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Clinical and Microbiological Characteristics of Intestinal Microbiocenosis Disorders In HIV-Positive Children with Acute Diarrhea

Muminova Makhbuba Teshaevna

Associate Professor of the Department of Infectious and Pediatric Infectious Diseases, Candidate of Medical Sciences, Uzbekistan

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Abstract: Acute infectious diarrhea (AID) is a frequent and serious complication in children living with HIV. This study aims to investigate the composition and severity of intestinal microbiota disruption (dysbiosis) in HIV-positive children with AID compared to HIV-negative peers. A total of 499 children aged 7–18 years were included. Dysbiosis was classified into four stages, and correlations with clinical parameters, immunodeficiency levels, and viral loads were assessed. Our results show that advanced dysbiosis (stages III–IV) is significantly more prevalent in HIV-infected children, particularly those with high viral loads and severe immunodeficiency. Early detection and correction of dysbiosis are essential to prevent further complications and support immune recovery.

Keywords: HIV infection, children, acute infectious diarrhea, intestinal microbiota, dysbiosis.

1.Introduction:

Relevance of the Problem. The data obtained in this study indicate that acute infectious diarrhea in HIV-infected children is accompanied by the development of deeper disorders of the intestinal microbiota compared to HIV-infected children without diarrheal syndrome. In particular, grade IV intestinal dysbiosis was registered in 26.4% of children in the study group, which is 2.4 times higher than in the comparison group [15].

Such results are consistent with the data of a number of foreign and domestic studies indicating that HIV infection is characterized by early and progressive damage to the intestinal immune system, primarily lymphoid tissue associated with the intestinal mucosa (GALT), which leads to a violation of the barrier function and a change in the composition of the microbiota [16, 22]. According to Brenchley et al., the destruction of the intestinal epithelium and the depletion of CD4+ T-lymphocytes in the intestinal mucosa in HIV infection contribute to bacterial translocation and the formation of pronounced dysbiosis, which persists even against the background of antiretroviral therapy [15]. Similar findings are presented in the reviews by Vujkovic-Cvijin et al., which showed a decrease in microbial diversity and an increase in the proportion of opportunistic microorganisms in HIV-infected patients [23].

A number of studies on children with HIV infection indicate that the presence of diarrheal syndrome significantly exacerbates changes in the gut microbiota. Thus, according to Montecino-Rodriguez et al., infectious diarrhea in HIV-infected children is associated with a pronounced suppression of obligate microflora and the rapid formation of severe forms of dysbiosis [12]. This is consistent with our results demonstrating a higher frequency of III–IV degrees of dysbiosis with the addition of acute infectious diarrhea.

Particular attention in the literature is paid to the relationship between the severity of dysbiosis and the stage of HIV infection. According to the WHO and a number of clinical studies, the progression of HIV infection, an increase in immunodeficiency, and a high viral load correlate with more pronounced microbiota disorders and an increased incidence of severe gastrointestinal complications [26, 27, 28]. Our study also found that in children with advanced clinical stages of HIV infection, moderate and severe degrees of immunodeficiency with the development of acute infectious diarrhea, severe degrees of intestinal dysbiosis were significantly more likely to be detected [15, 17].

Thus, the results obtained confirm the literature data that acute infectious diarrhea is an important additional pathogenetic factor exacerbating HIV-associated disorders of the intestinal microbiota. This emphasizes the need for early diagnosis and comprehensive correction of intestinal dysbiosis in HIV-infected children, especially in the presence of diarrheal syndrome and signs of severe immunodeficiency [14, 21, 25].

Objective: To study the state of the intestinal microbiota in HIV-infected children with acute infectious diarrhea.

2. Methods

The study was conducted from 2020 to 2025 on the basis of a specialized infectious diseases clinic at the Republican AIDS Center, in the HIV Infection Department of the Research Institute of Virology at the Scientific and Practical Medical Center for Epidemiology, Microbiology, Infectious and Parasitic Diseases of the Ministry of Health of the Republic of Uzbekistan.

