Facial Pain in the Recovery Room Secondary to Acute Parotitis

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Facial pain following anesthesia is a frequent complaint and can arise from a number of causes. The use of depolarizing muscle relaxants as an adjunct for intubation often results in muscular aches.1 Myalgia from the pterygoid, temporal, and masseter muscle produce painful mastication. Trismus occurs secondary to abnormal excitation or inhibition of the affected muscle groups. Temporomandibular joint dysfunction may result from excessive forward displacement of the condylar process and articular disc during laryngoscopy.2,3 Direct facial trauma may be caused by improper patient positioning, tight mask straps, or personnel leaning on a draped face.

This report describes a case of facial pain secondary to acute postoperative parotitis that developed in the recovery room. Anesthesia and surgery contribute to many factors that predispose to the development of this disorder. These include dehydration, the suppression of oral secretions by the restriction of fluids, the use of medication with antisialagogue properties, and inhalation of unhumidified gases.4-6

The clinical manifestations, predisposing factors, and treatment of parotitis are discussed. Acute postoperative parotitis occurs infrequently but should be considered as a possible cause of nonsurgical postoperative facial pain.

CASE REPORT

A 74-yr-old man was admitted for a transurethral resection of the prostate (TURP) for benign prostatic hypertrophy. Concurrent medical problems included hypertension, atrial fibrillation, and mild chronic obstructive pulmonary disease. His medications were hydrochlorothiazide 25 mg, triamterene 50 mg, digoxin 0.25 mg once daily, and aspirin 650 mg twice daily. He had no allergies and a review of systems was otherwise negative. Examination revealed a well-hydrated, edentulous, elderly man. His blood pressure was 160/90 mmHg and heart rate was irregular at 65 beats per min. His preoperative hemoglobin, electrolytes, urea creatinine, and conglomogram, including bleeding time, were normal. His electrocardiogram confirmed atrial fibrillation. The night prior to surgery he was ordered nph after midnight but he did not drink after 5 p.m. He requested that no preoperative sedation be ordered. At 10 A.M. the following morning he underwent a TURP under spinal anesthesia with spinal lidocaine 75 mg. A sensory block extended to the sixth thoracic vertebral level. He received fentanyl 50 μg, diazepam 5 mg iv, and 500 ml of 0.9% saline over the 1-h course of surgery. Supplemental oxygen was supplied by a face mask at 6 l/min. His vital signs remained stable and he tolerated the procedure well.

Fifteen minutes after admission to the postanesthetic care unit, he suddenly developed severe left-sided facial pain. The pain was throbbing, radiated to his left temple, and was made worse by mouth opening. He complained of a dry mouth and a tender jaw. He had never experienced a similar pain.

Examination revealed a firm, indurated swelling anterior to his left ear. Stensen's duct, located in the oral cavity opposite the second upper molar, was tender to bimanual palpation. No thickening of the duct could be felt nor pus or secretions expressed from its orifice. The left temporomandibular joint was examined by placing an index finger into the joint space and asking the patient to close his mouth. This did not elicit the pain. He appeared mildly dehydrated with dry mucous membranes and a jugular venous wave form visible at the sternal notch. He was afebrile and did not appear toxic. The diagnosis of nonsuppurative parotitis was made. He was rehydrated with 1000 ml of balanced salt solution over 4 h and received demerol 50 mg im for analgesia. He resumed oral fluids 6 h after surgery. The pain resolved gradually over the next 6 h and he made an otherwise uneventful recovery.

DISCUSSION

Acute parotitis or “surgical mumps” is characterized by the acute onset of facial pain, trismus, and xerostomia.6 It usually occurs on the second to fifth postoperative day but may occur in patients who have not undergone a surgical procedure. The parotid gland is located anterior and inferior to the external ear. It is composed of a superficial and deep lobe.7 The superficial lobe lies anterior to the masseter muscle and extends to the zygoma. The deep lobe lies posterior to the mandible and extends medially to the styloid process. The gland drains into Stensen’s duct which crosses through the buccinator muscle and opens opposite the second upper molar. Massaging the gland towards its orifice will normally express secretions.

