Correlation between Glycemic Control and the Incidence of Peritoneal and Catheter Tunnel and Exit-Site Infections in Diabetic Patients Undergoing Peritoneal Dialysis

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Background. Diabetes mellitus, especially if complicated by poor glycemic control, portends an increased risk of infection. The significance of this association in the case of diabetic patients undergoing peritoneal dialysis (PD) has not been assessed.

Methods. Using a retrospective observational design, we analyzed the association between glycemic control at the start of PD (estimated from glycosylated hemoglobin levels) and the risk of peritoneal and catheter tunnel and exit-site infections during follow-up in 183 incident patients on PD. We used the median value of glycosylated hemoglobin to classify patients into good (group A) or poor (group B) glycemic control groups. We applied multivariate strategies of analysis to control for other potential predictors of PD-related infection.

Results. Groups A and B differed significantly in age, dialysis vintage, use of insulin, and rate of *Staphylococcus aureus* carriage. Neither the incidence (0.60 episodes in group A vs 0.56 episodes in group B per patient–year) nor the time to a first peritoneal infection (median: 42 months vs 38 months) differed significantly between the study groups. In contrast, group B had a significantly higher incidence of catheter tunnel and exit-site infections (0.23 episodes vs 0.12 episodes per patient–year) and shorter time to a first infection episode (64 months vs 76 months, $p = 0.004$). The difference persisted in multivariate analysis (adjusted hazard ratio: 2.65; 95% confidence interval: 1.13 to 6.05; $p = 0.013$). We observed no differences between the study groups in the spectrum of causative organisms or in the outcomes of PD-related infections.

Conclusions. Poor glycemic control is a consistent predictor of subsequent risk of catheter tunnel and exit-site infection, but not of peritoneal infection, among diabetic patients starting PD therapy.

KEY WORDS: Peritonitis; catheter tunnel infection; exit-site infection; diabetes; hyperglycemia; glycosylated hemoglobin.

Diabetes mellitus (DM) is prevalent among patients undergoing chronic peritoneal dialysis (PD) (1). Mean comorbidity scores in diabetic individuals who initiate PD are significant, and compared with nondiabetic patients, patients with diabetes have a clinical course characterized by poor standards of rehabilitation and quality of life, a high incidence of cardiovascular events, and shortened life expectancy.

Diabetes mellitus has traditionally been labeled an infection-prone disease. Compared with their non-diabetic counterparts, people with diabetes experience higher hospitalization and mortality rates because of infection (2). The excess risk is particularly evident in certain settings (including surgical or skin and soft-tissue infections), but less apparent when other types of infection are considered (3). In the particular case of patients undergoing PD therapy, the role of DM as a risk factor for peritoneal or catheter tunnel and exit-site infections (TESIs) remains controversial (4-9). The variable efficacy of glycemic control might help to explain the discrepancies.

Hyperglycemia has been associated with *Staphylococcus aureus* carriage, and it may increase the incidence of community-acquired and nosocomial infections (3,10). Contrariwise, tight control of blood glucose levels may reduce the risk of infection, particularly in the surgical setting (11). An association has been demonstrated between poor glycemic control and increased mortality in diabetic patients undergoing PD therapy (12-14), but the specific impact of this factor on rates of PD-related infection has not been adequately assessed.

Our main aim in the present study was to analyze the correlation between glycemic control at the start of PD and the subsequent risk of PD-related infections in a relatively large sample of diabetic patients undergoing PD at our center over a period of 20 years.

METHODS

This retrospective observational study investigated the potential association between glycemic control and the incidence of dialysis-related infections in diabetic patients starting chronic PD in our unit between January 1991 and December 2010. That period

was selected because of relative homogeneity in the protocols for the prevention of peritonitis and TESIs, including screening for and treatment of *S. aureus* carriage. Follow-up was closed by December 2011.

