Nutritional Support in the Management of Surgical Complications Post-Attempted Laparoscopy for SBO: A Case Study

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Abstract

A small bowel obstruction (SBO) is characterized by a partial or complete blockage of the intestines. It usually presents with abdominal cramps and pain, bloating, nausea, and vomiting and can be caused by adhesions from prior surgeries, hernias, tumors, medications, or strictures from an inflamed intestine. The typical medical nutrition therapy for a small bowel obstruction includes intravenous fluids and bowel rest, and in less extreme cases, a liquid diet or a low fiber diet. This report reviews a case of an eighty three year old patient, GC, who came into Cedars Sinai Medical Center with a small bowel obstruction and faced different complications throughout her hospital stay, namely recurrent peritonitis and acute kidney injury.

Upon initial assessment with the registered dietitian (RD), the patient was diagnosed with unspecified severe malnutrition, which prompted the need for immediate nutrition. Total parenteral nutrition (TPN) was quickly initiated, indicated by previous SBO, recurrent peritonitis, a negative pressure dressing on the patient's abdomen, and frequent trips to the operating room, which would halt enteral feeds if started. Throughout the patient's hospital course, her TPN prescription remained fairly consistent, with the RD placing an emphasis on recommending custom values for protein and zinc at each visit to promote healing of a surgical wound and address uptrending blood urea nitrogen (BUN) values.

This report discusses the actions taken and recommendations given during GC's admission, and reviews some current literature pertinent to the patient's case. The purpose of this report is to discuss GC's case, highlight the rationale for decisions pertaining to nutrition, and focus on potential optimizations for future patients.

Keywords: small bowel obstruction, acute kidney injury, SMOFlipid, blood urea nitrogen, total parenteral nutrition, peritonitis, negative pressure dressing

Introduction

A small bowel obstruction occurs when the small intestine becomes partially or completely blocked. It can be caused by a multitude of factors, including adhesions from prior surgeries, hernias, colon cancer, certain medications, or strictures from an inflamed intestine in conditions like Crohn's disease or diverticulitis.[1] SBO can lead to severe complications such as bowel ischemia, perforation, and sepsis if left untreated. Therefore, prompt diagnosis and treatment are essential. This clinical case study delves deeply into a case of a patient who presented with symptoms of SBO and experienced surgical complications following a laparoscopy turned laparotomy in the operating room.

GC was an eighty three year old female with a significant past medical history of abdominal surgeries who presented to Cedars Sinai Medical Center with a small bowel obstruction. The patient's hospital course was a lengthy one, returning to the operating room four times within GC's stay. The patient's recovery was hindered by recurrent peritonitis. Upon admission, the patient went in for an exploratory laparoscopy which led to a laparotomy due to significant adhesions from previous abdominal surgeries.

The purpose of this clinical case study report is to provide a detailed analysis of the patient's hospital course, including the decisions made by the care team regarding the patient's care. The report will also review the relevant literature on the selected conditions that the patient experienced during GC's hospitalization. Additionally, this report aims to contribute to the existing literature on the nutritional approach in critically ill patients, serving as a valuable reference for healthcare professionals.

Background and Review of Medical Literature

Small bowel obstruction (SBO) is a complete or partial blockage of the small bowel, usually resulting from fibrous bands of tissue (adhesions) that form after abdominal surgery, hernias, tumors, certain medications, or from inflammation.[1] Usual symptoms include abdominal cramping, bloating, vomiting, nausea, dehydration, malaise, lack of appetite, severe constipation, and an inability to pass gas or stool.[1] SBO is commonly identified using a physical exam, abdominal x-ray, CT scan, ultrasound, and barium enemas. Some lab values to consider include a basic metabolic panel (BMP) to assess electrolyte balance in patients with vomiting, diarrhea, or fluid shifts and a complete blood count (CBC) which may indicate infection or inflammation.

In terms of nutritional management, the initial target is to provide intravenous fluids and bowel rest. Sometimes a nasogastric tube is required to decompress the small bowel. Antiemetics might also be considered in relieving nausea and vomiting. Once the obstruction has resolved or been surgically corrected (through intestinal obstruction repair), the patients are started on a gradual oral diet, usually starting with clear liquids and advancing as tolerated, while frequently being assessed for tolerance of the current diet. The usual medical nutrition therapy includes nothing by mouth (NPO) as SBO persists, small frequent meals, a liquid diet while solids are not tolerated, and a low fiber diet. If the obstruction is chronic or recurrent, patients may benefit from enteral or parenteral nutrition support. Per ASPEN guidelines, "Total Parenteral Nutrition should be considered for patients with SBO who are unable to tolerate enteral nutrition, have prolonged ileus or high-output fistulas, or are expected to have a prolonged period of bowel rest."[2] Management of SBO should be tailored to the individual patient's needs and requires a multi-disciplinary approach.

