Bowel bacterial overgrowth syndrome (BBOS) is an important cause of gastrointestinal (GI) abnormalities. Proinflammatory cytokines (PICs) are excessively produced and accumulate because of kidney failure in dialysis patients who experience chronic infections such as BBOS. We explored the association between GI function, BBOS, and the malnutrition, inflammation, and atherosclerosis (MIA) syndrome.

We studied GI malabsorption and maldigestion by analyzing fecal starch, sugar, fat, and nitrogen; intestinal protein permeability (α₁-antitrypsin fecal clearance); and fecal chymotrypsin. We evaluated BBOS by breath hydrogen test (BHT) after a 3-day fat-and-carbohydrate-overload diet.

Positive BHT was present in 10 patients, showing a high prevalence of GI macronutrient malabsorption and maldigestion, and compared with the other patients, the highest plasma levels of tumor necrosis factor α and interleukin 6 and lower levels of albumin and prealbumin. Those 10 patients were treated with a combination of several antibiotics, including neomycin, amoxicillin–clavulanate, and quinolones. Between 2 and 3 months later, the BHT, markers of nutrition, and PIC were re-tested. All treated patients showed an improvement in nutrition status and a lesser inflammatory pattern.

The BBOS infectious process is found frequently in dialysis patients in association with GI malabsorption and maldigestion, malnutrition, and systemic inflammation. Hyperproduction of PIC because of BBOS induces MIA through a double pathway: GI disorders and deleterious systemic effects.

Key words
Bowel bacterial overgrowth syndrome, gastrointestinal absorption, MIA syndrome

Introduction
Malnutrition, a multifactorial complication frequently found in peritoneal dialysis (PD) patients, is associated with elevated morbidity and mortality (1). The first step in achieving an adequate nutrition status is to maintain gastrointestinal (GI) integrity. Various GI disorders are frequently seen in patients with chronic renal failure, including anatomic disorders such as gastritis, nodular duodenitis, and bowel angiodysplasia (2,3). Functional abnormalities include increased absorption, mucosal enzymatic activity, and permeability of the bowel mucosa that lead to delayed motility disorders, and abnormalities in bile composition and GI peptide profile. Moreover, frequent chronic (and occasionally silent) GI infections such as Helicobacter pylori and microbiologic bacterial flora changes, with overgrowth, have been described (3–5). In the uremic milieu, persistent infection induces an accumulation of proinflammatory cytokines because of overproduction and especially because of retention on account of lack of renal excretion. In fact, infection with Helicobacter pylori is associated with high plasma levels of tumor necrosis factor α (TNFα) and severe anorexia and cachexia in PD patients; eradication of the infection has been shown to induce a dramatic nutritional recovery (5).

Bowel bacterial overgrowth syndrome (BBOS) is another infectious complication frequently found in
uremia (6) that has been associated with anatomic (2) and GI immuno-abnormalities (7) and with elevated cytokines (8). Directly or indirectly, cytokines induce malnutrition through their systemic effects on appetite (anorexia), muscle mass (wasting), and hepatic protein synthesis (diminishment). Proinflammatory cytokines also induce cardiovascular risk because of endothelial dysfunction and dyslipidemia, eventually leading to high morbidity and mortality. This clinical complex has been called the malnutrition, inflammation, and atherosclerosis (MIA) syndrome (9). In the present paper, we tried to establish a link between the presence of BBOS and MIA syndrome in PD patients.

Patients and methods
At the start of the study, we screened all active PD patients treated at our unit (n = 49) for malabsorption and maldigestion, bowel permeability, and BBOS and Helicobacter pylori [by breath hydrogen test (BHT)]. Although we found a wide spectrum of GI functional abnormalities (10), here we exclusively show the clinical course, before and after antibiotic therapy, of 10 patients diagnosed with BBOS.

