

Review Article

Impetigo in the Pediatric Population

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Submitted: 23 November 2016

Accepted: 04 February 2017

Published: 07 February 2017

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OPEN ACCESS**Abstract**

Impetigo is an endemic bacterial skin infection most commonly associated with the pediatric population; it is seen in more than an estimated 162 million children between the ages of 2 and 5 years old. Geographically, this infection is mostly found in tropical areas around the globe. Impetigo has the largest increase in incidence rate, as compared to other various skin infections seen in children. The major characteristic observed in this infection is lesions. They first appear as bullae that eventually form a honey-colored, thick crust that may cause pruritus. There are three forms of impetigo: bullous, non-bullous and ecthyma. The primary causative organisms for impetigo include *Staphylococcus aureus* and Group-A β -hemolytic streptococci (GABHS). Most impetigo infections resolve without requiring medication; however, to reduce the duration and spread of the disease, topical and oral antibiotic agents are utilized. A positive prognosis as well as minimal complications are associated with this disease state.

Keywords

- Impetigo
- Non-bullous impetigo
- Bullous impetigo
- Pediatric population
- *Staphylococcus aureus*
- Group-A β -hemolytic streptococci (GABHS)
- Topical antibiotics
- Systemic antibiotics
- Oral antibiotics

ABBREVIATIONS

SSTI: Skin and Soft Tissue Infection; GABHS: Group-A β -hemolytic Streptococci; SSSS: Staphylococcal Scalded Skin Syndrome; CA-MRSA: Community-Acquired Methicillin-Resistant *Staphylococcus aureus*; PVL: Panton-Valentine-Leucocidin; PSGN: Poststreptococcal Glomerulonephritis

INTRODUCTION

The evolution of bacteria and the widespread distribution of antibiotic-resistance have continued to increase and further validate the inevitable post-antibiotic era that has penetrated the consciousness of the healthcare world. Skin and soft tissue (SSTI) infections are a clinical priority, in part, because of the disproportionate effect that they have on the most vulnerable populations [1]. Impetigo is a highly communicable superficial skin infection commonly caused by gram-positive bacteria that includes either *Staphylococcus aureus* or a Group-A β -hemolytic streptococci (GABHS), such as *Streptococcus pyogenes*. Both organisms have been influential in the pervasive spread of bacterial resistance [2,3]. It is predominantly a pediatric infection that tends to occur in environments with hot, humid weather [4,5]. A global study on the population prevalence of impetigo concluded that more than an estimated 162 million children between the ages of two and five years old have suffered from the disease. The study showed that these children tend to reside in low-income countries located in tropical regions [4,6-8].

PATHOPHYSIOLOGY

Skin serves as the first line of defense between humans and their environment [9]. An imbalance of homeostasis between the

skin's microbiome and host has been associated with disease. Different factors responsible for the unique variability of the skin microbiome are only partly understood, but results suggest that host genetic and environmental influences play a major role [10,11]. Naturally, skin is colonized by a diverse assortment of bacteria [12]. Infections resulting from high concentrations of bacteria are rare due to the skin's own natural protective abilities. The human body's natural resistance to infection is due to both the skin and subcutaneous tissues' low pH of approximately 5.6, as well as sebaceous fluids that hydrolyze to form free fatty acids that strongly inhibit the growth of many bacteria and fungi, and skin possessing its own normal flora, helping to prevent the colonization of other pathogenic organisms [13]. Bacterial antimicrobial peptides, also called bacteriocins, are thought to be produced by many or most bacteria found in normal flora and plays a Major beneficial role in the relationship between bacteria and the skin. Bacteriocins do not protect against infection in the traditional sense; they contribute to the survival of individual bacterial cells by killing other bacteria that might compete for nutrients in the same environment [14,15]. Another example of a beneficial relationship between bacteria and the skin involves the innate capacity of the epithelium to detect microorganisms with Toll-like receptors (TLRs). Stimulation of TLRs induces distinct patterns of gene expression that lead to activation of a variety of immune responses. Traditionally, these immune responses were considered to be exclusively pro-inflammatory and designed to defend against the microbe causing infection [16]. However, under certain conditions pathogens can penetrate the integumentary barrier of a susceptible host and may cause tissue damage that may stimulate an inflammatory response. Conditions that may predispose a patient to the development

