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A Review of the Correlation between Disease Outcomes in Female Patients with Immune Thrombocytopenic Purpura and the Lung-Heat and Kidney-Deficiency Syndrome

Yaohan Yue, Qingqing Yu, Dongqing Zhou, Lei Wang*

Department of Traditional Chinese Medicine, Shanghai Putuo District People's Hospital, Shanghai 200600, China

**Corresponding author: Lei Wang, 18917792582@163.com*

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Abstract

Immune thrombocytopenic purpura (ITP) is an autoimmune hematological disorder characterized by excessive peripheral platelet destruction and impaired bone marrow megakaryocyte production. The disease outcomes vary significantly among different genders and age groups, with female patients often exhibiting a more complex disease progression due to factors such as unique endocrine changes, immune dysregulation, and traditional Chinese medicine (TCM) constitutional characteristics. Recent studies have suggested a potential significant association between the TCM Lung-Heat and Kidney-Deficiency syndrome and the progression and prognosis of ITP. However, the underlying mechanisms remain incompletely understood. This review summarizes the characteristics of disease outcomes in female patients with ITP, including clinical phenotypes, treatment responses, and long-term prognosis. It also delves into the pathological basis of the Lung-Heat and Kidney-Deficiency syndrome in TCM and its potential link to the disease outcomes of ITP. Additionally, this review evaluates the potential role of integrated traditional Chinese and Western medicine in improving disease outcomes for female patients with ITP and proposes directions for future research, aiming to provide evidence-based guidance for optimizing individualized treatment plans.

Keywords

Immune thrombocytopenic purpura

Female patients

Disease outcome

Lung heat and kidney deficiency syndrome

Integrated traditional Chinese and Western medicine treatment

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1. Introduction

Immune thrombocytopenic purpura (ITP) is a complex autoimmune disorder characterized by increased platelet destruction and decreased platelet production. Its pathogenesis involves multiple factors, including abnormal B-cell activation, autoantibody production, and T-cell immune dysregulation. Notably, gender plays a significant role in the clinical presentation and treatment response of ITP. Clinical data indicate that female patients, due to hormonal fluctuations and gender-specific immune system characteristics, often exhibit a more prolonged disease course and demonstrate lower response rates to glucocorticoid therapy. This may be associated with estrogen's regulatory effects on B-cell activation and autoimmune responses [1]. Particularly noteworthy is the significant fluctuation in platelet levels observed in pregnant patients with ITP, suggesting that hormonal environments exert a substantial impact on disease outcomes [2].

According to traditional Chinese medicine (TCM) theory, the pathological mechanisms of ITP are closely related to the "lung heat and kidney deficiency syndrome". TCM posits that the lungs govern "Qi" and regulate respiration; lung heat can lead to reckless movement of blood, presenting clinically as bleeding tendencies such as skin purpura and epistaxis. The kidneys govern bones and produce marrow; kidney deficiency impairs hematopoietic function in the bone marrow, resulting in insufficient platelet production. Clinical observations reveal that patients with this syndrome typically exhibit symptoms such as dry mouth and throat, tidal fever, night sweats, and soreness and weakness in the lower back and knees. Modern medical research confirms that patients with this syndrome often display pathological features such as Th1/Th2 cell imbalance, abnormally elevated levels of inflammatory cytokines, and impaired maturation of bone marrow megakaryocytes, aligning with the TCM pathogenesis of "lung heat and kidney deficiency" [1,3].

In recent years, the association between the use of immune checkpoint inhibitors such as PD-1 inhibitors and ITP has garnered attention. Case reports indicate that such drugs may induce immune thrombocytopenic purpura (ITP) or other thrombotic microangiopathies by disrupting immune tolerance, highlighting the central role of immune dysregulation in the pathogenesis of ITP [4]. Furthermore, ITP patients with genetic factors such as LRBA gene mutations and those with concurrent autoimmune diseases such as type 1 diabetes and arthritis exhibit more complex clinical phenotypes, further suggesting the interactive effects of the immune-endocrine network [5]. Future research directions should integrate multi-omics technologies, including genomics and proteomics, to deeply explore the molecular characteristics of the "lung heat and kidney deficiency" syndrome and provide evidence-based support for the integrated treatment of ITP with traditional Chinese and Western medicine.

