Peritoneal Transport in Peritoneal Dialysis Patients Is Not Affected by Transitorily Successful Renal Transplantation

Patients returning to peritoneal dialysis (PD) from failed renal transplantation are recognized to be inflamed, and this situation might produce a high peritoneal solute transport status. We wanted to determine if a period of time with a kidney allograft induces a change in peritoneal function.

We studied 19 PD patients who had been living with a graft for a mean of 47 ± 39 months. We studied their peritoneal function upon starting PD (baseline), immediately before transplantation (pre-Tx), and after returning to PD when the graft failed (post-Tx). We analyzed the peritoneal mass transfer coefficients for urea (U-MTAC) and creatinine (Cr-MTAC), the dialysate-to-plasma ratio of creatinine (D/P-Cr), and net ultrafiltration (UF).

We observed no significant differences in the various variables pre-Tx and post-Tx. The U-MTAC post-Tx was significantly lower than at PD baseline (25.9 ± 8 mL/min vs. 20.2 ± 5 mL/min, p = 0.03). The U-MTAC and Cr-MTAC post-Tx were not correlated with months on a graft or with MTAC values at baseline. In inherent high transporters (Cr-MTAC ≥ 11.5 mL/min at baseline, n = 8), we observed a significant reduction in Cr-MTAC post-Tx (15.2 ± 2 mL/min vs. 10.2 ± 4 mL/min, p = 0.03). Three of these patients remained high transporters post-Tx.

We conclude that peritoneal function upon reinitiating PD after transplantation is similar to function in the pre-transplantation phase; and that a high peritoneal transport status is more prevalent at first initiation onto PD than at return after transplantation, suggesting that inherently high transport is almost exclusively a feature of an intact, predialysis peritoneum.

Key words
Renal transplantation, peritoneal function

Introduction
Peritoneal small-solute and water transport during peritoneal dialysis (PD) treatment varies between individuals and is affected by many factors—age, time on PD, and peritoneal inflammation, among others. A period living with a renal allograft has not been previously studied as one of these factors, and up to now, the role of transplantation in change of peritoneal transport parameters has been unknown.

We wanted to determine whether spending a time with a temporarily successful renal allograft (Tx) induces changes in peritoneal membrane function upon return to PD.

Patients and methods
We studied 19 PD patients (8 men, 11 women) who were returning to PD from renal transplantation after a mean time of 47 ± 39 months with a functioning renal allograft. All patients had been treated with PD before receiving the graft, with a duration on PD of 25.7 ± 20 months. The mean age of the patients was 35.6 ± 12 years, and 21% had diabetes. The causes of end-stage renal disease in these patients were glomerulonephritis (n = 9), diabetic nephropathy (n = 4), tubulointerstitial nephritis (n = 2), unknown origin (n = 2), polycystic kidney disease (n = 1), and hemolytic uremic syndrome (n = 1).

Peritoneal function was evaluated at three different points: at first initiation onto PD (baseline), immediately before renal transplantation (pre-Tx), and when returning to PD after allograft failure (post-Tx). We used a previously-described mathematical model (1) to study the peritoneal mass transfer coefficients of urea (U-MTAC) and creatinine (Cr-MTAC), the

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dialysate-to-plasma ratio of creatinine (D/P-Cr), and net ultrafiltration (UF), measured as the negative balance after a glucose exchange of 3.86% for 4 hours of dwell time. We defined high transport status as a Cr-MTAC of 11.5 mL/min or higher.

Statistical analysis
We considered a $p$ value less than 0.05 to be statistically significant. We used the nonparametric Wilcoxon test to compare continuous variables over time.

Results
Table 1 shows the measured peritoneal function parameters for all patients. We observed no significant differences in the pre-Tx and post-Tx values. We found that the U-MTAC values were significantly higher at baseline than post-Tx (25.9 ± 8 mL/min vs. 20.2 ± 5 mL/min, $p = 0.03$). The U-MTAC and Cr-MTAC values did not correlate with months successfully living with a graft or with MTAC values at baseline.