The main independent variable was the blood level of glycosylated hemoglobin (HbA1C) at the initiation of PD. We also considered the average level of HbA1C during the first year on PD (median: 3 estimations; range: 1 - 6 estimations), but the results of this alternative strategy are not presented because they were very similar to those observed using the baseline HbA1C approach. We also took into consideration various control variables that might potentially influence the incidence of PD-related infections. Data for the latter were recovered from a prospective database that included all patients starting PD in our unit during the study period. The main study variables were the incidence rates of peritonitis and TESIs and the time to a first episode of infection during follow-up.

Our study complied with the ethics requirements for retrospective observational studies at our center.

STUDY POPULATION

During the study period, 672 patients started PD in our unit, 234 of whom had a diagnosis of DM. Our study used the following inclusion criteria:

- Diagnosis of DM at the initiation of PD (thus, we excluded patients in whom DM was diagnosed during follow-up on PD)
- Blood level of HbA1C available within ±1 month of PD initiation
- Follow-up on PD for at least 3 months
- Complete clinical records available

MAIN CLINICAL PROCEDURES AND DEFINITIONS

Systematic screening for *S. aureus* carriers was performed during the entire study period. Samples were taken from both nares and from the pericatheter area at 3 different times, separated by 1 month. The first sampling occurred either before or at the time of peritoneal catheter insertion. A patient was defined as a carrier whenever any of the

samples was positive for *S. aureus.* Carriers were treated with nasal and (from 1994) pericatheter mupirocin; they were then screened every other month for re-colonization. If re-colonization recurred, mupirocin was applied again.

Swan-neck double-cuff peritoneal catheters were used throughout the study period. Insertion was usually performed by a nephrologist. Surgical insertion was restricted to patients with suspected peritoneal adhesions or a need for a simultaneous surgical procedure (for example, hernia correction). Cefazolin was administered as antibiotic prophylaxis before catheter insertion. A delay of at least 2 weeks between insertion and initiation of PD was respected whenever possible. Exit-site care was usually based on povidone iodine, with 2% saline or simple cleansing with soap water reserved for specific indications (for example, skin irritation caused by povidone iodine).

Prevention of peritonitis is based on universally accepted protocols (15,16). Integrated Y-sets were the only systems used for continuous ambulatory PD (CAPD); for automated PD, HomeChoice cyclers (Baxter Healthcare Corporation, Deerfield, IL, USA) with Luer-lock connections were used in almost all cases. Most patients used classical lactate-buffered dialysis solutions; new low-GDP solutions have been used systematically since March 2008.

The diagnosis of peritoneal infection followed accepted standards (15). Empirical treatment was based on intraperitoneal ciprofloxacin between 1990 and 2007 and on intravenous vancomycin plus intraperitoneal cefotaxime between 2008 and 2011. Hospital admissions for peritonitis were individualized and not indicated on a routine basis. The diagnosis of TESI was made in the presence of at least 1 of 3 indications: purulent discharge from the exit site, a combination of serous or serohemorrhagic discharge and a positive microbial isolation from the exit site, or the presence of overt signs of inflammation either around the exit site or in the subcutaneous tunnel. Mild, non-exudative inflammatory signs around the exit site are not recorded as TESI at our center. The foregiong criteria are very similar to, but do not exactly match, current guidelines from the International Society for Peritoneal Dialysis (15). The variation is a consequence of the retrospective design and time span of the study. Systemic antibiotics (oral, intravenous, or intraperitoneal) were used, according to *in vitro* susceptibility, for the treatment of most TESIs. Local TESI management was based on hypertonic saline, 0.3% ciprofloxacin solution, 0.3% gentamycin solution, or 2% mupirocin ointment.

STUDY VARIABLES AND LABORATORY METHODS

The main independent variable was HbA1C (determined by high-performance liquid chromatography: Bio-Rad, Hercules, CA, USA). Using the median HbA1C value at the initiation of dialysis, we classified diabetic patients as metabolically well-controlled (below median, group A) or poorly controlled (above median, group B). The main dependent variables were the incidence of peritonitis and TESIs (in episodes per patient-year) and time to the first episode of peritonitis or TESI. We also compared the spectrum of causative organisms and the outcomes of infection in the study groups.

