

- vascular disease: 2006 update endorsed by the National Heart, Lung, and Blood Institute. *AHA; ACC; National Heart, Lung, and Blood Institute. J Am Coll Cardiol* 2006; 47: 2130–2139
15. Kasiske BL, Cangro CB, Hariharan S *et al.* American Society of Transplantation. The evaluation of renal transplantation candidates: clinical practice guidelines. *Am J Transplant* 2001; 1: 3–95
 16. Priori SG, Aliot E, Blomstrom-Lundqvist C *et al.* Task force on sudden cardiac death of the European Society of Cardiology. *Eur Heart J* 2001; 22: 1374–1450
 17. Rea TD, Pearce RM, Raghunathan TE *et al.* Incidence of out-of-hospital cardiac arrest. *Am J Cardiol* 2004; 93: 1455–1460
 18. Herzog CA, Mangrum JM, Passman R. Sudden cardiac death and dialysis patients. *Semin Dial* 2008; 21: 300–307
 19. van Dijk PC, Zwinderman AH, Dekker FW *et al.* Effect of general population mortality on the north-south mortality gradient in patients on replacement therapy in Europe. *Kidney Int* 2007; 71: 53–59
 20. Yoshino M, Kuhlmann MK, Kotanko P *et al.* International differences in dialysis mortality reflect background general population atherosclerotic cardiovascular mortality. *J Am Soc Nephrol* 2006; 17: 3510–3535
 21. Takeda K, Harada A, Okuda S *et al.* Sudden death in chronic dialysis patients. *Nephrol Dial Transplant* 1997; 12: 952–955
 22. Ritz E, Wanner C. The challenge of sudden death in dialysis patients. *Clin J Am Soc Nephrol* 2008; 3: 920–929

Received for publication: 13.12.09; Accepted in revised form: 26.8.10

Nephrol Dial Transplant (2011) 26: 1396–1401

doi: 10.1093/ndt/gfq568

Advance Access publication 17 September 2010

The increased risk of post-transplant diabetes mellitus in peritoneal dialysis-treated kidney allograft recipients

Katarzyna Madziarska¹, Wacław Weyde¹, Magdalena Krajewska¹, Dariusz Patrzalek², Dariusz Janczak², Mariusz Kusztal¹, Hanna Augustyniak-Bartosik¹, Przemysław Szyber², Cyprian Kozyra³ and Marian Klinger¹

¹Department of Nephrology and Transplantation Medicine, Wrocław Medical University, Wrocław, Poland, ²Department of Vascular, General and Transplantation Surgery, Wrocław Medical University, Wrocław, Poland and ³Department of Statistics, Wrocław University of Economics, Wrocław, Poland

Correspondence and offprint requests to: Katarzyna Madziarska; E-mail: kmadziarska@wp.pl

Abstract

Background. Post-transplant diabetes mellitus (PTDM) is a common metabolic complication in kidney allograft recipients, significantly contributing to the elevated cardiovascular morbidity after renal transplantation and increased risk of chronic transplant dysfunction. The aim of the present investigation was to evaluate the factors influencing PTDM development. Under particular consideration were the elements, existing before the transplantation, especially the modality of dialysis treatment significance, i.e. haemodialysis (HD) versus peritoneal dialysis (PD).

Methods. Three hundred and seventy-seven consecutive outpatients who underwent renal transplantation (RTx) in our institution between January 2003 and December 2005 were analysed. PTDM was diagnosed according to the current American Diabetic Association/World Health Organization criteria. Statistical inference was conducted by means of univariate methods (one factor versus PTDM) and multivariate methods in frames of generalized linear model.

Results. In the study group, 72 patients (23.4%) developed PTDM after RTx (55 HD and 17 PD patients). PTDM incidence at 3, 6 and 12 months was 15.9%, 22.1% and 23.4%, respectively. The mean interval from transplantation to the onset of PTDM was 3.08 ± 2.73 months. In univariate analysis, the factors associated with the elevated risk of PTDM appearance were older recipient age, positive family history of diabetes, hypertensive nephropathy as end-stage renal disease cause, higher body mass index at transplantation, treatment by PD, and the graft from an older donor. In multivariate verification, statistical significance remained: older recipient age ($P < 0.001$), positive family history of diabetes ($P = 0.002$), and treatment by PD ($P = 0.007$).

