

Commentary

Hydrocephalus is fairly common in children and the United States National Institute of Neurological Disorders and Stroke in 2011 estimated that hydrocephalus affects one in every 500 children.^[1] The most common and effective treatment of hydrocephalus involves the surgical placement of a shunt by neurosurgery. The shunt allows the excess and obstructed cerebrospinal fluid (CSF) to be drained from around the brain, thus preventing the dangerous accumulation of CSF and subsequent brain injury. Although overall, ventriculoperitoneal (VP) shunt has proved to be a simple and effective procedure with confirmed benefits to many patients, nevertheless as seen in the present series,^[2] the shunt procedure can be associated with many complications, for example, distal shunt obstruction, infection, and migration of the catheter.

The case of vaginal migration described by the authors is the second reported case in the English literature as the first case was reported in 1975. There is no doubt about the rarity of transvaginal migration as a complication of VP shunt and many neurosurgeons may not see such cases in their entire career. The authors should be congratulated for reporting such a rare case after a long gap (35 years). Apart from this, the major contribution of this article is to create awareness about the rare complications associated with VP shunt procedures in the neurosurgical world.

The literature shows that the probability of shunt failure increases with the passage of time. The risk of an individual experiencing shunt failure reaches its maximum within first few months after the surgery. At one year, it ranges from 25 to 40%. The annual

failure rate after one year remains about 4–5%. The mean survival for shunt had been estimated at five years.^[3] The migration of VP shunt reported in this article happened within one-and-a-half years after the initial VP shunt surgery, which is roughly within the most risky period according to Drake (1994).

Because of the uncommon nature of the complications, it is a little hard to determine the exact pathogenesis of VP migration through the vagina or anus. The authors proposed that poor nutritional status along with infection were the precipitating causes. The interactions between the tip of the lower end of the shunt and vaginal or colon wall may have caused chronic inflammation. The chronic pressure by the stiff and sharp end of the shunt exerted by the CSF pulsations on the vaginal or colon wall with inflammation may eventually cause the tip of the shunt to penetrate the walls. The speculated pathogenesis is logical, based on the observations of poor nutritional status (e.g., anemia in case 1, low body weight in case 3) and positive result of testing for bacteria in one case (case 1). However, in the other case (case 3), aerobic cultures were negative for both CSF and shunt tip. Therefore, there is not enough evidence to support a role of infection in case 3. Other factors^[4] that can contribute to the anal or vaginal migrations of VP shunt reported in this article may include:

- Proximal shunt malfunction due to ventricular catheter occlusion
- Distal (peritoneal) shunt obstruction due to improper placement at the time of initial procedure; low-grade infection
- Obstruction of valve due to bacterial proliferation

from the development of an immune-mediated cellular reaction and from contamination by clot, parenchyma, or debris during insertion.

- Fracture of the shunt due to poor design of the shunt, the material used within a shunt, and the initial surgical technique
- Disconnection of the shunt occurring at stress points where connectors are used
- Overdrainage of CSF through VP shunt related to valve
- Existing diseases of abdominal organs, for example, chronic infections of organs or abnormal organ development and/or malformation

The authors did not provide a thorough description (e.g., the completeness of shunt catheter, the condition of the shunt valve, the length of the shunt catheter, the initial surgical procedure) in the report. Thus, it is impossible to determine whether any of the above factors may also be involved in the rare migrations reported.

The anal or vaginal migrated shunt tubes were surgically removed in cases 1 and 3 followed by endoscopic third ventriculostomy to reduce intracranial hypertension. The authors claimed that in case 1, the child was fine after six months; however, there is no follow-up information for the third case. There is also no information as to whether any further treatments (e.g., VP shunt) were given to the two children for correction of hydrocephalus. The authors are encouraged to follow up the two cases further, given their rarity. The absence of follow-up

information as well as incomplete descriptions of the cases are the weaknesses of this article.

Yi Shao, Lei Zhang¹

*Department of Neurosurgery,
Qilu Hospital, Shandong University, Jinan, People's Republic of
China, ¹Cell and Tissue Therapies Unit, Biological Sciences Section,
Office of Scientific Evaluation, Therapeutic Goods Administration,
Woden, ACT, Australia*

Address for correspondence:

Dr. Yi Shao,
Department of Neurosurgery, Qilu Hospital,
Shandong University, Jinan, People's Republic of China.
E-mail: Shaoyi777@yahoo.com.cn

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