

a permanent catheter. The literature states that *P. phragmitetus* is resistant to multiple antibiotics. Our patient made very good progress after oral treatment with quinolones.

REFERENCES

- Zhou Y, Jiang T, Hu S, Wang M, Chen S. Genomic insights of *Pannonibacter phragmitetus* strain 31801 isolated from a patient with a liver abscess. *Microbiolgyopen*. 2017;6, e00515.
- McKinley KP, Laundy TJ, Masterton RG. Achromobacter group B replacement valve endocarditis. *J Infect*. 1990;20:262-3.
- Holmes B, Lewis R, Trevett A. Septicaemia due to Achromobacter group B: a report of two cases. *Med Microbiol Lett*. 1992;1:177-84.
- Jenks PJ, Shaw EJ. Recurrent septicaemia due to Achromobacter group B. *J Infect*. 1997;34:143-5.

- Wang M, Zhang X, Jiang T, Hu S, Yi Z, Zhou Y, et al. Liver abscess caused by *Pannonibacter phragmitetus*: case report and literature review. *Front Med (Lausanne)*. 2017;4:48.

Anna Gallardo ^{a,*}, María del Carmen Merino Bueno ^b, Cristina Sango Merino ^b, Ana María Suárez Laurés ^b, Miguel de la Torre-Fernández ^b, Emilio Sánchez Álvarez ^b

^a Nefrología, Hospital San Agustín, Avilés, Asturias, Spain

^b Nefrología, Hospital de Cabueñas, Gijón, Asturias, Spain

* Corresponding author.

E-mail address: annagallardoperez@gmail.com (A. Gallardo).
<https://doi.org/10.1016/j.nefroe.2020.08.008>

2013-2514/© 2020 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Peritoneal dialysis in non-renal solid organ transplants, experience in our center[☆]

Diálisis peritoneal en trasplantados de órgano sólido no renal: experiencia en nuestro centro

Dear Editor,

The risk of developing chronic kidney disease five years after a non-renal solid organ transplant varies in the range of 7%-21%.¹ The mechanisms involved include chronic dysfunction of the transplanted organ and previous acute kidney injury with incomplete recovery of renal function. However, the most important mechanism continues to be direct nephrotoxicity from calcineurin inhibitors, added to other factors derived from the use of these drugs that contribute to the progression of chronic kidney disease.²⁻⁴

It is estimated that 29% of this population will progress to advanced chronic kidney disease and therefore require renal replacement therapy.^{2,4,5} The use of peritoneal dialysis (PD) has been limited in these patients for fear of a higher incidence of infectious and non-infectious complications due to immunosuppression, and of possible calcineurin inhibitor-related peritoneal toxicity. Such toxicity can cause changes in the morphology of the peritoneal membrane (neoangiogenesis, vascular hyalinosis, profibrotic changes), but without

significant repercussions on peritoneal transport (demonstrated only in animal models).⁴⁻⁶

We describe here our centre experience with this group of patients on PD. This was a descriptive observational study that included all patients with a non-renal solid organ transplant who started PD from January 2012 to October 2019. The study group consisted of 10 patients: two liver transplant recipients, one double-lung transplant recipient and seven heart transplant recipients. Their characteristics are shown in Table 1. There were no cases excluded after they started on PD. The patients were referred by the transplant medical team of each speciality to our unit, where they were informed of the different renal replacement therapy techniques, ultimately opting for PD. No routine abdominal imaging tests were performed before starting the PD, although liver transplant patients had already undergone these studies as part of their regular monitoring.

According to their body mass index, 80% of the patients were of normal weight and 20% low weight; although albumin is not the best marker of nutrition, 30% had baseline hypoalbuminaemia. The mean time between the transplant and the start of PD was 7.2 years (86.4 months). The majority (90%) were taking a calcineurin inhibitor when the PD was started and no significant differences were observed in the pattern

DOI of original article:
<https://doi.org/10.1016/j.nefroe.2020.09.010>.

[☆] Please cite this article as: Andrade López AC, Bande Fernández JJ, Rodríguez Suárez C, Astudillo Cortés E. Diálisis peritoneal en trasplantados de órgano sólido no renal: experiencia en nuestro centro. Nefrología. 2022;42:210-212.

Table 1 – Baseline clinical-epidemiological characteristics of non-renal solid organ transplant patients.

Patient	Charlson	Age, years	Gender	BMI	HTN	DM type 2	Albumin, g/l	CRP, mg/dl	EGFR, ml/min	Residual diuresis, cc	Transplant	IS	Time from transplant- start PD, months	Time on PD, months	PD modality	Kt/V
1	7	67	Male	23	Yes	Yes	33	0.6	5	1,000	Heart	TC/EVE	55	9	CAPD	1.3
2	3	53	Male	23	Yes	Yes	39	0.1	7	1,500	Heart	TC/EVE	74	10	CAPD	1.9
3	4	63	Male	27	No	No	39	1.5	7	500	Liver	TC	50	26	CAPD	1.7
4	5	72	Male	34	Yes	No	29	0.7	12	1,000	Heart	MMF	228	28	CAPD	1.9
5	4	69	Male	23	Yes	No	38	0.5	8	600	Heart	TC	46	65	CAPD	2.5
6	4	71	Male	28	Yes	Yes	32	0.4	9	1,000	Liver	TC	20	29	APD	2
7	1	72	Male	29	Yes	Yes	33	1.2	16	2,000	Heart	TC/MMF	8	69	CAPD	2
8	5	75	Male	28	Yes	Yes	40	1	10	800	Heart	CisA/MMF	240	14	CAPD	2.1
9	1	42	Male	18	No	No	29	0.4	15	1,000	Heart	TC/MMF	144	6	CAPD	0
10	5	20	Female	18	Yes	No	41	0.8	9	1,000	Lung	TC/EVE	180	4	CAPD	3

