Recurrent bradycardia and asystole in a patient undergoing supratentorial tumor resection: Different types of trigeminal cardiac reflex in same patients

Sir,

Bradycardia is commonly reported complication in neurosurgical patients due to multi-factorial causes.[1] However, multiple episodes of bradycardia due to different causes in a same patient are rarely reported. Here, we have reported such a case and its possible mechanism.

A 47-year-old patient was admitted to our hospital with complaints of tingling sensation in right middle finger and deterioration of vision on right eye over a few months. Magnetic resonance imaging of brain revealed right side sphenoid ridge meningioma with brain edema and mass effect on right optic nerve. The patient was otherwise healthy and scheduled for right pterional craniotomy with tumor debulking under general anesthesia. Laboratory investigations were with in normal limits. Routine monitors were attached. Right radial artery was cannulated under local anesthesia. The patient was induced with remifentanil 1 mcg/kg, propofol 2-3 mcg/kg, and rocuronium 1 mcg/kg and trachea was intubated with portex endotracheal tube no 7. Anesthesia was maintained with propofol infusion 75-100 mcg/kg/min, remifentanil infusion 0.08-0.1 mcg/kg/min, and sevoflurane in oxygen and air mixture with constant end tidal concentration of 1-1.5. Normothermia was maintained. To minimize cerebral edema, dexamethasone 10 mg and mannitol 30 g were administered intravenously. The patient also received 1 g phenytoin intravenously over 1 h. At the time of tumor resection, sudden bradycardia (HR<32 bpm) was observed and the surgeon was notified immediately. He stopped the resection and heart rate reverted back to normal (HR=64 bpm). The surgeon was requested to continue with the resection. Bradycardia (HR<45 bpm) was again noticed. There were five episodes of such fluctuations in heart rate for approximately 5 min [Figure 1]. However, interestingly blood pressure was remained stable during these episodes of bradycardia; therefore, we did not give any anticholinergics medication. Urgent arterial blood gas analysis revealed no abnormality. The rest of the course of

tumor resection was uneventful. After the dural closure, propofol infusion was stopped. At the time of last suture, when the surgeon was approximating the skin edges with the forceps, patient developed sudden bradycardia (30 bpm) followed by asystole. The surgeon was asked to release the traction. Asystole lasted for 20 s and bradycardia for at least a minute. In this episode, interestingly, blood pressure was increased significantly following bradycardia [Figure 2]. Hemodynamics came back to normal within 3-5 min. After ascertaining neurological functions intact, trachea was extubated and patient was shifted to post-anesthesia recovery unit for further observation. Rest of the post-operative course was also uneventful.

The majority of bradycardic episodes in neurosurgical cases have been attributed to Cushing reflex (raised intracranial pressure (ICP), brain stem manipulation, hypothalamic stimulation, and trigeminocardiac reflex.[1] In our patient, bradycardic episodes during tumor resection can be attributable to Trigeminal Cardiac Reflex (TCR). There could be a possibility of stimulation of maxillary division

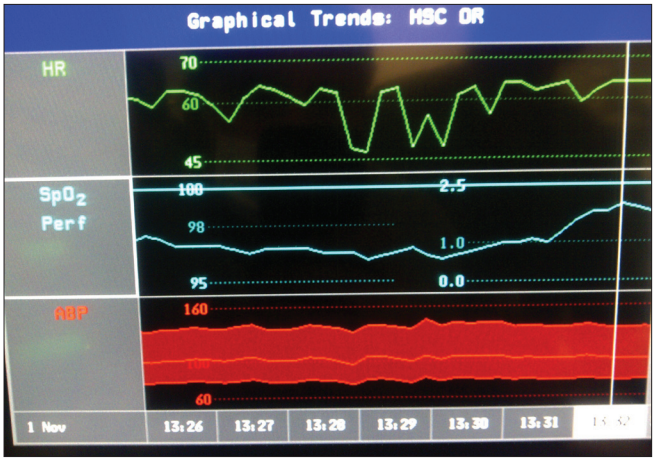

Figure 1: Hemodynamic changes during tumor dissection

Figure 2: Hemodynamic change (asystole) during skin closure

of trigeminal nerve stimulation during tumor manipulation as this tumor had extended up to cavernous sinus. The bradycardia and asystole event at skin closure could be seizure, raised ICP or TCR. The possibility of seizure was unlikely as this patient had received a loading dose of antiepileptic and had no further episodes post-operatively. The sudden increase in ICP in such cases could be due to subdural or epidural bleed; however, there was a subgaleal drain in this patient that was connected to low pressure suction during asystole/bradycardia episode and more so, the patient had also intact neurological functions at the end of procedure. Surprisingly, first event of bradycardia was associated with no change in blood pressure; however, second episode was presented with hypertension. The classical presentation of TCR is described as bradycardia, asystole, hypotension, and gastric hyper motility.[2] The exact mechanism is still unclear but there exist different subtypes of TCR including central (proximal) and peripheral (distal) reflex. In central TCR, there is stimulation of trigeminal nerve anywhere along Gasserian ganglion to brain stem (first event). The peripheral TCR is produced due to stimulation of trigeminal outside the cranium (second event). This reflex is usually provoked by hypoxia, hypercarbia, acidosis, and light plane of anesthesia. The peripheral TCR is further divided into oculocardiac (OCR) and maxillomandibular cardiac reflex. The second event was related to OCR. The OCR can sometimes present with bradycardia with hypertension as opposed to central TCR which usually present as bradycardia with hypotension.[2] In this patient, different sympathetic outflow response could be due to different depths of anesthesia.[3] In the first event, the patient was in deep plane of anesthesia; however, there was lightening plane of anesthesia during the second event. Thus, different depth of anesthesia coupled with different forms of TCR stimulation probably contributed to different hemodynamic responses in the same patient and none of the stimulation could elicit as classical TCR.

In conclusion, different forms of TCR can coexist in the same patient and can produce catastrophic consequences if not carefully observed throughout the surgical procedure. There is also possibility of depth of anesthesia related variable presentations of this reflex and require further confirmation.

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