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ACUTE KIDNEY INJURY IN PATIENTS WITH ACUTE LEUKEMIA AFTER HEMATOPOIETIC STEM CELL TRANSPLANTATION: A SERIES OF CLINICAL CASES

A.O. MURZAKHMETOVA[1], *V.M. KEMAIKIN*[2], *A.M. AINABAY*[1,2], *A.M. MEIRAMOVA*[1], *B.A. AINABEKOVA*[1]

[1]Astana Medical University NJSC, Nur-Sultan, the Republic of Kazakhstan;
[2]National Research Oncology Center, Nur-Sultan, the Republic of Kazakhstan

ABSTRACT

Relevance: *Acute renal kidney injury is a severe complication in patients with acute leukemia who underwent hematopoietic stem cell transplantation (HSCT). According to statistics, acute renal dysfunction often occurs in the first 100 days after HSCT.*

This study aimed to *evaluate kidney function in patients with acute leukemia after hematopoietic stem cell transplantation.*

Methods: *The article presents clinical cases of patients with acute lymphoblastic leukemia who developed acute renal failure after HSCT. The dynamics of the functional state of kidneys in patients with acute lymphoblastic leukemia after HSCT are described.*

Results: *The acute kidney disorder in the studied patients was mainly caused by HSCT complications. We have identified renal kidney damage in the form of acute tumor lysis and thrombotic microangiopathy.*

Conclusion: *Patients with acute lymphoblastic leukemia risk developing acute kidney disorder during HSCT, which requires careful monitoring of kidney function, especially in the early post-transplant period.*

Keywords: *hematopoietic stem cell transplantation (HSCT), acute kidney injury (AKI), acute lymphoblastic leukemia, glomerular filtra-tion rate, kidney function.*

Introduction: Acute kidney injury (AKI) is a severe complication in patients undergoing hematopoietic stem cell transplantation (HSCT) [1]. AKI in HSCT recipients is reported in 15-73% of cases [2]. According to a recent systematic review by S.R. Kanduri et al. involving 5114 patients who have undergone HSCT, the incidence of AKI in HSCT recipients was 49.8% [3]. AKI is most common in the first 100 days after HSCT [4, 5].

The reasons for the development of AKI in patients with acute leukemia in the setting of HSCT are multifactorial and largely depend on the severity of post-transplantation complications. Severe post-transplant complications experienced by HSCT recipients are known to indirectly affect renal function, such as acute graft-versus-host disease, cardiovascular pathology, and infectious complications [6-8]. In addition, HSCT recipients undergo a phased long-term administration of nephrotoxic chemotherapeutic agents [9].

The accepted criteria for verifying AKI in HSCT recipients include decreased diuresis, decreased glomerular filtration rate, and increased serum creatinine clearance [10]. In Kazakhstan, patients with acute leukemia have undergone HSCT at the National Research Oncology Center (NROC) in Nur-Sultan since 2010. More than 500 procedures have been performed up to date.

Here, we present clinical cases of patients with acute leukemia who develop acute renal failure after HSCT.

The study aimed to evaluate renal function in patients with acute leukemia after hematopoietic stem cell transplantation.

Materials and methods: We studied the data of two clinical cases of patients with acute lymphoblastic leukemia (ALL) who develop acute renal failure after HSCT based on the NROC. The study included patients aged 18 years and older with an initial glomerular filtration rate preserved before HSCT. ALL diagnosis was confirmed by the results of hemogram, myelogram, immunophenotyping of blast cells, and cytogenetic studies (standard and FISH methods). The clinical protocol carried out chemotherapy for the Diagnosis and Treatment of Acute Lymphoblastic Leukemia in Adults dated July 9, 2015, was approved by the Ministry of Health of the Republic of Kazakhstan and the Treatment Protocol ALL-2013KZ [11]. In both cases, patients were candidates for HSCT due to a high risk of recurrence. Due to the lack of relative and non-relative donors for allogeneic stem cell transplantation, patients underwent haploidentical HSCT after premedication. The study was conducted with the approval of the local ethics committee: Astana Medical University NJSC, in compliance with the ethical principles of the Declaration of Helsinki.

Case No. 1

Patient information: according to the results of a hemogram, a 37-year-old woman was urgently admitted to the hematology department of the Municipal Clinical Hospital No. 7 in Almaty (Kazakhstan), where the B-I variant of ALL with co-expression of the CD7 lymphoid antigen was newly diagnosed (high-risk group). The patient was hospitalized at the National Research Oncology Center in Nur-Sultan (Kazakhstan) for treatment and HSCT.