Of the 10 affected patients, 7 showed dyspepsia, nausea, and occasional vomiting; 5 were experiencing sporadic abdominal pain; and 4 had intermittent diarrhea or constipation. Varying degrees of anorexia were present in 4 patients, and 4 had a prior diagnosis of diverticulosis. In 3 patients, no GI disease had ever been diagnosed. No acute disorders were present in these patients during the 2 months before the study. Patients with known intestinal, pancreatic, or chronic liver diseases, neoplasms, abdominal radiation, infections, or obstructive lung diseases were excluded. No patient was receiving pancreatic supplements. The causes of chronic renal failure were diabetes in 3 patients, glomerulonephritis in 3, nephrosclerosis in 2, and chronic pyelonephritis in 1. Mean dialysis vintage in these patients at the start of the study was 34.1 ± 31.3 months (range: 3 – 66 months).

All 10 patients were using oral phosphorus binders; 9 were using oral calcium supplements; 6, vitamin D₃ derivatives; 6, osmotic laxatives (lactulose); 2, GI prokinetic drugs (domperidone); and 2, ranitidine. Omeprazole was withdrawn 15 – 10 days before study start.

Peritoneal ultrafiltration was considered to be normal in 8 patients (800 ± 200 mL daily, with a combination of 1.36% and 2.27% dextrose) and high in 1 patient (using mostly 1.36% dextrose). The local institutional ethics committee was informed about our aims and methods, and patients gave informed consent for the study.

Parameters of dialysis adequacy
In all patients, we determined parameters of dialysis adequacy: Kt/V urea, normalized protein equivalent of nitrogen appearance (nPNA), and residual renal function. We also measured (in fasting conditions) markers of nutrition: long-term markers—serum creatinine, albumin, and cholesterol [colorimetric method, Hitachi 704 analyzer (Boehringer Mannheim, Mannheim, Germany)]; and shorter-term markers—phosphorus, potassium, ferritin, nitrogen (colorimetric method, Hitachi 704 analyzer); iron (Hitachi 911 analyzer); prealbumin, retinol-binding protein, transferrin, fibronectin, α₁-antitrypsin [immunonephelometric method, Behring Nephelometer (Behringwerte AG, Marbus, Germany)]; and vitamin B₁₂ and folic acid (radioimmunoanalysis). Eating motivation was evaluated with an eating motivation visual analog scale (VAS) by Hill and Barkeling (11,12) that included 5 questions. The results were are marked on a horizontal scale of 0 to 100.

Intestinal absorption
To investigate intestinal absorption, all patients undertook a 3-day fat-overload diet (80 g daily) using a diet guide from our laboratory that described how to enrich food with olive oil. On day 4 (after the 3-day fat overload diet), the patients collected a 24-hour stool sample for these determinations: stool weight, water content, nitrogen, starch, sugar, and total fat. Malabsorption syndrome was evaluated by fecal near-infrared reflectance analysis (12). Fecal chymotrypsin (spectrophotometry) was used to evaluate pancreatic exocrine function [normal range: >23 U/g of stool (10)]. To evaluate GI protein losses, fecal clearance of α₁-antitrypsin (FCαAT) was determined by the immunonephelometric method (normal: <12 mL/24 h). High fecal values of nitrogen with normal FCαAT defines fecal protein maldigestion; high values of both variables indicate a protein-loss enteropathy [PLE (10,13)]. Fecal pH, reductase, glucose, and saccharose (Kelly method) were used to evaluate sugar absorption.

Breath hydrogen test
About 1 week after the measurements of intestinal absorption and digestion, a BHT was administered to check for BBOS. Before the BHT, the patients were asked
to observe dietary restrictions. Minimal intake of fiber and of poorly absorbed short-chain carbohydrates for 24 hours was advised, and the patients fasted overnight. The patients then consumed 15 g lactulose in solution with 100 mL water. The BHT samples were collected at baseline and every 15 minutes for at least 2 hours. An early rise in breath hydrogen after lactulose (ERBHAL) is expected approximately 90 minutes after consumption of the solution. The ERBHAL leads to one of two conclusions: either bacterial fermentation is occurring in the small intestine because of bacterial overgrowth, or rapid transit of the small intestine is occurring (15–17). An ERBHAL is defined as a rise in breath hydrogen of 10 points or more above baseline in two consecutive 15-minute samples after the ingestion of lactulose and before 90 minutes has elapsed since ingestion.

