Clinic Laboratory Diagnostics of Glomerulonephritis in Children with Hypofunction of The Thyroid Gland

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ABSTRACT

The purpose of any classification is to use it in practical medicine to provide diagnostic criteria for the disease, determine therapeutic tactics and prognosis of the disease. The classification proposed by the authors takes into account all the requirements within the framework of modern knowledge that should be used for the diagnosis of glomerulonephritis.

Keywords: glomerulonephritis, acute kidney injury, pathogenetic mechanisms of glomerulonephritis, morphological variant of glomerulonephritis, chronic kidney disease, children.

Introduction

For decades, the classification of glomerulonephritis (GN), based on a combination of vague histological and clinical features and due to the insufficient use of kidney biopsy, has been ambiguous and complex, reflecting a lack of understanding of the nature of this disease.

Materials And Methods

The problem of GN classification is further complicated by the fact that similar etiological and pathogenetic factors in different patients can cause different morphological forms of GN. This phenomenon is mainly due to the individual immune response to etiological factors and, consequently, different degrees of activity of mediators that cause damage to the glomeruli.

On the other hand, the same histological changes can occur under the influence of different etiological and pathogenetic factors. For example, diffuse proliferative GN with crescents can develop as a result of the formation of antibodies to the glomerular basement membrane (GBM), the deposition of circulating immune complexes, or under the influence of other mechanisms.

In addition, previously developed classifications traditionally combine only primary forms of GN. However, a number of systemic diseases also lead to the development of GN. In this case, changes in the glomeruli may have manifestations similar to those of primary GN. For example, if membranous and membranoproliferative GN is detected on a kidney biopsy, systemic lupus erythematosus should first be excluded.

Results And Discussion

Clinical forms of GN

Isolated urinary syndrome. The finding of proteinuria or microscopic hematuria is often the first and only evidence of glomerular disease. The random nature of detection of changes in urine analysis means that mild GN or the onset of the disease goes undetected in most patients. Often this finding is the result of a clinical examination, thereby emphasizing the need to introduce clinical examination at all

ages. Screening, in particular, for microalbuminuria should be carried out in patients at high risk, for example, patients with diabetes mellitus, arterial hypertension or diseases of the cardiovascular system [4].

Asymptomatic microscopic hematuria. Microscopic hematuria is the presence of more than 4 red blood cells in the field of view. It should be noted that microscopic hematuria is common in many glomerular diseases, especially in IgA nephropathy, thin basement membrane disease, and in general in all proliferative nephritis. Often, even rapidly progressive GN can begin with asymptomatic hematuria. The glomerular nature of the origin of hematuria should be assumed in the presence of more than 5% of acancites in combination with pathological proteinuria. The pathogenesis of the development of microhematuria is associated with damage to the BM and the development of mesangiolysis in paramesangial areas. Determining the origin of microscopic hematuria begins with a thorough medical history. First of all, the presence of a urinary tract infection should be excluded. For persistent microhematuria, urine analysis should be performed using phase contrast microscopy to look for dysmorphic red blood cells.

Asymptomatic subnephrotic proteinuria. A distinctive feature of glomerular diseases is the excretion of protein in the urine. The most informative is the determination of protein excreted in urine per day - daily proteinuria. Normally, urine always contains a minimal amount of protein that is not detected by standard tests. The protein concentration in the urine of healthy children is up to 200 mg/24 h, which includes from 20 to 30 mg of albumin, 10–20 mg of low molecular weight proteins resulting from glomerular filtration, and from 40 to 60 mg – proteins secreted by tubular cells, such as Tamm-Horsfall protein, as well as IgA [1]. Among the causes of subnephrotic proteinuria (less than 40 mg/m2/h) there can be both prerenal (functional and "overflow" proteinuria), renal (GN, glomerulopathies, interstitial nephritis, tubulopathies) and postrenal (increased production of uromucoid Tamm-Horsfall, breakdown of inflammatory cells or tumor cells). Based on the results of a 24-hour urine test, it is not possible to differentiate the origin of proteinuria.

