Spontaneous rupture of a metastatic brain tumor into the ventricular system

Report of two cases

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Two cases are reported in which spontaneous rupture of a metastatic brain tumor occurred into the ventricular system, leading to the dissemination of the tumor contents in the cerebrospinal fluid. The site of rupture was demonstrated by ventriculography in the first case and by surgical exposure in the second. These cases support the assumption that metastatic neoplasms within the brain may rupture into the ventricular system and spread via the cerebrospinal fluid pathways.

KEY WORDS: metastatic brain tumor · cerebrospinal fluid cytology · cerebral ventricles

It has been postulated that metastatic neoplasms within the brain may rupture or shed their cells into the ventricular system, thereby constituting one source of the tumor cells found in the cerebrospinal fluid in almost half of these cases. This hypothesis has also been advanced as one of the possible mechanisms by which meningeal carcinomatosis develops. We have recently encountered two unusual cases that add support to these theoretical considerations.

Case Reports

Case 1

This patient was a 47-year-old man with a history of chronic alcoholism who developed headaches in July, 1968. His family noticed changes in his mental status 4 months later. In December, 1968, he was admitted to a military hospital for evaluation of "syncope," and on January 23, 1969, was readmitted because of an episode of dysarthria, right-sided headaches, left hemiparesis, and left homonymous hemianopsia lasting approximately 24 hours. After a second transient neurological episode on January 29, consisting of apraxia of the left extremities, he was transferred to a neurosurgical center. Here it was noted that he was disoriented and confused, with a severe impairment of memory and a marked tendency to confabulate. In addition, he gradually developed a mild left hemiparesis. An electroencephalogram showed abnormal slowing of wave frequency bilaterally. A brain scan showed unusual retention in the left frontoparietal area. Right carotid arteriography was normal.

The patient was transferred to the Salisbury Veterans Administration Hospital on
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May 5 where an electroencephalogram (EEG) was slightly and diffusely abnormal, and x-ray films of the skull were normal. A neurologist evaluated the patient on May 14 and noted no abnormalities aside from disorientation. However, on June 9, the same neurologist observed mild bilateral papilledema. Another EEG showed left fronto-central suppression in addition to the previous bilateral slowing. Cerebrospinal fluid removed by lumbar puncture was clear and colorless, with 3 leukocytes per cu mm and a protein value of 53 mg%.

Examination. On June 12, the patient was transferred to the Durham Veterans Administration Hospital. He was combative and disoriented, and appeared to be having hallucinations. Mild bilateral papilledema was present. X-ray films of the skull were normal. A brain scan was technically poor because of head movement, but there appeared to be abnormal retention in the left frontal lobe. Bilateral carotid arteriograms also showed changes indicative of a mass in the left frontal lobe.

On June 15, while he was being prepared for a proposed left frontal craniotomy, he suddenly became lethargic and febrile, and developed neck stiffness. A lumbar puncture showed cloudy cerebrospinal fluid under a pressure of 500 mm. There were 300 cells per cu mm, and it was thought that 82% were polymorphonuclear leukocytes. Clinically, it appeared that the patient had a brain abscess in the left frontal lobe that had ruptured into the ventricular system. Antibiotic therapy was begun. Shortly after the lumbar puncture, a ventriculogram was performed through a twist drill hole in the right frontal bone. The cerebrospinal fluid was turbid and slightly yellow, with a pressure of 290 mm. A mass was demonstrated in the left frontal lobe, with a cavity in its center that communicated with the frontal horn of the left lateral ventricle (Fig. 1). In addition, there appeared to be second mass in the left thalamus. Since the possibility of metastatic tumors also existed, the CSF was studied cytologically as well as bacteriologically. Numerous neoplastic cells were found (Fig. 2), and cultures showed no bacterial growth.

Postmortem Examination. The patient was transferred back to the Salisbury Veterans Administration Hospital where he died on August 7, 1969. Postmortem examination disclosed a large adenocarcinoma of the gastric fundus, with metastases in the brain (Fig. 3), adrenal glands, liver, retroperitoneal lymph nodes, and left supraclavicular lymph nodes. There were numerous metastatic neoplasms bilaterally in the cerebrum and cerebellum, and the centers of many were necrotic. The ventriculographic findings were verified. The cranial meninges ap-

![Fig. 1. Case 1. Ventriculograms showing air within the evacuated center of the metastatic carcinoma in the left frontal lobe. Left: Anteroposterior view. Right: Lateral view.](image1)
Case 1

Cytological study of the CSF, showing a clump of tumor cells, some of which are vacuolated and resemble signet rings. Modified Papanicolaou stain, \( \times 550 \).

Fig. 2. Case 1.

A cerebral metastasis, demonstrating some vacuolated signet ring cells. H & E, \( \times 550 \).

Fig. 3. Case 1.

This 47-year-old man had had a left pneumonectomy in 1960 for an adenocarcinoma of the left upper lobe with metastasis to a hilar lymph node. He was admitted to the Durham Veterans Administration Hospital on November 13, 1969, with a 10-month history of diffuse headaches and indistinct vision to his left. On November 9, 1969, he had noted the onset of pain in the lumbosacral region with radiation into both thighs posteriorly. By November 12, he had developed urinary retention for which a urethral catheter was inserted at another hospital.

Examination. There was a left homonymous hemianopsia and early papilledema in the right eye. X-ray films of the skull, and of the thoracic, lumbar, and sacral spine were normal; x-ray films of the chest demonstrated the previous pneumonectomy, but were otherwise normal. A brain scan demonstrated an abnormal area of retention in the

Fig. 4. Case 2. Cytological study of the CSF demonstrating a cluster of reactive giant cells. Modified Papanicolaou stain, \( \times 650 \).

Case 2

peared grossly normal, but they were not studied microscopically. The spinal cord and spinal meninges were not examined.
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right occipital lobe. A right carotid arteriogram was normal, but the posterior cerebral artery was not visualized. A total myelogram was normal except for the appearance and composition of the cerebrospinal fluid, which was yellow and turbid, containing innumerable particles and glistening flecks that resembled crystals and tissue debris in suspension. Its protein content was 76 mg%, and cyto logical examination revealed clumps of cells interpreted as reactive giant cells (Fig. 4).

Operation. On November 17 a metastatic papillary adenocarcinoma (Fig. 5 left) was excised from the right occipital region. The anteromedial surface of the tumor extended into the occipital horn of the right lateral ventricle. Interestingly, microscopic examination of the necrotic center of this neoplasm disclosed spaces having the appearance of cholesterol clefts (Fig. 5 right).

Postoperative Course. Lumbar puncture on November 28 showed the CSF to be clear and colorless. By that date the patient was free of back and leg pain. A cystometrogram was normal and the urinary catheter was removed. The patient was given postoperative radiotherapy to the right occipital area before being discharged from the hospital.

Conclusions

The phenomenon of intraventricular rupture of a metastatic brain tumor had not been recognized previously at this medical center. As far as we can determine, only one other case has been reported in which this occurrence was diagnosed during life. These three cases together lend support to the assumption that metastatic neoplasms within the brain may occasionally rupture into the ventricular system and spread via the cerebrospinal fluid pathways.
References


Received for publication February 16, 1970.
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