CASE IN POINT Tubing in the Transverse Colon: An Uncommon Complication

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An 8-year-old boy with cerebral palsy and seizure disorder presented with a 1-day history of increased seizures. His mother reported that he had been in his usual state of health, without fevers or signs of illness, prior to having 3 episodes of tonic-clonic seizure activity within a 24-hour period. Each episode of seizure activity lasted 10 minutes. She also reported that the boy had missed 2 doses of phenobarbital 2 days prior to presentation.

The child had been born at 28 weeks of gestation, and his past medical history was significant for a history of grade III intraventricular hemorrhage and subsequent hydrocephalus requiring ventriculoperitoneal (VP) shunt placement. Four shunt revisions were required previously due to shunt infections and malfunctions, the last of which occurred 4 months prior to presentation. The boy had reported no sick contacts, and his review of systems was only positive for constipation.

On physical examination, the patient was lying in bed in no distress, and he easily aroused to voice and stimulation. He was afebrile with vital signs that were normal for his age, and he was at his neurologic baseline, according to his mother.

The boy's pupils were equal and reactive. His mucous membranes were moist, and his lungs were clear. The results of an abdominal examination were only notable for palpable stool in the left lower quadrant. He had spastic lower extremities bilaterally and 3+ reflexes in his lower extremities with ankle clonus. Shunt tubing was palpable over his scalp and neck. His neck was supple, and there were no meningeal signs.

His white blood cell count and differential were normal with a slightly elevated C-reactive protein at 28 mg/L (reference range 0-8 mg/L). A shunt series was obtained to evaluate continuity of tubing. Although tubing was intact, it was noted that the distal portion of the shunt tubing had perforated the transverse colon, with the tip lying in the descending colon (**Figure 1**). An abdominal computed tomography (CT) scan confirmed these findings (**Figure 2**).



Cerebral spinal fluid (CSF) analysis via shunt tap revealed cloudy fluid with 0 white blood cells, 656 / μ L red blood cells, glucose of 8 mg/dL, protein of 23 mg/dL, and visible bacteria. Gram staining showed gram-negative rods and gram-positive cocci in pairs and clusters.

The boy was initially started on cefoxitin. Surgery and neurosurgery externalized his shunt, and pediatric surgery repaired his colon perforation. His CSF culture ultimately grew significant amounts of 2 strains of *Escherichia coli*, as well as *Klebsiella pneumonia*, *Enterococcus avium*, and *Enterococcus gallinarium*. Antibiotics were tailored to the sensitivities, and the patient was treated until repeat CSF cultures were clear. The VP shunt was subsequently revised and internalized as a ventriculopleural shunt.

UISCUSSION

Since its introduction in 1905, VP shunt surgery has come to be the most widely used treatment for hydrocephalus.¹ Abdominal complications occur in 10% to 30% of patients with VP shunts, but spontaneous bowel perforation from the VP shunt catheters is rare, with an incidence of 0.01% to 0.07% based on most publications.²

The pathogenesis of bowel perforations by shunt catheters is unclear. One single-institution study notes that predisposing factors include nonambulatory status, history of meningeal infection or inflammation in the perinatal period, or a previous episode of shunt infection. Pediatric patients are also more likely to have this complication compared with adults.¹

Previous case reports have postulated 4 etiologic theories: 1) children with myelomeningocele and congenital hydrocephalus may be at increased risk due to bowel wall weakness from deficient innervation, 2) bowel wall weakness exists secondary to a preexisting but undeclared shunt infection,^{2,3} 3) bowel wall permeability increased due to inflammation secondary to an allergic reaction to catheter,² or 4) an encasing fibrosis around the end of the shunt tubing (found often upon surgery or autopsy) causes a pressure necrosis on nearby bowel wall.² All types of catheters have been linked to this complication, though hard-tipped peritoneal catheters increase the risk of performation.³ Our patient's only risk factors based on these published findings were his age and a history of previous shunt infections.

Timely diagnosis and a high degree of suspicion for this rare complication are important because perforations can lead to not only peritonitis but also meningitis and ventriculitis, with mortality rates approaching 15%.² Fever and abdominal symptoms such as diarrhea, vomiting, or pain are present in about 40% of patients, but signs of peritonitis are present in less than 25% of patients and are often a late finding.^{1,2}

Our patient had a longstanding history of constipation, which is common in patients with cerebral palsy and spasticity, but the constipation seemed unrelated to the tubing perforation. He also presented with seizures, which is found in only about 4% of presentations,², though it is reasonable to presume our patient had ventriculitis based on the spinal fluid findings. While the diagnosis is apparent when extrusion of distal catheter through the anus occurs, further evaluation with plain films of the chest and abdomen or abdominal CT with contrast may be necessary when the history is less clear.³ Shunt CSF cultures are positive in only about 50% of the cases; when positive, as was the case with our patient, cultures often grow 3 or more different organisms. The predominant organisms found in literature reports are enteric gramnegative organisms, with *E coli* being most common.^{1,2}

Bowel perforation from shunt tubing requires an individualized multi-step treatment approach. Initially, externalization of the shunt, broad-spectrum intravenous antibiotics, and bowel rest are necessary. Percutaneous removal of the shunt tubing is acceptable unless peritonitis or an abdominal abscess are suspected; a laparotomy is usually indicated in these cases based on the need for surgical drainage and likelihood of poor healing.

This initial treatment is later followed by narrowing of antibiotic choice based on organisms grown, if any, and a slow introduction of oral intake. As most of these patients are dependent on the shunt, the VP shunt may be replaced once CSF cultures are negative, though atrial or pleural shunts may be required if abdominal issues have not resolved at time of replacement.^{1,2} Due to the number of VP shunts our patient had previously had placed, a ventriculopleural shunt was placed prior to discharge.

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