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CASE REPORT

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Brain herniation and subsequent complications following partial resection of high-grade glioma: A case report

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Key Clinical Message

This case highlights the need for tailored strategies to address issues like brain herniation, subdural hygroma, and cerebrospinal fluid leak, which, if not managed promptly, can lead to long-term neurological deficits. Additionally, the role of specialized facilities in delivering highly specialized care for managing such intricate cases cannot be understated.

Abstract

Decompressive craniectomy-induced subdural hygroma (SDH) frequently coexists with external cerebral herniation, resulting in neurological impairments. The incidence of brain herniation through a craniectomy defect postoperatively is 25%. Brain herniation (BH), SDH, and cerebrospinal fluid leak require urgent neurosurgical management as they can lead to irreversible long-term neurological deficits. We report a case of a 42-year-old male who presented with headache and grand mal seizures. He was diagnosed with herniation of brain parenchyma through the surgical defect with a displacement of the bone flap by a heterogeneously enhancing lesion in the left parietal lobe along with SDH in the left frontoparietal region post partial resection of high-grade glioma. In this report, we discuss the pathogenesis and management strategies of brain herniation, wound infection, cerebrospinal fluid (CSF) leak, ipsilateral SDH, floating bone flap, and communicating hydrocephalus in an adult patient following partial resection of high-grade glioma. This particular case emphasizes the value of an individualized patient-centered surgical approach to minimize the risk of postoperative complications.

KEYWORDS

elevated intracranial pressure, glial cell neoplasms, glial cell tumor, intracranial hypertension, skull excision, subdural cerebrospinal fluid effusion, subdural cerebrospinal fluid leakage

1 | INTRODUCTION

Brain herniation (BH) most commonly occurs upto 5 days post craniectomy for traumatic brain injury or stroke. Protruded brain tissue more than 1.5 cm through the craniectomy defect has been labeled as BH, the incidence of which, postoperatively, is around 25%.[1,2] Increased hydrostatic pressure gradients from capillaries and cerebral

reperfusion post decompressive surgery are thought to be the mechanism related to BH postoperatively.[3] Decompressive craniectomy-induced subdural hygroma (SDH) frequently coexists with external cerebral herniation, resulting in neurological impairments.[4] SDH refers to fluid collections that are either clear, barely xanthochromic, or blood-tinged occurring within the subdural space. They may be brought on by venous congestion, mechanical damage to the body, or para meningeal or meningeal infection. Headaches, vomiting, nausea, fever, and confusion are common SDH symptoms.[5] The pathogenesis of SDH is not well understood. However, the most likely explanation is the tearing of the subarachnoid layer leading to the ball valve opening resulting in unidirectional cerebrospinal fluid (CSF) flow into subdural space, forming SDH.[6,7]

Neurosurgical procedures are associated with higher morbidity and mortality as compared to any other surgical procedure.[2] In this case report, we discuss the pathogenesis and management strategies of BH, wound infection, CSF leak, ipsilateral SDH, floating bone flap, and communicating hydrocephalus in an adult patient following partial resection of high-grade glioma. The sequence of events is strikingly similar to complications following craniectomy for the management of raised intracranial pressure following head injury.

2 | CASE REPORT

A 42-year-old man with history of chronic headache, seizures, and inability to speak was diagnosed with left frontal high-grade glioma in an outside facility. The magnetic resonance imaging (MRI) brain images from outside faciltiy were not available but report mentioned a 3 × 3 cm cortical-based heterogeneously enhancing lesion in the left parietal lobe with surrounding vasogenic edema. The patient underwent craniotomy and partial excision of the tumor which was reported as an isocitrate dehydrogenase (IDH) mutated grade 4 astrocytoma using fluorescence in situ hybridization (FISH) testing. Follow-up imaging as shown in Figure 1 showed residual disease in the pars opercularis of the inferior left frontal gyrus which was not suitable for further resection, and patient was referred to our institution for further management. On assessment, he was found to have expressive dysphasia and mild right arm weakness. He had swelling related to the craniotomy flap and cereberospinal fluid (CSF) leak from wound. He was admitted for urgent surgery. Computed tomography (CT) scan brain showed progressive disease with leptomeningeal enhancement and interval increase in SDH along the frontoparietal region. MRI showed changes in the left parietal craniotomy. There was herniation of brain parenchyma through the surgical defect with a displacement of the bone flap by a heterogeneously enhancing lesion in the left parietal lobe measuring approximately 2.9 × 3 cm (AP × TR). Adjacent pachymeningeal enhancement was noted. T2weighted hyperintense and fluid attenuated inversion recovery (FLAIR) hypointense signals were noted in the left frontoparietal region, characteristic of SDH. This had a maximum thickness of 1.5 cm (Figure 2).

