Impact of Recipient Obesity on Kidney Transplantation Outcome: A Retrospective Cohort Study with a Matched Comparison

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ABSTRACT

Background. The aim of this study was to evaluate the effect of a recipient's obesity on post-transplant complications and patient and graft survival.

Methods. A single-institution, retrospective study was performed on obese renal transplant recipients (BMI ≥ 30 kg/m^2, n = 102) from January 2010 to December 2018, matched with non-obese recipients (BMI < 30 kg/m^2, n = 204). For comparison, for every obese patient we selected 2 nonobese patients with a similar age, sex, and period of transplantation. The comparative analysis included patient and graft survival as primary outcomes and graft function and postoperative complications as a secondary outcome.

Results. Recipient demographics were comparable in both groups except for diabetic nephropathy in obese patients (P = .0006). Obesity was strongly related to a poorer patient survival (risk ratio [RR] = 2.83 confidence interval [CI] 95% 1.14-7.04; P = .020) but there was no observed difference in graft survival (P = .6). While early graft function was inferior in the obese population (RR = 2.41; CI 95% 1.53-3.79; P = .00016), during late follow-up, no statistically significant differences were observed between both groups (P = .36). Obese recipients had a significantly higher risk of delayed graft function (RR = 1.93; CI 95% (1.19-3.1), P = .0077), heart infarction (RR = 7; CI 95% 1.68-29.26; P = .0042), wound infections (RR = 8; CI 95% 1.96-32.87; P = .0015), diabetes aggravation (RR = 3.13; CI 95% 1.29-7.6; P = .011), and surgical revision for eventration (RR = 8; CI 95% 1.22-52.82; P = .026) when compared with nonobese recipients.

Conclusions. Despite the inferior early kidney graft function in obese recipients, there was no difference observed at the long-term follow-up. However, recipient obesity demonstrated a negative effect on patient survival and postoperative complications.

OBESITY, defined as a body mass index (BMI) of 30 kg/m^2 or greater, has increased markedly worldwide since 1980 [1,2]. This increase is also reflected in the population of patients with end-stage renal disease (ESRD) presented as candidates for kidney transplantation [3−5]. Therefore, the transplant community needs to balance the obesity-associated comorbidity of the transplant procedure for each individual obese kidney transplant candidate and needs to determine the right strategy to lose weight before transplantation.

The severity of a recipient's obesity is directly correlated with a longer transplant surgery and an increased incidence of complications afterwards [6,7]. Obesity is associated with a higher risk of delayed graft function (DGF), new-onset diabetes after transplant (NODAT), and the aggravation of preexisting diabetes [8−11]. However, the influence of a recipient's obesity on graft and patient survivals and on the associated increased

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risk for postoperative complications is still under debate [7,12,13].

The aim of this study was to evaluate the influence of a recipient's obesity after isolated kidney transplantation on patient and graft survival and immediate postoperative complications.

METHODS

Patients

Kidney transplant recipients with BMI ≥ 30 kg/m^2 (calculated at the day of transplantation) performed between January 2010 and December 2018 at the Université Catholique de Louvain, Cliniques Universitaires Saint-Luc, Brussels, Belgium, were included in this retrospective study and matched for comparison with 2 nonobese recipients (BMI < 30 kg/m^2) according to 3 demographic parameters: age, sex, and transplantation period. The study was approved by the local ethical committee.

The following donor and recipient characteristics were analyzed: donor age and sex, graft origin, primary nephropathy, duration of pretransplant dialysis, ischemia times, incidence of delayed graft function (DGF), graft failure and graft rejection, the length of hospitalization following transplantation, the hospital admissions up to 1 year after transplantation surgical, medical and metabolic complications, and patient and graft survival. The graft function was evaluated using the estimated glomerular filtration rate (eGFR, CKD-EPI) [14], and serum creatinine levels during the first 14 days, 3 and 6 months, and 1 and 2 years. DGF was defined as the need for dialysis during the first week after transplantation.

In order to identify the effect of a recipient's obesity on the early graft function, defined as the maximum eGFR measured during the first 14 days after transplantation, all patients, obese and nonobese, were stratified in 3 groups according to their initial kidney graft function: 1) "Poor" initial graft function defined as DGF or maximum eGFR for the first 14 days < 20 mL/min; 2) "Acceptable" initial graft function defined as the absence of DGF and a maximum eGFR between 20 and 40 mL/min during the first 14 days after transplantation; and 3) "Good" initial graft function defined as the absence of DGF and a maximum eGFR of > 40 mL/min during the first 14 days after transplantation. We then identified the number of obese patients in each group to correlate them with the non-obese patients.

