Presentation of Case

Dr. Jessica M. Rosenthal (Pediatrics): An 8-year-old girl who had required long-term tracheostomy because of airway stenosis caused by an injury in infancy was admitted to this hospital because of a chest-wall mass and a large pleural effusion.

The patient was born in China. At 7 months of age, she underwent emergency tracheostomy after a caustic injury from either ingestion or inhalation. At 2.5 years of age, she was adopted by an American family and moved to the United States. Her adoptive parents noted that she had stridor and snoring, and her pediatrician referred her to an otolaryngologist at the Massachusetts Eye and Ear Infirmary (MEEI) at 2 years 10 months of age. On examination, the nasopharynx was completely obstructed; there was fusion of the posterior tongue and hard palate and of the hard palate and epiglottis, with a narrow opening in the oropharynx. The supraglottic passage was normal beyond the stenosis; the subglottic airway was not evaluated.

A tracheostomy was performed, and the left nasal choanal obstruction was opened with a laser. Repeat bronchoscopy revealed that the larynx and subglottic trachea were normal. On two occasions, esophagogastroduodenoscopy revealed no evidence of gastroesophageal reflux. The tracheostomy tube was removed when the patient was 5.5 years of age; there was a residual tracheal–cutaneous fistula. When she was 7.5 years of age (7 months before this admission), another tracheostomy was performed because of worsening oropharyngeal stenosis that precluded intubation and because of concern about possible obstruction.

The patient had been in her usual state of health until approximately 6.5 weeks before this admission; during a 10-day period, her mother noted recurrent blood-tinged sputum in the tracheostomy tube, and an episode of bleeding at the tracheostomy site occurred at school. The patient was seen in the emergency department of the MEEI, where a flexible tracheoscopy, which was performed at the bedside after removal of the tracheostomy tube, revealed no exposed blood vessels or granulation tissue in the trachea or stoma. A chest radiograph showed consolidation in the right lower lobe, which was thought to be due to aspiration, pneumonia, or atelectasis. Gram's staining of the sputum showed abundant neutrophils, abundant gram-positive cocci in pairs and a few chains, abundant gram-negative diplococci, a few gram-positive rods, and a few thin gram-negative rods. Culture
of the sputum from the tracheostomy tube grew methicillin-susceptible Staphylococcus aureus, Moraxella catarrhalis, Haemophilus influenzae, and Streptococcus pneumoniae. The patient remained afebrile, and no antibiotic agents were administered. She was admitted overnight for observation and discharged home the next day (5 weeks before this admission). She had one more episode of blood-tinged sputum thereafter.

During the 2 weeks before this admission, the oxygen saturation, which was obtained during continuous overnight monitoring for sleep apnea, fell to approximately 85% (from the usual level of approximately 95%) for prolonged periods. Examination at school revealed decreased breath sounds at the base of the right lung. One day before this admission, while the patient was being bathed, her mother noticed that her chest was asymmetric, with the right nipple appearing lower than the left, and that blood vessels were prominent on the right side of the chest.

At the pediatrician’s office the next day, the patient described a recent episode of discomfort in her chest that occurred while she was being hugged. There was no history of respiratory distress, fevers, or cough. On examination, she was tearful and hesitant. The temperature was 36.1°C, and a tracheostomy tube was in place. The right lateral chest wall was more prominent than the left, had no crepitus, and was tender to touch. Breath sounds were normal on the left side and diminished on the right. The right upper quadrant of the abdomen was diffusely tender, and the remainder of the examination was normal. The patient was referred to another hospital for chest radiography, which revealed a large pleural effusion on the right side that tracked along the lateral chest wall, with evidence of loculation. The left lung and pleural space were clear. Computed tomography (CT) of the chest, performed on the same day after the administration of contrast material (Fig. 2), revealed asymmetry of the chest wall that was due to multiple heterogeneous soft-tissue masses and thickening of the soft tissue of the right chest wall. Consolidation was present in the right lower lobe and portions of the right middle lobe. Areas of low attenua-
tion in the right lower lobe were suggestive of decreased perfusion and possible necrosis. A large loculated pleural effusion, irregularly shaped parenchymal nodules in the right upper lobe, prominent enhancing ipsilateral axillary lymph nodes, and subtle periosteal elevation of the medial aspect of the right eighth rib were also present.

