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# The causes of death-censored graft loss among kidney transplant recipients

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## Abstract

**Aim:** This study presents the causes of death-censored graft loss among kidney transplant recipients.

**Materials and Methods:** Medical records of the patients, who had undergone kidney transplantation at a tertiary center between November 2010 and December 2018, were retrospectively reviewed. Death-censored graft loss was described as an irreversible graft failure signified by return to long-term dialysis (or re-transplantation). Inclusion criteria were: patients who had undergone kidney transplantation, and subsequently lost their first graft, and a follow-up of more than one year after kidney transplantation.

**Results:** Of 269 kidney transplant recipients, 33 recipients with a mean age of 33.54 ± 15.37 years (17 male and 16 female) were included in the study. The rate of death-censored graft loss was 12.26%. Of graft failures, 3.03% occurred in the hyperacute phase, 18.18% in the acute phase, and 78.78% in the chronic phase. Chronic allograft nephropathy was the leading cause of graft failure (48.48%). Other causes were medical problems (18.18), immunological problems (18.18%) and surgical complications (15.15%).

**Conclusion:** Identification of the true causes of graft failure described under the heading chronic allograft nephropathy is noteworthy. Comprehensive biochemical, physiological, pathological, immunological, and genetic research should be implemented to remove the obstacles in kidney transplantations.

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## Introduction

Although Russian surgeon, Yurii Voronoy, had reported human allogenic kidney transplantation (KT) from a deceased donor in 1933, the first temporarily successful KT was performed by Louis Michon and Jean Hamburger in 1952, using a kidney from a living related donor [1-3]. Michon and Hamburger had noted that the kidney was rejected and the patient had died within three weeks of transplantation. The plastic surgeon, Joseph Edward Murray, knowing the immunological obstacles associated with transplantation, conducted the first long-term successful KT between monozygotic twins in 1954. As stated by Murray, that was just the first step in overcoming failure in KT [2,3]. Today, KT has become the standard of care for patients with end-stage kidney disease due to its health and economic benefits over maintenance dialysis [4].

The evolution of KT is a long story that includes both disappointment and perseverance. Advances in immunosuppression and the medical care of recipients have resulted in an improvement in graft survival in the early post-transplant period, though long-term graft survival has not been achieved [4]. International studies show that the ratio of graft failure ranges between 2% to 5% [5]. According to the Joint Report of the Ministry of Health and the Turkish Society of Nephrology, as of the end of 2018, 17.220 patients were followed up with a functioning graft, and the graft failure rate was 1.84% in patients transplanted in 2018 [6]. Long-term graft survival may be influenced by several factors; among these, patients' death with a functioning graft (DWFG) is a major cause, which accounting for nearly half of the late losses [7]. It is usually associated with coexisting conditions, which may have been present before transplantation [7]. It is clinically distinct from graft loss due to progressive dysfunction. Thus, separation of DWFG from graft loss due to progressive dysfunction

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is suggested [7,8]. Among factors leading progressive graft dysfunction, chronic allograft nephropathy (CAN) is the main cause of death censored graft loss (DCGL) [8]. Donor related (age, hypertension, non-heart beating donors, delayed graft function) and recipient related (hypertension, recurrence of glomerular diseases, de novo disease, medical or surgical complications, infections) characteristics, immunologic factors and immunosuppressive regimen (dose-related nephrotoxicity of calcineurin inhibitors) are other causes of late graft loss [9,10]. This study presents the causes of DCGL among kidney transplant recipients.

## Materials and Methods

Medical records of the 269 patients, who had undergone KT for end-stage kidney disease at a tertiary center between November 2010 and December 2018, were retrospectively reviewed. DCGL was described as an irreversible graft failure signified by return to long-term dialysis (or re-transplantation). Inclusion criteria were: patients who had undergone KT, and subsequently lost their first graft, and a follow-up of more than one year after KT. Exclusion criteria included the recipients of multi-organ transplants, the recipients who had died with a functioning kidney, and recipients who had been followed-up for less than one year after KT. The failed re-transplants were also excluded. A total of 33 patients met these criteria and were included in the study. The data belonging to the donors of these recipients were also extracted. The study was conducted according to the principles set forth by the Helsinki Declaration of 1975. Approval from the Human Ethics Committee of the Institution was obtained (Inonu University Health Sciences Non-invasive Clinical Research Ethics Committee, approval number: 2018/7-11).