Conclusions. Treatment by PD appears to be a possible novel factor, not yet reported, which may increase the risk of PTDM development.

Keywords: modality of dialysis treatment; peritoneal dialysis versus haemodialysis; post-transplant diabetes mellitus; pre-transplant risk factors

Introduction

Post-transplant diabetes mellitus (PTDM) is a common metabolic complication in kidney allograft recipients appearing in 4–30% of patients [1–4].

It is also well known that PTDM contributes significantly to the elevated cardiovascular morbidity after kidney transplantation and increased risk of chronic transplant dysfunction [1,5,6]. Pathogenesis of PTDM embraces both impairment of insulin secretion and peripheral resistance [7].

In most studies, the main focus was paid to factors affecting PTDM occurrence in the post-transplantation period, such as the type of calcineurin inhibitor used (cyclosporine versus tacrolimus), acute rejection rate, cumulative steroid dose, viral infection presence, number of HLA mismatches and quality of the transplanted organ [1,8–11].

However, less attention was devoted to factors deriving from chronic dialysis treatment. Therefore, the aim of the present investigation was to evaluate the factors influencing the occurrence of PTDM, including those existing before transplantation in the dialysis period. Under particular consideration was the modality of dialysis treatment with the purpose of determination, whether being treated by peritoneal dialysis (PD) versus haemodialysis (HD) has significance for PTDM appearance.

Materials and methods

A retrospective cohort of 377 consecutive patients who underwent renal transplantation in our institution between January 2003 and December 2005 were analysed. All grafts were transplanted from deceased donors. Three hundred and fifty patients received a first kidney graft, and 27 received a second transplant. During the first 12 months, 5 patients changed transplant centre, 12 patients lost graft function and 4 patients died.

Finally, the study group encompassed 356 kidney allograft recipients with graft functioning >12 months. Forty-eight recipients (13.5%) were already diabetic during dialysis.

Therefore, the final analysis included 308 patients (all Caucasian) whose clinical characteristics (quantitative and qualitative parameters) are presented in Tables 1 and 2.

Statistical analysis was performed with Statistica 8.0 software by means of univariate methods (one factor versus PTDM): Pearson's chi-square test of independence (testing dependencies between categorical variables), non-parametric Mann–Whitney *U*-test (comparing means of qualitative variables in the two groups, when binary variable—PTDM—could be the cause of differences), and simple logistic regression (investigating influence of one qualitative variable on PTDM). Multivariate analysis was conducted in frames of generalized linear model (GLM) with the logit link function (investigating many qualitative and categorical variables to test their independent influence on PTDM). Statistical significance was recognized with *P*-value <0.05. Results with *P*-value greater than the significance level but <0.1 are also mentioned. The parameters of quantitative variables are given as mean ± SD.

In the cyclosporine subgroup, the steroid administration was as follows: Day 0—perioperative methylprednisolone 500 mg i.v. bolus, Day 1—250 mg i.v. bolus, Day 2—125 mg i.v. bolus, Day 3–14—40 mg prednisone p.o., Day 15–28—30 mg prednisone p.o., Day 29–42—20 mg prednisone p.o., Day 43–60—15 mg prednisone p.o., and from Day 61—10 mg prednisone.

In the tacrolimus subgroup, significantly smaller steroid doses were given: Day 0—perioperative methylprednisolone 500 mg i.v. bolus, Day 1—125 mg i.v. bolus, Day 2–14—20 mg prednisone p.o., Day 15–28—15 mg prednisone, Day 29–42—10 mg prednisone p.o., and from Day 43—5 mg prednisone p.o.

The mean yearly cumulative prednisone dose in cyclosporine-receiving patients was 5590 mg compared with 3000 mg in tacrolimus-treated recipients.

In addition, the tacrolimus-treated patients were significantly younger than those taking cyclosporine (mean age, 42.3 ± 14.4 and 47.8 ± 11.4 years, respectively, *P* = 0.0029).

