Pulmonary Infarction due to Embolized Ventriculoperitoneal Shunt Catheter in a Pediatric Patient: A Case Report

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ABSTRACT

Cerebrospinal fluid diversion serves as the primary therapeutic approach for managing hydrocephalus. Among the surgical interventions available, ventriculoperitoneal shunt placement is the preferred method. Occasionally, these procedures can result in complications, such as catheter migration to unexpected locations. This case illustrates a unique complication involving the migration of a ventriculoperitoneal shunt catheter into the pulmonary artery resulting in pulmonary embolism and infarction in a young adult patient, necessitating immediate catheter retrieval. Furthermore, the possible mechanisms of migration are discussed and a narrative review of the management of this rare complication is presented.

CASE REPORT

INTRODUCTION

In the United States, more than 40,000 cerebrospinal fluid shunts are implanted annually, primarily to treat hydrocephalus [1]. However, these procedures carry a risk of potential complications, some of which may require shunt removal or revision [2].

Complications associated with ventriculoperitoneal (VP) shunts include catheter blockage, infections, system disconnection, hardware malfunction, and pseudocyst formation [2,3]. In rare instances, VP shunt catheters can migrate to unexpected locations, such as the intestines, bladder, abdominal wall, thoracic cavity, or cardiovascular system [4].

Among these uncommon events, the migration of a distal VP shunt catheter into the pulmonary artery is exceedingly rare occurrence. This case illustrates a unique situation involving shunt migration into the pulmonary artery complicated by pulmonary emboli and infarction in a pediatric patient is presented and the subsequent management of this unusual situation is discussed.

CASE PRESENTATION

A 19-year-old male was transferred to the children's hospital from an outside facility for a higher level of care with a diagnosis of pulmonary embolism (PE) and infarction following an episode of severe chest pain and syncope. The patient has a history of myelomeningocele with neurogenic bladder and bowel s/p urostomy, ileostomy, Chiari malformation, and congenital hydrocephalus who had undergone multiple prior VP shunt placements and revisions bilaterally, with the most recent left sided catheter being placed five months prior to presentation for worsening of headache with non-contrast head CT (Figure 1A) showing asymmetric ventricular size with a slit like right lateral ventricle and asymmetrically prominent left lateral ventricle. Of note, extensive intrabdominal adhesions was noted during the VP shunt placement likely due to prior intrabdominal procedures including VP shunt placements and revisions.

Imaging obtained at the outside hospital was reviewed by the pediatric radiology department upon arrival to the children's hospital. The chest computed tomography angiography (CTA) at the outside hospital was notable for a curvilinear hyperdensity in the pulmonary trunk and right pulmonary artery with associated thrombus and a right middle lobe pulmonary infarction (Figure 2). In addition, on the scout topogram, there was clear absence of the smaller caliber left VP shunt catheter when compared to a prior shunt series following the most recent VP shunt revision (Figure 3). These findings were concerning for a possible foreign body, such as a catheter or wire, that had migrated to the right pulmonary artery resulting in thrombus formation and occlusion of the right middle lobe artery causing pulmonary infarction. A chest radiograph performed at the outside hospital also demonstrated a radiopaque wire or coiled tube projecting over the heart (Figure 4).

Additional evaluation was performed with an echocardiogram which demonstrated a linear echogenicity in the right ventricle extending into the right pulmonary artery, compatible with the previous imaging finding that a catheter curled within the main pulmonary artery extending into the right main pulmonary artery. The patient's ejection fraction and cardiac function appeared normal. Non-contrast head CT during admission showed stable position of the bilateral VP shunts with grossly stable ventricular size (Figure 5).

The patient was subsequently taken for foreign body retrieval by a pediatric interventional cardiology service. A VP shunt catheter was seen coiled in the right main and segmental pulmonary artery of the right middle lobe and the tip retrieved via the right femoral vein. The exteriorized portion of the catheter demonstrated active drainage of CSF and was confirmed by neurosurgery to be the VP shunt. The patient was then transferred to the operating room where neurosurgery and general surgery proceeded to perform an explantation of the left VP shunt catheter from the right femoral vein and revision of the left-sided VP shunt to a ventriculoatrial (VA) shunt without complication. Shunt location in the right atrium was confirmed with injection of Omnipaque through the shunt reservoir while compressing it proximally.

