Abdominal pseudocysts and ascites formation after ventriculoperitoneal shunt procedures

Report of four cases

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The authors report three patients with abdominal pseudocysts and one with cerebrospinal fluid ascites as late complications of ventriculoperitoneal shunts. The presenting signs and symptoms were those of an intraabdominal abnormality, with no neurological symptoms suggestive of shunt malfunction.

Key Words: infantile hydrocephalus • ventriculoperitoneal shunt • abdominal cysts • pseudocyst • ascites

Numerous abdominal complications have been reported subsequent to ventriculoperitoneal (VP) shunting procedures instituted for the treatment of hydrocephalus. Perforation of the gallbladder, intestine, vagina, umbilicus, and volvulus has been documented, as well as five cases of abdominal cerebrospinal fluid (CSF) encystation and two cases of CSF ascites. We are reporting three patients with CSF encystation and one with ascites; our cases differ from those previously reported both in histopathology and clinical presentation.

Case Reports

Case 1

A 3-week-old baby girl was referred for evaluation of an enlarged head. She was born after a 36-week gestation period, poorly developed and poorly nourished, with a greatly enlarged head (40 cm frontooccipital circumference). The anterior fontanel was tense and nonpulsatile. Ventriculography revealed noncommunicating hydrocephalus secondary to aqueductal stenosis. A VP shunt procedure was performed. When the patient was 26 months old, the shunt was revised due to omental obstruction of the peritoneal catheter.

One month later the patient was readmitted in an agitated, hyperactive state. She had a large, tender mass in the right upper abdominal quadrant in the area of the previous incision. This mass readily reduced with pressure and it was thought to be an incisional hernia. However, upon reopening the incision, 300 ml of clear CSF escaped from a large encystation around the peritoneal end of the catheter. No definite cyst wall was pres-
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ent, only adherent loops of small intestine, held together by inflamed serosal surfaces and granulation tissue. The “cyst” was evacuated and the wound reclosed.

Seven months later, the patient developed an enlarging tender mass in the left upper abdominal quadrant. It was believed to be a renal neoplasm; however, intravenous pyelography showed that it was not associated with the kidney. At laparotomy, a large amount of CSF was evacuated from an encystation around the peritoneal catheter. The “cyst” once again was formed by adherent intestinal loops. Postoperatively she was discharged asymptomatic.

At 3½ years of age, the patient had two nocturnal seizures. Physical examination revealed generalized abdominal dullness, a fluid wave, and distention of the subcutaneous veins of the trunk. The child was placed on diphyldantoin and phenobarbital. There were no neurological symptoms, so the shunt was not revised. Eleven months later she returned with another tender abdominal mass and symptoms compatible with bladder compression, thought to represent a recurrence of CSF encystation. The mass was percutaneously aspirated and 160 ml of clear CSF were obtained. One month later the VP shunt was converted to a ventriculoatrial (VA) shunt.

During the next 3 months she had two episodes of congestive heart failure. The VA shunt was subsequently reconverted to a VP shunt. When seen 6 months later, she remained asymptomatic.

Case 2

A 3-month-old baby girl presented for evaluation of an enlarged head, bulging anterior fontanel, vomiting, anorexia, and fever (39°C). She had been born 1 month prematurely, and subsequently developed neonatal jaundice, probably due to sepsis. On admission she was undernourished, with an enlarged head (45 cm frontooccipital circumference), tense anterior fontanel, widened suture lines, and exophthalmos. Ventriculography indicated communicating hydrocephalus, and a VP shunt was inserted. The child recovered and was discharged 3 days postoperatively.

The shunt had to be revised 3½ years later because of its migration from the peritoneal cavity due to growth of the child. At the age of 5 years, the child was readmitted; she had abdominal pain in the right lower quadrant and anorexia but no nausea, vomiting, or diarrhea. Physical examination was unremarkable except for temperature of 39.2°C and extreme abdominal guarding and tenderness. All laboratory values were within normal limits except for a leukocyte count of 12,000/mm³. Upper gastrointestinal films revealed no abnormality. The child was given ampicillin, became afebrile, and seemed to be recovering.

