Cerebrospinal fluid ascites complicating ventriculoperitoneal shunting

Report of four cases

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Four cases of cerebrospinal fluid (CSF) ascites secondary to ventriculoperitoneal shunting are described. It is possible to differentiate CSF ascites from a CSF-filled pseudocyst by the characteristic bowel gas pattern on films of the abdomen and by the presence of shifting dullness. Two of the patients had active shunt infections, and had ascitic fluid with a protein level greater than 3 gm% and a white blood cell (WBC) count greater than 1000/cu mm. Both were treated successfully with antibiotics and removal of the shunt from the peritoneum. Two other patients had no evidence of infection, protein levels of less than 2 gm%, and WBC count less than 100 cu mm. These disorders resolved spontaneously. A review of 18 cases reported in the literature shows that the etiology of CSF ascites in the absence of shunt infection is multifactorial, and no features are consistently present in all cases. Ascites without infection may resolve spontaneously without surgical intervention.

KEY WORDS • cerebrospinal fluid ascites • ventriculoperitoneal shunt • shunt infection

VENTRICULOPERITONEAL (VP) shunting has been associated with a wide variety of complications, a rare one being cerebrospinal fluid (CSF) ascites. We report here four cases of CSF ascites, and review the literature on this topic. The pathogenesis and treatment of CSF ascites are discussed.

Case Reports

Case 1

This 6-year-old girl had initially had shunt placement as an infant for communicating hydrocephalus. She had undergone elective distal revision of her VP shunt 8 months before her present admission. She was admitted with a 1-week history of progressive abdominal distention, anorexia, nausea, and vomiting.

She was alert and afebrile with a VP shunt reservoir that pumped and refilled well. Her abdominal girth measured 58.5 cm (Fig. 1). A fluid wave and shifting dullness were present. Bowel sounds were normal and there was no hepatosplenomegaly. The remainder of the physical examination was normal.

The peripheral white blood cell (WBC) count, total serum protein, liver function tests, routine cloting studies, electrolytes, blood urea nitrogen (BUN), and creatinine levels were all normal. The chest x-ray film was clear and abdominal films showed ascites. The cerebrospinal fluid analysis showed a red blood cell (RBC) count of 2/cu mm, WBC count of 59/cu mm with a differential of 14% polymorphonuclear leukocytes (PML), 84% lymphocytes, and 2% eosinophils, a glucose level of 65 mg%, and a protein value of 30 mg%. The CSF Gram stain was negative but subsequent cultures grew Staphylococcus epidermidis and a Micrococcus. Diagnostic paracentesis showed turbid straw-colored fluid with a cell count of 60 RBC’s/cu mm and 1520 WBC’s/cu mm (79% PML, 22% lymphocytes, and 7% monocytes), glucose 60 mg%, and protein 3.5 gm%. Cultures also grew S. epidermidis.

The patient was started on a 30-day course of oxacillin (300 mg/kg/day), penicillin G (300,000 units/kg/day), and gentamicin (3 mg/kg/day). After 3 days, her old shunt system was removed and a ventriculocholistic shunt was placed. Intraoperative cultures of ascitic fluid were negative. Her ascites resolved and she was well 18 months after discharge from the hospital.

Case 2

This 20-year-old woman initially underwent shunt placement in childhood for hydrocephalus of unknown etiology. For 4 months before her present admission...
Ascites with ventriculoperitoneal shunts

FIG. 1. Cerebrospinal fluid ascites secondary to a ventriculoperitoneal shunt infection in Case 1.

she had a low-grade fever, progressive abdominal distention, anorexia, and intermittent diarrhea. She had undergone multiple shunt revisions over the years, the last being 2 years earlier.

