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Parenteral Nutrition in Pancreatitis is Passé: But Are We Ready for Gastric Feeding?

A Practical Guide to Jejunal Feeding: Revenge of the Cyst –Part II

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(See September 2007 for Part I on evidence supporting jejunal vs parenteral or gastric feeding)

Nutrition support is required to prevent or reverse malnutrition in the 15%–20% of patients that develop severe or complicated pancreatitis who are unable to resume oral intake in seven-to-ten days. The best available data supports the use of jejunal feeding over parenteral nutrition in those patients. Jejunal enteral nutrition can be successfully achieved by using nasojejunal access (in those patients requiring <30 days of nutrition support) and either percutaneous endoscopic gastrostomy with jejunal extension or direct percutaneous jejunostomy access in patients requiring longer support. Symptoms such as diarrhea, nausea, vomiting, abdominal pain, and excessive gastric secretion may appear to be obstacles to successful enteral feeding, but our experience demonstrates that patients rarely remain intolerant to enteral feeding and require parenteral nutrition. The transient gastrointestinal symptoms associated with enteral feeding can be managed by the following recommendations outlined in this article. The use of long term enteral nutrition in patients with chronic pain, pseudocysts, malnutrition and other complications is increasing, but the efficacy of this practice still needs to be clearly demonstrated in randomized controlled trials.

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INTRODUCTION

The majority of patients with pancreatitis have a mild form of the disease and recover fully after a short period (3–5 days), while the remaining 15%–20% of patients will progress to a more complicated course, ultimately requiring nutritional support (1). In the past, parenteral nutrition (PN) was the mainstay of treatment; however, the evidence that has accrued in recent years has demonstrated that jejunal enteral feedings are, by far, the safest means to achieve this end (2). Although the decision to enterally feed may seem simple, the reality is that enteral feeding requires tenacity and clinical acumen. This article chronicles the evidence, as well as our experience, with jejunally feeding the patient with severe, complicated pancreatitis and the nutritional concerns that may arise long term.

PRACTICAL ASPECTS OF ENTERAL NUTRITION

Although reviews and practice guidelines have concluded that jejunal enteral nutrition (EN) is the preferred route for providing nutrition support during acute pancreatitis (3–7), our discussions with nutrition support professionals from across the nation suggest that routine use of PN in patients with pancreatitis remains quite common. Lack of technical expertise, difficulty in placement and maintaining jejunal access and perceptions of feeding intolerance that prevent successful EN continue to be barriers to successful EN during pancreatitis at many facilities.

ENTERAL ACCESS

Short-term jejunal enteral access can be achieved through the placement of a nasojejunal feeding tube in most patients. Fluoroscopy and endoscopy are frequently used to assist and ensure the placement of feeding tubes beyond the Ligament of Treitz (LOT). In our institution, fluoroscopic placement is the more cost-effective method, therefore, we reserve endoscopic tube placement for those patients that already require endoscopy, or in whom fluoroscopic placement has been unsuccessful. Our early experiences of attempting to drag or advance feeding tubes with an endoscope were not only time consuming, but frequently resulted in displacement of the tube when the endoscope was removed. More recently, when endoscopic placement is required, the use of a pediatric endoscope to place the guidewire then advancing the feeding tube over the guidewire, has been a more practical and successful approach.

Transnasal endoscopic placement of feeding tubes has been described, eliminating the need for intravenous sedation, but this method requires the use of an ultra thin endoscope (8). Wiggins has also described an endoscopically guided NJ placement push technique in which a 12 Fr Endotube stiffened by placement of two wires in its lumen is pushed into the small bowel under endoscopic visualization (9).

Magnetic guidance of feeding tubes (http://syncromedicalinnovations.com/content/section/4/45/) and use of modified feeding tubes that generate an electromagnetic signal recognized by an external receiver placed on the abdomen have reported success with placement of feeding tubes beyond the pylorus, but there is limited data about their effectiveness for placement of feeding tubes beyond the LOT (10).

