Skin Sores, Abscesses and Scabies Infections

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Skin infections are common health problems, with a higher frequency amongst children. In the NT, scabies and skin sores are amongst the commonest presentations in Aboriginal children to community health centres.[1] The bacteria on the skin do not usually cause infection, but the risk increases with humidity, hot weather, poor hygiene and especially if there is a break in skin integrity. Scabies is an extremely common factor in the NT, which underlies secondary impetigo or skin sores.[2] Less commonly, skin infections can lead to septicemia.

Impetigo/skin sores

The commonest causes of skin infections are Staphylococcus aureus, cultured in 86.7% of impetigo cases, and group A streptococcus (GAS), cultured in 29.2% of cases.[3] Other bacteria such as staph epidermis, E. Coli, group B streptococcus and gram-negatives cause skin infection in less than 5% of cases. There are very low rates of treatment failure due to these organisms.[3] Local studies have also confirmed the predominance of S. aureus in impetigo.[4] However, it is the GAS that is of greater concern, due to its potential to cause serious post streptococcal disease. Acute post streptococcal glomerulonephritis (APSGN) is strongly associated with skin infection, and NT communities regularly experience epidemics affecting large numbers of children.[5] Recent NT research has demonstrated a six-fold increased risk for individuals to develop adult renal disease if they have experienced APSGN as a child.[6] Acute rheumatic fever (ARF) and rheumatic heart disease (RHD) are also due to an abnormal immune response to GAS. The international literature links ARF/RHD to throat infections from GAS. However, local experience has shown low rates of throat carriage of GAS, but up to 70% of children either colonised or infected with GAS. Reducing GAS skin infections would appear to be an effective public health intervention to reduce the extremely high rates of ARF/RHD.[4,7,8]

Antibiotic selection for skin infections should reflect known local profiles of resistance, and also aim to minimise further development of antibiotic resistance.[9] This is especially important when treating common infections, and in our setting hyperendemic rates of skin infection. Inappropriate use of antibiotics can rapidly promote antibiotic resistance. Other factors which affect choice of antibiotic include the length of time, dosing frequency, side effects and cost. Unfortunately, there is limited strong evidence on which to make recommendations for many of the common childhood infections, including skin infections, but clinical guidelines show high levels of consensus for specific antibiotics and other treatments.[9]

Staphylococcus is a significant cause of skin infections. Methicillin-resistant S. aureus (MRSA) has been increasing in both hospital and community settings for many years. The majority of S. aureus are resistant to penicillins and, internationally, treatment failure rates with penicillin vary between 24–47%.[3,10] Erythromycin resistance in Australia is between 30–50%, strongly associated with high rates of use of this antibiotic. NT hospital studies have shown that MRSA causes 7% of all S. aureus infections, with 70% of these MRSA infections occurring on the skin.[11] Typing of these MRSA strains has shown increasing rates of WA MRSA (a strain first detected in the Kimberley). This is more likely to be a community-acquired, rather than hospital-acquired, infection.[12] A study of patients at Royal Darwin Hospital also demonstrated that Aboriginal patients had a higher risk of being infected with this strain, as did people who lived west of Darwin (adjacent to the Kimberley region) suggesting a spread across the border. This particular strain has also been associated with very high rates of resistance to mupirocin, a topical antibiotic.[11] Despite earlier studies demonstrating that mupirocin is highly effective in treating impetigo, it has been associated with rapid development of staphylococcal resistance.[13,14] In the early 1990s mupirocin was used widely in the Kimberley to treat skin sores in Aboriginal children, and antibiotic resistance developed within a short period.[12] Mupirocin resistance occurs more commonly amongst MRSA strains than methicillin-sensitive strains. A recent study in a rural Native American community showed very high rates of MRSA (55% of all S. aureus isolates), with the majority community-aquired.[15] There had been four deaths due to community-acquired MRSA in previously healthy children in the region which prompted the study. The authors note that careful antibiotic prescribing is important to avoid increasing resistance patterns.

