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BRIGHT'S DISEASE IN CHILDREN: REGIONAL FREQUENCY OPTIONS AND CLINICAL COURSE

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ABSTRACT

Many glomerular disorders, especially in children, have a temporal association with viral infections. When infection is clinically quiet in other cases of glomerulonephritis, viral syndromes can still be implicated as a cause. However, there is currently a lack of clear evidence for viral causation in the majority of glomerular diseases. While many case reports in children describe the onset of distinct kinds of glomerular illness following seroconversion to a variety of viruses, only a few give pathologic evidence of viral infection associated with glomerular lesions on kidney biopsy. Although there is a strong link between hepatitis viruses and glomerular injury in adults, the hepatitis C virus does not appear to be a cause in children. Treatment of hepatitis B virus, cytomegalovirus, and human immunodeficiency virus in children with membranoproliferative, membranous, and collapsing glomerulopathy plays a significant part in the treatment of glomerular disorders when they are detected. Otherwise, there is little indication that the discovery of a viral infection in a kid with glomerulopathy should alter the infection's or glomerulonephritis' care. As a result, more research into this subject is urgently required. -[6]

Keywords: children's, Bright's disease, frequency, clinical course, virus-associated, risk factors, disabled, chronic glomerulonephritis, end-stage kidney injury, dialysis.

INTRODUCTION

Many glomerular disorders, particularly in youngsters, are associated with the start of viral infections. Viral syndromes can still be implicated as a cause in some cases of glomerulonephritis, even if the infection is clinically quiet. In most glomerular disorders, however, there is currently a lack of clear evidence indicating viral causation.

Despite the fact that no single virus culture has been definitively linked to any specific renal pathology, infection and host antiviral responses are associated to a small number of glomerular disorders. The majority of these association studies are old and only involve a few patients.

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Now that novel molecular diagnostics are available, several glomerular disease classifications have been reviewed, and vaccinations have modified the patterns of viral infection, a review of these associations is required. Pediatric nephrologists may eventually learn that the results of viral-associated glomerulopathies differ significantly from those of true idiopathic glomerulopathies.

MATERIALS AND METHODS

According to the International Committee on Virus Taxonomy, viruses are classified into seven orders, 103 families, 455 genera, and over 2,800 species. The number of human viral pathogens continues to rise, despite the fact that only a portion of these species are known to infect people. Viruses are polytropic in nature, infecting several tissues or organ systems. Kidney cells are frequently infected during viral infections, but they appear to be more resistant to harm than other organs and tissues. During viral syndromes, viruria and viremia are frequently measured. Several viruses, including adenovirus, cytomegalovirus (CMV), Coxsackievirus, measles, and varicella viruses, have been cultured in the laboratory using human kidney cells. In contrast to viral arthritis, hepatitis, meningitis, otitis, pharyngitis, pericarditis, pneumonitis, and tonsillitis, the kidneys rarely bear the brunt of infection, either from cytotoxic consequences or from host antiviral responses. When kidney infection causes kidney injury, it can be difficult to tell the difference between infectious and non-infectious causes. -[6].

Multiple relapses of nephrotic syndrome (NS) in children are typically caused or related with infections. NS patients are more susceptible to bacterial and viral infections.[8]

Accumulating a viral-related glomerulopathy necessitates histopathological, viral-culture, or polymerase chain reaction-based proof of viral contamination in addition to clinical or pathologic proof of kidney disease (PCR). And that is undeniably a difficult task, and a considerable number of case reports or companion studies of renal contribution during viral diseases have failed to do so satisfactorily. Albuminuria and erythrocyturia are common in many febrile infections, and they aren't the only signs of glomerular impairment. Viruria, or the presence of incorporation-bearing cells in the urine, can be a cause or effect of glomerular damage, or it could actually reflect glomerular catching during viremia. Certain infections are common in the kidney and can be excreted without causing harm. Whereas polyoma infections (BK and JC infections) have been shown to infect glomerular epithelial cells in the kidney and cause interstitial nephritis and, in rare cases, crescentic glomerulonephritis (GN) in relocated kidneys, there have been no

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reports of these infections contaminating glomerular cells or causing glomerulopathy in local kidneys, even in immunocompromised patients.

