**Ganglionic trigeminocardiac reflex: A less known presentation of a well-known entity**

Sir,

The trigeminocardiac reflex (TCR) is portrayed as one of the cardinal causes of cardiac catastrophes encountered in neurosurgical patients.[1] This brainstem reflex triggered by manipulation of the trigeminal nerve manifests as acute haemodynamic perturbations in neurosurgical patients.[2] The literature is replete with evolving definitions and exhaustive descriptions about this peculiar neurocardiac kinship in various neurological settings.[3] We report a case of TCR seen during transorbital intracranial foreign body removal which presented as tachycardia and hypotension which is an unconventional presentation of TCR. Although TCR has been reported multiple times in the literature, clinical presentation in keeping with ganglionic TCR is not known in literature.

A 40-year-old male sustained an injury to right eye by iron rod while cutting rods in a factory. He presented with a history of vomiting, ear and nasal bleed after the injury with no history of seizures or loss of consciousness. He was conscious, oriented with a Glasgow Coma Scale (GCS) of E₄ V₅ M₆ and was vitally stable with unremarkable general physical examination. On local examination, there was an iron rod penetrating the right eye with a ruptured globe. Plain radiograph [Figure 1] and non-contrast computed tomography (NCCT) scan of head suggested metallic foreign body penetrating right orbit and orbital apex extending intracranially into sellar and suprasellar region.

A bifrontal craniotomy was planned to explore and remove the foreign body. The patient had stable vitals throughout the procedure but while extracting the foreign body transcranially, he had an episode of sudden severe hypotension [Invasive blood pressure (BP) 50/20 mm Hg] and tachycardia [Heart rate (HR) 120/min]. Surgeons at this time were manipulating and trying to remove the foreign body. They were informed to stop which led to the normalisation of BP and HR over a period of 20–30 s. As the surgeons manipulated the foreign body again, the patient had another similar episode of increased severity of tachycardia and hypotension [Invasive BP 40/20 mmHg, HR 150/min], which too resolved on ceasing the stimulus. Finally, the foreign body was successfully drawn outside transorbitally with no untoward haemodynamic disturbance.

During transcranial manipulation, the surgeons could have stimulated the trigeminal nerve at the gasserian ganglion which lead to the above event. Our case describes a rare and unique presentation of TCR which is described in literature but is seldom encountered. We ruled out variable causes of hypotension with tachycardia, that is, sudden massive blood loss from surgical field, dehydration, diuresis, sepsis, drug error, anaphylaxis, etc. Peak pressures were normal and bilateral air entry was equal excluding tension pneumothorax or pulmonary embolism as the cause of this haemodynamic instability.

Tumul et al. elucidated “plausibility” and “reversibility” as two major and “repetition” and “prevention” as two minor criteria to characterise TCR. A likely TCR event must satisfy these two main criteria, but not all criteria are required to be fulfilled in every case.[3] Plausibility entails the event to occur as a direct result of physical or chemical stimulation of the trigeminal nerve or its peripheral branches. In the present case, haemodynamic instability occurred as a direct consequence of transcranial manipulation of foreign body. Reversibility means abolition of TCR event on cessation of inducing stimulus. In our case, suspension of stimulus did improve the haemodynamics. Repetition and prevention are minor criteria.

Depending on the location of trigger point, TCR is classically divided into three subtypes: the central/proximal subtype which has an intracranial trigger point proximal to the Gasserian ganglion; the peripheral/distal subtype, due to stimulation of the extra-cranial course of the trigeminal nerve; and
the Gasserian ganglion subtypes.[2] Different subtypes have different haemodynamic presentations. TCR that is triggered at the Gasserian ganglion is an altogether distinct entity.

All variants of the TCR cause deceleration of HR barring the Gasserian ganglion type which has an atypical presentation where any combination of brady- or tachycardia, hyper- or hypotension and apnoea or hyperpnoea can occur, depending upon the stimulus.[4] This offbeat presentation is apparently because harmony between parasympathetic and sympathetic influences varies between the patients.[3]

We suggest that if the TCR is initiated at the Gasserian ganglion, it reacts in a different way from the better-known central or peripheral TCR. Cognisance about this rare entity allows us to anticipate, detect, and treat it adequately during neurosurgical cases.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
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