of acute mountain sickness of all manner of severity, that it is worthless as an indicator of cerebral mountain sickness. Their climbing performance falls off, they may be irritable and wish to be left alone. Appearance of ataxia, irritability, hallucinations or clouding of consciousness should alert one to the likelihood that the patient has now HACE. Other common symptoms are disturbed consciousness, pappilloedema, bladder dysfunction, abnormal plantar responses, VI nerve palsy, speech difficulty, stupor, paralysis, coma and death. Convulsions were not reported in the European trekkers, but seizures were said to have occurred in Indian soldiers. Cerebral mountain sickness is a rare variant, when seen against the background of a large number of cases of acute mountain sickness ranging from mild to fatal.

SIGNS

These depend upon stage of the condition. Probably the earliest sign is crackles at the lung bases. Pulse rate increases early, tachypnea and cyanosis may occur. Right ventricular heave and accentuated pulmonary second sound in about half of the patients. Systemic blood pressure is either normal or mildly elevated. Reflexes may be brisk and later plantars may be extensor. There may occur ocular muscle paralysis with diplopia, often there is also in an element of pulmonary edema with signs and symptoms of that condition as well. As the condition progresses headache becomes worse, the ataxia intensifies, coma sets in, breathing becomes irregular, death may occur in a few hours or in a day or two of untreated cases.

INVESTIGATIONS

Hematology

Menon[20] found E.S.R. was normal, W.B.C. count was raised in 75 of 95 cases. This increase was due to an increase in neutrophil count.

Blood Gases

PO_2 and arterial oxygen saturation is low compared with normals for that altitude. PCO_2 is very variable and is not significantly different from controls.

Radiological

Radiological examination demonstrates pulmonary edema as a coarse mottling which is confluent and prominent in the parahilar regions. The pulmonary vessels may be seen to be engorged.[21] Singh et al[9] and

Menon[20] reported that the shadowing was at first pronounced in the upper and middle lobes, especially on the right side.

Electrocardiographic Features

The ECG shows tachycardia. The P waves are often peaked (P pulmonale) and there is right axis deviation. Some patients show elevation of ST segment.[21] T waves may be inverted in precordial leads, but this may be seen in asymptomatic subjects at altitude.[22] After clinical recovery electrocardiogram may take 3-6 weeks to return to normal.[9]

Cardiac Catheter Studies

There have been a number of catheter studies carried out in patients with HAPE before treatment,[23] or soon after starting treatment.[24,25] There was found to be high pulmonary artery pressure compared with healthy subjects at the same altitude. The wedge pressure were normal.

Neurological Investigations

Lumbar puncture in this disease reveals an elevated cerebrospinal fluid pressure. The fluid is clear, colorless with normal content of sugar and protein.[12,26] Queckenstedt's test is negative.

Postmortem Appearances

Lungs in HAPE are grossly edematous and do not float in water. The lungs at necropsy do not collapse and are congested, so that on pressure they yield a foamy pink fluid.[27] There is commonly associated pulmonary thrombosis and infarction of lung. Sometimes there is bronchopneumonia. Histopathological examination shows dilatation of pulmonary capillaries leading to thickening of alveolar septa. Edema coagulum is found in alveolar ducts and space. There have been a few reports of postmortems in HACE.[12,26] The usual findings in the brain are of cerebral edema, with swollen, flattened gyri and compression of the sulci. There may be herniation of the cerebellar tonsils and unci. In many cases there were wide spread petechial hemorrhages, in some there were antemortem thrombi in the venous sinuses.

MANAGEMENT

Most cases of benign acute mountain sickness will get better in 24-48 hrs with no treatment. If there is progression of symptoms to those of acute pulmonary edema or serious cerebral edema, action is vital, since these two disorders are frequently fatal in matter of hours. Rest alone relieves symptoms of benign AMS.[27] Acetazolamide had been shown to be an effective treatment of AMS as well as a prophylactic.[28] For the headache of AMS, aspirin or paracetamol is often used. Prochlorperazine is preferred to other antiemetics for the treatment of nausea, because it is known to increase hypoxia ventilatory response.[29]

