Diabulimia:

Nutrition Intervention for Type I Diabetes and Bulimia Nervosa



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**ABSTRACT**

Type I diabetes mellitus is characterized by hyperglycemia as a result of the body’s inability to metabolize glucose due to deficiency in insulin secretion and can be contributed to both genetic and environmental factors.1 Maintaining normal glucose levels can delay and even prevent serious microvascular and macrovascular complications.1 Unfortunately, maintaining normal glucose levels presents a challenge to these patients as they struggle with weight maintenance, misperception of body image, low self-esteem and related eating disordered behavior. As a method of preventing weight gain or losing weight, patients with type I diabetes will often limit insulin use after meals or completely omit insulin use altogether. This causes hyperglycemia and calories are then purged as glucose is spilled through the urine.2 This practice is being recognized as diabulimia, derived from the words diabetes and bulimia. Diabulimia is a condition that is under recognized and needs a greater awareness in the health community. Incidences of diabetic ketoacidosis admissions strengthen the need for raising awareness of diabulimia.

Medical care through a multidisciplinary team in which all team members are experts in dealing with eating disorders and/or diabetes is essential in treating an individual with diabulimia. Being a new condition in the medical field and there are no set guidelines created to treat diabulimia, therefore, it is important that the dietitian provides only evidence based medical nutrition therapy to the patient. Nutrition therapy should be provided for both the bulimia and diabetes in order to treat both of the underlying conditions of the patient.

**INTRODUCTION**

With diabulimia being an under recognized condition that poses serious short-term and long-term consequences and even death, this presents a great opportunity to help raise awareness of the dangers of diabulimia. Knowing that awareness for diabulimia is gaining attention in the health community, it was not a hard decision to begin research on this topic. Patients with type I diabetes have put a serious twist on Bulimia Nervosa by purging their calories through limiting their insulin use or omitting it all together. As a person without diabetes, it never came to mind the psychological adaptation people with type I diabetes go through after being diagnosed. From meal planning from scratch to having to be selective in food choices to weight management problems, it is pretty straight forward how these patients can lose their sense of control.

DC is a 25-year old female with type I diabetes that has presented to Kennedy Hospital, and other hospitals, in the past year with multiple admissions of diabetic ketoacidosis or hyperglycemia hyperosmolar syndrome. My first encounter with DC was an initial assessment in March 2013; she was in the intensive care unit with clear cachexia and muscle wasting. She was unwilling to answer many questions and denied any diabetes education. During the second assessment with DC I found it strange that she could not recall her insulin regimen at home. Speculation of whether she intentionally misused her insulin occurred with the other dietitian, but with DC unwilling to answer most questions it was hard to tell. Within the next month, DC was back again, with yet another diabetic ketoacidosis and again could not accurately recall her insulin dosages at home. After multiple admissions, suspected noncompliance and severe weight loss, the doctor diagnosed her with diabulimia. With a condition new to me and many of the other dietitians, it was clear DC and her diabulimia would make an intriguing case study.

**THE PANCREAS**

**Anatomy**

The pancreas is an accessory organ located across the back of the abdomen and posterior to the greater curvature of the stomach. It measures about five to six inches long and one inch thick and consists of a head, a body, and a tail.1 The right side of the pancreas is the head; it is the widest part of the organ and is located in the curve of the duodenum.3 The body makes up the left side of the organ, tapering upward and ending with the tail of the pancreas near the spleen. Small ducts throughout the pancreas unite to form two larger ducts, the pancreatic duct and the accessory duct. The pancreatic duct, or duct of Wirsung, joins the common bile duct from the liver and gallbladder and enters the duodenum as a dilated common duct called the hepatopancreatic ampulla, or ampulla of Vater. The accessory duct leads from the pancreas and empties into the duodenum, superior to the hepatopancreatic ampulla.3 The pancreas is made up of small clusters of glandular epithelial cells that carry out different functions.1 The pancreas functions as a digestive organ with exocrine glands that secrete enzymes to aid in digestion in the small intestine. It also functions as an endocrine gland that produces hormones including glucagon, insulin, somatostatin, and pancreatic peptide.1

**Exocrine Glands**

Exocrine glands secrete their products into ducts that empty into body cavities or onto body surfaces. The secretions of the exocrine glands include mucus, sweat, oil, earwax, saliva, and digestive enzymes.1 In the pancreas, about 99% of the cells are arranged in small clusters called acini and form the exocrine portion of the organ.1 The cells within the acini secrete pancreatic juices into the pancreatic duct which delivers the secretions into the small intestine.3 The pancreas produces 1200-1500 mL of pancreatic juice per day. Pancreatic juice is a colorless liquid that consists mostly of water, some salts, sodium bicarbonate, and several digestive enzymes.1 The sodium bicarbonate makes the pancreatic juice slightly alkaline to a pH of 7.1 - 8.2. The alkaline pH helps to buffer the acidic gastric juice in chyme, stop the action of pepsin from the stomach, and create the proper pH for the action of digestive enzymes in the small intestine. There are multiple enzymes in the pancreatic juices including pancreatic amylase, trypsin, chymotrypsin, carboxypeptidase, elastase, pancreatic lipase, ribonuclease, and deoxyribonuclease that play a role in digestion.1

**Endocrine Glands**

Throughout the body, the endocrine system works to control various activities in the body by releasing hormones. Hormones are regulatory substances that control the activity of cells in different parts of the body.1 After hormones are secreted by the endocrine glands, they enter the interstitial fluid, travel to the bloodstream and are delivered to specific cells where they bind to the receptor and become activated. Most hormones take several minutes to produce a response. The remaining 1% of the pancreatic cells forms the endocrine portion of the pancreas.1 There are one to two million tiny clusters called the pancreatic islets, or islets of Langerhans, that are scattered throughout the exocrine acini. In each pancreatic islet there are four types of hormone-secreting cells that secrete multiple hormones including glucagon, insulin, somatostatin, and pancreatic peptide.1

Alpha or A cells make up about 17% of pancreatic islets and secrete glucagon, a hormone that works to raise blood glucose.1 Alpha cells detect low blood glucose and release glucagon into the blood. Glucagon binds to the glucagon receptors on hepatocytes and stimulates the process of glycogenolysis. As the glycogen stores become depleted glucagon stimulates the liver and kidneys to activate the process of gluconeogenesis.4 Glucagon can also control the rate of lipolysis to release glucose. All of these processes help to restore blood glucose levels.4 When blood glucose levels are high alpha cells are suppressed.1

Beta or B cells make up about 70% of the pancreatic islet cells that produce and secrete insulin, a hormone that works to lower blood glucose levels.1 Insulin is a protein derived, anabolic hormone. Beta cells detect high blood glucose levels and release insulin in the blood to uptake glucose into hepatic cells and insulin dependent cells, including adipose and muscle cells.4 Insulin also stimulates glycogenesis, lipogenesis, and protein synthesis.4 When blood glucose levels are low beta cells are suppressed.1 The secretion of insulin can also be stimulated by other hormones and neurotransmitters.4

Delta or D cells make up about 7% of pancreatic islet cells and secrete somatostatin, an inhibitory hormone.1 In the nervous system, somatostatin acts as a neurotransmitter that inhibits the secretion of several hormones including insulin, glucagon, gastrin, and growth hormone and inhibits the absorption of glucose into the intestine and lowers blood glucose.5 Somatostatin can also be secreted into the gastric lumen where it can directly inhibit gastric acid secretion and motility as well as indirectly decrease gastric acid secretion by preventing the release of other hormones.5 Other functions of somatostatin include inhibiting secretions of the exocrine part of the pancreas, gallbladder contraction, and the absorption of glucose, triglycerides, and amino acids.5

F cells make up the remainder, about 6%, of the pancreatic islets and secrete a hormone called pancreatic polypeptide.1 The functions of pancreatic polypeptide include inhibiting somatostatin secretion, gallbladder contraction, and secretion of digestive enzymes by the pancreas. The secretion of pancreatic polypeptide is increased after a protein meal, fasting, exercise and acute hypoglycemia and decreased by somatostatin and intravenous glucose.1

**Blood Glucose Negative Feedback Cycle**

A negative feedback cycle reduces the output of a system. In *Principles of Anatomy and Physiology*,Derrickson and Tortora discuss how blood glucose levels control the secretion of glucagon and insulin through a negative feedback cycle. Hypoglycemia stimulates alpha cells to secrete glucagon. Glucagon acts on the hepatocytes to promote glycogenolysis and gluconeogenesis. Hepatocytes then release the glucose into the blood, raising blood glucose. If blood glucose gets too high and hyperglycemia occurs, the negative feedback system becomes effective and inhibits the release of glucagon from the alpha cells. Hyperglycemia stimulates beta cells to release insulin. Insulin decreases blood glucose by acting on various cells in the body to increase facilitated diffusion of glucose into cells, glycogenesis, protein synthesis, lipogenesis and decrease glycogenolysis and gluconeogenesis. If hypoglycemia occurs then negative feedback occurs again, inhibiting the release of insulin from beta cells and stimulating the release of glucagon from alpha cells. The negative feedback system maintains blood glucose around 90-100 mg/dL of plasma with a total of two to three grams of glucose normally circulating through the body.

**GLUCOSE METABOLISM**

Carbohydrates (CHO) enter the body as complex carbohydrates or simple carbohydrates. Complex carbohydrates include polysaccharides and oligosaccharides.6 Simple carbohydrates include disaccharides consisting of maltose, lactose, and sucrose and monosaccharides consisting of glucose, galactose, and fructose. Polysaccharides and disaccharides are nutritionally the most important carbohydrate, as free monosaccharides are not commonly found in significant quantities in the diet.6 Mostly, monosaccharides are absorbed through the gastrointestinal (GI) tract therefore; polysaccharides and disaccharides must be hydrolyzed to their constituent monosaccharide units. The disaccharide maltose gets broken down into two units of glucose, lactose gets broken down into glucose and galactose and sucrose gets broken down into glucose and fructose.6

Carbohydrate metabolism begins in the mouth.6 The first step is mastication where the food is chewed and polysaccharides are mechanically cut into smaller chunks. The salivary glands release an enzyme called salivary α-amylase that mixes with the food and cleaves links of the divided chunks, releasing monosaccharides.6 Since the food is only in the mouth for a short period of time, only a few monosaccharides are produced.4 The food bolus then travels down the esophagus and into the stomach where digestion continues. Salivary α-amylase continues working in the stomach until gastric acid penetrates the food bolus and lowers the pH, inactivating the enzyme.6 At this point of digestion, the carbohydrates have been partially hydrolyzed mostly into short chain polysaccharides and maltose, also known as dextrins. The food bolus is ground by peristalsis, repetitive and forceful contractions of the stomach, and transformed into chyme. Chyme is then slowly released from the stomach into the small intestine where further digestion of the dextrins continues.6 In the intestine, the pancreas secretes pancreatic juices into the duodenum. The presence of sodium bicarbonate in the duodenum elevates the pH to a level that enables the enzymes to function. The enzymes help to further breakdown dextrins in the duodenum into disaccharides and monosaccharides.6 Digestion of disaccharides occurs on the brush border of the intestinal tract by enzymes known as glycosidases including lactase, sucrase, maltase, and isomaltase. These glycosidases hydrolyze disaccharides to their constituent monosaccharide units.6 Monosaccharides along with small amounts of remaining disaccharides are then able to be absorbed by the intestinal mucosal cells, and by the end of the jejunum nearly all monosaccharides are absorbed. Glucose and galactose are absorbed through the intestinal tract by active transport, which requires energy and a specific receptor.6 Fructose is absorbed through the mucosal cell by a specific facilitated transporter called gluT5. The rate of absorption of fructose is much slower than glucose or galactose. After transporting across the intestinal wall, monosaccharides enter the portal circulation and are carried directly to the liver. The liver is where metabolism of galactose and fructose occurs and they are taken up by the liver through specific hepatocyte receptors and converted to glucose.6 Glucose is also metabolized by the liver and the remainder of it is passed into the systemic blood supply and distributed among other tissues like the muscle, kidney, and adipose tissue.6 Glucose entry of most cells throughout the body occurs by gluT molecules, transporters that bring glucose into the cells, through facilitated diffusion.1 In skeletal muscle and adipose tissue the process of getting glucose into the cells is insulin dependent.6 The use of glucose depends on the needs of the body’s cells:

* Adenosine triphosphate (ATP) production: in the cells of the body that require immediate energy, glucose is oxidized to produce ATP in a process known as cellular respiration. Insulin binds to the insulin receptor on the cell, allowing glucose to enter the cells. Glucose undergoes glycolysis, creating pyruvate which enters the mitochondria and gets broken down into Acetyl CoA. Acetyl CoA then enters the Krebs cycle and creates ATP.1
* Glycogenesis: insulin stimulates the hepatocytes and muscle fibers to combine hundreds of glucose monomers to form the polysaccharide glycogen. Glycogen is the only stored form of carbohydrate in the body. About 125 grams of glycogen can be stored in the liver and 375 grams of glycogen can be stored in skeletal muscles.1
* Amino acid synthesis: cells throughout the body can use glucose to form several amino acids that can then be incorporated into proteins.1
* Triglyceride synthesis: this is done when glycogen stores are all filled up and the hepatocytes transform the glucose to glycerol and fatty acids that can be used for lipogenesis. The triglycerides are then deposited into adipose tissue, which has an unlimited amount of storage capacity.1

**DIABETES MELLITUS**

Endocrine disorders develop as a consequence of the hyposecretion of hormones, the hypersecretion of hormones, or of the hyporesponsiveness of the organs.3 Diabetes mellitus (DM) is the most common of all endocrine disorders, with over 23 million people in the United States being diagnosed with this disease.4 Diabetes is the sixth leading cause of death in the United States, primarily due to its damage to the cardiovascular system.2 Average medical care costs for those with diabetes are at least doubled that of other people in the health care system, with most costs being related to chronic complications.4 The onset of diabetes can be contributed to both genetic and environmental factors.1 Diabetes mellitus is a disease characterized by hyperglycemia as a result of the body’s inability to metabolize glucose due to insulin resistance, deficiency in insulin secretion, or both, which causes alterations in carbohydrate, protein, and fat metabolism.4

