

SUCCESSFUL EXPERIENCE WITH FAECAL MICROBIOTA TRANSPLANTATION IN A PATIENT WITH MYELODYSPLASTIC SYNDROME AND GRAFT-VERSUS-HOST REACTION WITH INTESTINAL LESIONS COMBINED WITH ENTEROCOLITIS CAUSED BY CLOSTRIDIUM DIFFICILE INFECTION: A CLINICAL CASE

**K.U. BATYRBEKOV¹, A.A. GALIAKBAROVA¹, V.M. KEMAYKIN¹,
A.S. SULEIMENOVA¹, A.V. KOLESNEV¹**

¹National Research Oncology Center, Astana, the Republic of Kazakhstan

ABSTRACT

Relevance: Faecal microbiota transplantation (FMT) is the transfer of intestinal contents from a healthy donor to a patient to restore normal microflora. The material contains beneficial bacteria, fungi, antibodies, prebiotics, and other biologically active components. FMT is most effectively used for recurrent *Clostridium difficile* infection, showing better results compared to traditional treatment, such as vancomycin. Studies also suggest the potential of FMT in treating inflammatory bowel diseases, obesity, metabolic syndrome, and gastrointestinal tract functional disorders. In recent years, the method has gained widespread recognition and is now considered a potential first-line therapy for *Clostridium difficile*.

The study aimed to present the first successful clinical experience of faecal microbiota transplantation in a patient with myelodysplastic syndrome and graft-versus-host disease, characterized by intestinal lesions.

Methods: A clinical case of a 46-year-old patient who underwent allogeneic bone marrow transplantation for myelodysplastic syndrome is described. In the early post-transplantation period, the patient developed severe manifestations of Graft-versus-host disease (GvHD) with predominant involvement of the gastrointestinal tract: severe diarrhoea, abdominal pain, weight loss, signs of dysbiosis, and nutritional deficiency. After ineffective therapy with steroids and supportive care, a decision was made to perform FMT using carefully selected donor material.

Results: The FMT procedure was clinically successful, with improvements in general condition, a decrease in the severity of diarrhea, stabilization of body weight, and restoration of appetite noted within several days. Endoscopic and histological examinations of the intestinal mucosa confirmed a reduction in inflammatory changes. No side effects, complications, or signs of systemic infection were recorded after FMT.

Conclusions: The successful application of FMT in this case demonstrates the potential of the method as an additional therapeutic tool in (GvHD) with intestinal involvement, particularly in steroid-resistant forms of the disease.

Keywords: Faecal microbiota transplantation (FMT), gut microbiota, myelodysplastic syndrome, graft-versus-host reaction (GvHD), dysbiosis.

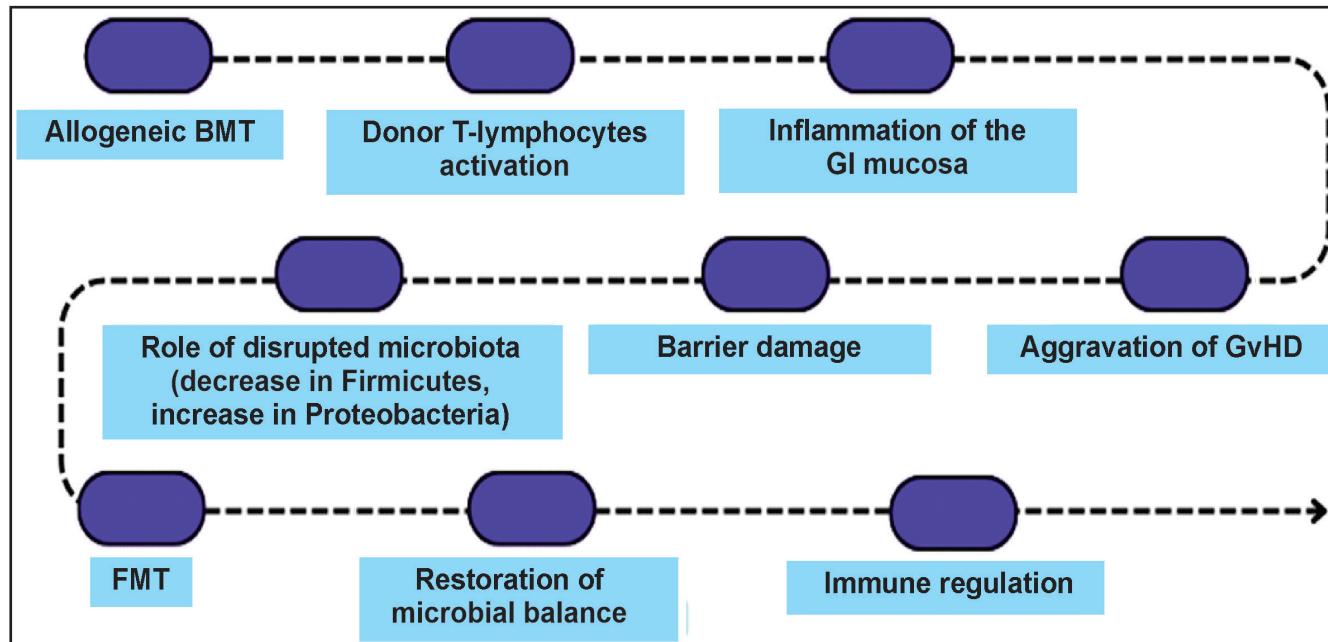
Relevance: Graft-versus-host disease (GvHD) is a severe complication of allogeneic hematopoietic stem cell transplantation (allo-HSCT), including transplantation for myelodysplastic syndrome (MDS). In GvHD, donor immune cells attack the recipient's tissues, recognizing them as foreign. GvHD can occur in acute or chronic forms, affecting various organs and systems of the body. Acute GvHD typically develops within the first 100 days post-transplantation and may manifest as skin rash, diarrhea, and liver damage. Chronic GvHD develops later and may present with skin changes, damage to the lungs, gastrointestinal (GI) tract, and other organs. Both forms of GvHD can be severe and