The main baseline control variables scrutinized were age, sex, dialysis vintage, treatment of diabetes, modality of PD (90th day), type of PD solution (90th day), *S. aureus* carriage, comorbidities (Charlson score), socioeconomic background, ability to perform self-dialysis, body mass index, hemoglobin, albumin (determined by autoanalyzer), glomerular filtration rate (determined as the mean of urea and creatinine clearances), peritoneal transport [dialysate-to-plasma ratio (D/P) of creatinine at 240 minutes of the patient's baseline peritoneal equilibration test], and C-reactive protein (determined by the immunoturbidimetry method: Roche Diagnostics, Mannheim, Germany).

STRATEGY OF ANALYSIS

We first compared the characteristics of diabetic patients with good and poor metabolic control. We then analyzed the episodes of peritonitis and TESI in both groups. No instance of peritonitis was diagnosed before the initiation of regular PD. On the other hand, 5 cases of TESI were diagnosed before PD start (median: 8 days; range: 2 - 28 days); those infections were recorded as occurring at time 0.

We applied multivariate strategies of analysis to correct for other factors that might mask or modify correlations between baseline HbA1C and the main study variables. Because of the long study timespan, dialysis vintage was systematically treated as a control variable. In addition, and according to previous analyses in our patients on PD (8), age, PD modality, glomerular filtration rate, and C-reactive protein were specifically scrutinized as control variables to illuminate the potential impact of glycemic control on the risk of peritoneal infection. Similarly, status as a carrier of *S. aureus* was included in the risk analysis for TESI.

STATISTICS

Numeric variables are presented as mean ± standard deviation unless abnormally distributed, in which case median and range are reported. We used the Student t-test, analysis of variance, and Mann-Whitney tests to compare numeric variables, and the chi-square distribution and Fisher exact test to compare categorical variables. Analysis of survival to first infection used Kaplan-Meier plots (log-rank) and stepwise Cox models. We used the SPSS statistical software (version 19.0: SPSS, Chicago, IL, USA) for data analysis.

RESULTS

Of 672 patients starting PD in our unit during the study period, 234 (34.8%) had diabetes, with 42 of the latter having a kidney disease diagnosis other than diabetic nephropathy. Only 183 of the diabetic patients were eligible for analysis. Reasons for exclusion included non-availability of HbA1C levels within 1 month before or after PD initiation *(n* = 39), follow-up of less than 3 months *(n* = 10), and incomplete clinical records *(n* = 2). A comparison of the included and excluded diabetic patients showed no significant difference in characteristics between the groups. Table 1 presents the baseline characteristics of the eligible patients.

The median HbA1C value in the study group was 7.1% (range: 4.4% - 13.4%). Consequently, patients with HbA1C values of 7.1% or less were included in group A, and those with a value greater than 7.1% were included in group B. Table 1 presents a comparison of the characteristics of both subgroups. The proportion of peritoneal catheters inserted surgically (9.9% in group A vs 10.9% in group B, $p = 0.88$) and the delay between catheter insertion and initiation of PD [median: 20 days (range: 2 - 144 days) in group A vs 22 days (range: 5 - 118 days) in group B,$p = 0.90$] were similar in the study groups.

Median HbA1C at the end of the first year was 7.4% (range: 4.5% - 13.0%; $p = 0.25$ vs baseline by paired t-test, $n = 138$), and the average during the first year on PD was 7.15% (range: 4.6% - 12.7%). Only 16 patients (8.7%) were differently allocated to groups A and B depending on whether baseline or average HbA1C was used to categorize glycemic

control. These data suggest an essential stability of glycemic control in most of the patients completing 1 year of follow-up.

PERITONITIS

We observed 237 episodes of peritoneal infection during follow-up. The overall incidence was 0.58 episodes/patient-year. This incidence declined during the study period, to 0.37 episodes/patient-year in 2006–2011 from 0.72 episodes/ patient-year in 1991–1995. The incidence of peritoneal infection was 0.60 episodes/patient-year in group A and 0.56 episodes/patient-year in group B. The spectrum of causative organisms was similar in both groups (Table 2).