Although there are no randomized studies supporting one type of nasojejunal access over another, our experience has been that the use of the largest size of the "small bore" feeding tubes (12 Fr as opposed to 8 or 10 Fr), results in less clogging without any discernable increase in patient discomfort. Double lumen gastrojejunal tubes that have 2 lumens are available (Tyco-Kendall Healthcare (http://www.kendallhealthcare.com/kendallhealthcare); the first lumen terminates in the stomach and the second in the jejunum. Double lumen tubes, which allow feeding into the distal opening and simultaneous gastric decompression/drainage through the proximal opening, may be useful to decrease nausea related to retention of endogenous gastric secretions without the need for a second nasal tube for nasogastric decompression. One potential disadvantage of double-lumen tubes is that in order to maintain an external diameter that is relatively comfortable for the patient (14-16 Fr); the jejunal portion of the tube is usually 6-8 Fr and may be prone to frequent clogging. In addition, because dual-lumen tubes are also used for decompression, the external diameter of the tube is significantly larger and stiffer than a small bore feeding tube and

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long-term patient comfort is an issue (personal experience of the authors).

When EN is required for periods of 30 days or less, many clinicians prefer to maintain nasojejunal access, reserving placement of percutaneous jejunal access for patients requiring long-term EN (11,12). Although it is possible to maintain nasal access for longer than 30 days, long-term nasojejunal tubes are not as desirable by most patients that are candidates for discharge to home (personal experience of the authors).

No randomized studies exist aimed at determining the optimal duration of EN support for patients with complicated pancreatitis. However, long-term EN with delayed introduction of oral intake (mean of 4.4 months) may be beneficial in patients with acute severe necrotizing pancreatitis (13) and in those with chronic recurrent pancreatitis with pseudocysts (13–16). There is a need for randomized studies to determine if there are outcome benefits (infectious complications, reduced hospitalizations, decreased surgical necessity) with extended jejunal EN and delayed oral intake in the setting of pancreatitis complicated by pseudocyst or necrosis.

Long-term jejunal access can be achieved by endoscopic placement of either percutaneous gastrostomy with jejunal extension (PEG-J) or by direct percutaneous jejunostomy (DPEJ). Although PEG-J has been criticized as having a significantly greater attrition rate than DPEJ in terms of tube patency (17), this limitation appears to primarily affect small-bore PEG-J devices. One case series that reported significantly more attrition from occlusion of the J-arm with PEG-J compared to direct percutaneous jejunostomy, utilized small bore (9 Fr) jejunal extension through a 20 Fr PEG (17). Another case series documented similar problems with small-bore jejunal extensions, reporting 83% of all occlusions in jejunal extensions occurring in the smaller tubes (8.5 Fr) (18). Those case series that have reported low malfunction rates with PEG-J tubes have utilized a 24 Fr PEG with a 12 Fr jejunal extension (13,16). Various techniques for PEG-J placement exist and have been described in various publications (11,13,17,19). Our practice is to pay particular attention to placement of the PEG (into the distal portion of the stomach, to the right of the spinal column, facing the pylorus) because we have found that this position decreases the distance the j-arm must traverse across the stomach and allows the jejunal extension to reach well beyond the LOT and appears to result in less displacement of the jejunal tube (13). One advantage of PEG-J tubes is that they allow decompression of gastric secretions while feeding into the jejunum. Although persistent gastric outlet obstruction occurred in only 14% of patients with complicated pancreatitis, in our experience, a much larger percentage of patients utilized the gastric port of the PEG-J to relieve symptoms of nausea during the initial period of jejunal feeding (13).

DPEJ tubes have also been successfully used to provide long-term EN (11,13,17,19). The major limitation with DPEJ's is their inability to facilitate gastric decompression in patients with functional gastric outlet obstruction. Patients that receive a DPEJ and have persistent gastric outlet obstruction may require a second percutaneous gastric tube for decompression and be exposed to the inherent risks that this may involve.

POSITION OF THE TIP OF THE TUBE

Positioning the tip of a feeding tube into the duodenum frequently allows successful EN in the setting of gastric dysmotility due to critical illness or gastroparesis. However, there is evidence that feeding into the duodenum is a strong stimulus to pancreatic secretions (20–22). Several studies have reported that infusing either elemental or polymeric feeding into the duodenum resulted in increased secretion of amylase, lipase, trypsin, bile acid, CCK and gastrin when compared to controls and those receiving PN (21,22). In contrast, when elemental or polymeric formulas were infused 40–60 cm beyond the Ligament of Treitz, there was actually an inhibition of pancreatic secretions compared to PN (21). Bedside techniques for blind placement of post-pyloric feeding tubes are rarely successful in placing feeding ports beyond the LOT. Most facilities use either endoscopic or fluoroscopic placement to ensure that feeding tubes are adequately distal to the LOT. A word of caution; it is essential that the clinician recognizes the location of the feeding ports in relation to the tip of the feeding tube. Feeding tubes that have several feeding ports proximal to the tip (frequently seen with weighted tubes) may appear to be beyond the LOT, while in reality the feeding ports remain in the duodenum and result in pancreatic

stimulation and worsening of pancreatitis/symptoms, leading clinicians to think that jejunal EN does not work. Feeding ports should be distal to the LOT to minimize pancreatic stimulation or reflux of formula when feeding patients with severe acute pancreatitis.