A triple-drug protocol consisting of tacrolimus (Advagraf, Astellas Pharma BV, Brussels, Belgium) with target trough levels of 10-14 mg/L after transplantation and with progressive reduction in the following months, mycophenolate mofetil (Cellcept, NV Roche SA, Brussels, Belgium), and methylprednisolone (Medrol, Pfizer NV, Brussels, Belgium) was used during the whole study period. In addition, induction therapy with basiliximab (Simulect, Novartis Pharma GmbH, Neurenberg, Germany) on days 0 and 4 after transplantation was used in recipients of a living donor graft. Plasmapheresis was applied in immunized recipients until 1 month after transplantation. Sensitization was based on the pretransplant patient panel reactive antibody historical sera.

Statistical Analysis

Categorical variables were compared with χ^2 tests; for the comparisons of 2 proportions, we used the Cook's correction; the confidence intervals of risk ratio were computed according to Koopman [15]. Relations of dichotomic variables were computed by logistic regressions. Ordinal variables were compared with the Kolmogorov-Smirnov test. For the regressions of ordinal variables, we used the maximum likelihood method according to Snell [16]. Continuous variables were compared with the Student t test, with the Welch's correction for unequal variances; if needed the data were normalized by logarithmic transformation. Relations of continuous variables were computed by linear regressions; in the case of repeated measurements, we used a mixed linear model, the individual patients being considered as a random variable. The survival curves are Kaplan-Meier estimates. For the regressions we used the Cox's proportional hazard model. In all statistics two-sided tests were used, and the results were considered statistically significant if P value was < .05. Analyses were carried out using the Statistical Package for the Social Sciences (SPSS) version 22 (IBM, Armonk, NY, USA).

RESULTS

Demographic Data

Between 2010 and 2018, 760 kidney transplant recipients were operated on at our institution. There were 102 recipients who were classified as obese (BMI ≥ 30) and matched with 204 nonobese recipients (BMI < 30) presenting a similar age, sex, and period of transplantation. The recipient demographics were comparable between both study groups except for a higher incidence of diabetic nephropathy in the obese group (P =.0006; Table 1). The median follow-up was 40.7 months (quartiles 1-3: 25.7-68.9).

Patient and Graft Survivals

Patient survival at 1, 3, and 5 years was 99.0% (± 0.7), 97.3% (± 1.2), and 96.0% (± 1.8) and 98.0% (± 1.4), 91.4% (± 3.2), and 87.2% (± 4.3), respectively, in the nonobese and the obese group (logrank P = .020). Both overall patient survival (risk ratio [RR] = 2.83 confidence interval [CI] 95% 1.14-7.04; P = .020) and patient survival with functioning grafts (RR = 2.05 CI 95% 1.05-4.01; P = .033) were significantly different between the 2 groups of recipients in favor of nonobese individuals (Fig 1).

The cumulative mortality risk is compatible with a linear trend in both groups (P = .81; Fig 2).

No critical time points were detected. Eleven patients died in the obese group and 8 in the control group. The most common causes of death were infections (4 out of 11 patients in the obese group and 4 out of 8 patients in the non-obese group) and tumors (3 of 8 patients in the obese group and 4 of 8 patients in the non-obese group). The remaining causes of death in the obese group were cardiovascular (n = 1), suicide (n = 1), car accident (n = 1), and of unknown cause (n = 1).

Graft survival at 1, 3, and 5 years after transplantation was 95.0% (± 2.2), 95.0% (± 2.2), and 90.5% (± 3.8) and 97.5% (± 1.1), 95.2% (± 1.6), and 94.1% (± 1.9), respectively, in obese and nonobese recipients (P = .6, NS). Death-censored graft survival demonstrated no significant difference between both study groups (P = .6), (Fig 3).

Graft Function

Early graft function affects graft survival as illustrated in Fig 4. An eGFR value below 20 mL/min or the need of dialysis during the first week after transplantation is associated with a poorer graft survival as compared to an acceptable (20 mL/min ≤ eGFR ≤ 40 mL/min) or good (eGFR > 40 mL/min) early renal function (HR 3.09, CI 95% 1.84-5.17). Table 2 summarizes the

Table 1. Kidney Transplant Demographics According TO Body Mass Index of 102 Obese Recipients Matched With 204 Nonobese Recipients for Age, Sender and Transplant Period in a Retrospective Single Center Transplant Program From January 2010 Until December 2018

	BMI < 30 n = 204	BMI ≥ 30 n = 102	*P* value
Recipient age, y	54.52 ±1.20	54.68 ±0.86	.92*
BMI, mean	23.93 ± 2.91	32.46 ± 2.27	
Sex, n			
Male	126	62	.87†
Female	78	40	
Pretransplant dialysis, n	165	93	.22‡
Pre-emptive transplant, n	39	9	.27‡
Primary diabetic nephropathy, n	20	25	.00064†
Primary non-diabetic nephropathy, n	184	77	.18†
Rank transplants, n			.012‡
1	183	99	
2	19	3	
3	2	0	
Donor Organ Source, n			.28†
Living	54	22	
DBD	119	69	
DCD	31	11	
Donor Age, y (+/-SD)	45.52 ± 0.80	46.69 ± 1.17	.42*
HLA Mismatches (A, B, DR), mean	2.51	2.80	.066*
CMV status, n			.87†
D+R-	48	21	
D+R+	68	32	
D-R-	35	20	
D-R+	47	26	
Sensitized anti-HLA patients, n	59	11	.88†
Immunosuppression, n			.24†
Tac, MMF, CS	115	67	
Induction	55	24	
HI protocol	34	11	
Total ischemia time, h (+/-SD)	13 (5.5-12)	12 (7.5-16,5)	.056‡
Observational time, mo (+/-SD)	39.3 (22.8-70.4)	35,8 (22.1-63.6)	.6‡

