of obstruction and symptomatology, but most authorities seem to agree to early intervention. The disease has a tendency to spontaneous regression, which complicates the evaluation of the efficacy of treatment. Systemic steroids are used frequently; however, the response may be disappointing. Other treatments have included local injections of depot steroids in the lesion with the risk of worsening the obstruction. Chloroquine-like drugs have been tried, as well as radiation therapy. Surgical approaches have included tracheostomy and the surgical removal of the obstructing lesion.

As this case demonstrates, the anesthesiologist must remain aware of the possibilities of laryngeal involvement in sarcoidosis. We conclude that instrumentation during endotracheal intubation increases the risk of obstructive respiratory arrest. Suspicion of the disorder requires a preoperative evaluation, including laryngoscopy and possible roentgenographic studies of the neck, such as tomography. A flow volume loop, or FEV$_1$, may also be useful preoperatively for assessment of airway obstruction. A small endotracheal tube and prophylactic dexamethasone may minimize airway problems. Careful postoperative observation for up to 36 h may be needed to avoid life-threatening postoperative airway obstruction and respiratory arrest.

REFERENCES


Submucosal Epiglottic Emphysema Complicating Bronchial Rupture

JOHN J. SACCO, M.D.,* DOUGLAS W. HALLIDAY, M.D.†

Rupture of the tracheobronchial tree is a relatively rare event when compared to other intrathoracic injuries resulting from blunt trauma. The true incidence of this lesion is difficult to establish, because many patients die from this injury prior to reaching the hospital, or diagnostic failure occurs antecedent to an early hospital death. Curiously, children seem particularly liable to suffer from this injury.

The acute clinical manifestations of this disorder are varied, and include: mediastinal, suprasternal and subcutaneous emphysema, Hamman’s sign (pericardial cracking sounds synchronous with the cardiac cycle in the presence of mediastinal air), hemoptysis, pneumothorax, tension pneumothorax, atelectasis, cyanosis, respiratory distress, shock, pain on swallowing, hoarseness, airway obstruction and suffocation from disruption, concomitant esophageal and cervical spine injuries, and, very rarely, hemorrhage from major pulmonary vessels.

We describe a pediatric patient who sustained a bronchial tree disruption and presented with some of the above signs and symptoms. Additionally, we believe her presentation is unique, in that submucosal epiglottic emphysema compounded her injuries.

CASE PRESENTATION

A previously healthy 7-yr-old 24 kg, female patient was admitted to our Emergency Unit approximately 40 min after falling across the handlebar of her bicycle. She sustained a contusion on the anterior neck at the level of the cervical trachea. This was further complicated

* Attending Anesthesiologist, St. Joseph’s Hospital.
† Assistant Clinical Professor, SUNY Health Science Center.
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Address reprint requests to Dr. Sacco: St. Joseph’s Hospital Health Center, 301 Prospect Avenue, Syracuse, New York 13203.
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by the development of massive face, neck, and anterior chest subcutaneous emphysema. She was cyanotic and conscious. Her respiratory rate was approximately 30 breaths per minute, and mild inspiratory and expiratory stridor were evident. Her arterial blood pressure was 140/80 mmHg and her heart rate was 140 bpm.

The head and neck were stabilized with sand bags during transport. Initial ER management involved the determination that the upper airway was patent, and 100% oxygen was administered with the patient breathing spontaneously. An intravenous line was inserted. Breath sounds were markedly diminished on the right and, following radiographic confirmation of a total pneumothorax on that side, the right chest was decompressed via needle aspiration. This needle was then attached to iv tubing and underwater seal. Because all the consultants involved suspected tracheal and/or major bronchial rupture, the patient was then immediately transferred to the OR for definitive treatment. Monitoring included EKG, BP, pulse oximetry, and precordial stethoscope. While 100% oxygen was administered to the spontaneously breathing child, infiltrative anesthesia of the anterior neck was attained using 1% lidocaine with 1:100,000 epinephrine, and a tracheostomy was performed. The tissue planes were grossly distorted, and anatomic identification was extremely difficult because of massive emphysema. Supplemental anesthesia was provided with the use of 15 mg of Ketamine given iv in three 5 mg doses over 20 min. Once the trachea was successfully cannulated with a 2 Shiley uncuffed tracheostomy tube, anesthesia was induced with halothane and oxygen. The vital signs were stable, and the oxygen saturation was 99–100% at all times.

A cross table lateral film of the neck was obtained to assess the continuity of the cervical spine. No fractures or displacement were noted to the C6 level. A right chest tube was then inserted, and negative pressure underwater seal drainage begun. A persistent air leak was not evident, and right lung inflation was sustained. Antibiotic prophylaxis with cephalothin was also started.

