Carol Rees Parrish, M.S., R.D., Series Editor

Post-Whipple: A Practical Approach to Nutrition Management

Nora Decher

Amy Berry

The classic pancreatoduodenectomy (PD), also known as the Kausch-Whipple, and the pylorus-preserving pancreatoduodenectomy (PPPD) are most commonly performed for treatment of pancreatic cancer and chronic pancreatitis. This highly complex surgery disrupts the coordination of tightly orchestrated digestive processes. This, in combination with a diseased gland, sets the patient up for nutritional complications such as altered motility (gastroparesis and dumping), pancreatic insufficiency, diabetes mellitus, nutritional deficiencies and bacterial overgrowth. Close monitoring and attention to these issues will help the clinician optimize nutritional status and help prevent potentially devastating complications.

A 63-year-old female, D.D., presented to the University of Virginia Health System (UVAHS) with weight loss and biliary obstruction. She was diagnosed with a large pancreatic serous cystadenoma and underwent a pancreatoduodenectomy (PD) (standard Whipple procedure with partial gastrectomy) with posterior anastomosis and cholecystectomy. Seven months later she was admitted to UVAHS with nausea, vomiting, diarrhea and a severe weight loss of 47lbs (33% of her usual body weight). She had not been able to take in much more than lemonade for 3 months. D.D. experienced a complicated hospital course requiring a stay in the ICU for management of sepsis, respiratory failure, clostridium difficile, heart failure, and volvulus, as well as multiple nutritional deficiencies, including copper, zinc, selenium, and potentially thiamine (thiamine was repleted before serum levels were checked). A percutaneous endoscopic gastrostomy with jejunal extension (PEG-J) was placed due to intolerance of gastric enteral nutrition (EN). After several more hospitalizations, prolonged rehabilitation in a nursing home, 7 months of supplemental nocturnal EN, and treatment of pancreatic insufficiency with pancreatic enzymes (with her meals and EN), D.D. was able to regain her strength, achieve a healthy weight, maintain good nutrition intake without the use of her PEG-J and have the tube removed.

Nora Decher MS, RD, CNSC, UVA Health System, Medical Nutrition Support Team
Amy Berry MS, RD, CNSC, UVA Health System Surgical Nutrition Support Team

Surgical Treatments for Pancreatic Cancer and Chronic Pancreatitis

Pancreatic cancer, most commonly ductal adenocarcinoma or exocrine pancreatic cancer, continues to have sobering statistics. The National Cancer Institute estimates 43,920 new cases and 37,390 deaths from

(continued on page 32)

(continued from page 30)

pancreatic cancer will occur in 2012.[1] Over half of patients are staged as distant or metastatic upon initial diagnosis and the 5-year survival among this group is only 1.8%.[1] Treatment of pancreatic cancer includes surgery, chemotherapy, radiation, or a combination of therapies. Surgery is the mainstay treatment, as it remains the only hope for a cure; however, only 15-20% of patients present with resectable disease. Most patients undergo pancreaticoduodenectomy (PD) with lymphadenectomy, or some variation of this procedure.[2] Other surgeries in the treatment of pancreatic cancer include a total pancreatectomy (TP) and distal pancreatectomy with or without splenectomy.

The diagnosis of both acute and chronic pancreatitis was the 8th highest among all reported digestive disorders in 2004. Incidence seems to be on the rise, as the age-adjusted rate of hospital discharges with the diagnosis of pancreatitis has increased 62% from 1988 to 2004.[3] Surgery may be considered in patients with chronic pancreatitis with at least one of the following: 1) pain refractory to medical treatment and/or narcotic dependency, 2) suspicion of malignancy, or 3) other structural abnormality (unresolved common bile duct stenosis and/or stricture, duodenal compression, portal vein compression, pseudoaneurysms not able to be alleviated by radiologic intervention, and/or pseudocysts unable to be resolved with endoscopic intervention).[4,5] The pathophysiology of pancreatitis pain is poorly understood and therefore is difficult to treat. The aim of surgery in chronic pancreatitis is to ameliorate pain while preserving as much of the pancreatic function as possible.

Two types of procedures are performed in chronic pancreatitis depending on the underlying cause of pain. Drainage procedures (such as lateral pancreaticojejunostomy or Puestow) may be used to relieve a dilated pancreatic duct.[6] Resection procedures include PD, pylorus preserving pancreatoduodenectomy (PPPD) and total pancreatectomy (often with islet cell transplant). Hybrids of these procedures (including Beger and Frey procedures) are also gaining popularity. Detailed descriptions of these procedures are described elsewhere.[5-7] This paper will focus on management of the patient post-PD or PPPD.

