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Sensitive Skin and Eczematous Dermatoses

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Introduction

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Sensitive skin is defined as a "skin less tolerant to frequent and prolonged use of cosmetics and toiletries and is self diagnosed, unaccompanied by any evident physical signs of irritation" (I). Currently, sensitive skin is a commonly encountered entity among cosmetic consumers and professionals. Sensitive skin subjects have been described as "people complaining of severe sensations of discomfort such as burning, stinging or inching after application of cosmetics or toiletries, such as suscreens and soays without any clinical stignata like scaling, induration and/or erythema that would be expected in known inflammatory or allergic processes" (2). Ezezmatous dermatosis are common dermatodogical disorders. The most common type of ezezmatous dermatitis is atopic dermatitis (AD). Other common ezezmatous dermatoses are allergic contact demantisis (ACD), irritant contact dermatitis (CID), and seborrheic dermatitis (SD). Principles of treatment of ezezmatous dermatous derma

Pathophysiology

Fattoppysiology

The etiology of sensitive skin is poorly understood. There is a decrease in the tolerance threshold of the skin without any immune or allergic mechanism (5). The condition is generally attributed to heightened neurosensory input and/or joopardized skin barrier (6).

The pathophysiological mechanism involved in sensitive skin.

The pathophysiological mechanism of the most common form of contact dermatūtis (CD), the ICD, is as follows (7). There is inflammation arising due to release of proinflammatory cytokines derived mainly from the keratinocytes, usually in response to chemical stimuli, resulting in direct recruitment and activation of T lymphocytes. The main pathophysiological changes are skin barrier disruption, epidermal cellular changes, and cytokine release.

Individuals with a history of AD are prone to develop ICD of the hands. Polymorphisms in the FLG gene, which result in loss of filagerin production, may alter the skin barrier and are a predisposing factor gene, which result in loss of filagerin production, may alter the skin barrier and are a predisposing factor gene, which result in loss of filagerin production, may alter the skin barrier and to the production of the p