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ORIGINAL ARTICLE

The influence of obesity on short- and long-term graft and patient survival after renal transplantation

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Summary

To determine short- and long-term patient and graft survival in obese [body mass index (BMI) ≥ 30 kg/m^2] and nonobese (BMI < 30 kg/m^2) renal transplant patients we retrospectively analyzed our national-database. Patients 18 years or older receiving a primary transplant after 1993 were included. A total of 1871 patients were included in the nonobese group and 196 in the obese group. In the obese group there were significantly more females (52% vs. 38.6%, $P < 0.01$) and patients were significantly older [52 years (43–59) vs. 48 years (37–58); $P < 0.05$]. Patient survival and graft survival were significantly decreased in obese renal transplant recipients (1 and 5 year patient survival were respectively 94% vs. 97% and 81% vs. 89%, $P < 0.01$; 1 and 5 year graft survival were respectively 86% vs. 92% and 71% vs. 80%, $P < 0.01$). Initial BMI was an independent predictor for patient death and graft failure. This large retrospective study shows that both graft and patient survival are significantly lower in obese renal transplant recipients.

Introduction

The prevalence of obesity in patients with end-stage renal disease (ESRD) is increasing rapidly [1]. In 2003, 60% of the renal transplant candidates in the United States were either obese or overweight [2]. In The Netherlands the prevalence of obesity at the time of transplantation increased from 5.9% in the early nineties to 10.6% in the last 4 years (J. Aalten, MH Christiaans, JW de Fijter, unpublished data). This increase is most probable because of an increase of the prevalence of obesity in ESRD patients, although we cannot exclude that a change in inclusion criteria is in part responsible. The increase of obesity at the time of transplantation underlines the importance to evaluate the policy regarding obese renal transplant candidates.

In the past in most centers obesity was an exclusion criterion for renal transplantation. This policy was based on a worse patient and graft survival and a higher incidence of post-transplantation complications [3–5]. In a review article from Pischon and Sharma [6], the authors confirmed this policy. They concluded that obesity [body mass index (BMI) ≥ 30 kg/m^2] in patients undergoing a renal transplantation was associated with a significantly higher overall mortality, a reduced allograft survival and a higher incidence of peri- and postoperative complications. They advised that before renal transplantation all patients with obesity should lose 5–10% of there weight with subsequent weight maintenance.

Since 2001 a few articles are published in which there was no difference in short-term graft and patient survival

between obese and nonobese patients. The authors stated that obesity alone should no longer be a contra-indication for renal transplantation [7–10]. Moreover, Glanton et al. [11] showed in a large historical cohort study that obese ESRD patients who received a renal transplant have a survival advantage compared to obese renal transplant candidates still on the waiting list.

In the Netherlands, as in the rest of the world, there is no consensus on the management of obese renal transplant candidates. In some transplantation centers obesity (BMI > 30 kg/m^2) is considered as a relative contra-indication for renal transplantation while in other transplant centers in the Netherlands obese patients are accepted for transplantation.

In order to find out what our policy in the future should be, we decided to study the patient and graft survival of obese and nonobese renal transplant patients in the Netherlands. Follow-up data of all renal transplantations in The Netherlands are stored in the Netherlands Organ Transplantation Registry (NOTR). We retrospectively analyzed these data. Aim of this study was to investigate if there is a difference in short- and long-term graft and patient survival between obese and nonobese renal transplant patients.

Patients and methods

In the NOTR database, data about all renal transplantations since 1966 are stored. Because the BMI from most patients transplanted before 1994 was not known, we only included patients transplanted after this date. Other exclusion criteria were age below 18 years or a previous transplantation.

In the database information was stored about recipient age and sex, donor age and sex, type of donor, cold and warm ischemia times, date of patient death and graft loss, cause of death, cause of graft failure, delayed graft function (DGF; defined as the need for dialysis in the first week after renal transplantation) and BMI at the time of transplantation and at 3 months, 1 year, 5 years, and 10 years after transplantation.