The classic PD (or Whipple) procedure involves removing the head of the pancreas along with the distal bile duct, gallbladder, duodenum, first couple centimeters of jejunum, and the distal stomach (often about 50%) along with the pylorus.[8] Intestinal continuity is reestablished by bringing up the remaining section of jejunum and creating a choledochojejunostomy and pancreaticojejunostomy (for bile and enzyme entry into the small bowel), and gastrojejunostomy (see Figure 1a).

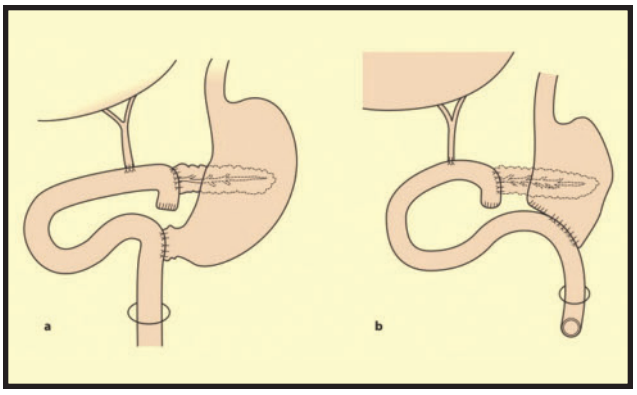

Figure 1. a) Pylorus Preserving Pancreaticoduodenectomy; b) Classic Pancreaticoduodenectomy or Whipple procedure [37]
Reprinted with Permission from Medicine, June 2007

The most common variation of the classic PD procedure is the PPPD (see Figure 1b), which was introduced with the aim of improving post-operative morbidity by avoiding gastrectomy. [9] Proposed benefits of the PPPD include shorter operating time, reduced intraoperative blood loss, avoidance of post-op dumping syndrome from partial gastrectomy, easier endoscopic access postoperatively and potentially improved quality of life and nutritional status.[10] Preservation of the duodenum may also have a positive impact on the hormonal profile regarding postprandial gastrin, cholecystokinin, and secretin. However, studies have not consistently shown a difference in post-operative morbidity between the two styles.

Mortality from the PD and PPPD have vastly improved in the last 1-2 decades, with high volume centers (>20 procedures/year) reporting mortality rates less than 5% and often <2%. However, post-operative morbidities such as pancreatic fistula, delayed gastric emptying, intra-abdominal abscesses, and drain/surgical site infections remain a significant problem, with reported rates as high as 30-60%.[11-12]

Post-Operative Nutrition

Hospital length of stays are quickly decreasing and moving towards a 'fast-track' approach, where stays are 5 -7 days. Often the nasogastric tube for

(continued on page 34)

(continued from page 32)

decompression is removed immediately post-op or within 24 hours, unless complications occur.[13] In the "fast track" progression, diet initiation is started with clear liquids as early as 1-2 days and advanced to regular diet by day 4 or 5. Discharge from the hospital will not occur until tolerance of a solid oral diet is demonstrated. A specialized diet after this procedure is often unnecessary and the diet should only be tailored for symptom management (small meals if patient reports early satiety, nausea/vomiting, etc); a regular diet with adequate caloric and protein provision is the goal. Other symptoms, such as fat malabsorption, most often will be medically managed and not require further diet restrictions.

Dependence on post surgical oral intake to fully meet nutritional needs can be problematic as nausea and decreased appetite are reported in 30% of patients; in addition, delayed gastric emptying can hinder progress.[14] If a patient requires nutrition support subsequent to surgery, the lack of enteral access can be problematic, as surgeons often will not want to place a feeding tube through a new anastomosis until after day 7 (surgeons at UVA). A standard of practice for postoperative EN has not been elucidated, as many surgeons feel placing a feeding tube in all patients may over-treat since some patients may not have postoperative complications. Post-operative EN has been shown to enhance the rate of pancreatic fistula healing, decrease length of stay, readmissions, and early and late overall complications.[15-18] There have been variable results on whether EN decreases incidence or exacerbates delayed gastric emptying. Conversely, the use of parenteral nutrition (PN) postoperatively in these patients has been associated with increased incidence of pancreatic fistulas, hemorrhages and infectious complications.[15,18] Enteral access most often used are J-tubes, PEG-Js and NJTs placed during surgery. Formulas studied have included immune enhancing and polymeric without definitive evidence to support one over the other, due to the lack of large, randomized studies. Our experience is polymeric EN is usually well tolerated.

