Cryptoccal granuloma of the choroid plexus in the temporal horn

Yohsuke Fukami

Follow this and additional works at: https://scholarlycommons.henryford.com/hfhmedjournal

Part of the Life Sciences Commons, Medical Specialties Commons, and the Public Health Commons

Recommended Citation
Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol25/iss3/12

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons.
Cryptoccal granuloma of the choroid plexus in the temporal horn

Yohsuke Fukami, MD*

A man developed sudden symptoms of increased intracranial pressure following a prolonged course of cryptoccal meningitis which appeared to be responding to treatment. A choroid plexus granuloma was found in the dilated and blocked lateral ventricle. The poor prognosis of this type of complication of the cryptoccal meningitis is discussed.

Cryptoccal infection of the central nervous system is a rare disease and when it occurs, the most common site of the involvement is the meninges. Sometimes it also causes granulomatous lesions, either single or multiple, in the brain.

We had a case of cryptoccal granuloma involving the choroid plexus of the lateral ventricle. Since only three similar cases are reported in the English literature, we report a detailed account of our case.

In our patient, the infection caused not only choroid plexus granuloma but also ventriculitis which effectively blocked the temporal horn from the rest of the ventricular system, thus acting as an expanding intracranial mass lesion.

Case report

The patient was a 49-year-old black man who had had intermittent low back pain for the past 5 years. In June, 1975, after he developed pain radiating into his right leg, he was seen by a local orthopedic surgeon, who performed lumbar laminectomy for apparent herniated nucleus pulposus in late November, 1975. Three weeks after the operation, he developed posterior neck pain and blurring of vision and the bilateral papilledema was detected. He was then referred to a local neurosurgeon. The evaluation there, including angiogram, brain scan and EMI scan, was negative except for slight dilatation of the ventricular system. Because of the persistence of his symptoms plus development of confusion, he was transferred to the VA Hospital on Christmas Eve of the same year.
On admission, his temperature was 98.6°F (37°C). He was somnolent, confused and disoriented and his neck was stiff. There was marked bilatera1 papilledema with flare hemorrhage. Neurological examination was otherwise nega-
tive. Because of the extensive scar in the lumbar area due to previous surgery, the spinal fluid was obtained through cisternal puncture. This revealed *Cryptococcus* on India ink preparation as well as fungal culture. Spinal fluid was xanthochromic and glucose count was 11/100 ml. He was treated with Amphotericine B, 10 mg once a day and 5-fluorocytosine 1 gm, three times a day. A week later, amphotericin B was increased to 30 mg, once a day. This dosage was maintained and the patient's condition gradually improved over the next month. His headaches had subsided and confusion had cleared, but he had continuous nausea and vomiting. Blood urea nitrogen (BUN) and creatinine remained somewhere near 35 mg/100 ml and 2.5 mg/100 ml, respectively. Electrolytes and liver function tests were within normal limits. Chest x-ray films and numerous sputum specimens failed to show cryptococcal infection in the respiratory system.

Spinal fluid on January 30, 1976, was xanthochromic with 55 cu mm lymphocytes. Glucose was 40 mg/100 ml. India ink and fungal cultures were negative. By this time, his papilledema had resolved.

Spinal fluid on March 3, 1976, became clear and colorless with only three lymphocytes/cu mm. Glucose was 65 mg/100 ml. India ink preparations and culture were again negative.

By early April, approximately four months after the onset of meningitis, he was completely asymptomatic and alert, without confusion. Headache, nausea, and vomiting had been resolved. Hemoglobin and hematocrit remained stable without any significant change of the hematopoietic system throughout the treatment with amphotericin B.

By April 21, 1976, the patient had received a total dose of 2,600 mg of amphotericin B and 120 mg of 5-fluorocytosine. Spinal fluid on May 6 showed only 12 cells and glucose was 54 mg/100 ml. A tentative decision was made by the infectious disease department to stop the medication when the total dose of amphotericin B reached 3,000 mg.

Shortly after medication was discontinued, he developed recurrence of the headache and nausea. Within two days, his condition rapidly deteriorated and he became semicomatose with right hemiparesis. The neurosurgical service was then consulted. Brain scan was negative, but EMI scan (Figure 1) showed marked midline shift with a mass lesion in the left temporal lobe which was radiolucent in appearance without contrast enhancement. Carotid angiogram showed a large avascular temporal mass on the left side.

In the operating room on May 13, after a left temporal burr hole was made, we encountered a cystic abscess by probing with a ventriculostomy needle. We removed 25cc of xanthochromic fluid and inserted an Ommaya reservoir for later injection of amphotericin B. The cyst fluid showed 750 white blood cells/cu mm, 80% lymphocytes/cu mm. No fungus was identified. The patient's condition improved postoperatively. He became awake and oriented but remained somewhat hemiparetic on the right side. The Ommaya reservoir was tapped every fourth day and approximately 10-15 cc of fluid was removed and 0.5 mg of amphotericin B was injected each time into the abscess through the Ommaya reservoir.

However, two weeks later, he became again stuporous and slightly dysphasic and his right hemiparesis increased. A ventriculogram showed obstruction of the body of the left lateral ventricle with a large mass in the temporal area. He was taken back to the operating room on May 25, and the left temporal craniectomy was performed. We entered what we had previously thought was the cavity of an abscess through the middle temporal gyrus. We found this to be the blocked and dilated temporal horn of the lateral ventricle, which was filled with gelatinous purulent fluid. In the middle of this mucoid material was the choroid plexus which showed a granulomatous change. This choroid plexus granuloma was totally removed. Histological examination showed numerous encapsulated organisms, *Cryptococcus neoformans*. (Figure 2) The right lateral ventricle was also tapped through a frontal burr hole but spinal fluid obtained from this side was clear and colorless containing only two cells.