On admission her temperature was 38°C and head circumference was 59.75 cm, greater than the 99th percentile. She was cachectic with marked abdominal ascites. Liver and renal function tests and the peripheral WBC count were normal. Chest x-ray films, oral cholecystogram, liver-spleen scan, endoscopic retrograde pancreateogram, and celiac and superior mesenteric artery arteriograms were all normal. A computerized tomography (CT) scan of the abdomen was normal except for ascites. Paracentesis revealed fluid with glucose 36 mg%, protein 4.8 gm%, and a WBC count of 1070/cu mm (86% PML). Cultures grew Propionibacter acnes. A radioisotope shuntogram revealed clear CSF under normal pressure and free flow of fluid into the abdomen. The CSF glucose level was 66 mg%, protein 18 mg%, and a WBC count of 2/cu mm. Cultures grew S. epidermidis and P. acnes. The patient was started on a 30-day course of nafcillin (12 gm/day).

After 2 days, she was taken to surgery where the shunt system was removed and a blind reservoir with a ventricular cannula was placed through which 50 mg of nafcillin was administered daily for 14 days. She developed a right hydrothorax secondary to her ascites which required thoracentesis on two occasions. A 2-week course of intravenous hyperalimentation was given because of nutritional depletion. Oral intake greatly improved and the ascites began to resolve. Because several taps of her reservoir showed the intraventricular pressure to be normal, the shunt was not replaced. Her neurological condition and ventricular size, ascertained by CT scanning, remained unchanged. She was well and without ascites 1 year after her discharge.

Case 3

This 4-year-old boy initially had shunt placement at the age of 17 months for communicating hydrocephalus. At the age of 4 years old he was found to have optic atrophy and a parasellar tumor on CT scanning. An open biopsy via right frontal craniotomy proved the lesion to be an optic nerve glioma. The postoperative course was initially uncomplicated except for a fever spiking to 39°C in the first 48 hours. On the 7th postoperative day abdominal distention was noticed. Shifting dullness was present and abdominal films revealed ascites. Upon paracentesis, 900 cc of clear xanthochromic fluid was removed; analysis showed a protein level of 1.5 gm%, a glucose value of 105 mg%, and 28 WBC's/cu mm. Cultures of the fluid were negative. A preoperative spinal tap had shown clear CSF with 97 mg% protein and 70 mg% glucose. The patient was put on a low-sodium diet with gradual resolution of his ascites. No further spinal taps were required. His VP shunt continued to function well 5 years later, and there was no further evidence of ascites.

Case 4

This 3-year-old girl initially had shunt placement for hydrocephalus at the age of 13 months. At the age of 2 years, she underwent craniotomy for biopsy of an optic nerve glioma. She was treated with cranial irradiation and was discharged. Five months later she was readmitted with diencephalic syndrome and Shigella enteritis, and was treated with a course of oral ampicillin. Moderate ascites was noted on physical examination at that time, but resolved spontaneously after a few days.

The patient was readmitted 3 months later with lethargy, irritability, and abdominal distention. She was thin, chronically ill, and blind, but afebrile. She had marked abdominal ascites. The shunt valve pumped and filled well, and the craniectomy site was soft. Sodium level was 131 mEq/liter; BUN, creatinine, and liver function tests were normal. A shuntogram showed free flow of fluid into the peritoneum without sheathing of the distal catheter. Cerebrospinal fluid analysis revealed a protein level of 1.3 gm%, a glucose value of 83 mg%, and 2 WBC's/cu mm. Aerobic and anaerobic CSF cultures were negative, and no malignant cells were seen. Her neurological condition remained stable and she was discharged on a fluid-restricted diet. The ascites gradually resolved and 5 years after the episode the shunt was functioning well, although the patient was bedridden from her primary disease.

