ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

IMPETIGO, DESCRIPTION, ETIOLOGY, EPIDEMIOLOGY, PATHOPHYSIOLOGY, EVALUATION, DIFFERENTIAL DIAGNOSIS, TREATMENT, PROGNOSIS AND COMPLICATIONS

Alexandra Elizabeth Lozano González[1], Carla Thaylee Pinos Cabrera[2], María Belén Gutama Baculima[3], Andreina Stefania Aguilar Lara[4], Carolina Elizabeth Aguilar Lara[5], Diego Leonardo Moreta Yauli[6], Carolina de la Nube Chasi Inga[7], Ricardo Andrés Vargas Álvarez[8], Andrea Estefanía Cañar Mendes[9], Bryam Esteban Coello García[10]

[1]General Practitioner in "Ministerio de Salud Pública", Faculty of Medical Sciences, Universidad Nacional de Loja. Saraguro- Ecuador ORCID https://orcid.org/0009-0005-1385-5416
[2]General Practitioner in "Hospital Misereor Gualaquiza", Faculty of Medical Sciences, Universidad Católica de Cuenca. Azuay- Ecuador ORCID https://orcid.org/0000-0003-2752-4667
[3]General Practitioner in independent practice, faculty of Medical Sciences, Universidad de Cuenca. Azuay- Ecuador ORCID https://orcid.org/0009-0000-4174-4928
[4]General Practitioner in "U.O San Vicente-Ministerio de Salud Pública", faculty of Medical Sciences, Universidad de Cuenca. Azuay- Ecuador ORCID https://orcid.org/0009-0006-8036-1878
[5]General Practitioner in "Hospital IESS CIEBOS, Centro de Salud Majua", Esmeraldas- Ecuador ORCID https://orcid.org/0009-0005-3382-8845
[6]Postgraduate Doctor in Internal Medicine at Faculdade de Ciências Médicas Minas Gerais. Belo Horizonte - Brasil. ORCID https://orcid.org/0009-0005-0895-2801
[7]General Practitioner in "Hospital Aida León de Rodríguez Lara", faculty of Medical Sciences, Universidad de Cuenca. Azuay- Ecuador ORCID https://orcid.org/0000-0003-3123-4718
[8]Resident physician in "Hospital General Julius Doepfner de Zamora, faculty of Medical Sciences, Universidad de Guayaquil. Guayas-Ecuador ORCID https://orcid.org/0009-0003-1929-6825
[9]Postgraduate in internal medicine in "Hospital das clínicas - Universidade de São Paulo". São Paulo-Brazil. ORCID: https://orcid.org/0000-0003-3205-8074
[10]Postgraduate doctor in orthopedics and traumatology at Faculdade de Ciências Médicas Minas Gerais. Belo Horizonte - Brasil. ORCID https://orcid.org/0000-0003-2497-0274

Corresponding Author: *Bryam Esteban Coello García,* ***Address:*** *Rua Tiradentes 266.Campo Belo. Minas Gerais. Brasil,* ***Postal Code:*** *37270-000*
Article DOI: https://doi.org/10.36713/epra13948
DOI No: 10.36713/epra13948

SUMMARY

Introduction: *Impetigo is a common infection of the superficial layers of the epidermis that is highly contagious and usually originated by gram-positive bacteria, this disease manifests with the presence of erythematous plaques with a yellow crust, which may become itchy or painful. Diagnosis is usually based on signs and symptoms alone. Treatment involves topical and oral antibiotics and symptomatic care.*
Objective: to detail current information related to impetigo, description, etiology, epidemiology, pathophysiology, evaluation, differential diagnosis, treatment, prognosis and complications.
Methodology: *a total of 37 articles were analyzed in this review, including review and original articles, as well as clinical cases, of which 28 bibliographies were used because the other articles were not relevant to this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: impetigo, cutaneous bacterial infection, S. aureus, streptococcal, bullous impetigo.*

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

Results: *Impetigo accounts for approximately 10% of skin complaints in the pediatric population and the main pathogens involved include Staphylococcus aureus and Streptococcus pyogenes. There are two common variants of impetigo: non-blistering (70%) and blistering (30%)Streptococcus pyogenes (group A streptococcus) is one of the most important bacterial causes of skin and soft tissue infections (STBI) worldwide. Impetigo is a non-life-threatening infection, but can lead to acute post-streptococcal glomerulonephritis.*

