Food is a Wonderful Thing — with Qualifications

The Division of Gastroenterology, Hepatology and Nutrition provides a focus on medical problems, such as nutritional deficiencies, the inability to eat, over- or under-eating, and diet/intervention consequences. The Division’s Nutrition Support team works to restore optimal nutrition status and health for patients in need.

In this issue, we highlight the clinical and research work of faculty and fellows interested in the broad topic of nutrition. John Scherer, MD, discusses a complex hepatobiliary case linked to diet. Julia B. Greer, MD, MPH, provides clear information on healthy eating for physicians to pass on to their patients. UPMC Digest editor Toby O. Graham, MD, provides an update on issues in clinical nutrition, and Stephen O’Keefe, MD, MSc, outlines his fascinating research on the effects of diet on colon cancer development. New faculty member, Joshua Novak, MD, highlights the use of percutaneous endoscopic jejunostomy tubes for those who cannot eat and may have a risk of aspiration or other complications.

Two more educational cases also are included. Elizabeth Blaney, MD, presents an interesting endoscopic unknown image, and Charles Gabbert, MD, presents a great case presentation of a woman with a complex neuroendocrine tumor.

We share the diversity and depth of our physician experts and fellows throughout all of our division’s educational endeavors, including UPMC Digest.

Bon Appétit,

David C. Whitcomb, MD, PhD
Giant Eagle Foundation Professor of Cancer Genetics
Professor of Medicine, Cell Biology & Physiology and Human Genetics
Chief, Division of Gastroenterology, Hepatology, and Nutrition
UPMC GI Research

UPMC is honored to be ranked number five in the nation for gastroenterology by U.S. News & World Report. While we are proud of this clinical recognition, we remain committed to educational and research objectives to continually enhance gastroenterology and hepatology advancements.

The faculty, fellows, and researchers at the University of Pittsburgh School of Medicine Division of Gastroenterology, Hepatology, and Nutrition were honored to provide numerous oral and poster presentations at Digestive Disease Week (DDW) 2012, which was held in San Diego this past spring. Pitt research focused on colon cancer screening, pancreaticobiliary disease, advancements in clinical hepatology, and new approaches to inflammatory bowel disease (IBD) pain management.

Robert Schoen, MD, MPH, professor of medicine and epidemiology, presented at the American Gastroenterological Association's Clinical Science Plenary Session to summarize the results of the colon portion of the Prostate, Lung, Colorectal & Ovarian Cancer (PLCO) Screening Trial (NIH/NCI). Stephen O'Keefe, MD, professor of medicine, also featured colon cancer prevention in his talk about how dietary modulation of intestinal microbiota could attenuate risk in African-Americans.

University of Pittsburgh pancreas research captured a large part of the spotlight at DDW 2012. An oral presentation by Randall Brand, MD, professor of medicine, highlighted advancements in pancreatic cancer risk stratification using a plasma SOMA panel to determine the need for EUS. SOMAmers are a unique class of DNA aptamers that bind their protein targets with high selectivity. Bridger Clarke, MD, a Year III GI fellow, presented two orals discussing the progression of recurrent acute pancreatitis (RAP) to chronic pancreatitis (CP) and the effectiveness of long-term endoscopic therapy on RAP.

Rawad Mounzer, MD, a Year I fellow, gave an oral presentation on the application of the revised Atlanta Classification for acute pancreatitis (AP) patients. Vijay P. Singh, MD, assistant professor of medicine, provided bench research advancements involving lethal lipotoxicity results from obese mice with cerulein-induced AP, as well as a reduction of pancreatitis severity by the presence of fibrosis in CP. Division Chief David Whitcomb, MD, PhD, provided two thoughtful presentations on the impact of early CP diagnosis on disease management and on advancements in predicting severe AP.

