Bilateral Lateral Rectus Palsies in High Altitude Illness

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ABSTRACT

A 50-year-old man with high altitude illness had bilateral lateral rectus palsies when he went to the Himalaya at 5,000 meters. He had no other neurological signs, except headache. He received acetazolamide, prednisone, and spent time in a hyperbaric chamber; but unfortunately he showed no improvement. Two months later, his diplopia completely resolved itself without any treatment, after his descent to a lower altitude.

Keywords: Lateral rectus palsy, cranial nerve palsy, high altitude illness, diplopia

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High altitude illness (HAI) comprises of acute mountain sickness (AMS), high altitude cerebral edema (HACE), and high altitude pulmonary edema (HAPE). Hypoxia is the primary cause of HAI. However, the pathogenesis of these syndromes remains unclear. AMS is the mildest form which develops at an altitude above 2,500 meters within 6 to 12 hours especially after rapid ascent to high altitude.[1-4] Diagnosis is clinical when typical symptoms occur in the setting of a rapid increase in altitude. The symptoms include headache, typically frontal and throbbing, and any one or more of the following: gastrointestinal symptoms (anorexia, nausea, or vomiting), insomnia, dizziness, and lassitude or fatigue.[1-3] The physical examinations in AMS are usually normal.[2] These nonspecific symptoms may be important warning signs because if ascent continues it may lead to HACE, the extreme spectrum of AMS.[2-3] It is characterized by ataxia, and alteration of consciousness in the form of confusion, drowsiness, stupor, and coma.[2-5] Other possible symptoms and signs include hallucinations, cranial nerve palsy, and occasionally, focal neurological deficits.[3] Ophthalmic examinations may reveal papilledema, cranial nerve sixth palsy, and retinal hemorrhages.[2-3,5] Treatment of AMS is with analgesics and acetazolamides.[4] Severe syndrome needs supplemental oxygen. Descent to a lower altitude, if no improvement occurs with medical therapy, is always an effective treatment. Dexamethasone is useful for HACE.[4]

CASE REPORT

A 50-year-old European man presented with a week's history of diplopia when he went to the Himalayas at 5,000 meters. Before he had developed diplopia, he had occipital headache while he was 3,500-5,000 meters high with oxygenation for the last 10 days. He twice received hyperbaric chamber treatment for one hour each time, acetazolamide (250 mg.) one tablet three times per day and prednisone (5 mg.) four tablets three times per day. Unfortunately, his diplopia was not improved. He came to our hospital without headache, nausea, vomiting, and hearing loss. He denied head trauma and his other past medical history was unremarkable. He had never smoked or abused alcohol.

Ophthalmic examination revealed visual acuity was 20/20 in both eyes. Ocular motility examination showed bilateral weakness of abduction (Fig 1) with a primary position 30 prism-diopter esodeviation (Fig 2). The anterior segment examination was unremarkable. Corneal sensation and facial nerve function were intact. The pupils were equal and reactive to light. Ophthalmoscopic examinations revealed normal disc and fundus. Humphrey visual field testing was normal in both eyes. General physical and neurological examinations were also normal. Brain magnetic resonance imaging (MRI) showed a few tiny nonspecific

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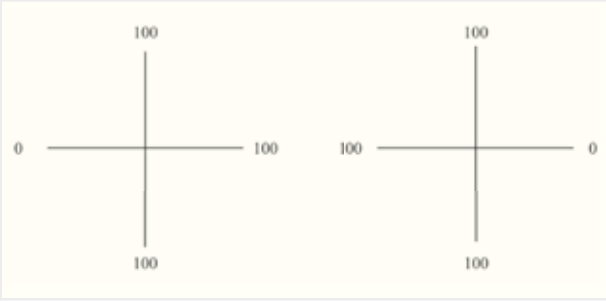

Fig 1. Ocular motility examination demonstrated bilateral weakness of adduction.

white matter changes. A brain magnetic resonance angiogram was unremarkable. Complete blood count, fasting blood sugar, erythrocyte sedimentation rate and blood chemistry were within normal limits.

