

Clinical Implications of “Near Total” Esophagectomy with Subtotal Gastrectomy

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Executive Summary

The esophagus is a straight, hollow tube that moves food boluses from the mouth into the stomach through a wave-like contraction of esophageal muscles called peristalsis. Gastroesophageal reflux disease (GERD) is described as the backward flow of the stomach or duodenal contents into the esophagus; may occur normally or as a chronic pathologic condition (2). It is possible that GERD, hiatal hernia, and prolonged hospitalization could lead to acute esophageal necrosis (AEN) and eventually lead to esophageal perforation (EP) (5).

While both AEN and EP are rarities, they both often require surgical intervention to correct the problem. A partial esophagectomy removes a portion of the esophagus and leaves the option of re-connection or diversion of the remaining esophagus to the stomach. Three surgical options exist: (1) a gastric pull-up procedure to reconnect a portion of the stomach with the remaining esophagus, (2) diverting the esophagus and creating a stoma in which the saliva and oral ingestions drain out of the remnant esophagus into an external ostomy bag or (3) colon interposition where lower end of remnant esophagus is attached to an inverted piece of healthy resected colon, and upper end of remnant stomach is attached to the other end of colon.

The following case study examines Mr. WC, who unexpectedly developed chest pain and was admitted to Anne Arundel Medical Center. He has a history of gastroesophageal reflux disease, gastrointestinal bleeding, hypertension as well as other documented medical conditions. Admitting test revealed an esophageal perforation as well as a necrotic esophagus that required emergent surgical repair. This report follows his hospital stay and recovery from the surgery. Medical complications, interventions and nutritional implications were addressed. A treatment plan was developed according to his specific nutritional needs that would allow for a successful transition back to a healthy life.

Case Report

General Information

Mr. WC is an 86 year old Caucasian male, admitted 10/19 to Anne Arundel Medical Center (AAMC) with epigastric/chest/abdomen pain, nausea and vomiting via Emergency Room. Initially he was thought to have an aortic dissection but upon further investigation, a CT scan revealed a paraesophageal hernia with a large volume of fluid and undigested food located in his thoracic region. Nasogastric tube was placed and patient had immediate relief from pain and shortly after aspirated which lead to intubation. Patient's height upon admission is 5'8" and weight is 165lbs (75kg).

Social History

Mr. WC is retired and lives with his wife. He is insured through Medicare Part A and B as well as Blue Cross Federal 190. He is a non-smoker since 1944, and currently uses alcohol in unspecified amounts.

Medical/Surgical History

- Medical history includes hypertension, gastroesophageal reflux (GERD), osteoarthritis, myocardial infarction, Benign prostatic hyperplasia, gastrointestinal bleeding, bladder obstruction, left bundle branch block, and melanoma.
- His surgical history consists of multiple incisional hernia repairs, appendectomy, Hartmann procedure and reversal on admission include: Atenolol (Antihypertensive) 25mg qd, Flomax (BPH treatment) 4mg qd, Detrol (Antimuscarinic, Bladder control agent) 4mg qd, Protonix (AntiGERD, Antisecretory) 40mg qd, Micalcin (Osteoporosis treatment) spray qd, Lipitor (Antihyperlipidemic) 10mg qd, Ocuville (Vitamin for eye health), Lidoderm patch (topical analgesic), Avodart (BPH treatment) 0.25mg qd, Ultracet (Analgesic) 37.5mg qid as needed and a daily aspirin (CVA or MI preventative) 81mg qd.
- Allergies – None.

Nutritional History

- Diet history (11/2) Due to paralyzed vocal cords, patient is difficult to understand and is not enthusiastic about talking in great lengths. Per his wife, he eats normal and somewhat large amounts, usually three times a day.
- Weight history (taken from electronic health record)

03/99	05/01	07/03	03/04	06/05
170	175	167	175	162

- Physical activity – WC has been very active most of his life, he sang and played in a band prior to hospitalization.
- Vitamin/Mineral/Herbal supplementation – Calcium 200mg qd, Centrum A-Z qd.
- Cultural attitudes – none to influence dietary intake.

