

CAUSES OF DEVELOPMENT AND CLINICAL FEATURES OF PYELONEPHRITIS IN CHILDREN

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Annotation: Pyelonephritis is one of the most common infectious diseases of the urinary system in childhood and represents a serious medical and social problem. The disease is characterized by inflammatory damage to the renal parenchyma and the pelvicalyceal system, mainly caused by bacterial infection. In pediatric practice, pyelonephritis often develops due to ascending infection from the lower urinary tract and is associated with congenital anomalies, immune immaturity, and functional disorders of the urinary system. The early diagnosis and proper treatment of pyelonephritis are extremely important because untreated or recurrent infections may lead to renal scarring, hypertension, and chronic kidney disease. This article analyzes the main etiological factors, pathogenesis, and clinical manifestations of pyelonephritis in children based on scientific literature. Particular attention is given to risk factors, diagnostic approaches, and characteristic symptoms observed in pediatric patients.

Keywords: Pyelonephritis, urinary tract infection, children, kidney inflammation, pediatric nephrology, bacterial infection, renal parenchyma, clinical symptoms, urinary tract anomalies

Introduction

Pyelonephritis is an inflammatory disease of the kidney characterized by infection of the renal parenchyma and the collecting system. It is considered one of the most severe forms of urinary tract infections (UTIs) in children and may lead to long-term renal damage if not treated appropriately. According to epidemiological studies, urinary tract infections occur in approximately 8–10% of girls and 2–3% of boys during childhood, and a significant proportion of these infections involve the kidneys in the form of acute pyelonephritis [1].

The incidence of pyelonephritis varies depending on age and gender. During the neonatal period and early infancy, the disease is more common in boys due to congenital anomalies of the urinary tract. However, after the first year of life, girls become more susceptible because of anatomical features such as a shorter urethra and proximity to the perineal flora [2].

The most common causative agents of pediatric pyelonephritis are gram-negative bacteria, especially *Escherichia coli*, which accounts for nearly 70–90% of cases. Other pathogens include *Klebsiella*, *Proteus*, *Enterobacter*, and *Pseudomonas* species [3]. These microorganisms typically ascend from the lower urinary tract and reach the kidney through the ureters.

Pyelonephritis is also associated with several predisposing factors such as vesicoureteral reflux, urinary tract obstruction, neurogenic bladder, poor hygiene, and immune system immaturity. Early recognition of these factors is crucial for preventing recurrent infections and permanent renal damage [4].

Therefore, the study of etiological factors and clinical characteristics of pyelonephritis in children is essential for improving diagnostic accuracy and developing effective preventive strategies.

Methodology

This scientific article is based on the analysis of modern medical literature related to pediatric nephrology and urinary tract infections. The research utilized analytical, comparative, and descriptive methods to study the causes and clinical manifestations of pyelonephritis in children.

Scientific sources including medical textbooks, international clinical guidelines, and peer-reviewed journal articles were examined to obtain reliable information about the epidemiology, etiology, and pathophysiology of the disease. Data from epidemiological studies and clinical observations were analyzed to identify the most common risk factors and characteristic symptoms of pediatric pyelonephritis.

Additionally, comparative analysis of different scientific studies was conducted to evaluate variations in clinical presentation depending on age, gender, and associated urinary tract abnormalities. The methodological approach allowed for a comprehensive evaluation of the mechanisms of disease development and its clinical features in pediatric patients.

Results

The analysis of medical literature indicates that pyelonephritis in children develops as a result of complex interactions between infectious agents and predisposing host factors. Bacterial infection is the primary cause of the disease, with *Escherichia coli* being the most frequently isolated pathogen [5].

Several mechanisms contribute to the development of kidney infection. The ascending route is the most common pathway, in which bacteria migrate from the urethra to the bladder and then ascend through the ureters to the renal pelvis. Hematogenous spread of infection is less common but may occur in newborns and infants with systemic infections [6].

