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IMMUNE-HEMATOLOGICAL FEATURES OF NEPHROPATHY COMPLICATIONS IN PATIENTS WITH HEMORRHAGIC VASCULITIS

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Abstract: Hemorrhagic vasculitis (Henoch-Schönlein purpura) is the most common manifestation of inflammation of the small vessels and may result in severe complications if nephropathy develops. The aim of this study is to investigate the correlation between immune-hematological markers and the development of nephropathy. Literature review demonstrates that IgA immunoglobulins, the complement factors, cytokine levels, and alterations in platelet function are determinants of nephropathy development. Results indicate that deposition of IgA deposits in glomeruli, decreased C3 and C4 complement levels, increased levels of IL-6 and TNF-α cytokines, and platelet function disorders correlate directly with nephropathy development.

Keywords: hemorrhagic vasculitis, nephropathy, immunoglobulin A, complement system, cytokines, platelet activity

GEMORRAGIK VASKULITLI BEMORLARNI NEFROPATIYA BILAN ASORATLANISHINING IMMUN-GEMATOLOGIK XUSUSIYATLARI

Annotatsiya. Gemorragik vaskulit (Henoch-Schönlein purpurasi) kichik qon tomirlari yallig'lanishining eng keng tarqalgan shakli bo'lib, nefropatiya rivojlanishi bilan og'ir asoratlanishi mumkin. Ushbu tadqiqot immun-gematologik ko'rsatkichlar va nefropatiya rivojlanishi o'rtasidagi bog'liqlikni tahlil qilishga qaratilgan. Adabiyotlar tahlili shuni ko'rsatdiki, IgA immunoglobulinlari, komplement tizimi komponentlari, sitokin profillar va trombosit faolligidagi o'zgarishlar nefropatiya rivojlanishida muhim rol o'ynaydi. Natijalarda IgA depozitlarining glomerullarda to'planishi, C3 va C4 komplementlar darajasining pasayishi, IL-6 va TNF-α sitokillari kontsentratsiyasining ortishi hamda trombositlarning funktsional holatidagi buzilishlar nefropatiya rivojlanishi bilan bevosita bog'liqligi aniqlandi.

Kalit so'zlar: gemorragik vaskulit, nefropatiya, immunoglobulin A, komplement tizimi, sitokinlar, trombosit faolligi

ИММУННО-ГЕМАТОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ОСЛОЖНЕНИЙ НЕФРОПАТИИ У БОЛЬНЫХ ГЕМОРРАГИЧЕСКИМ ВАСКУЛИТОМ

Аннотация. Геморрагический васкулит (пурпура Шенлейна-Геноха) представляет наиболее распространенную форму воспаления мелких сосудов и может привести к

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тяжелым осложнениям с развитием нефропатии. Данное исследование сосредоточено на анализе взаимосвязи между иммуно-гематологическими параметрами и прогрессированием нефропатии. Анализ литературы демонстрирует, что иммуноглобулины IgA, компоненты системы комплемента, цитокиновые профили и изменения активности тромбоцитов играют ключевые роли в развитии нефропатии. Результаты указывают на то, что накопление депозитов IgA в клубочках, снижение уровней комплементов C3 и C4, повышение концентраций цитокинов IL-6 и TNF-α, а также функциональные нарушения тромбоцитов непосредственно связаны с прогрессированием нефропатии.

Ключевые слова: геморрагический васкулит, нефропатия, иммуноглобулин А, система комплемента, цитокины, активность тромбоцитов

INTRODUCTION

Hemorrhagic vasculitis (HV) or Henoch-Schönlein purpura is an immune complex-dependent small-vessel vasculitis, the most frequent systemic vasculitis of children and adolescents [1]. Cutaneous purpura, arthralgia, gastrointestinal syndromes, and renal disease are its principal manifestations. Nephropathy development is the most severe complication of HV, occurring in 20-60% of patients and affecting long-term outcome [2]. Studies conducted within the Republic of Uzbekistan confirm renal involvement in 35% of HV children, with this symptom being based on regional characteristics [3].

Current studies confirm that nephropathy development in HV is a result of complex interactions between various immune-hematological factors. Defective IgA production, complement system activation, release of inflammatory cytokines, and platelet dysfunction are the key pathological features that collectively result in glomerular damage and progressive renal dysfunction. Russian investigators' large-scale research indicates that IgA nephropathy development mechanisms are multifactorial in nature and are due to both genetic and environmental factors [4]. Understanding these mechanisms is critical for the development of directed therapeutic approaches and improved patient outcomes.

Early diagnosis of immune-hematological predictors of nephropathy development cannot be overvalued, as it can avoid damage to the kidneys that is not reversible and the progression of chronic kidney disease. Longitudinal observational research in the Russian Federation indicates that 85% of patients detected and appropriately treated in early stages have favorable long-term prognosis. Central Asian regional population studies determine specific genetic polymorphisms affecting disease susceptibility and disease progression patterns, for which population-related research approaches are necessary.

