Case Report

An atypical pathway of infection in an adolescent with a deep neck space abscess

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Deep neck space infections are a potential complication of odontogenic infection, but rarely have been reported in children. In a recent study, 113 pediatric patients were hospitalized with maxillofacial infections with 32 from dental sources; however, the extension of these infections into deep neck spaces was not documented.[1] In a similar study of 67 children with abscess of the head and neck, only 15 cases were determined to have submandibular, lateral pharyngeal, or retropharyngeal space involvement, and only nine were attributed to dental infection.[2] Lateral pharyngeal space infections are more commonly odontogenic in adults; in contrast, the likely predisposing factors in children are nasopharyngitis or adenoiditis.[3] Eight pediatric patients with lateral pharyngeal space infections were identified by Broughton,[4] but no etiological determinations were given.

Early diagnosis and treatment of lateral pharyngeal infections are essential because further progression of infection can be rapid. Severe trismus, limiting direct examination of the oropharyngeal region, often complicates clinical evaluation. In addition, the limited value of conventional roentgenograms in locating the involved deep neck spaces makes definitive diagnosis difficult. This case report describes a 14-year-old African-American female who developed a left peritonsillar and lateral pharyngeal abscess from an odontogenic source. She was managed successfully with appropriate antibiotics and surgical intervention after diagnosis using computerized tomography (CT).

Case report

A previously healthy 14-year-old African-American female reported onset of dental pain in the left mandibular quadrant in 1992. Two weeks later, the dental pain was accompanied by left cheek swelling, dysphagia, odynophagia, and decreased oral intake. Her pediatrician recommended dental treatment for the offending tooth, but the family refused because of the patient's fear of needles. The patient received intramuscular penicillin and an oral aminopenicillin. Two days later she came to Children's Hospital of Wisconsin emergency department with a progression of symptoms and painful left neck swelling.

Upon initial examination, the patient had a temperature of 38.7°C (101.6°F) without evidence of airway distress. The oral cavity exam was limited by severe trismus, allowing only a 1-cm opening. The mandibular left permanent second molar had extensive decay with tenderness to percussion. No swelling or tenderness was present in either the floor of the mouth or in the buccal vestibule. The left tonsil was medially displaced. Externally, a 3-cm-diameter area of induration was present at the angle of the mandible with extension to the left jugulodigastric area. The white blood cell count was 18,200 with a leukocytosis. A lateral neck film indicated a good airway without retropharyngeal soft tissue swelling.

The patient was admitted to the otolaryngology service and intravenous clindamycin was initiated. A neck CT obtained 1 day after admission indicated soft tissue swelling of the left peritonsillar and lateral pharyngeal spaces without evidence of abscess formation. A panoramic radiograph confirmed extensive decay of the mandibular left permanent second molar with periapical radiolucencies consistent with an acute periapical-alveolar abscess. The joint services deferred tooth extraction until the deep neck cellulitis and trismus had resolved.

After 48 hr of parenteral antibiotic therapy, the physical findings remained unchanged and a low-grade fever of 38.4°C (101.1°F) persisted with nocturnal temperature spikes to 39.5°C (103.1°F). Induration became more localized at the left angle of the mandible and the submandibular area. Persistent unchanged trismus precluded a detailed oral exam. Although the involved tooth remained sensitive to manipulation, there were no associated gingival changes. A repeat neck CT scan was obtained to assess lack of clinical response. A large left lateral pharyngeal and peritonsillar abscess was identified with a tract originating from the posterior and inferior left mandibular border (Figs 1 & 2).

Fig 1. CT demonstrating sinus tract (arrow) from posteroinferior mandibular margin to lateral pharyngeal space abscess.

Fig 2. CT illustrating abscess (arrow) of lateral pharyngeal and tonsillar spaces with connection.

The patient had a quinsy tonsillectomy and extraction of the mandibular left permanent second molar under general anesthesia. No other treatment was performed. A bilobed abscess cavity involving the peritonsillar space with extension into the lateral pharyngeal space was confirmed intraoperatively. Cultures grew *Bacteroides melaninogenicus*. Close inspection of the alveolar socket failed to identify a sinus tract. Examination of the remaining dentition revealed minor caries on both maxillary second permanent molars and the mandibular right second permanent molar. The patient had an uneventful recovery, was discharged on the third postoperative day of a 10-day course of oral clindamycin, and had no postoperative sequelae. She was referred to her private dentist for restoration of the other carious teeth.