of skin infections include chickenpox, herpes simplex, insect bites, pediculosis, radiation therapy, scabies, scratching, surgery, thermal burns, trauma, high concentrations of bacteria, excessive moisture of the skin, an inadequate blood supply, the availability of bacterial nutrients, and damage to the corneal layer allowing for bacterial penetration [5,17-19]. The development of impetigo is dependent on the following three factors: bacterial adherence to host cells, invasion of tissue with evasion of host defenses, and the dissemination of toxins [13]. Bacterial invasion occurs initially in low numbers, with teichoic acid mediating adhesion in either of the impetigo causing microorganisms [20,21]. However, fibronectin, a cell adhesion molecule that allows for bacterial cells such as *Streptococcus pyogenes* (GABHS), to attach to collagen and invade disrupted skin surfaces is required in order to colonize skin [13]. In contrast, *Staphylococcus aureus* colonizes the nasal epithelium first and from this reservoir, colonization of the skin occurs [22]. As bacteria increase in number where the integumentary barrier is disrupted, invasion by these colonizing bacteria ensues and impetigo may develop. With the majority of SSTIs resulting from the dismantling of normal host defenses by processes such as skin puncture, abrasion, or underlying diseases, it must be understood that the nature and severity of the infection depends on both the type of microorganism present and the site of inoculation [9,17,19].

Etiology & Epidemiology

In general, the main causative pathogens of impetigo are *Staphylococcus aureus* and Group-A β -hemolytic streptococci (GABHS) [23]. Less common pathogens associated with impetigo include Group C streptococci, Group G streptococci, and anaerobic bacteria [23,24]. When focusing on the different types of impetigo however, there is a clear delineation of which pathogens predominate, as impetigo can be separated into non-bullous impetigo and bullous impetigo.

Non-bullous impetigo, also known as impetigo contagiosa [24,25] or pyoderma [23], is currently caused mostly by *S. aureus*. Following *S. aureus* are mixed infections of staphylococci and streptococci, and then streptococci alone. However, this has not always been the case. Over time the main causative agent has alternated between *S. aureus* and GABHS. According to Koning S et al., in moderate climates, *S. aureus* was the predominant causative organism in the 1940s and 1950s, after which GABHS became more prevalent; in the past two decades, *S. aureus* has become more common again. *S. aureus* alone or in combination with GABHS is responsible for about 80% of impetigo cases [25,26]. To further validate the higher prevalence of *S. aureus*, according to data from the Dermatology Department of Heim Pál Children's Hospital Budapest, more than 70% of the cases are caused by *S. aureus*, 20-25% are caused by a mixed infection of staphylococci and streptococci, and 5-10% of the cases are caused only by streptococci [27]. In contrast, there are instances when streptococcal infection is more common, such as in warmer, more humid climates [24,25].

Bullous impetigo is almost exclusively caused by strains of *S. aureus* that produce exfoliative toxins that cause a loss of cell adhesion in the superficial epidermis by targeting desmoglein1 [23,28,29]. The specific toxin is exfoliative toxin A, as opposed to exfoliative toxin B, which is produced by *S. aureus* in

Staphylococcal Scalded Skin Syndrome (SSSS) [26]. There are a small percentage of infections caused by GABHS [26].

Ecthyma, which is described either as a deeper form of impetigo [30], or as a separate but similar type of infection [31], extends through the epidermis and reaches the deep dermis. Similar to bullous impetigo, the primary pathogen is *S. aureus* [32], but streptococci may sometimes be the cause [27,31].

