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CORRESPONDENCE

First Report on the Use of Endoluminal FAS Brush to Restore Flow in Peritoneal Catheters

Editor:

We read with greater interest Tranter's report on the use of the FAS endoluminal brush in the management of peritoneal catheter occlusion, successful in five cases and unsuccessful in two (1). The use of such a brush for clearing peritoneal dialysis (PD) catheter obstruction has already been described by us, and has been suggested as an alternative approach to restoring catheter patency (2).

Since the initial case, we have used the FAS endoluminal brush in five other cases with PD catheter flow obstruction. Its use restored catheter flow in 4 cases (3 adults and 1 child) by successfully removing fibrin clots. Syringe manipulation with heparinized dialysate solution was attempted by trying to flush fluid in and out with a 10-mL syringe as a first step, in all cases without success. One patient had to restart dialysis 6 months after a renal transplant and flow could be obtained only following the use of the endoluminal brush. In another case, obstruction was associated with peritonitis, and in 2 patients, there was no apparent cause for fibrin clots. Failure to restore catheter flow occurred in 1 patient due to catheter malposition during an episode of peritonitis.

Our previous case and the ones now reported are in agreement with Tranter's suggestion that the brush might be useful only when the obstruction is caused by fibrin.

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High Incidence of Encapsulating Peritoneal Sclerosis in Pediatric Patients on Peritoneal Dialysis Longer Than 10 Years

Editor:

In our previous study (1), 11 cases (1.6%) of encapsulating peritoneal sclerosis (EPS) were found in 687 pediatric patients that started peritoneal dialysis (PD) in Japan between 1981 and the end of 1996 under 16 years of age. According to recent reports in adults on PD, an increased incidence of EPS has been reported (2). Using the data of all EPS cases, we investigated whether the incidence of EPS in pediatric patients had also increased recently.

Of 843 pediatric patients that had started PD by the end of 1999, 17 (2%) had developed EPS, including 11 cases diagnosed by 1996. Only 3 patients developed EPS from 1988 to 1993; however, 14 patients developed EPS from 1994 to 1999. Mean age was 8.0 ± 4.6 years at the start of PD and 18 ± 4.8 years at diagnosis of EPS. All EPS cases had received PD for longer than 5 years. Mean PD duration prior to EPS (10.3 ± 3 years) was much longer than that for patients not developing EPS (3.7 ± 4.6 years). The incidence of EPS was 6.6% in the patients on PD for longer than 5 years, and 22% in patients on PD longer than 10 years. The symptoms and signs included fever (8 patients; 47%), ascites (7; 41%), abdominal distension and/or abdominal tumor (7; 41%), and blood-stained effluent (7; 41%). The most frequently reported radiological findings were peritoneal calcification (13; 76%) and peritoneal thickening (10; 59%). Peritoneal biopsy was performed in 12 patients, and all confirmed peritoneal sclerosis. None of the 17 patients had residual renal function and 13 (76%) patients had impaired peritoneal ultrafiltration. The average peritonitis rate was 0.43 episodes/year. In 9 patients, EPS was diagnosed immediately following an episode of peritonitis. A peritoneal equilibration test (PET) was performed in 6 patients before the onset of EPS: 3 were categorized as

high, 2 as low-average, and 1 as high-average. Fourteen patients transferred to hemodialysis (HD) and 3 died prior to initiating HD. Five patients transferred to HD before the diagnosis of EPS and 9 transferred after the diagnosis. The only immunosuppressive therapy given was corticosteroids, which was tried in 6 cases. Only 1 patient received surgical treatment. The cause of death was septicemia in 2 patients and hepatic failure in 1 patient.

The incidence of EPS was 2% by the end of 1999, higher than that in our previous report, and had a tendency to increase with PD duration. The increased incidence of EPS found in this study could be a consequence of an increase in the number of pediatric patients on long-term PD in Japan. According to our registry data, in 1991, only 48 (11%) of 434 pediatric patients had received PD for longer than 5 years. However, in 1999, 259 (28%) and 50 (5%) of 931 pediatric patients had been on PD for longer than 5 and 10 years, respectively. Based on our experience, to avoid the development of EPS, special attention is required in pediatric patients continuing PD for longer than 10 years.

