



Case Report

Basal Cell Carcinoma Involving a Ventriculoperitoneal Shunt: Case Report and Literature Review

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Ventriculoperitoneal shunts (VPSs) are commonly used for the management of raised intraventricular pressure, especially in the context of hydrocephalus. Malignant invasion involving a VPS is an exceedingly rare association, only reported three times in the literature to date. We present the involvement of a VPS with a recurrent basal cell carcinoma (BCC), which has not been described previously. It was theorized that previously breached tissue planes associated with the VPS tract might facilitate local spread of the BCC. As such, this case represented an exceedingly rare association that has important conceptual implications for definitive surgical management. A 51-year-old paraplegic man with spina bifida and hydrocephalus, who had been immunosuppressed for 7 years following a renal transplantation, presented to our clinic with a recurrent BCC involving an inactive VPS in the right neck. Surgical management involved an excision of the scar with a peripheral margin of 1 cm, along with removal of the involved shunt tract and intraoperative pathologic assessment. Definitive histopathology revealed a focus of infiltrating sclerosing BCC involving the subcutaneous tissue and abutting the VPS. As the scope for synthetic materials in surgery continues to expand, so does the multitude of interesting complications that appear to arise from interactions between host tissue and foreign bodies. This case report describes a fascinating relationship between a recurrent BCC and a VPS, providing a guide for future management to ensure adequate surgical clearance. The case would be of interest to head/neck surgeons, neurosurgeons, and plastic surgeons.

Key words: Basal cell carcinoma – Ventriculoperitoneal shunt – Incomplete excision – Immunosuppression – Renal transplant

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Basal cell carcinoma (BCC) is a common skin malignancy in Australia. A recent systematic review revealed that Australia had the highest incidence of BCC in the world, with a rate greater than 1000 per 100,000 person-years.¹ Renal transplantation carries an independent elevated long-term risk for the development of BCC and other cutaneous malignancies.² The risk is still higher in recipients who begin immunosuppression before the age of 50 years, with the relative risk of nonmelanoma skin cancer being 200 times the baseline risk observed in those without transplants.³

BCCs arise from pluripotent basal keratinocytes of the epidermis and other adnexal structures. Clinical forms include nodular, superficial, and sclerosing types. The natural history is of local growth with direct invasion into, and destruction of, adjacent tissues, with invasion into cartilage and bone observed in neoplasms of a more aggressive nature.⁴ Perineural invasion is not uncommon, but metastases are rare.⁵ This may be due to the dependence of BCC on the surrounding stroma for survival.

Ventriculoperitoneal shunts (VPSs) are commonly used for the management of raised intraventricular pressure, especially in the context of hydrocephalus. The capsule that forms around implantable foreign materials may act as a low-resistance pathway for direct tumor extension. The ability of a VPS tract to facilitate this has been described for select malignancies.^{6–8} Importantly, the involvement of a VPS foreign body conduit by BCC has not been described previously. This case presents important conceptual implications for definitive surgical management of infiltrative tumors in the vicinity of a foreign body conduit.

Case Presentation

A 51-year-old paraplegic man with spina bifida and hydrocephalus, who had been immunosuppressed for 7 years following a renal transplantation, presented to our clinic in March 2013 with an incompletely excised infiltrating sclerosing BCC in the posterior triangle of the neck adjacent to an inactive VPS. Prior to referral, the BCC had been twice incompletely excised in depth over the palpable VPS and adjacent sternocleidomastoid (SCM) muscle. Neither excision had addressed the VPS or its capsule. In 2004, a VPS had been placed for management of hydrocephalus. It was replaced with a VPS on the contralateral side of the neck in 2009 when the ipsilateral VPS stopped functioning.