The study included data from 499 HIV-infected children aged 7 to 18 years with acute infectious diarrhea. The children were divided into three groups: the main group was 261 HIV-infected children with acute diarrhea of infectious etiology, the 1st comparison group was 247 HIV-uninfected children with infectious diarrhea, and the 2nd comparison group was 238 children without acute diarrhea, but with HIV infection. The study included the analysis of the clinical manifestations of HIV infection, the determination of the immunological status and virological profile of each patient.

The diagnosis of HIV infection was established on the basis of the Order of the Ministry of Health of the Republic of Uzbekistan No 270 dated 08/19/2023 "On protocols for the prevention and treatment of human immunodeficiency viral infection".

The diagnosis of "Acute infectious diarrhea" (ACD) was established on the basis of the order of the Ministry of Health of the Republic of Uzbekistan No 122 dated 25.03.2015 "On improving measures taken among the population of the republic against typhoid fever, paratyphoid, salmonellosis and acute intestinal diseases".

Distribution of patients into groups.

	Main group n=261		2nd comparison group n=238	
	Abs.	%	Abs.	%
Clinical stages of HIV infection				

Clinical stage II	105	40,2	95	39,9
Clinical stage III	123	47,1	114	47,9
Clinical stage IV	33	12,6	29	12,1
Degree of CD4+ Cell Immunodeficiency				
No immunodeficiency of more than 500 cells	160	61,3	148	62,2
Mild immunodeficiency	75	28,7	69	29
Moderate immunodeficiency of 350-499 cells	20	7,7	16	6,7
Severe immunodeficiency of less than 200 cells	6	2,3	5	2,1
Viral load 1 µL				
more than 100000 copies	8	3,0	7	2,9
10000-100000 copies	13	5,0	12	5,0
1000-10000 copies	57	21,8	53	22,3
less than 1000 copies	183	70,1	166	69,7

The WHO also distinguishes four clinical stages of HIV, regardless of CD4:

Stage I. Asymptomatic or persistent generalized lymphadenopathy.

Stage II. Moderate clinical manifestations: weight loss <10%, skin diseases, recurrent upper respiratory tract infections.

Stage III. Weight loss >10%, chronic diarrhea, prolonged fever, pulmonary tuberculosis, oral candidiasis.

Stage IV (AIDS). Severe opportunistic infections, oncological diseases; end-stage immunodeficiency.

The degree of immunodeficiency was assessed by quantifying the level of $CD4^+$-T lymphocytes in the peripheral blood. The study was carried out by flow cytometry using standardized reagents. $CD4^+$ values were interpreted according to the WHO classification:

1. ≥500 cells/µL — no immunodeficiency;
2. 350–499 cells/µL — mild immunodeficiency;
3. 200–349 cells/µL — moderate immunodeficiency;
4. <200 cells/µL is severely immunodeficient.

Viral load was determined by real-time PCR (RT-PCR) with quantitative detection of HIV RNA copies in 1 µl of blood plasma. Viral load was quantified using an automated real-time PCR platform. Certified diagnostic kits (Roche COBAS AmpliPrep/COBAS TaqMan, Abbott RealTime HIV-1 or similar) with high analytical sensitivity were used as follows:

1. Low viral activity – <1,000 copies/µl;
2. Moderate viral activity – 1,000–10,000 copies/µl;
3. High viremia – 10,000–100,000 copies/µL;
4. Severe viral replication – 100,000 copies/µl.

In the study, the genes of bifidobacteria, lactobacilli, typical and lactose-negative Escherichia coli, opportunistic and pathogenic enterobacteriaceae, non-fermenting gram-negative bacteria, staphylococci, hemolytic forms of microorganisms, yeast-like fungi, enterococci were isolated. Identification of anaerobic microorganisms, opportunistic and pathogenic enterobacteria was carried out using the VITEK 2 device Compact using ID cards: ANC (244147910), GN (bioMerieux France). Stool examination included the bacteriological method and polymerase chain reaction (PCR) with the determination of nucleic acids of Campylobacter spp., Salmonella spp., Shigella spp., Escherichia coli, Y. pseudotuberculosis, Y. enterocolitica, Norovirus, Adenovirus, Astrovirus, Rotavirus. The study was carried out using Xpect Clostridium difficile Toxin A/B (Oxoid) test systems.

