

Ventriculoperitoneal Shunt Migration into the Urinary Bladder and Prostatic Urethra Demonstrated on Imaging: A Case Report

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ABSTRACT

Ventriculoperitoneal (VP) shunting is the most common procedure for cerebrospinal fluid diversion in Hydrocephalus (HC); problems such as blockage, infection, and distal catheter migration can occur. Intravesical migration of the distal catheter is an extremely unusual complication that can result in chronic urine problems. The authors describe the case of a 44-year-old male with Normal Pressure Hydrocephalus (NPH) who presented with dysuria, burning micturition, and urine incontinence five months after being treated for recurrent urinary tract infections and genitourinary Tuberculosis (TB). Computed Tomography (CT) brain scans revealed ventriculomegaly, which is consistent with shunt malfunction. CT urography revealed the distal VP shunt catheter within the urinary bladder lumen, with delayed images revealing further extension into the prostatic urethra, demonstrating progressive intravesical migration. There were no related peritoneal pseudocysts, abscesses, or ascites. The present case emphasises the importance of cross-sectional imaging in discovering uncommon locations of VP shunt migration and permitting prompt surgical therapy.

Keywords: Computed tomography, Hydrocephalus, Urography, X-ray

CASE REPORT

A 44-year-old male arrived with a five-month history of burning micturition, dysuria, and urine incontinence. He also reported intermittent urinary frequency and urgency, which progressively worsened over the course of his symptoms. There was no history of haematuria, flank pain, or gastrointestinal distress. Despite many rounds of medication for suspected urinary tract infections, his symptoms remained.

The patient had a known diagnosis of NPH, for which a VP shunt was placed four years back. He also had genitourinary TB, which had been detected five months previously, and was receiving conventional Anti-Tubercular Treatment (ATT). Hypertension was identified two months ago and was managed with frequent antihypertensive medication. There was no history of diabetes, cancer, or other chronic conditions. Except for the VP shunt implantation, the patient had no previous surgical procedures.

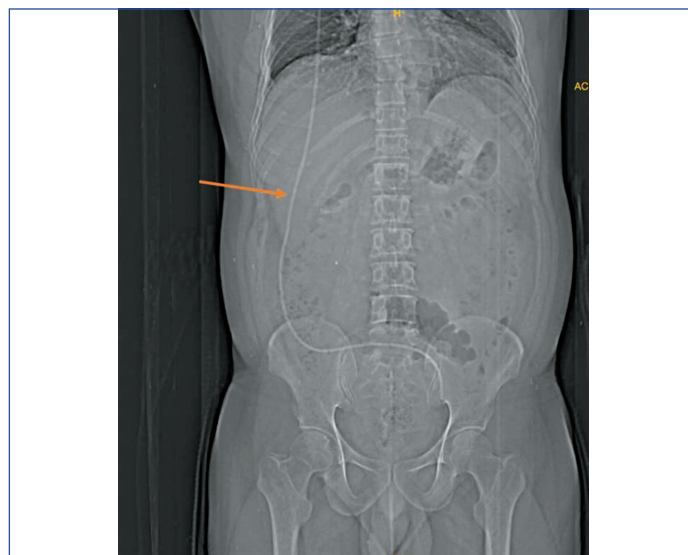
Vital signs were steady upon assessment (Blood Pressure (BP) 130/80 mmHg, Heart Rate (HR) 78 bpm, afebrile). There was no peripheral oedema, jaundice, or pallor. Upon inspection the abdomen was soft and non tender. There was no damage to the VP shunt's palpable subcutaneous route. The patient had a normal neurological evaluation, showing no focal impairments and being awake and focused.

Routine investigations showed a normal complete blood count (haemoglobin: 14.2 g/dL; total leukocyte count: $7.8 \times 10^9/L$; platelet count: $250 \times 10^9/L$), preserved renal function (serum urea: 28 mg/dL; creatinine: 0.9 mg/dL), and normal liver function tests (total bilirubin: 0.8 mg/dL; Aspartate Aminotransferase (AST): 28 U/L; Alanine Aminotransferase (ALT): 32 U/L; Alkaline Phosphatase (ALP): 96 U/L). Inflammatory markers were mildly elevated (C-Reactive Protein (CRP): 14 mg/L; Erythrocyte Sedimentation Rate (ESR): 32 mm/hr), consistent with an ongoing urinary tract infection. Urine analysis showed pyuria.

