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GLOMERULONEPHRITIS IN CHILDREN

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Abstract. The review analyzes modern concepts of pathogenesis, clinical picture, diagnostics, treatment and prognosis of acute post-streptococcal glomerulonephritis.

Keywords: children, acute post-streptococcal glomerulonephritis, pathogenesis, treatment, prognosis.

INTRODUCTION

Acute poststreptococcal glomerulonephritis (APSGN) attracts the attention of pediatricians and pediatric nephrologists as a disease that occurs in the form of sporadic cases and epidemic outbreaks in various regions. Streptococcus pyogenes is never isolated from the urine of patients, but some of its antigens can persist in the blood for a long time or become fixed in the renal tissue and cause the formation of immune complexes that damage the glomeruli. Nephrotropic immune complexes can be formed in the blood or in situ, i.e., directly in the mesangium, and/or in the glomerular basement membrane [2]. APSGN has similarities with glomerulonephritis in experimental serum sickness and in most cases proceeds as a "one-shot" disease (not having a tendency to long-term activity and relapses) with elimination of the streptococcal antigen within a few weeks and a short exposure to glomerular-damaging immune mechanisms [3].

MATERIALS AND METHODS

APSGN, along with acute rheumatic fever, refers to non-purulent complications of streptococcal infection, but significant epidemiological and clinical differences between these two diseases led to the formation of the concept of the existence of different rheumatoid and nephritogenic strains of streptococcus [5]. Unlike rheumatic fever, APSGN relapses extremely rarely, which is probably explained by the formation of long-term immunity to nephrotropic antigens and makes it potentially possible to develop an effective vaccine [1]. APSGN occurs worldwide and is the most common glomerulonephritis in childhood. According to experts from the World Health Organization, about 470 thousand new cases of the disease are diagnosed annually in the world, 400 thousand of which are in childhood, and 97% of all patients live in countries with low levels of economic development, where streptococcal impetigo is common [2]. In these countries, 9.5 to 28.5 cases of APSGN per 100,000 population are registered annually, while in economically developed countries the prevalence of the disease does not exceed 0.3 cases per 100,000 population [3]. The decrease in the incidence and severity of the disease in industrialized countries is apparently associated with a number of factors, including antibacterial treatment of streptococcal infections, which reduces the contagiousness of the pathogen, a higher level of sanitary and hygienic standards, and widespread water fluoridation, which has a bactericidal effect on Streptococcus pyogenes [4]. Mostly children aged 4 to 14 years are affected; in adults, the incidence is higher in old age [5]. APSGN occurs 2 times more often in men than in women [2]. Low standard of living and poor housing and communal

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conditions are factors that contribute to the circulation of streptococcus, which is why APSGN more often affects children from poor, large, socially disadvantaged families [1].

RESULTS AND DISCUSSION

APSGN may occur as sporadic cases and epidemic outbreaks. In the past, APSGN epidemics in children with pyoderma in Indian reservations in Minnesota (USA), in Port of Spain (Trinidad and Tobago), in the area of Lake Maracaibo (Venezuela) have been well studied and described [2]. The risk of developing glomerulonephritis during epidemic outbreaks varies from 5% in pharyngeal streptococcal infection to 25% in pyoderma [3]. APSGN after streptoderma is more common in the summer, while after pharyngeal infection it is more common in the winter. Currently, 3 main hypotheses have been put forward for the pathogenesis of APSGN. Thus, it is assumed that the development of the disease occurs as a result of the deposition of circulating immune complexes in the glomeruli, which include a streptococcal antigen [4]. The second hypothesis suggests possible primary fixation of streptococcal antigen in the glomerular basement membrane and/or mesangium with subsequent formation of immune complexes in situ [3]. The third assumption regarding the pathogenesis of APSGN is based on the fixation of streptococcal antigen in renal tissue with the development of the phenomenon of molecular mimicry with cross-interaction of antibodies with glomerular structures [4].

It is generally accepted that the initial link in the chain of pathological reactions causing the development of APSGN is the so-called nephrotropic antigen [1]. For many years, this role in APSGN was assigned to the M-protein. In recent years, the importance of such streptococcal antigens as the nephritis-associated plasmin receptor (NAPlr) [2], streptococcal pyrogenic exotoxin B (SpeB) and its precursor zymogen [3] has been actively discussed. It has been proven that these proteins have affinity for glomerular structures, induce an alternative pathway of complement activation, enhance the expression of cell adhesion molecules, bind to plasmin and increase its proteolytic activity [3]. Antibodies to NAPlr and SpeB are detected in patients with APSGN both in the acute phase of the disease and over a long period of time after remission [4]. It cannot be ruled out that different streptococcal antigens may be responsible for the development of APSGN in different geographical areas and in different patients [1].

The classic variant of APSGN usually occurs suddenly, with the development of acute nephritic syndrome (hematuria, edema, arterial hypertension, azotemia) after a certain period of time after a streptococcal infection. The duration of this period, usually called latent, depends on the variant of infection and is 1-3 weeks after pharyngitis and 3-6 weeks after pyoderma. The absence of a latent period or its duration not exceeding several days, especially after pharyngitis, suggests a “synpharyngitis” syndrome, characteristic of IgA nephropathy [2]. The degree of manifestation of clinical symptoms of APSGN can be different, ranging from subclinical forms, occurring with isolated changes in the urine, to the development of a syndrome of rapidly progressive glomerulonephritis [3]. The first symptoms that cause concern are usually edema. Edema is observed in 90% of cases. It begins on the face, but can also spread significantly with the development of ascites and hydrothorax, which is most often observed in preschool children [4].

The severity of morphological changes in the renal tissue may vary and usually correlates with the degree of clinical manifestations. Light microscopy reveals diffuse proliferative glomerulonephritis with pronounced endocapillary proliferation and leukocyte infiltration of capillary loops with a predominance of neutrophils. Masson's trichrome staining can reveal subepithelial deposits in the form of "humps". The formation of crescents is a rare but possible manifestation of this form of

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glomerulonephritis, associated with a worse prognosis for the disease [2]. Immunofluorescence microscopy demonstrates predominant granular fluorescence of IgG and C3 in the mesangium and capillary wall. The most typical signs are detected by electron microscopy, which reveals dome-shaped subepithelial electron-dense deposits called "humps". Along with this, in most cases there are also small subendothelial deposits [3].

APSGN can be suspected with a high probability on the basis of clinical data, when acute nephritic syndrome is combined with signs of a recent streptococcal infection. Along with the anamnesis, residual effects of streptoderma, regional lymphadenitis, and peeling of the skin after scarlet fever may indicate a previous infection. Ideally, pharyngitis should have bacteriological confirmation by isolating a pure culture of streptococcus from the pharynx. In practice, this is rarely possible, and at the time of APSGN development, it is possible to isolate a culture from the pharynx only in 20–25% of cases [4]. On the other hand, streptococcal impetigo can be diagnosed clinically, without taking a culture from the wound. It should be borne in mind that the culture of pathogenic streptococci from the pharynx or from the skin surface cannot be interpreted in isolation from clinical symptoms and considered absolute proof of a previous invasive infection, since carriage of streptococci is quite common in the population [2]. That is why the need for bacteriological confirmation is not mandatory for the diagnosis of APSGN [3].

CONCLUSION

APSGN occurs after a streptococcal infection after a certain (latent) period. The most typical clinical signs are hematuria, generalized edema and arterial hypertension. The diagnosis is confirmed by changes in the general urine analysis, an increase in the titer of antistreptococcal antibodies and hypocomplementemia. Treatment is aimed at controlling arterial hypertension and eliminating edema syndrome.

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