Oral presentations in liver and IBD were lauded. Swatha Ganesh, MD, instructor of medicine with the division’s Center for Liver Diseases, discussed the high hospital readmission rates of cirrhosis patients. Additionally, Matthew Coates, MD, PhD, a Year II fellow, was invited to discuss pain in quiescent ulcerative colitis and the potential involvement of irritable bowel syndrome (IBS) in its clinical course.

Division hepatologists will demonstrate their research productivity at the American Association for the Study of Liver Diseases (AASLD) meeting in Boston this November. Accepted abstracts include discussions about treatment prioritization for hepatitis C patients (Dr. Chhatwal), the spectrum of liver disease and inflammatory bowel disease (Dr. Dunn), pain and narcotic use in chronic liver disease (Dr. Rogal), and early treatment of depressive symptoms to improve post-transplant survival (Dr. Rogal).

More information about these University of Pittsburgh researchers and related studies may be found at www.dom.pitt.edu/gi.
Carbs Really Are the Enemy

By John A. Scherer, MD
Gastroenterology Fellow
Division of Gastroenterology, Hepatology, and Nutrition

One year before presentation to our clinic, a 75-year-old Caucasian female presented to another hospital with right upper quadrant pain and nausea. She had a history of hypertension and a prior cholecystectomy. She had elevated liver function tests (LFTs) (ALK 731, AST 121, ALT 212, TBili 2.5) and a dilated common bile duct (CBD) to 1.1cm on MRCP (Figure 1). An ERCP procedure found an ulcerated major papilla. She was diagnosed with papillary stenosis and was treated with a biliary sphincterotomy and placement of a plastic CBD stent.

Afterwards, the patient did well with complete normalization of LFTs. When she returned for stent removal one month later, an imbedded stent was found in the opposing duodenal wall. Subsequent stent removal resulted in free duodenal perforation, which was treated with endoscopic placement of two hemoclips. She developed pneumoperitoneum, pneumomediastinum, and subcutaneous emphysema, but recovered quickly and was discharged home in good condition.

The patient did well during the following year, until she developed frank steatorrhea two months prior to presenting at our hospital. She complained of 15 to 20 oily, loose bowel movements per day, nocturnal stools, and a 30-pound weight loss. Upon presentation at our hospital, she had elevated LFTs (ALK 719, AST 79, ALT 130, TBili 0.5), and stool C. difficile toxin was negative. Abdominal CT revealed dilated intra- and extra-hepatic bile ducts (CBD 1.3cm), a dilated main pancreatic duct (PD) of 6mm in the head of the pancreas, pancreatic parenchymal calcifications, pancreatic atrophy, and intact duodenal hemoclips (Figure 2). EUS confirmed these findings but did not locate evidence of cholecodolithiasis or a peri-ampullary/pancreatic mass to explain the “double duct sign” found on imaging. Subsequent ERCP was notable for an ulcerated major papilla consistent with recurrent papillary stenosis and was treated with biliary sphincterotomy and balloon dilation (Figure 3). Peri-ampullary biopsies were obtained and were notable for a high level of intraepithelial lymphocytes (>30/hpf). Follow-up labs demonstrated markedly positive celiac serologies (tTG IgA, gliadin IgA, and gliadin IgG, all 3 to 5 times beyond the upper limit of normal) and severely abnormal fecal elastase (7, normal >200). Genetic mutation analysis (CFTR, SPINK1, PRSS1) was normal. Of note, the patient denied alcohol or tobacco abuse, and her family history was unremarkable. She was started on a gluten-free diet (GFD). The steatorrhea resolved with pancreatic enzyme supplementation.

Celiac disease is associated with unexplained mild elevation in serum lipase and amylase, exocrine insufficiency, recurrent acute pancreatitis, and chronic pancreatitis. Improvement is seen with adherence to a GFD. A large Swedish population-based study retrospectively analyzed more than 14,000 patients with celiac disease and found a significantly increased risk of developing both recurrent acute pancreatitis (HR 3.3) and chronic pancreatitis (HR 19.8). Possible mechanisms to explain this association include papillary stenosis, autoimmunity, and malnutrition, with papillary stenosis providing the strongest supporting evidence.

