Patient Profile

A 75 year old white male was admitted to Baptist Medical Center on January 22, 2011 with a primary diagnosis of small bowel obstruction. The patient was transferred from a long term acute care facility with complaints of vomiting with subjective nausea and abdominal pain, per the patient's caregiver, as the patient is non-communicative. The patient is married and both he and his spouse usually live with his daughter in her 10,000 square foot home. The patient is bedbound and the large dining room was converted into a suite containing all of his medical supplies including a hospital bed, air-mattress, oxygen, nebulizer, enteral feeds and colostomy supplies. The patient is cared for by his daughter and two home health nurses who alternate caring for the patient during the weekdays. A doctor also visits the patient at home weekly.

According to the patient's home health nurse he had been fully ambulatory one year prior to this admission. At that time he received a skin tear to his coccyx that escalated into a sacral ulcer for which he continues treatment. There has been a parallel decrease in the patient's overall health; he has a history of type II diabetes, gastroparesis, past cerebrovascular accident (CVA), hypertension, congestive heart failure, and Alzheimer's disease with severe dementia, debility, and incontinence. Past surgical history includes PEG placement, new skin graft with wound debridement one week prior to current admission, and a recent colostomy on January 6^{th,} 2011.

The patient has a history of dysphagia secondary to his CVA is at high aspiration risk, and receives feedings through a PEG tube with a jejunal extension. The patient had a secondary diagnosis of malfunctioning PEG/J tube. He has had a prolonged, documented history of obstructions to the tube, leakage, migration, and high residuals. According to his daughter, he has had the PEG/J tube replaced 16 times in the past year.

Disease Background

Bowel obstruction is the term used for any condition that prevents the normal, forward flow of intestinal contents (1)(2). A mechanical obstruction occurs when something is visibly interfering with passage; this can include tumors, scar tissue from a previous surgery, hernias, concretions of fruit and vegetable fibers known as phytobezoars, or a twisting of the intestine upon itself known as a volvulus (1)(2)(3). A functional obstruction occurs when the muscles of the bowel are not working correctly, either completely paralyzed or simply inactive enough to prevent the normal passage of chyme; these obstructions are referred to as pseudo-obstructions or paralytic ileus(4). Myopathic causes of paralytic

ileus involve the musculature, for example, muscular dystrophy, lupus, or connective tissue disorders such as scleroderma (4). Neurogenic causes involve changes in the nerves in the gut, reasons include spinal cord injury, Parkinson's disease, diabetes, drug induced dismotility, and immune responses involved with paraneoplastic syndrome and certain viruses(4). Pseudo obstruction can be either acute or chronic (4).

Both forms of obstruction can be either partial or complete. The symptoms differ depending on the site and degree of obstruction. Proximal obstructions are more often accompanied by abdominal pain, nausea, and vomiting without marked distension (1)(2). Distal obstructions cause greater distension and abdominal pain with less vomiting (1)(2). If the obstruction is complete then constipation will occur, whereas a partial obstruction may result in diarrhea or constipation (1)(2). Bowel sounds can be either increased or decreased (1).

As intestinal secretions, chyme, and air build up around the obstruction the bowel becomes dilated. It is this dilation, usually visible on CT or X-ray images for diagnosis, which leads to the very serious complications of obstruction (5). This dilation or distention stimulates increased intestinal secretions and these increased secretions further distend the bowel, causing segments of the bowel proximal to the ileus to increase peristaltic activity in an attempt overcome the blockage (5). This is a vicious cycle and the spastic, amplified contractions are the cause of abdominal pain (5). Increased luminal pressure can cause edema leading to dehydration, ischemia and necrosis of tissue, and increased risk of perforation (5). Vomiting, gastric decompression, and loss of absorptive capability worsen dehydration and electrolyte abnormalities, cause metabolic acidosis, and can even lead to starvation ketosis(1)(4)(5). In addition, stasis of fecal matter can cause overgrowth of bacteria and translocation across the gut barrier (1)(4).

Treatment for obstruction focuses on correcting any complications caused by the obstruction and, if necessary, removing the cause of obstruction (1). Unless radiology confirms the presence of a high grade obstruction requiring immediate surgery, such as a hernial strangulation, then the first 24-48 hours involve conservative management while waiting to see if the obstruction resolves on its own(1). Conservative management involves replacing fluid and electrolytes and using a catheter to gauge the adequacy of resuscitation, gastric decompression, and antibiotics, especially with leukocytosis and fever (1). Management of pseudo obstruction involves the addition of prokinetic agents including reglan and erythromycin (4). Another pharmacologic therapy is the use of Octreotide; it interrupts the cycle of distension-secretion-contraction by inhibiting bilious secretions and decreasing intestinal contractions

by decreasing acetylcholine release (4)(5). This helps to relieve symptoms of colicky contraction and vomiting of intestinal secretions, stimulating an artificial bowel silence while the ileus resolves (5).

