Case Study #16
Pediatric Type 1 Diabetes Mellitus

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Intro

  Rachel Roberts is a 12-year-old 7th grader, previously in good health, who is admitted through the ED after fainting at soccer practice with a new diagnosis of acute-onset hyperglycemia. During the ED assessment, the patient was noted to have abnormally high serum glucose levels (724 mg/dL). She had recently healed from strep throat a few days ago. Rachel has lately experienced weight loss despite increased hunger, increased thirst, and frequent urination—" thirstier than I have ever been in my whole life and then I have had to use the bathroom a lot. . . . I even have to get up at night to go to the bathroom." After conducting several tests, the patient is presented with a new diagnosis of Type 1 Diabetes Mellitus. Additionally, her mother suffers from hyperthyroidism and her sister has celiac disease, which may increase Rachel's risk of developing these autoimmune diseases.

Understanding the Diagnosis and Pathophysiology

1. *What are the current thoughts regarding the etiology of type 1 diabetes mellitus (T1DM)? No one else in Rachel's family has diabetes—is this unusual? Are there any other findings in her family medical history that would be important to note?*

  Type 1A diabetes mellitus is one of the most common chronic diseases in childhood and results from autoimmune destruction of the insulin-producing beta cells in the islets of Langerhans. This process occurs in genetically susceptible individuals, may be triggered by environmental agents such as viral infections, and usually progresses over many months or years during which the individual is asymptomatic and euglycemic. Thus, genetic markers for type 1A diabetes are present from birth, immune markers are detectable after the onset of the autoimmune process, and metabolic markers can be detected with sensitive tests once enough beta-cell damage has occurred, but before the onset of symptomatic hyperglycemia. This long latent stage is a reflection of the large number of functioning beta cells that must be lost before hyperglycemia occurs. Hyperglycemia and symptoms develop only after 90% of the secretory capacity of the beta-cell mass has been destroyed. In contract, Type 1B diabetes mellitus, also known as idiopathic diabetes, refers to non-autoimmune islet destruction.[1]

The causes of type 1 diabetes are unknown, although several risk factors have been identified, including family history, genetics, geography, and age:[2]

Family History: The lifelong risk of Type 1 diabetes mellitus (T1DM) is markedly increased in close relatives of a patient with type 1 diabetes, with approximately 1-4% in offspring of an affected mother, 3-8% in offspring of an affected father, up to 30% in offspring with both parents affected, 5% in siblings, 50% in identical twins versus 0.4% in individuals with no family history.[1,2] Furthermore, a predisposition to develop T1DM is passed through generations in families, but the inheritance pattern is unknown.[3]

Genetics: The risk of developing T1DM is increased by certain variants of the HLA-DQA1, HLA-DQB1, and HLA-DRB1 genes. These genes provide instructions for making proteins that play a critical role in the immune system. The HLA-DQA1, HLA-DQB1, and HLA-DRB1 genes belong to a family of genes called the human leukocyte antigen (HLA) complex. The HLA complex helps the immune system distinguish the body's own proteins from proteins made by foreign invaders such as viruses and bacteria. HLA genes have many variations, and individuals have a certain combination of these variations, known as a haplotype. Certain HLA haplotypes are associated with a higher risk of developing T1DM, with specific combinations of HLA-DQA1, HLA-DQB1, and HLA-DRB1 gene variations resulting in the highest risk. These haplotypes may increase the risk of an inappropriate immune response to beta cells. HLA variations account for approximately 40% of the genetic risk for T1DM. Other HLA variations appear to be protective against the disease. Additional contributors, such as environmental factors and variations in other genes, are also thought to influence the development of this complex disease.[3]

Geography: The incidence of T1DM tends to increase as countries are located farther away from the equator, therefore being more common in cooler climates.[2]

Age: Although T1DM can occur at any age, it appears at two noticeable peaks. The first peak occurs in children from 4 to 7 years of age, and the second peak at puberty occurs in children from 10 to 14 years of age. Approximately 45% of children present before 10 years of age.[2]

Important findings in Rachel's family medical history entail her mother having hyperthyroidism and her sister diagnosed with celiac disease. As such, autoimmune

thyroid disease and celiac disease are two immune-mediated diseases that occur with increased frequency among children and adolescents with T1DM. Additionally, females are at greater risk for multiple autoantibodies and are therefore more susceptible to autoimmune diseases. Up to 20% of patients with T1DM have positive antithyroid antibodies (anti-thyroid peroxidase and/or anti-thyroglobulin). Patients with circulating antibodies may be euthyroid, or they may develop autoimmune hypothyroidism, with a prevalence of approximately 2-5% in patients with T1DM. Moreover, approximately 1-16% of patients with T1DM will develop celiac disease compared with 0.3-1% in the general population. Most cases of celiac disease are diagnosed within five years of diabetes onset.[4]

Overall, Rachel may have inherited a genetic predisposition to developing autoimmune thyroid disorders and celiac disease. Because of the high prevalence of thyroiditis and celiac disease and their potential clinical impact, Rachel like all children with T1DM should be screened for celiac disease and screened regularly for thyroid disease by measuring TSH.[4]

2. *What are the standard diagnostic criteria for T1DM? Which are found in Rachel's medical record?*

Diagnostic criteria for diabetes based upon the guidelines of the American Diabetes Association — diabetes mellitus is diagnosed based upon one of the following signs of abnormal glucose metabolism:[5]

- Fasting plasma glucose ≥126 mg/dL (≥7 mmol/L) on more than one occasion. Fasting is defined as no caloric intake for at least eight hours.

- Random plasma glucose ≥200 mg/dL (≥11.1 mmol/L) in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis.
    - *Rachel's medical record reports serum glucose of 724 mg/dL along with symptoms of unintentional weight loss, polydipsia (excessive thirst), polyuria (excessive urination), polyphagia (excessive hunger), and diagnosis of acute-onset hyperglycemia.*
    - *Note: most children and adolescents are symptomatic and have plasma glucose*

concentrations well above ≥200 mg/dL.

- Plasma glucose ≥200 mg/dL (≥11.1 mmol/L) measured two hours after a glucose load of 1.75 g/kg (maximum dose of 75 g) in an oral glucose tolerance test.

- Glycated hemoglobin (A1C) ≥6.5%. This criterion is more useful to the diagnosis of type 2 diabetes mellitus in adults and should be confirmed by hyperglycemia.
  - *Rachel's medical laboratory test presents with AIC 14.6% (high).*

- The presence of islet-specific autoantibodies supports the diagnosis of T1DM, but is not sufficient to make the diagnosis. The absence of pancreatic autoantibodies does not rule out the possibility of T1DM.
  - *Rachel tested positive for autoantibodies consistent with T1DM: Islet Cell Cytoplasmic Autoantibodies (ICA), Insulin Autoantibodies (IAA), Glutamic Acid Decarboxylase Autoantibodies (GADA), Insulinoma-Associated-2 Autoantibodies (IA-2A).*

3. *Dr. Cho requested these labs be drawn: Islet cell autoantibodies screen; TSH; thyroglobulin antibodies; C-peptide; immunoglobulin A level; hemoglobin A1c; and tissue transglutaminase antibodies. Describe how each is related to the diagnosis of type 1 diabetes.*

Islet cell autoantibodies screen
T1DM is a chronic autoimmune disease in which the immune system erroneously targets and destroys insulin-producing beta cells in the pancreas, eventually leading to absolute insulin deficiency.[6] In patients with clinical symptoms of T1DM, the presence of one or more islet autoantibodies indicates pancreatic autoimmunity and is consistent with a diagnosis of T1DM.[6] More specifically, islet autoantibodies are proteins produced by the immune system that recognize antigens found in insulin-producing pancreatic beta cells.[2]

TSH
Thyroid-stimulating hormone (TSH), is the most useful screening test for thyroid

dysfunction. TSH should be tested several weeks after the diagnosis of T1DM, when metabolic control has been established. This is because at least 20% of patients will have transient abnormalities of thyroid function when T1DM is first diagnosed, which resolves as the diabetes is treated. Approximately 2 to 5% of patients with T1DM are hypothyroid. Hashimoto's thyroiditis is an autoimmune disease where the thyroid cells are destroyed and type 1 diabetics have a greater risk of developing this specific form of autoimmune thyroid disease.[4]