Dr. Rosenthal: The patient was admitted to the pediatric intensive care unit. The temperature rose to 38.7°C, and acetaminophen was administered, with improvement.

On the second hospital day, a diagnostic procedure was performed.

Differential Diagnosis

Dr. Samuel M. Moskowitz: This 8-year-old girl had had a caustic exposure during infancy that resulted in upper-airway obstruction and tracheostomy dependence. I suspect that her airway injury may have caused mild dysphagia, with an increased risk of tracheal aspiration. She had a history of poor oral hygiene. Her immunizations included PCV7 and BCG, and a PPD skin test was negative after she immigrated to the United States. Six weeks before this admission, bacterial tracheitis with S. aureus, S. pneumoniae, and H. influenzae developed, and focal opacification of the right lower lobe was noted. Bacterial tracheobronchitis and pneumonia are common in children who have undergone tracheostomy, with an annual incidence of up to 88%.[1] The tracheitis abated spontaneously, without antibiotic treatment, whereas the lobar process progressed and was accompanied by hypoxemia, chest tenderness, decreased breath sounds, and pleural effusion but no fever.

On presentation, the patient had tachypnea, prominence and tenderness of the right lateral chest wall, and a tender right axillary lymph node. Laboratory test results showed elevated levels of acute-phase reactants, such as thrombocytosis and hyperglobulinemia. Imaging studies of the chest revealed opacification in the right lower lung zone, a loculated effusion, and an irregular chest-wall mass, with regional lymph-node enhancement and minimal bony changes.

The chest-wall mass is the most worrisome clinical feature of this child’s presentation. An acquired chest-wall deformity or mass can originate in bone or soft tissue; the differential diagnosis includes trauma, neoplasm, inflammatory and infectious processes, or a combination of these causes (Table 2). Before the results of imaging studies are available to rule in or rule out specific causes, the mass could represent a herniated right lung, a benign or malignant tumor, a sterile collection of inflammatory cells, or an abscess.

CHEST-WALL TRAUMA

Traumatic disruption of the skeletal components of the chest wall can result in flail chest, with paradoxical chest-wall motion and compromised gas exchange; these findings were not present in this child. Lung herniation is an uncommon chest-wall deformity that causes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference Range, Age-Adjusted†</th>
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<tbody>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>11.5–15.5</td>
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<tr>
<td>White-cell count (per mm³)</td>
<td>4500–13,500</td>
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<tr>
<td>Differential count (%)</td>
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<td></td>
</tr>
<tr>
<td>Neutrophils</td>
<td>33–59</td>
<td>73</td>
</tr>
<tr>
<td>Lymphocytes</td>
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<td>20</td>
</tr>
<tr>
<td>Monocytes</td>
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<td>5</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0–8</td>
<td>2</td>
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<tr>
<td>Platelet count (per mm³)</td>
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<td>Potassium (mmol/liter)</td>
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<td>Chloride (mmol/liter)</td>
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<tr>
<td>Protein (g/dl)</td>
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<tr>
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</tr>
<tr>
<td>Globulin</td>
<td>2.3–4.1</td>
<td>5.0</td>
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<tr>
<td>Phosphorus (mg/dl)</td>
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<td>4.0</td>
</tr>
<tr>
<td>C-reactive protein (mg/liter)</td>
<td>&lt;8.0 for inflammation</td>
<td>217.2</td>
</tr>
</tbody>
</table>

* To convert the values for phosphorus to millimoles per liter, multiply by 0.3229.
† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are age-adjusted for patients who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

Table 1. Laboratory Data.*
dyspnea, pain, and a bulge that varies with the respiratory cycle. It usually results from trauma but can also be congenital, arise spontaneously after a coughing paroxysm, or occur in patients with neoplastic, inflammatory, or infectious disorders of the chest wall.\(^2\) In this child, examination and imaging studies did not reveal the characteristic bulge of a herniated lung. Without a history of recent trauma, the diagnoses of hematoma and ruptured hemidiaphragm are also unlikely.