2. Main text

2.1. Pathological mechanisms and clinical manifestations of immune thrombocytopenic purpura

2.1.1. Immune dysregulation and platelet destruction

The core pathological mechanism of immune thrombocytopenic purpura (ITP) primarily stems from excessive platelet clearance triggered by immune system dysfunction. Studies have shown that ITP patients possess autoantibodies targeting platelet membrane glycoproteins such as GPIIb/IIIa, which are recognized and cleared by splenic macrophages through Fc receptor-mediated phagocytosis, thereby accelerating platelet degradation. Notably, clinical observations have revealed that immune checkpoint blockade therapy, particularly PD-1 inhibitors may induce ITP-like immune-related thrombocytopenia, such as the recently reported case of pembrolizumab-induced thrombotic thrombocytopenic purpura (TTP), whose mechanism is associated with ADAMTS-13 inhibitory autoantibodies. Immune dysregulation may also involve T-cell dysfunction, such as a shift in the Th1/Th2 balance and a reduction in regulatory T cells (Tregs), further exacerbating platelet destruction [6,7].

2.1.2. Mechanisms of reduced platelet production

In the pathogenesis of immune thrombocytopenia (ITP), in addition to increased platelet destruction, platelet production is also suppressed in ITP patients. Congenital immune deficiencies may interfere with platelet production through multiple mechanisms such

as chronic infections or autoimmune processes, with impaired maturation of bone marrow megakaryocytes being a significant contributing factor. This impairment may be related to direct targeting of megakaryocytes by autoantibodies or abnormal levels of cytokines including thrombopoietin and TPO. Clinical observations have revealed that in cases of LRBA gene deficiency complicated by immune thrombocytopenia (ITP), abnormal immune regulation not only leads to platelet destruction but may also indirectly inhibit platelet production by affecting megakaryocyte differentiation [8]. Additionally, when certain immune-mediated diseases such as autoimmune hepatitis or celiac disease coexist with ITP, the systemic inflammatory state may further suppress bone marrow hematopoietic function.

2.1.3. Unique clinical manifestations in female patients

Female patients with ITP exhibit distinct clinical characteristics. Epidemiological data indicate a higher incidence of ITP among women of childbearing age, suggesting a close association with estrogen-mediated immune regulatory mechanisms. For instance, female patients with 22q11.2 deletion syndrome are more prone to developing autoimmune diseases such as ITP and juvenile idiopathic arthritis, a phenomenon linked to immune tolerance defects resulting from thymic developmental abnormalities [9]. Furthermore, ITP may worsen during pregnancy in female patients, necessitating vigilance for the risk of neonatal thrombocytopenia. Reported cases of immune checkpoint inhibitor-related ITP also predominantly involve female patients, indicating a potential influence of gender factors in immune-mediated thrombocytopenia [10]. Elderly female patients often present with chronic ITP, characterized by a low rate of spontaneous remission, prolonged disease course, and significant impact from comorbidities, thereby increasing the complexity of diagnosis and treatment [11].

2.2. Characteristics and influencing factors of disease outcomes in female patients

2.2.1. Impact of hormonal levels on disease outcomes

Changes in female hormonal levels significantly influence the outcomes of various diseases. Sex hormones such as estrogen and progesterone not only participate in the regulation of the reproductive system but also affect immune homeostasis, inflammatory cascades, and vascular biological properties through multiple mechanisms. Research indicates that estrogen possesses immunomodulatory effects, potentially playing a protective role in certain autoimmune diseases by upregulating the expression of anti-inflammatory cytokines such as IL-10 and inhibiting the expression of pro-inflammatory factors such as TNF-α. However, excessively high levels of estrogen may also lead to overactivation of the immune system, exacerbating the severity of certain diseases [11].

2.2.2. Gender differences in the immune system and disease outcomes

Gender differences in the immune system are one of the important factors contributing to the characteristics of disease outcomes in female patients. Females typically exhibit stronger immune responses, including higher antibody production capacity, more active cellular immune responses, and more pronounced inflammatory responses. These gender differences in the immune system may be related to the effects of sex hormones, the expression of genes on the X chromosome, and epigenetic regulation [12]. Collectively, these studies suggest that the stronger immune response in females is shaped by a combination of sex hormones, genetic factors, and immune regulatory mechanisms, but this also leads to an increased risk of autoimmune diseases [13]. Therefore, when formulating individualized treatment plans, it is essential to fully consider gender differences in the immune system to optimize treatment outcomes and minimize adverse reactions.

