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Spontaneous Colonic Migration of a Ventriculoperitoneal Shunt Catheter

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 63-year-old
Final Diagnosis: Spontaneous colonic migration of a ventriculoperitoneal shunt catheter with retrograde CNS infection
Symptoms: Fever • drowsiness
Clinical Procedure: —
Specialty: Neurosurgery • Radiology

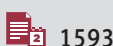
Objective: Unusual clinical course
Background: Silent and spontaneous colonic migration of a ventriculoperitoneal (VP) shunt is an extremely rare, life-threatening long-term complication, driven by chronic foreign-body erosion. It can precipitate severe retrograde central nervous system (CNS) infection insidiously, even in the complete absence of typical abdominal symptoms or peritonitis.

Case Report: A 63-year-old man with a history of VP shunt placement presented 2.5 years after the procedure with fever, somnolence, and elevated inflammatory markers. Brain computed tomography (CT) and contrast-enhanced magnetic resonance imaging (MRI) revealed progressive hydrocephalus and prominent peri-catheter cerebral edema. Subsequent abdominal CT demonstrated spontaneous distal shunt catheter perforation into the colon, with retrospective imaging ruling out iatrogenic injury. Although cerebrospinal fluid and blood cultures were negative, a diagnosis of retrograde CNS infection secondary to colonic perforation was clinically established based on direct anatomical continuity and localized neuroimaging abnormalities. The patient successfully underwent emergency shunt externalization and removal, external ventricular drainage placement, and broad-spectrum antibiotic therapy covering enteric flora, resulting in complete resolution of neurological symptoms without long-term sequelae.

Conclusions: Transmural colonic migration of a VP shunt is a critical late-onset complication that can present solely as a retrograde CNS infection without abdominal manifestations or positive cultures. High clinical vigilance for occult intra-abdominal shunt displacement, correlated neuroimaging and abdominal imaging, prompt surgical intervention, and targeted antimicrobial therapy are essential to prevent severe infectious morbidity and optimize neurological recovery.

Keywords: Hydrocephalus • Infections • Ventriculoperitoneal Shunt

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Introduction

Ventriculoperitoneal (VP) shunt placement is a common neurosurgical procedure performed for various indications, most notably hydrocephalus. Complications of the procedure include obstruction, infection, shunt migration, pseudocyst formation, and over-draining. Obstruction is the most common reason for shunt malfunction [1]. Early shunt infection is typically associated with the initial surgical insertion, involving the introduction of skin flora, whereas late-onset infection is usually secondary to intra-abdominal complications or hematogenous inoculation [2]. Although rare, distal catheter perforation into the gastrointestinal tract—including the stomach and colon, with some cases exhibiting trans-anal extrusion [3,4]—represents a severe yet insidious late complication. Crucially, a subset of these patients presents completely without peritoneal signs or systemic symptoms and surprisingly displays negative cerebrospinal fluid (CSF) cultures. Such silent presentations often mask the underlying visceral perforation, creating a diagnostic challenge and potential delay in surgical management. Here, we present a distinct case of delayed VP shunt migration into the colon occurring 2.5 years after implantation in an adult who presented entirely without abdominal symptoms. We also review the literature to elucidate the diagnostic and management considerations for this occult complication.

Case Report

A 63-year-old man was brought to the emergency department (ED) due to multiple traumatic injuries. Head computed tomography (CT) scans showed subdural hematoma in the left fronto-temporo-parietal, parafalcine, and supratentorial regions (Figure 1A, 1B). Craniotomy for hematoma removal was performed, with the follow-up series of CT during hospitalization

revealing unresolved hydrocephalus; therefore, a VP shunt was inserted (Figure 1C). The patient was discharged after a 50-day hospital stay.

