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Upper-airway Obstruction and Achalasia of the Esophagus

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Achalasia is a generalized disorder of esophageal motility characterized by aperistalsis, incomplete esophageal sphincter (LES) relaxation, and increased LES tension.¹ Obstruction at the esophageal hiatus results in dilatation of the thoracic esophagus, which can cause cough and dyspnea by compression of the trachea and main-stem bronchi, and predisposes to regurgitation and pulmonary aspiration of stagnant esophageal contents.² Because the upper esophageal sphincter at the level of the cricopharyngeus muscle can resist cephalad distending pressure of 100 cm H₂O while opening to pressures of 15–25 cm H₂O from above,³ air swallowing and positive-pressure ventilation can cause progressive dilatation of the thoracic and cervical esophagus, which may compromise the airway. Severe upper-airway obstruction caused by a dilated esophagus in a patient with achalasia is presented in this case report.

REPORT OF A CASE

A 73-year-old woman with known cerebellar ataxia was found in acute respiratory distress at home by her family physician, who arranged immediate transfer by ambulance to the hospital. Upon arrival she was semicomatose and cyanotic, with distended neck veins. Heart rate was 126 beats/min, arterial blood pressure 80/50 torr, and respiratory rate 36/min. Inspiratory stridor was unrelieved by oral or nasopharyngeal airways. Entry of air into the lungs was poor as judged by auscultation. The abdomen was distended and tympanic. While ventilation was manually assisted with 100 per cent oxygen, PaO₂ was 71 torr, PaCO₂ 58 torr, pH_a 7.09, and base excess (BE)-12 mEq/l. Sodium bicarbonate (90 mEq) was given intravenously. The electrocardiogram showed sinus tachycardia. Laryngoscopy with a #3 MacIntosh blade revealed a large cystic submucosal mass in the hypopharynx, which obliterated the piriform fossae and displaced the larynx anteriorly. The aryepiglottic folds were swollen and erythematous. Only the posterior fourth of the vocal cords could be visualized, and appeared normal.

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However, insertion of 7.0-mm and 6.0-mm orotracheal tubes met with resistance at the infraglottic region. A 5.5-mm tube subsequently was inserted into the trachea. Breath sounds improved with positive-pressure ventilation despite a large leak at the mouth. Radiogram of the chest revealed dilatation of the cervical and thoracic esophagus with displacement of trachea and upper airways. Increased densities were evident at both lung bases. A large volume of gas was removed via a nasogastric tube, which resulted in less labored breathing through and around the endotracheal tube. Upon reexamination of the pharynx, the cystic mass could not be identified. Two hours after intubation the trachea was extubated and the patient had no stridor. With administration of oxygen by face mask, PaO₂ was 60 torr, PaCO₂ 38 torr, pH_a 7.45, and BE +2.2 mEq/l. A second radiogram of the chest showed decompression of the cervical esophagus, increased pulmonary vascular congestion, and increased patchy, confluent densities at the right base. Digoxin, furosemide and sodium ampicillin were given. Barium swallow several days later demonstrated a dilated esophagus with residual secretions and aperistalsis consistent with achalasia.

The patient subsequently underwent esophagomyotomy and transthoracic hiatal hernia repair without recurrence of upper-airway obstruction. The trachea easily accommodated an 8.0-mm orotracheal tube at operation.

DISCUSSION

We presume that the patient had aspirated esophageal and gastric contents into the lungs at home. Subsequent air swallowing and positive-pressure ventilation administered in the ambulance caused esophageal, gastric and intestinal distention. Respiratory distress due to aspiration and abdominal distention was compounded by compression of the membranous trachea and bronchi by the dilated thoracic esophagus. The dramatic displacement of the pharynx by the tensely dilated cervical esophagus obstructed the airway above the glottis.

The distended neck veins could have been caused by compression of the superior vena cava by the tensely dilated esophagus or by acute biventricular failure. The large A-aDO₂ resulted from aspiration, atelectasis, and perhaps pulmonary edema, similar to those features encountered in association with other types of upper-airway obstruction.⁴ We are more certain, however, that tense dilatation of the esophagus in a patient subsequently proven to have achalasia caused severe upper airway obstruction, which was promptly relieved by venting the esophagus with a nasogastric tube.

In achalasia, lesions have been found in the myenteric and inhibitory ganglia of the esophageal wall,

vagal trunks, and dorsal vagal nuclei of the brain stem. Achalasia develops in 3 to 5.5 per cent of patients who have diffuse esophageal spasm. Benign inflammatory strictures from reflux esophagitis (with and without associated hiatal hernia), chemical burns, infection with *Trypanosoma cruzi* (Chagas' disease), scleroderma, and malignancies of esophageal, gastric, pancreatic and bronchogenic origin, as well as lymphoma, have caused achalasia.^{1,2}

Complications of achalasia include esophagitis with mucosal ulcerations and erosions, hemorrhage, perforations with periesophagitis and scarring; epiphrenic, pharyngoesophageal, and traction diverticula; malnutrition; and leukoplakia. Squamous-cell carcinoma develops in 5–10 per cent of patients who have chronic achalasia.^{2,1} Megaesophagus can cause compression of the recurrent laryngeal nerve, superior vena cava, and conducting airways. Chronic, silent regurgitation and aspiration of stagnant esophageal contents into the lungs may result in bronchopneumonia, pulmonary abscess, atelectasis, and chronic bronchiectasis.

During cardiopulmonary resuscitation of patients who have achalasia and megaesophagus, intubation of the trachea with an appropriately-sized, cuffed endotracheal tube should precede decompression of the esophagus. Because of the hazards of sudden release of esophageal contents into the pharynx or of injury to the esophagus, esophageal obturator airways should not be used in patients who have achalasia.

Even when carefully inserted, the standard 15-mm esophageal obturator airway with a 35-ml balloon cannot be relied upon to prevent esophageal air entry during positive-pressure breathing by face mask. In the dyspneic awake patient with achalasia, however, prompt venting of the esophagus with a nasogastric tube is indicated.

Patients with achalasia who need surgical procedures urgently or electively risk pulmonary aspiration, airway obstruction, and superior vena caval compression. The prevention of these complications involves emptying and decompressing the esophagus before, during, and after operation and the application of standard anesthetic principles⁵ to minimize the chance of aspiration of gastric contents before, during and after operation.

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