Patel et al. screened 169 consecutive patients with probable pancreatic-type sphincter of Oddi dysfunction type 1, and 7.3% had celiac disease.

Continued on Page 9.
What to Eat

by Julia B. Greer, MD, MPH

“What should I eat?” It’s a question that your patients, as well as friends and family members, ask you all the time. We are bombarded with mixed messages about nutrition from the media and product manufacturers, as well as conflicting research findings and pseudo-science regarding particular foods or nutrients. Adding to the confusion, there are more than 17,000 new food products on U.S. shelves each year, and you can imagine that these aren’t new types of apples.

With more than two-thirds of all adults overweight or obese, the first factor in achieving good health is appropriate adiposity. Maintaining a healthy weight in adulthood or losing just five to ten percent of excess body weight protects against heart disease, diabetes, and numerous types of cancer. So if weight isn’t ideal, aim to get BMI into a healthy range.

Focus on whole foods and foods that have undergone minimal processing. If a product’s ingredient list is longer than ten items, I typically avoid it. You do not need to munch on dandelion greens or become vegan to attain ultimate health. Rather, nutritious fruits, including citrus, apples, dark-colored grapes and berries, and green leafy vegetables, along with cruciferous vegetables such as broccoli, cauliflower, and Brussels sprouts should be the focus of the meal. Also included should be legumes such as beans and peas, soy foods, low-fat dairy products, and whole grains. Smaller portions of meat, poultry, fish, and seafood should round things out. There is no law stating that you need to eat meat every day. Recent studies show a significant link between red meat and increased mortality from cancer and heart disease. We all should go meatless a few days per week.

Omega-3 fatty acids are good for heart health due to their anti-inflammatory effects. North Americans have an imbalanced omega-6 to omega-3 ratio. The best ratio is about 3:1, and the average U.S. ratio is 25:1. Higher quantities of omega-3s are found in fatty fish such as salmon, tuna, mackerel, sardines, and herring, walnuts, nut oils, and flaxseed oil, or ground flaxseed meal. However, deep-water fish contain mercury, so don’t make this your only food source. Many brands of eggs and organic milk are enriched with DHA, an omega-3 fatty acid.

Finally, cut down on fat and salt when preparing food. Garlic, onions, shallots, leeks, and chives are rich in antioxidants and are wonderful seasonings. Add cancer-fighting spices, such as basil, rosemary, turmeric, and fenugreek, for flavor. If you’re looking for healthy recipes, you know where to find me.

Dr. Greer is an assistant professor of medicine with the University of Pittsburgh Division of Gastroenterology, Hepatology, and Nutrition and is interested in GI cancers, genetic epidemiology, dietary risk factors, and nutrition. She has authored The Anti-Cancer Cookbook, an acclaimed book with national sales distribution. A second cookbook on recipes for breast cancer prevention is in publication.
Physicians are often confronted with patients who are unable to consume adequate calories by mouth to maintain their weight or regain body mass losses. Establishment of enteral access to the gastric lumen is not an option in conditions such as gastroparesis and anatomical barriers, including gastric outlet obstruction, or due to the complications of prior gastrointestinal surgery. These situations present difficult dilemmas for gastroenterologists.

Traditionally, such patients would require placement of a nasoenteric feeding tube, percutaneous endoscopic gastrostomy tube with jejunal extension (PEG/J), surgical jejunostomy tube, or initiation of total parenteral nutrition (TPN). Although placement of a nasoenteric feeding tube is safe, it is limited by the availability of an experienced endoscopist and by patient tolerance. Additionally, these tubes do not have long-term durability, which can necessitate replacement. Placement of a PEG/J can be complicated by proximal migration of the jejunal extension back into the gastric lumen or clogging of the J-tube due to various reasons. Surgical jejunostomy tubes carry a higher morbidity rate of 6% to 25% as well as inherit surgery risks.1 TPN delivers nutrition to a patient effectively, but carries multiple risks, including infection, metabolic complications, increased expense, and even cirrhosis with long-term use.