Next, endoscopy of the pharynx, hypopharynx, and glottic structures was carried out with a Hollinger pediatric laryngoscope. This was significant in that the normal anatomic appearance of the hypopharyngeal and laryngeal structures was greatly altered by a massive amount of subcutaneous emphysema. The epiglottis was, at first, unrecognizable, because its lingual surface was inflated with bullous subcutaneous emphysema to about the size of a small walnut. This caused the epiglottis to reflect back upon the laryngeal inlet. Rigid bronchoscopy was then performed alongside the #2 Shiley trach tube. No intratracheal pathology was noted. Examination of the mainstem bronchi was significant for a 3–4 cm hematoma in the lateral aspect of the right mainstem bronchus just below the level of the carina. This was felt to most probably represent the site of rupture, because approximately 80% of these injuries occur within 2.5 cm of the carina. Additionally, no separation, mucosal flap, or luminal compromise were noted. This examination was complicated by a 2-min interval of increased airway resistance, difficulty in ventilating the patient (through the tracheostomy as well as the bronchoscope), and cyanosis. This problem was corrected by suctioning a relatively large blood clot from the lumen of the tracheostomy tube. Subsequent bronchoscopy proceeded in an uncomplicated manner. Esophagoscopy was then performed and found to be negative. The neck was packed with iodine-soaked gauze and, once emergence was complete, the patient was breathing via a 30% O2 tracheostomy collar and transported to the Pediatric ICU.

The postoperative course was uneventful. Chest tube drainage permitted continued re-expansion of the right lung. Increasing trans-laryngeal air exchange and return of normal vocal function allowed decannulation of the trachea on the third day. The right chest tube drainage was discontinued on the fourth day, and she was discharged on the seventh postoperative day.

She has been seen at appropriate intervals in follow-up clinic for 5 months after discharge, and examination has excluded any long-term complications.

**DISCUSSION**

This case poses several questions, some of which are: 1) what mechanisms are responsible for bronchial rupture? 2) how do we explain the presence of submucosal air in the epiglottis? 3) if one is presented with similar clinical circumstances, would case management be modified in any way? and 4) why was no primary closure of the bronchial tear performed?

The mechanisms of injury that involve the tracheobronchial tree following blunt trauma are diverse. They include shearing forces applied to the trachea and main bronchi by sudden compression and release of the resilient anterior chest wall, abrupt deceleration of the pendulous lung, sudden high tracheobronchial intraluminal pressure developing when expiration occurs against a closed glottis at the moment of impact, and sudden overstretch of the trachea. In this circumstance, the etiology of our patient's injuries might be attributed to any one, or all of, these forces. However, the manner of injury in this patient implicates the last two theories.

An anatomic explanation for the presence of the submucosal epiglottic emphysema most likely involves the passage of mediastinal visceral fascial air into the contiguous cervical visceral fascia. From there, the gasses moved superiorly within the fascial compartment that encloses the thyroid gland, pharynx, and larynx, thus permitting the balloning of the lingual epiglottic mucosa. This lingual surface was involved because of the rather loose set of connective tissue that is characteristic for this side of the epiglottis. This contrasts with the tight net of connective tissue present histologically within its glottic surface; and it is this structural idiosyncrasy that also explains the histopathology of infectious epiglottitis.

Given the presentation of massive subcutaneous emphysema of the face, neck, and anterior thorax, and because glottic and/or cervical tracheal injury could not be ruled out, tracheostomy was indicated in this case. Management of these injuries by endotracheal intubation may be complicated by further respiratory compromise, and involves the additional hazard that the tip of the tube can be accidentally placed extratracheally. Loss of the airway because of this or tracheal intubation difficulties that may have been experienced secondary to the massive submucosal epiglottic emphysema might have led to a disastrous outcome, because tracheostomy was technically complex. Additionally, tracheostomy may be beneficial, because it helps reduce the intrabronchial pressure and prevents an increase of subcutaneous emphysema. Although similar cases have been managed, successfully uti-
lizing rigid or flexible bronchoscopy with subsequent endotracheal intubation.\textsuperscript{19–23} Tracheostomy provided the safest and most practical solution to the circumstances presented by this patient.

Perhaps chest tube drainage should have been instituted once the diagnosis of pneumothorax was confirmed radiographically. Our thoracic consultant felt that, because thoracotomy was a very real possibility, the needle decompression of the right hemithorax was sufficient. Being aware of the possibility of recurrent pneumothorax, spontaneous ventilation was maintained throughout the procedure. This ensured minimal increases in airway pressure. Additionally, because time was allotted for a chest radiograph in the Emergency Unit, a lateral neck film could also have easily been obtained. This would have avoided diagnostic delay in the OR and would have been reassuring if acute airway obstruction occurred prior to placement of the tracheostomy tube.

Lastly, thoracotomy and primary closure of the bronchial rupture was not performed in this case, because the airway defect did not encompass more than one-third of the bronchial circumference, and underwater seal drainage resulted in complete and persistent re-expansion of the lung.\textsuperscript{24}

In summary, a pediatric case of bronchial rupture following blunt neck trauma is reviewed, and a critical discussion involving the mechanisms of injury, anatomic considerations explaining submucosal epiglottic emphysema, case management, and an alternative to primary repair is presented.

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