Primary endpoints were the difference in patient survival, graft survival and death-censored graft survival between patients with obesity (BMI ≥ 30 kg/m^2) and without obesity (BMI < 30 kg/m^2) at the time of transplantation [BMI = weight (kg)/height^2 (m)]. Because the relation between BMI and transplantation outcome is more complex than only the difference between obesity and normal weight, BMI was also analyzed as a more granulated category variable. For this analysis patients were divided in seven categories according to their initial BMI (BMI < 19 kg/m^2, from 19 to 34 kg at 3 unit increments and BMI ≥ 34 kg/m^2). A BMI between 22 and 25 kg/m^2 was considered as the reference group. Secondary end-points were cause of graft failure, cause of patient death, DGF and change of BMI during follow-up.

Statistical analysis

Statistical analysis was carried out using SPSS version 12.0.1. Normality of data was evaluated with the Kolmogorov–Smirnov test. Results are expressed as median (interquartile range) for continuous nonparametric data. Comparisons between groups were made using the Mann–Whitney *U*-test for continuous variables and the chi-square test or Fisher's exact test for categorical variables. Kaplan–Meier curves were constructed for patient survival, graft survival and death-censored graft survival. Differences between survival curves were calculated with the log-rank test. To evaluate the impact of obesity on short- and long-term graft and patient survival we analyzed them separately. For the short-term graft and patient survival we analyzed the difference in survival between obese and nonobese patients until 3 months. For the long-term survival, the conditional long-term survival in both groups was analyzed (conditional on being alive after 3 months with a functioning graft).

A multivariate Cox regression analysis was used to correct for potential confounders. All covariates which were significantly related to patient or graft survival in the univariate analyses were included in the multivariate analysis. In the Cox regression analysis BMI was assessed both as continuous and as categorical variable (seven categories). A backward stepwise method was used to define the final models.

Multivariate analysis of categorical outcome variables was performed using logistic regression. The influence of change in BMI on transplantation outcome was analyzed in a time-dependent Cox model. Initial BMI, BMI after 3 months, 1 year, and 5 year were included. A *P*-value <0.05 was considered as significant.

Results

A total of 4245 patients of 18 years or older received a first renal transplantation since 1994. From 2067 patients (48.7%) there were enough data to calculate the BMI at the time of transplantation. The median follow-up time was 2.0 years (interquartile range 0.25–5.0 years). Baseline characteristics for obese and nonobese patients at the time of transplantation are given in Table 1. Obese patients were significantly older and there were significantly more females in the obese group. The second warm ischemia time (anastomosis time) was significantly longer in obese patients.

Table 1. Baseline characteristics of the nonobese and obese patients (BMI < 30 kg/m^2 and BMI ≥ 30 kg/m^2).

	BMI < 30 kg/m^2 (n = 1871)	BMI ≥ 30 kg/m^2 (n = 196)
Recipient		
Age (year)	48 (37–58)	52 (43–59)*
Male (%)	61.5	48.0*
Weight (kg)	71 (62–80)	94 (85–103)*
BMI (kg/m^2)	23.8 (21.6–26.0)	32.2 (30.9–34.2)*
Donor		
Cadaveric donor (%)	67.3	68.4
Age (year)	48 (37–56)	48 (35–57)
Male (%)	47.1	52.0
Cold ischemia time (min)	1080 (181–1453)	1180 (291–1440)
Second warm ischemia time (min)	31 (25–38)	34 (28–40)*

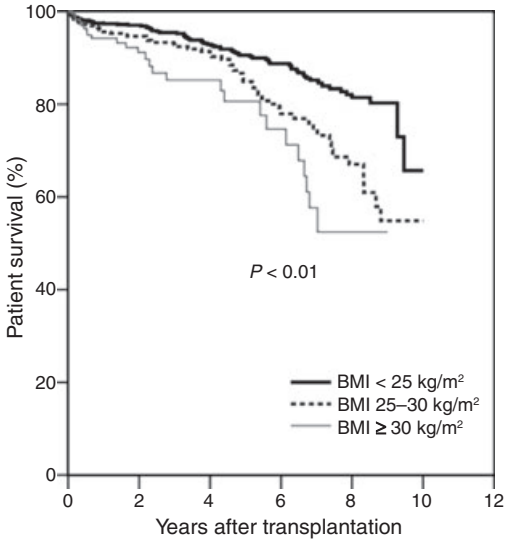
*P < 0.05.