BMI, body mass index; HLA, human leukocyte antigens; CMV, cytomegalovirus; CS corticosteroids; D, donor; DBD, donation after brain death; DCD, donation after circulatory death; HI, hyperimmunized; MMF, mycophenolate mofetil; R, recipient; Tac, tacrolimus.

* Student t test.

† χ^2 test.

‡ Kolmogorov-Smirnov test.

number of obese and nonobese patients in each category. Among patients who presented a poor early graft function, a statistically significant percentage of these were obese patients (RR = 2.41; CI 95% 1.53-3.79; *P* = .00016).

During the first 2 weeks the mean eGFR of obese patients who did not require dialysis rose progressively from 9.7 to 36.5 mL/min and from 11.8 to 42.8 mL/min for the nonobese patients. The eGFR ratio being approximatively constant with a mean value of 0.802 (CI 0.682-0.943; Fig 5). Mean ratio was significantly less than unity (*P* = .009). No statistically significant difference was identified in controls at 3 and 6 months, and at 1 and 2 years in both groups (*P* = .36), (Fig 6).

Postoperative Complications After Transplantation

Obesity demonstrated to be a risk factor of DGF (RR = 1.97; *P* = .008) without an effect on the hospitalization length (*P* = .11; Table 3). No differences were noted in terms of acute rejection during the follow-up in both groups (*P* = .99). The incidence of heart infarction after transplantation is statistically higher in obese patients compared with nonobese patients (RR = 7.00; *P* = .005). Obesity has not been demonstrated as a risk factor in the onset of de novo diabetes after transplantation, but obese diabetic transplanted patients become more frequently insulin-dependent (RR = 3.13; *P* = .011). An increased risk of infections in the obese population, in particular abdominal wall infections, has also been observed (RR = 8.00; *P* = .002) compared with the nonobese patients. No differences between both study groups were identified in terms of urinary tract infections, postoperative bleeding, or eventrations. Although there are no statistically significant differences in wound complications, obese patients who developed a dehiscence following transplantation had a higher risk of surgical revision (*P* = .026). No complications were noted in relation to vascular anastomosis or uretero-vesical anastomosis in the overall study population.

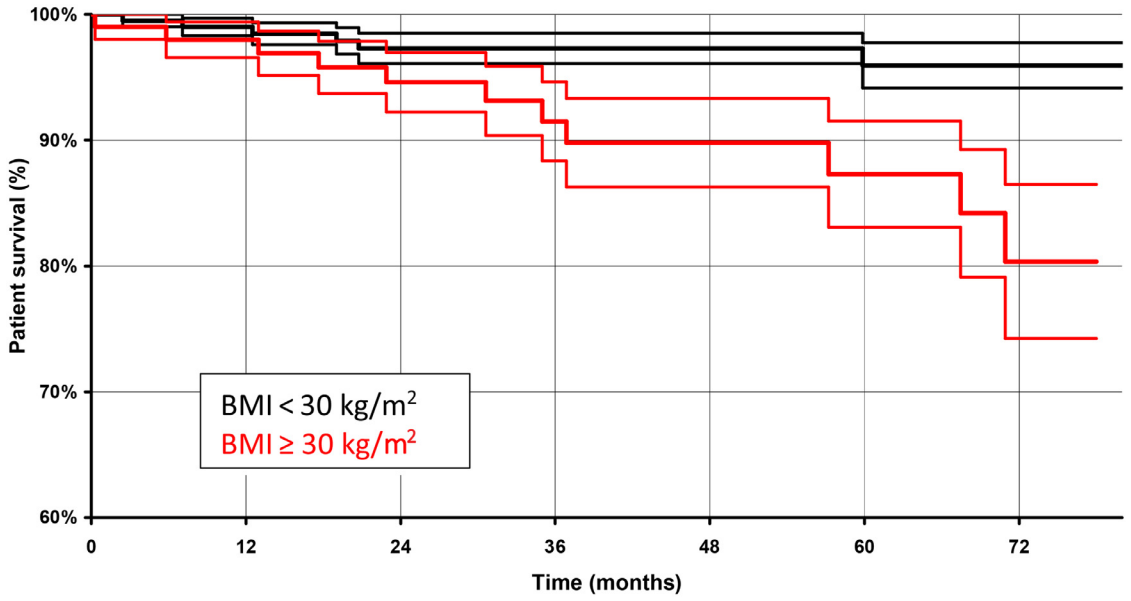

Fig 1. Patient survival of 102 obese recipients matched with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 until December 2018. Kaplan-Meier survival curves (thick lines); SD (thin lines); n = 204 nonobese patients (black lines); n = 102 obese patients (red lines); RR = 2.83 CI 95% 1.14-7.04; *P* = .020 (logrank). BMI, body mass index; SD, standard deviation; RR, risk ratio; CI, confidence interval; *P, P* value.