There was a dehiscence of the wound at the operation, leading to a CSF leak. The bone flap that had been fixed with silk sutures was pushed out by the herniated brain. The bone flap was removed, and the herniated brain was excised. A burr hole was made over the frontal edge of the craniectomy defect, and the SDH, which was under high pressure, was drained, allowing the brain to become lax and settle into the defect. The dural defect was reconstituted with temporalis muscle and fascia. Debulking of tumor was deferred due to brain swelling. The excised herniated cerebral tissue was reported as high-grade glioma. The specimens of fascia, bone, and subdural fluid sent for culture. The culture of specimens of galea aponeurotic, bone, and subdural fluid showed light growth of

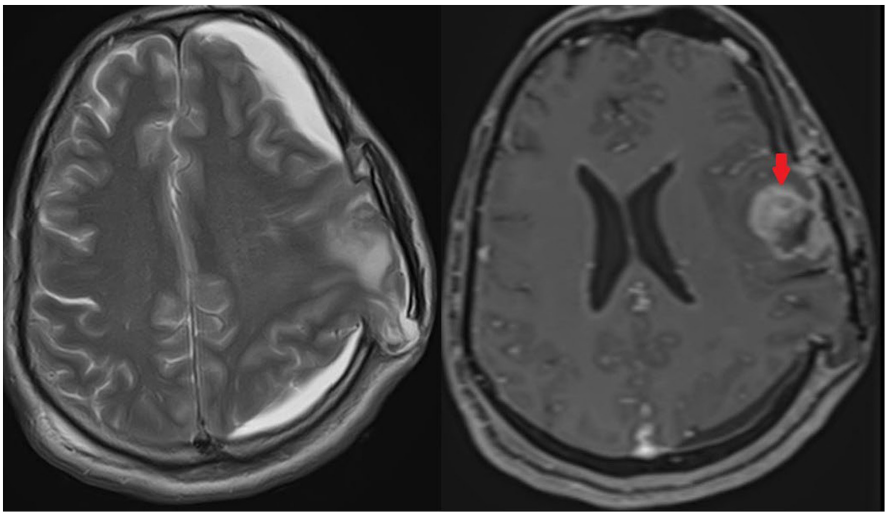

FIGURE 1 T1 and T2 section of magnetic resonance imaging (MRI) showing residual tumor in left inferior frontal gyrus (red arrow).

Pseudomonas Aeruginosa. The CSF culture was negative after 72 h of incubation. The infection was empirically treated with ceftriaxone followed by ciprofloxacin and piperacillin and tazobactum for 14 days once susceptibility report was back. Postoperatively expressive dysphasia improved, but CSF leak from the craniectomy wound recurred. Given the leakage of CSF from the wound, the patient was treated with lumbar CSF drainage for 5 days. Once the drain was removed, the craniectomy flap became tense. The CT scan brain without contrast showed progressive dilation of lateral ventricles and brain herniation through the craniectomy defect (Figure 3). The herniation resolved after the placement of a medium-pressure ventriculoperitoneal (VP) shunt with a mid-pressure burr hole valve, a 7 cm ventricular catheter, and a standard distal catheter with slits.

There was the resolution of the BH, SDH, and CSF leaks. The soft craniectomy flap wound was healing well without signs of infection. GCS improved to 14 and he had mild hemiparesis and expressive dysphasia. The patient was transferred back to referring hospital 3 days after VP shunt placement on antibiotics.

3 | DISCUSSION

A rare case of cerebral herniation through a surgical defect with coexisting SDH, post partial resection of high-grade glioma, has been described. The majority of these type of cases is reported in the pediatric population, with little data in the adult population.[8] High-grade gliomas are associated with raised intracranial pressure due to the tumor mass and vasogenic oedema.[3] Surgery is indicated to obtain tissue for histopathological diagnosis and achieve maximum safer resection; it has been reported that the incidence of complications increases where less than 50% of the tumor is resected as the residual tumor mass and associated edema can worsen in the postoperative period.[2] The size of the craniotomy is usually based on the extent of enhancing tumor mass. However, the region

