

LETTERS TO THE EDITOR

Diet and Dyspepsia: To Believe Patients or Studies?

TO THE EDITOR: We read with interest the article by Saito *et al.* in the December issue of this journal (1) and appreciate the study on the possible role of dietary nutrients and specific food items in functional gastrointestinal disorders (FGD). We would like to highlight certain major pitfalls of this study.

The symptoms in functional dyspepsia (FD) become worse after a meal and from the patient's perspective, food intolerance is a major offender. As clinical and experimental data show worsening of symptoms with fat ingestion in FD (2), it is strange to note that the cases in this study consumed more saturated fat than controls. It would have been appropriate to compare the consumption of various dietary items among the cases with and without symptoms and among the controls. The authors made no attempt to find out whether their patients reported more symptoms after ingestion of certain commonly implicated food items. They hypothesized that the symptomatic group may have reduced the consumption of offending food substances, which might have affected their observations, but they have not verified this.

In this study, both FD and irritable bowel syndrome (IBS) were grouped together and no attempt was made to differentiate diarrhea- and constipation-predominant IBS. The data from 20% of the patients who had a combined FD and IBS and 7% of patients probably with no FGD at the time of evaluation are included. Most of the earlier studies on the relationship between dietary factors and symptoms of FGD were on patient groups of 50 or less and the present study is also fraught with the same problem as the number of patients in the subgroup of IBS was 45 and 27 in the FD group. A study with a larger number of a more uniform sample is required to assess the role of dietary factors in FGD. So, contrary to the claim of the authors, it is highly likely that the selected group of a small number of patients with different types of diseases in their study may not be a true representation of the larger population with FGD.

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Response to Drs. Thomas and Kareem

TO THE EDITOR: We thank Drs. Thomas and Kareem for their interest in our article (1). They appropriately note that food intolerance has been linked to functional gastrointestinal disorders (FGIDs) such as functional dyspepsia (FD) and irritable bowel syndrome (IBS). Indeed, our population-based study observed that 17% of our cases with FD and/or IBS reported food allergy or intolerance, compared to only 9% of the controls. By definition, all cases were reporting symptoms and thus a comparison of diet between symptomatic and asymptomatic cases with controls could not be performed. Because the overlap of IBS and FD has been reported before (2, 3), our intent was to perform a population-based study of FGIDs; our study was not adequately powered to look at the connection between diet and specific disorders (*i.e.*, FD, IBS, or IBS subtypes). Nonetheless, the dietary and bowel symptom data were collected from 218 participants who were asked to participate based on an age- and gender-stratified random sampling approach utilizing the Rochester Epidemiology Project (REP) (4). We agree that the role of dietary components in specific FGIDs and subtypes still remains to be determined, and further study is certainly warranted.

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Transjugular Intrahepatic Portosystemic Shunt (TIPS), the Preferred Therapeutic Option for Budd Chiari Syndrome Associated with Portal Vein Thrombosis

TO THE EDITOR: We read with interest the article by Murad *et al.* about the pathogenesis and treatment of Budd

Author	Year	Patients	Cavernomas	Success	Complications	Follow-Up (months)	Reintervention
Radosevich	1993	10	1 (failed 1)	7/10 (70%)	No	12	1
Blum	1995	7	no	7/7 (100%)	No	8	0
Walsner <i>et al.</i> (5)	1998	14	2 (failed 2)	12/14 (85%)	No	10, 4	12
Ganger <i>et al.</i> (6)	1999	11	no	9/11 (81%)	2 (1 died)	13	"several"
Bilbao	2004	6	no	6/6 (100%)	No	15	ND
Jiang	2004	14	4 (failed 4)	10/14 (71%)	No	ND	ND
Senzolo <i>et al.</i> (4)	2006	28	9 (failed 3)	19/28 (73%)	1	21	7

ND = not defined.