Thyroglobulin antibodies
Thyroglobulin antibodies are also associated with autoimmune thyroid disease and can be used as an additional diagnostic marker. The appearance of antithyroglobulin autoantibodies in T1DM precedes thyroid dysfunction. Hyperthyroidism may worsen glycemic control while hypothyroidism alters carbohydrate metabolism.[4]

C-peptide
C-peptide (connecting peptide) is a short 31-amino-acid polypeptide that connects insulin's A-chain to its B-chain in the proinsulin molecule. In the context of diabetes, a measurement of C-peptide blood serum levels can help to distinguish between type 1 diabetes from type 2 diabetes. Testing for C-peptide helps to determine how much natural insulin is produced by the patient since it is secreted in equimolar amounts to insulin. Diagnosis of type 1 diabetes is characterized by insulin deficiency and accordingly by very low C-peptide levels.[2]

Immunoglobulin A
Immunoglobulin A (IgA) is an antibody that plays a vital role in mucosal immunity. More IgA is produced in mucosal linings than all other types of antibodies combined. Metabolic abnormalities and alteration of immunoglobulins levels are frequently observed in patients since T1DM is an immune-mediated disease associated with other autoimmune diseases.[2,6]

Tissue transglutaminase antibodies
Anti-tissue transglutaminase antibodies (tTGA), under IgA class, are the serological marker of celiac disease. Positivity for celiac disease-related antibodies allows the

identification of T1DM patients with suspected CD who will undergo a duodenal biopsy to confirm the diagnosis. The tTGA test displays the highest sensitivity, allowing the identification of approximately 98% of T1DM patients with untreated celiac disease.[7]

Hemoglobin A1c

Glycated hemoglobin (A1C) test indicates the average blood glucose level for the past two to three months. It measures the percentage of blood glucose attached to the oxygen-carrying protein in red blood cells (hemoglobin). The higher the blood glucose levels, then the more glycated hemoglobin levels the patient will have. An A1C level of 6.5% or higher on two separate tests indicates diabetes.[2]

4. *Using the information from Rachel's medical record, identify the factors that would allow the physician to distinguish between T1DM and T2DM.*

The initial step is to diagnose diabetes. The second step is to differentiate T1DM from other causes of diabetes. No set of criteria or diagnostic tests can consistently distinguish between T1DM and T2DM. Therefore, differentiating between the two types is based upon a combination of the clinical presentation and history, often supported by laboratory studies.[5] The following laboratory tests are usually helpful in differentiating between T1DM and T2DM especially when uncertain by clinical presentation:[5]

Antibodies

Although there is no specific test to distinguish between the two types of diabetes, T1DM is suggested by the presence of circulating, islet-specific, pancreatic autoantibodies.[2] However, the absence of pancreatic autoantibodies does not rule out the possibility of T1DM.[5] Up to 30% of individuals with the classical appearance and presentation of T2DM have positive autoantibodies and may have a slowly progressive type of autoimmune diabetes.[5] Rachel tested positive for autoantibodies consistent with T1DM including Islet Cell Cytoplasmic Autoantibodies (ICA), Insulin Autoantibodies (IAA), Glutamic Acid Decarboxylase Autoantibodies (GADA), Insulinoma-Associated-2 Autoantibodies (IA-2A).

Insulin and C-peptide levels

C-peptide is an indicative marker of insulin synthesis.[2] High fasting insulin and C-peptide levels suggest T2DM.[5] However, in T1DM, insulin and C-peptide levels are very low or in the normal range relative to the concomitant plasma glucose concentration.[5] As such, Rachel's C-peptide (0.10) is inappropriately low compared to normal levels (0.51–2.72) indicating T1DM.

Table 1[2,5] - Characteristics of type 1 and type 2 diabetes mellitus in children and adolescents		
	Type 1 diabetes	**Type 2 diabetes**
Prevalence	Common, increasing	Increasing
Age at presentation	Throughout childhood	Puberty
Onset	Typically acute severe	Insidious to severe
Ketosis at onset	Common	5 to 10%*
Affected relative	5 to 10%	75 to 90%
Female:male	1:1	Approximately 2:1
Inheritance	Polygenic	Polygenic
HLA-DR3/4	Strong association	No association
Ethnicity	Most common in non-Hispanic whites	All
Insulin secretion	Decreased/absent	Variable
Insulin sensitivity	Normal when controlled	Decreased
Insulin dependence	Permanent	Variable
Obese or overweight	20 to 25% overweight	>80% obese
Acanthosis nigricans	12%	50 to 90%
Pancreatic antibodies	Yes	No (although possible)

5. *Describe the metabolic events that led to Rachel's symptoms and subsequent admission to the ED (polyuria, polydipsia, polyphagia, fatigue, and weight loss), integrating the pathophysiology of T1DM into your discussion.*

The earliest symptoms of diabetes include polyuria (due to glucose-induced osmotic diuresis), polydipsia (due to increased urinary water losses), polyphagia (due to decreased energy stores), fatigue, and weight loss.[2]

Polyuria

Polyuria occurs when the serum glucose concentration rises significantly above 180 mg/dL, exceeding the renal threshold for glucose, which leads to increased urinary glucose excretion. Glucose in urine (glycosuria) causes osmotic diuresis or increased urination (polyuria) and hypovolemia. Polyuria can be identified by needing to urinate during the night and frequent bathroom trips.[5]

Polydipsia

Polydipsia is due to enhanced thirst because of increased serum osmolality from hyperglycemia and hypovolemia. Rachel also has some of the classic signs including dry mucous membranes of the throat and warm, dry skin indicating mild-moderate dehydration.[5]

Polyphagia

Polyphagia is characterized by excessive hunger or increased appetite. The lack of fuel kicks the body into a starvation state, stimulating hunger as a response. Simply eating will not get rid of polyphagia in those with uncontrolled/undiagnosed diabetes, as this will further exacerbate hyperglycemia.[2]

Weight loss

Weight loss is a result of hypovolemia and increased catabolism. Insulin deficiency in diabetic children impairs glucose utilization in skeletal muscle and increases fat and muscle breakdown. Initially, appetite is increased, but over time, children are more thirsty than hungry. Additionally, ketosis leads to nausea and anorexia, contributing to weight loss.[5]

The 3 P's of diabetes (polyuria, polydipsia, polyphagia) all stem from high blood glucose levels. Blood glucose is normally filtered by the kidneys and then reabsorbed

into the blood. When blood glucose levels are too high, the kidneys cannot reabsorb all of the glucose, so the excess glucose ends up being excreted in the urine. When there is glucose in the urine, excess body water is lost in the urine and thus polyuria develops. This loss of body water contributes to dehydration and increased thirst, or polydipsia. Therefore, polydipsia is a consequence of polyuria. Thus, an increase in fluid intake due to polydipsia can also contribute to increased urination. With impaired glucose utilization, the body is left without fuel. The body compensates by catabolizing muscle as well as fat (lipolysis); using ketones as fuel. Additionally, hepatic gluconeogenesis occurs as a response to the body's "starved" cells, resulting in excessive hunger (polyphagia). Fatigue may occur as a result of low energy stores, electrolyte imbalances, dehydration, and the body trying to regain glucose homeostasis. Mobilization of fat stores, muscle breakdown, and fluid loss due to polyuria contribute to weight loss.[1,2]

6. *Describe the metabolic events that result in the signs and symptoms associated with DKA. Was Rachel in this state when she was admitted? What precipitating factors may lead to DKA?*

      Diabetic ketoacidosis is an acute metabolic complication of diabetes. Signs and symptoms of DKA which include symptoms of hyperglycemia as well as nausea, vomiting abdominal pain are the result of acidosis, hyperglycemia, volume depletion, and electrolyte losses. First, hyperglycemia of DKA evolves through accelerated gluconeogenesis, glycogenolysis, and decreased glucose utilization, all of which are due to absolute insulin deficiency in T1DM. As a result, stress hormones (cortisol, glucagon, growth hormone, and catecholamines) increase the rate of lipolysis, whereby abundant free fatty acids are converted to ketone bodies for fuel; β-hydroxybutyrate, acetoacetate, and acetone. The production of ketones in blood ensues (hyperketonemia), resulting in an acidic blood pH. Additionally, hyperglycemia-induced polyuria leads to polydipsia, dehydration, hyperosmolarity, electrolyte loss, and subsequent decrease in glomerular filtration. With impaired insulin action and hyperosmolality, the utilization of potassium by skeletal muscle is markedly diminished leading to intracellular potassium depletion. Also, potassium is lost via polyuria and may cause total body potassium deficiency.[2,8]

