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Mini Review

An Insight into Impetigo: A Mini Review

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ABSTRACT

Impetigo is a highly contagious superficial bacterial skin infection commonly affecting infants and young children, particularly in warm, humid, and overcrowded environments. It primarily occurs in two forms: non-bullous impetigo, characterized by honey-colored crusts caused by Staphylococcus aureus and Streptococcus pyogenes, and bullous impetigo, marked by flaccid bullae produced by toxin-mediated epidermal separation. The pathophysiology involves disruption of the skin barrier, bacterial adhesion, toxin release, and inflammatory responses that lead to vesicles, pustules, and crust formation. Impetigo remains a global public health concern with significant disease burden, especially in low-resource settings. Diagnosis is usually clinical, while treatment depends on disease severity and resistance patterns. Topical antibiotics such as mupirocin, fusidic acid, retapamulin, and ozenoxacin are preferred for localized disease, whereas systemic agents are used for extensive or complicated cases. Recurrent infections require decolonization and hygiene measures. Emerging therapies, stewardship strategies, and vaccine research hold promise for improved future management.

INTRODUCTION

The skin forms an important defense barrier against the external environment and pathogens. The skin in children is quite delicate and sensitive and prone to several pathogenic conditions (1). Impetigo is a bacterial skin infection that affects the superficial cutaneous layers in children (2). It is also called school sores, i*nfantigo* and *impetigo contagiosa*. It initially starts as a minor infection and can worsen due to its infective nature (3). It is considered as a public health concern (4). It is classified into two types i.e., non-bullous and bullous (5). Non- bullous can be considered as an early stage of the infection. It is characterized by red sores transforming into honey-colored crust, mostly in chin regions (6,7). It is caused by *Staphylococcus aureus* and related microorganisms such as *Group A β- haemolytic Streptococcus, Methicillin- resistant Streptococcus* or in combination of these organisms. It may be frequently seen in children

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upto 5 years of age (8). Bullous impetigo has a *bullae* or blisters that are cloudy in appearance. The condition has signs of ecthyma at worsened state or severe infection (9). It is more common in children of lesser than 2 years (10,11).

PATHOPHYSIOLOGY

The pathophysiology of impetigo starts from bacterial entry into the epidermis leading to disturbance of the skin barrier. Primary infection is caused by direct bacterial entry into the skin. Secondary infection route can be through sites of wound, cuts etc.(12). The healthy skin comprises a microflora that normally coexists and maintains the functionality of the normal skin (1,13). The pathogenic bacteria change the structural integrity of the skin through degradation mediated by the acidic secretions, bacterial peptides and altered sebum release (14). It is enhanced by minor skin injuries. On crossing the skin, bacteria such as *S. aureus, S. pyogenes* attach firmly to skin cells through adhesive proteins. These proteins stick to host proteins and lead to bacterial colonization. It is enhanced in immunocompromised children. The bacterial colonization prevents skin fall. The release of toxic substances causes the formation of lesions. Neutrophils migrate to the infected site in response to bacterial invasion and release inflammatory mediators that cause vesicular and pustular formation. These fragile vesicles rupture easily and the resulting mixture of serum, neutrophils and bacterial debris- which dries to form the classic honey-colored crust on reddish blood base. Autoinoculation through scratching allows the infection to spread across the skin, often producing lesions even in areas where no visible break in the skin barrier is present (15).

Bullous impetigo, caused due to certain strains of *Staphylococcus aureus*, produce exfoliative toxin A that specifically targets and cleaves desmoglein-1, an adhesion molecule found in the epidermis. The reduction of desmoglein-1 disrupts cellular adhesion and creates a region of separation within the superficial epidermis, leading to the formation of large, flaccid *bullae* filled with clear or yellow fluid. Because the process occurs within the epidermis itself, there is minimal surrounding erythema or inflammation. When the bullae rupture, they leave behind a moist, red base bordered by a fine collar of scale, without forming honey-colored crusts (16,17).

The immunity resistance offered by the host also plays a major role in determining susceptibility to the infection. The acidic pH of the skin nature, lysozymal activity, production of proteins, defensins, fatty acids all contribute to antimicrobial protection. The protection is weakened by humidity, maceration, malnutrition, obesity, immune suppression or comorbidities. Additionally, skin pathogens grow better at warm temperatures and neutral pH level. These conditions are commonly observed in hot and humid climates. These biological and environmental factors set the stage for bacterial entry, multiplication and lesion formation (18,19).