The direct endoscopic insertion of a percutaneous endoscopic jejunostomy (PEJ) tube was first described in 1987 by Shike, et al.2 This procedure is a modification of the percutaneous endoscopic gastrostomy (PEG) tube that is often used for enteral access to the GI tract. The placement of a PEJ provides direct long-term enteral access to the patient’s jejunum. This enteric tube has a wider lumen tube compared to surgically-placed or nasoenteric jejunostomy tubes, and is less likely to clog. In addition, the placement of this tube distal to the ligament of Treitz decreases a patient’s risk of aspiration.1 PEJ placement success has been reported to be as high as 84% in some studies.3 A primary PEJ benefit includes reliable delivery of enteral nutrition to the GI tract. PEJ tubes also can facilitate the discontinuation of TPN, decreasing its associated long-term patient risks.

References:


Dr. Novak is an assistant professor of medicine with the University of Pittsburgh Division of Gastroenterology, Hepatology, and Nutrition. His research and clinical service concentrate on nutrition support advancements.
Environmental Influences in Colon Cancer Risk

by Stephen J. D. O’Keefe MD, MSc, FRCP

Analysis of epidemiological study results on the prevalence of GI cancers by Doll and Peto in the 1950s concluded that more than 90% were caused by environmental differences, with diet deemed to be the most powerful influence. Since that time, studies have indicated red meat and animal fat as the chief dietary risk factors, but also have revealed fiber, fruits, vegetables, fish oils, and calcium as protective (Table 1).

Most food-related non-GI diseases, such as coronary artery disease, occur after digestion and absorption of nutrients into the body by the small intestine. Yet diet affects diseases of the colon differently, since food residues enter the colon in an indigestible form.

Through evolution, microbes capable of digesting dietary residues have colonized the colon, with negligible quantities of nutrients exiting with the feces, a system termed “salvage.” Microbes then convert the residues to absorbable short chain fatty acids (SCFAs) to form a perfect symbiotic association with the host. SCFAs are the preferred energy source for colonocytes, not glucose, and one of the SCFAs, butyrate, is the key regulator of mucosal proliferation. Therefore, adequate SCFA supplies are essential to mucosal health. Extensive studies have demonstrated that butyrate is anti-inflammatory, and it suppresses epithelial proliferation, while inducing apoptosis. This makes butyrate a powerful suppressor of cancer risk.

Composition of microbial balances can be upset if a diet rich in carbohydrate residues changes to one low in fiber and rich in red meat and fat. When butyrate producers are suppressed, sulfate-reducing bacteria can be stimulated by the high sulfur content of meat to produce hydrogen sulfide (H2S) gas, rather than the usual hydrogen and methane — and H2S is genotoxic. Furthermore, protein residues are fermented to ammonia, branched chain fatty acids, and p-cresol, all of which are inflammatory. Therefore, a condition of chronic inflammation is displayed within the colonic milieu interieur, with this inflammation increasing epithelial proliferation rates and cancer risk.

Until recently, researchers knew of approximately 100 different microbial species in the colon determined through cultured fecal samples. Recent development of non-culture techniques base their analyses on conserved regions of microbial DNA, such as in the 16S region. These findings indicate that colonic microbiota number more than 2,500, much higher than previously thought. Measurements reveal that the gut contains more microbial cells (x10) and DNA (x100) than the body itself. The emerging field of metabolomics has shown that these microbiota are highly active metabolically, producing hundreds of metabolites that are mostly health-promoting. Under conditions of dysbiosis, however, these metabolites can be inflammatory and disease-precipitating in the colon and even in distal organs following absorption. Studies have verified the critical role of microbiota in the development of the gut immune system and in intestinal function and health. For example, germ-free mice do not thrive, and die prematurely, from an acute colitis that progresses to cancer in Il-10 knockout models.