The presence of MRSA as the causative agent of community-acquired impetigo is considered unusual and heterogeneous [26]. *Staphylococcal* induced impetigo is usually caused by *S. aureus* strains that possess the exfoliative toxin gene. Community-acquired methicillin-resistant *Staphylococcal aureus* (CA-MRSA) do not possess the exfoliative toxin gene, but instead have the Panton-Valentine-Leucodin (PVL) gene. *Staphylococci* that possess PVL usually cause abscesses and furuncles; therefore, concern of MRSA should be less in cases of impetigo [26]. Furthermore, no studies have identified a problem with MRSA-related impetigo in adults or children, but cultures may still be useful in some settings [24]. However, if present, MRSA that is associated with impetigo is usually seen in the non-bullous form [29].

CLINICAL PRESENTATION

Non-bullous impetigo may occur as a primary or secondary bacterial infection. Primary infection occurs via direct bacterial invasion of intact healthy skin [24]. Secondary infection, which is more common, occurs via bacterial infection of disrupted skin caused by trauma, eczema, insect bites, scabies, herpetic outbreaks, or other diseases [24]. Regardless of its primary or secondary nature, non-bullous impetigo initially presents as a maculopapular lesion that becomes a thin-walled vesicle located on an erythematous base. The vesicles tend to be <0.5cm as opposed to the bullae seen in bullous impetigo, which are typically >0.5cm [29]. Upon rupturing, the subsequent superficial ulceration is covered with purulent discharge that dries as a yellowish or honey colored crust [24,26]. The infection tends to occur in exposed areas, especially on the limbs and the face (e.g., nares, perioral region). Satellite lesions, caused by self-inoculation, are frequent; and regional lymphadenopathy is common [23,26]. However, systemic symptoms are unlikely [23-25] although fever can occur in severe cases [26]. Non-bullous impetigo tends to heal without scarring, and if left untreated, it may resolve spontaneously in 2-3 weeks [24-26].

Bullous impetigo initially starts as small vesicles, which become localized flaccid bullae or blisters measuring about 2cm in diameter; the blisters contain clear content that later becomes purulent [26]. These blisters do not rupture as easily as the vesicles seen in non-bullous impetigo, and may persist for several days [25]. Once the blister ruptures, the wet, erythematous base can be seen. Regional enlarged lymph nodes are usually absent, and systemic symptoms are uncommon but can include fever, diarrhea, and weakness [24,26]. The evidence supporting the presence of regional lymphadenopathy in bullous impetigo is conflicting. Some articles state that lymphadenopathy rarely occurs in bullous impetigo [26,33], while others do not mention lymphadenopathy in regards to bullous impetigo [24,25]. In contrast, one source states that lymphadenopathy is more

common in bullous impetigo than non-bullous impetigo [34]. The infection tends to occur on the trunk; the intertriginous regions such as the diaper area, axillae, and neck; and the extremities. However, other cutaneous areas can be affected as well [24,26]. As with the other form of impetigo, the infection generally resolves within 2-3 weeks without scarring [24].

Ecthyma is characterized by vesicles that rupture to produce circular, erythematous ulcers with adherent brown-black crusts. There is usually surrounding erythematous edema [30-32]. Itching is common and scratching can spread the infection [32]. Ecthyma mainly occurs on the legs and gluteus after a trauma or when insect bites have been scratched open [27]. In addition, unlike impetigo ecthyma heals with scarring.

DIAGNOSIS

Diagnosis of impetigo or ecthyma is typically completed via visual observation and clinical findings [29,32]. Gram stains and culture of the pus or exudates from skin lesions and ecthyma are recommended to help identify whether *S. aureus* and/or GABHS is the cause; treatment without these studies however, is reasonable in typical cases [31]. In addition, in patients suspected of having acute glomerulonephritis following impetigo, analysis of elevated anti-DNase B levels can provide supporting evidence of previous streptococcal infection [23].

