Case Report

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Stroke in Young at High Altitude

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Abstract

Mountaineering is a favorite adventure sport for many youngsters. In untrained individuals, sudden ascend to high altitudes can cause severe deleterious health issues. Individuals vary in their physiological response for adapting to the low oxygen conditions. Acute altitude illness can present as acute mountain sickness (AMS), high-altitude cerebral edema (HACE) and high-altitude pulmonary edema (HAPE). As emergency physicians, we stand as public health guards and it is our duty to educate people about risks involved in adventure sports.

Keywords: Acute mountain sickness; High-altitude cerebral edema; High-altitude pulmonary edema.

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Introduction

Mountaineering has become a favorite pastime for many youngsters. In untrained individuals, sudden ascend to high altitudes can cause severe deleterious health issues. There is a decrease in the barometric pressure at higher altitude resulting in decrease in the partial pressure of oxygen causing hypobaric hypoxia. This eventually results in a series of physiological responses, for adapting to the low oxygen conditions. However, when adaptation doesn't occur, it can result in acute altitude illness. There is an increased risk of developing acute altitude illness during the initial 1–5 days of ascent to an altitude ≥2500 m. Acute altitude illness can present as acute mountain sickness (AMS), high-altitude cerebral edema (HACE) and high-altitude pulmonary edema (HAPE). Following an ascent to a given altitude, the illness can range in severity from mild to life-threatening. AMS is a syndrome of nonspecific symptoms which include headache along with dizziness, GI disturbances, lassitude and/or sleep disturbance. HACE is potentially fatal condition characterized by ataxia and decreased consciousness with characteristic changes on magnetic resonance imaging. HAPE, a non-cardiogenic pulmonary edema results from excessive hypoxic pulmonary vasoconstriction and can be fatal if not recognised and treated promptly.[1]

Case report

A 25-year-old female was involved in adventure mountaineering. She had ignored the initial symptoms of dry cough and few episodes of vomiting and kept on ascending. Eventually she

developed shortness of breath and headache at 12000 feet. She was then made to descend under medical supervision in view of acute pulmonary edema and acute mountain sickness. Initial stabilization could be done after a descent to 6000 feet with oxygen supplementation, diuretics, intravenous fluids and antiemetics which stabilized her hemodynamics. Apart from these symptoms, weakness of left upper and lower extremities and slurring of speech was noticed and urgent decision to airlift her to the base hospital was planned. After 1 day, she was shifted to the Nizam's institute of medical sciences for further management. We received her with patent airway and ongoing fluid resuscitation. Her vitals on arrival were normal. Her ECG, arterial blood gas analysis and chest X-ray were normal. General examination was unremarkable except for a slight pallor noted in lower palpebral conjunctiva. On CNS examination, patient was conscious, opening eyes spontaneously, oriented to person but not to place; cranial nerve examination showed left upper motor nerve facial palsy with dysarthric speech; motor system examination revealed decreased tone in the left upper and lower limbs, power was 0/5 in left upper and lower limbs, deep tendon reflexes were diminished on left side with a positive Babinski's sign on left side.

A non-contrast CT Brain was ordered which revealed a right middle cerebral artery territory infarct. Immediate neurology referral was kept and she was started on antiplatelets. However, she was further investigated for neck vessel Doppler and Magnetic resonance angiography (MRA) to establish the cause of acute ischemic stroke. These investigations were suggestive of atherosclerosis, the chief cause of cerebral ischemia in this patient.