Subsequent follow-up chest CTA was obtained and showed no residual foreign body in the heart and the pulmonary artery with stable position of the right VP shunt and new left VA shunt (Figure 6).

DISCUSSION

The migration of a ventriculoperitoneal (VP) shunt to ectopic sites is a relatively infrequent occurrence in the context of shunt complications. Migration to the gastrointestinal system is the most commonly observed type of migration in pediatric patients. In contrast, there is a greater propensity for shunt migration to locations like the heart, breast, and abdominal wall in adults [5]. The time interval between the initial placement of a VP shunt and the diagnosis of migration can vary significantly, spanning from 7 days to as long as 4 years [6]. This wide range in diagnosis time makes it challenging to predict and anticipate this exceptional complication.

Migration of a catheter into the pulmonary arteries is an exceedingly rare event, and affected patients may or may not exhibit symptoms [7]. Individuals experiencing this rare type of migration, along with concurrent arterial thrombosis, may present with symptoms indicative of right heart strain in an emergency setting. These symptoms may include dyspnea, chest pain, and coughing, often accompanied by signs of respiratory failure. Arterial thrombosis can also give rise to PE and respiratory distress, necessitating emergent retrieval [8]. Additionally, if the catheter becomes tangled or knotted, it can lead to shunt dysfunction, and patients may develop signs and symptoms of increased intracranial pressure, such as headaches, altered mental status, focal neurological deficits, and vomiting.

Nevertheless, shunt migration into the pulmonary arteries remains a rarely reported phenomenon in the medical literature, and its underlying mechanism remains unclear. It has been suggested that iatrogenic damage to the internal or external jugular vein during the creation of the subcutaneous tract could be a contributing risk factor. Profuse bleeding at the neck may occur, although this unexpected damage can go unnoticed. Another predisposing factor may be the proximity of the shunt to veins [6,9]. Repetitive movement of the neck can potentially damage adjacent veins. Once the catheter has breached the internal or external jugular vein, a combination of negative intrathoracic pressure, positive intraabdominal pressure, and venous flow collectively contribute to the migration toward the heart [9]. Pulsatile cardiac impulses may further drive the catheter into the pulmonary arteries. If the catheter migrates far, it can become lodged and subsequently knotted within the pulmonary arteries, resulting in pulmonary emboli and infarction, typically presenting with symptoms of right heart strain. In addition, obesity, particularly in individuals with a body mass index exceeding 30 kg/m^2, and a history of multiple previous shunt procedures have been identified as independent risk factors for shunt migration [8]. In this case, it is possible the migration may have resulted from iatrogenic perforation of the external jugular vein, considering his relatively recent VP shunt placement.

In these rare cases of shunt catheter migration into cardiac or intravascular locations, the issue should be addressed expeditiously due to the potential for additional complications, such as thromboembolism, sepsis, and cardiac arrhythmias [10]. Diagnosis can be established through a radiographic shunt series to assess the position of the proximal and distal catheters. However, a chest CTA is recommended to precisely confirm the catheter's exact location when it is malpositioned. In addition, it is advisable to perform echocardiography to evaluate heart function and brain imaging to assess the possibility of associated shunt malfunction prior to any intervention [8].

