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Changes in Frequency of Delayed Graft Function in Deceased Donor Renal Transplant Recipient in a Tertiary Care Center in Mexico

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ABSTRACT

Background: Delayed graft function (DGF) is defined as the need for dialysis within the first seven days of transplantation. The frequency of DGF has decreased in the last five years compared with the previous 20 years of the kidney transplant program at a Mexican referral hospital. **Objective:** To determine the incidence and risk factors for DGF in the past five years (2009-2013). **Methods**: We analyzed a retrospective cohort of renal transplant recipients from deceased donors at our hospital between March 2009 and May 2013 (Period 2), and compared the results with a previously evaluated cohort (Period 1, between January 1990 and February 2009). **Results**: During the analyzed period, 78 deceased donor transplants were performed. The frequency of DGF was 9%. Multivariate analysis showed that recipient older age (OR: 1.074419; 95% CI: 1.0009-1.155116; p = 0.05), transoperative amines administration (OR: 7.73; 95% CI: 1.037-57.6; p = 0.046), and hypotension during surgery in the recipient (OR: 11.6; 95% CI: 1.33-100.8; p = 0.026) were risk factors for DGF. **Conclusion**: The incidence of DGF has significantly decreased in the past five years when compared to the previous 20 years in our hospital. (REV INVES CLIN. 2015;67:89-97)
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Key words: Delayed graft function. Renal transplantation. Graft survival.

INTRODUCTION

Delayed graft function (DGF) is a manifestation of acute renal injury resulting from multiple pathological processes, deriving from donor graft ischemia and inflammation as well as the recipient's reperfusion process and innate and adaptive immune responses[1]. The incidence of DGF, defined as the need for dialysis in the first seven days after transplantation[2], is between 21 and 50% in deceased donor (DD) recipients, including expanded criteria donors, and donors after cardiac death. These types of donors have become more common due to the scarce availability of organs required for a constantly growing number of patients on waiting lists worldwide. Reported results

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using expanded criteria donors and after cardiac death by several institutions are acceptable[3].

The impact of DGF on long-term graft survival has been a matter of controversy. Studies conducted in similar time periods have associated DGF to decreased graft survival rates, whereas others have not confirmed this relationship[4-8]. Moreover, some reports have suggested a 2.9-fold increase in the risk of losing the graft during the first year posttransplantation in patients with DGF[9].

More recent reports analyzing the U.S. Scientific Registry of Renal Transplant Recipients database have clearly associated prolonged cold ischemia time with an increased risk of developing DGF in grafts from both deceased donors with standard criteria and those with expanded criteria. However, long-term graft survival from both types of donors has not proven to be different when comparing those that developed DGF and those with immediate graft function[10,11].

One of the most important implications of DGF resulted from studies suggesting its association to acute graft rejection, where the probability of acute rejection was found to be 15% higher than in grafts without DGF[12]. According to an analysis of transplants performed in the USA between 1985 and 1992, the combination of DGF and acute graft rejection decreased five-year graft survival to 34%[5]. This increase in the acute rejection rate resulting from DGF has been suggested to be caused by the recipient's innate immune response secondary to ischemic injury and the release of free radicals, leading to increased expression of the major histocompatibility complex molecules and thus, graft antigen presentation, magnification of the inflammatory response, and cytokine production, all facilitators of graft foreign human leukocyte antigen allorecognition[1]. Additionally, DGF has been reported to be one of the risk factors more strongly associated with chronic graft injury[13]. This is related with the initial structural injury due to ischemic damage and manifested as cellular dysfunction and abnormalities in growth factor translation, leading to tissue replacement by connective tissue instead of functional tissue, and subsequent fibrosis with progressive loss of function[1,9].

Donor factors found to be related to the development of DGF include the use of inotropic agents, age > 55 years, which doubles the risk of developing DGF, grafts obtained from donors with expanded criteria (more vulnerable to DGF development), non-traumatic brain death, excessive weight, and female gender[9,11,14]. There is a tool available online through the Organ Procurement and Transplantation Network, the Renal Graft Donor Index, which assigns a numerical value that serves as an index reflecting the risk of graft failure based on the donor's clinical and demographic characteristics[15].

Brain death leads to sympathetic dysregulation of vascular tone, the initial immunological trigger of a generalized inflammatory status reflected in tissues as hyaline deposits with products of complement activation; similarly, free radical activity and the formation of microthrombi due to endothelial injury are also related to DGF development.

