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Gut Microbiota Features in Young Children with Acute Pyelonephritis Depending on The Type of Feeding

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Abstract

Background. The gut is a major reservoir of uropathogens, and its microbiota during the first years of life is substantially influenced by feeding type. This relationship has been insufficiently studied in young children with acute pyelonephritis.

Objective. To compare gut microbiota parameters in children aged 0–36 months with acute pyelonephritis who received breastfeeding or formula feeding.

Materials and methods. The study included 147 children: 57 breastfed patients with acute pyelonephritis (group I), 55 patients formula-fed from birth (group II), and 35 healthy breastfed children (group III). Fecal samples were examined before antibacterial therapy by real-time polymerase chain reaction using the Enteroflor Deti reagent kit.

Results. Group II had the lowest total bacterial mass (8.46±0.13 lg GE/g), proportion of normal microbiota (85.80±1.33%), taxonomic diversity, and levels of Bifidobacterium spp., Lactobacillaceae, the Clostridium leptum group, and Akkermansia muciniphila ($p \leq 0.05$ compared with group I). The proportion of opportunistic microbiota was

10.66±2.01%, versus 5.98±1.00% in group I and 3.61±0.19% in healthy children. Enterobacterales and Escherichia coli levels were higher in both patient groups than in controls.

Conclusion. In young children with acute pyelonephritis, formula feeding was associated with more pronounced dysbiotic shifts, whereas the microbiota composition of breastfed patients was closer to that of healthy children. The cross-sectional design does not allow causal inference.

Keywords: Acute pyelonephritis, young children, breastfeeding, formula feeding, gut microbiota, dysbiosis, Bifidobacterium, Akkermansia muciniphila, Enterobacterales, polymerase chain reaction.

1. Introduction

Acute pyelonephritis is a clinically important urinary tract infection in childhood. In infants and young children, it may lead to renal parenchymal damage, scarring, and later renal complications. The intestine is a possible reservoir for uropathogens: bacteria may colonize the periurethral region and then enter the urinary tract by an ascending route. For this reason, the gut microbiota is relevant when considering the development of urinary tract infections [1, 2].

During the first years of life, microbiota development is influenced by delivery mode, antibiotic exposure, living conditions, and particularly feeding type [3–5]. Human milk oligosaccharides selectively support bifidobacteria, including Bifidobacterium longum subsp. infantis [6]. With formula feeding, the microbiota may shift earlier toward an adult-like profile, with a possible reduction in protective symbionts and an increase in opportunistic bacteria [3, 4]. These changes may affect the intestinal barrier and colonization resistance.

Previous studies of pediatric urinary tract infection have often examined the gut microbiota and urinary microbiota separately. Less is known about how feeding type relates to protective and opportunistic gut microorganisms in children aged 0–36 months who have acute pyelonephritis [7].

The aim of this study was to compare gut microbiota parameters in children aged 0–36 months with acute pyelonephritis according to breastfeeding and formula feeding.

2. Methods

Between 2024 and 2026, we conducted a cross-sectional observational study with prospective recruitment at the pediatric nephrology department of the Tashkent State Medical University Clinic and Family Polyclinic No. 65 in Yangihayot district, Tashkent. The study included 147 children aged 0–36 months: 112 with a first episode of acute pyelonephritis confirmed clinically and by laboratory findings, and 35 healthy children who served as controls.

Group I included 57 patients who had received exclusive breast milk during the first 6 months of life and continued breastfeeding for at least 24 months. Group II comprised 55 patients who had been formula-fed from birth. Group III included 35 healthy children fed similarly to those in group I. Mean ages were 19.32±1.30, 17.95±1.33, and 17.06±1.94 months, respectively; the main groups did not differ significantly in age ($p>0.05$).

Inclusion criteria for the patient groups were age 0–36 months; confirmed acute pyelonephritis; absence of other acute infections and chronic diseases of the urinary, central nervous, digestive, or cardiovascular systems; no antibiotic use before examination; and written consent from a parent or legal representative. Controls were children without chronic diseases or functional abnormalities. The exclusion criterion was withdrawal of parental consent.

Feces were collected on hospital admission, before antibacterial therapy, after spontaneous defecation into a sterile disposable container, and delivered to the laboratory within 2 hours. If immediate delivery was not possible, the sample was refrigerated. Microbiota were examined by real-time polymerase chain reaction using the Enteroflor Deti reagent kit (DNA-Technology LLC, Moscow, Russia). Total bacterial mass, the proportion of normal microbiota, taxonomic diversity, major symbionts, butyrate-producing bacteria, and opportunistic microorganisms were assessed. Results were expressed in lg genome equivalents per gram (lg GE/g) and percentages.