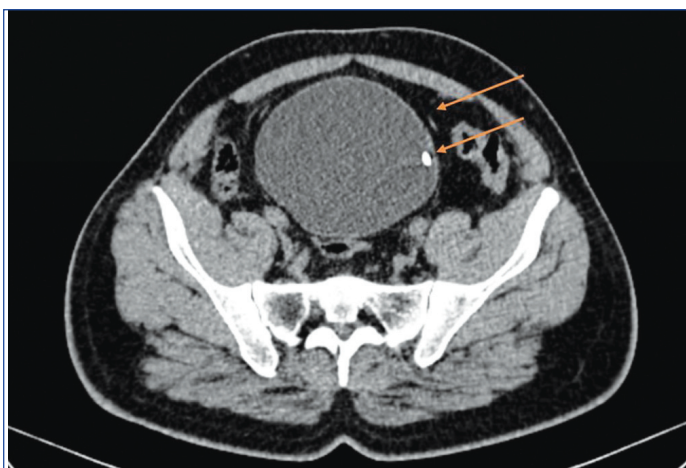
Contrast-enhanced CT urography was performed on a 128-slice multidetector CT scanner with axial acquisition (slice thickness 1-2 mm) and multiplanar reformations. Imaging was obtained in non

contrast, corticomedullary, nephrographic, and 10-minute delayed excretory phases following intravenous administration of iodinated contrast (100 mL, 3-4 mL/s).

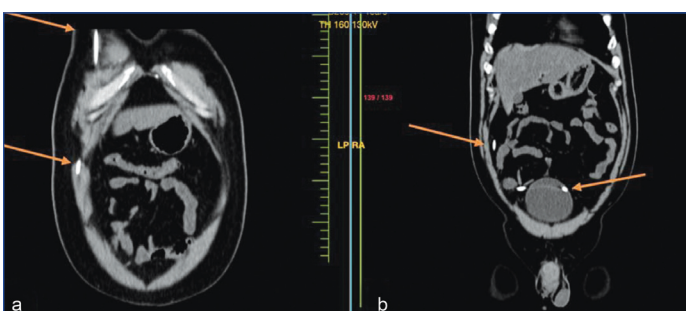
CT urography topogram delineates the course of the VP shunt [Table/Fig-1]. Multiplanar reformatted images demonstrate the distal catheter tip within the urinary bladder lumen, consistent with intravesical migration [Table/Fig-2,3a,b]. The right kidney demonstrated non-obstructive renal calculi as shown in [Table/Fig-4a,b]. Both kidneys had an extrarenal pelvis, with normal excretory function. No peritoneal collections, pseudocysts, ascites, or bowel perforation were noted. Delayed CT urography demonstrated progressive migration of the distal VP shunt tip from the urinary bladder into the prostatic urethra, as shown in [Table/Fig-5a,b], correlating with ongoing lower urinary tract symptoms, including dysuria and incontinence. No additional complications such as bladder wall perforation, pseudocyst, or bowel involvement were noted.



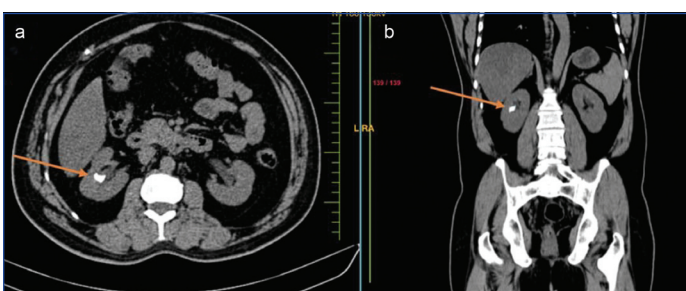
[Table/Fig-1]: Topogram showing the course of VP shunt with intravesical migration of distal tip.