Conclusions: *Impetigo is a common disease in early life, especially in warm and humid climates. The infection can be blistering or non-blistering and is usually caused by Staphylococcus aureus and Streptococcus pyogenes. The infection usually affects the face, but can occur anywhere else on the body with a laceration, abrasion, insect bite, or other trauma. History and physical examination are paramount for diagnosis, which is usually based on symptoms and clinical manifestations alone. Treatment strategies for impetigo differ, depending on whether the condition is localized or generalized, as well as resistance patterns to the causative agents and current guidelines. Topical antibiotics alone or in combination with systemic antibiotics are most frequently used for treatment. The prognosis is usually good and complications are rare.*

KEY WORDS: *impetigo, S. aureus, blistering, cutaneous, skin.*

INTRODUCTION

Normally the dermis is colonized by a large number of bacteria that dwell on its surface or in the hair follicles. Occasionally, excessive growth of these bacteria leads to skin diseases(1,2).

Impetigo is a common infection of the superficial layers of the epidermis that is highly contagious and usually caused by gram-positive bacteria, this disease manifests with the presence of erythematous plaques with a yellow crust, which may become itchy or painful. Diagnosis is usually based on signs and symptoms alone. Treatment involves topical and oral antibiotics, in addition to symptomatic care(3-6).

Primary infection with impetigo and secondary bacterial infection with scabies, presenting Staphylococcus aureus and Streptococcus pyogenes (Group A Streptococcus, GAS) bacteria lead to increased morbidity and mortality rates, as well as increased health care costs. Lesions generated in impetigo are highly contagious and spread easily(3,6).

Some factors will play an important role in the resistance of the infection and will depend on the host, such as the presence of sebaceous secretion (fatty acids, especially oleic acid), lysozyme and defensin production, the integrity of the cutaneous barrier with its acid pH and a decent nutritional status(1,2).

Impetigo is a disease frequently associated with infants living in warm, humid climates. Impetigo infection can be blistering or non-blistering. The infection mostly affects the face, but it can occur elsewhere on the body if there is a laceration, abrasion, insect bite or other trauma. Diagnosis is usually clinical, based on signs and symptoms. For treatment, topical and oral antibiotics are usually used, in addition to symptomatic management(3).

METHODOLOGY

A total of 37 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, of which 28 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: impetigo, cutaneous bacterial infection, S. aureus, streptococcal, bullous impetigo.

The choice of bibliography exposes elements related to impetigo, description, etiology, epidemiology, pathophysiology, evaluation, differential diagnosis, treatment, prognosis and complications.

DEVELOPMENT

Etiology and Epidemiology

Impetigo is a common superficial bacterial infection of the skin, with an approximate worldwide presence of more than 140 million and about 162 million children worldwide according to other bibliographies. This condition makes up about 10% of skin complaints in infants. If we take into account the totality of the age ranges, we can see that this disease presents similar incidence in males and females; however, only in adults, males are more affected. Its presentation in children from 2 to 5 years of age is the most common, although it can be observed at any age. Infants are more affected than adults and the incidence improves with age. In addition, impetigo is more common in summer and autumn. Bullous impetigo is more common in infants; children under 2 years of age account for 90% of individuals affected with bullous impetigo(3,7-11).

Streptococcus pyogenes or group A streptococcus is one of the most notable bacterial sources of skin and soft tissue infections (STBI) worldwide. No other pathogen gives rise to as many varied clinical entities as S. pyogenes. In particular, this bacterium causes infections, with different names depending on the site affected:

- Impetigo: in the superficial keratin layer.
- Erysipelas: in the superficial epidermis.
- Cellulitis: in the subcutaneous tissue.
- Necrotizing fasciitis: in the fascia.
- Myositis and myonecrosis: in the muscle.

It is also the etiologic agent of Streptococcal Toxic Shock Syndrome (StrepTSS) and scarlet fever. Although impetigo is generally not life-threatening, it can lead to acute post-streptococcal glomerulonephritis (AGN)(12-15).