Another focus of this case study includes nutrition and the use of a negative pressure dressing, an AbThera (a flexible perforated silicone sheet), to manage abdominal wounds. Indications for negative pressure wound therapy (NPWT) include chronic wounds, acute wounds (ex. surgical wounds), complex wounds, wounds with high infection risk, and wounds with high risk of dehiscence.[3] Depending on the patient's condition, tube feeding might be indicated. If this is the case, positioning of the tube should not interfere with the NPWT and should bypass the abdominal NPWT and feed into a different part of the abdomen. The WSES guidelines for the open abdomen in trauma and non-trauma patients recommend "immediate and adequate nutrition support" since patients with an open abdomen are hypermetabolic. Additionally, early enteral nutrition (EN) is recommended to start as soon as possible in a functioning gastrointestinal (GI) tract and should be delayed in patients with an intestinal tract in discontinuity, situations of high output fistulas, and with signs of intestinal obstruction.[4] If EN is contraindicated, "parenteral nutrition (PN) should be started as soon as possible."[4] Once the GI tract is viable again, it is recommended to start EN. A meta-analysis studying outcomes of EN versus PN suggests an overall reduction in morbidity and mortality in critically ill patients with EN.[5] Notably, the EN arm had fewer abdominal complications, specifically in the formation of fistule. On the other hand, early initiation of EN was associated with "increased time to abdominal closure, higher incidence of pneumonia and longer hospital stay."[5]

The third focus of this clinical case study is nutrition for acute kidney injury. Acute kidney injury (AKI) is characterized by an accumulation of waste products and fluids in the body due to sudden dysfunction of the kidneys. The two types of AKI pertinent to this case study are oliguric and non-oliguric AKI. Oliguric AKI is usually characterized by typically less than 400 ml urine output per day and patients may require dialysis.[6] Non-oliguric AKI usually presents with typically more than 400 ml urine output per day, less severe kidney damage, and these patients may not require dialysis. A common nutrition practice in either type of AKI is to restrict protein, sodium, and potassium intake and increase fluid intake as needed.[7] Schwartz et al. performed a literature review of four studies which gave insights into the ideal protein dosage for someone with an AKI. One review took place in the pediatric population and will not be further described in this case study. The Schwartz et al. case report highlights the need for clinicians to be aware of the potential for protein deficiency to exacerbate or mask kidney injury in malnourished patients. The first study that was reviewed was a prospective study measuring energy and protein needs using indirect calorimetry and PCR. "Results of the study showed a mean normalized PCR (nPCR) of 1.3 g/kg/d protein for adults with AKI not on renal replacement therapy (RRT)."[8] The second study, a randomized controlled trial, randomized patients to receive either standard of care ("according to standard hospital practices") or high-dose amino acids (2g/kg ideal body weight (IBW) to a maximum of 100g/d). Participants in the high-dose amino acid group had significantly higher urine output and GFR values than those in the standard of care group. The third study, a small randomized controlled trial, randomized patients into groups receiving either 75 g/d or 150 g/d of intravenous amino acids for three days. Serum urea nitrogen increased significantly from baseline in the moderate-dose amino group (75g/d), while the high-dose amino acid group required less diuretic to achieve goal urine output and had greater cumulative nitrogen balance.[8] The Schwartz et al. case report concluded that increased protein in critically ill patients may be beneficial to kidney function for patients with an AKI not on RRT. Current ASPEN guidelines are 1.2-2 g/kg usual body weight (UBW) or IBW for obese and critically ill patients.[8]

