INTRAVENTRICULAR MIGRATION OF PERITONEAL SHUNT CATHETER: A CASE REPORT

[1,2]Idowu OE, [2]Arowolo WB

[1]Neurosurgery Unit, Department of Surgery, Lagos State University College of Medicine and [2]Lagos State University Teaching Hospital, Ikeja, Lagos, Nigeria.

Correspondence : OE Idowu

E-mail:oeidowu412@yahoo.com

Abstract

Ventriculoperitoneal shunt is associated with various complications. A rare complication of ventriculoperitoneal (VP) shunt is intracranial migration of the peritoneal shunt catheter. We present a case of intraventricular migration of a peritoneal shunt catheter in a 26-month old male child who had ventriculoperitoneal shunt done when he was 14 months of age, but represented a year later with features of shunt malfunction. The clinical history, surgical treatment, and pathophysiological mechanism involved in shunt migration are discussed. The management of this very rare problem is discussed and the literature reviewed.

KEYWORDS: *Endoscopic third ventriculostomy, Shunt migration, Ventriculoperitoneal shunt*

Introduction

The definitive management of hydrocephalus is surgical intervention. This is usually done either via an endoscopic third ventriculostomy or ventriculoperitoneal shunt.

Ventriculoperitoneal shunt (VP) is one of the most common neurosurgical procedures done. The attractive feature of a ventriculoperitoneal shunt is its nearly universal applicability in the management of hydrocephalus, irrespective of aetiology. Unfortunately, it also has a high complication rate which varies widely[1]. This is divided into infective, mechanical failure and shunt migration.

Migration of ventriculoperitoneal shunt catheter is a rare complication after shunt surgery. When it occurs, the migration of the catheter may be proximal or distal. Unlike distal migration, proximal shunt migration is a rare event[2-6].

The sites reported for proximal migration include subgaleal space, scalp, subdural space, subdural haematoma cavity and ventricles[7-10]. When migration occurs it is noted to be more frequent with hard and spring loaded shunt tubes[2,3].

We report a case of intracranial ventriculoperitoneal shunt migration. The pathophysiological mechanism of the catheter migration is also discussed.

Case Report

F. F. is a 26-month old male child who had ventriculoperitoneal shunt (Slit and spring, Chhabbra shunt - medium pressure) done when he was 14 months of age (not by any of the authors). The aetiology of the hydrocephalus was congenital aqueductal stenosis. The patient was lost to follow up after the second month of surgery. He represented at the hospital a year after the shunt surgery with a 2-day history of refusal of feeds and vomiting. There was no history of antecedent head trauma, fever or abdominal swelling.

Examination revealed a conscious patient who was lethargic and afebrile. He had dilated scalp veins, macrocephaly (occipitofrontal circumference of 68cm) with bulging and tense anterior fontanelle. There was subgaleal collection of cerebrospinal fluid at the burr-hole site with non palpable shunt hardware subcutaneously. His pupils were 6cm dilated with minimal reaction to light.

The Shunt series showed proximal and distal shunt catheters within the intracranial compartment (Figure 1). This was also depicted by cranial computed tomographic scan. There was associated ventriculomegaly (Figure 2).

The patient was worked up for emergency surgery and subsequently had retrieval of the migrated shunt and insertion of another ventriculoperitoneal shunt with Chhabbra shunt - medium pressure. Intra-operatively the burr-hole site was noticed to be 2cm in diameter with the tip of the catheter just visible. The cerebrospinal fluid was clear and colourless, and under marked pressure. Though we now have facility for endoscopic third ventriculostomy, but this was not available when the patient presented. Post- operative period was uneventful. Patient is now 3 years post-surgery, and he remains stable with improvement in developmental milestones.

Discussion

Shunt complications have been frequently reported in the literature, but intracranial migration of ventriculoperitoneal shunt is one of the most rare complications and constitutes about 0.1- 0.4% of all shunt procedures [2] .

Two principal causes have been suggested to explain the shunt migration into the cranium: the mechanical force moving the shunt catheter into the cranium and the low resistance along the shunt tract.[11,12] Tortuous subcutaneous tract associated with neck movements, negative sucking intraventricular pressure and positive pushing intra-abdominal pressure have been thought to contribute to migration[10]. In childhood, vigorous flexion–extension movements of the head may act as a windlass, facilitating upward migration of the shunt catheter[13]. Apart from this, the distance between the ventricular and the peritoneal ends of the catheter is smaller than in adults, and thus proximal migration is easier[12].

The pressure gradient between the cranial and peritoneal cavities decides the direction of migration[11,14]. For migration to occur, the shunt needs to be under traction and able to move in the subcutaneous tissue. The traction requires a point of fixation and patient's growth. Inflammatory granulation tissue noted around migrated catheters might act as an anchoring point for the "windlass effect" for migration of the shunt[9]. The mechanism of shunt migration involves adhesion, necrosis, penetration, perforation, migration and extrusion[7,11,12].

Making a large dural hole around the ventricular catheter may predispose to periventricular cerebrospinal fluid (CSF) collection and easy migration of the valve system[9]. Mechanical

pressure over the valve by massaging may lead to intracranial migration of the shunt catheter. A mechanism of 'retained memory' of the shunt tubing has also been proposed as the appearance of the coiling was similar to that in the packaging when supplied[8]. Cerrón-Rojas et al reported a case of simultaneous cephalic migration into the intraventricular and subdural spaces[15]. These authors concluded that some factors are necessary: such as detachment of the shunt at the distal end (technical fault), underlying disease (porencephaly), dynamic factors causing expulsion (abdominal peristaltic movements), dynamic translocation factor (neck movements), dynamic attraction factor (increased cerebrospinal fluid reabsorption) and unishunt catheter (offering no resistance to passage through the burr hole site) [7,11,15].

