Use of dexmedetomidine infusion in anaesthesia for awake craniotomy

INTRODUCTION

Anaesthesia for awake craniotomy poses several challenges to an anaesthesiologist. The patient has to be anaesthetised to a level where he/she is able to tolerate the surgery and also needs to be fully alert and co-operative during neurocognitive testing.

The awake craniotomy technique has been used in patients undergoing surgical management of supratentorial tumours, AV malformations, etc. This technique aims to maximize lesion resection while sparing important areas of the brain.[1]

Dexmedetomidine has got good analgesic, sedative and anaesthetic-sparing properties without causing significant respiratory depression.[1] We wish to present a case of awake craniotomy where we used dexmedetomidine as an adjuvant to good effect.

CASE REPORT

A 50-year-old patient presented with complex partial seizures since 6 months and moderate degree of speech defect. The computed tomography (CT) scan revealed a brain tumour involving the left temporal lobe. The patient was explained about the awake craniotomy technique. The routine blood investigations were normal, the patient did not have any other systemic illnesses and he was weighing 80 kg.

Once in the operation theatre, glycopyrolate 0.2 mg, fentanyl 100 mcg and ondansetron 4 mg were administered intravenously and a simple O_2 mask was applied. Monitoring included ECG, pulse oximetry, non-invasive blood pressure (NIBP), end tidal CO_2 (sample tube under the O_2 mask) and urine output. The patient was placed in right lateral position with head resting on a horse shoe support. A bolus dose of propofol 100 mg was administered intravenously. Scalp block was performed with 20 mL of 0.5% bupivacaine. Infusions of propofol (60 mg/h) and dexmedetomidine (1 mcg/kg/h) were started. The scalp skin was infiltrated with 1% lignocaine along the line of incision and the surgery was begun. The patient was comfortably sedated and he tolerated the

procedure well during craniotomy, dural opening, tumour excision and during closure. The level of sedation was assessed by Ramsay sedation score. It was maintained between scores of 3 to 5 until neurocognitive testing was started. Two hundred millilitres of mannitol was infused about 15 min prior to completion of craniotomy. We observed a decrease in heart rate from 80 to 90 bpm to 50 to 60 bpm and blood pressure from 120 to 130/85 to 95 mmHg to 90 to 100/52 to 60 mmHg 30 min after initiation of dexmedetomidine infusion. The dexmedetomidine infusion rate was decreased to 0.5 mcg/kg/h to prevent further decrease in heart rate and blood pressure. Ten minutes prior to neurocognitive testing, the infusions of dexmedetomidine and propofol were stopped.

Once the dura was opened, brain was found to be relaxed and pulsating. During excision of the tumour, the patient underwent neurocognitive testing including naming, reading, counting and verbal fluency intermittently. The patient was adequately sedated but was easily arousable. Intra-operative arterial blood gas analysis showed normoxia and normocarbia. During closure, the infusions of propofol and dexmedetomidine were restarted at the same rate and 100 mcg of fentanyl was administered.

The procedure lasted for about 3.5 h. At the end of the surgery, the patient was fully awake and communicating and shifted to the intensive care unit for further observation. The patient received 1500 mL of normal saline and 500 mL of Ringer's lactate solution intra-operatively and the patient's haemodynamic parameters were maintained within normal limits throughout the procedure. The post-operative course was uneventful. Patient's memory and recognition ability were intact, with no speech defect, and he was discharged after 7 days.

DISCUSSION

During awake craniotomy, adequate anaesthesia and analgesia should be provided such that the patient does not feel the pain or discomfort of the surgical procedure. At the same time, he/she should be easily arousable in order to understand and obey the anaesthesiologist's commands. One should carefully balance the anaesthetic depth in order to avoid untoward incidents such as airway obstruction, respiratory depression, hypercarbia, coughing, hypotension, etc.[1,2]

Two standard techniques are practised to provide anaesthesia for awake craniotomy. One is the Asleep–Awake–Asleep (AAA) technique and the other is the Monitored Anaesthesia Care (MAC) technique, which was used in our patient.

We pre-medicated our patient with fentanyl as an analgesic, glycopyrolate as an antisialagogue and ondansetron as an antiemetic agent. Propofol is widely used for awake craniotomy because of its easily titratable sedative effect and rapid recovery with clear-headedness.[1,3]

Dexmedetomidine, a highly selective α_2 adrenoceptor agonist is the pharmacologically active dextroisomer of medetomidine.[4] Stimulation of α_2 adrenceptors by dexemedetomidine in pontine locus coeruleus (LC) results in decreased firing of LC neurons secondary to their hyperpolarisation. This appears to be the key factor in initiating the anaesthetic mechanism of dexmedetomidine. Dexmedetomidine has minimal effects on ventilation and does not cause respiratory depression even at very high infusion rates. Dexmedetomidine does not have a significant effect on intracranial pressure.[5]

Mack *et al.*[2] conducted a retrospective review of 10 cases who had received dexmedetomidine for awake craniotomy. All patients received a loading dose of dexmedetomidine of 0.5–1 mcg/kg over 20 min and infusion at the rate of 0.01–1 mcg/kg/h. All the patients underwent extensive neurocognitive testing. They concluded that dexmedetomidine is a useful sedative for awake craniotomy when neurocognitive testing is required.

Doyle *et al.*[6] reported a case series of 17 patients undergoing awake craniotomy. They administered a loading dose of dexmedetomidine (0.5 mcg/kg over 20 min) followed by infusion at the rate of 0.2 mcg/kg/h. All patients tolerated the procedure well and they concluded that dexmedetomidine is a good adjuvant to perform anaesthesia for awake craniotomy with high degree of safety and few side-effects.

Doyle *et al.*[7] reported a case of awake craniotomy managed using dexmedetomidine infusion. Dexmedetomidine was administered as a loading dose of 1 mcg/kg over 30 min and as infusion at the rate of 0.4 mcg/kg/h. They concluded that dexmedetomidine is an useful adjuvant for awake tumour resection and it provides good haemodynamic stability.

In our patient, with an infusion of dexmedetomidine 1–0.5 mcg/kg/h and propofol 60 mg/h, we could achieve adequate sedation and analgesia during craniotomy. For cognitive testing, the patient was fully co-operative and could perform all the tests successfully once the infusions were stopped 10 min prior.

We conclude that dexmedetomidine is a useful adjuvant during awake craniotomy for tumour resection.

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