pose a life-threatening risk to the patient. The pathogenesis scheme of GvHD is presented in Figure 1.

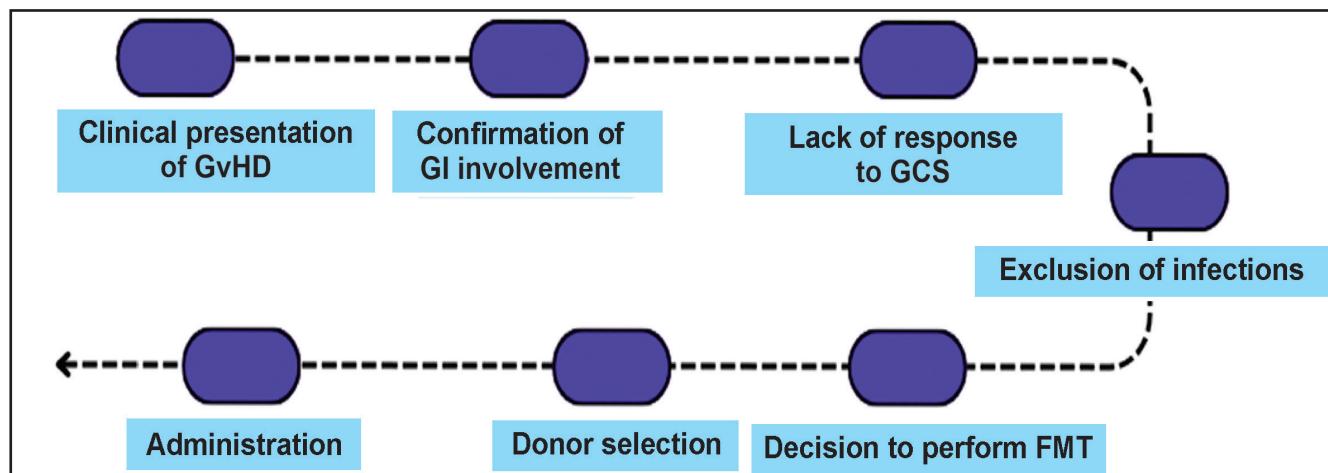
Faecal microbiota transplantation (FMT) is a medical procedure that involves transferring intestinal contents from a healthy individual into the patient's intestine. This is not merely the transfer of stool, but of an entire ecosystem that includes billions of bacteria, fungi, viruses (bacteriophages), prebiotics, natural antibiotics, secretory immunoglobulins (mainly IgA), mucin proteins, bile acids, and other biologically active components. This method gained widespread recognition due to its high efficacy in treating recurrent *Clostridium difficile* infection (CDI). Moreover,

preliminary data suggest the potential of FMT in treating inflammatory bowel diseases, obesity, metabolic syndrome, and functional GI disorders. Over the past decade, the method has been actively studied, and some experts now recommend FMT as first-line therapy for CDI, includ-

ing recurrent and treatment-resistant forms. Randomized controlled trials have confirmed that the effectiveness of FMT exceeds that of traditional vancomycin therapy for recurrent CDI [1]. The decision-making algorithm for FMT is presented in Figure 2.



