Case Report

A near catastrophe from trigeminocardiac reflex

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Abstract

Trigeminocardiac reflex is a brainstem reflex that results from stimulation of any branch of the trigeminal nerve along its course. It produces a constellation of signs and symptoms decrease in blood pressure (BP) and heart rate, dysrhythmias, apnoea and increased gastric motility. We present a case of 80-year-old female patient who developed alarming hypotension and bradycardia during craniotomy for meningioma excision resulting from this reflex. In the face of refractory hypotension despite administering ephedrine and phenylephrine, we had to resort to adrenaline to restore her normal BP.

Key words: Haemodynamics, meningioma excision, trigeminocardiac reflex

INTRODUCTION

Trigeminocardiac reflex (TCR) is a brainstem reflex that manifests by sudden decrease in blood pressure (BP) and heart rate (HR) along with apnoea and increased gastric motility. It may result in cardiac dysrhythmias and even asystole.[1] It is seen consequent to stimulation of any branch of trigeminal nerve.[2,3] It has been described during intracranial surgery,[4] and even during radiofrequency lesioning of Gasserian ganglion.[3,5] It requires quick recognition and intervention to forestall any serious complication. Here, we describe a near catastrophe resulting from TCR in a patient undergoing craniotomy for meningioma excision.

CASE REPORT

An 87-year-old female weighing 75 kg, presented to the neurosurgery clinic with the complaints of headache, moderately impaired memory, incontinence of urine and difficulty in walking. She was a known case of essential hypertension and diabetes mellitus, controlled with amlodipine and oral hypoglycaemic agent, respectively. Magnetic resonance image revealed a large bifrontal meningioma. The patient was admitted to the hospital for excision of the tumour. On examination, she was fully conscious and oriented. Her BP and blood glucose were within normal range. Echocardiography revealed a well-preserved heart with ejection fraction of 60%. Her all other investigations, including electrocardiography (ECG) were normal. On the day of surgery, her antihypertensive medication was continued, but hypoglycaemic therapy was withheld. Her morning blood glucose was 140 mg/dl and BP was 156/94 mmHg. Anaesthesia was induced with xylocaine, fentanyl, propofol and tracheal intubation was facilitated with rocuronium. Her radial artery and right internal jugular vein were cannulated. Anaesthesia was maintained on 50% oxygen in air and sevoflurane (0.8–1.0 minimum alveolar concentration). Lungs were mechanically ventilated to maintain an end-tidal carbon dioxide of 32–34 mmHg. Infusions of remifentanil (0.5–0.8 mcg/kg/min) and rocuronium (5 mcg/kg/min) were initiated. During skin flap elevation, her systolic BP, which hitherto was 160 mmHg, decrease suddenly to 50 mmHg along with decrease in HR from 70 to 45 beats/min. Simultaneously, there was drop in end-tidal carbon dioxide from 32 mmHg to 15 mmHg.

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The ECG rhythm was sinus with no signs of myocardial ischaemia. An initial diagnosis of massive venous air embolism (VAE) was made, and surgeon instructed to take steps to prevent further entrainment of air into the circulation. Immediately, two boluses of ephedrine, 5 mg each, were administered, and aspiration of air from central venous line was attempted, with no success. An arterial blood sample was drawn for analysis. HR though improved slightly following ephedrine administration, but BP remained unresponsive. It did not increase even following a bolus of 100 mcg of phenylephrine. Finally, to manage this resistant hypotension patient was given adrenaline in two boluses of 50 mcg each. This resulted in increase of BP to 120 mmHg. An arterial blood sample analysed at this point of time was normal except slight retention of carbon dioxide but without any decrease in oxygen tension. This ruled out air embolism as the cause of hypotension. Thus, a diagnosis of TCR was made. Subsequent surgical course was uneventful. The surgery lasted for 8 h. Post-operatively, she was electively ventilated in neurointensive care ward, and was investigated for any coronary event. However, her cardiac enzymes were normal and therefore, her mechanical ventilation was discontinued and she was extubated 4 h later. Her subsequent stay in the hospital was unremarkable and she was discharged to home after 2 weeks.

DISCUSSION

The TCR is a brainstem reflex that results in haemodynamic alterations including, sudden decrease in HR and BP, or cardiac arrhythmias, even asystole, apnoea and gastric hypermotility.