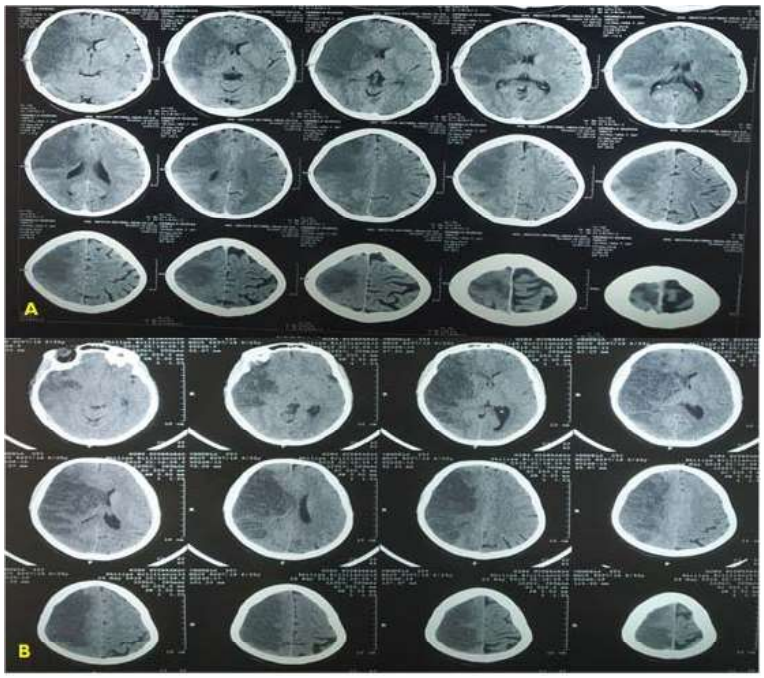

Fig. 1: (A) Non contrast CT brain showing large area of hypodensity involving the right fronto-parietal region suggestive of right middle cerebral artery (MCA) territory infarct. **(B)** Repeat CT showing increase in the size of infarct with effacement of adjacent sulcal spaces.

The MRA showed no flow in the right internal carotid artery. Her laboratory parameters showed low hemoglobin (9.2 gm/dl), subclinical hypothyroidism (raised TSH with normal T3/T4) and hyperhomocysteinemia (more than twice the normal value). Echocardiography was normal and the follow up in neurology ward showed normal vasculitis profile.

Our patient had cerebral infarction (acute ischemic stroke) with AMS and HAPE. Risk factors to be consider in our patient for causing an acute ischemic stroke was presence of anemia at the start of hiking, hyperhomocysteinemia and hypoxia at higher altitude. Her motor power in left upper and lower limb from the initial presentation of 0/5 [modified Rankin Score (mRS)–4] improved to 2/5 at 3 months follow up (mRS-3) and 5/5 at one year follow up (mRS-0) (Fig. 2).

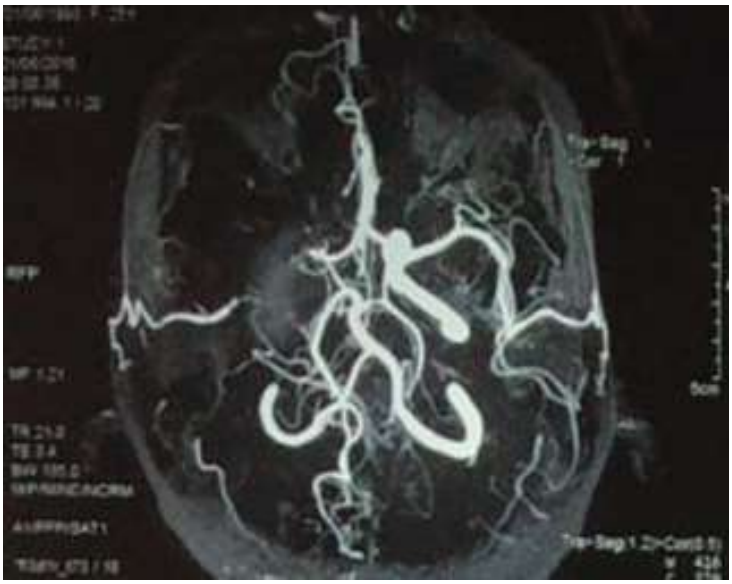

Fig. 2: MR angiogram brain showing non visualization of middle cerebral artery (MCA) and internal carotid artery (ICA) on right side.