Legend: BMT – bone marrow transplantation, GI – gastrointestinal; GvHD – graft-versus-host disease, FMT – faecal microbiota transplantation
 Figure 1 – Pathogenesis scheme of graft-versus-host disease with intestinal involvement and microbiota disruption



Legend: GCS – glucocorticosteroid, GI – gastrointestinal, GvHD – graft-versus-host disease, FMT – faecal microbiota transplantation.
 Figure 2 – Algorithm for decision-making regarding faecal microbiota transplantation in intestinal graft-versus-host disease

Aim of the study: To present the first successful clinical case in Kazakhstan of faecal microbiota transplantation in a patient with myelodysplastic syndrome and graft-versus-host disease with intestinal involvement, complicated by *Clostridium difficile* infection.

Materials and Methods: A retrospective analysis was conducted of a clinical case involving a patient with MDS who developed intestinal GvHD following allogeneic bone marrow transplantation (BMT), complicated by se-

vere dysbiosis and clinically significant symptoms. FMT was performed using donor material prepared in accordance with international safety protocols [2].

Clinical case:

Patient information: Patient T., 43 years old, upon admission complained of general weakness, periodic muscle pain in the legs, decreased appetite, and lack of weight gain. According to the medical history, the disease onset occurred in 2012, when intermittent bruises began to appear on the body.

Clinical data: In July 2018, in the hematology department of City Hospital No.1 in Astana (Kazakhstan), based on histological, cytological, flow cytometric, and FISH studies of the bone marrow, the diagnosis was confirmed: Myelodysplastic syndrome, hypoplastic variant with a paroxysmal nocturnal hemoglobinuria (PNH) clone, without signs of hemolysis.

Bone marrow histology from 05.07.2018: A morphological pattern consistent with hypoplasia of the megakaryocytic lineage is observed.

Bone marrow histology from 16.07.2018: Morphological features in the bone marrow are suspicious for MDS substrate.

Flow cytometry of peripheral blood for PNH from 27.06.2018: PNH clone on monocytes – 2.81%; granulocytes – 1.95%; erythrocytes type II – 0.23%; type III – 1.2% (total – 1.43%).

FISH from 02.08.2018: EGR1/D5S23, D5S721 -5;5q: not detected

FISH -7/7q- from 12.10.2022:

Karyotype: nucish (KMT2E, EZH2, CEP7) x2[200].

Conclusion: Deletion of 7q22/7q36 loci and monosomy of chromosome 7 were not identified in the analyzed interphase nuclei.

The patient received immunosuppressive therapy with Cyclosporin and blood component transfusions, but no positive effect was achieved.

After identifying a partially matched donor, the patient was referred to the National Research Oncology Center for HSCT.

On 17.05.2023, the patient was admitted to the National Research Oncology Center (NROC, Astana, Kazakhstan) for further evaluation. Bone marrow aspiration on May 18, 2023, showed no excess blasts or transformation; hypoplasia with signs of dysplasia persisted.

Myelogram from 18.05.2023: blasts – 2.4%. The bone marrow smear was cellular and polymorphic. Differential count performed on 500 myelokaryocytes. Erythropoiesis was of the normoblastic type with signs of megaloblastoid changes. The erythroid lineage was expanded to 30.6%, with preserved maturation and signs of dyserythropoiesis. The granulocytic lineage was preserved and represented evenly at all stages of maturation.

On May 22, 2023, Rituximab was administered at 375 mg/m²/day in monotherapy, two weeks prior to the planned conditioning regimen and three weeks prior to the haplo-HSCT transfusion.

The patient underwent all necessary pre-transplant examinations and was evaluated by a team of specialists. No absolute contraindications for haplo-BMT from her son were found.

On 14.06.2023, following conditioning (Bu 8 mg/kg + Flu 30 mg/m²) and premedication, the patient received a hematopoietic stem cell suspension transfusion: 330 mL, CD34 – 6.1 million/kg recipient's body weight, CD3 – 21.3

million/kg. The procedure was well tolerated.

On 13.06.2023 (day -1), prophylactic therapy for GvHD was initiated using the calcineurin inhibitor tacrolimus at 0.03 mg/kg/day, with serum level monitoring. Engraftment occurred on day 21 post-HSCT.

Diagnostics: On 31.08.2023, the patient was urgently hospitalized at the NROC due to a severe condition (ECOG score 3), caused by acute antral gastric ulcer pain syndrome, diarrhea (possible intestinal GvHD), and transfusion dependence. On 28.08.2023, the patient experienced a gastric ulcer exacerbation with bleeding. Esophagogastroduodenoscopy (EGD) showed an acute gastric ulcer with confirmed bleeding. On 05.09.2023, prednisolone was added to the immunosuppressive therapy at a minimal dose of 0.5 mg/kg due to increased stool volume (up to 750 mL), elevated calprotectin (448), and dyspeptic symptoms (nausea, vomiting). The condition was assessed as acute intestinal GVHD, with upper GI tract involvement. Gastrointestinal bleeding was noted.

On 11.09.2023, due to persistent GvHD symptoms and ulcer healing signs on EGD, methylprednisolone was prescribed at 1 mg/kg.