During the analysis period (12 months post-transplantation), steroid withdrawal was not attempted by any of the patients.

PTDM was diagnosed according to the current American Diabetic Association (ADA)/World Health Organization (WHO) criteria.

The current ADA criteria for diagnosis of diabetes mellitus are: symptoms of diabetes plus casual plasma glucose concentration ≥200 mg/dL (11.1 mmol/L), fasting plasma glucose ≥126 mg/dL (7.0 mmol/L), or a 2-h post-load glucose ≥200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test [12].

Table 1. Clinical characteristics of patients without diabetes mellitus at the day of transplantation (*n* = 308)—quantitative parameters

Characteristic	HD (<i>n</i> = 260)	PD (<i>n</i> = 48)	Mann–Whitney <i>U</i> -test <i>P</i> -value
Age at transplant (years)	46.10 ± 12.53	41.63 ± 14.98	0.0554
Duration of dialysis therapy (months)	30.15 ± 29.41	19.40 ± 14.82	0.0385*
BMI at transplant (kg/m ²)	24.02 ± 3.79	23.63 ± 3.86	0.5569
BMI at 12 months (kg/m ²)	25.63 ± 4.25	24.54 ± 4.00	0.0493*
Δ BMI (kg/m ²)	1.61 ± 2.45	0.91 ± 2.74	0.0285*
CIT (hours)	24.53 ± 6.97	23.26 ± 7.22	0.2219
Cumulative methylprednisolone i.v. dose at 12 months (g)	0.87 ± 1.23	0.77 ± 1.04	0.8600
Donor age (years)	45.25 ± 12.08	45.63 ± 13.40	0.4788
HLA matches	2.45 ± 0.82	2.52 ± 0.95	0.5306
HLA mismatches	3.55 ± 0.82	3.48 ± 0.95	0.5306

Results are given as mean ± SD. BMI, body mass index; CIT cold ischaemia time.

**P* < 0.05, statistically significant.

Table 2. Clinical characteristics of patients without diabetes mellitus at the day of transplantation (*n* = 308)—qualitative parameters

Characteristic	HD (<i>n</i> = 260)	PD (<i>n</i> = 48)	<i>P</i> -value
Gender of recipient (F)	97 (37.3%)	25 (52%)	0.0545
Family history of diabetes mellitus	58 (22.3%)	11 (22.9%)	0.9119
HCV infection at transplant	23 (8.8%)	4 (8.3%)	0.9081
Gender of donor (F)	101 (38.8%)	23 (47.9%)	0.2391
Primary diagnosis			
Interstitial nephropathy	32 (12.3%)	10 (20.8%)	0.1138
Chronic glomerular disease	134 (51.5%)	20 (41.7%)	0.2088
Polycystic kidney disease	44 (16.9%)	5 (10.4%)	0.2575
Hypertension	42 (16.2%)	10 (20.8%)	0.4265
Other	8 (3.1%)	3 (6.3%)	0.2764
Immunosuppressive therapy			
Cyclosporine	155 (60.5%)	20 (44.4%)	0.0211*
Tacrolimus	101 (39.5%)	25 (55.6%)	0.0866
Azathioprine	122 (46.9%)	13 (27.1%)	0.0109*
MMF	128 (49.2%)	34 (70.8%)	0.0059*
Rapamune	10 (3.8%)	2 (4.2%)	0.9160

HCV, hepatitis C virus; MMF, mycophenolate mofetil.

**P* < 0.05, statistically significant.

Table 3. Comparison of the qualitative parameters between groups (with PTDM versus without PTDM)