Long-Term Nutritional Considerations

The alteration of the gastrointestinal tract after undergoing the pancreaticoduodenectomy procedure can cause multiple long-term nutritional complications such as altered GI motility, exocrine pancreatic insufficiency, diabetes, nutrient deficiencies and small bowel bacterial overgrowth (see Table 1). Type and severity of complications will depend on many factors, including extent of pancreatic disease, how the anatomy is altered during surgery and subsequent surgical complications. Management of malnutrition and gastrointestinal symptoms are essential to the patient's quality of life and ability to thrive after surgery.[19] These nutritional complications along with long-term nutritional management of the patient following PD or PPPD will be discussed below. "PD" will be used to refer to both procedures unless otherwise specified.

Gastric Motility

Gastroparesis (GP) has been reported to occur in 25-50% of patients after PD (most common when a secondary complication is present) and usually resolves spontaneously by six months.[20] Persistent GP is not common but may occur. One proposed mechanism is that bypassing the duodenum reduces secretion of motilin therefore reducing activity of the migrating motor complex. Other factors that increase risk of GP include vagotomy or injury to the vagus nerve, anatomical alteration or torsion created during surgery, hyperglycemia, and medications (particularly narcotic use). Numerous studies suggest there is no significant difference between PD and PPPD on incidence of GP during the immediate post-operative period (14 days).[12,21] Long-term follow-up studies comparing rates of GP between other types of surgeries have not been done.

Diagnosis of GP is based on symptoms and objective quantification of delayed gastric emptying. The hallmark prominent symptoms are nausea, vomiting, bloating, early satiety and abdominal pain. The first step in evaluating for GP should include endoscopy to rule out mechanical obstruction, including gastric bezoar. The gold standard for quantifying rate of gastric emptying over time is gastric emptying scintigraphy (GES). Standards for GES have been published by the American Neurogastroenterology and Motility Society and the Society of Nuclear Medicine. Treatment options and nutrition management of these symptoms have been reviewed and published elsewhere.[22]

Prokinetics (ex. Erythromycin, Metoclopramide) and antiemetics are considered the mainstay of therapy for GP. The initial nutrition assessment should distinguish the patient who may undergo treatment on an oral diet alone from those who require early nutrition

(continued on page 36)

(continued from page 34)

support. A PEG-J or jejunostomy may be used in some cases. A glucose goal of < 200mg/dl is recommended by many clinicians to maximize gastric emptying and best nutrient utilization (www.ginutrition.virginia.edu under patient education materials for diet guidelines).

The standard PD surgery, which involves significant gastric resection, may put the patient at risk to experience dumping syndrome (DS). The loss of gastric reservoir and accelerated gastric emptying of osmotic contents into the small bowel provokes a host of untoward symptoms. Dumping syndrome is characterized as diarrhea, fullness, abdominal cramping and vomiting occurring 15-30 minutes after a meal. Weakness, flushing, dizziness and sweating may also be present (see www.ginutrition.virginia.edu under patient education materials for diet guidelines).

Exocrine Pancreatic Insufficiency

Pancreatic disease resulting in a loss of parenchyma, obstruction of the main pancreatic duct (resulting in destruction of parenchyma), decreased pancreatic enzyme secretion or acid-mediated inactivation of pancreatic enzymes may lead to pancreatic insufficiency (PI). A healthy pancreas has a large exocrine reserve capacity and studies have suggested that > 90% of acinar tissue must be lost before signs of steatorrhea are evident.[20] Although usually only ~30% of the pancreas is resected in a PD, exocrine function may be compromised by long-standing pancreatic disease and as a result, pancreatic insufficiency (PI) is common.