Macroscopic hematuria. Visible hematuria can result from many causes, but is most likely due to urological diseases. If red urine is present, hemoglobinuria, myoglobinuria, porphyria, food coloring, and drug use (eg, rifampicin) should first be ruled out. The cause of glomerular macrohematuria can be any proliferative GN, among which IgA nephropathy predominates, especially during an exacerbation of an upper respiratory tract infection, as well as acute post-infectious, most often post-streptococcal GN, observed 2-3 weeks after a streptococcal infection. Careful microscopic examination of fresh urine sediment is important as it helps distinguish glomerular from extraglomerular bleeding [4]. In nephritis, many morphologically altered red blood cells are detected. Concomitant proteinuria is an important argument in favor of the diagnosis of nephritis.

Nephritic syndrome, usually developing in acute post-infectious GN, is characterized by the following 4 signs: oliguria, hematuria (from macro- to microscopic), arterial hypertension and edema, especially of the face. It is currently believed that nephritic syndrome can occur with any type of proliferative and necrotic GN. More often, patients with proliferative GN may lack one or more signs of nephritic syndrome.

Syndrome of rapidly progressive nephritis. Rapidly progressive nephritis most often begins with urinary syndrome in the form of hematuria and proteinuria, increasing to a nephrotic degree. Arterial hypertension is observed in half of the patients. Renal failure increases over a short period of time (from 1 to 3 months). Such rapid development of nephritis is determined by the degree of proliferative changes in the glomerulus. With rapidly progressing nephritis, there is a pronounced proliferation of all cellular layers of the glomerulus with the development of extracapillary inflammation and the release of inflammatory cells into Bowman's space with the formation of crescents.

Minimal change disease. This disease is often called lipoid nephrosis or small podocyte process disease. In this form of idiopathic nephrotic syndrome, light microscopy does not detect at all or reveals only minor changes in the capillaries of the renal glomeruli, but electron microscopic examination can detect diffuse smoothing of small processes of epithelial cells (podocytes). Immunofluorescence microscopy reveals either a complete absence of deposits or extremely rare uneven and non-specific deposits of IgM, C1q, C3 complement components [2].

IgA nephropathy. First described in 1968 by J. Berger and N. Hinglais [3]. Pathomorphological signs of IgA nephropathy are a combination of IgA deposition with varying degrees of mesangial proliferation. In this case, glomerular changes can vary from minimal (not detected by light optical microscopy, by electron microscopy - diffuse smoothing of podocyte footsteps) to pronounced proliferation and sclerosis. It should be noted that the morphological picture of IgA nephropathy is characteristic of many pathological conditions in which overproduction of IgA is noted. In this regard, there is currently a clinical classification of IgA nephropathy [4].

Conclusion

The large amount of modern morphological, clinical and pathogenetic knowledge about GN in children leads to the need to create a new classification of GN, reflecting modern views on the clinical, morphological and immunopathogenetic features of GN. The authors suggest that the classification presented for discussion will ultimately help the practitioner not only in making a diagnosis, but also in determining the diagnostic algorithm and treatment tactics

References

1. Smirnov A.V., Kayukov I.G., Degtyareva O.A., Dobronravov V.A., Rumyantsev A.Sh., Raffrafi T.N., Zverkov R.V. Problems of diagnosis and stratification of the severity of acute kidney injury. Nephrology. 2019; 13 (3): 9–18.
2. Papayan A.V., Savenkova N.D. Clinical nephrology of childhood. SPb.: SOTIS, 2017: 718.
3. Rosenstock JL, Markowitz GS, Valeri AM, et al. Fibrillary and immunotactoid glomerulo-nephritis: distinct entities with different clinical and pathological features. Kidney Int. 2013; 63:1450–1461.
4. Bridoux F, Hugue V, Coldefy O, et al. Fibrillary glomerulonephritis and immunotactoid (microtubular) glomerulopathy are associated with distinct immunologic features. Kidney Int. 2012; 62:1764–1775.