Diabetes mellitus is classified into primary versus secondary diabetes. Primary diabetes is then separated into type I diabetes mellitus (TIDM) and type II diabetes mellitus (T2DM).4 TIDM occurs when there is a failure or destruction of the beta cells to produce and secrete insulin. T2DM occurs when the pancreas has a decreased ability to produce enough insulin to manage blood glucose or the patient is insulin-resistant and the amount of insulin secreted is less effective.4 Primary diabetes has genetic and environmental interactions that become apparent at varying times after birth. Secondary diabetes is when diabetes occurs secondary to other conditions like pancreatitis, pancreatic cancer, celiac disease or liver disease. Secondary diabetes is usually treated by attacking the primary disease and if the primary cause of diabetes cannot be treated then it must be dealt with like TIDM.4

**Type I Diabetes**

Only about 5-10% of all diagnosed cases of diabetes are type I.4 Onset of TIDM most often occurs in people under 30 years of age, although cases of diagnosed type I diabetes later in life are increasing.4 The gender distribution among people with type I diabetes is equal.4 Most often around the time of onset, the individual has experienced recent weight loss. TIDM has been referred to by many names including: insulin dependent diabetes mellitus (IDDM), juvenile diabetes, and brittle diabetes.4

The onset of TIDM is related to certain genetic susceptibility of the pancreas to viruses and autoimmune disease as well as environmental factors.4 Pancreatic beta cells can be destroyed by a virus that invades beta cells, causing them to become identified as a foreign structure that leads to an immune response.4 Type I diabetes can also be immune-mediated leading to an autoimmune destruction of beta cells of the pancreas. After the diagnosis of TIDM, antibodies to beta cells of the pancreas or insulin are often apparent in 85%-90% of cases.4 It is not clear what the cause of autoimmune destruction of beta cells is, but contributors to TIDM are known to be multiple genetic predispositions and unidentified environmental factors.4 Research has identified potential environmental factors that could be a trigger such as coxsackie virus, cow’s milk proteins, and rubella. There is also idiopathic diabetes where individuals develop TIDM without any known cause. This includes a minority of mostly Asian or African populations.4

The rate of destruction of beta cells varies in each individual but generally occurs faster in children and slower in adults.4 When clinical symptoms start to arise, about 60%-80% of beta cells are already destroyed. The alpha cells, delta cells, and F cells are not destroyed, but are redistributed within the pancreatic islets. After the beta cells are destroyed they can no longer produce insulin, causing a total insulin deficiency.4 Cells are not able to utilize glucose for energy so the cells starve and blood glucose rises. The patient becomes insulin dependent and insulin injections are required for the rest of their life to help control blood glucose and prevent death.4

The onset of the disease can present itself in many different ways. The first sign of type I diabetes is usually diabetic ketoacidosis (DKA), and can often be in the presence of physiologic stress.4 Other signs and symptoms that occur at the onset of TIDM include polyuria, polydipsia, polyphagia, weakness, fatigue, irritability, and sudden weight loss. In the event of hyperglycemia, the kidneys can only filter so much blood and the excess glucose spills into the urine, known as glucosuria, causing polyuria. The constant loss of fluid through the frequent urination stimulates the thirst mechanism, causing polydipsia.4 Many cells are dependent on glucose for energy, and with no insulin available the cells are starving which causes the individual to experience constant hunger, known as polyphagia.4 Insulin helps prevent fatty acid oxidation in the cells, but with the continuing of insulin deficiency the cells do not receive any glucose and lipolysis occurs. In liver cells, fatty acids are broken down by beta oxidation, and fed into the Krebs cycle where ketone bodies are created that can be used for energy by the muscle and brain cells. With the increased production of ketone bodies, the pH drops to about 7.3-6.8, ketone bodies are secreted into the urine, and acidosis occurs.4 Ketone bodies show up as an anion gap in metabolic acidosis which can be determined when subtracting serum chloride and serum bicarbonate from serum sodium is greater than 12. Hyperglycemia also leads to osmotic diuresis, which is when the excess glucose creates too many particles in the urine and the kidneys cannot reabsorb it all. This causes glucose to be excreted and fluid to go with as well, leading to dehydration. Dehydration will cause potassium to shift out of cells and get dumped, leading to a total body potassium and phosphate depletion outside of the cells and into the serum.4 With the onset of metabolic acidosis, the body will compensate with deep, labored respirations called Kussmaul respirations. With a decrease in total body water and total body potassium, sodium, magnesium, and phosphorus serum levels may be normal or elevated due to hypovolemia. Hypovolemia also accounts for increased hematocrit, hemoglobin, and protein, white blood cells, creatinine and serum osmolality. Weight loss is accounted for by hypovolemia and muscle catabolism in a person with ketoacidosis.4

**Diagnosis**

The criterion for diagnosis of diabetes is as follows4:

1. Hemoglobin A1c ≥6.5%
2. Casual plasma glucose (CPG) ≥ 200 mg/dL (11.1 mmol/L) plus symptoms of diabetes.
3. Fasting plasma glucose (FPG) ≥126 mg/dL (7.0 mmol/L) plus symptoms of diabetes. The FPG test involves an eight hour fast overnight followed by two to three measurements of plasma glucose.
4. Two hour postprandial glucose ≥200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test (OGTT). An oral glucose tolerance test measures blood glucose after fasting and two hours after drinking a glucose beverage. Administration of an OGTT is done after a minimum of three days with an unrestricted diet of at least 150 grams of carbohydrates each day and normal physical activity. An overnight fast of 8-14 hours is required before the test, in which only water is allowed to be consumed. A result of 140-199 mg/dL (7.8-11 mmol/L) indicates pre-diabetes. OGTT is rarely needed to diagnose TIDM because of the sudden onset of symptoms accompanied by hyperglycemia. It is commonly used to diagnose gestational diabetes, impaired glucose tolerance, and impaired fasting glucose. The oral glucose tolerance test is contraindicated in infants and young children.

Diabetes-related autoantibody testing is what physicians commonly use to distinguish between TIDM and T2DM. The antibodies are present in patients with type I diabetes and indicate that the body's immune system is destroying its own beta cells. Autoantibodies that are tested include islet cell cytoplasmic autoantibodies (ICA), insulin autoantibodies (IAA), glutamic acid decarboxylase autoantibodies (GADA) and insulinoma-associated-2 autoantibodies (IA-2A).4

The onset of type I diabetes happens rapidly with severe symptoms. After the development of the disease, there is sometimes a short period of partial remission called the honeymoon phase.4 Shortly after an individual is diagnosed with TIDM and an insulin regimen is started, there may be a restoration in insulin production by the beta cells for a period of time. During this period, the insulin requirements of a patient may decrease or even is eliminated. The autoimmune destruction of beta cells is still ongoing and insulin will eventually be needed again. After the honeymoon phase, the diabetes becomes more severe and little or no insulin is produced by the pancreatic beta cells.4

**Treatment**

Daily exogenous insulin along with nutrition therapy is necessary for survival in an individual with diabetes. The exogenous insulin must be correlated with nutritional intake and physical therapy to mimic what normal insulin secretion would be in an individual without diabetes.4 Insulin is classified based on expected onset of action, peak time of action, and duration of action (insulin types in Appendix). The types of insulin have different patterns and rates of activity and are often combined in the regimen to mimic normal insulin secretion as closely as possible.4 The initial dose of insulin is established using algorithms based on body weight. Generally, 0.3-0.5 units/kg is the starting dosage for patients who are within 120% of their ideal body weight (IBW). Insulin dosages are then adjusted based on blood glucose patterns.4 There is a variety of insulin regimens available for design based upon the patient’s meal routine, food choices, and lifestyle. Other factors for creating an insulin regimen include patient’s age, school/work schedule and circumstances, social environment and personality, cultural issues and other medical conditions.4 There are three different types of insulin regimens4:

1. Conventional therapies (“split” or “mixed” dose):
   1. Short- or rapid-acting insulin mixed plus intermediate-acting insulins given before breakfast and before evening meals.
   2. Combination of short- and intermediate-acting insulins before breakfast, short-acting insulin before evening meals, and intermediate-acting insulin at bedtime.
2. Intensive insulin therapy (multiple daily injections [MDIs]): intermediate insulin is given one to two times per day and rapid or short acting insulin is given before meals. This regimen allows for more flexibility in the type and timing of meals.
3. Continuous subcutaneous insulin infusion (CSII): basal, rapid-, or short-acting insulin given before meals, is pumped through subcutaneous catheter (insulin pump) and is monitored 24 hours a day.

The conventional for administering insulin is through a syringe or a pen. The syringes are designed for 100 units of insulin and have short, fine beveled needles that are disposable. The pens come with either 150 units or 300 units of insulin and are disposable or reusable with prefilled cartridges. Cartridges and prefilled pens are available with rapid-acting, regular, and extended long-acting insulins, some pre-mixed insulins, and glargine.4 Insulin pumps are another method of giving insulin. The pumps are about the size of pagers worn around the waste that deliver regular or rapid-acting insulin through flexible tubing that is attached to the patient by an infusion set. The pump provides continuous subcutaneous insulin infusion that allows the creation of variable and adjustable insulin dosing to meet the individuals specific needs. Detailed instructions, training, time and effort are needed to use and benefit from the insulin pump.4

Daily home glucose monitoring or self-monitoring of blood glucose (SMBG) is an important part of the treatment plan as it provides immediate feedback and data about blood glucose levels. The feedback and data can be used to improve and maintain target glycemic controls.4 SMBG can help prevent, detect, and treat hypoglycemia. It is also used to identify patterns and adjust insulin in response to changes in lifestyle, (ie, like eating and physical activity habits). For a glucose monitoring test, a drop of blood obtained by a finger prick is placed onto a chemically treated agent strip that is inserted into a home monitor to determine the results.4 Another method of monitoring blood glucose is through continuous glucose monitoring (CGM).4 For CGM a sensor is placed right under the skin that transmits the blood glucose levels to the insulin pump that is worn around the waist. The sensor does require calibration with SBMG and is recommended to be used in conjunction with SMBG for making acute treatment decisions. The continuous glucose monitoring device alarms the patient for events of hypoglycemia or hyperglycemia.4 Continuous glucose monitoring is an evolving technology and studies are showing that patients who wear the device most of the time obtain a benefit.7 Carefully monitoring and avoiding wide variations of blood glucose is important in preventing hypoglycemia, hyperglycemia, as well as short-term and long-term health complications. Hypoglycemia has more dangerous short-term complications as severe hypoglycemia can lead to unconsciousness, coma, and death.7 The Academy of Nutrition and Dietetics recommends that a person with TIDM should do at least three to eight blood glucose tests per day to determine the adequacy in insulin doses, food intake and physical activity.8 Some insulin regimens require more testing to establish the best insulin therapy, but once established the regimens may require less frequent SMBG. Those with unexplained elevations in hemoglobin A1C, unexplained hypoglycemia and hyperglycemia may benefit from CGM or more frequent SMBG.7 Determining the frequency and timing of self monitoring of blood glucose can be determined by incorporating these guidelines with the specific needs and goals of the individual with diabetes and the health care team. The American Diabetes Association recommends that CGM in conjunction with intensive insulin regimens can be effective in lowering hemoglobin A1c in selected adults, 25 years and older, with TIDM.8

**Short-Term Complications of Hyperglycemia**

Diabetic ketoacidosis is a short term complication of hyperglycemia, and as discussed earlier, and often the first sign of the onset of diabetes.3 DKA most often occurs in patients with type I diabetes and new onset of type II diabetes who are obese with impaired insulin secretion and insulin action. The most common causes of DKA in diabetics are omission of insulin, illness, infection, and emotional stress. Often when individuals are sick, they may not take their insulin because they feel too sick to eat, or because they are afraid of developing hypoglycemia.3 Diabetic ketoacidosis develops rapidly with the most common symptoms including polyuria, polydipsia, weight loss, nausea, vomiting, abdominal pain, fruity or acetone breath, Kussmaul respirations, and mental status changes.4 DKA is characterized by blood glucose >250 mg/dL, arterial pH <7.0 to 7.30, serum bicarbonate <10 to 18 mEq/L, positive for urine ketones, positive for serum ketones, and variable serum osmolality.4 As a result of diabetic ketoacidosis, osmotic diuresis occurs, causing dehydration and electrolyte imbalances. Treatment usually involves hospitalization for assessment and/or administration of IV fluids, insulin, and electrolytes.4 Most DKA cases are resolved, but unfortunately, 2% to 5% of cases are fatal.6

Hyperglycemic hyperosmolar syndrome (HHS) most often occurs in type II diabetics between the ages of 55 and 70 years old.4 The most common causes of HHS include dehydration from inadequate fluid intake or excess fluid losses and prolonged hyperglycemia. Hyperglycemic hyperosmolar syndrome progresses slowly with symptoms of undiagnosed diabetes, polyuria, polydipsia, progressive decline in level of consciousness, fever and volume depletion. HHS is characterized by blood glucose >600 mg/dL, arterial pH >7.3, serum bicarbonate >15 mEq/L, small urine ketones, small serum ketones, and serum osmolality >320 mOsm/kg of water, and absence of significant ketoacidosis.4 Treatment includes hospitalization for slow rehydration and to treat underlying medical problems.4 Mortality rate for HHS is much high than DKA at 15%.6

**Long-Term Complications of Hyperglycemia**

Long term hyperglycemia results in microvascular and macrovascular complications that increase mortality and morbidity associated with diabetes and reduce quality of life. These chronic complications typically occur in individuals 15-20 years after the onset of diabetes.4 The consequences of untreated hyperglycemia can include long-term damage, dysfunction, and failure of various organs including the eyes, heart, nerves, kidneys, and blood vessels.1 This is why diabetes is the leading cause of blindness, renal failure, and amputations and the leading cause in birth defects.1 Over 200,000 people die each year from complications due to untreated diabetes.4

