An Interesting Incidence Following General Anesthesia: Anesthesia Mumps?

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We report a case of 50 years old man with rapid onset of unilateral buccal and periauricular swelling after extubation of the trachea. Described by some pre-existing literature, the most likely cause is acute postoperative parotitis occurring due to various causes like pneumoparotitis, venous congestion, Stenson's duct obstruction, the retrograde passage of air, and excess saliva secretion in the perioperative phase. The resolution was relatively rapid, uncomplicated, and spontaneous in around 48 hours in our case. Most cases are managed with rehydration and anti-inflammatory drugs like ours while some mandate airway management due to obstruction. General anesthesia with endotracheal intubation demands caution as complications ranges from trivial to major. Awareness of benign complications helps prevent unnecessary worries.

Keywords: anesthesia mumps, retrograde parotitis.

Acute transient swelling of the parotid gland related to general anesthesia called postoperative parotitis or anesthesia mumps is relatively infrequent. It is an extremely rare complication. The occurrence is variable (0.2–17%) and the exact incidence is unknown. It is a benign complication. (1-3). The reason for its recent decline is the frequent use of antibiotics after major operations, combined with better oral hygiene, adequate control of fluid and electrolyte balance, and blood replacement. Presently postoperative parotitis occurs

mostly in uremic patients as they are prone to dehydration.[2]

Postoperative salivary gland swelling is typically mild and doesn't have many subjective symptoms. Most cases resolve spontaneously without being noticed and thus the discrepancies in the incidence rate. The exact cause and pathophysiology of anesthesia mumps remain unknown, but salivary duct occlusion, sialorrhea, venous congestion and venostasis, involvement of the autonomic nerves, a stormy induction or emergence from anesthesia and side effects of medications like belladonna, depolarizing neuromuscular-blocking drugs, succinylcholine, straining, coughing, and the Valsalva maneuver are few of the presumed causes.[2,3]

Salivary gland obstruction may be caused by physical compression in the lateral position, positions that rotate and flex the neck, compression by endotracheal tubes leading to mucosal lesions and edema. Pneumoparotitis is caused by the penetration of air by retrograde flow into the salivary duct due to positive pressure in the oral cavity by mask ventilation, pharyngeal reflex, and cough reflex; and dehydration may also cause duct obstruction. Use of atropine, and sympathetic nervous system activation due to invasiveness of surgery causing increased salivary viscosity, May in turn itself cause an occlusion. Intratracheal manipulation stimulates parasympathetic nerves and mediates the pharyngeal reflex leading to salivation, vasodilation and hyperemia in the salivary gland. Stimulation of the sympathetic nerves also causes salivation which is short-lasting, thick and mucinous, and vasoconstriction occurs. A noradrenaline infusion increases salivary alpha-amylase.

Most cases of postoperative parotitis resolve spontaneously with follow-up and observation alone. At the most rehydration therapy and anti-inflammatory drugs may be needed to treat it. In extremely rare conditions patients suffer from severe complications, including upper airway obstruction and thus may mandate airway management. Laryngoscopy and tracheostomy might both be difficult if there is massive edema and swelling of the neck.[3]

Case report

A 50-yrs-old, 55 kgs male presented to the operation room for MIPPO of left tibial fracture. His Hepatitis B antigen was positive but there was no active hepatitis. Also, he had a history of spinal instrumentation and subsequent removal under General anesthesia which was uneventful. He denied any past H/O Anesthetic complications. His baseline peripheral oxygen saturation(Spo2) was 99% on ambient air, heart rate was 76 beats