Characteristic	PTDM (–) (<i>n</i> = 236) (%)	PTDM (+) (<i>n</i> = 72) (%)	P-value	Odds ratio
Gender of recipient (F)	91 (38.6)	31 (43.1)	0.4947	1.205
Gender of donor (F)	100 (42.4)	24 (33.3)	0.1710	0.680
Modality of dialysis (PD)	31 (13.1)	17 (23.6)	0.0319*	2.044
Modality of dialysis (HD)	205 (86.9)	55 (76.4)	0.0319	0.489
Family history of diabetes mellitus	45 (19.1)	24 (33.3)	0.0046	2.366
HCV infection at transplant	20 (8.5)	7 (9.7)	0.7432	1.163
CMV infection at 12 months	37 (15.7)	10 (13.9)	0.7117	0.867
Herpes infection at 12 months	46 (19.5)	17 (23.6)	0.4481	1.277
Primary diagnosis				
Interstitial nephropathy	35 (14.8)	7 (9.7)	0.2689	0.618
Chronic glomerular disease	125 (53.0)	29 (40.3)	0.0595	0.599
Polycystic kidney disease	35 (14.8)	14 (19.4)	0.3488	1.386
Hypertension	32 (13.6)	20 (27.8)	0.0048*	2.452
Other	9 (3.8)	2 (2.8)	0.6785	0.721
Immunosuppressive therapy				
Cyclosporine	135 (57.2)	40 (55.6)	0.8048	0.935
Tacrolimus	95 (40.3)	31 (43.1)	0.6722	1.122
Azathioprine	105 (44.5)	30 (41.7)	0.6724	0.891
MMF	120 (50.8)	42 (58.3)	0.2655	1.353
Rapamune	11 (4.7)	1 (1.4)	0.2091	0.288

HCV, hepatitis C virus; CMV, cytomegalovirus; MMF, mycophenolate mofetil.

**P* < 0.05, statistically significant.

Results

Some disparities appeared between patients treated by HD and PD. Patients on maintenance PD were younger (*P* = 0.0554), with higher percentage of females (*P* = 0.0545) and shorter dialysis period (*P* = 0.0385). PD patients exhibited lesser weight gain during 12 months of post-transplantation follow-up (*P* = 0.0285) and received more frequently tacrolimus (*P* = 0.0866) and mycophenolate mofetil (*P* = 0.0059) than cyclosporine and azathioprine. The average cumulative annual prednisone dose in PD group was 4150 mg versus 4570 mg in HD group.

There was also difference in maintenance steroid dose between cyclosporine- and tacrolimus-treated recipients (smaller in tacrolimus group).

In the study group, 72 patients (23.4%) developed PTDM after RTx (55 HD and 17 PD patients). PTDM incidence at 3, 6 and 12 months was 15.9%, 22.1% and 23.4%, respectively. The mean interval from transplantation to the onset of PTDM was 3.08 ± 2.73 months.

The comparison of the recipients with and without PTDM is depicted in Tables 3–5.

The following factors not connected with transplantation procedure and immunosuppressive regimen used, already existing prior to transplantation, exerted significant impact on PTDM appearance: older recipient age (*P* < 0.001, OR = 1.050 per 1 year), positive family history of diabetes (*P* = 0.0046, OR = 2.366), hypertensive nephropathy versus glomerulonephritis, interstitial nephritis, polycystic kidney disease and others as the end-stage renal disease (ESRD) cause (*P* = 0.0048, OR = 2.452), higher body mass index (BMI) at transplantation day (*P* = 0.045, OR = 1.066 per 1 kg/m²), and treatment by

PD (*P* = 0.0319, OR = 2.044 with 95% confidence interval of 1.054–3.963).

From the elements operating after transplantation exclusively, older donor age was significantly associated with PTDM (*P* = 0.032, OR = 1.023 per 1 year). The other considered factors, particularly the type of calcineurin inhibitor used, duration of cold ischaemia time, number of HLA mismatches, cumulative methylprednisolone i.v. dose for acute rejection treatment, gender of donor and recipient, presence of hepatitis C virus (HCV) infection before RTx, cytomegalovirus (CMV) and herpes infections after RTx, and number of RTx (data not inserted), did not predict PTDM development.