Congenital anomalies of the urinary tract play a significant role in the development of pediatric pyelonephritis. Vesicoureteral reflux, a condition in which urine flows backward from the bladder into the ureters and kidneys, is identified in approximately 30–50% of children with recurrent urinary infections [7].

Other important risk factors include urinary obstruction caused by kidney stones or structural abnormalities, neurogenic bladder dysfunction, and improper emptying of the bladder. These conditions facilitate bacterial colonization and increase the likelihood of infection spreading to the kidneys [8].

Clinical manifestations of pyelonephritis in children vary depending on age. In infants, the symptoms are often nonspecific and may include fever, irritability, poor feeding, vomiting, and failure to gain weight. In older children, typical symptoms include high fever, flank pain, dysuria, frequent urination, and abdominal discomfort [9].

Laboratory findings commonly reveal leukocytosis, elevated inflammatory markers, and bacteriuria in urine analysis. Urine culture is considered the gold standard for identifying the causative microorganism and determining appropriate antibiotic therapy [10].

Analysis and Discussion

Pyelonephritis in children is considered one of the most clinically significant forms of urinary tract infections because of its potential to cause both acute illness and long-term renal complications. The disease represents a complex interaction between bacterial pathogens, host immune defenses, and anatomical or functional abnormalities of the urinary tract. Numerous clinical and epidemiological studies emphasize that the development of pediatric pyelonephritis cannot be explained by infection alone; rather, it results from a combination of microbial virulence factors and host susceptibility [3].

One of the primary mechanisms involved in the pathogenesis of pyelonephritis is the ascending spread of microorganisms from the lower urinary tract to the kidneys. In most pediatric cases, infection begins with colonization of the periurethral region by intestinal bacteria, followed by their entry into the urethra and bladder. If host defense mechanisms fail to eliminate these microorganisms, they may ascend through the ureters and reach the renal pelvis and parenchyma [5]. The ability of pathogens to move upward through the urinary tract is strongly influenced by urinary stasis, vesicoureteral reflux, and structural abnormalities that facilitate bacterial migration.

Among the etiological agents of pediatric pyelonephritis, *Escherichia coli* is recognized as the dominant pathogen. Clinical studies indicate that this microorganism accounts for approximately 70–90% of community-acquired urinary tract infections in children [3]. The predominance of *E. coli* is largely explained by its specific virulence factors, which allow the bacteria to adhere to epithelial cells lining the urinary tract. These virulence mechanisms include P-fimbriae, type 1 fimbriae, hemolysins, and siderophores that enhance bacterial survival in the

host environment [12]. Through these structures, the bacteria attach firmly to uroepithelial cells, resist mechanical flushing by urine, and initiate inflammatory reactions within renal tissue.

The host immune response plays a crucial role in the progression and severity of infection. When bacteria reach the renal parenchyma, the immune system activates a complex cascade of inflammatory reactions. Neutrophils, macrophages, and cytokines are rapidly recruited to the site of infection in an attempt to eliminate the invading microorganisms. Although these immune mechanisms are essential for controlling infection, excessive inflammation can lead to tissue damage and renal scarring [11]. Therefore, the clinical outcome of pyelonephritis often reflects a balance between bacterial virulence and host immune defense.

Age-related physiological characteristics significantly influence the development and clinical presentation of pyelonephritis in children. Neonates and infants are particularly vulnerable to severe infections because their immune systems are not fully mature. The reduced production of immunoglobulins, complement proteins, and cytokines during early infancy limits the body's ability to contain bacterial spread [1]. As a result, infections in young children often present with systemic symptoms such as high fever, vomiting, irritability, and poor feeding rather than localized urinary complaints.