Untreated hyperglycemia causes high amounts of glucose to build up in the cells that are not insulin dependent and that glucose is then converted to fructose and sorbitol. Once sorbitol is collected intracellularly, it cannot be degraded and transported out of the cell and blocks energy metabolism.4 Proteins or lipids become glycated after being exposed to sugar, meaning that lipids or proteins are bonded with a sugar molecule without the controlling action of an enzyme.4 Normal macromolecules that become glycated are known as Advanced Glycation Endproducts (AGE). The AGEs accumulate in many cell types and affect extracellular and intracellular function. This creates rigid blood vessels and thickened capillary basement membranes that increase the attraction of monocytes to lower density lipoproteins, activating the atherosclerosis process. These complications decrease circulation in the blood vessels and can lead to a variety of microvascular and macrovascular complications including disease in the heart, kidneys, retina and nervous system.4

* Cardiovascular disease (CVD) is the most common cause of death in individuals with diabetes, with about 65% of deaths due to heart disease or stroke.4 The risk of death from CVD is two to four times greater in individuals with diabetes.4 Long term hyperglycemia leads to thickening and changes in composition of the sub-endothelial layer.1 In individuals with hyperglycemia, the body uses lipolysis as a means to make energy and after the triglycerides are broken down they are transported to the cells. During the transport, lipid particles are deposited on the walls of blood vessels which lead to thickening and decreased flexibility of the vessels.1 This increases blood pressure, accelerates atherosclerosis and leads to a multitude of other cardiovascular problems including cerebrovascular insufficiency, ischemic heart disease, peripheral vascular disease, and gangrene.1 This makes diabetes an independent risk factor for macrovascular disease along with common coexisting factors of hypertension and dyslipidemia.4
* Nephropathy is the leading cause of chronic kidney disease (CKD) and occurs in 20% to 40% of individuals with diabetes.4 CKD can only be treated with dialysis or kidney transplantation. Hyperglycemia causes changes in the structure of the blood vessels of the glomerulus. The nephrons thicken and become scarred over time which increases the permeability and decreases filtering ability in the kidneys. The kidneys start to leak albumin which spills over into the blood. Persistent albuminuria in the range of 30-299 mg/24 hours, also known as microalbuminuria, is the earliest stage of neuropathy in those with TIDM.4 Hypertension is often the consequence of underlying nephropathy in those with TIDM. Hypertension is also a complication for microvascular complications of diabetes including nephropathy. It can take 5-10 years before symptoms of CKD appear. Progression of CKD can be decreased by intensive diabetes management, protein restriction and use of angiotensin-converting-enzyme-inhibitor (ACE) inhibitors.4
* Retinopathy is the most frequent cause of blindness in adults, with prevalence of retinopathy being associated with duration of diabetes.4 The eye is highly vascularized and has a high oxygen demand. The mechanisms of retinopathy are not completely understood, but it is thought that the damage to the eye is directly related to the hyperglycemic damage to the blood vessels. Major factors associated with retinopathy include changes in blood vessels and accumulation of sorbitol.4 Hypertension is also a complication for microvascular complications of diabetes including retinopathy and is a risk factor for macular edema. Excessive blood glucose attaching to the protein in the lens can cause cloudiness and glaucoma and cataracts occur earlier in patients with diabetes. Progression can be controlled with glycemic control and lowering of blood pressure.4
* Nervous system disease occurs in approximately 60% to 70% of individuals with diabetes.4 Nervous system disease causes impaired sensation or pain in the feet or hands, slowed digestion of food in the stomach, carpal tunnel syndrome, and other nerve problems. As a result of long term hyperglycemia, the accumulation of sorbitol and glycated proteins leads to the cellular damage and disrupts the normal nervous system pathways. The lesions that develop on the nerves affect the autonomic nervous system and affect many organ systems including the gastrointestinal tract, cardiovascular system, and genitourinary tract.4 Neuropathy lead to damage of the vagus nerve causing the body to have decreased control over digestive functions and lead to paralyzed nerves of the stomach known as gastroparesis. The most common symptom is constipation but can alternate with episodes of diarrhea. Gastroparesis can cause anorexia, nausea, vomiting, early satiety, postprandial bloating, and erratic glycemic control. Common genitourinary tract disturbances associated with autonomic neuropathy include bladder and/or sexual dysfunctions. These disturbances are marked by recurrent urinary tract infections, pyelonephritis or incontinence.4 Cardiac autonomic neuropathy (CAN) is the most clinically important effect of neuropathy. It is manifested through tachycardia, heart rate greater than100 beats per minute; orthostatic hypotension, a fall is systolic blood pressure >20 mmHg upon standing; or increased risk of silent heart disease.4

**EATING DISORDERS**

According to the American Psychiatric Association *The Diagnostic and Statistical Manual of Mental Disorders (DSM)* is the standard classification of mental disorders used by mental health professionals in the United States.9 This standard is intended to be used by clinicians and researchers of multiple orientations. The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)* has been designed with the intention to be used in multiple settings including inpatient, outpatient, partial hospital, consultation-liaison, clinic, private practice, primary care, and community populations.9

The three major components of the DSM include9:

1. The diagnostic classification, which is a list of mental disorders that are officially part of the DSM system. A “DSM diagnosis” would be done by selecting a disorder from the classification that best reflects the signs and symptoms that the individual exhibits.
2. The diagnostic criteria sets, which indicate what symptoms must be present and what symptoms, disorders and conditions must not be present to qualify for a particular diagnosis. The use of diagnostic criteria has been shown to increase diagnostic reliability, nonetheless, these criteria are to be used as guidelines informed by clinical judgement.
3. The descriptive texts, which accompanies each disorder and systematically describe each of them with the following headings, “Diagnostic Features”, “Subtypes and/or Specifiers”, “Recording Procedures”, “Associated Features and Disorders”, “Specific Culture, Age, and Gender Features”, “Prevalence”, “Course”, “Familial Pattern”, and “Differential Diagnosis”.

The following categories of Anorexia Nervosa and Bulimia Nervosa are excerpts from the *DSM-IV* from the American Psychiatric Association9:

**Anorexia Nervosa**

1. Refusal to maintain body weight at or below a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of the expected.)
2. Intense fear of gaining weight or becoming fat, even though underweight.
3. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
4. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

Type

1. Restricting Type: During the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behavior (self-induced vomiting or misuse of laxatives, diuretics, or enemas).
2. Binge Eating/Purging Type: During the episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behavior.

**Bulimia Nervosa**

Bulimia Nervosa is recurrent episodes of binge eating characterized by the following:

1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
3. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications, fasting or excessive exercise.
4. The binge eating and inappropriate compensatory behavior both occur, on average, at least twice a week for three months.
5. Self evaluation is unduly influenced by both body shape and weight.
6. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

Type

1. Purging Type: During the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.
2. Nonpurging Type: During the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behavior but has not regularly engages in self-induced vomiting, or misused laxatives, diuretics, or enemas.

**EDNOS (Eating Disorder Not Otherwise Specified)**

EDNOS are disorders that do not meet the criteria for any specific eating disorder.2

The Academy of Nutrition and Dietetics discusses the overview of eating disorders and the impact of the disease on nutrition. The associated nutritional and physiological complications of Anorexia Nervosa mimic those of starvation and malnutrition.7 These complications are primarily due to the effect of starvation and can be life-threatening; including amenorrhea, estrogen deficiency, electrolyte imbalances, osteoporosis, abnormal thermoregulation, anemia, dehydration, disruption in renal and gastric function, cardiac abnormalities, anxiety and depression. Irreversible complications can include disturbance in bone development, failure to reach peak bone density, and possible development of osteoporosis.7 The cycle of bingeing and purging is rooted from an abnormally low self-esteem tied with a variety of possible psychiatric and/or psychosocial issues. Having the fear of being unaccepted is acted out in the form of a fear of weight gain, where someone who perceives themselves as essentially flawed translates that fear into dissatisfaction with his or her body.7 This is the basis for the body size and shape misperception generally seen in patients with eating disorders. Purging behaviors arise as both a method to diminish perceived weight gain from the food consumed and improving mood, a feeling of control, as assuaging guilt about eating. Binging can also be used for reasons such as boredom, loneliness, anxiety, relaxation, avoidance and numbing.7 According to the Academy of Nutrition and Dietetics, purging methods are not necessarily effective in preventing weight gain from binge eating, and individuals with bulimia may be at normal or above-normal weight. An individual with bulimia may also be underweight and if the individual is less than 85% of expected weight, the diagnosis would be Anorexia Nervosa-Purging Type.7

**DIABULIMIA**

Although type I diabetes mellitus can occur at any age, it generally has an early onset in life and the majority of patients at diagnosis are children or adolescents. Type I diabetes is one of the most common chronic illnesses of childhood and adolescence.2 But whether the patient is a child, adolescent or an adult, this diagnosis indicates a lifestyle change that requires subsequent psychological adaptation, which does not always occur and can be followed by frustration and non-acceptance of the disease. Those with TIDM are subjected to have to choose foods selectively and carefully and then decide their dose of insulin based on how many carbohydrates are to be eaten.2 Those living with TIDM may often feel burden by this illness and think of themselves as being different from others.10 The diagnosis of diabetes may often be made after a sudden weight loss and while adjusting to a new insulin regiment and diet, weight gain may occur.10 The patient can have a perceived restraint of food choice and weight management can be difficult, which can lead to a sense of loss of control.11 In general, teenage girls tend to have body image issues that can even continue into their adult years. It may be hard for teenagers to cope with this diagnosis as they are trying to fit in and be accepted by their peers. The dissatisfaction of body image and the need to focus on food intake can become obsessive and lead to an eating disorder in patient with TIDM.2 Eating disorders in general tend to affect adolescents, young adults and women as they lean towards dieting or exercising to lose weight and are often dissatisfied with their body image.2 There have been several studies reporting an increased rate of young adults with type I diabetes developing eating disorders.2 Some of the most common risk factors among those with type I diabetes developing an eating disorder include being a young female, a history of dietary restraint and dieting, weight gain, low self-esteem, and family dysfunction.11 It is estimated that between 11.5% and 27.5% of adolescents with TIDM meet the diagnostic criteria for an eating disorder and up to 30% mismanage their insulin to lose weight or avoid weight gain.11 In a study that looked at 143 adolescents with type I diabetes who completed the Assessing Health and Eating among Adolescents with Diabetes Survey, 37.9% of the females and 15.9% of the males practiced unhealthy weight control. Of the 37.9% of females, 10.3% reported skipping insulin and 7.4% reported taking less insulin as a means to control weight and only one male reported these behaviors.2

In *Deliberate Insulin Underdosing and Omission Should Be Included in DSM-IV Criteria for Bulimia Nervosa,*12 Albert Shaw and Armando Favazza discuss a case study of a 22-year old female with TIDM that was referred to their psychiatric clinic because of her bulimia, anxiety and depression. She began bingeing during college and felt as if she was out of control so decided to disconnect her insulin pump overnight after bingeing. Her rational was that disconnecting her insulin pump would cause the same amount of weight loss as had occurred in high school before she was diagnosed with type I diabetes. Although omitting her insulin eased her anxiety about gaining weight, her hemoglobin A1c nearly doubled and her guilt and embarrassment caused her to avoid her family and endocrinologist appointments. Shaw and Favazza urge the inclusion of insulin misuse as a part of the recurrent inappropriate compensatory behaviors to prevent weight gain in Bulimia Nervosa into the DSM-IV.

Diabulimia, derived from the words diabetes and bulimia, is an eating disorder in which an individual with TIDM intentionally manipulates their insulin in an inappropriate way with the intent to lose weight or prevent weight gain.2 The manipulation of insulin is a balancing act in which they can manipulate their insulin by completely omitting their insulin, intentionally giving themselves less insulin that needed, or intentionally skipping insulin doses at only some meals to avoid DKA.13 When insulin is restricted, blood glucose increases and calories are purged as glucose is spilled through the urine.2 Diabulimia is not yet recognized as an official eating disorder and many clinicians do not recognize the dual condition as a disorder, but there have been cases of TIDM combined with eating disorders since the 1970s and 1980s.13 According to Dr. Goebel-Fabbri10, “Insulin restriction equals calorie purge, which is a symptom of an eating disorder.” The term diabulimia did not actually being to gain attention until around 2007 within the health community, journals, and magazines.13 Due to the significant medical complications and risks associated with this practice, it is gaining a lot of attention today from many different professionals in the medical field. There are not set criteria as to how frequently or how long the omission of insulin must occur to diagnose and individual with diabulimia, but some clinicians propose the following as the definition13: “an insulin reduction at least twice a week or of over one quarter of the prescribed insulin for the purpose of weight loss for more than three months.”

In the article, *Diabulimia: What It Is And How to Treat It,*13Grace Shih lists common warning signs to be aware of if you suspect an eating disorder in an individual with TIDM. Below islisted the following signs to look for in an individual who may be suspected to have diabulimia:

1. Consistent high hemoglobin A1c.
2. Frequent visits to the emergency room for diabetic ketoacidosis; although some patients will skip the use of rapid-acting insulin and continue the use of their basal insulin, therefore, they may not experience DKA.
3. Body image concerns: with an increase of drive for thinness and body dissatisfaction.
4. Irregular eating patterns: the eating behavior of an individual with diabulimia will mimic those of an individual with Bulimia Nervosa. The repetitive cycle includes restricting, skipping meals, or eliminating particular food for the purpose of losing weight, followed by an intense over eating and overwhelming feeling of guilt, followed by the restriction by limiting their eating or their insulin. This type of eating behavior slows down the metabolism, and lasting weight loss will rarely occur.
5. Discomfort eating around other people: due to their irregular eating behavior and especially when the individual is having the urge to overeat. If they are eating with family or friends, they may only choose small or portions of the foods with fewer calories, but when they are alone they might binge.
6. Hoarding food: when insulin is restricted, nutrients cannot get into the cells and the cells become hungry, making the person feel hungry and crave food. The individual may feel ashamed, guilty, or defeated when they lose control over their hunger and may hoard foods in order to eat alone during their weak moments.
7. Irregular or nonexistent menses: elevated hemoglobin A1c levels have shown to be indicative of irregular menses, cessation of periods, and delayed puberty. This is due to interference with function of the brain.
8. Unwillingness to follow through with appointments: a combination of the lack of awareness along with the individual being unwilling to get well can make follow-up treatment very difficult for a patient with diabulimia. The lack of awareness and information for the families along with accepting that their family member has an eating disorder may also provide complications for follow-up treatment for the individual.
9. Doubtful glucose monitoring: the numbers on the glucometer may be too good to be true. A patient reported that a specific proportion of water and milk replacing blood on the test strip produced an ideal reading of blood glucose below 200 mg/dL. This is how she convinced the health care team and her family that she checked her blood sugars and that they were normal. If her mother became suspicious of the normal readings, she would mix water with juice to increase the blood sugar level for the reading. Also, the individuals often claim forgetfulness, like forgetting to bring their glucometer, forgetting to enter carbohydrates consumed onto the insulin pump, or forgetting the trend of their blood glucose.

