Journal of Infection (2015) 71, S76—S79

BIAM British Infection Association

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Recent developments in the management of common childhood skin infections

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Accepted 23 April 2015
Available online 29 April 2015

KEYWORDS
Skin infections;
Clinical clues and tricks;
Resistance to antibiotics

Summary A literature review and clinical commentary on diagnosis and treatment of common childhood bacterial, fungal and viral skin infections is presented including impetigo, folliculitis, staphylococcal scalded skin syndrome, tinea capitis, warts and molluscum contagiosum.
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Introduction

At birth the skin is sterile. After one day colonisation occurs with bacterial, viral and fungal commensals. Per cm^2 100—1,000,000 microorganisms are present.[1] Recently the human cutaneous microbiome became in much scientific interest, although Anton van Leeuwenhoek already described it into 1683.[2] The human microbiome is an ecosystem composed of trillions of microorganisms (bacteria, yeast, viruses, fungi etc.), which colonise the stratum corneum. The skin microbiome and consequent biofilms interact by playing a dominant role in the occlusion of sweat ducts, leading to inflammation and pruritus.[3] It was reported that the microbiome or biofilm in AD, rosacea and psoriasis differs from that in normal skin. Probably and presumably this altered microbiome plays a pathogenic role in atopic eczema.[4]

The skin of AD patients is highly colonised by *Staphylococcus aureus* and other staphylococcal species persisting in areas of dry skin as well in mildly affected skin. Those bacteria form a biofilm. The current hypothesis is that staphyloccocal antigens act as super-antigens causing exacerbations of eczema. Therefore, increased staphylococci colonization/infection may be responsible for flares and reducing bacterial load may result in clinical improvement. This may also explain why regular baths of diluted bleach (sodium hypochlorite) and intranasal application of mupirocin ointment reduces the severity of atopic dermatitis in children.

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http://dx.doi.org/10.1016/j.jinf.2015.04.030
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In this article a short overview of the common bacterial and other common infections of the skin is given together with an update of developments.

Impetigo: general aspects and resistance

Impetigo is a common self-limiting superficial skin infection lasting 1—3 weeks usually caused by staphylococci, less frequently by streptococci or both. It mostly occurs in children aged 2—10 years, and sometimes in younger children (particularly bullous impetigo) or older people. In infancy impetigo can be more serious, especially when it occurs in the first months of life (neonatal impetigo).[5] Both primary and secondary cases occur, the latter is especially common in atopic eczema, where staphylococci may play a triggering super-antigen role. While in moderate climates *S. aureus* is the most common pathogen, *Streptococcus pyogenes* is much more prevalent in tropical impetigo.[6]

Impetigo occurs in about 1% of the children, but more often in tropical areas and secondary in atopic eczema. The following forms of impetigo are described:

Bullous impetigo

Bullous impetigo is caused almost exclusively by staphylococci, most commonly group II, phage types 71, 3a, 3c or 55. It can present as a few blisters (vesicles quickly developing in bullae and exfoliation of the skin) especially in the body folds. Generalisation may occur and may develop into Staphylococcal Scalded Skin Syndrome (SSSS) (see below).[7] Both conditions show a positive direct Nikolsky sign (the outer epidermis separates easily from the basal layer when slightly rubbed) in the erythematous areas. In this clinical scenario and in contrast to other types of impetigo, topical treatment is contra-indicated and systemic treatment is required, a fact which is often overlooked. By rubbing the skin you will induce a direct Nikolsky phenomenon making the exfoliation of the skin worse.

Bullous impetigo can occur in neonates, but is most common between the ages of 2—5 years. In neonates it may be life-threatening. Treatment with skin substitutes with intensive care in a burns centre is often indicated in such cases.[8]

Crusted impetigo

Crusted impetigo is caused by staphylococci, group A streptococci or by a mixed infection. It can present with small blisters (vesicles) which quickly turn into crusts as secondary efflorescences. The crusts are yellow and blisters are small and break easily. Some strains of streptococci may also infect the heart or more commonly the kidneys in serious infections leading to serious sequelae. Even in a small country like Holland the incidence may differ from region to region for reasons unknown.[9]

Therapy of impetigo

Treatment of superficial bacterial infections has been compromised by the development of antimicrobial resistance, to fluoroquinolones, macrolides, clindamycin, fusidic acid and mupirocin in *S. aureus*, and to macrolides and clindamycin in *S. pyogenes*.

Most cases of skin infections are treated by primary care physicians. Clearance of *S. aureus* and/or *S. pyogenes* is the main goal of treatment.[6] In general treatment is applied topically but uncontrolled over-the-counter use and treatment of ordinary wounds and minor infections promotes development of resistance to these agents. Fusidic acid cream is effective against superficial infections and is often first line treatment although it should not generally be used for longer than 10 consecutive days to reduce the risk of development of resistance. Retapumalin 2% ointment (see below) is effective in cases resistant to Fusidic acid cream.

Mupirocin 2% ointment is very effective against methicillin-resistant *S. aureus* (MRSA) colonisation. For this reason and because there is therefore a need to minimise development of resistance to it, it should be reserved for the elimination of nasal colonisation and not be used more generally. However, mupirocin use does not always lead to emergence of resistance and an integrated understanding of the factors that lead to it is lacking.[10]

Overall current therapy of impetigo is not well standardised and there is no general agreement about guidelines for treatment.[11] The lack of evidence of superiority of oral antibiotics over topical antibiotics may in part reflect the relative absence of studies of extensive impetigo. Evidence for the application of disinfectants in impetigo is also lacking.[8]

Local choice of drug varies in different regions. Van Bijnen et al. studied different national guidelines in Europe concluding that there is a lack of relevant national data on resistance.[12,13]

Mupirocin 2% nasal ointment is advised in the treatment of atopic eczema combined with diluted bleach baths as maintenance treatment to diminish the staphylococcal load.[14] In healthy adults Mupirocin nasal ointment is used for decolonisation of proven carriers. Wertheim et al. also conclude that it is less effective in decolonizing other sites such as the perineum and pharynx.[15]

Retapamulin cream is a semisynthetic novel pleuromutilin antibacterial that is the most recently developed topical drug.[16] It is a selective bacterial protein synthesis inhibitor which interacts with the 50S subunit of the bacterial ribosome in a manner distinct from other antibiotics.[17] This agent has *in vitro* activity against staphylococci and streptococci resistant to other classes of agents, including MRSA and is thought to have a low potential for development of mutational resistance.

Folliculitis and furunculosis

Folliculitis is a common skin disorder in which hair follicles become infected and inflamed. *S. aureus* is the commonest cause but it also be due to other microorganisms, including fungi and yeasts, and sometimes by an inflammation from ingrown hairs. The condition is classified as either superficial or deep, based on how much of the hair follicle it involves. Deep folliculitis is usually more severe. Some individuals are particularly susceptible. It may also be persistent and severe in particular in people with diabetes

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