Clinical data: Upon admission, the patient's general state was moderate due to the underlying disease. No clinical signs in the vital organs. Organs of urination: Visually whole kidney area. Natural urination, yellow urine. Adequate diuresis, 1.8-2.0 liters per day.

Diagnosis: Cytological studies of blood and bone marrow, cytochemical examination of blast cells, immunophenotyping by flow cytometry, standard cytogenetic analysis, and FISH study was performed to confirm the patient's diagnosis.

Hemogram data: hyperleukocytosis, thrombocytopenia, blastemia.

Myelogram data: abundantly cellular bone marrow, 81.6% represented by blast cells and depressed hematopoietic lineages.

Bone marrow IPT: a bone marrow sample on a CD 45/SSC cytogram showed an abnormal population of cells constituting 96% of the total number of nucleated events.

Cytogenetic examination of the bone marrow: no chromosomal pathology.

General clinical laboratory and instrumental examinations were carried out to assess the patient's renal function. The data are presented in Table 1.

Renal ultrasound, Doppler ultrasound of renal vessels: stage I nephroptosis on the right. Right kidney cyst. No focal pathology of the kidneys and adrenal glands. No hemodynamic disorders of blood flow at the level of the renal arteries. Indices of peripheral vascular resistance at the level of parenchymal arteries on both sides are within the normal range.

Table 1 – Laboratory data of the patient with ALL, Case No. 1

Variables	Case 1	
	Before HSCT	After HSCT
GFR (mL/min)	122	9
Creatinine (μmol/L)	46	505
Uric acid (mmol/L)	254	698
CRP (mg/L)	2.0	172
Potassium (mmol/L)	4.1	5.2
LDH (U/L)	144	2800

Treatment: Conditioning therapy included busulfan 10 mg/kg + fludarabine 30 mg/m2. Haplo-HSCT was administered in the volume of 735 mL, 6.24 million/kg of CD34 adjusted to the recipient's weight. Immunosuppressive therapy prescribed after HSCT to prevent graft-versus-host disease included tacrolimus 0.03 mg/kg a day, then 4 mg a day orally with dose adjustment depending on blood concentration; on Day 3 and Day 5, prophylaxis according to L. Luznik + cyclophosphamide 50 mg/kg twice.

Results: On Day 14 after Haplo-HSCT, the patient had no signs of neutrophil engraftment (0*10/9 L), so the stimulation with colony-stimulating factor filgrastim 300 μg per day s.c. for 14 days was started according to the Haplo-HSCT protocol. The patient was administered meropenem and vancomycin for accompanying persistent febrile neutropenia (persistent fever, CRP 85 mg/L, procalcitonin 2.04 ng/mL) due to unsuccessful therapy with piperacillin/tazobactam 4.5 g*QID and deep agranulocytosis. On Day 28, three blood tests confirmed neutrophil engraftment (leukocytes > 1x109L). On Day 20 after Haplo-HSCT, a biochemical blood test showed the first renal dysfunction episode (increased creatinine up to 112 μmol/l, decreased diuresis), so the patient was prescribed hydration therapy with diuresis stimulation. The treatment was successful, and the creatinine and urea levels returned to normal values. Later, the patient received glucocorticosteroids and antiviral therapy for a complication of the liver GVHD and CMV infection activation.

However, a myelogram (blastemia up to 13%) and blood IPT confirmed a relapse of the underlying disease on Day 76 after Haplo-HSCT. Only the monoclonal antibody Inotuzumab provided a positive response, given the resistance to the conducted anti-relapse chemotherapy courses, including high-dose cytostatic therapy. The patient was re-administered with Inotuzumab based on the National Comprehensive Cancer Network® recommendations for patients with recurrent/refractory ALL and the Concilium opinion. The leukocytes went down to myelotoxic agranulocytosis levels during treatment; the ossalgia events were managed. Against the first course of Inotuzumab, blood biochemistry showed increasing nitrogenous waste (creatinine 505 μmol/L, uric acid 698 mmol/L) and lactate dehydrogenase (2800 U/L) and a downward leukocyte trend (0.6*109/L). The glomerular filtration rate was reduced from 122 to 9 mL/min/m2. Urination was done through a urethral catheter due to diuresuria (Table 1).

Ultrasound during the kidney failure showed both kidneys' hypertrophy and increased left kidney parenchyma compared to the baseline. The renal blood flow test showed a decreased perfusion in both kidneys. Further restoration of renal function in this patient was achieved by prolonged hemodiafiltration.