A variety of procedures, ranging from blind removal to open-heart surgery, have been employed to address intravascular shunt migration [6,11]. In cases where the migrated catheter is impinged on the valves or firmly lodged within the pulmonary arteries due to knot formation or multiple loops [11,12], vascular intervention may be attempted. After the removal of the migrated catheter, the distal catheter can be repositioned into the peritoneum or converted into a VA shunt [8]. The insertion of a new distal catheter can be performed immediately or electively after an appropriate interval. Utilizing the contralateral side for revision may help mitigate the risk of further damage to already weakened vessels. Regardless, real-time visualization using fluoroscopy and intraoperative cardiac monitoring are recommended during the removal of the migrated catheter, especially if any resistance is encountered [6]. In our case, preoperative echocardiography and brain CT findings were unremarkable. The catheter was externalized via the femoral vein under fluoroscopic guidance by interventional

cardiology, without encountering any resistance. Subsequently, neurosurgery disconnected the distal catheter from the valve, removed the distal shunt valve entirely from the right femoral vein without difficulty and introduced a new distal catheter. Then, decision was made to convert it to VA shunt given extensive intrabdominal adhesions.

Here, we present a unique case highlighting distinctive features of VP shunt migration that have not been previously documented in the literature. Our patient, a 19-year-old, stands out as notably younger than the typical demographic experiencing VP shunt migration to the pulmonary artery and heart. The occurrence of pulmonary emboli and infarction due to migrated VP shunt is an exceedingly rare presentation, particularly within this age group. It is noteworthy that despite multiple shunt revisions on the right, it was the left VP shunt that migrated. However, it is possible that the presence of more than one peritoneal catheter and intraabdominal adhesions contributed for the migration. Furthermore, the potential association between a smaller caliber VP catheter and an increased risk of migration represents a novel aspect not observed in the cases reported in existing literature.

CONCLUSION

Reported here is an unusual case of shunt migration into the pulmonary artery possibly through the external jugular vein and its subsequent management. VP shunt catheter migration into the pulmonary artery can present with pulmonary emboli resulting in pulmonary infarct in pediatric patients. This report suggests that patient history and use of any available imaging is important to help reach into a diagnosis. Migrated catheters can be detected on different imaging modalities, like chest radiograph, though CTA is the preferred imaging modality to evaluate the extent of migration and associated pulmonary emboli and pulmonary infarct. Additionally, non-invasive endovascular retrieval is an option for migrated catheter retrieval. Moreover, risk of migration might be higher in smaller caliber VP shunt catheters compared to larger caliber catheters.

Therefore, radiologists should be aware of both the common mechanical and infectious complications of VP shunt placement as well as rare complications, such as cardiac and intravascular shunt migration that is addressed in this report. Awareness of this unusual yet hazardous complication is important for prompt diagnosis and management in order to prevent significant morbidity.

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FIGURES

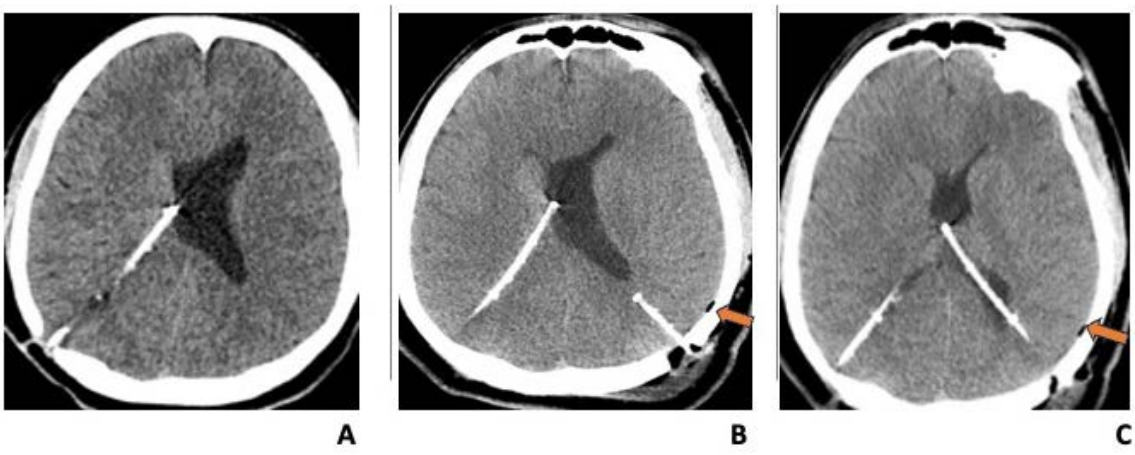

Figure 1: Non contrast Head CT axial image **(A)** shows slit like right lateral ventricle with a right parietal approach ventriculostomy catheter terminating along the right lateral ventricle and asymmetrically prominent left lateral ventricle. Non contrast Head CT post left side shunt placement axial image **(B & C)** shows new left posterior parietal approach ventriculostomy catheter crossing the left lateral ventricle, tip at the vicinity of the septum pellucidum with slight interval decrease in size of the left lateral ventricle. Stable right parietal approach ventriculostomy catheter. Pneumocephalus is present(arrow).

