Intraperitoneal Phosphatidylcholine Levels in Patients on Continuous Ambulatory Peritoneal Dialysis Do Not Correlate With Adequacy of Ultrafiltration[1,2]

Janine Beavis, John L. Harwood, Gerald A. Coles, and John D. Williams[3]

J. Beavis, G.A. Coles, J.D. Williams, Institute of Nephrology, University of Wales College of Medicine, Cardiff Royal Infirmary, Cardiff, United Kingdom

J.L. Harwood, Department of Biochemistry, University of Wales College of Cardiff, Cardiff, Wales, United Kingdom

(J. Am. Soc. Nephrol. 1993; 3:1954–1960)

ABSTRACT

A qualitative and quantitative study was undertaken to determine the lipid composition of dialysate effluent from patients maintained on continuous ambulatory peritoneal dialysis (CAPD). Effluent, after a 4-h, 2.27% dextrose dwell, was collected on ice, centrifuged and extracted for lipids with chloroform and methanol. Lipids were separated and identified by thin layer chromatography, and the constituent fatty acids were quantitated by gas liquid chromatography. Effluents from 10 patients were assayed at the commencement of CAPD treatment and again after 6 months of therapy. There was a significant fall in phosphatidylcholine and phospholipid concentrations ($P < 0.007$) with time, whereas the fatty acid compositions of these lipids remained constant. Dialysate phosphatidylcholine and phospholipid concentrations were not significantly different between seven patients with poor ultrafiltration and eight patients who had normal fluid removal. This study demonstrates that there is no relationship between dialysate phospholipid levels and the adequacy of filtration, although it corroborates previous reports of an inverse correlation between time on CAPD and dialysate lipid concentrations. These results do not support a rationale for ip phosphatidylcholine administration in patients with poor ultrafiltration.

Key Words: *Peritoneum, Continuous ambulatory peritoneal dialysis effluent, Type I ultrafiltration failure, surfactant, ultrafiltration rate*

Continuous ambulatory peritoneal dialysis (CAPD) is in widespread use for the treatment of chronic renal failure and serves as a long-term procedure for approximately 13% of dialysis patients worldwide. In a number of instances, however, there is a need for a switch to hemodialysis because of the loss of ultrafiltration capacity (1,2). The failure to remove fluid may be caused by a number of factors, but the most common reason is an increased membrane permeability, allowing rapid absorption of glucose and leading to the dissipation of the osmotic gradient.

It has been suggested by DiPaolo *et al.* (3) that this loss of ultrafiltration was associated with a reduced concentration of phospholipids in the dialysis effluent. This "surface-active material" was first described in the pleural cavity but lately has been characterized in the peritoneal cavity (4); its source has been identified as the peritoneal mesothelial cell (5). Peritoneal phospholipid has been likened to surfactant (6), which has unique hydrophobic properties that allow it to reduce surface tension at the air:water interface in the alveolar fluid. Within the peritoneal cavity, this water-repellent property is thought to facilitate ultrafiltration. Subsequently, it was demonstrated that the addition of phosphatidylcholine (PtdCho), either ip (3,7–9), iv (3) or po (3,10), resulted in improved fluid removal. Clearly, these results could be of considerable clinical significance. The nature of the proposed phospholipid deficit, however, including any changes in fatty acid composition, has not been examined in any detail.

We have, therefore, examined the phospholipid composition of CAPD effluent both quantitatively and qualitatively from patients commencing CAPD and after 6 months of therapy. We have also compared the dialysate phospholipid composition from patients exhibiting poor ultrafiltration with that of control subjects.

[1] Received July 2, 1992. Accepted March 24, 1993.
[2] Data from this work have been presented at the 24th Annual Meeting of the American Society of Nephrology.
[3] Correspondence to Dr. J.D. Williams, Institute of Nephrology, University of Wales College of Medicine, Cardiff Royal Infirmary, Cardiff, CF2 1SZ, Wales, United Kingdom.

