eISSN: 2229-3213 pISSN: 2229-3205
The Journal of Applied Science and Social Science
Volume 16,Issue 04 April, 2026

DYNAMICS OF URINARY TRACT INFECTIONS IN EARLY CHILDHOOD DURING ACUTE RESPIRATORY VIRAL INFECTION

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Introduction

In the structure of morbidity among pediatric patients, urinary system infections (USIs) rank second after acute respiratory viral infections (ARVIs). In children during the first three years of life, USIs occur more frequently than acute respiratory infections . The relevance of the problem of developing USIs against the background of ARVIs is determined by their high prevalence and the risk of severe complications in the urinary system (US), including chronic kidney disease (CKD), as well as the complexity of developing specific antiviral therapy in children, which is associated with etiological heterogeneity, high contagiousness of pathogens, and variability of viral antigenic properties .

ARVIs in early childhood contribute to heightened immune responses and provoke superimposed bacterial infections, including the development of pyelonephritis and infections of the distal urinary tract.

Keywords

Infection, Urinary System, Viral Infection, Immunity, Bacteriuria, Genferon Light

Immune Response Features in Children under Three Years of Age

Reduced immune response indicators in infants are considered an adaptive protective mechanism against overly intense cytokine reactions. Part of the passive protection is provided by immunoglobulins present in breast milk. However, this is often insufficient to protect against infections.

During immune system development in infants, two critical periods are identified. The first – the neonatal period – up to 29 days of life, when the child's body is protected only by maternal antibodies received via the placenta and breast milk . Neonates are highly susceptible to bacterial and viral infections during this period. Premature infants represent a particularly high-risk group . The second period occurs between four and six months of age, characterized by the loss of maternally acquired antibodies and weak synthesis of immunoglobulin (Ig) M. Insufficient local mucosal protection is linked to delayed accumulation of secretory IgA, resulting in high susceptibility to many airborne and intestinal infections.

During the neonatal period, both innate and adaptive immune responses are present . The innate response is characterized by reduced interferon (IFN) production, generation of immature early IFN-alpha, incomplete phagocytosis, and weak response to bacterial antigens. The adaptive response shows an excessive level of suppressor T cells, low cytotoxic activity of CD8+ lymphocytes and natural killer (NK) cells, a reduced number of plasma cells with normal B lymphocyte counts (cellular arm), delayed antibody synthesis, circulation of maternal IgG until six months, and insufficient IgA-producing plasma cells (humoral arm).

Key features of the immune system in infants include recovery of IgM levels, own IgG synthesis only by six months, a high CD4+/CD8+ lymphocyte ratio, relative immaturity of T cells, low capacity for memory cell formation, and relative IgA deficiency. The third critical period occurs during the second year of life, when children have increased contact with the external environment. The immune system functions more fully, lymphocyte activity is activated, IgG production increases significantly, and long-term immunity begins to form. However, a deficit of local protective factors remains, as evidenced by continued high susceptibility to bacterial and viral pathogens .

Thus, in early childhood, all major mechanisms of nonspecific protection against pathogenic bacteria and viruses are weakened, explaining the high susceptibility to bacterial and viral infections, development of severe infectious disease, complications, and infection generalization.

Urinary System Infection: Definition and Classification

The term “urinary system infection” (USI) refers to infection and inflammatory changes in any part of the urinary system without specifying the exact level (urinary tract or renal parenchyma) . Currently, there is no unified classification of USIs, which is due to different approaches to systematization: complicated or uncomplicated infection, persistent infection or reinfection, symptomatic or asymptomatic (Figure) . Persistent bacteriuria is usually the result of inadequately treated infection, most commonly due to microbial resistance to the administered drug. Persistence or reinfection refers to infections detected after documented negative urine cultures. Reinfection, unlike persistence, is caused by a different microorganism. Persistence typically occurs in children with anatomical abnormalities of the urinary system.

Epidemiology

USIs, including pyelonephritis, cystitis, urethritis, and asymptomatic bacteriuria, are the most common nephropathies in childhood . Annual population studies indicate a steady increase in morbidity among children. According to Rosstat, over the past ten years, the prevalence of nephropathies among children under 14 has increased 1.6-fold and among adolescents – twofold . USIs occupy a leading position in the spectrum of pediatric diseases, accounting for 18 cases per 1,000 children .

The mild and nonspecific clinical presentation of kidney and urinary tract diseases in children under three years complicates early diagnosis, delaying adequate therapeutic correction and leading to chronicity . The development of urinary system pathology in early childhood is associated with morphofunctional immaturity of the kidneys, especially in the presence of obstructive uropathies, infections, hypoxic conditions, or a history of intensive care .

Etiology and Pathogenesis

Bacteria, fungi, viruses, and opportunistic flora can be etiological agents of USIs. The most common pathogens are Enterobacteriaceae, primarily Escherichia coli, detected in approximately half of cases (41.3–83.3% depending on the region) . Prolonged and recurrent pyelonephritis may involve Mycoplasma, Chlamydia, and Candida species. In early childhood, pyelonephritis generally develops in the presence of urinary tract dysfunction or cytomembrane

instability.