2.2.3. The relationship between pregnancy and disease outcomes in ITP

Pregnancy has a complex impact on the disease outcomes of patients with immune thrombocytopenia (ITP). The platelet counts of newborns from patients newly diagnosed with ITP during pregnancy are significantly higher than those from patients diagnosed before pregnancy ($p < 0.05$), suggesting that ITP onset during pregnancy may represent a more benign subtype [14]. However, while 58% of pregnant women with ITP can maintain relatively stable platelet levels without treatment, some patients may experience a further decline

in platelet counts, increasing the risk of bleeding and requiring intervention until delivery [15,16]. The incidence of postpartum hemorrhage (PPH) in pregnant women with ITP is 23.2%, significantly higher than in the general population [17]. Clinical management should be stratified based on medical history, family history of the disease, and treatment responsiveness.

2.3. The pathological basis of traditional Chinese medicine's lung-heat and kidney-deficiency syndrome and its association with ITP

2.3.1. Relationship between lung-heat syndrome and uncontrolled blood heat

In traditional Chinese medicine (TCM) theory, the lungs govern "Qi" and regulate respiration, and are closely related to the body's defensive functions. When pathogenic heat invades the body externally, it first affects the lungs, causing impaired dispersion and descent of lung "Qi", which then transforms into heat. Alternatively, fire from other organs, such as liver fire or stomach fire, can ascend and affect the lungs, leading to lung heat. From a pathological perspective, when pathogenic factors invade the lung's defensive layer and disrupt the dispersion of lung "Qi", pathogenic heat accumulates in the lungs, condenses fluids into phlegm, and the combination of phlegm and heat damages the lung's collateral vessels. This, in turn, affects the circulation of "Qi" and blood throughout the body, resulting in uncontrolled blood heat, where heat pathogens force blood to flow irregularly, causing it to deviate from its normal pathways and overflow from the vessels. This shares similarities with the bleeding tendency observed in idiopathic thrombocytopenic purpura (ITP). Modern medicine suggests that ITP is associated with immune abnormalities, and the TCM concept of "uncontrolled blood heat" can partially explain the pathological process of accelerated platelet destruction and bleeding symptoms in ITP patients. Lung-heat syndrome may exacerbate abnormal platelet destruction by influencing immune regulation, thereby relating to the pathogenesis of ITP.

2.3.2. Relationship between kidney deficiency syndrome and bone marrow hematopoietic function

The kidneys are considered the foundation of prenatal constitution, governing the storage of essence and the production of marrow, and are closely related to bone marrow hematopoietic function. Kidney deficiency syndrome in TCM is characterized by symptoms such as soreness and weakness in the lower back and knees, dizziness, tinnitus, and fatigue. Its essence may be related to abnormalities in the bone marrow hematopoietic microenvironment or decreased hematopoietic stem cell function. In ITP patients, some cases exhibit impaired maturation of bone marrow megakaryocytes or reduced platelet production, which aligns with the TCM theory of "kidney deficiency and marrow depletion". Modern research indicates that kidney deficiency may indirectly lead to insufficient platelet production by affecting the secretion of hematopoietic growth factors or the proliferation and differentiation of hematopoietic stem cells. Therefore, kidney deficiency syndrome may represent an important pathological basis for thrombocytopenia in ITP patients [18].

2.3.3. Manifestations of lung-heat and kidney deficiency syndrome in female ITP patients

Among female ITP patients, the clinical manifestations of lung-heat and kidney deficiency syndrome are particularly prominent. Lung-heat syndrome may present with symptoms of uncontrolled blood heat, such as skin purpura, epistaxis, and gum bleeding. Meanwhile, kidney deficiency syndrome often manifests as heavy menstrual bleeding, prolonged menstrual periods, and soreness and weakness in the lower back and knees, indicating insufficient kidney "Qi". Due to physiological characteristics such as menstruation, pregnancy, childbirth, and lactation, which can easily deplete "Yin" and blood, women are more prone to developing complex pathological mechanisms involving concurrent lung heat and kidney deficiency. Therefore, when treating ITP in women, it is necessary to simultaneously clear lung heat and tonify kidney deficiency to regulate immune function and improve platelet production.