On 18.09.2023, methylprednisolone was discontinued due to a lack of clinical effect and the risk of profound immunosuppression. Tacrolimus IV was continued as immunosuppressive therapy. Colonoscopy under IV anesthesia revealed: Ulcerative terminal ileitis. Ulcerative colitis with total involvement.

On 19.09.2023, the patient was evaluated by a gastroenterologist. Diagnosis: Gastrointestinal GvHD? Pseudomembranous colitis? CMV colitis? Metronidazole therapy was initiated. A positive result for Clostridium difficile toxins was obtained.

On 25.09.2023, histological examination of the intestinal tissue showed: Colonic mucosa with ulceration, increased apoptosis, crypt distortion, and loss. This morphological pattern, taking into account the clinical and anamnestic data, most closely corresponds to GvHD with intestinal involvement (Grade IV according to Lerner).

Thus, based on endoscopic findings, Clostridium difficile toxin positivity, and intestinal biopsy, two concurrent intestinal pathologies were identified: enterocolitis caused by Clostridium difficile infection and severe intestinal GvHD. Clinical manifestations: Diarrhea up to 10 times per day, dark green mucous, mucous stool up to 1,100 mL, nausea, multiple episodes of vomiting, and abdominal pain requiring analgesia. Immunosuppressive therapy: IV tacrolimus under serum level monitoring. Frequent tramadol analgesia, symptomatic and syndrome-targeted therapy, and rectal mesalazine.

Treatment: On September 27, 2023, both EGD and colonoscopy were performed under IV anesthesia. Through the gastroscope, 50 mL of faecal microbiota

was administered into the descending part of the duodenum; during colonoscopy, 150 mL was introduced into the ileum. Starting from 20.10.2023, gradual clinical improvement was noted: Decrease in diarrhea frequency to 1 - 2 times/day, stool became pasty and brown. Pain resolved. Oral medication was resumed, and the patient began self-feeding. Intermittent dyspeptic symptoms persisted.

On 13.12.2023, the patient was hospitalized at the Hematology Center in Karaganda. An assessment of chronic GvHD activity was performed according to the criteria of the U.S. National Institutes of Health (NIH, Consensus Conference, 2014) [2]: ECOG – 2 points.

Skin: focal hyperkeratosis, peeling, dry skin syndrome – not included in score – 0 points.

Eyes: occasional dryness without visual impairment; uses drops as needed (Natural Tears/Dextrobrom) – 1 point.

GI tract: nausea, vomiting once daily, stool twice daily, pasty without pathological impurities – 2 points.

Results: Patient T., 43 years old, diagnosed with myelodysplastic syndrome, underwent allogeneic BMT. Post-transplantation, her condition worsened: febrile fever, diarrhea, hypotension, and severe neutropenia. Sepsis caused by *Staphylococcus epidermidis* was diagnosed, followed by severe diarrhea due to *Clostridium difficile* and subtotal pseudomembranous colitis. Intestinal GvHD developed, confirmed endoscopically and histologically (Grade IV).

Due to worsening condition, pronounced dysbiosis, and failure of conventional therapy, FMT was performed. The procedure was conducted on 27.09.2023, using a combination of: 50 mL via EGD and 150 mL via colonoscopy (Figures 3 and 4).

In the following days, marked positive dynamics were observed, including normalization of temperature, a progressive reduction in stool frequency, full normalization of stool characteristics, and laboratory improvements (leukocyte recovery, decreased C-reactive protein, and platelet stabilization). Detailed dynamics of clinical and laboratory parameters are presented in Table 1.