In GLM analysis of covariance, the occurrence of PTDM was significantly associated with the age of recipient (*P* < 0.001, OR = 1.051 per 1 year), family history of diabetes (*P* = 0.002, OR = 2.665), and PD treatment (*P* = 0.007, OR = 2.782) (Table 6). The lack of significant influence of other variables on PTDM risk could be the result of the relatively small number of patients for multivariate model. Referring to no discerned

Table 4. Modality of dialysis versus PTDM with confidence interval at the 95% level

Characteristic	PTDM (+) proportion	Confidence interval for proportion	Odds ratio	Confidence interval for OR
Modality of dialysis (PD) <i>n</i> = 48	35.4%	21.3–49.5%	2.044	1.054–3.963
Modality of dialysis (HD) <i>n</i> = 260	21.2%	15.8–26.6%	0.489	0.252–0.949

Table 5. Comparison of the quantitative parameters between groups (with PTDM versus without PTDM) and the P-values of non-parametric Mann–Whitney *U*-test and odds ratios from simple logistic regression

Characteristic	PTDM (–) (<i>n</i> = 236)	PTDM (+) (<i>n</i> = 72)	P-value	OR
Age at transplant (years)	43.74 ± 12.91	50.86 ± 11.89	0.000*	1.050
Duration of dialysis (months)	28.50 ± 27.91	28.38 ± 28.09	0.598	1.000
BMI at transplant (kg/m ²)	23.75 ± 3.76	24.67 ± 3.86	0.045*	1.066
ΔBMI (kg)	1.69 ± 2.52	0.87 ± 2.34	0.006*	0.866
CIT (hours)	24.43 ± 6.97	23.98 ± 7.22	0.558	0.991
Cumulative methylprednisolone i.v. dose at 12 months (g)	0.79 ± 1.18	0.83 ± 1.06	0.552	0.818
No. of HLA matches	2.44 ± 0.84	2.53 ± 0.86	0.806	1.125
No. of HLA mismatches	3.56 ± 0.84	3.47 ± 0.86	0.806	0.889
Donor age (years)	44.55 ± 12.47	47.78 ± 11.32	0.032*	1.023

Results are given as mean ± SD. BMI, body mass index; CIT, cold ischaemia time.

**P* < 0.05, statistically significant.

differences between the kinds of calcineurin inhibitor used, it should be underlined that the tacrolimus-treated patients were significantly younger than those taking cyclosporine.

Influence of PD on risk of PTDM is even higher in multivariate model because PD patients are younger than HD patients. The difference in risk of PTDM development between PD- and HD-treated patients is depicted in Figure 1 and with respect to the age factor in Figure 2. Considering the age of recipients, the probability of PTDM appearance was below the age 50 years in PD patients, similar to the rate of occurrence in HD group older than 50 years.

Discussion

In the cohort of kidney transplant recipients under this study, the appearance of PTDM was almost exclusively affected by patient pre-transplant clinical characteristics. The factors significantly augmenting PTDM occurrence were older recipient age (*P* ≤ 0.001), hypertensive nephropathy as the ESRD cause (*P* ≤ 0.01), higher BMI at transplantation day (*P* ≤ 0.05), and PD as the method of renal replacement therapy (*P* ≤ 0.05 versus HD). The only factor associated with transplant procedure significantly contributing to PTDM development was older donor age (*P* ≤ 0.05). The multivariate analysis revealed that the independent significant predictors of PTDM development were recipient age, positive family history of diabetes, and PD treatment. The novel finding, not reported in previous literature, is the observation of significantly higher rate of PTDM in the PD-treated patients compared with the number in HD group. The proportion of PD patients with PTDM was 35.4% with 95% confidence interval of 21.3–49.5% versus the proportion of HD patients equal to 21.2% with 95% confidence interval of 15.8–26.6%.

It is of particular note that this association was disclosed in multivariate analysis, albeit PD patients were younger (on the borderline of statistical significance, *P* = 0.055) and exhibited significantly (*P* = 0.03) lower weight gain post-transplantation (Table 1). Furthermore, the periods of dialysis treatment were significantly shorter in PD group (*P* = 0.04). The possible confounding effect of more frequent tacrolimus use in the PD group (on the borderline of significance *P* = 0.087) can be excluded based on the fact that there was no relationship between calcineurin inhibitor type and diabetes occurrence in the study group, probably due to extensively smaller accompanying steroid dose in the patients treated with tacrolimus (mean yearly cumulative steroid dose in the tacrolimus group was 3000 mg versus 5590 mg in the cyclosporine patients). In addition, the tacrolimus-treated patients were significantly younger than those taking cyclosporine.

