Distal Ventriculoperitoneal Shunt Failure Secondary to *Clostridium difficile* Colitis

Oren N. Gottfried, M.D., Mandy Binning, M.D., and William T. Couldwell, M.D., Ph.D.

Department of Neurosurgery, University of Utah, Salt Lake City, Utah

Corresponding Author:

William T. Couldwell, M.D., Ph.D.

Department of Neurosurgery

University of Utah

30 North 1900 East, Suite 3B409

Salt Lake City, Utah 84132

Email: william.couldwell@hsc.utah.edu

Phone: 801-581-6908

Fax: 801-581-4385

Addresses for co-authors: All authors share the same address. Email addresses for Dr. Gottfried (oren.gottfried@hsc.utah.edu) and Dr. Binning (mandy.binning@hsc.utah.edu) are the only differences.

Abstract

Distal ventriculoperitoneal shunt obstruction is typically associated with cerebrospinal fluid (CSF) infection, fluid pseudocysts, bowel obstruction, bowel perforation, or improper shunt placement in the abdomen. We describe a unique etiology for distal shunt obstruction secondary to *Clostridium difficile* pancolitis that occurred because of inflammation and ascites, which led to incomplete drainage and absorption of CSF. This case illustrates the importance of considering distal shunt obstruction in a patient with signs of abdominal pathology in the setting of mental status changes, and the effective treatment of this patient initially with distal catheter externalization followed by internalization of a new distal catheter after resolution of the pancolitis.

Key Words: colitis; hydrocephalus; pancreatitis; shunt failure; ventriculoperitoneal shunt

Running Head: Distal Shunt Failure from Colitis

Introduction

Ventriculoperitoneal shunt (VPS) obstruction or failure requires urgent management and surgical intervention. Distal shunt problems can often be confused with or complicated by abdominal pathology. Obstruction is the most common cause of distal shunt failure, followed in frequency by infection [9]. Typically, distal obstruction occurs secondary to shunt design flaws [6], fluid pseudocysts, bowel obstructions, bowel perforation, or improper shunt placement in the abdomen [7,11,19]. It is always important to consider the distal shunt in VPS patients who present with abdominal complaints or signs, as distal infection or obstruction can be either the cause of abdominal pain or the result of other abdominal pathology.

We report a case of distal shunt failure occurring in a patient with *Clostridium difficile* pancolitis and concomitant pancreatitis. In this patient, we postulate that the distal shunt failure resulted from increased intra-abdominal pressure from ascites and decreased absorption caused by the inflammatory response and hypoalbuminemia. The authors review the etiologies for distal shunt failure that occur in the setting of abdominal pathology.

Case Report

History and Physical Examination

A 56-year-old woman developed a grade III Kissler- Fisher subarachnoid hemorrhage because of rupture of a basilar tip aneurysm (Hunt/Hess grade II); she underwent a successful clipping of the aneurysm via a right subtemporal approach. Post-operatively,

the patient developed a communicating hydrocephalus and was treated with a right ventriculoperitoneal shunt (VPS). Computed tomography (CT) and a shunt series following shunt placement demonstrated a decrease in ventricle size and appropriate placement of the distal catheter into the peritoneum. The patient had an uneventful post-operative course, did not develop significant SAH-induced vasospasm, and remained neurologically intact.

One month after placement of the VPS, the patient developed decreased mental status with lethargy and difficulty following commands. In addition, she demonstrated signs of abdominal tenderness and distension. The patient had poor enteral intake during her time in the intensive care unit due to persistent pancreatitis, and on this day her amylase and lipase were still elevated, at 391 U/L and 3072 U/L, respectively. Her serum albumin level was low at 1.9 gm/dL. She had worsening diarrhea suggesting *Clostridium difficile* enterocolitis, given her recent treatment with ampicillin for a urinary tract infection. A head CT demonstrated an interval increase in ventricular size (Fig. 1a) and an abdominal CT showed diffuse thickening of the colonic wall, pericolic edema of the soft tissues, and free fluid (Fig. 2a). There was no evidence of an abscess, free air, or pseudocyst. The distal catheter was located in the peritoneal cavity.