Hospital Course of Patient

- Physical exam – Patient is intubated in Critical Care Unit (CCU), a nasogastric tube is in place and is draining a blood-tinged material. Lungs are coarse bilaterally; no obvious rales or heaves were identified. Abdomen is flat and soft however exam is limited due to the fact that pt is intubated. Vital signs – heart rate: 101, blood pressure: 140/103, on ventilator.
- Laboratory results – refer to Appendix A.
- Medications – refer to Appendix B.

Medical Treatment 10/20/08-11/08/08

WC was admitted to the Critical Care Unit on 10/19/2008, at that time was intubated and sedated. An esophagogastroduodenoscopy was performed which revealed a necrotic esophagus and stomach. Following the scope, he was emergently sent to the operating room (OR) for a partial esophagectomy with diverting esophagostomy and partial stomach resection ("partial gastrectomy"). He was empirically started on Zosyn and Levaquin then Fluconazole and vancomycin were added in the OR. A feeding gastrostomy was placed during surgery to allow for tube feedings post operatively. Pathology report revealed esophagus and stomach biopsies were not cancerous. He remained intubated and sedated on fraction of inspired oxygen (FIO2) of 70% and febrile at 102.7. Lines and drains include: left subclavian triple-lumen catheter, right femoral arterial line, right diverting esophagostomy Gastrostomy tube, peripheral intravenous line, urinary Foley catheter, endotracheal tube.

Mr. WC was finally exubated (11/1) after many unsuccessful attempts. He remained groggy and tired.

Modified Barium Swallow (11/05) – Upon swallowing, the majority of the contrast emptied into the esophagostomy bag, but minimal amounts entering the trachea – demonstrating silent aspiration.

Thus the patient was kept NPO and any further esophageal perforation or incision-related anastomotic leaks.

Laryngoscope (11/07) performed to evaluate hoarseness and difficulty swallowing demonstrated paralysis was secondary to his left vocal cord paralysis.

Vocal Cord injection (11/08) was performed per Ear, Nose and Throat (ENT) Surgeon to attempt to move the vocal cords back into a more normal medial position. While the results are temporary – quality of life is improved.

Nutritional Treatment

- Diet ordered – NPO upon admission through entire stay at AAMC.
- Comprehensive exam determined Resting Energy Expenditure (REE) to be 1885kcal/day via Penn State Equation and protein requirements 110gm/day. Caloric needs determined to be 2100 per day.
- 10/21/08 – Pharmacy was consulted to see patient in regards to Peripheral Nutrition (PN). He was started on a PN formula at 100mL/hr with 35gm of amino acids, 200gm dextrose and 0gm of lipids. A nutrition consult was also placed in which pt was assessed. His elevated triglyceride levels precluded use of IV lipids in PN.
- (10/21) An initial tube feeding (TF) was started with Peptamen PreBio (full strength) at 15 mL/hr which provided 15kcal/hr, 0.6grams of protein/hr.
- 10/22/08 – Following lab evaluation, Pharmacy maintained PN volume, but changed amino acids to 60gm, dextrose to 300gm and lipids to 20gm. TF held per surgeon, continues with elevated triglycerides. [Note - *TF was stopped on 10/22 and resumed 10/25 because his TG's were elevated and Dr was concerned.*]
- 10/23/08 – With TG levels dropping (274mg/dL), Pharmacy changed PN formula to 85gm of amino acids, 350gm of dextrose and 50gm of lipids.
- 10/24/08 – PN dextrose decreased to 300gm. Rate, amino acids and lipids stayed the same.
- 10/25/08 – Surgeon approved re-start of TF since triglycerides are now Within Normal Levels (WNL).
- 10/26/08 – TF residuals were elevated overnight and WNL by morning.
- 10/27/08 –PN changes included increased rate to 80mL/hr with composition of 50gm amino acids, 200gm dextrose and 40gm of lipids. TF increased to 30mL/hr.

- 10/28/10 – Transition from PN to TF began with PN decreased to 40mL/hr with composition of 50gm of amino acids, 200gm dextrose and 40gm of lipids, while TF increased to 45mL/hr. New orders written to advance TF rate 10mL/hr every six hours to target rate of 85mL/hr.
- 11/01/08 - Change TF to Fibersource HN at 65mL/hr, prosource supplement added one packet every 12 hours. Re-calculated nutrition needs as: REE changed to 1400kcal/day per Penn State Equation and protein requirements decreased to 80gm/day. Caloric needs estimated at 1800 per day. Elevated liver function test (LFT) and wt gain contributed to change in needs and decrease of TF.
- 11/04/08 – TF continues at goal rate (65mL/hr) with Prosource, pt tolerating TF without complications.
- 11/06/08 - TF continues at goal rate (65mL/hr) with Prosource, pt tolerating TF without complications.
- 11/09/08 - TF continues at goal rate (65mL/hr) with Prosource, pt tolerating TF without complications.

