Unusual Posttraumatic Porencephaly

—Case Report—

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Abstract

An unusual case of posttraumatic porencephaly preceded by neither overt cerebral contusion nor hemorrhage is reported. The cerebral cortex just above the porencephalic cyst was found intraoperatively to be partially herniated into a fracture line, while the cortex elsewhere was completely intact. The porencephalic cyst communicated with the lateral ventricle. Apparently, brain herniation and the cyst-ventricle communication can be causative factors in the occurrence and growth of post-traumatic porencephaly.

Key words: head injuries, porencephaly, fracture, brain herniation

Introduction

Acquired porencephaly can be caused by various factors such as trauma, infarction, hemorrhage, and focal encephalitis destroying cerebral tissue.^{3,6,10,11)} The development of posttraumatic porencephaly is usually preceded by cerebral contusion and/or hemorrhage causing severe focal damage of the brain.

Here, we report an unusual case of posttraumatic porencephaly. In this case, although computed tomographic (CT) scanning demonstrated no evidence for destruction of brain parenchyma, such as cerebral contusion and/or hemorrhage in acute to subacute stages of the head trauma, porencephaly did develop later. The mechanism by which this unusual development of posttraumatic porencephaly occurred is discussed.

Case Report

A 33-year-old male was hit on the frontal region during a motor-cycle accident in September, 1988. He suffered from loss of consciousness for a few minutes. On admission, he was alert and well oriented. The physical examination was unremarkable except for cerebrospinal fluid (CSF) rhinorrhea. Neurological examination showed no deficit. A

Fig. 1 Bone-window CT scan showing the left frontal bone fractures involving the inner table of the frontal sinus (arrow).

bone-window CT scan showed left frontal bone fractures involving the inner table of the left frontal sinus (Fig. 1). CT scans revealed the subdural space contained a little air, but there was no abnormality in the cerebral parenchyma. The CSF rhinorrhea was conservatively treated with antibiotics and disappeared 5 days after the trauma. He showed no symptom or sign of meningoencephalitis. Serial CT scans detected no abnormalities of brain parenchyma (Fig. 2). One month after the trauma, he was discharged without neurological deficit. Afterward, his general condition was uneventful.

In February, 1989, 6 months after the trauma, he complained of headache. A precontrast CT scan showed a wedge-shaped cystic lesion in the left frontal lobe with a slight mass effect (Fig. 3A). T₁-weighted magnetic resonance (MR) imaging revealed that the cystic lesion communicated with the lateral

Fig. 2 Precontrast CT scans taken 2 weeks (A) and 1 month (B) after the head trauma, showing no abnormality in the brain parenchyma.

Fig. 3 A: Precontrast CT scan at the second admission showing a wedge-shaped cystic lesion in the left frontal lobe with a slight mass effect. B: T₁-weighted MR image showing the cyst communicating with the lateral ventricle, but no opening into the subarachnoid space.

ventricle, but did not open into the subarachnoid space (Fig. 3B). Left internal carotid angiograms demonstrated slight displacement of the anterior cerebral arteries to the right across the midline.

A left frontal craniotomy was performed to explore the frontal lobe toward the anterior cranial fossa. A transverse tear in the dura about 1 cm in length was disclosed through which the left frontal lobe cortex had partially herniated into a fracture line of the inner table of the frontal sinus. The herniated portion of the left frontal lobe was discolored and appeared to have undergone cicatricial change. The other part of the cortex was apparently intact (Fig. 4). The herniated brain was resected and the dura tear repaired with lyophilized dura. A corticotomy of the left frontal lobe adjacent to the herniation was performed. The cyst was open-

ed and was found to communicate with the anterior horn of the left lateral ventricle. A cyst wall biopsy showed only brain tissue with slight gliosis. There was no epithelial lining. The cystic lesion was therefore diagnosed as porencephaly. The cyst fluid was identical to CSF.

Postoperative course was uneventful. Although postoperative CT scans showed no changes, the headache was remarkably relieved. Follow-up serial CT scans detected no change in the porencephaly size.