1046-6673/0312-1954$03.00/0
Journal of the American Society of Nephrology

MATERIALS AND METHODS

Patient Categories

A total of 25 patients, classified into three groups, were studied. The first group consisted of 10 patients who were investigated within 2 wk of starting CAPD and 6 months later. The second group comprised seven patients with poor ultrafiltration as judged by the need to use more than two bags containing 3.86% dextrose in 24 h to maintain fluid balance. They had been receiving CAPD for at least 11 months. The remaining eight subjects had normal fluid removal and had been treated for a period similar to that of Group II.

Materials

Sources of materials were as follows. Commercial dialysis fluid was supplied by Baxter Healthcare Ltd, (Egham, Surrey, United Kingdom). Chromatography-grade chloroform, methanol, petroleum spirit (60 to 80°C), diethylether, glacial acetic acid and sulfuric acid were all obtained from Merck Ltd (Eastleigh, United Kingdom), who also supplied Analar anhydrous sodium sulfate, potassium chloride, sodium chloride, di-potassium hydrogen orthophosphate and potassium di-hydrogen orthophosphate. Sigma Chemical Company Ltd (Poole, United Kingdom) supplied the following: L-α-phosphatidylcholine (egg yolk), L-α-phosphatidyl-L-serine (bovine brain), sphingomyelin (egg yolk), L-α-phosphatidylethanolamine (egg yolk), L-α-phosphatidic acid, heneicosanoic acid, diacylglycerol (1,3-dipalmitin), triacylglycerol (tripalmitin), nonesterified fatty acid and 8-anilino-1-naphthalene-sulfonic acid (ammonium salt; ANSA). The lipid standards were checked for purity before use and, where necessary, were purified by thin layer chromatography (TLC). Flat-bottomed developing chambers, silica gel 60 TLC plates (20 × 20 cm), calibrated disposable micropipettes, chromatography spray gun, and pyrex culture tubes with inert polytetrafluoroethane-lined screwcaps were all purchased from Merck Ltd.

Lipid Extraction

After an overnight exchange, a standard peritoneal equilibration test with fluid containing 2.27% dextrose was performed (11). After the 4-h dwell, the effluent was collected in a drainage bag surrounded by ice. The bag was weighed to assess ultrafiltration, and samples were taken for glucose and creatinine estimation. The remaining fluid was analyzed for lipid content. Aliquots of 100 mL were taken from the CAPD effluent, and any cellular material was removed by centrifugation at 2,720 × *g* for 35 min at 10°C, followed by centrifugation at 5,000 × *g* for 10 min at 4°C. Twenty-milliliter samples were taken and extracted for lipid by the method of Garbus *et al.* (12). To a 1-mL sample was added 3.75 mL of chloroform:methanol (1:2); the mixture was shaken and allowed to stand for 15 min. To this, a further 1.25 mL of chlorofirm and 1.25 mL of 2 M potassium chloride in 0.5 M potassium phosphate (pH 7.4) was added, shaken, and allowed to separate into a biphasic system overnight. The upper aqueous phase was separated from the lower lipid-containing chloroform phase, and the latter was dried under a vacuum at 25°C. The lipid extracts were resuspended in chloroform with a small amount of methanol and were stored in sealed glass tubes under nitrogen at −20°C until required. Analyses were always performed within 2 wk of the extraction procedure. TLC and gas liquid chromatography analyses confirmed the stability of acyl lipids under these storage conditions.