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Pneumococcal and Gonococcal Peritonitis Due to Vaginitis

Editor:

Peritoneal dialysis (PD) peritonitis associated with ascending vaginitis is very uncommon. Such peritonitis is typically caused by enteric pathogens, rarely by organisms that cause sexually transmitted diseases. We report a 31-year-old female receiving continuous ambulatory PD for end-stage renal disease due to hypertensive nephrosclerosis, who experienced pneumococcal peritonitis and a separate episode of gonococcal peritonitis, both associated with mild vaginitis.

The patient first presented with 1 day of abdominal pain, fevers, and cloudy PD effluent. Her peripheral leukocyte count was 18 700/mm^3 (46% bands). The PD effluent leukocyte count was 27 500/mm^3 (99% PMNs) and Gram stain had numerous gram-positive diplococci that grew *Streptococcus pneumoniae*. Abdominal/pelvic computed tomography (CT) scan was normal. Pelvic examination demonstrated cervical discomfort and showed scant vaginal secretions. A Papanicolaou smear had leukocytes. The wet prep demonstrated *Trichomonas vaginalis*; DNA probes on vaginal secretions were negative for *Neisseria* and *Chlamydia*. The peritonitis was successfully treated with intraperitoneal vancomycin. Thirteen months later, she experienced diffuse abdominal pain and cloudy PD effluent with a leukocyte count of 1925/mm^3 (93% PMNs). Intracellular gram-negative cocci were seen on the PD fluid Gram stain and culture grew *N. gonorrhea*. On pelvic examination, cervical discomfort was present. Leukocytes were present on a Papanicolaou smear. DNA probe was positive for *N. gonorrhea*. The patient demonstrated rapid clinical improvement following intravenous and intraperitoneal ceftriaxone.

This case describes episodes of PD peritonitis due to pneumococci and then gonococci. Although the patient did not report vaginal discharge, each peritonitis episode was associated with abnormal pelvic examinations and mild vaginitis. A focus of origin for the pneumococcal peritonitis other than vaginitis was not evident. Pneumococcal peritonitis, however, has been reported in women with retrograde menstruation while using intrauterine contraceptive devices (IUCD) (1,2). The IUCDs in those reports possibly caused a failure of normal uterine mucosal immunity, promoting ascending vaginal infection. Our patient did not have an IUCD. The episode of *N. gonorrhea* was associated with a positive vaginal DNA probe for *N. gonorrhea,* however, evidence for dissemination of the gonococcal infection was not present. Peritonitis due to *N. gonorrhea* is extremely rare (3,4), although cases of non-gonococcal *Neisseria* peritonitis, including *N. meningitides*, have been reported (5,6). Although the PD peritonitis episodes were due to encapsulated bacterial strains, she had normal liver and spleen anatomy on CT, had no prior personal or family history of infections with encapsulated bacteria, and demonstrated normal serum complement levels (C3, C4, and C9 were normal).

This report of PD peritonitis due to pneumococci and gonococci illustrates that vaginal infections can lead to PD peritonitis in women without IUCD use. We suggest a thorough gynecological history and pelvic examination is warranted in females with PD peritonitis due to atypical bacterial strains.

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Gastric Emptying in Renal Failure Patients Using the ^{13}C-Octanoic Acid Breath Test: Facts and Artifacts

Editor:

We read with interest the article by Van Vlem *et al.* (1). Although the hypothesis stated by the authors is very attractive, we feel that major methodological inaccuracies account for the differences found in the different test conditions. In our opinion, two major mistakes have been made:

