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Risk Factors of Delayed Graft Function in Patients with Kidney Transplantation

Oyunpurev Erdenechimeg[1], Tuvshinbayar Javzan[1], Uugan-Erdene Amarbold[1], Od-Erdene Lkhaakhuu[1], Kishigee Tseren[1], Nyamsuren Davaajav[1], Bayan-Undur Dagvadorj[1], Erdenesaikhan Mendbayar[1], Ganbold Gantulga[1], Javkhlantugs Dondog[1], Zolzaya Nyamsuren[1], Sarantsetseg Jigjidsuren[1], Oyunbileg Banzragch[1], Batsaikhan Batsuuri[1], Tumurbaatar Batchuluun[1], Tsogjargal Burentogtokh[1], Enkhtuya Jambaldorj[2], Enkhtamir Enkhtuvshin[2], Ariunbold Jamba[1]

[1]The First Central Hospital of Mongolia, Ulaanbaatar, Mongolia;
[2]Mongolian National University of Medical Sciences, Ulaanbaatar, Mongolia.

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Corresponding Author:
Ariunbold Jamba (M.D., Ph.D.)
Kidney Center, The First Central Hospital of Mongolia, Ulaanbaatar, Mongolia
E-mail: j.ariunbold@fchm.edu.mn

Objective: In Mongolia, since 2006, kidney transplantation has been performed regularly, with more than 400 people having undergone this surgery. This preliminary study aimed to investigate the incidence of delayed graft function after kidney transplantation in Mongolia and to identify the associated risk factors. **Methods:** This study used a retrospective approach, gathering data from monitoring records. We applied the Student t-test, one-way ANOVA, Wilcoxon rank sum test, Kruskal-Wallis rank test, Spearman correlation, multivariate linear regression, and logistic regression. **Results:** Of the 268 recipients, 198(74.44%) were male. There were 14 total cases of DGF, of which 13 were transplanted from brain-dead donors. Postoperative urine outcome decreases by 120 mL for a 1-year increase in donor age (b = -120, p= 0.033). A 1-minute increase in cold ischemia time results in a 20-mL decrease in urine output (b = -20, p= 0.002). Donor age>40 increased the risk by 1.2 times (OR=1.2, p= 0.05), and brain-dead donor increased the risk by 19.37 times (OR=19.37, p= 0.0001), while recipient age<35 reduced the risk by 5 times (OR=0.2, p= 0.039). **Conclusion:** Our study found that risk factors such as older donor and recipient age, prolonged cold and warm ischemia times, and brain-dead donor status were associated with an increased risk of delayed graft function.

Keywords: Kidney transplantation, Delayed graft function, DGF, Graft function, Kidney graft, Mongolia

Introduction

Kidney transplant surgery in Mongolia was first performed at the First Central Hospital of Mongolia in 1996. Between 1996 and 2006, a "National Kidney Transplant Team" was trained. Since 2006, kidney transplant treatment has been performed regularly and locally, with more than 400 people having undergone this surgery by the first half of 2025.

Delayed graft function (DGF) is one of the common early complications after kidney transplantation.[1] The incidence of delayed graft function following kidney transplantation from a brain-dead donor is 20-50%, compared to 4-10% following kidney transplantation from a living donor.[2,3] Delayed graft function is defined by several criteria, including an increase in serum creatinine of 43 µmol/L or a decrease in urine output of 30 mL/h within the first 24 hours, and the patient requiring dialysis within 7 days of surgery.[4]

Graft dysfunction occurs due to immune and ischemia-reperfusion injury during DGF. An article by Italian scientists, published in 2022, stated that the main factors influencing the development of DGF are ischemia-reperfusion injury, the source and quality of the donated kidney, and the recipient's clinical management. The pathophysiology of ischemia-reperfusion injury involves kidney hypoxia related to the duration of warm and cold ischemia, as well as the harmful effects of blood reperfusion on tubular epithelial cells and endothelial cells. Ischemia-reperfusion injury is more frequent and severe in kidneys from deceased donors than in those from living donors. Kidneys from living donors and those with normal function can provide better results. In the perioperative management of the recipient, great attention should be paid to hemodynamic stability and blood pressure; nephrotoxic medications should be avoided.[5]

DGF increases morbidity, hospital stay, and the cost of healthcare services.[6] Also, DGF is associated with acute and chronic renal rejection and increases the risk of graft nephropathy and failure.[7] One-year graft survival rates were 93.6% and 99.7%, respectively, for the groups with and without DGF.[8] Over time, patients with DGF may present lower graft function and survival compared to transplant recipients without DGF.[5]

