JANUARY CASE DISCUSSION

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I am passing my military service as a doctor in Andermatt (1600m), where young recruits are instructed to become mountain specialists.

On Wednesday, 3 March 1999, three weeks after begin of their training, the following incident happened. The troop, for the purpose of mountain instruction, were staying that day and the day before in the Nätschen-Gütsch area, at an altitude of 2300m. That night they spent in a snow-bivouac. In the afternoon, at about 14:00, the condition of a corporal increasingly deteriorated. He became somnolent within minutes and had several apneas with a maximal duration of 10-20 seconds. The corporal was a very fit man, but since two days before this incident he had been suffering from a common cold. The rescue was immediately organised. First with the help of his comrades and then by snow-cat, he was transported down to the valley. When I saw him at 15:30 he had a GCS of 8. Heart rate, blood pressure were normal. Auscultation of both lungs was without abnormalities. The pupils were dilated, but still reacting to light.

I informed the Swiss Air Rescue Guard; within several minutes they arrived by helicopter. The rescue guard doctor intubated the patient and transported him to the University Hospital of Zurich suspecting a subarachnoid hemorrhage. In Zurich the condition of the patient improved. Extubation at 18:00, CT scan of the head was normal, no signs of infection (including lumbar puncture). Normal blood sugar, no drugs could be detected. The next day the patient was fully conscious but he had a complete amnesia of this incident. He could leave the intensive care unit and returned then to Andermatt; he was very tired but otherwise healthy. Four days later I examined the patient again. He still suffered from a little headache and felt pain while breathing deeply. I could not find any pathologies in the clinical status.

Can such a condition have occurred due to exhaustion, in this case triggered by the common cold? Alternatively, could it have been a sort of high altitude cerebral edema (HACE), although the corporal was well acclimatized and it happened in a lower altitude zone (2300m). The patient fears that such an incident may occur again, as, in his civil life, he wants to become a mountain guide, it is understandable that he would like to know more about the causes of this occurrence.

May further examinations lead to a diagnosis? Is there any form of prevention necessary?

Bernard Marsigny, France
What was his central body temperature? We regularly receive, in the emergency ward, injured patients with a coma and mydriasis, whom body temperature is under 30°C and who recover fully within a couple of hours. Exhaustion greatly increases the risk of being hypothermic.

Gustavo Zubieta Jr, Bolivia
Interesting case. Unfortunately, no arterial blood gases, pulse oximetry or ventilation measurements at high altitude were made. This may be similar to a case of hypoventilation on arrival to high altitude we previously reported (ISMM Newsletter, Vol 8 #1, 1997/98 [resume] & http://www.geocities.com /CapeCanaveral/6280 [complete]). A Japanese subject that while in transit arrived at the La Paz airport (4100m) and within hours felt very sleepy, although responsive to stimulus with a GCS of 11. He had a very low alveolar ventilation 2384 cc/min BTPS, with a respiratory frequency of 8 per minute and a PaO_2 of 41 mmHg. When given oxygen during 24 hours he felt much better and his blood gases improved. These patients also hyperventilate on hyperoxic tests, which makes treatment much easier. A possible explanation is a temporary alteration of the respiratory center, possibly caused by bacterial or viral disease (in this case the common cold), thereby producing a limited respiratory response to hypoxia. These sudden hypoventilators on acute exposure to high altitude may confuse the physician, but there seems to be no reason to panic.

Intubation is definitely not needed. Oxygen administration and monitoring of pulse oximetry along with clinical observation, during 24 hours are necessary. There would probably be no risk in going back to altitude.

Jim Milledge, UK
Again this is an extra-ordinary case. I can only assume, in view of the rapid and complete recovery, that it must be a case of HACE. Possibly the virus infection, a common cold, was enough to cause a low saturation. This triggered the somnolence and periodic breathing thus lowering the O_2 saturation even more, causing HACE. However, at only 2300 m this sequence of events is most unusual.

Advice for the future is difficult. There is an impression that HACE is less a matter of susceptibility than is HAPE. But never-the-less he must be considered as being at higher risk of HACE than the average and future trips on to altitude he and his companions must be on the look out for trouble.

John Severinghaus, US
There is little one can do here except speculate. When the first physician saw him, was he cyanotic? Was a pulse oximeter used? One wonders if he had a severe cyclic apnea, preceded by hyperventilation such that a long apnea would occur before CO_2 rose enough to start him breathing. If so, it implies a low HVR, and he should have that tested before deciding to work at altitude. Also, if so, acetazolamide would be good to prevent a recurrence.

If a careful CT scan was really examined, and found no evidence of white matter edema, it will be hard to call it HACE. Did he have retinal petechiae? I can't relate this to a cold virus. So I guess he had post-anoxic unconsciousness. This is similar to the long period of unconsciousness we used to see after using hypothermic cardiac arrest for cardiac surgery in the 1950's. Patients whose circulation was stopped for about 15 min at 30°C often took 8-16 hours to really wake up after all anesthesia was thought to be out of the system.

We assumed it to be a post anoxic stunned brain (although we hadn't used "stunned" then).

Ken Zafren, US
This is a mysterious case. It doesn't really sound like HACE, except for the fact that it resolved after descent. Carbon monoxide poisoning is a possibility although the onset sounds quite rapid for this and we don't know from the history about exposure of others. Could this have been a seizure? An MRI of the brain might be helpful, even at this late date. The workup which included CT scan and lumbar puncture would exclude most cases of subarachnoid hemorrhage. The role of the upper respiratory infection in this case remains elusive.

Peter Hackett, US
I think this man's condition had nothing to do with altitude illness, and probably was not related to altitude at all. Assuming he had been at 1600 m for the 3 weeks of training before ascending to 2300 m, he was no doubt well acclimatized. In addition, 2300 m is barely high altitude. Since he had been staying in a snow shelter, could carbon monoxide poisoning have been a possibility? The clinical description is consistent with CO. Other possibilities include a drug not revealed by the testing undertaken, asphyxia (did one of his comrades try to suffocate him?), and atypical seizure disorder. Was he hypoxic at any time? Apparently hypoglycemia and other metabolic disturbances were ruled out, as was SAH, and presumably cerebral edema. Was an MRI done? Was MRA done to exclude any vascular abnormalities? His episode should not be considered due to altitude and warrants a thorough search for other etiologies. I would advise him to go to a higher altitude such as the Capanna Margharita or the Vallot Hut and his response to high altitude could be studied by one of our European colleagues.

Oswald Oelz, Switzerland
A Swiss soldier and mountain guide at an altitude of 2300 m. I have no idea what this guys problem was. However, I would definitely exclude HACE. The only thing that comes to my mind is some sort of seizure and I would recommend the appropriate examinations.

Robert Schoene, US
Have to think that this fellow had HACE. Unusual altitude to be sure. In HAPE there are good data (ours and the pediatric folks from Colorado) that a viral infection may predispose folks to getting sick and a large number have evidence of LTE4 in their urine as a marker of some inflammatory response that may make the endothelium more vulnerable to the subsequent pressures. Don't know about the brain but is possible. Was his serum sodium checked?

I have a couple of cases who drank a large amount of water (as read from the usual AMS brochures) and got HACE at very moderate altitudes (~2500m or so). I think that there may be a relationship between over-drinking of pure H_2O and HACE. My patient had a Na+ of 121 and was comatose.

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