Two and a half years later, the patient returned with a fever and drowsiness. After readmission, laboratory evaluations detected leukocytosis and an elevated C-reactive protein level. A brain CT performed in the ED showed progression of hydrocephalus compared with previous imaging, in which significant peri-catheter brain edema in the right parietal and temporal lobes and compression of the right lateral ventricle (Figure 2A, 2B) were noted. Occlusion of the VP shunt and catheter-related infection were initially suspected, and abdominal CT was arranged to identify the source of infection. Malposition of the VP shunt was detected. The distal part of the shunt catheter had perforated and entered into the transverse colon at the splenic flexure, with the tip terminating at the proximal descending colon (Figure 3). There was no intra-abdominal fluid, abscess, or free air. Magnetic resonance imaging (MRI) of the brain showed hyperintensity in the right temporo-parietal lobes on T2-weighted and T2-fluid attenuated inversion recovery (FLAIR) images (Figure 2C), with increasing enhancement over peri-catheter brain parenchyma also being revealed (Figure 2D). Upon reviewing the abdominal plain film just taken after the initial placement of the VP shunt 2.5 years prior, it was observed that the distal end of the catheter was situated on the right side of the abdomen and exhibited no correlation with colonic gas (Figure 4), thereby ruling out iatrogenic perforation. Based on the constellation of clinical and radiological findings, a malposition of the VP shunt inside the colonic lumen with a clinically inferred retrograde CNS infection was suspected. Lumbar puncture was subsequently performed. The results of the cerebrospinal fluid analysis and culture were unremarkable, and blood cultures drawn in the ED remained negative. Despite the lack of microbiological

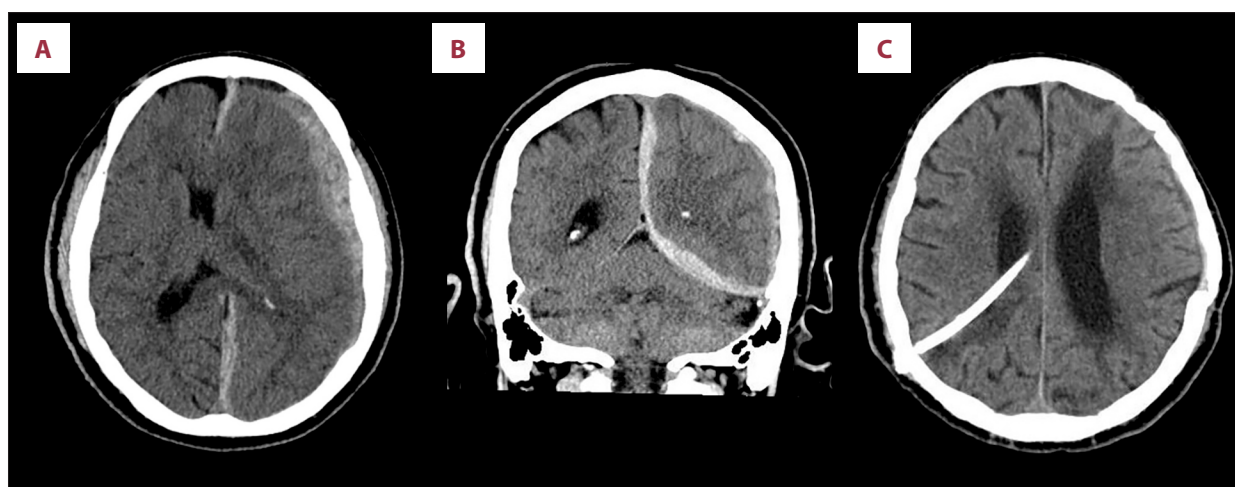


Figure 1. Computed tomography (CT) images of the first visit. Axial (A) and coronal (B) non-enhanced CT showed left fronto-temporo-parietal, parafalcine, and supratentorial subdural hematoma. (C) After ventriculoperitoneal shunt placement.