Precipitating Factors

Poor metabolic control or missed insulin doses: Insulin omission and other diabetes mismanagement accounts for the majority of DKA episodes in children with established diabetes.[2,9]

Illness: Intercurrent illnesses, particularly when associated with vomiting and dehydration, can precipitate DKA by increasing stress hormone levels that increase hepatic glucose output, cause peripheral insulin resistance, and promote ketogenesis.[2] Illnesses involving vomiting are particularly problematic because they interrupt food intake, often requiring a reduction in the amount of insulin administered.[9]

Medications: Certain medications, such as corticosteroids, atypical antipsychotics, diazoxide, and high-dose thiazides, have precipitated DKA in individuals not previously diagnosed with T1DM.[9] In children with established diabetes, use of corticosteroids can also lead to substantial insulin resistance with hyperglycemia and occasionally ketosis.[9]

Drugs and alcohol: In adolescents with T1DM, the use of illicit drugs and alcohol may interfere with adherence to good medical management recommendations, resulting in poor metabolic control, which increases the risk for DKA.[9]

Rachel's lab and physical screenings indicate the presence of ketones in the urine (ketones +1), acidic urine, low sodium/phosphate/anion gap (electrolyte and acid-base disturbances) as well as symptoms of polyuria, polydipsia, polyphagia, dehydration (water loss of 71.8 mL/kg), weight loss, weakness, fainting, and confusion all of which indicate diabetic ketoacidosis. Therefore, Rachel was most likely in a state of DKA at the time of admittance. Approximately 30% of children who present with newly diagnosed type 1 diabetes are ill with DKA.[9] Rachel was treated with insulin administered as an IV infusion at a rate of 0.1 unit/kg/hr, gradually increased to 5 units/hr to increase the rate of blood glucose decline to achieve the blood glucose target.

7. *Rachel will be started on a combination of Apidra prior to meals and snacks, with glargine given in the a.m. and p.m. Describe the onset, peak, and duration for each of these types of insulin. Her discharge dosages are as follows: 7 u glargine with Apidra prior to each meal or snack—1:15 insulin:carbohydrate ratio. Rachel's parents want to know why she cannot take oral medications for her diabetes like some of their friends do. What would you tell them?*

Multiple daily injections (MDI) combines a baseline level of insulin using a long-acting insulin analog (i.e. insulin glargine) with pre-meal/snack boluses of rapid- or short-acting insulin.[10] This approach results in more stable glycemic control and fewer episodes of hypoglycemia than the conventional approach in children.[10] Apidra is a mealtime insulin that is taken 15 minutes before consumption or within 20 minutes after starting a meal.[11] It is a rapid-acting insulin that has an onset action of approximately 15 minutes, peaks within an hour, and has a duration of 2 to 4 hours.[11] As such, Apidra can closely mimic normal physiologic insulin secretion at mealtime.[11] In contrast, Glargine is a long-acting insulin that has an onset of 1.5 to 2 hours, peaks at 5 hours, and has a duration of 24 to 26 hours.[12] The half-life may be shorter in some individuals, requiring division of the daily dose into two injections per day.[10] Accordingly, Galgrine mimics the natural physiological profile of basal endogenous insulin secretion with no pronounced peaks.[12] Furthermore, the basal/bolus insulin regimen has been documented to result in stable glycemic control and less hypoglycemia compared with regimens using intermediate and short insulin regimens.[13]

It is important to educate the family on the metabolic differences between type 1 diabetes and type 2 diabetes. Oral medications are only suitable for those who are able to secret insulin such as in T2D to help attenuate insulin resistance.[2] Additionally, challenges of oral medication include poor bioavailability due to inactivation of insulin by proteolytic enzymes, low permeability, and hydrophobicity.[2] In type 1 diabetes, the pancreas stops producing insulin altogether, relying solely on exogenous insulin.[2] Therefore, injecting insulin directly into the bloodstream is essential and is the most effective route of delivery.[10]

Although the MDI regimen is recommended because of its increased flexibility, it requires frequent blood glucose monitoring and insulin injections.[10] Rachel and her family are required to count dietary carbohydrates and accurately judge the effect of exercise on insulin requirements. Without this active management by a well-trained patient and family, the benefits of this regimen are not attained.[10] Before initiation of the MDI regimen, the patient and family must understand and accept the increased commitment and the need for increased frequency of blood monitoring and insulin injections required by this therapeutic approach.

8.   *Rachel's physician explains to Rachel and her parents that Rachel's insulin dose may change*

due to something called a honeymoon phase. Explain what this is and how it might affect
her insulin requirements.

A few weeks after the diagnosis and initiation of insulin therapy, a period of
decreasing exogenous insulin requirement occurs, commonly referred to as the
"honeymoon" or remission phase of diabetes.[2] During this period, the remaining
functional beta cells secrete some endogenous insulin resulting in reduced exogenous
requirement.[2] Rachel may require smaller doses of insulin during this time as symptoms
will not show during this period. Close monitoring of blood glucose is mandatory as
hypoglycemic episodes are likely if the insulin dose is not appropriately adjusted.[2] The
duration of this phase is variable and may last several months to several years.[10] Rising
blood glucose levels, A1C, and increasing exogenous insulin requirements indicates the
end of this phase.[2]

9. *How does physical activity affect blood glucose levels? Rachel is a soccer player and usually*
    plays daily. What recommendations will you make to Rachel to assist with managing her
    glucose during exercise and athletic events?

Regular exercise has important health and social benefits for children and
adolescents with T1DM and should be encouraged.[13] Exercise also presents several
important challenges, which require careful management in T1DM. Increased physical
activity increases hypoglycemia risk by approximately 30 to 50% both during and after
exercise (for up to approximately 12 hours) in children and adolescents with T1DM.[10]
Exercise induces hypoglycemia because it enhances insulin sensitivity in a temporal
pattern that depends on the time of day as well as the type and duration of exercise
performed.[14] When exercise is performed early in the day, heightened insulin sensitivity
is sustained for at least 11 hours after the exercise.[10] In contrast, exercise performed late
in the day is associated with a biphasic change in insulin sensitivity, such that
heightened insulin sensitivity occurs during the exercise and again 7 to 11 hours later
during the overnight hours.[10] This increases the risk for hypoglycemia late in recovery,
usually when the child or adolescent is sleeping.[10] As a result, afternoon exercise tends
to increase hypoglycemia risk compared with morning exercise.[2] With immediate
hypoglycemia, muscle contraction increases glucose uptake via insulin-independent

mechanisms.[2] Contributing factors include delayed glucose-lowering effects of exercise, sleep-induced defects in counterregulatory hormone responses to hypoglycemia, and missed bedtime snacks.[14]

Although exercise typically causes a decrease in BG concentration in individuals with T1DM, it can also cause an increase in BG if the exercise is of a very high intensity for a short duration, such as sprinting or resistance exercise. High-intensity interval exercise in a fasted state tends to promote a somewhat predictable rise in glucose concentration that may warrant insulin administration. Hyperglycemia and excessive ketosis during exercise are particularly undesirable as they can cause dehydration and may decrease blood pH, both of which impair exercise performance and place the child at risk for metabolic deterioration into ketoacidosis. High-intensity anaerobic exercise (>80 percent VO2max = maximal oxygen uptake) can cause hyperglycemia because it induces catecholamine release, which promotes hepatic glucose production and reduced glucose clearance. Management of glycemia is further complicated by participation in team sports that require bursts of intense activity punctuated by rests in play, such as baseball, soccer, basketball, or hockey), in which case the periods of inactivity also contribute to the risk for hyperglycemia.[2,10]

Consumption of extra carbohydrates. Children or adolescents with T1DM generally need to consume extra carbohydrates prior to, during, and after exercise that lasts more than 60 minutes. This strategy also is most practical when exercise is not always predictable, and when timely insulin reduction is not possible. The timing and dose of carbohydrate depends on the type, duration, and intensity of exercise, concurrent adjustments of the insulin dose, and the unique responses of the individual patient. The dose should be adjusted as needed depending on serial monitoring of BG. More specifically, individuals relying on exogenous insulin may need to ingest some added carbohydrate if pre-exercise glucose levels are 100 mg/dL (5.6 mmol/L).[14]

