Pyodermas: Diagnosis and Treatment

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The diagnosis and treatment of cutaneous infections appear to be rather straightforward at first glance. Major texts of pediatrics and dermatology indicate that the vast majority of pyodermas are due to either *Staphylococcus aureus* (*S. aureus*) or *Streptococcus pyogenes* and that antibiotics are curative. On closer scrutiny, however, one quickly becomes aware that there is a great deal of nomenclatural confusion with a corresponding lack of clarity regarding therapy—particularly on the point of whether or not topical antibiotics have any place in therapy.

Pyodermas can be divided into two main classes: primary and secondary. In a primary pyoderma, the skin is normal or at most has suffered a minor cut, abrasion, or insect bite. In such a situation, the invading organism is entirely responsible for the clinical picture. The situation is entirely different in a secondary infection. Here the underlying process is a chronic inflammatory process, for example, atopic dermatitis which first becomes colonized, usually by *S. aureus*, and secondary infection is present only when *S. aureus* has proliferated to rather high levels (see below).

Primary Cutaneous Infections.

Two distinct forms of primary pyodermas have been described: 1) Bullous impetigo caused by Group II *S. aureus* usually phage type 71, and 2) Streptococcal pyoderma or ecthyma due to Group A beta hemolytic streptococci. Both have distinctive clinical characteristics which can allow confident diagnosis.

Bullous impetigo presents as multiple, often coalescing superficial bullae which rupture easily and leave behind a thin varnish-like crust. This eruption is usually seen in children, most commonly on the face, although any part of the body may be involved. Fever and other systemic signs are infrequent. Therapy consists of oral antibiotics, either penicillin G 400,000 units q.i.d. for ten days or oxacillin 500 mg q.i.d. if a penicillinase producing organism is involved. Local care of compresses and topical antibiotic or steroid-antibiotic creams are useful. This form of pyoderma is rather contagious, but does not carry with it the potential for post-infection glomerulonephritis.

A severe and fortunately rare form of *S. aureus* infection is the toxic epidermal necrolysis syndrome. A toxin mediated by phage type 71 *S. aureus* causes widespread necrosis of the epidermis and results in a sloughing off skin, producing the so-called "scaled skin syndrome." The infecting organism is usually in the nasopharynx, ear, or conjunctiva and not in the skin. Treatment consists of intravenous penicillin (Nafcillin 50 mg/kg) until the lesion ceases to appear and then oral Nafcillin for seven to ten more days.

Streptococcal pyoderma begins as superficial vesicles, but quickly erodes and develops a central ulceration with a thick crust and an areola of erythema surrounding the ulceration. These lesions occur primarily on the lower extremities of children during the hot, humid summer months and are frequently associated with lymphadenopathy and fever. Streptococcal skin infections do not result from the strains responsible for throat infections and anti-streptolysin-0 (ASO) titers are feeble, although antibody response to other streptococcal antigens (DAN-use B and hyaluronidase) can be vigorous. Cultures reveal Group A streptococci under the crust and under the edges of the advancing ulceration while
the crust itself may reveal only *S. aureus*. Treatment studies in both humans and in hamsters support the view that streptococci are the important agents since the presence of penicillin-resistant staphylococci in infected mixed lesions does not interfere with effective treatment with penicillin G.\textsuperscript{3,4} It is this streptococcal variety of pyoderma which carries the risk of post-infection glomerulonephritis. There are no studies demonstrating the prevention of glomerulonephritis by penicillin treatment and in fact there is considerable clinical evidence to suggest that renal complications may occur despite penicillin treatment.\textsuperscript{5}

Treatment consists of benzathine penicillin 2.4 million units intramuscularly for extensive deeply seated lesions.\textsuperscript{6} The role of topical antibiotics is unsettled. Several studies comparing topical to systemic therapy concluded that systemic therapy was superior. However, no attempt was made to separate deep, extensive lesions from the early more superficial variety. Our own view is that in early lesions, compresses and topical antibiotics are quite effective.\textsuperscript{7}

**Secondary Infections: Impetiginization.**

Inflamed skin provides fertile soil for bacterial overgrowth, and it is not surprising that dermatomic skin can become colonized and then infected with various pathogens. In most cases, the organism involved is a *S. aureus*. A variety of phage types are found, but rarely are the highly virulent strains such as type 71 isolated. Beta hemolytic streptococci are rarely recovered from dermatoes: in several hundred studies cases we have found five examples of Group A beta hemolytic streptococci complicating an underlying disorder—two were in epidermolytic hyperkeratosis and the other three occurred as severe “athlete’s foot” infections.\textsuperscript{8} *Candida albicans* and gram-negatives are found in two situations, namely in dermatoes occurring in wet body regions and in patients on long-term antibiotic therapy.

The question arises of when does the recovery of an organism from a dermatosis indicate infection is present and that antibiotic therapy is indicated. Through our quantitative studies we have to date evolved to an understanding of the following guidelines for interpretation of routine non-quantitative culture reports.

