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Advances in Immune Profiling of Endometriosis and Infertility

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Introduction

Endometriosis is a chronic gynecological disorder characterized by the ectopic presence of endometrial-like tissue outside the uterine cavity, affecting an estimated 10% of women of reproductive age. The condition is associated with chronic pelvic pain, dysmenorrhea and infertility, representing a major cause of reproductive morbidity. While retrograde menstruation and hormonal imbalances have long been recognized as contributors to endometriosis, growing evidence underscores the critical role of immune dysregulation in disease pathogenesis and its impact on fertility. The immune system, responsible for distinguishing self from non-self and maintaining tissue homeostasis, exhibits aberrant responses in women with endometriosis, contributing to ectopic implantation of endometrial cells, chronic inflammation and impaired reproductive outcomes. Recent advances in immune profiling techniques including flow cytometry, single-cell RNA sequencing and multiplex cytokine assays have enhanced our understanding of the immunological landscape in endometriosis, offering insights into mechanisms of infertility and potential therapeutic targets [1].

Description

The pathophysiology of endometriosis involves complex interactions between ectopic endometrial tissue and the peritoneal immune environment. In a healthy peritoneal cavity, immune surveillance mechanisms, including Natural Killer (NK) cells, macrophages and cytotoxic T lymphocytes, eliminate ectopic endometrial cells that may reach the peritoneal space during retrograde menstruation. In endometriosis, these mechanisms are disrupted, allowing ectopic cells to evade immune clearance. Uterine NK (uNK) cells, critical for both implantation and endometrial remodeling, exhibit reduced cytotoxicity in women with endometriosis, impairing clearance of ectopic tissue and altering endometrial receptivity. Macrophages in the peritoneal fluid are often polarized toward an M2 phenotype, which promotes tissue repair and angiogenesis but can also facilitate survival and proliferation of ectopic endometrial cells [2].

Cytokine profiles in endometriosis further illustrate immune dysregulation. Pro-inflammatory cytokines such as InterLeukin-6 (IL-6), Tumor Necrosis Factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) are elevated in the peritoneal fluid and systemic circulation of affected women. These cytokines contribute to a pro-inflammatory microenvironment, promoting adhesion, angiogenesis and fibrosis. Concurrently, anti-inflammatory cytokines, including InterLeukin-10 (IL-10) and transforming growth factor-beta (TGF-β), are dysregulated, leading to an imbalance that favors chronic inflammation. Chemokines such as Monocyte Chemoattractant Protein-1 (MCP-1) and regulated upon activation, normal T cell expressed and secreted (RANTES) recruit immune cells to ectopic lesions, perpetuating local inflammation and creating conditions unfavorable for successful implantation [1].

Recent immune profiling studies have also highlighted the role of adaptive immunity in endometriosis-associated infertility. Alterations in T cell subsets, including increased Th17 cells and reduced regulatory T cells (Tregs), disrupt immune tolerance mechanisms essential for successful implantation. Th17 cells produce pro-inflammatory cytokines such as IL-17, which exacerbate peritoneal inflammation, while Treg deficiency diminishes the immune tolerance required for endometrial receptivity. B cell activation and autoantibody production have also been observed in some patients, indicating a potential autoimmune component contributing to impaired fertility. Emerging high-throughput immune profiling techniques have refined our understanding of these mechanisms.

Single-cell RNA sequencing allows for detailed characterization of immune cell populations within endometriotic lesions, revealing heterogeneity in macrophages, dendritic cells and lymphocytes that was previously unrecognized. Flow cytometry and mass cytometry enable quantitative analysis of cell surface markers, intracellular cytokines and functional states of immune cells in both peripheral blood and peritoneal fluid. Multiplex cytokine arrays allow simultaneous measurement of multiple soluble mediators, facilitating the identification of cytokine signatures associated with infertility, pain severity and disease progression. Collectively, these techniques provide a comprehensive map of the immune microenvironment, identifying potential biomarkers for diagnosis, prognosis and treatment response [2].

Conclusion

Endometriosis represents a complex interplay between ectopic endometrial tissue and the maternal immune system, with immune dysregulation contributing significantly to infertility. Aberrant function of innate immune cells, including NK cells and macrophages, imbalances in T cell subsets such as Th17 and Tregs, dysregulated cytokine profiles and chronic peritoneal inflammation collectively impair gamete function, endometrial receptivity and embryo implantation.

Advances in immune profiling techniques including single-cell sequencing, flow cytometry and multiplex cytokine analysis have elucidated these mechanisms, providing novel insights into pathogenesis and identifying potential therapeutic targets. Integrating immune profiling with clinical management allows for personalized, mechanism-based interventions aimed at restoring immune balance, enhancing implantation success and improving fertility outcomes. Continued research in this area promises to transform the understanding and management of endometriosis-associated infertility, offering hope for women affected by this challenging condition.

Acknowledgement

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Conflict of Interest

None.

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