Upward Herniation in Awake Craniotomy: A Case Report

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ABSTRACT

Awake craniotomy has been gaining popularity for the last decade. It allowed maximum tumor resection while avoiding neurological morbidity. However, this technique presents several challenges to both the neurosurgeon and anesthesiologist. In this case, we present a 33-year-old male who was diagnosed with low-grade glioma in the left parieto-occipital, which required surgical resection. Anatomically, the tumor was located in the language area. Hence, it was decided to perform an awake craniotomy excision of the tumor to allow intraoperative cortical mapping to preserve language functions. Intraoperative, a subdural hematoma was noted, and severe pain occurred. Eventually, this leads to an upward herniation of the brain parenchyma. The crisis was addressed promptly with maneuvers to decrease intracranial pressure. Awake craniotomy was abandoned, and the procedure was converted to general anesthesia without the benefit of intraoperative cortical mapping. It is important to note that complications may arise during the procedure, leading to significant harm and debilitation for the patient. Prompt crisis management is necessary to address these potential issues and ensure the highest level of care is provided.

Keywords: failed awake craniotomy, upward cerebral herniation, challenges in awake craniotomy

INTRODUCTION

Awake craniotomy for tumor resection located in eloquent areas presents several challenges. These areas are responsible for governing motor, language, and sensory. In this type of surgery, it allows neurosurgeons to maximize tumor resection while preserving neurological function. The role of anesthesiologists is to ensure a safe perioperative period, patient collaboration during the awake phase, and provide satisfactory analgesia. Awake craniotomy is a well-tolerated procedure. However, complications can arise in this situation, including seizures, neurologic deficit, air embolism, respiratory complications, cerebral edema, and pain that may eventually lead to an uncooperative patient.

CASE PRESENTATION

A 33-year-old right-handed male was brought into surgery for awake left temporoparietal craniotomy excision of a tumor. He presented with a two-month history of intermittent headaches associated with blurring of vision of the left eye. An ophthalmologic consult was done, and he was noted to have papilledema. On brain MRI, there were cystic masses at the left occipital (5.9 x 2.8 x 4.1 cm) and left temporoparietal (5.8 x 3.6 x 4.9 cm) areas and an enhancing irregularly-shaped nodular focus (2.5 x 1.8 cm) anteromedial to left occipital cyst (Figure 1). After a month, he sought consult with a neurologist. He was started on Dexamethasone 4 mg/tab, one tab once a day, and Levetiracetam 500 mg/

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tab, one tab twice a day. At this time, he was advised for a surgical intervention.

During the pre-anesthetic evaluation, the patient regularly takes dexamethasone 4 mg/tab, one tab once a day, and Levetiracetam 500 mg/tab, one tab twice a day for one month. He denied comorbidities, allergies, or previous surgeries. A lifestyle review revealed that he is a social alcoholic drinker. On physical examination, he weighs 72.2 kgs at a height of 157 cm and has a BMI of 29.29 kg/m^2; he is in good general condition, with normal pulses and regular heart sounds with no murmurs or adventitious sounds. Airway examination showed Mallampati class 1, neck movements within normal limits, and no difficult airway predictors. Neurologic examination revealed dyscalculia and shallow right nasolabial fold. All other aspects of the neurological examination were found to be normal. Laboratory tests were within normal limits. The planned procedure was explained thoroughly, and every detail of the planned procedure was walked through. The possibility of converting to general anesthesia was also discussed with the patient and relatives. The patient was given a chance to voice out his anxiety and fears and was reassured by the anesthesiologist.

Maintenance medications were taken with sips of water on the morning of the procedure. Upon arriving in the operating room, the patient was calm and oriented. The patient was attached to standard monitors, including electrocardiography (ECG), noninvasive blood pressure monitoring, and pulse oximetry. NIBP and pulse oximetry were placed on the ipsilateral side of the lesion. Supplemental oxygen at 4L/min (FIO_2 0.36) was administered via an O_2 nasal cannula with capnography. Baseline vital signs showed BP 140/90 mmHg, pulse rate of 90/min, RR 16, SpO_2 100%, and $EtCO_2$ (End-tidal carbon dioxide) 40 mm Hg.

General anesthesia was performed with a laryngeal mask airway (LMA). Anesthesia induction included fentanyl 50 mcg and propofol 150mg IV. LMA Supreme size 3 was inserted with good placement and was maintained in assisted ventilation. Target-controlled infusion (TCI) was initiated with propofol at 1 µg/ml (Marsh model), Dexmedetomidine infusion started at 0.5 mcg/kg/hr and one hour after Remifentanil at 1 ng/ml (Minto model). Scalp nerve blocks were performed using an anatomical approach with 0.25% bupivacaine + lidocaine 1% + 1:200,000 epinephrine mixture (total dose: 30 mL) before Mayfield frame installation. After induction, additional peripheral venous access (g18) and intra-arterial pulse pressure were obtained on the ipsilateral side of the brain lesion. A condom urinary catheter was also placed to measure urine output. The following medications were given: paracetamol 1 g IV and prophylaxis for nausea and vomiting with ondansetron 8mg IV and dexamethasone 4mg IV.