When diagnosing impetigo, it must be remembered that there can be other disorders that present with similar clinical manifestations. With non-bullous impetigo, diagnostic confusion can occur with a variety of skin disorders including shingles, cold sores, cutaneous fungal infections, and eczema [25]. With bullous impetigo, diagnostic confusion can occur with thermal burns, blistering disorders such as bullous pemphigoid, and Stevens Johnson syndrome [25].

THERAPY

There are various treatment options that may be utilized in the treatment of impetigo. Depending on the severity of the condition, the options range from topical disinfectants to topical and oral antibiotics. For uncomplicated impetigo, it has been observed that the infection is self-limiting and will resolve within a few weeks without scarring [29]. Because impetigo is highly contagious, it is best to treat with medications as opposed to only observation.

For local wound care, it is recommended that lesions are gently cleansed, remove the crusts of non-bullous impetigo using antibacterial soap and a washcloth, and frequent application of wet dressings to the affected areas [26]. Topical disinfectants are a good option for prevention of recurrence and serve as a great alternative to topical antibiotics for treatment. It has been demonstrated in studies that sodium hypochlorite baths have been effective at eradicating multiple community-acquired MRSA strains [35]. Although topical disinfectants are effective for prophylaxis, this option is not recommended for the treatment of impetigo in its active form. It is not recommended that this option be used in active disease.

In a review performed by the Cochrane Database of Systemic Reviews, it was noted that topical antibiotics showed better

cure rates than topical placebo [25]. Topical mupirocin has demonstrated superior efficacy to the oral agent erythromycin and has been proven to be a viable option in patients with erythromycin-resistant strains of *Staphylococcus aureus*. In other comparisons, cure rates between topical and oral agents show no significant difference [25]. According to Infectious Diseases Society of America guidelines, non-bullous and bullous impetigo can be treated with oral or topical antimicrobials for a duration of five to seven days, but oral therapy is recommended in patients with numerous lesions or outbreaks affecting several people to help mitigate the transmission of infection [31].

TOPICAL ANTIBIOTICS

Topical antibiotics are a viable option for limited impetigo disease. They are less likely to promote bacterial resistance and have less side effects. For infants, children, and adolescents in the United States, mupirocin and retapamulin are the two topical agents most commonly used (Table 1) [14,29]. Mupirocin inhibits bacterial protein synthesis by reversibly and specifically binding to transfer-RNA synthetase. It has tolerable side effects such as application site reactions (pruritus and stinging of skin) [14]. Retapamulin exhibits its action by inhibiting bacterial protein synthesis at the 50S ribosomal unit [14]. Due to the risk of epistaxis, retapamulin should not be used on the nasal mucosa [36]. Fusidic acid is also a common topical agent used worldwide, but it is not FDA approved in the United States.

Over-the-counter: Triple antibiotics (bacitracin-neomycin-polymixin) have some activity against the organisms, but they are not as effective for treatment [37]. Bacitracin and neomycin have also been known to cause contact dermatitis. These agents are not recommended for the treatment of impetigo.

SYSTEMIC ANTIBIOTICS

For lesions that are widespread, it is recommended that patients use oral antibiotics. Preferred agents for pediatric population include different types of penicillins and cephalosporins (Table 1); both of these classes inhibit bacterial cell wall synthesis. Dicloxacillin and cephalexin are used in infants, children, and adolescents (Table 1). If MRSA is suspected or confirmed by a culture and sensitivity test, the preferred antibiotics are clindamycin, doxycycline, or trimethoprim-sulfamethoxazole. Doxycycline is a tetracycline that exerts its function by inhibiting bacterial protein synthesis. It is not recommended in children less than 8 years of age due to teeth discoloration. Neonatal patients with bullous form of impetigo can be treated with nafcillin, oxacillin, or clindamycin. Erythromycin, a macrolide, is the drug of choice for neonates with non-bullous impetigo (Table 1). Intravenous vancomycin is recommended for MRSA cases [29]. Systemic antimicrobial agents are indicated when there is involvement of deep tissue structures, fever, lymphadenopathy, pharyngitis, infections near the oral cavity, and infections on the scalp and/or numerous lesions [26].

