

## Ascites and abdominal pseudocyst: two uncommon ventriculoperitoneal shunt complications in two cases

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Ascites and abdominal pseudocysts (APC) are two rare complications that can occur following placement of a ventriculoperitoneal (VP) shunt. Both complications are characterized by abnormal intraperitoneal cerebrospinal fluid (CSF) collections. Although various factors have been implicated, the exact pathogenesis of the two conditions remains elusive. This paper presents two cases of VP shunt placement resulting from hydrocephaly. The first patient presented with generalized ascites and the other with APC, both of whom were six years old. APC and ascites after VP shunt placement are rare and distinct conditions; therefore, they may require different management strategies.

**Key words:** abdominal pseudocysts, ascites, ventriculoperitoneal shunt, cerebrospinal fluid, child.

The accumulation of pathological fluid in the peritoneal cavity is called ascites. In childhood, ascites occurs most frequently due to liver and kidney diseases<sup>1</sup>. Ventriculoperitoneal (VP) shunt surgery is an extensively used operational technique in the treatment of hydrocephaly<sup>2,3</sup>. Complications such as displacement or dissociation of the shunt catheter, peritonitis and abscess can be seen in the abdomen as a result of VP shunt<sup>4</sup>. Abdominal fluid collections are relatively uncommon complications (2%) and mostly characterize in two forms: encysted collections, also known as abdominal pseudocysts (APC), and excess cerebrospinal fluid (CSF) accumulation, called CSF ascites. APC are more common than CSF ascites. Although APC and CSF ascites seem to be similar, the pathogenesis, clinical outcomes and treatment methods differ<sup>5,6</sup>. These two different forms of VP shunt complications are presented in this paper.

### Case Reports

#### Case 1

A six-year-old boy was admitted with complaints of an open wound in the

backbone, fever and abdominal distension that had been increasing for two months. The patient's medical history showed that he had myelomeningocele operation in the newborn period and VP shunt when he was nine months old. He underwent debridement of the myelomeningocele operation area as a result of decubitus ulcer and infection at the age of two years. At the age of four, he had a Fallot tetralogy total correction surgery. On the present admission, his physical examination revealed that he was paraplegic, his weight and height percentages were <3<sup>rd</sup> percentile, and he had a temperature of 37.8°C. There was a 3/6° systolic murmur on pulmonary focus, and ascites was determined on percussion; no organomegaly was observed. Decubitus ulcer and purulent discharge were observed on the previously operated lumbar area. Infection was superficial and did not cause meningitis. The laboratory findings were as follows: hemoglobin 8 g/dl, white blood cells count 7760/mm<sup>3</sup>, thrombocytes 557000/mm<sup>3</sup>, and C-reactive protein (CRP) 163 mg/L (0-5). Serum bilirubin and liver enzymes were all normal. The patient underwent diagnostic paracentesis, which appeared clear, and there

was no microorganism or leukocyte on the smear. Serum ascites albumin gradient was 2.75 g/dl; ascites fluid culture, tuberculosis culture and acid-fast bacilli were all negative. The patient's bone scintigraphy revealed osteomyelitis in the lumbosacral region, and he was placed on a broad-spectrum antibiotic therapy. The abdominal ultrasonography (USG) and cranial computerized tomography (CT) did not show any pathology other than generalized peritoneal fluid collection; no shunt dysfunction was observed, and portal vein Doppler USG was normal. Echocardiography showed operated Fallot tetralogy and residual pulmonary stenosis and deficiency. The patient's condition was not compatible with protein-losing enteropathy with serum albumin level of 3.5 g/dl. No varicose was observed in the upper endoscopy. Liver, kidney or cardiac-related ascites was not considered. The patient was thought to have CSF ascites. He was started on restricted fluid and diuretics for the treatment of ascites, and excess volume was drained with intermittent paracentesis, but it did not show any decrease. The cranial CT revealed that the ventricles were normal in size, and there was no mass of choroid plexus papilloma that might be the cause of oversecretion of CSF. The patient's VP shunt was replaced with a ventriculoatrial (VA) shunt. Following the operation, there was a significant decrease in the abdominal distension from the first day onwards, and on the third day it disappeared. The patient's ascites did not relapse in the follow-ups.

## Case 2

A six-year-old girl presented with constipation and abdominal distension, which had been increasing for 15 days. Her medical history stated that she was operated due to myelomeningocele when she was two days old, and VP shunt surgery was performed. At the age of three, she had shunt revision due to shunt dysfunction. At the present admission, her physical examination showed that she was paraplegic, and she had a temperature of 38°C. On abdominal examination, there was abdominal distension, but hepatosplenomegaly and the prevailing ascites were determined on percussion. Laboratory findings were as follows: hemoglobin 10.4 g/dl, white blood cells 10750/mm<sup>3</sup>, thrombocytes 704000/mm<sup>3</sup>, and CRP 43 mg/L (0-5). Serum liver and kidney function