Our lab’s current NIH-supported research hypothesizes that the composition of the colonic milieu interieur, rather than the diet itself, determine colon cancer risk. We are examining differences in colonic contents between low- and high-risk colon cancer populations. In America, the population with the highest colon cancer burden is in African-Americans. Interestingly, native Africans rarely get this disease, so an ideal high-risk and low-risk population model is available.

Our studies suggest that the difference in risk is related to dietary differences, since African-Americans have a
westernized diet rich in meat and fat, while Africans consume a diet high in complex carbohydrates, principally maize, with relatively small intakes of meat and fat. Our initial pyrosequencing measurements of fecal microbiota compositional differences (Figure 1) show dramatic differences in most of the predominant microbial phyla, with a predominance of Bacteroidetes in African-Americans and a predominance of Prevotella in native Africans. Prevotella are among the most numerous microbes in the rumen and hind gut of cattle and sheep and are utilized to ferment complex carbohydrates. Additionally, Prevotella is a preventive agent for rumen acidosis, a bovine disease that disrupts foregut fermentation. These findings support our hypothesis that the microbiota in Africans may be better adapted to fermentation and the production of mucosal anti-inflammatory and anti-proliferative SCFAs. Importantly, colonic SCFA content is significantly higher in Africans.

Of course, the colonic research is considerably more complex than may be discussed in this short article. Our studies have highlighted differences in bile acid metabolism and vitamin synthesis by the microbes as well. Consumption of a high-fat diet increases hepatic bile acid synthesis and delivery of primary bile acids to the colon. This process increases activity of specific colonic bacteria that convert primary bile acids to secondary bile acids, and which are carcinogenic in experimental models. Additionally, high rates of fermentation are associated with increased microbial synthesis of both folate and biotin, which play essential roles in the regulation of DNA synthesis. Our next step is to delve deeper into microbial metabolite analyses by employing NMR spectroscopy. We aim to discover currently unrecognized metabolites which may co-regulate mucosal epithelial proliferation, and thus increase cancer risk.

References:


A 23-year-old female with no significant medical history was transferred to our hospital with a six-month history of progressive weight gain, fatigue, and generalized edema. Polydipsia and polyuria led to a diagnosis of diabetes. She had melena and required several red blood cell transfusions. She denied NSAIDs, aspirin, or steroid use. Family and social histories also were unremarkable.

On physical exam, she was tachycardic and mildly hypertensive. She was obese with a moon facies, acne, hirsutism, abnormal dorsocervical fat pad deposition (a.k.a. buffalo hump), prominent abdominal striae, and LE edema. Laboratories were remarkable for WBC 17K, hemoglobin 9.7, and glucose 623. EGD revealed numerous cratered ulcers throughout the duodenum (Figure 1).

Gastrin level was elevated at 1599 pg/ml. The urinary free cortisol level was >1000 mcg/d (normal 10-100) with an ACTH level of >200 pg/dl (9-46) and an AM cortisol level of 116 ug/dl (7-25). Brain MRI was negative for a pituitary adenoma, and an ectopic source of ACTH production was highly suspected. Subsequent abdominal MRI noted a 6.0x4.0cm heterogeneous hepatic mass, suspicious for a possible metastatic lesion. An EUS revealed a 1.3cm mass in the pancreatic body (Figure 2). Cytology was positive for neoplastic cells, as concomitant immunohistochemical staining was positive for both ACTH and gastrin. The patient was diagnosed with a metastatic ACTH- and gastrin-secreting pancreatic neuroendocrine tumor (PNET) causing both Cushing’s and Zollinger-Ellison syndromes. Her MEN1 genetic analysis also was positive for pathogenic mutation. Metopirone was prescribed for ACTH suppression and octreotide for her neuroendocrine tumor. Chemoembolization was performed, followed by radiofrequency ablation, to treat her liver lesion. Resection of the pancreatic lesion was delayed, however, due to interval development of a pulmonary embolism and respiratory insufficiency.