1. The premise of the study is that the ^{13}C-octanoic acid breath test, validated in healthy subjects and patients with different gastrointestinal and hepatic diseases and metabolic abnormalities such as diabetes mellitus, is a valid test to be used in patients with severe chronic renal failure, and even in dialysis patients [both hemodialysis (HD) and peritoneal dialysis (PD) patients]. This is only true when "postgastric processing" of the ^{13}C-octanoic acid (2) is similar in these patients compared with normal subjects. But there are numerous pathophysiological processes present in dialysis patients that may interfere with this "postgastric processing": (a) Ultrafiltration decreases plasma volume and thereby portal blood flow, hence inducing a shift from hepatic to muscle oxidation of octanoic acid. (b) Ultrafiltration decreases plasma volume and dialysis interferes with the bicarbonate pool. Both factors are very important for the dynamic exchange of $^{13}CO_2$ with the fast and slow bicarbonate pool and the loss of the ^{13}C marker via feces, urine, ultrafiltrate, and incorporation in the bone. (c) Metabolic acidosis also interferes with different cell functions. Therefore, it cannot be assumed that "postgastric processing" of ^{13}C-octanoic acid is similar in dialysis patients compared with normal volunteers without doing validation studies (*e.g.*, intraduodenal instillation studies of ^{13}C-octanoic acid in uremic patients with and without ultrafiltration, bicarbonate substitution). Moreover, in severe uremic patients, electrolyte disturbances (especially of cationic electrolytes) may delay gastric emptying since they may interfere with smooth muscle contractility. It was recently shown that a single dose of $MgCl_2$ even in healthy volunteers provokes a dramatic decrease in gastric emptying rate, resulting in hampered protein digestion and lipolysis (3). The interpretation of gastric emptying without measuring (individually different) fluxes of calcium, potassium, and magnesium during dialysis is incorrect. All dialysis patients are subject to a multidrug regime, a lot of which may interfere with gastric emptying (*e.g.*, beta-blockers, tranquilizers, sedatives, benzodiazepines), accumulate, are not metabolized after an overnight fast, and are dialyzed only partially.
2. The most obvious methodological flaw, however, is that the authors did not check their dialysis fluids for background enrichment of $^{13}CO_2$. The advantage of ^{14}C-labeled breath tests is that, in nature, no background ^{14}C is present in the environment; this is not the case for ^{13}C, since it is incorporated in many of plants by photosynthesis. Therefore, a lot of substrates are "contaminated" with ^{13}C and, when using ^{13}C-labeled breath tests, interference has to be checked from natural ^{13}C enrichment of the test meal used (4),

but also by other substances given before or during the breath test. A well-known exclusion criterion for ^{13}C-labeled breath tests is concurrent intravenous solutions of glucose or bicarbonate. Also, the solutions used for HD (glucose-containing) and PD may be differently enriched, according to the substrates of glucose, amino acids, *etc.* used, and this may vary seasonally according to the crops used for making these substrates. This background ^{13}C enrichment should be subtracted from the $^{13}CO_2$ excretion curve obtained from the ^{13}C-octanoic acid used to dope the test meal. We noted that $^{13}CO_2$ excretion in the breath of PD patients *not* performing a ^{13}C-octanoic acid breath test increased significantly after intraperitoneal administration of PD solutions (both glucose- and icodextrin-containing). This bias is influenced not only by the substrate used (*e.g.*, glucose 1.36% vs icodextrin), but also by the permeability of the membrane (*e.g.*, high vs low transporter) and dwell time. To quantify this bias on the gastric emptying parameters of a $^{13}CO_2$ excretion curve obtained after a ^{13}C-octanoic acid breath test in traditional standard test conditions, we added the mean ^{13}C background enrichment of one 4-hour dwell of glucose 1.36% measured in 3 PD patients to a $^{13}CO_2$ excretion curve of 2 normal subjects (Figure 1). As noted, there is a shift of the $^{13}CO_2$ excretion curve, mainly in the descending part of the curve, that causes a substantial delay in the half emptying time ($t_{½}$ shifts from 72 to 98 and from 79 to 118 minutes) and is remarkably similar to the differences measured by the authors in different articles (1,5,6). Therefore, we are convinced that the ^{13}C-octanoic acid breath test is not a valid test in HD and PD settings, and that the differences seen in this and previous reports (1,5,6) merely reflect differences in fluxes of ^{13}C-enriched substrates rather than differences in gastric emptying.