DGF has a significant impact on short- and long-term outcomes in the kidney transplant recipients. Immediately after transplant, DGF is associated with higher costs due to frequent laboratory monitoring, imaging, biopsy, and the need for dialysis. The hospital stay is often prolonged, and DGF was associated with a higher OR (OR 2.4; 95% CI 1.3–4.5, $p < 0.0064$) of 30-day readmission.[9] Allografts with DGF are at increased risk of developing acute rejection. In a cohort study of 645 patients, DGF was associated with higher rates of 1-, 3-, and 5-year cumulative probability of rejection in those with DGF compared with those without DGF (16%, 22%, and 23% in the DGF group vs. 10%, 12%, and 16% in the non-DGF group).[12]

DGF impacts long-term graft function, as evidenced by higher serum creatinine levels in kidney transplant recipients with DGF at a mean follow-up of 3.5 years compared with non-DGF cases.[11] Graft half-lives were shown to be lessened by 3–5 years for recipients with DGF.[13] A cohort study of 18,149 patients from 17 French hospitals found that DGF increased the recipient's risk of developing major cardiovascular complications in the future (hazard ratio: 1.24, 95% CI: 1.10-1.40)—specifically, coronary artery disease, heart failure, and cardiovascular mortality, but no effect on stroke.[14]

A study[15] on children has found that post-transplant red blood cell transfusion was strongly associated with delayed graft function. Furthermore, the use of allografts with multiple arteries and cold ischemia time greater than 20 hours was a risk factor for delayed graft function (adjusted OR = 52.51 and 49.4; 95% CI, 2.57-1070.4 and 1.83-1334.2, respectively). Sex-matched transplants and living donors were protective factors for delayed graft function (adjusted OR = 0.043 and 0.027; 95% CI, 0.005-0.344 and 0.003-0.247, respectively). Total HLA mismatches <3 Played a protective role for delayed graft function (adjusted OR = 0.114; 95% CI, 0.02-0.6), whereas transplant within compatible but different blood types increased the risk of delayed graft function (adjusted OR = 20.54; 95% CI, 1.96 - 215.26). No significant correlation was shown between delayed graft function and allograft survival ($p = 0.190$). This study suggested delayed graft function as a key factor in allograft rejection-free survival (adjusted OR = 3.832; 95% CI, 1.18-12.37).[15] According to a meta-analysis study examining the risk of DGF after living donor kidney transplantation, following factors increased risk of DGF: older donors, male recipients, higher recipient body mass index, non-white recipients, pre-existing diabetes, pre-existing hypertension, history of dialysis, re-transplantation, unrelated donor/recipient, ABO incompatibility, higher panel reactive antibody (PRA) levels, utilization of right kidney, and longer cold ischemia time (CIT).[16]

A hormonal resuscitation package to manage the catecholamine "storm" that follows brain death is recommended. Donor pretreatment with dopamine before procurement lowers the rate of DGF. Hypothermic machine perfusion may offer a significant reduction in the rate of DGF vs simple cold storage.[17]

DGF is a common occurrence after kidney transplantation and affects the future prognosis of the recipient and graft survival. Compared to similar studies conducted in other countries, the

study in Mongolia has a smaller sample size due to the limited number of kidney transplantation surgeries performed. A better understanding of the predictive factors for DGF can lead to improved methods for preventing or mitigating the injury. Therefore, we consider it necessary to investigate this issue further, and this study serves as a starting point and foundation for future research in Mongolia. This preliminary study aimed to examine the incidence of delayed graft function after kidney transplantation in Mongolia and to identify the predictive risk factors for DGF in patients undergoing kidney transplantation.

Material and Methods

Research Design and Subjects

This retrospective study included patients who underwent kidney transplantation at the First Central Hospital of Mongolia between January 2018 and June 2025. Demographic, clinical, and laboratory data were collected from the postoperative follow-up checklists. Postoperative follow-up data before 2018 could not be collected, as the records from that period were maintained only in paper format and were not available electronically. Therefore, only data from 2018 onwards were included in this study. Delayed graft function was defined as the requirement for dialysis within the first 7 days following kidney transplantation, in accordance with the widely accepted definition in the literature. Patients who received a kidney transplant during the study period and had complete postoperative follow-up records were included in the analysis.