Therefore, our data suggest that the glucose load associated with PD therapy may increase the risk of PTDM development. Recently, Bergrem *et al.* reported that pre-transplant impaired glucose tolerance, revealed by a standard oral glucose tolerance test, was predictive for hyperglycaemia appearance 10 weeks post-transplant. However, they did not consider in the investigation the effect of dialysis modality. The major factor underlying PTDM occurrence in their study population was the recipient age which was previously reported [13]. The mention of PD treatment appears in the latest paper published by Hornum *et al.* after this manuscript submission [14]. In a smaller group of 57 kidney transplant recipients from live donors, they did not observe the impact of positive family history of diabetes and PD as treatment modality on PTDM occurrence.

Noteworthy, according to our data, the risk of PTDM in PD patients below age 50 years was similar to the risk in HD patients above 50 years, with starting point of risk increase at age 40 years.

The remaining elements, i.e. hypertensive nephropathy, higher BMI and older donor age, were strongly correlated with the age of recipients. In fact, it reflects the more frequent occurrence of hypertensive nephropathy and BMI increase in older recipients, and the higher probability to receive allograft from older donor. The lack of difference in PTDM frequency between cyclosporine- and tacrolimus-treated patients seems to be caused by higher

Table 6. GLM of analysis of covariance (influence both qualitative and quantitative variables on PTDM)

Effect	PTDM 0/1 logit link function			
	Estimate	OR	Wald statistics	P
Intercept	3.059		21.184	0.000
Recipient age at transplant (year)	0.050	1.051	13.392	0.000
Modality of dialysis treatment: PD/HD	0.512	2.782	7.365	0.007
Family history of diabetes mellitus	0.490	2.665	9.165	0.002

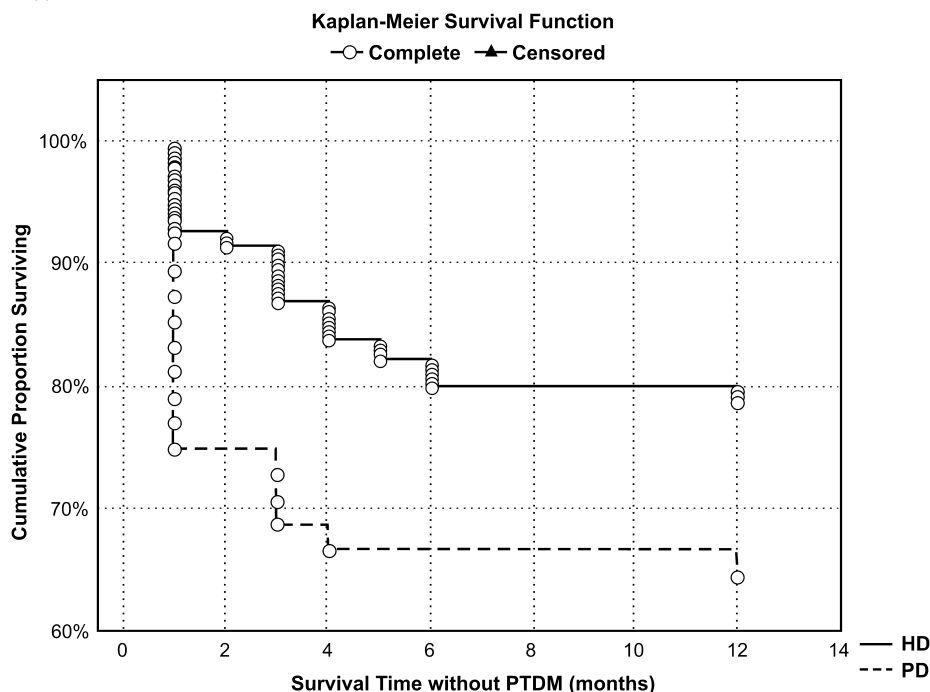


Fig. 1. Kaplan–Meier survival function. The difference in risk of PTDM development with respect to modality of dialysis treatment—PD versus HD.

accompanying prednisone doses in our cyclosporine-based immunosuppressive regimen.

