

Severe Hypertension with Dexmedetomidine Infusion during Awake Craniotomy

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A 50-year-old male weighing 70 kg was scheduled for awake craniotomy and excision of left temporal glioma under monitored anesthesia care. During the preoperative period, the heart rate (HR) was 67 beats/min (bpm) and blood pressure (BP) 132/68 mm Hg. Airway examination showed Mallampati Grade II. All the preoperative investigations including electrocardiogram (ECG) and chest X-ray were within normal limits. On the day of surgery, standard monitors such as ECG, non-invasive BP, pulse oximeter and oxygen nasal cannula with end-tidal CO₂ monitoring were connected. An arterial cannula was inserted in the left radial artery under local anesthesia. Loading dose of dexmedetomidine infusion 1 µg/kg over 10 min was started. Three minutes later, the BP started increasing along with simultaneous decrease in HR; the maximum effect occurred at 7 min (BP up to 211/96 mm Hg, mean BP 134 mm Hg, and HR up to 37 bpm). However, it was not associated with changes in ST-T wave in ECG. The patient was conscious without any complaints of chest pain or headache; respiratory rate and SpO₂ were 16 breaths/min and 98%, respectively. The loading dose of dexmedetomidine was stopped at 5 min and a maintenance infusion 0.5 µg/kg/h was continued. The hemodynamic parameters came back close to normal baseline values within 4 min and remained stable at 124/66-140/74 mm Hg and 50-55 bpm. Regional scalp block was given with 0.25% bupivacaine bilaterally, and surgery was carried out uneventfully. The hemodynamic parameters remained stable during the postoperative period.

Here, we present a case of severe hypertension during dexmedetomidine loading infusion in a patient undergoing awake craniotomy. The anesthesia technique for awake craniotomy should provide adequate sedation, analgesia and ensure respiratory and hemodynamic stability. A safe and acceptable analgesic/amnesic state for these procedures can be provided by the use of dexmedetomidine, with or without the addition of an opioid.¹ Dexmedetomidine is a highly selective α₂-agonist which has dose-dependent sedative, anxiolytic and analgesic effects without respiratory depression. In addition, sympatholytic and antinociceptive properties allow for hemodynamic stability at critical moments of neurosurgical stimulation.² The common side effects include hypotension, bradycardia,

worsening of heart block and nausea. Dexmedetomidine may evoke a biphasic response to BP; a short hypertensive phase followed by subsequent hypotension. An initial transient rise with a reflex fall in HR is brought about by stimulation of α₂B subtypes of receptors present in vascular smooth muscles. This is followed by fall in BP and HR due to inhibition of central sympathetic outflow and stimulation of presynaptic α₂A receptors. Hypertension associated with dexmedetomidine is usually mild and transient.³ In this patient, 7 min after initiation of the loading dose, the systolic BP increased to more than 200 mm Hg. The BP was normalized, after reduction of the infusion dose. The hypertensive response is a normal pharmacodynamic process and dose-dependent effect of dexmedetomidine. Hence, the side effects could be curtailed by reducing the infusion dose. There are other reports in literature of hypertensive urgencies after loading dose of dexmedetomidine requiring administration of antihypertensive medications.^{4,5} This could possibly be due to rapid distribution phase of dexmedetomidine, which has a distribution half-life of about 6 min and a terminal elimination half-life of 2 h.⁵

To conclude, episodes of severe hypertension must be kept in mind during bolus infusion of dexmedetomidine apart from the common side effects such as hypotension and bradycardia. The hypertensive response can be normalized by reducing the loading dose or discontinuation of drug with or without antihypertensive medications.

References

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