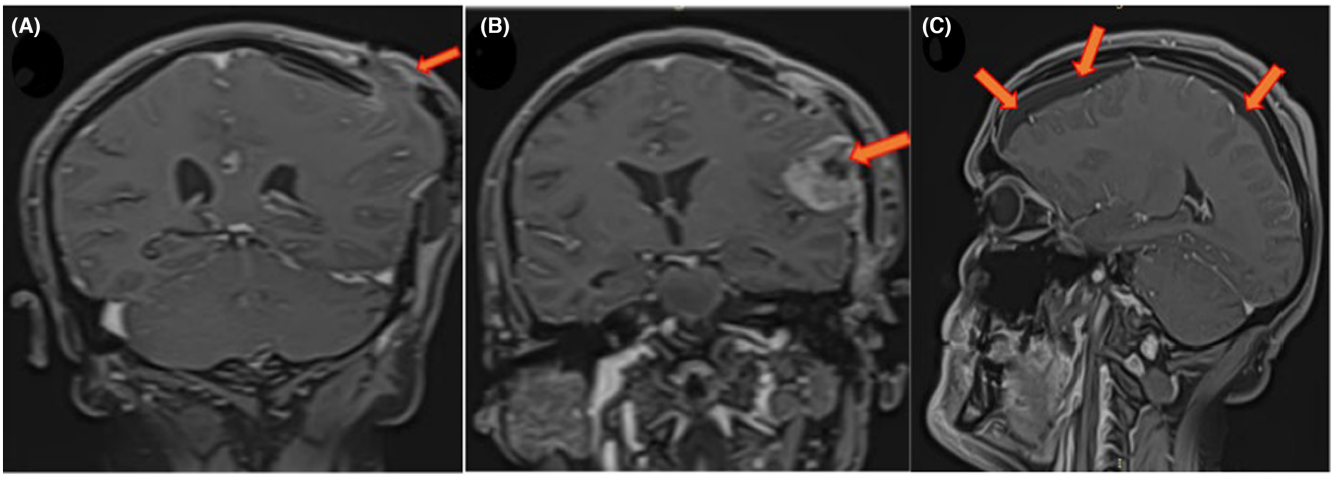

FIGURE 2 T1- Weighted coronal and sagittal views of magnetic resonance imaging (MRI) of the brain showing: (A) Displaced bone flap and herniation, (B) Residual tumor, and (C) Subdural hygroma.

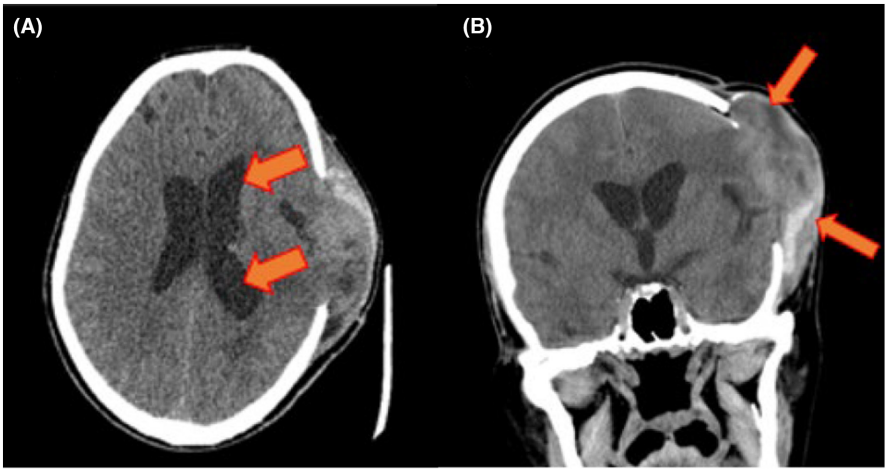

FIGURE 3 Computed tomography (CT) brain axial (A) and coronal views (B) showing hydrocephalus and herniation of the brain through the craniectomy defect.

of vasogenic edema extends well beyond the margins of the high-grade glioma, which continues to generate pressure effects, especially with incomplete resection,[3] which forces the surgeon to do a primary craniectomy or loosely replace the bone flap as in the above case.[9,10]

Furthermore, herniated tissue can compromise venous drainage after the veins get compressed against the edge of the craniectomy,[10] thus causing various neurological deficits which gradually improved after the membrane was resected and the herniated brain structure was excised, as mentioned above. In line with prior case reports, immediate surgical intervention of the herniated brain is advised to reduce the possibility of irreparable herniation with lasting neurological impairment. Craniectomy also leads to a dural defect and damage to the arachnoid membrane, which are associated with the development of SDH, CSF leak from the wound, poor wound healing, and infection[10] as in the case mentioned, which was alleviated by subsequent treatment of each complication including drainage for SDH and CSF leak along with intravenous antibiotic course for infection. The recurring SDH and leakage of CSF from the wound post-surgery could have obstructed the cerebrospinal fluid flow, which lead to hydrocephalus, and the placement of a VP or ventriculoatrial, or lumboperitoneal shunt is often necessary for treatment as done in this case.[11]

Following craniectomy for a head injury, the sequence of adverse events follows a recognized time course and pattern that is like the course of events in the above case indicating a common pathophysiological mechanism.[12] Postoperative wound complications following craniotomy for high-grade glioma can significantly increase the cost of treatment, increase patient morbidity, prolong hospital stay, and significantly delay radio-chemotherapy.[12,13]

Craniotomy for high-grade glioma requires careful planning to achieve maximum safe resection and reduce the potential for BH and subsequent complications related to large residual tumor volume. Based on morbidity and mortality postoperatively, an aggressive surgical approach can be taken to deal with high-grade glioma.[14] The meticulous surgical technique to achieve complete dural closure and firm fixation of the bone flap may have reduced the complications described in this case report. This case emphasizes the value of an individualized patient-centered surgical approach to minimize the risk of postoperative complications.

AUTHOR CONTRIBUTIONS

Sheheryar Hanif: Writing – original draft. **Irfan Yousaf:** Conceptualization; resources. **Maham Iqbal:** Writing – original draft. **Usha Kumari:** Methodology; project administration; writing – review and editing. **Salim Surani:** Supervision; writing – review and editing.

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DATA AVAILABILITY STATEMENT

Not applicable.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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