Shunt Malfunction Without Device Occlusion: A Case Report

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ABSTRACT

Shunt malfunction is the most common cause of ventriculoperitoneal shunt failure. In literature, occlusion of the tube with brain parenchyma, choroid plexus, blood, and proteinaceous debris has been suggested as a mechanism of obstruction. We herein report a case of shunt malfunction without any identifiable occlusion. Our case findings suggest that unapparent abdominal pathology, including inflammation and fibrosis, should be considered when treating shunt failures.

Keywords: Ventriculoperitoneal shunt; Hydrocephalus; Inflammation; Peritoneal cavity

INTRODUCTION

A ventriculoperitoneal shunt (VPS) is a common neurosurgical procedure to treat hydrocephalus. However, it is estimated that approximately 11%–25% of VPSs fail within the first year after initial placement.9,10,12 The most common cause of VPS failure is shunt obstruction.9 Many studies suggested possible mechanisms and contributing factors, yet its nature is still unknown. It has been speculated that the occlusion of the system with various materials including brain parenchyma, choroid plexus, blood, and proteinaceous debris is the main cause of the obstruction.9 Here, the authors introduce a case of shunt malfunction without occlusion of the device and a review of the related literatures.

CASE REPORT

A 25-year-old male patient was transferred from a rehabilitation hospital to our emergency department with a recurrent high fever. He had experienced a traumatic brain injury from a motorcycle accident 2 years earlier and currently was in a minimally conscious state. At the time of the initial injury, he underwent decompressive craniectomy for both sides of the cranium due to severe brain swelling and developed posttraumatic hydrocephalus. A VPS was placed on the left side 15 months earlier, which was removed 12 months ago due to ascending...
Conflict of Interest
The authors have no financial conflicts of interest.

Laboratory tests revealed a high serum C-reactive protein (14.35 mg/dL) level, elevated white blood cell count (711/mm$^3$), and protein (105.9 mg/dL) in cerebrospinal fluid (CSF) acquired from the shunt reservoir. Brain computed tomography (CT) scan revealed an enlarged ventricle size and the abdominal CT scan showed peritoneal fat stranding (FIGURE 1). Later, the CSF culture was reported to contain oxacillin-resistant Staphylococcus caprae.

Intravenous (IV) vancomycin was administered immediately. Initially, removal of the infected shunt device was considered. However, because of the patient's poor medical condition and the unwillingness of the family to have the patient undergo a surgical procedure, the authors tried to salvage the device. CSF (50 mL) was drained, and 10 mg of vancomycin was administered through the shunt reservoir twice a day along with standard IV vancomycin for a total of 2 weeks. Serum vancomycin was maintained in adequate level through therapeutic drug monitoring, even though CSF vancomycin concentration was not measured because of technical difficulty. CSF culture conversion was achieved after one week of treatment and the patient did not show any signs of infection. However, the shunt was still malfunctioning and a shunt evaluation test using technetium-99m DTPA revealed distal catheter malfunction (FIGURE 2).

The authors assumed distal catheter blockage due to proteinaceous material from the infected CSF and started flushing the device with 4 mg of tissue plasminogen activator (rt-PA) daily for one week by manually pressing the proximal occluder. The thrombolysis attempt failed, and a second isotope test revealed a more severe distal valve malfunction (FIGURE 2). The abdominal CT scan at this point showed cleared peritoneal fat stranding and no sign of complications including malposition, breakage, kinking, or pseudocyst formation.
The device was removed through an occipital skin incision. However, after careful examination of the recovered device, we concluded that it was fully functioning. Neither the proximal nor distal catheter showed signs of blockage. Saline pushed from the proximal catheter with slight pressure or by pushing on the reservoir drained well into the distal catheter. External ventricular drain was placed at the right Kocher’s point. Two weeks after removing the malfunctioning device, a new device was placed on the left side. The patient was stable and showed no signs of infection for 2 months and was transferred to a rehabilitation hospital.

**DISCUSSION**

The most common cause of VPS failure in both pediatric and adult populations is shunt obstruction followed by infection. Obstructions can occur anywhere in the system, including the proximal catheter, valve system, or distal catheter. Even though the proximal catheter was reported as the most common site of obstructions in most studies, peritoneal catheter obstructions occur in approximately 20% of the shunt failures.

![FIGURE 2. Technetium-99m DTPA shunt evaluation test. (A) Test before the thrombolysis attempt. The arrow indicates the obstruction of flow in the distal catheter. (B) Test after thrombolysis attempt. No flow is visible after the reservoir.](image-url)
Hypotheses regarding obstructions have been focused on occlusion of the tube with various materials including brain parenchyma, choroid plexus, blood, or proteinaceous debris. However, recent studies suggested inflammatory reactions to the device or CSF contents could play a key role in malfunctions. Studies have reported a visible fibrous capsule surrounding the peritoneal catheter tip removed by either laparotomic or laparoscopic approaches. Del Bigio et al. suggested that using a graphite preservative in a side-slit distal catheter could trigger an immune reaction, which is supported by the previously reported higher failure rate of side-slits.

In the treatment of an obstructed VPS, the mainstay treatment is to change the device. However, attempts to selvage non-functioning VPSs have been reported, especially for patients with poor medical conditions and a high risk of surgery. Fibrinolytic agents such as streptokinase, urokinase, and rt-PA have been used to treat obstructed shunts. Even though causes of obstruction in most of the forementioned cases were blood clot or proteinaceous debris from tumor, authors used thrombolytic agent because we suspected occlusion of the distal catheter where flow is blocked in shunt evaluation test (FIGURE 2A).

In our case, neither fibrinolytic agents nor flushing pressure from the reservoir cleared the obstruction. There was no identifiable occlusion or mechanical malfunction in the recovered shunt. After consultation with five general surgeons, the authors concluded that an inflammatory reaction and fibrous adhesion in the distal catheter caused a functional obstruction of the peritoneal catheter, rather than visible occlusion of the catheter. Considering S. caprae is a skin commensal, and increased peritoneal fat stranding was visible in the initial CT scan, authors also agreed that surgical infection of the shunt device is the probable cause of the abdominal pathology.

An interesting standpoint is that all of the consulted general surgeons preferred changing the device to exploring the distal catheter by either laparotomy or laparoscopy. The reported rationales were that 1) changing the device is a much quicker and simpler solution and 2) exploration does not guarantee functional recovery. However, consultation with a general surgeon is advised in the case of severe adhesion and when recovery from the proximal approach is not feasible.

CONCLUSION

We report a case of peritoneal catheter obstruction without an identifiable occlusion. When treating VPS dysfunction, unapparent abdominal pathology, including inflammation and fibrosis, should be considered. Thus, consulting a general surgeon is advised. More studies are required to understand the immunopathology of shunt obstructions.

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