Graft and patient survival

Patient survival was significantly lower in patients with an initial BMI ≥ 30 kg/m^2 compared to patients with an initial BMI < 30 kg/m^2 (1 and 5 year patient survival were respectively 94% vs. 97% and 81% vs. 89%, P < 0.01). Graft survival and death-censored graft survival were also significantly lower in obese patients compared to nonobese patients (1 and 5 year graft survival were respectively 86% vs. 92% and 71% vs. 80%, P < 0.01; 1 and 5 year death-censored graft survival were respectively 88% vs. 94% and 81% vs. 87%, P < 0.01). Patient survival, graft survival and death-censored graft survival were also significantly lower in patients with overweight (BMI 25–30 kg/m^2) compared to patients with normal weight (BMI < 25 kg/m^2). In Figs 1 and 2 patient and graft survival in patients with normal weight, overweight and obesity are shown. There was no difference in patient survival, graft survival and death-censored graft survival between patients with morbid obesity (BMI > 35 kg/m^2, n = 36) compared to patients with obesity (n = 160).

Graft loss after 3 months was significantly higher in obese patients than in nonobese patients (9.5% vs. 5.5%, P < 0.05). The conditional long-term graft and patient survival was also significantly lower in obese compared to nonobese patients (5 year graft survival was 90.8% vs. 85.7% in nonobese versus obese patients, P < 0.01; 5 year patient survival was 85% vs. 78.9%, P < 0.01).

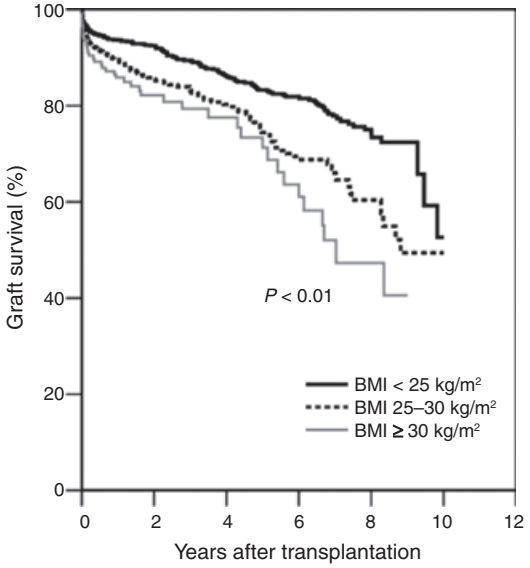
In a multivariate Cox regression analysis initial BMI was an independent predictor for patient death [hazard ratio (HR) 1.05 (95% CI 1.00–1.09) P < 0.05]. In the Cox regression model recipient age, donor age, type of donor and initial BMI were included. Initial BMI was also an independent predictor for graft failure (HR 1.04, 95% CI 1.01–1.07, P < 0.05]. In the Cox regression model for



Number of patients at risk

BMI < 25 kg/m²	1214	649	397	290	129	7
BMI 25–30 kg/m²	657	299	172	111	43	0
BMI ≥ 30 kg/m²	196	89	41	24	9	0

Figure 1 Patient survival in obese patients (BMI ≥ 30 kg/m^2), patients with overweight (BMI 25–30 kg/m^2) and patients with normal weight (BMI < 25 kg/m^2).