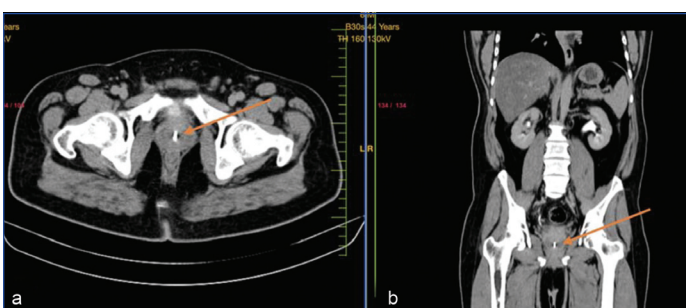
[Table/Fig-2]: Non contrast axial CT section showing distal tip of VP shunt in lumen of urinary bladder (arrow).



[Table/Fig-3a,b]: Non contrast coronal CT sections showing course of VP shunt with its distal tip in lumen of urinary bladder (arrow).



[Table/Fig-4a,b]: Non Contrast CT (NCCT) a) Axial b) Coronal sections showing non-obstructive right nephrolithiasis.



[Table/Fig-5a,b]: Delayed CT urography (10 minutes delayed) a) Axial b) Coronal images showing distal tip extending into prostatic urethra.

CT brain (captured during urography scan): Demonstrated ventriculomegaly of the lateral and third ventricles, consistent with HC as shown in [Table/Fig-6], and the proximal tip of the shunt in the left lateral ventricle as shown in [Table/Fig-7], indicating shunt dysfunction.

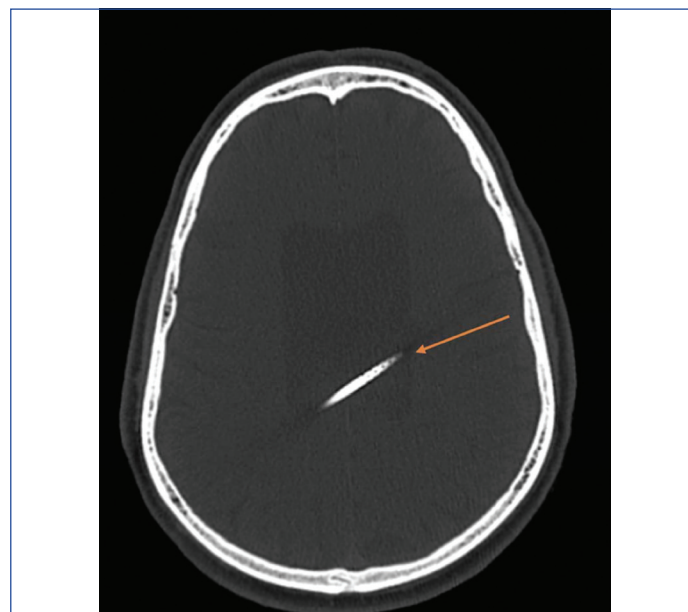
Radiological evaluation established that the patient's urinary complaints were attributable to progressive migration of the distal VP shunt tip from the urinary bladder into the prostatic urethra. Additionally, HC on brain imaging confirmed functional shunt failure.

The patient underwent surgical removal of the VP shunt. Postoperative CT brain revealed: Persistent ventriculomegaly with an Evans index of

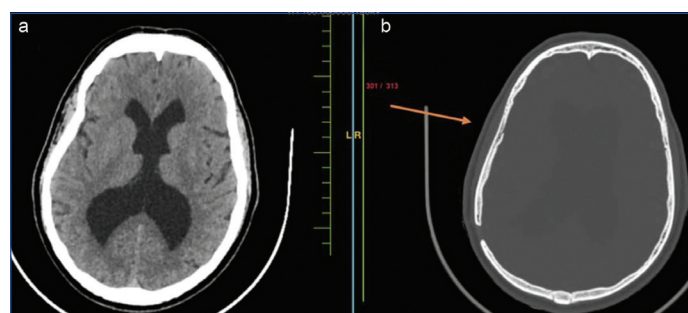
0.30, indicating residual ventricular enlargement as shown in [Table/Fig-8a]. Postoperative burr hole defect in the right parietal bone, as shown in [Table/Fig-8b]. The patient had an uneventful postoperative recovery and was discharged on the 5th postoperative day in stable condition. At the time of discharge, his lower urinary tract symptoms, including dysuria and incontinence, had significantly improved. At the 4-week short-term follow-up, the patient remained clinically stable, with no recurrence of urinary complaints.