Post-streptococcal glomerulonephritis (PSGN) alters the glomeruli and small blood vessels of the kidneys. It is characterized by a rapid deterioration of renal functions due to a

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

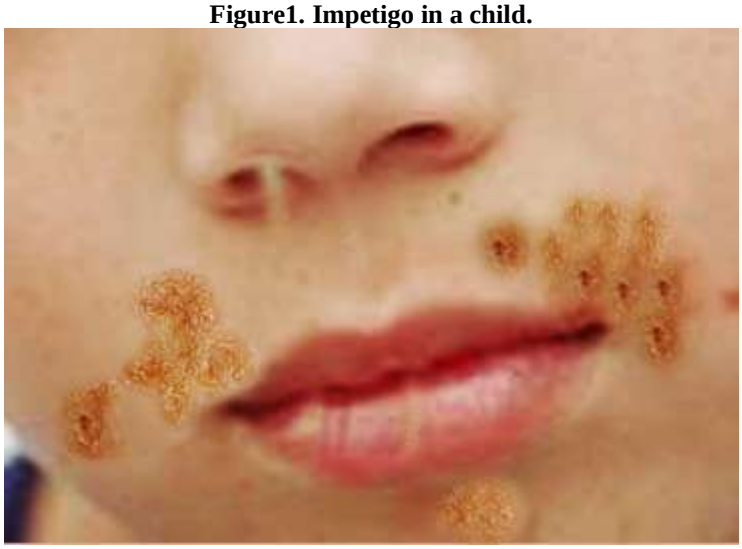
type III hypersensitivity reaction following a streptococcal infection; resulting from specific strains of group A beta-hemolytic streptococci called nephrogenic streptococci. PSGN is most common in children 1 to 2 weeks after a sore throat or 6 weeks after a skin infection such as impetigo. It usually presents clinically compatible with nephritic syndrome, showing hematuria, oliguria, hypertension and edema. Rarely, it may pass as a nephrotic syndrome with significant proteinuria(16).

Among the pathogens primarily involved are Staphylococcus aureus and Streptococcus pyogenes. Two variants of impetigo are frequently presented, non-blistering impetigo representing approximately 70% and blistering impetigo approximately 30%(10,13).

Non-blistering impetigo is commonly generated by S. aureus, which accounts for approximately 80% of the cases. Group A beta-hemolytic streptococcus (GABHS) is attributed to 10% of cases and the causative agent is a combination of S. aureus and GABHS 10% of the time. Methicillin-resistant S. aureus (MRSA) is currently more prevalent, particularly in hospitalized individuals. Community-acquired MRSA is currently increasing rapidly. The condition is more frequent in indoor populations, day-care centers and prisons(3,10).
Bullous impetigo is almost exclusively caused by S. aureus. Occasionally a deep ulcerative infection known as ecthyma may occur, which is a complication of bullous impetigo(3).

Risk factors for impetigo include crowding, young age, close contact, and warm, humid weather. In tropical climatic regions, GAS is considered the main pathogen and co-infection with S. aureus is common, while S. aureus has largely replaced GAS as the predominant pathogen in temperate climates(2,11,17).

Figure1. Impetigo in a child.



Source: image Courtesy S Bhimji MD. Nardi NM, Schaefer TJ. Impetigo(3).

Pathophysiology

Impetigo can be categorized as primary or secondary. Primary impetigo involves previously normal skin afflicted by direct bacterial colonization. Secondary impetigo involves the formation of an infection at a site with a previous skin wound(3). Any modification of the skin barrier leads to the introduction of fibronectin receptors by GABHS and S. aureus which require fibronectin for colonization. Cuts, insect bites, trauma, surgery, burns, atopic dermatitis, lice, herpes, scratching and chickenpox are common mechanisms of disruption of the natural skin barrier and therefore present increased likelihood and susceptibility to develop impetigo.Post-injury, auto-inoculation at other sites is common. Immunosuppression, malnutrition, overcrowding, diabetes, poor hygiene and access to day care centers promote susceptibility to impetigo(3).

Semiology

Non-blistering impetigo usually starts as a vesicle or pustule. Several vesicles coalesce and rupture, subsequently the emanating purulent exudate creates the characteristic honey-colored crust: it also has an erythematous base. There are usually many lesions on the face and extremities, most commonly on surfaces with a disruption of the skin barrier. The high speed of spread and the creation of satellite lesions come after autoinoculation, commonly on surfaces without apparent skin barrier disruption. Mild regional lymphadenopathy is a common related finding. Systemic symptoms, such as fever, are not usually present in non-blister impetigo(3,11).

