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Role of Pro-Inflammatory and Regulatory Cytokines in Pathogenesis of Graves' Disease in Association with Autoantibody Thyroid and Regulatory FoxP3 T-Cells

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Abstract : Background: Graves’ disease (GD) is an autoimmune thyroid disease. Imbalance of Th1/Th2 cells and T-regulatory (Treg)/Th17 cells was thought to play pivotal role in the pathogenesis of GD. Treg FoxP3 produced TGF-β to maintain regulatory function, and Th17 cells produced IL-17 as cytokines that were thought in mediating several autoimmune diseases. The aim of this study is to assess the role of IL-17 and TGF-β in the pathogenesis of GD and to investigate its correlation with Thyroid Stimulating Hormone Receptor Antibody (TRAb) and Treg FoxP3 expression. Method: 30 GD patients and 27 age and sex-matched controls were enrolled in this study. Diagnosis of GD was based on clinical and biochemical of GD. Serum IL-17, TGF-β, TRAb, and FoxP3 were measured by enzyme-linked immunosorbent assay (ELISA). Data were analyzed by using SPSS 21.0 (SPSS Inc.). Spearman rank correlation test was used for assessment of correlation. The statistical significance was accepted as P<0.05. Result: There was no significant correlation between IL-17 and TGF-β serum with expression of FoxP3 level in GD, but there was significant correlation between TGF-β and TRAb serum level (P<0.05). Serum levels of IL-17 and TGF-β were found to be elevated in patient group compared to control, where mean values of IL-17 were 14.43±2.15 pg/mL and TGF-β were 10.44±3.19 pg/mL in patients group; and in control group, level of IL-17 were 7.1±1.45 pg/mL and TGF-β were 4.95±1.35 pg/mL. Conclusion: Serum Il-17 and TGF-β were elevated in GD patients that reflect the role of inflammatory and regulatory cytokines activation in pathogenesis of GD. There was significant correlation between TGF-β and TRAb, revealing that Treg cytokines may play a role in pathogenesis of GD.

Keywords : IL-17, TGF-B, FoxP3, TRAb, Graves' disease
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