2.4. The impact of integrated traditional Chinese and Western medicine on the prognosis of ITP

2.4.1. Efficacy and limitations of Western medicine treatments

The primary treatment methods for ITP in

Western medicine include glucocorticoids and immunosuppressants. First-line treatment typically involves glucocorticoids such as prednisone, which can significantly increase platelet counts in the short term by inhibiting antibody-mediated platelet destruction, with approximately 70% of patients achieving remission in the initial stages of treatment. However, long-term use of glucocorticoids may lead to elevated blood sugar levels (20–30%), osteoporosis, and an increased risk of infections [19]. Second-line treatments include thrombopoietin receptor agonists (TPO-RAs, such as romiplostim/eltrombopag), which can enhance platelet production but carry risks of hepatotoxicity (eltrombopag) and thrombosis (with an incidence rate of 3–5%). Rituximab (an anti-CD20 monoclonal antibody) targets and clears B cells, proving effective in some refractory ITP patients with an efficacy rate of 40–60%; however, it poses an increased risk of infections and exhibits significant individual variations in efficacy [19].

Splenectomy may also be performed for patients who do not respond to drug therapy, achieving a long-term remission rate of over 60%, but its application is limited by surgical complications and postoperative infection risks [20]. For pregnant patients with ITP, intravenous immunoglobulin (IVIg) or low-dose hormones are preferred to reduce the risk of fetal malformations [21]. For patients with menorrhagia, a levonorgestrel-releasing intrauterine device (LNG-IUD) can reduce bleeding volume, with a 50% reduction in PBAC scores, although some patients may experience irregular uterine bleeding. Therefore, while Western medicine treatments can rapidly control symptoms, their long-term efficacy and safety still require optimization [22].

2.4.2. Clinical efficacy of traditional Chinese medicine syndrome differentiation and treatment

Traditional Chinese medicine classifies ITP under the category of "blood disorders" and emphasizes treatment based on syndrome differentiation. For the syndrome type of excessive heat-toxin, clinical practice often employs formulas such as Xijiao Dihuang Decoction with the effects of clearing heat and detoxifying, cooling blood, and stopping bleeding. For the syndrome type of deficiency in both the spleen and kidney, formulas such as Guipi Decoction with the effects of strengthening the spleen and tonifying the kidney, replenishing "Qi" and nourishing blood are mainly used. Data from multiple clinical studies have confirmed that traditional Chinese medicine (TCM) compound preparations can not only effectively alleviate patients' bleeding symptoms but also have a significantly lower incidence of adverse drug reactions compared to conventional Western medicine treatments [9]. The integrated traditional Chinese and Western medicine therapy for treating immune thrombocytopenia (ITP) demonstrates significant advantages across multiple clinical indicators, including increasing peripheral blood platelet counts, regulating levels of regulatory T cells (Tregs) and helper T cells ($CD4^+$); it also reduces traditional Chinese medicine (TCM) syndrome scores, levels of cytotoxic T cells ($CD8^+$), and platelet-associated immunoglobulin G (PAIgG) levels. Additionally, this therapy exhibits marked advantages in enhancing the stability of long-term efficacy and reducing adverse drug reactions. The core value of TCM in treating ITP lies in its individualized diagnosis and treatment model and holistic regulatory treatment philosophy. However, it has limitations such as a relatively slow onset and longer treatment duration. Currently, the TCM diagnosis and treatment system lacks standardized efficacy evaluation indicators, and the reproducibility of its clinical efficacy still requires further verification through large-sample, multicenter clinical studies [23].

2.4.3. Synergistic effects of integrated traditional Chinese and Western medicine therapy and improvement in disease outcomes

The integrated traditional Chinese and Western medicine therapy significantly improves disease outcomes in ITP through complementary advantages. Western medicine rapidly increases platelet counts, while TCM reduces hormone dosage and recurrence rates. For example, a retrospective study showed that the overall response rate in the combined therapy group was significantly higher than that in the Western medicine-only group, and the time for hormone reduction was significantly shortened [24]. Mechanistically, the active ingredients in TCM herbs such as *Astragalus* and *Angelica* can indirectly correct immune imbalances by regulating T cell subset differentiation such as increasing Tregs and

inhibiting inflammatory factors such as TNF-α and IL-17, forming a complement to Western medicine [25]. Long-term follow-up data indicate that the 5-year progression-free survival rate of patients receiving combined therapy is significantly higher, and their quality-of-life scores are significantly better than those of patients receiving a single therapy [23].