EPIDEMIOLOGY

The global market value of impetigo was valued at USD 1.09 billion in 2024 and is expected to grow to USD 1.8 billion by 2035, at a CAGR of greater than 4% (10,11). Impetigo is a widely prevalent condition, with more than 160 million cases reported annually across diverse geographical regions (20). As per Hawaii Department of Health, USA has about three million cases of impetigo (21). Warm climates in tropical and subtropical regions display disproportionately higher incidence rates due to ideal conditions for bacterial growth, survival and rapid skin barrier degradation (22). Socio-economic factors contribute to the epidemiology of impetigo. Overcrowded living conditions and regions, improper and unclean

sanitation facilities, no water access and low literacy and awareness increases the disease prevalence in affected communities. The impetigo spreads frequently in daycares, schools and institutionalized settings where close physical contact among children facilitates rapid transmission of the pathogens. Climate change, urban density and migratory shifts may further influence global trends of the disease prevalence (23).

Staphylococcus aureus is a ubiquitous organism. The new spread of methicillin-resistant strains has complicated treatment in impetigo cases, leading to greater dependence on culture-guided therapy and modified treatment guidelines. *Streptococcus pyogenes* continues to contribute substantially to disease spread in tropical regions where streptococcal skin infections remain endemic. The global burden of impetigo is not evenly distributed, reflecting the influence of environmental, socioeconomic and health-systems-related factors (24).

CLINICAL FEATURES AND DIAGNOSIS

The clinical presentation of impetigo varies based on bacterial subtype but has a predictable pattern that facilitates early diagnosis. Non-bullous impetigo is the more common form. It typically starts as small red macules or papules that form vesicular structures (25). These vesicles rupture to release serous or purulent fluid, sometimes too viscous. The fluid dries to become a honey-coloured crust. The crust confirms the presence of impetigo. The lesions can appear single or in clusters. The lesions are most commonly distributed on the face, arms and legs. Mild itching or discomfort may be caused during the rash. Bullous impetigo is the less common form. It is formed by larger, flaccid *bullae*, which is filled with clear or yellowish grainy fluid (26). These bullae arise on previously intact skin directly due to bacterial infection without any pre-existing skin conditions or injuries. They rupture and leave behind thin, varnish-like crusts. These crusts are different from the characteristic honey-colored crust seen in the non-bullous form. This bacterial subtype is mostly seen in infants and younger children.

The diagnosis of impetigo is usually clinical, based on the appearance and distribution of lesions on visual examination (27). Laboratory intervention is relevant in recurrent infections, outbreaks, failure of continuous therapy, or when methicillin-resistant *Staphylococcus aureus* is suspected as causative organism (28). The tested swabs may be collected from fresh, unruptured lesions for microbiological culture. Differential diagnosis includes comparative separation from herpes simplex infection, varicella, insect bite reactions, *eczema herpeticum*. It can also be differentiated in severe toxin-mediated cases, staphylococcal scalded skin syndrome among others (29,30).

PRINCIPLES OF MANAGEMENT

The management of impetigo is dependent on severity of lesion, extent of involvement, risk factors, presence and types of symptoms and occurrence of regional resistance patterns. In case of mild and localized disease, implementation of hygienic practices and topical therapy is sufficient (28). Complicated impetigo cases require systemic therapy. Primary management involves gentle cleansing with warm water and soap. The removal of crust facilitates better penetration of the drug through skin barrier. Infected children are usually advised to avoid school or daycare for at least the first 2 days after starting antibiotic therapy or until the crusts dry (31). The preventive methods include avoiding scratching; cutting fingernails, prevent sharing towels or clothing and maintaining clean environment. These measures promote healing, reduce the risk of recurrence and limit the

spread of infection. When impetigo occurs in clusters or institutional settings, public health management becomes necessary (32–34).

DRUG INTERVENTIONS

Topical therapy is the most used drug approach for localized impetigo and primary route of impetigo entry (35,36). Its mechanism directly targets the affected skin region, decreases the systemic exposure and reduces the risk of antibiotic resistance (37). Most commonly used topical interventions are mupirocin and fusidic acid. These drugs have established effectiveness against *Staphylococcus aureus* and *Streptococcus pyogenes* (38). The recent advancements include advent of antibacterial agents such as ozenoxacin and retapamulin (39). These newer topical antibiotics are developed which showcase improved activity against resistant strains, decreased time of action and enhanced bactericidal properties (40). Hydrogen peroxide, a non-antibiotic bactericidal agent, may be less potent than antibiotic agents, but useful for mild cases and for antibiotic resistance management (41). Topical therapy is usually prescribed for five to seven days. The signs of improvement can be seen within two or three days (42). It is characterized by drying of crusts and de-inflammation of lesions.

