Aseptic meningitis due to spontaneous rupture of craniopharyngioma cyst

Case report

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The occurrence of aseptic meningitis associated with extravasation of the contents of a craniopharyngioma cyst has been reported only rarely. Russell and Pennybacker referred to three cases of postoperative aseptic meningitis following surgery for craniopharyngioma, presumably due to the escape of the contents of the cyst into the subarachnoid spaces during surgery. They described three other cases of apparent rupture of a suprasellar cyst (craniopharyngioma) in association with head trauma.

Spontaneous rupture of a craniopharyngioma cyst is even more rare. Kaemmerer described a case of spontaneous "meningitis" in a patient with suprasellar calcification; as "meningitis" developed, there was a reduction in the size of the cyst on sequential angiograms. We are reporting a second such case.

Case Report

A 21-year-old freshman medical student was seen in the ENT Department, August 1970, because of recent headache presumably due to sinusitis. Skull and sinus films revealed a calcified mass measuring 11 x 12 mm located just above the sella turcica and assuming a configuration fairly characteristic of a craniopharyngioma (Fig. 1). Neurological examination and history revealed no abnormalities. There was no evidence of endocrine disorder other than that suggested by his height (5 ft 6 in.) compared to that of his father (6 ft 2 in.), paternal grandfather (5 ft 11 in.), and maternal grandfather (5 ft 9 in.). The headaches subsided, and no treatment was advised. Follow-up skull films in October, 1970, and 1971 revealed no change in the calcified mass. Visual fields remained normal. Annual follow-up was recommended, but no treatment or further investigation was felt warranted.

On January 26, 1972, the patient was examined because of tinnitus of several days' duration and stiff neck and morning headache relieved by aspirin for several weeks.
There was a small area of decreased sensation in the face above the right upper lip and in the right cheek, which had persisted for 2 to 3 days; it was described as an area of approximately $2 \times 3$ cm with only partial loss of sensation.

On the morning of January 28, 1972, while presenting a seminar paper, he experienced a very disagreeable pungent odor, which cleared after a few moments. On that same evening while strolling with a friend, he again experienced the same penetrating odor. He felt dizzy, attempted to speak, but found it very difficult to think clearly or express himself. His friend observed that he breathed deeply and rapidly while uttering incoherent words. His hands began to tremble followed by jerking of his head, and he lost consciousness. This lasted a few minutes after which he appeared to sleep.

The patient was admitted that evening to the University Hospital. He was lethargic and complained of severe headache and leg pains. The neck was stiff, Kernig and Brudzinski's signs were positive. A lumbar puncture was done; the CSF pressure exceeded 300 mm H$_2$O. The spinal fluid was brownish, turbid, and contained small and large fat droplets. Spinal fluid proteins were greatly elevated to 7500 mg/100 ml; the glucose was 25 mg/100 ml, and cholesterol 175 mg/100 ml. The CSF cell count was 23700, with 82% polymorphonuclear cells. CSF studies performed during the patient’s course are shown in Fig. 2.

The diagnosis of chemical meningitis associated with rupture of a craniopharyngioma cyst was made, and dexamethasone 4 mg four times a day was started. On the second hospital day, he became febrile and developed diplopia. On the fourth hospital day, CSF pressure was 440 mm; the fluid was clear except for a few fat globules. On this day fever and headache subsided but the patient developed a left facial paralysis and bilateral tinnitus. On the sixth hospital day a partial bilateral sixth nerve palsy appeared with decreased sensation in the second division of the right trigeminal. In addition, the tongue deviated to the left. CSF pressure was 300 mm and the fluid was clear. By the ninth hospital day, right facial sensation had returned to normal, tinnitus had become reduced, the left sixth and seventh nerve weakness had improved.

The patient was discharged after 13 days, ambulatory and without medication. He returned to his classes a few days later. All neurological deficit slowly cleared. Lumbar punctures were performed weekly, then bi-monthly. The last CSF study was made 4 months following the acute onset; the pressure was 180 mm of water and the protein content 53 mg%. A pneumoencephalogram done at this time revealed a small mass measuring less than 2 cm in its greatest diameter, coinciding with the area of calcification. The posterior contour of this mass was barely evident against the anterior inferior aspect of the third ventricle. One year later there were no residual neurological deficits and the EEG was normal.

**Discussion**

Meningitis following rupture of a craniopharyngioma cyst at the time of surgery is well known; emphasis has repeatedly been placed on carefully aspirating all contents of the cyst before opening it, as the cyst fluid is known to be irritating in the subarachnoid space and chemical aseptic meningitis has been produced by fluid leakage. The specific irritating substance in the craniopharyngioma cyst fluid has not been identified but presumably the suspended cholesterol crystals play some role in the
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The cyst fluid obtained at the time of surgery has been described as "motor oil" in consistency. It varies from light yellow to dark golden brown in color. Suspended in the fluid, are numerous cholesterol crystals which characteristically reflect transmitted light. The cyst also contains desquamated and liquified epithelial debris. Occasionally, however, the contents are firmer and the cyst contains crumbly, slightly soapy debris rich in cholesterol crystals.

Excluding the characteristic catastrophic onset and clinical history, the single most important laboratory procedure helpful in establishing a correct diagnosis is the spinal fluid lipid analysis. Previous studies from this laboratory have shown that the cyst fluid removed from a craniopharyngioma contains lipid materials ordinarily absent from normal brain. The cyst fluid from a craniopharyngioma, however, cannot be distinguished from that of a malignant glioblastoma by its lipid profile alone; normally, both contain traces of triglyceride, free fatty acid, cholesterol and cholesterol ester. Usually brain lipids contain only traces of triglyceride and free cholesterol and no cholesterol ester is present. The greatly elevated cholesterol fraction and the free fat droplets in the spinal fluid help to establish an early diagnosis of rupture of a craniopharyngioma cyst into the CSF.

There are many similarities between our case and that described by Kaemmerer. In both, follow-up studies at 1 year showed no filling of the cystic portion of the tumor. In our case only a small calcific mass could be discerned beneath the air-filled third ventricle.

References

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