Separation, Identification, and Quantitation of Lipid Classes

The lipid classes were separated by TLC on silica gel 60 TLC plates that had been activated at 120°C for 1 h. The solvent systems were freshly prepared, and the chromatograms were developed in sealed tanks lined with solvent-saturated filter paper. Oxidation of the polyunsaturated fatty acids was prevented by the addition of 0.05% butylated hydroxytoluene to the solvent systems. The phospholipids were separated in a polar solvent system (Plate 1) composed of chloroform:methanol:acetic acid:water (170:30:20:7 by vol), which allowed development to continue to within 2 cm of the top of the plate. The neutral lipids were separated into their classes after development in a neutral solvent system composed of petroleum spirit (60–80°C fraction):diethylether:acetic acid (90:10:1, by vol), while the phospholipids remained at the origin (Plate 2). Subsequent development of Plate 2 in the polar solvent system to a distance of 3 cm above the sample origin allowed the phospholipids to separate slightly, but to remain in a relatively compact area. After development, the plates were dried under nitrogen for 15 to 20 min and the lipid bands were detected under UV light (λ = 365 nm) after being sprayed with a 0.2% solution of ammonium salt (ANSA). The bands were identified by cochromatography with the following standards: PtdCho, sphingomyelin, phosphatidylserine, phosphatidylethanolamine, phosphatidic acid, triacylglycerol, diacylglycerol, and nonesterified fatty acid. From Plate 1, the duplicate PtdCho bands were scraped off, whereas from Plate 2, the total phospholipid bands were removed. The silica gel samples were scraped into pyrex culture tubes with Teflon-lined screwcaps to which 10 μg of the internal standard, heneicosanoic acid, was added for quantitation. To this, 2 mL of 2.5% sulfuric acid in dry methanol

was added, the tubes were sealed, and the samples were heated for 2 h at 70°C. The fatty acids were thereby converted to fatty acid methyl esters (FAME) by transmethylation. After being cooled, 5% saline was added to stop the reaction and the FAME were then extracted three times into petroleum spirit (60 to 80°C). Quantitation and identification of the total fatty acids present in the dialysate were also performed; after aliquots of the lipid-containing chloroform extract were dried, FAME were prepared as described above. The FAME extracts were then dried under nitrogen and resuspended in dried redistilled petroleum spirit; aliquots were analyzed by GLC. Fatty acid analyses were carried out by use of Perkin Elmer F33 gas liquid chromatography (Beaconsfield, United Kingdom), equipped with a 4 mm × 1.5 m glass column. Separations were achieved in PT 10% SILAR-10C on a 100-mesh Supelcoport (Supelco, Elysian, MN) with nitrogen as the carrier gas and isothermal runs in the range from 160 to 180°C. Fatty acids were detected by flame ionization. Identification of the peaks was by direct comparison of the retention times to commercial FAME standards and quantitation by comparison to the internal standard peak area.

Recovery experiments were carried out to determine the efficiency of the above procedures. Known amounts of egg yolk PtdCho (in the form of multilamellar liposomes) were added to used CAPD fluid, and the recoveries were calculated after the amount of PtdCho already in the dialysate was determined. Mean recovery was 81.4% ($N = 3$).

All data are represented as means ± standard deviations. Statistical evaluation for unpaired data was performed by the nonparametric, Mann Whitney U test and for paired data was performed by the nonparametric Wilcoxon's signed rank test.

RESULTS

Change in Phospholipid Concentration With Time on Dialysis

Ten patients starting CAPD were studied. Table 1 gives details of their age, sex, and diagnosis. Figure 1 shows the concentrations of total fatty acids, phospholipids, PtdCho, and neutral lipids at the start of dialysis and after 6 months of treatment. There was a significant fall in all lipid fractions, with mean values declining by more than 50%, despite a nonsignificant increase in ultrafiltration volumes (mean, 0 to 225 mL). The fatty acid composition of total lipids, total phospholipids, or PtdCho did not change during this time period. There was no difference in the proportion of PtdCho in the phospholipids with time on dialysis. PtdCho represented 85.0 ± 11.0% of the total phospholipids at the start of CAPD and 84.5 ± 11.2% after 6 months of treatment. In all samples studied, the following lipids were identified by TLC:

TABLE 1. Clinical details of patients commencing CAPD treatment

Patient No.	Age (yr)	Sex	Diagnosis
1	67	F	Amyloid
2	60	F	Unknown
3	59	M	Hypertension
4	35	M	Hypertension
5	52	F	Unknown
6	57	M	Diabetes
7	70	M	Unknown
8	67	M	Myeloma
9	41	M	Diabetes
10	72	M	Unknown