COMPLICATIONS

Complications are rare, but local and systemic spread of infection may result in cellulitis, lymphangitis, septicemia, guttate psoriasis, scarlet fever, and poststreptococcal glomerulonephritis (PSGN) [25]. PSGN is one of the most serious complications

Table 1: Pediatric Impetigo Treatment Regimens.

Medications	Age	Directions
<i>Topical Agents</i>		
Mupirocin 2% Ointment (Bactroban)	• Children ≥ 2 mo:	• Apply to lesions 2-3 times daily for 7-10 days
Retapamulin 1% Ointment (Altabax)	• Children ≥ 9 mo:	• Apply to affected areas twice daily for 5 days
<i>Systemic Agents</i>		
Amoxicillin-Clavulanic Acid (Augmentin)	• Infants < 3 mo: • Children ≥ 3 mo and < 40 kg • Children ≥ 40 kg	• 30 mg/kg/day po divided q12h x 10 days • 30 -90 mg/kg/day po divided q8-12h x 10 days • 250-500 mg po divided q8h or 875 mg q12h x 10 days
Cefuroxime (Ceftin, Zinacef)	• Infants and children 3 mo-12y • Children ≤ 12 yr • Adolescents	• 30 mg/kg/day (max 1 g/day) divided q12h x 10 days (suspension) • 250 mg q12h x 10 days (tablet) • 250-500 mg q12h x 10 days (tablet)
Cephalexin (Keflex)	• Children ≥ 1 yr:	• 25-100 mg/kg/day in divided doses every 6-8 x 10 days; Max 4 g/day
Dicloxacillin	• Neonates • Children < 40 kg Children • Children ≥ 40 kg	• Not recommended • 12.5-100 mg/kg/day divided q6h x 5-7 days • 125-250 mg q6h x 5-7 days
Erythromycin (E.E.S. 400, E.E.S. Granules, Ery-tab, EryPed 200, EryPed 400, Erythrocin Stearate)	Weight Based	• Erythromycin base: 30-50 mg/kg/day in 2-4 divided doses x 5-7 days; Max 2 g/day • Erythromycin ethylsuccinate: 30-50 mg/kg/day in 2-4 divided doses x 5-7 days; Max 3.2 g/day • Erythromycin base stearate: 30-50 mg/kg/day in 2-4 divided doses x 5-7 days; Max 2 g/day

and tends to occur as a result of streptococcal impetigo more commonly than streptococcal throat infection [25,26,32]. PSGN can occur in up to 5% of patients with non-bullous impetigo and tends to manifest approximately 2 weeks after infection [24,29]. Symptoms can include swelling in the face, especially around the eyes, oliguria, hematuria, and increased blood pressure. Most patients tend to recover without permanent kidney damage, but PSGN can lead to chronic kidney disease [29]. Currently there is no data to indicate that treating impetigo has any effect on preventing the development of acute PSGN [23-26,29]; however, treatment does reduce the dissemination of nephritogenic strains in the human population [23,25,26].

PROGNOSIS

Impetigo generally heals within 2-3 weeks, even without treatment. In randomized trials, it was demonstrated that in the placebo arms approximately 13% to 52% had spontaneous resolution within 7 to 10 days[14]. However, a higher cure rate is seen with the use of medications and it reduces the risk of spreading the infection[25,38].

PREVENTION

Proper hygiene is essential in the prevention of impetigo; washing hands with warm water and antibacterial soap and bathing regularly should help reduce chance of infection. Nails should be kept clipped and filed in order to avoid auto-inoculation by scratching sores. Patients infected with impetigo should use clean towels and washcloths every time [29]. Children may return to school 24 hours after beginning an effective antibiotic regimen, and draining lesions should be kept covered [39]. To limit the contamination of fomites, parents or guardians should wash children's toys and use disinfectant wipes on hard surfaces.