Because of the clinical suspicion for a shunt failure, the shunt was tapped. There was no evidence of problems with proximal drainage. The cerebrospinal fluid (CSF) white cell count was 3/μL, red blood cell count was 220/μL, glucose was 61 mg/dL, protein was 30 mg/dL, and the gram stain and culture were negative for organisms. Together, these findings led to the conclusion that the distal end of the VPS was failing to drain fluid

because of the pancolitis and ascites in this patient. The distal end of the shunt was externalized from the abdomen at the bedside, and there was good drainage from the catheter. Culture of CSF from the externalized distal catheter was also negative for organisms. Two days after externalization and drainage of the shunt, the patient was more alert with improved mental status, and a repeat CT scan of the head revealed a decrease in ventricular size (Fig. 1b). Subsequent CT scans demonstrated a return to normal ventricle size.

The patient had a positive *Clostridium difficile* stool toxin assay and her pancolitis was treated with intravenous (IV), then oral metronidazole over a two-week period. The patient's pancreatitis was treated conservatively by continuing the patient nothing *per orum* (NPO) and by initiating nasogastric suction and IV total parenteral nutrition. The patient's pancolitis and pancreatitis clinically improved. A repeat CT of the abdomen performed two weeks after externalization of the VPS demonstrated an interval improvement, and there was no evidence of bowel edema or free fluid (Fig. 2b). Additionally, the patient's pancreatic enzymes returned to normal. Three weeks after the onset of the distal shunt failure, a new sterile distal catheter was internalized into the contralateral peritoneum. A posterior auricular incision was made, the distal catheter was cut and removed, and a new sterile distal catheter was connected with a straight connector. The distal catheter was passed to a new incision made at the abdomen contralateral to the externalized catheter and was placed into the peritoneum. Post-operatively, the patient did well and did not demonstrate evidence of hydrocephalus clinically or on subsequent imaging. The patient did not develop any subsequent

abdominal difficulties. On follow-up examination at two months, the patient was asymptomatic and was not displaying any signs of shunt failure or shunt infection.

Discussion

The authors report the case of a 56-year-old woman with a distal shunt failure resulting from acute *Clostridium difficile* pancolitis and pancreatitis. Although there are rare reports of pancreatitis occurring as a result of a colitis [3,21], in the current case, the pancreatitis preceded the colitis and most likely occurred secondary to a more common etiology such as medications. The patient's pancreatitis and diarrhea, which led to poor nutrition and low protein levels, as well as the inflammatory response from the *Clostridium difficile* colitis and pancreatitis resulted in ascites. Ascites in the setting of infection or inflammation is known to decrease the functional absorptive surface area [5]. Thus, the authors suspect shunt failure occurred because of an increased intra-abdominal pressure and decreased osmotic gradient and absorptive area that limited CSF flow and absorption, respectively. Although the pancreatitis contributed, the diffuse colitis was the triggering event that resulted in the acute shunt failure.

In general, about 60% of new shunts remain patent after 1 year, but this proportion drops to 50% at 2 years [8,9,22,23]. Early shunt failure, which occurs within 2 years of shunt placement, is most often attributable to proximal failure, whereas distal shunt failure is more common after 2 years from shunt placement [8,12,16]. Obstruction is the most common cause of distal shunt failure, accounting for 74% of re-operations [9]; infection is the second most common cause. Although distal obstruction can result from a VPS infection, the shunt failure in the current case was not due to a shunt infection. Distal

obstruction often occurs because of pseudocysts and bowel obstructions, and sometimes because of bowel perforation or improper shunt placement in the abdomen [7,11,19]. It may also occur as a result of outgrown, fractured, disconnected, or occluded catheters [1].

