

coccus aureus, *Staphylococcus pyogenes*, *Streptococcus viridans*, and *pneumococci*.⁸ 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-spe-

cific 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.

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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.

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.

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Induction was smooth using halothane, nitrous oxide, and oxygen with spontaneous respiration. The blood pressure decreased to approximately 65/40 with 2% halothane but increased to 70/50 when halothane was decreased to 1.5%. Eventually there was slight upper airway obstruction that necessitated the use of an oral airway and occasional positive pressure. Intravenous catheter insertion proved very difficult and after 10 min. iv access had not been obtained. Because the stomach was becoming distended and the airway more difficult to maintain, it was felt that intubation was needed for airway protection at this time. Halothane concentration was at 1.5% and blood pressure was stable at 70/50. Oxygen saturation was 100%. Inspired oxygen concentration was 50% until just before intubation when 100% oxygen was given. The patient received an additional 0.2 mg im atropine and the trachea was intubated without difficulty with a 3.5-mm endotracheal tube while the child was spontaneously breathing. Tracheal intubation was confirmed with capnometry and bilateral breath sounds. At this time the heart rate suddenly slowed to approximately 70 beats per min with decreased heart tones and soon no pulse was palpable. Significant ST elevation was seen on the EKG monitor at lead II. Anesthetics were stopped and cardiopulmonary resuscitation (CPR) was started with external compressions of approximately 100 per min and ventilation with 100% oxygen. Continued attempts were made to insert an iv catheter. Atropine (0.2 mg, total) and epinephrine (1 ml of 1:10,000 solution, total) were administered intratracheally in two separate doses. Bradycardia continued at 70 beats per min without palpable pulses. After 8 min a right femoral iv catheter was inserted. The patient received iv bicarbonate (5 mEq), atropine (0.4 mg), and epinephrine (1 ml of 1:10000 solution) along with continued CPR. Twelve minutes after the arrest a femoral pulse and a heart rate of 190 beats per min were obtained.

Arterial blood gases (ABG) at this time were pH 7.11, P_{CO_2} 43, P_{O_2} 507, and O_2 saturation 99%. Five milliequivalents of bicarbonate were given. Later ABGs showed significant improvement in base deficit. The patient became responsive and was sent to the pediatric intensive care unit with the trachea intubated and the lungs mechanically ventilated. She became completely responsive and was judged neurologically intact; however, she could not be separated from the ventilator. Postoperative EKGs showed left ventricular hypertrophy with ST segment elevations in the anterior chest leads. ABG showed borderline oxygenation and chest x-ray showed mild pulmonary edema and an enlarged heart. Echocardiogram done for workup of cardiomegaly showed left atrial and ventricular enlargement and mitral regurgitation, and suggested that the LCA arose from the pulmonary artery. Subsequent thallium scan revealed decreased activity in the area perfused by the LCA. The patient was transferred to another institution for further cardiac evaluation. At this time cardiac catheterization showed an anomalous LCA arising from the pulmonary artery. The patient's condition continued to deteriorate and surgical correction was attempted. The patient expired intraoperatively.

DISCUSSION

Anomalous left coronary artery can cause intermittent discomfort in infants that is ascribable to angina.⁴ These children are generally too young to communicate about chest pain⁵ and often their symptoms are initially misinterpreted as reflecting a purely respiratory problem as was the situation here. This case illustrates that wheezing in infants, especially in the absence of coughing, can be a sign of congestive heart failure. An anesthesiologist presented with such a patient should be alerted to the possibility of cardiac involvement and should insist on an ad-

equated diagnostic workup to take all possibilities, including heart failure, into account. These points should be applied to all instances where infantile heart disease may be present.

Often, anomalous left coronary artery is not diagnosed until myocardial infarction has already occurred.⁶ The anomaly is quite rare, accounting for only 1 in 300,000 live births.⁷ Most patients are women.⁸ The condition takes two forms: the more serious infantile type (80% of cases) and a variant in which a patient survives to adulthood (20% of cases). In the former, mortality in the first year of life approaches 90%⁹ without early surgical intervention.

The pathophysiology of the infantile syndrome is unique. During fetal and early newborn life, pulmonary artery (PA) pressure closely approximates systemic pressure and the coronary artery arising from the PA is perfused but with venous (deoxygenated) blood. This suffices for a short period, but as PA pressure decreases with time, the perfusion pressure and thus blood flow in the LCA will decrease. Additionally, a steal phenomenon can develop where blood is shunted out of the coronary circulation into the pulmonary artery because of a reversed pressure gradient. If there are not adequate collaterals to supply blood from the normal right coronary artery, the patient will experience attacks of angina, usually beginning at 2–3 months of age. These episodes are usually paroxysmal and can be associated with feeding. Dyspnea, tachypnea, and restlessness are seen. At first these anginal equivalents are likely to be short lived and may not be distinguishable from routine childhood illnesses. The infants may appear well between episodes. However, over time the symptoms are likely to become more severe and may feature constant wheezing and other signs of CHF. Respiratory infections and mitral regurgitation often ensue. Chest x-ray will eventually show cardiac enlargement.^{3,10,5} Electrocardiogram will show ischemic changes; this is the most dependable early diagnostic tool⁴ and should always be obtained if there is any question of pre-anesthetic cardiac dysfunction in an infant. Echocardiograms show left ventricular dysfunction and left atrial dilatation, and can show directly the origin of the LCA from the PA. Thallium scans reveal localized abnormalities of uptake along the distribution of the LCA.¹¹ Definitive diagnosis is made by coronary angiography.⁴ In the past, patients who were symptomatic before the age of 6 months had an especially poor prognosis, often with death occurring within 1 month of presentation.⁶ However, recent evidence has suggested that creating a two coronary artery system with the Takeuchi procedure (intrapulmonary artery baffle) can lead to greatly improved survival in patients (including infants) with this anomaly.¹²

The "adult" syndrome simply implies survival with this disease past infancy. It is characterized by the development

of collaterals linking the right and left coronary circulations. These allow the left heart to receive oxygenated blood at an adequate perfusion pressure. It is not known why some patients develop collaterals; perhaps they simply survive earlier less-severe ischemic episodes long enough to form them. Depending on individual conditions, patients can remain virtually symptom free for variable periods (years or decades). Very few patients first present as adults; many more are first seen as older children. It is thought that the steal phenomenon, with the LCA acting as a fistula draining blood out of the coronary vasculature, may be especially important for causing ischemia in these patients.³ It is not understood why some patients do well for longer periods than others. Surgical correction is recommended soon after diagnosis because of a high frequency of sudden death among untreated patients.¹³

The immediate precipitating event for our complication was a bradycardia occurring at the time of tracheal intubation. There are several possible causes that must be considered in the differential diagnosis. Anesthetic overdose is one but is unlikely as there was no progressive drop in blood pressure. Hypoxia is another but is unlikely because the trachea was properly intubated. Acute ischemia (related to the underlying disease) after the stimulus of intubation certainly may have caused bradycardia. Vagal response to intubation is also a possibility. Because of the possibility for further dysfunction of an already compromised and ischemic myocardium, the patient with anomalous LCA is an exceedingly high anesthetic risk.

It must be understood that this type of rare medical condition is not the cause of most pediatric cardiac arrests. However, there are rare or initially undetected conditions that can have a devastating effect in yielding severe anesthetic complications. Anomalous LCA is one of these; it can present unexpectedly in infants or adults. It is clear that some familiarity with its related presentation may be helpful to anesthesiologists. This case should at the very

least alert anesthesiologists to the possibility of primary cardiac dysfunction in an infant with respiratory symptoms.

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