The standard small bore feeding tube at our facility (polyurethane 43 inch, 12 Fr Entriflex™ (http://www.kendallhealthcare.com/kendallhealthcare), placed through the jejunal port of a PEG tube, has allowed adequate jejunal access in the majority of our patients, however some patients have required a longer tube (55 inch, 12 Fr Entriflex™) (http://www.kendallhealthcare.com/kendallhealthcare), to reliably feed distal to the LOT.

FORMULA SELECTION

The initial studies of jejunal EN in acute pancreatitis used elemental or semi-elemental formulas, but several studies since have described successful jejunal EN using polymeric formulas with positive results (13,23–28). The conventional wisdom that elemental or semi-elemental formulas are better tolerated in patients with pancreatitis is based on two *assumptions*:

1. Standard EN formulas containing fat will stimulate the pancreas exacerbating the pancreatitis, and
2. Maldigestion from pancreatic insufficiency always accompanies pancreatitis and therefore, an elemental or semi-elemental formula is needed.

One early case report suggested that jejunal administration of a polymeric EN formula resulted in a five-fold increase in pancreatic lipase output compared to elemental EN (29). However, more recent research suggests that "pancreatic rest" can be achieved by administering a polymeric formula, as long as it is infused sufficiently distal to the LOT (21).

A study in healthy volunteers demonstrated that a polymeric liquid diet administered through a tube located just proximal to the LOT, resulted in a significant increase in lipase, amylase and trypsin output, while administration through a tube located 60 cm distal to the LOT did not result in a similar increase in output (30). Another study found that when polymeric formulas were infused 40–60 cm distal to LOT there was actually an inhibition of pancreatic secretions compared to PN (21).

In a randomized trial, Windsor, et al reported that polymeric EN resulted in significant reductions in C-reactive protein and APACHE II score compared to patients receiving PN (31). Pupelis, et al randomized patients to receive either jejunal EN with a polymeric formula, or standard therapy (npo receiving IV fluids) (32). Patients receiving polymeric formula via nasojejunal EN had significantly decreased mortality ($p = 0.05$) compared to standard therapy. Modena, et al in a study utilizing historical controls, reported that the group receiving polymeric jejunal EN had significant reductions in mortality ($p < 0.001$), less pancreatic necrosis ($p < 0.001$), organ failure or need for surgery ($p < 0.001$) than those patients receiving PN (33).

A retrospective study of patients with complicated pancreatitis receiving long-term (average 4.4 months) polymeric jejunal EN reported median CT severity index significantly improved ($p < 0.001$) while receiving polymeric jejunal EN. In addition, those patients with a BMI <18.5 at entry experienced a significant weight increase (13).

Although polymeric formulas appear to be well tolerated by the average patient with pancreatitis, there is a concern that those patients with pancreatic exocrine insufficiency may experience malabsorption or diarrhea. Several investigators have described the incidence of pancreatic exocrine insufficiency in patients with pancreatitis (34,35), but there is limited data regarding the incidence of malabsorption in patients receiving enteral feeding. A retrospective review of 127 patients with complicated pancreatitis who received jejunal EN reported that 19 of the 63 patients (30%) tested for fecal fat had evidence of steatorrhea (13). However, only two of 126 patients in this cohort received a semi-elemental EN; all other patients with steatorrhea were reported to tolerate and clinically progress well on polymeric EN after pancreatic enzyme powder was added to the feeding formula.