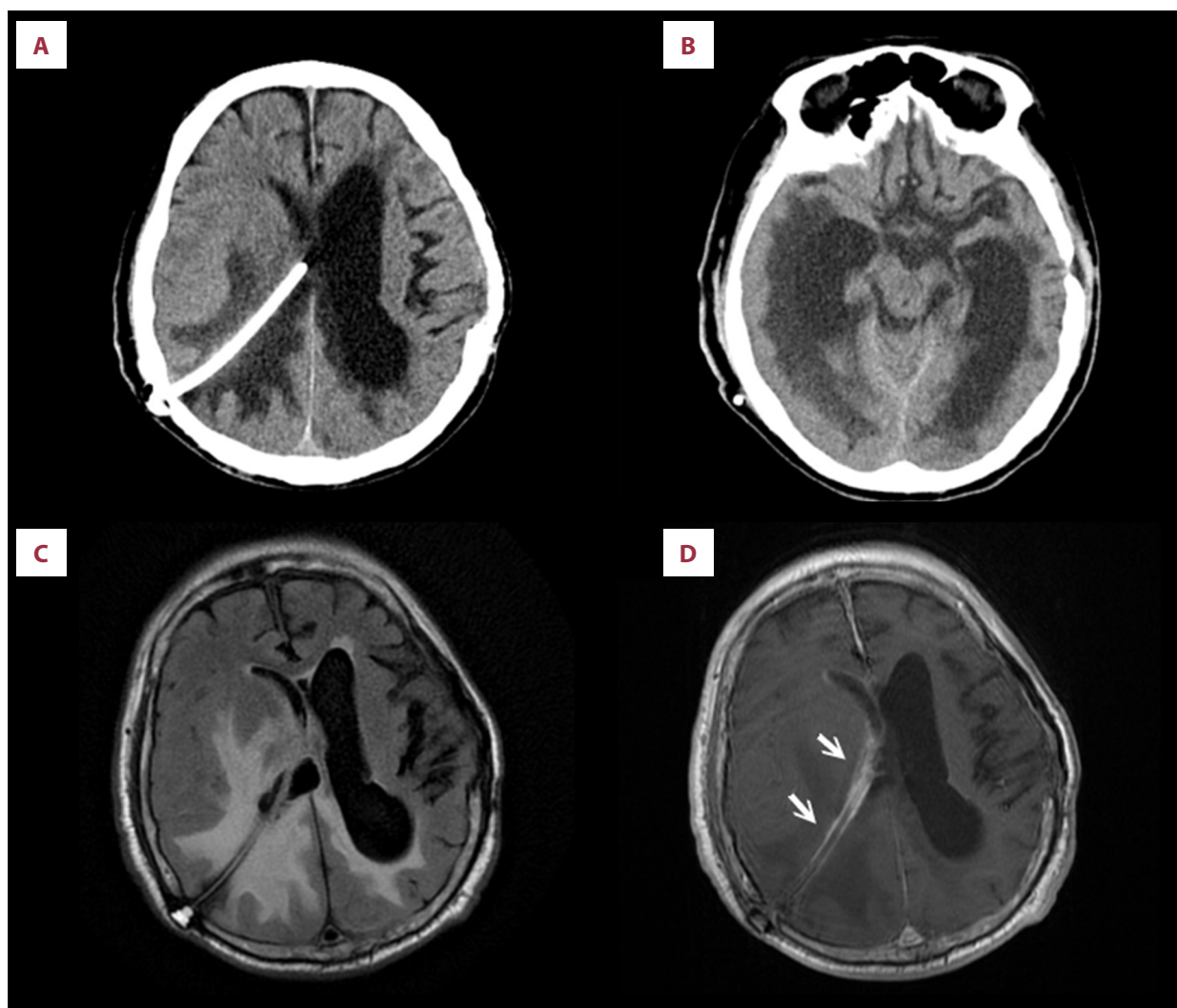


Figure 2. (A, B) Axial non-enhanced computed tomography (CT) shows hydrocephalus progression compared with prior CT (Figure 1C). Significant peri-catheter brain parenchymal edema in the right parietal and temporal lobes and compressed right lateral ventricle were noted. (C) Axial T2 FLAIR magnetic resonance images show hyperintensity in the right parietal and temporal lobes. (D) The lesion shows mild enhancement over peri-catheter parenchyma (arrows).

confirmation, the patient underwent immediate shunt removal and empirical antibiotic treatment covering potential retrograde translocation of intestinal flora.