Discussion

High-altitude syndromes usually result from rapid ascent in unacclimatized individuals. And all of them respond to the same therapy, i.e. descent to lower altitude and oxygen supplementation. Many of these syndromes are directly attributed to hypoxia, while others not necessarily related to hypoxia at higher altitudes include thromboembolic events, high-altitude pharyngitis, high-altitude bronchitis and ultraviolet keratitis. There is usually an overlap of different hypoxic clinical syndromes. Individuals given time to acclimatize for days to weeks can tolerate hypoxemia to varying degrees and therefore can have normal function. The carotid body modulates the hypoxic ventilatory response, senses a decrease in arterial oxygenation and signals the central respiratory center in medulla, thus increasing the ventilation. The bicarbonate diuresis compensates for the respiratory alkalosis, normalizing the pH, and ventilation continues to increase again. The baroreceptors are triggered by increased central blood volume due to the peripheral vasoconstriction, inhibiting secretion of antidiuretic hormone and aldosterone and inducing diuresis. Thus the plasma volume decrease results in hemoconcentration, thereby increasing the oxygen-carrying capacity of blood. Clinically, diuresis and hemoconcentration are considered to be a healthy response. Antidiuresis on the other hand is associated with AMS and may contribute to edema formation.

The symptoms of AMS occur with a usual delay of 4–12 h after arrival at a new altitude, more so after the first night spent and resolve spontaneously with appropriate measures taken.[2] The most common symptom is headache, required for the diagnosis of AMS.[3] However there are few exceptions where individuals presenting with symptoms due to higher altitude may not have headache at all (probably ~5%).[4] Progression of AMS to HACE should be considered when nausea and headache doesn't respond to the first-line antiemetics and analgesics and lassitude progressively increases.[5] HAPE also occurs after rapid ascent of unacclimatized individuals within 1–5 days at altitudes >2500–3000 m.[6] Early symptoms include mild cough, excessive exertional dyspnea, chest tightness and decreased exercise performance. Further, cough and dyspnoea worsen and orthopnea develops. There is patchy peripheral and nodular distribution of edema on chest radiographs and computed tomography scans in individuals with HAPE.[7] In many cases, symptoms of AMS usually precede HAPE. Other issues that can occur at higher altitude are retinal hemorrhages, cerebral infarction, seizures and funny turns, etc. Retinal hemorrhages resolve spontaneously and rarely cause visual loss.

Cerebral infarction at higher altitude occur more frequently than reported. Dehydration and hypoxia induced polycythemia are presumably involved in its pathogenesis.[8] In an Indian study of stroke at high altitude, longer the stay-higher was the risk

of associated stroke. Although all types of stroke were seen, ischemic stroke was the commonest. Also massive infarcts were common.[9] Several risk factors with increased risk to developing ischemic stroke at altitude included (a) polycythemia, (b) stronger platelet adhesiveness, (c) increased levels of thromboplastin, (d) blood stasis aggravated by the presence of polycythemia, endothelial damage and hyperviscosity (e) prolonged immobility and (f) increased chances of dehydration.[10]

Patients with subclinical hypothyroidism with higher levels of serum TSH tend to have a milder stroke on admission with a significantly better outcome and mortality was also found to be lower in these acute ischemic stroke patients.[11] Possible explanations for this association was ischemic preconditioning, reduced adrenergic tone and hypometabolic state. Subclinical hypothyroidism was associated with better modified Rankin scale scores at 1 and 3 months. The elevated initial TSH (clinical or subclinical hypothyroidism) may correspond to better functional outcomes, whereas low initial T3/fT3 might correlate with worse outcomes in acute ischemic stroke among clinically euthyroid patients.[12]

Hyperhomocysteinemia is one of the modifiable risk factor among causes of ischemic stroke in young. All the young patients with ischemic stroke needs to be investigated for homocysteine levels and hyperhomocysteinemia treatment may help in preventing recurrence of ischemic stroke.[13] Low hemoglobin level concentration is also an important risk factor for ischemic stroke.[14]

Conclusion

Mountaineering is a scientific art and not merely a physical activity. It is one of the finest sports but practicing it without proper training is a deliberate suicide. As emergency physicians, we stand as public health guards and it is our duty to educate people about risks involved in adventure sports.

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