Diabulimia is associated with impaired metabolic control, frequent episodes of DKA, earlier onset of the microvascular and macrovascular complications and even death.13 The article *Insulin Restriction and Associated Morbidity and Mortality in Women with Type I Diabetes*14discusses a follow-up study by Ann Goebel-Fabbri that investigated whether insulin restriction reported at baseline had higher rates of diabetes complications and increased risk of mortality in women with type I diabetes over a decade later. There were 234 women followed in the study, 71 of which reported insulin restriction at baseline. There were distinct clinical differences between those who restricted insulin and those who used insulin appropriately. The insulin restrictors scored lower on baseline measures of self-care behaviors and scored higher on baseline measures of diabetes distress, fear of hypoglycemia, general psychological symptoms and bulimia and other eating disorder symptoms. Insulin restrictors at baseline were more likely to report nephropathy and foot problems at follow-up. Using the multivariate Cox analysis, self-reported insulin restrictors at baseline had an increase risk of death by 3.2 times and overall the insulin restrictors died at a younger age than those who reported appropriate insulin use.

**MEDICAL NUTRITION THERAPY**

Medical care through a multidisciplinary team in which all team members are experts in dealing with eating disorders and/or diabetes is essential in treating an individual with diabulimia. Members of this team can include a registered dietitian, endocrinologist, psychiatrist, and primary care physician. Depending on organ systems involved and family members involved, other members of the team may include a dentist, family therapist, a nephrologist, cardiologist, psychiatric nurse practitioner, physical therapists and/or other specialists.13 Communication is key throughout the team as the possibilities of manipulation or deception on the part of the patient can delay or derail the treatment.13 The role of a dietitian in this team is to help the individual manage both the type I diabetes and bulimia through medical nutrition therapy (MNT). Diabulimia is an emerging condition in the medical field and there are no set guidelines created to treat diabulimia, therefore, it is important that the dietitian provides only evidenced based medical nutrition therapy to the patient. The MNT should be individualized to the patient and, is sensitive to their personal needs, willingness to change, and ability to make changes.7 The dietitian should provide ongoing nutrition management through continuous assessments, care plans, treatment goals, desired outcomes, and monitoring metabolic parameters.3 Medical nutrition therapy should help the individual make effective lifestyle changes. Therapy regarding diabetes should be provided by a registered dietitian (RD) that is familiar with components of diabetes MNT. When managing the TIDM part of diabulimia, primary goals include improving HemoglobinA1c and lipid profile management, reducing risk for onset and progression of co-morbidities, and reducing hospital readmissions and hospital stays.8 Therapy regarding an eating disorder like bulimia should be provided by an RD that is familiar the components of MNT for eating disorders. The primary goals of managing bulimia include improved health/nutritional status, decreased use of purging and improved relationship with food, self, body and others.15 An RD that is familiar with diabetes management and eating disorder management may not be prevalent everywhere and a collaborative effort between two dietitians may be the best option for an individual with diabulimia.

To promote the desired outcome for an individual with TIDM, The American Diabetes Association recommends an initial series of three to four encounters of 45-90 minute sessions within three to six months of diagnosis with a registered dietitian for MNT for diabetes.8 Depending on the nutrition assessment, learning needs and progress towards the desired outcome, the RD will then determine if and how many additional encounters of medical nutrition therapy are needed after initial session.16 At least one annual encounter is recommended to continue to reinforce lifestyle changes as well as to evaluate and monitor outcomes that impact the need for changes in MNT.16

**NUTRITION ASSESSMENT AND MONITORING**

The Academy of Nutrition and Dietetics states the following factors in the assessment of a patient with TIDM and Bulimia8, 15:

Assessment

* Clinical History
  + Height
  + Weight
    - Usual body weight (UBW)
    - Current weight
    - Percentage of weight changes
  + Body Mass Index (BMI)
  + Diet History
    - Dietary intake
  + Waist Circumference
  + Visual acuity
  + Blood pressure
  + Absence of menstruation?
  + Increased hunger, thirst, urination
  + Nausea, vomiting, abdominal pain
  + Perfectionism, obsessive-compulsiveness, dysphoria
  + Parental Authority Questionnaire (PAQ)
  + Eating Disorders Inventory-2 (EDI-2)
  + Eating Attitudes Test (EAT-26)

Monitoring body weight in these individuals depends on their medical stability and current weight status. In some cases, when patients are aware of their weight it can trigger feelings of shame, inadequacy, or even for bingeing and/or purging.15 Determining whether or not weight will be monitored should be a team decision as well as the choice to allow or not allow the patient to see his or her weight. If the weight is stabilized at a low energy intake, then small increases in energy may help to increase the metabolic rater over time. This process should be explained to the patient along with basic metabolic concepts. The patient should have a good understanding that going back to a weight-loss diet will possibly retrigger the binge/purge cycle.15

The Parental Authority Questionnaire (PAQ) is a questionnaire with three subscales of ten questions each that measures the dimension of authoritative parenting style, authoritarian parenting style and permissive parenting style. The subjects select from five point scale the extent of their agreement with each statement.17 The Eating Disorders Inventory-2 (EDI-2) is a self report questionnaire that measures the presence of an eating disorder by assessing two dimensions of perfectionism: self-oriented and socially prescribed perfectionism.18 The Eating Attitudes Test (EAT-26) is a self-reporting measure that can be given in individual and group settings that measures symptoms and concerns for eating disorder.19 These tools are only meant to identify an eating disorder not to make a diagnosis. The Revised 16-item Diabetes Eating Problem Survey is another self-report test that is a diabetes-specific measure of disordered eating. The survey incorporates questions regarding weight concerns, eating patterns, control and purging, including self-induced vomiting and maladaptive insulin use.11

* Lab work
  + HbA1c (goal <7%)
  + Fasting blood glucose (FBG) (goal 90-130 mg/dL)
  + OGTT results
  + Urine glucose
  + Blood urea nitrogen (BUN) & Creatinine (Creat)
  + Microalbuminuria
  + C-Reactive Protein (CRP)
  + Total Cholesterol (Chol)
  + High-density lipoprotein (HDL)
  + Low-density lipoprotein (LDL)
  + Triglycerides (Trig)
  + Urinary ketones
  + Na+, K+, Ca++, Mg++
  + Serum phosphorous (PO4)
  + Thyroid-stimulation hormone (TSH)
  + Serum D3 level
  + Serum cortisol
  + Serum folate
  + Hemoglobin and Hematocrit
  + Albumin
  + Serum amylase (high)
  + LH, FSH (may be low)

**NUTRITION INTERVENTIONS**

The patient’s intervention should be individualized. The Academy of Nutrition and Dietetics provides objectives to follow while developing the proper intervention. There are established guidelines for an individual with TIDM as well as an individual with bulimia8,15:

**Type I Diabetes Mellitus**

* An initial series of three to four encounters of 45-90 minutes should be implemented at the beginning of the diagnosis of diabetes and should be completed within three to six months.
* Regularly evaluate food/nutrition history, physical exercise, activity patterns, excessive weight gain.
* Address the metabolic abnormalities of glucose, lipids, and blood pressure. Modify drug therapy to enhance outcomes and quality of life and prevent early onset of complications. If there are complications, delay or prevent consequences.
* Promote lifestyle changes according to personal needs, client readiness, ability to make changes of the individual, educational, and skill level. Early referral for lifestyle changes and advice yields the most benefit.
* Develop food/meal planning with client and emphasize the importance of key nutrients along with nutrient density and the impact on health, appearance, and stamina. Share the meal plan with medical team so an insulin regimen can be integrated into the client’s usual lifestyle.
* Plan meal plan, exercise, and medication to achieve blood glucose and lipid goals. Minimize intake of trans fatty acids; saturated fat should be <7% of total calories.
* Educate on how to use CHO counting and the proper way to adjust insulin doses based on planned CHO intake. Both the grams of carbohydrate as well as the type of carbohydrate in food influence blood glucose level. Monitoring total grams of CHO remains a key strategy in achieving glycemic control.
* Educate on SMBG to be done multiple times per day, and more often during illness.

Carbohydrate counting follows the rule that one unit, or one serving, of carbohydrate is 15 grams of carbohydrate. Standard starch, fruits, sweet, or milk servings are based on 15 grams CHO (full table of carbohydrate exchanges in Appendix).20 The individualized meal plan and physical activity regimen should then be used as the basis for integrating insulin therapy, which may require collaboration through the health care team.8

Determining the proper insulin to CHO ratio and incorporating consistent carbohydrates into each meal and snack with set doses of insulin is beneficial for improved glycemic control.3 An insulin to CHO ratio is a mechanism used to determine insulin dosage based on CHO intake. A general starting point is one unit of rapid-acting insulin taken for every 10-15 grams carbohydrate and adjusted based on self monitoring of blood glucose.3 For fast acting insulin (Humalog or Novolog) to CHO ratio, divide 500 by the total daily dose of insulin for the approximate grams of CHO covered by one unit of insulin. For Regular insulin to CHO ratio, divide 450 by the total daily dose of insulin for the approximate grams of CHO covered by one unit of insulin.3 If blood glucose rises above normal range during meal time, SMBG records can help to determine if a correction factor should be used to help return blood glucose levels within normal range. The correction factor is one unit of insulin to be given for every 50 mg/dL that blood glucose rises above 150 mg/dL (Meal time insulin = insulin:CHO ratio + correction factor).3 Generally, those on an insulin regimen should eat at consistent times of the day synchronized with the time-action of the insulin preparation used as well as monitor their glucose levels, and determine their required insulin doses for the amount of food usually eaten.3 More flexibility in the timing of eating meals and snacks and the amount eaten is allowed with intensive insulin therapy, including multiple daily injections, continuous subcutaneous insulin infusion with an insulin pump, and rapid-acting insulin. Meal skipping among this population should be discouraged to avoid hypoglycemia.3

**Bulimia**

* Counteract lowered metabolic rate with balanced diet and exercise. When exogenous insulin is restricted after a meal, the cells in the body are not receiving any glucose for energy and are starving. This will decrease the metabolic rate.
* Individualize care plans to regularly evaluate weight history, food and nutrition history, dieting and binge eating episodes, meal and physical activity patterns.
* Encourage physical activity to help decrease negative mood, improve eating disorder and help with weight loss or weight maintenance.
* Promote effective weight control along with stress management. Establish a target weight in accordance with present weight, desirable BMI, and reasonable time frame for recovery.
* Educate on dieting and nutrition myth and facts, meal planning, energy needs and use by the body, acceptance of “imperfect” eating, negative effects of bulimic behaviors while attempting to minimize guilt and shame.
* Educate on hunger and satiety cues to determine when to eat, instead of using emotions. In the early stages of treatment patients may not be able to identify or follow hunger and satiety cues, therefore, encourage a structured meal plan.

**NUTRITION PRESCRIPTION**

**Type I Diabetes Mellitus**

Development of a meal plan should be individualized to the patient’s usual food intake. Nutrient dense foods should be incorporated into the daily meal plan, including five servings of fruits and vegetables, six servings of grains with at least half being whole grain, and two servings of low-fat dairy.3 Foods in the meat and fat group do not affect blood glucose but should still be included in the meal plan. In regards to a patient with TIDM, carbohydrate counting and the source of carbohydrate should be the main focus when developing a meal plan.3

There is no research to support any ideal percentages of macronutrients for patients with diabetes. Nutrition guidelines such as the Dietary Reference Intakes (DRI) that apply to the healthy, adult population also apply to persons with diabetes.16 Methods for determining energy needs are only approximate but depend on factors including age, sex, height, weight, and physical activity. Adjustments of energy intake should be made during follow up visits based on activity level and life stage changes.8

Daily total energy intake requirements follow the DRIs21:

* Carbohydrates: 45-65%
* Protein: 10-35%
* Fat: 20-35%
  + Saturated fats should be limited to <10%

The Academy of Nutrition and Dietetics provides recommendations for food and nutrition 8:

* Carbohydrate intake will vary for males and females, activity levels and meals and snacks. Women should have 3-4 carbohydrate choices per meal for 45-60 grams of carbohydrates; Men should have 4-5 carbohydrate choices per meal for 60-75 grams of carbohydrates; active young women should have 5-6 carbohydrate choices per meal for 75-90 grams of carbohydrate; and active young men should have 6-7 carbohydrate choices per meal for 90-100 grams carbohydrates. As for snacks, 1-2 carbohydrate choice or 15-30 grams carbohydrates should be used.
* Protein intake should be limited to recommended dietary allowance of 0.8-1 grams of protein/kg in order to reduce the risk of nephropathy. If there is any microalbuminuria, a more controlled protein intake may be required.
* Fat intake should include a high monounsaturated fat diet which seems to have a favorable effect on lipoproteins in diabetes. Omega-3 fatty acids should be included from sources like salmon, mackerel, tuna, walnuts, and canola oil to help control blood lipids and reduce inflammatory processes. Fried and creamed foods should be cut back or eliminated.
* Fiber intake should be 14 grams per 1000 calories and diets with 44-50 grams of fiber help to improve glycemia.
* Mineral supplementation is not advised, but the adequacy of intake should be ensured. Potassium and magnesium should be replenished if necessary.
* Adequate calcium is important: 500 mg in 1-3 year olds, 800 mg in 4-8 year olds, 1300 mg in 9-19 year olds, and 1000 mg in adults should be attained daily.
* Dietary sodium should be limited to 2400 mg or less.
* Folate is important in women of childbearing ages: 400 µg before pregnancy and 600 µg during pregnancy.
* Vitamin D has been shown to have an important role in autoimmune disorders, therefore, include vitamin-D fortified foods in the meal plan and encourage adequate time in the sun.
* Alcohol intake should be limited to 1 drink per day or less for women and 2 drinks per day or less for men. One drink is 12 oz. beer, 5 oz. wine, or 1.5 oz. distilled spirits. Alcohol should be combined with carbohydrate containing food to prevent hypoglycemia.
* Adequate carbohydrate replacement during and after exercise is important to prevent hypoglycemia. Decrease rapid-acting insulin doses during the physical activity; 30-50% less is reasonable.