Statistical analysis was performed using MS Excel 2019. Data are presented as the arithmetic mean and its error (M±m). Student's t-test was used; the Mann–Whitney U test was applied when individual variability was high, and Pearson's χ^2 test was used for categorical variables. Differences were considered statistically significant at $p\leq0.05$.

The study was conducted in accordance with the legislation of the Republic of Uzbekistan on the protection of citizens' health and the bioethical requirements of the Ministry of Health. The manuscript source indicates that ethics committee approval details (institution, protocol number, and date) should be supplied if applicable. Written informed consent was obtained from parents or legal representatives of all children.

3. Results

Total bacterial mass exceeded the reference boundary in all groups; however, it was lower in formula-fed patients than in group I and controls ($p \leq 0.05$). The proportion of normal microbiota and taxonomic diversity were also lowest in group II (Table 1).

Table 1. Integral parameters of the gut microbiota

Parameter	Group I: AP, BF (n=57)	Group II: AP, FF (n=55)	Group III: healthy (n=35)	P
Total bacterial mass, lg GE/g	9.11±0.11	8.46±0.13	9.27±0.56	≤0.05
Normal microbiota, %	92.31±1.25	85.80±1.33	95.03±0.89	≤0.05
Taxonomic diversity	10.34±0.37	9.33±0.36	11.00±0.51	≤0.05
Bifidobacterium spp., lg GE/g	7.54±0.12	6.25±0.22	8.10±0.19	≤0.05
Metabolically active bifidobacteria, %	12.61±0.53	10.36±0.52	16.71±0.83	≤0.05
Lactobacillaceae, lg GE/g	5.15±0.26	4.08±0.22	6.12±0.29	≤0.05
Firmicutes/Bacteroidetes ratio	1.89±0.10	1.51±0.08	2.18±0.06	≤0.05
Opportunistic microbiota, %	5.98±1.00	10.66±2.01	3.61±0.19	≤0.05
Candida spp., lg GE/g	4.83±0.17	4.97±0.12	4.19±0.20	>0.05

Note. Data are presented as M±m; lg GE/g = decimal logarithm of genome equivalents per gram of feces; AP = acute pyelonephritis; BF = breastfeeding; FF = formula feeding. P indicates the comparison between groups I and II; differences from controls are described in the text.

Bifidobacterium spp. levels and the proportion of metabolically active bifidobacteria were lowest in group II; the latter was 1.6-fold lower than in controls. Levels of metabolically active "infant-type" species in formula-fed patients were 5.61±0.25 lg GE/g, compared with 6.48±0.08 and 6.50±0.09 lg GE/g in groups I and III, respectively.

B. longum subsp. infantis levels in groups I, II, and III were 6.23±0.26, 5.14±0.22, and 6.80±0.15 lg GE/g, respectively; B. longum subsp. longum levels were 6.20±0.19, 4.89±0.40, and 8.15±0.19 lg GE/g. Levels of B. bifidum and B. breve in group II (5.99±0.19 and 5.05±0.21 lg GE/g) were significantly lower than in controls ($p \leq 0.05$). Conversely, metabolically active "adult-type" species increased in group II to 6.37±0.22 lg GE/g, accounting for 31.40±3.78%.

Among butyrate-producing bacteria, the Clostridium leptum group was significantly lower in group II than in group I and controls. Faecalibacterium prausnitzii and Lachnospiraceae levels were lower in both patient groups than in controls, but did not differ significantly between groups I and II (Table 2).

Table 2. Protective symbionts and potential pathobionts

Microorganism	Group I: AP, BF (n=57)	Group II: AP, FF (n=55)	Group III: healthy (n=35)	P
Clostridium leptum group, lg GE/g	8.01±0.15	7.01±0.23	8.23±0.17	≤0.05
Faecalibacterium prausnitzii, lg GE/g	5.95±0.26	5.39±0.29	6.22±0.30	>0.05
Lachnospiraceae, lg GE/g	7.57±0.15	7.12±0.24	8.13±0.19	>0.05
Alistipes spp., %	1.11±0.12	2.13±0.23	1.21±0.07	≤0.05

Bacteroides spp., lg GE/g	7.14±0.31	7.03±0.29	7.22±0.23	>0.05
Akkermansia muciniphila, lg GE/g	3.73±0.39	2.39±0.57	3.95±0.48	≤0.05
Desulfovibrio spp., lg GE/g	3.86±0.32	4.08±0.21	3.51±0.30	>0.05
Enterobacterales, lg GE/g	7.09±0.32	7.47±0.36	6.05±0.24	—
Escherichia coli, lg GE/g	6.25±0.12	6.51±0.13	5.89±0.13	—

Note. Abbreviations are as in Table 1.

