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Obstructive hydrocephalus caused by intraventricular collapse of malacotic brain

Case report

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The authors present the case of a 68-year-old man who experienced acute obstructive hydrocephalus after having suffered an infarction in the occipital lobe. Histopathological and serial neuroimaging examinations revealed that portions of a large disintegrating occipital infarct had entered the lateral ventricle and obstructed the passage of cerebrospinal fluid (CSF). Ventricular drainage was performed for 2 weeks until the patient's hydrocephalus resolved. The CSF initially contained a high concentration of protein (1070 mg/dl), a high leukocyte count of 115 cells/mm^3, and a rich fibrinous exudate.

Findings in the present case indicate that collapse of a periventricular ischemic lesion into the ventricles may sometimes occur not only after cerebral hemorrhage but also after cerebral infarction.

KEY WORDS • cerebral infarction • noncommunicating hydrocephalus • encephalomalacia • ventricle drainage

ACUTE obstructive hydrocephalus can be caused by various brain lesions that disturb the passage of CSF through the ventricular system. To our knowledge, however, there seems to have been no previous reports of obstructive hydrocephalus arising from spontaneous intraventricular collapse of malacotic brain tissue. The peculiar clinical course of the patient whom we describe emphasizes that periventricular ischemic lesions have the potential to collapse into ventricles and may cause obstructive hydrocephalus or adhesive subarachnoiditis.

Case Report

History. This 68-year-old man had complained about a defect in his left visual field for approximately 10 days. This defect was followed by a sudden onset of limb weakness and fecal incontinence. Because the patient had suffered from chronic renal failure due to diabetic nephropathy for many years and had undergone hemodialysis three times per week, his symptoms were first considered to be caused by uremic neuritis, and dialysis was performed immediately. Despite the treatment, his level of consciousness continued to decrease.

Examination. Computerized tomography scanning performed 6 hours after sudden onset of limb weakness revealed a low-density area in the occipital lobe with scattered high-density spots and prominent ventriculomegaly. In addition, the patient's right lateral ventricle and third ventricle were filled with isodense material (Fig. 1). We believed that the occipital low-density area containing high-density spots was a large occipital infarct with scattered petechial hemorrhages and that hydrocephalus had been induced by obstruction of the aqueduct by the intraventricular isodense mass.

Operation and Findings. To treat the patient's obstructive hydrocephalus, we immediately performed ventricular drainage. When the anterior horn of the right lateral ventricle was initially punctured with a ventricular needle and catheter, no CSF was obtained, but a gelatinous mass (5 mm in size) was removed by aspiration. The mass was histopathologically shown to be necrotic brain tissue with a rich fibrinous exudate (Fig. 2). After deeper insertion of the drainage catheter, xanthochromic fluid and fragments of gelatinous tissue squirted out of the catheter at an opening pressure of 250 mm Hg. Postoperative CT scans demonstrated that the catheter had penetrated the ependyma of the right anterior horn and that the tip of the catheter lay in the left posterior horn. No air was detected in the right anterior horn, except for that contained within the catheter (Fig. 3). The patient's CSF initially contained a high concentration of protein (1070 mg/dl), a high leukocyte count of 115 cells/mm^3, xanthochromia, and a few red blood cells (33 cells/mm^3).

These findings indicated that the right lateral ventricle

Abbreviations used in this paper: CSF = cerebrospinal fluid; CT = computerized tomography.

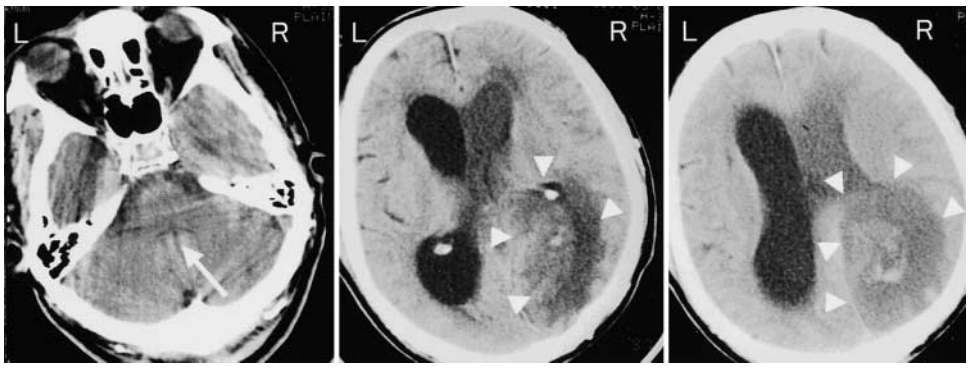

FIG. 1. Computerized tomography scans of the brain obtained 6 hours after admission revealing a large right occipital infarct *(arrowheads)* as well as pronounced ventricular dilation, except for the fourth ventricle *(arrow)*.

was filled with nonliquid matter, which was assumed to be tissue from the necrotic occipital infarct.

Postoperative Course. Within 4 hours after the operation, the patient recovered consciousness. Although the drainage catheter frequently became obstructed by tissue fragments, drainage was maintained by manual flushing with small amounts of sterile saline and by aspiration of fragments through the catheter. It seemed that the left lateral ventricle was also packed with fibrinous exudate and malacotic brain components, based on our observation of material that had been removed through the catheter. After 2 weeks of ventricular drainage, the catheter was removed and the patient experienced no subsequent neurological deterioration. Magnetic resonance imaging performed 1 month after the operation demonstrated that the lateral ventricles were almost normal in size (Fig. 4). Magnetic resonance angiography revealed localized thrombosis in the quadrigeminal segment of the right posterior cerebral artery, which was believed to have caused the large occipital infarction, after which the artery recanalized.

