Subarachnoid Hemorrhage Presenting an "Abnormal Movement"

—Case Report—

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Abstract

A 61-year-old male presented with subarachnoid hemorrhage manifesting an abnormal movement as the initial symptom. The movement was rhythmic with phases: tongue protrusion with eyes wide open, and tongue retraction with eyes closed, lasting for about 10 minutes. Neuroradiological methods identified a small aneurysm as the origin of the hemorrhage. The movement never after recurred clipping the aneurysm and clot drainage. Transient increase in the intracranial pressure was thought to be the cause.

Key words: subarachnoid hemorrhage, abnormal movement

Introduction

Involuntary movement rarely occurs in patients with subarachnoid hemorrhage. We describe a patient presenting with a transient abnormal involuntary movement after subarachnoid hemorrhage. The movement is described in detail and possible mechanisms are discussed.

Case Report

A 61-year-old right-handed male was well until February 20, 1991, when he suffered onset of severe headache and was rapidly transferred to our hospital. His personal and family history included no episodes of epilepsy, hysteria, or habits of drug abuse. On admission, he was semicomatose, but his general condition was stable. Laboratory data were all within normal limits. Neurologically, no laterality was observed. Several hours later, an abnormal two-phase involuntary movement occurred. In the first phase, he protruded his tongue straight out with the eyes open, knitting his brows, and both eyes directed slightly upward. In the second phase, he retracted his tongue with the eyes closed tightly. The orbicular oral muscle was not contracted while the ocular muscle contracted tightly (Fig. 1). This two-phase movement appeared rhythmically with 1 Hz, lasting for about 10 minutes. The movement stopped after administration of 10 mg diazepam. During the movement he was unconscious without verbal response. After he regained consciousness, the abnormal movement did not recur.

Computed tomographic (CT) scans immediately after admission revealed subarachnoid hemorrhage mainly in the basal cistern and bilateral Sylvian fissures (Fig. 2 upper). There were no abnormalities in the intracerebral areas. Angiograms revealed a small aneurysm at the A1 segment of the left anterior cerebral artery (Fig. 2 lower).

His consciousness improved to Hunt and Kosnik grade 2 during the day of admission, so he underwent surgery. Left frontotemporal craniotomy through the left pterional approach exposed the small aneurysm, and the neck of the aneurysm was clipped. The subarachnoid clots were removed by cisternal drainage for 2 weeks postoperatively.

His postoperative condition was good except for lower paraparesis which lasted for 2 months, possibly due to vasospasm of the bilateral anterior cerebral arteries which was confirmed by angiography 1 week after operation. However, no in-
Voluntary movement occurred after surgery. He was discharged ambulatory 2 months later.

Discussion

Involuntary movements following organic central nervous system disorders are rare. Sequelae to subarachnoid hemorrhage include chorea and athetosis. In both cases, the mechanisms causing the involuntary movements were probably circulatory disturbance or ischemic changes in the basal ganglia, especially in the caudate nucleus. However, the abnormal movement seen in our case has never been described. This abnormal movement occurred only in the territory of cranial nerves, i.e., facial and hypoglossal nerves without involvement of the pyramidal and extrapyramidal tracts. The first phase, knitting his brows with the eyes open, tongue protrusion, and upward gaze, indicates activation of the facial and hypoglossal nerves and tectal area. In the second phase, eye closing and tongue retraction indicates activation of the facial nerve. However, the oral muscle was not contracted tightly. The eye closing and knitting brows occurring alternately were related to the facial nerve. Generalized convulsive seizure was associated with subarachnoid hemorrhage in 26% of 100 consecutive ruptured aneurysm cases. However, the movement in our case, although occurring rhythmically and repeatedly, was localized only in the face and tongue, and so was not a type of seizure.

The lesion responsible is difficult to localize in the brainstem. However, considering the characteristic features of the movement, reciprocal and alternate, and responsible cranial nerves, the brainstem may have been stimulated resulting in the movement occurred. Why the whole brainstem was not affected remains unclear, but differences in the vulnerability of the structures including cranial nerves might be a factor. Neuroradiological studies including CT and angiography showed no abnormal findings in the intracerebral regions. No magnetic resonance imaging or single photon emission CT studies were obtained.

Possible mechanisms for such an abnormal move-
ment are as follows: 1) a rapid increase in intracranial pressure after subarachnoid hemorrhage, especially in pressure on the brainstem, and 2) ischemic changes after subarachnoid hemorrhage. As 1) the abnormal movement occurred acutely and temporarily, and 2) it disappeared when he recovered consciousness, the former explanation is more probable, because ischemic changes can occur several weeks after onset of subarachnoid hemorrhage as well as in the acute stage. In addition, this elderly patient may have had an underlying subclinical condition such as an arteriosclerotic circulatory or metabolic disturbance in the extrapyramidal system.

While no direct evidence for the location of the movement and the mechanism was found, this is the first case of abnormal movement associated with subarachnoid hemorrhage.

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References


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