Intestinal neuroendocrine tumors (NETs) were first differentiated from other malignancies in 1907. Named carcinoid tumors due to their slow growth, they were considered to be “cancer-like” (karzinoide), rather than true cancers.

Carcinoid tumors comprise two thirds of gastroenteropancreatic neuroendocrine tumors (GEP-NETs) and arise from multiple organ systems, which may include small bowel, appendix, rectum, and stomach.

The remaining one-third of neuroendocrine tumors are of pancreatic origin (PNETs). PNETs are classified as either functional (50 to 60%) or non-functional (40% to 50%), depending on hormonal hypersecretion. Functional PNETs are named according to the hormone they secrete: insulinoma, gastrinoma, VIPoma, glucagonoma, somatostatinoma, or the rare CRH/ACTH-oma. In general, non-functional PNETs are usually larger and have higher rates of metastases at presentation. Once suspected, the best method for tumor localization of PNETs is endoscopic ultrasonography (EUS), with an overall sensitivity and accuracy of >90%.

In general, treatments are aimed at curing the disease and relieving symptoms based on the hormone hypersecreted. Somatostatin analogs (i.e. octreotide and lanreotide) control symptoms in roughly 75% of patients effectively. Cytotoxic chemotherapy is often the first-line therapy for poorly differentiated or rapidly progressive PNETs. Surgery should be considered even when hepatic metastasis is present, although treatment strategies remain controversial in MEN1 patients with small PNETs (<2cm) given their relative good prognosis with medical management and probable inability to cure surgically. Enucleation is recommended for isolated PNETs, tumors <2cm, and small gastrinomas.

The overall five-year survival rate for PNETs is estimated to be approximately 60% to 70%, but may be highly variable, depending on clinical stage (World Health Organization/WHO, European Neuroendocrine Tumor Society/ENETS, or American
Many of these subjects experienced acute recurrent pancreatitis, and a high concordance with papillary stenosis. These patients were treated with sphincterotomy and GFD, and 75% had complete resolution at two-year follow-up.

In this case, celiac disease with its chronic duodenal inflammation caused recurrent papillary stenosis and chronic pancreatitis.

References:


Dr. Scherer is an Assistant Professor of Medicine with the University of Pittsburgh Division of Gastroenterology, Hepatology and Nutrition. His research and clinical service concentrate on nutrition support advancements. Dr. Scherer graduated from the University of Pittsburgh School of Medicine and completed his fellowship in Gastroenterology at the University of Pittsburgh. Dr. Scherer is an Assistant Professor of Medicine with the University of Pittsburgh Division of Gastroenterology, Hepatology, and Nutrition. His research and clinical service concentrate on nutrition support advancements.
Parenteral Nutrition (PN) Product Shortages Impact Patient Safety

by Toby O. Graham, MD

I have been honored to serve as an editor for UPMC Digest for the past eight years. Upon my retirement from medicine, I have been asked to present one of the major challenges confronting the nutrition support subspecialty, parenteral nutrition (PN). Since 2010, product shortages have been an increasingly critical issue in PN. These shortages create daily obstacles for UPMC’s multidisciplinary Total Parenteral Nutrition Safety Team, which approves, orders, and monitors very ill patients requiring PN.

A PN order involves more than 40 components, including dextrose, amino acids (AA), lipid emulsions, electrolytes, vitamins, and trace elements. Over the past two years, all PN products, except hypertonic dextrose and water, have been in short supply. Product shortages and discontinuations, manufacturing issues, and diminished supplies of raw materials all clash with growing demand. The complexity of PN formulations predisposes patients to significant safety issues when products are unavailable. In spring 2011, commercial AAs were in short supply, and nine patients died from Serratia infections linked to contaminated AA products manufactured by an infusion pharmacy.