The data included demographics, clinical findings, laboratory findings and therapeutic interventions for both recipients and their donors. The following characteristics were recorded: Demographics (age and gender), comorbid factors (cardiovascular diseases, diabetes mellitus, endocrine diseases, central nervous system diseases), etiology and origin of the kidney disease, the presence of preemptive transplant and/or pre-transplant dialysis (peritoneal dialysis and/or hemodialysis), duration of dialysis treatment, the type of KT (deceased donor and/or living-donor transplant), the type of donor relationship (relative and/or non-relative), the presence of extended criteria donor, the presence of delayed graft function, the presence of surgical complications, choice of immunosuppressive regimen, the time from transplantation to DCGL, and causes of the DCGL.

Hyperacute graft failure was defined as graft loss, which had developed within the first 72 hours of the transplantation. Acute graft failure included graft losses, which had developed within the period from the 72$^{nd}$ hour to the 3$^{rd}$ month of the transplantation. Chronic graft failure included graft losses, which had developed after the 3$^{rd}$ month of the transplantation. Early-surgical complications were described as surgical complications, which had developed within the first 28 days of the KT. Late-surgical complications were described as surgical complications, which had developed after day 28 of the KT. Delayed graft function was defined as the need for dialysis during the first week after transplantation. Extended criteria donor was defined as shown in Table 1.

*Surgical technique*

*Donor nephrectomy:* It was performed via either an open surgical (before 2017) or a 'pure' transperitoneal laparoscopic (through 2017 and beyond) approach. The left kidney was preferred for living-donor nephrectomy, unless it contradicted the donors' benefits, because the renal vein is longer on the left. When vascular, urological or other abnormalities were present, the right kidney was procured.

*Kidney transplantation:* The right iliac fossa was preferred for extraperitoneal placement of the transplanted kidney because the iliac vessels were more superficial than on the left side. When vascular or other abnormalities were present, or the right iliac fossa had been used before for renal transplantation, the left side was used. The left iliac fossa was also preferred in cases where the right iliac fossa had been reserved for pancreas transplantation in diabetic patients.

*Statistical analysis*

Data were analyzed using SPSS 22.0 for Windows (Inc, Chicago, USA). Descriptive frequencies were obtained for the demographic characteristics of the donors and recipients. The data were reported as mean ± standard deviation for normally distributed variables.

## Results

Two hundred and sixty-nine KTs were performed between November 2010 and December 2018. Of them, 33 recipients (17 male and 16 female) with a mean age of 33.54 ± 15.37 (range: 3-60) years were included in the study. The rate of DCGL was 12.26%. Mean duration of graft survival was 36.5 ± 31.98 (range: 0-102) months (Table 2). Mean length of hospitalization was 10.15 ± 7.87 (range: 4-42) days.

In the majority of cases (n:16; 48.4%) the cause of the kidney disease was unknown (idiopathic). Nephrocalcinosis, membranoproliferative glomerulonephritis (MPGN), hypertensive nephropathy, polycystic renal disease, diabetic nephropathy, and distal renal tubular acidosis secondary to vesico-ureteric reflux (VUR), renal agenesis, neurogenic bladder, Familial Mediterranean Fever, and Alport Syndrome were the other causes of renal failure (Table 2). Seventeen of the patients (51.5%) had at least one additional disease; hypertension was the most common (45.4%), followed by diabetes mellitus (9.09%) and epilepsy (9.09%). Twenty-five patients (75.75%) were on dialysis (18 on hemodialysis, 3 on peritoneal dialysis, and 4 on both forms of dialysis) with a mean duration of 60.8 ± 76.38 (range: 2-276) months.

