Bacterial Tracheitis

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Occurring almost exclusively in children, bacterial tracheitis is a rare but rapidly life-threatening upper airway disease with reported mortality rates as high as 20%. Clinicians must recognize this disease promptly to prevent impending airway obstruction and its associated complications, which include acute respiratory distress syndrome and septic shock. Approximately 80% of patients with bacterial tracheitis require intubation, and 94% are admitted to the intensive care unit. Bacterial tracheitis affects 2 groups of pediatric patients: those with a native, intact airway and those with an artificial airway established with an endotracheal tube or tracheotomy tube.

Bacterial tracheitis without tracheotomy is also known as membranous laryngotracheobronchitis, bacterial laryngotracheobronchitis, and pseudomembranous croup. As implied by these alternative names, the hallmark findings of bacterial tracheitis are ulceration and pseudo membrane formation in the trachea, mucopurulent exudate, and sloughing of the mucosa, which obstruct the tracheal lumen and can lead to significant airway obstruction. Historically, the leading causative organisms include *Staphylococcus aureus, Haemophilus influenzae,* α-hemolytic *Streptococcus,* and *Streptococcus pyogenes. Moraxella catarrhalis* has been implicated with increasing frequency and is associated with a more severe course. Gram-negative organisms are rare, yet *Pseudomonas* and *Klebsiella* species have been responsible in a few cases. It is also widely believed that tracheitis may represent a bacterial superinfection of viral tracheobronchitis most commonly caused by influenza, parainfluenza, or respiratory syncytial virus.

A thorough history and physical examination can often lead to the diagnosis of bacterial tracheitis. Its occurrence peaks in the fall and winter months, with an incidence of 4 to 8 per 1,000,000 children. The mean age at diagnosis is 5 years. Frequently, the child presents with a several-day prodrome of an upper respiratory tract viral infection (cough, coryza, and sore throat) and then develops rapid onset of high temperatures (>101.3°F [38.5°C]), stridor, hoarseness, and a toxic appearance. Clinical deterioration typically occurs within 2 to 10 hours. Odynophagia, dysphagia, drooling, and "sniffing" posturing are usually absent. Some children may have concurrent pneumonia, sinusitis, otitis media, or pharyngitis. Leukocytosis or leukopenia is often present.

The differential diagnoses include croup, viral laryngotracheobronchitis, epiglottitis, bronchiolitis, angioedema, and diphtheria in nonvaccinated patients. However, suspicion of bacterial tracheitis should increase when a patient with respiratory distress fails to respond to racemic epinephrine and/or systemic corticosteroids, which typically improve croup and viral laryngotracheobronchitis.

When the history and physical examination lead to suspicion of bacterial tracheitis, the next step in the workup depends on the status of the patient's airway. In patients with a stable airway, clinicians should perform anteroposterior and lateral neck radiography followed by examination with flexible laryngoscopy, if

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tolerated. Radiographs classically reveal irregular borders of the tracheal margins and haziness of the tracheal column, indicative of pseudomembrane detachment. A steeple sign (superior tapering of the trachea manifested as an inverted V on anteroposterior neck radiograph) may also be present, as seen in viral laryngotracheobronchitis. Flexible laryngoscopy reveals mucopurulent secretions in the glottic or subglottic regions. Patients with minimal airway symptoms and normal flexible laryngoscopy examination findings can be treated medically. If symptoms worsen, rigid bronchoscopy with the patient under general anesthesia is warranted.

On the contrary, the priority for patients in severe respiratory distress with airway compromise is to secure the airway. Endotracheal intubation may be necessary, and to account for airway inflammation and thick secretions, the inner diameter of the endotracheal tube should be 1-mm smaller than what is normally indicated. Urgent diagnosis and treatment should then proceed in the operating room with direct rigid bronchoscopy, preferably performed by a pediatric otolaryngologist. Endoscopic findings include edema and erythema of the subglottis, mucopurulent tracheal secretions, and thick pseudomembranes along the tracheal wall mucosa. Definitive treatment entails aggressive debridement of the mucopurulent debris with suction and foreign-body forceps. Gram stain, bacterial culture, and sensitivities of tracheal secretions and viral cultures are performed to help direct medical therapy.

Postoperatively, patients are admitted to the intensive care unit for further monitoring and aggressive supportive care. Children who are not intubated still need close observation by medical personnel skilled in airway management. In some cases, transfer to a tertiary care center may be indicated. For all patients, treatment with intravenous broad-spectrum antibiotics should be initiated and eventually tailored based on culture results. Reasonable empiric antibiotic options include vancomycin or clindamycin plus ampicillin-sulbactam, a third-generation cephalosporin (ceftriaxone or cefotaxime), or a fluoroquinolone in patients with penicillin allergy. Inhaled or systemic steroids and racemic epinephrine have no proven efficacy in relieving airway obstruction from bacterial tracheitis.

