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"Sunken Brain Syndrome"

—Case Report—

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Abstract

An elderly male became semicomatose, with aggravation of right hemiparesis, due to massive air collection in the subdural space immediately after craniotomy for wrapping of an aneurysm. A subdural tap disclosed very low pressure within the cranial cavity. The neurological manifestations were not caused by tension pneumocephalus, but were presumed to be secondary to downward deviation of the brain parenchyma. Rapid resolution of the neurological signs was achieved by replacement of the air with saline. In this pathological condition, which has been referred to as "sunken brain syndrome," neurological symptoms develop secondarily to downward displacement of the brain parenchyma. This complication should be kept in mind as a neurosurgical risk in patients with brain atrophy.

Key words: brain atrophy, cerebral infarction, intracranial pressure, neurosurgical complications, tension pneumocephalus

Introduction

It has recently been recognized that there is a high incidence of complications due to cerebral ischemia following surgery for intracranial aneurysm incidentally detected in patients with cerebral infarction.[5] Moreover, in patients with brain atrophy, there is a risk of such complications as intracerebral hemorrhage and tension pneumocephalus with neurosurgical procedures that entail drainage of a large amount of cerebrospinal fluid.

Recently, the author operated on an intracranial aneurysm that had been detected incidentally in a patient with cerebral infarction. The postoperative course was complicated by deterioration of consciousness and worsening of a preoperative hemiparesis due to the collection of a large amount of air in the subdural space. These neurological manifestations were not caused by tension pneumocephalus, but were considered to be secondary to downward deviation of the brain parenchyma in the cranial cavity. The symptoms abated following replacement of the air with saline. There have been no previous reports concerning this pathological process.

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Case Report

A 68-year-old normotensive male who had undergone a right pneumonectomy for tuberculosis 30 years earlier had been in good health until February 23, 1986. At that time he noticed clumsiness of his right hand and, on the following day, presented at a nearby hospital. Left carotid angiography revealed no stenotic or occlusive lesions, but demonstrated an aneurysmal dilatation of the left internal carotid artery at the branching of the posterior communicating artery. He had not experienced sudden onset of severe headaches. He was transferred to Tokyo Metropolitan Fuchu Hospital on March 1, 1986 for clipping of the aneurysm.

On admission, the physical examination disclosed no abnormalities other than clumsiness of the right hand and the state after the right pneumonectomy. Results of laboratory studies were all within the normal range. A computed tomographic (CT) scan revealed only moderate cortical atrophy (Fig. 1). Although the aneurysm did not appeared to have ruptured, he underwent surgery on April 11, 1986.

Under general anesthesia with nitrous oxide, oxygen, and fluothane, he was placed in the supine position with the head turned to the right. The left chiasmatic cistern was approached by separation of

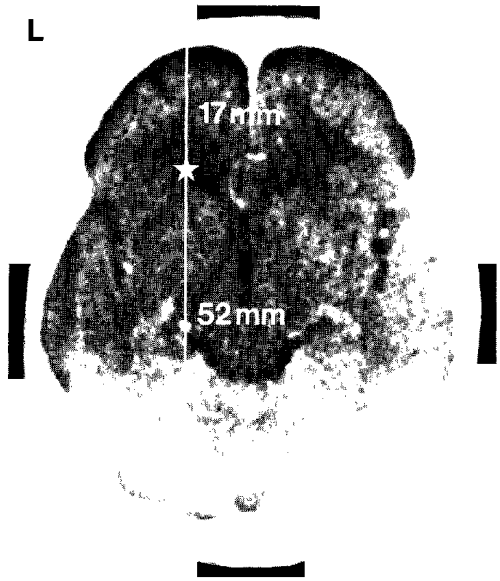

Fig. 1 Preoperative CT scan showing moderate cortical atrophy. The distances of the upper lateral corner of the anterior horn of the lateral ventricle from the top and bottom of the inner table of the skull are 17 and 52 mm, respectively.

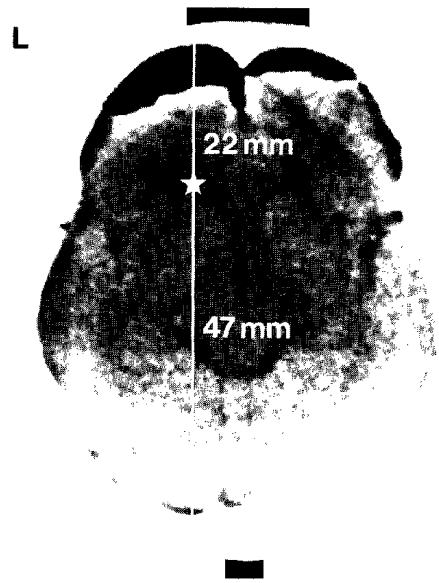

Fig. 2 Postoperative CT scan demonstrating a large volume of air and fluid levels in the subdural space. The anterior horn is 22 mm from the top and 47 mm from the bottom of the inner table of the skull, indicating that the brain parenchyma has sunk within the cranial cavity.

the Sylvian fissure. No evidence of rupture was observed in the aneurysmal dilatation of the internal carotid artery. Because the posterior communicating artery was found to originate at the apex of the aneurysmal dilatation, neck clipping was abandoned in favor of wrapping with muscle pieces. The surgical procedure took 2 hours and 55 minutes. Systemic arterial hypotension was not observed during the period of anesthesia.