In providing nutrition to critically ill patients, there are cases in which parenteral nutrition is indicated over enteral nutrition in patients who require nutrition support. The current recommendation is to feed enterally if the GI tract is viable. Some examples of when PN would be indicated include patients with severe pancreatitis, bowel obstruction, or short bowel syndrome. PN may also be used when the patient has a high metabolic demand or is unable to tolerate EN. TPN provides nutrients straight into the bloodstream through a central line. TPN typically consists of carbohydrates, proteins, and fats, with potential to add electrolytes, vitamins, and trace elements. The fat in TPN is usually administered separately and comes in different forms. Some are soybean-oil based, some fish-oil based, and some provide a combination of different types of oils. SMOFlipid is a combination lipid emulsion that contains soybean oil, medium-chain triglycerides (MCTs), olive oil, and fish oil.[9] The omega-3 to omega-6 fatty acid ratio in SMOF is 1:2. Omega-3 fatty acids have been shown to have anti-inflammatory properties. Compared to other lipid infusions, a short-term use of SMOFlipid showed significant decreases in liver function test labs (LFT), total bilirubin, triglycerides, C-Reactive protein, and hospital length of stay.[9] Similarly, 2 g/d of EPA and DHA (types of omega-3 fatty acids) showed an anti-inflammatory effect on cytokines, reactive oxygen species (ROS) production, and NF-KB activation. Intralipids, another type of lipid emulsion containing soybean-oil, may promote inflammation and affect immune function due to their high omega-6 to omega-3 fatty acid ratio.[9] The usual dose of Smoflipid for adults is 1 to 2 grams per kilogram of body weight per day, whereas the recommended dose for Intralipids is no greater than 1g/kg/d body weight.

Patient Hospital Course

GC was admitted to Cedars Sinai Medical Center on March 17, 2023 for a laparoscopic lysis of adhesions. While in the operating room, the scope could only view so far into the intestine due to adhesions from prior surgeries, leading the medical team to perform a laparotomy. The patient's past medical history included an esophageal perforation and repair (2011), hiatal hernia repair (2012), Nissen fundoplication (2012), bowel resections and extensive lysis of adhesions (2012), pandiverticulosis and rectal polyp removal post-colonoscopy (2016), pacemaker insertion (2021), and COVID-19 related dysphagia (2023). GC also had a history of type 2 diabetes mellitus, gastrointestinal reflux disease (GERD), hyperlipidemia, chronic obstructive pulmonary disease (COPD), and iron deficiency anemia. At home, she was taking calcium citrate-vitamin D3, a multivitamin, protonix, metformin, rosuvastatin, propranolol, metoprolol succinate, eliquis, levothyroxine, and amlodipine. GC's primary care provider (PCP), a Cedar's doctor, conducted frequent telehealth visits with the patient. Per GC's conversation with her PCP, she reported a lack of appetite, that it has been "getting harder for her to eat," and a weight loss of thirty pounds in three months. GC also experienced increasing nausea since January 2023 and was drinking mostly liquid soups starting February 2023 prior to admission.

By the time GC was first seen by a dietitian, the patient was status post (s/p) open lysis of adhesions with multiple enterotomies, a small bowel resection, ileocecectomy with anastomosis, and ventral hernia repair. Upon the initial visit with the RD on March 22, GC was intubated, on pressors (norepinephrine), and sedation (propofol at 2.4mL/hr, fentanyl). The medical team also treated the patient with lasix, a lactated ringer's bolus, iron sucrose, insulin, and antibiotics. GC also had a nasogastric tube in place for suction and a negative pressure dressing over her abdomen. Visual moderate temporal wasting and potential moderate wasting of the orbital fat pads noted. With the visual NFPE and the knowledge of the prior conversations with her PCP, GC was diagnosed with unspecified severe protein-calorie malnutrition evidenced by NPO status for 5 days and severe body weight loss. The PES statement for this patient was: malnutrition related to previous SBO as evidenced by NPO for 5 days (with reported difficulty eating prior to hospital admission) and 16% body weight loss in 2.5 months. A stated weight from an encounter with the PCP on March 8th was used to estimate nutrient needs. GC's weights from prior encounters can be found in Figure 1. GC's estimated nutrient needs were based on a 162 pound (73.6 kilogram) body weight. The Penn State equation was used to estimate calorie needs since the patient was on ventilation. GC's estimated calorie needs were 1641 calories (kcal), using an average Ve (minute ventilation recorded from ventilator in L per minute) of 11.73 and a 38 degree Celsius maximum temperature in the twenty four hours prior. Indirect calorimetry was requested by the dietitian, but never performed. The patient's protein needs were estimated at 99-109 grams of protein (1.2 g/kg + 15-30 g/L vac output per day).[11] At the time, the average vac output was 700mL/day. Elevated BUN was taken into consideration while estimating protein needs and instructions to the team included monitoring renal function. Fluid needs were estimated to be 25 mL/kg/day or per the ICU team. At the initial visit, TPN was determined to be the most appropriate route of nutrition due to the previous SBO, current open abdomen with negative pressure dressing, an NGT for suction, an immediate need for meeting nutrition requirements, and frequent trips to the operating room that could halt enteral feeding at nutrition goal rate. The recommended TPN formula to the MD was as follows: 220 g dextrose (750 kcal), 1.2 g/kg protein of 80.6kg dosing weight (400 kcal), SMOFlipid 20% at 20.8mL/hr for 12 hours (500 kcal, 250mL), and recommended multivitamin, trace element, thiamine (300mg), and zinc (5mg) supplementation. This would provide 1650 kcals (100% nutrition goal) and 100 g protein

