CASE REPORT

A Case of Cerebral Sinus Thrombosis Developed During a High-Altitude Expedition to Gasherbrum I

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We report a case of cerebral sinus thrombosis, which is a rare but serious complication that may develop during high-altitude climbing. A climber who reached the summit of Gasherbrum I (8068 m) suffered from motor weakness and gait disturbance as he descended from the peak. Within a few days, the symptoms progressed until he could not walk. Following a difficult and week-long rescue mission by his colleagues, he was hospitalized and diagnosed with thrombosis at the superior sagittal and right transverse sinuses. Climbers and rescuers should know the risk factors of this complication, such as hemoconcentration, procoagulability, and dehydration.

Key words: sinus thrombosis, cerebral infarction, high-altitude expedition, dehydration, coagulation disorder, cerebral edema

Introduction

High-altitude pulmonary edema and high-altitude cerebral edema are well-known serious diseases that can develop during high-altitude climbing.¹ Cerebral sinus thrombosis, which is one of the consequences of high-altitude dehydration, is not frequently observed, but is another serious complication of a stay at high altitude. Rescuing the victim from a remote and high-altitude location is very difficult. Also, the victim may have significant disability related to acute symptoms. We present a case in which the patient experienced motor weakness and severe gait disturbance while descending from the summit of Gasherbrum I.

Case report

A 37-year-old man who had already climbed 4 summits higher than 8000 m was a member of a 4-member expedition aiming at Gasherbrum I and Chogorisa. He had no preexisting disease before the expedition, and no abnormality had been identified in previous health checks.

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He had been a habitual smoker (1 pack per day) for 15 years. The party's activities during the expedition are listed in the Table. On the 45th day after the start of climbing activity above 5000 m, the man showed signs of neurological dysfunction, including gait disturbance and difficulty speaking. On the 46th day, he experienced paresthesia in his upper extremities, visual field narrowing, and coordination disorder. These neurological symptoms developed one by one over 48 hours. None of the symptoms improved, and all slowly progressed during the following 10 days of rescue activities. Since he could not stand up by himself and could not walk, his party and cooperative members from another expedition rescued him. The rescue work was extremely difficult due to hazardous conditions on the unstable glacier.

SYMPTOMS AT OUR HOSPITAL

On the 13th day after the onset of symptoms, the patient was admitted to our hospital (Fujioka General Hospital, Fujioka, Japan). He could not walk because of motor weakness in his lower extremities and dysequilibrium. Articulation disorders and agraphia were also observed. Laboratory examinations revealed a high hemoglobin concentration (18.1 mg/kg) and hypoproteinemia (5.4 mg/kg). Platelet count, prothrombin time, activated partial thromboplastin time, bleeding time, and whole blood clotting time were all within normal ranges. There were

List of activities

June 25	Started from the base camp (altitude: 5100 m) (Between the base camp and the first camp, the route went across a big unstable glacier)
July 1	Set up the first camp (5800 m)
July 2	Set up the second camp (6400 m) (For 21 days total, the climbers stayed at the base camp because of bad weather)
August 5	Arrived at the summit (8068 m) and sheltered overnight in an emergency tent at 7250 m
August 6	Returned to the third camp (7150 m)
August 7	Patient experienced gait disturbance at 6400 m Patient unable to keep standing position and experienced difficulty in speaking at 6000 m Colleagues orally administered dexamethasone (5 mg) and nifedipine (10 mg)
August 8	Patient experienced paresthesia in his upper extremities, visual field narrowing, and coordination disorder Patient descended to the first camp with support from his colleagues
August 9	Stayed at the first camp Colleagues fetched an oxygen cylinder and medicine from the base camp
August 10	Patient received an injection of dexamethasone (8 mg intramuscular) Patient crawled 100 m further down in altitude with help
August 11	Party descended 250 m further in altitude
August 12	Returned to the base camp in the evening
August 13–15	Patient used oxygen and waited for a military rescue at the base camp
August 16	Patient transported to a local hospital by an air-rescue helicopter
August 17–18	Physicians administered intravenous crystalloid infusion at a local hospital
August 19	Patient transported to a hospital in Japan (Fujioka General Hospital)

no abnormalities in chest radiographic examinations or electrocardiography. Brain magnetic resonance imaging (MRI) revealed ischemic changes in the cerebrum and frontal cortex (Figure 1). Narrowing of the superior sagittal and right transverse sinuses was observed in the patient's angiography, which was obtained on the second day after admission (Figure 2). Cerebral arteries appeared completely normal in the image. Single-photon-emission computed topography showed decreased blood flow in the cerebellar cortex, frontal cortex, and the border of the temporal and occipital cortex.

TREATMENT AFTER ADMISSION

Low-molecular-weight (40 000) dextran (1000 mL/d) was infused for 5 days to reduce the blood viscosity. On the third day after admission, physiotherapy and occupational therapy were started for rehabilitation. The patient's speaking and writing capabilities gradually improved. Dysequilibrium was ameliorated, and he could walk without any support when he was discharged from the hospital 3 months later. At that time, ischemic changes on MRI were not observed, but the ventricles were slightly enlarged, implying minor atrophy in the cerebrum and cerebellum. Six months later, he was working on the rehabilitation program as an outpatient.

