Journal of Pediatric Sciences

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Journal of Pediatric Sciences 2016;8:e252
http://dx.doi.org/10.17334/jps.78460

How to cite this article:

Stallings MW. Buccal Cellulitis in 3 Infants. Journal of Pediatric Sciences. 2016;8:e52

CASE REPORT

Buccal Cellulitis in 3 Infants

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Abstract:

Three patients are reported with buccal cellulitis. All were between 4 months and 6 months of age. Two patients had received two sets of immunizations, the other patient had received three sets, but the third set was only three days prior to onset of illness. This is an age where protective maternal antibodies would be absent or at a low level. Active immunization with Pentacel is effective after three injections starting at two months of age. All infants were using a pacifier with this thin hard plastic "wings" that contacted the skin sites where cellulitis developed. A break in the skin surface predisposes to development of cellulitis. It is felt that their unprotected antibody status and probable minor skin abrasion from the pacifier wings contributed to their acquiring buccal cellulitis. Antibiotic selection, immunization schedules, and pacifier usage were discussed.

Keywords: *Buccal Cellulitis, pacifier usage, Immunizations*

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Case series

Patient One is a 4 month old black female who had a small red rash on her left cheek for two days prior to being seen. The night previously it had become warm and hard feeling. There had been no recent respiratory infection or known exposure to sickness. There had been no fever. She did use a pacifier, and the mother had noted previously that this left cheek area had been red at times. The past medical history was normal. She had received one Pentacel and one Prevnar 13 vaccine at 2 months of age. On physical exam she was nontoxic with temperature of 99 degrees. There was a discrete 2 ½ by 3 ½ centimeter area of redness of her left cheek that was warm , very indurated and hard, with no fluctuance . The rest of the physical exam was normal. It was noted that the distal left wing on her pacifier was overlying the area of cellulitis and that the pacifier rim was of a thin hard plastic construction. She was given ceftriaxone 500mg (60mg/kg) IM and began on Amoxicillin-clavulanate (600/42.9), 2.5 mls twice a day which is 75mg per kg per day of amoxicillin. The next day the area of redness was smaller and less red with smaller area of induration. Four days later only 1 cm of redness and ¾ x 1 ½ cm of induration remained. She completed ten days of oral antibiotic, and the area resolved. However, eleven days after finishing her antibiotic treatment, she became fussy, and the left cheek area was warm to touch and slightly pink. She was restarted on the amoxicillin-clavulanate (600/42.9) at 72mg/kg per day of

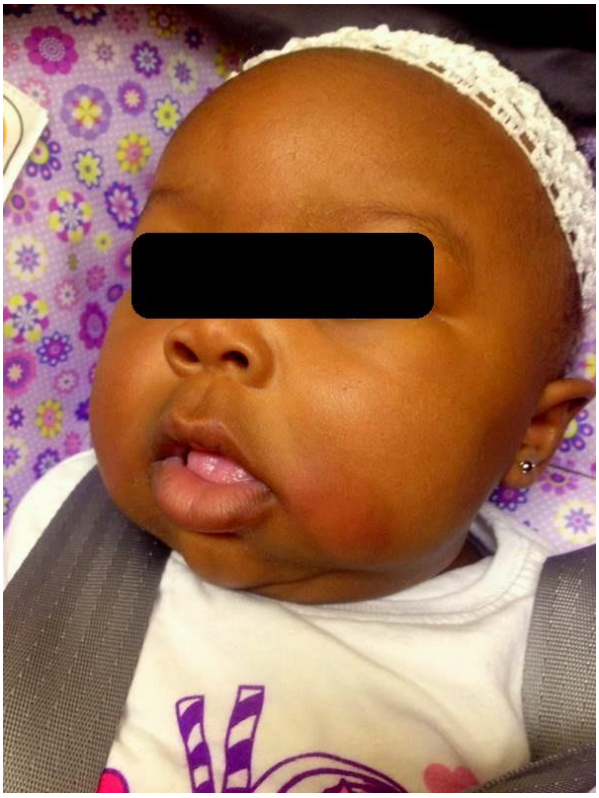

Figure 1. Hard induration and redness in patient one

amoxicillin component. The next day she was afebrile with 1 ½ cm of hard induration and redness which resulted in crying when palpated (Figure 1). Ceftriaxone 500mg IM was given. The next day there was no redness nor increased heat, and the area of induration was smaller. Ceftriaxone 500mg IM was given, and she was begun on cefdinir 125mg daily (15mg/kg per day) as she was refusing the amoxicillin-clavulanate. Five days after the recurrence there was no redness nor induration, but the area had slight hyperpigmentation. She completed ten days of cefdinir and has remained well.