Also, parents should follow-up with a primary care physician if they notice that the lesion is continuing to spread, the sore is not beginning to heal, or the child is developing systemic symptoms [29].

DISCUSSION & CONCLUSION

Impetigo is a highly contagious bacterial infection that mainly affects children between the ages of 2 to 5; however, people of any age can acquire the infection. Impetigo is principally caused by *Staphylococcus aureus*, and *Streptococcus pyogenes* (GABHS). Diagnosis is generally made through visual observation of clinical manifestations; cultures may be drawn to tailor treatment. Limited infection is treated with topical antibiotics such as Mupirocin or Retapamulin, while more extensive disease is treated with oral or IV antibiotics. Although rare, complications are possible; however, most cases of impetigo resolve completely without complications.

REFERENCES

1. Hay RJ, Johns NE, Williams HC, Bolliger IW, Dellavalle RP, Margolis DJ, et al. The Global Burden of Skin Disease in 2010: An Analysis of the Prevalence and Impact of skin conditions. *J Clin Investig Dermatol*. 2014; 134: 1527-1534.
2. Fish DN. Chapter 42. Skin and soft tissue infections. In: Wofford MR, Posey LM, Linn WD, O'Keefe ME, eds. *Pharmacotherapy in Primary Care*. New York, NY: McGraw-Hill; 2009.
3. Mahé AH, Hay RJ. *Epidemiology and Management of Common Skin Diseases in Children in Developing Countries*. Geneva: World Health Organization; 2005.
4. Steer AC, Jenney AWJ, Kado JH, Batzloff MR, La Vincente S, Waqatakirewa L, et al. High Burden of Impetigo and Scabies in a Tropical Country. *Lancet Infect Dis*. 2009; 3.