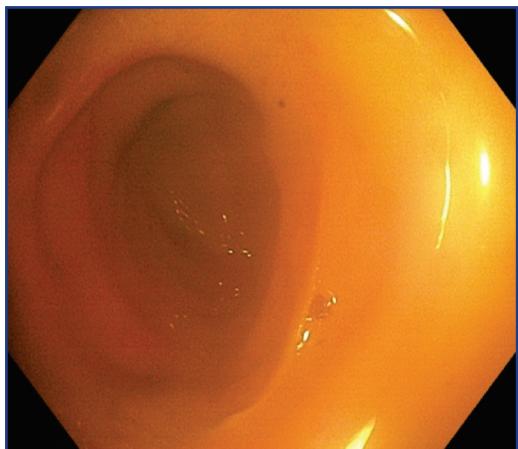


Figure 3 – Faecal microbiota transplantation into the duodenum lumen



Figure 4 – Faecal microbiota transplantation into the colon lumen

Table 1 – Dynamics of Clinical and Laboratory Parameters

Date	Event / Condition	Temperature, °C	Leukocytes ($\times 10^9/L$)	Neutrophils (%)	Platelets ($\times 10^9/L$)	CRP (mg/L)	Diarrhea	Notes
31.08.2023	Hospitalization	37.5	1.2	38	12	102	+++	Anemia, hepatosplenomegaly
14.09.2023	Sepsis (Staph. epidermidis)	39.2	0.8	25	8	250	+++	Hypotension
24.09.2023	<i>Clostridium difficile</i> infection (CDI)	38.7	1.1	40	10	198	++++	Pseudomembranous enterocolitis
25.09.2023	Diagnosis: Intestinal GvHD, Grade IV	38.4	1.3	42	9	175	++++	Enteritis, ulcers, GIT involvement
27.09.2023	FMT (IV and endoscopic administration)	37.8	1.7	48	20	132	++	FMT via EGD and colonoscopy was administered
30.09.2023	Improvement after FMT	37.1	2.4	55	45	84	+	Improved appetite and general condition
10.10.2023	Reactivation of CMV infection	37.9	2.1	50	60	98	-	CMV: 6.5×10^2 IU/mL

Note: CMV – cytomegalovirus; EGD – esophagogastroduodenoscopy; CRP – C-reactive protein; GIT – gastrointestinal tract; GvHD – graft-versus-host disease; FMT – faecal microbiota transplantation

The timeline of disease progression is presented in Table 2.

Table 2 – Timeline of faecal microbiota transplantation in a patient with myelodysplastic syndrome and graft-versus-host disease with intestinal involvement and Clostridium difficile infection

Дата	Event	Note / Diagnosis
2012	Disease onset	Appearance of bruises
27.06.2018	Immunophenotyping	PNH clone detected: monocytes 2.81%, granulocytes 1.95%, total 1.43%
05.07.2018	Bone marrow histology	Hypoplasia of the megakaryocytic line-age
16.07.2018	Bone marrow histology	Suspected myelodysplastic syndrome
02.08.2018	FISH (5q)	Negative result
July 2018	Diagnosis confirmed: "Myelodysplastic syndrome, hypoplastic variant with PNH clone, without hemolysis"	
12.10.2022	FISH (7q)	No 7q deletion or monosomy 7 detected
17.05.2023	Hospitalization at NROC	Pre-transplant evaluation, planned ritux-imab therapy
18.05.2023	Bone marrow aspiration and myelogram	Hypoplasia, dysplasia, blasts – 2.4%
22.05.2023	Rituximab administration	375 mg/m ² , two weeks before conditioning
13.06.2023	Tacrolimus initiated	GvHD prophylaxis
14.06.2023	Haplo-BMT after conditioning	Donor – son, Bu+Flu, CD34 – 6.1 mil-lion/kg
31.08.2023	Emergency hospitalization at NROC	Diarrhea, gastric ulcer, suspected GIT GvHD
05.09.2023	Prednisolone started	0.5 mg/kg. Diarrhea up to 750 mL, dys-pepsia
11.09.2023	Methylprednisolone started	1 mg/kg. GIT GvHD – clinical symptoms persist
18.09.2023	Methylprednisolone discontinued, tacrolimus continued; colonoscopy performed	Ulcerative ileitis, ulcerative colitis (total involvement)
19.09.2023	Diagnosis clarification, Metronidazole therapy started	Clostridium difficile confirmed
25.09.2023	Intestinal histology	Grade IV intestinal GvHD per Lerner
27.09.2023	Faecal microbiota transplantation performed	50 mL – duodenum, 150 mL – ileum
20.10.2023	Clinical improvement noted	Diarrhea reduced to 1 - 2 times/day, mushy stool, pain syndrome resolved
13.12.2023	Hospitalization at Hematology Center, Kara-ganda; evaluation of chronic GvHD	NIH criteria: ECOG 2; eyes – 1 point; GIT – 2 points; skin – 0 points
26.11.2024	Hospitalization at NROC, general condition evaluation	NIH criteria: ECOG 1; eyes – 1 point; GIT – 0 points; skin – 0 points
29.11.2024	Patient discharged with improvement for outpatient follow-up at the place of residence.	

Note: GIT – gastrointestinal tract; BM – bone marrow; NROC – National Research Oncology Center (Astana, Kazakhstan); PNH – paroxysmal nocturnal hemoglobinuria; GvHD – graft-versus-host disease; BMT – bone marrow transplantation; FMT – faecal microbiota transplantation; CMV – cytomegalovirus; EGD – esophagogastroduodenoscopy; NIH – U.S. National Institutes of Health

Discussion: Graft-versus-host disease (GvHD) remains one of the leading causes of adverse outcomes following allo-HSCT. When the gastrointestinal tract is affected – especially in steroid-resistant cases – the risk of fatal outcomes increases significantly, necessitating the search for new therapeutic approaches.