Number of patients at risk

BMI < 25 kg/m²	1214	645	396	289	129	7
BMI 25–30 kg/m²	657	296	168	108	42	0
BMI ≥ 30 kg/m²	196	88	41	24	10	0

Figure 2 Graft survival in obese patients (BMI ≥ 30 kg/m^2), patients with overweight (BMI 25–30 kg/m^2) and patients with normal weight (BMI < 25 kg/m^2).

Table 2. Unadjusted and adjusted hazard ratios for the risk factors related to graft failure and patient death.

	Graft survival		Patient death	
Covariate	Unadjusted hazard ratio	Adjusted hazard ratio	Unadjusted hazard ratio	Adjusted hazard ratio
Recipient age	1.03 (1.02–1.04)	1.03 (1.01–1.04)	1.07 (1.06–109)	1.07 (1.05–1.08)
Donor age	1.01 (1.00–1.02)	1.01 (1.00–1.02)	1.02 (1.00–1.03)	1.01 (1.00–1.02)
Cadaveric donor	2.27 (1.68–3.01)	1.96 (1.11–3.46)	1.77 (1.22–5.58)	1.23 (0.83–1.82)
Initial BMI	1.07 (1.04–1.10)	1.04 (1.00–1.07)	1.09 (1.05–1.13)	1.05 (1.00–1.09)
Delayed graft function	1.51 (1.38–2.001)	0.86 (0.62–1.18)		
Second warm ischemia time	1.01 (1.01–1.02)	1.00 (0.99–1.01)	–	–
Cold ischemia time	1.02 (1.01–1.04)	1.00 (0.98–1.02)	–	–

graft failure recipient and donor age, type of donor, DGF, cold and warm ischemia times, and initial BMI were included. In Table 2 the entire Cox regression model for graft failure and patient death are shown. After correction for the same variables as in the model for graft failure initial BMI was no independent predictor for death-censored graft failure (HR 1.04, 95% CI 1.00–1.09, $P = 0.06$).

In a multivariate Cox regression model we also analyzed the influence of BMI as a categorical variable (seven categories) on graft and patient survival. In this analysis both a high and a low BMI were related to patient death, graft survival and death-censored graft survival. In Figs 3 and 4 the adjusted hazard ratios for graft survival and patient death categorized for BMI are given. The risk for graft failure was significantly increased for patients with a low and a high BMI compared to the reference group, with the highest risk for patients with a BMI ≥ 34 kg/m^2. For death-censored graft survival a similar pattern was found. When we removed DGF from the Cox regression model the results were not significantly different. The risk for patient death was increased for all patients with a BMI > 28 kg/m^2 with the highest hazard ratio for patients with a BMI ≥ 34 kg/m^2. For patients with a BMI under 19 kg/m^2 there was a trend to an increased risk for death.

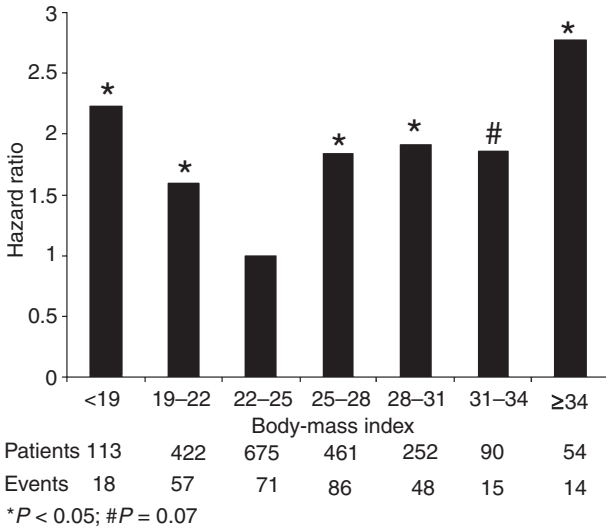


Figure 3 Hazard ratios for graft survival by categorized BMI group (model is corrected for recipient and donor age, donor type, DGF and cold and warm ischemia times).