DISCUSSION

In this retrospective single center analysis, recipient obesity demonstrated no influence on late graft function compared with nonobese patients. However, we observed an inferior early graft function, patient survival, and a higher incidence of postoperative complications in the obese population.

In line with our findings, a recipient's obesity was associated with an increased risk of death compared with recipients with a

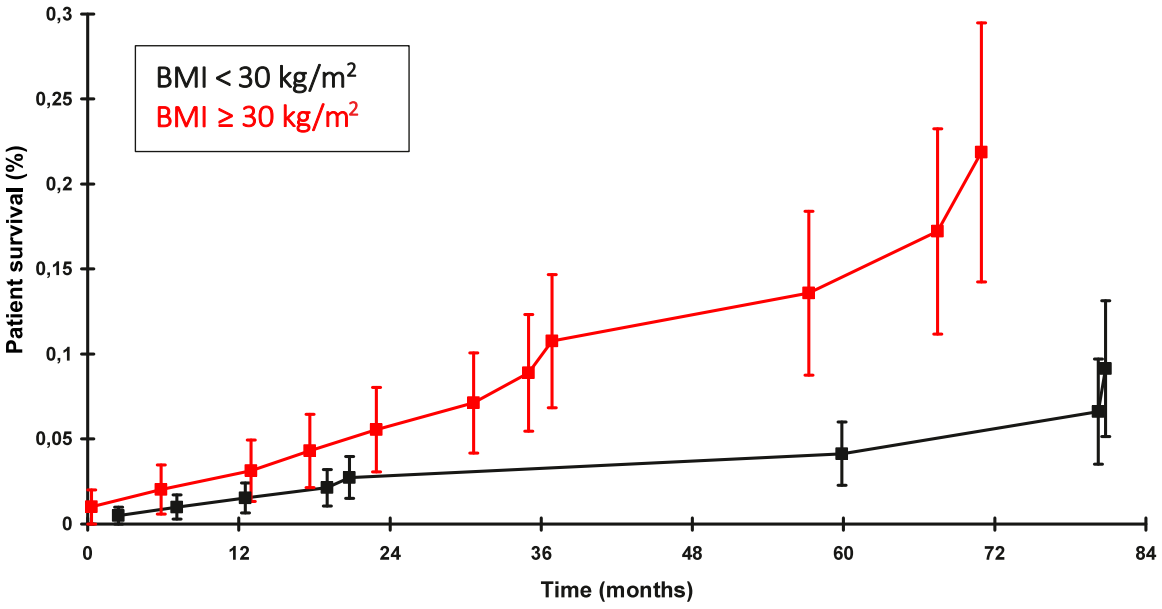

Fig 2. Cumulated risk of death of 102 obese recipients matched with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 to December 2018. n = 204 nonobese recipients (black); n =102 obese recipients (red); *P* = .81. SD, standard deviation; *P, P* value.