Medical Considerations

Understanding the normal anatomy and physiology of the upper gastrointestinal (GI) tract is essential to understanding pathophysiology. The upper GI tract is composed of the mouth, pharynx, esophagus and stomach, while the lower GI tract includes the small and large intestine (1). Accessory or ancillary organs include the liver, biliary system and pancreas (1). The upper GI tract as well as the lower GI tract is responsible for moving, digesting, absorbing and excreting food and nutrients.

The esophagus is a straight, hollow tube approximately 25 cm long and 2 cm in diameter with two sphincter muscles at either end. The upper esophageal sphincter is responsible for preventing air ingestion and to prevent esophageal reflux. The lower esophageal sphincter plays a more important role; it controls the release of foodstuff from the esophagus to the stomach and prevents stomach contents from backing up into the

esophagus. The esophagus's main function is to move food boluses from the mouth into the stomach via wave-like contractions of esophageal muscles termed "peristalsis".

The normal anatomy and physiology of the stomach is more complex than the esophagus; it has a greater role in digestion. Portions of the stomach (fundus, corpus, antrum, and pylorus) differ by anatomy and function (1). Motility, secretion, digestion and absorption are the major functions of the stomach.

By medical history this patient had Gastroesophageal reflux which may occur normally or as a chronic pathologic condition (2). Symptoms vary but may include reflux of gastric secretions, heartburn with episodes of substernal pain, belching and esophageal spasm (2). The severity and complications associated with these symptoms is not well understood. Some patients display significant findings, but live with minor complications; others have few findings but have considerable discomfort. Variation in pain tolerance may play a role in this difference. Prolonged erosive disease causes esophagitis, esophageal erosions, ulceration, scarring, stricture, and in some cases dysphagia (2). Factors associated with esophagitis development include: radiation exposure, immunosuppression, GERD, recent chemotherapy, recent antibiotic therapy, corticosteroid therapy, neutropenia, presence of malignancy, advanced age, general debilitation, prematurity, and diabetes (3). The prevalence of esophageal reflux varies with the description of the symptoms, but about 20% to 40% of adults report symptoms of GERD at least one time per week (Talley and Wiklund, 2005) (2).

GERD management has three major goals: (1) increasing lower esophageal sphincter (LES) competence; (2) decreasing gastric acidity to lessen symptom severity; and (3) improving clearance of contents from the esophagus (1). Decreasing gastric acidity involves use of medication and nutrition therapy. Medications fall into five major categories: (1) antacids or buffering agents, (2) histamine blocking agents, (3) prokinetic agents, (4) proton pump inhibitors, and (5) mucosal protectants (1). If medical management of GERD is unsuccessful, surgical procedure (e.g. fundoplication) can help increase LES competence.

Nutritional therapy includes avoidance of those foods that typically cause symptoms. So patients are advised to limit foods relax LES pressure (peppermint, chocolate, etc.), decrease consumption of foods that may increase gastric secretions (coffee, alcohol, pepper), and avoiding other foods that could potentially cause a symptomatic response (milk, fried foods, hot-dogs, and others) – if it gives them discomfort. Smaller and more frequent meals may prove beneficial as well as remaining up-right post-prandial (1) for some individuals.

While GERD has an excellent prognosis with 80 to 90 percent of improvement noted with medication, relief can be partial or temporary in nature. Severe GERD has also been associated with esophageal ulcer, strictures and Barrett's esophagus (6).

Acute esophageal necrosis (AEN), also known as “black esophagus” or “necrotizing esophagitis” presents on endoscopy as a circumferential black esophagus; it is more common in the distal/ lower portion of the organ, but ends sharply at the gastroesophageal border. The disease affects both mucosa and submucosa layers; necrosis is diffuse and severe and occurs in the absence of caustic or other injurious agents (4). Diagnosis of AEN is made endoscopically through the observation of a diffusely black esophageal mucosa (5).

