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Perioperative Laryngospasm – Uncovering the Concealed

Charu Shrotriya*, I. C. Devaraj, N. Kiran Chand, H. Bala Subramanya and D. Srinivasulu

Department of Anaesthesiology, Vijayanagara Institute of Medical Sciences, Bellary, Karnataka, India; shrotriya.charu2190@gmail.com

Sir,

Laryngospasm in the perioperative period is an emergency, which accounts for significant morbidity and mortality, especially in the paediatric age group[1]. There is an increased incidence among patients presenting for emergency surgeries, those requiring nasogastric tubes, patients undergoing tonsillectomy, cervical dilation, hypospadias correction, oral endoscopy, or excision of skin lesions[2]. Once the diagnosis is made, the offending stimulus should be identified and removed, airway manoeuvres to open the airway may be attempted, and general anaesthesia may need to be administered to relieve the obstruction.

We report a case of hypocalcemia induced post-operative laryngospasm following adeno-tonsillectomy. A 9 year old male weighing 20 kg was posted electively for adenotonsillectomy. History was unremarkable and preoperative examination revealed grade II tonsillar enlargement. Other physical examinations and routine investigation were unremarkable. Patient was taken to OT, standard monitors and iv fluid were established. Patient was induced with Inj propofol 50 mg, Inj fentanyl 40 mcg and Inj vecuronium 2 mg IV and intubated nasally with 5.0 mm cuffed ETT. Anaesthesia was maintained with halothane and inj vecuronium 0.5 mg intermittently. Intraoperative period was uneventful. Upon completion of the surgery, neuromuscular blockade was reversed and extubation was attempted after thorough suctioning, once patient achieved clinical criteria for extubation and there was no bleeding from the surgical site. After extubation, patient had an episode of stridor which was managed by Inj Propofol 10 mg and bag and mask ventilation with CPAP. Post operative analgesia was managed with paracetamol intravenous 300 mg thrice a day and diclofenac 20 mg if pain did not subside with paracetamol. After 30 minutes of observation, once patient was awake and spontaneously breathing, patient was shifted to ICU. After 2 hours, patient again had stridor which failed to respond to conservative management and he had to be re-intubated and ventilated on SIMV mode in propped up position and Inj Hydrocortisone 40 mg I V was given. Patient was reassessed after 24 hours and trial extubation was attempted. Patient had another episode of stridor. Inj Lignocaine 30 mg IV, Inj. Propofol 10 mg IV and Larson manoeuvre were tried but stridor could not be relieved.

To evaluate the cause of stridor, video laryngoscopy (C-MAC) was performed under sedation with inj propofol 50 mg IV and patient was reintubated. Video laryngoscopy revealed edema of arytenoids and hypomobile, partially erected vocal cords, suggestive of laryngospasm (Figure 1). Flexible bronchoscopy done via the endotracheal tube did not reveal any foreign body or stenosis. Blood investigation suggested hypoparathyroidism (S.PTH 9.1 mg/dl) and hypocalcaemia (S.Ca^{+2} 8.0 mg/dl, ionic Ca^{+2} 3.2 mg/dl). Arterial blood gases were normal. Keeping in mind laryngospasm, patient was treated with continued mechanical ventilation, Inj calcium gluconate IV, adrenaline nebulisation and Inj. hydrocortisone IV with antibiotic and antacid cover. After 48 hours postoperatively patient was reassessed and extubated. Patient was observed for another 24 hours and was then discharged from the ICU for further evaluation of hypoparathyroidism and hypocalcaemia.

*Author for correspondence

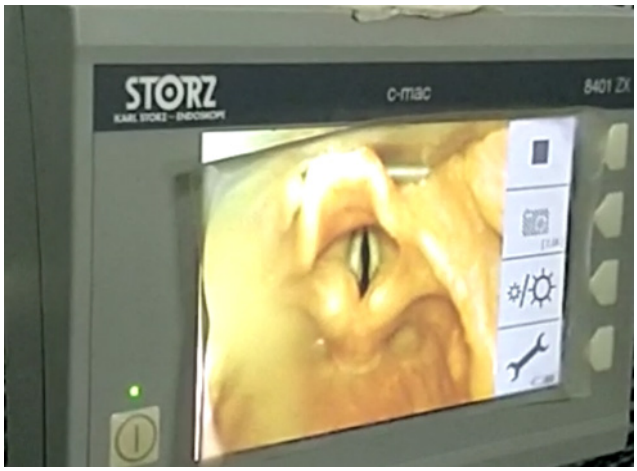

Figure 1. Videolaryngoscopic view of the glottis showing oedema of arytenoids with hypomobile and partially erected vocal cords.

Laryngospasm, mediated by the superior laryngeal nerve, is a reflexive closure of the glottis as a result of abnormal stimulus. The incidence of laryngospasm has been found to range from 1/1,000 up to 20/100 in high-risk surgery (*i.e.*, otolaryngology surgery)[3]. It may be aggravated by incomplete reversal of neuromuscular blockade, hypocalcemia or hypermagnesemia[4]. Laryngospasm is more often a clinical diagnosis made by the anaesthesiologist by identifying various signs of airway obstruction like laboured breathing or stridor. Although various etiologies are attributed to laryngospasm like hyperreactive airway, inadequate depth of anaesthesia, irritants like blood, mucus, laryngoscope blade or endotracheal tube, it may also present as a rare complication of hypocalcemia, especially without the overt signs of tetany or seizures which may be due to increased neuromuscular irritability[5]. Immediate intervention in form of jaw thrust manoeuvre, 100% O_2 with CPAP, deepening the plane of anaesthesia with inj. propofol 0.25-0.8 mg/kg and inhalational anaesthetics improve the outcome. Refractory cases may require Inj Suxamethonium 0.1-3 mg/kg. Children are more prone to airway related events especially those with URTI because of hyper reactivity lasting upto 6 weeks. Calcium levels should be assessed in patients with laryngospasm not responding to standard treatment and should be corrected promptly. Knowledge of the risk factors, anticipation of laryngospasmic episodes and measures for prevention of such an event are vital to decreasing the morbidity.

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