Streptococcus pyogenes showed rapid development of resistance to erythromycin in Japan and Finland, where this antibiotic was widely used. The data prompted recommendations in Japan for restricted use of erythromycin for skin infection, and the erythromycin resistance subsequently decreased. International guidelines commonly recommend erythromycin or cephalexin for impetigo. The current Australian Therapeutic Guidelines: Antibiotics recommend penicillin or roxithromycin.[10]

Despite high use of penicillins for a number of infections amongst Aboriginal people, GAS remains very sensitive to penicillin.[16] Due to this continuing penicillin sensitivity of GAS, and the requirement to eradicate GAS to prevent post streptococcal disease, penicillins are the recommended first choice for impetigo/skin sores in Aboriginal children, despite the known resistance of S. aureus. Many years of experience by health staff and families anecdotally confirms the rapid resolution of skin sores following a single dose of benzathine penicillin. This is the preferred antibiotic as it is effective in both treating the clinical infection, and in eradicating the GAS, due to its long action. If oral treatment is preferred by families, or required due to penicillin allergy, a longer course of an oral antibiotic for ten days is necessary to achieve effective GAS eradication.

Cellulitis is usually due to streptococci, but can be caused by S. aureus, similar to skin sores. Recommended treatment is penicillin, usually procaine penicillin. Elevation of affected area (usually leg or arm) is an important principle of treatment which reduces swelling.[13]

Abscess/boils

The principal treatment for abscess (boils) is incision and drainage rather than antibiotics.[3] Antibiotics penetrate the abscess cavity very poorly, although they will improve surrounding cellulitis. S. aureus is almost exclusively responsible for causing boils. Flucloxacillin is not currently recommended due to rare but potentially fatal cholestatic hepatitis.[17] This has occurred mainly in older patients, and those treated with longer courses (two weeks or more), with the incidence estimated between one in 400 and one in 20 000 courses. The current recommendation is to use dicloxacillin, which still has a risk of hepatitis, but this risk is approximately half that seen with fluclocaxillin.[18] There is no paediatric preparation of dicloxacillin, and hepatotoxicity is rare in this age group, thus flucloxacillin syrup is still recommended for children. Recurrent boils due to Staphylococcus are seen in a small group of patients. These patients are carriers of Staphylococcus, usually in the nose and occasionally in the perineal region. Carriage status should be confirmed with swabs prior to treatment. Eradication with topical mupirocin has been successful in a number of trials, and restricting the use of mupirocin for chronic carriers with recurrent infection may prevent development of resistance. Treatment is recommended as a single course for between five to 10 days, although one small study showed reduced recurrences amongst people with frequent infection using prophylactic nasal mupirocin for five days each month over a one-year period.[19,20] In difficult cases with continued reinfection, oral rifampicin has been successfully trialled.

[Editor: In the Top End wet season, a person with risk factors for melioidosis, such as diabetes, renal failure, chronic alcohol abuse may have cellulitis or abscess caused by melioidosis. If suspected, talk to a doctor. (See the Melioidosis chapter in this book.)]

Scabies

Scabies is caused by an obligate human parasitic mite, Sarcoptes scabiei, which is transmitted from person to person through close contact. A common misconception is that dogs are responsible for the high scabies prevalence in the NT, however genetic typing studies done at Menzies School of Health Research in the mid 1990s, showed that dog scabies and human scabies are different subspecies.[21,22] The pregnant scabies mite burrows and deposits about two to three eggs a day in the stratum corneum of the skin. The nymphs emerge as adults on the surface of the skin after a series of moults which takes about two weeks. The mature mites mate and reinvade the skin of the same or another host.

[Editor: The dog subspecies can still cause an itchy condition (delayed hypersensitivity) in humans, however it does not reproduce on humans and was not isolated from humans in the Top End studies mentioned (possibly not common). It may be unwise to tell people they are wrong in their belief that dogs are important, as a person who frequently has close contact with scabies infected dogs (e.g. sleeps with them) may still suffer a chronic scabies condition. In theory this would be repeated infection with canine scabies rather than self-sustaining human scabies infection.

There have been a number of successful community scabies control programs that did not treat dogs. Person to person transmission is the key aspect of scabies control.]

