

Research Advances in the Integrated Treatment of Purpura Using Traditional Chinese and Western Medicine

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Abstract:

Purpura is a common immune-related disease in children, primarily characterized by purplish-red skin rashes that do not blanch upon pressure. It may be accompanied by other complications such as joint pain, gastrointestinal bleeding, and purpura nephritis. The exact cause of purpura remains unclear. Clinically, it is classified into allergic purpura and thrombocytopenic purpura based on symptoms and laboratory findings. Treatment primarily involves symptomatic management, adjusted according to the severity of the child's condition and accompanying symptoms. This article systematically reviews both Western and Traditional Chinese Medicine approaches to treating purpura, combining their respective advantages and disadvantages to provide a reference framework for the clinical management of childhood purpura.

Keywords:

Purpura, Immune Thrombocy-topenic Purpura, Henoch-Schönlein Purpura, Traditional Chinese Medicine, Treatment

1. Introduction

Purpura, classified as a blood disorder in traditional medicine and also known as ecchymosis, is primarily characterized by the leakage of blood beneath the skin and mucous membranes, resulting in petechiae and ecchymoses that do not blanch upon pressure. This condition is similar to the diseases described in ancient medical texts as "muscle hemorrhage," "purple toad," and "grape plague." Modern medicine categorizes purpura into immune thrombocy-topenic purpura (ITP) and Henoch-Schönlein purpura (HSP).

ITP, also known as immune thrombocytopenia, is an autoimmune disorder caused by increased platelet destruction and insufficient production due to immune intolerance. ITP accounts for 25% to 30% of pediatric bleeding disorders. Most cases present with mild hemorrhagic symptoms, including ecchymoses and petechiae,

occasionally accompanied by mucosal bleeding. These symptoms may resolve spontaneously as platelet counts improve [1,2].

HSP, also known as IgA vasculitis, is a vasculitic disorder caused by immune complex deposition in small blood vessels (including glomeruli), leading to leukocyte rupture. It is characterized by cutaneous purpura and commonly involves the abdomen, joints, and kidneys, resulting in joint swelling and pain, abdominal pain, and hematologic disorders. Among pediatric HSP patients, renal involvement is predominant, with a nephritis incidence rate reaching 20%-60% [3,4].

2. Diagnosis

The diagnosis of ITP involves ruling out all known causes of secondary ITP and meeting the following criteria: bleeding; at least two platelet counts $<100 \times 10^9/L$ with normal hematopoietic morphology; generally unenlarged spleen; bone marrow examination showing normal or increased megakaryocytes with maturation defects; exclusion of other secondary thrombocytopenias. Most children experience spontaneous remission within weeks to months, but the condition may occasionally progress to chronic ITP, defined as persistent thrombocytopenia lasting over 12 months [1,5].

Currently, there are no specific diagnostic serological laboratory tests or biomarkers for HSP. Diagnosis is primarily based on its clinical presentation, along with meeting at least one of the following criteria: arthritis or arthralgia, acute abdominal pain attacks, hematuria or proteinuria, and histopathological evidence of leukocytoclastic vasculitis with IgA deposition [6,7].

3. Etiology

3.1. Thrombocytopenic purpura

Several studies [8,9] indicate that ITP is primarily associated with antiplatelet antibodies in the body. Immune cells mistakenly identify platelets as pathogens, leading to the production of autoantibodies. When these autoantibodies bind to platelet antigens, the reticuloendothelial system (primarily the spleen) removes platelets, leading to their premature destruction. Since platelets play a crucial role in blood clotting, children with ITP exhibit peripheral blood platelet counts below normal levels. Consequently, their bleeding time progressively lengthens, significantly increasing the risk of hemorrhage.

3.2. Henoch-Schönlein purpura

HSP exhibits a higher incidence among children under 10 years of age, with a peak onset between 4 and 7 years old [10]. It is largely self-limiting and shows an epidemic tendency during spring and autumn. The typical histopathological features of HSP include endothelial cell damage and leukocyte infiltration within the vascular wall, though its etiology and pathogenesis remain incompletely understood. Song Y et al. [11] indicated that medications and vaccines can trigger HSP. Similarly, Nikolaishvili M et al. [12] reported that genetic predisposition, environmental factors, and infectious agents are closely associated with HSP onset. Elevated serum IgA levels and the release of pro-inflammatory cytokines during the acute phase suggest that infection is also a significant cause of HSP, with respiratory infections being the most common, followed by gastrointestinal infections, cellulitis, and urinary tract infections.