In summary, the present study has identified the features of the recipients associated with an elevated risk of PTDM development, such as older age, family history of diabetes, hypertensive nephropathy as ESRD cause, higher BMI at

transplantation, treatment by PD, and the graft from an older donor. The recipients carrying these features should undergo detailed pre-transplant glucose metabolism evaluation with oral test for glucose intolerance, and be considered for calcineurin inhibitors or steroid-minimization protocols.

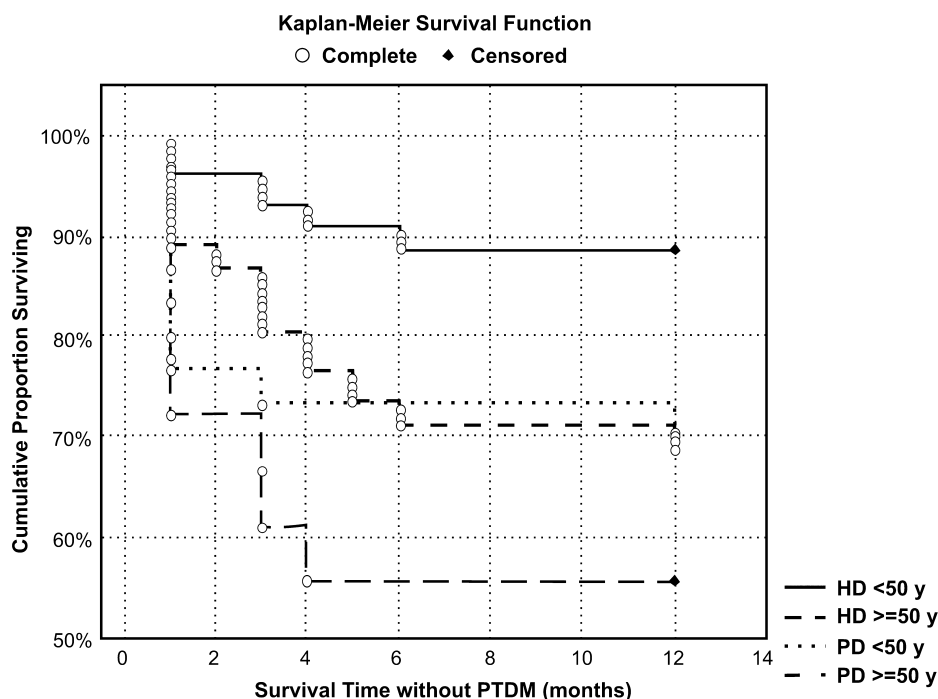


Fig. 2. Kaplan–Meier survival function. The difference in risk of PTDM development with respect to the age factor.

It is of note that 49 cases of PTDM from the total number of 72 cases during 1 year were developed during the first 3 months post-transplantation.

Conclusions

In evaluating the risk of PTDM occurrence, more attention should be paid to the stratification of the predisposing abnormalities present during the dialysis period. Treatment by PD may increase the risk of PTDM development.

Conflict of interest statement. None declared.