Statistical data processing was carried out using parametric and non-parametric statistical methods using Excel computer programs. The significance of the differences was determined at $P<0.05$ by calculating the chi-squared test and the error of match.

3. Results and discussion

Information is presented on the presence of viruses and bacteria in the etiological structure of acute diarrhea in children with HIV infection, as well as on their presence

in the form of mono- and mixed infection. A study of the degree of overlap of viral and bacterial agents in the overall pattern of diarrhea in HIV-infected children showed that viruses were found in 51.7% (135) of 261 children aged 7 to 18 years with acute diarrhea, and bacteria were found in 48.3% (126); in the control group, viruses were detected in 60.3% (149), bacteria in 39.7% (98 people).In clinical practice, intestinal dysbiosis is classified according to the degree of severity of microbiota disorders.

It is customary to distinguish four degrees that characterize the depth of quantitative and qualitative changes in the composition of the intestinal microflora.

Stage I (mild disorders of microbiota). There is a moderate decrease in the population of obligate microorganisms — bifidobacteria and lactobacilli. Quantitative indicators of opportunistic pathogenic flora remain within the normal range or are slightly increased. Clinical manifestations, as a rule, are absent or minimally expressed.

Stage II (moderate changes in microflora). It is characterized by a more pronounced decrease in the levels of protective microflora with the simultaneous growth of individual representatives of opportunistic pathogens (Proteus spp., Klebsiella spp., Staphylococcus spp., etc.). Functional intestinal disorders are possible: flatulence, stool instability, abdominal discomfort.

Stage III (pronounced disorders of microbiota). There is a significant suppression of obligate microflora and a significant increase in the concentration of opportunistic bacteria. Pronounced clinical symptoms are observed: diarrheal or constipation syndrome, abdominal pain, signs of inflammatory changes in the mucous membrane.

Stage IV (severe dysbiosis). It is characterized by a sharp decrease in the quantitative indicators of obligate bacteria and a massive increase in pathogenic and opportunistic flora. Toxico-infectious manifestations, pronounced inflammatory syndrome, impaired digestion and absorption processes are often revealed.

The initial stage of our study was the analysis of the degree of disorders of the intestinal microbiota in the examined children. That in children in the study group (HIV+ AID+), grade I intestinal dysbiosis was 2.6 times less common than in children in the first comparison group (HIV-AID+), and 4.1 times less often than in children in the second comparison group (HIV+AID-), while in children in the study group, grade II intestinal dysbiosis was 1.7 times less common. than in children in the first comparison group (OR=2.05; 95% CI 1.34–3.12; P<0.001) and 2.5 times less often than in children in the second comparison group (OR=3.60; 95% CI 1.34–3.12; P<0,001). Almost half of the children in the study group had grade III intestinal dysbiosis, which was 1.3 times less common in children in the first comparison group (OR=1.60; 95% CI 1.13–2.28; P<0.001) and 2 times less often in children in the second comparison group (OR=3.06; 95% CI 2.10–4.47; P<0,001). Stage IV intestinal dysbiosis developed in 26.4% of the children of the study group, while in the children of the 1st control group, this indicator developed 1.5 times less often (OR=1.70; 95% CI – 1.11–2.62; P<0.001), and in children of the 2nd control group – 2.4 times less often (RR – 2.93; 95% CI – 1.79–4.79; P<0,001).

In children with AID against the background of HIV infection (the main group), mild degrees of intestinal dysbiosis (I and II) are much less common than in the 1st comparison group. At the same time, severe degrees (III and IV) develop much more often, especially in comparison with the 1st comparative group. These results indicate that the coexistence of HIV and AIDs leads to the development of serious disorders of the intestinal microflora.

Intestinal dysbiosis in acute diarrhea in HIV-infected children has been studied according to the severity of diarrhea, the degree of dehydration, the number and duration of attacks.

In mild acute diarrhea, there were no statistically significant differences between the indicators of both groups for stage I and II intestinal dysbiosis (OR = 1.27; 95% CI 0.52–3.12; P>0.05 and RR=1.23; 95% CI – 0.49–3.05; P>0,05). The incidence of grade III intestinal dysbiosis in children of the study group was 4.3 times higher than in children of the 1st comparison group. (OR=4.91; 95% CI 0.84–28.65; P>0.05), but no statistically significant differences between these indicators were found. In the mild course of acute diarrhea in children of both groups, cases of intestinal dysbiosis of the IV degree were not detected.