There is only one randomized study that has directly compared the use of semi-elemental to polymeric EN in acute pancreatitis (36). The pilot study enrolled 30 subjects and found that both formulas were well tolerated without a significant difference in stool fat or protein loss between the two groups. Furthermore, no significant differences in pain scores, amylase or C-reactive protein were noted, implying lack of increased

inflammation in the polymeric group. The semi-elemental EN group had a significantly shorter hospital stay (23 ± 2 versus 27 ± 1, p = 0.006) and less weight loss. However, the difference in weight, although statistically different (2.4 lbs), could be attributed solely to transient changes in fluid status known to occur in this patient population. Considering the small number of patients, and the apparent lack of pain exacerbation, increase in inflammatory markers, or any sign of increased feeding intolerance or malabsorption of polymeric feeding, a much larger, double-blind study would be required to know if there is a meaningful advantage to elemental formulas that would justify the significantly increased cost of these formulas (Tables 1 and 2).

MANAGEMENT OF "FEEDING INTOLERANCE"

Diarrhea

Pancreatic Insufficiency

Diarrhea is not an uncommon finding in patients with pancreatitis that receive EN, but diarrhea does not equal feeding intolerance, nor does it necessarily equate to malabsorption (37). Pancreatic insufficiency may be associated with the occurrence of steatorrhea that may present as diarrhea. Pancreatic exocrine insufficiency occurs in 35%–86% of patients with severe acute pancreatitis (35,38,39) and pancreatic endocrine impairment (hyperglycemia) was documented in 25% of patients in one study (40). A greater frequency of both exocrine and endocrine pancreatic insufficiency has been reported in patients with alcohol-induced acute pancreatitis compared to gallstone-associated pancreatitis (35,39). Migliori, et al (35) found that 84% of patients with acute alcoholic pancreatitis and 22% with acute biliary pancreatitis demonstrated evidence of exocrine pancreatic insufficiency using duodenal intubation and the amino acid consumption test. However Bozkurt, et al further found that while biochemical pancreatic insufficiency occurred in 80%–85% of cases, clinical signs of exocrine insufficiency occurred only in 5%–10% (34). A retrospective review of patients with pancreatitis who received jejunal feeding suggests that only a small percentage of those patients are diagnosed with exocrine insufficiency (41).

Depending on the extent of pancreatic damage, pancreatic enzyme insufficiency may display early on, or may occur much later as fibrosis and pancreatic cal-

Table 1
Nutrition Information of Various Elemental and Semi-Elemental Formulas

Product	*Source*	*Calories per mL*	*CHO g/L*	*Pro g/L*	*Fat g/L*	*MCT:LCT*[1]	*Price/ 1000 kcal**
F.A.A.	Nestle	1.00	176	50	11	25:75	$29.92
Optimental	Ross	1.00	139	51	28	45:55	$25.43
Peptamen	Nestle	1.00	127	40	39	70:30	$24.90
Peptamen 1.5	Nestle	1.50	191	60	58	70:30	$24.20
Peptinex DT	Novartis	1.00	164	50	17	50:50	$25.00
Perative	Ross	1.30	177	67	37	40:60	$12.41
Tolerex	Novartis	1.00	230	21	1.5	No MCT	$16.67
Vital HN	Ross	1.00	185	42	11	45:55	$21.18
Vivonex TEN	Novartis	1.00	210	38	2.8	No MCT	$18.33

*Phone numbers used to obtain pricing information on June 1, 2007:
Ross: (800) 544-7495
Nestle: (800) 776-5446
Novartis: (800) 828-9194

[1]Medium chain triglyceride (MCT): Long chain triglyceride (LCT)
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Table 2
Cost Comparison of Elemental Formulas versus Standard Formulas with Pancreatic Enzymes Added

Formula	*Price/1000 kcal**
Elemental	
F.A.A.	$29.92
Peptamen 1.5, unflavored	$24.90
Vital HN	$20.28
Vivonex TEN	$18.33
Standard formula ***with and without*** ½ teaspoon Viokase powder added per can**	
Jevity 1.5	$14.02 / 11.02
Two Cal HN	$6.38 / 3.78
Promote with Fiber	$10.73 / 7.73
Probalance	$11.76 / 8.76

*Cost information obtained from company using toll free number (2007 prices)
**Cost based on « teaspoon of Viokase powder (8 oz.—Wal-Mart) added per can formula (1/2 teaspoon = 0.75 cents)
Note: ½ teaspoon contains ~1.4 g Viokase powder
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Table 3
Addition of Pancreatic Enzymes to Enteral Formula