Discussion

Acquired porencephaly frequently results from various pathological conditions of the cerebral parenchyma, such as infarction, hemorrhage, focal encephalitis, contusion, and degenerative disease.^{3,6,10,11)} The essential factor for the development of porencephaly is focal destruction of the brain tissue. In our case, the clinical course and serial neuroradiological findings did not indicate a progressive destructive process in the brain tissue. Intraoperatively, the frontal cortex was found to be partially herniated into a fracture line, but there was no evidence of contusion in other cortex areas. A possible precursor of the porencephalic cyst was a cavity in the white matter, probably formed by a negative pressure difference between the paraventricular white matter and the intraventricular CSF at the time of head injury, thus sparing the cortex.⁴⁾ Serial CT scans, however, detected no abnormalities in the brain parenchyma during the first

Fig. 4 Intraoperative photograph showing discolored left frontal lobe cortex herniating into a fracture of the inner table of the frontal sinus (arrowheads). The cerebral cortex (C) around the herniation (H) appears to be intact.

hospitalization. A delayed infection secondary to the frontal sinus fracture is unlikely to have caused the porencephaly, as no symptoms or signs of meningo-encephalitis were observed throughout the first hospitalization and after discharge. Intraoperatively, the only abnormal finding apart from the subcortical cavity was the brain herniation into the frontal bone fracture. This brain herniation is therefore a possible initiator of the brain tissue destruction process.

The possible pathogenesis of posttraumatic syringomyelia provides indicators to the mechanism of occurrence and growth of the posttraumatic porencephaly in our case. Posttraumatic arachnoiditis is postulated to encroach upon the segmental blood supply to the spinal cord, causing cystic myelomalacia to develop with subsequent formation of an intramedullary cavity.^{1,7-9)} Enlargement of this cavity is considered to occur by various mechanical factors such as stretching of the cord. The spinal cord is relatively fixed at the trauma site due to arachnoidal adhesion, so the cord is stretched by vertebral movement. Repeated neck movement slowly causes expansion of the cavity.^{2,9)}

The postulated mechanism for the development of posttraumatic porencephaly in our case is as follows. The brain herniation into the fracture involved the cortical segments of the long perforating arterioles. Consequently, chronic hypoperfusion of the white matter caused ischemic encephalomalacia to develop slowly. The encephalomalacia, probably followed by cystic degeneration, was the precursor of the porencephalic cyst (Fig. 5A). The enlargement of the cyst is presumably due to mechanical factors. As the brain is fixed at the herniation site, the cerebral parenchyma around the cyst is stretched by the pulsation of the brain, increasing the intracavitary pressure (Fig. 5B). This caused enlargement of the cyst and the communication with the lateral ventricle, with a pulsatile flow of CSF from the ventricle into the cyst. The CSF flow may also have increased the intracavitary pressure, contributing to the development of headache as well as further enlargement of the cyst. Although such a destructive process may not occur in all patients with a brain herniation into a fracture line, this hypothesis does explain the formation of posttraumatic porencephaly in our case.

Leahy and Singer⁵⁾ emplaced a cystoperitoneal or a ventriculoperitoneal shunt in cases of expansive porencephaly with or without hydrocephalus. Xiang and Sheng-yu¹²⁾ opened porencephalic cysts to improve symptoms and signs of increasing intracranial pressure. In our case, opening of the porencephalic cyst by a corticotomy released the intracavitary pressure, averting further cyst enlargement and re-

Fig. 5 Illustrations of the occurrence and growth of the porencephalic cyst. A: Brain herniation into the fracture involved the cortical segments of the long perforating arterioles, leading to hypoperfusion of the white matter. Ischemic encephalomalacia developed with subsequent formation of the intraparenchymal cyst. B: The brain is fixed at the herniation site, resulting in stretching of the brain tissue around the cyst with pulsation of the brain. The stretching increases the intracavitary pressure and enlarges the cyst.

lieved the headache.

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