Variables

The following preoperative variables were included: patient characteristics (age, sex, body weight, height, ABO type, rhesus factor, HLA-mismatch count), laboratory results (hemoglobin, hematocrit, white blood cell count, neutrophil percentage, lymphocyte count, C-reactive protein, serum creatinine). Intraoperative data included cold ischemia time and warm ischemia time. Postoperative data included urine output on the first day after transplantation, hospital stay, and postoperative laboratory results (hemoglobin, hematocrit, white blood cell count, neutrophil percentage, lymphocyte count, C-reactive protein, and serum creatinine).

Statistical Analysis

Variables were statistically analyzed using the Stata 17 program. The distribution of variables was assessed using the Shapiro-Wilk test, and differences between means were evaluated using the Student t-test and one-way ANOVA test when the distribution was normal, and using the Wilcoxon rank sum test and Kruskal-Wallis rank test when the distribution was non-normal. Logistic regression analysis was used to estimate odds. Demographic data were presented as descriptive statistics. Statistical significance was considered to be achieved when p values were ≤ 0.05.

Results

There were 14 total cases of DGF or a prevalence of 5.2%, of which 13 were transplanted from brain-dead donors and 1 from a living donor.

A total of 268 recipients (n=268) were included in the study, comprising 68 females (25.56%) and 200 males (74.44%). While 128 (51.61%) of the donors were female, 120 (48.39%) were male. Also, 45 were transplanted from brain-dead donors, and 223 were transplanted from living donors. The mean age of the recipients was 36 (35-39) years, the mean age of the donors was 43.8 ± 0.68 years, the mean BMI of the recipients was 22.8 (22.3-23.6) kg/m2, the mean length of hospital day 14 (14-15) days, the urine output on the first postoperative day was 5015 (4680-5323) ml, the mean cold ischemia time was 71.5 (60.3-83.3) min, and the mean warm ischemia time was 42 (36-44) min. Preoperative blood tests showed hemoglobin 10.5 (10.3-10.7) (g/dl), hematocrit 31 (30.5-31.7), white blood cell count 15.3 (14.5-15.6)*109, neutrophils 88.7 (88.2-89.5) %, CRP 30.2 (27.3-35.4) mg/l, preoperative serum creatinine 8.4 (8-9) mg/dl, and serum creatinine at discharge 1.08 (1.03-1.12) mg/dl.

Table 1. Demographic and clinical characteristics

Variables	Mean
Donor age (year)	43.8 ± 0.68
Donor sex (male)	120 (48.39%)
Recipient age (year)	36 (35-39)
Recipient sex (male)	198 (74.4%)
Recipients of kidney from brain dead donor	45 (16.8%)
Recipients of kidney from living donor	222 (83.2%)
Recipient BMI (kg/cm2)	22.8 (22.3-23.6)
Length of hospital stay (day)	14 (14-15)
Urine output on the 1st postoperative day	5015 (4680-5323)
Cold ischemia time (min)	71.5 (60.3-83.3)
Warm ischemia time (min)	42 (36-44)
Hemoglobin (g/dl)	10.5 (10.3-10.7)
Hematocrit (%)	31 (30.5-31.7)
WBC (*109)	15.3 (14.5-15.6)
NEUT (%)	88.7 (88.2-89.5)
CRP (mg/l)	30.2 (27.3-35.4)
Serum creatinine (preoperative) (mg/dl)	8.4 (8-9)
Serum creatinine (at discharge) (mg/dl)	1.08 (1.03-1.12)

Table 2. The difference between a living donor and cadaver donor

Variables	Living donor (N=222)	Cadaver (N=45)	*p* value
Recipient age (year)	36.25±12.7	48.6±11.49	0.0004*
Donor age (year)	43.6±11.14	48.1±11.87	0.15
Recipient BMI (kg/m2)	23.25±4.9	24.8±3.8	0.22
Urine output on the 1st postoperative day (ml)	5583±1945	2876±2168	0.0001*
Hemoglobin (mg/dl)	9.2±1.26	8.4±0.98	0.012*
Length of hospital day (days)	13 (12-14)	18 (14-23.3)	0.0003*
Cold ischemia time (min)	60 (45.5-86.1)	383 (340.8-483.3)	0.0001*
Warm ischemia time (min)	42 (10-46.5)	49 (21.2-62.5)	0.24
Serum creatinine (preoperative) (mg/dl)	8.2 (7.4-9.2)	10.48 (8.02-13.2)	0.01*
Serum creatinine (at discharge) (mg/dl)	1.06 (0.92-1.13)	1.45 (1.18-1.65)	0.0001*
CRP (mg/l)	14.3 (10.5-20.3)	37.52 (21.5-63.7)	0.0007*

