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Research Progress on Cytokine-Mediated Fibrosis in Intrauterine Adhesions

Lin Jin[1], Xu Ding[2], Yanhong Huang[2,*]

[1]*Shaanxi University of Chinese Medicine, Xianyang, Shaanxi, 712046, China*
[2]*Xi'an International Medical Center Hospital, Xi'an, Shaanxi, 710100, China*
**Corresponding author: 381874548@qq.com*

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Abstract: Intrauterine Adhesion (IUA) is a common intrauterine trauma disease in gynecology. Clinically, it mainly presents as amenorrhea, reduced menstrual flow, periodic abdominal pain, infertility, and recurrent miscarriage, seriously damaging the reproductive health of women of childbearing age. Recent studies have confirmed that immune inflammation imbalance is the core initiating mechanism mediating the occurrence and development of intrauterine adhesions. Cytokines, as the core regulatory mediators of the immune microenvironment, can participate in the transformation from physiological repair to pathological fibrosis after uterine endometrium injury by regulating the inflammatory cascade reaction, polarization of immune cells, activation of fibroblasts, and deposition of extracellular matrix. This article systematically reviews the mechanisms of normal intrauterine immune homeostasis and immune imbalance after injury, and summarizes the regulatory roles of various immune factors such as pro-inflammatory factors, anti-inflammatory factors, and chemokines in intrauterine adhesions and their downstream signaling pathways. The aim is to provide theoretical references for in-depth research on the mechanism of intrauterine adhesions and precise targeted immunotherapy.

1. Introduction

Cervical adhesions, also known as Asherman syndrome, is a pathological state where the basal layer of the endometrium is irreversibly damaged, resulting in abnormal adhesions between the uterine muscle layer and endometrial tissue, and partial or complete closure of the uterine cavity [1]. With the widespread popularity of uterine cavity operations such as induced abortion, curettage, and hysteroscopic surgery, the incidence of cervical adhesions has been increasing year by year, becoming an important cause of secondary infertility in women. Currently, the clinical treatment mainly focuses on hysteroscopic adhesion separation surgery, supplemented by estrogen to repair the endometrium and the placement of anti-adhesion materials, etc. However, the postoperative recurrence rate of patients with severe cervical adhesions can reach 30-60% [2-3]. Cytokines are the core molecules for signal transmission between immune cells, including pro-inflammatory factors, anti-inflammatory factors, and chemotactic factors. They precisely regulate the entire process of inflammation initiation, amplification, and resolution. After mechanical injury to the endometrium, local immune cells secrete

various cytokines, which successively mediate excessive activation of inflammation and remodeling of the matrix fibers, ultimately inducing the formation of cervical adhesions. This cascade reaction runs through the entire process of disease occurrence and development. [4]. Clarifying the specific mechanisms and interactive regulatory relationships of various cytokines in cervical adhesions is of great significance for revealing the pathogenesis of cervical adhesions and exploring new diagnostic and therapeutic targets.

2. Physiological Repair of the Endometrium and Uterine Cavity Immune Homeostasis

2.1. Physiological Characteristics of Endometrial Repair

The endometrium is a functional tissue with extremely strong regenerative capacity. Under normal physiological conditions, it can complete the cyclical process of proliferation, secretion, and shedding during the menstrual cycle, and can achieve scarless repair even after minor trauma. The physiological repair of endometrial damage mainly consists of three stages: the early stage of brief and controllable inflammatory activation, where a small number of immune cells infiltrate the damaged area to remove necrotic tissues and foreign substances; the middle stage of cell proliferation repair, where endometrial epithelial cells and stromal cells proliferate orderly to fill the damaged defects; the later stage of matrix remodeling, where the extracellular matrix is moderately synthesized and degraded to complete the reconstruction of the tissue structure, ultimately restoring the normal physiological morphology and function of the endometrium, with no excessive fibrosis or scar tissue formation throughout the process [5].