The patient was placed in a sniffing position to ensure airway patency. Clear drapes were positioned to maintain unimpeded access to the airway and face. The surgical procedure started one hour and 15 minutes after induction.

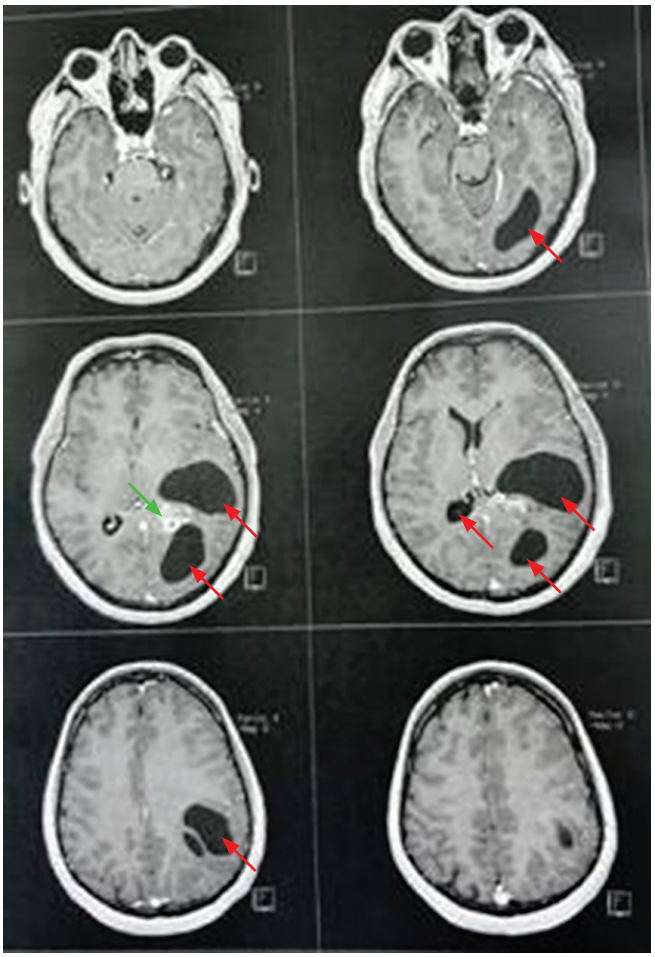

Figure 1. ***Cranial MRI.*** Cystic masses *(red arrows)* at left occipital (5.9 x 2.8 x 4.1 cm) and left temporoparietal (5.8 x 3.6 x 4.9 cm) areas, enhancing irregularly shaped nodular focus *(green arrow)* (2.5 x 1.8 cm) anteromedial to left occipital cyst with hemosiderin stains and no restricted diffusion. Perilesional vasogenic edema and secondary slight compression of ipsilateral lateral ventricle and midbrain. Intraocular protrusion optic nerve head suggests papilledema. Tortuous vessels left temporal convexity, likely middle meningeal and superficial temporal veins.

The remifentanil, propofol, and dexmedetomidine infusions were continued as previously, with no significant changes in hemodynamic variables and maintenance of BIS from 54-55.

Vital signs before incision were heart rate of 69 BPM and blood pressure of 101/48 mmHg. Vital signs after skin incision and during craniectomy remained stable, with a systolic range of 90-110 mm Hg over a diastolic range of 40 to 60 mm Hg; heart rates were 60 to 70/min and $ETCO_2$ maintained at 30-35 mmHg. Before the opening of the dura, the neurovascular bundle from the middle meningeal artery was infiltrated with lidocaine 2%. Anesthetic drugs were discontinued to allow the return of consciousness.