Most patients in previously published series with distal shunt failure and associated abdominal pathology had localized peritonitis caused by a distal shunt infection or a focal intra-abdominal process such as appendicitis [15,17,18]. Distal shunt failure secondary to other intra-abdominal or pelvic etiologies has also been described. Lee et al. [10] described a case of distal shunt failure secondary to ovarian hyperstimulation syndrome. In that case, shunt dysfunction was attributed to intra-abdominal hypertension caused by ascites, which led to limited CSF flow. VPS failure in pregnancy is a well-documented occurrence with a reported incidence of 27.5%; the shunt failure is usually secondary to increased intra-abdominal pressure during gestation [13]. Snow et al. [20] noted several cases of shunt failure that were due to a hypersensitivity-like reaction around the shunt that led to malfunction. Necrotizing enterocolitis (NEC) in one cohort was shown to be associated with more distal obstruction in patients with posthemorrhagic hydrocephalus who underwent placement of a shunt than in similar patients without NEC [14]. Cases of distal shunt failure following intraperitoneal, urological procedures have also been reported [2]. Thirty-one percent of the patients in one series had complications requiring shunt revisions; most complications occurred because of peritoneal contamination, catheter obstruction, abdominal pseudocyst, or distal catheter disconnection [2]. VPS failure has also been reported after laparoscopic surgery; this was thought to be a result of peritoneal insufflation through an incompetent shunt valve, which caused impaction of soft tissue and or air within the distal catheter [4].

The shunt failure secondary to *Clostridium difficile* pancolitis and concomitant pancreatitis in our patient is unique in the literature. After resolution of the pancolitis and pancreatitis, a new sterile distal catheter was internalized into the peritoneum without complications or evidence of further shunt failure. This case not only demonstrates an interesting cause of distal VPS failure, but also points to the importance of considering distal etiologies in patients with acute neurological decline and concomitant diverse abdominal pathology.

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Figure Legends

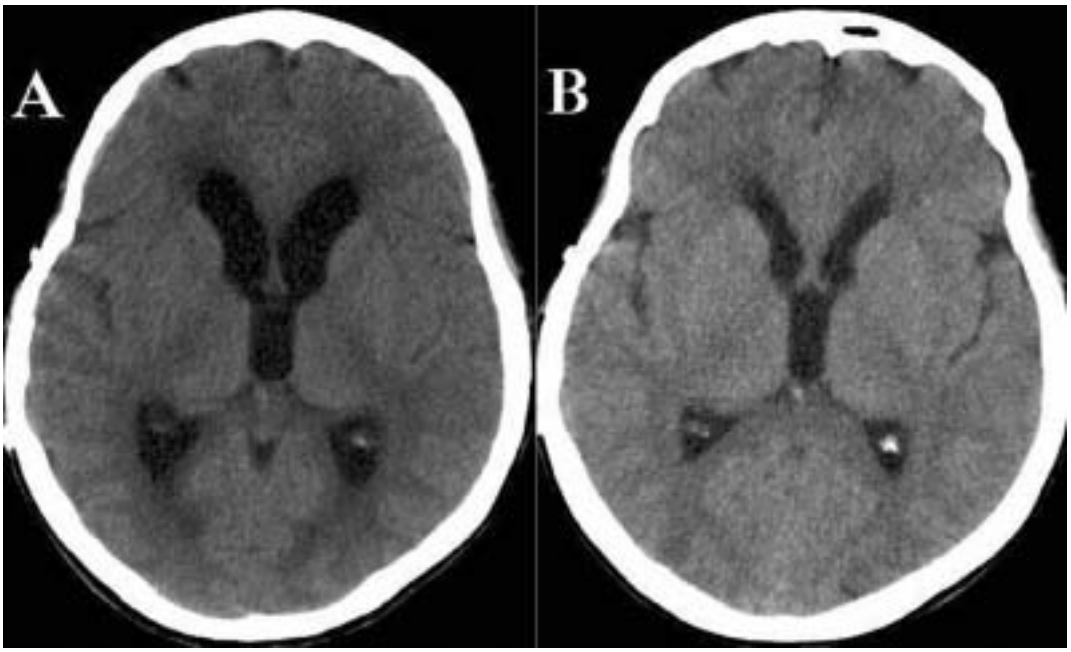

Figure 1. A: Head CT scan displaying ventriculomegaly obtained the day the patient developed decreased mental status. B: CT scan performed two days after externalization of the VPS demonstrating decreased ventricular size.