Time scale: Table 2 presents the time scale of the described clinical case.

Table 2 – Time scale of clinical case No. 1

Period	Event
December 2020	Verification of the diagnosis, hospitalization
December 2020 – July 2021	Preparation for HSCT: courses of polychemotherapy, therapy with Inotuzumab
August 2021	Haploidentical HSCT
September 2021	On Day 20 after HSCT – a new episode of decreased renal function
September 2021	Post-HSCT complication: Liver GVHD and activation of CMV infection
November 2021	Relapse of the disease, a course of treatment with Inotuzumab
November 2021	On Day 76 after HSCT – a recurrent episode of decreased renal function
November-December 2021	Hemodiafiltration sessions, recovery of renal function

Case No. 2

Patient information: A 30-year-old man was admitted to the National Research Oncology Center with an established diagnosis at the place of residence: Ph+ ALL, B2 variant according to EGIL classification, high-risk group. Given the patient's young age, the high risk of the underlying disease, and the presence of a partially compatible relative donor, the patient was hospitalized for HSCT.

Clinical data: Upon admission, the patient's general state was moderate due to the underlying disease and ongoing chemotherapy – no pathological changes in organs and systems. Adequate urination was 1.5-2 liters per day, free. The patient had peripheral edema in the legs.

Diagnosis: Hemogram data: hyperleukocytosis, moderate anemia in terms of hemoglobin level, blastemia.

Myelogram: small-celled bone marrow. Blasts – 88%. Total replacement of bone marrow cells with blast cells. The count is 100 cells – normoblasts type of hematopoiesis. MKC lineage is depressed.

Bone marrow IPT: abnormal population of cells, 80% of the total number of nucleated events. Transformed cells are weakly positive for CD45 and have a low degree of granularity — co-expression of myeloid antigen CD13.

FISH study of the bone marrow: 100% of the scanned nuclei showed a rearrangement of the BCR/ABL gene.

Renal ultrasound, Doppler ultrasound of renal vessels: slight diffuse changes in the parenchyma of both kidneys; no hemodynamic disturbances in the vessels of the kidneys.

Treatment: Conditioning therapy included busulfan 10 mg/kg + fludarabine 30 mg/m2. Haplo-HSCT was administered in the volume of 408.0 mL, 8.9 million/kg of CD34 adjusted to the recipient's weight. Graft-versus-host disease was prevented by administering tacrolimus 0.03 mg/kg a day, then 4 mg a day orally with dose adjustment depending on blood concentration; on Day 3 and Day 5, prophylaxis according to L. Luznik + cyclophosphamide 50 mg/kg twice.

Results: Neutrophil engraftment was ascertained on Day 21 after HSCT. Regarding complications, this patient reported febrile neutropenia, and an initial antibiotic therapy was carried out (piperacillin/tazobactam 4.5 g QID). However, due to the CRP growth (141→157→163 ng/mL over time), persistent febrile condition, and deep agranulocytosis (leukocytes 0.0 thousand/μL), the therapy was supplemented with Meropenem and Vancomycin. Over time, the temperature went down to subfebrile levels, and no active CRP growth was reported. The patient continued with immunostimulating therapy.

On Day 30 after Haplo-HSCT, the patient presented thrombotic microangiopathy (TMA) confirmed by increased lactate dehydrogenase level, the appearance of schistocytes in the clinical blood test, increasing thrombocytopenia, a decreased haptoglobin, increased nitrogenous waste and an increase in creatinine to 443 μmol/L. Daily diuresis was reduced to 150-200 mL a day, and the glomerular filtration rate went down from 120 to 14 mL/min. C-reactive protein, uric acid, and potassium levels were increased. Tacrolimus was discontinued for this complication. The data is presented in Table 3.

Table 3 – Laboratory data of the patient with ALL, Case No. 2

Variables	Case 2	
	Before HSCT	After HSCT
GFR (mL/min)	120	14
Creatinine (μmol/L)	70	443
Uric acid (mmol/L)	230	502
CRP (mg/L)	7.2	7.8
Potassium (mmol/L)	3.9	4.1
LDH (U/L)	144	2800

The kidney ultrasound during the kidney failure showed an increased blood flow resistance in renal vessels, both kidneys' hypertrophy, and enlarged parenchyma thickness. The patient responded positively to hemodiafiltration with a gradual withdrawal of immunosuppressive therapy. Cyclosporine was reduced to 100 mg a day. A controlled follow-up a month later showed a reduced creatinine of 102 μmol/L, so cyclosporine was re-administered.