Chiari syndrome (BCS) associated with portal vein thrombosis (PVT) with/without other splanchnic vein thrombosis (SVT) (1), which is reported in about 20% of BCS patients (2). The authors conclude that anticoagulation should be used in these patients whenever possible, because derivative shunt procedures appear almost impossible. However, one-third of the patients had apparent contraindications to anticoagulation. We have some comments about this conclusion. Firstly, anticoagulation should be used in all patients with BCS regardless of the presence of PVT/SVT, the type of previous specific shunt or stent therapy used, or the ability to diagnose a known thrombophilic disorder. The use of anticoagulation has been shown to ameliorate prognosis; in almost 50% of BCS a defined etiological factor is not identified even using sophisticated techniques (2). If high-risk esophageal varices are present they can be banded first to eradicate them and minimize the risk of bleeding. However, it is unlikely that anticoagulation alone will resolve hepatic and portal vein thrombosis when present together, especially if the PV thrombus is long standing and extensive (3) (*i.e.*, this was the case in more than 50% of the reported patients with PVT (1)). In addition, if anticoagulation is specifically contraindicated, then, in these cases and in general, TIPS should be the preferred therapeutic option, because it can treat both BCS and PVT. In experienced centers, TIPS for PVT is safe and has a success rate of about 70% (Table). We have successfully treated three patients with BCS associated with PV and SVT (1 also had added thrombosis of the previous surgical mesocaval shunt); two previously reported in our series of 28 patients with PVT (9 cavernomas and 18 with SVT) were treated with TIPS (4). The success of TIPS was 73% regardless of the extension of the thrombus or the presence of cavernomas and it was successful in all three BCS patients. There were no life-threatening complications. We believe that PVT on its own can be an indication for TIPS and in particular when present with BCS. Relief of portal hypertension and decongestion of the liver can be treated at the same time, and improve the poor prognosis of BCS associated with concomitant PVT (2).

A summary of the reported series of TIPS for PVT is reported in the table below.

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Response to Dr. Senzolo et al.

TO THE EDITOR: We would like to respond to the letter by Senzolo *et al.*, in which our recently published article "Pathogenesis and treatment of Budd-Chiari syndrome combined with portal vein thrombosis" (1) is discussed. Senzolo *et al.* advocate the use of TIPS for PVT, based on the results of a study they have recently performed in 28 patients with various forms of PVT, among which two exhibited combined BCS-PVT (2). In their view, the successful results of the TIPS placement in these two patients oppose the results of our study. We believe, however, that results of both the studies are very much consistent. In our study, we have treated four

BCS-PVT patients with TIPS, with a success rate of 75% after 4–43 months of follow-up. Given the fact that thrombosis in both hepatic and portal veins clearly jeopardizes successful catheterization, these results were interpreted by us as remarkable. Thus, in our article, TIPS was clearly regarded as a promising therapeutic option, provided adjunctive measures are employed to overcome technical problems.

The second point brought up by the authors concerns the use of anticoagulation. They are of the opinion that anticoagulation should be used in all patients with BCS, regardless of the severity of thrombosis, therapeutic management, or underlying disease. Furthermore, varices should accordingly be treated by early endoscopic treatment and should therefore never be a contraindication. In our study with a follow-up from 1984 to 2001, 61% received anticoagulant therapy. As previously described, the use of anticoagulation for BCS has, in particular, been recognized as a cornerstone for treating patients with BCS during the last decade (3, 4). Previously, risk of bleeding in the presence of portal hypertension was thought to outweigh the risk-benefit ratio in favor of withholding. In our long-term study, the main reason for withholding anticoagulation was indeed a well-documented history of or presentation with variceal hemorrhage, or spontaneous elevated INR. In fact, the authors used the same contraindications in their study on TIPS in PVT and therefore administered anticoagulation to only two patients (7%). However, we agree with the author's general opinion to use anticoagulant therapy in all patients with BCS whenever possible. The reasons hereof we have already mentioned in our paper: (1) if given early, there is a considerable possibility for recanalization (3, 4), and (2) since extension of thrombosis is related to increased mortality, anticoagulation may play an important role in preventing progression of thrombosis.

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Crohn's Disease in Common Variable Immunodeficiency: Treatment with Antitumor Necrosis Factor Alpha

TO THE EDITOR: Common variable immunodeficiency (CVID) is one of the most prevalent primary immunodeficiency disorders, and is characterized by low levels of most or all of the immunoglobulin classes (hypogammaglobulinemia), a lack of B lymphocytes, and some T-cell defects. Its clinical manifestations include recurrent infections and an increased incidence of malignant and autoimmune disease (1, 2). Immunodeficiency and autoimmune phenomena may occur concomitantly. In Spain, the prevalence of inflammatory bowel disease in CVID in a retrospective series was 3.2% (3). A persistent activation of the tumor necrosis factor system has been reported in a subgroup of patients with CVID, and this may contribute to the pathogenesis of the disease (4). Clinically, tumor necrosis factor alpha (TNF- α) inhibitors such as infliximab have demonstrated a remarkable efficacy in a wide range of autoimmune and inflammatory disorders. However, these benefits are accompanied by an increased susceptibility to intracellular bacterial infections, reactivation of chronic viral infections, and mycosis (5). There are no data regarding the efficacy and safety of anti-TNF- α in patients with CVID and inflammatory bowel disease. We report on two patients with CVID and concomitant Crohn's disease (CD) who have been treated with infliximab.