Figure 1 depicts time to the first episode of peritoneal infection in the two groups (median: 42 months in group Avs 38 months in group B, $p = 0.15$). Multivariate analysis (Table 3) disclosed baseline glomerular filtration rate, age, and dialysis vintage as independent predictors of the risk of peritonitis in the study population. Charlson score showed a minor, nonsignificant trend to the same association. In contrast, baseline glycemic control did not appear to correlate with the later risk of peritoneal infection.

Table 4 compares peritonitis outcomes, showing no apparent effect of glycemic control, aside from a marginal benefit with respect to the relapse rate.

CATHETER-RELATED INFECTIONS

We observed 71 episodes of TESI during follow-up. The overall incidence was 0.17 episodes/patient–year, and as with peritonitis, the incidence tended to decline with time, to 0.15 episodes/patient–year in 2006–2011 from 0.22 episodes/patient–year in 1991–1995. The overall incidence was 0.12 episodes/patient–year in group A compared with 0.23 episodes/patient-year in group B. Table 2 presents the organisms causing TESI. As with peritoneal infections, the spectrum of causative organisms was comparable in the two groups.

Figure 2 depicts time to the first episode of TESI in the two groups (median: 76 months in group A vs 64 months in group B, $p = 0.004$). In group B, 5 patients (compared with 0 in group A) were diagnosed with a TESI before initiation of PD. Multivariate analysis (Table 5) disclosed elevated baseline C-reactive protein as an independent predictor of

the risk of TESI and, importantly, confirmed a consistent association between baseline glycemic control and the later risk of TESI (hazard ratio: 2.65; 95% confidence interval: 1.13 to 6.05; $p = 0.013$).

DISCUSSION

In the population at large, DM is associated with an increased risk of infection (2). That risk has been particularly documented for nosocomial infections, especially in the surgical setting (11). On the other hand, the diabetic milieu appears to predispose to some types of community-acquired infections, including skin and soft-tissue infections, *S. aureus* carriage, and periodontal disease; the association with urinary tract or respiratory infections is less consistent (3). Diabetes mellitus may predispose patients to infection through several mechanisms. First, ischemia-and neuropathy-related tissue damage sets a favorable scene for infection. In addition, dysfunction of both the innate and the adaptive immune systems has consistently been demonstrated in patients with DM (3). Importantly, polymorphonuclear leukocyte function is markedly affected by the diabetic milieu (3). Humoral immunity is also affected, at least in part because of glycosylation of immunoglobulins, but the significance of disorders of T-cell function is more controversial (3). Hyperglycemia also boosts the growth and virulence of many microbes, and promotes inflammation and endothelial dysfunction, which may cooperate indirectly to increase the risk of infection (10).

The incidence of peritoneal infections in patients with and without diabetes undergoing PD is a longstanding matter of controversy (17). Most recent reports suggest a moderately but significantly higher incidence in diabetic patients (6,7). Data from the Canadian registry suggest that the observed risk might be restricted to diabetic women (9). However, other studies have not detected an association between DM and the risk of PD-related peritonitis (8,18). Moreover, consecutive reports from the ANZDATA registry have not shown DM to be a predictor of peritoneal infection caused by *S. aureus* (19), coagulase-negative staphylococci (20), *Pseudomonas* species (21), or yeasts (22). In fact, patients with diabetic nephropathy have been shown to present a relatively low incidence of non-pseudomonal gram-negative peritonitis (23). The outcomes of peritoneal infections in diabetic and nondiabetic patients are probably similar (24). On the other hand, the influence of DM on the incidence of TESI has not been reassessed in recent

years, and earlier data suggest a similar or, at most, a slightly increased incidence in diabetic patients (17).

The potential influence of glycemic control on the incidence of infections in diabetic patients deserves particular attention. Unequivocal data indicate that hyperglycemia during the perioperative period increases the likelihood of surgical infection (3). Conversely, tight glycemic control may reduce the incidence of postoperative infections (11). The evidence linking long-term glycemic control (as estimated from HbA1C levels) with such infections is less compelling, but has been observed in several studies (10). In contrast, the association between glycemic control and nonsurgical infections is less apparent, although poor glycemic control appears to increase the risk of *S. aureus* carriage and mucocutaneous candidiasis (3). With respect to diabetic patients undergoing PD, poor glycemic control has been shown to predict overall and cardiovascular mortality (13,14), but its impact on PD-related infections has not been assessed.