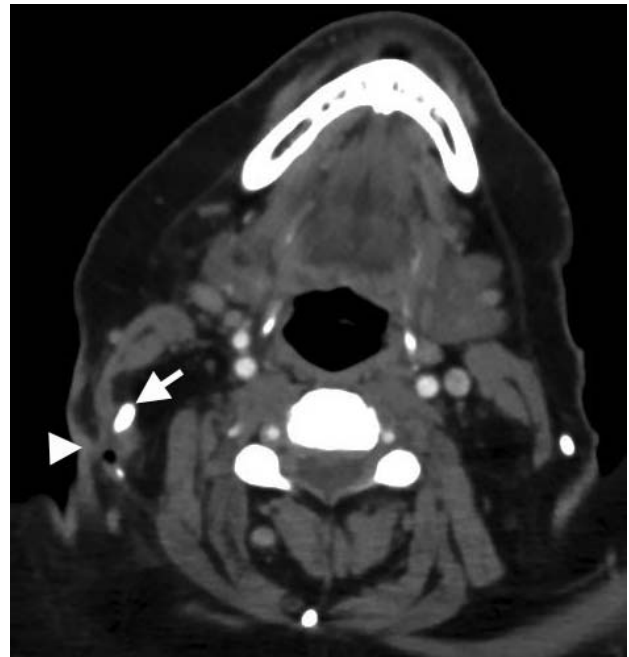


Fig. 1 Preoperative CT of the neck. Image shows a 3 × 2 cm lesion (white arrowhead) abutting the residual VPS tubing (white arrow) in the area deep to the SCM muscle in the right neck.

The calvarial part of the ipsilateral shunt was removed at the time, but the portion in the neck remained *in situ* because of technical difficulties encountered during attempts to remove it. The patient had also undergone a renal transplantation in 2006 secondary to longstanding obstructive nephropathy and was under immunosuppressive management with tacrolimus, mycophenolate mofetil, and prednisone. Other past medical history was significant for total cystectomy with an ileal conduit for neurogenic bladder and multiple non-melanoma skin cancers.

On examination, the patient had a 4-cm scar in level Va of the right neck, just posterior to the margin of the SCM muscle. There was no clinical evidence of BCC superficially. On palpation, a 3 × 2 cm mass could be felt under the scar and was fixed to the underlying SCM and adjacent VPS, which was also palpable in the inferior neck. There was no palpable cervical lymphadenopathy. Computed tomography (CT) revealed a soft tissue mass extending from the cutaneous scar and abutting the residual VPS capsule deep to the SCM muscle in the neck (Fig. 1). No other regional disease or metastatic spread was present. The patient was assessed in our multidisciplinary head and neck meeting, and the consensus opinion was for surgery

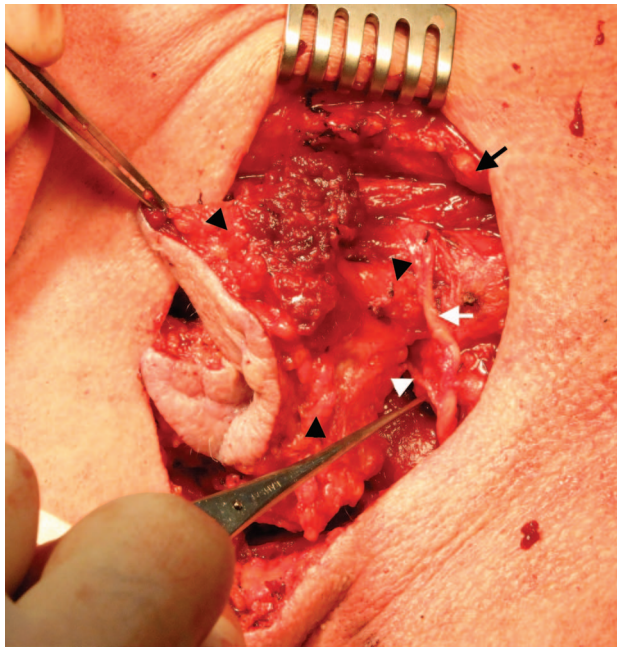


Fig. 2 Intraoperative view of the right neck with the excised lesion and residual defect. Image shows the relationship of the lesion (black arrowheads) to the adjacent SCM muscle (black arrow), spinal accessory nerve (white arrow), and cervical plexus contribution to the spinal accessory nerve (white arrowhead).

with wide excision of the lesion and adjacent VP shunt tract, using intraoperative frozen section assessment.