The host's features also contribute to a high risk of urinary system infections, particularly congenital anomalies and urinary flow disturbances. Morphological anomalies of the urinary tract underlie 90.3% of pediatric pyelonephritis cases . Viruses also play a role in acute cystitis and pyelonephritis, triggering inflammatory exacerbations with subsequent bacterial invasion.

Key factors in viral etiology of USIs in children include :

- Immature immune system (reaches adult levels at 10–12 years)
- High contagiousness of viral infections
- Unstable immunity to certain pathogens
- Diversity of viral serovars
- Viral suppression of interferon production

Transmission is primarily airborne, but contact and household routes (e.g., via unwashed hands) are also possible.

In children, ascending infection is typical; hematogenous infection is characteristic of neonates and infants with reduced immunity. The lymphatic route has not been convincingly documented. Periurethral colonization is an important contributing factor, with microorganisms translocating from the intestines to the bladder . Urinary flow disturbances are critical in the pathogenesis of kidney inflammation .

Clinical Presentation

Pyelonephritis in early childhood often presents latently or with mild symptoms, delaying diagnosis and potentially leading to CKD .

In neonates, nonspecific signs dominate: intoxication, subfebrile temperatures, poor sucking, insufficient weight gain, and bowel dysfunction. Children up to five years may present with anorexia, vomiting, dyspepsia, and nonspecific abdominal pain . Latent pyelonephritis reflects reduced immune function. Infection generalization is common, and even acute inflammatory processes can cause rapid tissue destruction in neonates.

Diagnostics

Key laboratory analyses confirming pyelonephritis include:

- **Urinalysis:** leukocyturia (15–20 cells per field for distal tract involvement; full fields for pyelonephritis), bacteria, neutral/alkaline pH, turbidity, salts, mild proteinuria (0.06 g/L, higher with urinary tract malformations or reflux)
- **Complete blood count:** leukocytosis, elevated ESR
- **Urine culture:** E. coli most frequent, less commonly Klebsiella, Pseudomonas, Proteus

Significant bacteriuria: ≥10^5 CFU/mL in freshly voided urine. Catheterized urine: ≥10^3 CFU/mL. Repeat cultures in neonates under three months are indicative of infection regardless of count . Additional diagnostics: detection of rare pathogens (Chlamydia, Mycoplasma,

Ureaplasma, fungi, Mycobacterium tuberculosis), humoral and cellular immunity assessment, urine biochemistry, serum biochemistry, kidney ultrasound, cystography, urography, renoscintigraphy, and dynamic/static scintigraphy . Recent studies highlight procalcitonin as a highly sensitive marker for USIs, with specificity of 89.7% compared to 18.5% for C-reactive protein .

Treatment and Recurrence Prevention

USI therapy in children includes general and specific measures:

- **General:** diet, physical therapy, massage, hygiene, increased fluid intake (+50%)
- **Specific:**
  - Prompt antibacterial therapy with renal-excreted drugs targeting major pathogens
  - Long-term antimicrobial prophylaxis for reflux or recurrent infections
  - Correction of urinary flow disturbances and bowel motility
  - Antifungal and antiviral therapy
  - Desensitizing and membrane-stabilizing therapy
  - Immunomodulatory and anti-sclerotic therapy

Cephalosporins (2nd/3rd generation) and protected penicillins are preferred. Treatment duration: pyelonephritis 10–14 days, cystitis seven days. Oral antibiotics preferred for outpatient cystitis . Routine measures to restore urinary flow: hydration, scheduled voiding, double voiding at night. Post-antibiotic therapy: uroantiseptics.

Interferons

Interferons are the most studied immunomodulators approved from birth. They have potent antiviral activity .

Neonates have early IFNs in circulation, differing biologically from adult IFNs. Early IFN-alpha plays a key role in antiviral defense, while IFN-gamma coordinates innate and adaptive immunity .

Human leukocyte interferon was developed in Russia in 1967, with studies confirming rectal and oral efficacy . Recombinant IFNs allow precise dosing and higher purity.

Mechanism of action :

- Induce antiviral proteins
- Enhance phagocytosis and killer cell activity
- Stimulate secretory IgA
- Restore cytokine balance
- Prevent infection of healthy cells
- Immunomodulation

IFN-alpha-2 induces early antiviral defense and adaptive immunity activation .

Genferon Light, a pediatric suppository containing IFN-alpha-2b and taurine, is approved from birth and during pregnancy (≥13 weeks). Dosage: 0–7 years: IFN-alpha-2b

125,000 IU + taurine 5 mg; >7 years: IFN-alpha-2b 250,000 IU + taurine 5 mg. Administered rectally twice daily for five days . Its use is pathogenetically justified in ARVIs and USIs in neonates and infants, including mixed etiology infections or coexisting ARVI, allowing age- and weight-adjusted dosing and minimizing drug load.

Conclusion

Children under three with USIs should be monitored by pediatricians and nephrologists due to subtle clinical signs and higher risk of kidney damage [31]. Proper antibacterial, antifungal, antiviral, and anti-sclerotic therapy promotes faster recovery and reduces chronicity risk for both upper and lower urinary tract infections.

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