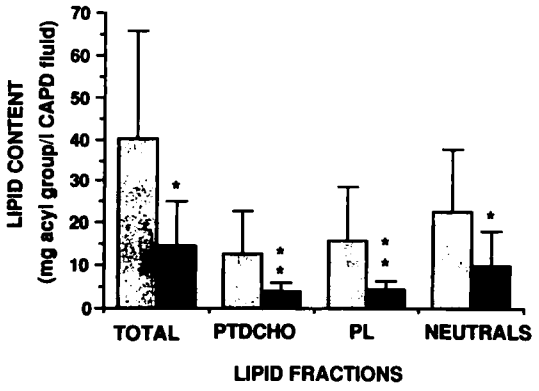

Figure 1. Changes in lipid content of CAPD fluid during a 6-month dialysis period. Stippled box, patients just starting CAPD ($n = 10$); closed box, the same patients after 6 months of treatment. Results are expressed as means ± SD. * $P < 0.011$; ** $P < 0.007$ (Wilcoxons signed rank test). PL, total phospholipid.

PtdCho, sphingomyelin, phosphatidylserine, phosphatidylethanolamine, triacylglycerol, and nonesterified fatty acid. There was no relationship between ultrafiltration volume and phospholipid concentrations (or total amount) in the dialysate at either time.

Dialysate Lipid Analysis in Poor Ultrafiltrators

Table 2 compares the clinical details and peritoneal equilibration test results of the seven patients with poor ultrafiltration with those of the eight subjects who had undergone CAPD for similar periods but who did not have problems with fluid removal. As can be seen, the net ultrafiltration value was significantly lower ($P < 0.001$) in the poor ultrafiltration group and was often negative. The maximum value was 90 mL less than the minimum result in the patient group without ultrafiltration problems. Similarly, the 4-h dialysate-to-plasma creatinine concentration ratio was significantly higher ($P < 0.001$) in

TABLE 2. Clinical details and peritoneal equilibration test results for patients on CAPD for extended periods[a]

Patient No.	Age (yr)	Sex	Diagnosis	Time on CAPD (months)	Net UF (mL)	D/P creatinine	D/Do glucose
Normal Filtration							
11	29	F	Pyelonephritis (reflux)	40	210	0.64	0.59
12	75	F	Unknown	33	345	0.75	0.32
13	76	F	Amyloid	25	380	0.76	0.28
14	66	F	Unknown	21	560	0.67	0.44
15	65	M	Glomerulonephritis	36	180	0.73	0.32
16	63	M	Hypertension	24	400	0.70	0.34
17	64	F	Glomerulonephritis	19	490	0.68	0.35
18	65	F	Diabetes	27	230	0.52	0.50
Mean ± SD	62.9 ± 14.6			28.1 ± 7.5	349 ± 136	0.68 ± 0.08	0.39 ± 0.11
Poor Ultrafiltration							
19	31	F	Glomerulonephtiris	56	−60	0.89	0.28
20	52	F	Polycystic	30	80	0.77	0.34
21	63	F	Unknown	37	−100	0.95	0.25
22	51	F	Pyelonephritis	73	−160	0.93	0.22
23	63	M	Hypertension	16	−100	0.89	0.20
24	65	M	Obstruction	48	−260	0.98	0.22
25	57	F	Glomerulonephritis	11	90	0.77	0.32
Mean ± SD	54.6 ± 11.8			38.7 ± 22.1	−73 ± 125	0.88 ± 0.08	0.26 ± 0.05
P^b	<0.048			NS	<0.001	<0.001	<0.01

[a] UF, ultrafiltration; D/P, dialysate to plasma; D/Do, r- to 0-h dialysis; NS, not significant.

[b] Mann-Whitney *U* test.

all subjects with poor ultrafiltration than in any patient with normal ultrafiltration. The 4-hour/0-h dialysis ratios for glucose in the two groups overlapped, but the values for the normal patients were significantly higher ($P < 0.01$).