Malabsorption of fat can quickly lead to weight loss and assessment for PI should be at the forefront of consideration for the clinician. The process of fat digestion and absorption is more complex and easily

Table 1. Nutritional Complications Following Pancreatoduodenectomy

	Functional Problem	Symptoms/Diagnosis	Treatment Options
Stomach	Gastroparesis	Nausea, vomiting, weight loss, dehydration, electrolyte abnormalities	Fluid and electrolyte repletion, prokinetics, antiemetics, check for and replete vitamin and mineral deficiencies, nutrition support
	Dumping		Nutrition education (small frequent meals, avoid osmotic foods)
Duodenum	Poor mixing, Inadequate digestion, Nutritional deficiencies	Foul smelling stool, weight loss, failure to thrive, fat-soluble vitamin or mineral deficiencies	Treat small bowel bacterial overgrowth, check for and replete vitamin and mineral deficiencies
Pancreas	Exocrine insufficiency (Fat malabsorption)	Foul smelling stool, weight loss, failure to thrive, fat-soluble vitamin or mineral deficiencies /fecal fat testing, fecal elastase 1	Pancreatic enzyme therapy, check for and replete vitamin and mineral deficiencies
	Endocrine insufficiency (Diabetes mellitus)	Fasting plasma glucose levels > 126mg/dL or HgA1C > 6.5%, classic symptoms (blurry vision, excess thirst/hunger, fatigue, frequent urination, weight loss) plus random blood glucose >200mg/dl (American Diabetes Association and WHO criteria)	Insulin therapy, blood glucose monitoring and DM education

interrupted than digestion of other macronutrients. Pancreatic synthesis and secretion of lipase is impaired more rapidly than amylase and proteases due to short intraluminal survival and high susceptibility to denaturing by acid and proteolysis.

Testing for pancreatic insufficiency may not be necessary prior to starting enzyme therapy if clinical signs of PI are present, such as presence of foul-smelling or oily stools or weight loss despite adequate calorie intake. It is rare for patients with pancreatic insufficiency to present with all of the classic manifestations of malabsorption (pale, greasy, voluminous, and foul-smelling stools). Additionally, signs of steatorrhea may be masked by chronic opioid or anti-diarrheal medications use. Hence, steatorrhea may be present despite formed stools.

The most commonly used tests for PI include quantitative fecal fat, qualitative fecal fat and fecal elastase 1 (FE-1). The 72-hour quantitative fecal fat test is considered to be the gold standard for diagnosis of steatorrhea. During this study, the patient is instructed to consume a diet that includes 100g of fat daily for 3-5 days and collect all stool for 72 hours during this period. Presence of an amount of fat in the stool greater than 7% of the total amount of fat consumed in the diet during this period is indicative of fat malabsorption. A detailed description of how a 72-hr fecal fat test is conducted at the University of Virginia Health System is available online at www.GInutrition.virginia.edu (Patient Education Materials → Other → 100 gm Fat Diet and Instructions for 72-hour Fecal Fat Collection). This test is limited by patient and nursing staff compliance, as it relies heavily on the patient to record a very detailed diet log (much easier if the patient is enterally fed with tube feeding and/or guided by a dietitian) and may require collection of a large volume of stool.

Less cumbersome (and less sensitive) qualitative fecal fat tests, such as the Sudan III stain, may useful in some cases. FE-1 is a measure of pancreas-specific proteolytic enzyme that binds to bile salts and remains intact during passage through the gut. FE-1 has been shown to correlate with pancreatic output of amylase, lipase, trypsin and other pancreatic enzymes. This test may be useful as a surrogate marker for pancreatic insufficiency. Drawbacks of this test include: 1) normal values are not well established and 2) watery diarrhea can have a dilutional effect (resulting in a false negative).[24]

One common misconception is the idea that patients with pancreatic insufficiency require a low-fat diet to control steatorrhea and pain. Because fat is such a dense calorie source, a low-fat diet may exacerbate weight loss in a population with already increased energy needs and inadequate intake. In our clinical experience, those patients who experience abdominal discomfort associated with fat intake are often able to achieve relief with adequate enzyme therapy.

To treat pancreatic insufficiency, pancreatic enzymes should be initiated specifically with all fat-containing oral intake and EN. Enzymes should be administered during meals, or every few hours during tube feeding, to allow for adequate mixing. Available preparations are enteric-coated microspheres. The enteric coating protects the enzymes from degradation in gastric acid, allowing it to remain inactive until meeting food in the small bowel. However, non-enteric coated forms (powder or tablets—currently not available in the U.S.) may be preferred in patients who lack gastric acid (ex. atrophic gastritis, high dose PPI) or are solely jejunally fed, where powdered forms may be mixed with the formula. Postprandial pattern of lipase occurs in a mean output rate of 2000–4000 IU/min after ingestion of a normal mixed meal in healthy subjects.[24] Dosing pancreatic enzymes should aim to match physiologic rates, however existing guidelines vary greatly, possibly due to a lack of standardization of enzyme preparations in the past (see Table 2).