The results of our study show that baseline glycemic control is an independent predictor of the risk of TESI but not of peritoneal infection during follow-up of patients with DM undergoing PD. The effect persisted after controlling for some significant imbalances between the patient groups with HbA1C levels falling above or below median (Table 1). Those imbalances included dialysis vintage (the quality of glycemic control seemingly improved in the second decade) and insulin-based treatment for DM. *S. aureus* carriage was also more frequent among poorly controlled patients, in agreement with previous reports (3). Interestingly, the latter condition was not associated an increased risk of peritonitis or TESI, probably because carriers were systematically scrutinized and treated with nasal or pericatheter mupirocin (or both) as needed (25).

The association between glycemic control and TESI should not be unexpected, given the correlation between DM and skin and soft-tissue infections, particularly in a surgical setting. In the group with poor glycemic control, 5 patients experienced a TESI before the initiation of regular PD (compared with 0 patients in the well-controlled group), suggesting an origin during catheter insertion. On the other hand, the poor association between glycemic control and later risk of peritoneal infection should not be unexpected either, because PD-related peritonitis is commonly a consequence of intraluminal or endogenous (abdominal or blood-borne) contamination, expectedly less affected by the diabetic milieu than are catheter-related peritoneal infections.

It could be argued that our study lacks statistical power to detect minor associations, but if that were the case, the bias would be expected to affect TESI rather than peritonitis, because of the markedly lower incidence of the former infections in PD patients.

The main limitations of our study include its retrospective design, the significant imbalances between the study groups at baseline, and limited statistical power, which precluded a more comprehensive analysis of the factors predicting PD-related infections in patients with DM. Moreover, only baseline HbA1C levels were used to classify patients as well-controlled or poorly controlled (although analysis of the same variable at 1 year suggested an essential stability in most patients). On the other hand, our study provides clear results not reported previously, and the quality of data collection for control variables was good, mainly because of the prospective approach.

CONCLUSIONS

Poor baseline glycemic control is a consistent predictor of the risk of catheter-related infection among diabetic patients starting PD therapy. In contrast, glycemic control does not appear to be associated with the incidence of peritoneal infections during follow-up. Further studies will be necessary to assess the importance of tight glycemic control, during and after insertion of the peritoneal catheter, to ensure good healing and to prevent later infection.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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Table 1 Study Population

		Patient HbA1C group		
Variable	Overall	A: ≤7.1%	B: >7.1%	*p* Value (Avs B)
Patients (*n*)	183	91	92	—
Age (years)	60.3±12.4	62.5±11.9	58.5±12.7	0.03
Sex (%)				
Men	67.8	68.6	66.7	0.78
Women	32.2	31.4	33.3	
Use automated PD (%)	30.9	32.6	29.2	0.62
Started on low-GDP solution (%)	13.2	15.9	10.5	0.23
PD vintage (%)				
1991–2000	49.9	42.8	57.0	0.035
2001–2010	50.1	57.2	43.0	
CCI score	5.5±1.3	5.7±1.5	5.4±1.2	0.14
Treatment of hyperglycemia				
Diet ± oral antidiabetics	20.5	29.1	12.0	0.002
Insulin	79.5	70.9	88.0	
Glycemia (mg/dL)				
Median	149	141	158	0.03
Range	40–445	44–340	40–445	
Glycosylated hemoglobin (%)				
Baseline	7.3±1.7	5.9±0.7	8.8±1.2	0.0005
End of first year (*n*=138)	7.5±1.7	6.4±1.2	8.4±1.5	0.0005
Average of first year	7.3±1.6	6.1±0.8	8.5±1.2	0.0005
Socio-economic level (% low)	37.6	36.4	38.9	0.92
Self-dialysis (%)	48.5	46.6	50.5	0.60
S. aureus carriage (%)	46.8	38.5	54.4	0.006
Body mass index (kg/m^2)	26.4±4.8	27.3±5.5	25.7±4.0	0.025
Albumin (g/L)	35.1±5.4	34.6±6.1	35.6±4.6	0.20
Hemoglobin (g/dL)	10.5±1.6	10.5±1.8	10.5±1.4	0.96