The patient experienced a relatively satisfactory clinical course and, approximately 2 months after onset of symptoms, was discharged home with sequelae of left homonymous hemianopsia and spatial agnosia.

Discussion

Cerebral infarction is occasionally followed by hydrocephalus, which is generally categorized as either acute obstructive hydrocephalus, which is caused by compression and obstruction of the ventricles secondary to a prominent midline shift, or chronic communicating hydrocephalus, which is associated with normal or moderately high intraventricular pressure (normal-pressure hydrocephalus). Although it is considered possible for periventricular malacotic ischemic brain to collapse into the ventricles and induce obstructive hydrocephalus in the same manner after intracerebral hemorrhage, there have been no reports on this type of hydrocephalus. This may be because the ependyma are generally strong enough to prevent malacotic brain from disintegrating into the ventricles, or because brain fragments that are released are generally too fragile and small to obstruct the flow of CSF.

Our patient more or less continued to lead his usual life for approximately 10 days after the onset of infarction before he required hospital admission. The only difference in his daily activities was that he frequently hit his head on obstacles because he experienced a left visual field defect due to obliteration of the right strial region. Because the patient had cataracts as well as multiple old cerebral in-

FIG. 2. Representative photomicrograph showing a section of gelatinous tissue aspirated from the patient's right anterior horn. Necrotic brain tissue with degradation of the neuropil, destruction of neurons, and proliferation of macrophages are findings consistent with the subacute stage of cerebral infarction. Note the rich fibrinous exudate *(arrows)*. H & E, original magnification × 100.

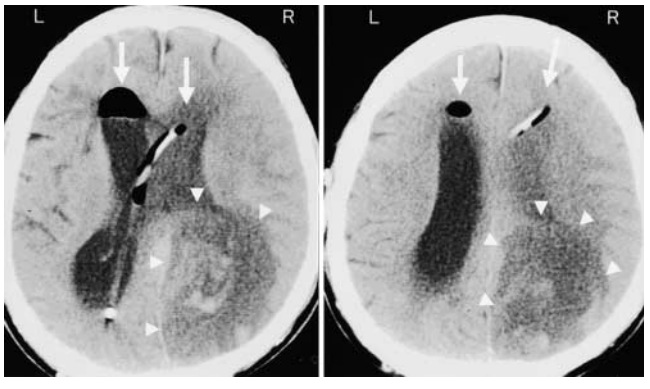

FIG. 3. Postoperative CT scans of the brain demonstrating the draining catheter penetrating the ependyma of the right anterior horn, with the tip in the left posterior horn. There is no air in the right anterior horn, except that inside the catheter *(left arrow on each scan)*, although there is some air in the left anterior horn *(right arrow on each scan)*. *Arrowheads* mark the boundaries of large right occipital infarction.

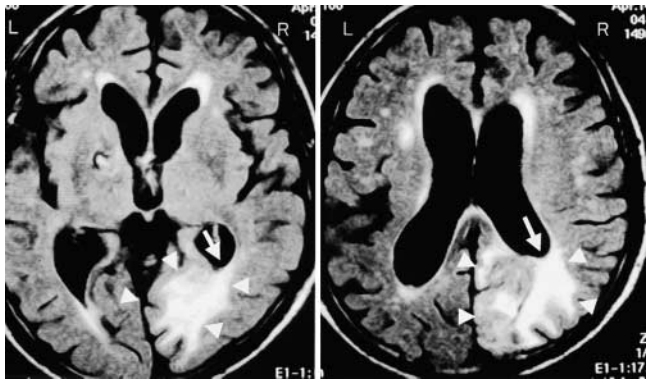

FIG. 4. Axial fast low-angle inversion recovery magnetic resonance images of the brain obtained 1 month after symptom onset clearly demonstrating a large right occipital infarction *(arrowheads)*, as well as the damaged ependyma of the right posterior horn *(arrows)*.

farcts, lumbar disc herniation, and chronic renal failure, he already suffered from visual impairment, moderate disorientation, gait disturbance, and bilateral lower limb numbness. Thus, despite the large size of the new lesion in his occipital lobe, the extent of his neurological deficits was masked.

Okazaki[3] and others[1,2] have reported that infarcts 7 to 10 days old are soft and slightly friable (coagulative necrosis), whereas those 2 to 3 weeks old are friable and mushy, with small areas of cavitation (liquefactive necrosis). After approximately 3 weeks, numerous cavities become evident in the affected parenchyma (the cavitation phase). Therefore, during the liquefactive necrosis phase (10 days–3 weeks after cerebral infarction), lesions may be soft enough to disintegrate spontaneously. In our patient symptoms developed during this period.

Fortunately in our case, the intraventricular brain tissue could be removed easily by aspiration and continuous drainage through the ventricular catheter, because the tissue was degenerating and undergoing spontaneous liquefaction. If the ventricular masses had been more robust and difficult to remove, endoscopic aspiration may have been effective.

Findings in the present case indicate that collapse of periventricular ischemic lesions into the ventricles may sometimes occur, although such a lesion is likely to be less extensive than that observed in our patient.

Conclusions

In this paper we described the clinical case of a patient who suffered from obstructive hydrocephalus after having suffered a right occipital lobe infarction.

Histopathological and serial neuroimaging examinations revealed that malacotic brain from the right occipital lobe had collapsed into the lateral ventricle and obstructed the flow of CSF.

Findings in the present case indicate that collapse of periventricular ischemic brain tissue into the ventricles may sometimes occur after not only cerebral hemorrhage but also cerebral infarction.

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