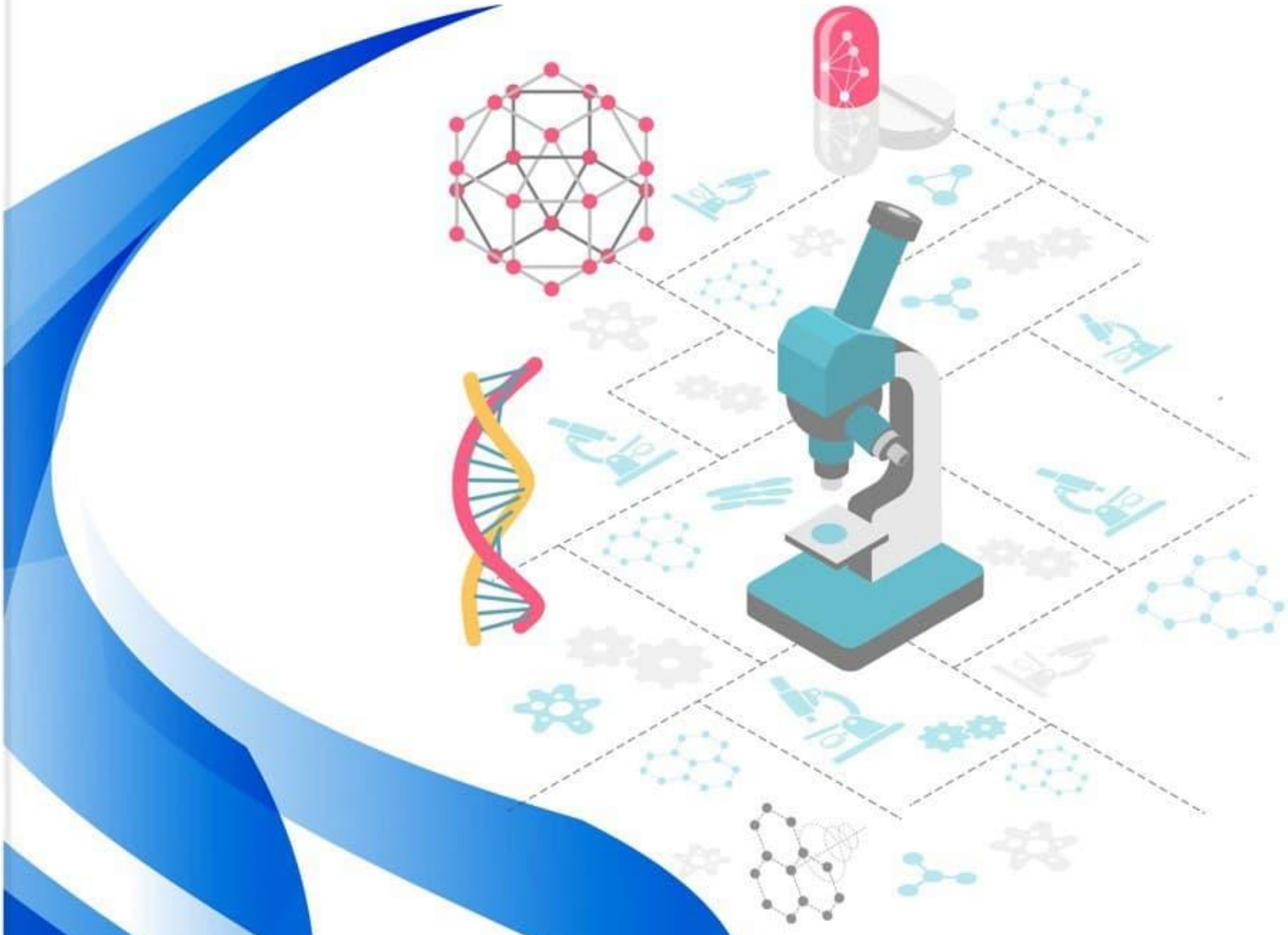
ISSN 2231-2218

ASIAN JOURNAL OF PHARMACEUTICAL AND BIOLOGICAL RESEARCH

AJPBR



Indexed by:

ROAD Crossref Universal Impact Factor IMPACT FACTOR SEARCH

Asian journal of Pharmaceutical and biological research 2231-2218
http://www.ajpbr.org/
Universal IMPACT factor 7
SJIF 2022: 4.465
Volume 13 Issue 2
MAY-AUG. 2024

Asian journal of Pharmaceutical and biological research 2231-2218
http://www.ajpbr.org/
Universal IMPACT factor 7
SJIF 2022: 4.465
Volume 13 Issue 2
MAY-AUG. 2024