(100% nutrition goal). The TPN ordered by the Nephrologist included: 235g dextrose, 1.2 g/kg dosing weight protein, 250 mL SMOFlipid 20%, calcium gluconate, multivitamin, trace elements, and insulin. This prescription was meeting 102% of the patient's calorie goal and 110% of GC's protein goal. TPN was monitored according to ASPEN guidelines and the patient was monitored for refeeding syndrome.[10]

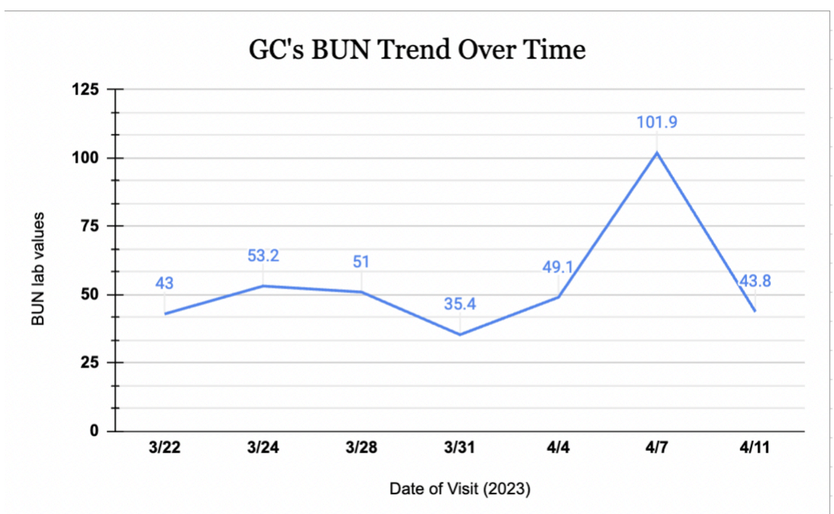
At the second visit by the registered dietitian, the patient was s/p a diverting loop ileostomy, closure of the fascia, and remained intubated and sedated. To estimate GC's needs at this visit, the Penn State equation was used again, now calculating her calorie needs to be 1660 kcals. The patient's needs once she no longer required the ventilator were estimated to be 1537 kcal, calculated using the Mifflin St. Jour equation with a total 1.3 injury and activity factor. With the absence of a negative pressure dressing, her protein needs decreased to 88 g protein (1.2g/kg of 73.6kg). At this visit, the nephrologist noted an AKI secondary to pre-renal state. GC's BUN increased to 53.2, up 10 units since the last RD visit two days prior and her urine output averaged 557 mL over the last five days. Recommendations from the RD included: adjusting TPN protein dosage to 1.1g/kg of dosing weight (80.6kg), supplementing 5 mg zinc in TPN, and recommending a peptide-based formula if eventually safe to feed through the gut. At both the initial and follow-up visit, the RD evaluated the goals set, and at the follow-up, the TPN was meeting within 10% of the patient's estimated needs, the patient's labs were monitored, and she remained on TPN with no imminent plans to use the enteral route.

Since the second follow-up, the patient was transferred to the floor, briefly put on a clear liquid diet which was soon discontinued due to bloating and gas per patient and some dysphagia. Zinc was recommended in the TPN for high ostomy output and to promote healing of the surgical wound. GC's labs started to trend to normal. Another negative pressure dressing was placed on her abdomen, in which the care team found feculent matter. The surgery MD requested a higher protein dosage of 1.5 g/kg/d in the TPN prescription in response to the delayed healing of the abdominal surgical wound. This value is higher than the current recommended guidelines from ESPEN and on the higher end of the ASPEN guidelines, but falls within many of the values discussed within the Schwartz et al. article.[8] At this time, GC's BUN started to rise again. BUN values over time are found in Figure 2. At the second to last follow-up, the patient was transferred to the MICU for acute on chronic hypoxic respiratory failure. She was receiving propofol, which provided 660 kcal. The RD recommended discontinuing IV lipids while on high-dose propofol (>15ml/hr). GC's BUN was 101.9 and her GFR was 36, the nephrologist noted that the patient was in acute renal failure. The patient's dosing weight was changed to 100.2 kg, which affected the amount of protein in one bag of TPN. This was soon corrected by the nephrologist. At the last follow-up, the patient had been transferred to the SICU and had been started on continuous renal replacement therapy (CRRT). GC was taken back into the operating room another time for an abdominal washout and drainage of abscess in response to peritonitis. At this point, supportive care started following the patient. GC's care was discontinued on April 12 after care team discussions with the family, and she passed away that afternoon.