Figure 2 compares the concentrations of the different classes of lipid in the 4-h effluent from these patients. Total fatty acids were significantly higher in the poor ultrafiltrator subjects ($P < 0.008$). This was because of an increase in both phospholipids and neutral lipids, but only the latter rise was statistically significant ($P < 0.028$). The differences in the lipid fractions seen between the two groups of patients were the same when expressed as total amount of lipid in the dialysate (data not shown). There was no significant difference in the proportion of PtdCho in the phospholipid fractions. PtdCho represented 77.9 ± 10.7% of the total phospholipids in the normal patients, whereas in the poor ultrafiltrators, PtdCho represented 62.0 ± 22.5%. There was no difference in the fatty acid composition of the total lipids or total phospholipids. There was, however, a slight but significantly lower amount of stearic acid (18:0) in the PtdCho isolated from the poor ultrafiltration patients (Figure 3), whereas linolenic acid (18:3) was only detected in the PtdCho fraction of these same patients.

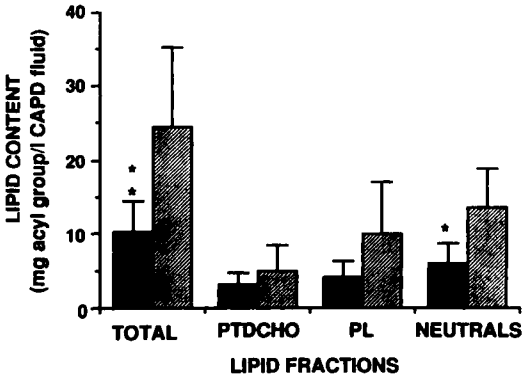

Figure 2. Lipid content of CAPD fluid from eight patients with normal ultrafiltration (closed box) and seven patients with poor ultrafiltration (hatched box). Results are expressed as means ± SD. * $P < 0.028$; ** $P < 0.008$ (Mann-Whitney *U* test). PL, total phospholipid.

DISCUSSION

This study clearly demonstrates that there is a time-dependent fall in the lipid content of the CAPD effluent. These changes affect phospholipid and neutral lipid fractions, but the overall distribution of different fatty acids in each class remains unchanged. In addition, the quantity of PtdCho, when expressed as a percentage of the total phospholipids,

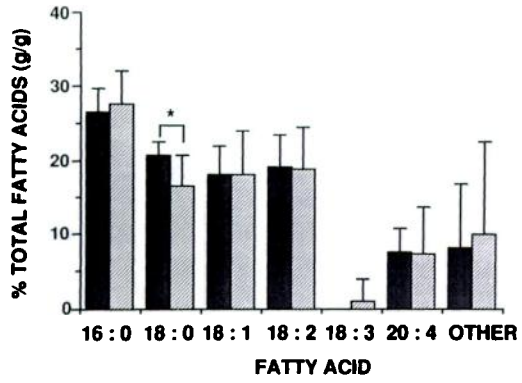

Figure 3. Fatty acid composition of the PtdCho fraction; closed box, patients with normal ultrafiltration; hatched box, patients with poor ultrafiltration. Results are expressed as means ± SD. * $P < 0.028$ (Mann-Whitney *U* test).

is relatively constant. Despite this marked fall in measured lipids, there was a nonsignificant rise in ultrafiltration during the same period, suggesting that there is no direct link between phospholipid concentration and fluid removal. In a separate group of patients, the lipid composition of dialysate from subjects with poor ultrafiltration was compared with that of individuals with good fluid removal. In this instance, there was a significantly higher concentration of neutral lipids in the dialysate of patients with poor ultrafiltration, whereas there were no significant differences in the phospholipid or PtdCho content. Fatty acid compositions of the lipid fractions were generally similar in the two groups.

Grahame *et al.* (4) first demonstrated the presence of phospholipids in peritoneal dialysis fluid. The composition bore a superficial resemblance to that of lung surfactant (6,13–15), with PtdCho forming 80% of the total phospholipid. It was proposed that the phospholipids formed a layer over the mesothelium, acting as a water-repellent barrier. However, as we have demonstrated in this study, the fatty acid composition of dialysate PtdCho is different from that found in alveolar surfactant (Figure 3). In the latter, palmitic acid (16:0) forms 73 to 81% of the acyl groups in PtdCho (15,16), whereas our results reveal that palmitic acid forms only 27% of the acyl groups in dialysate PtdCho, the remaining being made up of nearly equal amounts of stearic (18:0), oleic (18:1), and linoleic acids (18:2). Of the acyl groups in the total peritoneal phospholipids, palmitic acid represents 33%, a result that is comparable to that of Zeigler *et al.* (17). This difference in the proportions of fatty acids will markedly alter their physiologic behavior. A minimum level of 65% palmitic acid in PtdCho is required for normal surfactant function (15). It is these high levels of palmitate in lung surfactant that allow it to form stable monolayers at the air/water interface. The situation in the peritoneum is different because it is an aqueous compartment. There is no need for fatty acids, which are resistant to oxidation, nor is there a requirement for the phospholipids to be stable to constant expansion and contraction cycles (18). It is likely that peritoneal phospholipids, of the composition described in this study, would exist as bilayers at the cell surface or as micelles in the peritoneal fluid. As such, they could well have a lubricating function.

