IMMUNO-INFLAMMATORY ALTERATIONS OF THE PLACENTA IN LATE PREGNANCY LOSS

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ABSTRACT

Late spontaneous abortion, occurring after the first trimester, represents a complex clinical entity associated with high maternal and fetal morbidity. While endocrine and anatomical causes have traditionally been emphasized, recent research highlights the importance of immune-mediated placental injury. Successful pregnancy requires a finely balanced immune tolerance at the maternal–fetal interface. Disruption of this balance can activate inflammatory pathways, leading to placental insufficiency and fetal demise.

Introduction

Late spontaneous abortion, occurring after the first trimester, represents a complex clinical entity associated with high maternal and fetal morbidity. While endocrine and anatomical causes have traditionally been emphasized, recent research highlights the importance of immune-mediated placental injury. Successful pregnancy requires a finely balanced immune tolerance at the maternal–fetal interface. Disruption of this balance can activate inflammatory pathways, leading to placental insufficiency and fetal demise.

Materials and Methods

Placental samples from 100 women with late spontaneous abortion were retrospectively analyzed. Immunohistochemical staining was performed to evaluate key inflammatory and angiogenic markers, including IL-6, TNF-α, IL-1β, IL-10, and CD34. Expression levels were assessed semi-quantitatively based on staining intensity and distribution within villous and intervillous regions. Statistical significance was defined at $p < 0.05$.

Results

Placental tissues from late pregnancy loss cases demonstrated a predominance of pro-inflammatory cytokine expression. IL-6, TNF-α, and IL-1β showed moderate to high expression in a substantial proportion of samples, indicating persistent inflammatory activation within the placental microenvironment.

In contrast, the anti-inflammatory cytokine IL-10 exhibited reduced expression, suggesting impaired immunological tolerance mechanisms. This imbalance between pro- and anti-inflammatory mediators reflects a shift toward a hostile intrauterine immune milieu.

Angiogenic activity, assessed via CD34 expression, was decreased in many samples, pointing to compromised endothelial integrity and suboptimal placental vascular

development. These findings imply that inflammatory signaling and angiogenic failure may act synergistically, exacerbating placental hypoperfusion.

Discussion

The observed cytokine profile supports the concept that late pregnancy loss is closely linked to immune-mediated placental dysfunction rather than isolated structural abnormalities. Elevated pro-inflammatory cytokines can promote endothelial damage, thrombogenic tendencies, and impaired trophoblast invasion. Concurrent suppression of IL-10 further weakens maternal immune tolerance toward fetal tissues.

Reduced CD34 expression underscores the vulnerability of placental vasculature under inflammatory stress. Insufficient angiogenesis limits oxygen and nutrient delivery, creating conditions incompatible with continued fetal development.

Importantly, this study highlights immune-inflammatory mechanisms independently, without duplicating morphological analyses, emphasizing their standalone pathogenic relevance.

Conclusion

Late spontaneous abortion is strongly associated with immune-inflammatory disturbances at the placental level. A dominance of pro-inflammatory cytokines combined with weakened anti-inflammatory regulation and impaired angiogenesis may represent a critical pathway leading to placental failure. Targeted assessment of these immunohistochemical markers may offer new opportunities for risk stratification and preventive strategies in high-risk pregnancies.

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