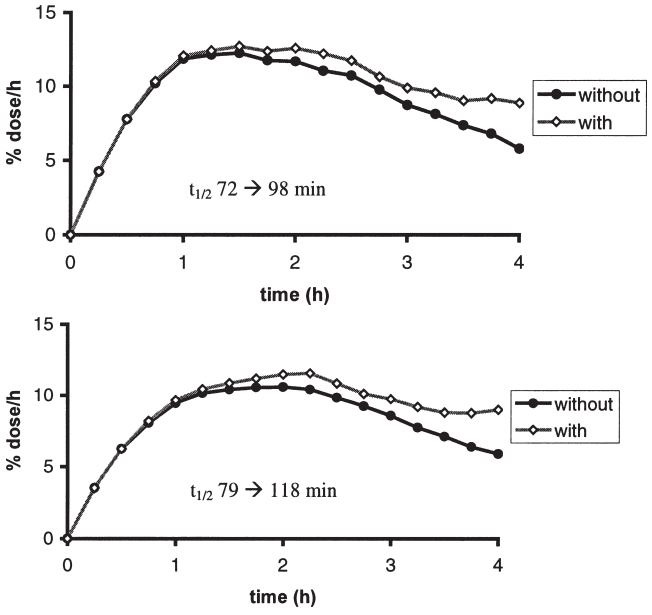

Figure 1 — Effect of the addition of the mean ^{13}C background of a 4-hour dwell of 2 L glucose 1.36% in 3 peritoneal dialysis patients on the $^{13}CO_2$ excretion curve in 2 normal healthy volunteers.

Part of the problems discussed in both paragraphs may be elucidated by showing $^{13}CO_2$ excretion curves and individual gastric emptying data of several gastric emptying parameters (not only half emptying time, but also lag phase, gastric emptying coefficient). However, the addition of multiple interferences (see item 1) may be the cause of the wide variability noted in the half emptying time given by the authors.

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Reply to Maes *et al.*

Editor:

We thank Dr. Maes *et al.* for their comments regarding our paper, "Influence of Dialysate on Gastric Emptying Time in Peritoneal Dialysis Patients" (1). The criticism by Dr. Maes *et al.* suggests that methodological inaccuracies would account for the differences in gastric emptying between the various test conditions, and that these differences would merely reflect differences in fluxes of ^{13}C-enriched substrates.

We agree that, for our conclusions to be valid, the postgastric processing of octanoic acid in peritoneal dialysis (PD) patients should be similar to other populations in which this gastric emptying breath test has been validated (2,3). After all, the ^{13}C-octanoic acid breath test measures the time between the oral ingestion of ^{13}C-octanoic acid and the expiration of $^{13}CO_2$. Calculation of gastric emptying time from this ^{13}C recovery relies on the fact that gastric emptying is the true rate-limiting step, whereas the process of duodenal absorption, portal transport, hepatic oxidation, transport to the lungs, and exhalation (together called "postgastric processing") is both fast and stable in various populations (2,3).

Doctor Maes *et al.* suggest that several pathophysiological processes could interfere with "postgastric processing" in PD patients. Plasma volume changes during a single dwell of PD when an ultralow sodium concentration is used, but not when a normal sodium concentration is used (4). It is therefore highly unlikely that, in PD patients, the rate and the completeness by which the absorbed octanoic acid is transported from the duodenum to the liver via the portal vein and hepatic extraction, and consequently the liver/muscle oxidation ratio, would change during PD. Since there is no physicochemical difference between ^{13}C and ^{12}C, all buffering processes are identical for both isotopes per unit of time, so that no differences in $^{13}C/^{12}C$ ratio can occur. In addition, the gastric half emptying time results are independent of both the mass of $^{13}CO_2$ administered and the endogenous net CO_2 production. The liver capacity for β-oxidation of medium-chain fatty acids is very high. Even in cirrhosis and hepatic failure, the ^{13}C-octanoic acid breath test has been validated (Schoonjans *et al.*, unpublished observations). Their further criticism concerning the possible interference of acidosis is, in our opinion, not valid because continuous ambulatory PD patients are usually in normal acid–base balance.

The next arguments (electrolytes, medication) do not address the alleged methodological bias, but both factors can have a real impact on gastric emptying. Electrolyte disturbances can be present in chronic renal failure patients, and many patients indeed take multiple drugs. However, both items are an intrinsic part of uremic syndrome and could contribute to both the delay in gastric emptying *per se* and the variability between patients, but not to differences within patients observed between glucose and icodextrin, or glucose and "empty abdomen" conditions.

