

The predominance of Th17 lymphocytes in contrast to the decreased number and function of T regulatory cells in pre-eclampsia

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Objective

The aim of the study was to estimate the prevalence of CD3+CD4⁺ T lymphocytes producing IL-17, IL-2, IFN- γ and IL-4, as well as CD4+CD25+FoxP3⁺ T regulatory cells (Tregs) in peripheral blood of patients with pre-eclampsia and healthy women in the third trimester of normal pregnancy. Furthermore, the purpose of our study was to assess the immunosuppressive activity of Treg cells of patients with pre-eclampsia in comparison with the controls.

Methods

Thirty four patients with pre-eclampsia and 27 healthy women in third trimester of pregnancy were included to the study. The percentage of CD4+CD25+FoxP3⁺ Treg cells and CD3+CD4⁺ T lymphocytes with intracellular expressions of cytokines were estimated using monoclonal antibodies and flow cytometry. The in vitro functional assays were performed with the use of Treg Cells Isolation Kit and 3H-thymidine.

Results

The percentages of T CD3+CD4⁺ lymphocytes producing IL-17A were significantly higher in pre-eclampsia when compared to healthy normotensive pregnant women in the third trimester of normal pregnancy ($p < 0.001$). The population of CD4+CD25+FoxP3⁺ Treg cells was significantly lower in the study group compared to the control group ($p < 0.05$). There were no changes in the stimulation index of CD3+CD4+CD25⁻ T lymphocytes of patients with pre-eclampsia during the in vitro assay without Treg cells and after the addition of autologous Tregs. In normal pregnancy the stimulation index of CD3+CD4+CD25⁻ T lymphocytes was significantly higher without Treg cells when compared to this response after addition of autologous Tregs ($p < 0.05$).

Conclusion

The results obtained suggest the up-regulation of Th17 immune response in pre-eclampsia. It seems that the decreased number and function of Treg cells may be responsible for the activation of inflammatory response in this disorder. In pre-eclampsia the predominance of Th17 immunity can act through the modulation of Th1/Th2 immune response.