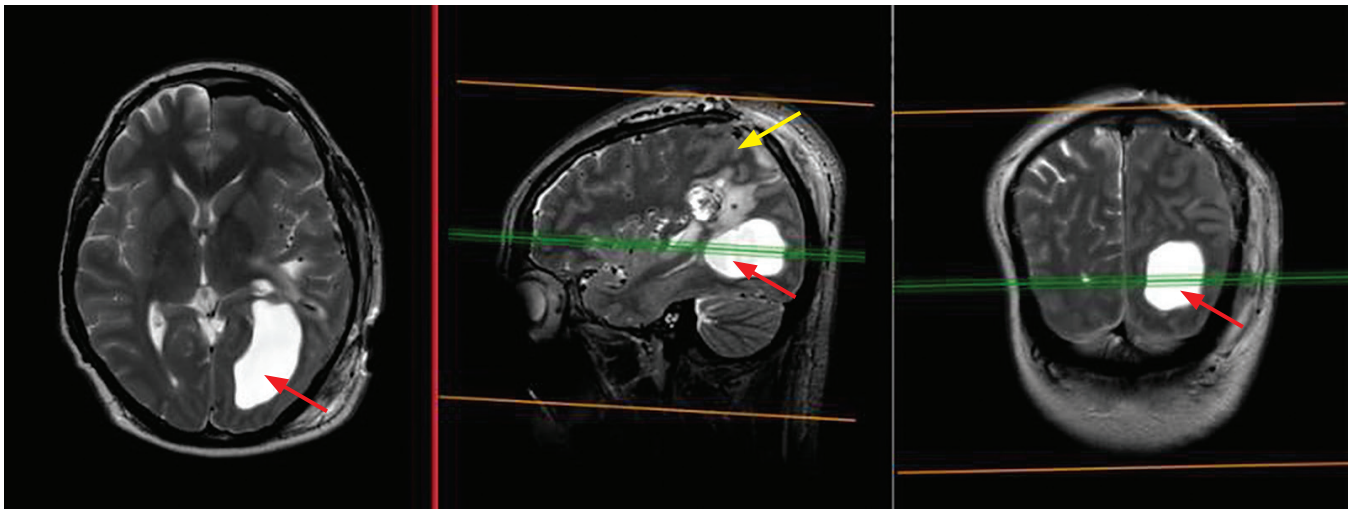

Figure 2. Parenchymal defect with hyperintense signals likely representing bleeding post-op, with associated perilesional vasogenic edema *(yellow arrow)* still and minimal midline shift. Still with left occipital cystic lesion *(red arrows)* with no apparent change in size.

The patient was awake 10 minutes after discontinuation of anesthetics. LMA was then removed successfully. He remained calm, comfortable, and followed instructions. The patient was then on O_2 support via nasal cannula at 4 LPM (FIO_2 0.36). Ultrasound was used to locate the tumor. The cystic component was located at the subcortical level. The surgery proceeded with the aspiration of 20 ml of a cystic component located in the left parietal-temporal area slowly and carefully using a g 18 abocath. After decompression, the patient complained of extreme headache and was restless. Upon opening the dura, a subdural hematoma was observed along with upward herniation of brain parenchyma through the surgical opening.

The neurosurgical team proceeded to perform evacuation of subdural hematoma. At this point, there was a marked increase in the vital signs, blood pressure as high as 165/80 mm Hg, heart rate 95 BPM, SPO_2 100%, and $ETCO_2$ 42. He was given fentanyl 100 mcg IV with no resolution of symptoms. Then remifentanil was started at 0.5 ng/ml and was gradually titrated up to 2.25 ng/ml. Still, the patient was restless. The neurosurgical team decided to abandon the awake technique and stabilize the patient. Conversion to general anesthesia was achieved by propofol bolus 1 mg/kg IV. The airway was secured using an LMA, and proper placement of LMA was confirmed with equal chest rise and breath sounds.

Cerebral edema was addressed with mannitol 0.27 g/kg IV and hyperventilation to decrease $ETCO_2$ 42 to 35 mmHg. In addition, the patient was placed in a head-up position of 45 degrees to decrease cerebral edema further. Vital signs were stabilized, and there was a resolution of cerebral edema. Surgery proceeded without further complications. Corticectomy was done above the supramarginal and angular gyrus. After the surgery, all anesthetics were discontinued, and LMA was removed. The patient was shifted to O_2 support via nasal cannula at 4LPM (FiO_2 of 0.36). The patient was transferred to the post-anesthetic care unit (PACU) fully awake, conversant, calm, and without pain.

Postoperative brain MRI (Figure 2) confirmed the removal of the left parietotemporal cystic lesion and irregularly shaped nodular lesion. There is still a cystic lesion located on the left occipital region. The rest of the peri-operative course was uneventful, and he was discharged on the 5th post-op day.

DISCUSSION

Awake craniotomy is the gold standard of tumor resection located in the eloquent areas.[1] In most cases, the procedure is well tolerated.[2] The patient is purposedly kept awake for active participation during cortical mapping and tumor resection. This is because it can prevent injury to the eloquent brain region while excising the tumor. Furthermore, it is perceived that the brain parenchyma has no or minimal pain fibers.[3] This allows neurosurgeons to perform painless intracranial surgery in awake patients. Despite the growing prevalence of awake craniotomies, there are still few studies documenting complications of an awake craniotomy, such as intraoperative pain, subdural hematoma, and upward herniation.