While AEN is a rare disorder, its cause is generally believed multi-factorial. Some experts feel that ischemia is the major factor in rapid development of esophageal lesions as the lower esophagus is less vascularized and is thus more prone to ischemic injury. While vascular compromise alone could be responsible for AEN, its pathogenesis is generally more complex (5). AEN incidence remains very low, in autopsy studies (0- to 0.2%) and clinical trials (0.01% to 0.2%). Less than 90 cases to date have been reported in the literature (5).

Alcohol-related GERD, gastric stasis, and dilatation, and decreased mucosal protection may contribute to AEN development. Post-operative dysmotility, preexisting lower esophageal sphincter dysfunction with reflux, and recumbent position all may contribute to the massive reflux of the gastric contents into the esophagus causing further damage to the mucosal layers (5). Additionally, prolonged contact of non-steroidal anti-inflammatory

drugs may occur with advanced age, recumbence, and motility disturbance will worsen any pre-existing reflux. (7).

Prognosis with AEN remains poor. Overall mortality in patients with black esophagus was high (31.8%) and was commonly associated with underlying diseases (5). Conservative treatment that addresses the underlying disease, intravenous hydration, establishing supportive parenteral alimentation, and intravenous proton pump inhibitors (PPI) together with sucralfate can help reverse the progression of AEN (5). Alternatively, histamine receptor blocker can be used and anemia should be corrected by blood transfusions (5).

Esophageal perforation (EP) exists when a hole in the esophagus occurs, allowing fluids and foodstuff to escape into the surrounding chest cavity. The most common cause of EP remains iatrogenic (8). Spontaneous perforations (Boerhaave's syndrome) account for 15% of all esophageal ruptures, whereas perforations from trauma (penetration or blunt injuries, caustic injuries, and swallowed foreign bodies) account for about 20% of cases (8). Once the esophagus had been perforated symptoms such as pain, blood-tinted vomit, or hematemesis, dysphagia, odynophagia, and dyspnea are observed.

Diagnosis of EP can be made in 90% of cases with radiographic change documented in the neck, chest, and/or abdomen. (8). EP is generally considered a medical emergency with immediate surgical intervention recommended for any one medically able to undergo surgery. Radovanovic and associates (1995) reported a mortality that increased from 2.8% when patients were treated within 24 hours to 18.4 % when treatment was delayed more than 24 hours (8).

Surgical treatment is aimed at (1) preventing further thoracic leakage, (2) controlling and/or eliminating perforation-induced infection, (3) restoring the GI integrity and continuity, and (4) maintaining esophageal function (8). Extensive debridement of all nonviable tissue in the mediastinum and pleural cavity is undertaken. A myotomy is often necessary beyond the extent of the mucosal tear to expose the total length of

the mucosal injury (8). Wide stitches in healthy tissue are used to reapproximate the edges of the perforation and staples have been suggested to close the lacerated mucosa (8).

Nutritional Therapy

Nutritional therapy for esophagectomy patients consists of two phases, the preoperative phase and the postoperative phase. The preoperative phase, aimed at improving nutritional status in known esophageal cancer patients, is not relevant since this patient had an acute non-cancerous perforation necessitating the partial esophagectomy.

Following removal of the part or the entire esophagus – patients can either be re-connected (gastric pull-up or colon interposition) or have stomas made in the esophagus for salivary secretion removal, and a second stoma in the form of a feeding gastrostomy. Some patients undergo an initial surgery with stoma creation, and when more medically and nutritionally stable undergo a second reconnection procedure. In this case, a gastric pull-up was considered, but was not done.

WC, was NPO throughout his hospitalization, but may later be a candidate for a gastric pull-up procedure. Post-operatively a barium swallow was done to look for potential further perforation or anastomotic leaks at the cervical esophagostomy site. WC was transitioned from PN onto TF as his primary source of nutrition support. While the stomach was partially resected, the surgeon determined a feeding gastrostomy, rather than a feeding jejunostomy was the best choice.