Nineteen of 33 (57.57%) transplants were from a living donor. Seventeen (51.5%) grafts were recruited from non-relatives. Delayed graft function developed in 8 (24.2%) recipients (Table 2).

Of graft failures, 3.03% occurred in the hyperacute phase, 18.18% (n:6) in the acute phase, and 78.78% (n:26) in the chronic phase. CAN was the leading cause of graft failure (48.48%). Other causes were medical problems in six

**Table 1.** The definition of the extended criteria donor.

| Deceased Donor | Living Donor |
|---|---|
| Age ≥ 60 and < 5 | Age ≥ 60 |
| Vascular or anatomic variations | Vascular or anatomic variations |
| Kidney with simple cysts and/or stones | Simple kidney cysts and/or stones in one kidney, which is planned to be recovered. Donors with multiple cysts in one kidney or simple cysts and/or stones in both kidneys are not eligible for donation. |
| Presence of infection (except sepsis) | |
| Ischemia time longer than 24 hours | |
| Grafts with ATN (especially when CPR applied) | |
| ABO incompatible donors | ABO incompatible donors |
| * It is not applicable in our country | * It is not applicable in our country |
| Donors aged (≥ 50 - < 60), who have at least two of the following criteria. | Donors aged (≥ 50 - < 60), who have at least one but no more than two of the following criteria. |
| - Cerebrovascular accident | - Previous history of cerebrovascular accident without serious sequelae |
| - Hypertension | - Hypertension (uncomplicated) |
| - Diabetes Mellitus | - Diabetes Mellitus (uncomplicated) |
| - Serum creatinine >1.5 mg/dL at time of donation | - Connective tissue disease (uncomplicated) |
| Donors aged (≥ 5 - < 50), who have at least one of the following criteria. | Donors aged (≥ 30 - < 50), who have only one of the following criteria. Donation is not eligible if the potential donors have two or more of the criteria. |
| - Cerebrovascular accident | - Hypertension (uncomplicated) |
| - Hypertension | - Diabetes Mellitus (uncomplicated) |
| - Diabetes Mellitus | - Connective tissue disease (uncomplicated) |
| - Serum creatinine >1.5 mg/dL at time of donation | |
| | *** Our clinic recommends not to recover kidney from the potential living donors aged (≥ 18 - < 30) securing donor interests. If it is preferred, it would be appropriate for donors not to have additional diseases. |

ATN: Acute tubular necrosis, CPR: Cardiopulmonary resuscitation.

(18.18%) patients, immunological problems in six (18.18%) patients and surgical complications in five (15.15%) patients (Table 3).

Hyperacute rejection developed in one patient (3.03%) due to ABO incompatible renal transplantation. Acute and chronic rejections accounted for 6.06% and 9.09% of graft failures, respectively. Non-adherence to immunosuppressive medication resulted in allograft rejection in 2 patients.

Surgical complications, which were the underlying causes of graft failure in five (15.15%) patients, developed in 21 (63.6%) recipients. None of the patients developed intraoperative complications. Nearly half of surgical complications (n:10) developed in the early postoperative period (within 28 days of renal transplantation). Seven of the 21 (33.3%) patients underwent reoperation within the same week. Table 4 shows surgical complications and their management. Vascular thrombosis, which developed in two patients, who had undergone en-bloc and dual KT, and renal artery pseudoaneurysm, which developed in two patients, who had received the graft from the same deceased donor, in whom pseudomonas species were isolated from the tracheal aspirate, were the surgical causes of early-graft loss. İliac artery dissection was the surgical cause of chronic graft loss.

Immunosuppressive therapy consisted of induction with [antihuman T-lymphocyte immunoglobulin (n:29) or Basiliximab (n:4) and methylprednisolone (n:33)], and triple therapy with methylprednisolone (n:32), antiproliferative agents [mycophenolate mofetil (n:21) or enteric-coated mycophenolate sodium (n:11)], and calcineurin inhibitors [immediate-release tacrolimus (n:30) or cyclosporine-A (n:2)]. Triple therapy could not be used in one patient due to hyperacute rejection.

