Severe acute mountain sickness

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Summary

The experiences of acute mountain sickness (AMS) as it has presented to a physician working in a general hospital at 1370 m in Kathmandu, Nepal, are described. The features of 39 cases are analysed. It is suggested that AMS should be classified into benign and malignant forms.

Introduction

The experience of acute mountain sickness (AMS) at a general hospital lying at an altitude of 1370 m in Kathmandu, Nepal, is now described.

There are a number of limitations on the value of the observations. Firstly, patients are not seen at high altitude, but only after evacuation, generally from the Everest Base Camp route by which heights of anything up to 5800 m may be achieved without mountaineering or even scrambling. Secondly, patients are of such diverse nationality and language that history-taking is often difficult or impossible. Thirdly, Shanta Bhawan is a busy service-orientated mission hospital in which it is hard to find time for adequate observational and analytical work.

From the epidemiological point of view, the figures presented suffer from a further limitation, namely that there is no adequate information available regarding the total number of persons going to altitude and their distribution with regard to age, sex, nationality and rate of ascent. In other words, the epidemiological denominator is missing. An analysis of 278 trekkers passing through Pheriche on the Everest route in one month was attempted by Hackett, Rennie and Levine (1976). This put the overall incidence of AMS at 53%, although it is possible that the criteria for diagnosis were insufficiently strict. The incidence of life-threatening disease was 4.3% of the total group.

The data given here are intended to represent only one individual's experience of severe AMS under the conditions described. As an aid to true epidemiological work in the future, it is suggested that there is a need for agreement on a 'unit of altitude stress' that will take into account both the absolute altitude achieved and the rate at which it is reached.

Definitions

AMS may conveniently be divided into benign and malignant forms. The symptoms and signs of benign AMS are well known, and are listed in Table 1. Peripheral oedema and retinal haemorrhages

TABLE 1. Features of benign acute mountain sickness

Headache	Discomfort in the chest
Nausea, vomiting	Peripheral oedema
Vertigo, insomnia, weakness	Retinal haemorrhages

may be seen in persons with no subjective symptoms, but a macular haemorrhage may cause a scotoma or blindness. It seems that all persons will suffer from at least one of these symptoms if they go high enough rapidly enough. See, for example, Gray *et al.* (1971). However, these features are not life-threatening and recovery can be expected. Benign AMS may progress into one of the 2 malignant forms, affecting the lungs and the brain, or into a combination of the two. Either of these forms may be fatal, especially if evacuation to a lower altitude is delayed. While there is no doubt that the pulmonary form is due to pulmonary oedema, it is not always possible to find evidence for raised intracranial pressure in the cerebral form. Post-mortem may show petechial and larger haemorrhages, but Houston and Dickinson (1975) have summarized the evidence that brain disease is actually due to cerebral oedema, and high altitude cerebral oedema (HACO) is the standard name for the condition. Thus malignant AMS includes high altitude pulmonary oedema (HAPO) and HACO.

It is by no means clear what determines the development of HAPO and HACO. The fact that one member of a party may become deeply unconscious or severely dyspnoeic while others proceed with little or no discomfort argues for an element of idiosyncrasy. Whilst any person may suffer benign AMS symptoms if exposed to sufficient altitude stress, this may not be true of the malignant forms. A study is being planned at Pheriche this autumn which may throw light on the factors predisposing a person to the effects of altitude.

Analysis of 39 cases of malignant AMS

Type of disease

Of 39 documented cases seen between 1969 and April 1978, only 3 were pure HAPO, 10 were pure HACO and 26 showed features of both forms. The low number of HAPO cases is probably because the disease generally (but not inevitably) responds very promptly to descent to lower altitudes, and patients do not need to come to hospital. It is quite possible that the apparently 'pure' HACO cases would have shown features of pulmonary oedema if they had been examined at altitude. There were 5 deaths in the series (12.8%).