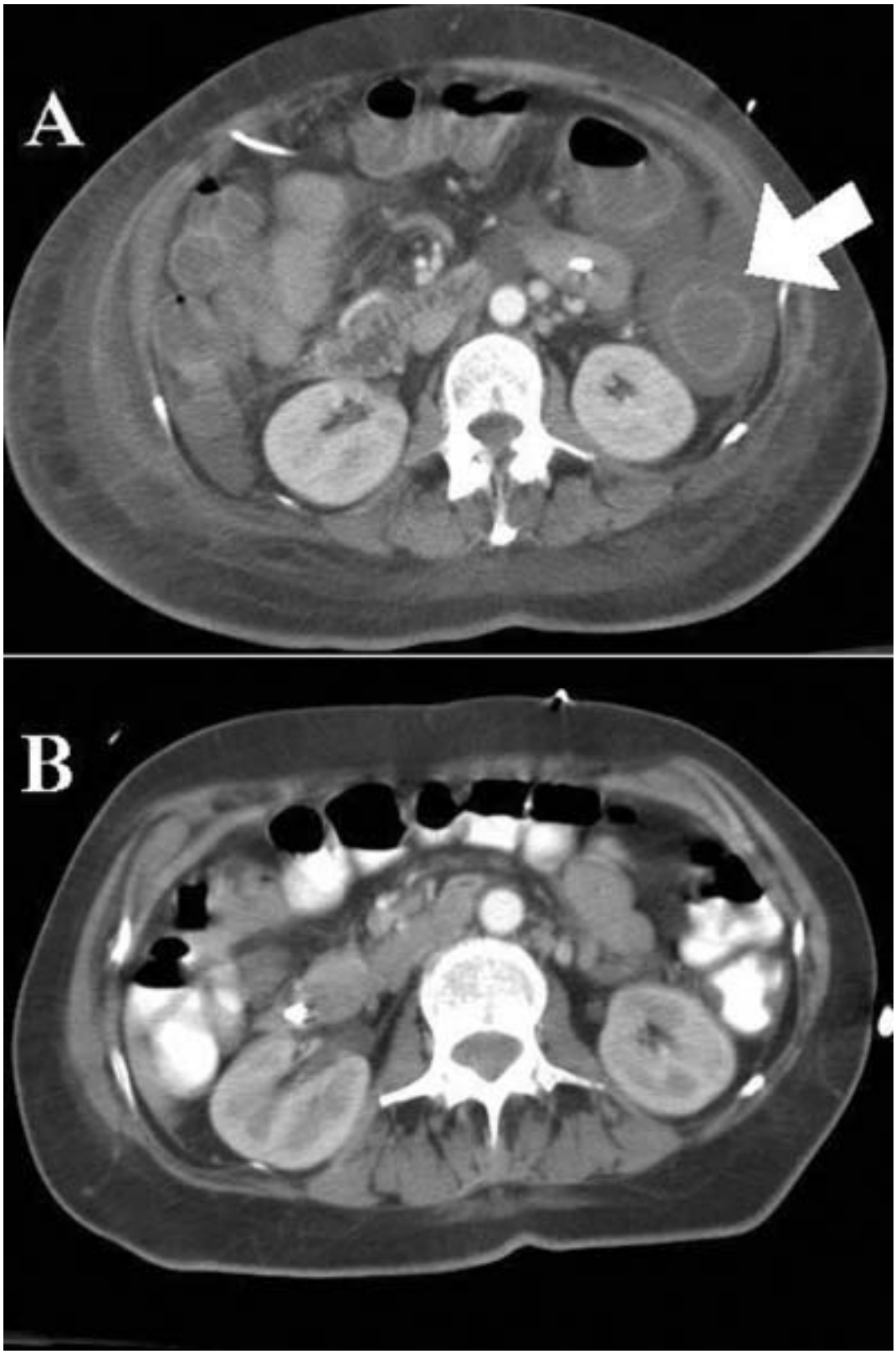
A
B

Figure 2. A: Abdominal CT scan with intravenous contrast performed on the day the patient developed decreased mental status. The image demonstrates diffuse colon wall thickening and edema (arrow) and ascites. B: Abdominal CT scan with oral and intravenous contrast taken two weeks later displaying a marked interval improvement in colon wall thickening and ascites.