ISSN 2181-

EARLY DIAGNOSIS AND PROGNOSIS OF HEMORRHAGIC VASCULITIS USING MODERN LABORATORY BIOMARKERS

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ANNOTATION. Hemorrhagic vasculitis (Henoch–Schönlein purpura, IgA vasculitis) is a common immune-inflammatory disease in children, affecting the skin, joints, gastrointestinal tract, and kidneys. The clinical heterogeneity and severity of the disease determine the prognosis and treatment strategy. Traditional laboratory tests-urinalysis, C-reactive protein, ESR assist in assessing disease activity, but their ability to detect severe forms early is limited. Modern laboratory biomarkers play a key role in early diagnosis, identification of severe forms, and prediction of renal complications. A comprehensive clinical and laboratory approach enables timely diagnosis of hemorrhagic vasculitis in children, prevents the progression of severe forms, and reduces the risk of long-term complications.

Keywords: hemorrhagic vasculitis, laboratory biomarkers, endothelial dysfunction, inflammation**.**

Introduction. According to the World Health Organization (WHO), hemorrhagic vasculitis represents the most prevalent form of systemic vasculitis in the pediatric population, affecting approximately 10-20 children per 10,000 annually. This

ISSN 2181-

immune-mediated inflammatory disorder manifests in multiple clinical forms, with disease progression and prognosis largely dependent on the extent of renal involvement. The wide spectrum of clinical presentations makes early diagnosis a significant clinical challenge, highlighting the importance of evaluating modern diagnostic biomarkers.

As a systemic microvascular vasculitis, hemorrhagic vasculitis impacts the skin, joints, gastrointestinal tract, and kidneys. Its clinical course is heterogeneous, and both severity and long-term outcomes are primarily determined by renal involvement. Recent years have seen an increase in incidence in pediatric practice, most frequently affecting children between 4 and 11 years of age, with a higher prevalence among boys. Viral infections, environmental exposures, and genetic predisposition of the immune system contribute significantly to disease development. The underlying pathogenesis involves deposition of IgA1-containing immune complexes in the vascular endothelium, complement system activation, and subsequent endothelial dysfunction.

Hemorrhagic vasculitis in children can manifest in multiple clinical forms. The mildest and most frequently observed is the cutaneous form, characterized by symmetrically distributed palpable purpura. Skin lesions are typically the earliest and most persistent clinical sign, appearing in nearly all affected children. In this form, the general health status is usually satisfactory, and standard laboratory tests often remain within normal limits.

The cutaneous-articular form, present in a majority of pediatric patients, is distinguished by purpura accompanied by joint pain, swelling, and restricted mobility. This form is generally reversible and does not result in permanent joint deformities, though it requires careful differential diagnosis from certain rheumatologic conditions.

ISSN 2181-

The cutaneous-abdominal form is among the most variable and diagnostically challenging presentations. It involves hemorrhagic inflammation of the intestinal wall, leading to severe abdominal pain, vomiting, diarrhea, and signs of gastrointestinal bleeding. In some cases, this presentation can mimic an “acute abdomen,” posing a risk of misdiagnosis and potential unnecessary surgical intervention.

The cutaneous-renal form represents the most severe and prognostically critical variant of hemorrhagic vasculitis. It is characterized by the onset of glomerulonephritis, with urinalysis often demonstrating hematuria, proteinuria, and cylindruria. Renal involvement typically develops several weeks after disease onset and may remain clinically silent, which can result in delayed diagnosis. This form carries a high risk of progression to chronic kidney disease.

In the generalized form, cutaneous, articular, abdominal, and renal manifestations occur concurrently. Occasionally, the vasculature of the cardiovascular, pulmonary, or central nervous system may also be affected. Laboratory investigations complement clinical assessment in diagnosing hemorrhagic vasculitis. Common inflammatory markers, including C-reactive protein and erythrocyte sedimentation rate (ESR), are typically elevated during active disease. Although increased levels of these markers indicate systemic inflammation, they are not specific to hemorrhagic vasculitis.

Immunological assessments commonly demonstrate elevated IgA levels, which constitute one of the most distinctive laboratory findings in hemorrhagic vasculitis. In particular, increases in IgA1 fractions and the formation of IgA-C3 immune complexes are closely linked to renal involvement. Alterations in complement components, including C3 and C4, also provide valuable information for evaluating disease activity.

Urinalysis remains the primary tool for early detection of the nephritic form. The presence of proteinuria and hematuria reflects the initiation of immune-

mediated inflammatory processes in the kidneys and underscores the need for long-term patient monitoring.

In recent years, modern biomarkers have received considerable attention for their role in the early diagnosis and prognosis of severe forms of hemorrhagic vasculitis. Inflammatory cytokines such as IL-6, IL-8, and TNF-α serve as sensitive indicators of disease activity, with elevated levels correlating with more severe clinical courses, particularly in patients developing abdominal or nephritic manifestations.

Markers of endothelial dysfunction, including VCAM-1, ICAM-1, and E-selectin, serve as indicators of vascular endothelial injury. Elevated levels of these markers are associated with purpura activity, microangiopathy, and the generalized forms of hemorrhagic vasculitis. The von Willebrand factor is a critical biomarker for assessing both endothelial damage and hemostatic disturbances. Oxidative stress markers, particularly malondialdehyde (MDA), reflect the severity of the disease; in nephritic forms, MDA levels are markedly increased and are accompanied by diminished activity of antioxidant defense enzymes.

Additionally, modern renal biomarkers such as NGAL, KIM-1, and cystatin C demonstrate high sensitivity for the early detection of kidney injury. These markers often become elevated prior to the onset of clinical symptoms or standard laboratory abnormalities, allowing for timely recognition of nephritic complications and enabling appropriate adjustments to therapeutic strategies.

In conclusion, hemorrhagic vasculitis in children represents a clinically heterogeneous condition with a substantial risk of complications. Timely diagnosis and effective patient monitoring necessitate not only thorough evaluation of clinical signs but also the integration of laboratory assessments and modern biomarkers. These biomarkers facilitate the assessment of disease activity, enable early detection of severe clinical forms, and allow for the prediction of renal involvement. A comprehensive approach that combines clinical evaluation with laboratory and

biomarker analyses enhances treatment efficacy and helps reduce the likelihood of long-term complications in pediatric patients with hemorrhagic vasculitis.

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