Reduction in exogenous insulin dose. Reducing the insulin dose given prior to exercise reduces the risk for hypoglycemia and the need for exogenous carbohydrates.[14] Rachel plays soccer daily which entails moderate to vigorous-intensity aerobic exercise (continuous or intermittent exercise lasting more than 60 minutes), thus adjustment of the pre-exercise insulin regimen is recommended, in addition to extra carbohydrate

intake.[14]

Post-exercise recovery period and protein intake. During the recovery period after moderate or vigorous exercise, it is generally helpful to consume a snack consisting of mixed protein and carbohydrates.[14] Examples of suitable recovery snacks include dairy-based fruit smoothies, low-fat milkshakes, yogurt drinks, and fruit mixed with yogurt, with an estimated carbohydrate:protein ratio of 2:1.[10] The snack should generally be taken with insulin to avoid post-exercise hyperglycemia. The mixed composition of the snack helps to avoid post-exercise, late-onset hypoglycemia and also supports protein synthesis.[10]

BG monitoring. BG should be monitored frequently before, during, and after exercise because glucose levels can change rapidly, and good glycemic control is important to maintain performance and safety.[14] In episodic monitoring, at least two measurements of BG should be taken in the hour prior to exercise so that the direction of change in BG concentrations can be assessed and so that preemptive interventions can occur before the start of the activity.[14] It is advised to measure BG every 30 minutes during the exercise to help anticipate and prevent hypo- or hyperglycemia.[14] Increased frequency of monitoring during the recovery period after exercise is also recommended to avoid post-exercise, late-onset hypoglycemia.[2] On the other hand, continuous glucose monitoring is performed using a subcutaneous sensor that continuously measures glucose levels in interstitial fluid. CGM is reasonably accurate during both aerobic and anaerobic exercise.[10] Directional rates of change on CGM systems and sensor-augmented pumps can inform individuals if glucose concentrations are outside of the target range, and alerts and alarms are useful to alert the patient to hypo- or hyperglycemia.[10]

Rachel should fully participate in physical education classes and team sports at school, provided that there is good collaboration between her, the health care provider, parents, the school nurse, and the physical education instructor or team coach and good adherence to a well-designed regimen for glycemic control during and after exercise. The supervising staff must be trained to recognize and treat hypoglycemia, and the student should have ready access to BG monitoring equipment and fast-acting carbohydrates.[14]

10. *At a follow-up visit, Rachel's blood glucose records indicate that her levels have been consistently high when she wakes in the morning before breakfast. Describe the dawn phenomenon. Is Rachel experiencing this? How might it be prevented?*

Certain time periods during the day may require higher, while other periods may require lower insulin intake depending on individual factors including lifestyle as well as the dawn phenomenon.[2] The dawn phenomenon is characterized by an abnormal early-morning increase in blood glucose, usually between 2 AM and 8 AM.[2] The dawn phenomenon is thought to result from increased secretion of hormones, particularly growth hormone, that tend to antagonize the actions of insulin resulting in an increase in blood glucose concentrations.[10] The amount of carbohydrates and insulin intake at bedtime can be adjusted to maintain the pre-breakfast blood glucose in the target range.[10] Overall, Rachel's elevated blood levels during the morning may be indicative of the dawn phenomenon. However, more information is needed to confirm this assumption.

To avoid the dawn phenomenon one can:[2]
- Avoid carbohydrates at bedtime.
- Adjust dose of medication or insulin.
- Change the time of taking insulin from dinnertime to bedtime.
- Administer extra insulin during early-morning hours.

Understanding the Nutrition Therapy

11. *The MD ordered a carbohydrate-controlled diet for when Rachel begins to eat. Explain the rationale for monitoring carbohydrates in diabetes nutrition therapy.*

Patients with diabetes along with hyperglycemia who are eating should be on a consistent-carbohydrate diet, and glucose monitoring should be ordered before each meal and at bedtime. Carbohydrates are the main energy source in the diet and include starches, vegetables, fruits, dairy products, and sugars. Most meats and fats do not contain any carbohydrates. Carbohydrates have a direct impact on the blood sugar level

whereas proteins and fat have little impact. The amount of carbohydrate ingested is regarded as the major factor of postprandial glucose and insulin response. Therefore, eating a consistent amount of carbohydrates at each meal can help to control blood sugar levels, especially if the patient is taking long-acting insulin. There are several ways to calculate the carbohydrate content of a meal, including carbohydrate counting and exchange planning. Carbohydrate counting is a method based on the principle that all types of carbohydrates, with the exception of fiber, are digested with the majority being absorbed into the bloodstream as glucose molecules and that the total amount of carbohydrates has a greater effect on blood glucose elevations than the specific type. The RD usually helps to determine the number of carbohydrates needed at each meal and snack, based upon the patient's usual eating habits, insulin regimen, body weight, nutritional goals, and activity level. Also, the way carbohydrates are divided up for each meal or snack is based upon personal preferences, meal timing and spacing, and type of insulin regimen.[2]

The carbohydrate-controlled diet (CCHO) helps patients with diabetes keep their carbohydrate consumption at a steady level, through every meal and snack. Thus keeping carbohydrate intake approximately the same throughout the day, and every day of the week. This prevents blood sugar spikes or falls. The CCHO diet assigns units of measurements called "choices" to foods using the exchange system. About 15g of carbohydrates equals one carb "choice." Some patients receive fixed insulin regimens such as with the use of premixed insulins and do not need to adjust their mealtime insulin doses, having day-to-day consistency in the timing and amount of carbohydrates eaten.[2]

Rachel is initially prescribed a controlled carbohydrate diet using appropriate insulin regimens for when she begins eating. More specifically, Rachel is on an NPO diet except for ice chips and medications. After 12 hours, she can begin with clear liquids if stable and then advance to a CCHO diet order consisting of 70-80 g CHO breakfast and lunch; 85-95 g CHO dinner; 3-15 g CHO snacks. The amount of carbohydrates is individualized to her nutritional needs which is covered accordingly with appropriate insulin doses. It is necessary to consult the diabetes education team for self-management training for Rachel and her parents and begin education after stabilization.

12. Outline the basic principles for Rachel's nutrition therapy to assist in control of her T1DM.

Medical nutrition therapy is the process by which the nutrition prescription is tailored for people with diabetes based on medical, lifestyle, and personal factors and is an integral component of diabetes management and diabetes self-management education.

Basic principles of nutritional management
Diet and physical activity are critically important in the treatment of T1DM. The nutrition prescription for patients with T1DM should aim to optimally manage the "ABCs" of diabetes control: glycated hemoglobin (A1C), blood pressure and low-density lipoprotein cholesterol.[2] The prescription must also be tailored for the individual patient to address diabetes complications and other concomitant conditions. The nutritional goals for people with T1DM are to:[15]
- Maintain as near-normal blood glucose levels as possible, by integrating insulin therapy into each individual's diet and physical activity patterns.
- Achieve optimal blood pressure and lipid levels.
- Provide adequate calories for achieving and maintaining a reasonable body weight, normal growth, and development.
- Manage risk factors and prevent complications of diabetes, both acute (hypoglycemia and short-term illness) and long-term (hypertension, hyperlipidemia, renal disease, cardiovascular disease, and other micro- and macrovascular complications).
- Improve overall health through healthful food choices.
- Address individual nutrition needs, incorporating personal and cultural preferences, willingness to change, and maintaining the pleasure of eating by restricting choice only when clearly appropriate.

MNT for type 1 diabetes should consider five key aspects[15]
- Consistency in day-to-day carbohydrate intake at meals and snacks
- Adjusting insulin for variations in blood glucose, food, or activity
- Weight management (caloric intake balanced with caloric expenditure)
- Nutritional content (balance of selected protein, carbohydrates, and fats)

- Meal-insulin timing

Recommended macronutrient intake ranges[2]
- Carbohydrates: 45 to 65% of total caloric intake
- Protein: 15 to 20% of total caloric intake
- Fat: 25 to 35% of total caloric intake
  - Monounsaturated fat: Up to 20% calories
  - Polyunsaturated fat: Up to 10% calories
  - Saturated fat: < 10% of calories
  - Cholesterol: < 200 mg/day
  - Trans fat: < 1% of calories

Randomized controlled trials of MNT have demonstrated decreases in A1C of up to 1.9% in three to six months in patients with type 1 diabetes. In the Diabetes Control and Complications Trial (DCCT), specific diet behaviors were associated with achieving up to a 1 point lower mean A1C (8 versus 7%) in the intensive treatment group:[15]
- Adherence to the negotiated meal plan (diet consistency)
- Adjusting food and/or insulin in response to hyperglycemia
- Adjusting insulin dose for meal size and content
- Appropriate treatment of hypoglycemia (not overtreating hypoglycemia)
- Consistent habits with regard to consumption of a bedtime snack and avoidance of extra nighttime snacks

Nutrition Assessment
13. Assess Rachel's ht/age; wt/age; ht/wt; and BMI. What is her desirable weight?