Mr. WC lab values

	OCT													NOV							
	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	7	Normal Range	
BUN	17	21	32	27	23	24	27	29	27	28	25	28	23	18	21	25		33	28	5-25 mg/dL	
CO2	19	17	20	27	32	26	28	24	25	22	24	24	22	21	26	31		34	31	18-32 mmols/L	
GLU	205	143	125	94	106	138	145	156	131	334	143	132	179	106	144	151		113	117	70-115 mg/dL	
Ca	7.2	8.4	8.4	7.9	8.4	8.0	8.5	8.7	8.9	8.2	8.8	8.7	8.7	8.6	9.1	9.1		9.6	9.3	8.1-10.2 mg/dL	
AST			57	49		34								78	15					10-40 IU/L	
ALT			52	42		31								112	95					4-44 IU/L	
Total Pro			4.2	3.9		4.8								6.0	6.2					5.7-8.1 gm/dL	
Albumin			2.2	2.1		2.4								2.7	3.1					3.2-5.0 gm/dL	
TG			401	349	274	192	160	175	156	253										<250 mg/dL	
WBC	7.6	11.7		13.8		8.5	9.8	11.6		13.1	16.7	13.5	11.9	11.0		7.6	8.9	8.8	8.7	4.8-10.8 10 ³ /u	
RBC	5.08	5.25		3.48		3.52	3.38	3.32		3.26	3.37	3.28	3.18	3.10		4.27	3.50	3.58	3.77	4.50-8.90 10 ⁶ /L	
HGB	12.1	16.2		10.5		10.6	10.2	10.0		10.0	10.3	9.9	9.7	9.3		12.6	10.7	10.7	11.3	13.5-17.1 gm/dL	
HCT	36.1	47.2		31.1		31.6	30.5	30.7		30.1	31.1	30.6	29.8	28.4		40.6	32.7	33.3	35.1	41.0-51.0 %	

*Labs with three or more abnormal results were considered.

Medication	Dosage	Dates of Administration	Medical Function	Nutrition Side Effects
Albuterol 0.5%	2.5 mg	10/19-11/17 Less freq admin later in admission	Bronchodilator	May cause peculiar taste, sore/dry throat, anorexia or diarrhea.
Caspofungin (Cancidas)	70 m 50 mg	10/21 10/22-11/6	Antifungal	May cause N/V, dyspepsia or diarrhea.
Diphenhydramine (Benadryl)	50mg 25 mg	11-1 and 11-8 11/1- 11/17 Protocol driven	Antihistamine, Sleep Aid	May cause anorexia, dry mouth/throat, epigastric distress, constipation or diarrhea.
Docusate syrup (Colace)	20 mg	11/5-11/15	Stool Softener	Alters intestinal absorption of water & electrolytes
Haloperidol (Haldol)	5-10 mg	11/2-11/17 Protocol driven	Antipsychotic	Take with food or milk. May increase appetite and wt or may decrease appetite and wt
Hydralazine (Apresoline)	10 mg	10/19-11/17 Protocol driven	Antihypertensive	Decreasing Na ⁺ and Ca may be recommended. Food increase bioavailability, interferes with Pyruvic acid metabolism
Imipenem (Primaxin)	500 mg	10/28-11/15	Antibiotic	May cause N/V, cramps or diarrhea
Insulin Glargine (Lantus)	8-16 units daily	10/27-11/17 D/C on 15 units	Antidiabetic, Hypoglycemic	May increase wt, use caution with alcohol. Need meal plan for carbohydrate consistency.
Ipratropium 0.02% (Atrovent)	15 mg	10/19-11/17 Protocol driven	Bronchodilator	May cause dry mouth/throat, metallic/bitter taste, N, dyspepsia.
Potassium Chloride		Protocol driven electrolyte replacement	Electrolyte	Take with meals but not with salt subs. May cause GI irritation, N/V, diarrhea, abd pain.
Lorazepam (Ativan)			Antianxiety	Limit caffeine, caution with grapefruit. May cause anorexia, dry mouth, or increase in