2.2. Normal Characteristics of the Uterine Cavity Immune Microenvironment

There exists a stable innate and adaptive immune system within the uterine cavity, which is the core foundation for maintaining the homeostasis and repair of the endometrium. The innate immune cells mainly include macrophages, neutrophils, and natural killer cells (NK cells), and they are the main cells for the early response to uterine cavity injuries; the adaptive immune cells are mainly composed of CD4+ T cells, CD8+ T cells, and B cells, and they participate in the precise regulation and resolution of inflammatory responses [6]. In a normal uterine microenvironment, pro-inflammatory immune factors and anti-inflammatory immune factors are in a dynamic equilibrium state. Low levels of pro-inflammatory factors can initiate normal injury repair signals and activate cell proliferation; anti-inflammatory factors can promptly inhibit excessive inflammation and prevent continuous tissue damage; chemokines orderly regulate the recruitment and disappearance of immune cells, ensuring the timeliness of the inflammatory response. This immune homeostasis can ensure that the inflammatory response after uterine endometrial injury is moderate and the repair process is orderly, fundamentally avoiding the occurrence of pathological fibrosis [7-8].

3. The Core Pathological Mechanism of Cytokine-Mediated Uterine Cavity Adhesion

Gynecological surgeries and infections can directly damage the basal layer structure of the endometrium, disrupt the immune homeostasis of the uterine cavity, induce abnormal expression of various cytokines, and trigger persistent and uncontrolled inflammatory responses. This pathological immune imbalance will form a vicious cycle of "inflammatory infiltration - tissue damage - fibrotic remodeling", inhibit the regeneration of endometrial stem cells, induce abnormal activation of fibroblasts, promote the accumulation of extracellular matrix, and ultimately lead to the formation of uterine cavity scar adhesions [9-10]. Different types of cytokines play differential and mutually coordinated regulatory roles in this pathological process.

3.1. Pathogenic Mechanism of Pro-inflammatory Cytokines

Pro-inflammatory immune factors are the core for initiating and amplifying the inflammatory response in intrauterine adhesions. They are abnormally highly expressed after injury and mediate the damage and fibrotic process of the endometrium through multiple pathways. Tumor necrosis factor-α (TNF-α) is the core pro-inflammatory factor in the early stage of intrauterine injury. During the normal repair process of the endometrium, TNF-α is transiently expressed at a low level and participates in the clearance of necrotic tissues. However, in the post-traumatic intrauterine tissues, TNF-α is persistently highly expressed. On the one hand, it can directly induce apoptosis of endometrial epithelial cells and stromal cells, disrupting the basis of endometrial regeneration; on the other hand, it can activate local fibroblasts, promoting the massive synthesis and secretion of type I and type III collagen, while inhibiting collagen degradation, resulting in abnormal deposition of extracellular matrix [11]. Moreover, TNF-α can further activate downstream inflammatory pathways, amplifying the inflammatory cascade reaction and aggravating intrauterine tissue damage [12]. Studies have reported that the endometrium in patients with IUA exhibits a state of persistent inflammatory activation, which is distinctly different from the transient inflammation observed during physiological cyclical endometrial shedding. Compared to normal endometrium at the same stage, although CD138 is negative, levels of inflammatory factors such as TNF-α are significantly elevated in the late proliferative phase endometrium of IUA patients [13].

The interleukin family members IL-1β and IL-6 are important pro-inflammatory factors. IL-1β is mainly secreted by activated macrophages and can directly mediate local inflammation infiltration in the uterine cavity, disrupt the uterine endometrial barrier structure, and simultaneously induce the expression of fibrosis-related genes, promoting scar tissue formation [14]. IL-6 can exacerbate immune imbalance by regulating the differentiation of immune cells, promoting the proliferation of Th17 cells, inhibiting the function of regulatory T cells, and activating fibroblasts to transform into myofibroblasts, thereby significantly enhancing collagen synthesis capacity and accelerating the process of uterine cavity fibrosis, ultimately leading to permanent scar adhesion in the uterine cavity [15].

3.2. The Mechanism of Imbalance in Anti-inflammatory Cytokine Protection

Anti-inflammatory cytokines play a negative regulatory role in the repair of normal endometrium, capable of limiting excessive inflammation activation and inhibiting the progression of fibrosis. However, when intrauterine adhesions occur, the expression of these factors significantly decreases, and their anti-inflammatory protective function is lost, further exacerbating immune imbalance.

