Neurology Asia 2013; 18(1) : 99 – 101

Delayed parkinsonism following high mountain climbing: A case report

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Abstract

Acute mountain sickness is an illness caused by climbing to a high altitude without prior acclimatization. Neurological consequences, like parkinsonism following acute mountain sickness without lesion of brain MRI have been reported rarely. A healthy 56-year-old man presented with dysarthria and gait disturbance. Neurological examination revealed tremor of hands, limb rigidity, and bradykinesia. The symptoms developed approximately 30 days following a 3,500 m climb of the Annapurna in the Himalayas. Brain MRI did not reveal any abnormalities including globus pallidus. The parkinsonism symptoms persisted for about 3 months before a complete recovered was made. We suggest that parkinsonism can develop after climbing to a high altitude but that the symptoms can be transient if a brain MRI detects no abnormalities.

INTRODUCTION

Acute mountain sickness (AMS) is an illness caused by climbing to an altitude above 2,500 meter without prior acclimatization. The clinical symptoms of AMS include headache, nausea, malaise, dizziness, and insomnia within 6 to 12 hours of reaching the high altitude.[1] Symptoms commonly improve within 1 to 3 days; neurological consequences, like cognitive dysfunction and parkinsonism are rarely reported.[2,3] For the reported cases, globus pallidus lesions were seen in magnetic resonance imaging (MRI) and prolonged symptoms. Herein, we report a case of parkinsonism presenting with bradykinesia and limb rigidity following AMS and without lesions of the globus pallidus.

CASE REPORT

A healthy 56-year-old Korean man presented with dysarthria and gait disturbance. He has by then having symptoms for 10 days. He was on medication for hypertension, diabetes, and hyperlipidemia. The vital signs were stable. About a month before symptom onset, he climbed the Annapurna of the Himalayas. Prior to that endeavor, he often went hiking and had completed a full marathon. He often climbed to the Daecheongbong, the highest peak of Mount Sorak in South Korea; however, he had never climbed above 1,700 m. He stayed in the Annapurna for 5 days with the intent of mountaineering. On the first day, he climbed to a base camp located about 1,200 meter above sea level. On the second day, he climbed to 3,000 meter, during which he had no symptoms. However, when he began climbing in the early morning of the third day, he suffered from headache, heaviness of both legs, and dizziness. Upon reaching 3,500 m, he turned back and began descending the mountain. After returning home, he experienced no disturbances in his daily activities. Approximately one month later, he experienced some loss in his facial expressions, and became slower and imbalanced when he walked. He also complained of subjective dysarthria and general weakness. A neurological examination showed that he was mentally alert. The Unified Parkinson's Disease Rating Scale (UPDRS) Part III (motor examination) was 11 points. The following parameters were scored: speech (1), facial expression (1), tremor at rest (2), action or postural tremor of hands (1), rigidity (1), finger taps (1), hand movements (1), rapid alternating movements of hands (1), leg agility (1), and gait (1). Routine blood tests only revealed hyperlipidemia. His electrocardiogram and echocardiography were normal. The 3.0-Tesla MRI (Achieva 3.0T, Phillips, Dutch) with

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T2-weighed and FLAIR images revealed no brain lesions (Figure 1). The patient improved progressively without the use of any specific anti-Parkinson medication. One month following symptom onset, the UPDRS improved to 3 points, and by 3 months, it was 0 point.

DISCUSSION

AMS was defined by the Lake Louise Consensus Group as the onset of symptoms in healthy individuals, such as headache, insomnia, dizziness, malaise, poor oral intake, nausea, and vomiting following ascent to an altitude above 2,500 meter.[1] In severe cases, AMS sufferers can develop high altitude pulmonary edema (HAPE) or high altitude cerebral edema (HACE), which in turn can be life threatening.[4,5] HACE is clinically diagnosed when ataxia or mental changes is present in a patient with AMS or HAPE. MRI imaging studies have shown that most AMS sufferers have had some degree of cerebral edema.[6,7] It has also been shown that the occurrence of AMS depends on an individual's ability to compensate or overcome cerebral edema.[8] There have been also a few case reports of lesions in the globus pallidus following high altitude climbing.[2, 3]