Faecal microbiota plays an important role in regulating the immune response and maintaining the barrier function of the mucosal lining. In patients with GvHD, particularly in the context of immunosuppression and antibiotic therapy, severe dysbiosis develops, characterized by the loss of beneficial commensal bacteria, which worsens the disease course and reduces treatment effectiveness [3, 4].

FMT helps restore microbial diversity, reduces pro-inflammatory cytokines, enhances the production of short-chain fatty acids, and contributes to the restoration of intestinal epithelial integrity. Although data on the use of FMT in GvHD remain limited in the literature, a Chinese pilot study (Qi et al., Suzhou, 2018) on steroid-refractory acute intestinal GvHD included eight patients who received FMT. All patients showed significant improvement, including reduced stool frequency, pain resolution, and restoration of microbial flora, with none of the procedures causing serious side effects [5]. Additionally, a review pub-

lished in *Biology of Blood and Marrow Transplantation* (2019) described several cases in which FMT "cured" steroid-refractory intestinal GvHD [6].

Thus, accumulated clinical observations, including the presented case, confirm the safety and potential efficacy of this method in this patient population.

It is essential to emphasize the importance of strict adherence to safety protocols when selecting a donor, performing infection screening, and preparing biological material, particularly in immunosuppressed patients. Multidisciplinary management is essential for such patients, involving gastroenterologists, infectious disease specialists, and hematologists.

Conclusion: This clinical case demonstrates the successful application of FMT in treating severe post-transplant dysbiosis. The observed clinical improvement supports the theoretical rationale for using FMT as adjunctive therapy in immune-mediated gastrointestinal diseases, including GvHD. The described case highlights the importance of an individualized approach and opens new perspectives to introduce FMT into clinical practice in oncohematology, particularly in intestinal GvHD. However, randomized controlled trials are necessary to determine the efficacy, optimal regimen, frequency of procedures, and long-term safety of FMT in immunocompromised patients.

The successful experience of using FMT in a patient with GvHD and MDS demonstrates the potential of the method to expand its indications beyond CDI. Further studies involving a larger number of patients are required to assess the efficacy and safety of FMT under conditions of immunosuppression.

References:

1. Zhang Y., Wu J., Xu Z., Qin T., Qu S., Pan L., Wang H., Sun Q., Li C., Jia Y., Cai W., Yan X., Gong J., Gao Q., Li B., Gale R.P., Xiao Z. *Impact of the International Consensus Classification of myelodysplastic syndromes* // Br. J. Haematol. – 2023. – Vol. 201(3). – P. 443-448. <https://doi.org/10.1111/bjh.18628>
2. Cammarota G., Ianiro G., Kelly C.R., Mullish B.H., Allegretti J.R., Kassam Z., Putignani L., Fischer M., Keller J.J., Costello S.P., Sokol H., Kump P., Satokari R., Kahn S.A., Kao D., Arkkila P., Kuijper E.J., Vehreschild M.J.G., Pintus C., Lopetuso L., Masucci L., Scaldaferri F., Terveer EM., Nieuwdorp M., López-Sanromán A., Kupcinskas J.,
3. Jiang H., Zhao X., Zang M., Fu R., Shao Z., Liu C. *Gut Microbiome and Plasma Metabolomic Analysis in Patients with Myelodysplastic Syndrome* // Oxid. Med. Cell Longev. – 2022. – Vol. 2022. – Art. no. 1482811. <https://doi.org/10.1155/2022/1482811>
4. Simunic M., McGraw K., Pavletic S.Z., Rashidi A. *Intestinal microbiome and myelodysplastic syndromes: Current state of knowledge and perspectives for future* // Semin. Hematol. – 2024. Vol. 61(6). – P. 442-448. <https://doi.org/10.1053/j.seminhematol.2024.10.006>
5. Qi X., Li X., Zhao Y., Wu X., Chen F., Ma X., Zhang F., Wu D. *Treating Steroid Refractory Intestinal Acute Graft-vs.-Host Disease With Fecal Microbiota Transplantation: A Pilot Study* // Front. Immunol. – 2018. – Vol. 9. – Art. No. 2195. <https://doi.org/10.3389/fimmu.2018.02195>
6. Henig I., Yehudai-Ofir D., Zuckerman T. *The clinical role of the gut microbiome and fecal microbiota transplantation in allogeneic stem cell transplantation* // Haematologica. – 2021. – Vol. 106(4). – P. 933-946. <https://doi.org/10.3324/haematol.2020.247395>