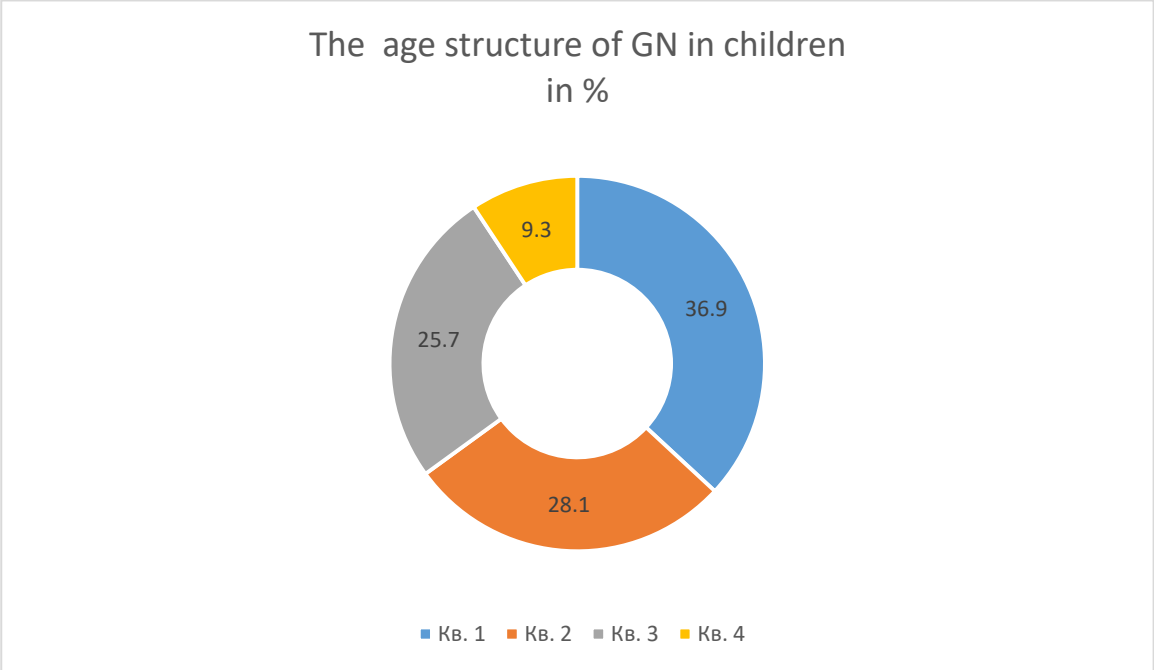
The outcome for viral infections with CG ranges from spontaneous remission to FSGS and chronic renal disease development. There is no indication that NS relapses triggered by viral infection respond any faster or slower than relapses triggered by other factors, or that an association between NS onset and specific infections impacts steroid sensitivity or dependency.

The goal of this study is to examine the geographical alternatives for GN frequency and clinical course in children residing in the Republic of Uzbekistan's Bukhara region..

The health of 249 sick children with GN who were admitted to the Bukhara regional children's multifunctional medical center for inpatient examination and treatment were tracked. General blood tests, urine tests, Nechiporenko and Zimnitsky urine tests, biochemical testing, and functional research methods were all performed on all patients.

There were somewhat more boys (161 (64.6%) than girls (88%) among those polled (35.4 percent). The sick children examined ranged in age from one year to eighteen years, with children under five years accounting for 70 percent of the total, 6-10 years for 92 percent, 11-15 years for 64 percent, and 16-18 years for 23 percent (9.3 percent).

Diag. 1



Patients were separated into two groups for a comparative investigation of the impact of risk factors:

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1-group: 138 (55.5%) sick children with GN caused by a virus;
2-group: 111 (44.5%) sick children with GN not caused by a virus.
The data of the official medical statistics of the regional Childrens' Health Department of the Bukhara region for 2017-2019 were studied retrospectively.
The "case-control" method was used to calculate the risk variables for the development of GN. The odds ratio (or) was calculated as follows: if the OR is greater than 1, it suggests that the chances of identifying a risk factor are higher in the group with an outcome, and the factor has a direct association with the likelihood of an outcome. An OR of less than one shows that the chances of discovering a risk factor are higher in the second group, and that the factor has an inverse association with the likelihood of a specific result- [8].