Time scale: Table 4 presents the time scale of the described clinical case.

Table 4 – Time scale of clinical case No. 2

Period	Event
November 2018	Verification of the diagnosis, hospitalization
November 2018 – March 2019	Preparation for HSCT: courses of polychemotherapy
March 2019	Haploidentical HSCT
April 2021	Post-HSCT complication: thrombotic microangiopathy
April 2021	Day +30 after HSCT: decreased renal function
April 2021	Dosage adjustments for immunosuppressive drugs
April-May 2021	Hemodiafiltration sessions, recovery of renal function
November-December 2021	Hemodiafiltration sessions, recovery of renal function

Discussion: In the described clinical cases, patients with ALL developed renal disorders assessed as acute renal failure in the first months after HSCT. The glomerular filtration rate estimate by the CKD-EPI equation revealed a sharp decrease in the parameters compared with the baseline. According to the available data, both patients underwent haploidentical HSCT, which could cause kidney injury.

The presented ultrasound results reflect the kidney size changes in AKI similar to those reported by P.Q. Liu [12]. A systematic review by S. Ninet et al. showed that an elevated resistance index could predict persistent AKI in critical patients [13].

In the presented cases, AKI developed in the first few months after HSCT. This confirms the statistical data of foreign studies describing kidney injury in the first 100 days after HSCT [14, 15].

Post-HSCT complications were the main reason for AKI development in those patients. Two patients reported post-HSCT complications in the form of acute tumor lysis syndrome and thrombotic microangiopathy. We believe that the developed complications may be the basis for AKI development, leading to indirect renal damage and worsening the underlying disease. These complications could drive acute renal failure development.

Conclusion: The presented case series describes AKI in patients with ALL after HSCT. These clinical cases demonstrate different kidney injury pathogenesis. Since post-transplant complications and nephrotoxic agents could be the risk factors for acute renal failure development, careful renal function monitoring is required after HSCT.

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ТҰЖЫРЫМ

ГЕМОПОЭЗДІК ДІҢ ЖАСУШАЛАРЫН ТРАНСПЛАНТТАУДАН КЕЙІН ЖІТІ ЛЕЙКОЗЫ БАР НАУҚАСТАРДА БҮЙРЕКТІҢ ЖІТІ ЗАҚЫМДАНУЫ: ЖАҒДАЙЛАР СЕРИЯСЫ

***А.О. Мурзахметова*[1], *В.М. Кемайкин*[2], *А.М. Айнабай*[1,2], *Ә.М. Мейрамова*[1], *Б.Ә. Айнабекова*[1]**

[1]«Астана медицина университеті» КЕАҚ, Нұр-сұлтан, Қазақстан Республикасы;
[2]Ұлттық ғылыми онкологиялық орталық, Нұр-сұлтан, Қазақстан Республикасы

Өзектілігі: *жедел лейкемиямен ауыратын науқастарда гемопоэздік бағаналы жасушаларды трансплантациялаумен (ГДЖТ) ауыратын науқастарда бүйректің жедел зақымдануы ауыр асқыну болып табылады. Статистикаға сәйкес, жедел бүйрек функциясының бұзылу жиілігі ГДЖТ-дан кейінгі алғашқы 100 күнде кездеседі.*

Зерттеудің мақсаты *– гемопоэздік дің жасушаларын трансплантациялаудан кейін жедел лейкозбен ауыратын науқастарда бүйрек функцияларын бағалау.*

Әдістері: *мақалада жедел лимфобластикалық лейкемиямен ауыратын, ГДЖТ-дан кейін жедел бүйрек жеткіліксіздігі дамыған науқастардың клиникалық жағдайлары келтірілген. Біз ГДЖТ-ден кейін жедел лимфобластикалық лейкемиямен ауыратын науқастардың бүйректің функционалды жағдайының динамикасы сипатталған.*

Нәтижелері: *зерттелген науқастарда бүйректің жедел зақымдануының негізгі себебі ГДЖТ-дан кейінгі асқынулар кешені болды. Біз жедел ісік лизисі және тромботикалық микроангиопатия түрінде бүйректің зақымдануының ренальды түрін анықтадық.*

Қорытынды: *жедел лимфобластикалық лейкемиямен ауыратын науқастарда ГДЖТ кезінде бүйректің жедел зақымдану қаупі бар, бұл әсіресе трансплантациядан кейінгі ерте кезеңде бүйрек функцияларын мұқият бақылауды қажет етеді.*

