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Case presentation

Scalp necrosis overlying a ventriculoperitoneal shunt: a case report and literature review

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Abstract

Background: The use of ventriculoperitoneal (VP) shunts has become ubiquitous in neurosurgery for the treatment of hydrocephalus. VP shunts work by creating a conduit for cerebrospinal fluid (CSF) to flow from the cerebral ventricles to the peritoneum and thus relieving pressure. Although typically safe, VP shunt complications are extremely common, occurring in up to 29% of adult cases and approximately half of pediatric cases. These complications may require patients to undergo several revisions throughout their lifetime.

Purpose: We describe a man who developed scalp necrosis overlying his VP shunt. We also summarize the potential complications of VP shunts. We discuss the presentation and pathogenesis of scalp necrosis in these patients.

Materials and methods: A PubMed search of the following terms was performed and relevant citations were assessed: ventriculoperitoneal shunt, VP shunt, shunt complications, scalp necrosis, skin necrosis, ventriculoperitoneal shunt induced scalp necrosis, ventriculoperitoneal shunt induced skin necrosis.

Results: A 73-year-old man developed VP shunt-induced scalp necrosis leading to loss of skin overlying the shunt valve. The patient was emergently referred to neurosurgery and admitted for revision of the shunt due to the significant risk for infection. The VP shunt was replaced with an external drain and a skin flap was used to repair the defect.

Conclusion: VP shunts are devices that are frequently used for reducing intracranial pressure associated with hydrocephalus. The insertion of the shunt beneath the scalp sets the stage for potential pressure-induced scalp necrosis. The early recognition and diagnosis of VP shunt-induced scalp necrosis is essential to prevent infections and future complications.

Abbreviations: VP – ventriculoperitoneal; CSF – cerebrospinal fluid

Keywords: Ventriculoperitoneal shunt, VP shunt, scalp necrosis, scalp, skin necrosis, pressure

Introduction

Ventriculoperitoneal (VP) shunts are devices commonly used in the management of hydrocephalus. VP shunts relieve increased intracranial pressure caused by excessive cerebrospinal fluid (CSF) by creating a conduit for CSF to flow from the cerebral ventricles to the peritoneum [1,2]. Indeed, VP shunts have revolutionized the management of hydrocephalus; however, these devices come with their own set of risks and complications (Table 1) [3-5].

We present a 73-year-old man with a history of hydrocephalus secondary to a subependymoma who developed scalp necrosis overlying his VP shunt, a known risk factor for central nervous system infection. We also summarize the potential complications of VP shunts. In addition, we discuss the clinical presentation and pathogenesis of VP shunt-induced scalp necrosis.

Table 1. Complications associated with ventriculoperitoneal shunts [1-3]

Common (a):

Infection

Proteus mirabilis

Staphylococcus aureus

Staphylococcus epidermidis

Mechanical failure

Breakage

Disconnection

Malpositioning

Migration

Obstruction

Functional complication

Overdrainage

Underdrainage

Uncommon:

Cerebrospinal fluid ascites

Intraventricular hemorrhage

Scalp necrosis

Status epilepticus

Subdural hematoma

(a) Complications listed in order of frequency

Case synopsis

A 73-year-old man presented to the dermatology clinic for a total body skin exam; he had a history of multiple actinic keratoses and non-melanoma skin cancers. His past medical history was also significant for a subependymoma with secondary hydrocephalus diagnosed and treated in 2003; the tumor recurred in 2013 and was resected. Subsequently, the patient was treated symptomatically with a VP shunt.

Physical exam showed multiple keratotic plaques on the face, chest, back and arms consistent with actinic keratoses that were treated with cryotherapy using liquid nitrogen. In addition, there was an area of erythema and crusting on the scalp overlying the VP shunt.

Warm soaks followed by application of mupirocin 2% ointment, three times a day, was initiated for the crusted area overlying the shunt, and the patient was advised to follow-up with his neurosurgeon.

Follow-up examination, two months later, again showed numerous keratotic plaques on the face, chest, backs and arms consistent with actinic keratosis. The erythema and crusting overlying the VP shunt had progressed—despite therapy—and there was a large area of full-thickness skin necrosis; the shunt valve was visible without overlying scalp skin (Figure 1). The patient was immediately sent to his neurosurgeon for evaluation and was hospitalized. The VP shunt was surgically removed in the operating room and replaced with an external drain; a skin flap was used to repair the cutaneous defect. He was also treated for possible infection with intravenously administered cefazolin. His post-operative course was unremarkable.



