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TRIGEMINOCARDIAC REFLEX INDUCED BRADYCARDIA DURING DURAL CLOSURE: A CASE REPORT

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ABSTRACT The trigeminocardiac reflex (TCR) is a brainstem reflex characterized by sudden onset of hemodynamic perturbations including bradycardia, hypotension, apnea, and gastric hypermotility during stimulation of any branch of the trigeminal nerve. This case report describes an unusual presentation of TCR occurring during dural closure in a 55-year-old male undergoing elective craniotomy for a left frontotemporal lesion. The patient experienced significant bradycardia that resolved following cessation of manipulation and administration of atropine. This report highlights the importance of recognizing TCR during various stages of neurosurgical procedures, particularly during dural closure, which is a less commonly reported trigger point. Early recognition and appropriate management of TCR are crucial for preventing potentially life-threatening complications during neurosurgical procedures.

KEYWORDS : Trigeminocardiac Reflex; Dural closure; Bradycardia; Craniotomy; Neurosurgery; Atropine

INTRODUCTION

The trigeminocardiac reflex (TCR) is a well-recognized brainstem reflex characterized by the sudden onset of hemodynamic changes including bradycardia, hypotension, apnea, and gastric hypermotility upon stimulation of any branch of the trigeminal nerve[1,2]. First described by Schaller et al. in 1999, TCR is considered a subtype of the diving reflex[3]. The afferent pathway involves the sensory nucleus of the trigeminal nerve, and the efferent pathway is carried by the vagus nerve, resulting in parasympathetic stimulation[4].

Although TCR is commonly reported during direct manipulation of the trigeminal nerve or structures it innervates, its occurrence during dural closure is less frequently reported[5,8]. The incidence of TCR during neurosurgical procedures ranges from 8% to 18%, with higher rates in procedures involving direct trigeminal nerve manipulation[6,7]. This case report describes an unusual presentation of TCR manifesting as significant bradycardia during dural closure in a patient undergoing craniotomy for a left frontotemporal lesion. This report aims to highlight the importance of recognizing and appropriately managing TCR during this critical phase of neurosurgical procedures.

Case Report

A 55-year-old male presented to the neurosurgery outpatient department with complaints of intermittent headaches and progressive speech difficulties over the past three months. The patient had a medical history significant for controlled hypertension (on amlodipine 5mg daily) and type 2 diabetes mellitus (on metformin 500mg twice daily). He had no known drug allergies, no history of cardiac disease, and no previous surgical history. The patient was a non-smoker and reported occasional alcohol consumption. Family history was non-contributory.

On examination, the patient was alert and oriented with a Glasgow Coma Scale score of 15/15. His vital signs were within normal limits: blood pressure 132/78 mmHg, heart rate 74 beats per minute, respiratory rate 16 breaths per minute, and temperature 36.7°C. Neurological examination revealed mild expressive aphasia and right upper limb weakness (power 4/5). The remainder of the neurological examination was unremarkable, with intact cranial nerve function, normal sensory examination, and no cerebellar signs.

Magnetic resonance imaging (MRI) of the brain revealed a 3.5 × 4.0 cm heterogeneously enhancing mass in the left frontotemporal region with surrounding edema, suggestive of a high-grade glioma. Laboratory investigations, including complete blood count, coagulation profile, liver and renal function tests, and serum electrolytes, were all within normal limits. Electrocardiogram showed normal sinus rhythm, and chest X-ray was unremarkable. Preoperative cardiac evaluation was normal with an ejection fraction of 60%.

The patient was scheduled for an elective left frontotemporal craniotomy and tumor excision. After obtaining informed consent, the patient was taken to the operating room. Standard ASA monitors were applied, and anesthesia was induced with propofol (2 mg/kg), fentanyl (2 µg/kg), and vecuronium (0.1 mg/kg) for muscle relaxation. Anesthesia was maintained with sevoflurane (MAC 0.8-1.0) in an oxygen-air mixture and continuous infusion of remifentanil (0.1-0.2 µg/kg/min). The patient was positioned supine with the head turned 30° to the right and fixed in a Mayfield head clamp.

A left frontotemporal craniotomy was performed, and the dura was opened in a cruciate fashion. Tumor excision proceeded uneventfully, with hemodynamic parameters remaining stable throughout the resection phase. The estimated blood loss was 350 ml, and the patient received 2000 ml of crystalloid fluids during the procedure.

During dural closure, approximately 4 hours into the surgery, the patient developed sudden, severe bradycardia with a heart rate dropping from 68 beats per minute to 35 beats per minute within a few seconds. Blood pressure concurrently decreased from 126/74 mmHg to 92/56 mmHg. Oxygen saturation remained stable at 99%, and end-tidal CO2 was 35 mmHg.