In cases of donors after cardiac death, injury depends on the duration of warm ischemia, and the period between extubation and asystole. In this time period, kidneys switch to anaerobic metabolism and biopsies of these grafts show acute tubular necrosis and perivascular edema even after cortical perfusion[1]. The probability of developing DGF increases 23% for every added six-hour period of cold ischemia[2].

In recipients, abnormalities such as transoperative hypovolemia, hemodialysis on the day before transplantation, thrombophilia-associated vascular thrombosis, use of OKT3 antibodies as induction therapy, presence of the antiphospholipid syndrome, excessive body weight, dialysis duration, and the number of previous transplants, among others, have been associated with DGF development[3,9].

Another relevant aspect is that graft recipients developing DGF are at a higher risk of vascular, infectious, and wound complications that may, in turn, increase almost fivefold the risk of death with a functional graft at two years[16].

In a previous study conducted at our institute between 1990 and 2009, the incidence of DGF in recipients of DD grafts was 21%[17]. The perception of the Institute's transplant team was that this incidence has tended to decrease over the past five years. Thus, the aim of this study was to determine the incidence of DGF in the past five years and the factors associated with it.

METHODS

Graft recipients

We retrospectively analyzed a cohort of renal transplant recipients whose graft was obtained from deceased donors at the Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán between March 2009 and May 2013 (Period 2), compared with the previously reported cohort of 20 years of the Kidney Transplant Program at our institute (Period 1, January 1990-February 2009)[16]. During this five-year period, the same surgical team was in charge of both organ procurement and renal transplantation.

Demographic data, pre-transplant baseline, transoperative information (including the surgical aspects of the renal transplant), and data related to the follow-up in the first year after the transplant were obtained from the medical charts. The need for dialysis (hemodialysis or peritoneal) in the first week posttransplantation (our primary study outcome) was ascertained in every transplanted case. Information pertaining to complications in the wound site (seroma, dehiscence, infection, hematoma) and to specific infections (urinary tract infection or pneumonia) occurring during the posttransplant hospitalization period, as well as all biopsy-documented rejection events during the first posttransplant year and graft and/or patient losses in that same time period, were collected. Graft loss was defined as a return to continuous dialysis treatment after recovery of renal graft function post-DGF.

Deceased donors

Information on the donors was collected from charts designed by profession for every donor and kept at the institute's transplantation department. This information included demographic characteristics (age, sex), cause of brain death, use of amines before procurement, body mass index, diuresis (ml/kg/hour) and the last serum creatinine value previous to organ procurement. The Kidney Donor Profile Index was calculated using the tool available in the Organ Procurement and Transplantation Network.

Statistical analysis

Statistical analyses were performed using Stata software (Statistics/Data Analysis, Edition 11.0. StataCorp, Texas, USA). Quantitative variables, nominal variables, and the outcome (DGF) were evaluated with Mann-Whitney *U* test, Chi square test, and multivariate analysis, respectively. We also compared both time periods, where period 1 corresponds to January 1990 to February 2009, and period 2 from March 2009 to May 2013.

RESULTS

During period 2, 78 DD renal transplants were performed in the same number of recipients, with grafts obtained from 46 donors. Demographic characteristics of the recipients and the donors as well as the variables related to the surgical procedure are shown in table 1. During the first posttransplant week, 7/78 (9%) patients required dialysis support as a result of DGF.

Recipients with grafts obtained from expanded criteria donors had a greater incidence of DGF than those with grafts obtained from criteria donors (p = 0.05). This result agreed with the calculated score obtained from each donor using the Kidney Donor Profile Index, where the expanded criteria donor grafts had worst outcomes (p = 0.03) (Table 2).

Among recipients (Table 3), the variables that showed a difference with the development of DGF included recipient age (p = 0.03), transoperatory amines administration (p = 0.02), and hypotension during surgery (p = 0.008).

According to the surgical procedure, the only significantly different variable was the duration of ureteral implantation (p = 0.03); neither ischemia duration nor the graft's anatomical characteristics were associated with DGF (Table 4).