IL-10 is a core protective anti-inflammatory factor in the uterine cavity, mainly secreted by regulatory T cells and M2-type macrophages. Its main function is to broadly inhibit the synthesis and release of pro-inflammatory factors (such as TNF-α, IL-1β, IL-6, etc.), blocking the inflammatory cascade reaction; at the same time, it can inhibit the excessive activation and proliferation of fibroblasts, reduce collagen deposition, and alleviate tissue fibrosis. In physiological endometrial repair, IL-10 can timely terminate the inflammatory response, ensuring the orderly completion of the repair process. However, in patients with intrauterine adhesions, the expression level of IL-10 at the damaged site is significantly reduced, the anti-inflammatory barrier is damaged, and it is unable to effectively counteract the excessive inflammatory response, resulting in persistent inflammation in the damaged area and promoting the progression of adhesions [16].

Transforming growth factor-β (TGF-β) exhibits a typical bidirectional regulatory function and is a key molecule connecting inflammation and fibrosis. At normal physiological concentrations, TGF-β participates in the proliferation of endometrial cells, tissue repair, and matrix remodeling, playing a positive role in maintaining the integrity of the endometrium; however, after uterine cavity trauma,

the local microenvironment disorder will induce abnormal high expression of TGF-β. At this time, its repair function is lost and it instead exerts a strong pro-fibrotic effect [17]. High concentrations of TGF-β can activate the TGF-β/Smad signaling pathway, induce endometrial epithelial-mesenchymal transformation, promote the activation and proliferation of fibroblasts, significantly increase the synthesis efficiency of extracellular matrix components such as collagen and fibronectin, and simultaneously inhibit matrix degradation [18].

3.3. Mechanism of Immune Cell Infiltration Mediated by Chemokines

Chemokines (CCL, CXCL family) are the core mediators that regulate the directional migration of immune cells. They precisely recruit various immune cells to infiltrate the damaged site of the uterine cavity, reshape the local immune microenvironment, and indirectly regulate the process of uterine cavity adhesions. During normal repair, chemokines are expressed briefly, orderly recruit immune cells to complete the repair of the injury and then rapidly fade away; while after uterine cavity trauma, chemokines such as CCL2, CCL5, and CXCL8 are continuously highly expressed, which can massively recruit neutrophils, M1 type pro-inflammatory macrophages, Th17 cells, etc., to gather at the injury site. The infiltrated immune cells can continuously secrete a large amount of pro-inflammatory factors, further amplifying the inflammatory injury and inducing macrophages to polarize towards the pro-fibrotic M2 type, which can massively secrete factors such as TGF-β and IL-6, continuously promoting the progression of uterine cavity adhesions [19-21]. Thus, although chemokines do not directly mediate fibrosis, they regulate the infiltration of immune cells and are an important hub for maintaining chronic inflammation and driving the continuous progression of adhesions.

3.4. Interacting Regulatory Network of Cytokines

Various cytokines do not function independently but form a complex cross-regulatory network that jointly determines the outcome of endometrial repair. Pro-inflammatory factors TNF-α and IL-1β can mutually induce expression, amplifying the inflammatory response and simultaneously inhibiting the secretion of anti-inflammatory factors such as IL-10, disrupting the anti-inflammatory balance; anti-inflammatory factor IL-10 can negatively feedback to inhibit the expression of all core pro-inflammatory factors, limiting the excessive progression of inflammation; chemokines act as upstream regulatory molecules, mediating the recruitment of various immune cells, providing a cellular basis for the continuous secretion of inflammatory factors; TGF-β can synergize with various pro-inflammatory factors to further strengthen the fibrotic signal [22-23]. The regulatory network of multiple factors that antagonize and cooperate with each other ultimately leads to the normal repair of minor damage or the pathological adhesion remodeling after severe damage.

4. Application Value of Cytokines in the Diagnosis and Treatment of Intrauterine Adhesions

4.1. Diagnostic Markers and Prognostic Evaluation Indicators

The abnormal expression of cytokines is closely related to the occurrence, severity, and recurrence risk of intrauterine adhesions. It has the potential value for non-invasive diagnosis and prognosis assessment. Studies have shown that in the endometrium tissue of intrauterine adhesions, the expression of genes related to cell migration and cell-to-cell communication is mainly downregulated. The downregulation of IL1B expression is significantly associated with endometrial adhesions; the activation of MAPK and NOD-like receptor signaling pathways and gene alternative splicing events are active in the endometrium tissue at the adhesion site [24]. Clinical research has found that the

expression levels of TNF-α, IL-6, and TGF-β in serum and uterine lavage fluid increase significantly with the increase in the grade of intrauterine adhesions, while the expression level of IL-10 decreases with the aggravation of the disease. Multi-factor combined detection can effectively distinguish mild, moderate, and severe intrauterine adhesions [25]. Compared with traditional invasive hysteroscopy examination, immune factor detection is simple to operate, has less trauma, and can be used for early screening, dynamic monitoring of the condition, and prognosis assessment.