APD: automated peritoneal dialysis; BMI: body mass index; CAPD: continuous ambulatory peritoneal dialysis; CisA: ciclosporin A; CRP: C-reactive protein; DM: diabetes mellitus; EGFR: estimated glomerular filtration rate; EVE: everolimus; HTN: hypertension; IS: immunosuppression; MMF: mycophenolate mofetil; PD: peritoneal dialysis; TC: tacrolimus.

of immunosuppression between the different transplanted organs.

With regard to the baseline peritoneal equilibrium test in these patients, 44% were fast transporters, 33% fast average and 11% slow average. There were no significant changes in the peritoneal equilibrium test at one year compared to baseline. We believe that most of these patients are fast transporters due to a more protracted chronic inflammatory state, with increased peritoneal surface area. Although we did not have inflammatory cytokine measurements, we observed a tendency towards having elevated C-reactive protein.

There were no significant differences compared to the rest of the population on PD at our centre (38% fast transporters, 41% fast average, 13% slow average, 1% slow).

Regarding infectious complications, there were four episodes of peritonitis due to *Staphylococcus aureus* in the same patient and three exit site infections: two due to *Staphylococcus aureus* and one due to *Serratia marcescens*. There were no fungal infections. At present, the rate of peritonitis in the PD population at our centre corresponds to a ratio of 0.38 episodes per patient-year, and the ratio in the solid organ transplant group was 0.1 episodes per patient-year, suggesting that there is no direct relationship with immunosuppression.

One heart transplant patient had an inguinoscrotal hernia a month after starting PD, requiring a temporary transfer to haemodialysis; four weeks after the surgical repair, he restarted the technique without incident. There were no other non-infectious complications.

During follow-up, four patients left the programme: two received a kidney transplant; and two died. The causes of death were complications unrelated to the technique.

PD is now considered comparable to haemodialysis in terms of survival, and may even have advantages over haemodialysis due to the better preservation of residual renal function and less haemodynamic stress.^{3,4} Those factors could even make it particularly indicated in this group of patients, in whom preserving residual renal function can be complicated in clinical practice.

In conclusion, in our experience, with the limitation of the small sample size, patients with non-renal solid organ transplants do well on PD without any added risk of infectious or non-infectious complications, with good outcomes in terms of safety and adequacy of the dialysis, and no differences in peritoneal transport regardless of the transplanted organ.

REFERENCES

- Ojo AO, Held PJ, Port FK, Wolfe RA, Leichtman AB, Young EW, et al. Chronic renal failure after transplantation of a nonrenal organ. *N Engl J Med.* 2003;349:931–40, <http://dx.doi.org/10.1056/NEJMoa021744>.
- Bloom RD, Reese PP. Chronic kidney disease after nonrenal solid-organ transplantation. *J Am Soc Nephrol.* 2007;18:3031–41, <http://dx.doi.org/10.1681/ASN.2007040394>.
- Buffet A, Guillouët S, Lobbedez T, Ficheux M, Lanot A, Béchade C. Safety of peritoneal dialysis after nonrenal solid-organ transplantation. *Perit Dial Int.* 2018;38:37–43, <http://dx.doi.org/10.3747/pdi.2017.00125>.
- Perl J, Bargman JM, Jassal SV. Peritoneal dialysis after nonrenal solid organ transplantation: clinical outcomes and practical considerations. *Perit Dial Int.* 2010;30:7–12, <http://dx.doi.org/10.3747/pdi.2008.00215>.
- Cornelis T, Rioux JP, Bargman JM, Chan CT. Home dialysis is a successful strategy in nonrenal solid organ transplant recipients with end-stage renal disease. *Nephrol Dial Transplant.* 2010;25:3425–9, <http://dx.doi.org/10.1093/ndt/gfq373>.
- Van Westrenen R, Aten J, Hajji N, de Boer OJ, Kunne SC, deWaart DR, et al. Cyclosporin A induces peritoneal fibrosis and angiogenesis during chronic peritoneal exposure to a glucose-based, lactate-buffered dialysis solution in the rat. *Blood Purif.* 2007;25:466–72, <http://dx.doi.org/10.1159/000112475>.

Ana Cristina Andrade López *, José Joaquín Bande Fernández, Carmen Rodríguez Suárez, Elena Astudillo Cortés

Área de Gestión Clínica de Nefrología, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain

* Corresponding author.

E-mail address: anacristina.andrade@sespa.es

(A.C. Andrade López).

<https://doi.org/10.1016/j.nefroe.2020.09.011>

2013-2514/© 2020 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).