On the 12th hospital day, the patient developed a distended abdomen with hyperactive bowel sounds. A midline lower abdominal mass was palpated on the 14th hospital day. Later that day the patient’s temperature rose to 40°C and she had a leukocyte count of 15,000/mm³. A diagnosis of appendicitis with rupture and abscess formation was made, and exploratory laparotomy was then undertaken. An 8 × 8-cm “cystic” mass was encountered in the peritoneal cavity, its walls composed of intestinal loops matted together by granulation tissue. The tip of the peritoneal catheter was within the cystic cavity. The shunt was repositioned within the peritoneal cavity. The fluid from the cyst grew Staphylococcus aureus, coagulase-negative. Two days postoperatively, the abdomen was no longer distended, bowel sounds were normal, and the shunt was apparently functioning well. The child was given cephalosporin and chloramphenicol.

Six weeks postoperatively CSF cultures yielded no bacterial growth. The patient was in good condition neurologically. The shunt was removed and a new VP shunt mechanism placed. No neurological symptoms or cyst recurrence have been evident in the following 12 months.

Case 3

A 3-year-old boy was admitted because of increasingly severe lethargy. He had been 10 weeks premature and had had an enlarged head at birth. Ventriculography at that time revealed hydrocephalus secondary to a Dandy-Walker cyst, and a VP shunt was inserted. When the patient was 7 weeks old, the original shunt malfunctioned and was replaced. The child recovered and was well for 3 years.
Upon his current admission, the patient was markedly lethargic and had a right abducens nerve palsy; funduscopic examination revealed early papilledema. Revision of the shunt revealed that the abdominal catheter was in the preperitoneal fat rather than the peritoneal cavity. The catheter tip was repositioned and all signs and symptoms cleared rapidly.

One month later, the child returned to the clinic. He was vomiting and had abdominal distention, diffuse abdominal tenderness, absent bowel sounds, and a tender mass in the right lower abdominal quadrant. All vital signs were normal and laboratory studies disclosed only a slight leukocytosis (71,000 WBC/mm³). A provisional diagnosis of appendicitis with possible abscess formation was made and exploratory laparotomy undertaken. A pseudocyst which contained approximately 300 ml of clear CSF was found in the peritoneal cavity. The cyst cavity was formed by the visceral surface of the liver and loops of matted small bowel. The intestinal serosal surfaces were inflamed, but not purulent. Cultures of the CSF and serosa later revealed no bacterial growth. The peritoneal end of the catheter was brought out to drain externally as it was thought unwise to replace the catheter within a possibly infected peritoneal cavity. Two weeks later, the catheter was replaced in the abdomen. When seen in the outpatient clinic 6½ months later, the patient had no abdominal complications and the shunt was functioning well.

Case 4

A 7-month-old baby boy was referred for evaluation of a bulging anterior fontanel and an enlarged head (46 cm frontooccipital circumference). He had been born 8 weeks prematurely. Ventriculography suggested communicating hydrocephalus, and a VP shunt was performed. Postoperatively the anterior fontanel was flattened but did not pulsate.

One week later the patient's abdomen became distended and he developed a large fluid collection extending caudally along the course of the catheter beneath the scalp. At surgery, 300 ml of clear CSF poured forth from the abdomen, and CSF dripped freely from the peritoneal end of the catheter. The shunt was then replaced in the abdomen. Cultures of the ascitic CSF were later reported negative for bacterial growth. Three days postoperatively, the abdomen again became distended; however, since the patient was asymptomatic, it was elected to follow his progress closely as an outpatient.

