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Increased Expression of Interleukin-17A in the Lesional Skin Indicates Increase of Serum Antibody Anti-phenolic Glycolipid-I in Leprosy Patients

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Abstract

Background: In lepromatous type of leprosy, the serum titer of the antibody against phenolic glycolipid (PGL)-I-specific antigen in leprosy that is produced by B cell is high. Whereas, the interleukin (IL)-17A expressed by T-helper 17 cells could induce B-cell differentiation. The role of IL-17A in leprosy is still unknown. Hence, this study aimed to assess a correlation between IL-17A expression in the lesional skin and anti-PGL-I immunoglobulin (Ig)M serum levels in leprosy patients. **Methods:** This study was performed in Leprosy Clinic, Dr. Hasan Sadikin Hospital Bandung, Indonesia, using a cross-sectional analytical study. A punch biopsy obtained from 49 leprosy patients was included through consecutive sampling for measurement of IL-17A expression in the skin by immunohistochemistry scoring (histoscore). Furthermore, the anti-PGL-I IgM level in serum is evaluated by enzyme-linked immunosorbent assay. **Results:** The IL-17A expressions in the skin biopsies of tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), and lepromatous leprosy (LL) patients using histoscore were 1.00, 2.31, 4.63, 5.06, and 10.14, respectively, and they showed significant differences ($P = 0.0001$). The titer of anti-PGL-I IgM levels was 89 pg/ml, 555 pg/ml, 1.244 pg/ml, 1.920 pg/ml, and 23.591 pg/ml in TT, BT, BB, BL, and LL patients, respectively, and they showed significant differences ($P = 0.005$). The results of Rank-Spearman correlation analysis between IL-17A expression in the skin and anti-PGL-I IgM serum levels were as follows: $r = 0.767$ and $P = 0.0001$. **Conclusion:** These results suggested that the increase of IL-17A expression in the lesional skin indicates the increase of anti-PGL-I IgM serum levels in leprosy.

Keywords: Anti-phenolic glycolipid-I immunoglobulin M, interleukin-17A, leprosy

INTRODUCTION

Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae*,¹⁻³ which mainly affects the peripheral nerves and the skin.¹⁻³ The important diversity of clinical and pathological findings related to leprosy is a result of the variable levels of cellular and humoral immunity to *M. leprae* among the patients affected by it.^{10,4} The abnormality of CD⁺ T-lymphocyte immunological function is the most important pathogenesis in leprosy. The T-helper (Th) 17 cell, a new kind of CD⁺ T-cell subpopulation found recently, highly produces interleukin (IL)-17 or IL-17A.⁵ The presence of IL-17A mRNA in the skin of lepromatous patients but not in tuberculoid (TT) type was performed by Cirse *et al.*⁶ However, the role of IL-17A in leprosy is still unknown.⁷

Lepromatous type of leprosy is known for high anti-phenolic glycolipid (PGL)-I immunoglobulin (Ig) M serum levels⁸

and the *M. leprae*-specific antigen.⁹⁻¹¹ This anti-PGL-I IgM is produced by B cells,¹⁰ while IL-17A also expressed by Th17 cells that could induce B-cell differentiation.¹² Thus, we want to know the correlation of IL-17A expression in the skin and anti-PGL-I IgM in leprosy patients.

METHODS

Patients

This study was an observational analytical study within cross-sectional design. Forty-nine leprosy patients (9 females

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