Infectious agents encompass both bacterial and viral pathogens, including *Streptococcus*, *Helicobacter pylori*, *Mycoplasma pneumoniae*, human parvovirus B19, and hepatitis A virus. These pathogens can cause extensive damage to the vascular endothelium, ultimately leading to systemic vascular inflammation and permeability [13,14].

3.3. Traditional Chinese Medicine's Understanding of Purpura

Traditional Chinese medicine holds that both HSP and ITP are associated with “heat, stasis, and deficiency,” with heat being the primary cause. Heat originates from multiple sources, including both external and internal factors. External factors primarily involve exogenous cold invasion and irregular dietary habits, while internal factors include yin deficiency and qi deficiency [15,16]. Pediatric organs are delicate and underdeveloped, making children susceptible to external pathogens. Once invading pathogens enter the body, they transform into heat and fire within the child's constitution. This internal heat and fire force blood to flow erratically, scorching blood vessels and causing blood leakage beyond the vessels—resulting in purpura. Simultaneously, fire pathogens deplete qi and can also boil the blood, impairing qi's ability to propel blood circulation and leading to blood stasis. Children often exhibit spleen deficiency, and since the spleen is the foundation of acquired constitution and the source of qi and blood generation, recurrent infections like colds, coughs, and fevers can impair spleen function in the middle and late stages, diminishing appetite and disrupting the generation of vital substances. Children often exhibit signs of qi deficiency, such as fatigue, poor appetite, and a sallow complexion. When spleen qi is deficient and unable to retain blood, it may lead to blood deviating from its channels, overflowing beyond the vessels, and manifesting as hemorrhagic disorders like epistaxis, hematochezia, or skin ecchymoses and petechiae.

4. Treatment

4.1. Treatment of ITP

Children with ITP exhibit a certain degree of self-limiting nature. Recent British research on treatment developments for pediatric ITP patients categorizes bleeding severity as follows: Mild: Minor ecchymoses and small bruises (<5 cm), epistaxis (bleeding stops with pressure application); Moderate: Numerous ecchymoses and large bruises (>5 cm), epistaxis lasting over 20 minutes, intermittent bleeding from gums, lips, buccal mucosa, oropharynx, and gastrointestinal tract without hypotension or blood pressure drop; Severe: Severe epistaxis (requiring nasal packing or cauterization for hemostasis), persistent bleeding from gums, buccal mucosa, and oropharynx, suspected internal hemorrhage (lung, muscle, joint, or other sites) leading to hypotension and decreased hemoglobin; life-threatening hemorrhage or intracranial hemorrhage requiring fluid resuscitation and blood transfusion therapy (>10 mL/kg) [17]. Children with mild bleeding that does not impact daily life should be primarily observed; if it does impact daily life, glucocorticoids are the preferred treatment. Children with moderate bleeding volume increase accompanied by mucosal damage may receive intravenous immunoglobulin therapy. First-line treatment for ITP children involves corticosteroids and intravenous immunoglobulin (IVIG). IVIG primarily acts by saturating Fc receptors on splenic macrophages, thereby inhibiting Fc-mediated antibody-mediated phagocytosis of platelets. Corticosteroids reduce proinflammatory cytokines and enhance T-regulatory cell activity, contributing to

overall immunosuppression and achieving rapid platelet count elevation [18]. For children with no significant symptom improvement or persistent thrombocytopenia, second-line therapies may be considered. These include thrombopoiesis-stimulating agents, immunosuppressants (azathioprine, hydroxychloroquine, mycophenolate mofetil, and rituximab), and splenectomy. Some children exhibit poor tolerance to thrombopoiesis-stimulating agents, reducing clinical efficacy. In such cases, rituximab may be selected as monotherapy, with thrombopoiesis-stimulating agents reserved for combination therapy when other agents prove ineffective [1,2,19].