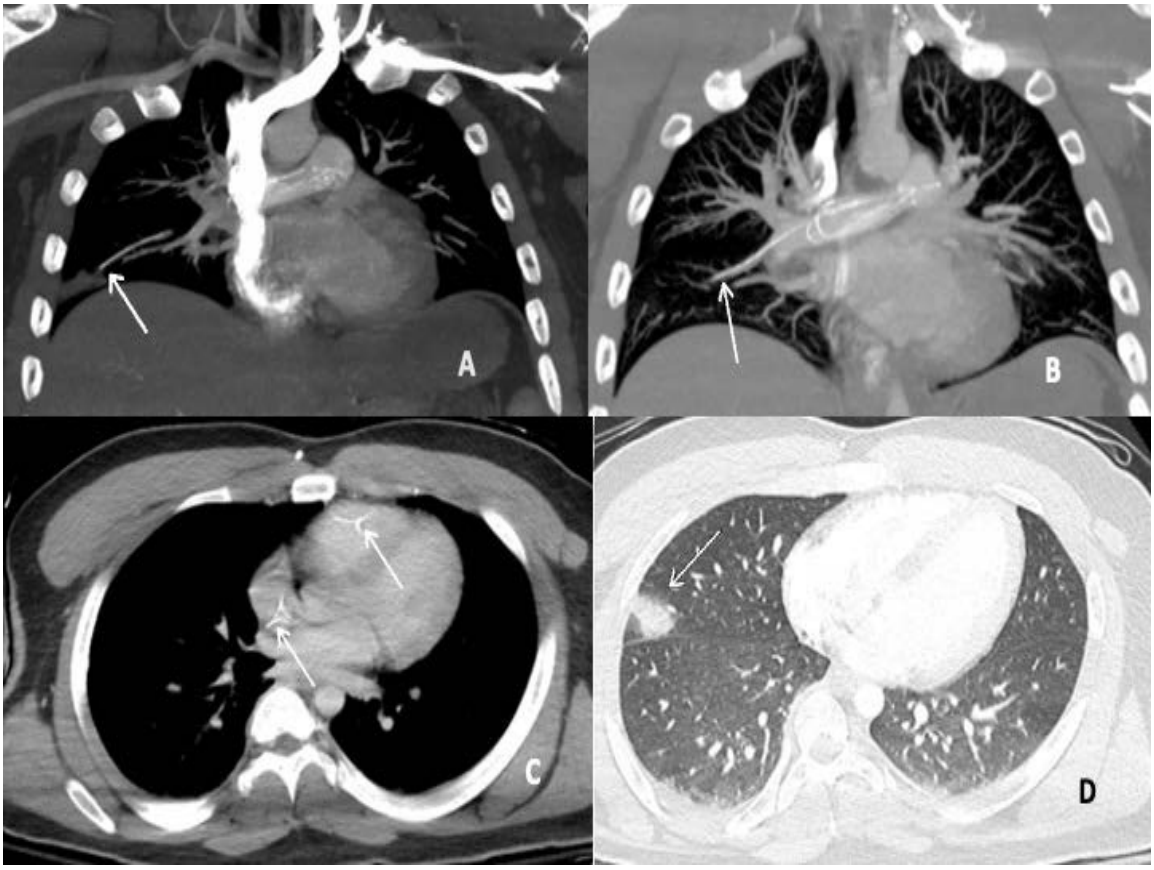

Figure 2: Initial outside hospital chest CTA demonstrating a foreign body within the right atrium, right ventricle, and pulmonary trunk, with associated thrombus resulting in complete pulmonary artery occlusion and pulmonary infarction. Coronal images windowed to the soft tissues demonstrate a fine curvilinear hyperdensity in the pulmonary trunk **(A&B)** extending to a segmental right middle lobe pulmonary artery with a distal pulmonary infarction. Axial image windowed to the soft tissues demonstrate a fine curvilinear hyperdensity in the right atrium and ventricle **(C).