
Sodium overload is the main factor underlying symptomatic fluid retention and hypertension in CAPD patients. Lowering the sodium concentration of peritoneal dialysis enhances sodium removal by diffusive transport and short term (1 week) observations have shown that ultra-low sodium (98 mg/dL) may be effective in decreasing BP in over-hydrated CAPD patients (Nakayama M et al. Clin Nephrol 1996:45, 188). In this double blind, randomised, crossover study we tested the effect on arterial pressure of ultra-low sodium (UL-Na) dialysate in a group of patients on CAPD with inadequate arterial pressure control. Seven CAPD patients (M 5 F 2; mean age: 67 years, range 42–76) were selected on the basis of clinical signs of fluid retention and hypertension requiring drug treatment. Patients were studied during two experimental periods, each lasting 1 month (control period and ultra low Na dialysate (UL-Na)). Convective transport was kept identical in the two periods. In the control period the UL-Na was infused at a maximum rate of 400 mL/h. In the UL-Na period, during the first two weeks we introduced one low sodium (Na=98 mmol/L) exchange during night and a further UL-Na exchange during daytime during the next two weeks. The wash-out period between the two experimental phases lasted 2 weeks. Blood pressure data [10 measurements over 30 minute by an automatic recorder (Dynamap)] are reported in the Table (Mean (SD), *p<0.05 (week 4 vs 0)).

<table>
<thead>
<tr>
<th>Weeks</th>
<th>0</th>
<th>2</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diast BP (mmHg)</td>
<td>146(24.3)</td>
<td>141(21.5)</td>
<td>147(17.9)</td>
</tr>
<tr>
<td>Syst BP (mmHg)</td>
<td>74(11)</td>
<td>74(10.1)</td>
<td>77(8.7)</td>
</tr>
<tr>
<td>Body weight (Kg)</td>
<td>68.9(12.0)</td>
<td>68.8(10.2)</td>
<td>69.3(12.0)</td>
</tr>
</tbody>
</table>

Diastolic and Systolic pressure fell significantly in the experimental period allowing reduction or discontinuation of drug treatment. Serum sodium, osmolality and pH did not differ between the two periods. The decrease of arterial pressure in apparently drug-resistant overhydrated CAPD patients. This study represents a sound basis for designing a larger scale trial aiming at optimising arterial pressure control in these patients.

Hypertension in CAPD patients. Lowering the sodium concentration of peritoneal dialysis (CAPD) with inadequate arterial pressure control in these patients. The purpose of this study is to detect low risk group of patients for peritonitis and to evaluate the capacity of priming of PM0 from an infection free period (IFP) have an increased capacity (are "primed") to release pro-inflammatory cytokines.

In conclusion: the CAPD peritoneum appears to be a state of severe overload of carbonyl compounds both in CAPD solutions and peritoneal effluents from patients with end-stage renal failure, as a result of an increased protein synthesis (PS), breakdown (PB) and protein balance (NB). Muscle protein synthesis (PS) was increased in CAPD patients compared to controls (232±29 vs 151±4 at 0 week, p<0.01).

In conclusion, the CAPD peritoneum appears to be a state of severe overload of carbonyl compounds derived from a CAPD solution and from uremic creatinine. The accumulated carbonyl compounds may cross-link peritoneal matrix or cellular proteins and alter their structures and functions, implicating carbonyl stress in peritoneal sclerosis.

T238 (PS) T1051 (FC)

Previously we reported, that PM0 isolated from CAPD-patients during P compared to those from an infection free period (IFP) have an increased capacity (are "primed") to release IL-1β after stimulation in vitro with LPS. In order to investigate the molecular mechanisms of priming of PM0, we studied the gene expression, as well as the production and secretion of active mature IL-1β (mIL-1β) and its precursor pro-IL-1β by infection-free and infection PM0’s. PM0’s were isolated from IFP’s and P’s and incubated with 100U/ml LPS. Total IL-1β production (supernatants + lysates of PM0 isolated during IFP and P) was measured in supernatants in patients undergoing PD during IFP (P) and in patients undergoing CAPD with inadequate arterial pressure control. IL-1β production during IFP’s and P’s and incubated with LPS was increased compared to IFP and P (382 and 97 attomoles/10^5 cells, N.S.). The fraction of total IL-1β released that remaining in the cell), expressed in pmol, did not differ significantly, between these groups in line with the findings on IL-1β mRNA levels in PM0 from IFP and P (382 and 97 attomoles/10^5 cells, N.S.). The fraction of total IL-1β released as mIL-1β was 23 ± 4% (in ± SEM) and 55 ± 8% during IFP and P (p < 0.05).

In conclusion: In LPS stimulated PM0 from peritonitis patients processing and export of IL-1β rather its production are increased, pointing to the key role of processing in the regulation of IL-1β activity in P during infection.

A1438

Advanced glycation end products (AGEs) accumulate in serum and tissue proteins of patients with end-stage renal failure, as a result of an increased protein modification by carbonyl compounds derived from autoxidation of carbohydrates and lipids ("carbonyl stress"). Carbonyl stress has been implicated in the pathogenesis of long-term complications in hemodialysis patients. Carbonyl stress appears relevant not only to hemodialysis but also to CAPD, because peritoneal tissue and cellular proteins are continuously exposed not only to carbonyl compounds transferred from dialysis solutions but also to high glucose CAPD solution which is a highly reactive disease causing compound generated during the heat sterilization process or formed within the peritoneum. To address this hypothesis, we measured the levels of carbonyl compounds both in CAPD solutions and peritoneal effluents from CAPD patients. The reaction of 2,4-dinitrophenylhydrazide with carbonyl compounds yields stable hydrazone adducts which can be measured by a spectrophotometric assay. The levels of carbonyl compounds measured by this method in CAPD solutions were increased after the heat sterilization (pre- and post-heat sterilization 2.5 % glucose CAPD solution) which is a highly reactive disease causing compound. The levels of carbonyl compounds are markedly higher in effluents after overnight CAPD than in the original CAPD solution (5.45 vs. 30.76 nmol/mL, P < 0.001).

In conclusion, the CAPD peritoneum appears to be a state of severe overload of carbonyl compounds derived from a CAPD solution and from uremic creatinine. The accumulated carbonyl compounds may cross-link peritoneal matrix or cellular proteins and alter their structures and functions, implicating carbonyl stress in peritoneal sclerosis.