Patients with DGF developed at least one complication, mostly at the wound site (p = 0.001). Complications included seroma (n = 4), dehiscence (n = 1), hematoma (n = 3), and infection (n = 2). There was one case of graft loss in the DGF group, leading to a statistically significant difference between groups (n = 1, 14%; p = 0.001); this graft loss occurred after one month of follow-up of acute rejection. In the non-DGF group there was one reported death with a functional graft during the first year posttransplantation

Table 1. Characteristics of recipients, donors and surgical technique

General demographic variables of receptors, donors and surgical technique			
Variables in recipients (n = 78)		**Variables of surgical technique (n = 78 grafts)**	
Male, n (%)	40 (51)	Pre-implantation biopsy, n (%)	33 (42.3)
Age, years (SD)	40.6 ± 11.9	Extravesical implantation, n (%)	66 (86)
Pretransplant creatinine mg/dl (SD)	10.6 ± 4.1	Left kidney, n (%)	35 (47)
Weight kg (SD)	62.6 ± 11.4	Ureteral implantation time, min (SD)	44.3 ± 23.6
Second transplant, n (%)	8 (10.2)	Anastomosis time, min (SD)	57.4 ± 19.1
PRA class 1/ class 2, % (SD)	7.3 ± 18.4/ 7.6 ± 21.6	Cold ischemia time, hours, SD	19.4 ± 5.3
DSA, n (%)	15 (19.2)	Immediate uresis, n (%)	47 (77)
Cause of renal disease, n (%)		Induction, n (%)	
Glomerulonephritis	6 (8)	Thymoglobulin	58 (79)
SLE	9 (11)	Basiliximab	15 (19.2)
Polycystic kidney disease	13 (17)	Daclizumab	5 (6.4)
Diabetes mellitus	14 (18)	Multiple vessels, n (%)	18 (23)
Unknown	26 (33)	**Variables in donors (n = 46)**	
Other	10 (13)		
Past history, n (%)		Male, n (%)	39 (62)
Hypertension	56 (82.3)	Age, mean(SD)	32.9 ± 12.5
Dyslipidemia	27 (39.7)	Extended criteria donor, n (%)	7 (9.8)
Heart disease	20 (29.4)	BMI, mean (SD)	24.2 (2.4)
Hypotension during surgery, n (%)	4 (6)	Pre-ablation creatinine mg/dl, mean (SD)	1.2 ± 0.78
Transoperative amines administration, n (%)	5 (7.3)	Cerebrovascular cause of death, n (%)	9 (16)
Creatinine 1 yr posttransplant mg/dl mean (SD)	1.2 ± 0.31	Kidney Donor Profile Index, mean (SD)	0.86 (0.25)
DGF, n (%)	7 (8)		
Complications, n (%)	32 (41)		
Postoperative infection, n (%)	10 (13)		
Complications at wound site, n (%)	14 (18.1)		
Acute rejection in 1st year, n (%)	11 (14.1)		
Graft loss in 1st year, n (%)	1 (1.2)		
Death during 1st year, n (%)	1 (1.2)		

SD: standard deviation; PRA: panel of reactive antibodies; DSA: donor-specific antibody; SLE: systemic lupus erythematosus; BMI: body mass index.

Table 2. Donor features associated with delayed graft function

Donor variables	DGF (n = 7, 9%)	No DGF (n = 71, 91%)	p
Expanded criteria, n (%)	2 (28)	5 (7.6)	0.05
Male donor, n (%)	5 (71.3)	35 (48.6)	0.8
Donor age, mean (SD)	46 ± 10.3	40.1 ± 11.8	0.3
Pre-ablation serum creatinine mg/dl, mean (SD)	1.09 (0.33)	1.23 (0.81)	0.12
Donor BMI, mean (SD)	24.3 ± 2.1	24.1 ± 2.4	0.4
Amines, n (%)	4 (80)	35 (77)	0.1
Cerebrovascular cause of death, n (%)	2 (28)	7 (14)	0.3
Kidney Donor Profile Index, mean (SD)	1.08 (0.43)	0.84 (0.22)	0.03

DGF: delayed graft function; SD: standard deviation; BMI: body mass index.

in a patient with long-standing diabetes mellitus who presented mesenteric thrombosis (Table 5).

Induction therapy with biological agents, either by lymphocyte depletion (thymoglobulin) or with anti-CD25 antibodies, was used in all patients during period 2. Thymoglobulin was more frequently used (74.4%) than anti-CD25 (basiliximab or daclizumab, 25.6%). No significant differences were observed in the frequency of DGF according to the induction modality used.