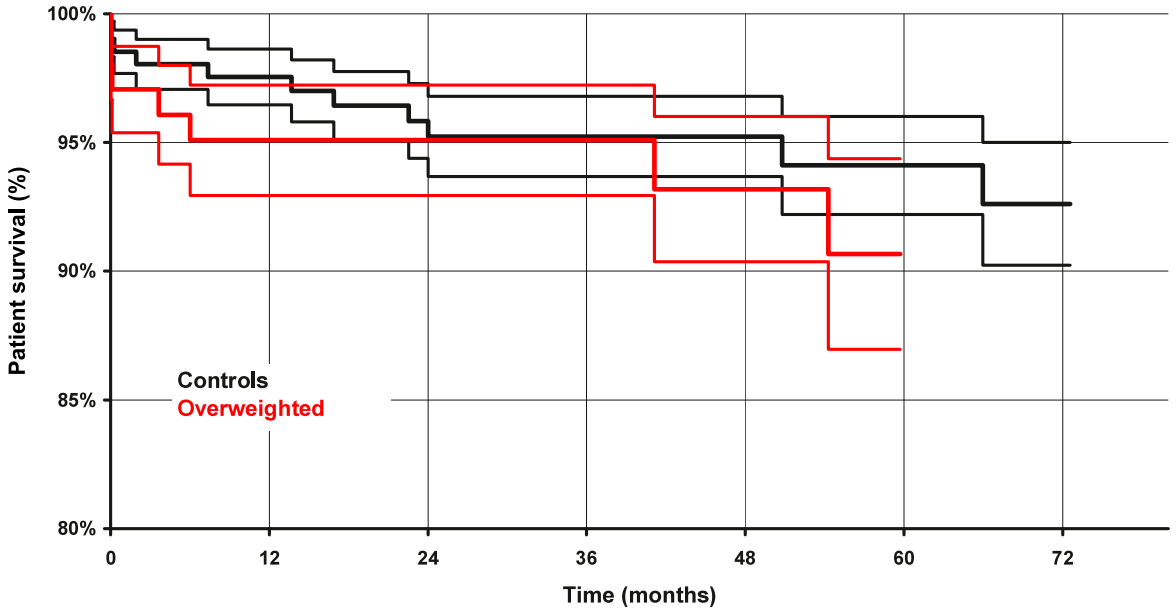

Fig 3. Death censored graft survival of 102 obese recipients matched transplant with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 to December 2018. Kaplan-Meier survival curves (thick lines); ± SD (thin lines); n = 204 nonobese patients (black lines); n = 102 obese patients (red lines), *P* = .6 (logrank).

normal BMI [7,17]. This appears intuitive because of the increased prevalence of common comorbidities such as hypertension and diabetes mellitus in obese individuals [18−20]. However, it is interesting to highlight 2 considerations. First, the causes of death found in our study were not directly related to cardiovascular or metabolic causes but to infections and tumors in obese patients as well as in the control group. This could be related to the immunosuppression dosage, and drug monitoring in obese patients, including variations in the distribution of medication and the accurate estimation of renal

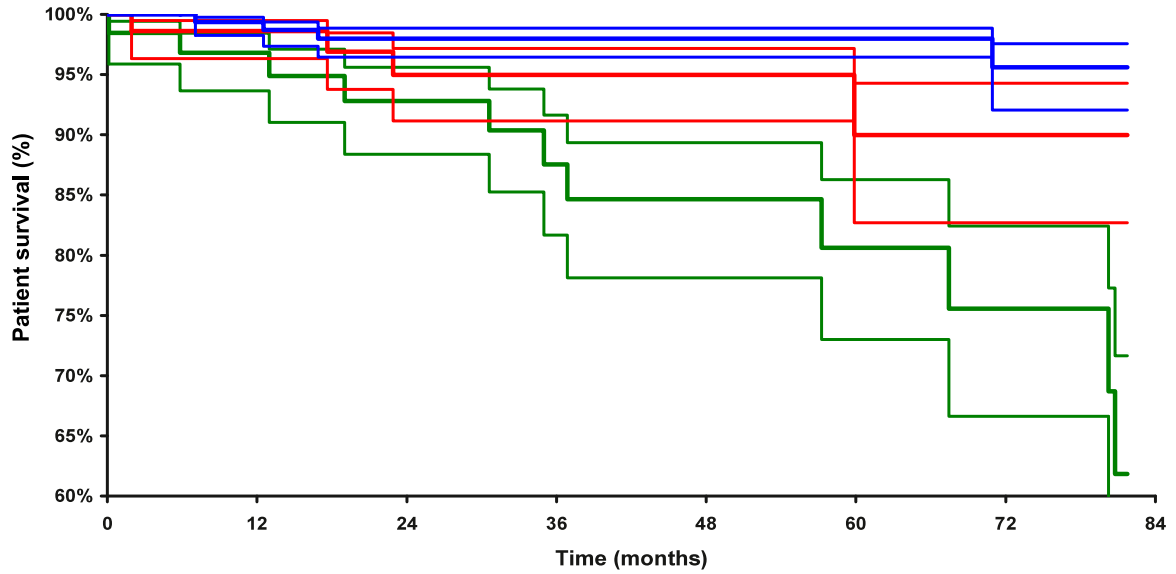

Fig 4. Initial graft function predicts late graft survival of 102 obese recipients matched with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 to December 2018. Kaplan-Meier survival curves (thick lines); ± SD (thin lines); n =161 patients, eGFR > 40mL/min (blue lines); n = 73 patients, 20mL/min ≤ GFR ≤ 40mL/min (red lines); n =71 patients, eGFR < 20mL/min or need of dialysis (green lines); *P* < .0001 (logrank); n =1 patient was excluded because of the lack of available data. SD, standard deviation; eGFR, estimated glomerular filtration rate; GFR, glomerular filtration rate; *P, P* value.

Table 2. Early Graft Function Defined as Maximum eGFR During the First 14 Days Posttransplantation.