Patients are to remain without oral or enteral feedings for the first 24 to 48 hours of treatment, as stimulation at this time can aggravate the obstruction or cause ischemia(1). If the obstruction does not clear on its own then the degree of diet modification will depend on the location and degree of obstruction. Patients with only a partial obstruction will likely tolerate an oral or enteral diet, whereas a full obstruction will require parenteral nutrition (2)(4). A liquid or pureed diet, in smaller more frequent meals, may be tolerated in patients with gastric involvement; however severe gastroparesis will require jejunal feedings (2)(4). Patients without gastric involvement may be able to tolerate a low-fat, low-fiber diet with small frequent meals (4). The low fiber diet helps to prevent formation of gas , cramps, and phytobezoars, which is more likely to occur during partial obstruction or paralytic ileus because fibrous foods may not be small enough to pass though narrowed segments of the intestine(2)(4).

Because acute obstruction either resolves on its own, or is treated surgically in a small time frame, malnutrition is not as much of a concern. However, preventing or correcting malnutrition is the focus of care for chronic intestinal pseudo-obstruction (CIPO) (4). Patients with CIPO often have a prolonged avoidance of food in order to minimize symptoms, causing malnutrition (4). Case studies have shown that nighttime enteral feedings may be necessary while the patient's oral intake improves (7). Increasing oral intake may require therapy and counseling to overcome fears (7). As with acute cases, a low-fat, low-residue diet, with smaller, more frequent meals is better tolerated.

Dysfunction of PEG/J tubes appears to be a common complication and continued concern about aspiration risk in these patients appears justified by at least one study (6). Wolfsen and colleagues defined dysfunction as peritube leakage, fracture, plugging, or migration (6). In a study with 191 patients followed for a mean of 275 days after placement of either a PEG or PEJ, 36% of patients experienced tube dysfunction(6). 14 percent experienced plugging, 9% experienced leakage, 8% experienced migration, and 4% had a fracture of the tube (6). Replacement or removal of the tube occurred in 80% of the patients who had complications (6). The average replacement in PEG patients occurred at 4 months from placement and 2 months after placement for PEJ (6). Within 30 days of placement 5% of PEG patients and 17% of PEJ patients aspirated (6). Authors noted that pre-existing conditions warranting the use of PEJ over PEG feedings, such as gastroparesis, gastro-esophageal reflux, and recurrent aspiration, was likely the cause of increased aspiration in these patients compared to gastrically fed patients(6). However the American Society for Parenteral and Enteral Nutrition (ASPEN), guidelines cite

grade C evidence in two studies for reducing risk of aspiration with small-bowel feedings(8). Of particular interest to this case study, ASPEN guidelines cite grade E evidence for not using blue food coloring as a surrogate marker of aspiration(8). This is due to FDA issuing a mandate against its use for this purpose as it has been associated with mitochondrial toxicity and patient death(8).

Current Admission: Tests and Procedures

Upon admission the patient underwent a Computed Tomography (CT) of the abdomen to investigate a possible small bowel obstruction. The CAT scan did not identify any dilation or obstruction of the bowel but it did show distended loops of bowel. This was noted in the doctor's initial transcription to be a questionable obstruction or ileus. This same CT scan also revealed erosion of the sacrum at the site of the ulcer, suggested by the radiologist to be evidence of osteomyelitis.

Four days after admission the patient underwent replacement of his PEG tube with jejunal extension because the current tube was found to have a malfunctioning seal and leaking. An X-ray of the abdomen was performed after the procedure to confirm placement of the tube. The radiologist also noted mild diffuse dilatation of the small bowel at this time. On February 28th, 2 days after the PEG/J tube was replaced, another X-ray of the abdomen was completed; this time showing no evidence of bowel obstruction.

On the January 27th the patient's daughter began requesting a surgical jejunostomy placement for enteral feedings and complained of difficulty in checking residuals through the new PEG/J tube. Nursing reported the highest residuals since the new PEG/J tube placement as 60ml. However, it was noted that an inadequate seal made it difficult to check the residuals and the nurse noted that she believed actual residuals to be higher. After an appropriate adapter for checking residuals was sourced, residuals as high as 180ml were recorded. The gastroenterologist noted that the output appeared bilious.