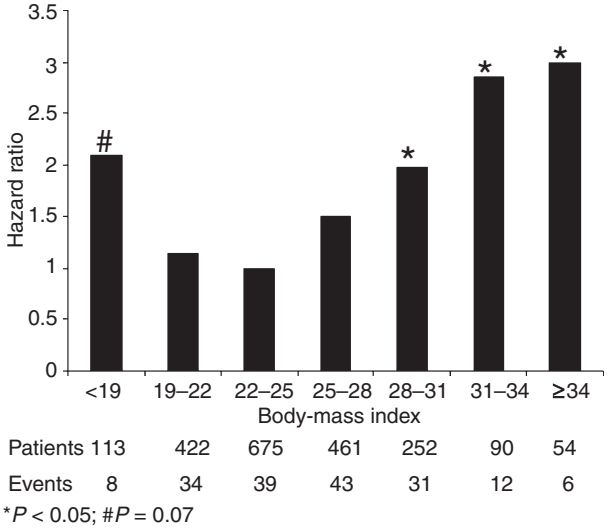


Figure 4 Hazard ratios for patient death by categorized BMI group (model is corrected for recipient and donor age and donor type).

Because data about cardiac risk factors and disease at baseline were missing we did a subanalysis in patients with a lower cardiovascular risk. In this analysis we included all patients under 55 years of age. In this subanalysis obesity was also significantly related to a decreased patient and graft survival.

Cause of graft failure and patient death

In the obese group significantly more patients lost their graft through an infection. Permanent nonfunctioning was also significantly more frequent in obese patients. There were no significant differences in cause of patient death, although there was a trend to a higher infectious ($P = 0.05$) and cardiovascular mortality ($P = 0.09$) in the

obese group. Causes of graft failure and patient death are given in Tables 3 and 4.

Change in BMI after transplantation

After transplantation the mean BMI increased from 24.7 ± 4 at baseline to 25.7 ± 4.3 after 1 year and 26.2 ± 4.6 kg/m^2 after 5 years ($P < 0.01$). During this period the percentage of patients with obesity increased from 9.5% to 14.7%. In a time dependent Cox model increase in BMI was significantly related to graft loss (HR 1.03, 95% CI 1.00–1.05, $P < 0.05$). Increase in BMI was not related to patient death and death-censored graft survival.

Delayed graft function

Delayed graft function was significantly more frequent in obese patients compared to nonobese patients (respectively 31.1% compared to 21.1%, $P < 0.01$). In a logistic regression model initial BMI was an independent predictor for DGF (HR 1.08, 95% CI 1.05–1.11, $P < 0.01$).

Table 3. Causes of graft failure in patients with and without obesity.

Cause of graft failure	BMI < 30 kg/m^2 ($n = 1871$)	BMI ≥ 30 kg/m^2 ($n = 196$)
Rejection (%)	73 (4.0)	8 (4.1)
Died with functioning graft (%)	95 (5.1)	13 (6.6)
Vascular (%)	10 (0.5)	3 (1.5)
Thrombosis (%)	20 (1.1)	5 (2.6)
Technical problem (%)	7 (0.4)	1 (0.5)
Removal of functioning graft (%)	4 (0.2)	0 (0)
Recurrence (%)	13 (0.7)	1 (0.5)
Permanent nonfunctioning (%)	10 (0.5)	4 (2.0)*
Infection (%)	4 (0.2)	3 (1.5)*
Other (%)	28 (1.5)	6 (3.0)

*$P < 0.05$.

Table 4. Causes of death in patients with and without obesity.

Cause of death	BMI < 30 kg/m^2 ($n = 1871$)	BMI ≥ 30 kg/m^2 ($n = 196$)
Cardiovascular (%)	43 (2.3)	9 (4.6)
Infection (%)	35 (1.9)	8 (4.1)
Malignancy (%)	25 (1.3)	0 (0)
Gastrointestinal disease (%)	6 (0.3)	2 (1.0)
Pulmonary embolus (%)	4 (0.2)	0 (0)
Renal failure (%)	1 (0.1)	1 (0.5)
Other (%)	6 (0.3)	3 (1.5)
Unknown (%)	27 (1.4)	4 (2.0)

None of the differences was statistically significant.

