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Case Study Presentation

**Pediatric Type I DM Case Study**

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?**

Diabetes mellitus of all forms shares one universal characteristic: defects in insulin production, insulin action, or both, which results in hyperglycemia (482). More specifically, pediatric type 1 diabetes mellitus is characterized as an autoimmune disease in which the immune system destroys pancreatic β cells. The function of these cells is to produce insulin, which essentially determines the metabolic fate of carbohydrate, protein, and lipid. High blood glucose levels catalyze insulin secretion. The β cell destruction results in insulin deficiency, therefore leaving some circulating blood glucose not metabolized, ultimately leading to hyperglycemia (482). This also leads to systemic acid/ base imbalance, insufficient glucose delivery to the brain and retina, inadequate blood supply to the tissues, widespread vascular degeneration, and neuropathy (Strayer, 2013). The rate of β cell destruction varies, and trends suggest that this destruction rate is generally fast in infants and children and slow in adults. The first sign of T1DM tends to be ketoacidosis, which is defined as acid-base imbalanced caused by an increased concentration of ketones in the blood. This is evidenced by biochemical lab values. Autoantibodies, which are the cause of the destruction of the pancreatic β cells, are present in 85-89% of T1DM diagnoses (483). The remainder of cases are classified as idiopathic, in which they cannot produce insulin and are prone to ketoacidosis, yet their lab values display no evidence of antibodies. It is not entirely unusual that no one else in Rachel’s family has diabetes. The cause of β cell destruction lacks sufficient research, yet recognized contributing factors to this disease are multiple genetic predispositions and unidentified environmental stimuli. Genetic predispositions include the presence of a specific protein that may be associated with antibody formation, existence of T1DM in first-degree relatives, and specific human leukocyte antigen types. There are many other contributing risk factors for developing T1DM other than first-degree relatives having the disease, therefore Rachel’s case is not usual. Two significant findings in Rachel’s family history that should be noted is that her mother is diagnosed with hyperthyroidism and her sister is diagnosed with celiac disease, both of which are autoimmune diseases. If an autoimmune disease is present in a family, their first-degree family members may be at risk for developing the same or a different autoimmune disease

Strayer, D., & Schub, T. (2013). Diabetes Mellitus, Type 1.

(2004). Autoimmune Disorders. *Emory University School of Medicine.*

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

Standard diagnostic criteria for diabetes mellitus are symptoms of diabetes such as polyuria, polydipsia, and unexplained weight loss in addition to a casual plasma glucose concentration of greater than or equal to 200 mg/dL, a fasting plasma glucose concentration of greater than or equal to 126 mg/dL, or a two hour post-prandial glucose level of greater than or equal to 200 mg/dL during an oral glucose tolerance test (485). Rachel’s history reported that she did experience symptoms such as unexplained weight loss, polyuria, and polydipsia. Her serum glucose was 724 mg/dL during her ER assessment, which is well above 200 mg/dL. Her casual glucose concentration as well as her symptoms reveals that she meets the criteria for diagnosis of diabetes mellitus.

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

The criteria used in testing for type 2 diabetes mellitus in children is a BMI of greater than the 85th percentile for age and gender, weight for height greater than the 85th percentile, or weight greater than 120% of ideal for height plus any two of the following risk factors: family history of T2DM in first or second degree relatives, race/ethnicity (Native American, African American, Latino, Asian American, or Pacific Islander), or signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, or polycystic ovarian syndrome)(499) . Since Rachel meets the criteria for diabetes mellitus, yet meets none of these criteria for T2DM in children, the physician would deduce that she has T1DM. Rachel’s lab results also show the presence of islet cell cytoplasmic antibodies (ICA antibodies), glutamic acid decarboxylase autoantibodies (GADA antibodies), and insulin antibodies (IAA), which are all indicative of T1DM as opposed to T2DM.

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

As a form of compensation for high blood glucose levels, excess glucose is lost in urine. This is due to the fact that the kidneys can only filter a finite amount of glucose from the blood. This results in glycosuria and polyuria. In turn, this loss of fluid from frequent urination stimulates the thirst mechanism, resulting in the symptom polydipsia. T1DM results in cell starvation because glucose cannot enter cells because of the lack of insulin. The body responds to these “starving” cells by promoting hunger, or polyphagia. Production of certain hormones increases to promote lipolysis for energy, since glucose is no longer readily available for energy. The fatty acids resulting from the breakdown of stored fat are transformed into keto acids in the liver. This breakdown leads to a decrease in pH to an acidic classification and the secretion of ketone bodies in the urine. Bicarbonate concentration is consequently reduced, therefore metabolic acidosis develops and ketoacidosis results. The body attempts to counteract metabolic acidosis with deep, labored breathing, yet this leads to fatigue. Individuals experiencing ketoacidosis experience weight loss due to decreased blood volume (hypovolemia) and muscle catabolism.

