Alerts, Notices, and Case Reports

Subarachnoid Hemorrhage at High Altitude

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RAPID ASCENT TO ALTITUDES over 2500 meters can result in acute mountain sickness (AMS). AMS may progress to severe forms of altitude illness, including that known as high-altitude cerebral edema (HACE) with symptoms of severe headache, vomiting, ataxia, confusion, localized paralysis, coma, and death.[1] With timely descent, the syndrome resolves. Severe neurologic impairment presenting at altitude is usually attributed to HACE, but other acute life-threatening neurologic illnesses need to be considered.

Neurologic symptoms at high altitude that persist after descent have been associated with drug-induced hypoglycemia,[2] brain tumors,[3] migraine with aura,[4] transient ischemic attacks,[5] and cerebral infarction.[6] Subarachnoid hemorrhage (SAH) presenting at high altitude has been previously noted in an autopsy series of seven altitude-related deaths.[7] To our knowledge, there is no other mention in the medical literature of SAH occurring under these circumstances. We report a case of SAH following recent ascent to high altitude.

Report of a Case

A previously healthy 72-year-old retired forest worker flew from Sweden to Kathmandu (elevation 1400 meters) in preparation for a trek in the Mt. Everest region of Nepal. After staying in Kathmandu for 3 nights, the patient flew to 2900 meters and hiked to an altitude of 4700 meters over the next 9 days. On day 2, after ascending to 3400 meters, he experienced mild frontal headache and weakness. Suspecting mild AMS, he chose to delay any further ascent for 4 nights until symptoms completely resolved. The patient remained symptom-free through the remainder of his climb to 4700 meters.

After 2 nights at 4700 meters, the patient descended to 4200 meters, at which point one of the authors noted that he appeared remarkably well given his age, and the patient reported feeling better than at any other time during his expedition at high altitude. At 6 a.m. the following day, the patient reported that he had been awakened suddenly by a severe occipital headache, although he had otherwise slept well. He took two paracetamol tablets and remained in bed. An hour later, the patient was found to be unresponsive by a companion. One of the authors residing at the nearby clinic was urgently summoned.

The patient was lying in bed and unresponsive to stimuli. His pulse was 118 beats per minute and regular, his respirations 18 per minute, and his oxygen saturation 78% (normal at 4200 m is 88%). His neurologic examination was otherwise unremarkable, including round, equal, and reactive pupils. After several minutes, the patient regained consciousness but was markedly confused and agitated. He was given 8 mg of dexamethasone via intramuscular injection and placed in a portable hyperbaric bag at 104 mmHg over ambient pressure. After 20 minutes, the patient was confused but no longer agitated. After 2 hours, the patient was alert and oriented, although he complained of occipital headache and nausea. He said that it was not the worst headache of his life. After hyperbaric treatment was discontinued, further examination yielded nonfocal neurologic results, including a supple neck, with the exception of slight ataxia found on heel-to-toe walking. His pulse was 100 beats per minute and regular, his blood pressure 155/85 mmHg, his respirations 16 per minute, his oxygen saturation 85%, and his oral temperature 98.5°F. An additional hour of hyperbaric treatment was administered while awaiting helicopter evacuation to Kathmandu. The suspected diagnosis was SAH, possibly with concurrent HACE. Lumbar puncture was not performed because of potential complications associated with this procedure in the setting of cerebral edema and because findings would not have directed a change in management during evacuation from the mountains.

On arrival in Kathmandu, the patient underwent physical and neurologic examination by a second physician and was found to be in normal condition. He did complain of neck pain, however, which was attributed to spondylosis of his cervical vertebrae based on radiographic findings. Because of the patient's history of headache and sudden loss of consciousness, a magnetic resonance imaging (MRI scan) of the head was obtained the following day to rule out a space-occupying lesion; findings were unremarkable. This physician suspected HACE that had resolved with descent to Kathmandu. As a precautionary measure, prompt return to Sweden for cerebral vascular imaging was recommended. Six days after the initial episode, while still in Kathmandu, the patient suddenly became confused, agitated, and ataxic. Lumbar puncture revealed bloody fluid under high pressure (not quantified) with a xanthochromic supernatant. An electrocardiogram and chest x-ray film were normal. The patient was medically evacuated to Sweden, where a cerebral angiogram revealed bleeding from a left middle cerebral artery aneurysm.

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ABBREVIATIONS USED IN TEXT

AMS = acute mountain sickness
HACE = high-altitude cerebral edema
MRI = magnetic resonance imaging
SAH = subarachnoid hemorrhage

Discussion

Global neurologic deterioration leading to unconsciousness is a prominent feature of both HACE and SAH. In cases of AMS sustained by unacclimatized individuals ascending to high altitudes, MRI routinely demonstrates increased intracranial pressure caused by diffuse brain swelling; this finding is associated with decreased extracerebral cerebrospinal fluid and ventricular volume resulting from hypoxia.[8] Subarachnoid hemorrhage results in blood escaping into the subarachnoid space surrounding the brain, causing meningeal irritation and an increase in intracranial pressure. Spontaneous SAH usually results from rupture of a congenital intracranial aneurysm. Less commonly, it may occur as the result of a mycotic or arteriosclerotic aneurysm, arteriovenous malformation, hemorrhagic disease, or head trauma.[9] Because the blood from an SAH surrounds the brain rather than flowing from within it,[10] global impairment of consciousness rather than focal neurologic deficit is characteristic of the syndrome, as with HACE.

In the case presented here, several clinical features differentiated high-altitude SAH from HACE. First, there was a notable absence of progressive mild symptoms of AMS with ascent; instead, the patient suddenly became symptomatic during descent. Second, sudden loss of consciousness without a gradual progression of neurologic symptoms is uncharacteristic of HACE. Third, the patient regained consciousness, although he was markedly confused and combative, before he received hyperbaric therapy. Finally, the persistent occipital headache following actual descent was alarming; HACE is known to clear rapidly with descent. These clinical features are consistent with the warning leaks frequently seen in patients with SAH prior to a disabling or fatal episode.[11]

Several factors may contribute to the risk of SAH at high altitude. The incidence of spontaneous SAH may increase with decreased barometric pressure.[12] The increase in cerebral blood flow that occurs at higher altitudes may make cerebral aneurysms and arteriovenous malformations more liable to bleed, perhaps in the same manner as retinal hemorrhages, which are very common at high altitude.[13] The response of blood pressure to acute altitude exposure is not clearly understood, but may also be involved in the onset of SAH.

MRI, which is less sensitive to subarachnoid blood than computerized tomography (CT) scanning,[10] yielded normal findings in this case. Results of spinal puncture, which is extremely sensitive to the presence of disseminated subarachnoid blood,[14] were positive after a subsequent bleeding episode in this patient while awaiting evacuation from Kathmandu to Sweden.

Conclusion

Any severe neurologic deterioration at high altitude calls for immediate and rapid descent to decrease cerebral edema and maximize oxygenation. However, recognition of acute intracranial bleeding or other neurologic emergency is also critical, because it may speed the evacuation from a remote area to a place where life-saving definitive care is available. Diagnosis of these neurologic conditions requires a careful history and examination early in the presentation, with attention to the possibility of neurologic illness other than AMS in the differential diagnosis of neurologic deterioration at high altitude. Any signs and symptoms of distress at high altitude that are notable for atypical onset, appear in unusual combinations, or persist after descent should alert health care providers to other possible emergencies in addition to high altitude illness.

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