**BENIGN AND MALIGNANT TUMORS OF THE CHEST WALL**

Various benign and malignant tumors can arise from the bones of the chest wall (Table 2). How-

**Figure 1. Chest Radiographs.**

Radiographs of the chest were obtained on admission. Frontal and lateral chest radiographs (Panels A and B, respectively) show air-space opacification in the right lower lung zone (asterisks) and a large right pleural effusion (arrows). Decubitus chest radiographs (Panel C [right side down] and Panel D [left side down]) show that pleural fluid on the right side is not freely layering (arrows).
ever, in this case, chest CT revealed only a minimal rib lesion, and thus a bony tumor is unlikely. Some benign tumors that occur in children, such as lymphangioma (cystic hygroma) or hemangio-
ma, arise in the soft tissue of the chest wall and are generally confined to it, without the pleural and pulmonary components that were seen in this case. Other benign tumors originating in soft tissue, such as lipoma, are more common in adults than in children. Peripheral-nerve tumors can be associated with effusion and pleural nodules but would be more likely to occur in a patient with known neurofibromatosis. Desmoid tumor, a histologically benign fibromatosis that can be
locally invasive, is more likely to occur in the shoulder region than in the anterolateral chest.\(^3\) Benign mesothelioma is rare in children and usually confined to the pleural space.

Patients with an inflammatory myofibroblastic tumor can present with a soft-tissue mass of the chest wall. It arises from lung parenchyma and can extend into the chest wall, with localized mass effect.\(^4\) These lesions, which are the most common lung tumors in children, are typically benign but can occasionally undergo malignant transformation. They are typically associated with dyspnea, cough, hemoptysis, and fever. Laboratory evaluation may reveal iron-deficiency anemia, thrombocytosis, and hypergammaglobulinemia. However, chest-wall invasion is inconsistent and generally occurs no sooner than 12 to 18 months after the onset of symptoms, pleural effusions are uncommon, and lymphadenopathy is rare. Associated chest CT reveals a varying nonspecific pattern, with heterogeneous enhancement and attenuation.\(^5\) The laboratory test results and imaging findings in this patient were suggestive of an inflammatory myofibroblastic tumor, but her course was unusually rapid for this lesion and she did not have several of the usual signs and symptoms.

This child did not have persistent systemic symptoms suggestive of a malignant tumor; however, because of the consequences of missing a malignant neoplasm, these lesions deserve consideration. In children, rhabdomyosarcoma is the most common malignant tumor arising in the soft tissue of the chest wall, but the associated chest CT would typically reveal a discrete mass,\(^4\) which is unlike the finding in this child. Other soft-tissue sarcomas are relatively rare in children. Pleuropulmonary blastoma arises from the pleura or lung and can invade the chest wall, but it usually occurs in children 5 years of age or younger. Malignant fibrous histiocytoma is usually confined to the chest wall and is more common in elderly persons than in children.

Lymphomas in the thorax typically arise in the mediastinum and can extend into the anterior chest wall, and thus a locally invasive lymphoma needs to be considered in this case. Patients with thoracic lymphoma typically present with chest pain, dyspnea, and lymphadenopathy, sometimes without fever. Early-stage Hodgkin’s or non-Hodgkin’s lymphoma that arises in the mediastinum can invade the lung parenchyma and chest wall, but this invasion usually is contiguous with a tumor in the anterior mediastinum and does not involve the lower lateral chest (the location of the chest-wall lesion seen in this child).\(^6,7\) In addition, lymphomatous chest-wall invasion usu-