## Discussion

International studies show that the ratio of graft failure ranges between 2% to 5% [5]. According to the Joint Report of the Ministry of Health and the Turkish Society of Nephrology, as of the end of 2018, the graft failure rate was 1.84% in patients transplanted in 2018 [6]. In this study, DCGL occurred in 12.26% of the KTs, which was greater than that of national and international values. It should be noted that our national registry report only shows the graft failure within the first year of transplantations, which were performed on 2018. It does not include the rate of graft failure over the years. In concordance with the literature, CAN was the main cause of DCGL (48.48%) [8]. Of graft failures, 78.78% developed after 3 months of transplantation and CAN constituted 61.53% of them. CAN is the histologic description of the fibrosis, vascular and glomerular damage occurring in kidney allografts, and is independent of underlying etiology [11,12]. Actually, it refers to a clinical situation that causes graft loss, which could not be fully elucidated, with these gathered together under the collective heading. Transplant programs are based on monitoring the change in serum creatinine for identification of the patients at risk for CAN, but this change occurs

**Table 2.** The characteristics of the donors and recipients.

| Characteristics | Mean ± SD or (n) |
|---|---|
| Age (recipient, year) | 33.54 ± 15.37 |
| Gender (recipient) | |
| Female | 16 |
| Male | 17 |
| Co-morbid disease (recipient) | |
| Yes | 17 |
| No | 16 |
| Causes of ESRD | |
| Idiopathic | 16 |
| Hypertensive nephropathy | 2 |
| Diabetic nephropathy | 1 |
| MPGN | 3 |
| Nephrolithiasis | 4 |
| Polycystic kidney disease | 2 |
| dRTA secondary to VUR | 1 |
| Renal agenesis | 1 |
| Neurogenic bladder | 1 |
| Alport Syndrome | 1 |
| Familial Mediterranean fever | 1 |
| Pre-transplantation RRT | |
| Preemptive | 8 |
| HD | 18 |
| PD | 4 |
| HD-PD | 3 |
| Mean duration of dialysis (month) | 60.8 ± 76.38 |
| Sources of Donor Kidneys | |
| Living donors | 19 |
| Deceased donors | 14 |
| Age (donor, year) | 46.57 ± 22.08 |
| Gender (donor) | |
| Female | 18 |
| Male | 15 |
| Extended criteria donor | |
| Yes | 19 |
| No | 14 |
| Ischemia time | |
| Warm ischemia time (second) | 180.5 ± 73.5 |
| Cold ischemia time (minute) | 1299 ± 378 |
| Delayed graft function | |
| Yes | 8 |
| No | 25 |
| Mean duration of graft survival (month) | 36.5 ± 31.98 |

MPGN: Membranoproliferative glomerulonephritis, dRTA: Distal renal tubular acidosis, VUR: Vesico-ureteric reflux, RRT: Renal replacement therapy, HD: Hemodialysis, PD: Peritoneal dialysis.

usually late in the course of the disease, and overlooks the severity of the pathological change. Histological evaluation prior to transplantation and afterwards may not be possible in every transplant center as in our center.