- Use ½–1 teaspoon of pancrealipase powder (Viokase in patients who are not achieving clinical response) per can of enteral formula given.
- First mix the powdered enzyme in 25–50mL of lukewarm water to increase solubility when added to enteral product.
- In those patients who prefer to crush pancrealipase tablets (or who have a large supply at home), 4 tablets of Viokase-8 (equivalent to 1/2 teaspoon of powder), or any other equivalent tablet that provides 8,000 units of lipase activity/tablet, can be crushed fine, mixed with tube feeding and infused via jejunal tube (a mortar and pestle is an inexpensive way to do this at home). **NOTE: Do not use sustained release preparations for this purpose!**
- After adding the enzyme mixture, mix it up in the bag by hand—it will look "icky" as the enzyme starts to breakdown the formula. We have our patients squish the bag by hand periodically.
- Some patients prefer to give a "dose" of enzyme before bed, and maybe once during the night, and again in the am before the tube feeding runs out, rather than adding to the formula itself. However, if the clinical response is not adequate, then it may need to be given with the feedings for better delivery with nutrients.
- In those patients who are transitioning to PO intake with persistent pancreatic insufficiency, acid suppression may be needed to keep the gastrically delivered enzymes from being denatured by gastric acid. More than one patient has started PO in the hospital with enzyme powder being given with enteral feeding, but none ordered with their meals.

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cification occur. Our experience has indicated that not all patients with pancreatic exocrine insufficiency exhibit overt diarrhea. In particular, during the early stages of severe pancreatitis the use of narcotics with resultant gut slowing may prevent diarrhea despite the presence of steatorrhea. See Table 3 for our institution's guidelines for adding pancreatic enzymes to standard enteral formulas.

Small Intestinal Bacterial Overgrowth

Another likely cause of diarrhea, especially in patients with complicated acute on chronic pancreatitis, is small intestinal bacterial overgrowth (SIBO) (42–45). SIBO has been reported in 34%–40% of patients who have pancreatic insufficiency (42,43). Animal studies have also demonstrated a positive correlation between duodenal bacterial overgrowth and acute necrotizing pancreatitis (46). This association has been attributed to a combination of factors including: decreased intestinal motility as a result of narcotic pain medication, reduction of gastric acid from the use of proton pump inhibitors (PPIs), inflammatory or obstructive changes to the proximal small intestine resulting from pancreatic enlargement, loss of bactericidal effects by pancreatic enzymes as well as a decrease in bicarbonate secretion further decreasing the efficacy of the pancreatic enzymes that are present. SIBO causes diarrhea in two ways:

1. By creating a functional bile salt deficiency by way of denaturing the bile salts. The end result is an alteration in micelle formation and steatorrhea, and
2. The denatured bile salts are caustic to the colon causing a secretory (cholereic) diarrhea. Of interest, normal enzymes and biliary secretion result in 97% fat

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absorption within the first five feet of the jejunum; bile salt deficiency (or "disabled" bile salts) decreases this efficient process to 40%–50% (47).

Diagnosis and treatment of SIBO is beyond the scope of this article but has been detailed in previous publications (48).

Other Causes of Diarrhea

Patients with pancreatitis receiving antibiotics are at risk for *C. Difficile* infection as well as osmotic diarrhea from medications. In our population, those patients with persistent diarrhea that test negative (or are treated for) *C. Difficile* and pancreatic exocrine insufficiency based on a quantitative fecal fat (assuming adequate fat calories infused), either receive empiric treatment for SIBO or undergo a hydrogen breath test.

Abdominal Pain. Persistent abdominal pain is not uncommon with severe pancreatitis, and many patients continue to experience abdominal pain after jejunal EN is initiated. The presence of abdominal pain should not serve as a contradiction to enteral feeding as long as the feeding does not exacerbate pain or other clinical parameters that might indicate worsening of pancreatitis (respiratory status, CT scan, etc.). If patients experience an acute increase in abdominal pain after initiation of EN (that is not relieved with venting of gastric secretions), our practice is to temporarily hold EN infusion and verify that the feeding ports are located beyond the LOT. Some patients have required placement of a feeding tube even more distal in the jejunum before they tolerated jejunal EN. In our cohort of 127 patients, only one patient remained intolerant to jejunal EN requiring transition to PN (13).