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Originates in or Affects Bone</th>
<th>Originates in or Affects Soft Tissue</th>
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</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>Eosinophilic granuloma (Langerhans’-cell histiocytosis), osteochondroma (exostosis), chondroma,</td>
<td>Lung herniation, hematoma, ruptured hemidiaphragm</td>
</tr>
<tr>
<td></td>
<td>chondroblastoma, fibrous dysplasia, osteoid osteoma, osteoblastoma, mesenchymal hamartoma</td>
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</tr>
<tr>
<td>Benign tumor</td>
<td>Osteosarcoma of the rib, chondrosarcoma of the rib, multiple myeloma, metastasis</td>
<td>Rhabdomyosarcoma, other sarcomas (neurofibrosarcoma, fibrosarcoma, leiomyosarcoma, angiosarcoma,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>liposarcoma), pleuropulmonary blastoma, lymphoma, malignant fibrous histiocytoma, metastasis</td>
</tr>
<tr>
<td>Malignant neoplasm</td>
<td>Hyperostosis, SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis), chronic recur-</td>
<td>Sebaceous cyst</td>
</tr>
<tr>
<td></td>
<td>rent multifocal osteomyelitis (nonpurulent inflammation of bone), Friedrich’s disease (aseptic</td>
<td></td>
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<tr>
<td></td>
<td>necrosis of the sternoclavicular joint)</td>
<td></td>
</tr>
<tr>
<td>Inflammatory process</td>
<td>Osteomyelitis (tuberculosis, actinomycosis), septic arthritis</td>
<td>Aabscess, phlegmon, cellulitis, empyema necessitatis</td>
</tr>
<tr>
<td>Infectious process</td>
<td></td>
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ally develops more slowly than did the process in this child.

In summary, a neoplastic process cannot be ruled out in this case, but an inflammatory or infectious process is more likely.

**INFLAMMATORY AND INFECTIOUS PROCESSES OF THE CHEST WALL**

Primary inflammation of bone, such as that seen with hyperostosis or chronic recurrent multifocal osteomyelitis, can cause a chest-wall mass. Various pathogens can cause osteomyelitis or septic arthritis with localized chest-wall swelling and induration. However, the localization and imaging findings in this child were not consistent with an osseous lesion.

Patients with a sebaceous cyst can present with a soft-tissue mass, but it would remain confined to the chest wall and would not invade the pleura or lung. Soft-tissue infections of the chest wall can be manifested by a solitary abscess or cellulitis, with associated inflammation of adjacent tissues. However, several findings in this child were consistent with an infection originating in the chest cavity and extending into the chest wall.

**EMPYEMA NECESSITATIS**

The clinical and radiographic features seen in this case are most consistent with empyema necessitatis, a process characterized by extension of pleural empyema into the chest wall. This condition was first described by Gullan de Baillon in 1640. Empyema necessitatis typically develops over a period of 4 to 8 weeks and is associated with pain, swelling, and lymphadenopathy of the anterolateral chest, often without fever; these features were seen in this case. It can also result in a pleurocutaneous or bronchopleurocutaneous fistula, which was not seen in this case. René Laennec succinctly described its pathogenesis in 1819 as follows: “When, as the result of chronic pleurisy, a gangrenous eschar forms on the pleura, it sometimes happens that the effusion infiltrates through the intercostal muscles at this point, and comes to form under the skin an abscess whose natural or artificial opening provides, in a few rare cases, a healing of the empyema. These species of abscess, known since the origin of the art, have been observed from time to time by the surgeons, and their opening constitutes what is commonly called the empyema of necessity. This case is also very rare; my friend Mr. Recamier told me that he has observed twice; I have encountered it but once.”

The patient in Laennec’s case is said to have been a 12-year-old boy. In 1869, William Moore described empyema necessitatis in a 10-year-old girl. The publication of only a few relevant case reports since the 19th century indicates that this condition remains extremely rare in children; nonetheless, I believe that this child had empyema necessitatis.

Empyema necessitatis was more common in the era before antibiotics. At that time, it was associated with an overall mortality of approximately 66%; the most common pathogens were *Mycobacterium tuberculosis* (mortality, 87%) and *S. pneumoniae* (mortality, 28%). Since antibiotics have been in use, cases of empyema necessitatis are rarely fatal, and actinomycosis species are a more common cause than is *S. pneumoniae*. Less common pathogens include *S. aureus*, *S. milleri*, *Fusobacterium nucleatum*, *M. avium*, *M. intracecalum*, *Burkholderia cepacia*, blastomyces species, and *Nocardia asteroides*.