2.5. Future research directions and clinical practice recommendations

2.5.1. Research on the molecular mechanisms of lung-heat and kidney-deficiency syndrome

As a complex syndrome in Traditional Chinese Medicine (TCM) theory, the molecular mechanisms underlying Lung-Heat and Kidney-Deficiency Syndrome remain in the exploratory stage. Existing literature suggests that immune dysregulation may be a core pathological feature of this syndrome. For instance, adverse events associated with immune checkpoint inhibitor therapy (e.g., PD-1 inhibitors), and thrombocytopenic purpura, indicate a link between excessive immune activation and tissue damage [26]. Future research could focus on the following directions: first, utilizing multi-omics technologies (e.g., transcriptomics, proteomics) to analyze immune biomarker profiles in patients with Lung-Heat and Kidney-Deficiency Syndrome, clarifying the relationship between Th1/Th2 cell imbalance, inflammatory cytokines including IL-6, TNF-α, and the syndrome; second, exploring the role of coagulation-related molecules, such as ADAMTS-13, in the development of bleeding tendencies associated with this syndrome, providing potential targets for integrated TCM-Western medicine therapies. Additionally, monogenic immune dysregulation disease models, such as LRBA gene deficiency , may offer molecular biological evidence for the TCM concept of “congenital endowment deficiency” in this syndrome [27].

2.5.2. The necessity of multicenter clinical research

Current research on Lung-Heat and Kidney-Deficiency Syndrome is limited by small sample sizes and inconsistent diagnostic criteria. A cohort study of Iranian patients with DCLRE1C gene deficiency demonstrated the need for multicenter collaboration to accumulate data on rare diseases. The following recommendations are proposed, where establishing unified TCM-Western medicine diagnostic criteria such as combining tongue and pulse diagnosis with ADAMTS-13 testing and designing prospective registry studies; analyzing clinical characteristics and prognostic differences across age groups (children/adults/elderly) by referencing age-specific management strategies for thrombotic thrombocytopenic purpura (TTP); leveraging real-world data including electronic health record databases to uncover patterns of syndrome evolution [28]. Furthermore, international collaboration could validate the immunomodulatory effects of TCM herbal formulas and advance mechanistic research through shared biobanks.

3. Conclusion

The disease outcomes of immune thrombocytopenic purpura (ITP) in female patients are influenced by multiple factors, including hormonal levels, immune regulation, and the characteristics of Traditional Chinese Medicine (TCM) constitutional types. Among these, the syndrome of lung-heat and kidney deficiency is closely associated with the onset and progression of ITP. From an expert perspective, integrated traditional Chinese and Western medicine (ITCWM) demonstrates unique advantages in improving the prognosis of female patients with ITP, with mechanisms potentially involving multi-target effects such as immune regulation, inflammation suppression, and platelet production promotion. However, current research still has certain limitations, including small sample sizes, insufficient follow-up durations, and a lack of standardized evaluation systems, leading to variations in conclusions across different studies.

Future research should focus on the following aspects. Firstly, delve into the molecular mechanisms of the lung-heat and kidney deficiency syndrome to clarify its specific associations with the onset and progression of ITP, providing a theoretical basis for precision medicine. Secondly, optimize individualized treatment plans by combining targeted therapies from modern medicine with syndrome differentiation and treatment from traditional Chinese medicine to enhance efficacy and reduce adverse reactions. Finally, conduct multi-center, large-sample clinical studies using unified evaluation criteria to verify the long-term efficacy and safety of ITCWM.

Overall, the treatment strategy for ITP should balance the precision of modern medicine with the holistic perspective of traditional Chinese medicine, offering more effective personalized management plans for female patients through multidisciplinary collaboration. Future research should emphasize the integration of basic and clinical studies to drive innovation in ITP diagnosis and treatment models, ultimately improving patients' quality of life and long-term prognosis.

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