Initial infestation is asymptomatic. After four to six weeks the host becomes sensitised to the excreta of the mites and an itch and rash develops, although in some people the latency period can last for several months.[23] With subsequent reinfestation the host will immediately develop a hypersensitivity reaction and become symptomatic. Young children have a poorly developed immune response to the scabies mite and carry greater numbers of mites. The most common symptom is an itchy rash, which usually has a classic distribution. In young children, the lesions are usually widespread, from head to toe, including 'pustular' blisters on the palms of their hands and soles of their feet. These are not infected with bacteria but are caused by the host immune response to the mite. Older children and adults usually have lesions at the wrists, in the interdigital space between fingers and toes, the buttocks and around the ankles. Scabies lesions may also be found on the head in older children and adults, although this is much less common. The lesions include vesicles, excoriation (from scratching), nodules and, less commonly, burrows.[24]

Investigations are rarely performed for diagnosis because the clinical picture is usually clear. The diagnosis can be confirmed, if the rash is atypical, by skin scrapings from the lesions and identification of mites and eggs using microscopy. Differential diagnosis could include eczema, mild cases of psoriasis or contact dermatitis. A swab of associated skin sores will invariably grow Streptococcus and Staphylococcus.

Treatment involves topical application of scabicide ointments, lotions or creams. Treatment of the affected individual and the close contacts is recommended, although there are no randomised control trials (RCTs) on contact treatment.[25] A Cochrane review has identified RCTs that have compared the multiple treatments available for scabies, which have varying efficacy and ease of application. The clinical cure rates of crotamiton, lindane, benzyl benzoate and sulphur showed no difference in small RCTs. Permethrin has similar efficacy to lindane (91.5% cure vs 88%), although permethrin is more effective in reducing itch. Permethrin was more effective than crotamiton (89% vs 60%). Oral ivermectin has been trialled and has similar efficacy to benzyl benzoate and lindane. Ivermectin is not currently approved for scabies treatment, although may be used 'off label' for crusted scabies (see below). There are rare serious adverse effects from lindane (convulsions, aplastic anemia), permethrin (convulsions) and ivermectin (apparent increased risk of death in elderly people, although uncertain association), which have been identified from case reports, although not from the RCTs.[25]

Permethin 5% is the currently recommended treatment internationally, based on small studies and expert opinion. It was introduced in the NT in 1994, and due to low toxicity and ease of application it is the preferred treatment.[26] It is usually prescribed as a single treatment for eight to 12 hours (overnight). People with multiple lesions (usually children) require a second treatment after two weeks to ensure eradication of newly hatched mites. People may continue to have itching for two to four weeks after treatment, although most people experience relief of symptoms within three days.[24]

Scabies may be transmitted by fomites (bedding, clothes, linen), and it is recommended that washing clothing and linen used in recent days is included as part of the treatment.[24] For ordinary cases of scabies environmental transmission is not as important as person-to-person transmission. However, for people with crusted scabies who carry millions of mites it is essential to eradicate the mites from the house.

Crusted scabies

Previously called Norwegian scabies, as it was first described in a leprosarium in Norway. Crusted scabies is a severe form of scabies infestation, in which the mite multiplies in the millions. In most studies people with crusted scabies have some form of immune deficiency, which is usually well documented, such as HIV infection.[27,28] However, in the NT the underlying immune problems may be more complex and subtle as they are rarely identified, although there are documented cases of people with HTLV1 and crusted scabies in Central Australia.[29,30] In crusted scabies the affected person cannot immunologically contain the scabies mite and they become infested with millions of mites, developing a thick, crusted skin in response. They are highly infectious to others and also highly susceptible to reinfestation.[31]

The rash from crusted scabies can vary. Mild cases may have localised patches on the buttocks, upper thighs, upper arms and occasionally on the dorsum of the hands and the feet. Severe cases may be covered from head to toe with thick, elevated crusted lesions, which may also have fissures. Crusted scabies is often not itchy and it is commonly misdiagnosed as eczema, psoriasis or other dermatitis.[32] Recurrent cases of scabies in people may be an indicator of contact with a case of crusted scabies. It is important to confirm the diagnosis with skin scrapings to detect the mite microscopically, and exclude serious immune deficiency. The NT-CDC guidelines on community control of scabies and skin infection includes guidelines on management of crusted scabies.[33]