Early-graft loss is a catastrophic event that is assumed to occur as a result of vascular thrombosis, acute rejection, primary non-function, and urological complications [13]. In current practice, graft loss due to rejection in the first 3 months of KT is unusual [13]. However, in this study, early-graft loss constituted 21.21% of all cases, and immunological rejections accounted for 42.85% of early-graft loss. As a result of immunological advances, graft loss due to hyperacute rejection has been considered so unusual that some authors did not deem it worthwhile to comment on it [13]. Unfortunately, in our series hyperacute rejection developed in one patient (3.03%) due to an ABO-incompatible KT. In fact, this case was one of medical malpractice rather than immunological rejection. Our clinic adopted current strategies, which were defined to prevent allograft rejection. However, ABO incompatible KTs are not performed in our center because they are not covered by the requisite medical insurance. The information regarding blood groups of the transplant pairs had been uploaded incorrectly to the laboratory information system. Since we assumed that we would perform ABO-compatible KT, the immunosuppressive protocol was not adjusted to that of an ABO-incompatible one. It was one of the preventable factors for graft loss, which transplant team has to resolve. From that point onwards, which was in the early stage of the KT program, the transplant team has adopted a comprehensive control mechanism, including final confirmation of the laboratory results just before transplantation, irrespective of the previous laboratory records. If ABO-incompatible KT is to be planned, optimization of desensitization and patient-tailored immunosuppressive regimens are required to achieve better outcomes [14]. The other two patients with early graft loss were treated as if the underlying factor was immunological rejection, after all other causes were excluded, though protocol biopsy could not be performed. In our center, immunological rejections accounted for 18.18% of graft loss, half of those developed in early period. According to the Joint Report of the Ministry of Health and the Turkish Society of Nephrology, the rate of acute rejection within the first 6 months of transplantation was 13.38% [6]. Lack of protocol biopsy was an important obstacle to obtain better outcome. We think that protocol biopsy should be applicable in all transplant centers.

Non-adherence to the immunosuppressive regimen leads to rejection, graft loss and even death [15]. Its prevalence in KT recipients varies between 20% and 70% [16]. Non-adherent recipients to immunosuppressive medications have 7-fold increase in risk of graft failure [17]. It is a preventable cause of the graft loss, and accounted for 33% of graft rejection in the current study, all of them developed in late period. Early detection of risk factors, which were explained by Belaiche et al, [15], (age below 50, male gender, low social support, unemployment, poor education, more than 3 months after transplant, living donor, re-transplant, more than 6 co-morbidities, more than 5 drugs/day, more than 2 intakes/day, negative beliefs, negative behaviors and negative satisfaction, depression, and anxiety) is essential to overcome non-adherence.

**Table 3.** The causes of death-censored graft loss among kidney transplant recipients.

| | | | Hyperacute phase (n) | Acute phase (n) | Chronic phase (n) | Total (n) |
|---|---|---|---|---|---|---|
| CAUSES | Pre-renal | Medical problems | | | | 7 |
| | | Congestive heart failure | (-) | (-) | 2 | |
| | | Surgical complications | | | | |
| | | Vascular thrombosis | (-) | 2 | (-) | |
| | | Renal artery pseudoaneurysm | (-) | 2 | (-) | |
| | | Iliac artery dissection | (-) | (-) | 1 | |
| | Renal | Medical problems | | | | 25 |
| | | Recurrence of the primary disease | (-) | (-) | 1 | |
| | | BKVN | (-) | (-) | 2 | |
| | | Immunological problems | 1 | 2 | 3 | |
| | | CAN | (-) | (-) | 16 | |
| | Post-renal | Medical problems | | | | 1 |
| | | VUR + BKVN | (-) | (-) | 1 | |
| | Total (n) | | 1 | 6 | 26 | 33 |

CAN: Chronic allograft nephropathy, BKVN: BK virus nephropathy, VUR: Vesico-ureteric reflux.

**Table 4.** The surgical complications and their management.

| | Surgical complications | (n) | Management | (n) |
|---|---|---|---|---|
| Lymphovascular | Vascular thrombosis | 2 | Reoperation (explantation of the renal graft) | 2 |
| | External iliac artery dissection | 1 | Reoperation (the internal iliac artery was used for reanastamosis) | 1 |
| | Lymphocele | 2 | Percutaneous drainage & Sclerotherapy | 2 |
| | Renal artery pseudoaneurysm | 2 | Reoperation (explantation of the renal graft & resection of pseudoaneurysm with vascular reconstruction) | 2 |
| | Lymphocele with intra-abdominal abscess | 1 | Percutaneous drainage & Reoperation | 1 |
| Renal & Perirenal | Hematoma | 4 | Conservative management | 1 |
| | Perirenal abscess | 1 | Reoperation | 3 |
| | | | Drainage | 1 |
| Urological | Ureteral leakage | 1 | Percutaneous nephrostomy and double -J- catheter placement | 1 |
| | Ureteral obstruction | 5 | Percutaneous nephrostomy and double –J- catheter placement | 4 |
| | Ureteral obstruction with pyelitis | 1 | Percutaneous nephrostomy and double –J- catheter placement & Reoperation | 1 |
| | Ureteral obstruction with VUR | 1 | Percutaneous nephrostomy | 1 |
| | | | Percutaneous nephrostomy and double –J- catheter placement & Reoperation | 1 |