METHODOLOGY AND LITERATURE ANALYSIS

This comprehensive literature review was conducted through systematic analysis of peer-reviewed publications. The analytical framework employed in this review encompasses several key methodological approaches. First, systematic evaluation of immunoglobulin profiles, particularly IgA subclasses and their glomerular deposition patterns, was examined across

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multiple studies to establish consistent patterns associated with nephropathy development. Second, complement system analysis focused on classical and alternative pathway components, their activation patterns, and correlation with renal involvement severity. Third, cytokine profile assessment included pro-inflammatory and anti-inflammatory mediators, their temporal variations, and relationship with disease progression. Fourth, hematological parameter evaluation encompassed platelet function, coagulation factors, and endothelial markers associated with microvascular damage.

Russian Federation research findings demonstrate that IgA immunoglobulins, specifically IgA1 subclass with aberrant galactosylation, represent the primary pathogenic factor in HV nephropathy [5]. These abnormal IgA molecules form immune complexes that deposit preferentially in glomerular mesangium, initiating inflammatory cascades leading to glomerular damage. Complement system activation, particularly through the alternative pathway, amplifies the inflammatory response and contributes to tissue injury. Studies consistently demonstrate that patients developing nephropathy exhibit significantly higher levels of circulating IgA-containing immune complexes compared to those without renal involvement.

Research conducted in Uzbek medical institutions reveals that clinical manifestations of HV and immune indicators among the local population correspond with international data, although certain genetic characteristics exist [6]. Regional studies indicate that a comprehensive approach is necessary for early identification of patients at high risk of nephropathy development in local conditions. These findings emphasize the importance of population-specific biomarker validation and risk stratification protocols.

RESULTS AND DISCUSSION

Analysis of available evidence reveals distinct immune-hematological patterns associated with nephropathy development in hemorrhagic vasculitis patients. The most consistent finding across multiple studies is the elevation of serum IgA levels, particularly the IgA1 subclass, in patients who subsequently develop renal complications [7]. These elevated IgA levels correlate directly with the severity of glomerular involvement and long-term renal outcomes. Immunohistochemical analysis of renal biopsies consistently demonstrates predominant IgA deposition in glomerular mesangium, often accompanied by C3 complement and fibrin deposits.

Comprehensive studies conducted in the Russian Federation demonstrate that complement system alterations represent another crucial aspect of the pathophysiological process. Patients with HV nephropathy demonstrate significantly reduced serum levels of C3 and C4 complement components, indicating active complement consumption during the inflammatory process [8]. The alternative complement pathway appears particularly important, with factor B and properdin levels showing inverse correlation with nephropathy severity. These findings suggest that complement activation contributes to glomerular damage through direct cytotoxic effects and enhancement of inflammatory cell recruitment.

Cytokine profile analysis reveals a predominantly pro-inflammatory pattern in patients developing nephropathy. Research conducted in Russian pediatric centers demonstrates elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β) consistently observed in patients with renal involvement compared to those with isolated

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cutaneous manifestations [9]. These cytokines promote endothelial activation, increase vascular permeability, and facilitate immune complex deposition in glomerular capillaries. Conversely, anti-inflammatory mediators such as interleukin-10 (IL-10) and transforming growth factor-beta (TGF-β) show variable patterns, with some studies suggesting inadequate compensatory responses in patients developing severe nephropathy.

Hematological parameters demonstrate significant alterations in HV patients with nephropathy. Studies from Uzbek medical centers reveal platelet function abnormalities, including increased platelet aggregation and enhanced release of pro-thrombotic mediators, contributing to microvascular thrombosis and glomerular damage [10]. Coagulation system activation, evidenced by elevated fibrinogen levels and shortened activated partial thromboplastin time, reflects the prothrombotic state characteristic of severe vasculitis. Endothelial dysfunction markers, including von Willebrand factor and soluble intercellular adhesion molecule-1, show significant elevation in nephropathy patients, indicating widespread microvascular involvement.

Therapeutic implications of these findings are substantial. The identification of specific immune-hematological patterns associated with nephropathy risk enables more targeted treatment approaches. Research from Central Asian medical institutions indicates that patients demonstrating high-risk profiles may benefit from earlier and more intensive immunosuppressive therapy, potentially preventing irreversible renal damage. Additionally, monitoring of specific biomarkers may guide treatment duration and intensity, optimizing therapeutic outcomes while minimizing adverse effects.

CONCLUSION

The comprehensive analysis of immune-hematological features in hemorrhagic vasculitis patients reveals distinct patterns associated with nephropathy development. Elevated IgA levels, particularly IgA1 with aberrant glycosylation, represent the primary pathogenic mechanism, while complement system activation and pro-inflammatory cytokine elevation contribute to glomerular damage progression. Hematological abnormalities, including platelet dysfunction and coagulation system activation, further exacerbate microvascular injury. These findings support the implementation of immune-hematological monitoring as an integral component of patient management, enabling early identification of nephropathy risk and guiding targeted therapeutic interventions. Future research should focus on developing standardized protocols for biomarker assessment and establishing evidence-based treatment algorithms based on individual risk profiles. The integration of immune-hematological parameters into clinical decision-making represents a promising approach for improving long-term outcomes in hemorrhagic vasculitis patients across diverse populations.

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