INTRODUCTION

Acute mountain sickness (AMS) occurs commonly in hikers who are rapidly exposed to high altitude environments (1). Symptoms of AMS include headache, nausea, irritability, insomnia, dizziness, and vomiting. In some individuals, AMS can progress to high altitude cerebral edema (HACE) in which patients suffer from somnolence, dysarthria, diplopia, nystagmus, truncal ataxia, pyramidal signs, or even coma (2). Despite the numerous reports of AMS and HACE, few studies have reported pallidal lesions associated with altitude sickness (3). We report a case of bilateral globus pallidus lesions that developed during mountain climbing. Our case suggests that globus pallidus injury should be included in the differential diagnosis of patients with personality or cognitive change after recovery from AMS.

CASE REPORT

A 49-yr-old Korean man with 16 yr of school education was brought to our Memory Disorder Clinic because of change in personality. Symptoms developed abruptly after climbing a high mountain one month prior to evaluation. He was previously healthy without prior history of smoking or vascular disease. Past medical history was also negative for carbon monoxide poisoning or hypoxic insult. On August 4, 1998, he and his wife started climbing Mount Kilimanjaro and reached 4,700 m on the third day. At that time, he complained of fatigue and asked to rest every 10 meters while climbing. After abandoning further climbing later that day, he immediately fell asleep. When he woke up the next morning, he acted exhausted and did not speak spontaneously; he spoke only when directly addressed. He was evacuated to 3,700 m and slept throughout the fourth day. On the next day, he climbed down to 1,700 m without assistance but looked unsteady while walking. His wife later noted that he could not recall events between the fourth and fifth days of the expedition. During the whole trip, the patient and his wife stayed in the same tent or hotel and denied of being exposed to other sources of hypoxic injury (e.g., use of butane gas stove in the tent).

After returning home on August 13, the patient required no assistance with activities of daily living, but he was slow to respond and displayed lassitude, preferring to stay in bed. He also required prompting to execute personal hygiene. Prior to the climb, he was talkative and humorous; upon return, the quantity and volume of his speech were markedly reduced and he showed minimal sense of humor. Premorbidly, as an owner of a small business, he was strong-willed and rather impatient with others in his office. After the climb, his co-workers noted that he became generous, indifferent, and indecisive when making important decisions.

His elemental neurological examinations were unremarkable and his MMSE score was 30/30. Results of neuropsychological tests performed 34 days after the onset were as follows: verbal IQ 141, performance IQ 116, and full scale IQ 131 (98th percentile); digit span forward plus backward 14 (96th percentile); fluent speech with intact comprehension, repetition, and naming (Boston Naming Test 58/60 [98th percentile]);
flawless pantomiming of tool use and tool-free gestures; Rey-Osterrieth Complex Figure copy 36/36, immediate recall 28/36 (95th percentile), and 20 min delayed recall 30/36 (90th percentile); Hopkins Verbal Learning Test immediate free recall over 3 trials 27/36 (99th percentile) with 20 min delayed recall 11/12 (99th percentile); intact controlled oral word association; flawless letter and color reading on the Stroop test; achieved all 6 categories in Wisconsin Card Sorting Test. Minnesota Multiphasic Personality Inventory revealed a normal profile with no evidence of depression. Blood work-ups including hemoglobin were normal. T2-weighted and FLAIR magnetic resonance (MR) imaging performed on September 2, 1998, revealed a focal high signal intensity lesion in the globus pallidus bilaterally (Fig. 1). MR angiography showed no diagnostic abnormalities.

In January 1999 (6 months after the onset), the patient’s wife reported that the patient could manage to run his own business but needed co-workers’ help because of lack of confidence, indecisiveness, and decreased judgment in making important decisions. Brain 18F-fluoro-2-deoxy-D-glucose positron emission tomography (PET) performed at that time revealed no regions with abnormal metabolism. We lost further follow-up of the patient.

**DISCUSSION**

After ascent to 4,700 m, our previously healthy patient suffered symptoms consistent with AMS that might have progressed to HACE. After descent from the high altitude, the patient showed changes in personality characterized by abulia, indifference, and indecisiveness. Our patient’s behavioral alterations may be explained by a medial frontal and dorsolateral frontal lobe syndrome that is associated with globus pallidus injury (5). Alternatively, our patient’s behavioral changes can be interpreted as the regional brain hypometabolism associated with high-altitude hypoxia reported by Hochachka et al. (6), who performed PET scans in 6 healthy lowlanders before climbing and after descent of high altitude. According to this study, the PET scan performed one day after the descent showed decreased regional brain metabolism on the frontal and occipital lobes and thalamus, compared to the baseline. However, this frontal hypometabolism may not be consistent with our patient’s frontal lobe dysfunction because the glucose hypometabolism was asymptomatic, occurring as a defense adaptation against chronic hypoxia. Furthermore, the frontal hypometabolism in that study was transient, whereas our patient’s symptoms lasted over 6 months. There has been only one report of globus pallidus damage associated with AMS (7). In this case, the time interval between AMS and identification of the pallidal lesion was one year, rendering the association less convincing than in our patient.

Despite the behavioral alterations in everyday life of our patient, the neuropsychological tests were inconclusive. A similar dissociation, i.e., the combination of profound changes of behavior and superior intelligence has been reported previously (8). The reason for this discrepancy remains unclear. One parsimonious explanation may be that conventional frontal lobe tests including Wisconsin Card Sorting Test may not be sensitive for detecting subtle changes of executive functions, and medial frontal lobe syndrome (e.g., abulia and indifference) which was the main symptoms of our patient.

Numerous etiologies have been known to be associated with bilateral globus pallidus necrosis (9, 10). These include hypox-
ia, hypotension, hypothermia, and intoxication with disulfiram (11) or cyanide (12). Of these, the pallidal lesion of our patient could be explained by altitude-related anoxic brain damage as often observed in ischemic-hypoxic encephalopathy caused by CO poisoning (12) or cardiac arrest with successful resuscitation.

Occurrence of the severe form of AMS or HACE depends on the speed of ascent, the altitude attained, and age (4). Of these factors, AMS in our patient might have resulted from failure to observe recommendations to avoid abrupt ascent to altitudes over 3,000 m and to spend 2-3 nights at the intermediate altitudes prior to further ascent (1). Therefore, our case emphasizes the necessity for gradual ascent to high altitude. Our experience also suggests that globus pallidus injury should be included in the differential diagnosis of patients with personality or cognitive change after recovery from AMS.

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