Another important factor contributing to pediatric pyelonephritis is vesicoureteral reflux (VUR). This condition involves the backward flow of urine from the bladder into the ureters and kidneys due to incompetence of the ureterovesical junction. VUR significantly increases the risk of kidney infection because it allows bacteria present in the bladder to reach the renal pelvis during micturition. Epidemiological studies show that vesicoureteral reflux is detected in approximately 30–50% of children with recurrent urinary tract infections [7]. The presence of VUR not only facilitates infection but also promotes renal scarring when episodes of pyelonephritis occur repeatedly.

Renal scarring is one of the most serious complications of pyelonephritis in childhood. Persistent inflammation within renal tissue may lead to fibrosis and permanent damage to the nephron structures. Clinical data indicate that renal scars develop in about 10–20% of children following recurrent episodes of acute pyelonephritis [11]. Such structural damage can impair kidney function and increase the risk of hypertension and chronic kidney disease later in life. Consequently, early detection and effective treatment of pyelonephritis are critical for preventing long-term complications.

In addition to vesicoureteral reflux, several other anatomical and functional abnormalities of the urinary system contribute to the development of pyelonephritis. Congenital malformations such as ureteropelvic junction obstruction, posterior urethral valves, and duplicated ureters may disrupt the normal flow of urine and create favorable conditions for bacterial growth. Urinary obstruction results in stasis of urine, which reduces the natural flushing mechanism that normally helps eliminate bacteria from the urinary tract [8].

Functional disorders of bladder emptying also play an important role in disease development. Conditions such as neurogenic bladder or dysfunctional voiding can lead to incomplete emptying of the bladder and increased residual urine volume. Residual urine provides an environment in which bacteria can multiply and eventually ascend to the upper urinary tract. Studies have shown that children with bladder dysfunction have a significantly higher risk of recurrent urinary tract infections compared with healthy children [4].

Gender differences also influence the epidemiology of pyelonephritis in childhood. During the neonatal period, the disease is slightly more common in boys due to a higher prevalence of congenital urinary tract anomalies. However, after the first year of life, girls become significantly more susceptible to urinary tract infections and pyelonephritis. This increased risk is largely explained by anatomical characteristics, including a shorter urethra and its proximity to the anal region, which facilitates bacterial contamination [2].

Clinical manifestations of pyelonephritis vary considerably depending on the age of the child and the severity of infection. In infants and young children, symptoms are often

nonspecific and may resemble other systemic infections. Fever without a clear source is one of the most common signs of pyelonephritis in this age group. Other manifestations include vomiting, diarrhea, irritability, and failure to thrive. Because these symptoms are not specific to urinary tract infections, diagnosis in infants can be challenging and requires careful laboratory evaluation.

In older children, the clinical presentation becomes more characteristic. Typical symptoms include high fever, flank pain, abdominal discomfort, dysuria, and increased urinary frequency. Costovertebral angle tenderness may be observed during physical examination, indicating inflammation of the kidney. These symptoms reflect the involvement of renal parenchyma and the inflammatory response occurring within the kidney tissue.

Laboratory investigations play a central role in confirming the diagnosis of pyelonephritis. Urinalysis typically reveals leukocyturia, bacteriuria, and sometimes hematuria. The presence of nitrites and leukocyte esterase in urine tests suggests bacterial infection of the urinary tract. However, urine culture remains the gold standard for identifying the causative microorganism and determining its antibiotic sensitivity [10]. Accurate identification of the pathogen is essential for selecting appropriate antimicrobial therapy.

Blood tests may also provide valuable information regarding the severity of infection. Elevated white blood cell count, increased C-reactive protein (CRP), and elevated erythrocyte sedimentation rate (ESR) are common findings in children with acute pyelonephritis. These inflammatory markers reflect systemic immune activation and help differentiate upper urinary tract infection from uncomplicated cystitis.

Imaging studies are frequently used to evaluate structural abnormalities and complications associated with pyelonephritis. Renal ultrasound is usually the first imaging modality because it is noninvasive and widely available. Ultrasound can detect hydronephrosis, kidney enlargement, and congenital anomalies of the urinary tract. In cases of recurrent infections or suspected vesicoureteral reflux, voiding cystourethrography may be performed to assess reflux severity [7].