Our patient suffered AMS symptoms, with headache, nausea, and malaise. He subsequently experienced tremor of his hands, limb rigidity, and bradykinesia. In a previous case report, the patient developed mild akinetic-rigid parkinsonism after climbing the 4,876 m Himalayan peak, Bhagirathi. The brain MRI revealed bilateral globus pallidus lesions. The patient progressively improved with dopamine medication and recovered 20 months after the event.[2] In comparison to that case, our patient's symptoms of parkinsonism improved quickly and without medication, and 3.0-Tesla MRI revealed no lesions in the brain. The UPDRS initially scored 11 points, but 3 months later, the patient had completely recovered. We would like to suggest that parkinsonism occurred by transient regional hypometabolism due to hypoxia in the globus pallidus. However, we did not perform functional imaging. There was another case previously reported with personality changes that occurred due to hypoxic damage of the globus pallidus after climbing 4,700 m of Mount Kilimanjaro. In that case, parkinsonism did not occur, although MRI imaging revealed bilaterally symmetrical lesions on the brain, involving the globus pallidus region.[3] We did not implement any neuropsychological tests because our patient did not complain of cognitive dysfunction or personality changes. During his symptomatic period, he continued to perform his daily activities in spite of motor disturbances. However, we cannot exclude the possibility that subtle changes in cognitive functions could have been highlighted by neuropsychological tests.

As altitude increases, barometric pressure falls. This causes a corresponding drop in the partial pressure of oxygen, resulting in hypobaric hypoxia. Neurological consequences vary greatly from person to person and with rate of ascent.[9] The mechanism of cognitive dysfunction or parkinsonism that can occur after high altitude climbing may be due to a medial frontal and dorsolateral frontal lobe syndrome associated with globus pallidus injury. Regional brain hypometabolism associated with high altitude hypoxia is believed to be one of the possible causes.[10,11]

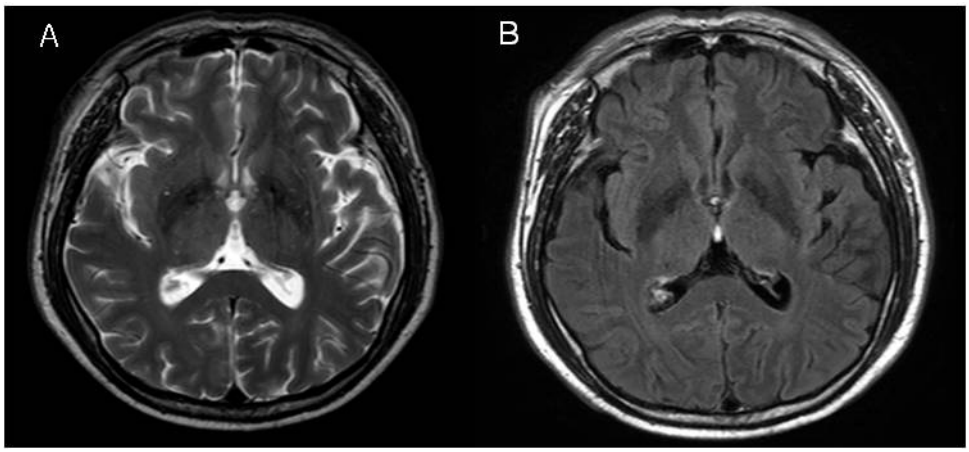

Figure 1. Brain MRIs of the patient. Axial T2-weight image (A) and FLAIR image (B) show no definite abnormality in basal ganglia.

Clinically, the long-term prognosis of AMS is good unless it is accompanied by other complications, such as pulmonary or cerebral edema. A slow ascent with sufficient time for acclimatization is the best way of preventing AMS and HACE.[12] For example, climbers should ascend less than 600 m every 24 hours if they are climbing at altitudes above 2,500 meter. When traveling above 3,000 meter, the recommended ascent rate is less than 300 m a day, with a rest day for every 1,000 m climbed.[12] Our patient had no acclimatization period before climbing the Annapurna, and he ascended 1,800 m within 24 hours.

We suggest that parkinsonism can develop after climbing to a high altitude, the symptoms of which can be transient if a brain MRI detects no abnormalities. However, people who plan to climb altitudes above 2,500 meter need sufficient acclimatization before climbing and must pay attention to their speed of ascent.

ACKNOWLEDGEMENT

This work was supported by a research grant from Soonchunhyang University in 2012.

DISCLOSURE

Conflict of interest: None

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