Bullous impetigo starts with small vesicles that turn into flaccid blisters. The exfoliative toxin A produced by S. aureus generates loss of cell adhesion in the superficial epidermis. The blisters have in their interior a clear or yellow liquid that later becomes purulent or dark. Edema and erythema around the blisters are

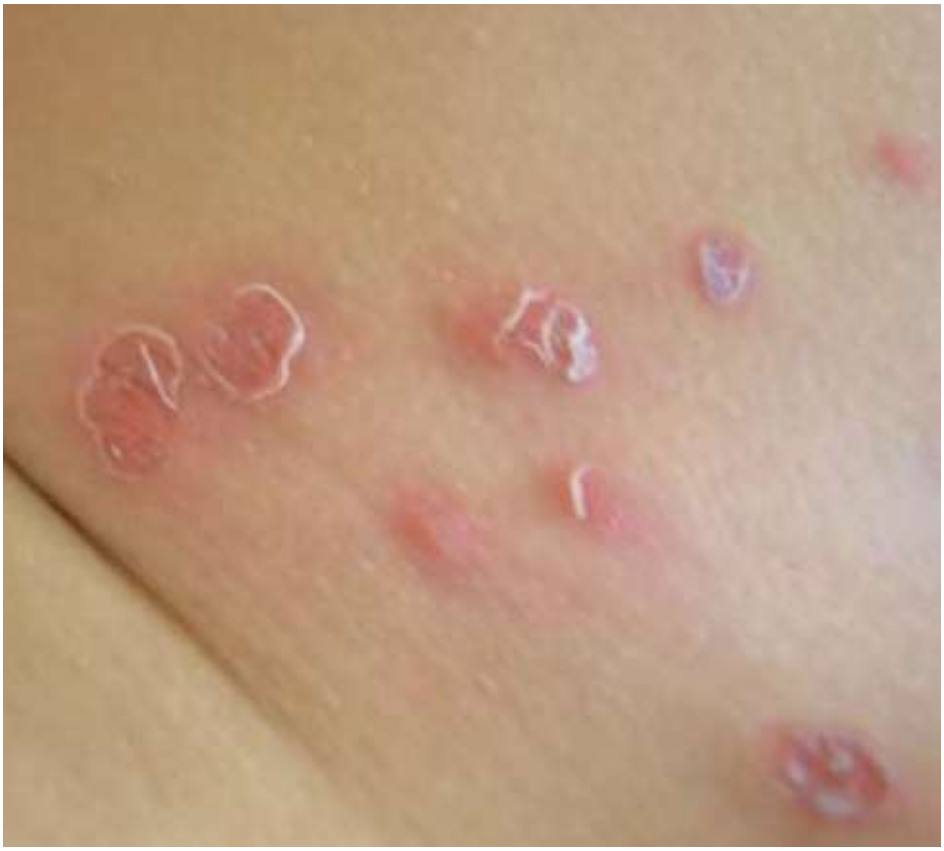
ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

usually absent. After the blisters break, an erythematous base with a ridge remains. Bullous impetigo does not give a honey-colored crust. Lesions usually appear in the intertriginous regions and on the trunk, in contrast to non-blistering impetigo, they may appear on the buccal membranes. Commonly, there are fewer lesions in non-blister impetigo. Regional lymphadenopathy is not present. Systemic symptoms, such as fever, are more common than in non-blister impetigo(3,18-20).

S. aureus produces exfoliative toxins, which are proteases that selectively hydrolyze one of the intracellular adhesion molecules, desmoglein-1, found in the desmosomes of keratinocytes located in the epidermal granular layer. Toxins are the most important virulence factor of S. aureus , generating the separation of epidermal cells with the creation of blisters. The blisters are located in bullous impetigo and spread in scalded skin syndrome. There are at least 2 different variants of exfoliative toxins, so that exfoliative toxin A is linked to bullous impetigo and toxin B to scalded skin syndrome(2,21).

Ecthyma is a deep tissue configuration of impetigo. Ulcerative lesions enter through the epidermis and into the dermis. Ulcers appear as punch lesions with violaceous borders. The crusts may be honey-colored or brownish-black and the lesions may have pus(3).

Figure 2. Bullous Impetigo –Desquamation Collarette and Flaccid Blisters.