Since corn is used as the base material for the production of both glucose and icodextrin PD dialysates, these could indeed contain an enhanced concentration of ^{13}C. To the best of our knowledge there are no published data evaluating the effect of glucose or icodextrin dialysates *per se* on fasting $^{13}CO_2$ expiration. Therefore, we performed a fasting analysis of ^{13}C in PD patients. In contrast to what Maes *et al.* report in their letter, the fasting isotopic delta values after the exchange of an overnight icodextrin for glucose 1.36% or 2.27% did not rise at all (Figure 1). Therefore, the "baseline" on which the isotopic delta of ^{13}C after ingestion of ^{13}C-octanoic acid has to be added is flat and stable.

We provide in Figure 2 the isotopic delta curves as asked for by Maes *et al.* The shape of our curves (Figure 2) show both a slower increase and a slower decrease in ^{13}C expiration in the glucose curve compared to the icodextrin and the "empty abdomen" curve. The delay in Maes *et al.*'s curves is due to a slight increase in the values beyond the first hour. It is clear that the biased addition curves simulating the effect of an eventual ^{13}C source in the PD solution (as shown by Maes *et al.*) show a completely different pattern than the curves we obtained in our experiments demonstrating a true delay in gastric emptying.

Several previous studies evaluated gastric emptying in PD patients (5–8). Despite a marked heterogeneity in methods for the assessment of gastric emptying, in the PD populations studied, and in the statistical methods, all the studies have reported a delay in gastric emptying in PD patients compared to healthy volunteers when glucose dialysate was present in the peritoneal cavity. Our findings in glucose-treated PD patients are therefore in concordance with the remaining literature. We are convinced that the data and conclusions reported in our paper are valid, that the octanoic acid breath test is applicable in stable PD patients, and that the glucose data can serve as a valid comparative marker for alternative, nonglucose osmotic agents.

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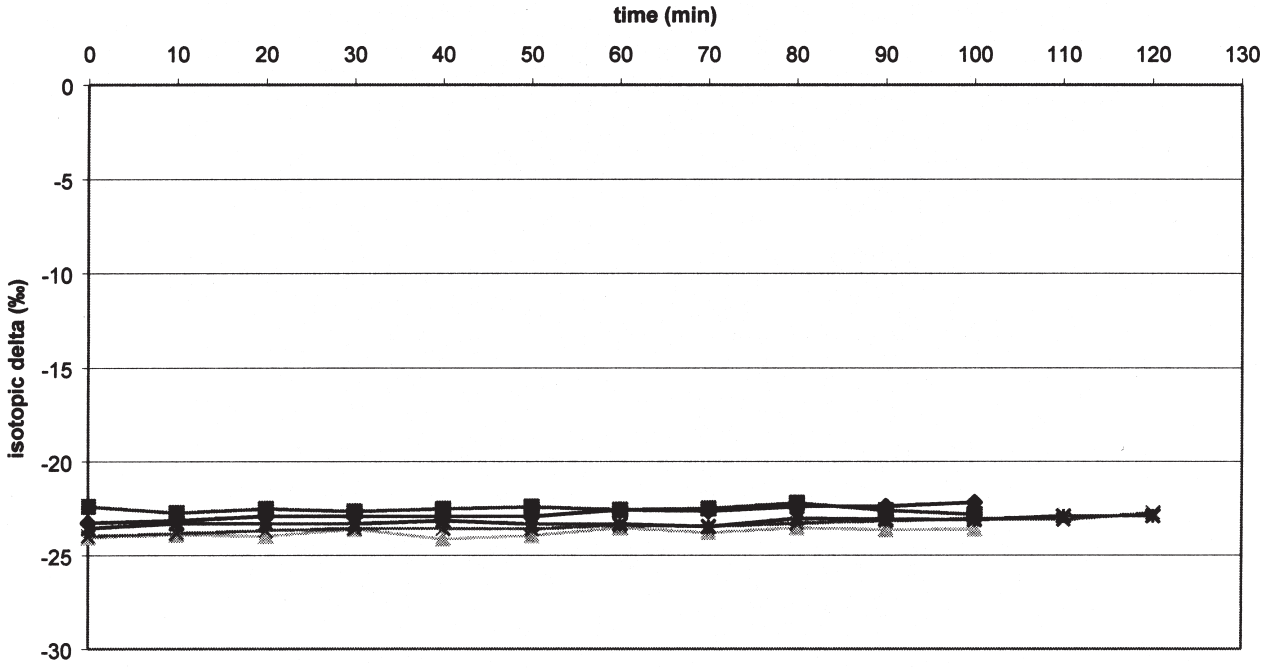