Treatment for HACE is very similar to that of HAPE, that is, get the patient down the altitude as soon as possible. Early diagnosis is of utmost importance. If any one looks uncharacteristically weak or fatigued, he should be brought down the mountain immediately, one should not wait for obvious rales or bubbling crackles to indicate the onset of severe pulmonary edema. The intellectual debate on the validity of the diagnosis can then take place at a lower and safer altitude. Delaying descent for anything but the mildest cases is likely to be highly dangerous. The condition of descent should be made as comfortable as possible. If necessary, descent should not be delayed until morning, but undertaken by moonlight or torchlight. For those in remote mountainous areas the recommendation is urgent descent, if the patient is able to walk, he may do so, otherwise he must be carried on a porter's back or on a yak. Descent must be made immediately by car, by dirt track, mountain roads, no matter how hazardous. Even a descent of as little as 300 m may improve a patient's condition dramatically. Prompt evacuation to levels below 2400 m usually brings about rapid recovery.

Oxygen

Oxygen is a vital component in treatment, high flow rates are necessary. Menon[20] gave the gas continuously at 6-8 L/min, finding that an intermittent flow below 4 lt./min was ineffective. However others claim that oxygen at 5 to 1 L/min. may be useful.[30] It relieves hypoxia and reduce pulmonary artery pressure. There was response to oxygen therapy with in 30 min to 2 hrs, with relief of cough, cyanosis, chest pain and a diminution of pulse rate. The Gamow bag is a portable (5.5 kg) recompression chamber, which can be inflated in two minutes with a foot pump and is reported to be

effective in treating AMS. Use of portable hyperbaric bags (Gamow bags) in HACE is less well documented then in HAPE, but if available they should certainly be used. Recovery after descent may not be as rapid as is usually the case with HAPE.[31]

Patients who do not improve within few hours of oxygen therapy and those with severe symptoms may be given low doses of furosemide (20 mg x i.v. x 8 hourly), usually one to two doses are sufficient. Indian physicians initially claimed that furosemide in a dose of 80 mg daily for two days or more may prevent HAPE, but others have found that such a dose give rise to headache, vomiting, ataxia and even coma.[32] Although diuretics may seem to be an ideal agent for clearing cerebral and pulmonary edema, yet if has to be kept in mind that the resulting hemoconcentration may induce thrombosis which may characterize the pathology of severe cases of HACE and HAPE, so one may unwittingly make the condition worse.

In established cases of acute mountain sickness, treatment with acetazolamide relieves symptoms, improves arterial oxygenation and prevents further impairment of pulmonary gas exchange.

Morphine has a traditional use in the treatment of pulmonary edema with a cardiac basis. However, in severe forms of acute mountain sickness and particularly if cerebral edema is present respirationary depression can occur. Morphine certainly helps to ally anxiety and restlessness and may have a more direct effect by ensuring peripheral pooling of blood and thus decreasing pulmonary blood volume.

Antibiotics

Antibiotics may be needed to control superadded respiratory infection in HAPE. Necropsy studies of subjects dying form HAPE commonly show evidence of bronchopneumonia.

Dexamethasone is preferred to diuretics in doses of 8 mg initially followed by 4 mg 6 hourly. If dexamethasone is used, the patient should not reascend until it has been discontinued for 1 to 2 days without recurrence of symptoms as rebound effects are known to occur.[33]

Vasodilator Therapy

Vasodilator therapy is a new method for treatment of acute HAPE. Treatment of HAPE patients with nifedipine results in a reduction of pulmonary arterial pressure, clinical improvement, improved oxygenation, decrease of alveolar arterial oxygen gradient and progressive clearing of pulmonary edema on chest x-ray.[34,35] Nifedipine is given 10 mg orally every 4 hr until improvement or 10 mg orally once and then 30 mg extended release every 12-24 hrs. In another study sodium nitroprusside was shown to be effective in the treatment of HAPE and dilatation of small pulmonary arteries by sodium nitroprusside may explain its efficacy.[36]

PROPHYLAXIS

Mechanical transport by car, train or aircraft is worst means of reaching high altitude, for the simple reason that the subject can not control his rate of ascent. Overexertion should be avoided during early hours and days of arrival at high altitude. Milledge[37] advocates that care had to be taken at altitude exceeding 3000 m, with an ascent of 300 m/day for 2 days, followed by an ascent of 300 m over 2 days. The idea of "snatching a quick holiday" may be alright for sea side, but it does not give respect to the mighty mountains.

> Do not go "too fast too high"
> Beware of the "do or die" altitude in the mountains. All too often it has meant more "die" then "do"

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