The scar was excised with a 1-cm peripheral margin. The excision included the skin, subcutaneous tissue, platysma, the posterior aspect of the SCM muscle, the great auricular nerve, the VPS capsule, and tubing that lay deep to the SCM (Fig. 2). The tract of the nonfunctional VPS was noted to pass between the spinal accessory nerve and its cervical plexus contribution. This was dissected free with a cuff of surrounding fatty tissue to include 2 cm of VPS proximal and distal from the involved capsule while preserving the spinal accessory nerve in its entirety. Those parts of the VPS outside of this field were fixed when firm traction was applied and were therefore left *in situ*. The caudal limits of the excised capsule and VPS were ligated. Additional biopsies of the distal limit of the VPS capsule were sent for frozen section and were clear of malignancy. The wound was closed directly and the postoperative course was uneventful.

Definitive histopathology revealed a focus of infiltrating sclerosing BCC involving the subcutaneous tissue and abutting the VPS (Fig. 3). There was

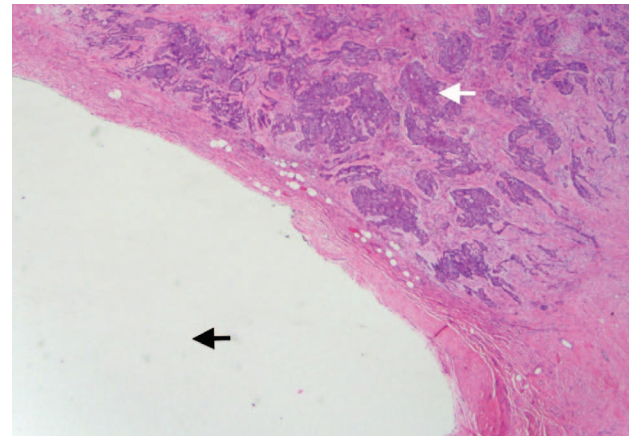


Fig. 3 Histopathology (×200) of the BCC and VPS specimen. This slide demonstrates the involvement of an infiltrating sclerosing-type BCC (white arrow) as it invades the tissue external to the VPS (black arrow).

no associated perineural or perivascular invasion, and all margins were clear of BCC. The VPS capsule was heavily calcified, with surrounding tissue showing extensive scarring.

At 24-month follow-up, the patient remained clinically disease free.

Conclusion

Involvement of a foreign body conduit by a BCC, such as a VPS, has not previously been described. Because this presented a unique association there were no guidelines by which to plan treatment. It was hypothesized that the VPS or its capsule could form a conduit by which the local or distant spread of the BCC could be facilitated. Significantly, similar routes of dissemination have been described for other types of malignancies, such as breast cancer,⁶ primary cerebrospinal fluid lymphoma,⁷ and pancreatic carcinoma.⁸ The theoretical mechanisms by which a patent foreign body conduit, such as a VPS, could promote tumor spread include direct invasion of the capsule with proximal and/or distal extension or breach of the capsule to access the potential space around the VPS, which could act as a low-resistance pathway for tumor spread.

In light of the unknown potential for tumor spread by a foreign body conduit and the immunosuppressed state of the patient, our multidisciplinary clinic advised radical surgery as a single modality. To ensure adequate resection we employed the use of wide margins of excision and

intraoperative frozen section histopathologic examination. Using this method, we were able to define the required margins for a complete excision.

We recommend the use of intraoperative pathologic specimen examination and the use of wide clinical margins for infiltrating sclerosing BCC involving a VPS and its associated capsule. Such an approach is justified because of the unacceptable risk of further local dissemination associated with an incomplete excision of BCC in the vicinity of a VPS, especially in the context of immunosuppression. The value of the multidisciplinary meeting in the planning and execution of this outcome was paramount to solving this unique surgical problem.

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