In the living donor group, significant differences were observed, including the recipient age was younger (36.25±12.7 vs 48.6±11.49, *p*= 0.0004), the urine output on the 1st postoperative day was higher (5583±1945 vs 2876±2168, *p*= 0.0001), the hemoglobin was higher (9.2±1.26 vs 8.4±0.98, *p*= 0.012), and the length of hospital day was shorter (13 (12-14) vs 18 (14-23.3), *p*= 0.0003), shorter cold ischemia time (60 (45.5-86.1) vs 383 (340.8-483.3), *p*= 0.0001), lower creatinine (8.2 (7.4-9.2) vs 10.48 (8.02-13.2), *p*= 0.01), lower creatinine at discharge (1.06 (0.92-1.13) vs 1.45 (1.18-1.65), *p*= 0.0001), and lower CRP (14.3 (10.5-20.3) vs 37.52 (21.5-63.7), *p*= 0.0007).

Table 3. The difference between delayed and normal graft function

Variables	Normal graft function (N=253)	Delayed graft function (N=14)	*p* value
Recipient age (year)	37±13.48	48.35	0.38
Donor age (year)	43.47±11.1	60.2±8.31	0.0015*
Recipient BMI (kg/m2)	23.17±4.72	26.93±4	0.026*
Urine output on the 1st postoperative day (ml)	5115±2038	2051±1608	0.003*
Hemoglobin (mg/dl)	9.16±1.21	7.9±0.94	0.005*
Length of hospital day (days)	13 (12-14)	23 (18.1-29.8)	0.0001*
Cold ischemia time (min)	70 (54-98)	420 (106-533.9)	0.0003*
Warm ischemia time (min)	43 (27-53)	27 (5.23-59.14)	0.7
Serum creatinine (preoperative) (mg/dl)	8.17 (7.4-9.2)	11.62 (9.1-13.3)	0.011*
Serum creatinine (at discharge) (mg/dl)	1.09 (0.98-1.14)	2.03 (1.36-2.58)	0.0006*
CRP (mg/l)	18.23 (12.7-22.12)	51.48 (21.8-120.5)	0.006*

In the group with delayed function, following differences were observed donor age being older (43.47±11.1 vs 60.2±8.31, *p*= 0.0015), the recipient BMI being higher (23.17±4.72 vs 26.93±4, *p*= 0.026), lower urine output on the 1st postoperative day (5115±2038 vs 2051±1608, *p*= 0.003), lower hemoglobin level (9.16±1.21 vs 7.9±0.94, *p*= 0.005), longer hospital bed days (13 (12-14) vs 23 (18.1-29.8), *p*= 0.0001), longer cold ischemia time (70 (54-98) vs 420 (106-533.9), *p*= 0.0003), higher preoperative serum creatinine (8.17 (7.4-9.2) vs 11.62 (9.1-13.3), *p*= 0.011) and higher serum creatinine at discharge (1.09 (0.98-1.14) vs 2.03 (1.36-2.58), *p*= 0.0006), higher CRP levels (18.23 (12.7-22.12) vs 51.48 (21.8-120.5), *p*= 0.006).

Recipient age (r=-0.17, *p*= 0.005), donor age (r=-0.18, *p*= 0.006), cold ischemic time (r=-0.29, *p*= 0.0001), length of hospital days (r=-0.18, *p*= 0.002), creatinine level at discharge (r=-0.14, p= 0.02), NEUT% (r=-0.16, *p*= 0.006) and CRP (r=-0.19, *p*= 0.016) were inversely correlated with urine output on the first postoperative day, while hemoglobin level (r=0.2,*p*= 0.0008), hematocrit (r=0.23, *p*= 0.001) were directly correlated with urine output on the first postoperative day.

Recipient age (r=0.18, *p*= 0.002), donor age (r=0.32, *p*= 0.0001), recipient BMI (r=0.23, *p*= 0.001), cold ischemia time (r=0.43, *p*= 0.0001), length of hospital day (r=0.43, *p*= 0.0001), preoperative serum creatinine (r=0.37, *p*= 0.0004), WBC (r=0.13, *p*= 0.023), NEUT% (r=0.26, *p*= 0.0001) and CRP level (r=0.35, *p*= 0.0001) were directly related to the creatinine level at hospital discharge, but were inversely associated with urine output on the first postoperative day (r=-0.29, *p*= 0.006).