Given this child’s history of BCG immunization and the negative PPD skin test, tuberculosis is unlikely. Because a culture of tracheal secretions was positive for *S. pneumoniae*, it would seem to be a prime suspect; it is found in pleural fluid in approximately 50% of children with uncomplicated empyema, and other pathogens are much less common in these patients. However, the patient’s history of pneumococcal vaccination makes this pathogen unlikely. Other aerobic bacteria in her tracheal secretions could cause pneumonia; of these, only *S. aureus* has been reported in patients with empyema necessitatis. Both *S. pneumoniae* and *S. aureus* would be expected to cause fever, which was not seen in this case.

Poor oral hygiene promotes growth of bacteria that can cause pneumonia, including aerobes such as *S. pneumoniae* but also anaerobes that cannot be detected on a routine culture of tracheal secretions. Chief among anaerobes that could cause empyema necessitatis is *A. israelii*. Additional factors that increase the likelihood of pulmonary actinomycosis in this case include the risk of aspi-
ration, subacute course, and absence of fever. Pulmonary actinomycosis is often polymicrobial, with gram-positive bacteria or anaerobes as common coisolates, and its parenchymal component may cavitate. Small-to-medium-sized pleural effusions may occur, and chest-wall invasion may result in early rib erosion, which was seen in this child. I suspect that an actinomyces species was her primary pathogen.

If a neoplasm were likely in this case, I might suggest the use of positron-emission tomography (PET) or magnetic resonance imaging (MRI) to delineate the tumor burden or ultrasound-guided or CT-guided percutaneous aspiration to obtain a tissue sample. In cases of infection or inflammation that is confined to the pleural and parenchymal compartments, my preferred approach would be video-assisted thoracoscopic surgery to facilitate pleural drainage and decortication and to obtain a lung-biopsy specimen, as indicated. For this chest-wall lesion, I would favor exploratory thoracotomy to obtain a biopsy specimen and to permit open débridement or resection.

**Clinical Diagnosis**

Possible malignant tumor of the chest wall or pleura.

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**Dr. Samuel M. Moskowitz’s Diagnosis**

Empyema necessitatis, with pneumonia of the right lower lobe and empyema of the right pleural cavity caused by actinomycetes species.

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**Pathological Discussion**

*Dr. Eugene J. Mark: The diagnostic procedure was a biopsy of the soft-tissue mass of the chest wall. Histopathological examination revealed an abscess with purulent inflammation (Fig. 3A), surrounded by granulomatous inflammation and fibrosis (Fig. 3B). Staining with hematoxylin and eosin revealed a sulfur granule in the abscess (Fig. 3A). Filamentous structures were visible at the periphery of the sulfur granule; on silver staining, the filamentous structures could be seen both creating the sulfur granule (Fig. 3C) and extending as bacillary organisms from the periphery of the sulfur granule (Fig. 3D). These morphologic findings are typical of actinomycetes species.

Both actinomycetes species and nocardia species are filamentous bacteria, with branching segments that often vary as positive or negative on Gram’s staining. Actinomycetes are found in the sulfur granules and not in the surrounding inflammation.

 Cultures of the tissue and abscess specimens grew *A. israelii* and *Aggregatibacter* (formerly *Actinobacillus*) *actinomyctetemcomitans* (Fig. 3E and 3F). Aggregatibacter is a gram-negative facultative nonmotile rod that is frequently isolated together with actinomycetes. It is an oral commensal facultative nonmotile rod that is frequently isolated together with actinomycetes. It is an oral commensal facultative nonmotile rod that is frequently isolated together with actinomycetes.

It is not clear to me in this case whether the pleuropulmonary infection is due to aspiration from the oral cavity or whether the trachea or the soft tissue around the trachea is infected with actinomycetes. The closest analogy that I have found is involvement of tissue around the innominate artery that results in a pseudoaneurysm. The patient had a classic presentation of...
The findings in the soft tissue of the chest wall, indolent clinical progression, absence of fever, signs and symptoms of acute infection, and radiologic evidence of an apparent thoracic mass led to the initial consideration of a malignant process. The diagnosis of thoracic actinomycosis.