Inflammatory disorders of the parotid gland can be classified as supplicative and nonsuppurative.7 Nonsuppurative parotitis can result from obstruction of the duct with mucus plugs and inspissated secretions. Recurrent obstruction can lead to chronic inflammation of the gland and permanent fibrous enlargement. The etiology of this disorder is unknown. Patients usually complain of a dry mouth and pain which resolve spontaneously over several hours following oral rehydration. Suppurative parotitis results from a bacterial infection of the gland. It is believed that oral micro-organisms ascend in a retrograde manner up Stensen’s duct. The common pathogens are Staphylo-

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coccus aureus, Staphylococcus pyogenes, Streptococcus viridans, and pneumococci.6 Facial pain is often accompanied by fever, headache, malaise, and leukocytosis. Edema may extend to periorbital area and cheek. Suppurative parotitis can be differentiated from nonsuppurative parotitis by signs of sepsis and the ability to milk purulent material from the gland orifice.

Factors that predispose to the development of parotitis include dehydration, malnutrition, vomiting, and poor oral hygiene. Mouth breathing, the suppression of oral intake, antihistamines, diuretics, and phenothiazines are major contributing factors. The majority of patients are middle age or older.

In the case presented, the prolonged period of fasting, the concurrent diuretic therapy, and the use of dry inspired gas may have contributed to the onset of this disorder. Spinal anesthesia results in a relative hypovolemia by blocking sympathetic nerve fibers causing peripheral vasodilation.

The recommended treatment of parotitis includes analgesics, rehydration and improved oral hygiene.8,9 If suppurative parotitis is suspected, smears and cultures should be obtained from material milked from the Stensen's duct and antimicrobial therapy initiated. Type-specific antibiotics are essential. If abscess formation occurs, incision and drainage may be necessary.

In summary, a case of nonsuppurative parotitis is described following anesthesia. The diagnosis should be considered in all patients that complain of unexpected postoperative facial pain.

REFERENCES


Cardiac Arrest and Resuscitation in a Child with Undetected Anomalous Left Coronary Artery

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Anatomic abnormalities of the coronary circulation are uncommon and often undetected conditions that can precipitate life-threatening anesthetic complications. Among the best described of these conditions is an anomalous left coronary artery (LCA) arising from the pulmonary artery. Rarely seen by anesthesiologists, this variant can lead to sudden cardiovascular collapse. The early symptoms are nonspecific and often appear to be purely respiratory in nature.1–3 The following is a case of cardiac arrest associated with this condition.

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CASE REPORT

A 3-month-old, 4.5-kg girl had episodic discomfort including a "hoarse cry" and intermittent wheezing. The symptoms, initially ascribed to an upper respiratory infection, had persisted for 1 month, and while still relatively mild, were increasing in frequency. The patient had been seen in an emergency room at another hospital where mild respiratory distress was noted. Subsequently the infant was transferred to our institution and scheduled for diagnostic bronchoscopy to rule out subglottic and pulmonary pathology. The patient was the product of a normal gestation with no problems at birth. There were no abnormalities in feeding or activity noted. A previous workup included hemoglobin (10.8), hematocrit (33.5), and chest x-ray described as clinically unremarkable. There was no preoperative electrocardiogram. The patient had a recent low-grade fever not noted in the previous two days. Her distress had been noted to be paroxysmal and intermittent. Upon presentation for bronchoscopy the patient appeared comfortable with no evidence of wheezing, intercostal retractions, cyanosis, or tachypnea. Breath sounds were clear. Preanesthetic medication consisted of atropine 0.1 mg im. Monitors included electrocardiogram (EKG), automated blood pressure cuff, pulse oximeter, capnometer, and precordial stethoscope. Preinduction blood pressure was 80/50, and EKG monitor showed normal sinus rhythm rate of 130 with a normal-appearing lead II configuration.