Discussion

While shunt dysfunction typically occurs early, late-onset complications pose a distinct diagnostic challenge. Although obstruction and infection are the primary causes of shunt failure [1,2], spontaneous transmural bowel perforation and distal migration are exceptionally rare (0.1%-0.7%) [1]. This complication predominantly affects pediatric patients due to thinner bowel walls, making adult cases particularly unusual, with the colon being the most common site [5]. Crucially, in silent bowel migration,

the absence of classical peritonitis masks the intra-abdominal etiology, often presenting with insidious neurological symptoms such as lethargy or low-grade fever, which can easily lead clinicians to misclassify the event as routine mechanical malfunction or primary intracranial pathology. However, beyond shunt migration, dysfunction can also manifest through various other delayed mechanisms that carry severe clinical and medicolegal implications. As highlighted by Modgil et al [6], sudden clinical deterioration or fatal outcomes can arise from insidious failure across any component of the shunt system. This underscores the necessity of long-term clinical surveillance, comprehensive clinicopathological correlation, and systematic evaluation of the entire VP shunt assembly, from the ventricular catheter to the distal peritoneal tubing, when evaluating patients presenting with subtle or acute neurological symptoms.



Figure 3. Axial (A), coronal (B), and sagittal (C) non-enhanced abdominal computed tomography showing malposition of the ventriculoperitoneal shunt into the colonic loop (arrows), from transverse colon, splenic flexure to proximal descending colon.

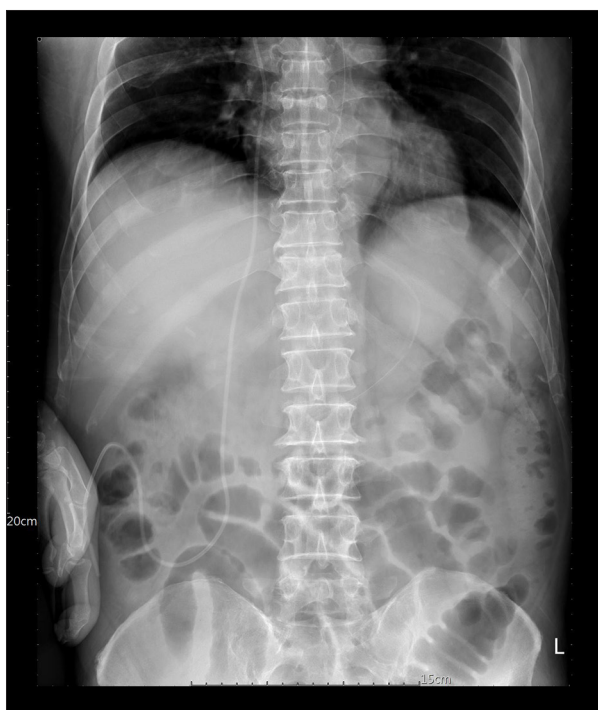


Figure 4. The abdominal plain film, taken just after the initial placement of the ventriculoperitoneal shunt 2.5 years ago, revealed that the distal end of the catheter was located on the right side of the abdomen and showed no correlation with colonic gas.

The mechanical sequence leading to transmural colonic migration is believed to be a localized, step-wise process [5]. Initially, continuous mechanical friction from the free distal catheter tip against the intestinal serosa triggers a localized foreign-body response. This chronic low-grade inflammation induces localized fibrous adhesions, anchoring the catheter to the colonic wall. Subsequently, continuous mechanical pulsation combined with progressive tissue fibrosis facilitates gradual transmural erosion. Over time, the catheter penetrates the muscular and mucosal layers into the bowel lumen, completing intraluminal migration [7]. Because this erosive process is

slow and localized, the surrounding fibrotic seal prevents the leakage of intestinal contents, explaining why patients rarely exhibit overt pneumoperitoneum or generalized peritonitis. Crucially, a thorough review of the initial postoperative imaging confirmed correct placement, ruling out iatrogenic causes and confirming spontaneous, delayed migration.