The surgeon was immediately notified, and all surgical manipulation was temporarily halted. With cessation of dural manipulation, the heart rate gradually increased to 45 beats per minute. However, as the surgeon resumed dural closure, bradycardia recurred, with the heart rate dropping to 32 beats per minute. Atropine 0.5 mg was administered intravenously, resulting in prompt normalization of the heart rate to 78 beats per minute. The dural closure was then completed without further bradycardic episodes. The bone flap was replaced, and the scalp was closed in layers.

Following the procedure, the patient was transferred to the neurosurgical intensive care unit for close monitoring. Postoperative electrocardiogram showed normal sinus rhythm, and cardiac enzymes were within normal limits, ruling out any myocardial injury. Neurological assessment revealed no new deficits compared to the preoperative status. The patient's immediate postoperative course was uneventful, with stable hemodynamic parameters and no recurrence of bradycardia.

The patient was discharged on the seventh postoperative day with improvement in his preoperative symptoms. Histopathological examination of the resected tumor confirmed the diagnosis of a grade III anaplastic astrocytoma. The patient was referred to the oncology department for adjuvant chemoradiotherapy. At the one-month follow-up, the patient's neurological status had improved, with no recurrence of bradycardia or other cardiac events. The patient is currently undergoing adjuvant therapy and is being followed closely by both neurosurgery and oncology teams.

DISCUSSION

This case illustrates an uncommon presentation of the trigemino-cardiac reflex occurring specifically during dural closure in a patient undergoing craniotomy for a left frontotemporal tumor. While TCR is well-documented during various neurosurgical procedures, its manifestation during dural closure is less frequently reported and may be overlooked as a potential cause of intraoperative bradycardia[9].

The dura mater is richly innervated by sensory branches from the trigeminal nerve, specifically from the meningeal branches of the

ophthalmic (V1) and maxillary (V2) divisions[10]. Manipulation, stretching, or traction of the dura can stimulate these nerve fibers, triggering the trigeminocardiac reflex arc. In our case, the traction applied to the dura during closure likely stimulated the trigeminal afferents, initiating the reflex.

The clinical manifestation of TCR can range from mild bradycardia to severe hemodynamic instability, asystole, and cardiac arrest[11]. In our patient, the reflex manifested as severe bradycardia with moderate hypotension. The temporal relationship between dural manipulation and bradycardia, along with recurrence upon resumption of manipulation, strongly supports the diagnosis of TCR.

TCR should be distinguished from other causes of intraoperative bradycardia such as the Cushing's reflex, which is typically associated with elevated intracranial pressure, or hypoxia, which presents with systemic desaturation. In contrast, TCR is characterized by a direct temporal relationship between trigeminal nerve stimulation and sudden bradycardia.

Management of TCR follows a stepwise approach[12]. The first step is immediate cessation of the surgical stimulus, which in many cases leads to spontaneous resolution of the hemodynamic changes. If the bradycardia persists or recurs upon resumption of the surgical manipulation, as observed in our case, anticholinergic agents such as atropine (0.5-1 mg) or glycopyrrolate (0.2-0.4 mg) are indicated[13]. For refractory cases, epinephrine or cardiac pacing may be necessary[14].

Several risk factors for TCR have been identified, including young age, light plane of anesthesia, hypercapnia, hypoxemia, and pre-existing cardiac disease[15]. Our patient did not have apparent risk factors apart from manipulation of the trigeminal nerve territory. Preventive measures include maintaining adequate depth of anesthesia, normocapnia, and infiltration of the surgical site with local anesthetics to block the afferent pathway[16].

While TCR during direct trigeminal nerve manipulation is well-documented, its occurrence during dural closure is less commonly reported. This case highlights the importance of considering TCR in the differential diagnosis of sudden bradycardia during any phase of cranial neurosurgery, including dural closure. Surgeons and anesthesiologists should be aware of this possibility and be prepared to manage it promptly to prevent potentially life-threatening complications.

CONCLUSION

This case demonstrates the importance of recognizing the trigeminocardiac reflex as a potential cause of bradycardia during dural closure in neurosurgical procedures. Prompt recognition and appropriate management, including cessation of the surgical stimulus and administration of anticholinergic agents when necessary, are essential to prevent adverse outcomes. Neurosurgeons and anesthesiologists should maintain a high index of suspicion for TCR during all phases of cranial procedures, including dural closure, to ensure patient safety. Future neurosurgical protocols may consider incorporating prophylactic strategies, such as deepening anesthesia or using anticholinergics in high-risk patients, to minimize TCR episodes.

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