Crusted scabies involves both topical treatment with permethrin and a keratolytic cream to soften the crust as well as oral ivermectin. The protocol for crusted scabies also includes treatment of the entire household and environmental health officers working with family members to clean the house and use insecticide bombs to kill all scabies mites. Severe cases of crusted scabies usually need treatment in hospital. The main complication of crusted scabies is septicemia. A study done at Royal Darwin Hospital showed that people with severe crusted scabies had a five-year mortality rate of 50% due to septicemia, although improved antibiotic protocols has reduced this mortality.

[Editor: Given that those with crusted scabies are likely to have an underlying immune-deficiency or be otherwise debilitated, this high mortality may not all be attributable to the scabies itself, but scabies may be important in creating portals of entry for infection or increasing immune suppression. Prof Bart Currie believes the multiple organisms that cause the septicemia are directly due to the skin fissures allowing unimpeded access, not the underlying immunodeficiency see: Currie B, Huffam S, O'Brien D, Walton S. Ivermectin for scabies. Lancet 1997; vol 350:1551.]

Skin conditions	Baseline screening	Follow-up 1 (7 months)	Follow-up 2 (21 months)
Scabies	56%	28%	48%
Skin sores	14%	35%	41%

Adapted from; Ewald D, Hall G. Housing and Health: Evaluation of the National Aboriginal Health strategy — Environmental Health program in a Central Australian Community. Darwin: CRC for Aboriginal and Tropical Health, 2002.]

Community prevalence and public health programs

There is limited historical documentation about the prevalence of scabies in the NT. There were reports in 1815 of scabies being problematic on missions but not amongst tribal people. In Central Australia in 1957 it was reported to be common, however, in 1960 a scientific expedition in Arnhem Land demonstrated that scabies was quite rare. This is consistent with the experience of leprosy nurses who worked throughout the NT and recalled that scabies and skin sores were rare around that time. Long-term health professionals in the Top End believe the rates of scabies and skin infections have been increasing since the 1970s (pers. comm. Joan Fong, leprosy nurse, CDC).

Currently, community surveys show high but variable prevalence rates ranging from 30–60% in children under the age of 15. Surveys of adults have shown similar levels of at least 30% at any one time, although adults generally have milder clinical manifestations. A successful model to control high rates of scabies in a community setting has been adapted from Panama and used effectively in several remote NT communities.[15] The Panama program involved simultaneous supervised treatment of all community members (the community was on an island), with 5% permethrin cream. This led to a reduction in prevalence rates of scabies from 33% of the population to very low levels of 1.5% that were sustained for three years. The pyoderma (skin sores infection) prevalence rate in children decreased from 32% to 2% without use of specific antibiotics.

An adapted Panama model was then formally trialled at Minjilang in 1994, which was successful in maintaining low rates of scabies and skin infection for up to five years using regular community screening and treatment with permethrin.[7] The scabies rate decreased from 29% to below 10% at two years. Following the success of this program, guidelines for health staff were developed with NT Centre for Disease Control.[17]

A number of communities throughout the NT have subsequently run the program.[34–37] Three communities have successfully maintained low prevalence rates of scabies and skin infection for at least 12 months (Minjilang, Wadeye and Kunbarllanjnja). The common element amongst these programs was ongoing surveillance and community awareness of rates of scabies and skin infections. In other communities that have available data, the initial period following the Healthy Skin day has shown a significant reduction in scabies. The surveillance is important to maintain lower rates and

sustainability of the program. Further research on this issue is currently occurring through the Cooperative Research Centre for Aboriginal and Tropical Health.

[Editor: There is some evidence that scabies is not always the major cause of skin sores in Aboriginal communities. For example, in one series of surveys and treatment of children under 13 years old, in a Central Australian community, the prevalence of scabies changed quite differently to the prevalence of skin sores. This can be seen in the table below. However, the calculated population attributable risk of skin sores from scabies was stable at around 34% in each round.]

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