After this procedure, the patient recovered slowly and eventually became awake and fully ambulatory. His right hemiparesis disappeared, but he remained slightly dysphasic and had right homonymous hemianopsia. He also showed inappropriate behavior, wandered around in the hospital, got lost and could not find his way back to his room on several occasions.

Spinal fluid was obtained through cisternal approach on several occasions postoperatively. The cerebrospinal fluid, as time progressed, showed slow but steady improvement in appearance, cell count, glucose and protein content. For example, spinal fluid on July 16, two months postoperatively, was clear and had only one lymphocyte and 40 mg/100 ml glucose. *Cryptococcus* was never demonstrated in these spinal fluids.

EMI scan on July 7 showed disappearance of a previously seen mass lesion and no midline shift. The patient's condition during the first three months postoperatively was one of slow improvement. His dysphasia and hemiparesis had completely cleared by the beginning of August, although he continued to have right field cut and a severe degree of inappropriate behavior.
Cryptococcal granuloma

However, in the middle of August, the patient's condition again deteriorated fairly rapidly and in a few days he became disoriented and stuporous. Nausea and vomiting also recurred. EMI scan on August 16 revealed slight left-to-right shift with dilatation of the left frontal horn. The patient's family refused further surgical procedure and the patient died on August 21, 1976.

**Autopsy**

Major autopsy findings were limited to the brain. The lungs showed bilateral pulmonary congestion but there was no evidence of cryptococcal infection in the lungs or other organ systems, except the central nervous system.
The brain was examined by Dr. Jose Bebin. It weighed 1,420 gm. The leptomeninges were generally clear however they were thickened and cloudy with slight yellowish discoloration at the base of the brain. There were bilateral tonsillar and a right uncal herniation. There was a well-healed operative scar over the left middle temporal gyrus measuring 3 x 1 cm.

Sectioning of the brain (Figure 3) showed the left frontal horn was dilated and the septum pellucidum was displaced from left to right. The entire ventricular system appeared to show changes of ventriculitis with thickening of the wall and the left foramen Monro was obstructed. In addition, the right lateral ventricle contained thick, yellowish-brown mucoid material. The right choroid plexus was thickened and granulomatous. This material from the right lateral ventricle contained large numbers of cryptococci with many budding forms (Figure 4). The left thalamus had a small area of softening.

The aqueduct, brain stem and the cerebellum showed no focal abnormality.

**Discussion**

Cryptococcosis is the most common fungal infection of the central nervous system. *Cryptococcus* is widely distributed in the environment but most commonly isolated from the soil contaminated by the droppings of fowl. This may account for higher incidence of the infection among individuals who handle birds.4

The most common site of primary infection is the lung, but about 70% of the cases show involvement of the central nervous system at the time of the initial diagnosis. Thirty percent to 50% of proven cryptococcosis cases are associated with debilitating diseases or immuno-suppressive conditions. Many cases of cryptococcal granuloma in the central nervous system have been reported previously.5 Focal granulomata have
Cryptococcal granuloma

Figure 3
The dilated left frontal horn due to obstruction of the foramen Monroe. Another granuloma of the choroid plexus in the right side.
Cryptococci from the right lateral ventricle.

been described in the cerebrum, the cerebellum, and the spinal cord. They usually form cysts with firm capsules and are filled with xanthochromic fluid, but some reports describe a solid mass containing necrotic, foamy, soap-suds-like tissue. Some of these granulomata were reported to be successfully removed, if there was no communication with the ventricular system.

On the other hand, there are only three cases of intraventricular cryptococcal granuloma reported in the English literature.

The case reported by Manganiello (Medical College of Georgia) lived 10 days postoperatively.

Vijayan, of the Sacramento Medical Center, California, reported another patient who remained unresponsive postoperatively and died 10 weeks later.

Both of these cases were treated surgically and the granulomatous choroid plexus was removed.

The third case reported by Shelby, of Cook County Hospital, had cystic granuloma with communication with the lateral ventricle and this patient also died three days after the surgery.

We believe our patient to be the fourth reported case of intraventricular cryptococcal granuloma.

There are a few points of interest in our case compared to the three cases previously reported.

First of all, our patient developed blockage of the ventricular system presumably due to the ventriculitis around the infected choroid plexus. Hence, the left temporal
Cryptococcal granuloma

horn was isolated from the rest of the ventricular system, became dilated and acted as an acutely expanding intracranial mass lesion.

Secondly, the port of entry of infection was vague in this case. He had lumbar laminectomy by an orthopedic surgeon three weeks prior to the onset of the meningitis, but no apparent complication was reported. However, our patient worked at a chicken farm in rural Mississippi for a number of years prior to the onset of the disease. Even though the pulmonary cryptococcal infection was never established clinically or at the time of autopsy, the fact that he had bilateral choroid plexus involvement clearly suggests hematogenous spread. Although the pulmonary lesion was not found in many cases of cryptococcal meningitis, the lung is commonly thought of as the port of entry. This concept can be probably applied to our case, also.

Thirdly, the postoperative course of our patient was remarkably good even though he did succumb later from extensive infection. Following the craniectomy and removal of the infected choroid plexus, he became awake and fully ambulatory, and his clinical improvement was supported by serial spinal fluid examinations and EMI scans until his condition took a rapid downturn at the end of the 13th postoperative week.

We had hoped that we could present at this meeting of Dr. Knighton's alumni residents the first successful treatment of this condition because the patient did so well after the surgical procedure for nearly three months. We regret that we were not able to achieve this goal.

References

