Anaesthetic Management of Left Temporal Glioma Undergoing Awake Craniotomy

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

For an awake craniotomy, a 49 year old (ASA 2), 78 kg woman with type II DM was given regional anaesthesia (scalp block) with monitored anaesthesia care (MAC). She had a headache, which was primarily caused by a left temporal glioma. She was very apprehensive about having this procedure done while she was awake. Fentanyl and Dexmedetomidine infusions in combination with scalp block initially provided adequate operating conditions. Because the patient needed to be fully awake, alert and cooperative during the language and motor mapping, all sedation was turned off. Patient was cooperative and obeyed commands during motor and language mapping as well as during tumour excision. Patient underwent complete excision of tumour without any postoperative neurological deficit. The success of the awake craniotomy is dependent on the patient cooperation, anaesthesiologist's experience, adequate intraoperative analgesia coverage, careful sedation titration, and meticulous planning.

Keywords: Awake craniotomy; conscious sedation; dexmedetomidine, scalp block.
1. INTRODUCTION

Sir Victor Horsely performed the first awake craniotomy in 1886 to localise the epileptic focus with cortical electrical stimulation [1]. Awake craniotomy has been a 'gold standard' for tissue resection close to the eloquent cortex, deep brain stimulation surgery, neuroendoscopic surgery, ventriculostomy and excision of small lesions over the last few decades. It is a difficult task to perform awake craniotomy with an uncooperative patient and an inexperienced anaesthesiologist [2,3,4]. Awake craniotomy has advantages such as reduced general anaesthesia complications, maximum tumour excision with minimal neurological deficits, shorter ICU and hospital stays; and disadvantages such as an unprotected airway, excessive sedation, respiratory depression. Sometimes patients may become uncooperative during the procedure, which may require conversion to general anaesthesia [5,6,7,8,9,10,11]. There are various techniques for performing awake craniotomy; the best choices for surgeons and anaesthesiologists are MAC, Asleep–Awake–Asleep and Asleep–Awake technique. Optimal anaesthesia care is a critical step in the success of awake craniotomy [10,11].

2. CASE REPORT

A 49 year old female (78 kg, 155 cm) with type 2 diabetes was admitted with a left temporal glioma and was scheduled for a left temporal craniotomy and tumour resection. She had been complaining of headaches for three months and had no other complaints. She had a CT scan, which revealed a 43*31*35mm left temporal lobe lesion involving the hippocampus with subtle diffusion restriction, heterogeneous enhancement, and moderate oedema, mild mass effect over the midbrain, with a 4mm midline shift to the right. Awake craniotomy was planned to perform intraoperative cortical mapping for speech and motor function testing. General examination and vital parameters were within normal limits. The patient was grade 1 obese (BMI 32.46 Cm2). Patient's comfort and cooperation was assured. Patient was given final position for surgery. Awake anaesthesia care was a critical step in the success of awake craniotomy.

On the day of surgery, the patient was counselled and reassured in the preoperative room. Informed verbal and written consent was obtained, one 18 G IV cannula was secured, and Inj. Ondansetron 4 mg, Inj. Metoclopramide 10 mg and Inj. Ranitidine 50 mg were administered intravenously. Prior to taking the patient into the operating room, difficult airway trolly (LMA, ET tube, bougie, laryngoscope, airways), medications for general anaesthesia, 50 ml Dexmedetomidine syringe (concentration of 4 mcg/ml) were kept ready. AC was turned off and all OT staff was informed to maintain noise free environment. In the operating room, standard anaesthetic ASA monitoring were attached and 0.9 % NS was started (100 ml/hr), Dexmedetomidine infusion loading dose was given at 1mcg/kg over 10 min followed by maintenance at 0.2 – 0.7 mcg/kg/hr. Inj. Fentanyl 1 mcg/kg was given iv before scalp block. Scalp block was performed using 0.5 percent bupivacaine 2 to 3ml at bilateral Supraorbital nerve, Supratrochlear nerve, Zygomaticotemporal nerve, Auriculotemporal nerve, Lesser occipital nerve, Greater occipital nerve and Greater auricular nerve. Vital parameters were normal during and after scalp block. Oxygen at 4L/min was started via face mask with capnography monitoring. Urinary catheterisation and right radial arterial line placement was done post sedation. The patient was comfortably positioned and covered with a warming air blanket. Dexmedetomidine infusion was stopped and patient was awakened before Mayfield head holder pins placement. Fentanyl 1 mcg/kg was administered prior to the application of pins. Local anaesthetic infiltration was also performed at the site of the Mayfield head holder pins with 2-3 ml 2% lignocaine at each site. Patient was given final position for surgery. Patient's comfort and cooperation was assured during the positioning. Dexmedetomidine infusion maintenance was resumed again. Inj. Leviteracetam 1 gm iv was given as prophylaxis for seizure control and Inj. Mannitol 0.75 gm/kg was given for brain relaxation. Surgeons were told to infiltrate site of skin incision with 2% lignocaine. Before opening the dura, pellets soaked in 2% lignocaine were placed over the
durameter at the site of the dural opening. When the dura was opened, dexmedetomidine infusion was stopped. The patient was immediately awakened. Using a Medtronic Nim eclipse neuromonitoring system, direct cortical stimulation was done to perform motor and speech mapping, following which tumour resection was started. Patient was asked to perform various tasks rehearsed during preop visit for motor and speech functions intraoperatively and any changes in motor and speech function were noted and informed to surgeons. Following the total tumour resection with conservation of motor and speech functions, dexmedetomidine infusion was restarted again. During dural closure, Inj. Fentanyl 0.5 mcg/kg was administered again. Dexmedetomidine infusion was stopped during the skin closure. The entire surgery was completed in about 4 hours and 30 minutes. Total doses of Dexmedetomidine and Fentanyl required were 218.8 mcg and 240 mcg respectively. RBS was within normal limits throughout the surgery. There were no complications or complaints throughout the procedure except for positional discomfort. Postoperatively, the patient was transferred to the Neuro ICU and given Inj. Paracetamol 1gm iv 8 hourly for postoperative analgesia. Patient didn’t complain surgical site pain for almost 4-5 hours postoperatively. The patient was discharged on the fourth day with no speech or motor disability.