АНДАТПА

**МИЕЛОДИСПЛАСТИКАЛЫҚ СИНДРОММЕН ЖӘНЕ ШЕК ЗАҚЫМДАНЫМЕН
ЖҮРЕТІН «ТРАНСПЛАНТАТЫҢ ИЕСІНЕ ҚАРСЫ РЕАКЦИЯСЫ» БАР,
КЛОСТРИДИЯЛЫҚ ИНФЕКЦИЯМЕН ШАҚЫРЫЛГАН ЭНТЕРОКОЛИТПЕН
ҚАТАР ЖҮРЕТІН НАУҚАСҚА ФЕКАЛЬДЫ МИКРОБИОТА
ТРАНСПЛАНТАЦИЯСЫН ЖҮРГІЗДІҢ СӘТТІ ТӘЖІРИБЕСІ:
КЛИНИКАЛЫҚ ЖАҒДАЙ**

К.У. Батырбеков¹, А.А. Галиакбарова¹, В.М. Кемайкин¹, А.С. Сүлейменова¹, А.В. Колеснев¹

¹«Ұлттық ғылыми онкологиялық орталық» ЖШС, Астана, Қазақстан Республикасы

Озекмілігі: Нәжіс микробиотаны трансплантациялау (HMT) — бұл қалыпты микрофлораны қалпына келтіру ушин сау донордан пациенттеге ішек мазмұнын беру. Материалда пайдалы бактериялар, саңырауқұлақтар, антиденилер, пребиотиктер және басқа биологиялық белсенді компоненттер бар. Ен тиімді HMT қайталанатын *Clostridium difficile* инфекциясында қолданылады, бұл Ванкомицин сияқты дәстүрлі еммен салыстырганда жақсы нәтижеле көрсетеді. Зерттеулер сонымен қатар ішектің қабыну ауруларын, семіздікті, метаболикалық синдромды және асқазан-ішек жолдарының функционалдық бұзылыстарын емдеудегі HMT әлеуетін көрсетеді. Соңғы жылдары бұл әдіс кеңінен қабылданды және *Clostridium difficile*-де мүмкін болатын бірінші қатардагы терапия ретінде қарастырылды.

Зерттеу мақсаты – Миеодиспластикалық синдромы бар және ішек зақымдануы бар трансплантаттың иесіне қарсы реакциясы (ТИКР) бар науқаста нәжіс микробиотасын трансплантациялаудың алғашқы сәтті клиникалық тәжірибесін ұсыну.

Әдістері: Миеодиспластикалық синдром ушин сүйек кемігін аллогенді трансплантациялаудан откен 46 жастагы науқастың клиникалық жағдайы сипатталған. Трансплантациядан кейінгі erteme кезеңде науқаста асқазан-ішек жолдарының қатысуымен ТИКР ауыр диарея, іштің ауыруы, салмақ жоғалту, дисбиоз белгілері және қоректік заттардың жетістіпешілігі. Стероидтермен тиімсіз терапиядан жоне демеуіш күтімнен кейін мүжіттің таңдалған донорлық материалды пайдалана отырып, HMT жүргізу туралы шешім қабылданды.

Нәтижелері: HMT процедурасы клиникалық түргыдан сәтті отті: бірнеше күн ішінде жалпы жағдайдағы жақсаруы, диареяның ауырлығының томендеуі, деге салмагының тұрақтануы және тәбеттің қалпына келуі байқалды. Ишектің шырышты қабығын эндоскопиялық және гистологиялық зерттеу қабыну озгерістерінің томендеуін растады. HMT-ден кейін ешқандай жанама әсерлер, асқынудар немесе жүйелік инфекция белгілері тіркелген жоқ.

Корытынды: Бұл жағдайда HMT-ны сәтті қолдану ішектің қатысуымен, әсіресе ауруудың стероидтерге тозімді түрлерінде трансплантаттың иесіне қарсы реакциясында қосынша емдік құрал ретінде әдістің әлеуетін көрсетеді.

Түйінді сөздөр: Нәжіс микробиотаны трансплантациялау (HMT), ішек микробиотасы, миеодиспластикалық синдром, трансплантаттың иесіне қарсы реакциясы (ТИКР), дисбиоз.

АННОТАЦИЯ

**УСПЕШНЫЙ ОПЫТ ТРАНСПЛАНТАЦИИ ФЕКАЛЬНОЙ МИКРОБИОТЫ
ПАЦИЕНТУ С МИЕЛОДИСПЛАСТИЧЕСКИМ СИНДРОМОМ
И РЕАКЦИЕЙ «ТРАНСПЛАНТАТ ПРОТИВ ХОЗЯИНА»
С ПОРАЖЕНИЕМ КИШЕЧНИКА В СОЧЕТАНИИ С ЭНТЕРОКОЛИТОМ,
ВЫЗВАННЫМ КЛОСТРИДИАЛЬНОЙ ИНФЕКЦИЕЙ:
КЛИНИЧЕСКИЙ СЛУЧАЙ**

К.У. Батырбеков¹, А.А. Галиакбарова¹, В.М. Кемайкин¹, А.С. Сүлейменова¹, А.В. Колеснев¹