In the moderate course of acute diarrhea in children of both groups, stage I intestinal dysbiosis was not observed. Grade II intestinal dysbiosis in moderate acute diarrhea in children of the study group was recorded 1.6 times less often compared to children of the 1st comparison group. (OR=1.82; 95% CI 1.10–3.0; P<0,05). In the children of the study group, in comparison with the children of the comparison group, in the moderate course of acute diarrhea, intestinal

dysbiosis of the III and IV degrees was recorded more often, but no statistically significant differences between these indicators were revealed. (OR=1.40; 95% CI 0.92–2.13; P>0.05 and OR=1.14; 95% CI 0.68–1.91; P>0,05).

In severe acute diarrhea in children of both groups, intestinal dysbiosis of I and II degrees was not observed; there were no statistically significant differences between grade III and IV intestinal dysbiosis (OR = 0.57; 95% CI 0.20–1.64; P>0.05 and OR=1.74; 95% CI 0.61–5.00; P>0,05).

According to the WHO, in adolescents, the degree of dehydration (in relation to body weight) is classified as follows: mild dehydration (thirst, dry mouth, decreased frequency of urination) - weight loss up to 5%; moderate dehydration (sunken eyes, dry skin, crying without tears, dizziness) – weight loss from 5% to 10%; severe dehydration (impaired consciousness, cold extremities, low blood pressure, convulsions) – weight loss of more than 10%.

In mild dehydration, grade I intestinal dysbiosis was 2.2 times less common in the children of the study group compared to the children in the comparison group (OR=2.70; 95% CI – 1.32–5.55; P<0.05), grade II — 1.3 times less often (OR=1.75; 95% CI – 1.00–3.09; P<0.05), while grade III intestinal dysbiosis was recorded 4.7 times more often (OR=6.79; 95% CI – 3.04–15.12; P<0,05). In both groups, no cases of grade IV intestinal dysbiosis were observed in children. In children with moderate dehydration, grade I intestinal dysbiosis was not detected in children of both groups, while grade II intestinal dysbiosis in the children of the study group developed 6.1 times less often compared to the children of the comparison group (OR=6.58; 95% CI – 1.43–30.29; P<0,05). In moderate dehydration, there were no statistically significant differences between the indicators of grade III and IV intestinal dysbiosis in both groups (OR=1.05; 95% CI – 0.64–1.71; P>0.05 VA OR=1.31; 95% CI – 0,79–2,17; P>0,05). In severe dehydration in children of both groups, the development of intestinal dysbiosis of I and II degrees was not noted. Grade III intestinal dysbiosis in the children of the study group was recorded 2 times less often compared to the children of the comparison group, while grade IV intestinal dysbiosis was recorded almost 3 times more often (OR=5.82; 95% CI – 1.32–25.56; P<0,05).

Further, we studied the dependence of the degree of intestinal microbiota disorders in HIV-infected children (with and without AID) on the clinical stage of HIV infection.

According to the data obtained, at the second clinical stage of HIV infection in children of the main group, stage I intestinal dysbiosis was recorded almost 3 times less often, and grade II intestinal dysbiosis was 1.7 times less common than in children of the comparison group. At the same time, advanced stages of intestinal dysbiosis (grades III and IV), on the contrary, were detected in children of the study group almost 3 times more often (P<0.05).

In children with stage III clinical HIV infection, stage I intestinal dysbiosis developed only in 2 (1.6%) children of the study group and in 19 (16.7%) children from the comparison group, the difference between the groups in indicators of stage II intestinal dysbiosis was 3.8 times (P<0.05). In stage III clinical HIV infection, stage III intestinal dysbiosis was recorded 1.9 times more often, and stage IV – 2.7 times more often in children of the study group compared to children in the comparison group (56.1% vs. 30.9% and 29.8% vs. 11.4% of cases, respectively, P<0.05).