THE CORRELATION BETWEEN CLINICAL LABORATORY INDICATORS AND KIDNEY STRUCTURAL CHANGES IN DIFFERENT TYPES OF CHRONIC GLOMERULONEPHRITIS WITH NEPHROTIC SYNDROME

Ismatova M.N.
Bukhara State Medical Institute, Bukhara, Uzbekistan

Annotation.A study of the specific severity of each nosological form of chronic glomerulonephritis that contributes to the development of nephrotic syndrome.The results of 104 biopsies from patients with neurotic syndrome were studied. The diagnosis was made based on the results of a comprehensive clinical and morphological examination of patients.

Keywords:chronic glomerulonephritis, clinical and laboratory examination, result.

Introduction. Glomerulonephritis (GN) is a group of kidney diseases with different etiologies and morphological patterns, which are based on immunoinflammatory damage mainly to the glomeruli. Depending on the morphological changes, GN is divided into 2 main groups - proliferative and non-proliferative GN. The group of proliferative GN includes: diffuse endocapillary GN, extracapillary GN with the formation of crescents, membranous proliferative GN (MPGN), mesangioproliferative GN (MezPGN). The group of non-proliferative GN in the English-language literature is positioned as nephropathies, which include minimal change nephropathy (MCNE), membranous nephropathy (MNP) and focal segmental glomerulosclerosis (FSGS). These morphological forms of GN can have various clinical manifestations, independent of either the etiology or the morphological features of glomerular damage. There are 4 variants of the clinical course of GN: nephrotic, hypertensive, mixed (triadic extended form of GN in the acute course of the disease) and latent (urinary variant, or GN with isolated urinary syndrome) [1]. The mechanisms of formation of individual GN syndromes - nephrotic, nephritic - have been sufficiently fully characterized, combinations of which in varying variations are observed in various clinical variants of GN. However, the factors that have a decisive influence on the formation of the clinical variant of GN have not been described. We paid attention to the information available in the literature about the association of nephrotic syndrome with hypofunction of the thyroid gland [2]. On the other hand, there is data on the influence of pro-inflammatory cytokines on indicators of thyroid status [3]. We hypothesized that the state of thyroid function may influence the development of not only nephrotic syndrome, but also other GN syndromes. In order to test this hypothesis, we

conducted a study of the thyroid status and cytokine profile in patients with various clinical variants of GN [4].

A comprehensive clinical and laboratory examination of 78 patients was carried out (table). Of these, 58 (74.4%) had a proteinuric form, 19 (24.3%) had a mixed form, and 1 (1.3%) had a hematuric form of chronic glomerulonephritis. The patients were predominantly under 35 years of age (average age 24.9+5.2 years). The number of men and women was approximately the same (53.4% and 46.6%, respectively). All patients with glomerulonephritis with nephrotic syndrome accompanied by severe swelling of the face, trunk, limbs up to the anatomy hypoproteinemia, dysproteinemia (hypoalbumin-nemia, hyperglobulinemia, hypergamma-2-globulinemia, hypergamma-lobulinemia), hypergammalipidemia, hypercholesterolemia, character-urinary sediment (hematuria, lymphocyturia, cylindruria), protein Nuria more than 3.5 g/day, hyperfibrinogenemia, decreased glomerular levels kovy filtration.

Results. We conducted a correlation analysis between the studied morphometric parameters and routine laboratory tests. It turned out that there are direct and inverse correlative connections between these indicators. Moreover, each form of CGN, manifested by nephrotic syndrome, had several different correlative connections.

Thus, with MpGn, between the amount of mesangial cells, blood creatinine concentration (g = 0.65) and pro-teinuria (r = 0.56) a direct correlation was established. The fact that in MpG there is a direct correlation between the volume of deposits and hematuria, as well as between the density of deposits and proteinuria new connections, indicate Danielewicz, Wagrowska-Danielewicz.

With MMG, there are inverse correlative connections between the areas glomeruli of the kidney and the level of total protein in the blood (g = –0.74), pro-teinuria (r = –0.69), between the number of convoluted tubule cells and the blood creatinine content (r = –0.73), between the number of glomerular cells and the blood creatinine level (r = –0.88). Direct correlations tive connections exist between the cross-sectional area of glomerular capillaries and glomerular filtration rate (r = 0.54), the number of convoluted tubule cells and the level of total blood protein (r = 0.57), between the number of glomerular cells and the number of hyaline cylinders in urine (r = 0.56), between the section extraglomerular capillaries and the level of total protein in the blood (r = 0.63).