Table 4. Correlation with urine output after surgery

Variables	Correlation coefficient (r)	*p* value
Recipient age	-0.17	0.005*
Donor age	-0.18	0.006*
Recipient BMI	-0.06	0.3
Cold ischemia time	-0.29	0.0001*
Warm ischemia time	-0.1	0.14
Length of hospital day	-0.18	0.002*
Preoperative serum creatinine	-0.003	0.96
Serum creatinine at discharge	-0.14	0.02*
Hemoglobin	0.2	0.0008*
Hematocrit	0.23	0.0001*
WBC	0.01	0.8
NEUT%	-0.16	0.006*
CRP	-0.19	0.0016*

Table 5. Correlation with serum creatinine at discharge

Variables	Correlation coefficient (r)	*p* value
Recipient age	0.18	0.002*
Donor age	0.32	0.0001*
Recipient BMI	0.23	0.001*
Cold ischemia time	0.43	0.0001*
Warm ischemia time	0.08	0.22
Length of hospital day	0.43	0.0001*
Preoperative serum creatinine	0.37	0.0004*
Urine output on the 1st postoperative day	-0.29	0.006*
Hemoglobin	-0.2	0.054
Hematocrit	-0.16	0.13
WBC	0.13	0.023*
NEUT%	0.26	0.0001*
CRP	0.35	0.0001*

Table 6. Factors affecting urine output after surgery

Variables	(b)	(95% CI)		*p* value
Recipient age	-22.4	-69.3	24.5	0.34
Donor age	-120	-979.8	739	0.033*
Recipient BMI	30.7	-88.5	150	0.61
Cold ischemia time	-20	-34	-6.2	0.002*
Warm ischemia time	-8.4	-14.8	-1.96	0.005*
Creatinine	198.5	15.9	380.9	0.84
Hemoglobin	69	-293	552.1	0.18
Hematocrit	37	7.1	57	0.021*
WBC	-12.4	-38	8.2	0.047*
Neut%	-18.7	-80.5	50.3	0.59
CRP	-4.4	-19.3	10.6	0.5

According to multiple regression analysis, postoperative urine output decreases by 120 mL for every 1-year increase in donor age (b = 120, 95% CI: [-979.8; 729], *p*= 0.033). A 1-minute increase in cold ischemia time results in a 20-mL decrease in urine output (b = -20, 95% CI: [-34,- 6.2], *p*= 0.002). Urine output decreases by 8.4 ml with a 1-minute increase in warm ischemic time (b=-8.4, 95% CI: [-14.8;-1.95], *p*= 0.012. A 1-unit increase in hematocrit was associated with a 37 mL increase in urine output (b=37, 95% CI: [7.1;57], *p*= 0.021). For every 1 increase in white blood cell count, urine output decreases by 12.4 ml (b=-12.4, 95% CI: [-38.8;8.2], *p*= 0.047).

Table 7. Logistic regression analysis for delayed graft function

Independent variable	(b)	(95% CI)	*p* value
Donor age >40	1.2	1-1.5	0.04*
Donor male	3.2	0.18-55.85	0.4
Brain dead donor	19.37	-11.3-36.5	0.0001*
Recipient age <35	0.2	0.04-0.92	0.039*
Recipient male	1.14	0-10.9	0.38
Recipient BMI >25	1.23	0.9-1.66	0.17

As assessed by logistic regression analysis, donor age >40 increased the risk by 1.2 times (OR = 1.2, *p*= 0.05; 95% CI 1-1.5), and brain-dead donors increased the risk by 19.37 times, while recipient age <35 reduced the risk by 5 times.

Discussion

In this study, we sought to determine the frequency of DGF after kidney transplant and its risk factors in Mongolia. There were 14 cases of DGF found in 268 patients; the incidence is similar to usual rates of DGF.[18] Of importance, 13 of these cases were in brain-dead donor kidney recipients, and there was a strong

correlation between donor type and the likelihood of developing DGF. Out of 45 transplants from brain-dead donors, DGF occurred in almost a third of patients, indicating the increased susceptibility in this donor group.[19]

Several clinical and perioperative factors were also found to be associated with DGF in our study. Older donor and recipient age, prolonged cold ischemia time and warm ischemia time, as well as perioperative laboratory parameters such as hematocrit level and white blood cell count, were significantly related to graft dysfunction. These findings are consistent with prior studies that have identified ischemia–reperfusion injury, donor age, and cold ischemia time as major contributors to the development of DGF.[20]

The strong association observed between brain-dead donor status and DGF in our study is noteworthy. Brain death is known to trigger a cascade of hemodynamic, hormonal, and inflammatory changes that may impair organ quality and predispose to early graft dysfunction.[19,20] Our results confirm earlier findings that brain-dead grafts, used to increase the donor pool but with a worse outcome in comparison to living donors, are at higher risk of developing complications. Thus, attention to perioperative care and donor maintenance may be crucial in minimizing this potential risk.