VUR: Vesico-ureteric reflux.

Vascular thrombosis, which developed in two patients, who had undergone en-bloc and dual KT, and renal artery pseudoaneurysm, which developed in two patients, who had received the graft from the same deceased donor, in whom pseudomonas species were isolated from the tracheal aspirate, were the surgical causes (57.15%) of early-graft loss. Early-graft loss after KT may be more likely after the use of kidneys from suboptimal donors [18]. There is

an organ supply shortage in our country and living kidney donation is the main graft source. Thus, we have to procure the kidney grafts from extended criteria donors to increase the organ pool. Extended criteria donor kidneys constituted 57.6% of the graft pool in the current study. There are no universal criteria defining extended criteria donors. It refers higher risk compared with a standard donor. The risk could be a disadvantage in the future not only for recipients but also for living donors. According to our criteria, which were previously described, any deceased donor candidate with infection (except those with sepsis) is eligible for donation under antimicrobial coverage. As known, the culture results of deceased donors can mostly be obtained after transplantation, as in the current study, due to laboratory procedures. Pseudomonas species were isolated from the blood cultures of both recipients. Although a culture-specific antibiotic regimen was started as soon as the culture result of the donor was obtained, renal artery pseudoaneurysm was not inevitable in either of the recipients. Renal artery pseudoaneurysm after KT is a rare complication and usually secondary to infection or technical default [19]. Infectious anastomotic pseudoaneurysms described in the literature (usually an opportunistic microorganism in origin) are limited to case reports and/or case series and do not exceed 30 cases [20]. Early diagnosis is a challenge, especially in fungal infections, which may be indolent for months [20]. Serial radiologic investigations and routine blood cultures may be implemented, especially in recipients of infected donors. Graft nephrectomy and resection of pseudoaneurysm with vascular reconstruction is the gold standard therapy in patients with large vascular defects [20].

Medical problems accounted for 23.07% of late-graft loss, and BKVN was responsible for half of these cases. With the use of more potent immunosuppressive regimens, BKVN has increasingly been recognized as resulting in allograft damage [21]. Recurrence of the primary disease and congestive heart failure were the other medical problems. The incidence of de novo congestive heart failure in kidney transplant recipients is 2–5 times higher than the incidence in the general population [22]. Hypoperfusion led to late-graft loss in two patients with de novo congestive heart failure.

This was a descriptive study without a comparator. Thus, predictors of the graft loss were not evaluated. The lack of protocol biopsy was another limitation of the study.

## Conclusion

In conclusion, there are several preventable factors for graft loss, which transplant teams have to resolve, these include creation of an effective training program to prevent malpractice, ensuring that patients are fully informed before transplantation to improve their adherence to immunosuppressive therapy, close monitoring of the patients to resolve their medical problems, adoption of a protocol biopsy and immunological support, and improving the management of the potential organ donor. There are many unresolved variables in KT and many problems, which remain to be elucidated in order to achieve long-term graft survival. Identification of the true causes of graft failure described under the heading CAN is noteworthy. Comprehensive biochemical, physiological, pathological, immunological, and genetic research should be implemented to remove the obstacles in KTs.

### *Ethical approval*

Ethical approval was received for this study from Inonu University Health Sciences Non-invasive Clinical Research Ethics Committee (approval number: 2018/7-11).

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