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COURSE OF DYSENTERY WITH PATHOGENIC FUNGI AND TREATMENT APPROACHES IN CHILDREN

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Abstract: The dynamics of the pathogen's biological properties, infection conditions, and the biological state of the host organism determine the constant evolution of clinical manifestations and outcomes of infectious diseases (Pokrovsky V.I. et al., 2003). The previously held view of dysentery as a disease with a mild course and decreasing lethality, prevalent by the mid-1980s, was revised due to a sharp worsening during the epidemic spread of Shigella flexneri 2a in the 1990s (Solodovnikov Yu.P., 1992; Kartsev A.D., 1992; Pokrovsky V.I., Yushchuk N.D., 1994; Belyaeva T.V., 1995, 2003; Kondrasheva L.N. et al., 2003). Literature analysis indicates that Shigella dysentery caused by Flexner bacilli in recent years has been marked by an increased proportion of severe cases and unfavorable outcomes (Brodov L.E. et al., 1996; Rakhmanova A.G. et al., 1996).

Keywords: Shigellosis in children, Flexner dysentery, Candida infection, intestinal microbiota, dysbiosis, antibiotic-associated diarrhea (AAD), pathogenic fungi, antimicrobial resistance, coprological analysis, immunological response

Dysentery (shigellosis) is an acute intestinal infection caused by bacteria of the genus Shigella, transmitted via the fecal-oral route. It manifests as anatomical and functional damage to the large intestine and systemic intoxication, sometimes with primary neurotoxic syndrome. According to estimates, around 140 million shigellosis cases occur globally each year. Susceptibility to the infection varies by age group, with children under 2–3 years being most vulnerable. One factor determining the severity, duration, outcome, and pathogen clearance is the state of the intestinal microbiocenosis (Nisevich N.I.; Mazankova L.N.). The intestinal flora forms during the early days of life and includes anaerobes (bifidobacteria, lactobacilli, bacteroides), aerobes (E. coli – indigenous flora), facultative flora (staphylococci, fungi), and transient (allochthonous) flora (Klebsiella, Clostridia, etc.) (Bondarenko V.M.; Parfenov A.I.). This flora comprises 1–4% of the intestinal microbial biomass (N.I. Nisevich; V.F. Uchaikin; D.J. Hentges; G.W. Elmer; F. Bäckhed).

The structure, prevalence, and dynamics of dysentery among children in Uzbekistan in recent years show the persistent circulation of dysentery and outline its modern clinical features. Pathogen sensitivity patterns reveal that Shigella flexneri 2a and Shigella sonnei are highly susceptible to 3rd and 4th generation cephalosporins, ciprofloxacin, and amikacin, but resistant to kanamycin, gentamicin, tetracycline, chloramphenicol, and ampicillin.

Comprehensive clinical-immunological studies of invasive intestinal infections in children revealed early predictors of disease severity and outcomes. Laboratory diagnosis prioritizes bacteriological analysis. Fecal sampling is advised before initiating antibiotics, ideally bedside, with delivery to the lab within 2 hours. Results may be negative by day 3–5 or positive by day 5–7 post-sampling.

Modern treatment standards have encouraged widespread use of antibacterial agents, which raises the problem of antibiotic-associated diarrhea (AAD). AAD is defined as three or more episodes of loose stool over two or more days during or after antibiotic therapy (1). Most antibiotic classes reduce beneficial gut flora and increase pathogenic flora. Dysbiosis may persist and become pathogenic (Seredina E.Yu., 2002; Rozhanets A.N., 2002; Dmitrachkov V.V., 2000; Gorelov A.V., 2005). Candida, Klebsiella, Enterobacter, and resistant strains often increase after antibiotics (Kharchenko G.A., 1997).

Objective: To optimize comprehensive therapy for shigellosis in children based on modern clinical characteristics, regional pathogen profiles, and immune response patterns.

Research Objectives:

1. To study the clinical features and structure of invasive dysentery over the past decade in children of different ages in Uzbekistan.

2. To analyze regional Shigella strains in dysentery cases complicated by fungal infection, with attention to antibiotic resistance.

3. To evaluate cellular and humoral immunity parameters by age, severity, and clinical outcome.

4. To investigate coprological and bacteriological parameters in children with fungalassociated dysentery.

5. To analyze the clinical course of Candida co-infection in Shigella flexneri 2a dysentery.

Materials and Methods:

40 children aged 1–14 were studied: 20 with acute dysentery hospitalized in Infectious Disease Hospital No. 4, Fergana, and 20 with Candida-associated dysentery (control). Diagnosis was confirmed by bacteriological culture (mostly Shigella flexneri). Based on disease severity, they were divided into two groups: 10 with Flexner dysentery, and 10 with fungal dysentery. Acute dysentery showed signs of intoxication and colitis in 80.7%, with hemorrhagic colitis in 70.2%. Patients received traditional treatment including etiological, pathogenetic, and symptomatic therapies.

Results and Discussion:

Monooxygenase system activity correlated with disease severity (see Table 1). Amidopyrine demethylation decreased significantly in patients, indicating impaired detoxification. Post-treatment, recovery of biotransformation was noted only in mild cases, with acetylation levels still below normal. The monooxygenase system showed no full restoration even after treatment. This indicates an ongoing deficit in xenobiotic biotransformation, limiting chemical homeostasis in recovering children.

Conclusions:

1. Dysentery with Candida in children suppresses intestinal flora function, especially evident in dysbiosis severity correlating with disease severity.

2. Standard therapy improves gut microbiota, yet flora remains below normal during recovery. Restoration lags behind Candida reduction, disrupting microbial balance.

3. Targeted pathogenetically justified therapy is needed to reduce toxic load during fungal and bacterial dysentery.

Gastrointestinal involvement in shigellosis was marked by severe colitis (abdominal pain, tenesmus, spastic sigmoid colon, loose stools with mucus and blood). Treatment includes bed rest, rehydration, dietary management, pathogenetic therapy (sorbents, probiotics, prebiotics, enzymes), etiotropic therapy (antimicrobials, oral immunoglobulins), and symptomatic agents (antipyretics, spasmolytics, hemostatics).

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