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Viral and Bacterial Interplays in Children's Psoriasis: Overlooked Pathways to Flare-ups

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Dear Editor,

Pediatric psoriasis differs from adult disease in age of onset, clinical presentation, and trigger profile. Infections, particularly bacterial and viral, are among the best-documented precipitants of acute flares in children, yet their synergistic interaction remains underappreciated (1). Group A *Streptococcus* (GAS) is the most established trigger, preceding up to 80% of new-onset guttate psoriasis cases 1 - 3 weeks after pharyngitis or perianal dermatitis. Streptococcal pyrogenic exotoxin superantigens drive polyclonal T-cell activation and marked expansion of skin-homing CLA+ T-cells, amplifying the IL-23/Th17/IL-17 pathway central to psoriatic inflammation. Molecular mimicry between streptococcal M-protein and human keratin further sustains auto-reactivity (2). Viral infections are less consistently emphasized but repeatedly implicated. Influenza, rhinovirus, varicella-zoster, parvovirus B19, and SARS-CoV-2 have all been linked to de novo psoriasis or acute exacerbations in children, often via type I interferon-driven activation of myeloid dendritic cells and subsequent IL-23 production (3). Of greater concern is the frequent co-occurrence of viral and bacterial infection in children. Viral compromise of mucosal barriers facilitates secondary streptococcal invasion, creating a “two-hit” scenario in which viral-induced interferon responses and bacterial superantigens converge on the same inflammatory cascade. Skin and gut microbiome dysbiosis may further perpetuate Th17 polarization. Although direct pediatric evidence of such synergy is still limited, the temporal clustering of mixed infections before flares is well recognized clinically (4).

Current management often focuses on streptococcal screening (throat swab) while viral testing is rarely performed. When GAS is confirmed, penicillin or other appropriate antibiotics remain first-line in trigger-directed therapy. Biologics targeting IL-17 (for example, secukinumab) or IL-23 are approved and effective in children ≥ 6 years of age with moderate-to-severe disease, but their specific role in infection-triggered flares has not been studied separately (5). The use of probiotics or other microbiome-modulating strategies is theoretical at present and lacks pediatric trial data (6).

In summary, viral-bacterial interplay represents an under-recognized pathway for psoriasis flares in children. Routine consideration of both classes of pathogens, combined with targeted antimicrobial therapy when indicated, may reduce recurrence rates and the need for systemic immunosuppression. Prospective studies incorporating multiplex respiratory/viral PCR and streptococcal testing during flares are warranted.

Footnotes

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