5. RothRR, James WD. Microbial Ecology of the Skin. *Anna Rev Microbial*. 1988; 42: 441-464.
6. Bowen AC, Mahé AH, Hay RJ, Andrews RM, Steer AC, Tong SY, et al. The global epidemiology of impetigo: a systematic review of the population prevalence of impetigo and pyoderma. *PLoS One*. 2015; 10.
7. Carapetis JR, Steer AC, Mulholland EK, Weber MW. The Global Burden of Group A Streptococcal Diseases. *Lancet Infect Dis*. 2005; 5: 685-694.
8. Andrews RM, McCarthy JS, Carapetis JR, Currie BJ. Skin disorders, including pyoderma, scabies, and tinea infections. *Pediatr Clin North Am*. 2009; 56: 1421-1440.
9. Cogen AL, Nizet V, Gallo RL. Skin Microbiota: A Source of Disease or Defence? *Br J Dermatol*. 2008; 158: 442-455.
10. Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. *Nature*. 2012; 486: 207-214.
11. Costello EK, Lauber CL, Hamady M, Fierer N, Gordon JI, Knight R. Bacterial community variation in human body habitats across space and time. *Science*. 2009; 326: 1694-1697.
12. Hancock RE, Lehrer R. Cationic peptides: a new source of antibiotics. *Trends Biotechnol*. 1998; 16: 82-88.
13. McAdam AJ, Sharpe AH. Infectious diseases – bacterial infections. In: Kumar V, Abbas AK, Fausto N, editors. *Robbins & Cotran Pathologic Basis of Disease*. Philadelphia: Elsevier Inc; 2005; 371-396.
14. Klaenhammer TR. Bacteriocins of lactic acid bacteria. *Biochimie*. 1988; 70: 337-349.
15. Riley MA. Molecular mechanisms of bacteriocin evolution. *Annu Rev Genet*. 1998; 32: 255-278.
16. Lai Y, Di Nardo A, Nakatsuji T, Leichter A, Yang Y, Cogen AL, et al. Commensal bacteria regulate Toll-like receptor 3-dependent inflammation after skin injury. *Nat Med*. 2009; 15: 1377-1382.
17. Gould D. Skin flora: Implications for nursing. *Nurs Stand*. 2012; 26: 48-56.
18. Granato PA. Pathogenic and Indigenous Microorganisms of Humans. In: Murray PR, Baron EJ, Jorgensen JH, et al., eds. *Manual of Clinical Microbiology*, 9th ed. Washington, DC: ASM Press 2007; 46-56.
19. Reichel M, Heisig P, Kampf G. Identification of Variables for Aerobic Bacterial Density at Clinically Relevant Skin Sites. *J Hosp Infect*. 2011; 78: 5-10.
20. Weidenmaier C, A Peschel. Teichoic Acids and Related Cell-Wall Glycopolymers in Gram-Positive Physiology and Host Interactions. *Nat Rev Microbiol*. 2008; 6: 276-287.
21. Sutcliffe IC, N Shaw. Atypical Lipoteichoic Acids of Gram-Positive Bacteria. *J Bacteriol*. 1991; 173: 7065-7069.
22. Darmstadt GL, Lane AT. Impetigo: an overview. *Pediatr Dermatol*. 1994; 11: 293-303.
23. Stevens DL, Bryant AE. Impetigo, Erysipelas and Cellulitis. 2016 Feb 10. In: Ferretti JJ, Stevens DL, Fischetti VA, editors. *Streptococcus pyogenes: Basic Biology to Clinical Manifestations* [Internet]. Oklahoma City (OK): University of Oklahoma Health Sciences Center; 2016.
24. Hartman-Adams H, Banvard C, Juckett G. Impetigo: diagnosis and treatment. *Am Fam Physician*. 2014; 90: 229-235.
25. Koning S, van der Sande R, Verhagen AP, van Suijlekom-Smit LW, Morris AD, Butler CC, et al. Interventions for impetigo. *Cochrane Database Syst Rev*. 2012; 1.
26. Pereira LB. Impetigo-review. *An Bras Dermatol*. 2014; 89: 293-299.
27. Zitás É, Mészáros J. The most common childhood skin diseases. *Our Dermatol Online*. 2016; 7: 213-218.
28. Amagai M, Matsuyoshi N, Wang ZH, Andl C, Stanley JR. Toxin in bullous impetigo and staphylococcal scalded-skin syndrome targets desmoglein1. *Nat Med*. 2000; 6: 1275-1277.
29. Sahraoui S, Akiyode O. Impetigo in Children and Adolescents. *US Pharm*. 2013; 38: 68-71.
30. Davis S. Impetigo: a review with a focus on retapamulin. *S Afr Pharm J*. 2015; 82: 22-25.
31. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2014; 59: 10-52.
32. Behesti M, Ghotbi Sh. Impetigo, a brief review. *Shiraz E-Medical Journal*. 2007; 8: 138-141.
33. Dagan R. Impetigo in childhood: changing epidemiology and new treatments. *Pediatric Annals*. 1993; 22: 235-240.
34. NHS. Health A-Z. Impetigo. Overview.
35. Fisher RG, Chain RL, Hair PS, Cunnion KM. Hypochlorite killing of community-acquired methicillin-resistant *Staphylococcus aureus*. *Pediatr Infect Dis J*. 2008; 27: 934-935.
36. Altanax (retapamulin) ointment. FDA Medwatch. September 2010.
37. Bass JW, Chan DS, Creamer KM, Thompson MW, Malone FJ, Becker TM, et al. Comparison of oral cephalexin, topical mupirocin and topical bacitracin for treatment of impetigo. *Pediatr Infect Dis J*. 1997; 16: 708-710.
38. George A, Rubin G. A systematic review and meta-analysis of treatments for impetigo. *Br J Gen Pract*. 2003; 53: 480-487.
39. Baddour. Impetigo. Up to Date.
40. Impetigo Treatment & Management. Medscape.

Cite this article

Ghazvini P, Treadwell P, Woodberry K, Nerette E Jr, Powery H II (2017) Impetigo in the Pediatric Population. *J Dermatolog Clin Res* 5(1): 1092.