Source: Pereira LB. Impetigo - review. An Bras Dermatol(2).

Evaluation

Anamnesis and physical examination are crucial to the correct diagnosis of impetigo, especially inspection, which is classically evidenced by distinctive honey-colored crusty lesions. Bacterial cultures can be used to confirm the diagnosis and should be sought in the presence of evidence of methicillin-resistant staphylococcus aureus (MRSA) or in an outbreak of impetigo. In addition, a skin biopsy may be considered in refractory cases if necessary(3,10).

The anti-streptolysin O (ASO) response is weak for impetigo alone. Therefore, streptococcal antibody serological tests are not often indicated for its diagnosis. However, it can be used when post-streptococcal glomerulonephritis is suspected in an individual with a new outbreak of impetigo(3).

It is important to consider human immunodeficiency virus (HIV) testing when a previously healthy adult individual presents with clinical manifestations of bullous impetigo(3).

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

Treatment and Management

Management strategies for impetigo differ, according to whether the condition is localized or generalized, patterns of resistance to the causative agents, and prevailing models(17).

Topical antibiotics alone or in addition to systemic antibiotics are used to treat the disease. Topical antibiotics are often considered the treatment of choice for impetigo, however the clinical efficacy of these treatments are declining at an alarming rate because of the rapid emergence and spread of resistant bacteria. Systemic antimicrobials are recommended when deeper structures such as subcutaneous tissue and muscle fascia are involved, accompanied by fever, pharyngitis, infections close to the oral cavity, adenopathies, scalp infections and very numerous lesions (2,11).

The antibiotic used should cover the spectrum where S. aureus and S. pyogenes are present. As previously mentioned, impetigo is usually self-limited, however antibiotics reduce the permanence of the disease and the spread of lesions to other areas. In addition, antibiotic treatment reduces the chances of bacterial involvement of the kidneys, bones, joints and lungs, as well as acute rheumatic fever(3,22-24).

When the impetigo is regionalized, uncomplicated and non-blistering, topical therapy is suggested. The scab should be removed with soap and water prior to topical antibiotic therapy. Mupirocin, retapamulin and fusidic acid are the first-line treatments. Treatment for limited impetigo is topical and the use of oral systemic antibiotics is left for more extensive cases. The antibacterial action of Retapamulin is generated through inhibition of protein synthesis by selectively binding to bacterial ribosomes(2,3,10).

Ozenoxacin or ozenoxacin, a striking topical antibacterial agent with strong bactericidal activity against gram-positive bacteria, exists in cream presentations with 1% of active drug to manage impetigo, this drug is effective and well tolerated in individuals 2 months and older. It presents a rapid onset of response and a remarkable clinical and microbiological response compared to placebo. Topical ozenoxacin is an ingenious alternative for the treatment of impetigo(25).

Systemic antibiotics are indicated for all patients with bullous impetigo, as well as for non-blistering impetigo with more than 5 lesions, lymphadenopathy, systemic signs of infection, deep tissue involvement and lesions in the oral cavity. The antibiotics of choice are beta-lactamase resistant antibiotics such as cephalosporins, amoxicillin-clavulanate and dicloxacillin. Cephalexin is frequently used. If there is a positive culture only for streptococci, oral penicillin is the therapy of choice(3,26).

In places with a high prevalence of MRSA, as well as if cultures are positive for MRSA, the antibiotic of choice is clindamycin or doxycycline. The drug trimethoprim-sulfamethoxazole has a commensurate effect against MRSA, but should be used when group A streptococci are not the causative agent, or in conjunction with an anti-streptococcal antibiotic(3).

Antibiotics are currently the first line treatment when multiple lesions are present, however with an increasing prevalence of antibiotic resistant bacteria, the successful management of impetigo in the future is of concern, the widespread use of these antibiotics contributes to antimicrobial resistance and presents its negative effects for individuals and communities.

Some studies agree that new antimicrobials and topical antiseptics are required as an alternative treatment strategy due to this drawback(27).

It is recommended to maintain good personal hygiene in all affected children, in addition to trying not to have contact with other infants during the active outbreak. It is important to emphasize the importance of cleaning hands, bedding, clothing, and affected areas likely to have had contact with infected fluids. Sores can be covered with a bandage to reduce distribution by contact. If the impetigo is reiterative, it is convenient to make an evaluation of the bacterial carriage that gives origin. Frequently the nose acts as a reservoir; carriers can be given mupirocin applied to the nostrils(3).