The first patient is a 20-yr-old man with CVID who had been diagnosed 4 yr earlier with CD, which proved to be refractory to conventional therapies. The patient underwent a colonic resection with ileorectal anastomosis when he suffered a severe flare-up, but did not respond to full doses of corticosteroids. One year after resection, the disease recurred at neoleum and a severe perianal disease with large ulcers in the anus was detected. Systematic screening was performed to detect local or systemic infections (tuberculosis and viruses included) and the results were negative. The patient had slightly decreased IgM, IgG, and IgA immunoglobulin levels and periodically received intravenous IgG replacement treatment. The perianal disease progressed and treatment with antibiotics (metronidazole and ciprofloxacin) did not produce a positive response. Infliximab treatment was initiated, following the usual scheduled program (at 0, 2, and 6 wk and every 8 wk for 1 yr), and azathioprine was concomitantly prescribed. Remission was achieved and 1 year after the last infliximab infusion the patient remained asymptomatic and without perianal lesions. No concomitant infections were detected during or before infliximab treatment and the patient requires less intravenous IgG replacement than he did the previous year.

The second patient is a 24-yr-old man with CVID who was diagnosed 1 year ago with ileocolonic CD. After the first flare-up he developed a moderately refractory CD with active disease, and so the treatment with azathioprine was started. The

disease continued to be endoscopically and clinically active despite immunosuppressive therapy at the appropriate doses. Infections were excluded through systematic analysis (culture of stools and investigation of viruses via colonic biopsies), tuberculosis screening was negative, and infliximab treatment was started (at 0, 2, and 6 wk and every 8 wk for 1 yr) while azathioprine therapy was maintained. Though the initial response was positive, the patient subsequently declined and we continued to administer 10 mg/kg infliximab every 4–6 wk. An ileocolonic resection was necessary because of persistent activity. Levels of immunoglobulin and periodicity of infusions of intravenous IgG during infliximab treatment were maintained as previously. No more concomitant infections than usually were detected.

To our knowledge, these are the first cases of patients suffering both CVID and CD, and who have received infliximab treatment. A case of CVID with juvenile rheumatoid arthritis was previously reported to have been treated effectively with etanercept, which moderated the disease (6). This case provides evidence that infliximab could be a safe treatment option in such clinical circumstances. Nevertheless, the risk of developing infection is considerable and so caution is paramount. Specific inhibition of TNF- α could improve CVID, as a subgroup of patients with CVID were reported to present an increased production of TNF- α , which suggests that this cytokine plays a part in the immune deregulation of this disease.

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Baclofen Facilitates PEG Placement in Paraplegia-In-Flexion Syndrome

TO THE EDITOR: Percutaneous endoscopic gastrostomy (PEG) has revolutionized the nutritional management of patients unable to eat and swallow normally because of severe neurologic impairment or other medical conditions (1). PEG is less invasive, less costly, and safer than the alternative of surgical gastrostomy (2). A simple, new treatment to reverse an absolute technical barrier to PEG that could render PEG available to thousands of new patients per year in the United States is therefore potentially clinically important. Patients with spastic contractures of the arms, knees, and hips (spastic contraction-fetal position syndrome, or paraplegia-in-flexion syndrome, or fixed flexor posture syndrome(3)) generally require PEG because of neuromuscular dysphagia from profound neurologic incapacity. This syndrome can, however, block percutaneous access to the abdominal wall (folded legs covering the abdominal field) and until now, when severe, has been an absolute and insurmountable technical barrier to performing PEG. A novel, simple, and noninvasive treatment is presently reported which permitted PEG in this situation.

Among 300 referrals for PEG during 16 yr in a hospital-based gastroenterologic practice of Dr. Cappell, three otherwise appropriate candidates for PEG (1%) could not undergo PEG because of poor abdominal wall access because of this syndrome (distance from folded legs to abdominal wall ranged from 20 to 24 cm). In the first two cases the contracted legs could not be withdrawn from over the abdomen even after intravenous administration of diazepam and meperidine. The third case is presently reported.