Shimizu et al suggested that the cause of migrated shunt catheter had been related to stress due to seizures, constipation and calvarial osteolysis[12]. In these patients there was no history of constipation, seizures or radiological evidence of calvarial osteolysis. Migration of shunt is not prevented by locks and slip clips[7,16]. Gupta and Mann reported a case of shunt migration in a child with Dandy Walker cyst[7]. Absence of raised intracranial pressure in this case suggests equilibration of cerebrospinal fluid pressure gradient.

Most migrations are said to occur in the early postoperative periods, up to 3 months[7,10]. The time that the shunt migrated in our patient is difficult to ascertain due to non compliance to follow up. There was no history of seizure in our patient, neither radiological features of calvarial osteolysis.

The treatment for ventricular shunt migration is removal of the migrated shunt tube and endoscopic third ventriculostomy or replacement with another shunt if the patient is symptomatic. If the diagnosis was incidental the patient is followed up expectantly.

In developing countries, shunt malfunctions and its complications and are less likely to be addressed in a timely fashion. Avoiding shunt dependency in an environment such as ours is crucial. Hence the option of endoscopic third ventriculostomy may avoid shunt dependency and its complication in many patients.

References

1. **Murali R, Ravikumar V**. Transoral migration of peritoneal end of ventriculoperitoneal shunt: A case report of a rare complication and review of literature. *Pediatr Neurosci* 2008: 3: 166-168

2. **Acharya R, Bhutani A, Saxena H, Madan VS**. Complete migration of ventriculoperitoneal shunt into the ventricle. *Neurol Sci*. 2002; 23:75-77.
3. **Albala DM, Danaher JW, Huntsman WT**. Ventriculoperitoneal shunt migration into the scrotum. *Am Surg* 1989; 55:685-688.
4. **Aoki N**. Lumboperitoneal shunt: clinical applications, complications and comparison with ventriculoperitoneal shunt. *Neurosurgery* 1990; 26:998-1004.
5. **Besson R, Hladky JP, Dhellemmes P, Debeugny P**. Peritoneal pseudocyst-ventriculo-peritoneal shunt complications. *Eur Pediatr Surg* 1995; 5:195-197.
6. **Pereira CU, Silva AD, Barreto AS, Duarte GC, Braz PC**. Peritoneal catheter extrusion through the scrotal sac. Case report. *Rev Chil Neurocirug* 2002; 70-71.
7. **Gupta PK, Dev EJ, Lad SD**. Total migration of a ventriculoperitoneal shunt into the ventricles. *Br J Neurosurg*. 1999; 13:73-74.
8. **Dominguez CJ, Tyagi A, Hall G, Timothy J, Chumas PD**. Subgalial coiling of the proximal and distal components of a ventriculoperitoneal shunt. An unusual complication and proposed mechanism. *Childs Nerv Syst* 2000; 16: 493-495.
9. **Kim KJ, Wang KC, Cho BK**. Proximal migration and subcutaneous coiling of a peritoneal catheter - report of two cases. *Childs Nerv Syst* 1995; 11: 428-431.
10. **Eljamel MS, Sharif S, Pidgeon CN**. Total intraventricular migration of unisystem ventriculo-peritoenal shunt. *Acta Neurochir (Wien)* 1995; 136: 217-218.
11. **Choudhury AR**. Avoidable factors that contribute to the complications of ventriculoperitoneal shunt in childhood hydrocephalus. *Child's Nerv Syst* 1990; 6:346-349.
12. **Shimizu S, Machizuki T, Nakayama K, Fujii K**. Visual field defects due to valve migration into the cranium. *Acta Neurochir (Wien)* 2002; 144:1055-1056
13. **Kanai M, Kawano K, Uehara S**. Upward migration of the L-P shunt catheter into the cranial base. *Osaka City Med J*. 1999; 45:123-127.
14. **Serlo W, Fernell E, Heikkinen E, Anderson H, Von Wendt L**. Functions and complications of shunt in different etiologies of childhood hydrocephalus. *Child's Nerv Syst* 1990; 6:92-94.

15. **Cerrón-Rojas VR, Vega-Basulto S, Varela-Hernández A, Montejo-Montejo J, Gutiérrez-Muñoz FG**. Simultaneous Cephalic Migration of a ventriculoperitoneal shunt into the intraventricular and subdural spaces. *Rev Neurol* 2001; 33:437-439.
16. **Gaskill SJ, Marlin AE**. Pseudocysts of the abdomen associated with ventriculoperitoneal shunts: a report of twelve cases and a review of the literature. *Pediatr Neurosci* 1989; 15:23-26.

Figure 1: Shunt series (skull X-ray) showing coiled intracranial shunt catheter

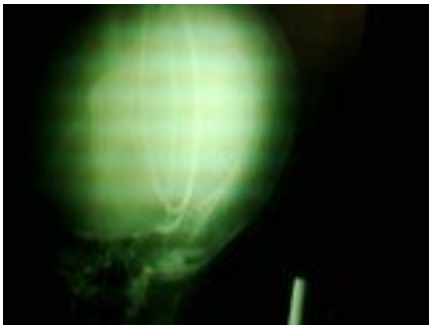

Figure 2. Computed tomography showing intraventricular shunt catheter