Nausea. Patients with severe acute pancreatitis frequently have delayed gastric motility. The inflammatory responses to illness, as well as the use of narcotic pain medication are both contributing factors. In addition, duodenal compression from an inflamed pancreas further contributes to a functional gastric outlet obstruction, hence, a significant percentage of patients with complicated pancreatitis exhibit a partial or complete functional gastric outlet obstruction (13). See Figure 1 for a depiction of the relationship between the pancreas and the compressible duodenum. Patients that exhibit symptoms of nausea with jejunal EN may benefit from "venting" off endogenous gastric secretions via nasogastric suction or through the g-port of a PEG-J (see section below on reinfusing). Our experience has been that, although transient, nausea during initial EN is common. In most patients it resolves within several days to a couple weeks as pancreatic swelling decreases and the body adapts to enteric nutrient infusion, while jejunal EN continues.

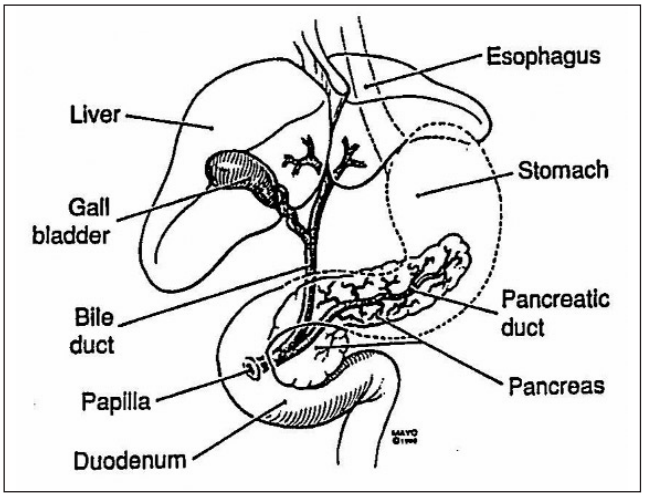

Figure 1. Biliary system: normal pancreas anatomy (55). By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

Theoretically, administration of a PPI may be helpful to decrease nausea related to delayed emptying of gastric secretions by virtue of decreasing the sheer volume of gastric secretions produced. In addition, some have suggested that raising gastric pH may be of benefit because low gastric pH has a stimulatory effect on pancreatic secretion. However, there is no data that administration of PPIs alters the course of pancreatitis. Other measures to decrease the stimulation of pancreatic secretions by gastric acid, such as nasogastric suction or administration of H_2 antagonists have not been shown to improve outcomes of acute pancreatitis (49–52). In addition, it is worth noting that PPIs are known to increase gastrin secretion, which can also stimulate pancreatic exocrine secretions.

Reinfusion of Vented Gastric Secretions. Some patients with pancreatitis may develop gastric outlet

obstruction (GOO) as a result of duodenal compression from inflammation of the pancreas and surrounding tissue and have large amounts of gastric secretions necessitating gastric venting. In patients with both gastric and jejunal access (PEG-J, NG and NJ, or NG-J), this can be accomplished by venting gastric secretions from the gastric access port into a gravity bag. In our institution, if gastric output exceeds 500 mL/24 hours, secretions are reinfused jejunally to prevent dehydration and metabolic alkalosis. Secretions can be reinfused via the jejunal feeding port by gravity infusion using a gravity feeding bag or bolus method using a 60 mL syringe. Although acid secretion or inhibition does not influence the course of pancreatitis, it is now standard therapy at UVAHS to prescribe a liquid PPI administered jejunally for patients with pancreatitis after a PEG-J is placed. The rationale for using liquid PPI as adjunctive therapy includes the following:

- To reduce the total volume of gastric secretions in patients who require gastric venting
- To increase the pH of gastric secretions (>6) reinfused into the jejunum to mimic the physiologic pH that the jejunum is accustomed to
- To reduce the likelihood of gastritis or ulceration while under physiological stress and without oral intake

A gastric pH is checked two-to-three days after PEG-J placement to ensure adequate acid suppression therapy. See Table 4 for the standard protocol for patients with pancreatitis who receive a PEG-J at UVAHS.

