Our skin does not merely envelop our bodies. Its functions are more profound than a sum of its metabolic activities, its protective and barrier properties, participation as a heat sensor and endocrine organ, and as a critical component of the immune system. Our skin provides subjective information to our brain for the development of our self-image. The psychological burden of skin disease and its effect on quality of life is greatly under-appreciated.

Skin disease negatively affects quality of life for children more significantly than many other chronic diseases. In one study, inflammatory skin diseases such as atopic dermatitis (AD) and psoriasis caused the greatest impairment. Only cerebral palsy scored higher in its negative consequences on quality of life.1

Children affected by inflammatory skin diseases are not only burdened with the daily intensive and time-consuming application regimen of numerous topical agents, but also they are afflicted with pruritus that seemingly never abates, resulting in chronic sleep deprivation. This in turn may result in daytime drowsiness, decreased productivity, and poor attention in school.2

Appreciating the concept of a “vulnerable” skin barrier in those with AD helps us intuitively understand why emollients and “gentle skin care” are the basis for AD therapies. Sorting through the wide range of newer barrier repair products and separating fact from marketing claims can be difficult. Wynnis L. Tom, MD, in her article “Atopic Dermatitis: Recent Findings and Insights” (see page 20), reviews and updates us on the pathogenesis of AD and shares important new information regarding topical therapies for AD.

LeAnna Lane, MD, and Jonathan Dyer, MD, discuss the reciprocal relationship between certain infections and pediatric inflammatory skin diseases such as AD (see page 21).

It is important for the reader to remember that Staphylococcus aureus colonizes most, if not all, of those with AD and it cannot be eradicated. Thus, I discourage my patients with AD from using harsh antibacterial soaps and cleansers such as chlorhexidine; not only are these agents not capable of maintaining a sterilized skin environment, they further disrupt the already vulnerable skin barrier, often leading to clinical exacerbation of skin disease.

In clinical practice, one often questions whether there is enough clinical deterioration to warrant the administration of oral antibiotics. I have always found the term “superinfection” to be vague and subject to one’s subjective clinical impression. The presence of pustules or extensive crusting, beneath which is a
nafunctional skin barrier usually coincides with significant increase in S. aureus counts.

In less obvious clinical scenarios, it is very important to know your patient. Worsening of AD, decreasing efficacy of standard treatments, and increased body surface involvement are all clues that systemic antibiotics may be required as an adjunctive therapy in AD management. Additionally, I routinely culture before starting oral antibiotics (usually a generic cephalosporin), as this reduces the diagnostic confusion if your patient does not respond well to the treatment regimen.

As Drs. Dyer and Lane note in their paper, there are a variety of treatment options available to the clinician for molluscum. My personal favorite is cantharidin, as it is painless to apply and very effective. However, I would caution the pediatrician who is unfamiliar with this product to seek instruction before using it, as it is very dose-dependent. The application of too much material (or concentrated material from near the bottom of a bottle that has not been mixed by agitation before each use) can result in severe blistering.

Additionally, while not supported by evidence, I advise my patients to avoid prolonged bathing (ideally not more than 5 minutes in the tub) until their molluscum is resolved. It may be that prolonged immersion leading to decreased skin barrier function allows the virus to spread more readily, making it more difficult to eradicate.

In “Management of Pediatric Psoriasis,” Tina Bhutani, MD, and colleagues present a very practical review on the management of pediatric psoriasis (see page 22). Pediatric psoriasis in the diaper area can be particularly challenging to diagnose, because its location in an occluded area reduces or eliminates the associated scale characteristic for psoriasis. Management is often challenging due to the limitations of using more potent topical corticosteroids in an occluded area. As Dr. Bhutani points out, this is an ideal location to add a calcinurin inhibitor if necessary for disease control and patient safety.

Evidence-based systemic treatment options for moderate and severe pediatric inflammatory skin diseases such as AD and psoriasis are sorely lacking. The recognition of the need for such data has led to the creation of a national collaborative by a group of pediatric dermatologists, the Pediatric Dermatology Research Alliance (PeDRA). The initial goals of PeDRA include gathering information on pediatric patients who are receiving systemic treatments for severe AD at several medical centers across the US. This will hopefully generate safety and efficacy data that can be used to select the “best” agents and use them in a more uniform protocol.

There are very effective systemic agents such as azathioprine and cyclosporine for the management of AD, as well as biologic agents for the treatment of psoriasis, but use of these agents is hampered by lack of information on optimizing their use in children.

Furthermore, without the indication for use in this age group by the Food and Drug Administration, insurance companies are often reluctant to pay for these often costly treatments. Collaboration is the most efficient and sometimes the only way to study uncommon or rare diseases.

REFERENCES

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