IL-31 and IL-33 circulating levels in allergic contact dermatitis

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Introduction

Allergic contact dermatitis (ACD) involves epidermal cells, such as keratinocytes and Langerhans cells, fibroblasts and endothelial cells, as well as invading leukocytes, interacting amongst themselves under the control of cytokines and mediators network (1). Interleukin (IL)-31, a recently discovered cytokine, is involved in both innate and adaptive immunity in tissues in close contact with the environment, such as the skin (2). IL-31 is produced by human mast cells, monocytes, macrophages, monocyte-derived dendritic cells, human epidermal keratinocytes and dermal fibroblasts upon various stimulations (2). Enhanced IL-31 expression in skin biopsies is present in pruritic diseases as atopic dermatitis, ACD and prurigo nodularis, and elevated serum IL-31 levels have been found in chronic urticaria and pruritic skin lesions related to epidermal growth factor receptor-tyrosine kinase inhibitors treatment (3-7).

IL-33, a cytokine of the IL-1 cytokine family has recently been attributed to the epithelial “alarmin” defense system. IL-33 is released by the epithelial cells in various tissues and organs, including keratinocytes, endothelial cells, and immune cells and, as other cytokines of IL-1 family, by necrotic structural cells, as fibroblasts and keratinocytes (8,9).

The aim of this study was to measure the circulating IL-31 and IL-33 levels in patients with ACD to test the hypothesis that these cytokines could have a systemic involvement in this pruritic skin disease.

Material and Methods

We enrolled 20 patients (15 females, 5 males), with ACD (mean age 36.84 ± 12.62 years; range 18-62 years), diagnosed by patch testing with SIDAPA (Italian Society of Allergological, Occupational and Environmental Dermatology) series, in accordance