Age: 12 years
Height: 5' (152.4 cm)
Usual weight: 90 lbs (41 kg)
Current weight: 82 lbs (37.2 kg)
Current BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) × 10,000 = 15.93 kg/m^2
BMI is calculated using weight and stature measurements for a child aged 2 to 20 years[5]
% Weight Change: 8.89%

Desirable weight: 93-100 lbs (42-45.4 kg)
Desirable BMI: 18-19.55 kg/m^2

Using CDC growth charts for girls 2 to 20 years (5th to 95th percentile)[2]		
Weight-for-age	between 25th and 50th percentiles	Normal
Stature-for-age	between 50th and 75th percentiles	Normal
BMI-for-age	between 10th and 25th percentiles	Lower end of normal

Tracking growth patterns over time can help the RD make sure the patient is achieving or maintaining a healthy weight. A single BMI-for-age calculation is not enough to evaluate long-term weight status because height and weight change with growth.[16] Rachel is currently placed between the 10th and 25th percentiles (18th percentile to be exact) on the CDC BMI-for-age growth chart, which means that 82% of children of the same age and sex in the reference population have a higher BMI-for-age. Based on the below cut-offs Rachel is in the healthy weight category, however, her BMI less than the 25th percentile may indicate that she is somewhat underweight. The patient experienced an unintentional weight loss of 8 lbs (approximately 9% body weight loss) from the time of her strep throat infection (about 10-14 days ago) leading up to her ED admittance with a T1DM diagnosis. Therefore, the patient's T1DM-related weight loss in a very short time period may indicate mild or at risk for malnutrition. Immediate intervention and management of her T1DM condition is crucial to prevent further weight loss and T1DM related complications.

It is more important to maintain a healthy weight for children with T1DM, as weight can influence diabetes and diabetes can influence weight. Rachel and most other children, when diagnosed with T1DM are underweight as undiagnosed or untreated T1DM can result in weight loss despite a normal or increased appetite.[2] Once diagnosed and treated properly, their weight usually returns to normal levels.[2] The patient's desirable weight should range from 93 lb to 100 lbs which would place her in the desirable weight-for-age (50th to 65th percentile) and BMI-for-age (50th to 75th percentile).[2] Of note, the patient should regain the 8 lbs that she lost recently to reach

her usual body weight, with an additional 3 to 10 lbs. It is very important to consider that as children approach puberty, their weight can vary significantly. During puberty, children grow taller, by as much as 10 inches, before reaching their full adult height. They also gain muscle and develop new fat deposits as their bodies become more like those of adults.[2]
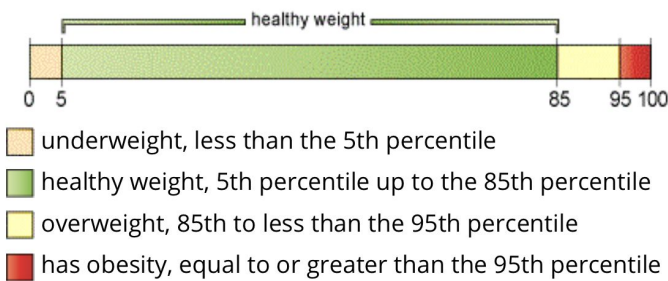

healthy weight

underweight, less than the 5th percentile
healthy weight, 5th percentile up to the 85th percentile
overweight, 85th to less than the 95th percentile
has obesity, equal to or greater than the 95th percentile

Figure 1[16] - Plotted measurements are based on the percentile ranking of the CDC growth charts (5th to 95th percentile) with the percentile cutoff value corresponding to the nutrition indicator

5th percentile	68 pounds
10th percentile	72 pounds
25th percentile	81 pounds
50th percentile	92 pounds
75th percentile	106 pounds
90th percentile	123 pounds
95th percentile	135 pounds

Figure 2[16] - The average weight of a 12-year-old female.

14. *Identify any abnormal laboratory values measured upon her admission. Explain how they may be related to her newly diagnosed T1DM.*

Chemistry	Ref Range	Upon Admission 5/4 1780	Explanation of abnormal lab values in T1DM diagnosis
Sodium, (mEq/L)	136–145	126 (low)	Electrolyte imbalance as a result of altered distribution of electrolytes

			related to hyperglycemia (induced osmotic fluid shifts)[2]
Glucose, (mg/dL)	70–99	683 (high)	Hyperglycemia due to lack of insulin
Phosphate, inorganic, (mg/dL)	2.2–4.6	1.9 (low)	Electrolyte imbalance as a result of altered distribution of electrolytes related to hyperglycemia (induced osmotic fluid shifts)[2]
Osmolality, (mmol/kg/H2O)	275–295	295.3 (slightly high)	Slightly hyperosmolar due to hyperglycemic state; dehydration, dry mucous membranes[2]
HbA1C, (%)	3.9–5.2	14.6 (high)	Hyperglycemia over the last 90 to 120 days depending on how fast red blood cells are replaced)[2]
C-peptide (ng/mL)	0.51–2.72	0.10 (low)	Indicates inadequate insulin production from pancreas[10]
ICA	—	+ (present)	Presence of multiple islet cell autoantibodies strongly indicative of T1DM[1,3]
GADA	—	+ (present)	
IA-2A	—	+ (present)	
IAA	—	+ (present)	Signal an autoimmune pathogenesis of β-cell killing[4]
Urinalysis			Proteinuria, glycosuria, ketonuria due to hyperglycemia as a result of insulin deficiency.[2,9]
Specific gravity	1.001–1.035	1.036 (slightly high)	
pH	5–7	4.9 (slightly low)	Slightly low urine pH due to ketones in urine (acidic waste)[9]
Protein (mg/dL)	Neg	+4 (present)	
Glucose (mg/dL)	Neg	+3 (present)	When high blood glucose levels exceed renal threshold, excess glucose and water are lost in urine.[2]
Ketones	Neg	+1 (present)	Ketones in urine may be due to breakdown of fats for energy since carbohydrate metabolism is defective.[9] Protein in urine may be due to hyperfiltration of kidneys as a result of hyperglycemia.[2]

15. Determine Rachel's energy and protein requirements. Be sure to explain what standards you used to make this estimation.

Institute of Medicine Equation for Normal weight children (Females 9-18 yrs):[17]

Estimated energy requirements (kcal/d) = 135.3 - 30.8 x Age [y] + PA x (10.0 x Weight [kg] + 934 x Height [m]) + 25

PA = 1.56 if very active (PAL ≥1.9 and <2.5)

> EER = 135.3 - 30.8 x 12 [y] + 1.56 x (10.0 x 37.2 [kg] + 934 x 1.524 [m]) + 25
>
> EER = **2591.55 kcal/day**

The factor of 25 is included to account for the energy cost of growth.

