IMAGES

Clinical Images

A mystery

A 34-year-old man was in a hurry to reach the summit of Mount Everest. He experienced a headache, extreme fatigue, and weakness at about 5500 meters on the ascent. Disregarding these symptoms, he continued to ascend while carrying a 20 kg load. At 6450 m, the next day, his headache worsened and he became confused. He began hallucinating and was almost combative. He was treated by assisted descent on foot. He received oxygen and dexamethasone (8 mg orally three times daily) during the initial descent until his mental status normalized. He eventually reached Kathmandu on foot and by road. An MRI scan of the brain (Figure 1) was obtained several days later.

What is your diagnosis?

Figure 1. MRI scan of the brain obtained with fluid attenuation inversion recovery (FLAIR) shows extensive increased signal intensity, especially in the splenium of the corpus callosum.

Figure 2. MRI scan of the brain obtained 7 months later (on a different machine), shows complete resolution of the signal abnormalities seen in Figure 1.

Answer

The MRI scan (Figure 1) reveals extensive, prominently increased signal intensity, especially in the splenium of the corpus callosum. This image is consistent with the diagnosis of High Altitude Cerebral Edema (HACE).¹

Figure 2 reveals complete resolution of the signal abnormalities 7 months later.

HIGH ALTITUDE CEREBRAL EDEMA

The history suggests that the patient had Acute Mountain Sickness (AMS) that progressed to HACE. The exact pathophysiology of HACE remains elusive, but as shown in Figure 3,² hypoxia leading to cerebral hypoxemia may trigger 3 mechanisms that culminate in vasogenic cerebral edema.³ The splenium of the corpus callosum is a region of the brain particularly susceptible to vasogenic edema resulting from cerebral hypoxemia and has been suggested as a classic MRI feature of HACE.¹

With prompt diagnosis and correct care, HACE is usually a gratifying disease to treat. Despite severe edema, as depicted in Figure 1, the patient in this case went on to complete recovery in about a month's time. Figure 2 shows no radiologic sequelae. Two years later, the patient successfully climbed Mount Everest on the 50th anniversary of its first ascent.

LESSONS

Although the patient eventually went on to climb Everest successfully, he had a narrow escape. He should have "listened to his body" and descended to 5500 m or even lower when he started to experience severe symptoms of AMS. Perhaps his climbing companions should have persuaded him to descend. However, he seems to have been determined to get to the top, "come hell or high

Figure 3. Possible mechanisms in the causation of acute mountain sickness (AMS) and its complication, High Altitude Cerebral Edema (HACE). BBB indicates blood brain barrier; VEGF, vascular endothelial growth factor; Pcap, capillary hydrostatic pressure; iNOS, inducible nitric oxide synthase; and HVR, hypoxic ventilatory response.²

water.” His motivation to reach the summit may have clouded his judgment. This is a common mindset in many climbers attempting Everest.

The most effective treatment for HACE is immediate descent. While not effective without descent, dexamethasone may have an additional beneficial role in the treatment of HACE. If you are going to be trekking or climbing in remote high altitude areas, don’t leave home without dexamethasone, and use it judiciously.⁴

References

1. Hackett PH, Yarnell PR, Hill R, Reynard K, Heit J, McCormick J. High-altitude cerebral edema evaluated with

magnetic resonance imaging: clinical correlation and pathophysiology. *JAMA*. 1998;280:1920–1925.

2. Hackett PH, Roach RC. High altitude medicine. In: Auerbach PS, ed. *Wilderness Medicine*. 4th ed. St. Louis, MO: Mosby; 2001:2–43.
3. Hackett PH, Roach RC. High altitude illness. *N Engl J Med*. 2001;345:107–114.
4. Basnyat B, Murdoch DR. High altitude illness. *Lancet*. 2003;361:1967–1974.

Buddha Basnyat, MD, MSc, FACP
Kathmandu, Nepal

David Tingay, MBBS, FRACP
Melbourne, Australia

Geeta Basnyat, MD
Kathmandu, Nepal