In the group of patients with high transport status at baseline (baseline Cr-MTAC ≥ 11.5 mL/min, $n = 8$; Figure 1), we observed a significant decrease in the post-Tx Cr-MTAC value as compared with baseline values (15.2 ± 2 mL/min vs. 10.2 ± 4 mL/min, $p = 0.03$), but

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Functional peritoneal parameters in all patients ($n = 19$)</th>
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<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Urea MTAC (mL/min)</td>
<td>25.9±8</td>
</tr>
<tr>
<td>Creatinine MTAC (mL/min)</td>
<td>11.2±5</td>
</tr>
<tr>
<td>Ultrafiltration (mL/4 h)</td>
<td>800±372</td>
</tr>
<tr>
<td>D/P creatinine</td>
<td>0.71±0.1</td>
</tr>
</tbody>
</table>

$^a$ $p < 0.05$ versus baseline. No differences were found between pre-transplantation and post-transplantation data. Tx = transplantation; MTAC = mass transfer area coefficient; D/P = dialysate-to-plasma ratio.

FIGURE 1 Evolution of functional parameters in high-transport patients at baseline [mass transfer area (MTAC) creatinine (Cr) ≥ 11.5 mL/min; $n = 8$]. Tx = transplantation; U = urea; UF = ultrafiltration; D/P = dialysate-to-plasma ratio.
no significant differences in the remaining parameters. Only 3 of the 8 patients who were high transporters when initially starting PD persisted with high transport post-Tx. When we analyzed the group of patients with high transport pre-Tx (pre-Tx Cr-MTAC ≥ 11.5 mL/min, n = 7) separately, we found no significant differences between their pre-Tx and post-Tx values. However, we observed a tendency toward normalization of small-solute transport parameters after the transplant period, with a decrease in the U-MTAC values to 22.2 ± 7.7 mL/min from 25.2 ± 4.1 mL/min [p = nonsignificant (NS)], Cr-MTAC to 10.2 ± 4.1 mL/min from 13 ± 1.6 mL/min (p = NS), and D/P-Cr to 0.72 ± 0.08 from 0.76 ± 0.05 (p = NS). Peritoneal transport of water did not significantly change (to 950 ± 180 mL/4 h from 1150 ± 229 mL/4 h, p = NS).

Discussion
Peritoneal small-solute and water transport are not significantly changed post-Tx relative to pre-Tx upon PD reinitiation after a period of successful renal transplantation. We also observed that high peritoneal transport status is much more frequently observed at the beginning of PD than after time spent with a kidney allograft.

Peritoneal transport parameters vary among individuals and change during time on PD. Longevity on treatment and peritonitis are the main factors associated with development of a high transport status. At the beginning of PD, heterogeneity is usually seen in peritoneal transport parameters (2), and the number of high-transport patients varies depending on the series. During the first year on PD, peritoneal small-solute transport tends to decrease in high transporters and to increase in low transporters (3). In contrast, in patients with hyperpermeable peritoneum, a peritoneal rest of at least 4 weeks has been described as a good treatment for reducing higher acquired small-solute transport and for increasing lost ultrafiltration capacity (4–7). However, the role of time spent with a renal allograft on the outcome of peritoneal function has not been explored.

In the present study, we observed a tendency toward decreased small-solute transport after renal transplantation, although the differences were nonsignificant. However, our small series cannot exclude that this peritoneal rest period—represented by the transplantation period with immunosuppressive treatment—may have some beneficial effects on peritoneal transport. Larger studies should be performed to confirm our findings.

Our data suggest that high peritoneal transport as defined by the presence of Cr-MTAC ≥ 11.5 mL/min is much more prevalent in patients with intact, predialysis peritoneal membranes. In the present study, 8 patients were high transporters at baseline, but only 3 presented high transport status when returning to PD. In addition, we observed that Cr-MTAC values at PD baseline were significantly higher than those seen post-Tx (15.2 ± 2 mL/min vs. 10.2 ± 4 mL/min, p = 0.03). Those findings suggest that inherent peritoneal high transport has characteristics or causes that exclusively affect the peritoneum upon first initiation onto PD.

Conclusions
Peritoneal function upon reinitiating PD after failure of a renal allograft is similar to that seen pre-Tx. A high peritoneal transport status is much more frequently observed when initiating PD for the first time than post-Tx.

References

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