A bed-bound 74-yr-old woman was referred for PEG because of inability to eat orally secondary to neuromuscular dysphagia associated with chronic, end-stage Parkinsonism and profound multiinfarct dementia. Physical examination revealed an emaciated patient weighing only 42 kg, who was incontinent of urine and feces, nonverbal, and minimally responsive to verbal stimuli. There was severe temporal muscle wasting, intercostal muscle wasting, and muscle wasting in all four extremities. The abdomen was soft and nontender, with normoactive bowel sounds. The abdominal examination was, however, severely limited by the spastic contraction-fetal position syndrome (knees fixed in flexure <22 cm above abdominal wall).

For PEG, an EGD was first performed after intravenous administration of 50 mg of fentanyl and 3 mg of midazolam. EGD revealed no lesions and was uncomplicated. An ideal site for percutaneous transabdominal needle puncture for PEG was identified in the left upper quadrant by manual compression of the abdominal wall and endoscopic transillumination. The PEG was, however, canceled because percutaneous access to the site was prevented by this spastic syndrome, despite attempts by the endoscopic staff to manually pull the extremities away from the abdominal wall. A Dobhoff nasoenteral feeding tube was placed for short-term enteral feeding, but was unacceptable for long-term feeding because

of its attendant long-term risks. A surgeon was reluctant to perform surgical gastrostomy for long-term enteral feeding because of the restricted abdominal access.

By coincidence, 3 days after the failed PEG, treatment with baclofen 5 mg twice daily was initiated via the Dobhoff tube for the muscular spasticity. Five days later, the patient had near complete relief of this spastic syndrome. This serendipitously provided abdominal access for PEG. PEG was successfully completed after intravenously administering 50 mg of fentanyl and 3 mg of midazolam, with neither difficulty nor complications. No side effects of the baclofen therapy occurred. The patient was transferred 1 day postprocedure to a hospice using the PEG for feeding. The spastic syndrome gradually recurred after baclofen and other medications were discontinued as per the family's request. The PEG has remained functional without complications and the patient has gained weight during 1 month of follow-up.

Bilateral upper and lower extremity spasticity with this syndrome is an end-stage sequela of severe upper motor neuron injury from multiple sclerosis, spinal cord injury, or catastrophic brain injury (4, 5). Patients typically undergo PEG before this syndrome occurs, but are sometimes referred for PEG afterwards. The currently reported 1% rate provides an estimate of ($1\% \times 200,000$ PEGs per annum (6)) 2,000 technical failures to perform PEG because of this syndrome per annum in America.

Evidence that the currently reported novel treatment can reverse this syndrome to enable PEG includes a direct temporal relationship between baclofen initiation and syndromic relief, a direct temporal relationship between baclofen cessation and syndromic recurrence, and the known antispasmodic action of baclofen, a GABA (gamma aminobutyric acid) B receptor agonist that reduces muscle spasticity (7). For the small additional risks of oral baclofen therapy, surgical gastrostomy was replaced by PEG, a procedure with less morbidity. For the small additional cost of about \$5.00 for the 8 doses of 5 mg of baclofen (Mosby Drug Consult, on the MD Consult Web site, 2004), thousands of dollars were saved by substituting PEG for the more expensive surgical gastrostomy, and by earlier hospital discharge.

Baclofen is recommended to treat severe spasticity, including this syndrome, from causes that include spinal cord trauma and disease or brain trauma and other brain injury (4, 8). Baclofen reversed spastic rigidity from end-stage Parkinsonism and multiinfarct dementia in this case. The drug should be started 3 or more days before PEG for optimal syndromic relief. The usual initial dose is about 5 mg orally three times daily, with gradual titration until the desired effect is achieved up to a maximum total dose of 40–80 mg per day. The patient does not have to be monitored when administered oral therapy, but should be monitored for the first dose if administered intrathecally. When high-dose therapy is discontinued after PEG, the dose should be slowly tapered over several days to prevent seizures or hallucinations from abrupt drug withdrawal. Baclofen is relatively safe, particularly when administered short-term, orally, and

in low doses (8, 9). It can cause sedation or lethargy; can exacerbate psychiatric disorders, particularly schizophrenia; can exacerbate confusional states; and can rarely cause hypotonia, dizziness, or paresthesias. It is contraindicated in the rare patient with known drug hypersensitivity. It should be used cautiously in pregnant or nursing females as drug safety in the fetus or nursing infant is inadequately analyzed.