To determine the actual source of residuals, and whether the patient was actually refluxing up the formula into the stomach, the gastroenterologist ordered methylene blue dye to be added to the formula. The patient was also placed on Nexium at this time. After addition of the blue dye it was noted that the gastric residuals were not blue, indicating that the formula was not the cause. The residuals normalized for the following two days. However, on the third day after treatment with Nexium, February 4th, residuals increased again to as high as 130ml. At this time Octreotide was also ordered. The following day residuals began decreasing to 30ml and by the patient's discharge on February 7th there

was consistently less than 10ml of residuals. Before the patient was discharged, a final x-ray of the abdomen on February 5th showed no signs of obstruction or ileus.

Current Admission: Medications

Because this patient was at such high risk for Aspiration, It was very important to maintain a low residual volume and prevent reflux of stomach contents. As Discussed in the patient's treatment, he was placed on both Nexium and Octreotide. Nexium is a Proton Pump inhibitor which decreases gastric acid secretion. Because Nexium also decreases gastric pH, it has the side-effect of causing decreased absorption of Iron, B12, and Calcium (9).

Octreotide is a mimic of the natural hormone somatostatin. It is most commonly prescribed for acromegaly, as it is an inhibitor of growth hormone, and to control diarrhea caused by carcinoid tumors(8). It actually suppresses the release of a wide range of hormones including many in the gastrointestinal tract; thereby decreasing exocrine secretions from the pancreas and contraction of the gallbladder, these effects were the reason for using Octreotide in this patient(5). There are many negative side effects when using such a wide reaching drug. Gastrointestinal side effects include decreased motility and decreased absorption of fat and fat soluble vitamins; this can lead to diarrhea, steatorrhea, abdominal pain, bloating, nausea and vomiting. It also inhibits glucagon and insulin, causing periods of both hypoglycemia, and hyperglycemia especially in diabetics (9). It can also cause hypothyroidism, cardiac arrhythmias, and buildup of biliary sludge causing gallstones, pancreatitis, and cholecystitis (8).

Other Medications of nutritional interest include Insulin, Reglan, Digoxin, daily multivitamin and 500mg of Vitamin C twice daily. Reglan is used as a treatment for the patient's diabetic gastroparesis. It increases gastric emptying, also helping to prevent gastroesophageal reflux. Digoxin is a treatment for Congestive heart failure and interacts with certain electrolytes including potassium, Calcium, and Magnesium (9). A high potassium, low sodium diet with adequate magnesium and calcium is recommended (9). However, calcium and Vitamin D supplements should only be used with caution as they increase the drug's affects and the potential for arrhythmias (9). Hypokalemia and hypomagnesemia may result (9). Anorexia, weight loss, diarrhea, nausea and vomiting are all common side effects (9). The daily multivitamin and Vitamin C are used to promote wound healing.

Nutrition Care Process

Upon admission to the hospital, dietary was consulted to evaluate the patient's tube feeding. The patient is 6 feet 2 inches tall and the admitting weight was 90.7 kg. The patient is 105% of ideal body weight, putting the patient at a borderline overweight BMI of 25.6. The patient's needs were estimated to be 24-28kcal per kilogram and 1.1-1.5g/kg of protein.

The patient's diet, per the long-term acute care (LTAC) facility records was 2CalHN at 55ml/hr which would provide 30kcal/kg and 2.2g of protein per kilogram of actual body weight. Records also showed that the patient was having difficulty tolerating the tube feeding and that the PEG/J was clogging at the LTAC. The current diet order was one can Glucerna, bolus, with one scoop of Promod every 4 hours. The current order would provide 22kcal/kg and 1.3g/kg of protein. However, according to the patients nurse, he had not yet received any bolus or continuous feeds since being admitted to the hospital. The nutrition diagnosis at this time was increased nutrient needs related to wound healing as evidenced by albumin of 2.0. The recommendations were to change to Glucerna 1.5 bolus q 4 hours and 1 packet of Prostat daily with 200ml free water flushes QID. These recommendations would provide 24kcal/kg and 1.5g/kg of protein. The goals at this time were to preserve lean body mass, promote wound healing, and for albumin to trend toward normal.

Four days after the initial assessment a follow up visit occurred. The patient had undergone replacement of his PEG/J tube and was now on Glucerna 1.0 running at 20ml per hour. The ordered rate in the chart was 30ml/hr with a goal of increasing to 70ml/hr. However, according to the patient's nurse, his daughter was decreasing the rate due to high residuals. The RD spoke with the daughter and explained that the patient was not meeting his needs at the current tube feeding rate. The recommendations at this time remained as stated on the initial assessment. The diagnostic statement at this time was inadequate energy intake related to daughter decreasing rate as evidenced by enteral intake.