Recurrent catheter-related bloodstream infection (CRBSI) is a common and life-threatening complication of home PN. Ethanol lock therapy (ELT) has been a UPMC Pharmacy and Therapeutics Committee-approved protocol since 2010, and has proven safety and efficacy records for CRBSI prevention. ELT is inexpensive, bactericidal, and fungicidal, and can avoid concerns of bacterial resistance resulting from antibiotic treatment and antibiotic lock therapy. Sterile ethanol, used for maintenance of 70% ELT, was a PN product in short supply as recently as last year.

A recently published case series of six home PN patients with CRBSI histories documents the recurrence of CRBSI when ELT was withheld due to a national sterile ethanol shortage. Though anecdotal, UPMC’s PN safety team cared for one home PN patient with recurrent CRBSI who was treated successfully with ELT for eight months, until the national product shortage of sterile ethanol prompted ELT cessation. Only two weeks later, the patient was admitted with a gram-negative CRBSI.

In the face of PN product shortages, the UPMC safety team is working to find new ways to provide safe and efficacious PN. Initiatives include the use of premixed, commercially prepared PN, as well as engagement of skilled nutrition specialists to evaluate patients prospectively to assure PN appropriateness. Additionally, enteral nutrition should be used whenever feasible, and PN products should be conserved during product shortages following the guidelines established by the American Society for Parenteral and Enteral Nutrition (www.nutritioncare.org) and other government regulatory entities.

Dr. Graham served as an associate professor of medicine with the Division of Gastroenterology, Hepatology, and Nutrition, where she specialized in nutrition support and was an inaugural editor for this newsletter. Dr. Graham retired in June 2012.
Save the Date!

PSC Partners Seeking a Cure

NINTH ANNUAL CONFERENCE
APRIL 26 TO APRIL 28, 2013
PITTSBURGH, PENNSYLVANIA

PSC Partners Seeking a Cure’s ninth annual conference for PSCers and caregivers will take place in Pittsburgh, Pennsylvania, April 26-28, 2013 in association with UPMC.

Recognition goes out to Kapil Chopra, MD, for securing the permission for UPMC to be the host member along with the City of Pittsburgh. Ricky Safer and Joanne Grieme, will be the 2013 Conference Co-Chairs.

PSC Partners Seeking a Cure is a growing, unique, volunteer organization including laypeople, medical professionals, and researchers with a strong emphasis in finding better treatment and a cure for Primary Sclerosing Cholangitis.

If you would like to find out more about PSC, go to www.pscpartners.org or email contactus@pscpartners.org.

Registration will begin in January 2013.

Online CME Opportunities

Sepsis: Systemic inflammation gone wrong
Derek C. Angus, MD, MPH, discusses the strengths and weaknesses of current pathophysiological models in the management and treatment of systematic sepsis in the hospital setting.

Risk factors and Markers for Severe Acute Pancreatitis
Georgios Papachristou, MD, reviews important markers for the diagnosis of severe acute pancreatitis, including the risk factors.

Visit UPMCPhysicianResources.com/GI to view these courses.

Discovery Channel
Physician Podcasts

Personalized Care: A New Approach to Diagnosing & Treating Pancreatic Disease
David Whitcomb, MD, PhD, Giant Eagle Foundation professor of Cancer Genetics and division chief, and Adam Slivka, MD, PhD, professor of medicine and associate chief for Clinical Services, discuss their team’s approach to clinical treatment and research advances.

Crohn’s Disease: A New Paradigm for Patient care
Miguel Regueiro, MD, discusses medical advancements which can, in some patients, lessen IBD surgery.

Visit UPMCPhysicianResources.com/Discovery to view these videos.
A 77-year-old man with past history of myelodysplastic syndrome (MDS) presented with maroon stools. Labs were significant for WBC 3.7, hemoglobin 6.5, and platelets 106. An upper endoscopy revealed a 12x8 mm lesion in the gastric body.

Compare your answer to Dr. Blaney’s on page 9.