In an early study, DiPaolo *et al.* (3) reported a decrease in dialysate phospholipid with time on CAPD. Our results confirm this finding but further demonstrate that the proportion of PtdCho in the total dialysate phospholipid pool remains unchanged. In addition, the fatty acid composition of the PtdCho is constant with time on dialysis. A similar decline in protein concentrations has been previously described (19). This latter fall has been attributed to the relatively high permeability of the peritoneal membrane early on in dialysis treatment, which disappears with time (20). Whether this is the explanation for the fall in phospholipid is not known, because the source(s) of all of the dialysate lipids has not been clearly defined. Furthermore, there are no data available on plasma levels of phospholipids during CAPD treatment that would allow a clearance to be calculated. Because mesothelial cells themselves may synthesize phospholipid (5), the change could, in part, represent an alteration in mesothelial metabolism.

We found no differences in the phospholipid or PtdCho content of the dialysate effluent of patients with poor ultrafiltration dur to hyperabsorption of glucose (Type I ultrafiltration failure) as compared with that of controls. The discrepancy with the results of DiPaolo *et al.* (3) remains unexplained, but there are considerable methodologic differences. We used specific extraction and assay procedures. In addition, we studied only patients with increased peritoneal permeability. No details of the type of ultrafiltration failure were provided in the previous report. The biologic significance of the increased neutral lipids in our poor ultrafiltration group is at present uncertain. The concentration of phospholipids in the effluent is assumed to reflect production at the level of the peritoneum; however, there is no way of validating this. Equally, the loss of ultrafiltration could reflect a decrease in the surface area and this could also account for the decline in phospholipid release. Type I ultrafiltration loss, however, is associated with an increased absorption of glucose and an increased appearance of creatinine in the peritoneal cavity. This would imply a functional increase in surface area rather than a decrease. The ip administration of PtdCho has been reported to improve ultrafiltration by either directly affecting peritoneal water transport (3,7) or by decreasing lymphatic flow (8,9). Improvements were originally only observed in

patients with impaired ultrafiltration capacity (3), although subsequently, it has been suggested there is an effect on ultrafiltration rate in patients with normal fluid removal (21). This discrepancy has been found in rabbit studies, where the ip administration of PtdCho had no effect over 1 h (22) but did seem to increase ultrafiltration after at least 2 h (7). Of concern, however, was the observation that PtdCho increased the formation of intraabdominal adhesions in rats (23), although a later report from the same group concluded that PtdCho could reduce adhesion formation (24).

The po administration of PtdCho has also been reported by some authors (3,10) to improve ultrafiltration, although others (25,26) were unable to confirm this finding. As PtdCho is degraded in the gut (27), any such treatment could only work by supplying supplementary fatty acids and/or choline. The results in this study would indicate, however, that substrate availability is adequate and is not a rate-limiting step in the formation of phospholipid. In addition, none of the published studies suggesting a therapeutic benefit of added phospholipid have given details of the fatty acid composition of the administered preparation. Whether or not large pharmacologic doses of ip PtdCho can enhance fluid removal remains uncertain, but our data certainly exclude a deficiency of this phospholipid in patients with Type I ultrafiltration failure or any direct relationship between PtdCho concentration and fluid removal.

ACKNOWLEDGMENTS

This work was supported by grants from Baxter Healthcare and the Kidney Research Unit Foundation for Wales. The authors thank Cheryl Patterson for typing the manuscript.

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