Abdominal distention was noticeable at every clinic visit. Eleven months after the operation, the patient became irritable and had numerous episodes of vomiting. Examination again revealed a large subgaleal fluid collection surrounding the flushing device. The anterior fontanel was tense and bulging. The abdomen was protuberant, dull to percussion, and demonstrated a fluid wave. The shunt was revised on the suspicion that the abdominal catheter was occluded. At surgery, approximately 500 ml of clear CSF was found in the abdomen. The next day the fontanel was bulging more than before and the abdomen remained distended. A VA shunting procedure was then performed. The child had a smooth postoperative course and no further malfunction of the shunt has developed for 8 months.

Discussion

Ventriculoperitoneal shunts are the procedure of choice in the treatment of hydrocephalus at our institution. We have presented here two unusual complications of this procedure. In each of our cases, the initial symptoms seemed to indicate an intraabdominal problem; consequently, the initial diagnostic possibilities considered were incisional hernia, appendicitis, or polycystic kidney.

Five cases of abdominal encystation of CSF following a VP shunting procedure have previously been reported. Fischer and Shillito reported three cases and Scott, et al., reported one case in which the peritoneal end of the catheter was encased in a thick-walled fibrous cyst. Jackson and Snodgrass reported a case in which an omental cyst formed. In all these cases, at least one abdominal procedure had been performed in addition to the original shunt insertion. Previous authors have incriminated these additional procedures as being significant factors in cyst formation. Dean and Keller, in reporting a patient with sterile CSF ascites, speculated that the peritoneal malabsorption was due to an antigen-antibody response after routine diphtheria-pertussis-tetanus immunization. However, their patient's abdomen had previously been subjected to an omphalocoele.
Abdominal pseudocysts and ascites after VP shunting repair and another laparotomy for correction of large bowel malrotation. Although it is possible the small amount of manipulation could have been a contributing factor, our patients with pseudocyst had undergone only a shunt revision. Our patient (Case 4) with CSF ascites had never had abdominal surgery except for the original shunt insertion.

The patients reported previously have all exhibited one or more of the usual manifestations of increased intracranial pressure: headache, vomiting, irritability, head enlargement, or lethargy. None of our patients initially demonstrated any signs or symptoms of increased intracranial pressure due to an obstructed shunt. In addition, the flushing mechanism in each case was examined by the bubble palpation method of Ames and was thought to be functioning well.

Our cases of “encystation” differ from those previously reported in yet another way. Previously reported cases of encystation of CSF have had a thick-walled fibrous capsule. In each of our “cysts,” the capsule was formed solely of inflamed intestinal serosal surfaces. They were, in fact, pseudocysts and not true cysts. In each of our operative procedures, the abdominal incision was large enough (10 to 11 cm), and abdominal exposure adequate, to exclude the possibility of a true cyst in relation to the loculation or any other abdominal disease or deformity.

In one of our patients (Case 2), Staphylococcus aureus, coagulase-negative, was isolated from the abdominal CSF. This organism was also cultured by Fischer and Shillito from two of their cases. The CSF in our Cases 1 and 3 was sterile; however, these patients had been on high doses of antibiotics. We are postulating that the walls of the pseudocysts were formed as an inflammatory reaction around the catheter tip, secondary to a low-grade shunt infection and localized peritonitis.

Fischer and Shillito have suggested that cyst formation is a poor prognostic sign regarding future use of the peritoneal cavity for shunting. In their series, 3 months was the longest period of time a shunt remained functional after replacement in the abdomen. Except for Case 1, this has not been our experience with pseudocyst formation. Following replacement of the catheter in the abdomen, the patients in Cases 2 and 3 demonstrated no sign of cyst recurrence or shunt malfunction.

It is of interest, though of questionable significance, that all of our patients were at least 4 weeks premature at birth.

Abdominal pseudocyst should be included in the differential diagnosis of an acute abdominal complaint for any patient with a VP shunt. Pseudocyst or CSF ascites should also be considered as a possible explanation for deteriorating neurological status in such a patient even when the shunting mechanism seems to be functioning satisfactorily. Awareness of this condition among all neurosurgeons could save valuable time and decrease the chance of serious retrograde shunt infection.

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References

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