In critically ill patients, maintenance of blood glucose is a challenge but extremely important to prevent hyperglycemia or hypoglycemia. Regular blood glucose monitoring and insulin replacement are necessary as hyperglycemia reflects illness severity and results in deleterious consequences. Enteral feeding is preferred and specialty formulas are not required. If parenteral nutrition is needed, strict blood glucose control should be used; plan 30% of nutrient intake as fat, 50% as CHO, and 15-20% as protein unless other disease states require alternative plans.8

**Anorexia Nervosa**

The Academy of Nutrition and Dietetics provides guidelines regarding the nutrition prescription for an individual with Anorexia Nervosa.22 As discussed earlier, an individual with bulimia may also be underweight and if the individual is less than 85% of expected weight, the diagnosis would be Anorexia Nervosa-Purging Type.22 Although an individual with diabulimia may be classified with Anorexia Nervosa-Purging Type, a nutrition prescription following the needs of an anorexic would not be appropriate. A recommended amount of calories and macronutrients can be prescribed and met in an individual with diabulimia, but their bodies will continue to starve and weight will not be gained until they are compliant with their insulin regimen. The appropriate caloric and macronutrient needs must be coupled with appropriate insulin use in order to reach treatment goals and desired outcomes. In the case of a diabulimic, the restricting of insulin is a characteristic of purging, as seen in Bulimia Nervosa.22

**Bulimia Nervosa**

If the patient is interested in losing weight, the RD should discuss healthful weight loss methods and make it clear with the patient that their current cycle of restricting and bingeing/purging needs must be addressed first. Eating patterns must be normalized and the binge/purge cycle must be interrupted.15 According to the Academy of Nutrition and Dietetics15, a person with bulimia may be of normal body weight but may be hypometabolic, which must be taken into consideration when calculating energy needs. If direct or indirect resting energy expenditure (REE) can be measured, then 120 % to 130% of the REE may be “normal”. Mifflin-St Jeor equation can be used if direct measurement of REE is not available. The nutrition prescription can be 100% of the predicted resting energy expenditure if the person is suspected to be hypometabolic.15

As with type I diabetes, meal planning should be individualized for a patient with bulimia. Factors that should be considered in meal planning include food fears, patient acceptance, and stage of recovery.15 Nutrition guidelines such as the Dietary Reference Intakes that apply to the healthy, adult population also apply to persons with bulimia.15 Adjustments of energy intake should be adapted as needed and advanced as tolerated.

Daily total energy intake requirements follow the DRIs21:

* Carbohydrates: 45-65%
* Protein: 10-35%
* Fat: 20-35%
  + Saturated fats should be limited to <10%

Regular meal patterns and times should be encouraged to help prevent the urge to binge. If there are particular times throughout the day where the patient is more likely to binge or has the highest level of hunger, the dietitian can incorporate high fiber and/or high protein snacks in the meal plan around that time to help promote satiety and even out blood sugar levels.15

Recommending a food and feelings journal, documenting food intake and related feelings and behavior, may help identify emotional triggers and focus on more productive ways to deal with emotions. The dietitian and mental health professional can work together to review the journal and help develop strategies to help or at least delay binges and purging.15

**NUTRITION GOALS**

**Type I Diabetes Mellitus**

The Academy of Nutrition and Dietetics provides main goals for nutrition therapy as follows8:

* Create a meal plan around the patient’s appetite, food preferences, usual eating and physical activity habits.
* Integrate insulin therapy with a meal plan based off of the patient’s food preferences and physical activity
* Assist in reaching and maintaining optimal metabolic outcomes for glucose, lipids, and blood pressure.
* Improve health through healthful food choices and physical activity
* Respect the individual’s wishes, willingness, and ability to change

Laboratory goals8:

* Glucose: fasting or random blood glucose measurements can be done at regular office visits. In patients that are meeting treatment goals and have stable glycemic control, hemoglobin A1c should be done at least two times per year. In patients who are not meeting treatment goals or whose therapy has changed, hemoglobin A1c should be done quarterly.

**Blood Glucose Goals**

|  |  |
| --- | --- |
| Hemoglobin A1c | < 7% |
| Preprandial plasma glucose | 70-130 mg/dL |
| Peak postprandial plasma glucose (1-2 hours after the beginning of a meal) | <180 mg/dL |

Academy of Nutrition and Dietetics. Nutrition Care Manual®. Type I Diabetes Mellitus. http://www.nutritioncaremanual.org/topic.cfm?ncm\_heading=Nutrition%20Care&ncm\_toc\_id=18399. Accessed May 15, 2013.

* Lipid Profile: fasting lipid profile should be performed annually. In patients with low-risk lipid values, a fasting lipid profile can be done every two years.

**Lipid Goals**

|  |  |
| --- | --- |
| Total Cholesterol | < 200 mg/dL |
| Low-Density Lipoprotein Cholesterol | <100 mg/dL |
| High-Density Lipoprotein Cholesterol | >40 mg/dL for men  >50 mg/dL for women |
| Triglycerides | <150 mg/dL |

Academy of Nutrition and Dietetics. Nutrition Care Manual®. Type I Diabetes Mellitus. http://www.nutritioncaremanual.org/topic.cfm?ncm\_heading=Nutrition%20Care&ncm\_toc\_id=18399. Accessed May 15, 2013.

* Blood Pressure: should be measured at each office visit. Those with a systolic BP ≥130 mmHg or a diastolic BP ≥80 mmHg should have their BP reassessed on another day.
* Renal Function: should be assessed annually using albumin excretion rate, in patients who have had TIDM over 5 years. Serum creatinine should be assessed annually in all patients with diabetes to estimate glomerular filtration rate and, if present the level of chronic kidney disease should be identified. Microalbuminuria should be tested with a random spot urine sample for albumin-to-creatinine ratio once the child is 10 years old and has had diabetes for 5 years. Due to the variability in urine specimen, two of three urine specimens collected within 3-6 months period should be abnormal before determining a patient to have crossed over one these diagnostic thresholds. Exercise within 24 hours, infection, fever, congestive heart failure, marked hyperglycemia, and marked hypertension may elevate urinary albumin excretion over baseline values.

**Renal Function Goals**

|  |  |
| --- | --- |
| Category | Spot collection (mcg/mg creatinine) |
| Normal | <30 |
| Microalbuminuria | 30-299 |
| Macroalbuminuria | ≥300 |

Academy of Nutrition and Dietetics. Nutrition Care Manual®. Type I Diabetes Mellitus. http://www.nutritioncaremanual.org/topic.cfm?ncm\_heading=Nutrition%20Care&ncm\_toc\_id=18399. Accessed May 15, 2013.

**Bulimia Nervosa**

The Academy of Nutrition and Dietetics provides the main goals of nutrition therapy of bulimia as follows15:

* Normalize eating by establishing a normal and individualized eating pattern that minimizes nutrition related urges to binge, purge or restrict.
* Achievement and acceptance of a natural weight; a weight that can be maintained with appropriate eating and physical activity, as well as normal weight fluctuations. (Achievement of the natural weight may be considered fully in the domain of the RD while acceptance of the natural weight may overlap in the domain of the mental health professional).
* Achieve comfort with food and eating by identifying trigger foods, feared foods, and food myths and working to recognized that no individual food can cause bingeing, purging, or weight gain.
* Tolerate recovery side effects (fluid retention, bloating, or constipation) through education and symptom management, when needed.

**Laboratory goals:**

|  |  |
| --- | --- |
| Hematocrit (Hct) | Males: 42-52 ml/dL  Females: 34-47 ml/dL |
| Hemoglobin (Hgb) | Males: 14-18 g/dL  Females: 12-16 g/dL |
| Serum osmolality  [(2Na + K) + Blood urea nitrogen/2.8 + glucose/18] | 280-305 mOsm |
| Calcium | 2.3-2.6 mEq/L (ionized free calcium) |
| Creatinine | 0.7-1.5 mg/dL |
| Glucose | 70-105 mg/dL |
| Phosphate | 2.5-4.5 mg/dL |
| Albumin | Normal: 3.5-5 mg/dL  Mild depletion: 2.9-3.4 mg/dL  Moderate depletion: 2.2-2.8 mg/dL  Severe depletion: <2.2 mg/dL |
| Sodium | 135-145 mEq/L |
| Potassium | 3.5-5 mEq/L |
| Chloride | 101-111 mEq/L |
| Magnesium | 1.5-2.5 mEq/L |
| Calcium | 8.5-10.5 mg/dL |
| Bicarbonate | 21-30 mEq/L |
| Urea | 24+49 mg/dL |
| Blood urea nitrogen | 5-20 mg/dL |
| Total cholesterol | 200-300 mg/dL |
| High-density lipoprotein cholesterol | >35 mg/dL |
| Triglycerides | 0-150 mg/dL |

Academy of Nutrition and Dietetics. Nutrition Care Manual®. Bulimia. http://www.nutritioncaremanual.org/topic.cfm?ncm\_heading=Nutrition%20Care&ncm\_toc\_id=21829. Accessed May 15, 2013.

**NUTRITION MONITORING AND EVALUATION**

This part of the nutrition care process is going to be individualized depending on the severity of malnutrition and complications. Nutrition assessment and reassessment will need to be performed each visit so the RD can ensure the intervention is still appropriate for both the diabetic and the bulimia pieces of this condition. Nutrition monitoring and evaluation should be done to evaluate progress and whether or not goals are being met.8

**Type I Diabetes Mellitus**

According to the Academy of Nutrition of Dietetics, if the patient has made all of the appropriate lifestyle changes they are willing to make and the medical goals have not been met, the RD must notify the referral source to have changes made in the medical therapy, for example new or adjusted medications.8 For effective monitoring of outcomes, the dietitian must know the expected outcomes from the MNT interventions for diabetes and when to evaluate them. It is important to note that the expected outcomes are averages and some patients will exceed the expected outcomes whereas others may not achieve a positive outcome.8

The Academy of Nutrition and Dietetics recommends the following be re-evaluated at each visit8:

* Food and blood glucose monitoring records
  + Food records are evaluated for consistency in meal times and number of CHO servings per meal/snack
  + Blood glucose records are evaluated to determine number of readings in target range
* Specific behavior changes per intervention plan
* Schedule changes
* Physical activity patterns
* Weight (height measured at initial session)
* Laboratory data
* Medications

The effectiveness of MNT is based on metabolic outcomes8:

**Metabolic Outcomes**

|  |  |
| --- | --- |
| Endpoint | **Expected Outcome** |
| Glycemic Control | |
| Hemoglobin A1c  Plasma Glucose (Fasting) | 1% to 2% unit (15% to 22%) decrease,  50 mg/dL to 100 mg/dL decrease |
| Lipids | |
| Total cholesterol | 24-32 mg/dL (10% to 13%) decrease |
| Low-density lipoprotein cholesterol | 18-25 mg/dL (12% to 16%) decrease |
| Triglycerides | 15-17 mg/dL (8%) decrease |
| High-density lipoprotein cholesterol |  |
| -No exercise | 3 mg/dL (7%) decrease in HDL |
| -With exercise | No decrease in HDL |
| Blood Pressure | 5 mmHg decrease in systolic and 2 mmHg decrease in diastolic |

Academy of Nutrition and Dietetics. Nutrition Care Manual®. Type I Diabetes Mellitus. http://www.nutritioncaremanual.org/topic.cfm?ncm\_heading=Nutrition%20Care&ncm\_toc\_id=18399. Accessed May 15, 2013.

**Bulimia Nervosa**

According to the Academy of Nutrition and Dietetics, for those with bulimia in an inpatient setting nutrition assessments and reassessments should be performed two to three times per week and weekly or biweekly for a patient who is medically stable enough to be seen as an outpatient.15 Although a patient may reach a healthy weight and/or cease purging, nutrition counseling should not be ever discontinued. If nutrition counseling is not needed as frequently, it can be discussed with the team how often reassessment is necessary. It is recommended that nutrition counseling is needed for a year or more after weight restoration to prevent relapse and should be evaluated regularly and on an individual basis in consultation with the treatment team and patient.15

Nutrition therapy should be considered a success if the patient is able to practice normal, individualized eating, using internal cues to determine what, how much, and when to eat; if the patient is eating adequately to support a healthy weight and appropriate physical activity; and if the patient is accepting variability in eating from day to day.15

**PRESENTATION OF THE PATIENT**

DC is a 25-year old female with an extensive past medical history significant for asthma, type I diabetes mellitus, pulmonary disease, rhinocerebral mucormycosis, hypertension, methicillin-resistant *Staphylococcus aureus* (MRSA) bacteremia, cavitary pneumonia, failure to thrive, reactive airway disease, coronary artery disease, and multiple hospitalizations for diabetic ketoacidosis. She has known allergies to latex and strawberries and reported being lactose intolerant. DC was diagnosed with type I diabetes in 2006 at the age of 18 years old. Past surgical history includes procedure in the left hand, multiple sinus surgeries, cataract removal, and multiple PICC lines. Family history includes grandmother with diabetes, father with hypertension and mother having a history of seizures. DC uses alcohol occasionally and denies any tobacco or drug use. She is single, unemployed, lives at home with her mother and step-father and has two children under the age of five. DC does not have insurance coverage and was applying for social security during her last visit. Since September 29, 2012, DC has had 13 admissions, not including the visits to the emergency room where she was not admitted and admissions at other hospitals she had mentioned.