Table 4
Post PEG-J placement protocol for patients with pancreatitis at the University of Virginia Health System

- NPO except 8 ounces of ice chips per 24-hour period
- Start 30 mg PPI via J-port q pm day of PEG-J placement
- Check gastric pH (via G-port) Day #2 after PEG-J placed
- If <6, increase PPI to BID dosing, recheck gastric pH in 2 days and reevaluate
- Tube feedings to start via the J-port upon return to the unit per Nutrition Support Team recommendations
- G-port to gravity for the first 24 hours after PEG-J placed; record output q shift during this time
  - If <500 mL drainage, clamp G-port and monitor for nausea/vomiting
  - If >500 mL/hours ***AND*** gastric pH ≥6, reinfuse gastric secretions via j-port
- Check quantitative fecal fat if pancreatic insufficiency suspected and adequate volume of enteral nutrition (fat) has been infused
- Once tube feedings start, accucheks at 1800, 2200, 0600 × 2 nights, then stop if adequately controlled
- Pill crusher/splitter to unit now (or advise patient to obtain a mortar and pestle—an inexpensive way to do this at home)
- Instruct patient for medication delivery via j-tube

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RESUMING ORAL INTAKE

The resumption of oral intake following a bout of pancreatitis is largely an empiric decision. Most clinicians take into consideration the presence or absence of pain, the severity of pancreatitis, biochemical markers and radiologic improvement based on CT scan (13). Well-nourished patients with mild pancreatitis can be kept NPO for five-to-seven days until the pain disappears, but patients with severe pancreatitis should receive early EN, that continues until they are pain free (4). In both mild and acute pancreatitis, a trial of oral feeding should be initiated when the patient is pain free (off narcotics) and continued as long as the patient remains pain free (4). To date, no studies have assessed the criteria to be used in the decision to resume oral feeding; however, a few studies have identified factors that increase the risk of pain relapse after initiating oral intake. These include a higher Balthazar's CT score, a longer period of initial pain (53,54) and/or a serum lipase three times above normal (53) the day before resumption of oral intake. In both studies, refeeding pain resulted in a longer length of hospital stay and the longer length of stay occurred after the first attempt of oral nutrition.

Although serum amylase and lipase levels are often used during diet advancement, both lack sensitivity and specificity as a marker of worsening pancreatitis (55). Also, in chronic pancreatitis, it is possible to have such extensive damage to the pancreas that even in an acute event ("acute on chronic"), elevated serum levels of amylase and lipase are not possible.

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No published studies exist that demonstrate the best diet to begin after a bout of pancreatitis, however, conventional wisdom is to start a diet low in fat (<30% of total energy intake) (4). Our practice is to initiate a healthy diet without fried or greasy foods, but we do not restrict fat per sé.

OTHER NUTRITIONAL CONSIDERATIONS

Long Term

Once the acute pancreatic insult is resolved, a certain percentage of patients will go on to develop varying degrees of chronic pancreatic changes with some component of fat malabsorption. Ongoing monitoring of these patients for fat-soluble vitamin deficiency is prudent, even for those who are on pancreatic enzyme supplementation. Osteoporosis is well documented in patients with chronic pancreatitis (56–58). Given the average age of onset of pancreatitis, and the length of time between transition from acute and chronic changes, much bone loss can occur. Patients with known chronic pancreatitis should have a DEXA scan at baseline once the acute insult has resolved.

Because it is unclear which patients are developing early changes of chronic pancreatitis, we have begun to obtain baseline vitamin A and 25-OH vitamin D levels in patients admitted to UVAHS with a diagnosis of pancreatitis. A remarkable number of patients may actually have significantly low serum levels of fat-soluble vitamins. Over a 3 month period (July–Sept 2007) at our institution, 15 of 21 (71%) patients admitted or seen in GI nutrition clinic with a diagnosis of pancreatitis were found to have vitamin A levels ranging from 56-345 and 18 of 21 (86%) patients had 25-OH vitamin D levels ranging from 2.6 to 23.3 ng/mL. The etiology and clinical implications are unclear, but clearly this is an area worthy of further investigation.

Even though the low levels of these vitamins may have preceded the insult that brought the patient into the hospital, by checking baseline levels we are able to identify the deficiencies and manage them appropriately; it would therefore behoove clinicians to identify those at risk early on and provide early intervention.

CONCLUSION

The best available data demonstrate that jejunal EN is associated with improved outcomes compared to PN in patients with pancreatitis. However, successful implementation of enteral feedings in pancreatitis requires a dedicated effort to overcome the real and perceived barriers of enteral access and tolerance.

Research continues into the optimal route and type of feeding to achieve the best clinical outcomes in the most cost-effective manner. Greater attention should be placed on the potential role of enteral nutrition to modify long-term outcomes and recurrences in this population. Pancreatitis can create long-term health concerns in many patients, and identifying and treating nutrition deficiencies may improve the health risks, quality of life and economic burden created by this disease. ■

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