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DIFFERENTIATED IMMUNOPATHOGENETIC MECHANISMS OF ABNORMAL UTERINE BLEEDING DUE TO OVULATORY DYSFUNCTION: THE ROLE OF CONNECTIVE TISSUE DYSPLASIA AND PCOS

Background

AUB-O remains one of the leading causes of chronic blood loss, iron deficiency anemia, and reduced quality of life among women of reproductive age. According to the contemporary FIGO concept, in the absence of structural uterine pathology, functional and regulatory mechanisms become predominant, including immune-mediated and vascular factors [1]. The presence of undifferentiated connective tissue dysplasia is associated with congenital vascular fragility, impaired extracellular matrix organization, and defective tissue remodeling. These alterations support the hypothesis that immune-inflammatory processes play a pivotal role in the pathogenesis of AUB-O, contributing to endometrial instability and abnormal hemostatic responses [3].

Objective

To identify the specific features of immunopathogenetic mechanisms of abnormal uterine bleeding due to ovulatory dysfunction depending on the presence of undifferentiated connective tissue dysplasia and polycystic ovary syndrome.

Materials and Methods

The study included 93 women of reproductive age, divided into three groups: patients with AUB-O associated with UCTD (n=30), patients with AUB-O and PCOS without signs of connective tissue dysplasia (n=35), and a control group of healthy women with regular menstrual cycles (n=28). Serum levels of pro-inflammatory cytokines (IL-6, IL-8, TNF- α) and angio-regenerative factors (VEGF-A, TGF- β 1) were measured using standardized immunoassay techniques. Statistical analysis was performed using both parametric and non-parametric methods; differences were considered statistically significant at $p < 0.05$.

Results

Women with AUB-O in combination with UCTD demonstrated a significant increase in the concentrations of IL-6, IL-8, and TNF- α compared both to the control group and to patients with PCOS ($p < 0.001$), indicating marked activation of the systemic inflammatory response. In parallel, this group exhibited a substantial elevation of VEGF-A levels accompanied by a decrease in TGF- β 1, reflecting an imbalance between excessive pathological angiogenesis and insufficient reparative activity of the endometrium.

In contrast, patients with AUB-O associated with PCOS showed less pronounced immunological alterations, which are likely secondary to endocrine and metabolic disturbances. The observed increase in TGF- β 1 in this group may represent a compensatory mechanism aimed at maintaining endometrial regenerative processes and limiting excessive tissue damage.

Conclusion

AUB-O in women with UCTD is characterized by more pronounced immune-inflammatory and angio-regenerative disturbances compared to AUB-O associated with PCOS. The identified differences confirm the presence of distinct immunopathogenetic mechanisms underlying these conditions and substantiate the need for a personalized approach to diagnosis and treatment. The use of modern immunological biomarkers may significantly improve clinical decision-making and optimize therapeutic outcomes in women with abnormal uterine bleeding.

References:

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