4.2. Research Progress in Immunotargeted Therapy

Based on the core pathogenesis of immune imbalance, immune-targeted regulation has become a new research direction for the prevention and treatment of intrauterine adhesions. Basic research has confirmed that by blocking the activities of pro-inflammatory factors such as TNF-α and IL-6 with inhibitors, it is possible to effectively reduce intrauterine inflammatory infiltration and inhibit the progression of fibrosis [26]; in addition, stem cell therapy can regulate the intrauterine immune microenvironment through paracrine effects, balance the expression of pro-inflammatory and anti-inflammatory factors, and achieve multiple effects such as anti-inflammation, inhibition of fibrosis, and promotion of endometrial regeneration, and has currently entered the preliminary clinical exploration stage [27]. Various immune-targeted intervention methods provide new ideas for the treatment of intrauterine adhesions.

5. Current Problems and Controversies in Research

At present, the related research on cytokines in intrauterine adhesions has achieved some phased progress, but there are still many deficiencies and controversies, which limit the clinical application and transformation. First, some core cytokines have bidirectional regulatory characteristics, with TGF-β being the most typical. The concentration thresholds for repair and fibrosis promotion, as well as the temporal and spatial expression characteristics of these cytokines, have not been fully clarified. There are also some controversies regarding the expression patterns in different studies. Second, the existing research mainly focuses on basic cell experiments, animal experiments, and small-sample clinical observations. There is a lack of large-sample, multi-center, and long-term follow-up clinical studies, and the diagnostic thresholds, effectiveness, and safety of cytokines have not been fully verified. Third, the regulatory network of cytokines is highly complex, with various factors interacting and influencing each other. Current research mostly focuses on the effects of a single or a few factors, lacking a systematic analysis of the overall regulatory network. The combined targeted intervention mechanism is still unclear. Fourth, there is currently no unified standard for cytokine detection, sampling timing, reference thresholds, and diagnostic evaluation systems, making it difficult to apply them in routine clinical diagnosis and treatment.

6. Summary and Outlook

The essence of intrauterine adhesions is the pathological fibrotic remodeling mediated by the imbalance of immune homeostasis after endometrial injury. Cytokines play a core regulatory role in this process. Pro-inflammatory factors (TNF-α, IL-1β, IL-6, IL-17) initiate and amplify the inflammatory response, driving the progression of fibrosis; anti-inflammatory factors (IL-10, low concentration TGF-β) maintain the homeostasis of endometrial repair, and their deficiency exacerbates the condition; chemokines maintain the chronic inflammatory microenvironment by regulating the infiltration of immune cells. Various cytokines form a cross-regulatory network through core signaling pathways such as NF-κB, JAK/STAT, and TGF-β/Smad, jointly mediating the transformation of the endometrium from physiological repair to pathological adhesions. Future

research focuses on the shortcomings of existing studies to further deepen the mechanism research and clinical translation. Firstly, explore the spatiotemporal specific expression patterns and bidirectional regulatory mechanisms of immune factors, clarify the threshold and regulatory conditions of factors such as TGF-β, and resolve current research controversies; secondly, conduct large-sample clinical studies to verify the clinical diagnostic and therapeutic value of immune factors and establish standardized detection and evaluation systems; thirdly, promote the clinical translation of new therapies such as immune-targeted drugs and stem cell immune regulation, explore multi-target combined intervention schemes, and provide new strategies for the precise prevention and treatment of intrauterine adhesions.

In conclusion, the in-depth study of the immune imbalance mechanism will completely improve the theory of intrauterine adhesions pathogenesis, providing important support for breaking through clinical treatment bottlenecks and improving the reproductive prognosis of patients.

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