Table 3. Characteristics of recipients with/without delayed graft function

Recipient variables	DGF (n = 7, 9%)	No DGF (n = 71, 91%)	p
Age, mean (SD)	49.4 ± 10	39.7 ± 11.7	0.03
Male, n (%)	11 (50)	39 (47)	0.7
Second transplant, n (%)	0 (0)	8 (11)	0.3
PRA class I/II, mean (SD)	1.5± 3.3/0.28 ± 0.75	7.9 ± 19.2/8.4 ± 22	0.5/0.2
DSA, n (%)	3 (42)	12 (16.9)	0.09
Past history, n (%)			
Systemic arterial hypertension	6 (85.7)	50 (81.9)	0.8
Dyslipidemia	1 (14.2)	26 (42.6)	0.14
Heart disease	3 (42.8)	17 (27.8)	0.41
Transoperative amines administration n (%)	2 (28.5)	3 (4.9)	0.02
Hypotension during surgery n (%)	2 (33.3)	2 (3.2)	0.008
Induction, n (%)			
Thymoglobulin	6 (85.7)	50 (70)	0.62
Basiliximab	1 (14)	14 (19.7)	0.12
Daclizumab	0	5 (7.04)	0.52

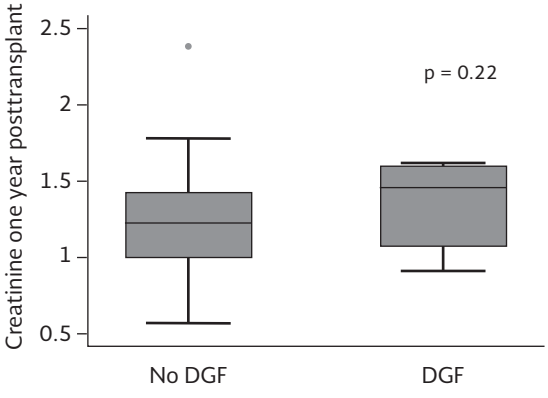
DGF: delayed graft function; SD: standard deviation; PRA; panel of reactive antibodies; DSA: donor-specific antibody.

Table 4. Graft and surgical technique variables

Graft and surgical technique variables	DGF (n = 7, 9%)	No DGF (n = 71, 91%)	p
Extravesical implantation, n (%)	5 (71)	61 (88)	0.2
Left kidney, n (%)	4 (66)	31 (46)	0.3
Multiple anastomoses, n (%)	2 (28)	16 (22)	0.7
Vascular anastomosis time (min, SD)	51.1 ± 16.4	58 ± 19	0.3
Cold ischemia time (hours, SD)	18.8 ± 5.1	19.4 ± 5.4	0.7
Ureteral implantation time (min, SD)	61.1 ± 20.8	42.5 ± 23.4	0.03

DGF: delayed graft function; SD: standard deviation.

Figure 1. Posttransplant serum creatinine.



In the multivariate analysis, the risk factors significantly associated with the development of DGF were again the patient's age (OR: 1.074419; 95% CI: 1.0009-1.155116; p = 0.05), transoperatory amines administration (OR: 7.733333; 95% CI: 1.037304-57.65373; p = 0.046), and hypotension during surgery (OR: 11.6; 95% CI: 1.334562-100.8271; p = 0.026) (Table 8).

Average serum creatinine levels one year after transplantation were similar in patients with and without DGF (Fig. 1, Table 3).

In the period analyzed in this study (period 2), no significant differences in induction therapy were found between the groups with or without DGF (Table 3). However, the difference in the use of induction treatment between both periods is striking (period 2, 100% vs. period 1, 34.3%; p < 0.0001) (Table 7). During period 1, when most patients' induction therapy was based on anti-CD25 antibodies, 10/36 (28%) developed DGF compared with 7/78 (9%) in period 2, when patients were commonly treated with thymoglobulin. We calculated the risk of developing DGF in both periods and significant differences were found in the thymoglobulin group (OR: 17.33; 95%

Table 5. Recipient outcomes

Recipient variables	DGF (n = 7, 9%)	No DGF (n = 71, 91%)	p
Graft loss, n (%)	1 (14.2)	0 (0)	0.001
Complications, n (%)	7 (100)	25 (35)	0.001
Infectious complications, n (%)	1 (14.2)	9 (12.8)	0.9
Complications at wound site, n (%)	3 (42.8)	11 (15)	0.07
More than one complication, n (%)	3 (42.8)	25 (34)	0.001
Acute rejection, n (%)	1 (14.2)	10 (14.1)	0.9
Death, n (%)	0 (0)	1 (2)	0.7
Average SCr one year posttransplant (SD), mg/dl	1.9 ± 1.6	1.2 ± 0.28	0.22

DGF: delayed graft function; SD: standard deviation; SCr: serum creatinine.