TABLE 2. Nationalities of 39 cases of malignant AMS

German	9	French	2
Japanese	4	New Zealand	1
Swiss	4	American	1
Austrian	4	Italian	1
British	4	Spanish	1
Nepalese	2	Korean	1
Norwegian	2	Polish	1
Swedish	2		

Distribution by nationality (Table 2)

This will help to emphasize the language difficulty in studying these trekkers. The proportion of Japanese is probably grossly underestimated since in many cases communication was so poor as to prevent determination of the history at all. There are only 2 Nepalese: one was a lady who went on pilgrimage to the shrine of Mukthinath, which lies at about 4250 m. She became unconscious 5 min after arrival at the shrine, and even after air evacuation she was stuporous, her arms were rigid and her legs flaccid and she remained incontinent of urine for about 10 days. The second Nepalese was a Sherpa who developed cerebral oedema at about 7000 m on the 1973 Italian Everest expedition. Since then Clarke and Duff (1976) have reported 2 more cases in Sherpas on an expedition at about 7300 m. It is worth noting that Sherpas do not suffer from any form of mountain sickness when returning to their homes in the Solo-Khumbu at 3350–4900 m, unlike the natives of the high Andes and the residents of Leadville, Colorado, who frequently suffer re-entry disease, especially pulmonary oedema. This may be connected with the fact that chronic mountain sickness (Monge's disease) has never been reported from the Himalayas, and the observation of Morpurgo *et al.* (1976) that Sherpas achieve a high affinity of the blood for oxygen without increasing the haemoglobin content of the blood. In fact they appear to become anaemic by normal standards when they descend to lower altitudes. The authors

consider this to represent a genetic adaptation to hypoxia, but have not been able to discover a physiological basis for it.

The Austrian, Swiss and German patients included several experienced Alpine guides, who had no idea that Himalayan altitudes held dangers for them.

TABLE 3. Age distribution of 39 cases

	No.	(%)
20–29 years	8	21
30–39 „	11	28
40–49 „	8	21
50–59 „	7	18
60–69 „	5	13

Age distribution (Table 3)

This is unremarkable except in so far as it draws attention to the fact that no age group is immune to acute mountain sickness. Hackett *et al.* (1976) showed, however, that younger trekkers are more at risk from mountain sickness than older people, even if adjustment is made for a slower rate of ascent.

Sex distribution

There were 33 males (84.6%). This probably reflects the sex distribution of the trekking population, which was 71% male in the paper of Hackett *et al.* (1976).

TABLE 4. Altitude of onset

in 39 cases	
Below 3000 m	2
3000–3650 m	4
3650–4550 m	17
4550–5500 m	13
above 5500 m	3

Altitude of onset (Table 4)

Only 2 of the illnesses developed below 3050 m and 3 above 5500 m.

One of the 2 'low' cases also had severe pneumonia, pleuritis and empyema at post-mortem. It is often difficult to know if a pulmonary infection has preceded altitude sickness and precipitated it by contributing to the hypoxia, or if hypostatic pneumonia has been super-added upon waterlogged lungs. In this case the history was at least compatible with the former explanation. If this is so, it may be an explanation of the observation that respiratory infections increase the risk of altitude sickness.

The second case is most surprising. This 27-year-old man asserted that he had not been above 2150 m, yet he developed severe headache, extreme weakness

and an unco-ordinated gait. After evacuation, he was found to have moderate ataxia and an extensor right plantar response. These features were reversed in a few days. Previously no case has been reported below 2450 m.

TABLE 5. Features of high altitude pulmonary oedema

Dyspnoea at rest	Tachypnoea
Cough	Cyanosis
Sputum – white frothy	Basal crepitations
– pink frothy	

Features of pulmonary oedema

Table 5 shows that the features of HAPO are similar to those of pulmonary oedema from any other cause. It is well known that pulmonary wedge, left atrial and left ventricular end-diastolic pressures have been normal in HAPO whenever it has been possible to measure them, and that cardiac glycosides do not provide any relief to the patient. It is therefore perhaps a little surprising that 'pink frothy' haemoptysis can occur in HAPO in spite of the lack of raised pulmonary venous pressure. Presumably it results from capillary damage.

X-ray changes are sometimes predominantly unilateral and can often be detected when all symptoms and signs of pulmonary oedema have disappeared.