   *Staphylococcus epidermidis* (*S. epidermidis*), lipophilic diptheroids.
2. Gram-negatives.
   The relative virulence of these organisms has not been fully established as yet. *Pseudomonas* certainly appears to be relatively virulent, but the density required to aggravate an underlying dermatosis and the density of *Escherichia coli* (*E. coli*), *Proteus*, and others have not been elucidated. These organisms can be found in low numbers in wet body areas such as the groin, axilla, and toe space, and at low numbers these organisms do not damage intact human skin. Present evidence would suggest that a useful guideline would be that if a culture reveals the predominant organism to be a gram-negative, then it most likely is contributing to the clinical picture. The choice of an antibiotic would be one that would include activity against the gram-negative isolated.

3. *Candida albicans*.

Our experience with this organism in experimental infection situations has taught us that it is extremely toxic to human skin. A far lower inoculum is needed to induce an infection than with any other organism we have studied to date. For example, as low as ten *Candida* cells will result in a severe reaction after three to four days of growth under saran wrap while thousands of cells and prior suppression of the resident flora are required for experimental *S. aureus* infections.\textsuperscript{9,10} The isolation of *C. albicans* from a dermatitis such as diaper dermatitis is therefore a very significant finding, and anti-yeast measures are indicated.

4. *Propionibacterium*.

Two main groups have been identified: *P. acnes* which is susceptible to *P. acnes* bacteriophage is more prevalent and found in greater densities in the sebaceous areas than is the phage-resistant *P. granulosum*. Recovery of this organism from a skin culture does not indicate an infection, rather it is a member of the resident flora. This organism does, however, appear to play a central role in acne vulgaris. Acne patients carry a much greater density of this organism than do aged-matched controls.\textsuperscript{11} The current hypothesis is that this organism influences the acne process by virtue of production of free fatty acids through hydrolysis of sebaceous gland triglycerides. Those antibiotics which lower *P. acnes* in vivo such as tetracycline, erythromycin, and clindamycin are also judged to be useful in the management of acne, while penicillin and sulphonamides neither lower *P. acnes* nor help in the treatment of this disorder. To date, we have not encountered *P. acnes* resistant to antibiotics, and clinical failure during antibiotic therapy does not indicate that another factor is involved, for example, hormonal disturbance, emotional dis-
tress, or other factors. To date, only systemic antibiotics have been found to lower P. acnes levels. Recently, however, there is some evidence suggesting that topical benzoyl peroxide may be an effective agent for suppressing P. acnes.

5. S. aureus.

*Staphylococcus aureus* is the usual organism recovered from inflamed skin. A variety of phage types are recovered: virulent strains such as type 71 are not frequent. In a series of studies, we have established that a density of one million *S. aureus* organisms per sq cm is the level at which antibiotic therapy will produce clinical improvement. Our definition of a *S. aureus* secondarily infected dermatosis is therefore expressed in quantitative terms. Certain eruptions far more commonly harbor levels of *S. aureus* exceeding our established quantitative criteria for presence of a secondary infection. In atopic dermatitis, as many as 45% of even the chronic lichenified variety and 100% of the acute exudative form will harbor more than a million *S. aureus* per sq cm. In such cases, even though overt clinical signs of infection are lacking, topical or systemic therapy will produce a rapid suppression of this organism in one week’s time and result in clinical improvement. Other conditions frequently harboring more than a million *S. aureus* per sq cm include numular eczema, neurodermatitis, exfoliative erythroderma, and chronic familial benign pemphigus, and again antibiotic therapy will often be useful in these conditions. In psoriasis and seborrheic dermatitis, *S. aureus* very rarely achieves a high enough density to be clinically significant, and antibiotic therapy is rarely useful except when *S. aureus* growth has been stimulated, which can happen, for example, during occlusive therapy with impermeable plastic film. An important corollary of this principle of the quantitative aspect of *S. aureus* secondary infections is the role of steroids in such conditions. One might predict that topical steroids could lead to an aggravation of a condition heavily colonized with *S. aureus*. Clinical experience does not support the theory, however. The explanations for this apparent paradox is that topical steroids are effective in suppressing the inflammation which promotes *S. aureus* colonization and proliferation. By removing the conditions favoring *S. aureus* growth, the return of the normal protective flora is promoted rather than *S. aureus* overgrowth.

**Therapeutic Approaches.**

In primary infections such as bullous impetigo, eczema, and cellulitis, systemic antibiotic therapy is curative because invading pathogenic bacteria are responsible for the entire clinical picture. Local compresses to remove crusts and topical steroids to suppress cutaneous inflammation are useful adjuncts, but the main thrust of therapy is to remove the offending pathogen.

In secondarily infected dermatoses, a variety of therapeutic approaches are available and often several modalities must be simultaneously utilized. Both topical and systemic antibiotics are effective, and choice is determined principally by the extent and severity of involvement. Steroids are indicated for suppression of the underlying inflammatory condition, which promotes a more normal skin and the return of the normal flora. The combination of steroids and antibiotics appears to offer a rational approach.

In excessively wet areas such as the axilla, groin, and toe web space, and in severely exudative lesions anywhere, therapy must also include “drying” agents. Excessive moisture promotes bacterial growth and must be controlled in order to obtain satisfactory results. The use of compresses is often indicated. Dyes such as gentian violet and Castellani’s paint which have both astringent and antimicrobial activity provide a double-pronged approach.

The object of therapy should be to restore the skin to a noninflamed status and to promote return of the normal protective resident microflora. Antibiotics are used to remove pathogens, steroids suppress inflammation, and compresses remove excessive moisture. Successful therapy involves attacking all aspects of a disorder.

**REFERENCES**


