Aseptic Localized Peritonitis in a Patient with Chemical Meningitis Associated with Craniopharyngioma Cysts

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We report a rare case of a young woman with cystic craniopharyngioma who developed not only aseptic chemical meningitis as an initial sign but also aseptic localized peritonitis which was confirmed by the presence of ascites.

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Introduction

Chemical meningitis is rarely caused by spontaneous rupture of a cystic tumor of the brain or spinal cord (1). We describe a young woman with cystic craniopharyngioma who had not only chemical meningitis but also aseptic localized peritonitis. There was a marked increase in concentrations of the 19-9 carbohydrate antigenic determinant (CA19-9) in serum as well as ascites. To our knowledge, this is the first case of craniopharyngioma with a combination of chemical meningitis and aseptic localized peritonitis.

Case Report

A 20-year-old woman was admitted to the hospital because of headache, vomiting and fever of recent onset. Physical examination revealed neck stiffness but no abnormalities in the heart, lungs or abdomen. A lumbar puncture yielded cerebrospinal fluid (CSF) that contained 2029 white cells/mm³, of which 85% were neutrophils and 15% lymphocytes. Protein was 386 mg, and glucose 37 mg per 100 ml. Microscopical examinations of stained smears disclosed no microorganisms; cytologic examination was negative for tumor cells and culture gave negative results.

A computed tomographic (CT) scan of the brain disclosed a calcified mass with cystic lesions located just above the sella turcica (Fig. 1).

On the fourth hospital day, her signs and symptoms completely disappeared and another lumbar puncture revealed clean CSF associated with a marked reduction in cell counts to 23 cells/mm³; protein was 40 mg/100 ml, and glucose was 18 mg/100 ml. However, she complained of lower abdominal discomfort. Therefore, she underwent echograms of the lower abdomen which revealed a quantity of ascites in the Douglas’ pouch but no abnormalities in ovaries or uterus (Fig. 2). An exploratory puncture demonstrated bloody ascites which contained 4.0 g of protein per 100 ml. Cytologic examination was negative for tumor cells and microscopic examination showed no microorganisms. Concentrations of CA19-9 (2) and CA125 (3) in ascites were 38 and 2300 U/ml, respectively. Serum levels of the two tumor markers were 260 and 50 U/ml, respectively (normal range, CA19-9: less than 37 U/ml, CA125: less than 35 U/ml). Although there were no tumor cells in smears of ascites stained with Papanicolaou’s method, abdominal CT, upper gastrointestinal endoscopy, endoscopic retrograde cholangiopancreatography, barium enema, and gallium scintigraphy were performed. All gave negative results. Another lower abdominal echogram revealed no ascites on the 14th hospital day, when there was a decrease in serum CA19-9 and CA125 to 82 and 30 U/ml, respectively. This was followed by further decreases to 20 and 19 U/ml, respectively, on the 25th day. She was discharged and symptom free.

Three months later, she had surgical removal, although partial, of the suprasellar tumor due to rapid growth of the tumor. She was discharged again symptom free 2 months after operation. Pathological examinations demonstrated a craniopharyngioma which was partly

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Fig. 1. A computed tomographic scan showing a calcified mass with cystic lesions located above sella turcica in a patient with chemical meningitis, followed by aseptic localized peritonitis.

Fig. 2. An echogram showing a quantity of ascites in a patient with chemical meningitis associated with cystic craniopharyngioma.

cystic and partly solid. The volume of cystic fluid was too small to be studied.

Comments

Aseptic chemical meningitis has been reported to be associated with extravasation of the contents of cystic intracranial tumors. This complication rarely occurs spontaneously and almost always follows attempted surgical removal of the tumor (4–9). The patient described here had meningismus of catastrophic onset, a sterile pleocytosis with reduced glucose and increased protein concentrations, and a craniopharyngioma, indicating that she had aseptic chemical meningitis due to a leaking craniopharyngioma. The abrupt improvement in CSF findings was presumably due to cessation of the discharge of the cystic contents of the craniopharyngioma into the subarachnoid space. There is no explanation for spontaneous leak of cystic contents, or for cessation of the leak.

In addition to aseptic chemical meningitis, a quantity of ascites in the Douglas pouch was indicated by lower abdominal echograms, and was demonstrated by an exploratory puncture. Because ascites was bloody and two tumor markers, CA19-9 and CA125, were elevated in serum, we wondered if the patient had carcinoma of any sites in the abdominal cavity, especially that of ovaries and pancreas. However, ascites spontaneously disappeared and the increased serum levels of the tumor markers returned to normal values within 3 weeks. In addition, further examinations of gastrointestinal and genitourinary tracts gave negative results. Therefore, we concluded that the ascites found in the patient was not associated with malignancy in the abdominal cavity. To our knowledge, there is no report on chemical meningitis in association with aseptic localized peritonitis. Although we have no explanation for the association, we believe that this is a unique case that adds to the spectrum of chemical meningitis associated with cystic craniopharyngioma.

References