Discussion

Odontogenic infections tend to spread to soft tissues via planes of least resistance to adjacent potential spaces and must perforate bone before spreading to the deeper fascial spaces. The most common neck spaces involved in mandibular odontogenic infection include the submandibular, masseteric, and sublingual.[5] In the young child, the primary root apices are located superficially and anteriorly within the mandible and usually drain to the buccal aspect of the alveolus intraorally. The permanent first and second molar apices lie deeper and more posteriorly in the mandible and drainage is more complex. Since the lingual bone of the posterior mandible is thinnest in relation to molar apices, infections of the first and second permanent molars tend to drain lingually. The first molar has apices which are located superior to the mylohyoid muscle attachment, and therefore drain to the sublingual space. The second and third molar apices are below the mylohyoid attachment and typically drain to the submandibular space with out sublingual involvement. Therefore, the spread of infection to deep neck spaces through the submandibular space must be anticipated in the teenager.[6] A lateral pharyngeal space infection can occur by extension of the infection from the internal compartment of the masseteric space or, rarely, from the submandibular space.[7]

The lateral pharyngeal space (parapharyngeal space, pharyngomaxillary space) is cone-shaped, with its base at the skull base and its apex at the greater cornu of the hyoid. The styloid process and its muscles divide the lateral pharyngeal space into two compartments: 1) an anterior (prestyloid) space containing connective tissue, muscles, and lymph nodes, and 2) a posterior (retrostyloid) space containing the great vessels and nerves of the neck.[7-9] An abscess of the anterior compartment results in trismus from irritation of the internal pterygoid muscle, medial deviation of the lateral pharyngeal wall, dysphagia, torticollis, swelling over the parotid gland, and possible neck swelling with loss of the space behind the mandibular angle. An abscess of the posterior compartment has no trismus or tonsillar prolapse, but rather parotid swelling and medial deviation of the posterior lateral pharyngeal wall.[8] Respiratory distress can ensue with significant lateral pharyngeal wall swelling.

The peritonsillar space is a potential space defined medially by the fibrous capsule of the palatine tonsil and laterally by the superior constrictor muscle. Breakdown of the tonsillar capsule during tonsillitis is the classic source of infection of this space. Purulent material in the midportion of the peritonsillar space appears to be prone to penetrate the superior constrictor muscle and enter the lateral pharyngeal space. Trismus occurs from reflex irritation of the adjacent muscles of mastication.[10]

Prompt and accurate diagnosis and treatment of deep neck space infections are crucial. The classic symptoms of neck abscesses rarely are present today because oral antibiotics can modify symptoms. Thus the differentiation of diffuse cellulitis from a distinct abscess cavity is complicated. In addition, the progres-

sion of infection may be difficult to ascertain clinically due to significant local edema and trismus. The atypical aspect of this case was the direct involvement of the anterior compartment of the lateral pharyngeal space via a sinus tract in the mandible rather than the more typical intervening masseteric space infection.

The limitations of conventional roentgenograms can hinder prompt diagnosis of head and neck-space infections. CT has been used extensively to identify and manage various maxillofacial pathoses.[11–15] The diagnostic value of CT for head and neck infection include evaluating the degree of displacement of parapharyngeal soft tissues, differentiating between cellulitis and abscess formation, and locating an abscess anatomically.[15] An abscess on a CT scan can be characterized by single or multiple loculations; low density air and/or fluid at the center of the abscess; contrast enhancement of the abscess wall; and tissue edema surrounding the wall and anatomic boundaries that correspond to known fascial planes.[12] In this case, the atypical direct path of infection to the lateral pharyngeal space could not have been detected with conventional roentgenograms. Incorporating a CT allowed prompt determination of abscess formation and delineated the abscess location to facilitate drainage.

Serious complications may develop from a lateral pharyngeal space infection. Contiguous spread to the retropharyngeal space is ominous, often seriously compromising the airway and progressing to mediastinitis due to its inferior communication with the posterior mediastinum. Along with septicemia, other complications of deep neck space infection include jugular thrombosis, hemorrhage of the great vessels, laryngeal edema, osteomyelitis of the mandible, pneumonia, vagal nerve involvement, meningitis, parotid abscess, and aspiration of purulent material following spontaneous rupture of the abscess.[3, 16]

Management of a lateral pharyngeal space infection of odontogenic origin must be prompt and accurate, with removal of the odontogenic infection nidus and parenteral antibiotic therapy. In cases of diffuse cellulitis without localization, parenteral antibiotics alone often are curative.[17] Surgical intervention is advised if no improvement is seen after 48–72 hr of parenteral antibiotic therapy. Exploration and drainage of the abscess and removal of the offending tooth should then be accomplished at the same time. In one recent study, more than 50% of children with deep neck infections did not require surgical drainage.[4] The eight nonsurgical patients in this study became afebrile in a mean of 3.8 days (range 1–7 days) after instituting antibiotic therapy. The author's recommended therapy paradigm utilizes neck CT for differentiation of cellulitis from abscess and the presence or absence of respiratory distress to determine the need for early surgical incision and drainage.