Table 6. Delayed graft function development by study period according to used induction therapy

Type of induction n (%)	Period 1		Period 2		
	DGF (n = 22, 21%)	No DGF (n = 83, 79%)	DGF (n = 7, 9%)	NoDGF (n = 71, 91%)	OR; 95% CI (p)
Anti-CD25 (n)	8	25	1	19	6.08; 0.99-52.86 (0.10)
Thymoglobulin (n)	2	1	6	52	17.33; 1.36-220.86 (0.02)
Any type of induction (n)	10	26	7	71	3.9; 1.34-11.32 (0.012)
No induction (n)	12	57	0	0	–

DGF: delayed graft function.

Table 7. Comparison between Period 1 and Period 2 of the available variables in patients with delayed graft function

Variables in transplants with DGF	Period 1 (n = 22, 21%)	Period 2 (n = 7, 9%)	p
Recipient			
Age, mean (SD)	42 ± 13.2	49 ± 10	0.1
Male, n (%)	11 (50)	4 (57)	0.02
Second transplant, n (%)	3 (13)	0	0.9
Past history, n (%)			
Systemic arterial hypertension	1 (4.7)	5 (71)	0.28
Hyperlipidemia	9 (40.9)	1 (14.2)	0.62
Heart disease	2 (9)	3 (42.8)	0.85
With induction, n (%)	10 (55)	7 (100)	0.001
No induction, n (%)	12 (54.5)	0	–
Transoperatory amines administration, n (%)	4 (19)	2 (28.5)	0.17
Hypotension during surgery n (%)	4 (19)	2 (28.5)	0.40
Donor and ischemia duration			
Male, n (%)	13 (59)	4 (66.6)	0.99
Donor age, mean (SD)	37 ± 12	45.5 ± 13.4	0.13
Cold ischemia time, hours (SD)	20.8 ± 6.1	18.8 ± 5.1	0.47

DGF: delayed graft function; SD: standard deviation.

Table 8. Results of multivariate analysis of factors associated with delayed graft function

Variables relating to DGF development	Univariate	Estimated	Standard error	OR	95% CI	p
Recipient age (increase per year)	0.03	0.1243804	0.059244	1.074419	1.0009-1.155116	0.050
Transoperatory amines administration	0.02	–13.25452	2917.756	7.733333	1.037304-57.65373	0.046
Hypotension during surgery	0.008	16.37151	2917.756	11.6	1.334562-100.8271	0.026
Donor with expanded criteria	0.03	1.176988	0.0247766	4.72	0.7225252-30.83408	0.105
Ureteral implantation time	0.02	0.0116026	0.0020577	1.02817	0.9963326-1.061025	0.083

Chi square of the probability coefficient (11.37); p = 0.030 with 43 degrees of freedom.
DGF: delayed graft function.

CI: 1.36-220.86; p = 0.02) as well as in the group treated with any form of induction (OR: 3.9; 95% CI: 1.34-11.32; p = 0.012) (Table 6).

The frequency of DGF in period 1 was 21% (22/105), decreasing to 9% (7/78) in period 2 (OR: 2.7; 95% CI: 1.084749-6.663164; p = 0.033). Differences in recipient management included the frequency of use and type of induction therapy, as previously mentioned. No differences were found in other variables analyzed in patients who developed DGF in periods 1 and 2 (Table 7).

DISCUSSION

This study on DD renal transplant recipients, conducted between 2009 and 2013 at our institute, supports the perception that the incidence of DGF has significantly decreased during period 2 compared with the previous 20 years (period 1)[16]. Our interest in documenting what has occurred in the past five years has relevant implications since DGF can impact several outcomes, including increasing the incidence of acute rejection[12] and longer hospital stays that may lead to increased costs[1,3]. The DGF also raises the risk of graft loss 2.3-fold[17] as well as the risk of infections, particularly at the wound site as in our series.