For children who do not improve clinically with antibiotics, a subsequent endoscopy may be indicated to remove reaccumulated debris. The decision to intubate and when to extubate is individualized, with a plan formulated by the pediatric intensivist in conjunction with the endoscopist. Extubation is performed when fever, toxic appearance, and tracheal secretions have decreased and a positive air leak is present. Intubation usually lasts 2 to 7 days, and length of hospital stay varies from 5 to 12 days. Patients who continue to improve after extubation should be transitioned to oral antibiotic therapy for 10 to 14 days after discharge. Most patients make a full recovery.

Patients with preexisting intubation or a tracheotomy who develop bacterial tracheitis comprise a unique entity that warrants special clinical assessment and consideration. These patients have an increased risk of tracheitis because an artificial airway provides an easy portal of entry for bacteria and also disrupts innate mucosal immunity. Furthermore, the tube may cause ulceration and tracheal denudation, increasing the risk of infection. Bacterial biofilms that contain live bacteria have also been demonstrated to colonize the internal surface of tracheotomy tubes.

No specific risk factors have been identified for ventilator-associated tracheitis (VAT) or tracheotomy-associated tracheitis. In patients with an artificial airway, symptoms of tracheitis typically have an onset more indolent and subacute than in patients with an intact airway. Clinical suspicion should heighten when a patient exhibits increasing purulent tracheal secretions that require more frequent airway suctioning; with changes in the color, viscosity, or odor of secretions; with a decrease in oxygen saturations; and, if applicable, when changes in mechanical ventilation settings are needed to maintain gas exchange. Bacterial tracheitis in these patients may be challenging to distinguish from an associated lower respiratory tract infection, such as pneumonia, but the absence of pulmonary infiltrates on chest radiograph can support the diagnosis. Nonetheless, isolated tracheitis is *uncommon* in patients with an artificial airway; a concurrent bronchial and/or pulmonary infection is frequently present. If untreated, VAT is also considered a precursor to ventilator-associated pneumonia.

Similar to bacterial tracheitis of the intact airway, the diagnosis of bacterial tracheitis in children with an artificial airway is based on clinical, microbiologic, and endoscopic findings. Patients may have fever without an obvious source, and an increase in neutrophils and the presence of new microbes on tracheal aspirates can help distinguish new bacterial infection from normal flora colonization. However, tracheal aspirates do not differentiate tracheal from pulmonary infection. Tracheoscopy performed with a flexible fiberoptic scope can confirm the diagnosis with evidence of tracheal inflammation. The most common colonizing microbes are *S aureus, H influenzae, Streptococcus pneumoniae, Pseudomonas*

aeruginosa, and other gram-negative organisms. Antibiotic treatment should again be directed at the most common pathogens for 7 to 10 days, taking into account the possibility of multidrug-resistant organisms. If feasible, serial surveillance of endotracheal aspirate specimens can identify multidrug-resistant pathogens and help tailor antibiotic therapy.

The risk of VAT peaks at approximately 4 days after intubation, with an estimated incidence of 2% to 11%. The onset of bacterial tracheitis in children with long-standing tracheotomies is less predictable, with a reported incidence of less than 1%. In general, tracheotomy-associated tracheitis is not aggressive and, if there is close follow-up, can be treated in the outpatient setting with oral antibiotics, topical ciprofloxacin and dexamethasone, or tobramycin and dexamethasone drops directed into the tracheotomy for several days. Recurrence of tracheitis is more common in children with a long-term tracheotomy.

During the past several decades, the incidence, diagnosis, and management of bacterial tracheitis have been influenced by new vaccines and by the heightened awareness among physicians of the alarmingly high morbidity associated with delayed recognition. Despite its low incidence, clinicians must consider the possibility of bacterial tracheitis in any patient with acute respiratory decompensation and be able to distinguish it from viral croup, epiglottitis, and other causes of upper airway obstruction.

COMMENTS: As Drs Kuo and Parikh point out, bacterial tracheitis is rare in children with normal airways. For a child with a tracheotomy tube, however, it is an ever-present worry. With the defense systems of the upper airway bypassed and a direct opening to the environment, the trachea with a tube becomes akin to the throat—readily colonized by a host of bacteria. The problem for the clinician faced with a febrile child with respiratory symptoms is how to distinguish colonization from infection. Periodic surveillance tracheal cultures while the child is well may help. They define the usual flora and through information on sensitivities provide at least a starting point if empiric antimicrobial therapy seems advisable. Culture at the time of illness may then identify a new potential pathogen, with its sensitivities to antibiotics. In addition, the Gram stains performed with the surveillance cultures can usually establish a baseline neutrophil presence. A significant increase in the tracheal neutrophil count on a Gram stain performed when the child is ill suggests active inflammation, likely in the context of fever and symptoms to be infection.

– Henry M. Adam, MD
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