Discussion

In this large, retrospective study we found that obesity and overweight are significantly related to a decreased short- and long-term patient and graft survival. We also found that obesity and overweight had a negative impact on death-censored graft survival. Next to that we found that the initial BMI was an independent predictor for patient death and graft failure. Both patients with an increased and decreased BMI compared to the reference group (BMI 22–25 kg/m^2) had a significantly increased risk for graft failure and death-censored graft failure. The increase in risk was most pronounced for patients in the highest BMI group (BMI ≥ 34 kg/m^2). For these patients the risk for graft failure increased more than twofold. Risk for patient death also increased more than twofold for obese patients. These results are in agreement with the large study from Meier-Kriesche et al. [12]. When the initial BMI was analyzed as a continuous variable there also was a significant relation between BMI and graft failure and patient death. Because both a high and low BMI are related to a worse transplantation outcome this relation was less strong than for BMI as a categorical variable.

A lot of other studies also found a decreased patient and graft survival in obese renal transplant patients [3–5,12,13]. In contrast there are some recent studies in which no significant differences in graft and patient survival between obese and nonobese patients were found [7–10,14]. There are a few differences between our study and the recent studies that found an equal patient and graft survival. One possibility is the difference in sample size. Most of these recent studies are much smaller and differences in graft survival and patient survival could have been missed because of a lack of power. In the study from Marks et al. [10] for instance there was a trend to a decreased 3 year graft survival in morbid obese patients. Another possibility could be a difference in patient selection. In the study of Johnson et al. [7] and Massarweh et al. [14] all obese patients underwent a rigorous cardiac screening before inclusion and patients were excluded when significant abnormalities were found. Although most transplantation centers in the Netherlands are reluctant in accepting obese renal transplant candidates, we do not know the exact screening policy of all transplantation centers. Besides Johnson et al. [7] included diabetes, cardiovascular disease and smoking in their multivariate analysis. Unfortunately these data were lacking in our database. As a surrogate analysis for the correction of possible differences in cardiovascular risk factors and disease at baseline we repeated our analysis in patients with a lower cardiovascular risk. In this analysis we only included patients under 55 years of age. In this subgroup obesity was also significantly related to a decreased patient

and graft survival. Although these results do not exclude that the difference in patient and graft survival is in part because of a difference in cardiovascular disease at baseline, it supports the finding that obesity is an independent risk factor for patient death and graft failure. Because diabetes mellitus and an increased BMI are strongly associated, data still could be biased by a higher incidence of diabetes mellitus in obese patients.

In most of the studies, that found a decreased patient survival in obese patients, this was the result of a higher cardiovascular or infectious mortality [3,5,12]. Although the difference in cardiovascular mortality in our study was not statistically significant, there was a trend to a higher cardiovascular mortality in the obese group ($P = 0.09$). This observation is not surprising as obesity is an independent risk factor for cardiovascular disease [15]. Moreover, other important risk factors for cardiovascular disease like diabetes mellitus, hypertension and hyperlipidemia are increased in obese patients [12]. With this data and the results from Johnson et al. [7] in mind it is possible that a more rigorous cardiac screening of obese patients could improve patient and graft survival in obese patients.

There also was a trend to a higher infectious mortality in obese patients ($P = 0.05$). In many studies obesity was related to a higher frequency of wound complications or infections [3,4,7]. It is conceivable that an increased frequency of wound complications in patients treated with immunosuppressive medication could lead to a higher infectious mortality.

As we stated before many studies reported a decreased graft survival or death-censored graft survival in obese patients [3–5,12,16]. In our study obese patients had a significantly decreased graft survival and death-censored graft survival. Significantly more obese patients lost their graft as the result of an infection or primary nonfunction. Next to that significantly more obese patients lost there graft within 3 months after transplantation. A higher incidence of early graft loss has been described in a few other studies [6]. This higher incidence of early graft loss has been related to an increased frequency of postoperative complications in obese patients [4].