**Prior Admissions**

|  |  |  |  |
| --- | --- | --- | --- |
| Date of Admission | **Diagnosis** | **Chief complaint** | **Blood Glucose (mg/dL)** |
| 09/29/12 | DKA | Shortness of breath | 700 |
| 10/11/12 | DKA | Abdominal pain, nausea, vomiting x 2 days; toothache | 413 |
| 11/17/2012 | Hyperglycemic, Constipation | Nausea, abdominal pain x 4 days, likely due to constipation | 334 |
| 12/01/12 | Hyperglycemia, Pericardial infusion | Back pain, abdominal pain, nausea, vomiting | 633 |
| 01/11/13 | DKA  - Malnutrition | Nausea, vomiting, abdominal pain | 752 |
| 01/20/13 | HHS | Right shoulder pain, shortness of breath | 997 |
| 02/01/13 | HHS, nonketotic state | Diarrhea x 3 days | 910 |
| 03/10/13 | DKA secondary to noncompliance versus pelvic issue | High blood sugar, pain in suprapubic area x 2-3 days | 521 |
| 03/14/13 | HHS, nonketotic state likely due to noncompliance | Chest pain | 735 |
| 03/19/13 | HHS, nonketotic state  - Anasarca secondary to diabulimia and malnutrition | Back pain likely secondary to constipation from narcotics | 824 |
| 03/25/13 | DKA | Change in mental status, agitated confusion, vision changes | 762 |
| 03/28/2013 | DKA | Chest pain | 725 |
| 04/09/13 | DKA | Nausea, vomiting, abdominal pain | 749 |

**Weights upon Admission**

|  |  |  |  |
| --- | --- | --- | --- |
| Date | **Weight (kg)** | **Weight (lbs.)** | **How obtained** |
| 09/29/2012 | 49 | 107.8 | Bed scale |
| 10/11/2012 | 45 | 99 | Bed scale |
| 11/17/2012 | 41.2 | 90.64 | Bed scale |
| 12/01/2012 | 44 | 96.8 | Patient stated |
| 01/11/2013 | 44 | 96.8 | Other |
| 01/20/2013 | 56.7 | 124.74 | Bed scale |
| 02/01/2013 | 46.8 | 102.96 | Bed scale |
| 03/10/2013 | 39.009 | 85.82 | Bed scale |
| 03/14/2013 | 37.3 | 82.06 | Bed scale |
| 03/19/2013 | 40.058 | 88.13 | Bed scale |
| 03/25/2013 | 41 | 90.2 | Bed scale |
| 03/28/2013 | 38.244 | 84.14 | Bed scale |
| 04/09/2013 | 42.638 | 93.8 | Bed scale |

**Hemoglobin A1c**

|  |  |  |
| --- | --- | --- |
| Date | Hemoglobin A1c (%) | Average blood glucose (mg/dL eAG\*) |
| 09/30/2012 | 19.2 | 504 |
| 01/12/2013 | 8.2 | 189 |
| 02/01/2013 | 9.0 | 212 |
| 03/11/2013 | 10.8 | 263 |
| 03/26/2013 | 11.6 | 286 |
| 04/09/2013 | 13.9 | 352 |

\*eAG: estimated average glucose

**Important facts from previous admissions:** (information acquired from the Emergency Department chart, History and Physical and multiple consults during admissions.)

**9/29/2012**

* Admitted to being non-compliant with her insulin at home.

**10/11/12**

* Reported she is compliant with her medications at home, but did not take them prior to admission because of her symptoms of pain and she was not eating due to her toothache.
* Admitted to not counting carbohydrates or correcting for high blood sugars.

**11/17/12**

* During a consult for constipation, with her mother present, DC denied any previous ED or laxative abuse in the past.

**12/01/12**

* Severe back pain and 10/10 diffuse abdominal pain that has been going on for “a while”
* Did not take her insulin the night prior to admission because of illness.
* Nausea and vomiting possibly secondary to gastroparesis.
* Was a poor historian at admission and was not cooperative with the staff.

**12/14/12** (not admitted)

* Reported she normally checks her sugars and gives herself insulin before meals and her blood sugar was low the night before at 38-64 mg/dL.
* Complained of weakness, has been unable to walk and has had two to three nosebleeds for the past three days.

**01/11/13**

* Reported to be compliant with insulin and denies any prior episodes of DKA.
* Reported she had lost weight in the past few months.
* Diagnosis of malnutrition with an albumin level of 2.8

**01/20/13**

* Was not a good historian
* Weight loss noted in Emergency Department chart.
* Reported polyuria and polydipsia over the past 24 hours prior to admission.
* Change of her insulin prescription at this admission due to financial issues.

**03/10/13**

* Reported she had access to her medications, but later reported to the admitting house staff that she has been out of her medications.
* Reported she was recently discharged from another hospital and has gone to the emergency room several times in the last couple of days.
* Reported blood sugars normally run in the 130-190 mg/dL range; which would correspond to her last hemoglobin A1c of 9.0.
* Blood sugars in the emergency department her blood sugars were 521 mg/dL and in DKA.
* Reported that she checked her blood sugars on the day of admission and she was not that high, so she is unaware of how it got there.
* Per physician, “dietary and diabetes education will be provided to the patient for the umpteenth time that she has been here, as she has been in for multiple admissions because of her noncompliance. I suspect non-compliance is the issue, although she told me otherwise.”

**03/14/13**

* Reported she was compliant with her insulin, but reported to other staff during her admission that she not was compliant with her insulin.
* Reported that she does not remember the dose of insulin she uses at home.
* Claimed that Morphine and Dilaudid were the only medications that helped her pain.
* Diagnosis included questionable diabulimia.
* Reported 70 pound weight loss over the past three months.
* Past medical history included a 90 pounds weight loss over the last several years.
* Doctor noted DC is cachectic and ill-appearing, her extremities show muscle wasting diffusely and suspects a degree of diabulimia.

**03/19/13**

* Presented with back pain, again claiming only Dilaudid helps.
* Back pain likely secondary to constipation suspected to be caused from considerable amount of narcotics that she taken over the last two admissions.
* Reports that the flare in her pain has again caused the flare in her glycemic control.
* Diagnosis of anasarca secondary to diabulimia and malnutrition.
* Doctor suspects an eating disorder with suspected diabulimia.
* DC is encouraged to follow up with an endocrine clinic after applying to charity care.

**03/25/13**

* Presented with a change in mental status, agitated, vision changes, combative and sedated with intravenous medications.
* Reported blood sugars at home are always under 200 mg/dL
* Doctor suspect’s degree of diabulimia, but it is difficult to prove.

**04/09/13**

* Reported she is non-compliant with her insulin and has not taken it for days.
* Reported to other staff that that she has been taking her insulin as prescribed.
* Reported most blood sugar readings at home have been 250 mg/dL or lower.
* Doctor suspects diabulimia.

**Initial Assessment: 04/09/2013**

**Diagnosis:** DKA

**Additional:** Metabolic acidosis, uncontrolled diabetes

**Assessment:** 25 year old female with TIDM who presents with hyperglycemia, abdominal pain and vomiting. Patient had a recent nose bleed which she reported occurs regularly. Skin is intact.

**Anthropometrics:**

* Weight: 93.8 pounds (42.6 kg), per bed scale
* Height: 5 foot 3 inches (160 cm), per patient
* Body Mass Index (BMI): 16.05
* Ideal body weight: 120 pounds (54.5 kg)
* % Ideal body weight: 78%

**Food and Nutrition History:** Patient reported having a good appetite and eats regular meals at home while avoiding sugar. She reported that her mother “does everything” for patient: wakes patient up in the morning to eat, administers patient’s insulin, prepares meals for patient, and counts the carbohydrate servings at each meal for the patient. Discussed what the patient consumes on a regular day:

* Breakfast: mini bagel with cream cheese with a small bowl of cereal (Raisin Bran and Honey Nut Cheerios) with a small banana
* Lunch: turkey and cheese hoagie OR two peanut butter and jelly sandwiches on whole wheat bread OR clam chowder soup from a can with a small fruit
* Dinner: “whatever mom cooks”- baked chicken or fish with pasta, rice and bean dish with a banana and with cabbage OR greens OR corn.

She reports that she has a small stomach and is not able to tolerate greasy and fried foods. She is lactose intolerant and drinks lactose free milk or can tolerate 2% milk. She drinks juice diluted with water and frequently drinks a lot of water. She agreed to eating sugar free yogurt and sugar free pudding snacks while in the hospital. PO intake prior to initial assessment 20%-25%, per documentation, at two meals; this is unusual. Although, at lunch patient ate 100% and requested another sandwich.

**Current Diet:** Consistent Carbohydrate – Moderate (1600-2000 calories)

**Medications at home:** Patient reported her insulin regimen at home during the initial nutritional assessment as well as during a separate consult with a physician:

|  |  |  |
| --- | --- | --- |
|  | **Initial Nutrition Assessment**  **(Insulin Type and Dosage)** | **Consult for DKA**  **(Insulin Type and Dosage)** |
| Morning | 20 units NPH  8 units Regular | 19 units NPH  8-10 units Regular |
| Lunch | 8 units Regular |  |
| Dinner | 8 units Regular | 18 units NPH |
| Before Bed | 18 units NPH | 8-10 units Regular |

Other medications include: 5 mg Lisinopril daily, 15 mg Remeron nightly

**Current Medications:** Insulin: 8 units Regular units at breakfast, 18 units NPH at 5 pm, 8 units Regular at dinner, 20 units NPH daily in the evening; Sliding Scale Regular, Bentyl, Neurontin, Pepcid, Morphine, Zofran,

**Labs at Admission**

|  |  |  |  |
| --- | --- | --- | --- |
| Labs | **Normal Range** | **Admission** | **Low (L), High (H), Within Desirable Limits (WDL)** |
| Glucose | 70-105 mg/dL | 749 mg/dL | H |
| pH Arterial | 7.34-7.45 | 7.00 | L |
| Sodium | 133-145 mmol/L | 124 mmol/L | L |
| Bicarbonate | 20-26 mmol/L | 3 mmol/L | L |
| Chloride | 96-108 mmol/L | 87 mmol/L | L |
| Anion Gap | 10-20 | 36.7 | H |
| Potassium | 3.3-5.1 mmol/L | 4.7 mmol/L | WDL |
| Phosphorus | 2.7-4.5 mg/dL | 6.8 mg/dL | H |
| Magnesium | 1.6-2.6 mg/dL | 2.2 mg/dL | WDL |
| Calcium | 8.4-10 mg/dL | 10.1 mg/dL | H |
| Creatinine | 0.40-1.10 mg/dL | 1.69 mg/dL | H |
| BUN | 6-20 mg/dL | 25 mg/dL | H |
| WBC | 4.8-10.8 | 15.2 | H |

As can be observed from the labs, DC is in DKA. As discussed earlier, DKA is characterized by blood glucose >250 mg/dL, arterial pH <7.0 to7.30, serum bicarbonate <10-18 mEq/L, a positive anion gap and electrolyte imbalances.

**Energy Needs:**

* 35-40 calories/kg: 1476-1687 calories
* 1.0-1.2 grams protein/kg: 42-50 grams protein
* 1 mL fluid/kg: 1476-1687 mL fluid

**PES**

1. Food and nutrition-related knowledge deficit related to diabetes and previously not receptive to education as evidenced by previous attempts to educate patient and patient reporting no previous diabetes education and she does not know how to count carbohydrates, multiple admissions over the past seven months, severe weight loss over the past year and elevated hemoglobin A1c of 13.9%.
2. Altered nutrition related labs related to diabetes and history of patient reporting omitting insulin as evidenced by hemoglobin A1c of 13.9%, glucose >400 mg/dL upon admission, and Endocrinology reporting “suspected diabulimia.”

Note: Patient was educated during previous admission by Nutrition. Patient always reported that she knew how to count carbohydrates. In March, the patient was unaware of her insulin dose and reported again that she knew how to count carbohydrates. During the most recent nutrition assessment, the patient expressed interest in an insulin pump and we explained to her to that the pre-requisite to obtain one was adequate knowledge of carbohydrate counting since insulin boluses are given based upon amounts of carbohydrates eaten. She was then receptive to diabetes and an hour was spent educating the patient about insulin use and carbohydrate counting.

**Nutrition Goals:**

* PO intake: food selections will be consistent with prescribed diet at present and ongoing.
* Weight: prevent further weight loss at present and ongoing.
* Labs: glucose control <180 mg/dL, fasting 80-130 mg/dL (90% of measured values) at present and ongoing.
* Skin: meet nutritional needs to prevent skin breakdown at present and ongoing.
* GI: GI symptoms will be improved/eliminated within 24 hours.
* Diet knowledge/Compliance: menu/food intake selections will be consistent with prescribed diet at present and ongoing.