Figure 1: Periauricular swelling when viewed form the side

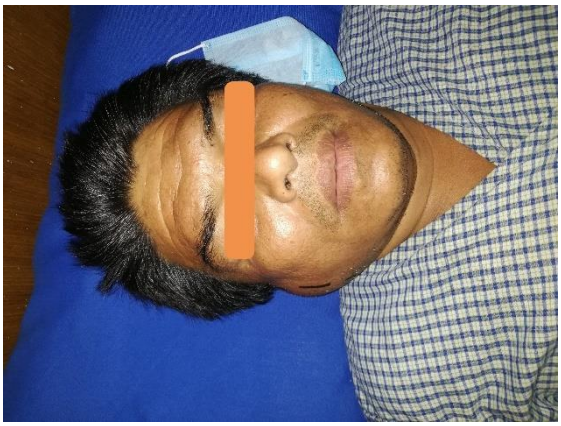

Figure 2: Frontal view of periauricular swelling of the face

per minute, blood pressure was 140/95 mmHg and respiratory rate was 16 breaths per minute before induction. Preoperatively, his chest examination results were normal and a chest radiograph indicated clear lung fields. His baseline lab report including hemoglobin, total leucocyte count, platelet count, blood sugar, renal function tests, electrolytes, and liver function tests were all within normal range. The patient was premeditated with 2mg intravenous (IV) midazolam, and 150 mcg IV fentanyl was given for analgesia, and anesthesia was induced with 180 mg iv propofol, and 7 mg iv vecuronium for facilitation of tracheal intubation. The trachea was intubated with a 7.5-mm internal diameter (ID) endotracheal tube using a no. 4 Macintosh laryngoscope with direct visualization of the vocal cords. The vocal cord visualization required two attempts at laryngoscopy with BURP (backward, upward, right, and posterior) maneuver, and Cormach Lehane grade was III. Bilateral breath sounds were confirmed. Intraoperatively, the lung mechanics, as well as the oxygenation, were normal. The patient was hemodynamically stable with minimal blood loss and was easily ventilated and oxygenated. Maintenance of anesthesia was done with isoflurane, vecuronium, oxygen, and positive pressure ventilation. A total of 1400 ml lactated Ringer's solution was administered during the 2hrs 30-mins surgical procedure. Reversal of neuromuscular (NM) blocker was done with 2.5 mg IV neostigmine and 0.6 mg iv glycopyrrolate. Suctioning of the oropharynx was done and the trachea was extubated. The patient coughed vigorously post-extubation.

The patient gradually developed swelling over the right periauricular and submandibular region which was prominent after 1.5 hours post-extubation in the evening **(Figure 1, 2)**. There was no

pain over the region of swelling but he developed minimal pain the other day. On examination, there was fluctuant swelling over the right cheek with no tenderness or rise in temperature. There was no fever. The next morning there was slight tenderness present as the swelling had increased. The intraoral hygiene was poor and parotid swelling was observed. The maxillofacial team was consulted. The impression of retrograde parotitis secondary to general anesthesia was made. The patient was managed with intravenous fluids, NSAIDs, and prophylactic antibiotics were given considering the patient's poor oral hygiene and the chance of developing a secondary bacterial infection. At 36 hours post-extubation, the swelling had subsided and there was no pain. On examination too minimal parotid swelling was present, no tenderness was elicited. On manipulation, there was no pus or serous discharge from the right parotid duct opening and saliva flow was present. All lab parameters were within normal range. Serum lipase was normal but serum amylase was raised.

Discussion

Unilateral or bilateral swelling of parotid glands can appear rapidly during the perioperative period, but has an uncomplicated course and resolves spontaneously. Few cases with massive swelling leading to airway obstruction and subsequent need for airway management have also been described in the literature. In our patient, the onset of swelling was 1.5 hours after extubation of the trachea and was progressive. Fortunately, our patient did not develop airway obstruction or any major distress physically. The patient was psychologically affected due to sudden swelling around the facial region which as per him was quite unrelated to the course of his primary illness that he presented with to the hospital.

In a case report an extensive swelling in the left parotid region, extending to the buccal and cervical areas, developed in a 30-year-old man immediately after a partial nephrectomy which was performed under general anesthesia. Radiological examination immediately after the onset revealed no abnormality, but a large swelling was detected by computed tomography in the left parotid region. Serum amylase was significantly elevated. The clinical signs had almost disappeared approximately 2 weeks after the onset, following intravenous infusion of antibiotics and transfusion. This parotid swelling was considered to be acute postoperative parotitis induced during induction of anesthesia by luxation of the temporomandibular joint or by the positioning of the patient during operation.[4,5] The literature available

describes many causes of perioperative parotitis. Mechanical trauma and parasympathetic nerve stimulation during endotracheal intubation can be the cause. Obstruction of glandular ducts by calculi or thick secretion can cause bacterial infection and thus purulent sialadenitis. Increased airway pressure during mask ventilation combined with muscle relaxation causes air to enter the parotid orifice and obstruct the ducts. This could have been the potential cause in our case. Adverse drug reactions can be of type A that is an augmentation of a pharmacologically known effect with drugs like morphine infusion or of type B that is abnormal immune-mediated or non-immune-mediated reactions with

Mechanical trauma due to endotracheal intubation is also likely in our case as there were multiple attempts at laryngoscopy and swelling developed quite early on after extubation. Excretory duct obstruction could not be ruled out due to a lack of imaging. An adverse drug reaction was also unlikely because none of the drugs already associated with drug-induced sialadenitis was administered. Although intraoperative venous stasis is less likely as the patient was in the supine position, it could have happened during laryngoscopy due to head extension. Also, vigorous coughing immediately post-extubation could be another potential cause in our case.

Based on the findings and as suggested in many kinds of literature, this condition resolves spontaneously without a complicated course. The management modality involves anti-inflammatory drugs and hydration. In our case too the swelling resolved within 48 hours, was not much of a problem for the patient and the management involved NSAIDs and intravenous and oral fluids along with good oral hygiene and close monitoring of the course of the condition.[4, 5]

Conclusion

We report a case of postoperative parotid sialadenitis which resulted in unilateral facial oedema but without worrisome airway obstruction. Hypothetically, intra-operative mechanical trauma and head position or increased airway pressures during mechanical ventilation can best describe the genesis of swelling in our case. Our report suggests that physician anesthesiologists and surgeons must know this condition. The unawareness about this complication could bring about unnecessary doubts and worries and also there could be a lack of preparedness due to the absence of anticipation of potential progression to airway obstruction.

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