Differential Diagnosis

- Scabies.
- Herpes simplex.
- Candidiasis.
- Varicella zoster.
- Atopic dermatitis.
- Contact dermatitis.

Prognosis

The disease is most of the time mild and self-limited; however, antimicrobial treatment is frequently started to decrease the spread and improve the clinical course. Without any treatment, the natural course of the disease lasts 14 to 21 days and it is considered cured. Approximately 20% of cases resolve spontaneously. Scarring is infrequent, however some affected individuals may present pigmentary alterations, others may develop ecthyma. With medical treatment, healing takes up to 10 days. Newborns may develop meningitis. An unusual complication is acute post-streptococcal glomerulonephritis, which occurs 2-3 weeks after skin infection. Approximately 5% of the affected individuals with this disease will develop the associated glomerulonephritis, nevertheless at the moment there is no conclusive evidence if antibiotics support or not to diminish the incidence of post-streptococcal glomerulonephritis, this usually can happen after 1 or 2 weeks of the streptococcal infection, presenting clinically fever, hypertension, edema and hematuria(3,10).

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

Complications

Complications are infrequent, however they may occur, some of the affected individuals may present renal failure, it is more common to present with an infection generated by streptococci; the renal alteration is shown 7 to 14 days after infection. The presence of blood in the urine and transient proteinuria may remain for weeks or even months. In addition, complications such as sepsis, meningitis, scarlet fever, septic arthritis and staphylococcal scalded skin syndrome may occur(3,10,28).

CONCLUSIONS

Impetigo is a common disease in early childhood, especially in warm and humid climates. The infection can be blistering or non-blistering and is usually caused by Staphylococcus aureus and Streptococcus pyogenes. The infection usually affects the face, but can occur anywhere else on the body with a laceration, abrasion, insect bite, or other trauma. History and physical examination are paramount for diagnosis, which is usually based on symptoms and clinical manifestations alone. Treatment strategies for impetigo differ, depending on whether the condition is localized or generalized, as well as resistance patterns to the causative agents and current guidelines. Topical antibiotics alone or in combination with systemic antibiotics are most frequently used for treatment. The prognosis is usually good and complications are rare.

