Sudden sustained asystole during a cerebellopontine angle surgery

Sir,

Cerebellopontine angle (CPA) surgeries are encountered with numerous potentially life-threatening cardiovascular complications. Cardiovascular complications are usually in the form of arrhythmias with hypertension or hypotension.[1] Asystole without any alerting signs is a very rare presentation during these surgeries.[1,2] We report a rare presentation of sudden sustained asystole without preceding bradycardia during excision of a tumor in the CPA.

A 50-year-old lady, well-controlled hypertensive, was posted for surgical excision of acoustic schwannoma at right CPA. General endotracheal anesthesia with standard monitors including central venous (CV) (right subclavian vein) and intra-arterial (right dorsalis pedis artery) blood pressure monitoring was induced intravenously with fentanyl 200 μg, propofol 150 mg, vecuronium 8 mg, and maintained with morphine 8 mg intravenous, isoflurane 1.0-2.0% in 50% air and oxygen with controlled ventilation. In left lateral position, the tumor was removed with blood loss ≈ 500 ml with no cardiorespiratory events. When the surgeon was removing the last part of the tumor which was close to the brainstem, a sudden drop in heart rate from 72 to 0 beats per minute (bpm) was noticed with flattening of electrocardiogram (ECG), invasive blood pressure, plethysmogram, and capnography waves (pattern of $ETCO_2$ fall: 35 → 10 → 5 → 0 mmHg within 20 s). Immediately the surgical team was informed and manipulations were stopped. Atropine (1.2 mg, intravenous) bolus was administered. Surgical area was flushed with saline, inspiratory oxygen concentration was increased to 100%, and patient's head was lowered below the level of heart. Blood was aspirated from the CV catheter which did not show any air bubbles. When asystole persisted for 40 seconds with above measures, adrenaline (1 mg, intravenous) bolus was administered. Within 5 seconds, sinus rhythm was seen with overshooting of the heart rate to 180 bpm and blood pressure to 196/110 mm Hg with $ETCO_2$ of 60 mm Hg. Within 5 min the heart rate and blood pressure were brought down to 100 bpm and 144/90 mm Hg, respectively with esmolol (30 mg, intravenous) bolus and $ETCO_2$ also became normal (35-40 mm Hg). Arterial blood gas analysis done was within normal limit. Surgery was restarted once patient was hemodynamically stable. Surgery was completed uneventfully without recurrence of such episodes with gentle surgical handling. Trachea was extubated after adequately reversing the neuromuscular blockade. Patient was monitored in intensive care unit (ICU) for 24 h which was uneventful.

The probable causes for the event may have been a massive venous air embolism (VAE) or an intense trigeminocardiac reflex (TCR).

We could not retrieve air bubbles from CV catheter. Although aspiration of air from the CV catheter confirms air embolism, the absence of air in aspirate does not rule it out.

TCR is a well-understood brainstem reflex triggered by electrical, mechanical or chemical stimulation of the trigeminal nerve pathways[3] and characterized by abrupt onset of arrhythmias usually sinus bradycardia terminating in asystole, rarely asystole with no preceding bradycardia[1,2], hypotension, apnea, and gastric hypermotility.[4] In our patient, sudden asystole developed without preceding bradycardia when the surgeon was manipulating near the brainstem. Asystole persisted for ≈ 40 seconds in spite of administration of anticholinergic drugs but responded to adrenaline and did not require chest compressions.

Although the clinical presentation in our patient resembled TCR, VAE could not be ruled out completely.

The preparation of clinicians to foresee, prevent, recognize, and manage any clinical event is possible only when they know about the possibility of such an event. So this report alerts the clinicians about possibility of a sudden sustained asystole without warning signs and also persuades them to be better prepared to handle such episodes.

M C B Santhosh,
Rohini Bhat Pai, Raghavendra P Rao,
Varun Byrappa

Department of Anesthesiology, Shri Dharmasthala Manjunatheshwara College of Medical Sciences and Hospital, Dharwad, Karnataka, India

Address for correspondence: Dr. M C B Santhosh,
Department of Anesthesiology, Shri Dharmasthala Manjunatheshwara
College of Medical Sciences and Hospital, Dharwad,
Karnataka - 580 009, India.
E-mail: mcbsanthu@gmail.com

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