Besides the increased early graft loss there also was an increased late graft loss. This is in agreement with some previous studies. In these studies obesity was significantly related to chronic allograft nephropathy [12,17,18]. In our database we had no information about the frequency of chronic allograft nephropathy. One of the theories for the increased prevalence of chronic allograft nephropathy in obese patients is a disparity between donor and recipient weight which could lead to hyperfiltration in the donated kidney [12]. Unfortunately donor weight is not included in our database. There was no significant difference in graft loss caused by rejection between the nonobese and obese group. Earlier studies also did not find a difference in the frequency of rejections between obese and nonobese patients [4,7,12,13,18].

Delayed graft function was also significantly more frequent in obese patients. This is in agreement with some former studies [4,12,19]. In contrast there are other studies that did not find a difference in DGF between obese and nonobese patients [3,5,7,8,18]. In our study initial BMI was an independent predictor for DGF. Although the second warm ischemia time was significantly longer in obese patients in the multivariate analysis second warm ischemia time was no independent predictor for DGF or graft failure.

We further observed that an increase of BMI after transplantation is related to a decreased graft survival. In the study of El-Agroudy et al. [20] an increase of the BMI after transplantation was related to a higher incidence of hypertension, diabetes mellitus and ischemic heart disease. This underscores that it is not only important to start with a BMI under 30 kg/m^2, but that it is also very important to prevent obesity after transplantation.

Although our study has many limitations, for several reasons we still think that we can draw some hard conclusions. The first limitation of our study is that BMI was missing in half of the patients. This might have influenced our results. Although we cannot exclude bias there were no baseline differences between patients with known BMI and unknown BMI. Next to that we still had information about 196 obese renal transplant patients, which is, apart from the study of Meier-Kriesche et al. [12] the largest published cohort of obese renal transplant patients in the literature.

The second limitation is the lack of data on patient history, cardiac risk factors, and HLA matching. For this reason we do not know whether there were besides the difference in age and sex other significant baseline differences between obese and nonobese patients which could be responsible for the difference in transplantation outcome. In a low risk group for cardiovascular disease (patients under 55 years) obesity was also related to a decreased patient and graft survival.

What are the implications of our results for daily practice? The study from Glanton et al. [11] gave fair evidence that obese patients have a better outcome after transplantation compared to staying on dialysis. From this point of view it would not be fair to exclude all obese patients from renal transplantation. Additionally, renal transplantation is accepted in other groups of patients with a decreased patient and graft survival, for example diabetes mellitus or elderly. Some authors state that it is not ethical to make a difference between these groups [10].

In our opinion there is a difference between these groups of patients. In contrast to age and diabetes mellitus, obesity is a preventable and fundamentally curable situation. Therefore it would be fair to motivate patients to lose weight before they get on the transplantation list. Losing weight is not only beneficial to the patient, but it is also important to give scarce organs to patients with the lowest risk. Unfortunately the experience is that it is very difficult for obese ESRD patients to lose weight.

In conclusion we can state that obesity is related to a decreased patient and graft survival. Nevertheless obese patients still do better after transplantation than on dialysis. With this data in mind we suggests that it is not fair to withhold obese patients a transplantation. On the other hand we should not disregard the increased risk for obese patients after transplantation. Patients with obesity have to be fully informed about their increased risk and should be motivated to lose weight. If weight loss is impossible (for instance through immobility) or does not succeed the transplant surgeon should judge if a transplantation is technically possible. If a transplantation is technically possible and cardiac screening is negative obese patients have to be accepted for transplantation. Because the prevalence of obesity is increasing rapidly we should focus on the prevention and treatment of obesity in ESRD patients. Next to that the effects of weight loss in obese patients before transplantation has to be analyzed further.

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Conflict of interest

None.

Reference

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