Table 1 Study Population

		Patient HbA1C group		
Variable	Overall	A: ≤7.1%	B: >7.1%	*p* Value (Avs B)
GFR(mL/m)	6.7±3.6	6.9±3.5	6.5±3.7	0.35
D/P creatinine[a]	0.67±0.15	0.67±0.15	0.67±0.14	0.95
C-Reactive protein (mg/L)				
Median	7.0	9.2	6.1	0.13
Range	1.0–223.0	1.0–223.0	1.0–167.0	
Follow-up (months)	26.6±21.5	26.9±21.1	26.3±21.7	0.55

PD = peritoneal dialysis; GDP = glucose degradation product; CCI = Charlson comorbidity index; S. aureus = Staphylococcus aureus; GFR = glomerular filtration rate.

[a] At 240 minutes of a peritoneal equilibration test.

Table 2 Causes of Infection According to Baseline Glycated Hemoglobin

	Peritonitis by HbA1C group [*n* (%)]		Catheter-related infection by HbA1C group [*n* (%)]	
Organism	≤7.1%	>7.1%	≤7.1%	>7.1%
Staphylococcus aureus	11 (8.9)	8 (7.0)	12 (50.0)	23 (48.9)
Coagulase-negative *Staphylococcus*	27 (22.0)	35 (30.7)	0 (0)	6 (12.8)
Other gram-positive	34 (27.6)	27 (23.7)	1 (4.2)	4 (8.5)
Non-fermenting gram-negative	3 (2.4)	0 (0)	4 (16.8)	5 (10.6)
Other gram-negative	12 (9.8)	18 (15.9)	5 (21.0)	6 (12.8)
Fungal	4 (3.3)	2 (1.8)	0 (0)	0 (0)
Polymicrobial	13 (10.5)	13 (11.5)	2 (8.4)	3 (6.4)
Other	2 (1.6)	0 (0)	0 (0)	0 (0)
Culture-negative	17 (13.8)	11 (9,7)	0 (0)	0 (0)
TOTAL	123 (100)	114 (100)	24 (100)	47 (100)
	p=0.18[a]		*p*=0.66[a]	

[a] By chi-square test.

Table 3 Predictors of Peritoneal Infection in Multivariate Analysis

Variable	Reference	HR	95% CI	*p* Value
Model 1				
Baseline				
GFR (mL/min)	Per unit	0.94	0.88 to 1.00	0.05
Age	Per year	1.03	1.00 to 1.04	0.045
PD vintage	1991–2000	0.67	0.54 to 0.93	0.009
Model 2				
With main study variable				
GFR (mL/min)	Per unit	0.94	0.84 to 0.99	0.04
Age	Per year	1.03	0.99 to 1.05	0.06
PD vintage	1991–2000	0.70	0.40 to 1.26	0.60
Glycemic control	≤7.1%	0.83	0.60 to 1.39	0.83
Other variables scrutinized				
C-Reactive protein	<7 mg/L	1.46	0.90 to 2.41	0.14
Mode of PD	CAPD	0.84	0.60 to 1.69	0.90
Sex	Men	1.29	0.77 to 2.17	0.34
CCI score	Per point	1.22	0.99 to 1.50	0.06
Insulin therapy	No	0.81	0.47 to 1.41	0.44
Socio-economic level	Low	0.76	0.52 to 1.11	0.16
Albumin (g/L)	Per unit	0.98	0.95 to 1.04	0.85
Body mass index (kg/m^2)	Per unit	1.03	0.98 to 1.08	0.32
S. *aureus* carriage	No	1.16	0.78 to 1.74	0.46

