Scientific Article

Comparison of odontogenic and nonodontogenic facial cellulitis in a pediatric hospital population

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Abstract

Facial cellulitis in the pediatric hospital population can be classified as odontogenic and nonodontogenic. Emergency departments welcome timely diagnosis from consultants as cellulitis is associated with significant morbidity in children. The purpose of this retrospective study is to assist pediatric dentists in recognizing differences between odontogenic and nonodontogenic facial cellulitis and to determine whether odontogenic infections make up a major portion of facial swellings seen upon admission to the hospital. The completed medical records of 100 patients admitted to Children's Hospital of Pittsburgh from 1980–1989 with an ICD-9 diagnosis of facial cellulitis were reviewed. The types of cellulitis were differentiated using admission data. The information reviewed included age, sex, temperature, white blood cell count, location of facial infection, and season of the year. Odontogenic cellulitis comprised approximately 50% of the total hospital facial infections of the records reviewed during the 10-year period. Upon admission, patients with odontogenic and nonodontogenic facial cellulitis have similarities (season of onset during the year, febrile temperature, and location of infection) and differences (mean admission temperature, age at time of affliction, white blood cell count, and most commonly occurring microorganisms). (Pediatr Dent 19:476–79)

Facial cellulitis in the pediatric population is a problem frequently seen in hospital emergency rooms. Patients presenting with facial cellulitis can be dehydrated and have impaired central nervous system responsiveness, airway obstruction, and systemic illness. Children with such infections are often seen by several medical consultants before a diagnosis is made. Prior to admitting patients, hospital medical services should seek consultation from pediatric dentists to enhance diagnostic capabilities. Delineating pediatric odontogenic from nonodontogenic facial cellulitis presents a challenge to both dental and medical personnel.

In this study, odontogenic cellulitis refers to orofacial infections arising or resulting from the dentition and its adjacent supporting periodontal structures. The infection then disseminates beyond its source, e.g., dentoalveolar abscess, and into the surrounding connective tissues. The purpose of this retrospective study is to 1) determine whether odontogenic infections make up a major portion of hospital facial cellulitis seen upon admission and 2) assist dental consultants in differentiating odontogenic from nonodontogenic facial cellulitis by identifying differences and similarities between the two types of facial infection.

Methods and Materials

The medical records of patients admitted to Children's Hospital of Pittsburgh from 1980 to 1989 with an ICD-9 diagnosis of infection from the oral cavity, jaws, maxillary ethmoid and frontal sinuses, nasal cavity, or facial skin were reviewed at random. One investigator compiled data sheets on the first 100 charts containing complete admission information and a clinician's statement that the patient had facial cellulitis. Admission data reviewed included age, sex, temperature, white blood cell count, location of facial infection, and season of the year. All temperatures were converted to axillary recordings by subtracting 2°C from rectal and 1°C from oral temperature chartings for standardization.[1] Axillary temperatures greater than 36.5°C were considered febrile. The cellulitis location was divided into upper and lower facial regions based on the admitting clinician's (dentist or physician) description. The infection location scheme was modified from that used by Dodson.[2] Upper facial cellulitis consisted of a swelling emanating from the tissues of the frontal, maxillary, or nasal regions or maxillary dentition. Lower facial cellulitis included swelling emanating from the mandible, floor of the mouth, or mandibular dentition. A cellulitis originating from the neck, eyes, or ears was excluded from our study.

Season was defined as summer (June through August), autumn (September through November), winter (December through February), and spring (March through May). Additional data reviewed were source of infection, culture results, and white blood cell count. Origin or source of infection was assigned an odonto-

genic or nonodontogenic label. Documented cultures were collected by aspiration, leading edge, blood, urine, cerebral spinal fluid, or incision for aerobic and anaerobic tests. Results were evaluated using the Statview statistical package (Abacus Concepts, Berkeley, CA). Statistical analyses included the Kruskal-Wallis (equivalent to a one-way analysis of variance by ranks) and chi-square nonparametric tests.

Results

The total sample of 100 patients was divided into odontogenic or nonodontogenic origin of facial cellulitis. The odontogenic group contained 47 patients (22 males and 25 females; 30 white and 17 black); the nonodontogenic group contained 53 patients (30 males and 23 females; 44 white and 9 black) (Table 1). There was no statistical difference between cellulitis groups regarding gender or race.

Analysis of mean axillary temperature upon admission revealed a significant temperature difference between the two groups. The nonodontogenic group (38.4°C) had a significantly higher mean temperature than the odontogenic group (37.5°C) at the time of admission ($P \leq 0.0001$).