** Axial images windowed to the lungs demonstrate a right middle lobe pulmonary infarction distal to the occluded pulmonary artery **(D).**

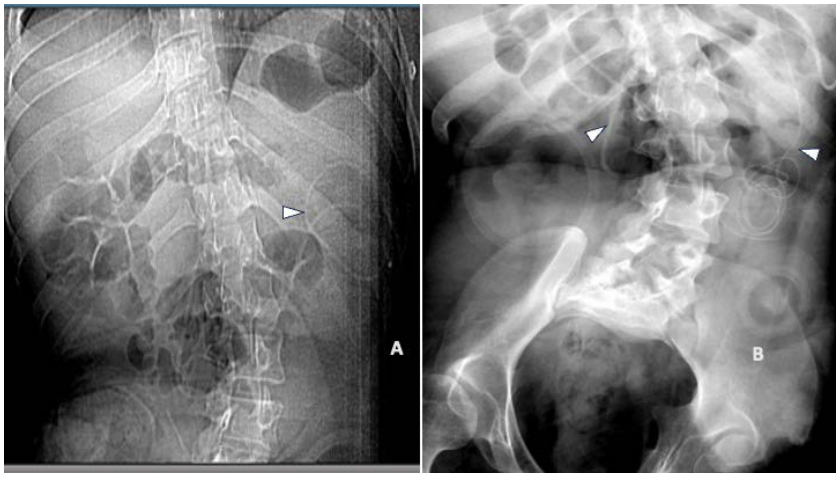

Figure 3: Comparison of chest CTA scout topogram **(A)** with prior ventriculoperitoneal shunt series abdominal radiograph **(B)** demonstrating notable absence of the recently placed small caliber distal left VP shunt catheter.

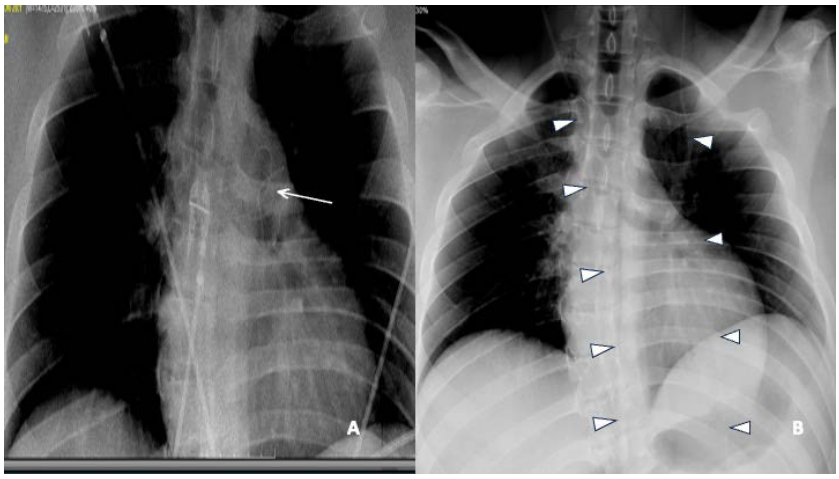

Figure 4: Comparison of chest radiograph **(A)** with prior ventriculoperitoneal shunt series chest radiograph **(B)** demonstrating a curvilinear density coiled and projecting over the pulmonary artery shadow.