[Table/Fig-6]: CT brain showing dilated bilateral lateral ventricles suggestive of ventriculomegaly (arrow indicates VP shunt).



[Table/Fig-7]: CT bone window showing proximal tip of VP shunt in left lateral ventricle.



[Table/Fig-8a,b]: Postoperative CT brain a) Axial sections showing ventriculomegaly in brain window, b) Postoperative burr hole defect in right parietal bone in bone window.

DISCUSSION

The Ventriculoperitoneal Shunt (VPS) procedure is one of the most frequently performed neurosurgical interventions for the treatment of HC, redirecting CSF from the ventricular system to the abdominal cavity. Despite its benefits, VPS is associated with a range of adverse outcomes, particularly those involving the distal catheter [1].

Radiological evaluation of shunt complications: Plain radiographs remain the first-line screening method for assessing gross migration and shunt continuity. However, they are unable to accurately depict organ perforation or related complications due to inadequate soft-tissue contrast. In a child's bladder migration instance, ultrasonography confirmed radiography results of catheter course alteration by detecting a bladder fistula with the shunt tip inside the lumen[2].

The CT imaging provides a definitive assessment by allowing multiplanar visualisation of the catheter, quantification of ventricular dilatation, and evaluation of adjacent organs. Notably, CT brain imaging revealed chronic ventriculomegaly associated with shunt dysfunction, as measured by indices such as the Evans ratio, which may be used to determine HC severity [3].

It is uncommon for a VP shunt to cause bladder perforation. Pohlman GD et al., reported a case of a 14-year-old male with a history of cerebral palsy, severe developmental delay and HC who presented with VP shunt tubing protruding from his urethral meatus [4]. A summary of a few similar cases is seen in the [Table/Fig-9] [2,5-8].

Author	Patient	Site of migration	Clinical presentation	Imaging findings	Management
Obaidy Y et al., [6]	Paediatric	Urethra	Irritability, fever, vomiting, bulging fontanelle	Catheter seen extending into urethra	Shunt removal and revision
Guimarães AS et al., [2]	Paediatric	Urinary bladder	Dysuria, urinary symptoms	Intravesical catheter with knotted formation	Surgical removal
Yang X et al., [7]	Adult	Bladder with urethral extrusion	Chronic urinary complaints	Catheter perforating bladder wall with urethral extrusion	Surgical management
Chen T et al., [5]	Adult	Urethra	Recurrent urinary tract infections	CT revealed that the shunt penetrated the urinary bladder from the intra-abdominal cavity and then went into the urethra.	Operative intervention

Kataria R et al., [8]	Paediatric	Bladder → urethra	Urinary symptoms, catheter extrusion	Intravesical knotting with urethral extrusion	Surgical management
[Table/Fig-9]: Summary of reported cases of distal Ventriculoperitoneal (VP) shunt migration into urinary bladder and urethra [2,5-8].					

If an abnormal catheter trajectory is seen on imaging, there should be a high index of suspicion for visceral perforation or dysfunction, especially in patients who have recently developed or are worsening symptoms such as urgency, frequency, or infection. In addition to adding depth to radiological evaluation, quantitative data like ventricular indices, bladder wall thickness, and contrast extravasation patterns also aid in directing urological and neurosurgery procedures [2].

CONCLUSION(S)

The VP shunt underwent an uncommon intravesical migration that led to both shunt dysfunction and ongoing urinary complaints. To precisely locate the migrated catheter and evaluate any associated HC, multimodality imaging- specifically, CT urography and CT brain- was crucial for guiding the appropriate surgical procedure.

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