WHO and Schofield[18]

EER (kcal/day) = Resting Energy Expenditure X Activity Factor X Stress Factor

> **WHO Equation (Females 10-18 yrs):**[18]
>
> Estimated energy requirements (kcal/d) = 12.2W [kg] + 746 x PA
>
> PA = 1.56 very active (120 min. daily moderate activity OR 60 mins. moderate + 60 mins. vigorous activity)
>
> > EER = 12.2 x 37.2 [kg] + 746 = 1199.84 kcal/day
> >
> > TEE = 1199.84 kcal/day [EER] x 1.56 [PA] = **1871.75 kcal/day**

> **Schofield Equation (Females 10-18 yrs):**[18]
>
> Estimated energy requirements (kcal/d) = 8.365W + 4.65H + 200.0
>
> PA = 1.56 very active (120 min. daily moderate activity OR 60 mins. moderate + 60 mins. vigorous activity)
>
> > EER = 8.365 x 37.2 [kg] + 4.65 x 152.4 [cm] + 200.0 = 1219.84 kcal/day
> >
> > TEE = 1219.84 kcal/day [EER] x 1.56 [PA] = **1903 kcal/day**

Therefore,

Rachel's energy requirements will range from **2100-2600 kcal/day**

Dietary Reference Intakes for Total Protein[18]
Recommended Dietary Allowance for females 9-13 yrs = 0.95 g/kg/day

Protein RDA = 0.95 [g] x 37.2 [kg] = **35.34 g protein/day**

The Institute of Medicine Prediction Equation estimates the energy requirements of children and adolescents, ages 9 through 18 years, taking into account the physical activity coefficient. Total energy expenditure was predicted from age, height, and weight based on the stable isotope method, doubly-labeled water, and an average of 25 kcal/d for the energy cost of growth based on rates of weight gains from the Fels Longitudinal Study and rates of protein and fat deposition for adolescents. This equation was also used in the 2005 Dietary Guidelines for Americans and the new food pyramid, MyPyramid.[17]

Marked variability exists in the energy requirements of adolescents because of variable growth rates and physical activity levels.[2] A systematic review was conducted to examine the effect of puberty on energy expenditure.[17] Puberty was associated with a 12% increase in basal metabolic rate and an 18% increase in total energy expenditure compared with prepuberty.[17] The higher rates of total energy expenditure were accounted for largely by the pubertal increase in fat-free mass.[17]

Dietary estimated energy requirements of adolescents have been based on their total energy expenditure and requirements for growth, taking into account habitual physical activity level (PAL) and lifestyle consistent with the maintenance of health, optimal growth and maturation, and social and economic demands. The recommendations allow for four categories of PAL (sedentary, low active, active, and very active). Rachel's consistent soccer practice correlates with active or very active levels (at least 60 minutes of physical activity on most days of the week). PAL levels were defined based on the ratio of total energy expenditure:BMR.[2,17]

The WHO and Schofield predictive equations are also reliable and commonly used to predict resting energy expenditure in children and adolescents.[18] The REEs calculated using the WHO and Schofield equations were used as a reference for the lower end cut-off for Rachel's energy requirements. Overall, all these equations provide an estimate of energy requirement and relative body weight (i.e., loss, stable, gain) is the preferred indicator of energy adequacy.[18] It is recommended that the patient consume a range of 2100 to 2600 kcal/day and 35.34 g protein/day to achieve and maintain a healthy

and desirable weight.

Nutrition Diagnosis

16. prioritize two nutrition problems and complete the PES statement for each.

Food-and-nutrition-related knowledge deficit related to lack of exposure to diabetes education/management as evidenced by patient's new diagnosis of type 1 diabetes mellitus and inconsistent carbohydrate intake in patient's usual dietary intake.

> *Breakfast—cereal w/ milk or Pop-Tart® w/ milk*
> *Lunch for school—peanut butter and jelly or turkey and cheese sandwich, chips, carrots*
> *Before soccer practice—cereal or granola bar*
> *Dinner at mom's—salad, meat, and pasta, potato, or rice*
> *Dinner at dad's—pizza or Chinese food. Snacks include cereal, ice cream, yogurt, some fruits (apples, bananas), popcorn, chips, or cookies.*

Unintended weight loss related to undiagnosed type 2 diabetes mellitus as evidenced by approximately 9% weight loss (8 lbs) in 1-2 weeks; serum glucose of 724 mg/dL (70–99 mg/dl); HBA_{IC} 14.6% (3.9–5.2%); presence of autoantibodies ICA, IAA, GADA, IA-2A; symptoms of polydipsia, polyuria, and polyphagia; diagnosis of acute-onset hyperglycemia.

Nutrition Intervention

17. Determine Rachel's initial nutrition prescription using her usual intake at home as a guideline, as well as your assessment of her energy requirements.

Macronutrient Breakdown of usual daily intake					
Meal	Food items	kcal	CHO (g)	Protein (g)	Fat (g)
Breakfast	Cereal (1 C) w/ milk (1 C whole) Pop-Tart® (1 pkg) w/ milk (1 C whole)	281 561	40 91	10 12	10 19
Lunch	Peanut butter and jelly (1 sandwich) Turkey and cheese (1 sandwich) Chips (1 individual bag) Carrots (1 kg carrot)	300 250 180 30	32 29 15 7	10 17 3 1	15 7 12 0

Before Soccer Practice	*Cereal (1 C)* *Granola bar (1 bar)*	*130* *100*	*27* *18*	*2* *2*	*1* *2*
Dinner at Mom's	*Salad (1.5 C)* *Meat (Chicken, 3oz)* *Pasta (1 C)* *Potato (1 C)* *Rice (1 C)*	*15* *150* *166* *113* *205*	*3* *0* *30* *26* *45*	*1* *18* *5* *3* *4*	*0* *9* *3* *0* *0*
Dinner at Dad's	*Pizza (1 slice)* *Chinese (general tso chicken ½ order)*	*285* *789*	*36* *64*	*12* *34*	*10* *44*
Snacks	*Cereal (1 C)* *Ice Cream (1 C)* *Yogurt (1 container 6oz)* *Apple (1 med)* *Banana (1 med)* *Popcorn (1 C)* *Chips (1 individual bag)* *Cookies (1 med)*	*130* *273* *107* *95* *105* *44* *180* *148*	*27* *31* *12* *25* *27* *4* *15* *20*	*2* *5* *9* *0* *1* *0* *3* *2*	*1* *15* *3* *0* *0* *3* *12* *7*
	Total Daily Range	*1448-2633*	*174-300*	*60-102*	*49-105*

*Information adapted from https://fdc.nal.usda.gov/

We recommended that the patient consume a range of 2100 to 2600 kcal/day and 35.34 g protein/day to achieve and maintain a healthy and desirable weight. Additionally, based on Rachel's usual intake and on recommendations, she should consume approximately 45-60% of total calories from carbohydrates.[2]

(2100+2600kcal/day) ÷ 2 = 2350kcal/day × 45% total kcal from CHO = 1057kcal ÷ 4kcal/g = 265 g CHO/day ÷ 15 g CHO per serving = 17.6 ≈ 18 servings of CHO/day

(2100+2600kcal/day) ÷ 2 = 2350kcal/day × 60% total kcal from CHO = 1410kcal ÷ 4kcal/g = 350 g CHO/day ÷ 15 g CHO per serving = 23.5 ≈ 24 servings of CHO/day

Average CHO requirement: (265 g CHO/day + 350 g CHO/day) ÷ 2 = 300 g CHO/day
Apidra dosages are measured by 1-unit increments, thus 330 g CHO/day will be used to formulate nutrition prescription using below calculations. Although, estimates will vary depending on Rachel's individualized CHO intake.

Nutrition Prescription: Carbohydrate-controlled diet

2100 to 2600 kcal/day

35.34 g protein/day

330 g CHO/day breakdown:

90 g CHO breakfast ÷ 15 g CHO per serving = 6 servings of CHO

90 g CHO lunch ÷ 15 g CHO per serving = 6 servings of CHO

90 g CHO dinner ÷ 15 g CHO per serving = 6 servings of CHO

30 g CHO snacks (× 2) ÷ 15 g CHO per serving = 2 servings of CHO/per snack

- One snack before PE class
- One Snack before soccer practice

Rachel's nutrition/medication prescription consists of a carbohydrate-controlled diet with a Multiple Daily Injection insulin regimen using Galgrine and Apidra insulin. Rachel is newly diagnosed and presented with weight loss, as many patients typically experience weight loss when T1DM is first diagnosed. The lost weight is generally regained during the first few weeks of therapy due to insulin, hydration, and adequate energy intake.[2] During this time of increased consumption, children often require large amounts of insulin to control their blood glucose levels.[2] After the weight loss is corrected, ongoing assessment of growth, (weight, height, BMI) is necessary to monitor the adequacy of dietary intake and glycemic control.[2] Therefore, Rachel's initial nutritional recommendations are based on adequate calories, proper hydration, and exogenous insulin to restore and maintain appropriate body weight.[2] Individualized meal planning is highly recommended, thus it is preferable to use the patient's food and nutrition history of typical daily intake to further tailor energy needs.[2] Other general recommendations for Rachel are outlined below;