4.2. Treatment of HSP

Several medications are used clinically to treat HSP, but no specific cure has been identified. Treatment primarily follows principles of anti-allergy management, infection control, and allergen avoidance to select appropriate therapeutic strategies [20]. For children with mild HSP, first-line treatment involves symptomatic management focused on pain relief, fluid replacement, and rest. Some children develop severe, life-threatening, or debilitating complications such as acute gastrointestinal bleeding or nephropathy. In such cases, treatment primarily involves glucocorticoids, often combined with anticoagulants (dipyridamole, heparin, etc.) and vasculoprotective agents (rutin tablets, vitamin C, etc.) based on the severity of the condition. Some severely ill children require adjunctive immunosuppressants, intravenous immunoglobulin, or combined treatment with *Tripterygium wilfordii* preparations [21]. Allergic purpura nephritis is a common complication of HSP. If the child presents primarily with hematuria and proteinuria, it is typically mild, presenting only as pathological urine. First-line treatment involves oral glucocorticoids. For children with moderate HSP nephritis, intravenous glucocorticoids may be administered. Children with severe HSP nephritis should receive initial treatment combining glucocorticoids with intravenous cyclophosphamide [22].

The primary medications commonly used in Western medicine include corticosteroids, immunosuppressants, and antihistamines. Hormone therapy for HSP primarily reduces immunoglobulin synthesis, lowering immune function to decrease IgA production and prevent its deposition on capillary walls, thereby alleviating inflammatory responses. Long-term use is not recommended due to recurrence risks and potential developmental effects in children. Immunosuppressants mitigate clinical manifestations by inhibiting excessive immune responses, with common agents including cyclophosphamide and mycophenolate mofetil. Antihistamines block histamine receptors to alleviate allergy-induced symptoms like skin purpura and joint swelling/pain. Commonly used drugs include cetirizine and loratadine. As noted in Chen Sihan et al.'s article [23], cimetidine injection can inhibit skin and mucosal hemorrhage, reduce vascular permeability, prevent persistent gastric mucosal damage, alleviate intra-abdominal bleeding and edema, protect skin and mucosa, and reduce renal injury in children with allergic purpura. Additionally, gamma globulin, as an immunomodulator, can be used in purpura treatment. Rich in immunoglobulins, it not only regulates the concentration of immune factors within the body but also blocks immune cascade reactions caused by pathogens such as bacteria and viruses. By adjusting the ratio of antibodies to antigens within the patient's body, gamma globulin alters the volume of immune complexes, reduces IgA concentration, protects platelets, and inhibits the formation of platelet antibodies [24].

4.3. Traditional Chinese Medicine Treatment for Purpura

Traditional Chinese medicine attributes the onset of purpura primarily to heat, stasis, and deficiency. “Heat” is the most common pathogenic factor in the early stages of the disease, as it readily agitates blood and causes hemorrhage. Therefore, treatment focuses on clearing heat and cooling the blood. Given that “heat” has an upward-moving nature, it is necessary to combine this with herbs that draw heat downward. Pediatric formulations should be simplified, favoring herbs with broad therapeutic coverage such as *Achyranthes* root, *Rehmannia* root, and *Salvia* root. “Stasis” is a persistent factor throughout the disease process, serving both as a cause and a pathological product. Thus, herbs that activate blood circulation and resolve stasis must be consistently incorporated into treatment. In the later stages of the disease or when the child's constitution is weak, deficiency patterns predominate. Traditional Chinese Medicine holds that the kidneys represent the root of innate constitution, while the spleen represents the root of acquired constitution. Thus, treating deficiency often begins with the spleen and kidneys, selecting herbs that fortify the spleen and nourish the kidneys to support therapy. Additionally, purpura falls under the category of blood disorders. Treatment can incorporate Professor Tang Rongchuan's Four Methods for Blood Disorders (hemostasis, stasis resolution, blood stabilization, and deficiency tonification) into formula design, with flexible modifications based on clinical manifestations.

In the article by Zhang Shaoqing et al. [25], Professor Zhang Jun proposed that “hidden toxins” are closely associated with the onset of purpura. Therapeutic principles involve constitutional regulation, clearing the lungs, tonifying the spleen, and nourishing the kidneys, alongside activating collaterals and resolving stasis. The Six Flavor *Rehmannia* Decoction serves as the foundation for constitutional adjustment, augmented with herbs to activate collaterals and resolve stasis. This approach combines pungent herbs and vine-like herbs, supplemented with agents to clear the lungs, tonify the spleen, nourish the kidneys, clear heat, and resolve toxins for treating purpura nephritis. Professor Zhang Shiqing [26] posited that heat-stasis damaging collaterals and blood overflowing outside vessels constitute the core pathogenesis of pediatric HSP. Thus, treatment primarily involves clearing heat, cooling blood, resolving stasis, and unblocking collaterals. He created the “Four Herbs for Eliminating Spots Decoction” by modifying the Rhino Horn and *Rehmannia* Decoction to treat HSP. Professor Chen Guangming [27] advocates distinguishing between internal and external factors, deficiency and excess, and the severity of the condition when treating purpura. The primary approach involves consistently cooling the blood while also dispelling wind and promoting blood circulation to resolve stasis. For prolonged cases, dampness-eliminating herbs should be incorporated to protect the child's spleen and stomach, with emphasis on dietary regulation. Additionally, herbal fumigation as an external treatment method can effectively reduce the number of skin purpura lesions with a low incidence of adverse reactions[28].