Figure 1: GC's weights from prior encounters

Wt Readings from Last 15 Encounters	
03/20/23	87.8 kg (193 lb 9 oz)
03/08/23	73.5 kg (162 lb)
02/16/23	79.4 kg (175 lb)
01/13/23	86.2 kg (190 lb)
11/28/22	86.2 kg (190 lb)
09/28/22	83.9 kg (185 lb)
08/19/22	83.9 kg (185 lb)
07/18/22	86.2 kg (190 lb)
06/09/22	83.9 kg (185 lb)
05/24/22	83.9 kg (185 lb)
05/12/22	83.9 kg (185 lb)
04/08/22	81.6 kg (180 lb)
03/01/22	83.9 kg (185 lb)
09/28/21	83.9 kg (185 lb)
06/10/21	85.1 kg (187 lb 9.8 oz)

Figure 2: GC's BUN Trend Over Time



Summary and Conclusions

GC was an eighty three year old female with a nutrition-pertinent past medical history of COPD, hypertension, hyperlipidemia, type two diabetes mellitus, and GERD. The patient was admitted to Cedars Sinai Medical Center for SBO, and had a complicated hospital course. For over ten years, she was in and out of the hospital for procedures. In the patient's most recent hospital course, she underwent four different procedures including a laparoscopy turned intestinal obstruction repair for an SBO which removed two to three feet of small bowel and anastomosed the ileum to the cecum, a peritonitis washout, a second abdominal irrigation turned diverting loop ileostomy, and a final peritonitis washout.

At the initial visit with the dietitian, it was decided that TPN should be initiated due to the history of SBO, recurrent peritonitis, the patient's frequent trips to the operating room which would halt enteral feeding, and the urgency to feed at goal rate at the initial assessment with the RD. The patient had already been NPO for most of her hospital admission (5 days) and had gotten minimal nutrition since then. This patient was malnourished, and while with an open abdomen would have only been able to receive 10-20 mL of feeds per hour enterally (per preference of the SICU) which would not have met her needs. Therefore, it was clear that TPN should be initiated as soon as possible to provide the patient with her estimated needs. For the course of the patient's hospital stay, the RD and GC's nephrologist worked closely to adjust the TPN prescription. When recommending the TPN prescription, the RD recommended to the physicians to monitor for refeeding syndrome, which includes monitoring potassium, phosphorus, and magnesium closely and to replete as needed. Zinc supplementation was recommended for surgical wound healing and potential losses from the negative pressure dressing. The dietitian also decided to be conservative with the protein dosage recommendation for a patient who was malnourished and had a negative pressure dressing in place (1.2 g/kg), since the patient had uptrending BUN and creatinine values. In light of the recent evidence on protein during AKI, the RD could have been more lenient with the protein recommendation, potentially using the PCR estimated amount of 1.3 g/kg/d protein, in efforts to hopefully promote kidney function and urine output.[9] Since this patient was malnourished, had a surgical wound, and a negative pressure dressing, GC could have potentially benefitted from a higher protein prescription.

SMOFlipid was recommended in the TPN prescription due to its relative anti-inflammatory effect when compared to Intralipids.[9] Short-term use of SMOFlipid shows a significant decrease in LFTs, total bilirubin, triglyceride levels, CRP, and hospital length of stay. The combination of different fat sources, including omega-3 fatty acids, has been seen to have an anti-inflammatory effect on cytokines, ROS production, and NF-KB activation. The high omega-6 to omega-3 fatty acid ratio in Intralipids may promote inflammation and affect immune function.[9] At a certain point in GC's hospital admission, intravenous lipids were discontinued due to her propofol prescription and the calories from fat it provided.

Throughout GC's hospital stay, she was monitored for tolerance to the TPN and for the ability to transition to enteral feeds. GC's nutrition goals included TPN meeting ~90-110% of the patient's estimated needs, a point of care glucose value of 70-180 mg/dL, triglyceride levels less than 400, and no increase in LFTs. The patient's labs were also monitored according to ASPEN's criteria.[11] In GC's case, when trialing enteral feeds through a clear liquid diet, the patient reported bloating and gas, so the medical team decided to discontinue an oral diet and continue solely with TPN. For the remainder of GC's hospital stay, she was NPO on customized TPN.

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