BIBLIOGRAPHY

1. *Oumeish I, Oumeish OY, Bataineh O. Acute bacterial skin infections in children. Clin Dermatol. 2000;18(6):667–78.*
2. *Pereira LB. Impetigo - review. An Bras Dermatol. 2014;89(2):293–9.*
3. *Nardi NM, Schaefer TJ. Impetigo. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 Jul 9]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK430974/*
4. *Department of Paediatrics and Adolescent Medicine, The University of Hong Kong, Pokfulam, Hong Kong, Leung TN, Hon K, Leung AK. Group A Streptococcus disease in Hong Kong children: an overview. Hong Kong Med J [Internet]. 2018 Nov 9 [cited 2023 Jul 9]; Available from: http://www.hkmj.org/abstracts/v24n6/593.htm*
5. *Clebak KT, Malone MA. Skin Infections. Prim Care Clin Off Pract. 2018 Sep;45(3):433–54.*
6. *May PJ, Tong SYC, Steer AC, Currie BJ, Andrews RM, Carapetis JR, et al. Treatment, prevention and public health management of impetigo, scabies, crusted scabies and fungal skin infections in endemic populations: a systematic review. Trop Med Int Health. 2019 Mar;24(3):280–93.*
7. *Castro MCR de, Ramos-E-Silva M. Cutaneous infections in the mature patient. Clin Dermatol. 2018;36(2):188–96.*
8. *Sahu JK, Mishra AK. Ozenoxacin: A Novel Drug Discovery for the Treatment of Impetigo. Curr Drug Discov Technol. 2019;16(3):259–64.*
9. *Breyre A, Frazee BW. Skin and Soft Tissue Infections in the Emergency Department. Emerg Med Clin North Am. 2018 Nov;36(4):723–50.*
10. *Johnson MK. Impetigo. Adv Emerg Nurs J. 2020 Oct;42(4):262–9.*
11. *Abrha S, Tesfaye W, Thomas J. Intolerable Burden of Impetigo in Endemic Settings: A Review of the Current State of Play and Future Directions for Alternative Treatments. Antibiotics. 2020 Dec 15;9(12):909.*
12. *Stevens DL, Bryant AE. Streptococcus pyogenes Impetigo, Erysipelas, and Cellulitis. In: Ferretti JJ, Stevens DL, Fischetti VA, editors. Streptococcus pyogenes: Basic Biology to Clinical Manifestations [Internet]. 2nd ed. Oklahoma City (OK): University of Oklahoma Health Sciences Center; 2022 [cited 2023 Jul 9]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK587091/*
13. *Stevens DL, Bryant AE. Impetigo, Erysipelas and Cellulitis. 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 [cited 2023 Jul 22]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK333408/*
14. *Chelsom J, Halstensen A. [Streptococcus group A infections of skin, soft tissue and blood]. Tidsskr Den Nor Laegeforening Tidsskr Prakt Med Ny Raekke. 2001 Nov 20;121(28):3310–4.*
15. *Brouwer S, Barnett TC, Rivera-Hernandez T, Rohde M, Walker MJ. Streptococcus pyogenes adhesion and colonization. FEBS Lett. 2016 Nov;590(21):3739–57.*
16. *Rawla P, Padala SA, Ludhwani D. Poststreptococcal Glomerulonephritis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 Jul 22]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK538255/*
17. *Gahlawat G, Tesfaye W, Bushell M, Abrha S, Peterson GM, Mathew C, et al. Emerging Treatment Strategies for Impetigo in Endemic and Nonendemic Settings: A Systematic Review. Clin Ther. 2021 Jun;43(6):986–1006.*
18. *Bangert S, Levy M, Hebert AA. Bacterial Resistance and Impetigo Treatment Trends: A Review: Impetigo. Pediatr Dermatol. 2012 May;29(3):243–8.*
19. *Sladden MJ, Johnston GA. Common skin infections in children. BMJ. 2004 Jul 10;329(7457):95–9.*
20. *Cole C, Gazewood J. Diagnosis and treatment of impetigo. Am Fam Physician. 2007 Mar 15;75(6):859–64.*
21. *Brazel M, Desai A, Are A, Motaparthi K. Staphylococcal Scalded Skin Syndrome and Bullous Impetigo. Medicina (Mex). 2021 Oct 24;57(11):1157.*
22. *Rush J, Dinulos JG. Childhood skin and soft tissue infections: new discoveries and guidelines regarding the management of bacterial soft tissue infections, molluscum contagiosum, and warts. Curr Opin Pediatr. 2016 Apr;28(2):250–7.*
23. *Smith DRM, Dolk FCK, Pouwels KB, Christie M, Robotham JV, Smieszek T. Defining the appropriateness and inappropriateness of antibiotic prescribing in primary care. J Antimicrob Chemother. 2018 Feb 1;73(suppl_2):ii11–8.*
24. *Loadsman MEN, Verheij TJM, van der Velden AW. Impetigo incidence and treatment: a retrospective study of Dutch routine primary care data. Fam Pract. 2019 Jul 31;36(4):410–6.*
25. *Rosen T, Albareda N, Rosenberg N, Alonso FG, Roth S, Zsolt I, et al. Efficacy and Safety of Ozenoxacin Cream for Treatment of Adult and Pediatric Patients With Impetigo: A Randomized Clinical Trial. JAMA Dermatol. 2018 Jul 1;154(7):806–13.*
26. *Koning S, van der Sande R, Verhagen AP, van Suijlekom-Smit*

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 7| July 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

LWA, Morris AD, Butler CC, et al. Interventions for impetigo. Cochrane Database Syst Rev. 2012 Jan 18;1(1):CD003261.

27. *D'Cunha NM, Peterson GM, Baby KE, Thomas J. Impetigo: A need for new therapies in a world of increasing antimicrobial resistance. J Clin Pharm Ther. 2018 Feb;43(1):150–3.*
28. *Walker MJ, Barnett TC, McArthur JD, Cole JN, Gillen CM, Henningham A, et al. Disease Manifestations and Pathogenic Mechanisms of Group A Streptococcus. Clin Microbiol Rev. 2014 Apr;27(2):264–301.*

Conflict of Interest Statement

The authors report no conflicts of interest.

Funding

The authors report no funding by any organization or company.