Figure 1 — Fasting analysis of ^{13}C in peritoneal dialysis patients. The fasting isotopic delta values [$\delta^{13}C = (R_{sample} - R_{standard}) / R_{standard}$, where $R = {}^{13}C/{}^{12}C$ and $R_{standard} = 0.011\ 237\ 2$) after the exchange of an overnight icodextrin for glucose 1.36% or 2.27% ($n = 5$).

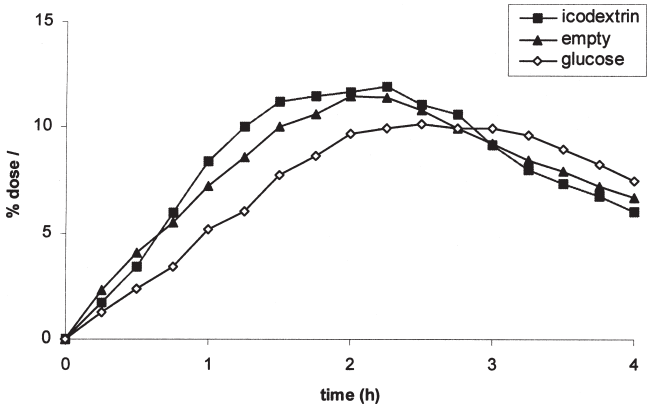

Figure 2 — Isotopic delta curves of a peritoneal dialysis patient after eating the ^{13}C-octanoic acid-containing test meal, comparing gastric emptying after drainage of the overnight dwell (empty t½ = 104 minutes), gastric emptying when icodextrin was used (t½ = 85 min), and gastric emptying when glucose 1.36% dialysate was used (t½ = 153 min).

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Is Icodextrin Peritonitis a Reaction to Bacterial Endotoxins?

Editor:

Goffin and co-workers recently observed a sterile peritonitis as a possible delayed manifestation of icodextrin hypersensitivity after an episode of acute peritonitis due to *Streptococcus viridans* in a 31-year-old woman on continuous ambulatory peritoneal dialysis (CAPD) (2-L icodextrin bag during the night). Effluents of icodextrin bags, but not of dextrose bags, were cloudy and contained 100 white cells (WBC)/mm^3 (82% macrophages) (1). We report a case of cloudy effluent with prevalence of macrophages in a patient with diverticulosis after 4 months of Extraneal (7.5% icodextrin; Baxter Healthcare, Deerfield, Illinois, USA).

A 42-year-old man with end-stage renal disease due to autosomal dominant polycystic kidney disease had been on automated peritoneal dialysis since March 2001. Extraneal was used to increase both ultrafiltration and total clearance and to avoid abdominal pain when his abdomen was empty. He had no previous history of atopy or allergies.

The patient was admitted to our hospital on 20 July suffering from mild abdominal pain and malaise; he had no fever. His peritoneal effluent contained 180 WBC/mm^3 (90% monocytes, 10% neutrophils). Intraperitoneal antibiotic therapy was started with cefazolin and tobramycin. The next peritoneal effluent sample showed a white cell count ranging from 40 to 180 WBC/mm^3 (70% – 90% monocytes, 10% – 30% neutrophils). There were no bacteria, fungi, or acid-fast bacilli grown in cultures of the effluent. Plain abdominal x ray was normal. Enema showed signs of sigmoid diverticulosis.

We hypothesized that the peritoneal inflammation could have been induced by icodextrin and its use was stopped, with a dramatic improvement in signs and symptoms in the following 24 – 48 hours.