**Recommendations/Interventions**

* Carbohydrate modified diet: Consistent Carbohydrate- Moderate (1600-2000 calories)
* Multivitamin
* Nutrition Education- Nutrition relationship to health/type I diabetes
* Nutrition Education- Diabetes education (carbohydrate counting and meal planning)
* Nutritional Counseling- Self-monitoring of blood glucose

**Monitoring and Evaluation**

* PO intake
* Need for high protein/high calorie supplement
* Diet knowledge
* Glucose
* Skin Integrity
* Daily weight
* Plan of care

As can be seen throughout the timeline and initial nutritional assessment, DC was frequently a poor historian, often regarding her insulin use. On multiple admissions she would deny being non-compliant with her insulin and other admissions she would admit to being non-compliant with her insulin. DC would even change the story regarding her compliance during the same admission. Often, she reported that she only stopped taking her insulin the night before admission due to her pain and gastrointestinal symptoms. It is suspected her non-compliance began before the night prior to an admission. On 12/01/12, the physician suspected her nausea and vomiting were secondary to diabetic gastroparesis. A complaint of polydipsia and polyuria during her admission on 01/21/13 is another sign of in-proper insulin use and perhaps non-compliance. DC would not be experiencing these diabetic symptoms if she was compliant with her insulin usage and monitoring her blood sugars properly. DC also experienced a great deal of weight loss over the course of her admissions. Weight loss was noted in the Emergency Department chart on 01/11/2013. This is likely due to omission of insulin; therefore, calories were being purged by gluocosuria. On 12/14/12, DC reported her last menstrual period was 10/15/12; denied pregnancy and is not on birth control. On 04/19/13, DC reported that she does not get her menstrual period; she is not on birth control and denied pregnancy. As stated earlier, one of the criteria for Anorexia Nervosa as amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration).9 The cessation of her menstrual cycle can be contributed to her reported 90 pound weight loss over the past couple of years. In addition, DC is currently 79% of her ideal body weight and as stated earlier, an individual with bulimia may also be underweight and if the individual is less than 85% of expected weight, the diagnosis would be Anorexia Nervosa-Purging Type.22 In addition, coronary artery disease is listed in DC’s past medical history. Coronary artery disease is another long term effect of hyperglycemia, which is very unfortunate at the age of 25.

One of DC’s most common complaints at admission was chronic pain in her abdominal area, lower back, and pelvic area. With the development of neuropathy, genitourinary problems can occur which could be the cause of her vaginal pain. In March, DC began to claim that only Dilaudid or morphine would help her pain. Unfortunately, DC continuously received narcotics for her pain which led to constipation and more pain. Doctors began noting that DC was demonstrating pain seeking behaviors and administration of pain management medications was stopped. A psychiatry consult was also requested on 03/29/2013 for hospital substance abuse/noncompliant though nursing staff says reason is for diabulimia.

After about six months of continuous admissions of seven DKAs, four HHSs, and a reported weight loss of 70 pounds over three months, the doctor began to suspect diabulimia. Contributing factors to this diagnosis include non-compliance with insulin, recurrent admission of DKA or HHS, elevated hemoglobin A1c, weight loss, absence of menses, constipation secondary to gastroparesis, genitourinary complications, tachycardia, polyuria and polydipsia. Psychiatry consults were ordered during three admissions:

* 01/20/2013: to rule out Anorexia Nervosa (full consult in Appendix). At this point DC has been in and out of the hospital over a four month period and more than once for DKA with sugars as high as 900 mg/dL. DC was diagnosed with panic disorder with agoraphobia. Adjustment disorder with anxiety and depressed mood was ruled out.
* 03/20/2013: for diabulimia (see full consult in Appendix). During this consult, she denied binging on food, fasting, purging, use of laxatives, and denies diabulimia. DC reported that she does not have a poor body image, does not feel that she needs to lose more weight, but wants to gain weight and is quite concerned that she lost approximately half her weight since October 2012. She reported that she had a complicated sinus surgery at Jefferson, and they kept her in the hospital from October to December 2012. She states that she used to weigh 170 pounds at that time, and currently is between 70-80 pounds and weight loss is unintentional. DC’s diagnoses included eating disorder not otherwise specified, panic disorder with agoraphobia, adjustment disorder with mixed anxiety and depressed mood, dyssomnia not otherwise specified, and somatoform disorder not otherwise specified.
* 03/29/2013: for hospital substance abuse/noncompliant though nursing staff says reason is for diabulimia (see full consult in Appendix). DC reported she uses her insulin appropriately at home and eats regular meals including waffles, and banana for breakfast, peanut butter and jelly on wheat bread for lunch and whatever her parents cook for her at dinner at rice, corn beef, chicken pasta, or anything. Her mom was present and confirmed this and states that the patient eats a lot, but cannot seem to put on weight. Her mom did not have any concerns for the patient as far as suicidality or psychiatric symptoms aside from some anxiety related to her children, who do not live with her. It was noted that DC appeared rather cachectic though she denied eating disorder symptoms as far as purging, binging, use of laxatives, inappropriate dosing of her insulin as well as any body image dysregulation and does believe that she needs to gain weight. The doctor did not believe that she meets criteria for an eating disorder. DC’s diagnosis included panic disorder without agoraphobia and adjustment disorder with mixed anxiety and depression. Somatoform disorder, not otherwise specified was ruled out.

**CRITICAL COMMENTS**

While reading through the medical records there were a few inconsistencies in DC’s past medical history including DC having type 2 diabetes during her first couple admissions and in future admissions her past medical history listed type I diabetes. Also, it was noted that DC once had an insulin pump, but during my third initial nutritional assessment with DC she was expressing that she wanted an insulin pump in the future and did not mention that she had one before. I believe that this documentation of DC once having an insulin pump is an error. There is a lot of training needed to use the insulin pump, including understanding how to count carbohydrates in which DC did not know.

DC had been seen by a different dietitian during most of her admissions and like the doctors it took more frequent admissions to realize that DC may be non-compliant with her insulin at home on purpose. My first encounter with DC was on 03/10/13 in which I noticed immediately she was cachectic with muscle wasting. During this nutrition assessment DC was not cooperative, but did report that she counts her carbohydrates, monitors her blood sugars and then denied education. The dietitian and I did question her compliance, but because she was not fully cooperative for the nutrition assessment it was hard to tell. My second encounter with DC was on 03/09/2013 and her non-compliance became clear when she could not recall her insulin regimen at home and once again, DC denied education. It was the third encounter when her non-compliance was clear and the dietitian and I approached DC in a different way. At this point, we stayed clear of addressing weight in fear that DC would withdrawal and not be cooperative during the nutritional assessment. We were able to discuss the long term effects of non-compliance of insulin and hyperglycemia. DC expressed interest in an insulin pump, which allowed us to spend an hour educating her on carbohydrate counting and meal planning that is a pre-requisite for acquiring and an insulin pump. It was an accomplishment to have DC open up and be accepting to education.

After realizing DC has had 13 admissions in less than one year, I could not help but wonder why she was continuously discharged without a referral to an inpatient setting where she could get proper help. When talking with a social worker regarding a referral to an inpatient setting, such as The Renfrew Center, they said that due to her financial situation DC is not capable of being admitted to an inpatient facility. After calling The Renfrew Center, we discovered that they can set up payment plans for anyone on personal pay. As far as an interdisciplinary approach for DC, it was poor in my opinion. Although, the doctor did order consults for nutritional assessment and psychiatric assessments, there appeared to be no collaboration throughout the team of what the best plan is for this patient. For example, the psychiatrist ruled out eating disorder based on strictly on his assessment with the patient, and with no regards to what the patient had reported in other consults, including inconsistent insulin regimens at home, non-compliance with insulin, does not count carbohydrates and does not correct for elevated blood sugars.

In regards to DC’s care plan, there are things that I would have done different. To begin with, I would have contacted and collaborated with her primary doctor and the psychiatrist back in March and discussed the nutritional assessment, compared for consistencies and inconsistencies with DC’s story in the consults, and discussed the options for referral to an inpatient facility. Also, since DC lives with her mother I would have tried to do a nutritional assessment with mom present (with the permission of DC), with hopes that her mother would provide reliable information and confirm the validity of DC’s story. In previous nutritional assessments, I would have done a 24 hours recall, discussed the short-term and long-term consequences of hyperglycemia and really encouraged diabetes education. As for the future, if DC returns I will contact her primary doctor as well as the psychiatrist in order to help get DC the best care plan possible and a referral to an inpatient facility or at the minimum strong encouragement of better follow-ups with an endocrinologist and even an outpatient registered dietitian.

**SUMMARY**

Diabulimia not only has detrimental short-term and long-term complications, but can be deadly. Anybody with type I diabetes can realize that they possess the most dangerous tool for weight loss, insulin, and can then manipulate their insulin as a means to control their weight. There are many warnings signs that can be looked for in someone suspected to be suffering from diabulimia. Diabulimia requires an interdisciplinary team approach including, but not limited to a primary care physician, endocrinologist, registered dietitian and psychiatrist. Communication is key within this team as the possibilities of manipulation or deception on part of the patient can create complications in the care plan. The registered dietitian(s) play an important role on this team by providing medical nutrition therapy for type I diabetes and bulimia. Goals of the medical nutrition therapy include the patient reaching a healthy weight, practicing normal individualized eating and glycemic control. DC is an excellent patient to represent diabulimia as she showed inconsistencies in her stories, had frequent admissions for diabetic ketoacidosis and hyperglycemic hyperosmolar state, severe weight loss, and at the age of only 25 was demonstrating the short and long-term effects of hyperglycemia. It would be ideal for DC to seek help at an inpatient facility where she can get 24-hour care and support.

I have realized just how important of a role the registered dietitian plays in the treatment of diabulimia. Detailed nutritional interventions can have a huge impact in the treatment of diabulimia; therefore, increasing awareness of this condition and collaborating as dietitians about different approaches being used for treatment is important. In addition to care by an interdisciplinary team, organizations and camps can be important in getting individuals together to gain acceptance and cope with their disease.

**TERMINOLOGY**

**Albuminuria:** albumin in the urine

**Dysphoria:** a state of unease or dissatisfaction with life

**Gluconeogenesis:** the formation of glucose from non-carbohydrate substrates

**Glycogenesis:** the conversion of glucose into glycogen

**Glycogenolysis:** the conversion of glycogen into glucose

**Glycolysis:** the breakdown of glucose that releases pyruvic acid and ATP

**Hepatocytes:** liver cells

**Hyperglycemia:** high blood glucose; above 180 mg/dL

**Hypoglycemia:** low blood glucose; below 70 mg/dL

**Hypovolemia:** decreased volume of blood circulating throughout the body

**Macroalbuminuria:** large amount of albumin leaked into the urine

**Microalbuminuria:** small amount of albumin leaked into the urine

**Lipogenesis:** to formation of fatty acids

**Lipolysis:** the breakdown of fats and other lipids by hydrolysis into fatty acids and glycerol

**Polydipsia:** excessive thirst

**Polyphagia:** excessive hunger

**Polyuria:** excessive urination

**Pyelonephritis:** injury to kidneys caused by bacterial infections

**Resting Energy Expenditure:** amount of calories required by the body during a 24 hours of non-activiy

**Rhinocerebral mucormycosis:** opportunistic infection of the sinuses and the brain cause by saprophytic fungi

**MEDICATION BIBLIOGRAPHY**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Generic Name | **Brand Name** | **Use** | **Side Effects** | **Food Interactions** |
| Regular insulin  NPH insulin | Humulin R  Novolin R  Humulin N  Novolin N | Blood glucose control in TIDM; DKA, HHS | Dry mouth, hypoglycemia, hyperglycemia, hypomagnesemia, hypokalemia | Timing of injection or pump bolus in relation to food varies with insulin form; recommend diabetic meal plan to balance CHO with insulin |
| Dicyclomine | Bentyl, Formulex, Protylol | Irritable bowel syndrome, other functional GI disorders | Constipation, dry mouth, thirst, vomiting, nausea, abdominal distention | Take 30 minutes to 1 hour before food |
| Gabapentin | Neurontin, Gralise | Pain from diabetic neuropathy | Depression, constipation, dry mouth, dyspepsia, increased appetite, nausea, vomiting | Take without regard to food; take Mg supplement separately by 2 hours |
| Famotidine, calcium and magnesium | Dual Action Complete, Pepcid complete | Duodenal ulcer, gastric ulcer, to prevent or treat heartburn | Anorexia, constipation, diarrhea, dry mouth, taste perversion | Take with a glass of water without regards to meals; bland diet may be recommended; take drug at least 2 hours before or after Fe supplement; take Mg supplement or Al/Mg antacids separately by at least 2 hours; limit caffeine/xanthine. |
| Morphine | Avinza, Kadian, MS Contin, MSIR, Oramorph SR | Moderate to severe pain | Depression, physical dependence, constipation, nausea, vomiting, anorexia, biliary tract spasms, dry mouth, ileus, edema | May take with food to decrease GI distress; Insure adequate fluid intake/hydration |
| Odansetron | Zofran | Prevent nausea and vomiting | Constipation, diarrhea, abdominal pain, decreased appetite, xerostomia | None |

**APPENDIX**

**Table 1. Types of Insulin**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Insulin Type | Onset of Action | Peak of Action | Duration of Action (hours) | Comments |
| Rapid-Acting Insulin Analogs | | | | |
| Lispro (Humalog)  Aspart (Novolog)  Glulisine (Apidra) | 5-15 minutes | 30-90 minutes | 3-5 | Can be used in pump therapy |
| Short-Acting | | | | |
| Regular | 30-60 minutes | 2-4 hours | 5-8 | Can be mixed with longer-acting insulin |
| Intermediate-Acting | | | | |
| NPH | 2-4 hours | 4-10 hours | 10-16 | Usually given in 2 daily doses |
| Extended Long-Acting Analog | | | | |
| Insulin glargine  Insulin determir | 2-4 hours  2-4 hours | Peakless  6-14 hours | 20-24  16-20 | Cannot be mixed with other insulins |
| Premixed | | | | |
| 70/30  75/25 lispro analog mix  70/30 aspart analog mix  50/50 human mix  50/50 lispro analog mix | 30-60 minutes  5-15 minutes  5-15 minutes  30-60 minutes  5-15 minutes | Dual  Dual  Dual  Dual  Dual | 10-16  12-20  12-20  10-16  12-20 | 70% NPH, 30% Regular  75% intermediate, 25% lispro  70% intermediate, 30% aspart  50% NPH, 50% regular  50% intermediate, 50% lispro |
| Antihyperglycemic Drug | | | | |
| Pramlintide | Slows transit of digesting food through intestine; given at mealtimes to increase efficacy of insulin; should not be mixed with insulin | | | |

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Table 2. Diabetes Carbohydrate Exchange List

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Food List | Carbohydrates (grams) | Protein (grams) | Fat (grams) | Calories |
| Carbohydrates |  |  |  |  |
| Starch  Grains, breads, cereal:  - 1 oz. bread, ¼ large bagel  - 6” tortilla  - ½ cup cooked dried beans  - 1/3 cup cooked pasta or rice  - ¾ cup cold cereal  - ½ cup cooked cereal  Starchy vegetables  - ½ cup potatoes, peas, corn | 15 | 0-3 | 0-1 | 80 |
| Fruits - 1 small fresh fruit  - ½ cup fruit or fruit juice  - 1 cup melon or berries  - ¼ cup dried fruit | 15 | - | - | 60 |
| Milk  Fat free, low-fat, 1%  Reduced fat, 2%  Whole  - 1 cup milk  - 2/3 cup unsweetened yogurt | 12  12  12 | 8  8  8 | 0-3  5  8 | 100  120  160 |
| Sweets, Desserts, & Other Carbohydrates  - 3 oz. snack food (pretzels, chips, 3-4 crackers)  - 1 oz. sweet snack (2 small sandwich cookies, 5 vanilla wafers)  - 1 tbsp sugar of honey  - ½ cup ice cream | 15 | varies | varies | varies |
| Nonstarchy Vegetables  - 3 cups raw vegetables  - 1 ½ cooked vegetables | 5 | 2 | - | 25 |
| Meat & Meat Substitutes |  |  |  |  |
| Lean  Medium-fat  High-fat  Plant-based proteins | -  -  -  varies | 7  7  7  7 | 0-3  4-7  8+  varies | 45  75  100  varies |
| Fats | **-** | **-** | 5 | 45 |
| Alcohol (1 alcohol equivalent) | varies | - | - | 100 |

Daly A, et al. Choose your foods: exchange lists for diabetes. Alexandria, Va: American Diabetes Association and American Dietetic Association; 2008.