Inflammatory markers
The inflammatory marker C-reactive protein (CRP) was determined by the immunonephelometric method (ELISA, Vectastin: Vector Laboratories, Burlingame, CA, U.S.A.; normal value: 0 – 5 mg/dL); TNFα was measured by ELISA (Easial: Medgenix Diagnostics SA, Fleurus, Belgium; normal values: 3 – 20 pg/mL); and interleukin 6 (IL-6) was measured by ELISA (Easial; normal values: 0 – 16 pg/mL).

Re-evaluation
All determinations of markers of nutrition, inflammation, and BBOS were repeated at between 30 and 45 days, after adequate antibiotic treatment. We also recorded GI and general symptoms in the patients.

Statistical analyses
Statistical analyses were performed using the Mann–Witney U-test or the Wilcoxon test for non-paired data, and regression analysis. A probability of 95% was considered statistically significant. Results are given as mean ± standard deviation.

Results
The screening for malabsorption, maldigestion, and bowel permeability, and the breath test for BBOS and *Helicobacter pylori* were administered to all patients in our PD unit (*n* = 49). A positive ERBHAL was noted in 10 patients (7 women, 4 men), ranging in age from 43 to 78 years (mean: 59 ± 17.2 years). Of the 10 patients, 3 were on continuous ambulatory PD, and 7, on automated PD.

**Parameters of dialysis adequacy**
The mean weekly urea Kt/V in the 10 patients was 2.19 ± 0.4, the nPNA was 1.06 ± 0.22 g/kg daily, and the renal creatinine clearance was 2 ± 2.3 mL/min.

**General analytical data**
Mean laboratory values included hemoglobin 12 ± 1.6 g/dL, triglycerides 176.2 ± 94 mg/dL, ferritin 258 ± 198 ng/mL, blood urea 138 ± 42 mg/dL, creatinine 9.1 ± 3 mg/dL, potassium 4.6 ± 0.56 mEq/L, calcium 9.8 ± 0.88 mg/dL, and phosphorus 5.6 ± 2 mg/dL. Table I shows the changes in markers of nutrition before and after antibiotic therapy.

**Protein digestion and absorption**
Of the 10 patients with BBOS, 4 showed elevated fecal nitrogen. Notably, the patients with higher fecal nitrogen also had higher fecal fat as shown by a positive linear correlation (*r* = 0.6, *p* < 0.05). High fecal nitrogen also showed a significant negative linear correlation with serum albumin (*r* = 0.48, *p* < 0.05). We found no relationship between abnormally elevated fecal nitrogen and blood urea. After specific treatment for BBOS, fecal nitrogen normalized in 2 patients (Table I), but persisted high in 2 others (24-hour stool).

**Pancreatic exocrine function**
In 6 patients, values of fecal chymotrypsin were lower than normal, including in 3 with diabetes. These patients showed a statistically nonsignificant (NS) negative linear correlation for fecal chymotrypsin with fecal fat (*r* = −0.4, *p* = 0.09) and with fecal nitrogen (*r* = −0.37, *p* = 0.1, NS). Fecal chymotrypsin and serum albumin showed a significant positive linear correlation (*r* = 0.45, *p* < 0.05). No relation was found for fecal chymotrypsin with venous pH, parathyroid hormone, calcium, or phosphate levels. After BBOS treatment, no patient had an elevated fecal chymotrypsin (Table I).

**Intestinal protein losses**
An elevated FCαAT (>12 mL/24-h stool) was present in 5 patients. All also had high fecal nitrogen, denoting the presence of PLE. This condition was associated with poor nutrition status, characterized by lower serum albumin (3.54 ± 0.6 g/dL vs. 3.99 ± 0.36 g/dL in the remaining patients, *p* < 0.05) and transferrin (246 ± 71 mg/dL vs. 274 ± 44 mg/dL in the remaining patients, *p* < 0.05). On the other hand, we observed a positive linear correlation for FCαAT with fecal fat (*r* = 0.48,
p < 0.05) and statistically nonsignificant tendencies to correlation with fecal starch \((r = 0.40, p < 0.07, \text{NS})\) and fecal nitrogen \((r = 0.41, p < 0.07, \text{NS})\). The antibiotic treatment induced a decrease in FCαAT in 4 patients, but values did not return to normal (<12 mL/24-h stool).