The factors for anti-impetigo drug choice depend on patient's age, comorbidities, pregnancy or breastfeeding status and possible antimicrobial resistance patterns (43). The factors ensure the therapy is safe, effective and prevent antibacterial resistance. Usually, the systemic therapy is administered for patients with high extent of lesions, blister formation, spread of infection to multiple body areas, fever, lymphadenopathy and associated complications (44). Also, oral antibiotics are used in people who are resistant to topical therapy; in case of community outbreaks, nasal or distant bacterial colonization may be contributing and extensive. First-line drugs include agents active against methicillin-sensitive *Staphylococcus aureus* such as flucloxacillin and dicloxacillin (45). Cephalexin and amoxicillin-clavulanate serve as additional alternatives. For patients with contraindications in case of penicillin allergies, clarithromycin can be considered as the best alternative (46). In regions with high prevalence of methicillin-resistant *Staphylococcus aureus,* clindamycin, doxycycline, or trimethoprim-sulfamethoxazole can be used (47). Normally, a five to seven-day course of oral antibiotics is sufficient (8).

Supportive and palliative measures such as fever reduction, hydration, nutritional maintenance and preventing corticosteroids are important adjuncts to antimicrobial therapy (48). Some of the marketed products to treat impetigo through topical route of administration as discussed in Table 1.

Table 1: Some of the marketed products to treat impetigo

S. No.	Brand name	Active composition	Dosage form	References
1.	T-Bact	Mupirocin 2% w/w	Ointment	(49)
2.	Supirocin	Mupirocin 2% w/w	Ointment	(50)
3.	Fucigold	Fusidic acid 2% w/w	Cream	(51)
4.	Bactroban	Mupirocin 2% w/w	Ointment	(52)
5.	Altabax	Retapamulin 1% w/w	Ointment	(53)
6.	Xepi	Ozenoxacin 1% w/w	Cream	(54)

RECURRENT, COMPLICATED CASES AND PREVENTION

Recurrent impetigo means repeatedly and frequently occurring impetigo infections (55). It poses a greater risk and is due to various factors such as extreme colonization, warm and humid conditions, increased perspiration, inadequate hygiene or pre-exisiting skin disorders. *Staphylococcus aureus* colonization is a known source of repeated infections (56). The techniques to minimize intranasal bacterial colonies using mupirocin and antibacterial bodywashes (57). These methods can be truly useful when approached with changes in household, personal hygiene and surroundings (58). The rare complications can be in the form of cellulitis, lymphadenitis, abscesses, and, glomerulonephritis (59). Early diagnosis can only curb the recurrence of such conditions and prevent these outcomes. High-risk groups include infants, immunocompromised patients should be given extra care. The preventive techniques include maintenance of skin integrity, reducing environmental risk factors. Regular handwashing, avoiding sharing personal things, daily cleaning and managing underlying skin conditions reduces impetigo. Public health campaigns, streetplays, programmes play an important role in affected areas for raising awareness and make people know about early methods of disease cause and spread (60).

CONCLUSION

The management of impetigo is evolving, exposed to antimicrobial resistance, environmental changes and global health. Newer topical antibiotics can offer better efficacy and activity against antibiotic-resistant bacterial strains. Exact target vaccines for impetigo are not currently available (61). There has been research on vaccines targeting *Staphylococcus aureus* and *Streptococcus pyogenes*. StreptAnova is a vaccine that targets Group A *Streptococcus (GAS)* organisms (62). New strategies are trying to improve diagnosis, treatment optimization and trace the antibiotic resistance mechanisms of the bacterial pathogens. It is also important step for antimicrobial stewardship. Thus, impetigo is a clinically underexplored but significant, manageable skin infection. The disease burden is due to various environmental, socioeconomic and public health factors. There is a need for an integrated and balanced approach that includes clean practices, proper topical or systemic treatments and public health measures can significantly reduce its impact. Improved therapeutic options and sustained efforts to stewardship and timely management of impetigo are gaining importance.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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