DISCUSSION

According to the findings of a three-year retrospective study, 43293 (76%) children out of the total number of children were admitted to the hospital with disorders of the genitourinary apparatus (UTD) throughout the study period. The prevalence of GN in the examined population is 17.3 percent. It was discovered that the frequency of hospitalization of children with gastrointestinal tract disorders accounts for 7.61 percent of all children's hospitalizations.
Children residing in rural areas were more likely to be hospitalized, according to an analysis of morbidity and hospitalization at their place of residence - 204. (81.9 percent).
The number of children hospitalized to hospitals with UTD increased by about one-sixth during the same time period. In children, the nosological organization revealed a prevalence of chronic GN. As a result of our research, we discovered that: CGN - 137 (55.0%), AGN - 75 (30.2%), and initial syndrome (NS) - 37. (14.8 percent) **Tab. 1**

Nosological structure of GN		
Chronic GN	Acute GN	1st NS
137	75	37
55.0%	30.2%	14.8%

Children in this class had comorbid pathology, according to the test. CGN is usually caused by the following circumstances. 68 (49,6 percent), oral cavity disorders – 68 (49,6 percent), skin redness – 18 (13.2 percent), and edema – 78 (56,9 percent), herpes infection – 55 (40,2 percent), diarrhea – 28 (20,4 percent), convulsive

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syndrome – 1 (0,73 percent), haemorragic vasculature – 1 (0,73 percent), haemorragic va (0,73 percent).**Tab.2**

Comorbid patholgy structure of CGN									
IDA	RRI	delayed physical development	oral cavity disorders	skin redness	edema	herpes infection	diarrhea	convulsive syndrome	vasculitis
64, 3%	89,0 %	50,4%	49,6%	13.2%	56,9%	40,2%	20,4 %	0,73%	0,73%

In AGN, the frequency of comorbid pathology was as follows:: reccurent respiratory viral infections (RRVI))- forty six (61,4%), iron deficiency anemia I-IIdegree--twenty one (28,0%), allergic reaction - two (2,7%), class Insecta allergic - one (1,4%), haemorragic vasculitis-1 (1,4%), pox -1 (1,4%), diarrhea-1 (1,4%), measles-1 (1,4%).

Tab. 3

Acute GN comorbid pathology structure						
RRVI	IDA	allergic reaction	haemorragic vasculitis	pox	diarrhea	measles
61,4%	28,0%	2,7%	1,4%	1,4%	1,4%	1,4%

Primary NS happens in comorbidity with RRVI-23 (62.2%), iron deficiency anemia of I-IIdegree-5 (13.5%), undetermined-6 (16.2%), herpes infection-1 (2.7%), food allergy-1 (2.7%), dental caries-1 (2.7%)

Tab.4

Primary NS comorbidity structure					
RRVI	IDA	undetermined	herpes infection	food allergy	dental caries
62.2%	13.5%	16.2%	2.7%	2.7%	2.7%

CONCLUSION

As a conclusion of the study, various geographical options for GN in children were discovered: GN is much more common in rural children; comorbid kinds of GN predominate in the clinical picture; GCN predominates in the structure of urinary organ illnesses. Comorbid pathology is a predictor of GN development in children, and thus the method's transfer to a chronic course. Bright's disease in children has

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several regional characteristics: The prevalence of GN in boys is much higher than in girls; the clinical course is overtaken by comorbid GN pathologies; CGN is ubiquitous in the composition of renal diseases; pathological pregnancy and childbirth, viral and allergic diseases of the child, and the nonspecific factor of hypothermia (P0.01) are risk factors for the development of virus associated GN in children. Allergic disorders in the family, previous vaccine reactions, diarrhea, and convulsive syndrome in the kid are all risk factors for the development of non-virus associated GN in children (P0.01).

As a result, identifying key risk factors for the progression of GN in children, particularly in early life, is theoretically significant. Renal pathology can be reduced by increasing preventative efforts during the prenatal and postnatal periods of pregnancy and delivery, especially when GN is related with the virus in children.

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