### Intestinal sugars

One patient had a fecal pH below 5, and 7 patients (2 with diabetes) showed positive fecal starch. These patients showed a negative statistically nonsignificant linear correlation for fecal starch with albumin and prealbumin \((r = -0.35\) and \(r = -0.39\) respectively, both NS). Fecal starch declined in 4 patients, but none reached a normal level.

### Fecal fat losses

Fecal fat loss was elevated in 5 patients (>6 g/24-h stool) and was negatively related with parameters of nutrition. As compared with the remaining patients, patients with fecal fat losses had lower prealbumin \((27 \pm 8.8 \text{ mg/dL vs. } 36.1 \pm 7.9 \text{ mg/dL}, p < 0.05)\) and fibronectin \((36.6 \pm 7.7 \text{ ng/mL vs. } 45.1 \pm 6.4 \text{ ng/mL}, p < 0.05)\).

The BHT for BBOS was negative in all patients at re-test.

### Inflammatory markers

Table I shows an important decrease in CRP, IL-6, and TNFα from before to after antibiotic treatment for BBOS.

### Gastrointestinal symptoms

In some patients, GI symptoms improved after BBOS treatment. Improvements occurred mainly in patients who had had abdominal pain, discomfort, and intermittent diarrhea. All of the patients with anorexia experienced important improvements in food intake as measured by daily nPCR \((0.98 \pm 0.2 \text{ g/kg before vs. } 1.12 \pm 0.3 \text{ g/kg after})\).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal range</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily nPNA (g/kg)</td>
<td>≥1.1</td>
<td>1.09±0.2</td>
<td>1.12±0.19</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>3.8–5</td>
<td>3.45±0.32</td>
<td>3.81±0.22</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Prealbumin (mg/dL)</td>
<td>≥30</td>
<td>28.8±8</td>
<td>34±7.7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Retinol binding protein (mg/dL)</td>
<td>3–6</td>
<td>11.4±5.6</td>
<td>12.1±6.1</td>
<td>NS</td>
</tr>
<tr>
<td>Fibronectin (mg/dL)</td>
<td>25–40</td>
<td>38±14.6</td>
<td>37.2±12.3</td>
<td>NS</td>
</tr>
<tr>
<td>Serum α₁-AT (mg/dL)</td>
<td>150–350</td>
<td>254.5±76</td>
<td>248±65</td>
<td>NS</td>
</tr>
<tr>
<td>Transferrin (mg/dL)</td>
<td>≥200</td>
<td>234.8±54.7</td>
<td>249±61.8</td>
<td>0.08</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>&gt;100</td>
<td>212.8±54</td>
<td>225.3±99</td>
<td>NS</td>
</tr>
<tr>
<td>Serum iron (μg/dL)</td>
<td>50–145</td>
<td>72.3±38.5</td>
<td>78.6±24</td>
<td>NS</td>
</tr>
<tr>
<td>Vitamin B₁₂ (pg/mL)</td>
<td>150–750</td>
<td>881±238</td>
<td>798±291</td>
<td>NS</td>
</tr>
<tr>
<td>Folic acid (ng/mL)</td>
<td>2–10</td>
<td>8.2±3.4</td>
<td>7.8±2.9</td>
<td>NS</td>
</tr>
<tr>
<td>Venous pH</td>
<td>7.35–7.45</td>
<td>7.36±0.05</td>
<td>7.38±0.12</td>
<td>NS</td>
</tr>
<tr>
<td>C-Reactive protein (mg/dL)</td>
<td>0–5</td>
<td>6.77±2.2</td>
<td>5.44±3</td>
<td>NS</td>
</tr>
<tr>
<td>Interleukin 6 (pg/mL)</td>
<td>0–16</td>
<td>21.4±16</td>
<td>12.7±8.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TNFα (pg/mL)</td>
<td>3–20</td>
<td>67.4±14</td>
<td>48±17.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fecal pH</td>
<td>6–8</td>
<td>6.3±0.81</td>
<td>6.9±0.9</td>
<td>0.07</td>
</tr>
<tr>
<td>Fecal chymotrypsin (U/g)</td>
<td>≥23</td>
<td>12±9.8</td>
<td>14±10.1</td>
<td>NS</td>
</tr>
<tr>
<td>Fecal sugar (g/24 h)</td>
<td>0</td>
<td>2.99±2b</td>
<td>2.5±1.8</td>
<td>NS</td>
</tr>
<tr>
<td>Fecal fat (g/24 h)</td>
<td>0–6</td>
<td>6.3±2.8</td>
<td>5.1±3.2</td>
<td>0.09</td>
</tr>
<tr>
<td>Fecal N (g/24 h)</td>
<td>0–2</td>
<td>2.1±1.12</td>
<td>1.8±1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Fecal starch (g/24 h)</td>
<td>0–2</td>
<td>1.7±2.8</td>
<td>1±0.55</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Fecal water (%)</td>
<td>65–85</td>
<td>73.1±11.9</td>
<td>78±14.3</td>
<td>NS</td>
</tr>
<tr>
<td>α₁-AT clearance (mg/24 h)</td>
<td>&lt;12</td>
<td>8.3±6.7</td>
<td>7.8±6.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

