Sir,

Large tongue (macroglossia) of the patients remains an important concern for securing the airway not only for anesthetic induction but also during the postoperative period. We report a case of persistent postoperative macroglossia after posterior fossa surgery in sitting position; the possible causes are discussed. A 7-year-old boy weighing 22 kg was admitted with complaints of generalized tonic–clonic seizure, outward deviation of right eyeball for 1 year, and severe headache for 2 months. A magnetic resonance imaging of brain showed a solid cystic mass in the pineal region extending into posterior third ventricle with hydrocephalus. A ventriculoperitoneal shunt was inserted in view of emergent situation. Two days later, suboccipital craniotomy and excision of the tumor in sitting position was planned under anesthesia. A standard anesthetic technique was followed, which included induction with propofol and fentanyl, facilitation of endotracheal intubation with 5.0 ID cuffed tracheal tube after rocuronium. Anesthesia was maintained with $O_2$, $N_2O$, isoflurane, fentanyl, and rocuronium. The child was monitored for invasive blood pressure and central venous pressure along with the routine monitoring modalities. Rest of the surgical procedure was uneventful. At the end of surgery, the trachea was not extubated in view of severe tongue edema [Figure 1]. The child was sedated with fentanyl and shifted to the intensive care unit for further management. The child was kept in head up position and steroid ointment was applied locally over the tongue to reduce edema, which persisted even after 5 days of surgery. Hence, elective tracheostomy was performed on bedside, after which the edema resolved. Thereafter, the tracheostomy tube was decannulated and the wound site was closed. On 12th postoperative day, the child was discharged with an advice for follow-up.

Macroglossia is a rare complication occurs after neurosurgical procedures carried out in sitting position, although the exact etiology is unclear. It is presumed to be complicated by an extreme neck flexion and a resultant bilateral lingual vein thrombosis, lymphatic obstruction, or arterial compromise. Lingual obstruction may occur after use of airway, endotracheal tube, supraglottis device, esophageal stethoscope, and transesophageal echocardiography. Macroglossia may occur in conditions without neck flexion due to abnormal brain stem signaling. In our patient, soft airway (cotton made) was used and adequate (two fingers) distance between the chin and the sternum was maintained to prevent the consequences of severe neck flexion. It seems, abnormal discharges from brain stem were responsible for the causation of tongue edema with a mechanism similar to the formation of pulmonary edema. Abnormal impulses may be provoked by surgical manipulation at brainstem or tumor infiltration. The management in such a situation remains emergency tracheostomy albeit if the possibility of extreme neck flexion is ruled out.

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Sir,

Larsen syndrome was first described by Dr. Loren J. Larsen in 1950 as a syndrome with multiple joint dislocations, ligamentous laxity, club foot, dysmorphic facies, and depressed nasal bridge.[1] Patients with this syndrome require repeated anesthetics. Because of the various musculoskeletal abnormalities in these patients, they are at increased risk during perioperative period.[2] We present a case of malignant hyperthermia (MH)-like episode in a child with Larsen syndrome undergoing surgery for deformity correction.

A 4-year-old boy weighing 15 kg was scheduled to undergo corrective surgery under general anesthesia. The child had multiple musculoskeletal abnormalities and dysmorphic facies with depressed nasal bridge, suggestive of Larsen syndrome.

After premedication with oral midazolam, anesthesia was induced with sevoflurane in oxygen. He was paralyzed with vecuronium 1.5 mg intravenous (IV). In view of the possibility of cervical spine instability in Larsen syndrome, laryngoscopy and intubation were done with manual inline stabilization. Thirty minutes through surgery, endtidal CO2 (EtCO2) had increased to 55 mmHg. Bilateral equal air entry into lungs was reconfirmed and ventilatory settings were re-adjusted to increase the minute ventilation. The EtCO2 did not decrease with the ventilatory changes and had increased to 60 mmHg in another 15 min. The nasopharyngeal temperature had increased to 39.1°C and the heart rate was 160 beats/min in spite of adequate depth of anesthesia and analgesia. Tepid sponging with cold water was given. In view of the raising temperature, EtCO2 and heart rate with underlying musculoskeletal abnormality,