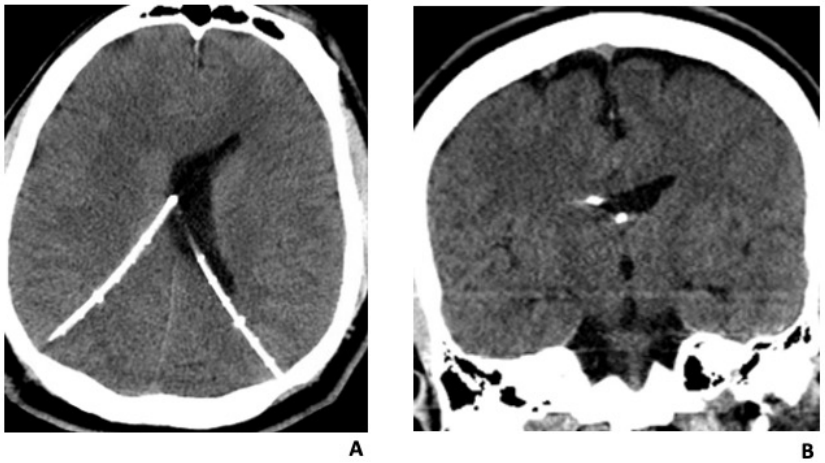

Figure 5: Non-contrast Head CT on admission axial (A) and Coronal (B) demonstrated grossly stable ventricular sizes with stable right parietal approach ventriculostomy catheter terminating along the right lateral ventricle and left posterior parietal approach ventriculostomy catheter crossing the left lateral ventricle and terminates at the vicinity of the septum pellucidum.

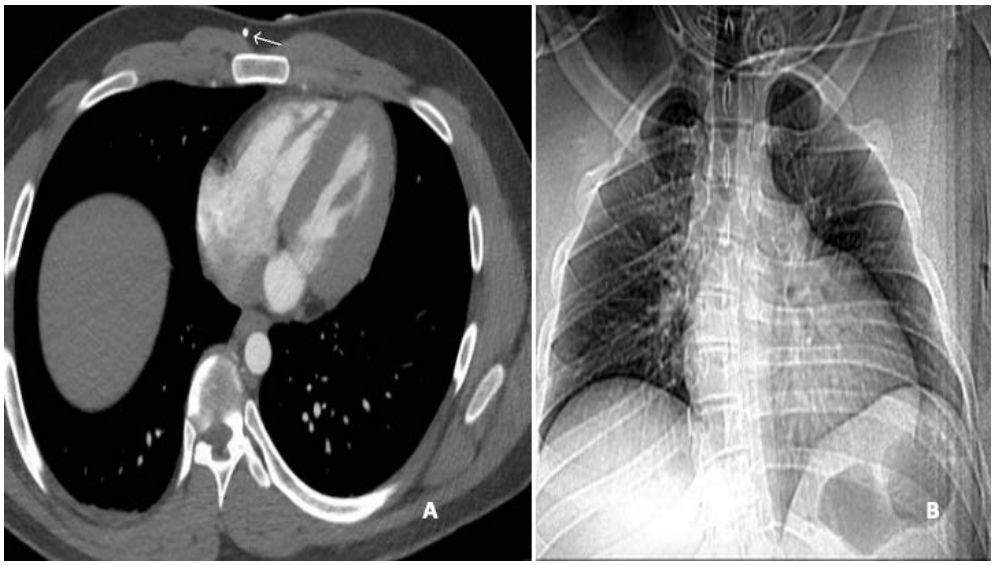

Figure 6: Follow-up images demonstrating appropriate positioning of the right VP shunt and new left VA shunt without complications. Postoperative chest CTA **(A)** and chest radiograph **(B)** with no evidence of residual catheter within the heart or main pulmonary arteries.

KEYWORDS

Pulmonary Infarct; Pulmonary Embolus; Ventriculoperitoneal Shunt; Hydrocephalus; Migration

ABBREVIATIONS

CT = COMPUTED TOMOGRAPHY
CTA = COMPUTED TOMOGRAPHY ANGIOGRAPHY
PE = PULMONARY EMBOLISM
VA = VENTRICULOATRIAL
VP = VENTRICULOPERITONEAL

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