Since its creation, the renal transplant program at our institute has undergone several changes leading to key improvements. The first is, undoubtedly, the increase in the number of DD renal transplants; the numbers have increased 2.3-fold when comparing the period between 2004 and 2008 with period 2. Another is the significant decrease in the incidence of DGF when comparing period 1 with period 2 (21 vs. 9%; p = 0.03), confirming the perception of the transplant team. In a retrospective cohort study it is difficult to demonstrate which other dependent variables could have played a role in the higher number of DD renal transplants and in the decrease in the frequency of DGF cases. But among these we must consider the role played by organ donor coordinators, whose participation and direct interaction with other donor hospitals collaborating with our institute has allowed shortening the time between the diagnosis of brain death in the donors and the organ procurement process by our Institute's surgical team. Organ donor coordinators have also optimized the management of potential donors and the interaction with intensive care unit physicians at the donor's hospital, as well as coordinated the full time availability of our institute´s transplant surgeons who conduct the organ procurement and perform the transplantation following a refined and standardized technique. The technique employed for extraction and multiorgan perfusion also appears to have a positive effect on the decline in DGF frequency[18-20].

A point to consider in the integral management of DD renal transplant recipients is the universal use of induction therapy in the past few years at our institute. During period 2, reported herein, all patients were treated with some form of biological induction: 75% received thymoglobulin and 25% was treated with anti-CD25 antibodies, whereas only one-third of patients in period 1 (36/105) were given induction therapy, mostly based on anti-CD25 monoclonal antibodies[16]. It is difficult to evaluate the contribution of induction therapy in the prevention of DGF and the incidence of acute rejection in period 2 in the absence of a side-to-side comparison with a group with no induction treatment. The fact that the use of any

induction therapy improves transplant results has been well documented[21]. Regarding the biological agent used, another study demonstrated that the use of rabbit antithymocyte globulin (thymoglobulin) decreases the incidence of DGF when compared with anti-CD25 antibodies (daclizumab) as an induction agent[22]. This, however, was not confirmed in another study conducted with thymoglobulin as an induction agent in moderate to high-risk patients when comparing thymoglobulin and basiliximab[23]. Randomized trials conducted in the USA and Europe have shown that when at least one of the following risk factors is present, cold ischemia time > 24 hours, donors after cardiac death or older than 50 years, or donors with pre-ablation acute renal failure, the incidence of acute rejection, graft loss and/or death is lower when using thymoglobulin instead of anti-CD25 as induction therapy[9]. All DD transplant recipients at our institute are currently treated with thymoglobulin as an induction agent, except for unsensitized donors (panel of reactive antibodies, 0%).

As reported by other centers, we found that in period 2, expanded criteria donor was a factor for DGF development. Grafts obtained from these donors have a lower number of functional nephrons (due to frequent biopsies) and are therefore more susceptible to ischemia-reperfusion injury. It has been recommended that the cold ischemia time in these grafts be shortened as much as possible[9]. There are measures that can be taken in the donor's case to decrease the frequency of DGF such as the administration of furosemide and mannitol as well as the preferential use of vasopressin, levothyroxine, or steroids instead of amines. These measures reduce the risk of developing renal microthrombi, usually occurring approximately 24 hours after establishing the diagnosis of brain death or after > 40 hours of hospitalization in the intensive care unit. Another intervention is prophylactic anticoagulation since it decreases the endothelial concentration of free radicals. Other experimental free radical inhibitors include heme-oxygenase-1 and propionyl-l-carnitine or anti-inflammatory agents such as Ginkgolide (BN 52021), a specific inhibitor of ICAM-1 expression, as well as the use of statins to prevent cholesterol-mediated injury. These management alternatives remain to be accepted[19].

In DD renal transplant recipients, factors including the use of albumin, obesity, dialysis duration, coexisting diabetes mellitus, and/or heart disease have been associated with DGF. In our series, hypotension and the use of amines during the recipient's surgery were significantly associated with DGF development. Hypotension after completion of vascular anastomoses and graft revascularization leads to hypoperfusion of the transplanted organ and fosters DGF development[24]. Furthermore, the need for transoperative amine support to increase blood pressure leads to constriction of the graft's vessels, promoting ischemic injury[9].

In summary, the frequency of DGF in DD renal transplant recipients at our institute has significantly decreased in the last five years, thus confirming the transplant group's perception. In the multivariate analysis, risk factors associated with DGF development included the recipient's age, transoperative amine administration, and hypotension during surgery in the recipient. Factors increasing the risk of DGF vary by center, but their detection and, when possible, their control, are essential. Among the changes made in our program that could have helped to lower the incidence of DGF are the graft procurement technique and the universal administration of induction therapy to all deceased donor recipients. The impact of decreased DGF frequency on long-term patient and graft outcomes remains to be ascertained.

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