References

1. Kasiske BL, Snyder JJ, Gilbertson D *et al.* Diabetes mellitus after kidney transplantation in the United States. *Am J Transplant* 2003; 3: 178–185
2. Woodward RS, Schnitzler MA, Baty J *et al.* Incidence and cost of new onset diabetes mellitus among U.S. wait-listed and transplanted renal allograft recipients. *Am J Transplant* 2003; 3: 590–598
3. Davidson J, Wilkinson A, Dantal J *et al.* New-onset diabetes after transplantation: 2003 international consensus guidelines. *Transplantation* 2003; 75: SS3–SS24
4. Gourishankar S, Jhangri GS, Tonelli M *et al.* Development of diabetes mellitus following kidney transplantation: a Canadian experience. *Am J Transplant* 2004; 4: 1876–1882
5. Revanur VK, Jardine AG, Kingsmore DB *et al.* Influence of diabetes mellitus on patient and graft survival in recipients of kidney transplantation. *Clin Transplant* 2001; 15: 89–94
6. Cosio FG, Pesavento TE, Kim S *et al.* Patient survival after renal transplantation: IV. Impact of post-transplant diabetes. *Kidney Int* 2002; 62: 1440–1446
7. Hagen M, Hjelmestaeth J, Jenssen T *et al.* A 6-year prospective study on new onset diabetes mellitus, insulin release and insulin sensitivity in renal transplant recipients. *Nephrol Dial Transplant* 2003; 18: 2154–2159
8. Gentil MA, Luna E, Rodríguez-Algarra G *et al.* Incidence of diabetes mellitus requiring insulin treatment after renal transplantation in patients with hepatitis C. *Nephrol Dial Transplant* 2002; 17: 887–891
9. Ducloux D, Kazory A, Chalopin J-M. Posttransplant diabetes mellitus and atherosclerotic events in renal transplant recipients: a prospective study. *Transplantation* 2005; 79: 438–443
10. Sulanc E, Lane JT, Puumala SE *et al.* New-onset diabetes after kidney transplantation: an application of 2003 international guidelines. *Transplantation* 2005; 80: 945–952
11. Sato T, Inagaki A, Uchida K *et al.* Diabetes mellitus after transplant: relationship to pretransplant glucose metabolism and tacrolimus or cyclosporine A-based therapy. *Transplantation* 2003; 76: 1320–1326
12. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diab Care* 2004; 27: S5–S10
13. Bergrem HA, Valderhaug TG, Hartmann A *et al.* Glucose tolerance before and after. *Nephrol Dial Transplant* 2010; 25: 985–992
14. Hornum M, Jørgensen KA, Hansen JM *et al.* New-onset diabetes mellitus after kidney transplantation in Denmark. *Clin J Am Soc Nephrol* 2010; 5: 709–716

Received for publication: 23.2.10; Accepted in revised form: 25.8.10

Nephrol Dial Transplant (2011) 26: 1401–1407

doi: 10.1093/ndt/gfq592

Advance Access publication 21 September 2010

HIV infection and renal transplantation

Auxiliadora Mazuecos¹, Ana Fernandez², Amado Andres³, Ernesto Gomez⁴, Sofia Zarraga⁵, Dolores Burgos⁶, Carlos Jimenez⁷, Javier Paul⁸, Alberto Rodriguez-Benot⁹ and Constantino Fernandez¹⁰

¹Department of Nephrology, Hospital Puerta del Mar, Cadiz, Spain, ²Department of Nephrology, Hospital Ramon y Cajal, Madrid, Spain, ³Department of Nephrology, Hospital Doce de Octubre, Madrid, Spain, ⁴Department of Nephrology, Hospital de Asturias, Oviedo, Spain, ⁵Department of Nephrology, Hospital de Cruces, Barakaldo, Spain, ⁶Department of Nephrology, Hospital Carlos Haya, Malaga, Spain, ⁷Department of Nephrology, Hospital La Paz, Madrid, Spain, ⁸Department of Nephrology, Hospital Miguel Servet, Zaragoza, Spain, ⁹Department of Nephrology, Hospital Reina Sofia, Cordoba, Spain and ¹⁰Department of Nephrology, Hospital Juan Canalejo, A Coruña, Spain

Correspondence and offprint requests to: Auxiliadora Mazuecos; E-mail: mauxiliadora.mazuecos.sspa@juntadeandalucia.es

Abstract

Background. Some aspects of kidney transplant outcome in human immunodeficiency virus (HIV)-infected patients are still controversial. Besides, published experience is scarce in Europe.

Methods. A multicentre case–control study was designed to analyse the outcome of renal transplant in HIV+ pa-

tients in Spain. Twenty HIV+ patients were compared with a matched cohort of 40 HIV– recipients.

Results. Post-transplant follow-up period was 39.98 ± 36.51 months. Pre-transplant dialysis duration and the incidence of pre-transplant opportunistic infections were significantly higher for HIV+ patients. Following transplantation, HIV+ recipients presented lower incidence of