As of April 2010 all pancreatic enzyme supplements were required to either obtain FDA approval to ensure effectiveness, safety and manufacturing consistency, or be removed from the market. Over-the-counter pancreatic enzyme products are available without a

Table 2. Pancrealipase Dosing Guidelines (units indicate the amount of lipase)

- 1000-2000 units lipase / kg / meal or 2000-4,000 units lipase / gram dietary fat
- Dose should not exceed 2,500 units of lipase / kg / meal or 10,000 units lipase / kg / day
- Ultra-high doses of enteric coated tablets in have been associated with fibrosing colonopathy in cystic fibrosis patients[23]

Adapted from Creon dosing instructions (www.rxabbott.com) and clinical experience at UVAHS

Table 3. Summary of Long-Term Nutrition Management Guidelines Following Pancreatoduodenectomy

Evaluation

- Evaluate nutritional status through careful weight and diet history
- Determine patient's anatomy (understand type of PD, discuss w/ surgeon or review of op-reports if available, ask patient about other prior GI surgeries)
- Evaluate for nutritional complications (fat malabsorption, diabetes, nutrient deficiencies, gastroparesis, dumping)
- Consider baseline DEXA scan if not already done (unless palliative)
- Hydration

Maintain Optimal Nutritional Status

- Set weight goal.
- Provide diet education to manage gastroparesis, early satiety, dumping, hyperglycemia, and/or lactose intolerance, if present
- Daily multivitamin with minerals with a plan to reevaluate status to prevent unnecessary supplementation.
- Additional calcium (500-1000mg/ day) and vitamin D (600-1000 IU/day), as warranted
- Continued nutrition intervention for at-risk patients
- Nutrition support if initial interventions fail and/or weight loss continues or repletion thwarted

Treatment of Fat Malabsorption

- Check C. Difficile in those presenting with diarrhea
- Determine level of suspicion for steatorrhea (consider symptoms, extent of pancreatic disease, ask patient about history of alcohol consumption).
- Stools may contain fat even if they appear normal (particularly w/opioid or antidiarrheal use)
- Test for PI or treat empirically with pancreatic enzymes.
- Use gut-slowing agents if needed
- Treat bacterial overgrowth if present
- Monitor and supplement fat soluble vitamins and minerals as needed

Diabetes Mellitus

- Educate patient about symptoms
- Check HgA1C
- Refer to endocrinologist or primary care physician for insulin therapy
- Refer to diabetes educator for comprehensive teaching

prescription; however, these products are classified as dietary supplements rather than drugs. Because of this classification they are not controlled by the FDA for manufacturing consistency from one batch to the next and are not recommended.

Formulations that are currently approved by the FDA include: Creon® capsules, Pancreaze™ capsules, Zenpep™ capsules, Ultrase™ capsules and Viokace™ tablets or powder (still pending market availability). Pancrecarb® capsules are pending approval.

For instructions on how to administer enteric-coated formulations with tube feeding, refer to: www.GInutrition.virginia.edu → Resource for the Nutrition Support Clinician → Tube Feeding and Pancreatic Enzymes

Diabetes Mellitus

Twenty to fifty percent of patients will develop diabetes mellitus (DM) after pancreatic resection.[20] Studies suggest that 80% of individuals with pancreatic cancer have DM preceding the diagnosis.[26] In some populations of patients with pancreatic cancer, hyperglycemia has been shown to improve after surgery.[27] However, in patients with existing chronic pancreatitis, new DM after surgery is common.[28] There is limited information available to guide clinicians in predicting which

Table 3., continued

Vitamin / Mineral Monitoring

- Careful physical exam to look for signs of nutrient malabsorption
- Check baseline and after 1 year (or sooner if malnutrition/symptoms of deficiency present) (based on UVA experience):
  - Vitamin B12; methylmalonic acid also if SIBO present or highly suspected
  - Folate
  - Ferritin
  - 25OHD
- If symptoms of nutrient deficiency (reported below in parenthesis) or severe malnutrition/malabsorption present, consider checking:
  - Zinc (dermatitis enteropathica, glossitis, chelilitis, stomatitis)
    - If large volume stool losses are present, in our clinical experience we would consider treating empirically with 220mg zinc sulfate daily for 4 weeks then reevaluate; fix diarrhea
  - Copper (neuropathy, gait disturbance)
  - Selenium (cardiomyopathy, hypothyroidism)
  - Vitamin E (visual impairment, gait disturbance)
  - Vitamin A (night blindness, skin disorder/dermatitis)
  - Vitamin K (bleeding, stomach pain, cartilage calcification, atherosclerosis)
- Supplement thiamine when severe malnutrition is present and/or if significant alcohol ingestion is on going.
  - 100-200mg daily IV x 3-5 days
  - Then transition to 100mg daily PO x 5-7 days.
  - Higher IV doses are needed if pt is symptomatic
- If a patient has multiple nutrient deficiencies, it should be assumed that others are present. A complete multivitamin with minerals should be provided in addition to repletion of specific nutrients.