Admissions for both types of cellulitis peaked in the spring ($P \leq 0.0001$, Table 2), and the majority of facial infections were located in the upper facial area. Spring accounted for 39% of total annual facial cellulitis admissions. Upper facial infections accounted for 65% of the conclusive data. For the odontogenic group, 71% of the cases were in the upper face. In the nonodontogenic group, 92% of the cases were located in the upper face.

The range of patients' ages extended from approximately 2 months to 19 years. The mean age for the odontogenic group was 8.8 years, significantly older than the 4.4 years mean age for the nonodontogenic group ($P \leq 0.0001$).

In the nonodontogenic group, 74% had an elevated white blood cell count (> 15 000 WBC/mm^3). In the odontogenic group, 43% experienced leukocytosis. This difference was statistically significant at the $P \leq 0.0009$ level.

The three microorganisms most commonly isolated from the odontogenic group were alpha streptococcus (47%), Neisseria (28%), and diphtheroids (26%). The most frequently cultured microorganisms in the nonodontogenic cellulitis were *Hemophilus influenza* type B (36%), alpha streptococcus (17%), and coagulase-negative staphylococcus (15%).

Discussion

The review of completed charts revealed that odontogenic infections can constitute a large portion of facial cellulitis in pediatric hospital patients (47%). This study supports the finding of an earlier investigation, which reported that upper facial infections outnumber lower facial infections in the pediatric hospital population.[2] Of the conclusive data, upper facial infections outnumbered lower facial infections by more than 4:1 in our investigation. When cellulitis did occur in the lower facial region, odontogenic cellulitis predominated—13 cases (81%) compared with 3 cases (19%) of nonodontogenic infection. Nineteen cases, however, could not be differentiated as to location.

TABLE 1. ODONTOGENTIC VERSES NONODONTOGENIC CELLUTLITIS

Parameter	*Odontogenic* N = 47	*Nonodontogenic* N = 53	*Significance Between Types*
Gender	M = 22 F = 25	M = 30 F = 23	n.s.
Race	White = 30 Black = 17	White = 44 Black = 9	n.s.
Age	Mean = 8.8 SD ± 4.4 Range 0.2 to 19.1 years	Mean = 4.4 SD ± 4.7 Range 0.2 months to 16 years	P = < .0001
Temperature	Mean = 37.5°C SD ± 1.1°C	Mean = 38.4°c SD ± 1.1°c	P = < .0001
Dentition	Anterior = 15 (35.7%) Posterior = 27 (64.3%) Missing Observ. = 5		
Leukocytosis	20 (42.6%)	39 (73.6%)	P = < .0009
Microorganisms			
Alpha strep.	22 (46.8%)	9 (17.0%)	
Neisseria	13 (27.6%)		
Diphtheroids	12 (25.5%)		
H. Flu. type B		19 (35.8%)	
Coag. Neg. Staph.		8 (15.1%)	
Location			Total
Upper Right	14	15	29
Upper Left	17	19	36
Total	31	34	65
Lower Right	3	3	6
Lower Left	10	0	10
Total	13	3	16
•Description inconclusive	3	16	19

TABLE 2. SEASONAL DISTRIBUTION OF CASES

Season	Odontogenic N = 47	Nonodontogenic N = 53
Summer	7	9
Fall	10	11
Winter	10	14
Spring*	20	19

*Chi Sq (3DF) $P = < 0.0001$

Although Dodson and associates[2] reported that males are affected more than females, our study found no significant difference between genders or races.

Cultures of nonodontogenic infections revealed three main causative microorganisms: *H. influenza* type B, alpha streptococcus, and coagulase negative staphylococcus. While coagulase-negative staphylococcus may be considered a contaminant, it has been reported to be opportunistic.[3] The three most prevalent odontogenic cellulitis bacteria included alpha streptococcus, Neisseria, and diphtheroids. Facial cellulitis in the odontogenic group was generally secondary to a periapical infection resulting from cariogenic bacteria. The therapeutic implications from this study include the need to treat these two distinct types of facial cellulitis differently in the absence of reliable cultures. An example would be incorporating a ß-lactamase inhibitor in antibiotic therapy when addressing upper facial infections to cover for *H. influenza.*

Leukocytosis (> 15 000 WBC/mm^3) has been associated with *H. influenza* infections[4] and nonodontogenic facial cellulitis.[5,6] Our study also found white cell counts to be elevated more often in the nonodontogenic group.

