APJAI
Asian Pacific Journal of
Allergy and Immunology

ORIGINAL ARTICLE

Interleukin-18 correlates with interleukin-4 but not interferon-γ production in lymphocyte cultures from atopic dermatitis patients after staphylococcal enterotoxin B stimulation

Oki Suwarsa,[1] Sudigdo Adi,[1] Ponpon Idjradinata,[2] Endang Sutedja,[1] Erda Avriyanti,[1] Adlina Asfara,[1] Hendra Gunawan[1]

Abstract

Background: *Staphylococcus aureus (S. aureus)* triggers exacerbation of atopic dermatitis (AD) and causes chronic inflammation through the action of various proteins such as staphylococcal enterotoxin B (SEB). SEB has a role in activating interleukin (IL)-18, an important regulator of interferon (IFN)-γ and IL-4, in regards to a therapeutic strategy.

Objective: To determine the correlation of IL-18 level with the IL-4 and IFN-γ level in lymphocyte cultures from AD patients following SEB stimulation.

Method: Twenty patients with AD based on the Hanifin and Rajka criteria and 20 healthy subjects as a control group were selected. A 5 ml blood sample from each subject was taken for lymphocyte culture. The culture was stimulated with SEB for two days and the outcomes were assessed by enzyme-linked immunosorbent assays (ELISA) to evaluate the levels of IL-18, IL-4, and IFN-γ.

Results: In the AD group, the levels of IL-18, IL-4, and IFN-γ in lymphocyte cultures with SEB were significantly increased compared with non-SEB exposed cells (each $p<0.001$); similar results were found in the control group. The level of IL-18 was significantly elevated in lymphocyte cultures with SEB stimulation in AD vs. control ($p<0.05$) and without SEB in AD vs. control ($p<0.05$). Furthermore, IL-18 levels were significantly correlated with IL-4 levels and score atopic dermatitis (SCORAD) values in AD patients with SEB ($r=0.41$, $p<0.05$; and $r=0.70$, $p<0.05$, respectively); on the in contrary, there was no correlation between IL-18 and IFN-γ levels ($p=0.469$).

Conclusions: Our results suggest that IL-18 is correlated with increased of IL-4 levels in SEB-stimulated AD lymphocyte cultures.

Keywords: atopic dermatitis, interleukin-18, interleukin-4, interferon-γ, lymphocyte culture, staphylococcal enterotoxin B

From:
[1] Department of Dermatology and Venereology, Faculty of Medicine, Universitas Padjadjaran – Hasan Sadikin General Hospital, Bandung, West Java, Indonesia.
[2] Department of Pediatrics, Faculty of Medicine, Universitas Padjadjaran – Hasan Sadikin General Hospital, Bandung, West Java, Indonesia.

Corresponding author:
Oki Suwarsa
Department of Dermatology and Venereology
Faculty of Medicine, Universitas Padjadjaran – Hasan Sadikin General Hospital
Jl. Pasteur No. 38 Bandung, West Java, 40161, Indonesia
E-mail: okispkk@yahoo.co.id

Introduction

Atopic dermatitis (AD), also known as atopic eczema, is a chronic, relapsing, highly inflammatory skin disease characterized by pruritic eczematous skin lesions that usually presents in people with respiratory allergy. The prevalence of AD in children is 10% to 20%, and in adults is 1% to 3%.[1] The causes and mechanisms of AD are not completely understood.[2]

Antigen-specific T cells play an important role in AD pathogenesis at a cellular level,[3] and have been found in lesional skin of AD to produce different cytokines.[4,5] T helper (Th) 1 cells secrete interleukin (IL)-2 and interferon (IFN)-γ,[6] while Th2 cells secrete IL-4, IL-5, and IL-13.[6,7] IL-4 is the major factor regulating immunoglobulin (Ig) E production by B cells, and is required for optimal Th2 differentiation.[8] Furthermore, IL-4