Ventricular Fibrillation as an Uncommon Manifestation of Trigeminocardiac Reflex during Simultaneous Surgery for Aneurysmal Clipping and Trigeminal Schwannoma Resection

Kumari Pallavi1 – Amit Goyal1 – Sriganesh Kamath2

1 Department of Neuroanesthesia and Neurocritical Care, Medanta-The Medicity, Gurgaon, Haryana, India
2 Department of Neuroanesthesia and Neurocritical Care, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India

Address for correspondence: Sriganesh Kamath, DM, Department of Neuroanesthesia and Neurocritical Care, National Institute of Mental Health and Neurosciences, Bengaluru 560029, Karnataka, India (e-mail: drsri23@rediffmail.com).

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Trigeminocardiac reflex (TCR) from stimulation of sensory branches of trigeminal nerve during neurosurgeries may produce various types of arrhythmias, including bradycardia and asystole. Rarely, TCR can present with unusual manifestations such as tachyarrhythmias secondary to sympathetic stimulation. Ventricular tachycardia (VT) and fibrillation (VF) are rare in neurosurgical patients without preexisting cardiac etiology. We report such presentation during simultaneous surgery of cerebral aneurysmal clipping and trigeminal schwannoma excision that was managed successfully.

A 52-year-old female patient with a history of hypertension presented with headache and one episode of loss of consciousness. Her admission Glasgow Coma Scale score was E4V5M6. On radiological evaluation, she was diagnosed with subarachnoid hemorrhage (aneurysmal) from ruptured anterior communicating artery aneurysm measuring 1.1 × 0.8 cm and coexisting trigeminal schwannoma measuring 4.3 × 4.1 cm (►Fig. 1A). She was scheduled for craniotomy to treat both pathologies simultaneously. Her blood investigations were normal. Electrocardiogram (ECG) showed left axis deviation, ST depression in II, aVF and V3 to V5 leads and two-dimensional-echocardiogram revealed concentric left ventricular hypertrophy with apical wall motion abnormality, ejection fraction of 45%, and mean pulmonary arterial pressure of 43 mm Hg. Anesthesia was induced with fentanyl 120 µg, propofol 100 mg, preservative-free lidocaine intravenous 60 mg, and tracheal intubation with 7.5 mmID tube was facilitated with vecuronium 5 mg. Anesthesia was maintained with oxygen/air/sevoflurane between 0.7 and 1.1 minimum alveolar concentration and intermittent fentanyl and vecuronium. Scalp block was performed before skull-pin application. Apart from standard monitoring, cardiac output was monitored using FloTrac/Vigileo. Aneurysm clipping was performed uneventfully. One hour into schwannoma excision, during tumor decompression, ECG showed sudden VT (►Fig. 1B). Surgeon was alerted and surgical stimulation was stopped. However, VT rapidly progressed to VF leading to absent pulses and unrecordable blood pressure (BP); hence, cardiopulmonary resuscitation was started. Cardiac defibrillation with 150J (biphasic) restored sinus rhythm (heart rate 90/min) and BP to 70/40 mm Hg. As cardiac index was 1.5 L/min/m2, dobutamine (10 µg/kg/min) was started that improved BP to 120/70 mm Hg and cardiac index to 4 L/min/m2. Surgery was completed uneventfully in next 90 minutes. Dobutamine was tapered and stopped at the end of surgery and trachea extubated. Patient was monitored in the neurointensive care unit and discharged from the hospital after 7 days in good neurological status and no new cardiac event or fresh changes in ECG or echocardiogram. In view of the transient and resolved intraoperative event, cardiologist could not conclude definitive etiology for the event postoperatively and did not recommend initiating any new treatment other than follow-up after discharge.

Arrhythmias are common in patients with neurosurgical pathologies such as aSAH,\textsuperscript{1} traumatic brain injury,\textsuperscript{2} and posterior fossa lesion,\textsuperscript{3} but VT/VF is uncommon. TCR in our patient probably occurred from surgical stimulation of trigeminal nerve as this event occurred during decompression of schwannoma and not during clipping. TCR has a complex neurophysiological mechanism with sensory interaction at Gasserian ganglion and various brainstem nuclei. It is associated with stimulation of parasympathetic (more prominent) and sympathetic (less prominent) pathways. TCR-induced tachyarrhythmias may occur secondary to coactivation of sympathetic pathway.\textsuperscript{4} Coronary vasospasm due to vagal stimulation initiated by TCR might be another possible explanation for VT/VF.\textsuperscript{5} Hypertension and aSAH-related myocardial injury and alterations in cardiac electrical activity might have contributed to the worsening of surgical stimulus-induced TCR and aggressive manifestation of VT/VF unlike the classical bradycardia/asystole. We attributed the occurrence of VT/VF to TCR as other causes such as drugs (epinephrine, aminophylline, halothane), hypoxia, hypercapnia, hypovolemia, extreme temperatures, dyselectrolytemia, inadequate anesthetic depth or pain, and any apparent cardiac disease that can precipitate arrhythmias were absent in our patient. Cessation of surgical stimulus often terminates arrhythmia, but occasionally cardiac defibrillation may be required, as in our case.

To conclude, life-threatening cardiac arrhythmia, though rare, can occur during neurosurgery involving trigeminal nerve in susceptible individuals. The arrhythmias were likely due to exaggeration of surgical stimulus-induced TCR in the background of aSAH-related cardiac changes. Anesthesiologists should be aware of and be prepared to manage such rare but potentially fatal manifestations of TCR.

\textbf{Conflict of Interest}
None declared.

\textbf{References}
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