Because pulmonary oedema contributes further hypoxia to an already hypoxaemic patient, it represents a dangerous, self-exacerbating state. Fortunately, the response to a few thousand feet of descent is usually dramatic, as illustrated by the following case:

A 28-year-old Spaniard stumbled into the Base Camp of an Everest Expedition in severe pulmonary oedema. He required continuous oxygen overnight, and helicopter evacuation was arranged. The following morning, descent of only 1000 m permitted him to remove the oxygen mask and enjoy the rest of the flight taking photographs of the mountains. He refused admission to hospital, which seemed reasonable as there were no signs in his lungs or elsewhere.

One case has provided an exception to the rule of quick recovery from HAPO. He was a 62-year-old Austrian who had been evacuated from Mount Kenya a few months before with 'lung oedema'. His history was unusual from the beginning in that it commenced while descending from 5500 to 4000 m. Further descent by air to 2750 m and the next day to Kathmandu gave no relief. On examination he was in severe pulmonary oedema and was pale and sweating and jugular venous pressure was slightly elevated. Blood pressure (150/70 mmHg) was lower than

previously recorded levels, X-ray showed some cardiomegaly as well as pulmonary oedema, ECG showed incomplete left bundle branch block and the serum aspartate aminotransferase was very high. All the factors seemed to point to cardiogenic pulmonary oedema, and he was accordingly given digitalis. However, he deteriorated and died the day after admission and post-mortem revealed severe pulmonary oedema, thrombosis of several major branches of the pulmonary arteries and no evidence of myocardial infarction in spite of moderate coronary artery arteriosclerosis. It seems that he suffered from HAPO, the only uncertainty being the pulmonary arterial thrombosis, a feature that had been noted in a previous post-mortem (Houston and Dickinson, 1975).

Features of cerebral oedema

HACO is very often associated with HAPO, and it may even be that some degree of pulmonary oedema is necessary to provide the extra degree of hypoxaemia that triggers HACO. As already mentioned, apparently 'pure' HACO cases may have had some HAPO that was missed in the mountains and had resolved by the time of admission to hospital.

TABLE 6. Common features of high altitude cerebral oedema

Severe headache	Coma
Vomiting	Papilloedema
Ataxic gait	Extensor plantar responses
Abnormal behaviour	

Table 6 shows the commonest feature of HACO. Probably the heel-toe walking, finger-nose, and Romberg's tests are the most useful in detecting early disease. Papilloedema is often mild or absent. Transient hemiparesis has been noted in association with HACO, but it is important to realize that a cerebrovascular accident may occur at altitude independent of HACO, as happened to a Sherpa on a recent Everest expedition. Some less common findings are listed in Table 7.

TABLE 7. Less common features of high altitude cerebral oedema

Hallucinations	Hemiparesis
Blurred vision	Rigidity or flaccidity
Speech difficulty	Convulsions
Urinary incontinence	Visual field defect
Cranial nerve palsies	Coarse tremor

A Japanese was recently admitted with typical severe mixed HACO and HAPO, and among other

signs he had a gross coarse flapping tremor indistinguishable from that of hepatic encephalopathy or carbon dioxide retention. It disappeared gradually in a few days.

Most victims of HACO recover fully, although one case was unconscious for 6 weeks and another for 3 weeks. Late results may include amnesia for the illness, subsequent forgetfulness and mental slowness.

the illness, because he claimed that he had followed the recommendations of the Himalayan Rescue Association regarding the rate of ascent and the need for spending 2 nights at the same altitude at about 3650 or 4250 m. This was so, but he had ignored the advice not to go higher if suffering symptoms of altitude sickness, and this had been his undoing.

FIG. 1. Altitude-time diagram of a patient with high altitude cerebral oedema (see text).