At the IV clinical stage of HIV infection, intestinal dysbiosis of the first and second degrees was not observed in the children of the main group, only one child (3.4%) from the comparison group developed intestinal dysbiosis of the first degree, and 7 (24.1%) children developed intestinal dysbiosis of the second degree (P<0.05). There were no significant differences between the groups for grade III intestinal dysbiosis at this clinical stage of HIV infection (P>0.05). However, the significant difference between the groups for grade IV intestinal dysbiosis was 1.9 times (P<0.05).

The World Health Organization (WHO) distinguishes the degrees of immunodeficiency based on the level of $CD4^+$ lymphocytes. This classification is used to assess the depth of damage to the immune system in patients, including those with HIV infection.

In severe and moderate degrees of immunodeficiency, intestinal dysbiosis of I and II degrees was not recorded in children in both groups. Grade III intestinal dysbiosis in severe immunodeficiency developed 2.4 times less often, and grade IV, on the contrary, 3 times more often in children of the study group compared to children in the comparison group (33.3%; 80% and 66.7%; 20.0% of cases, respectively, P<0.05). There were no significant differences between the indicators of grade III intestinal dysbiosis in the comparison groups with moderate immunodeficiency (P>0.05), grade IV intestinal dysbiosis was recorded 1.8 times more often in the children of the study group compared to the children of the comparison group (P<0.05).

Although grade I intestinal dysbiosis with mild immunodeficiency in HIV infection was not observed in children of the study group, this indicator was detected only in 2 (2.9%) children from the comparison group. Grade II intestinal dysbiosis was detected in 2 (2.7%) children of the study group with mild immunodeficiency in HIV infection, and was significantly more often recorded in 1/3 of the children of the comparison group (P<0.05). There were no significant differences between the indicators of both comparison groups for grade III intestinal dysbiosis in mild immunodeficiency, the significant difference between grade IV was 2.1 times (P<0.05).

A comparative analysis of the indicators of intestinal dysbiosis in children with HIV infection in the absence of immunodeficiency (the main group) and in children without HIV infection (comparison group) revealed significant differences in the distribution of the degrees of intestinal microbiota (P<0.05). In patients of the main group, stage I dysbiosis was detected 3.9 times less often, stage II — almost 2 times less often, while stage III was observed 4.7 times more often, and stage IV — 3.2 times more often compared to the comparison group. The results obtained indicate the prevalence of severe forms of dysbiosis in HIV-infected children even with minimal manifestations of immunodeficiency.

In both groups with a pronounced viral load (more than 100,000 copies/μl), as well as with a high viral load (in the range from 10,000 to 100,000 copies/μl), dysbiosis of the I and II degrees was not detected. At the same time, there were no significant differences between the groups in the incidence of stage III and IV dysbiosis in children with a pronounced viral load (over 100,000 copies/ml, P>0.05). Children of the study group with a high viral load of HIV infection (ranging from 10,000 to 100,000 copies/μL) had a significantly higher incidence of grade IV intestinal dysbiosis compared to the comparison group (P<0.05). This indicates the similarity of the severity of intestinal microbiota disorders with a more pronounced level of viremia.

In children of the study group with a moderate viral load of HIV infection (in the range of 1000 to 10,000 copies/μL), grade I intestinal dysbiosis was not detected, while in the comparison group this degree of dysbiosis was recorded in one patient (1.8%). Grade II dysbiosis in the patients of the study group was 2.9 times less common compared to the comparison group, while grade IV, on the contrary, was almost 1.5 times more common (P<0.05). There were no significant differences in the incidence of grade III dysbiosis between the groups (P>0.05).

In children of the study group with a low viral load of HIV infection (less than 1000 copies), grade I intestinal dysbiosis was detected 4.5 times less often, and grade II intestinal dysbiosis was 2.4 times less common than in children of the comparison group (6.6%; 29.5% and 20.8%; 50% of cases, P<0.001), while grade III intestinal dysbiosis was observed 3.1 times more often, and grade IV intestinal dysbiosis was 4.8 times more common (49.7%; 15.7% and 23.0%; 4.8% of cases). P<0.001).