a Values accepted as normal in PD patients.
b Different from normal range.
NS = nonsignificant; α₁-AT = α₁-antitrypsin; TNFα = tumor necrosis factor α.
Appetite evaluation
Antibiotic treatment for BBOS led to a decrease in inflammatory markers and to improvements in markers of nutrition and in eating motivation (Table II).

Discussion
The most important finding of present study is the connection between BBOS, proinflammatory cytokines overproduced and retained because of a lack of renal function, and the deleterious systemic effect of those cytokines in PD patients. Another important way in which BBOS may contribute to malnutrition is through intestinal malabsorption and maldigestion induced by bacteria overgrowth in the GI tract (8). The small intestine is the usual site of the excessive bacterial growth in BBOS; the disorder is less frequent in the colon, which is normally rich in bacteria [the bacterial concentration in the small bowel is usually less than \(10^4\) organisms per milliliter (15)].

The diagnostic BHT relies on bacterial metabolism (converting carbohydrates to hydrogen), by detecting byproducts from the digestion of carbohydrates that are not usually metabolized. In the BHT, patients are given a carbohydrate load (usually in the form of rice or lactulose). A diagnosis of BBOS is frequently made by using a combination of the BHT and a d-xylose test to measure GI malabsorption. In the d-xylose test, the patient drinks a quantity of d-xylose and then d-xylose levels are measured in urine and blood; if there is no evidence of d-xylose in the urine and blood, malabsorption in the small bowel is suspected (16). Although the combined use of the BHT and the d-xylose test improves sensitivity for a BBOS diagnosis, the main problem in adding the d-xylose test in dialysis patients is that most of those patients are anuric. The BHT alone is therefore widely used in clinical practice (17). The “gold standard” test for diagnosing BBOS is finding an excess of bacteria (>\(10^5\)/mL) in aspirate from the jejunum (15).

Patients with BBOS typically develop symptoms including nausea, bloating, vomiting, and diarrhea—all resulting from malabsorptive and inflammatory mechanisms. Surprisingly, all of the study patients had at least one GI malabsorption or maldigestion disorder. Antibiotic treatment corrected those disorders in only a few cases, indicating that BBOS was not the sole cause of those symptoms.

Previously, we demonstrated that in PD patients, severe GI abnormalities of both digestion and absorption are present. The contributions of those GI disorders to malnutrition have not previously been reported (10). In recent years, a great many anatomic (gastritis, esophagitis, peptic ulcer, intestinal ulceration, pancreatitis) and functional (poor motility, reduced enzymatic activity, intestinal permeability, low dipeptidase and disaccharidase, bile composition) abnormalities of the GI tract have been associated with uremia (2,7).