	BMI < 30 n = 203*	BMI ≥ 30 n = 102	*P* value	RR	CI 95%
Poor (GFR < 20 mL/min or need of dialysis)	36	35	.00016	2.41	1.53-3.79
Moderate (20 mL/min < GFR < 40 mL/min)	45	28			
Good (GFR > 40 mL/min)	122	39			

BMI, body mass index; eGFR, estimated glomerular filtration rate; GFR, glomerular filtration rate; RR, risk ratio; CI, confidence interval.
* One nonobese patient was excluded because of lack of available data.

function [21−23]. Second, the cumulative mortality risk evolved with a linear trend, suggesting a stable instantaneous risk of death during the follow-up. This means that the mortality risk is not related to a precise moment after transplantation even during the postoperative period. In contrast, other authors have related the increased mortality of obese recipients directly to the surgical procedure, which evidence shows to be longer, more complex, and more at risk of complications [24]. According to our experience, the risk of mortality appears independent of the early phases after transplantation.

Our study did not demonstrate the negative effects of obesity on graft survival, but clearly demonstrated that obese patients have a higher risk of developing poor initial graft function. The obese kidney transplant recipient with good initial graft function will have a similar graft survival to nonobese patients. These findings contrast with previous comparative studies concerning the possible relationships between obesity and graft survival showing the negative effect of excess weight on kidney survival duration [25,26].

These results are likely because of some combination of visceral fat effect, lean muscle mass or sarcopenia, frailty, propensity for diabetes, and other obesity-related comorbidities.

The association between obesity and transplantation outcomes has been also recently investigated in several meta-analyses and systematic reviews. A large study of 51,927 US renal transplant recipients presented a very strong association between BMI and kidney transplant outcome [27]. Both very high and very low BMI values were significantly correlated with worse patient and renal graft survival.

Several authors linked this relationship to the increased occurrence of chronic allograft nephropathy (CAN) usually related to chronic rejection. Many reasons have been cited to explain why obese recipients are more often affected by CAN [28,29]. Increased glomerular filtration rates to maintain homeostasis, leading to glomerulosclerosis, diabetic nephropathy, and high blood pressure and dyslipidemia, occurs more frequently among obese patients; lack of weight proportionally

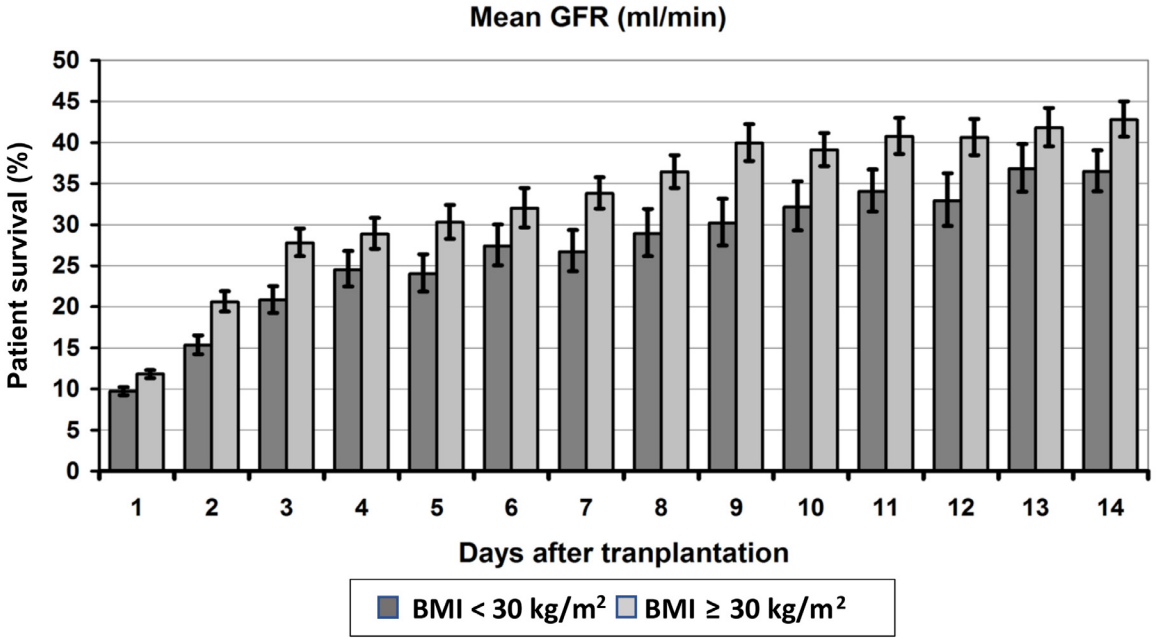

Fig 5. Early graft function predicts late graft survival of 102 obese recipients matched with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 to December 2018. Mean eGFR during the first 14 days after transplantation in obese (n = 102) and nonobese (n = 203) patients. Kolmogorov-Smirnov test, *P* = .016. GFR, glomerular filtration rate; *P, P* value.