On 12 August 2001, Extraneal was restarted. After 38 days, the abdominal effluent became cloudy again with 190 WBC/mm^3 (100% monocytes). He had neither pain nor fever.

During the course of this peritonitis episode, there were no changes in ultrafiltration volume, permeability pattern, or blood biochemistry. On withdrawal of icodextrin, the cell count in the effluent dropped dramatically. In summary, our patient showed refractory peritonitis under icodextrin, with spontaneous recovery after its discontinuation and recurrence on re-exposure.

Reichel *et al.* reported a patient that developed sterile peritonitis, after 9 months on Extraneal, just after an episode of peritonitis due to *Streptococcus agalactiae*; dialysate effluent contained 1570 leukocytes/mm^3 (36% neutrophils, 25% lymphocytes, 32% monocytes). Extraneal was discontinued and leukocytosis quickly returned to normal without using antibiotics. These authors suggested a possible association of this sterile peritonitis with the previous bacterial peritonitis caused by a bacterium infrequent in peritoneal dialysis peritonitis (2).

Finally, Pagniez *et al.* reported two cases of sterile peritonitis that were probably both due to icodextrin. In both cases, an opaque enema showed sigmoid diverticulosis. There had been a transient response to antibiotics, that is, a decrease in the number and a disappearance of WBC in the dialysate only for some days after the antibiotic treatment. Their interpretation was that those two episodes of peritonitis were inflammatory reactions to bacterial endotoxins (3).

Other authors have suggested that sterile peritonitis in patients on icodextrin might be allergic reactions similar to skin lesions (4,5).

Another possible explanation, according to Pagniez *et al.*, is that the ultrafiltration caused by icodextrin might induce translocation of bacterial endotoxins through the weakened wall of infected diverticula. We believe that this hypothesis explains why, in some episodes of sterile peritonitis, there are the following findings: (1) a previous peritoneal infection due to occasional commensals of the bowel (*Streptococcus agalactiae*, *Streptococcus viridans*), (2) prevalence of macrophages in the peritoneal effluent, (3) a long time lag between initiation of icodextrin and these complications (8 – 450 days), and (4) a temporary effect of antibiotic therapy that could have reduced intestinal bacteria and endotoxin concentration.

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Treatment with Sevelamer Decreases Bicarbonate Levels in Peritoneal Dialysis Patients

Editor:

Sevelamer is a new aluminum- and calcium-free molecule with proven efficacy in lowering phosphate without raising calcium levels (1). Some studies report a decrease in bicarbonate levels following initiation of sevelamer treatment (2). In this regard, our group has recently confirmed that sevelamer decreases bicarbonate levels in hemodialysis patients (3). At present, there is not much information about the use of sevelamer in peritoneal dialysis (PD) patients (4).

We investigated the effects of sevelamer on bicarbonate levels in 13 stable chronic PD patients. Six patients had been previously treated with calcium acetate (mean dose 1.9 g), 6 patients were on calcium carbonate (mean dose 2.4 g), and 4 were on aluminum hydroxide (mean dose 800 mg). Seven patients were on "low" calcium dialysate (1.25 mmol/L) and 6 patients were on "standard" calcium dialysate (1.75 mmol/L).

Analysis of variance (ANOVA) was used to compare baseline with treatment controls (1, 2, 4, and 6 months after initiation of sevelamer). The average dose of sevelamer was 2.12 g and 2.8 g at the time of the first- and sixth-month controls, respectively. Sevelamer was well tolerated in 11 patients (84.6%). In 2 patients, it had to be discontinued because of gastrointestinal problems. During the 6-month follow-up period, there was an early significant reduction in bicarbonate levels: from 24.9 mmol/L [95% confidence interval (CI) 23.3 – 27.3] to 23.32 mmol/L (95% CI 22.1 – 25.7) at first-month control, and to 22.59 mmol/L (95% CI 21.4 – 24.8) at second-month control (Figure 1). This decrease in bicarbonate levels was partially recovered at successive months. However, in most patients, $CaCO_3$ was added from the third month onwards, which may be responsible for this recovery. Patients on standard calcium dialysate (lactate 35 mmol/L) had lower baseline bicarbonate levels (bicarbonate 23.9 mmol/L) compared to patients on low calcium dialysate (lactate 40 mmol/L) (bicarbonate 26.5 mmol/L). Therefore, those patients also achieved lower minimum bicarbonate levels (22.3 vs 24.06 mmol/L).