Our patient presented with fever, drowsiness, poor appetite, leukocytosis, and elevated C-reactive protein, but lacked classical physical signs of peritonitis or meningitis. Furthermore, CSF analysis, CSF culture, and 2 sets of blood cultures were entirely normal and negative. In the literature, direct microbiological confirmation can sometimes be elusive in shunt-related infections. Previous studies have also revealed that less than 25% of patients had signs of peritonitis [1]. While fever and more than 10% neutrophils in the CSF have high specificity (99%) and positive predictive value (93%) for predicting shunt infection [1], normal CSF cell counts and negative culture results do not entirely exclude a clinical diagnosis of infection, especially when masked by previous antibiotics or when the inflammatory response is localized. Similar to reported cases of colonic perforation or anal extrusion presenting with sterile CSF cultures [3,4], the absence of active bacterial growth in CSF does not rule out ascending microbial translocation or localized CNS inflammation. Despite these negative culture findings, a retrograde CNS infection was clinically established based on persistent fever, elevated inflammatory markers, progressive hydrocephalus, peri-catheter edema and enhancement on neuroimaging, and confirmed colonic catheter migration. While aseptic chemical meningitis or localized sterile inflammation were considered alternative differential diagnoses, the rapid clinical and biochemical resolution following shunt removal and targeted antimicrobial therapy strongly corroborated an infectious etiology over a purely mechanical reaction.

During hospitalization, the patient was treated in the intensive care unit and immediately started on empirical intravenous antimicrobial therapy with CNS-therapeutic doses of vancomycin (1 g IV every 12 hours) and meropenem (2 g IV

every 8 hours) for a total duration of 16 days. On hospitalization day 2, the patient underwent prompt surgical extraction of the infected and migrated shunt system, primary repair of the colonic perforation, and placement of a temporary external ventricular drain. On day 11, a new contralateral (left) VP shunt was successfully implanted. The patient was discharged at his baseline neurological status and has remained free of new neurological deficits during more than 1 year of regular outpatient follow-up.

This case offers several distinct educational lessons that refine our clinical recognition of long-term shunt complications beyond their mere rarity. First, it demonstrates that delayed colonic shunt migration in adults can progress silently and present primarily as routine shunt malfunction or a clinically significant, retrograde intracranial inflammatory process, even when cultures remain negative and typical abdominal signs or peritonitis are completely absent. Second, it highlights a crucial, imaging-led diagnostic workflow: utilizing abdominal CT to identify the exact site of migration, combined with a meticulous retrospective comparison against early postoperative imaging to rule out iatrogenic injury and confirm a spontaneous, transmural erosion mechanism. Ultimately, early detection and proper management remain the keys to preventing morbidity, and clinicians must maintain a high index of suspicion for such occult complications when a patient's neurological status unexpectedly deteriorates years after the procedure.

This study has several limitations. First, as a single-case report, the findings and clinical trajectory may have limited generalizability to all cases of distal shunt migration. Second, direct microbiological confirmation of the retrograde CNS infection was absent, relying instead on a clinically inferred diagnosis based on radiological and clinical parameters. Consequently, causal inferences regarding the exact pathway and pathogen involved should be interpreted with caution.

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Conclusions

Transmural colonic migration of a VP shunt represents a critical late-onset complication that can progress silently without overt abdominal symptoms or positive microbiological cultures. Clinicians should remain vigilant for occult intra-abdominal shunt displacement in patients presenting with late, unexplained neurological deterioration or retrograde CNS inflammation. Prompt diagnostic workup, including abdominal and neuroimaging, coupled with early surgical intervention and targeted antimicrobial therapy, remains essential to prevent severe infectious morbidity and optimize neurological recovery.

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Institution Where Work Was Done

Kaohsiung Medical University Hospital, Kaohsiung, Taiwan.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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