Akkermansia muciniphila levels were lowest in group II, whereas the proportion of Alistipes spp. was highest ($p \leq 0.05$). Differences in Desulfovibrio spp. and Bacteroides spp. were not statistically significant.

The proportion of opportunistic microbiota increased from the control group to group I and then to group II. Enterobacterales and Escherichia coli levels were higher in both patient groups than in healthy children ($p \leq 0.05$). Candida spp. levels were also higher in patients than in controls, but did not differ between groups I and II ($p > 0.05$).

4. Discussion

The gut microbiota of children aged 0–36 months with acute pyelonephritis differed from that of healthy peers, with more pronounced differences among formula-fed patients. Group II showed a lower proportion of normal microbiota and lower taxonomic diversity, while the proportion of opportunistic microorganisms was almost three times that in controls. The Firmicutes/Bacteroidetes ratio decreased to 1.51±0.08 and approached the lower reference boundary. This ratio should not be considered a diagnostic criterion on its own; together with other indicators, it characterizes a shift in microbiota composition.

The reduction in Bifidobacterium spp., B. longum subsp. infantis, B. longum subsp. longum, B. bifidum, and B. breve—most marked in formula-fed children—is consistent with evidence that feeding type influences bifidobacterial development in early life. Human milk oligosaccharides provide selective substrates for B. infantis and other infant-associated bifidobacteria [3, 6]. This may explain why, during acute infection, bifidobacterial levels in breastfed children, although lower than in healthy children, were better preserved than in formula-fed patients.

Reduced levels of the Clostridium leptum group, F. prausnitzii, Lachnospiraceae, and Lactobacillaceae indicate depletion of microbiota components involved in intestinal epithelial trophism and colonization resistance. Since F. prausnitzii and Lachnospiraceae did not differ significantly between groups I and II, their reduction cannot be attributed solely to feeding type. The acute infectious-inflammatory process, feeding characteristics, and other early-life factors may all contribute.

The markedly lower level of Akkermansia muciniphila in group II reflects altered microecological conditions in the intestinal mucus layer. In this group, higher Enterobacterales, E. coli, and Alistipes proportions co-occurred with depletion of protective flora. The role of Alistipes is complex: both protective properties and associations with inflammation have been described [8]. The gut can serve as a reservoir of uropathogens, and E. coli may colonize the periurethral area and ascend into the urinary tract [1, 2, 7]. However, genomic matching of fecal and urinary isolates was not performed in this study; therefore, it cannot be claimed that fecal E. coli was the strain that caused pyelonephritis.

Candida spp. levels were higher in children with acute pyelonephritis than in healthy children, but the difference between the two patient groups was not statistically significant. Thus, an independent effect of feeding type on this parameter was not confirmed. Differences in Bacteroides spp. and Desulfovibrio spp. were also not statistically significant.

These results suggest that formula-fed young children with acute pyelonephritis may be more likely to show intestinal dysbiosis. Stool microbiota testing, however, is not a substitute for antibacterial treatment, and PCR findings alone should not guide clinical decisions. They need to be considered together with the child's clinical condition, urine culture results, and inflammatory markers.

The study has several limitations. Microbiota were assessed at a single time point, before antibacterial treatment, so the findings cannot establish a causal relationship. Feeding groups were defined from the children's histories, and other factors—such as delivery mode, diet, previous infections, and living conditions—may have influenced the results. Further studies with follow-up measurements, genetic comparison of E. coli isolates from urine and stool, and evaluation of microbiota changes alongside the clinical course are needed.

5. Conclusion

In children aged 0–36 months with acute pyelonephritis, the gut microbiota showed reduced levels of normal flora and protective symbionts and increased levels of opportunistic microorganisms. Dysbiotic changes were more pronounced with formula feeding: these patients had the lowest total bacterial mass, proportion of normal microbiota, taxonomic diversity, and levels of Bifidobacterium spp., Lactobacillaceae, the Clostridium leptum group, and Akkermansia muciniphila, while opportunistic microbiota, Enterobacterales, E. coli, and Alistipes indicators were highest. In breastfed children with acute pyelonephritis, the microbiota profile remained closer to that of healthy children. The findings indicate an association between feeding type and gut microbiota status; longitudinal, multivariable studies are needed to confirm causality.

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