
PROINFLAMMATORY CYTOKINES ARE MARKERS OF FETAL GROWTH RETARDATION

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Objective: To investigate the immune interrelationships between mother and fetus in chronic placental failure which leads to intrauterine fetal growth retardation (ILJGR).

Methods: In order to study the immune mechanisms of IUGR pathogenesis, we examined the levels of proinflammatory cytokines such as interleukin-1-alpha (IL-1a), interleukin-1-beta (IL-1b), interleukin-8 (IL-8) and tumor necrosis alpha factor (TNF-a) in maternal blood and in amniotic fluid. Cytokines were tested by double-antibody enzyme immunoassay. Amniotic fluid was obtained by amniocentesis before cesarean section or induced labor.

Results: The study was performed on 10 pregnant with preeclampsia, on 10 women with preeclampsia combined TUGR and on 10 females with uncomplicated pregnancy (control group) in 28-38 weeks of gestation period. The blood levels of IL-1a, IL-8 and TNF-a in patients with preeclampsia were more elevated than in healthy pregnant women. In pregnant with IUGR the blood levels of IL-1b, IL-8 and TNF-a were elevated more considerably than in women with preeclampsia without IUGR. Fetal funic blood from mothers with IUGR was characterized by higher levels of inflammatory cytokines (especially TNF-a and IL-1b) than those in fetuses of other groups. Women with IUGR had the most elevated amniotic fluid levels of IL-1b, IL-8 and TNF-a than women with normal pregnancy and pregnant with preeclampsia. High levels of inflammatory cytokines led to the intensification of hypercoagulation, decreased levels of antithrombin III and protein C, plasminogen consumption, increased levels of Fibrin degradation products.

Conclusions: These results suggest that the levels of proinflammatory cytokines can serve as diagnostic criteria for IUGR. Cardinal signs of IUGR are elevated levels of IL-1a and TNF-a in maternal blood and IL-1b, IL-8 in amniotic fluid.

INTERLEUKIN-8 IS AN INDICATOR OF INTRAAMNIOTIC INFECTION

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Objective: The purpose of this study was to examine the relationship between asymptomatic intrauterine infection and levels of proinflammatory cytokines in amniotic fluid, maternal and fetal blood.

Methods: Amniotic fluid of 23 pregnant with high risk of intrauterine infection was received by amniocentesis before amniotomy or cesarean section. Anhydrous period lasted from 1 till 6 hours. All patients had no sexually transmitted diseases and pathogenic microorganisms in cervical/vaginal culture. Cesarean sections were performed in 8 cases for the usual obstetric indication. 15 women delivered per vias naturales. Proinflammatory cytokines such as interleukin-1-alpha (IL-1a), interleukin-1-beta (IL-1b), interleukin-8 (IL-8) and tumor necrosis alpha factor (TNF-a) were tested by double-antibody enzyme immunoassay.

Results: All patients were divided in to two groups according to the results of placenta's histology examination. Group 1 was composed of women (n=12) without histologic responses of intraamniotic infection. Group 2 comprised (n=11) women with histologic evidence of inflammation (parietal deciduitis or choriodeciduitis, funiculitis). In these pregnant amniotic fluid levels of all three cytokines were elevated. In maternal blood elevated levels of IL-8 (253 ± 12 pg/ml, $p < 0,001$) and TNF-a (245 ± 11 Pg/ml, $P < 0,001$) were identified. Fetal funic blood from mothers of 1-st group was characterized by higher levels of TNF-a (306 ± 24 pg/ml, $p < 0,001$) and IL-1b (48 ± 4 pg/ml, $p < 0,001$). Only amniotic fluid level of IL-8 (786 ± 81 pg/ml, $p < 0,001$) was directly correlated with the presence of fetal inflammatory responses and the absolute neutrophil count of funic blood.

Conclusions: Interleukin-8 is a cytokine produced by human decidua in response to bacterial products. High levels of interleukin-8 in amniotic fluid can be regarded as an indicator of intraamniotic infection.