HR = hazard ratio; CI = confidence interval; GFR = glomerular filtration rate; PD = peritoneal dialysis; CAPD = continuous ambulatory PD; CCI = Charlson comorbidity index; *S. aureus* = *Staphylococcus aureus.*

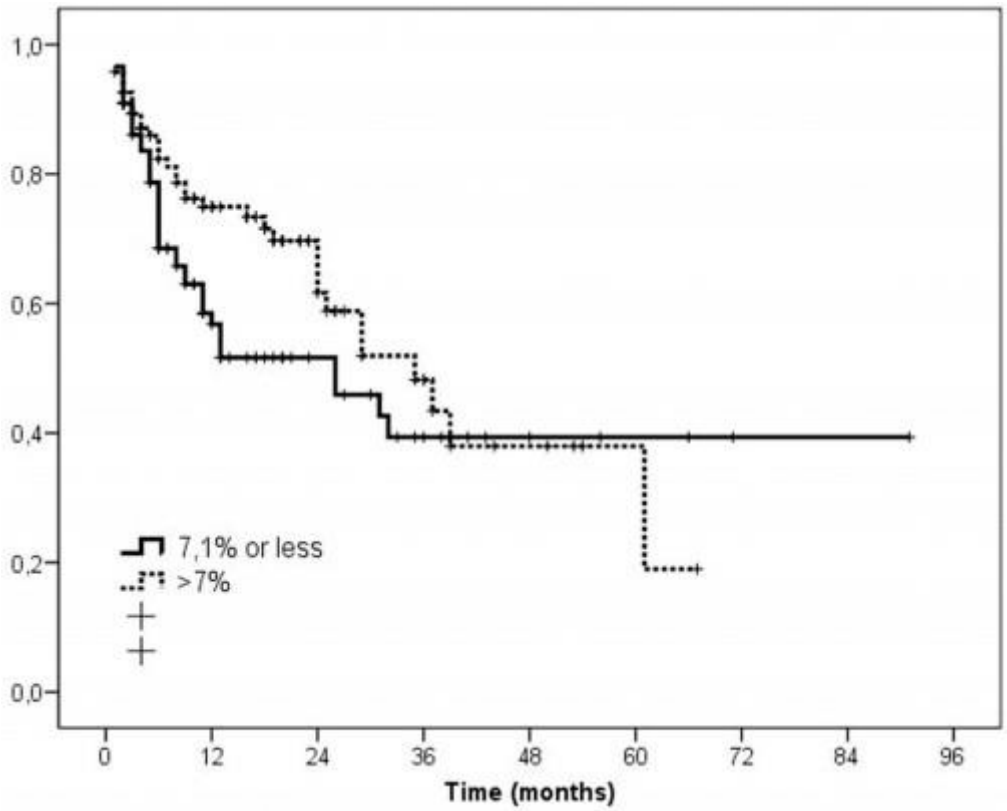

Figure 1 Survival to the first episode of peritonitis by baseline level of glycosylated hemoglobin. Log rank $p = 0.15$.

Table 4 Outcomes of Peritoneal Dialysis-Related Infections

	Peritonitis by HbA1C group (%)		Catheter-related infection by HbA1C group (%)	
Variable	≤7.1%	>7.1%	≤7.1%	>7.1%
Hospital admission	24.0	18.7	19.0	29.8
Catheter removal	8.5	14.6	14.3	29.8
Relapse	7.0	24.4[a]	14.3	14.9
PD drop-out	3.9	3.3	0	2.1
Death	6.9	5.4	4.2	0

[a] $p = 0.001$ versus ≤7.1% group (Fisher exact test). Other differences were nonsignificant.

