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아토피피부염 바르는 약: 언제까지 어떻게?

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The dysfunctional barrier function is one of the main feature of atopic dermatitis(AD). The mutation of filaggrin gene is well-known genetic cause of barrier dysfunction in AD. However, the mutation is detected only in 20% of AD patients, which means genetic factors are not the only reason for the barrier dysfunction. Inflammatory mediators (IL-4, 13, 25, 31, histamine) in AD can cause impairment of the epidermal barrier. The breakdown of epidermal barrier facilitates entry of environmental allergens, and also cause skin inflammation, further aggravating epidermal barrier dysfunction. The breakdown epidermal barrier even increases the risk for asthma and allergic rhinitis.

In the treatment of AD, a step-up approach is used far too often: patients receive mild topical corticosteroid without subsequent maintenance therapy. This is often repeated for years to treat exacerbations. Instead, atopic dermatitis should be treated efficiently from the beginning, as early as the first signs and symptoms occur, to clear the inflammation and restore the barrier function. After intensive topical treatment, the dosage and dosing intervals can be reduced to the level that are able to keep control of the disease. Effective long-term treatment of atopic dermatitis restores the skin barrier function and reduces the load of environmental antigens into the skin which in turn decreases skewing towards Th 2 type of inflammation and increases Th type 1 reactivity towards normal.

Topical corticosteroids (TCS) and topical calcineurin inhibitors (TCI) are the mainstay of anti-inflammatory therapy in AD. TCS act on a variety of immune cells, including T lymphocytes, monocytes, macrophages, and dendritic cells, interfering with antigen processing and suppressing the release of proinflammatory cytokines. TCS also act on structural cells, including epithelial cells, endothelial cells, fibroblasts. TCI mainly act on T cells and also influence dendritic cell and mast cell function. TCS and TCI show very different penetration ability into the skin. TCS can easily penetrate the skin and are often detected in peripheral blood. In contrast, TCI can only penetrate the damaged epidermal barrier and penetration depth is also shallow. Systemic absorption of TCI is negligible. For acutely exacerbated swollen lesions, or for chronic lichenified lesions, TCS is a better choice

because of its good penetration. To increase the efficacy of TCS, wet-wrap therapy or soak and smear technique can be used. As prolonged TCS use is associated with skin barrier damage, switching to TCI after an intensive clearance period with TCS can be a good strategy.

For those patients who experience frequent, repeated outbreaks at the same body, a proactive approach to maintenance has been advocated. Applying low-mid potency TCS twice weekly (consecutive days or Thursday and Sunday) for the prevention of flares in stabilized AD has reduced rates of relapse and increased time to the first flare. The use of twice weekly TCS up to 52 weeks has not been shown to cause skin atrophy. The proactive use of TCI twice or three times weekly also has been shown to effective and safe.

Patients who flare frequently, despite TCS induction followed by a proactive management, or who need for repeated courses of systemic steroids would be candidates for systemic therapy.