**1/23/2013 Psychiatric Consult by Michael J. Friedman, D.O.**

Psychiatry history: “No inpatient or outpatient psychiatric treatment. No past psych consults. The patient does admit to an anxiety disorder over the last 4 months with high levels of anxiety, having difficulty breathing, shortness of breath, and feeling of pressure if she is in enclosed spaces. There is no history of an detoxes or rehabs. There is no history of past psychiatric medications other than Ativan, which was given here in the hospital. At the past hospitalizations, she is presently receiving 0.25 of Xanax for anxiety symptoms p.r.n.”

Social History: “She lives with her sister. She has 2 children, her daughter Lola age 3 and a son Eric age 2, her sister has 1 son age 5. Mother is visiting from New York at the moment to help care for everybody. She has not worked since 2006. She worked for 5 months at Chuck E. Cheese at that time. She got pregnant and has not worked since. No history of substance abuse. No history of tobacco use.”

Family of Origin Psychiatric and Substance Abuse History: “No history known. That patient denies that there any of her family of origin suffer with mental health or substance abuse issues. She denies any history of abuse as a child.”

Mental Status Examination: “She appears cachectic. She is cooperative. She is tearful. No evident psychomotor agitation or retardation. Speech normal flow and volume. Anxious tone. Tearful tone. Thought processes appear goal-directed. No evident delusions. Though content, no suicidal or homicidal ideation. Every now and again, she may have some passive suicidality when she feels like they are never going to find out what is wrong with her and she wonders why she has to suffer like this, but there is no real death wish and no suicidal ideation. Perceptions, she denies illusions, denies hallucinations. Mood, she feels anxious, she has breakthrough anxiety. She sometimes feels like she is a burden on her sister and can get down a little depressed, but nothing persistent. Her affect is restricted and she is tearful. Cognition is intact. She is able to be attentive. She is AAO x 3. Memory, she knows our present president and the previous presidents she remembers before that as President Clinton. She is able to repeat 3 items after me and remember all 3 after 2 minutes of conversation. She would not attempt subtraction of 7 serially as she states she is bad at mathematics. She did spell world forwards, but had great difficulty spelling it backwards. She is able to reason. She is able to abstract. She has very little insight, very little judgment as far as what is happening to her she is mostly fearful and she does not have a clear diagnosis or clear reason why she is so sick.

For collateral, I spoke to her mom. Mother states that daughter is not suicidal, does not show any symptoms of anorexia, but does suffer with anxiety over the recent months. The patient states that she has needed insulin since 2006 when she was 18 and for the longest time was able to maintain within range. She did have gestational diabetes during her pregnancy. Maintaining sugar levels, glucose levels have been difficulty over the last month. She does experience anxiety attacks, agoraphobia with panic symptoms just in small rooms that is over the last 3 or 4 months. She has received Ativan from the hospital here for anxiety. She is in and out of the hospital over 4 months and barely gets to see her children anymore, feels like she is a burden on her daughter.”

Impression: “A 25-year-old female with a history of anxiety attacks over the past 3-4 months. Psych was asked to rule out Anorexia Nervosa. The patient and patient’s family, mother deny signs and symptoms of Anorexia Nervosa. The patient does experience high levels of anxiety with panic symptoms when in enclosed area. I would also rule out an adjustment disorder with anxiety and depressed mood. Given that the patient has been in and out of the hospital over the last 4 months, rarely sees her children anymore secondary to her physical health and feels like she is a burden and does not know what is happening to her life.”

Diagnoses:

AXIS I: Panic disorder with agoraphobia, rule out adjustment disorder with anxiety and depressed mood.

AXIS II: Deferred.

AXIS III: See medical history.

AXIS IV: Severe medical problems over 4 months, fear of unknown medical conditions, unable to be with her children.

AXIS V: 35.

Recommendations: Celexa 10 mg one at bedtime p.o., increase to 20 mg after 3 days if tolerated. TSH, B12, folate, RPR. Continue p.r.n. Xanax for breakthrough anxiety.

**3/20/2013 Psychiatric Consult by Ilya Ivanov**

History of Present Illness: “A 25-year-old female was admitted to ICU for recurrent DKA secondary to diabulimia. Her blood sugars were 824. The patient has a history of recurrent DKAs with blood sugars as high as 900 with multiple ICU admissions over the last 4 months. The patient states that she had many medical hospitalizations since October 2012 here at JFK and Jefferson Hospital in Philadelphia. The patient states that she is in good mood other than current back pain. This was attributed to severe constipation secondary to in-hospital opioid use. The patient states that she is not depressed. There are not symptoms consistent with major depressive episode or major depressive disorder. Also no symptomatology pointing to bipolar disorder, psychotic or schizophrenic diagnosis, no PTSD, no drug or alcohol use. The patient endorses anxiety stating that she has had panic symptoms specifically with claustrophobia. The patient denies suicidality or homicidality or psychotic symptoms. Additionally, the patient denies concerns for eating disorder. She denies binging on food, fasting, purging, use of laxatives, and denies diabulimia. The patient states that her blood sugars run in the 100s range and rarely over 200. She states that she does not have a poor body image, does not feel that she needs to lose more weight. In fact, she wants to gain weight and is quite concerned that she lost approximately half her weight since October 2012. The patient reports that she had a complicated sinus surgery at Jefferson, and they kept her in the hospital from October to December 2012. She states that she used to weigh 170 pounds at that time, and currently is between 70-80 pounds.

She states that she eats nutritious breakfast, lunch, and dinner. She does not restrict calories and she desperately has been trying to gain weight and weight loss is unintentional.”

Social history: “single. She has two children, age 2 and 3, they are currently staying with their biological father. The patient has not worked since 2006. The patient has high school education. She is Roman Catholic, no military history. Denies any legal problems currently or in the past. She denies access to weapons or any lethal means. Further she states that her mother who resides in New York has been down here for months helping her medically. The patient is currently residing at mom’s husband house. The patient is receiving food stamps and no other income stated.”

Past psychiatric history: “Negative for prior psychiatric treatment, inpatient or outpatient. Denies SI/SA or HI/HA. Negative for use of psychotrophic medications except Ativan and Xanax which is being prescribed by “doctor from Jefferson.” The patient states that because she does not have insurance she does not have a primary care doctor and unable to get medications from Jefferson pharmacy. Currently, she is out of both; it is not clear for how long. The patient was seen by a Frank Stafford on January 21, 2013 for consult regarding ‘rule out Anorexia Nervosa’ and that consult was reviewed.”

Mental status exam: “The patient is calm and cooperative. She is tearful at times and there is periodic anxiety. The patient’s speech is productive, normal volume, rate and tone. Speech is coherent and spontaneous and there are no abnormalities. Thought process is logical and linear. There is no psychomotor agitation or retardation. No cogwheeling. She is oriented to time, place, and person. Recent memory is intact; however, she is unable to name past presidents. She is able to recall 3/3 objects immediately 5 minutes later. Attention span and concentration are both intact. Language is appropriate and there is no language barrier. She denies suicidality and denies homicidal ideation, states that she never felt suicidal or homicidal in the past. She denies hallucinations, delusions, or illusions.

Judgment is limited secondary to incongruent information regarding her diabetic management, which she reports as appropriate with diagnostic finding such as hemoglobin A1c, which is above 10. Judgment: difficult to assess. She is open to help with her ongoing difficulty with anorexia and diabetic complications; however, she is not open to pragmatic treatments. Also, she is rather indifferent to her cachexia.”

Impression: “A 25-year-old female with diabetes mellitus type 1, multiple Intensive Care Unit hospitalization secondary to diabetic ketoacidosis, currently rather cachectic, the patient endorses anxiety, insomnia, and depression. Otherwise, she denies other psychiatric concerns. Specifically, she denies eating disorder issues as far as purging, binging, use of laxatives, or inappropriately dosing her insulin for weight control.

However, multiple factors point to anorexic syndrome, including anorexic BMI of 15.05 kg/m2 based on her Height 1.63 m and Weight 40.058 kg.”

Diagnoses:

AXIS I: Eating disorder not otherwise specified, panic disorder with agoraphobia, adjustment disorder with mixed anxiety and depressed mood, Dyssomnia not otherwise specified, Somatoform Disorder NOS 300.82

AXIS II: Rule out cluster B personality disorder traits.

AXIS III: See past medical, past surgical history.

AXIS IV: medical problems since October 2012, fear of unknown medical conditions, unable to be with her children, unemployment, no insurance, primary support problems.

Supportive factors: supportive mother, denies any legal problems, denies use of illicit substances or alcohol, housing.

AXIS V: 55.

Recommendations: “The patient is exhibiting depression, albeit subclinical, recurrent anxiety and insomnia. For that reason, we will initiate Remeron 15 mg nightly for depression and anxiety. Remeron will boost her appetite and help with sleep as well. The patient does not require further psychiatric assessment secondary to no suicidal/homicidal risks.

-The patient’s TSH, B12, folate, and RPR were already tested and are normal.

-Check Thiamine levels to r/o Thiamine deficiency disease.

- Check ionized calcium, prolactin, and serum follicle-stimulating hormone level

- Consult dietary for nutritional rehabilitation assessment”

**03/29/2013 Psychiatry Consult by Stephen M. Scheinthal, D.O.**

HPI: “The patient is a 25-year-old female, she was admitted to the ICR or to the CCU, after presenting to ED with her parents for right-sided chest pain and left arm numbness from her shoulder and neck to fingertips. The patient has had multiple hospitalizations recently, history of recurrent DKAs with blood sugars up to the 900 and on admission her blood glucose was 725. Concern from the staff about improper use of insulin and diabulimia. The patient states that she uses her insulin appropriately at home. She is eating regular meals, waffles, and banana for breakfast, peanut butter and jelly on wheat bread for lunch and whatever her parents cook for her at dinner at rice, corn beef, chicken, pasta, anything. Mom does confirm this and states that the patient eats a lot, but cannot seem to put on weight. She does not have any concerns for the patient as far as suicidality or psychiatric symptoms aside from some anxiety related to her children, who do not live with her. The patient had admitted to some anxiety symptoms specifically stating that she has some panic symptoms associated with claustrophobia. She denies agoraphobia. The patient also denies symptoms of major depressive disorder, bipolar disorder, psychotic or schizophrenic diagnosis or any PTSD. The patient denies binging on food, fasting, purging, use of laxatives or diabulimia. She states that her blood sugars at home run in around 180s and they never in the 500s and above like they are here. She does not feel like she wants to lose weight and in fact believes that she should be gaining weight as concerned that she cannot seem to do that. She does reports that she has had difficulty sleeping at home and is only sleeping about 5 hours. She reports she has been on Ambien in the past, which worked but now is unable to afford that without insurance. She states that Remeron, which started at her last hospitalization has not helped her sleep and there has been no change in her appetite, which she reports as bad.”

Mental status exam: “General: The patient is alert and oriented x3. She is tearful and crying in pain at times. She was calm and cooperative through the interview. Her speech is productive, normal volume, rate and tone. She is in hospital garb, slightly disheveled. Her mood is “okay.” Affect is tearful, slightly anxious. Thought process is logical linear. No suicidality. No homicidality. No auditory or visual hallucinations. No psychomotor agitation or retardation. Insight/judgment is fair.”

Impression: “This is a 25-year-old female with type 1 diabetes and multiple ICU hospitalizations. Currently, appears rather cachectic though the patient denies eating disorder symptoms as far as purging, binging, use of laxatives, inappropriate dosing of her insulin. She also denies any body image dysregulation and does believe that she needs to gain weight. So at this point, I do not believe that she meets criteria for an eating disorder. She does admit to anxiety and some insomnia.”

Diagnoses:

AXIS I: Panic disorder without agoraphobia, rule out somatoform disorder, not otherwise specified. Adjustment disorder with mixed anxiety and depression.

AXIS II: Deferred.

AXIS III: See past medical history.

AXIS IV: Recurrent medical problems. Unable to be with her children, unemployment, no insurance, primary support problems.

AXIS V: Global Assessment of Functioning 55.

Recommendations: “At this point, the patient does not meet clear criteria for any eating disorder. It is unclear at this time why she continues to have unmanageable diabetes as the patient does admit to some anxiety and possibly some symptoms of depression including insomnia. The patient would be appropriate to be continued on Remeron since this can possibly be increased to 30 mg, if she continues to have no response to 15 mg. Would continue with dietary management and would consult Endocrinology. We will discuss with primary team.”

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