As an illustration of the development of HACO, Fig. 1 shows the progression of events in a 31-year-old male Norwegian patient on an altitude/time diagram. The patient himself drew the rough graph from which this figure was adapted. He suffered some diarrhoea and vomiting in the first few days of his trek, noted Cheyne-Stokes breathing at Namche Bazaar 3450 m, headache at Thyangboche 4200 m getting worse at Dingboche 4250 m on the fifth day with nausea and some unsteadiness on the feet. However, he pressed on to Gorakshep 5650 m and there collapsed with such severe weakness and inco-ordination that he could no longer walk. Had he remained at Dingboche, it is almost certain that he could have descended on his own feet, but the extra day's ascent caused him to be so helpless as to need to be carried down on a Sherpa's back using portable oxygen, and then air evacuation to Kathmandu. On arrival he was alert and coherent, but grossly ataxic and had mild papilloedema. Full recovery took about 3 days. He was indignant about

Treatment

Descent to a lower altitude is clearly the mainstay of treatment. The current recommendation in Nepal, no matter how serious the case, is to start evacuation by foot. 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. This is generally simpler on mountain trails than using a stretcher. If necessary, the descent should not be delayed till morning, but undertaken by moonlight or torchlight. Using these methods it is rarely necessary to go to the expense of helicopter evacuation, which in any case may be much delayed because of slow communications. Even if a helicopter is summoned, arrangements should be made for foot evacuation in the meanwhile, and the helicopter service advised of various possible picking-up points. Oxygen may be life-saving temporarily, but should not be regarded as a substitute for descent.

The role of diuretics has been questioned on the grounds that the patients are often already dehydrated. However, it seems reasonable to accept

a degree of peripheral dehydration for the sake of drawing fluid out of the lungs and brain, provided a careful watch is kept for evidence of hypovolaemia.

Agents for reducing cerebral oedema include corticosteroids, such as betamethasone and dexamethasone, and osmotic agents such as hypertonic glycerol, mannitol and urea. It is generally difficult to separate the effects of these agents from the effect of evacuation, and there is little direct evidence that they are of value.

Morphine has been used for the treatment of HAPO with apparent benefit, but caution is needed if there is an element of cerebral disease as morphine may further impair consciousness and increase the risk of inhalation of vomit.

Prevention

The Himalayan Rescue Association, centred in Kathmandu, has been educating the trekking companies and the trekking public by means of a leaflet in various languages which describes the features of altitude sickness and steps to be taken if it occurs.

It also gives recommended schedules for some of the main routes. However, it is almost impossible to devise a schedule that will be safe for everyone in view of the enormous variation in susceptibility to altitude effects. The most important recommendation is the one advising that people with mild symptoms should go no higher until they have recovered, and those with severe symptoms should go down at once. Thus the more severe forms of the disease may be avoided.

All this valuable propaganda has recently been threatened by the publicity given to the American long distance runner who ran from Kathmandu to Kala Pattar, above the Base Camp of Everest, in 4 days and 14 hours without ill effect. This, though a very fine athletic performance, sets an unfortunate precedent which may affect trekkers as well as potential runners.

Although diuretics, especially acetazolamide and frusemide, have been shown to prevent at least benign AMS, there is no definite evidence that they prevent the malignant forms. Just as rapid descent is the mainstay of treatment, so gradual ascent should be the essential element in preventative advice.

Finally, a rather bizarre case is reported, more on account of the interesting metabolic questions posed than because of any practical relevance to AMS. Many years before, and for unknown reasons, a 25-year-old Frenchman had undergone surgery to

implant both his ureters into his rectum. This is, of course, well known to produce a hyperchloraemic acidosis as well as a tendency to renal damage by calculi and infection. He was evacuated unconscious from Namche Bazaar, 3450 m, but may well have been higher than that, and detailed history was not available. His respiration was deep and stertorous, he frothed at the mouth and did not respond to painful stimuli. His pupils were widely dilated, but reactive, and there was increased tone in all 4 limbs. Although Hoffman's sign was positive bilaterally, the plantar responses were absent. The lungs were clear. Blood urea was 8.0 mmol/l. Serum potassium was 3.5 mmol/l, sodium 148 mmol/l, chloride 145 mmol/l, and bicarbonate 5 mmol/l. In spite of treatment he died 15 hr later.

The problem is – what happens to a person with an obligatory hyperchloraemic acidosis who goes to altitude and must hyperventilate and therefore tends towards respiratory alkalosis? The experience of this single case would suggest that he develops severe hyperchloraemia and goes into a coma and dies.

Post-mortem showed the results of the surgery described above, the expected consequent renal damage and moderate bronchopneumonia affecting both lungs. It seems probable that his death had a metabolic cause in view of the grossly deranged serum electrolytes in the presence of only moderate uraemia.

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