Gastroparesis [22]

- Fluid and electrolyte repletion
- Decision for oral nutrition (Small, frequent meals w/ focus on liquids) vs nutrition support (if unable to maintain nutrition/hydration orally)
- Anti-emetic and prokinetic therapy
- Treat bezoars [37]
- Treat bacterial overgrowth [34]

patients will develop DM post-operatively. However, pre-operative HgA1C has been suggested as a helpful screening tool.[29]

Patients should be educated regarding how to identify signs and symptoms including excessive thirst and hunger, frequent urination, fatigue and unintentional weight loss. Measurement of HgA1C or fasting blood glucose may be considered if the patient develops significant weight loss despite maintaining their usual food intake. Hyperglycemia during EN is often overlooked and patients newly started on EN may benefit from measuring blood glucose levels at the start, mid-infusion, and immediately following the end of feeding. Referral to a DM education program is essential for patients who are newly diagnosed with DM.

Nutrient Deficiencies

For many patients, a healthy diet including lean meats, legumes, whole grains, dairy products, fruits and vegetables, in addition to a multivitamin with minerals, is sufficient. However, some patients will benefit from additional nutrient supplementation. Understanding the patient's baseline nutritional status and surgical anatomy are critical to assessing the risk for specific nutrient deficiencies.

Nutrient deficiencies may result from lack of intake, malabsorption, or maldigestion of nutrients. With bypass of the duodenum and upper jejunum, the tightly orchestrated digestive processes between the stomach, duodenum and pancreatobiliary system are disrupted. Production of gastrin, cholecystokinin and secretin are reduced, leading to diminished pancreatic secretion of bicarbonate and inadequate gastric acid neutralization. The duodenum and proximal jejunum are also important sites for absorption of iron, folate, fatty acids, proteins and trace elements. Bypass of this part of the small bowel may result in impaired absorption of iron, calcium, zinc, copper and selenium. In addition to bypass of absorptive surface area, a PD may lead to impaired digestion by creating a "mismatch" of mixing of bile and pancreatic secretions with foodstuffs.

Available Evidence

Armstrong et al examined serum levels of fat soluble vitamins (retinol, 25-OHD, alpha-tocopherol), iron status (iron, ferritin, transferrin, transferrin saturation) and trace elements (selenium and zinc) in 37 patients > 6 months after PD for periampullary neoplasia. Frank selenium deficiency (indicated by serum levels) was identified in 56% of patients, and serum 25-OHD and alpha-tocopherol were reduced compared to the controls (but not outside of the normal range).[30] Yu et al studied 48 patients an average of 4 years after surgery and found 69% to be zinc deficient. Of these patients, 52% exhibited symptoms of zinc deficiency (including acrodermatitis enteropathica, purulent bulla, poor wound healing, photophobia, night vision blurred, and glossitis) and deficiency was strongly associated with pancreatic exocrine insufficiency. [31] Unfortunately, neither of the above studies measured nutrient levels prior to surgery.

Vitamin/mineral deficiencies are often overlooked, particularly when other complications are present. Clinicians being alert to patients who are at risk for deficiencies and providing repletion as necessary can prevent more severe and costly consequences. Several nutrients (zinc, ferritin, calcium, copper) are bound to albumin in the serum and therefore, checking serum levels may not reflect whole body stores when the patient is acutely ill. Caution should be taken to change patients from repletion doses to maintenance doses, once repletion has been achieved, to avoid over-supplementation. A comprehensive overview of signs/symptoms of nutrient deficiencies and recommendations for repletion (specifically in the gastric bypass population) is available.[32]