4. Discussion

The results of our study demonstrate that acute infectious diarrhea significantly aggravates disturbances in the intestinal microbiota of HIV-infected children. Children with diarrheal syndrome exhibited deeper degrees of dysbiosis, particularly grade IV dysbiosis in 26.4% of cases, which is 2.4 times higher than in HIV-infected children without diarrhea [15]. This finding aligns with previous research indicating that HIV infection is associated with early and progressive impairment of the intestinal immune system, particularly in gut-associated lymphoid tissue (GALT), leading to barrier dysfunction and altered microbiota composition [16, 22].

The observed depletion of CD4+ T-lymphocytes in the intestinal mucosa facilitates bacterial translocation and the persistence of dysbiosis, even under antiretroviral therapy [15]. Moreover, reductions in microbial diversity and increases in opportunistic microorganisms have been reported in both adult and pediatric HIV patients [23]. These changes can exacerbate systemic immune activation, promoting chronic inflammation and further weakening the host immune defense [18, 26].

Our findings support previous studies that have emphasized the impact of diarrheal episodes on gut microbiota in HIV-infected children. Montecino-Rodriguez et al. reported that infectious diarrhea leads to rapid suppression of obligate microflora and the development of severe dysbiotic forms [12]. This is consistent with our results showing a higher prevalence of severe dysbiosis in children with acute diarrhea.

The severity of intestinal dysbiosis was also closely associated with the stage of HIV infection and immunodeficiency. Children in advanced clinical stages, with moderate or severe immunodeficiency and high viral loads, were more likely to develop severe dysbiosis [15, 17, 27]. These findings underscore the interplay between immune status and gut microbiota composition, suggesting that HIV-induced immunodeficiency facilitates the progression of

dysbiosis, which in turn can exacerbate gastrointestinal complications.

The pathophysiological mechanisms underlying these changes involve both structural damage to the intestinal mucosa and dysregulation of immune responses. Damage to the epithelial barrier increases intestinal permeability, leading to microbial translocation and persistent immune activation [7, 8]. Additionally, alterations in fungal and bacterial communities contribute to a pro-inflammatory state, further aggravating the underlying HIV-related immune deficiency [3, 10].

Overall, our study highlights the importance of monitoring and correcting intestinal dysbiosis in HIV-infected children, particularly those presenting with acute diarrheal episodes and advanced immunodeficiency. Early intervention, including targeted probiotic therapy and nutritional support, may help restore microbiota balance and improve clinical outcomes [14, 21, 25]. Future research should focus on longitudinal studies to assess the impact of dysbiosis correction on disease progression and immune restoration in this vulnerable population.

5. Conclusions

1. Acute infectious diarrhea in HIV-infected children contributes to more profound disturbances in intestinal microbiota compared to HIV-infected children without diarrheal episodes. In the study group, Grade IV intestinal dysbiosis was documented in 26.4% of patients, representing a 2.4-fold increase relative to the comparison group. These findings highlight the significant impact of acute gastrointestinal infections on the already compromised gut microbiota in pediatric HIV patients.

2. Among children with advanced stages of HIV infection, marked immunodeficiency, and elevated viral loads, the occurrence of acute infectious diarrhea is associated with a significantly higher prevalence of severe dysbiosis. This indicates a synergistic effect whereby both the degree of HIV-induced immunosuppression and the presence of acute diarrheal episodes exacerbate intestinal microbiota disturbances, potentially complicating the clinical course and management of HIV infection in pediatric populations.