This study has several limitations. First, the data source was restricted to postoperative term follow-up forms, which may not fully capture all perioperative and long-term clinical information. As a result, some potentially essential variables (donor-related factors: donor BMI, ethnicity, comorbidity, primary cause of donor death, and donor serum creatinine; recipient-related factors: dialysis vintage, HLA mismatch, and operative note) might have been missed, leading to an incomplete assessment of risk factors. Second, the retrospective nature of the study may introduce information and selection bias. Finally, the study was conducted at a single center, which may limit the generalizability of our findings to other settings.

Despite these limitations, the findings offer valuable insights into the risk profile of kidney transplant recipients in Mongolia, highlighting the need for tailored donor and recipient selection strategies.

A meta-analysis published in 2025 identified risk factors for DGF as prolonged cold ischemia time (CIT) (OR=1.05, 95% CI=1.03 to 1.07, $p< 0.0001$), elevated donor end-stage serum creatinine (OR=1.54, 95% CI=1.26 to 1.87, $p< 0.0001$), extended dialysis vintage (OR=1.02, 95% CI=1.00 to 1.02, $p= 0.014$), increased human leucocyte antigen (HLA) mismatch number (OR=1.19, 95% CI=1.06 to 1.33, $p= 0.004$), higher donor body mass index (BMI) (OR=1.07, 95% CI=1.03 to 1.11, $p< 0.0001$), advanced donor age (OR=1.02, 95% CI=1.01 to 1.03, $p= 0.003$) and recipient diabetes mellitus (OR=1.52, 95% CI=1.40 to 1.64, $p< 0.0001$).[21]

Our findings, which demonstrated a significantly higher incidence of delayed graft function (DGF) among recipients of kidneys from brain-dead donors, are in line with the observations of Jakubiv, et al. who reported that acute kidney injury (AKI) in deceased organ donors is a critical determinant of post-transplant outcomes. In their study, donor-related factors such as hemodynamic instability, ischemia, and inflammatory responses were identified as contributors to AKI, ultimately leading to impaired graft function after transplantation. Similarly, in our study, donor type and ischemia times emerged as significant risk factors for DGF, suggesting that donor-related injury mechanisms play a central role in early graft dysfunction. Taken together, these findings highlight the need for careful donor assessment and optimization, particularly in the management of brain-dead donors, to improve post-transplant kidney function.[22]

In conclusion, we have demonstrated that donor age, recipient age, ischemia times, and hematologic parameters, particularly the status of brain-dead donors, significantly contribute to DGF. The present study results demonstrate the need to reduce ischemia time and optimize donor management for better graft performance. More multicenter, prospective studies with larger populations are required to verify this association and establish preventive measures.

Conflict of Interest

The authors declare that they have no conflict of interest.

Authors Contribution

Oyunpurev Erdenechimeg: Conceptualization, data curation, formal analysis, methodology, writing original draft
Ariunbold Jamba: Conceptualization, formal analysis, methodology, validation, writing original draft
Od-Erdene Lkhaakhuu: Conceptualization, methodology, validation
Tuvshinbayar Javzan: Conceptualization, writing original draft
Nyamsuren Davaajav: Supervision, validation
Bayan-Undur Dagvadorj: Supervision, validation
Erdenesaikhan Mendbayar: Project administration, validation
Ganbold Gantulga: Data curation, resources
Javkhlantugs Dondog: Data curation, resources
Zolzaya Nyamsuren: Data curation, resources, software
Khishigee Tseren: Data curation, software
Uugan-Erdene Amarbold: Data curation, software
Sarantsetseg Jigjidsuren: Resources, software
Oyunbileg Banzragch: Resources, software
Batsaikhan Batsuuri: Resources, software
Tumurbaatar Batchuluun: Supervison
Tsogjargal Burentogtokh: Supervison
Enkhtuya Jambaldorj: Supervison
Enkhtamir Enkhtuvshin: Methodoly

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