The incidence of a subdural hematoma in awake craniotomy is not very well discussed. There are only two case reports detailing a subdural hematoma during an awake craniotomy in Pubmed and Google Scholar databases. It can manifest with loss of consciousness, apnea, isocoria, headache, and nausea. The location of subdural hematoma for both these cases was on the contralateral side.[4,5] This contrasts with our case, which was on the ipsilateral side.

The aspiration of the cystic component before opening the dura may have a decompressive effect. This may have resulted in stretching and tearing of bridging veins and subsequent subdural hematoma. The resultant hematoma can cause an elevation in intracranial pressure (ICP), which in turn, can lead to an increase in intracranial venous pressure. That increase in intracranial venous pressure would transmit an increased pressure in the raptured bridging veins, causing more bleeding into the subdural layer.[6] The formation of subdural hematoma causes a displacement of the brain. Traction on intracranial structures can eventually lead to a headache, as these structures are sensitive to pain. Pain can be caused by pain-sensitive structures such as the meninges, meningeal blood vessels, large cerebral arteries, and veins.[3] This may explain the intense pain felt by the patient after aspiration of the cyst.

Published studies have shown that intraoperative pain reported ranged from 20-56%.[1,7] Pain intensity ranges from moderate by 20-24% and severe by 5%. In a prospective postoperative survey, 25 out of 105 (23.8%) patients reported a visual analog scale (VAS) above 3/10.[8] Intraoperative pain can be managed with short-acting opioids. However, administration of opioids can lead to sedation and respiratory depressant effects.[9] This can interfere with providing an awake, cooperative patient and leads to cerebral swelling from hypercapnia.

Brain herniation is often thought about as a downward phenomenon. However, it can also manifest with an upward movement. It is described as displacement of brain tissue because of mass effect.[10] Different types of herniation depend on their location and the structures involved. PubMed database search for keywords with "transcalvarial herniation AND awake craniotomy" and "upward herniation AND awake craniotomy" yielded no results. However, three cases have been described with a transcalvarial herniation after a post-elective craniotomy. All three cases were reported to have delayed neurologic deterioration, and this was addressed with the repair of dural defect.[11]

Various factors have contributed to brain herniating through the surgical defect. One of which is an increased ICP. As stated by the Monro- Kellie doctrine, the sum of volumes of blood, cerebrospinal fluid (CSF), and brain remains constant. Any change in one of the constituents necessitates a reciprocal change in one or both compartments.[12] The two main factors that significantly impact ICP are CSF and blood. Cerebral blood volume (CBV) is directly associated with cerebral blood flow (CBF). Increasing CBF by increasing blood pressure eventually leads to a rise in ICP.[13] In this case, elevated blood pressure secondary to pain might have been a factor contributing to herniation of the brain. Furthermore, the patient has an intracranial pathology (brain tumor) that would aggravate ICP from the vasogenic edema. In the presence of brain tumors, there is a disruption of blood-brain-barrier and an increased permeability of vessels around the tumor, causing an influx of solutes into the brain parenchyma.[14,15] The extravasated fluid, when accumulated, increases ICP.[16] When the ICP reaches a certain threshold, the compensatory mechanism becomes exhausted and can significantly elevate ICP, which can lead to cerebral ischemia and herniation.[17]

Brain herniation through the defect can strangulate viable brain parenchyma over dural edges.[11] A rapid intervention is needed to address the underlying cause of increased ICP to avoid further injury to the brain parenchyma. It is interesting to note that there was no loss of consciousness or weakness despite an upward herniation. Between a transcalvarial and a downward transtentorial herniation, the latter would present a more debilitating effect. In a downward transtentorial herniation, the diencephalon's involvement alters the sensorium by disrupting the reticular activating system.[18]

A scenario of brain herniating through the defect poses difficulties to neurosurgeons. In an awake craniotomy, this can interfere with tumor resection and eventually abandonment of the awake procedure. Various strategies can be utilized to control cerebral edema and brain herniation. Positioning the patient in a head-up tilt of 30-45 degrees would facilitate venous drainage.[19] Hyperventilation reduces the partial pressure of arterial carbon dioxide, leading to cerebral vasoconstriction. The use of propofol provides a reduction in cerebral metabolic consumption rate ($CMRO_2$) and lowers ICP via decreasing CBF. Furthermore, it has neuroprotective, anticonvulsant, and antiemetic properties. Propofol is the optimal anesthetic agent for dealing with increased ICP. Osmolar agents such as mannitol reduce cerebral edema by excreting water content from intracellular tissue into the intravascular compartment.[20]

CONCLUSION

Awake craniotomy has become common in neurosurgical centers. As with any surgical procedure, awake craniotomy presents its own set of complications. These complications can be debilitating for the patient. Management of these complications requires prompt crisis management, continuous team communication, and experienced anesthesia and surgical teams.

Informed Consent

Informed consent was obtained for the publication of the patient's clinical information.

Statement of Authorship

Both authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

Both authors declared no conflicts of interest.

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