Ventriculoperitoneal Shunt Malfunction in a Pediatric Patient Due to Compression by Skull Growth: A Case Report

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Abstract

There are various causes of ventriculoperitoneal shunt (VPS) failures. Patients who receive shunt placement during childhood need follow-up for decades as they grow, especially in the early periods of life. Herein, we report a rare case of mechanical shunt obstruction in a pediatric patient in whom a cramped burr hole and skull growth compressed the tube and obstructed cerebrospinal fluid flow. A 6-year-old girl presented to our hospital with nausea and headache. She was born preterm and developed intraventricular hemorrhage followed by VPS placement for hydrocephalus; thereafter, she had no need for shunt revision until this admission. After careful evaluation of the patency of the shunt system, the presence of tube stenosis was suspected at the site of the shunt tube penetrating the burr hole of the skull. During the operation to revise the shunt tube, a compressed tube was observed at the exit from the skull. After enlarging the narrowed burr hole and reconstructing the proximal catheter, her symptoms immediately improved. Previously, only one case of shunt malfunction due to tube compression from bone growth has been reported in a pediatric patient with osteopetrosis. To the best of our knowledge, such a condition has never been described in pediatric patients with no metabolic bone disease. Although it is rare, obstruction at the exit from the skull due to bone growth should be included in differential diagnoses for young patients during a long follow-up after VPS.

Keywords: bone growth, hydrocephalus, neonate, shunt malfunction, ventriculoperitoneal shunt

Introduction

Hydrocephalus is a common neurosurgical disorder in both pediatric and adult populations. Venticuloperitoneal shunt (VPS) is a treatment for hydrocephalus used in 98% of pediatric hydrocephalus treatments.3,5 However, pediatric patients frequently require multiple revision surgeries after VPS, which are often performed within 2 years of the initial procedure.2,3 The average number of revision surgeries for pediatric shunts is reportedly 2.66 per case.7 The common reasons for shunt failures include infections, catheter obstruction, and the disconnection of the shunt tube due to the increase in the body size.2,7,8

Herein, we present a rare case of a pediatric patient with shunt malfunction that occurred 6 years after the initial VPS placement. In the present case, the compression of the tube occurred at the site at the exit from the skull. We conceive that compression by the growing bone may not be well recognized as a cause of shunt malfunction because of its rare incidence.

Case Report

A girl was born at 23 weeks of gestation and developed a grade 3 intraventricular hemorrhage that was initially treated with a subgaleal shunt and then converted to a VPS using Codman Hakim Programmable Valve with SiphonGuard (Codman Corporation, Raynham, MA) at the age of 3 months. Subsequently, the patient showed normal growth and development. After 6 years of follow-up, the patient presented with headache and nausea for 10 days and visited our hospital. Compared with her previous series of computed tomography (CT) scans, the size of the ventricles increased (Fig. 1). The valve opening pressure was adjusted to a lower value. Her symptoms tentatively improved for the next 24 hours but worsened again. On
the pumping test, the silicon dome of the device was easily compressed with a moderate force. However, it reinfated extremely slowly, suggesting problems at the proximal catheter. A shuntography was also performed to evaluate the patency of the VPS. The injected contrast medium entered both the proximal and distal tubes (Fig. 2A); how-

**Fig. 1** Axial computed tomography scans obtained during follow-up (A) and at admission (B) showing an increase in the size of ventricles.

**Fig. 2** A, Shuntography demonstrated that the contrast medium entered both the proximal and distal tubes. B, Magnification of the squared portion in A. Severe stenosis of the tube (black arrow) was observed where it passed the narrowed burr hole. C, Three-dimensional (3D) CT reconstruction showing the narrowing burr hole (red circle). D, Computed tomography (CT) scan immediately after the shuntography presented a reflux of the contrast medium in the ventricles. E, An axial CT scan 24 hours after the shuntography demonstrating disappearance of the contrast medium in the ventricles. N, needle; V, valve.
ever, some resistance was noted when injecting the contrast medium toward the ventricular tube with manual compression on the tube distal to the valve. Severe stenosis of the ventricular tube was suspected at the site of the shunt tube penetrating the burr hole of the skull (Fig. 2B). Three-dimensional (3D) CT reconstruction images were further obtained, which demonstrated that the tract of the proximal shunt tube adjacent to the burr hole was not clearly traceable distally to the burr hole (Fig. 2C). The intraventricular contrast medium was washed away on serial CT scans in 24 hours after the shuntography, indicating that the obstruction was not complete (Fig. 2D and E). These situations prompted us to explore the possibility of the malfunction of the proximal catheter. Since her symptoms persisted and worsened, shunt revision was planned under the suspicion of local stenosis of the shunt tube.

After opening the cranial incision, the granulated tissue along the tube was removed. After exposure, the tube was observed to be strongly compressed by the bone at the exit from the skull, where the previous burr hole had been narrowed by growth for years (Fig. 3A). To release the obstruction, the previous burr hole was enlarged using a rongeur. The stenotic portion between the ventricle catheter and the valve was replaced with a new one (Fig. 3B). When cutting the tube just at the site of the dura mater for replacement, active drainage of the cerebrospinal fluid was observed, indicating that the ventricle catheter tip was not occluded. The prompt reexpansion of the silicon dome after pumping was also confirmed.

Her postoperative course was unremarkable with immediate resolution of symptoms. A follow-up of head CT for 1 week after the revision surgery showed a reduction in the size of the ventricle (Fig. 3C). A postoperative 3D CT reconstruction image showed no stenosis of the shunt tube with an enlarged burr hole (Fig. 3D). The patient showed no recurrence of symptoms during a 6-month follow-up period. A written agreement for the publication of the case report was obtained from the parents of the patient.
Discussion

Shunt malfunction can occur under various mechanisms at different intervals after shunt surgery. According to previous reports, the causes may include obstruction of the ventricular catheter, abdominal catheter, disconnection, and infection accounting for 27%, 15%, 11%, and 9% of the cases, respectively. There was a single case report of shunt malfunction due to compression of the tube by bone growth in a pediatric patient with osteopetrosis. In addition, a case of a partially collapsed shunt valve 9 years after VPS due to the bony-hard scar tissue around the valve has been reported. However, shunt malfunction due to bony compression has never been reported in patients with no metabolic bone disease. Shuntography is effective to diagnose the occlusion of the shunt tube; however, the findings must be carefully evaluated in cases with incomplete occlusion or stenotic tube. In the present case, 3D CT reconstruction imaging was useful to detect the local stenosis of the shunt tube. Notably, the burr hole opening for ventriculo-subgaleal shunt and VPS was conducted when she was 23 weeks old and 3 months old, respectively. Stretching of shunt tube due to the subsequent increase of body size might have caused localized reactive bone growth at the site of the shunt tube penetrating the burr hole of the skull, resulting in the bony engulfing of the tube. Given that the shuntography revealed incomplete occlusion, the further temporary stretching of the tube caused by changes in body position might have exacerbated the blockage. However, the reasons why this phenomenon specifically occurred to this patient are uncertain. Although it is rare, shunt tube obstruction could occur due to bone growth even during the normal process of skull growth for pediatric patients who undergo burr hole opening and shunt tube placement in the very early neonatal or infantile period.

Conclusion

We report a rare case of shunt malfunction due to bone growth unrelated to metabolic bone diseases. This condition, although rare, should be included in the possible causes for delayed shunt malfunction. Based on our experience, 3D CT reconstruction imaging effectively detected the narrowing of the tube due to bony compression.

Acknowledgments

None

Conflicts of Interest Disclosure

The authors have no conflict of interest to declare.

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