Carbohydrate Intake and Insulin Dose Adjustment
Education should be provided by a team of certified professionals, including physician, nurse, dietitian, diabetes educator, and mental health professional, that is dedicated to communicating basic diabetes management skills within a context that addresses family dynamics and issues.[2] After Rachel has learned and practiced the skills needed, she can assume the daily diabetes management tasks, such as insulin injections and blood glucose testing with supervision and support from family and trained adults.[2] Education

on the patient's new basal/bolus-MDI insulin regimen will be imperative. Rachel and her family should learn the concept of the insulin: carbohydrate ratio, and gain the needed skills to cover her CHO intake with appropriate insulin doses. Rachel will be on two insulin medications, Apidra and Glargine. Her pre-meal and pre-snack bolus doses of a rapid-acting insulin (Apidra) are based upon:[10]

- Pre-meal blood glucose level
- Estimated amount of carbohydrates to be consumed
- Expected level of exercise after the meal
- Exercise before the meal

Frequency of Blood Glucose Monitoring
Learning and implementing continuous blood glucose monitoring is crucial. Frequency and timing are dependent on MNT goals, diabetes medications, and physical activity.[2] For patients with type 1 diabetes on insulin therapy, at least three to eight blood glucose tests per day are recommended to determine the adequacy of the insulin doses and guide adjustments in insulin doses, food intake, and physical activity.[2,10] Some insulin regimens require more testing to establish the best integrated therapy (insulin, food, and activity). Intervention studies that include self-management training and adjustment of insulin doses based on SMBG result in improved glycemic control.[13] However, if the patient later experiences unexplained hypoglycemia and hyperglycemia she may benefit from the use of continuous glucose monitoring (CGM) or more frequent SMBG.[13]

Physical Activity and Insulin/Insulin Secretagogue Use
The RD should instruct Rachel on insulin or insulin secretagogues on the safety guidelines to prevent hypoglycemia (frequent blood glucose monitoring and possible adjustment in insulin dose or carbohydrate intake).[2] Research indicates that the incidence of hypoglycemia during exercise may depend on baseline glucose levels.[13]

Community Education
Teach family members, teachers, coaches, friends on how to recognize symptoms and how to treat hypoglycemia if and when it happens.[13] Trained school personnel can help to ensure that Rachel will stay safe and able to participate in all school-sponsored events.[13] Rachel should wear a diabetes identification tag or bracelet at all times

especially at school or in public.[10]

Diabetes Diary

For Rachel's follow up appointments having a more detailed picture of her daily routine is essential. At a more advanced level, carbohydrate counting focuses on adjustment of food, insulin, and activity based on patterns from detailed logs.[10] The patient can keep a logbook, journal, or use a diabetes tracking mobile app to record the following information;[2,10,15]

- Time of waking
- Usual meal and eating times
- Amounts of CHO consumed per meal
- Amount and types of food consumed per meal (Measuring or weighing foods is helpful in the beginning to learn what common food portions look like)
- Type, amount, and timing of exercise
- Type, dosage, and timing of diabetes medication (insulin)
- Self-Monitoring blood glucose (SMBG) data
- Work schedule or school hours
- Usual sleep habits
- Any hypoglycemia/hyperglycemia symptoms experienced

18. *What is an insulin:CHO ratio (ICR)? Rachel's physician ordered her ICr to start at 1:15. If her usual breakfast is 2 pop-Tarts and 8 oz skim milk, how much Apidra should she take to cover the carbohydrate in this meal?*

For normal-weight persons with T1DM, the required insulin dosage is about 0.5 to 1 unit/ kg of body weight per day.[2] Two methods are used to calculate insulin-to-carbohydrate ratios which are the 450 to 500 rule and the weight method.[10] These methods do not take into account individual variation and, therefore, are not as accurate as using detailed records.[10] For example, rapid-acting insulin-to-carbohydrate ratio = 500 divided by TDD of insulin.[10] About 50% of the total daily insulin dose is used to provide for basal or background insulin needs.[2] Therefore, if Rachel's total daily insulin dose is 33 units, her long-acting insulin Glargine will equal 16.5 units.

In contrast, insulin to CHO Ratio (ICR) equals the number of grams of carbohydrate that 1 unit of rapid-acting insulin will cover.[2] This ratio may vary person to person and may vary from meal to meal.[2] For Rachel, the remainder rapid-acting insulin (Apidra) will be divided proportionally among the CHO content of her meals so that 1 unit of insulin will cover 15 g of carbohydrates, known as the insulin:carbohydrate ratio (ICR). If her usual breakfast consists of 2 Pop-Tarts (75 g CHO) and 8 oz skim milk (12 g CHO) = 87 g CHO total, she will need 87 g CHO/15 g CHO = 5.8 ≈ 6 units of Apidra to cover this meal. Rachel and her family will learn how to effectively adjust insulin doses to match her carbohydrate intake (ICR).[13] This can be accomplished by comprehensive nutrition education and counseling on interpretation of blood glucose patterns, nutrition-related medication management and collaboration with the healthcare team.[13] Adjusting insulin dose based on planned carbohydrate intake improves glycemic control and quality of life without any adverse effects.[13]

19. Dr. Cho set Rachel's fasting blood glucose goal at 90–180 mg/dL. If her total daily insulin dose is 33 u and her fasting a.m. blood glucose is 240 mg/dL, what would her correction dose be?

As a result of higher levels of counterregulatory hormones in the morning, most individuals may require larger doses of mealtime insulin for carbohydrates consumed at breakfast than meals later in the day.[2] A commonly used formula which determines the insulin sensitivity, or correction factor (CF), defines how many milligrams per deciliter a unit of rapid- or short-acting insulin will lower blood glucose levels over a 2- to 4-hr period.[2] The CF is determined by using the "1700 rule," where 1700 is divided by the total daily dose (TDD) of insulin that the individual typically takes.[2] For Rachel, the TDD is 33 u, therefore CF = 1700/33 = 51.5. Additionally, 1 unit of rapid-acting Apidra insulin should decrease Rachel's blood glucose level by 51.5 mg/dl. Therefore, Rachel's correction dose should consist of an additional 1 to 2 units of rapid-acting insulin to lower her blood glucose to a fasting blood glucose goal of 90–180 mg/dl.

 240 mg/dl fasting a.m. blood glucose MINUS fasting blood glucose goal of 180 mg/dl = 60 mg/dl ÷ CF of 51.5 = approximately 1 unit of Aprida to lower her BG level to 180 mg/dl.

240 mg/dl fasting a.m. blood glucose MINUS fasting blood glucose goal of 100 mg/dl = 140 mg/dl ÷ CF of 51.5 = approximately 2.7 units of Apidra to lower her BG level to 100 mg/dl.

Nutrition Monitoring and Evaluation

20. When Rachel comes back to the clinic, she brings the following food and blood glucose record with her.
    a. Determine the amount of carbohydrates she is consuming at each meal.
    b. Determine whether she is taking adequate amounts of Apidra for each meal according to her record.

Time	*Diet*	*g of CHO*	*Exercise*	*BG (mg/dL)*	*Insulin Doses*	
					What patient took	*What we would recommend*
7:30 a.m.	2 Pop-Tarts 1 banana 16 oz skim milk with Ovaltine (2 Tbsp)	137g		(Pre) 150	5 u Apidra	9 u Aprida (137g CHO/15 = 9.1 units total)
10:30 a.m.				None given - she should have a BG here		
12 noon	2 slices pepperoni pizza 2 chocolate chip cookies Water	110g		(Pre) 180	6 u Apidra	7 u Apidra (110 g CHO/15 ≈ 7 units total)
2 p.m.	Granola bar	18g	PE – 30 min			None given because of exercise
4:30 p.m.	Apple 6 Saltines with 2 Tbsp Peanut butter	47g		(Pre) 110		None given because her BG may be dropping and soccer practice coming up

5-6:30 p.m.	*16 oz Gatorade*	*31g*	*Soccer – 1.5 hours*	*(Pre) 140*		*None given because of soccer practice*
6:30 p.m.	*Chicken with broccoli stir-fry (1 c fried rice, 2 oz chicken, ½ c broccoli)* *1 Egg roll* *2 C skim milk*	*99g*		*(Pre) 80*	*5 u Apidra*	*5.5 - 6 u Apidra* *Typically this meal would require 99 g CHO/15=6.5 u but we recommend 5.5 to 6 units total because her preprandial BG is considerably low as per her BG goals*
8:30 p.m.	*2 C ice cream* *2 Tbsp peanuts*	*70g*		*(Pre) 150*	*4 u Apidra*	*4 u Apidra* *Typically this meal would require 70 g CHO/15=4.5 u but we round down to 4 units total nearing bedtime to avoid low BG while sleeping*
10:30 p.m.	*Bed*					
Total		*512g*			*20 u Apidra*	*26 u Apidra*

According to Rachel's food record, her blood glucose levels are within the set goal of 90-180 mg/dL prior to each meal. The patient has also managed to successfully avoid hypoglycemia throughout the day despite having two hours of vigorous exercise, which increases the risk of hypoglycemia in T1DM. She consumes a carbohydrate snack before each exercise event, thus keeping in line with exercise recommendations. On another note, the patient seems to incorrectly estimate her carbohydrate intake during meals especially with breakfast, lunch, and dinner, excluding the bedtime snack. Therefore, she is taking less insulin than the appropriate amount. This may indicate a knowledge deficit on carbohydrate counting techniques. The RD should reinforce her education on diabetes self-management and teach her how to accurately and precisely count carbohydrate exchanges corresponding to correct insulin doses. She is also consuming 512g CHO which is higher than her recommended intake (265-350 g

CHO/day), thus slightly lowering her CHO intake may be a better and safer option.