Discussion

To date, 18 cases of CSF ascites due to VP shunting have been reported in the literature (Table 1). All of the patients, including ours, had normal hepatorenal function with the exception of an infant who died of Hurler's syndrome. Two patients had a history of shunt infections. Two of our patients (Cases 1 and 2) had
**TABLE 1**  
Summary of 18 cases of CSF ascites due to VP shunting*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age at Onset</th>
<th>Diagnosis</th>
<th>Time from Shunt to Onset</th>
<th>Ascitic Fluid</th>
<th>CSF</th>
<th>Shunt Infection</th>
<th>Treatment</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adegbite &amp; Khan, 1982</td>
<td>11 yrs</td>
<td>craniopharyngioma</td>
<td>1 mo</td>
<td>prot: 1.2 gm%</td>
<td>WBC: 16/cu mm</td>
<td>prot: 620 mg%</td>
<td>no</td>
<td>convert to VA then VP</td>
</tr>
<tr>
<td>Adeloye &amp; Olumide, 1977</td>
<td>16 mos</td>
<td>Hurler's syndrome</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>no</td>
<td>—</td>
</tr>
<tr>
<td>Ames, 1967</td>
<td>2 yrs</td>
<td>aqueductal stenosis</td>
<td>2 yrs</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>no</td>
<td>convert to VA</td>
</tr>
<tr>
<td>Cummings, et al., 1976</td>
<td>53 yrs</td>
<td>aqueductal stenosis</td>
<td>18 mos</td>
<td>prot: 1.1 gm%</td>
<td>WBC: 9/cu mm</td>
<td>prot: 100 mg%</td>
<td>WBC: 2/cu mm</td>
<td>no</td>
</tr>
<tr>
<td>Dean &amp; Keller, 1972</td>
<td>9 mos</td>
<td>aqueductal stenosis, recent DPT vaccine; prior abd. surgery</td>
<td>2 mos</td>
<td>prot: 900 mg%</td>
<td>WBC: 9/cu mm</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Herrera, et al., 1980</td>
<td>two cases reported, no details</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Lees, et al., 1978</td>
<td>5 yrs</td>
<td>myelomeningocele, recent influenza vaccine</td>
<td>2 yrs</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Little, et al., 1972</td>
<td>two cases reported, no details</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Mori, et al., 1977</td>
<td>2 yrs</td>
<td>medulloblastoma</td>
<td>4 mos</td>
<td>prot: 300 mg%</td>
<td>—</td>
<td>—</td>
<td>no</td>
<td>repeated taps ip carbouquone</td>
</tr>
<tr>
<td>Noh, et al., 1979</td>
<td>5 mos</td>
<td>post. fossa cyst</td>
<td>2 mos</td>
<td>prot: 1.6 gm%</td>
<td>WBC: 90/cu mm</td>
<td>—</td>
<td>—</td>
<td>no</td>
</tr>
<tr>
<td>Odeku, et al., 1970</td>
<td>15 mos</td>
<td>hydrocephalus</td>
<td>9 mos</td>
<td>prot: 1.3-1.7 gm%</td>
<td>WBC: 1/cu mm</td>
<td>—</td>
<td>—</td>
<td>no</td>
</tr>
<tr>
<td>Ohaegbualm, 1980</td>
<td>6 yrs</td>
<td>craniopharyngioma</td>
<td>7 mos</td>
<td>prot: 110 gm%</td>
<td>WBC: 1/cu mm</td>
<td>—</td>
<td>—</td>
<td>no</td>
</tr>
<tr>
<td>Parry, et al., 1975</td>
<td>7 mos</td>
<td>hydrocephalus</td>
<td>24 hrs</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>no</td>
<td>convert to VA tumor excision</td>
</tr>
<tr>
<td>Ray &amp; Peck, 1956</td>
<td>4 mos</td>
<td>choroid plexus papilloma</td>
<td>1 wk</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>no</td>
<td>—</td>
</tr>
<tr>
<td>Rosenthal, et al., 1974</td>
<td>2 yrs</td>
<td>astrocytoma</td>
<td>10 mos</td>
<td>prot: 100 mg%</td>
<td>WBC: 0</td>
<td>—</td>
<td>—</td>
<td>convert to VA</td>
</tr>
<tr>
<td>Weidmann, 1975</td>
<td>3 mos</td>
<td>aqueductal stenosis</td>
<td>2 mos</td>
<td>prot: 700 mg%</td>
<td>WBC: 10/cu mm</td>
<td>prot: 110 mg%</td>
<td>no</td>
<td>convert to VA</td>
</tr>
<tr>
<td>Yount, et al., 1984</td>
<td>Case 1</td>
<td>6 yrs</td>
<td>hydrocephalus</td>
<td>8 mos</td>
<td>prot: 3.5 gm%</td>
<td>WBC: 1520/cu mm</td>
<td>prot: 30 mg%</td>
<td>WBC: 59/cu mm</td>
</tr>
<tr>
<td>Case 2</td>
<td>20 yrs</td>
<td>hydrocephalus</td>
<td>2 yrs</td>
<td>prot: 4.8 gm%</td>
<td>WBC: 1070/cu mm</td>
<td>prot: 18 mg%</td>
<td>WBC: 2/cu mm</td>
<td>yes</td>
</tr>
<tr>
<td>Case 3</td>
<td>4 yrs</td>
<td>optic nerve glioma</td>
<td>1 wk</td>
<td>prot: 1.5 gm%</td>
<td>WBC: 28/cu mm</td>
<td>prot: 97 mg%</td>
<td>WBC: 4/cu mm</td>
<td>no</td>
</tr>
<tr>
<td>Case 4</td>
<td>3 yrs</td>
<td>optic nerve glioma</td>
<td>2 yrs</td>
<td>prot: 1.3 gm%</td>
<td>WBC: 2/cu mm</td>
<td>prot: 63 mg%</td>
<td>WBC: 0</td>
<td>no</td>
</tr>
</tbody>
</table>