Table 5 Predictors of Catheter-Related Infection by Multivariate Analysis

Variable	Reference	HR	95% CI	*p* Value
Model 1				
Baseline				
S. aureus carriage	No	1.19	0.67–2.11	0.55
C-Reactive protein	<7 mg/L	2.23	1.07–4.65	0.033
PD vintage	1991–2000	0.51	0.25–1.04	0.065
Model 2				
With main study variable				
S. aureus carriage	No	1.17	0.65–2.08	0.60
C-Reactive protein	<7 mg/L	2.56	1.21–5.41	0.009
PD vintage	1991–2000	0.64	0.29–1.47	0.46
Glycemic control	≤7.1%	2.65	1.13–6.05	0.013
Other variables scrutinized				
Age	Per year	0.99	0.96–1.01	0.37
Mode of PD	CAPD	1.35	0.69–2.65	0.39
Sex	Men	0.75	0.37–1.74	0.43
CCI score	Per point	1.23	0.94–1.76	0.15
Insulin therapy	No	1.51	0.53–4.35	0.44
Socio-economic level	Low	0.90	0.52–1.51	0.68
GFR (mL/min)	Per unit	0.98	0.87–1.10	0.98
Albumin (g/L)	Per unit	0.98	0.91–1.05	0.51
Body mass index (kg/m^2)	Per unit	1.03	0.95–1.12	0.49

HR = hazard ratio; CI = confidence interval; *S. aureus* = *Staphylococcus aureus*; PD = peritoneal dialysis; CAPD = continuous ambulatory PD; CCI = Charlson comorbidity index; GFR = glomerular filtration rate.

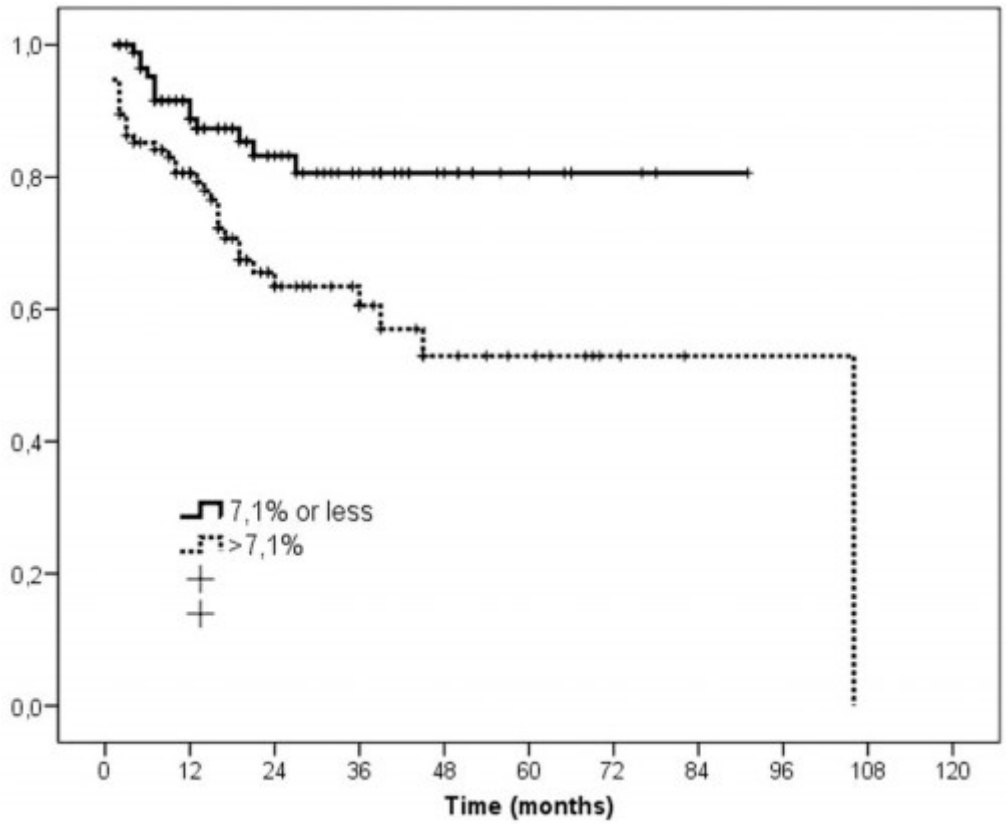

Figure 2 Time to the first episode of catheter-related infection by baseline level of glycosylated hemoglobin. Log rank $p = 0.004$.