Complications Related to the Afferent Limb

Afferent limb syndrome is defined as chronic, mechanical obstruction of the afferent limb resulting in pancreatobiliary complications.[33] These complications can be caused by kinking, hernias, compression of the afferent limb from adhesions or radiation enteritis. The afferent limb creates a "blind loop," which can serve as a nidus for bacteria to thrive. Small bowel bacterial overgrowth (SIBO) is quite common and may accompany mechanical complications or exist alone. Symptoms of SIBO include nausea, gas, bloating, diarrhea (due to deconjugation of bile salts), B12 deficiency and elevated folate.[34] SIBO is an under-recognized cause for non-specific gastrointestinal symptoms; these symptoms alone can be embarrassing (flatulence, belching, fecal incontinence, etc.), can create a malabsorptive process, severely reduce quality of life, and inhibit nutritional intake. Treatment with broad spectrum antibiotics is effective, however repeated courses may be needed in some patients.[34]

Nutritional Monitoring

Ongoing nutritional assessment is essential to optimize the patient's recovery and long-term success. Assessment of patient's weight over time is a simple and valuable tool for determining nutritional status.[35] A euvolemic weight should be established so that any fluid changes over time can be accounted for. Comparing a patient's current weight to their ideal weight may underestimate malnutrition (leading to a false assumption that "patient appears well-nourished") or overestimate malnutrition (as in the case of a person who has been thin all his or her life). A detailed diet history is also valuable for assessing changes in nutritional status as well as nutritional adequacy. Ideally, a 3-day diet log should be completed by the patient and reviewed by a registered dietitian. However, a 24-hr recall of a typical day is the most practical method if the patient is unable to complete a 3-day log. Weight history (especially recent unintentional loss), diet history, physical exam and functional status are the main factors considered in assessment of the patient's nutritional status. Initial assessment will help guide the level of nutritional support that is appropriate and if emergent intervention is necessary.

(continued on page 42)

(continued from page 40)

CONCLUSION

The PD procedure has many variations and can alter many normal digestive and metabolic processes. Identifying and correcting these abnormalities is paramount to preserving nutritional status in patients undergoing PD. PD surgery alters the fine coordination of the digestive process setting the patient up for nutritional compromise such as altered motility (gastroparesis and dumping), pancreatic insufficiency, diabetes mellitus, nutritional deficiencies and bacterial overgrowth. Close monitoring and attention to the clinical signs and symptoms if they arise, will help the clinician optimize nutritional status and help prevent potentially devastating complications. For suggested trouble-shooting tips, see Table 3. ■

