v. 5, n. 2, 2025

International Journal of Health Science

# FRACTURE OF A VENTRICULOPERITONEAL CATHETER CAUSING INTRACRANIAL HYPERTENSION

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*Acceptance date: 07/01/2025*

**Abstract: Introduction:** Hydrocephalus is a condition characterized by excessive cerebrospinal fluid (CSF) accumulation in the ventricles, leading to increased intracranial pressure (ICP). The standard treatment involves the placement of ventriculoperitoneal (VP) shunts, and immediate evaluation for potential malfunction is essential when signs of elevated ICP emerge. **Case Report:** A 43-year-old female patient, with a history of VP shunt placement since childhood due to obstructive hydrocephalus, presented with headache, blurred vision, vomiting, and papilledema. The chest X-ray revealed a fracture of the VP catheter between the second and third left rib arches. The patient underwent a new VP shunt placement on the right side, with immediate improvement in ICP symptoms and reduction of thoracic bulging. **Discussion:** Approximately 40% of shunts fail within the first year after placement, with ventricular catheter obstruction being the most common cause. Catheter fractures occur in 15% of cases, often due to silicone biodegradation, leading to calcification and fragility. Such failures require immediate diagnosis and surgical intervention to prevent further complications. **Conclusion:** VP shunt fracture is a significant mechanical dysfunction that may be difficult to diagnose but requires immediate investigation and correction, even during routine follow-ups.

**Keywords:** Intracranial Hypertension; Ventriculoperitoneal Shunt; Catheter Fracture

## INTRODUCTION

Hydrocephalus is a disorder characterized by the excessive accumulation of cerebrospinal fluid (CSF) in the brain's ventricles and/or subarachnoid spaces, leading to ventricular dilation and increased intracranial pressure (ICP) (1). Ventriculoperitoneal (VP) shunts have long been the standard treatment for hydrocephalus (2). Immediate evaluation for potential shunt malfunction should be performed in patients who develop new or aggravated signs of increased ICP, such as headache, vomiting, lethargy, papilledema, and irritability (3, 4).

## CASE REPORT

A 43-year-old female patient, with a history of VP shunt placement since childhood due to obstructive hydrocephalus, presented with headache, blurred vision, projectile vomiting, and papilledema. Additionally, she had a bulge in the left thoracic region, with fluctuating points on palpation and localized pain. A cranial CT scan revealed enlargement of the lateral ventricles and the third ventricle, while the fourth ventricle was of normal size. A chest X-ray showed a calcified and fractured VP catheter between the second and third left rib arches (Figure 1).

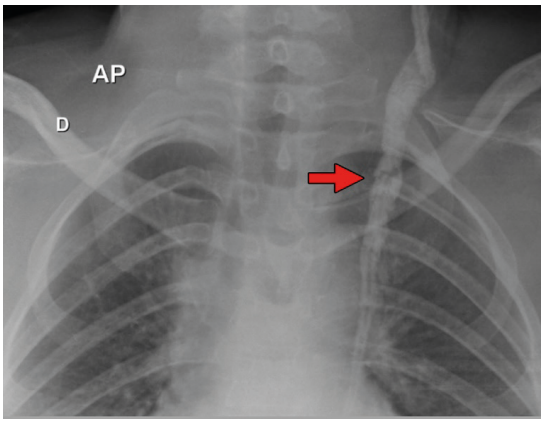


**Figure 1:** Anteroposterior chest X-ray showing a ventriculoperitoneal shunt catheter with a path in the left hemithorax, calcified, with a fracture point (red arow) at the level of the second/third rib arches.

Urgent surgical intervention was chosen, and a new VP shunt (medium-pressure valve) was placed on the right side, with an opening pressure of 50 $cmH_2O$ for CSF. The patient showed immediate improvement in intracranial hypertension symptoms in the immediate postoperative period, as well as a gradual reduction of the bulging in the left thoracic region, the site of the catheter fracture.

## DISCUSSION

Approximately 40% of standard shunts malfunction within the first year after placement, and 5% per year experience failure in the subsequent years (5). Evaluation of shunt malfunction typically includes a detailed neurological exam, neuroimaging (CT scan or MRI), and radiographs of the catheter path (6, 7). Malfunction can result from infection or mechanical failure (8).

Mechanical failure of the shunt is more common during the first year after placement, with ventricular catheter obstruction being the most frequent cause (9). Fractured tubing accounts for about 15% of shunt failure cases (10). Mechanical failure requires immediate recognition and surgical intervention.

Studies report the occurrence of biodegradation of the catheter when implanted over long periods. Silicone degradation results in the loss of its dynamic properties within the first year of implantation, leading to calcification, rigidity, fragility, adhesion to subcutaneous tissue, loss of function, or fracture of the catheter wall (11, 12).

The presence of calcium accumulation sites, linked to proteins or protein denaturation, may become a nidus for subsequent catheter calcification (13). The brain parenchyma and the peritoneal cavity do not degrade the silicone catheter; localized inflammatory reactions have been associated with T-cell granuloma formation, indicating both cell-mediated immunity and humoral immune responses (14). The release of silicone particles causes a granulomatous inflammatory response of the antigen-antibody type, with macrophage-mediated attacks (15).

Mechanical failure of the shunt, such as disconnection and fracture, is a significant cause of malfunction and requires immediate surgical intervention (16-17).

## CONCLUSION

Disconnection and fracture are two significant mechanical dysfunctions of the ventriculoperitoneal shunt that should be thoroughly investigated and understood, even during routine follow-up. A disconnected or fractured shunt may still be functioning, and it is unsafe to assume the shunt is no longer necessary without proper investigation

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