Figure 3. Chest-Wall–Biopsy Specimen.
Hematoxylin and eosin staining shows purulent and histiocytic inflammation in adipose tissue and a sulfur granule (dark blue) amid the inflammation (Panel A); there is fibrosis in the adipose tissue that resulted from the histiocytic inflammation (Panel B). Methenamine silver staining of the tissue sample shows the filamentous forms of the organism creating a relatively solid nodule (the sulfur granule) (Panel C); the filamentous forms also can be seen extending from the periphery of the nodule (Panel D). Gram’s staining of smears of the cultures shows gram-positive rods, which were identified as *Actinomyces israelii* (Panel E), and gram-negative rods identified as *Aggregatibacter actinomycetemcomitans* (Panel F). (Panels E and F are courtesy of Dr. JiYeon Kim [Pathology].)
infection was established only at the time of surgical exploration.

Dr. Rosenthal and I were consulted postoperatively, after the Gram’s stain of the chest-wall abscess revealed pleomorphic gram-positive rods. In many cases of actinomycosis, cultures remain negative, perhaps because of previous administration of antibiotics or exposure of the specimen to air. However, in this case, the culture grew not only *A. israelii* but also the fastidious oral gram-negative pathogen *A. actinomycetemcomitans*.

Since the child was known to have virulent pathogens in preoperative respiratory secretions, including methicillin-susceptible *S. aureus* and *Pseudomonas aeruginosa* (in a culture that was obtained on the day of admission), we initiated single-agent parenteral antibiotic therapy with imipenem. A peripherally inserted central catheter was placed for the administration of intravenous antibiotic therapy at home. After nearly 5 weeks of therapy, acute fevers developed, with temperatures exceeding 40°C. The patient was readmitted to this hospital. Blood cultures were negative, and follow-up CT showed resolution of the chest-wall lesions, with residual pleural thickening and improving consolidation in the right lower lobe. Her fever persisted despite discontinuation of imipenem and initiation of ceftriaxone therapy. She defervesced after her therapy was transitioned to oral doxycycline and she underwent removal of the catheter.

The patient had a favorable response to the doxycycline therapy and received a 1-year course of therapy. Three years later, she is currently well, and she and her family are considering reconstructive surgery that might permit removal of the tracheostomy tube.

**Dr. Harris:** Are there questions or comments for any of our discussants? Dr. Moskowitz, do you have any comments?

**Dr. Moskowitz:** I was concerned that there was a polymicrobial component to this infection, but I thought the discussion had gone far enough with actinomyces and elected not to delve into six-syllable coinfectants.

**A Physician:** Because of the upper-airway abnormalities, is this patient at risk for a recurrence of this disease?

**Dr. Pasternack:** This is clearly a very rare complication of tracheostomy, and one would have to think, as Dr. Moskowitz has suggested, that aspiration led to this complication. The patient certainly might be at risk for reaspiration.

**Dr. Harris:** In retrospect, do you think that the episodes of bleeding that occurred 6 weeks before admission, with the finding of consolidation in the right lower lobe on imaging studies, represented the beginning of this process?

**Dr. Pasternack:** I suspect that this was the case. I reviewed the films from the admission at MEEI, and the possibility that this was atelectasis of the right lower lobe was reasonable. The upper-airway symptoms improved, so the imaging finding in the lung was thought to not be related and was not evaluated at follow-up.

## Anatomical Diagnosis

Infection of the chest wall (empyema necessitatis) with *Actinomyces israelii* and *Aggregatibacter actinomycetemcomitans*.

This case was discussed at Pediatric Grand Rounds.

Dr. Moskowitz reports receiving fees for serving on advisory boards from Vertex Pharmaceuticals; consulting fees from EnBiotix, MCIC Vermont, Kala Pharmaceuticals, Pfizer, and Cowen Group; and grant support from Gilead Sciences, Novartis, Rempex Pharmaceuticals, Kala Pharmaceuticals, Vertex Pharmaceuticals, and Savara Pharmaceuticals; as well as holding a pending patent for intermediate-metabolism products that potentiate aminoglycoside antibiotics in bacterial infections (U.S. application number, 61,871,554). Dr. Mark reports providing expert testimony in litigation regarding asbestos exposure. No other potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Drs. Bernard Kinane, Mark Pasternack, and Jessica Rosenthal for assistance with preparing the case history.

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