The contribution of silent or obvious infections to malnutrition in PD patients has been recognized. Infections of the GI tract can cause anorexia and systemic cachexia by a double pathway: lack of GI integrity, and overproduction of proinflammatory cytokines (18). Our patients showed a great improvement

<table>
<thead>
<tr>
<th>Visual analog scale questions</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>(p) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desire to eat before lunch</td>
<td>66±7.6</td>
<td>75.8±7.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Desire to eat after lunch</td>
<td>12.4±4.1</td>
<td>22.6±7.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hunger before lunch</td>
<td>58±7.4</td>
<td>72.7±8.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hunger after lunch</td>
<td>10.2±2.4</td>
<td>24.4±4.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fullness before lunch</td>
<td>19.8±10.5</td>
<td>20.5±12.3</td>
<td>NS</td>
</tr>
<tr>
<td>Fullness after lunch</td>
<td>78.8±15.6</td>
<td>69.3±19.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Prospective consumption before lunch</td>
<td>61±13.9</td>
<td>68.6±8.7</td>
<td>NS</td>
</tr>
<tr>
<td>Prospective consumption after lunch</td>
<td>12±4.8</td>
<td>18±5.1</td>
<td>0.08</td>
</tr>
<tr>
<td>Palatability</td>
<td>64±13.1</td>
<td>78.4±16.5</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

\(^{a}\) Scored from 0 to 100.

NS = nonsignificant.
in eating motivation (by the VAS) and in markers of nutrition (Table II) after treatment.

Three categories of factors that predispose to BBOS have been proposed: disordered small-bowel motility (stasis), disorders of the local immune system, and conditions that cause penetration into small bowel of bacteria from the colon (15). The dialysis population can be considered at high risk for BBOS (6,19). Disorders of the three predisposing mechanisms have frequently been reported in uremia. A decrease in intestinal motility, leading to increased bacterial concentrations, with subsequent overgrowth, is one of the most important causes. In fact, constipation (sometimes associated with phosphate binder use) is commonly mentioned by dialysis patients (2). The resulting decrease in GI motility may be associated with disordered levels and release of GI peptides (inhibiting rather than stimulating) (6). This disorder is generally conditioned by drug intake, retention of renal peptides, insulin resistance in diabetic and nondiabetic patients with uremia, high plasma levels of TNFα, and disorders of the peripheral nervous system such as vagus axonal degeneration and neuropathies (20). Gastric emptying can be inhibited by TNFα, possibly inducing macronutrient GI malabsorption and central anorexia through the hypothalamus (18,21). We found high plasma levels of TNFα that declined after BBOS treatment in all patients whose nutrition status improved and food intake increased.

The second mechanism predisposing to BBOS is disordered local and systemic immunity. Lower and dysfunctional immunoglobulin A in disorders of the GI epithelium, dysfunction in T cells and lymphocytes, and iron excess predispose to infections by both gram-negative and gram-positive bacteria (7,19). Conditions that cause bacterial proliferation into the small bowel from the colon include anatomic small-bowel problems such as outpouchings (diverticula) that can cause bacterial accumulation (22). In fact, 4 patients in our series had diverticulosis.

Other risk factors for BBOS include chronic pancreatitis, immunosuppressant drugs, and GI surgery. Pancreatitis induces BBOS through a decrease in bile quality, limiting the body’s capacity to digest nutrients. Diabetes mellitus (typically the first or second cause end-stage renal disease leading to dialysis), with its high frequency of pancreatic exocrine insufficiency [up to 50% (10)], can create similar problems. In a large group of PD patients (with and without diabetes), we observed lower fecal chymotrypsin (10). Significant numbers of patients on dialysis also take immunosuppressant drugs either temporarily or over the long term. And importantly, uremic patients frequently take proton pump inhibitors, anti-H2 agents, and antacids such as phosphate binders that may decrease the acid in the stomach, decreasing the antibacterial defense (23). Notably, we found a strong tendency toward lower fecal pH in the study patients (Table I).

Conclusions

The infectious process called BBOS is frequently found in PD patients and is associated with GI malabsorption and maldigestion, malnutrition, and systemic inflammation. Antibiotic treatment for BBOS was associated with improvement in GI disorders, anorexia, and deleterious systemic effects.

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