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Impetigo and scabies — Disease burden and modern treatment strategies

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Summary Impetigo and scabies both present different challenges in resource-limited compared with industrialised settings. Severe complications of these skin infections are common in resource-limited settings, where the burden of disease is highest. The microbiology, risk factors for disease, diagnostic approaches and availability and suitability of therapies also vary according to setting. Taking this into account we aim to summarise recent data on the epidemiology of impetigo and scabies and describe the current evidence around approaches to individual and community based treatment.

Introduction

Both impetigo and scabies are common infections of the skin with a large global burden.[1,2] In the industrialised world, significant complications from impetigo and scabies are rare whilst in resource-poor settings and certain marginalised communities, their collective impact is much greater. There are several effective options for the treatment of both impetigo and scabies. Despite this, challenges remain in addressing the burden of disease on a community level in regions where infection is endemic.

Impetigo

Background

Impetigo is a common superficial skin infection which predominantly affects young children.[3,4] It is estimated that more than 162 million children are suffering from impetigo at any one time.[2] The burden of disease is highest in low-income countries and within marginalised populations in developed nations.[2] Infection is caused by invasion of the epidermis by bacteria colonising the skin following

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minor trauma. Autoinoculation is common and the infection is highly transmissible. Hot and humid climatic conditions, poor access to water and possibly overcrowding are factors which play a role in frequent impetigo transmission in endemic areas.[4]

The bacterial aetiology of impetigo varies according to region and continues to change over time. In tropical climates *Streptococcus pyogenes* (Group A Streptococcus or GAS) remains the major pathogen[3,4] and co-infection with *Staphylococcus aureus* is common.[5] In temperate climates *S. aureus* has largely replaced *S. pyogenes* as the predominant pathogen in impetigo[6] and community acquired methicillin resistant *S. aureus* (CA-MRSA) is of increasing importance worldwide.[6–8]

Clinical manifestation, complications and diagnosis

Impetigo can present as bullous lesions or non-bullous, papular lesions that go on to form a crust. Bullous impetigo is caused by *S. aureus* whilst non-bullous lesions are associated with both *S. pyogenes* and *S. aureus* as described above. Ecthyma is a deep form of impetigo in which ulceration extends into the dermis. In the developed world impetigo is a common reason for presentations to primary health care providers but it is generally a self-limiting condition in this setting.[9] In resource-limited settings severe disease and complications of impetigo remain problematic[3,4,10]

Invasive infections such as erysipelas (involving the dermis and lymphatics), cellulitis (involving subcutaneous tissue), osteomyelitis, septic arthritis and bacteraemia can all complicate impetigo. *S. pyogenes* bacteraemia and streptococcal toxic shock syndrome are commonly preceded by skin and soft tissue infection.[11,12] *S. aureus* bacteraemia carries a high mortality and skin infection is an important risk factor in settings where impetigo is common.[8,13]

Where *S. pyogenes* is the predominant pathogen, impetigo can also lead to significant immune-mediated complications. In endemic settings most cases of acute post-streptococcal glomerulonephritis (APSGN) are preceded by impetigo.[14,15] Individuals with a history of APSGN in childhood are at increased risk of developing ongoing albuminuria and chronic kidney disease in later life.[16,17] There is also a plausible link between *S. pyogenes* skin infection and acute rheumatic fever.[18] This hypothesis is supported by the presence of very high rates of rheumatic fever and rheumatic heart disease in Aboriginal populations in Australia wherein impetigo is pervasive and *S. pyogenes* throat infection is uncommon.[19]

The diagnosis of impetigo is generally made clinically. The use of clinical algorithms may aid in the identification and treatment of impetigo in resource-limited settings. For example, the WHO Integrated Management of Childhood Illness (IMCI) skin algorithm has been assessed in Fiji and demonstrated improvement in the clinical recognition of impetigo.[20] Elsewhere, flipcharts using high quality photographs and clinical descriptions are used to train health care workers in diagnosing impetigo.[21] Gram stain and culture of skin swabs to confirm the aetiological agent are often recommended[22] but adequate laboratory resources are not always available in resource-limited settings and treatment of typical cases without microbiology is empiric.[22] Nonetheless, in the current milieu of increasing antimicrobial resistance,[6] regional data on causative bacteriological agents and their antibiotic sensitivity profiles remain vital to best direct empiric therapy and to monitor for changing patterns of resistance.[4]

Treatment

When determining impetigo treatment, there are several important factors including the extent of disease, community wide prevalence, likely adherence to treatment and known antimicrobial resistance. Most of the clinical trials for impetigo treatment relate to limited or uncomplicated impetigo, defined as fewer than 5 lesions. Where, impetigo is extensive (greater than 5 lesions) or community prevalence is high, refer to the treatment section on extensive impetigo.

Limited or uncomplicated impetigo

A Cochrane systematic review concluded that topical antibiotics are the most effective treatment for limited impetigo.[23] This review included 68 randomised control trials representing 5578 participants,[23] finding that mupirocin, fusidic acid and retapamulin were all superior to placebo and there was no difference demonstrated between the most commonly studied topical agents: mupirocin and fusidic acid. In addition, there was no significant difference found in 7-day cure rates between topical and oral antibiotics (excluding erythromycin which is inferior to topical mupirocin) and topical antibiotic use was associated with fewer adverse events.[23] The review also cited a lack of supportive evidence for the use of disinfectant solutions in the treatment of impetigo.[23]

There are several factors to consider when selecting a topical antibiotic. Resistance to mupirocin and fusidic acid among *S. aureus* isolates is increasing in association with increased use of these agents.[6,24] Although retapamulin has demonstrated good *in vitro* activity against methicillin resistant *S. aureus* (MRSA), its efficacy in clinical trials against MRSA infections has been variable[25,26] and it is not approved for the treatment of MRSA infections. Moreover, *S. aureus* isolates with elevated minimum inhibitory concentrations (MICs) to retapamulin have been described, although the clinical significance of this is uncertain.[27] There are calls to restrict the use of topical fusidic acid in order to preserve the oral formulation as a useful agent, in combination with rifampicin, for difficult-to-treat MRSA infections.[24] Topical fusidic acid is not available for use in the USA and this is reflected in the Infectious Diseases Society of America (IDSA) guidelines for skin and soft tissue infection which recommend topical retapamulin or mupirocin for uncomplicated impetigo.[22]

Extensive impetigo

Determining the optimal treatment of extensive impetigo, particularly in resource-limited settings where the burden of disease is highest, remains a challenge.[4] It is generally accepted that the use of systemic antibiotics for extensive disease is practical and appropriate, yet there are limited