

IFN- γ VERSUS IL-10 *IN SITU* EXPRESSION IN RECURRENT SPONTANEOUS ABORTION

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Abstract

Background: The possible immunological bases of recurrent spontaneous abortion (RSA) are still largely unknown, aberrant type 1 cytokine production; interferon- γ (IFN γ), and a defective type 2 cytokine; Interleukin-10 (IL-10) has been suggested to be related to the incidence of unexplained RSA.

Objective: To study the relation between the *in situ* expression of IFN γ and IL-10 in women with recurrent spontaneous abortion.

Materials and Methods: The study included three groups of women; Group A: patients had recurrent abortion (n=24), Group B: patients had spontaneous abortion for the first time (n=10), Group C: women with elective pregnancy termination (n=6). Curate samples obtained from these women were subjected for *in situ* hybridization technique to detect and determine the *in situ* expression of IFN- γ and IL-10.

Results: The *in situ* expression of IFN- γ was significantly higher in women with RSA as compared with normal pregnant and first abortion groups ($p=0.000$ and 0.002 respectively), while IL-10 expression was significantly lower in women with RSA as compared with first abortion group ($p=0.005$), and the ratio of IFN- γ /IL-10 was 1.97 in women with recurrent abortion, while that of normal pregnant and first abortion groups were 0.67 and 0.73 respectively.

Conclusion: The data of this study strengthened the possibility that type-1 immune response may have the upper hand in the pathology of RSA in association with reduction in the type-2 immune response.

Key words: RSA, IFN- γ , IL-10

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Introduction

Human pregnancy represents a semi-allograft to the maternal host. It is very interesting that the semi-allogeneic embryo/fetus is not rejected by the mother ⁽¹⁾. T helper (Th1)-dependant effector mechanisms such as cytotoxic T lymphocytes (CTL) activity play a central role in acute allograft rejection ⁽²⁾. The production of Th2-type cytokines or regulatory cytokines such as TGF- β and IL-10 may be central to the induction and maintenance of allograft tolerance ^(2, 3). So that, the physiological protection from maternal rejection, was hypothesized to be due to a Th2-type response at the materno-fetal interface ^(4, 5).

IL-10 was proposed to be a factor that might protect the semi-allogeneic fetus from maternal allo-recognition and rejection by driving the maternal (both local and systemic) immune reaction toward a Th2-type immune response ^(6,7), IL-10 is believed to play a major role in directing Th₀ cell differentiation toward a Th2 phenotype ^(8,9). IL-10 inhibits pro-inflammatory cytokines production including IL-1 β , IL-6, IL-8, TNF- α and IFN- γ ⁽¹⁰⁻¹²⁾, therefore prevents the development of Th1-type immune reactions deleterious for the maintenance of pregnancy ⁽⁵⁻¹³⁾.

In 1995, Th1-type cytokine secretion was observed for the first time in women with RSA, when peripheral blood mononuclear cells were activated by a trophoblast cell line ⁽¹⁴⁾. This finding was also supported by other reporters ⁽¹⁵⁻¹⁹⁾. Th1-type cytokines (IL-2, TNF- α , IFN- γ) can boost, and Th2-type cytokines (IL-3, IL-4, IL-10) can reduce abortion

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