Patient Two is a 6 month old black male with a one day history of a rash on his left cheek. Hewas afebrile with a head cold and was fussy. Physical exam revealed a nontoxic infant with a 1 ½ cm area of redness on his left cheek, with hard induration underlying it, slightly lateral to the corner of his mouth. There was no fluctuance. Bilateral otitis media with thick middle ear fluid was present. He was given an injection of ceftriaxone 500mg IM (62mg/kg) and started on amoxicillin-clavulanate (600 /42.9), 2.5 ml twice a day, (75mg/kg per day of amoxicillin). He also used a pacifier with a thin hard plastic rim. The next day revealed improvement in the cellulitis with less redness and slightly smaller area of induration. One week later there was no redness nor induration, but was slightly hyperpigmented. He completed ten days of the amoxicillin-clavulanate. However, two days after finishing this, he developed red areas on both cheeks. These increased, in size and he developed tactile fever one day later. He was given acetaminophen 80mg. The next day, his exam revealed red areas of both cheeks, 2x3 cm in size. These areas were bright red and had hard induration beneath them (Figure 2). These areas were warm to touch and tender with no fluctuance. His tympanic membranes where normal as was the rest of his exam, and he was nontoxic. He was given ceftriaxone 500mg IM. The next day these areas where smaller, and he continued Daily ceftriaxone for 2 more days. Cefidinir 125mg per day (15mg/kg per day) was

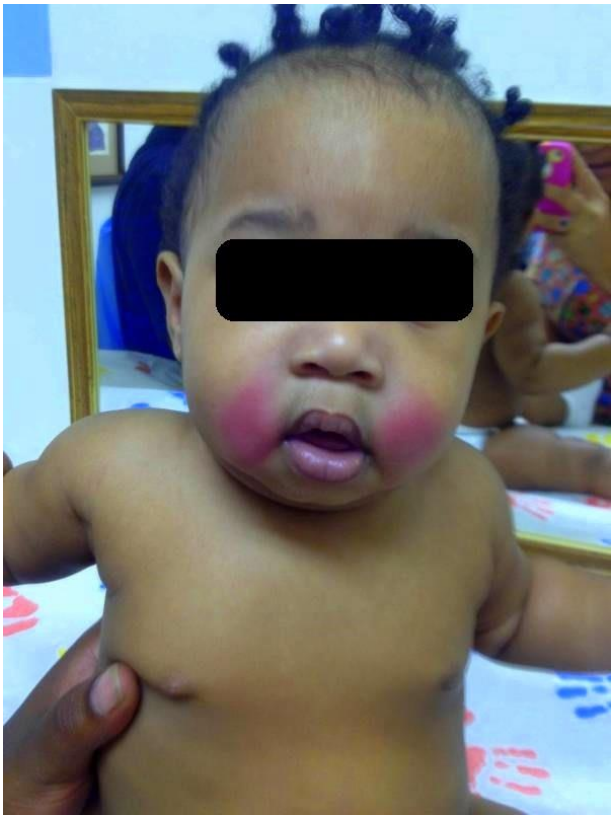

Figure 2. The bright red areas on the cheeks and hard induration beneath them

started then. An NP culture taken prior to antibiotics grew a Streptococcus pneumoniae and a Moraxella catarrhalis, β lactamase positive. A Throat culture was taken and was negative for Streptococcus. A mixed flora grew that unfortunately was not identified. After thirteen days of treatment the induration and redness had resolved and only hyperpigmentation remained. He had received 2 sets of immunizations prior to onset of this illness including Pentacel and Prevnar13.

Patient Three is a black female who had Bilateral Otitis Media at her six month exam with thick middle ear fluid and no motion on pneumatoscope exam. No rash nor fever were present. She was started on Cefdinir 125mg /5ml, 2.5ml twice a day (16mg/kg/day). She also received her third set of immunizations, including Prevnar and Pentacel. Three days later, she developed red areas on both cheeks. Mother thought that the "pacifier hard plastic had rubbed cheeks". On exam, there was a 1 ½ x 1 ½ cm red area with deep firm induration just lateral to right corner of mouth, which was tender to touch. No fever was present. The middle ears still had straw colored, thick middle ear fluid with no motion. The left cheek was normal. She was given Ceftriaxone 500mg IM (66mg/kg) and begun on Amoxicillin/clavulanate (600/42.9) per 5ml, 2 ½ ml twice a day, (80mg/kg/day of amoxicillin). Patient was continued on daily Ceftriaxone 500mg IM. Two days later, the cheek was less red and firm but still indurated. Nine days after diagnosis, there was no buccal redness nor induration.