In MGN, a direct correlative relationship has been established between the area glomeruli and the level of total blood protein (r = 0.65), and between the

glomerular density and glomerular filtration rate (r = 0.52), as well as the number of mesangial cells and proteinuria (r = –0.80) revealed a feedback correlative connection has been established. The same direction was observed between the cross-sectional area of extraglomerular capillaries and the blood creatinine level (r = –0.53), between the cross-sectional area of ex-raglomerular capillaries and the level of proteinuria (r = –0.52).

The presence of correlations between the area of the glomeruli with the level of blood protein and glomerular filtration, proteinuria and the density of deposits in MGN is reported by others. In MGN, direct connections between the cross-sectional area are found glomerular capillaries and the level of total blood protein (r = 0.74), the number of convoluted tubules of the kidney and the level of total blood protein (r = 0.62), the number of mesangial cells and the number of unchanged red blood cells product in the urine (r = 0.56), the area of the convoluted tubule cells of the kidney and the level of total protein in the blood (r = 0.70). Inverse correlative connections They were established between the cross-sectional area of glomerular capillaries and the number of unchanged red blood cells in the urine (r = –0.57), the area of convoluted tubules and the level of proteinuria (r = –0.53), the area of convoluted tubule cells and the level of proteinuria (r = –0. 56), between the planes cross-sectional area of extraglomerular capillaries and the level of total protein in the blood (r = –0.79). About the presence of correlations in MCG between the level of blood creatinine and the number of cells containing the enzyme tryptase, as well as between the level of proteinuria and deposits, lo-localized in the basement membrane, Danielewicz reports, Wagrowska-Daniele.

In FibGn, direct correlations were found between the area of the glomeruli and the level of glomerular filtration (r = 0.65), the level of total blood protein (r = 0.51), between the cross-sectional area of the glomeruli merular capillaries and the number of leukocytes in urine (r = 0.72), area the number of convoluted tubule cells and the number of hyaline casts in the urine (r = 0.77). An inverse correlation was registered between the indicators of the number of convoluted tubule cells and the level of reabsorption (r = –0.96), and exceptionally young forms of Tr, which from the 3rd day were replaced by pathological gical forms of irritation.

Our results confirm the position that the development of CS is accompanied by endothelial dysfunction with activation of the vascular-platelet component of hemostasis, the development of disseminated intravascular coagulation and increased consumption of Thr. The developing TP acts as a trigger that triggers the proliferation of the megakaryocytic growth of the BM. Due to the increased consumption of Tr and the large demand However, in them, BM MCs do not have time to mature, as evidenced by the early

predominance of immature forms in the megakaryocytogram. As a result of this, immature MKs are released into the bloodstream, which we observed during CS in the microvasculature of the lungs. Availability of megaca-Ryocytosis of the lungs indicates that during CS the lungs become a hematopoietic organ in which the production of Thr is carried out. Under these conditions, immature MK of the lungs produce immature Thr, which enter the bloodstream and are not able to fully perform their functions. At the same time, during blood loss, changes in the "megakaryo-
cyt - platelet" are more pronounced and develop earlier.

Conclusions. Thus, in the "megakaryocyte-platelet" system in critical conditions associated with sepsis and blood loss, pronounced quantitative and qualitative changes occur, reflecting affecting the formation and subsequent progression of its functional nal insufficiency/insolvency. These changes are not contribute to the achievement of adequate compensation, favor pro-progression of the CS and require therapeutic correction.

Bibliography

1. Nephrology. Clinical recommendations / Ed. EAT. Shilova, A.V. Smirnova, N.L. Kozlovskaya. - M.: GEOTAR-Media, 2016. - 816 p. [Nephrologiya. Klinicheskie rekomendacii. Ed. by Shilov EM, Smirnov AV, Kozlovskaya NL Moscow: GEOTAR-Media; 2016. 816 rub. (In Russ).

2. Anderson JL, Gruppen EG, van Tienhoven-Wind L, et al. Glomerular filtration rate is associated with free triiodothyronine in euthyroid subjects: Comparison between various equations to estimate renal function and creatinine clearance. Eur J Intern Med. 2018;48:94-99.

3.Mario F, Pofi R, Gigante A, et al. Hypothyroidism and nephrotic syndrome: why, when and how to treat. Curr Vasc Pharmacol. 2017;15(5):398-403.

4. Marchiori R, Pereira L, Naujorks A, et al. Improvement of blood inflammatory marker levels in patients with hypothyroidism under levothyroxine treatment. BMC Endocr Disord. 2015;15:32.