Based on varying clinical manifestations, Traditional Chinese Medicine categorizes purpura into four major syndromes: Blood Heat with Uncontrolled Circulation, Yin Deficiency with Excessive Fire, Qi Deficiency with Blood Stasis, and Spleen-Kidney Yang Deficiency. Blood Heat with Uncontrolled Circulation Type: Primarily manifests as bright red spots, potentially accompanied by epistaxis, gingival bleeding, or hematuria. Clinical features include a red tongue with yellow coating and a rapid pulse. Treatment principles involve clearing heat, cooling blood, detoxifying, and resolving stasis. Yin Deficiency with Excessive Heat Pattern: Characterized by

recurrent dark red purpura, accompanied by irritability, night sweats, dry mouth and throat, and a red tongue with thin coating. Treatment focuses on nourishing yin, clearing heat, stopping bleeding, and consolidating yang. Qi Deficiency with Blood Stasis Pattern: Manifested by pale complexion, diffuse pale spots over the body, accompanied by fatigue, pallor, decreased appetite, red tongue with white coating. Spleen-Kidney Yang Deficiency Pattern: Typical clinical presentation includes recurrent purpura, prolonged course with persistent symptoms, pale complexion with ecchymoses, aversion to cold, soreness in waist and knees, swollen tongue with teeth marks, slow pulse.

Additionally, external therapies in traditional Chinese medicine play a significant role in treating purpura, including acupuncture, herbal fumigation and washing, and herbal poultice application. Acupuncture helps unblock meridians, relieve pain, and regulate qi and blood flow, making it an effective adjunct therapy for various conditions. When treating purpura, primary acupoints such as blood sea (Xuehai), three yin junction (Sanyinjiao), diaphragm shu (Geshu), and foot three miles (Zusanli) are selected. Additional auxiliary points with specific functions are then chosen based on the patient's individual subtype. Herbal steam bath therapy primarily works through skin absorption and steam penetration to unblock meridians, promote blood circulation, and relieve pain. It is also one of the commonly used external treatments. Chinese herbal plaster application: This involves formulating specific medicinal ingredients into a paste and applying it to designated acupoints to achieve effects such as pain relief and promoting blood circulation to remove stasis. It offers the advantage of being simple to administer and is more readily accepted by pediatric patients. This approach has also been validated in articles by Fan Xiaojiao et al.[29].

5. Discussion

After a child falls ill, the disease progresses rapidly. Differences in constitutional constitution can lead to varying symptoms for the same illness, and conditions prone to deficiency or excess, or a mixture of deficiency and excess, are common. Therefore, we must integrate the physiological characteristics of children with their constitutional constitution, analyze the symptom patterns of patients at different stages of the disease through pattern differentiation, and thereby select an appropriate treatment plan. Western medicine currently lacks specific drugs for treating purpura. Treatment primarily involves corticosteroids, immunosuppressants, or symptomatic supportive care. While these approaches yield rapid results, the disease course is prolonged, recurrence rates are high, and complications are common. Furthermore, purpura most frequently occurs during childhood school years, and corticosteroid use may adversely affect subsequent growth and development. Traditional Chinese medicine treats purpura based on syndrome differentiation and classification. The general therapeutic principles involve clearing heat and cooling the blood, as well as strengthening the spleen and boosting qi, adjust dosage according to symptoms; onset of action is relatively slow. The author does not recommend its use during the acute or critical phases of the disease, but it demonstrates significant therapeutic efficacy in other stages. During clinical management, a combination of Chinese and Western medicine can be employed. This approach not only effectively addresses the limitations of each modality but also maximizes relief for the child, reduces the incidence of sequelae, and alleviates the burden on the family and financial resources. In summary, we must dynamically monitor the child's condition and adjust treatment promptly.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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