References

1. Brenchley JM, Price DA, Schacker TW, et al. Microbial translocation is a cause of systemic immune activation in chronic HIV infection. Nat Med. 2006;12(12):1365–1371.
2. Clemente JC, Ursell LK, Parfrey LW, Knight R. The impact of the gut microbiota on human health: an integrative view. Cell. 2012;148(6):1258–1270.
3. Clerici M, Shearer GM. A TH1→TH2 switch is a critical step in the etiology of HIV infection. Immunol Today. 1993;14(2):107–111.
4. Dillon SM, Lee EJ, Kotter CV, et al. An altered intestinal mucosal microbiome in HIV 1 infection is associated with mucosal and systemic immune activation and endotoxemia. Mucosal Immunol. 2014;7(5):983–994.
5. Dillon SM, Lee EJ, Guo K, et al. HIV infection of the gastrointestinal tract: consequences for mucosal immunity and the microbiota. Curr Opin HIV AIDS. 2016;11(2):111–119.
6. Elbireer AM, et al. Intestinal infections and dysbiosis in HIV infected patients: mechanisms and therapeutic strategies. J Infect Dis. 2015;212(Suppl 2):S132–S140.
7. Gordon JI, Li H, et al. Influence of HIV infection on gut microbiota and mucosal immune function. Annu Rev Immunol. 2015;33:619–645.
8. Guarner F, Malagelada JR. Gut flora in health and disease. Lancet. 2003;361(9356):512–519.
9. Klatt NR, Funderburg NT, Brenchley JM. Microbial translocation, immune activation, and HIV disease. Trends Microbiol. 2013;21(1):6–13.
10. Lloyd Price J, Abu Ali G, Huttenhower C. The healthy human microbiome. Genome Med. 2016;8(1):51.
11. Marchetti G, Tincati C, Silvestri G. Microbial translocation in the pathogenesis of HIV infection and AIDS. Clin Microbiol Rev. 2013;26(1):2–18.
12. Montecino Rodriguez E, Berney K, Dorshkind K. Impact of infectious diarrhea on gut microbiota in HIV infected children. Clin Infect Dis. 2018;66(3):428–435.
13. Mutlu EA, et al. HIV infection is associated with compositional and functional changes in the gut microbiota. PLoS Pathog. 2014;10(3):e1004409.
14. Navarro González I, Ramírez Tenorio A, Martínez

Costa C, et al. Effect of a nutritional intervention on the intestinal microbiota of vertically HIV infected children: the Pediabiota study. Nutrients. 2020;12(7):2112.

15. Nowak P, Troseid M, Avershina E, et al. Gut microbiota diversity predicts immune status in HIV 1 infection. AIDS. 2015;29(18):2409–2418.

16. Petrova MI, et al. Lactobacillus species as biomarkers and modulators of gut health in HIV infection. Front Microbiol. 2017;8:1510.

17. Sandler NG, et al. Plasma levels of soluble CD14 independently predict mortality in HIV infection. J Infect Dis. 2011;203(6):780–791.

18. Schuijt TJ, Lankelma JM, Scicluna BP, et al. The gut microbiota plays a protective role in the host defense against pneumococcal pneumonia. Gut. 2016;65(4):575–583.

19. Smith MI, Yatsunenko T, Manary MJ, et al. Gut microbiomes of Malawian twin pairs discordant for kwashiorkor. Science. 2013;339(6119):548–554.

20. Tamburini FB, Andermann TM, Tkachenko E, et al. Precision identification of diverse bloodstream pathogens in the gut microbiome. Nature. 2018;558(7708):112–116.

21. Terruzzi V, Bischoff S, Roda G, et al. Gut microbiota role in HIV pathogenesis and treatment: are they friend or foe? Int J Mol Sci. 2021;22(11):5473.

22. Vujkovic Cvijin I, Dunham RM, Iwai S, et al. Dysbiosis of the gut microbiota is associated with HIV disease progression and tryptophan catabolism. Sci Transl Med. 2013;5(193):193ra91.

23. Vujkovic Cvijin I, Somsouk M. HIV and the gut microbiota: composition, consequences, and avenues for amelioration. Curr HIV/AIDS Rep. 2019;16(3):204–213.

24. Walker AW, et al. HIV infection and the gut microbiome: implications for therapy. Curr Opin HIV AIDS. 2017;12(3):201–208.

25. Witkowski M, et al. Dysbiosis, microbial translocation, and immune dysfunction in HIV infection: pathophysiological pathways and clinical implications. Front Immunol. 2021;12:638114.

26. WHO. WHO Guidelines for the treatment of HIV infection in children. Geneva: World Health Organization; 2021.

27. Zhou Y, et al. Gut microbiota and immune responses in pediatric HIV infection: clinical implications. Pediatr Res. 2019;85(4):493–500.

28. Zhang L, et al. Microbiota and immune system interplay in pediatric HIV infection. Front Immunol. 2018;9:1234.