Both cellulitis types showed febrile (> 36.5°C) axillary mean temperatures. Children with nonodontogenic cellulitis had a greater temperature elevation upon admission to the hospital than did the odontogenic cellulitis patients (38.4°C versus 37.5°C). We may have reduced febrile oral and rectal temperatures too much.[7,8] (One degree Celsius was used to reduce oral to axillary temperature and 2°C to reduce rectal to axillary temperature.) If this were the case, mean febrile temperatures may actually be higher. Febrile rectal temperature elevations greater than 38.5°C can be considered diagnostic for nonfacial *H. influenza* infections and may suggest *H. influenza* for facial infections.[4]

Odontogenic cellulitis generally occurred in older children (mean 8.8 years) in contrast to nonodontogenic infections (4.4 years). It is interesting that the peak age of incidence for invasive *H. influenza* disease occurs between 6 and 12 months. During the 1980s, only the polysaccharide influenza vaccine (licensed in April 1985 in the United States) was available for infants older than 17 months. The immunogenicity and protective efficacy of the vaccine were poor for children younger than 24 months. The more efficacious conjugated *H. influenza* type B vaccine was not available to younger infants prior to October 1990 in the United States (HibTiter, Lederle-Praxis).[9] This fact may account for the lower mean age in the nonodontogenic group as more than one-third of the cellulitis resulted from *H. influenza.* Increasing use of the *H. influenza* vaccine may necessitate further evaluation of the data presented. Additional investigation will also be needed to determine whether a shift in the bacterial mix of cellulitis cases occurs in immunized patients.

The seasonal variation and preference of posterior teeth being responsible for the highest number of cellulitis cases could not be explained from our data. However, a seasonal preponderance for spring was noted in contrast to a previous study by Chartland.[10] Seasonal shifts may correlate with nonodontogenic sources of infection such as *H. influenza.* This bacillus is transmitted via secretory droplets of the respiratory system. Children in day care may be exposed to the bacterium in closed quarters during late winter and express symptoms that require hospitalization in spring. Posterior teeth may be affected more than the remaining dentition because of narrow root canal systems that, when infected, may be susceptible to thrombosis that leads to pulpal ischemia, necrosis, and periapical infection.

Retrospective studies provide baseline data for further investigation and treatment recommendations. However, they have limitations. Limitations in our study include results from only one children's hospital, multiple culture sites and techniques, interpretations from multiple practitioners, and possible incorrect diagnoses. Incomplete medical chart data restricted review of all charts during the evaluation period.

The baseline data in this study, which consulting dental clinicians can use, include both similarities and differences in odontogenic and nonodontogenic facial infections. Similarities suggest that odontogenic infection should not be omitted when contemplating a differential diagnosis for pediatric facial infection. For example, the highest number of odontogenic as well as nonodontogenic infection cases occur in spring and usually in the upper face. A lower facial swelling is usually secondary to dental disease. In addition, both nonodontogenic and odontogenic forms of cellulitis can be febrile on admission. Practitioners encountering young febrile patients with upper facial infections should entertain the possibility of *H. influenza.* This is important in the precooperative age group where radiographic evidence required to rule out odontogenic infection may not be possible because of behavioral considerations.

Differences can assist dental consultants in discriminating the two types of facial cellulitis. Odontogenic facial infections are more likely to affect children with mixed dentition, whereas nonodontogenic cellulitis is more common in younger children prior to the eruption of teeth or

in the primary dentition. White blood cell count (> 15 000/mm^3) and axillary temperatures (> 36.5°C) are higher in nonodontogenic facial cellulitis.

Conclusions

Odontogenic facial infection composed almost 50% of facial cellulitis during the 1980s at Children's Hospital of Pittsburgh. Hence, odontogenic facial infections should not be excluded in the differential diagnosis. Clinical differences between nonodontogenic and odontogenic facial cellulitis are:

1. Dental facial infection has a propensity for older children of mixed dentition age ($P \leq 0.0001$)
2. Nonodontogenic cases generally have white blood cell counts > 15 000/mm^3 ($P \leq 0.0009$)
3. The most commonly occurring organisms differ, with *H. influenza* type B (36%) being found in the nonodontogenic group and alpha streptococcus (47%) in the odontogenic group
4. Nonodontogenic febrile axillary temperatures are likely to be higher than the odontogenic group ($P \leq 0.0001$).

The two forms of facial cellulitis have three elements in common.

1. All had mean febrile axillary temperatures on admission (>36.5°C)
2. The greatest number of cases occur in spring ($P \leq 0.0001$)
3. Cellulitis is more likely to afflict the upper facial region (65%).

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