Despite severe neurologic compromise, patients with this syndrome often are not acutely ill, and often survive more than 1 month. Nasoenteral tubes are not recommended as an alternative to PEG in this situation because of their long-term risks, and because of rejection by nursing homes or hospices of patients fed by nasoenteral tubes (10). This novel application of baclofen to reverse this spastic syndrome could potentially benefit 2,000 patients per year in America currently denied the manifest nutritional benefits of PEG. Baclofen may also improve abdominal wall access for abdominal surgery, such as surgical gastrostomy or cholecystectomy in patients with this syndrome.

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A Psoas Abscess Caused by *Pseudomonas aeruginosa* Due to Diverticular Perforation

TO THE EDITOR: Psoas abscess is a condition traditionally associated with tuberculosis of the spine. In recent years it has been reported in gastrointestinal (GI) diseases such as appendicitis, malignancy, Crohn's disease, and less frequently, diverticulitis (1). Associated microorganisms include those of normal GI flora as well as *Staphylococcus aureus*. Regardless of the etiology, clinical symptoms usually develop over a period of several weeks (2). We report the acute clinical presentation of a *Pseudomonas aeruginosa* psoas abscess because of perforated sigmoid diverticulitis.

A 72-yr-old man with a history of chronic obstructive pulmonary disease (COPD), hypertension, diabetes mellitus, and mild chronic renal insufficiency presented to our emergency room with severe pain in the left hip. The pain steadily worsened over 2 days, did not radiate, and was exacerbated by movement. Patient had a distant history of appendicitis. He denied abdominal pain, change in bowel movements, nausea, vomiting, or fever. Physical examination was remarkable for left hip tenderness to palpation and severe distress with passive range of motion. There were no abdominal findings. A mild leukocytosis (13.4 k/mm^3) and elevated alkaline phosphatase (194 units) were present. Computed tomography (CT) scan of the abdomen demonstrated a $9 \times 6 \times 18 \text{ cm}$ abscess along the psoas muscle adjacent to the left sigmoid colon and into the left groin (Fig. 1). Gas and debris were present. Adjacent sigmoid colon showed an inflammatory reaction. These findings were interpreted as ruptured diverticulitis with retroperitoneal diverticular abscess. The patient was admitted to the hospital and treated with ampicillin-sulbactam, levofloxacin, and CT-guided iliopsoas abscess drainage. Fluid culture yielded *B-hemolytic Streptococcus* and *Pseudomonas aeruginosa*. Subsequently, the patient underwent exploratory laparotomy and diverting ileostomy.

Because of the acute clinical course and unusual organisms, this case reveals an infrequent presentation of psoas abscess. Although diverticulitis has been reported to cause abscess of the psoas muscle, it is an extremely rare finding. Moreover, in the few reported cases, symptoms developed over 2 wk to 3 months (1, 3). Interestingly, fluid culture grew *Pseudomonas aeruginosa*, which is not commonly associated with diverticulitis and is usually associated with immuno-

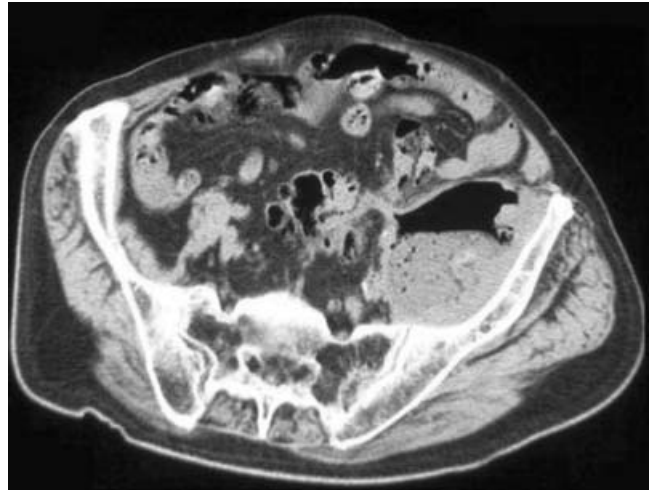


Figure 1. CT-Scan of the lower abdomen showing a Psoas abscess extending from the spine into the left groin, adjacent to reactive sigmoid colon (arrow).

suppressing states and manifests with diarrhea. This patient was not taking steroids or other immune-modulators. To our knowledge this is the first report of *Pseudomonas aeruginosa* psoas abscess because of perforated diverticulitis. Our case alerts physicians of potential acute presentation, changing etiology, and evolving microbiology of psoas muscle abscess.

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