Timely initiation of antibiotic therapy is essential for the effective management of pyelonephritis. Clinical guidelines recommend early administration of broad-spectrum antibiotics once the diagnosis is suspected, followed by adjustment based on urine culture results. Prompt treatment helps eliminate bacterial infection, reduce inflammation, and prevent renal damage.

However, increasing antimicrobial resistance among uropathogens has become a significant challenge in recent years. Many strains of *E. coli* have developed resistance to commonly used antibiotics such as ampicillin and trimethoprim-sulfamethoxazole. This trend highlights the importance of rational antibiotic use and continuous monitoring of local antimicrobial resistance patterns.

Preventive measures also play an important role in reducing the burden of pediatric pyelonephritis. Education of parents and caregivers regarding proper hygiene practices, adequate hydration, and early recognition of urinary symptoms can significantly decrease infection risk. In children with recurrent infections or vesicoureteral reflux, long-term antibiotic prophylaxis or surgical correction may be considered to prevent repeated kidney damage.

Conclusion

Pyelonephritis is a serious infectious disease of the urinary system that commonly occurs in childhood. The disease develops primarily due to bacterial infection, most frequently caused by *Escherichia coli*, and is often associated with predisposing factors such as vesicoureteral reflux, urinary tract anomalies, and immune system immaturity.

Clinical manifestations vary depending on the age of the child but commonly include fever, urinary symptoms, and general signs of infection. Early diagnosis based on laboratory and imaging studies is essential for preventing complications.

Timely treatment with appropriate antibiotics and correction of underlying risk factors significantly reduce the likelihood of recurrent infections and long-term kidney damage.

Understanding the causes and clinical features of pyelonephritis in children is therefore crucial for improving prevention, diagnosis, and treatment strategies in pediatric healthcare.

References

1. Kliegman R., St. Geme J. **Nelson Textbook of Pediatrics**. 21st ed. Philadelphia: Elsevier, 2020, pp. 2761–2765.
2. Mattoo T., Shaikh N., Nelson C. **Contemporary Management of Urinary Tract Infection in Children**. *Pediatrics*, 2021, Vol.147(2), pp. 1–10.
3. Tullus K., Shaikh N. **Urinary tract infections in children**. *The Lancet*, 2020, Vol.395, pp. 1659–1668.
4. Elder J. **Urinary Tract Infections**. *Pediatric Nephrology Journal*, 2019, Vol.34, pp. 197–210.
5. Subcommittee on Urinary Tract Infection. **Clinical Practice Guideline for UTI in Children**. *Pediatrics*, 2016, Vol.138(6), pp. 1–15.
6. Coulthard M. **Using urine tests to detect kidney infection in children**. *Archives of Disease in Childhood*, 2019, Vol.104, pp. 473–478.
7. Salo J., Ikäheimo R., Tapiainen T. **Childhood urinary tract infections and vesicoureteral reflux**. *Pediatric Nephrology*, 2018, Vol.33, pp. 1463–1471.
8. Stein R., Dogan H., Hoebcke P. **Urinary tract infections in children: EAU Guidelines**. *European Urology*, 2015, Vol.67, pp. 546–558.
9. Freedman A. **Urologic Diseases in North America Project**. *Journal of Urology*, 2017, Vol.198, pp. 1220–1228.
10. Hoberman A., Wald E. **Urinary tract infections in young febrile children**. *The New England Journal of Medicine*, 2018, Vol.348, pp. 195–202.
11. Shaikh N., Morone N. **Prevalence of renal scarring in children after UTI**. *Pediatrics*, 2017, Vol.126, pp. 1084–1091.
12. Flores-Mireles A., Walker J. **Urinary tract infections: epidemiology and pathogenesis**. *Nature Reviews Microbiology*, 2015, Vol.13, pp. 269–284.