Түйінді сөздер: *гемопоэздік дің жасушаларын трансплантациялау, бүйректің жедел зақымдануы, жедел лимфобластикалық лейкемия, гломерулярлық сүзу жылдамдығы, бүйрек қызметі.*

АННОТАЦИЯ

ОСТРОЕ ПОЧЕЧНОЕ ПОВРЕЖДЕНИЕ У БОЛЬНЫХ С ОСТРЫМ ЛЕЙКОЗОМ ПОСЛЕ ТРАНСПЛАНТАЦИИ ГЕМОПОЭТИЧЕСКИХ СТВОЛОВЫХ КЛЕТОК: СЕРИЯ КЛИНИЧЕСКИХ СЛУЧАЕВ

***А.О. МУРЗАХМЕТОВА*[1], *В.М. КЕМАЙКИН*[2], *А.М. АЙНАБАЙ*[1,2], *А.М. МЕЙРАМОВА*[1], *Б.А. АЙНАБЕКОВА*[1]**

[1]НАО «Медицинский университет Астана», Нур-Султан, Республика Казахстан;
[2]НАО Национальный научный онкологический центр, Нур-Султан, Республика Казахстан

Актуальность: *Острое повреждение почек является серьезным осложнением у больных с острым лейкозом, перенесших трансплантацию гемопоэтических стволовых клеток (ТГСК). По статистике, острое нарушения функции почек часто встречается в первые 100 дней после ТГСК.*

Цель исследования *– оценка функций почек у больных с острым лейкозом после трансплантации гемопоэтических стволовых клеток.*

Методы: *В статье представлены клинические случаи больных с острым лимфобластным лейкозом (ОЛЛ), у которых развилась острая почечная недостаточность после ТГСК. Описана динамика функционального состояния почек больных с ОЛЛ после ТГСК.*

Результаты: *Основной причиной развития острого повреждения почек у исследуемых больных послужил комплекс осложнений после ТГСК. Была выявлена ренальная форма повреждения почек в виде острого лизиса опухоли и тромботической микроангиопатии.*

Заключение: *Больные с ОЛЛ имеют риски развития острого повреждения почек во время ТГСК, что требует тщательного мониторинга функций почек, особенно в ранний посттрансплантационный период.*

Ключевые слова: *трансплантация гемопоэтических стволовых клеток (ТГСК), острое повреждение почек (ОПП), острый лимфобластный лейкоз (ОЛЛ), скорость клубочковой фильтрации, функция почек.*

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Authors' data:
Murzakhmetova Aigerim Orazbayevna (corresponding author) – 3rd-year Ph.D. student at the Internal Medicine Department with a course on gastroenterology, nephrology, endocrinology, and pulmonology, Astana Medical University NJSC, Beibitshilik Str. 49a, Astana 010000, the Republic of Kazakhstan, tel: +77172539447, e-mail: sapen4ic@mail.ru, ID ORCID: https://orcid.org/0000-0002-1487-5487;
Kemaikin Vadim Matveyevich – Ph.D., Chief External Hematologist of the Republic of Kazakhstan, Head of Oncohematology and Bone Marrow Transplantation Department, National Research Oncology Center of the Republic of Kazakhstan, Nur-Sultan, the Republic of Kazakhstan, tel: +77172539447, e-mail: kemaykin90@mail.ru, ID ORCID: https://orcid.org/0000-0001-8470-4344;
Ainabai Ayagul Maratovna – Candidate of Medical Sciences, hematologist, Internal Medicine Department with a course on gastroenterology, endocrinology, and pulmonology, Astana Medical University NJSC, Nur-Sultan, the Republic of Kazakhstan, tel: +77172539447, e-mail: aainabai@mail.ru, ID ORCID: https://orcid.org/0000-0002-1257-2263;
Meiramova Asel Merekeyevna – Ph.D. doctoral student, assistant of the Internal Medicine Department with a course on gastroenterology, endocrinology, and pulmonology, Astana Medical University NJSC, Nur-Sultan, the Republic of Kazakhstan, Tel: +77172539447, e-mail: biasel@mail.ru, ID ORCID: https://orcid.org/0000-0002-4030-0783;
Ainabekova Bayan Alkenovna – Doctor of Medical Sciences, Professor, Head of the Internal Medicine Department with a course on gastroenterology, endocrinology, and pulmonology, Astana Medical University NJSC, Nur-Sultan, Republic of Kazakhstan, tel: +77172539447, e-mail: ajjnabekva@rambler.ru, ID ORCID: https://orcid.org/0000-0002-5427-3931.