His postoperative recovery was slow; he was unable to follow verbal commands until 1 hour and 20 minutes after withdrawal of the anesthetic agents. In the intensive care unit, where he was placed on his back with his head elevated 30 degrees, he underwent progressive deterioration of consciousness and was semicomatose within 1 hour. He also developed right hemiplegia.

An emergency CT scan demonstrated a massive quantity of subdural air and fluid levels over bilateral frontal lobes, more on the left side. The CT scan also disclosed downward deviation of the cortical mantle and the lateral ventricles (Fig. 2). The presumptive diagnosis was tension pneumocephalus, and a percutaneous subdural tap of the left frontal region was performed at the bedside with using Aoki's subdural needle for adults (Muraishi Co., Ltd., Japan). Although the needle penetrated the dura mater, no escape of air was observed. Needle aspiration with a syringe yielded first air and then thin, bloody cerebrospinal fluid. Because it was judged that his subdural pressure was very low or even negative, his head was lowered to a flat position and the air was replaced with 20 ml of saline. Shortly after this procedure, he rapidly regained consciousness and gradually attained the preoperative extent of right-side mobility.

A CT scan obtained after the subdural tap showed reduction of air and elevation of the cortical mantle and the lateral ventricles (Fig. 3). His subsequent clinical course was uneventful.

Discussion

As the indications for neurosurgery increase, the problem of postoperative complications grows more complex. It is clear that individuals with cerebral atrophy[1] or infarction are at risk for postoperative complications. Recently, Kitahara *et al.*[5] reported a high incidence of complications following surgery for unruptured intracranial aneurysms in 20 patients with known cerebral infarction. Seven of their patients suffered worsening of neurological symptoms due to cerebral ischemia, and in two cases this complication resulted in death. In Addition, among five

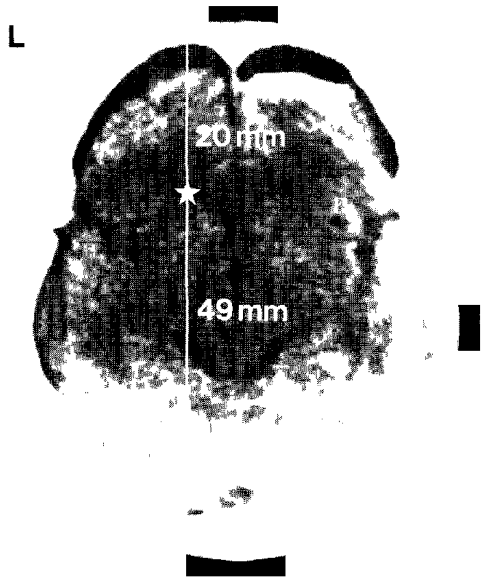

Fig. 3 CT scan obtained after replacement of the air with 20 ml of saline. The anterior horn is 20 and 49 mm, respectively, from top and bottom of the inner table of the skull, indicating elevation of the brain parenchyma.

patients who appeared to have substantially recovered by the time of discharge, four had intracerebral hemorrhages and one had an intraventricular hemorrhage.

Intracranial gas accumulation resulting in neurological manifestations — tension pneumocephalus[2,3,6] — is a well known, although infrequent, complication of neurosurgery. There are several hypotheses concerning its pathomechanism, and each suggests that gas exerts a mass effect within the cranial cavity.[2,3,6] The patient presented here, a 68-year-old male with a cerebral infarction and brain atrophy, underwent intracranial exploration for an unruptured, incidentally discovered aneurysm. Unexpectedly, his postoperative course was marked by delayed recovery from anesthesia and then was complicated by progressive deterioration of consciousness and worsening of right hemiparesis. Despite a massive accumulation of air in the subdural space according to postoperative CT, a subdural tap revealed very low intracranial pressure, which ruled out tension pneumocephalus. His neurological symptoms rapidly resolved after replacement of the air with saline.

In this case it appears that the patient's symptoms were attributable to downward displacement of the brain parenchyma in combination with profound intracranial hypotension. This condition, which is graphically described as "sunken brain syndrome," can be appreciated by comparison of the position of the anterior horn of the lateral ventricle on successive CT scans — preoperative (Fig. 1) and before (Fig. 2) and after (Fig. 3) replacement of the air with saline. The postoperative 30-degree head elevation, which is routinely employed by Durward *et al.*,[4] may have contributed to the patient's neurological deterioration by further lowering the intracranial pressure.

Profound intracranial hypotension associated with accumulation of air is considered to play an important role in the development of "sunken brain syndrome." The resultant downward deviation of the brain parenchyma might lead to compression of the brain against the skull base, shifting of the brain stem, or vascular compromise. Future investigations will no doubt clarify the processes involved in this condition.

As demonstrated in the case reported here, this complication can be successfully treated by filling the cranial cavity with saline. In addition, it seems preferable to keep the patient's head level with the body, rather than elevating it. Finally, it should be noted that, in patients with brain atrophy or infarction, a delay in recovery from anesthesia may portend the development of "sunken brain syndrome."

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