Discussion

In the present study, MRI indicated blood circulation disorder at the frontal cortex and cerebellum. Gait disturbance and speech problems were explained by dysfunction of the motor cortex and Broca center. Dysequilibrium, articulation disorders, and agraphia were possibly derived from cerebellar dysfunction. Relatively slowly developing symptoms were consistent with cerebral infarction induced by venous occlusion. The results of angiography implied that the cause of the circulation disorder was occlusion in the superior sagittal and right transverse sinuses. However, these images were obtained 10 to 12 days after the onset of symptoms. Also, the patient had already been treated at a local hospital. (Since neither the patient nor the person attending him remembered the name of the local hospital or the doctors, we were unable to obtain details of their diagnosis and therapy regimen. The patient only remembers that he had an intravenous drip infusion of Hartmann solution.) Thus, it is possible that the original circulation disorders were largely modified before the time of our assessment. We speculated that symptoms consistent with cerebral infarction were incurred by thrombosis of the cerebral sinuses and that the thrombus was partially lysed and the occluded sinuses were partially recanalized

Figure 1. Diffusion weighted image (DWI) magnetic resonance image at the time of admission. Brain magnetic resonance imaging revealed ischemic changes in cerebellum (A) and frontal cortex (B).

within the 12 days before his admission to our hospital. Another possibility is that the narrowing observed in this patient may have been present before the accident, since 10% to 30% of the population have anomalies in their brain sinuses.² Based on the diagnosis of cerebral venous sinus thrombosis and because the patient had hyperhemoglobinemia when he was admitted, we prescribed a drip infusion of low-molecular-weight dextran.

The literature contains few reports describing cerebral sinus thrombosis during high-altitude climbing.^{3–6} Considering that it is very difficult to obtain a definitive diagnosis due to the circumstances of high-altitude climbing, it is possible that many patients may have had this disorder misdiagnosed. Song et al⁵ analyzed their case and previous case reports and concluded that sinus thrombosis at high altitude might not be rare. Recently, Boulos et al⁶ reported a case in which this syndrome developed at 3000 m above sea level. When a climber has a procoagulation disorder (such as protein C deficiency, fibrinolytic enzyme deficiency, or antiphospholipid antibody syndrome) or atherosclerosis, sinus thrombosis can develop even at a moderate altitude.⁶ Jha et al⁷ analyzed cases describing cerebral infarction at high altitudes and proposed that hemoconcentration and hypercoagulability were major risk factors. In the present case, we found no coagulation disorder in our rou-

tine laboratory examinations; however, it is possible that further assessment regarding coagulation factors and fibrinolytic enzymes might reveal some abnormality in coagulation and fibrinolysis cascades.

Climbers staying at high altitudes for weeks have several risk factors for thrombosis. Red blood cell counts and hemoglobin concentrations increase to maintain oxygen transport in the hypobaric environment. Despite large amounts of body water loss while climbing, fluid intake is restricted by the difficulty of obtaining water. Mountaineers easily fall into high-altitude dehydration in spite of it being a well-known phenomenon. These conditions induce hyperviscosity in the circulating blood. Garvey et al⁸ demonstrated that erythremia in the setting of a hypobaric environment provoked procoagulability in a rhesus monkey model. Cerebral edema induced by vasodilatation under hypoxic conditions elevates intracranial pressure and compromises cerebral blood flow. Venous low pressure circulation may be easily interrupted by the increased intracranial pressure. The climber described in this report had all of these risk factors.

Symptoms of cerebral sinus thrombosis depend on the vessels involved and the chronicity of development. Although the onset may be acute, the symptoms slowly progress over days or weeks, according to the growth of

Figure 2. Angiography performed on the second day after admission. Narrowing of superior sagittal (A) and right transverse (B) sinuses was observed.

thrombus. Also, venous collateralization and recanalization modify the progress of symptoms. Hyperintensity on T2-weighted MRI images reveals cerebral infarction and venous edema in the area involved. The parenchymal signal change is typically the subcortical and/or overlying cortex. The symptoms and radiographic findings of cerebral sinus thrombosis and those of cerebral infarction are similar. The differential diagnosis may be based on rapidity of manifestation of symptoms and on the damaged area involved. Cerebral infarction provoked by cerebral sinus thrombosis could not be explained by the occlusion of an artery. However, it is possible that there are many cases in which establishing the differential diagnosis is clinically difficult. High-altitude cerebral edema also presents similar neurological dysfunctions as observed in cerebral sinus thrombosis.¹⁰ Without radiographic data, it may be impossible to differentiate these syndromes. In the setting of a high-altitude expedition, it is highly possible that attending doctors or accompanying members suspect high-altitude cerebral edema first when a member presents with neurological disorders after a long stay at high altitude. In the present case, the accompanying colleagues suspected high-altitude cerebral edema and gave dexamethasone to the patient.

The therapy given our patient after admission to a hospital in his home country was mostly rehabilitation. In the acute phase, treatment of cerebral thrombosis consists of (1) correction of dehydration; (2) thrombolysis by urokinase or tissue plasminogen activator; (3) prevention of further coagulation with anticoagulant or antiplatelet drugs; (4) intracranial pressure reduction with osmotic diuretics, steroids, and oxygen, and (5) for extreme cases, surgical craniotomy. However, it seems unlikely that a climber who had experienced thrombosis during a high-altitude expedition would be transported to an appropriate hospital immediately after the onset of symptoms.^{1,11} Possible emergency treatments on site are oral or parenteral hydration and therapy for cerebral edema by oxygen, steroids, and careful use of diuretics. In the chronic phase, treatment of these patients is directed mainly toward rehabilitation and prevention of secondary disorders, such as pneumonia and decubiti.

The patient in this case was rescued by the strenuous and dedicated actions of his 3 colleagues. However, rescuing a patient with severe neurological symptoms at

high altitudes is extremely difficult. To prevent this syndrome, climbers have to take appropriate amounts of fluids and minimize the duration of their high-altitude stay. Climbers should know that polycythemia induced by dehydration increases the risks of thrombosis at high altitudes.

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