				salivation, N/V/D, constipation.
Metoprolol (Lopressor)	2.5 mg 5 mg	10/18-11/8 11/8-11/17	Antihypertensive, Antiangina, CHF treatment	Take with food to increase bioavailability. Decrease Na+ and Cal may be recommended. May cause dry mouth, N/V, dyspepsia
Morphine Sulfate	4 mg	Protocol driven for sedation	Analgesic	May cause anorexia, increase thirst, dehydration, dry mouth, taste changes, etc. May take with foods to decrease GI distress.
Nystatin	5 ml	10/26-11/17	Oral Candidiasis Treatment	Retain drug in mouth as long as possible, do not chew, let dissolve in mouth. May cause GI distress, N/V, stomach pain and diarrhea.
Pantoprazole	40 gm	10/19-11/4	CC protocol prophylaxis	May decrease absorption of Fe, Vit B12. Increases gastric pH, may cause diarrhea, nausea or abd pain.
Piperacillin-Tazobac (Zosyn)	3.375 mg	10/19-10/28	Antibiotic	May cause anorexia, black hairy tongue, dry mouth, oral candidiasis, N/V, epigastric distress, etc.
Promethazine	6.25-12.5 mg	10/19-11/17 Protocol driven, rarely given	Antihistamine, Antivertigo, Antiemetic, Sedative	Take with meals or HS with water or milk to decrease GI irritation. Increased need for riboflavin. May cause dry mouth, N/V, constipation.
Aspart Novolog	units	High dose sliding scale, protocol driven	Antidiabetic, Hypoglycemic	May increase wt, use caution with alcohol. Need meal plan for carbohydrate consistency.
Trypsin-Balsam (Granulex)		Topically given as needed during admission	Promotes skin healing	None noted.
Lansoprazole (Prevacid)	30 mg	11/4-11/17	Anitulcer, AntiGERD, Antisecretory	May decrease absorption of Fe, Vit B12. Increases gastric pH, may cause diarrhea, nausea or abd pain.

Vancomycin	900-1200 mg	10/19-11/2	Antibiotic	Little GI absorption. May cause bitter taste, nausea.
Enoxaparin (Lovenox)	40 mg	11/2-11/10	Anticoagulation	May affect lab values, caution with DM & ESRD, and increased concern of Vitamin K foods.
Furosemide (Lasix)	40 mg	10/22-11/18 for 20 total doses	Loop Diuretic	Take on empty stomach, decrease Na intake may be recommended, may cause increased thirst.
Propofol	protocol driven dosing	10/19-11/1	lipid based anesthesia used for sedation	Adds calories, 30 mL/hr = 790kcal/fat/day, will affect tube feeding calculations.
Albumin 25%	25 mg	10/24-10/26	Oncotic agent	Given with low blood volume, would affect Alb level on labs.

Appendix C

PN History

WC PN History

Date	Rate	Amino Acids	Dextrose	Lipid
10/21	100mL/hr	35gm/hr	200gm/hr	0gm/hr
10/22	100mL/hr	60gm/hr	300gm/hr	20gm/hr
10/23	100mL/hr	85gm/hr	350gm/hr	50gm/hr
10/24	100mL/hr	85gm/hr	300gm/hr	50gm/hr
10/27	80mL/hr	50gm/hr	200gm/hr	40gm/hr
10/28	40mL/hr	50gm/hr	200gm/hr	40gm/hr

PharmD consulted 10/20.

TPN D/C'ed 10/28 when bag expired.

References

1. Nelms, M, Sucher, K, Long S, *Nutrition Therapy and Pathophysiology*. 2007; 423-427.
2. Mahan, LK, Escott-Stump, S. *Krause's Food and Nutrition Therapy*. 2008; 655-657.
3. Fenoglio-Preiser, CM, Noffsinger, AE. *Gastrointestinal Pathology, An Atlas and Text*. 1999; 44, 72.
4. Mishkin, D S, Gelrud, D. Acute esophageal necrosis (black esophagus). *Up to Date*: online, accessed 25 November 08. <http://www.uptodate.com>.
5. Gurvits, GE, et al. Acute esophageal necrosis: a rare syndrome. *Journal of Gastroenterology*. 2007; 42:29-38.
6. Mayo Clinic. *Gastroesophageal Reflux Disease*. <http://www.mayoclinic.org/gerd/treatment.html>.
7. Hiroshi, Y, et al. Acute necrotizing esophagitis: role of nonsteroidal anti-inflammatory drugs. *Journal of Gastroenterology*. 2006; 41:193-197.
8. Pearson, FG, Ginsberg, RJ. Esophagectomy for Benign Disease. *Esophageal Surgery*. 2002; 459-462.
9. Knight, CE. Nutrition Considerations in Esophagectomy Patients, *Nutrition in Clinical Practice*. 2008; 521-528.
10. ADA Nutrition Care Manual via Anne Arundel Medical Center.