



**SCARLET FEVER IN YOUNG CHILDREN AND ITS DISTINCTIVE CLINICAL
COURSE**

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Abstract : Scarlet fever is an acute infectious disease caused by Group A β -hemolytic streptococcus (*Streptococcus pyogenes*), predominantly affecting children. This scientific article provides a comprehensive analysis of the epidemiology, etiology, pathogenesis, immune and inflammatory mechanisms, morphological and histopathological changes, clinical manifestations, diagnostic approaches, treatment strategies, and preventive measures of scarlet fever in young children. Particular attention is paid to age-related characteristics of the disease course, which are associated with the immaturity of the immune system. The importance of early diagnosis and timely antibiotic therapy in preventing severe complications is emphasized based on current scientific literature.

Keywords: scarlet fever, *Streptococcus pyogenes*, young children, erythrogenic toxin, superantigen, immune response, antibiotic therapy.

Significance of the Topic

Scarlet fever remains a significant infectious disease in pediatric practice, especially among children aged 3–10 years. Rapid transmission in child communities, acute intoxication, and the risk of severe late complications such as rheumatic fever, myocarditis, and post-streptococcal glomerulonephritis highlight the relevance of this condition. In young children, functional immaturity of the immune system often contributes to a more severe clinical course.

Epidemiology and Etiology

Scarlet fever occurs more frequently during the autumn–winter season. The source of infection is an infected child or an asymptomatic carrier of *Streptococcus pyogenes*. Transmission mainly occurs via airborne droplets, though household contact transmission is also possible. The etiological agent is toxin-producing strains of Group A β -hemolytic streptococci capable of producing erythrogenic exotoxins.

Main Body

The pathogenesis of scarlet fever involves a complex interaction between infectious agents and immunological responses. After entering the body, streptococci colonize the pharyngeal mucosa and release toxins that induce both local inflammation and systemic intoxication.

Pathogenesis and Immune Mechanisms

Erythrogenic exotoxins play a central role in the pathogenesis of scarlet fever. These toxins function as superantigens, causing massive, non-specific activation of T lymphocytes without antigen presentation. This leads to excessive release of cytokines such as interleukin-1, interleukin-6, tumor necrosis factor- α (TNF- α), and interferon-gamma. The resulting cytokine storm contributes to fever, systemic inflammation, and intoxication. In young children, insufficient immune regulation increases the risk of severe disease manifestations.

Inflammatory Reactions Inflammatory processes predominantly affect the pharynx, tonsils, and skin. Vasodilation and increased capillary permeability result in the characteristic fine, punctate rash. The pharynx typically shows catarrhal or necrotic inflammation.

Morphological and Histopathological Changes

Morphological examination reveals hyperemia, edema, and fibrinous or purulent exudates on the tonsils. Initially, the tongue is coated with a white film, which later desquamates, revealing a bright red surface known as the “strawberry tongue.” Histopathological findings include



lymphoid hyperplasia, perivascular inflammatory infiltration, and increased vascular permeability.

Clinical Course

Scarlet fever usually has an acute onset, with body temperature rising to 38–40 °C, accompanied by sore throat, headache, vomiting, and general weakness. The rash typically appears within 1–2 days, starting on the neck, chest, and flexural surfaces. In young children, irritability, decreased appetite, and sleep disturbances are common.

Clinical Forms of Scarlet Fever

The disease may present in mild, moderate, or severe (toxic or septic) forms. Severe cases are characterized by pronounced intoxication, cardiovascular disturbances, and neurological symptoms, requiring urgent medical intervention.

Differential Diagnosis

Scarlet fever should be differentiated from measles, rubella, enteroviral infections, allergic dermatitis, and drug-induced rashes. The presence of a strawberry tongue, acute tonsillitis, and a fine punctate rash are characteristic diagnostic features.

Diagnosis

Diagnosis is based on clinical findings supported by laboratory investigations. Throat swab cultures identifying *Streptococcus pyogenes*, leukocytosis and elevated erythrocyte sedimentation rate (ESR) in blood tests, and increased antistreptolysin O (ASO) titers are diagnostically significant. Rapid antigen detection tests for streptococcus are widely used in modern clinical practice.

Treatment and Prevention

The cornerstone of treatment is antibiotic therapy, primarily penicillin-based antibiotics administered for 10 days. In cases of penicillin allergy, macrolides are recommended. Symptomatic therapy includes antipyretics, detoxification measures, and local antiseptic treatment. Preventive strategies involve early detection, isolation of infected individuals, and strict adherence to hygiene measures in child-care institutions.

Complications and Prognosis

Without timely treatment, scarlet fever may lead to complications such as rheumatic fever, myocarditis, otitis media, sinusitis, and post-streptococcal glomerulonephritis. With early diagnosis and appropriate therapy, the prognosis is generally favorable.

Conclusion

Scarlet fever in young children results from a complex interplay of immune and inflammatory mechanisms. Early diagnosis, rational antibiotic therapy, and effective preventive measures significantly reduce the risk of severe disease and long-term complications.

References

- Nelson W.E. Nelson Textbook of Pediatrics. 21st ed. Elsevier; 2020.
- Murray P.R., Rosenthal K.S. Medical Microbiology. 9th ed. Elsevier; 2020.
- World Health Organization. Scarlet fever: clinical guidelines.
- Centers for Disease Control and Prevention (CDC). Group A Streptococcal Infections.