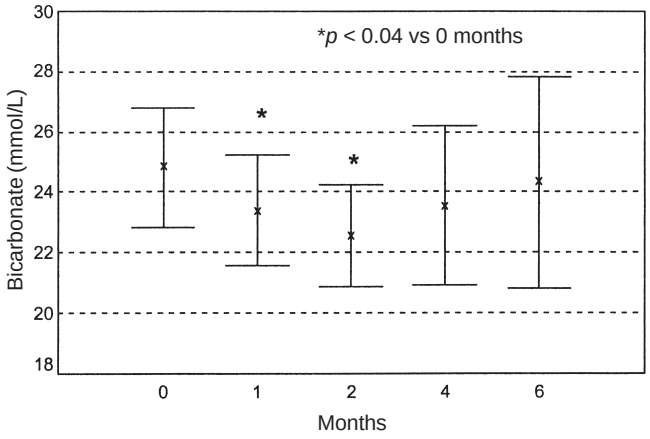

Figure 1 — Bicarbonate levels at baseline (0 months) and after the introduction of sevelamer.

According to these results, in PD there is an early reduction in bicarbonate levels when sevelamer substitutes calcium salts as phosphate binder. The discontinuation of calcium salts probably justifies the worsening of acidosis. In fact, partial recovery of bicarbonate levels was observed from the third follow-up month when calcium salts were added. However, sevelamer may also directly play a role in acidosis due to its biochemical structure. It is composed of partially protonated amines that interact with phosphate anions, releasing HCl. Supporting this fact, the 2 patients that discontinued sevelamer had an increase in bicarbonate levels that preceded the reintroduction of calcium salts. Irrespective of the mechanism, bone buffering may be present and long-term deleterious effects of acidosis on mineral metabolism cannot be excluded. Thus, attention must be paid to the acidosis risk of sevelamer use, especially in PD patients using standard calcium dialysate, without calcium salts.

Based on our clinical observation, we recommend strict monitoring of bicarbonate in PD patients on sevelamer.

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No Need to Measure Serum Aluminum in Patients Starting Chronic Ambulatory Peritoneal Dialysis

Editor:

Aluminum is one of the most abundant minerals in the Earth, but usually its content in the human body is very low (no more than 30 mg). This phenomenon is due to its low tissue incorporation and its prompt renal excretion (1). However, in patients with chronic renal failure, aluminum can accumulate and be deposited in both bone and brain and can lead to several complications, such as adynamic bone disease, microcytic anemia, and neurological toxicity (2). Increased aluminum blood levels may be observed in patients with chronic renal failure if they ingest aluminum hydroxide to bind phosphorus, and are taking other phosphorus-containing medications, such as sucralfate, and are receiving aluminum in their drinking water (3,4).

Even though we have not used aluminum-containing phosphate binders in our area in the past 5 to 6 years, it has been our practice to measure blood aluminum levels in all our new peritoneal dialysis (PD) patients to ensure that we are not missing a case of aluminum overload. Recently, we reviewed this practice and this letter presents our findings.

We recorded serum aluminum measurements in 92 patients during their first month of PD. We also recorded other parameters for correlation with aluminum levels, such as parathyroid hormone, transferrin, ferritin, hemoglobin, hematocrit, total iron, iron saturation, serum phosphates, total calcium, corrected calcium, and ionic calcium.

The mean (±SD) serum aluminum value was 178.9 ± 156.3 nmol/L, range 26 – 812 nmol/L (normal values < 560 nmol/L). Only 4 patients had values above the upper limit of normal (797, 789, 704, 812 nmol/L). The only parameters that had a small but significant correlation with serum aluminum levels were ferritin, iron saturation, and total iron. The correlation with these entities can be explained because the body handles both of these divalent cations (iron and aluminum) in a similar manner (5).

Thus we have concluded that serum aluminum levels are not elevated in new patients that have not received aluminum-containing drugs previously, and therefore there is no need to measure aluminum levels in this population.

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