tests were normal. Cranial CT revealed enlarged ventricles. Abdominal USG and CT showed a locular collection of 17x10x21cm that filled the entire right quadrant and pelvis. The shunt catheter was observed inside the collection, and it also contained thin septations in some places. Repression was observed in the liver, right kidney, pancreas, and bowels. Three hundred ml of fluid was aspirated from the cyst with ultrasound guidance. The fluid appeared clear, the microscopic examination did not show any microorganism, and the culture was negative. Serum ascites albumin gradient was 3.09 g/dl. The patient was started on broad-spectrum antibiotic, and the shunt catheter was externalized. The follow-up ultrasound revealed that the cyst had shrunk in size (14x7x16 cm) but not disappeared. In order to extract the cyst, an 8 French catheter was placed into the cyst with ultrasound guidance. One week later, the fluid had disappeared and the catheter was removed. Four weeks later, no APC was observed in the follow-up USG, and the VP shunt was re-inserted. The patient did not have any complaints in the follow-ups.

## Discussion

Ascites frequently occurs as a complication of portal hypertension and nephrotic syndrome in childhood, and occurs rarely as a result of conditions such as tuberculous peritonitis, peritoneal carcinomatosis, and cardiac, pancreatic or biliary disorders<sup>1,7</sup>. Intraabdominal complications of VP shunt are often abdominal abscess, peritonitis and dissociation of the shunt, while excess collection of CSF is rare<sup>1,8</sup>. CSF ascites that develops in patients with shunt generally tends to be locular<sup>5</sup>. The mesentery that encloses the shunt catheter causes CSF accumulation in this area and prevents CSF from spreading to the other areas of the abdomen<sup>5</sup>. This is called CSF APC<sup>9</sup>. It was defined first by Harsh<sup>10</sup> in 1954 and its frequency is reported to be between 0.33% and 68%. The etiology of APC is still not clear<sup>3,6</sup>. These patients generally have an infection (17-80%), but culture is positive in only half of them<sup>5,6</sup>. APC fluid is confined by a fibrotic wall that includes many inflammatory cells histologically that decrease the CSF absorption<sup>3,5</sup>. Previous abdominal surgeries and shunt revision cause a predisposition to APC formation<sup>5</sup>. Sixty percent of the patients

present with shunt dysfunction symptoms<sup>6</sup>. Although abdominal symptoms were noticed in our patient (Case 2), shunt dysfunction was detected as well. She had a shunt revision history that could set up a predisposition to CSF APC formation. The ascites fluid did not contain any bacterial agent or tuberculosis; however, since the patient had a fever and CRP, she was started on broad-spectrum antibiotics. Although the culture was negative, it was considered that an infection inside the abdomen may have led to the APC formation.

Cerebrospinal fluid (CSF) ascites is a slightly different condition from APC, which is cystic ascites accumulation<sup>5</sup>. CSF accumulates extensively in the peritoneal cavity generally because of the excessive CSF production or insufficient absorption capacity of the peritoneum; however, the pathophysiology of CSF ascites is still not fully explained<sup>4,5,11</sup>. CSF overproduction can be associated with suprasellar region tumors such as optic glioma and craniopharyngioma<sup>5,12</sup>. Due to such neoplastic conditions and infections, heightened CSF protein level causes an increase in the temporary oncotic powers in the peritoneum<sup>4,11</sup>, and thus absorption of CSF from the peritoneum can be disrupted and can cause CSF ascites<sup>9</sup>. It has been reported that CSF ascites can also result from the massive immune response against shunt material<sup>13</sup>. Kariyattil et al.<sup>6</sup> reported that major abdominal surgery and shunt revision, which tend to cause abdominal infection, are seen in APC other than CSF ascites. Our CSF ascites patient (Case 1) did not have a history of shunt revision, he had no abdominal infection, abscess or peritonitis, his brain scan did not show tumor or choroid plexus pathology that can cause excessive CSF production, and there was no CSF culture positivity to support central nervous system infection.

Ultrasound is an easy means for a quick and reliable diagnosis of APC and ascites<sup>14</sup>. Although treatment of the two entities is quite similar, treatment of APC consists of shunt externalization, antibiotics treatment if there is shunt infection, and replacement of the shunt with a second surgery<sup>5</sup>. APC aspiration with ultrasound is an effective method in showing the infection agent and relieving the patient's abdominal symptoms<sup>3,14</sup>.

Generally, displacement of a shunt catheter causes spontaneous resorption of the APC<sup>14</sup>. In our patient, the shunt was extracted and the fluid was drained by placing the catheter under ultrasound guidance. After the patient's clinical findings had improved, the VP shunt was re-inserted, and there was no fluid collection on further examinations.

The treatment of CSF ascites requires the CSF drainage to be placed somewhere other than the peritoneal cavity. VP shunt can be converted to ventriculoatrial, ventriculopleural or ventriculo-gallbladder shunts. VA shunt is frequently preferred<sup>6</sup>. After our patient's VP shunt was converted to VA shunt, his complaints resolved completely.

In conclusion, CSF ascites and APC are rare complications of VP shunt. While looking for the causes of ascites in children with VP shunt, it should be kept in mind that the cause can be collection of CSF.

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