Editorial

The Overlap of Inflammation and Infection

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Dermatologists, pediatricians, and pediatric dermatologists treat skin and subcutaneous tissue infections on a daily basis. The pathogenic organism (whether bacterial, viral, or fungal) is not isolated until days to weeks after presentation. Anti-infective therapy is initiated empirically until an etiology is known. Much scientific effort has been devoted to finding virulence factors that can cause direct cytopathic effects and, therefore, greater morbidity and mortality of skin infections.[1] On the flip side, host response to infection (ie, inflammation) also is associated with excess tissue destruction. Classically, the host response to potential bioterrorism agents (eg, anthrax, smallpox), exacerbated by virulence factors, is believed to be one of the major causes of morbidity and mortality in those conditions. Host response, or inflammation, is manifested by erythema, edema, pruritus, and low-grade fever. This inflammation is a by-product of released T-helper type 1 cytokines. Cytokines released in chronic infections include interleukins 1 and 12, interferon γ, and tumor necrosis factor α. However, bacterial infection also can be worsened by concurrent chronic allergic disease.[2] Thus a T-helper type 2 phenotype also may contribute to infection and likely does in conditions such as eczema herpeticum and super-infection of atopic dermatitis. From a clinical standpoint, inflammation may overlap with and exacerbate the symptoms of infection, including edema, pruritus, fluctuance, tenderness, and warmth.

Other infections evade host response by producing factors that block important events in cell-mediated immunity and cytokine cascade. One such infection is caused by molluscum contagiosum virus, a poxvirus with skin-limited infectivity. The molluscum infection produces substances that block immune recognition of the virus, prolonging the length of infection. Inflammation often is ineffective when it begins. One third of patients will experience symptoms that mimic atopic dermatitis localized around the molluscum, the so-called molluscum dermatitis.[3] For years, practitioners have treated this dermatitis with topical anti-inflammatory creams and occasionally with oral antibiotics when the swelling of lesions mimicks bacterial infection. Other viral infections are treated with anti-inflammatory agents as well. Treatment of extensive herpes zoster infection with corticosteroids has been shown to reduce post-herpetic neuralgia and recovery time.[4] In pediatrics, the use of anti-inflammatory agents in systemic viral infections was stopped for many years because of Reyes syndrome induced by aspirin therapy,[5] which has been replaced by ibuprofen therapy.

It is rare for dermatologists to treat the inflammation in bacterial infections. In this issue of *Cutis*®, Dall and colleagues[6] examine the benefits of adding an oral anti-inflammatory medication to an antibiotic regimen given for cellulitis. In this preliminary study, the recovery time of the dual-treatment patients was significantly faster ($P<.05$), and symptomatic improvement was remarkably rapid, beginning in 1 to 2 days. These findings are very important and pertinent to dermatologic therapeutics. Should physicians add anti-inflammatory medications to other infectious treatment regimens such as staphylococcal abscesses or panniculitis?

Clearly further clinical trials must be done to assess the role of anti-inflammatory agents in the treatment of skin and subcutaneous tissue infections. Until this data is available, practitioners should consider anti-inflammatory agents as potentially remitting, not just symptomatic, therapy for infections. Adjunctive use of anti-inflammatory agents should be contemplated with all infections complicated by erythema, edema, pruritus, and low-grade fever.

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