Rachel, being newly diagnosed with type 1 diabetes, has been prescribed a controlled carbohydrate diet using appropriate insulin regimens. Rachel's goals include maintaining near-normal BG levels, achieving appropriate blood lipid and pressure levels, achieving a normal BMI, maintaining normal growth and development as well as managing risk factors and improving overall health through food choices. Day-to-day, Rachel will need to maintain consistency in her CHO intake through carbohydrate counting, appropriately adjust insulin amounts, and appropriately time insulin with meals and exercise. Initially presenting with unintended weight-loss related to undiagnosed type 1 diabetes mellitus as evidenced by approximately 9% weight loss (8 lbs) in 1-2 weeks, it is important that Rachel reaches and maintains an appropriate weight. Therefore, it is acutely important that Rachel and her parents have continued MNT intervention to ensure proper nutrition, growth, and development all while preventing hyperglycemic and hypoglycemic episodes. It is also crucial to distinguish between types of fat in the diet, lowering saturated fat and avoiding hydrogenated fats, while consistently including monounsaturated and omega-3 fatty acids in the diet.[15] In addition, Rachel needs to ensure that her diet is adequate in fiber content and essential vitamins and minerals.[15]

Diabetes self-management education and support are integral components of care for all patients with diabetes which improves diabetes-related self-care behaviors and health outcomes, such as hemoglobin A1C.[10] After the initial management phase, the diabetes team should continue to provide care, teaching, and support to Rachel and family. Sessions with individual team members (endocrinologist, nurse educator, dietitian, and a mental health professional) allow more in-depth education and care directed toward the goal of maintaining excellent glucose control.[10,14] The concepts that are required for glycemic control should be taught and reinforced.[2] These include the interaction of insulin, diet, and exercise on blood glucose concentrations. A management regimen specific for each patient is designed to achieve the best possible glucose control. In addition, the clinician should explain that strict glycemic control helps to prevent long-term consequences of diabetes; this discussion should be repeated and reinforced as often as necessary, particularly if glycemic control is suboptimal.[10,14]

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ADIME		
PATIENT SUMMARY/CLIENT HISTORY 12 y.o. 7th grader Previously healthy Admitted to ED after fainting at soccer practice Recent strep throat **Dx:** acute-onset hyperglycemia and type 1 diabetes mellitus **Family History**: Father – HTN Mother – hyperthyroidism Sister – celiac disease	ADMITTING HISTORY/PHYSICAL Report of unexplained weight loss of about 8 lbs Serum glucose high – 724 mg/dL Patient reports increased hunger (polyphagia), increased thirst (polydipsia), and increased urination (polyuria) Throat: Dry mucous membranes Skin: Warm and dry	ANTHROPOMETRIC MEASUREMENTS *Height: 5' (152.4 cm)* *Usual weight: 90 lbs (41 kg)* *Current weight: 82 lbs (37.2 kg)* *Current BMI: 15.93 kg/m2* *% Weight Change: 8.89%* *Desirable weight: 93-100 lbs (42-45.4 kg)* *Desirable BMI: 18-19.55 kg/m2*

| | Skin Color: pale
Stool: soft, light brown
Urine appearance: cloudy, amber | |

FOOD/NUTRITION RELATED HISTORY

Recent report of increased appetite and feeling very hungry (polyphagia)

Usual dietary intake:
≈1500-2650 kcal/day
≈175-300 gCHO/day

Parents report Rachel is kind of a picky eater. She eats only chicken and fish—eats salad, broccoli, carrots, tomatoes, and asparagus as her only vegetables.
Breakfast—cereal w/ milk or Pop-Tart® w/ milk
Lunch for school—peanut butter and jelly or turkey and cheese sandwich, chips, carrots
Before soccer practice—cereal or granola bar
Dinner at mom's—salad, meat, and pasta, potato, or rice
Dinner at dad's—pizza or Chinese food. Snacks include cereal, ice cream, yogurt, some fruits (apples, bananas), popcorn, chips, or cookies.

Assessment
Weight loss (9%<2 weeks)
Reason: uncontrolled and undiagnosed diabetes

Energy Requirements

TEE 2100-2600kcal/day

PROTEIN 35-36 g protein/day

CHO 260-350 g CHO/day

BIOCHEMICAL DATA, MEDICAL TESTS & PROCEDURES

Chemistry
Sodium 126 (low)
Glucose 683 (very high)
Phosphate 1.9 (low)
Osmolality 295.3 (slightly high)
HbA1C 14.6% (high)
C-peptide 0.10 (very low)
Presence (+) autoantibodies ICA, IAA, GADA, IA-2A

Urinalysis
Specific gravity 1.036 (slightly high)
pH 4.9 (slightly low)
Protein+4 (proteinuria)
Glucose +3 (glycosuria)
Ketones +1 (ketonuria)

DIAGNOSIS (PES)

1. *Food-and-nutrition-related knowledge deficit* related to lack of exposure to diabetes education/management as evidenced by patient's new diagnosis of type 1 diabetes mellitus and inconsistent carbohydrate intake in patient's usual dietary intake.

2. *Unintended weight loss* related to undiagnosed type 1 diabetes mellitus as evidenced by approximately 9% weight loss (8 lbs) in 1-2 weeks; serum glucose of 724 mg/dL (70–99 mg/dl); HBAIC 14.6% (3.9–5.2%); presence of autoantibodies ICA, IAA, GADA, IA-2A; symptoms of polydipsia, polyuria, and polyphagia; diagnosis of acute-onset hyperglycemia.

INTERVENTION

Discharge Insulin Dosage
7 u glargine
Apidra prior to each meal or snack
1:15 insulin:carbohydrate ratio (ICR).

Nutrition Prescription
Carbohydrate controlled diet using appropriate insulin doses.
Rough Guideline using 330 g CHO/day:
90 g CHO breakfast ÷ 15 g CHO per serving = 6 servings of CHO
90 g CHO lunch ÷ 15 g CHO per serving = 6 servings of CHO
90 g CHO dinner ÷ 15 g CHO per serving = 6 servings of CHO
30 g CHO snacks (×2) ÷ 15 g CHO per serving = 2 servings of CHO/per snack (before PE class + before soccer practice)

Goals after discharge
Fasting blood glucose goal set at 90-180 mg/dL

	A1C ≤8% (level ~1% higher than the adult standard, is recommended for Children 6–12 years old)[13] Long-term followup encounters with RD to ensure sustained improvements in A1C at 12 months and longer.[13]
MONITORING AND EVALUATION	
During hospitalization Change IVF to D5.45NS with 40MEq K @ 135 mL/hr. Begin Apidra 0.5 u every 2 hours until glucose is 150–200 mg/dL. Begin glargine 6 u at 9 pm. Progress Apidra using ICR 1:15. Continue bedside glucose checks hourly. Notify MD if blood glucose >200 or <80.	**Follow Up** **Monitor:** Metabolic control (glycemia, lipids, and blood pressure) Adherence Education/knowledge on self-management Evaluate food intake Medication management Anthropometric measurements (weight, BMI) **Assess Diabetes Diary Info** Time of waking Usual meal and eating times and types of foods consumed Work schedule or school hours Type, amount, and timing of exercise Usual sleep habits Type, dosage and timing of diabetes medication Self-Monitoring blood glucose (SMBG) data