3. DISCUSSION

The concept of awake craniotomy arose from epilepsy surgery. Awake craniotomy is used to improve neurological outcomes when operating on tumours near eloquent areas of the brain. Because of advances in anaesthetic management and equipments, performing awake craniotomy isn’t difficult in modern times, but it requires good team work, proper patient selection, and patient cooperation. There are several methods for performing awake craniotomy, including Asleep-Awake-Asleep (AAA), Asleep-Awake and Monitor anaesthesia care (MAC) with scalp block. These techniques are equally effective, but each has some disadvantages like need to insert LMA, excessive sedation in AAA and AA technique and unprotected airway, patient discomfort in MAC. The most difficult task for anaesthesiologists is providing adequate analgesia, sedation, stable hemodynamics, airway management, and patient comfort during craniotomy and neurological mapping. Despite this difficulty, it has many advantages like better outcome, greater extent of resection, fewer postoperative neurological deficits, shorter hospital and ICU stay. We decided to perform awake craniotomy with MAC as it is comparatively safer than other techniques provided sedation is titrated well. To provide analgesia and hemodynamic stability, regional anaesthesia in the form of a scalp block was used. It is crucial to select a long-acting local anaesthetic of choice and to avoid exceeding the maximum toxic dose. The primary sedation and hypnotic drug used for the procedure is Dexmedetomidine, a selective alpha-2 receptor agonist with advantages such as not suppressing respiratory drive, not interfering with electrophysiological testing, and earlier arousal; all of which favour the use of Dexmedetomidine during awake neurosurgical procedures. The most common side effects are bradycardia and hypotension which can occur at higher doses. Close monitoring of the depth of sedation is critical in the awake technique, and should be done using hemodynamic monitors and capnography and depth of anaesthesia monitors like Entropy or Bis. Capnography can indicate about respiratory depression, hypoventilation. Oversedation can lead to respiratory depression causing hypercarbia, which can lead to increased intracranial pressure and brain bulge. While undersedation can lead to uncooperation, anxiety, tachycardia, hypertension, and nausea. MAC with sedation is associated with less agitation compared to other techniques. Airway obstruction, seizure, and venous air embolism are the most lethal complications from anaesthesia and the surgeon’s side during awake craniotomy. Airway obstruction may occur during MAC with sedation. Most cases of airway obstruction can be relieved with proper airway management or the placement of an LMA or an ETT, but this can be difficult due to the Mayfield pins being fixed to the patient's head and limited access to the patient from the head side. Fortunately there was no episode of airway obstruction or desaturation during the procedure. The key to prevent airway obstruction is constant monitoring of patient’s chest movement and capnography. The incidence of seizures ranges from 3% to 16%. Most seizures can be controlled by ceasing the cortical stimulation by surgeons and irrigation of the cortex with ice cold saline. Sometimes benzodiazepines (Lorazepam, Midazolam) or antiepileptic drugs (Phenytoin, Levetiracetam, Sodium Valproate) may be required. Venous air embolism can be detected by ETCO2 monitoring.
precardial doppler or hemodynamic monitoring and managed by covering open area with saline soaked mops, 100% oxygen, head low position, fluids and vasopressors. Hypothermia should be avoided by using warm air devices. In postoperative care, the patient should be observed in Neuro ICU and pain management can be taken care of by administering small doses of opioids intravenously, including patient controlled analgesia combined with paracetamol [11,13,14].

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

Informed verbal and written consent was obtained from patient.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES


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