The diagnosis was bilateral lateral rectus palsies. He was treated with trental to improve blood circulation of the nerve and brain and one eye was patched to relieve his diplopia. He stayed in the hospital for three days then flew back to his country.

Three months later, we got his email. He stated his diplopia got progressively better and recuperated completely from diplopia after two months. He went to check with an ophthalmologist there. The doctor said his ocular motility was normal. He went to the Alps to progressively climb to 3,300 meters for skiing.

DISCUSSION

Acclimation is a complex process by which climbers adjust to high altitude hypoxia to increase their oxygen. Barometric pressure and the partial pressure of oxygen decreases rapidly at increasing altitude. Oxygen comprises a constant 21% of air at any altitude; therefore the partial pressure of oxygen declines along with barometric pressure on ascent to high altitudes. Hyperventilation is an important process to increase oxygen consumption, although, it also reduces the alveolar partial pressure of carbon dioxide and causes respiratory alkalosis, resulting in a decrease in ventilation.[5-7] Alkalosis is corrected by excreted bicarbonate in urine, and thereafter results in a further appropriate increase in the ventilatory response. Inadequate hyperventilation in non-acclimatized persons will develop to HAI after ascent to higher altitudes. The risk of HAI increases directly with the rate of ascent and the altitude reached. In addition to hyperventilation, the heart rate and cardiac output increases and cerebral blood flow also increases to deliver oxygen to the tissues.

Our patient had an occipital headache when he ascended to high altitude. He developed binocular horizontal diplopia later at 5,000 m. height. Unfortunately, the symptoms were not improved with acetazolamide and prednisolone. It might be difficult to diagnose that he had severe AMS or HACE. The only focal neurological deficit which was isolated, was bilateral lateral rectus palsy. He did not show any sign of disc edema, although he was examined seven days after his descent from the high altitude.

The causes of lateral rectus palsy were proposed as increased intracranial pressure from a cerebral edema and an ischemiatic process of the sixth nerve.[8] For our patient, we judged this might be a sixth nerve injury, or stretch, from elevated intracranial pressure where it enters the cavernous sinus through Dorello's canal. It is unlikely to be ischemiatic to both the sixth nerve trunks simultaneously. He also did not have any medical history of diabetes mellitus, hypertension, or dyslipidemia that could be the cause of the ischemia. It was not sixth nerve nucleus lesion in the pons, because our patient neither developed any signs of internuclear ophthalmoplegia, contralateral long tract signs, nor cranial nerves V,VII, VIII involvements. Brain MRI also showed a normal brainstem. The possible pathophysiologic mechanisms of cerebral edema and increase of intracranial pressure in severe AMS and HACE may involve cerebral vasodilatation, increased cerebral blood flow, raised cerebral capillary pressure and increased permeability of the blood-brain barrier through cytokine activation.

Our case demonstrated bilateral sixth nerve palsies after ascent to a high altitude. We postulate that the sixth nerves were injured from increased intracranial pressure in HAI. However, this was resolved completely after descent to a lower altitude after two months. Finally, we were interested in this case because it is uncommon. Nowadays sport climbing has become more popular. We may encounter more patients coming with bilateral cranial sixth nerve palsies. Well this condition is another cause, of which we should be aware.

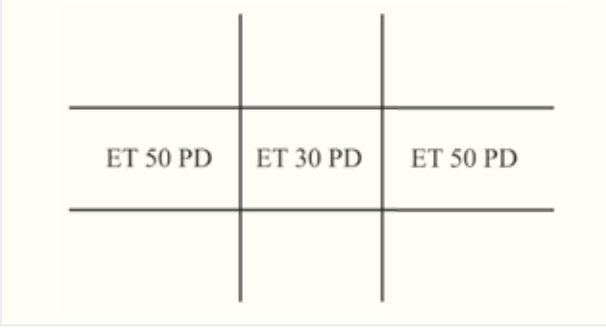

Fig 2. The patient had esotropia 30 prism-diopter in primary position and 50 prism-diopter on right and left gaze.

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