*CSF = cerebrospinal fluid; VP = ventriculoperitoneal shunt; VA = ventriculoatrial shunt; DPT = diphtheria toxoid; VCC = ventriculocholecystic shunt; WBC = white blood cell count; ip = intraperitoneal; abd = abdominal.

Active shunt infection, both with marked elevation of ascitic fluid WBC count and ascitic fluid protein exceeding 3 gm%, although CSF protein levels were normal. These facts point to peritonitis with impaired resorptive capacity of the peritoneum. Ascites is a relatively uncommon sequela of shunt infections. We have found that by far the most common intra-abdominal response to infection is sheathing of the peritoneal catheter. The CSF draining into these sheaths produces large abdominal fluid-filled cysts that are distinguished from ascites by their characteristic displacement of the bowel gas pattern on abdominal films and by the absence of shifting dullness.

Most non-infected cases have less marked elevation of ascitic fluid protein with relatively low ascitic WBC counts, as in our Cases 3 and 4. These patients also lack the clinical signs of peritonitis. An immune reaction has been postulated to be the etiology in two previous cases in which ascites developed shortly after vaccinations. Elevated CSF protein has been implicated as a factor in the production of CSF ascites. However, we have seen several cases of patients with CSF proteins of over 700 mg% who did not develop ascites after VP shunting. In addition, three of the cases presented here had normal CSF protein levels at the time of their ascites. Low ascitic fluid protein has been reported in two cases. In another case, excess CSF production from a choroid plexus papilloma was thought to cause ascites. Malignant seeding of the peritoneum from a medulloblastoma was implicated by Mori, et al., in a
Case treated by intraperitoneal chemotherapy. They were unable, however, to demonstrate the presence of malignant cells despite several paracentesis procedures. In general, conversion of the shunt to atrial drainage results in resolution of the ascites. 

Two cases have been reported in which the ascites resolved without the need for operative intervention. Our Cases 3 and 4 both had sterile ascites which resolved spontaneously.

We suggest the following approach to the management of CSF ascites in patients with no evidence of hepatorenal dysfunction on routine blood chemistry. Peritoneal fluid and CSF should be obtained for analysis of protein, cell count, and culture. If cytological examination of the fluid and abdominal CT reveal no evidence of malignancy, further abdominal diagnostic studies are unnecessary. Ascitic fluid protein levels of 3 gm% or greater are highly suspicious of infection. If cultures are positive, the patient should be treated appropriately for a shunt infection. This treatment should include removing the distal catheter from the peritoneum. Once the infection has resolved, shunting to a space other than the peritoneum may be expected to give the best results. Cases without infection generally have ascitic fluid protein levels of less than 2 gm%. The etiology of the ascites in these cases is usually obscure, and probably multifactorial. Under these circumstances the ascites may resolve spontaneously without the necessity for operative revision of the shunt system.

References


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