Shelly and Church[6] coined the term TCR, and Schaller *et al*. first described the central component of the TCR in humans and pioneered the definition/concept of the TCR in humans.[7] Subsequent to the pioneering work of Schaller *et al*. in 1999,[7] this unique reflex has gained a lot of interest in neurosurgery. In addition, Schaller *et al*. further explored the possibilities of different clinical variants of the TCR[8,9] and presented the generally accepted classification of peripheral and central subtypes of the TCR.[10] It has been demonstrated that the TCR may occur with manipulation of all the branches of the trigeminal nerve anywhere along its (intracranial or extracranial) course. According to Schaller, the TCR occurs in 10%–18% of the patients. The incidence of TCR is as high as 18% in patients undergoing microvascular decompression of the trigeminal nerve for trigeminal neuralgia.[11] It has also been reported in transsphenoidal pituitary adenoma excision.[12] In recent times, literature on the TCR has provided substantial evidence on the different subtypes of TCR. Of these, the three main important subtypes include peripheral, central and Gasserian ganglion-type TCR.[13] As haemodynamic changes have different presentations in different subtypes, it is not feasible to use a definition based on the smaller changes of HR/mean arterial BP only, the unique cause–effect relationship based criteria for TCR episode has been highlighted. According to this, they have focussed on four major domains including plausibility, reversibility, repetition, and prevention. Further to this, these have been divided into two major (plausibility and reversibility) and 2 minor criteria (repetition and prevention). For defining TCR, there should be at least 2 major criteria. Minor criteria may or may not be present.[13]

In our patient, sudden decrease in BP and HR were accompanied with sudden fall in end-tidal carbon dioxide giving rise to suspicion of massive VAE. However, the arterial blood carbon tension did not show any increase and it corresponded to the end-tidal carbon dioxide tension. In VAE, a decrease in end-tidal carbon dioxide tension is associated with rise in arterial blood carbon dioxide tension because of the inability of the lungs to excrete it due to ventilation-perfusion mismatch. Furthermore, VAE of such magnitude would usually result in some degree of decrease in arterial blood oxygen tension, which was not the case in our patient. Thinking it to be VAE episode we did not administer vagolytic agent. Moreover, atropine may fail in terminating the episode.[14] Therefore, fearing the worst case scenario, we administered ephedrine followed by phenylephrine initially. We subsequently administered adrenaline due to the failure of these vasoactive drugs to restore BP. We hypothesise that failure of ephedrine as well as phenylephrine to restore normotension may be due to hitherto unknown another variant of TCR.

We did not suspect any coronary event because ECG rhythm remained unchanged and was sinus throughout the episode and even after resolution of the episode. There were no ST segment disturbances. Moreover, subsequent to adrenaline administration the patient remained haemodynamically stable for the entire duration of the surgical procedure. Furthermore, her cardiac enzymes tests were normal in the post-operative period.

The possible mechanism of TCR lies in that the stimulation of any sensory branch of the trigeminal nerve, results in the signal transmission to the sensory nucleus of the trigeminal nerve through the Gasserian ganglion. This afferent pathway continues along the short internuncial nerve fibres in the reticular formation to connect with the efferent pathway in the motor nucleus of the vagus nerve. This terminates in the cardiac ganglia from which the post-ganglionic fibres are sent to the conduction system, leading to

autonomic changes that usually manifest as negative chronotropy.[15,16] The mechanism of TCR involves both sympathetic and parasympathetic pathways. The trigeminal depressor response includes both activation of vagal inhibitory fibres and inhibition of adrenergic vasoconstriction. Therefore, the use of vagolytic atropine is not the only treatment modality. TCR that is refractory to vagolytic (atropine or glycopyrrolate) may need to be managed with adrenaline.

Various factors which may give rise to TCR include high vagal tone, light plane of anaesthesia, use of opioids, beta-blockers or calcium channel blockers, dural stimulation, hypoxia, hypercarbia and acidosis. In our patients, only risk factor was history of amlodipine use, a calcium channel blocker, prescribed for hypertension. Recent experiments have suggested that exposure to nicotine may be another risk factor.

The TCR in the majority of the reported literature seems a mild manifestation that is usually transient in nature.[17] However, our case as well as previously cited reports emphasise that it may be a potentially serious phenomenon.[1,18] Therefore, a quick recognition and prompt management is must to prevent any potentially serious consequence of this innocuous looking reflex.

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Conflicts of interest

There are no conflicts of interest.

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