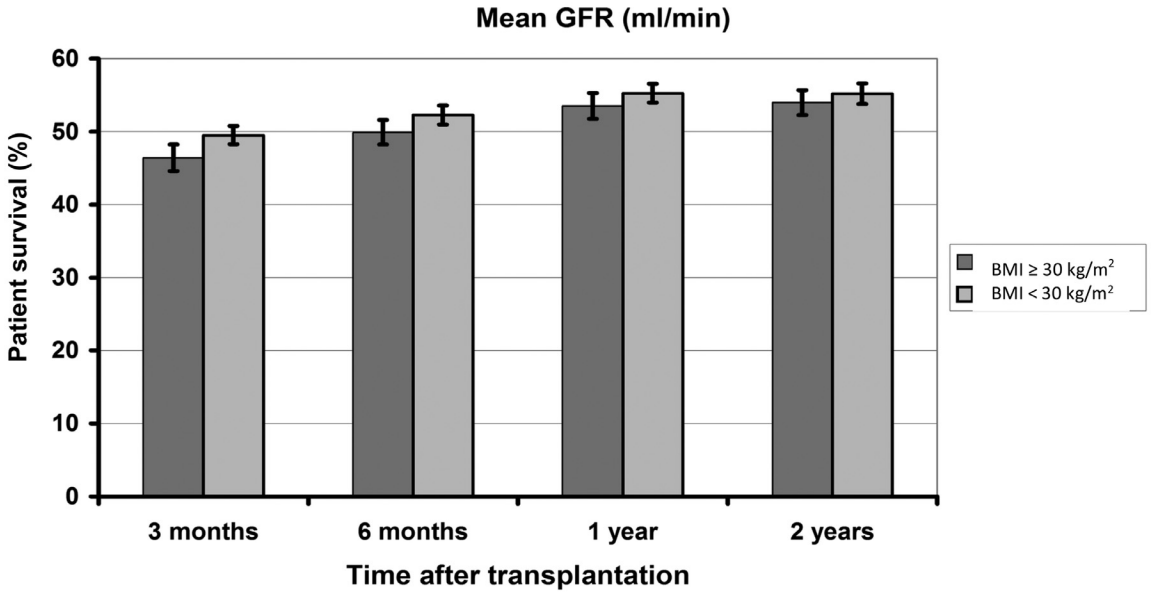

Fig 6. Late graft function predicts late graft survival of 102 obese recipients matched with 204 nonobese recipients for age, sex, and transplant period in a retrospective single center kidney transplant program from January 2010 to December 2018. Mean eGFR during the first 14 days after transplantation in obese (n = 102) and nonobese (n = 203) patients. Kolmogorov-Smirnov test, *P* = .36. eGFR, estimated glomerular filtration rate; *P, P* value.

between recipient and donor and DGF seem to be the principal risk factors for CAN. On the other hand, no significant difference in graft loss in obese recipients was found in more recent studies [12,30]. A meta-analysis by Lafranca et al presented confusing results because some studies showed that 3-year patient and graft survival were significantly worse in recipients with BMI 30 kg/m2, and regression analysis indicated better patient survival in the group of obese patients [31].

Very few studies have evaluated the problem of early posttransplant function in the group of obese kidney recipients. In our study early graft function immediately after transplantation was inferior in the obese compared to nonobese patients. This may be explained by a higher rate of complications that could negatively affect initial graft function. Obesity did not influence middle term results in our study compared with non-obese patients. It is conceivable that it can be explained by the advances in

Table 3. Overall Comorbidity of 102 Obese Recipients Matched With 204 Nonobese Recipients for Age, Sex, and Transplant Period in a Retrospective Single Center Kidney Transplant Program From January 2010 to December 2018

	BMI < 30 n = 204	BMI ≥ 30 n = 102	*P* value	RR (95% CI)
Length of hospitalization, d	10	11	.11	-
Acute rejection, n (%)	12	6	.99	1 (0.4-2.48)
DGF, n (%)	27	26	.0077	1.93 (1.19-3.1)
Cardiovcascular, n (%)	14	11	.24	1.57 (0.75-3.27)
heart infarction, n (%)	2	7	.0042	7 (1.68-29.26)
dysritmia, n (%)	8	3	.67	0.75 (0.22-2.53)
Metabolic, n (%)	11	17	.00064	1.88 (1.33-2.53)
de novo diabete, n (%)	4	6	.07	3 (0.93-9.7)
diabete aggravation, n (%)	7	11	.011	3.13 (1.29-7.6)
Infections, n (%)	31	25	.048	1.61 (1.01-2.56)
Urinary Infections, n (%)	20	14	.31	1.4 (0.74-2.61)
Wall Infections, n (%)	2	8	.0015	8 (1.96-32.87)
Surgical, n (%)	26	18	.26	1.38 (0.8-2.37)
Eventrations, n (%)	5	6	.13	2.4 (0.79-7.24)
Surgical revisions for eventration, n (%)	1	4	.026	8 (1.22-52.82)
Urinary Complications, n (%)	18	7	.56	0.77 (0.34-1.73)
Surgical revisions for bleeding, n (%)	11	8	.41	1.45 (0.62-3.39)

BMI, body mass index; CI, confidence interval; DGF, delayed graft function; RR, risk ratio.

immunosuppressive therapy along with the improvement in general medical practice exemplified by better control of comorbidities such as lipid disorders, hypertension, diabetes, cardiac conditions, and others and the gain of experience in kidney transplantation [30].

