Letters to the Editor

Cerebral edema in the Himalayas: too high, too fast!

To the Editor:

A 55-year-old woman was trekking in Nepal. On day 1, she took a flight from 1350 m to 2500 m for her first overnight stop. The next day, she trekked to 3250 m above sea level and developed headache and tiredness, so she descended that evening to 3000 m. On day 3, feeling much improved, she trekked to 3800 m and developed further headache and shortness of breath. That evening, she received compressed oxygen by mask. At 4 AM the following day, she had developed slurred speech and shortness of breath. Two hours later she was unconscious. She was immediately evacuated to Kathmandu (1350 m).

The patient was afebrile on examination. Her eyes opened to pain, she made incomprehensible sounds, and her limbs withdrew to painful stimuli. Her pupils were reactive to light, and fundi were normal. No neck stiffness was present; however, there was increased tone in all limbs with reflexes symmetrical, apart from a right up-going plantar reflex. Blood pressure was 140/90 mm Hg, heart rate was 85 bpm, and no murmurs were noted. The patient was mildly tachypneic, and she had fine bibasilar crepitations in her lungs.

Oxygen saturation (room air) was 85%, rising to >97% with 2 L oxygen/min. A blood count revealed a hematocrit of 47% and a white blood cell count of 8700×10^3 . Electrolytes were normal, and glucose level was 6.7 mmol/L.

Lumbar puncture, echocardiography, and chest radiograph were all normal. Electrocardiogram showed left ventricular hypertrophy. Urine and blood cultures were negative. Computed tomography of the head without contrast was normal. Magnetic resonance imaging (MRI) showed discrete, high-intensity T2 signal in the white matter of the corpus callosum.

With a diagnosis of high-altitude encephalopathy and pulmonary edema, the patient received oxygen by facemask and dexamethasone, 8 mg tid for 9 days, which resulted in improvement in orientation. One month later, she had mild persisting dysarthria. Repeat MRI was normal.

High-altitude cerebral edema is a well-recognized outcome for trekkers with initial acute mountain sickness

who climb too high, too fast.^{1,2} Symptoms of high altitude cerebral edema (HACE) include headache, confusion, focal neurological deficits and occasionally progression to coma. In addition, high-altitude encephalopathy is often associated with coexistent high-altitude pulmonary edema.³

This case report is important for the following reasons: 1) It supports a recent small study investigating MRI changes associated with HACE.⁴ Hackett et al identified a clinical imaging correlate for high-altitude encephalopathy and found in 7 of 9 patients an intense T2 signal in the white matter of the splenium of the corpus callosum. The selective involvement of HACE in the less densely packed white matter suggests vasogenic edema as the pathophysiologic mechanism, rather than cytotoxic edema, which would be a likely mechanism of edema in the tightly packed gray matter.^{4,5}

2. This climber unwisely ascended 800 m in 1 day and slept at this height. Recommendations stipulate not to sleep higher than 400 m from previous day's sleeping altitude.⁶

High-altitude cerebral edema is a life-threatening form of acute mountain sickness and is a clinical diagnosis that in most cases does not require an MRI. It is 100% preventable if climbers ascend slowly and "listen to their bodies" and descend in the face of increasing symptoms of headache, nausea, and tiredness.

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