Figure 1. Distant (a) and closer (b) views of an area of erythematous crust on the mid-scalp overlying the ventriculoperitoneal shunt; adjacent to the crust, there is complete scalp necrosis and the shunt can be seen.

Six months later, a well-healed depressed scar was observed on his scalp at the site that had previously undergone necrosis (Figure 2).



Figure 2. Distant (a) and closer (b) views show a depressed scar following repair of the scalp defect using a skin flap.

Discussion

Hydrocephalus is a disorder characterized by increased intracranial pressure due to an excessive accumulation of CSF in the cerebral ventricles and subarachnoid space [6]. Several etiologies can contribute to the development of hydrocephalus (Table 2) [7]. Hydrocephalus typically presents clinically with headache, nausea, and vomiting that are worse in the morning [8]. Untreated hydrocephalus is associated with significant morbidity and mortality. However, with the introduction of surgical management and the use of shunts, the overall mortality rate is now less than 5% over a 10-year follow-up [2].

Table 2. Causes of hydrocephalus [4]

Excessive production
Choroid plexus papilloma
Impaired absorption
Infection
Obstruction
Aqueductal stenosis
Chiari malformation

Treatment options for hydrocephalus may include diuretics, corticosteroids, shunts, and third ventriculostomy [9]. VP shunts continue to be the mainstay of treatment for hydrocephalus in adult and pediatric patients. In the United States, approximately 36,000 shunt procedures are performed each year with healthcare related costs for pediatric hydrocephalus approaching \$2 billion dollars annually [10,11].

VP shunts work by creating a conduit for CSF to flow from the cerebral ventricles to the peritoneum. This effectively decreases the volume of CSF in the cerebral ventricles and subarachnoid space—thus relieving the increased pressure [1]. Despite their utility, VP shunt complications are extremely common, occurring in up to 29% of adult cases and approximately half of pediatric cases [3,12].

Common complications, in order of frequency, associated with VP shunts include infection, mechanical failure, and functional defects (Table 1) [3-5]. Infection of a VP shunt is typically caused by *Staphylococcus epidermidis* or *Staphylococcus aureus*; these are biofilm-forming bacteria that can frequently be found attached to the surface of implanted devices such as VP shunts [13]. Factors frequently associated with VP shunt infection include younger age, poor preoperative skin condition, type of operation, and the presence of wound dehiscence or scalp necrosis [14]. Mechanical failure of a VP shunt can be caused by breakage, disconnection, malpositioning, migration, or obstruction of the catheter. Obstruction is the most common cause of mechanical shunt failure occurring in over half of cases involving a mechanical failure [3]. Functional defects of VP shunts refer to over-drainage or under-drainage of CSF fluid leading to either increased or decreased intracranial pressure [3].

Scalp necrosis is an uncommon complication of VP shunt placement in adults [14,15]. However, skin necrosis is actually a common complication associated with VP shunts in neonates due to the inherent fragility of the skin in combination with increased vascular stasis due to localized pressure from decreased head movement in infants [16]. In contrast, scalp necrosis is not as frequently reported in older children and adults; yet, it has been linked to an increased risk for infection, morbidity, and mortality [14]. Therefore, early identification and treatment of scalp necrosis overlying a VP shunt are essential.

The pathogenesis of scalp necrosis overlying a VP shunt likely arises from skin pressure necrosis due to the underlying device. Indeed, skin pressure necrosis has also been reported with other implanted devices such as pacemakers and first-generation tissue expanders [18,19]. In a VP shunt, a catheter is passed through a small hole drilled in the skull into a cerebral ventricle. The catheter then progresses subcutaneously across the skull, behind the ear, and down to the abdomen where it drains into the peritoneal cavity [1]. The relatively superficial nature of the shunt as it passes across the scalp increases the risk of pressure necrosis in this area. Patients with particularly friable skin or those with prolonged external pressure on the scalp are at increased risk for developing pressure necrosis overlying a VP shunt [16,17].

Conclusion

Ventriculoperitoneal shunts are devices that are frequently used for reducing intracranial pressure associated with hydrocephalus. The insertion of the shunt beneath the scalp sets the stage for potential pressure-induced scalp necrosis. Our patient's scalp necrosis initially presented as an erythematous crust overlying his VP shunt valve. However, progressive erosion of the overlying skin resulted in an exposed and unprotected VP shunt. It is important to recognize the early clinical presentation of scalp necrosis in a patient with a VP shunt; full-thickness necrosis of the scalp overlying a VP shunt can result in significant and potentially lethal consequences. Therefore, in a patient with a VP shunt, we suggest that a complete examination be performed to evaluate for possible scalp necrosis.

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