However, as recently suggested by Schold et al [32] the obesity-associated risks in transplant recipients may vary significantly depending on age, ethnicity, diagnosis, and sex. Therefore, contraindications should not be based on absolute BMI thresholds but modified based on patient characteristics.

Obesity and Outcomes After Transplantation

As reported by others [11], we observed a higher incidence of DGF in obese recipients compared to nonobese individuals. The increased DGF rate in the obese transplanted population may be related to a longer operative time and a longer warm ischemic time [33−35], higher sympathetic activity which results in renal vasoconstriction [36,37], increased risk of overdose of calcineurin inhibitors [23], and the increased prothrombotic activity and endothelial dysfunction [38] linked to the obese population.

Technical issues during the transplant procedure are additional risks for complications in obese recipients [24] (eg iliac veins access, iliac arteries when involved by an atherosclerotic process, and even the uretero-vesical anastomosis). Caval extension in deceased donor right kidneys could be a solution to lengthen the graft vein leading to easer graft positioning and grafting. Among our series occurrence of urinary or vascular complications did not seem to be increased in obese patients compared with nonobese patients.

Nevertheless, our study demonstrated a statically higher rate of wound infections and a higher requirement for surgical revision in obese patients in line with previously reported studies [7,12]. These increased complication rates seem to be related to multiple factors: longer intervention times, larger incisions, reduced resistance of fat tissues to in situ infection, and stronger retraction of the abdominal wall during surgery.

Moreover, obesity leads to the development of insulin resistance and, therefore, the appearance of diabetes mellitus, atherosclerosis, and the onset of cardiovascular diseases [9,18]. In line with other authors [2,8], in our experience, renal transplantation exposes obese patients to a deterioration of these preexisting comorbidities, particularly related to the effects of immunosuppressive treatment, more frequently than with nonobese patients. In our analysis, we did not identify differences in the rates of acute rejection episodes. A possible correlation between obesity and acute rejection is discussed in some studies and may be caused by inflammation and a modified immune response [7,39].

Limitations

This study has limitations because of the retrospective nature of the data collection. Not all possible risk factors of kidney recipients, especially environmental, behavioral, and psychological, were available to be included in the analyses. Obese patients were considered as a single group, and not considering the effect of the degree of severity of obesity and outcome. Moreover, we were limited to taking a "picture" of a patient's BMI, obese and nonobese, at the time of transplantation, without considering the evolution of a patient's weight during the follow-up. Finally, over the last decade, it has been increasingly recognized that BMI is an imperfect measure of adiposity and that the inclusion of the muscle mass or the waist circumference would allow for more analysis [13]. There are more patients who have a metabolic syndrome in the obese recipient group than in the non-obese recipient group. However, although the metabolic syndrome may be a confounding factor, from a clinical point of view, obesity must be considered in its complexity, considering all the comorbidities of which obesity is a risk factor.

Therapeutic Strategy for Kidney Transplant Candidates With Obesity

Various options should be proposed to obese patients who are candidates for kidney transplantations, including behavioral, pharmacologic, and surgical approaches. Behavioral interventions that address both diet and physical activity show small but significant benefits on weight loss maintenance [40]; however, a considerable number of patients cannot reach the target weight either because of poor compliance [41] or because of inadequate therapeutic plans [42]. Kidney transplant candidates should be referred to a dietician as soon as it is practicable, with regular follow-ups to monitor the weight variance. Dietary advice should be individualized and include meal plans, exercise plans, and specific goals. Pharmacotherapy may be a useful adjunct to a comprehensive lifestyle modification program for obesity; however, its usefulness is limited in particular because of the risk of malabsorption and the potential development of kidney oxalate nephrolithiasis and interference with immunosuppressive drugs [43].

Although bariatric surgery has been thought to be a risky procedure for patients with chronic kidney disease, it may increase access to the transplant waiting list and transplantation [44]. Whenever possible, the preferred approach should move from gastric bypass to sleeve gastrectomy, particularly for patients requiring dialysis [45,46]. The patient should be informed about the benefits and disadvantages of each approach, which should be a part of comprehensive care.

CONCLUSIONS

In conclusion, obesity is not an absolute contraindication for transplantation because obese recipients demonstrated no negative influence on late graft function compared to nonobese patients. However, early graft function, patient survival and the incidence of postoperative complications were negatively influenced by a recipient's obesity. A comprehensive strategy should be proposed to obese candidates for renal transplantation toward a sustainable control of a patient's weight.

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