Two days later a nutrition consult was requested for a continuous feeding rate. At this time the feeding was changed to Glucerna 1.5 and running at 20ml/hr despite current orders to be at 40ml/hr and increase to a goal of 50ml/hr. The daughter again cited concern for high residuals as the reason she continued to decrease the formula delivery rate. As the patient had multiple problems tolerating tube feedings in the past, a trial of an elemental formula was suggested to the daughter. She agreed. The

recommendations at this time were to change the tube feeding to Vivonex at 50ml/hr and increase to a goal of 75ml/hr with 30ml of Prostat TID. This would provide 22kcal/kg and 1.5g/kg of protein.

The following day a follow-up was necessary. The doctor ordered Vivonex as suggested, however the hospital was out of stock. At this time recommendations were made to continue to use Glucerna 1.5 for continuous feeding until Vivonex arrived. The dietitian also advised the nurse at this time not to check residuals from the J-tube and to ensure that feedings remained continuous rather than bolused.

Three days later an informal follow-up, for patient monitoring and changes to the formula occurred. At this time the patient had been on Vivonex for over 24 hours. The tube feeding was dyed with methylene blue and running at 40ml/hr. According to the doctor's notes, gastric residuals were not running blue, indicating that they did not contain formula and were likely gastric or intestinal secretions. The patient was placed on Nexium in order to decrease gastric output. The Vivonex was a powdered mix, requiring daily mixing, inherently less sanitary than a sealed and ready to hang formula. Because ready to hang Perative, a semi-elemental formula, was now in stock, the Vivonex was replaced with Perative and recommendations were to continue increasing to the goal rate of 75ml/hr. This would provide 28kcal/kg and 1.8g/kg of protein.

Two days later the scheduled follow-up occurred. The patient was on Perative at 50ml/hr with plans to increase by 10ml every day to a goal of 75ml/hr. The patient was meeting about 67 percent of his caloric needs and 90% of originally estimated protein needs at this time due to the use of Prostat. The patient was tolerating the formula well with less than 10ml of residuals according to his nurse. The decrease of gastric residuals following treatment with Nexium, coupled with the fact that the residuals were not blue, indicated that the previous formula was not likely the cause of the high residuals. This was discussed with the daughter along with recommendations to change back to Glucerna 1.5. The daughter strongly believed that Perative was better tolerated by her father and had already arranged to have it supplied through home health. The nutrition diagnosis was inadequate energy intake related to delayed progression of TF rate as evidenced by enteral intake. The recommendations made were to continue increasing Perative to a goal of 75ml/hr as tolerated. On February 7th, 2011 the patient was discharged home with his daughter; tolerating Perative at 70ml/hr with residuals less than 10ml.

Summary and Conclusion

There is a great deal of variation between all of the issues that share the diagnosis of bowel obstruction. The treatment is very dependent on the individual's history and the location and extent of obstruction. It is important that the Dietitian take the time to carefully review the notes, patient history, and if possible remain in contact with the doctor as the full diagnosis develops.

This patient's struggles with PEG/J dysfunction requiring frequent replacement were not uncommon. These are lifelong issues that anyone with a PEG or PEJ likely deals with. To my amazement the daughter's concern for aspiration, even with jejunal feedings, was not unfounded. Many experts believe that the real risk is aspiration of throat contents and saliva, rather than aspiration of formula (2). Checking residuals does not protect the patient from aspiration and oftentimes residuals contain more gastric fluids and secretions than formula (2). Standard precautions include elevating the head of the bed 30-45 degrees, continuous subglottic suctioning, using continuous rather than bolus feeding, using prokinetic agents, and feeding past the pylorus (2) (8). There are many sources of aspiration besides enteral formula and medication can be just as important as standard aspiration precautions. Residuals should not be checked out of the jejunal portion of the tube; because the jejunal lumen is small the mucosa can be suctioned into the tube, injuring the patient and clogging the tube. Indicators besides residuals that are a good indication of tube feeding tolerance include monitoring for abdominal pain, distension, and stool output and consistency (2) (8).

The most interesting aspects of this case study were quality of life issues and the daughter's involvement in the patient's care. The daughter was self- educated, providing her own care while the patient was in the hospital, despite lack of a medical background. This made it difficult for the entire multidisciplinary team to ensure the best care for the patient. A very confident and assertive dietitian is needed to both address concerns and educate an equally assertive family member such as this. I believe that this confidence comes from knowledge and experience and this is why I chose this case study for myself, so that I may have that confidence in the future. It is the quality of life issue that I know I will struggle with for some time. Providing such aggressive care to a patient with such a limited existence is a matter of ethics. However it is not the dietitian's role to make these decisions; rather the dietitian needs to provide expert knowledge, patience and reassurance given the caregiver's emotional attachment and wishes for the patient

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