The microbiology of deep space neck infections continues to evolve. In 1983, *Streptococci* (61%) and *Staphylococcus* species (32%) comprised the majority of pure cultures in maxillofacial infections.[18] More recently, Brook[2] found that the most common isolates in pediatric head and neck infections are *Staphylococcus aureus* (aerobic) and *Bacteroides* species. In patients with dental infections as the predisposing factor, anaerobic isolates either as pure cultures or in a mixed flora predominate. This is consistent with the observation that anaerobic bacteria outnumber aerobic species in the oral cavity by 10 to one.[19]

Greater resistance of bacteria to antibiotics, especially penicillin, has been another trend. Recent findings could preclude penicillin as an automatic choice in head and neck infections. Beta-lactamase-producing organisms can survive penicillin therapy and may even protect penicillin-susceptible organisms by enzymatic release within the abscess. Brook[2] found beta-lactamase activity in 46% of head and neck infections in children. Halula et al.[20] also described specific mechanisms for the transfer of clindamycin resistance in *Bacteroides* species.

Penicillin and clindamycin have been shown to produce similarly good results in treating odontogenic infections when the rate of penicillin resistance among oral anaerobic bacteria is relatively low. Penicillin is felt by some authors to still be the antibiotic of choice for maxillofacial infections of odontogenic origin.[1, 21] Clindamycin is indicated in cases of penicillin allergy, recent exposure to penicillin, failure of an infection to respond to penicillin therapy, or in cases where the known pathogen is *Bacteroides fragilis*.[21]

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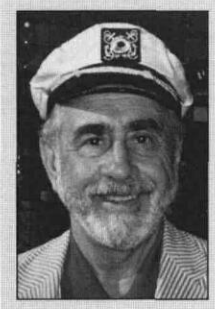
M. MICHAEL COHEN, SR., DMD, 1905–1994

His legacy was pediatric stomatology



M. Michael Cohen, Sr.

Dr. M. Michael Cohen, Sr., an internationally renowned clinician, researcher, and teacher in pediatric stomatology, was born in Boston in 1905 and died in Cambridge, Mass., on October 17, 1994. He graduated from Tufts in 1928, practiced dentistry in Boston and Brookline, and was affiliated with Beth Israel Hospital, Boston Floating Hospital, New England Center Hospital, Children's Hospital Medical Center, Eunice Kennedy Shriver Center, and Lakeville Hospital.

Most of Dr. Cohen's academic career was at Tufts University School of Dental Medicine. After his retirement from Tufts in 1971, he joined the faculty of Harvard School of Dental Medicine, where in 1978 the Michael Cohen, Sr. Teaching and Research Fund was established to help support the departments of pediatric dentistry and oral medicine and oral pathology. He was the 1982 recipient of the American Society of Dentistry for Children's Award of Excellence.

Dr. Cohen was the first dentist in the U.S. to hold professorial rank in a pediatrics department in a medical school (Tufts University) and the first in the U.S. to be appointed stomatologist at a pediatric hospital (Boston Floating Hospital). Together with Dr. Manuel Album, he was instrumental in establishing the Academy of Dentistry for the Handicapped (now the Academy of Dentistry for Persons with Disabilities).

He was also instrumental in establishing the Massachusetts Cerebral Palsy Association and was its first President. He was a member of the Massachusetts Committee on Childhood and Youth and a Consultant in Dentistry for Children to the U.S. Public Health Service. He served as President of the Brookline Community Council and presided over a wide range of activities during the 1950s. He was a member of numerous organizations, including the International Association for Dental Research, Society for Research for Child Development, American Diabetic Association, New York Academy of Sciences, and the Royal Society of Health (UK). He was a Diplomate of the American Board of Pedodontics and held Fellowship in the American Public Health Association and American Association for the Advancement of Science.

His first book, *Pediatric Dentistry*, went through two editions in 1957 and 1961. The first edition was translated into Spanish. His second book, *Minor Tooth Movement in the Growing Child*, appeared in 1977 and was translated into Italian, Portuguese, and Japanese.

Dr. Cohen's academic legacy was pediatric stomatology. He was the first dentist to look at the mouth as a window of pediatric disease and lectured widely on the subject. Early papers dealt with juvenile diabetes, ectodermal dysplasia, the oral manifestations of erythroblastic anemia, dental development in pituitary dwarfism, dental age in the adrenogenital syndrome, the dentition in congenital syphilis and rubella syndrome, and various aspects of trisomy 21 syndrome. For years, he taught oral manifestations of systemic disease to pediatrics house staff and dental students.

Dr. M. Michael Cohen, Sr. was truly a man for all seasons who reached out to help others. His academic legacy lives on in the endeavors of his students and the memories of the many people he influenced. He will be greatly missed by all those whose lives he touched.