¹ТОО «Национальный научный онкологический центр», г. Астана, Республика Казахстан

Актуальность: Трансплантация фекальной микробиоты (ТФМ) — это перенос кишечного содержимого от здорового донора пациенту для восстановления нормальной микрофлоры. Материал содержит полезные бактерии, грибки, антитела,

пребиотики и другие биологически активные компоненты. Наиболее эффективно ТФМ применяется при рецидивирующей инфекции *Clostridium difficile*, показывая лучшие результаты по сравнению с традиционным лечением, например ванкомицином. Исследования также указывают на потенциал ТФМ в терапии воспалительных заболеваний кишечника, ожирения, метаболического синдрома и функциональных нарушений ЖКТ. В последние годы метод получил широкое признание и рассматривается как возможная терапия первой линии при *Clostridium difficile*.

Цель исследования – представить первый в Казахстане успешный клинический опыт трансплантации фекальной микробиоты у пациента с миелодиспластическим синдромом и реакцией «трансплантат против хозяина» с поражением кишечника и клострдиальной инфекцией.

Методы: Описан клинический случай 43-летнего пациента, перенесшего гаплоидентичную трансплантацию костного мозга по поводу миелодиспластического синдрома. В раннем посттрансплантиационном периоде у пациента развились тяжёлые проявления реакции «трансплантат против хозяина» с преимущественным поражением желудочно-кишечного тракта: выраженная диарея, боль в животе, потеря массы тела, признаки дисбиоза и нутритивной недостаточности. После неэффективной терапии стероидами и поддерживающими средствами было принято решение о проведении ТФМ с использованием тщательно отобранного донорского материала.

Результаты: Проведенная процедура ТФМ оказалась клинически успешной: в течение нескольких дней отмечено улучшение общего состояния, снижение выраженности диареи, стабилизация массы тела, восстановление аппетита. Эндоскопическое и гистологическое исследование слизистой кишечника подтвердило снижение воспалительных изменений. Никаких побочных эффектов, осложнений или признаков системной инфекции после ТФМ не зафиксировано.

Заключение: Успешное применение ТФМ в данном случае демонстрирует потенциал данного метода как дополнительного терапевтического инструмента при наличии реакции «трансплантат против хозяина» с поражением кишечника, особенно при стероид-резистентных формах заболевания.

Ключевые слова: трансплантация фекальной микробиоты (ТФМ), кишечная микробиота, миелодиспластический синдром (МДС), реакция «трансплантат против хозяина» (РТПХ), дисбиоз.

Transparency of the study: Authors take full responsibility for the content of this manuscript.

Conflict of interest: The authors declare no conflict of interests.

Funding: The authors declare no funding for the study.

Authors Contribution:

Conceptualization – K.U. Batyrbekov, A.S. Suleimenova; Project Administration – K.U. Batyrbekov, A.A. Galiakbarova; Investigation – K.U. Batyrbekov, A.A. Galiakbarova, A.S. Suleimenova, A.V. Kolesnev; Validation – K.U. Batyrbekov, V.M. Kemaykin; Writing – Original Draft Preparation – K.U. Batyrbekov, A.S. Suleimenova.

Information about the Authors:

K.U. Batyrbekov (Corresponding author) – PhD, Head of the Department of Expert Endoscopy and Interventional Radiology, National Research Oncology Center, Astana, Kazakhstan, tel. +7 707 474 4980, e-mail: dr.kanat77@gmail.com, ORCID: 0000-0003-4837-0775;

A.A. Galiakbarova – Endoscopy doctor at the Department of Expert Endoscopy and Interventional Radiology, National Research Oncology Center, Astana, Kazakhstan, tel. +77072676316, e-mail: ainura-endo@mail.ru, ORCID: 0000-0002-9588-0025;

V.M. Kemaykin – Head of the Center for Oncohematology and Bone Marrow Transplantation, National Research Oncology Center, Astana, Kazakhstan, tel.: +77013501621, e-mail: ke-maykin90@mail.ru, ORCID: 0000-0001-8470-4344;

A.S. Suleimenova – Head of the Department of Hematopoietic Depression with Day Hospital of the Center for Oncohematology and Bone Marrow Transplantation, National Research Oncology Center, Astana, Kazakhstan, tel.: +77024995250, e-mail: aizatik507@gmail.com, ORCID: 0009-0003-3467-7626;

A.V. Kolesnev – Head of the Bone Marrow Transplantation Department of the Center for Onco-hematology and Bone Marrow Transplantation, National Research Oncology Center, Astana, Kazakhstan, tel.: +77015865436, e-mail: koles.sasha1966@gmail.com, ORCID: 0009-0005-2054-5547.

Address for Correspondence: K.U. Batyrbekov, National Research Oncology Center, Kerey Zhanibek khandar St. 3, Astana 010017, Kazakhstan.