Discussion

Prior to effective vaccines, Haemophilus influenza, type b, was the most common cause of buccal cellulitis, and a frequent cause of epiglottitis, sinusitis, orbital cellulitis, meningitis, pneumonia and otitis media. Fortunately serious bacterial infections from Haemophilus influenza, type b are now very unusual (1). Adequate immunization produces a protective anti-PRP titer. A titer ≥ 0.15 mcg per ml is considered adequate to provide short term protection against invasive disease and to reduce colonization of the throat and nasopharynx (2). In unprotected infants, this periodical colonization can lead to infections as buccal cellulites, epiglottis, sinusitis, orbital cellulitis, and otitis media (3,4). It is thought that bacteremia from the upper respitory tract leads to seeding and infection of distal sites. A break in the skin due to previous trauma, surgery, or underlying skin lesion predisposes to cellulitis at any site (4). Interestingly, these infants used pacifiers with a thin hard plastic rim. The site of the buccal cellulitis was where the "wing" of the pacifier rim contacted the cheek surface. If H. influenza organisms colonize the nasopharynx and throat, it is possible that these bacteria would be in nasal secretions and saliva. These organisms could then be rubbed into the cheek surface. The thin hard plastic rim of the pacifier could cause microscopic disruptions of the epithelial surfaces, making it susceptible to bacterial inoculation and infection. As these infants had not received their full immunizations due to their age, they were still susceptible to Haemophilus influenza, as well as other bacteria. Protective Maternal antibodies would have decreased by this age, and the vaccinations which they received would not have led to protective level of anti-PRP. These patients received Pentacel vaccines which would require 3 doses to achieve a level ≥ to 0.15mcg per ml of anti-PRP titer (5).

Cellulitis is a clinical diagnosis manifested as an area of edema, warmth, erythema and tenderness. Aspiration of the cellulitis margins yields the best results in obtaining causative bacteria, but still is only occasionally successful (4). Bacteremia can be a complication of cellulitis. Positive blood cultures would be less likely due to the short time from onset of cellulitis to parental treatment in these cases. Other occasional causes of buccal cellulitis are Streptococcus pneumonia and Staphylococcus aureus (4). Also, since the introduction of effective Hib vaccines, there has been an increase in nontypable Haemophilus influenza, type b invasive infections (5,6). Up to 63% of H.

flu infections were due to nontypable H flu in 2006 in the United States (7). There has also been an increase in Haemophilus influenza infections in adults who may not have received vaccinations as infants or who's naturally acquired immunity has waned. Adults with diabetes, renal failure, COPD and myeloma are at particular high risk (8).

Recurrence of Buccal cellulitis would be an extremely uncommon event. One wonders if the decreased amount of clavulanate potassium in the 600mg amoxicillin component per 5ml preparation would be less effective against B-lactamase producers. It is one half the ratio that is in the 200 and 400 amoxicillin component preparations. Resistance of Haemophilus influenza is usually by beta lactamase production but can occur by other mechanisms (5).

Conclusion

Clinicians need to be aware that H. flu type b can still be a cause of serious infections in unprotected infants. A window of vulnerability exists when maternal antibodies decline and active immunization hasn't reached protective levels. Some HIB vaccines provide protective anti-PRP titers with two injections two months apart. In treating bacterial infections in not fully immunized infants, an antibiotic effective against Haemophilus influenza should be selected. Inoculation of pathogenic bacteria into a skin surface that has been compromised seems a more likely event than a child becoming bacteremic from nasal carriage and then seeding to the buccal surface, as opposed to all other skin surfaces. The use of pacifiers with a hard thin plastic rim should be discouraged.

References

1. Fisher RG, Benjamin Jr DK. Facial cellulitis in childhood: A changing Spectrum. South Med J. 2002; 95 : 672-4
2. Hallander HO, Lepp T, Ljungman M, Netterlid E, Andersson M. Do we need a booster of Hib vaccine after primary vaccination? A study on anti-Hib seroprevalence in Sweden 5 and 15 years after the introduction of universal Hib vaccination related to notifications of invasive disease. APMIS. 2010;11:878-887
3. Barbour LB. Conjugate Vaccines and the carriage of Haemophilus influenza Type b. J Infect Dis. 1984;149:1034
4. Nelson Textbook of Pediatrics. 19th Edition. P. 940-2, 2300-1.
5. Anderson P. The protective level of serum antibodies to the capsular polysaccharide of Haemaphilus Influenza type b. J Infect Dis. 1984;149:1034.
6. Dworkin MS, Park L, Borchardt SM. The changing epidemiology of invasive Haemophilus influenza disease, esp. in persons over 65yrs old. Clin Infect Dis.2007; 44:810-816.
7. Center for Disease Control and Prevention Active Bacterial core surveillance (ABCs) Report, emerging infections Program Network, Haemophilus influenza, 2006. Http://www.cdc.gov/abcs/reportsfindings /surveports// hib 06.pdf.
8. Nix EB, Hawdon N. Risk of invasive Haemophilus influenza type (Hib) Disease in adults. CVI 2012; 19.5:766-771.