References

1. Cancer of the Pancreas - SEER Stat Fact Sheets [Internet]. Available from: http://seer.cancer.gov/statfacts/html/pancreas.html
2. Hidalgo M. Pancreatic Cancer. N Engl J Med, 2010;362: 1605–1617.
3. Everhart JE, Ruhl CE. Burden of Digestive Diseases in the United States Part III: Liver, Biliary Tract, and Pancreas. Gastroenterology, 2009;136:1134–1144.
4. Rückert F, Kersting S, Fiedler D, et al. Chronic pancreatitis: early results of pancreatoduodenectomy and analysis of risk factors. Pancreas, 2011; 40:925–930.
5. Aimoto T, Uchida E, Nakamura Y, et al. Current surgical treatment for chronic pancreatitis. J Nihon Med Sch, 2011;78:352–359.
6. Andersen DK, Frey CF. The evolution of the surgical treatment of chronic pancreatitis. Ann. Surg, 2010;251:18–32.
7. Reddy SK, Tyler DS, Pappas TN, et al. Extended resection for pancreatic adenocarcinoma. Oncologist, 2007;12:654–663.
8. Niedergethmann M, Shang E, Farag Soliman M, et al. Early and enduring nutritional and functional results of pylorus preservation vs classic Whipple procedure for pancreatic cancer. Langenbecks Arch Surg, 2006;391:195–202.
9. Uravić M, Zelić M, Petrošić N, et al. Effect of pyloric dilatation on gastric emptying after pylorus-preserving pancreaticoduodenectomy. Hepatogastroenterology, 2011;58:2144–2147.
10. Paraskevas KI, Avgerinos C, Manes C, et al. Delayed gastric emptying is associated with pylorus-preserving but not classical Whipple pancreaticoduodenectomy: a review of the literature and critical reappraisal of the implicated pathomechanism. World J. Gastroenterol, 2006;12:5951–5958.
11. Horstmann O, Markus PM, Ghadimi MB, et al. Pylorus preservation has no impact on delayed gastric emptying after pancreatic head resection. Pancreas, 2004;28:69–74.
12. Kurahara H, Shinchi H, Maemura K, et al. Delayed gastric emptying after pancreatoduodenectomy.J Surg Res, 2011;171:e187–192.
13. Willcutts K. Pre-op NPO and traditional post-op diet advancement: Time to move on. Practical Gastroenterology, 2010; series 90:16-27.
14. Warren J, Bhalla V, Cresci G. Postoperative Diet Advancement: Surgical Dogma vs Evidence-Based Medicine. Nutrition in Clinical Practice, 2011;26:115–125.
15. Liu, C., Du, Z., Lou, C., et al. Enteral nutrition is superior to total parenteral nutrition for pancreatic cancer patients who underwent pancreaticoduodenectomy. Asia Pac J Clin Nutr, 2011;20:154–160.
16. Grizas, S., Gulbinas, A., Barauskas, G., et al. A comparison of the effectiveness of the early enteral and natural nutrition after pancreatoduodenectomy. Medicina (Kaunas), 2008;44:678–686.
17. Baradi, H. Postoperative jejunal feeding and outcome of pancreaticoduodenectomy. Journal of Gastrointestinal Surgery, 2004;8:428–433.
18. Gianotti, L., Braga, M., Gentilini, O., et al. Artificial nutrition after pancreaticoduodenectomy. Pancreas, 2000; 21:344–351.
19. Carey S, Storey D, Biankin AV, et al. Long term nutritional status and quality of life following major upper gastrointestinal surgery - a cross-sectional study. Clin Nutr, 2011; 30:774–779.
20. Tran TCK, van Lanschot JJB, Bruno MJ, et al. Functional changes after pancreatoduodenectomy: diagnosis and treatment. Pancreatology, 2009;9:729–737.
21. Kunstman JW, Fonseca AL, Ciarleglio MM, et al. Comprehensive Analysis of Variables Affecting Delayed Gastric Emptying Following Pancreaticoduodenectomy. Journal of Gastrointestinal Surgery. EPub March 2012.
22. Parrish C, McCray S. Gastroporesis and Nutrition: The Art. Practical Gastroenterology 2011;26–41.
23. Layer P, Keller J, Lankisch PG. Pancreatic enzyme replacement therapy. Curr Gastroenterol Rep, 2001;3:101–108.
24. Lieb JG 2nd, Draganov PV. Pancreatic function testing: here to stay for the 21st century. World J Gastroenterol, 2008;14: 3149–3158.
25. Domínguez-Muñoz JE. Pancreatic enzyme replacement therapy for pancreatic exocrine insufficiency: When is it indicated, what is the goal and how to do it? Advances in Medical Sciences, 2011;56:1–5.
26. Fang W-L, Su C-H, Shyr Y-M, et al. Functional and morphological changes in pancreatic remnant after pancreaticoduodenectomy. Pancreas, 2007;35:361–365.
27. Gullo L, Pezzilli R, Morselli-Labate AM. Diabetes and the risk of pancreatic cancer. N. Engl J Med, 1994;331:81–84.
28. Riediger H, Adam U, Fischer E, et al. Long-term outcome after resection for chronic pancreatitis in 224 patients. J. Gastrointest Surg, 2007;11:949–959.
29. Hamilton L, Jeyarajah DR. Hemoglobin A1c can be helpful in predicting progression to diabetes after Whipple procedure. HPB, 2007;9:26–28.
30. Armstrong T, Strommer L, Ruiz-Jasbon F, et al. Pancreaticoduodenectomy for peri-ampullary neoplasia leads to specific micronutrient deficiencies. Pancreatology, 2007;7:37–44.
31. Yu H-H, Yang T-M, Shan Y-S, et al. Zinc deficiency in patients undergoing pancreatoduodenectomy for periampullary tumors is associated with pancreatic exocrine insufficiency. World J Surg, 2011;35:2110–2117.
32. O'Donnell K. Severe Micronutrient Deficiencies in RYGB Patients: Rare but Potentially Devastating. Practical Gastroenterology, 2011;13–22.
33. Pannala R, Brandabur JJ, Gan S-I, et al. Afferent limb syndrome and delayed GI problems after pancreaticoduodenectomy for pancreatic cancer: single-center, 14-year experience. Gastrointest Endosc, 2011;74:295–302.
34. DiBaise JK. Nutritional consequences of small intestinal bacterial overgrowth. Practical Gastroenterology, 2008;15-28.
35. Jeejeebhoy KN. Nutritional assessment. Gastroenterol Clin North Am, 1998;27:347–369.
36. Sanders M. Bezoars: From mystical charms to medical and nutritional management. Practical Gastroenterology, 2004;37-50.
37. Spalding D, Williamson RCN. Pancreatic cancer. Medicine, 2007;35:325–329.