1. **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?**

 Production of certain hormones increases to promote lipolysis for energy, since glucose is no longer readily available for energy. The fatty acids resulting from the breakdown of stored fat are transformed into keto acids in the liver. This breakdown leads to a decrease in pH to an acidic classification and the secretion of ketone bodies in the urine. Bicarbonate concentration is consequently reduced, therefore metabolic acidosis develops and ketoacidosis results. The body attempts to counteract metabolic acidosis with deep, labored breathing, yet this leads to fatigue. Individuals experiencing ketoacidosis experience weight loss due to decreased blood volume (hypovolemia) and muscle catabolism. Rachel was experiencing rapid respirations upon admittance. A mental status change might also occur. Upon admittance Rachel was described as “alert but slightly confused”, which is most likely a result of ketoacidosis. Precipitating factors for ketoacidosis include lack of blood glucose self-monitoring, severe illness or infection, insulin omitted, increased insulin needs with growth spurts, and inappropriately stored insulin.

6. **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: carbohydrates ratio. Rachel’s parents want to know why she cannot take oral medications for her diabetes like some of her friends do. What would you tell them?**

Glargine has an onset on 2-4 hours. This specific type of insulin is peakless. The duration is 20-24 hours. Apidra is also known as insulin glulisine. The onset of action is 5-15 mins. The peak of action is 30-90 minutes. The duration of action is 3-5 hours (488). I would tell Rachel’s parents that only people with type 2 diabetes can take pills to manage their insulin, therefore her only option is insulin injections. Her case is severe and insulin injections are more dependable because it goes straight into the bloodstream.

(2013). Can Diabetes Pills Help Me. Retrieved 7 November 2013 from <http://www.diabetes.org/living-with-diabetes/treatment-and-care/medication/oral-medications/can-diabetes-pills-help-me.html>

7. **Rachel’s physician explains to Rachel and her parents that Rachel’s insulin dose may be due to something called a honeymoon phase. Explain what this is and how it might affect her insulin requirements.**

The honeymoon phase is characterized by relative self-sufficient insulin production after the T1DM is clinically diagnosed and insulin treatment is initiated. This occurs because the remaining beta cells that have not been affected by the autoimmune response produce insulin in variable amounts and react in an unpredictable manner to the newly prescribed insulin. This is only a temporary state. This self-sufficient insulin production generally translates to a lower insulin prescription, however this prescribed dosage is only temporary and will most likely be increased.

Coppieters, K. T., Van Belle T.L., & Von Herrath M. G. (2011). Type 1 Diabetes: Etiology, Immunology, and Therapeutic Strategies. *American Physiological Society.* 91: 79-118

8. **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?**

The benefits of physical activity for individuals with diabetes include the following:improved glycemic control; improved blood lipids and blood pressure, with subsequent low cardiovascular risks and overall mortality; positive impact of metabolic abnormalities, prevention or delay of onset T2DM for individuals at high risk for developing diabetes or with pre-diabetes mellitus; reduced risk of development of cardiovascular disease, since physical inactivity and diabetes are independent risk factors for it; improved coping and stress management and reduced feelings of depression; improved physical fitness and functional capacity; and enhanced quality of life (495). This being said, Rachel should definitely monitor her blood glucose levels when she does exercise because hypoglycemia and hyperglycemia are acute risks of exercise. There is a possibility of hypoglycemia during exercise that lasts longer than 60 minutes and for up to 24 hours after strenuous, prolonged, and/or sporadic exercise (495). Hyperglycemia is also a possibility in individuals that are underinsulinized. Exercise catalyzes an increase in counter-regulatory hormones that cause an increase in hepatic glucose production as well as free fatty acids. Cellular uptake of glucose is minimal due to the lack of adequate insulin, which translates to hyperglycemia. I would recommend that Rachel monitor her glucose levels before and after exercise. I would advise her to follow the general rule of thumb in which she should consume an additional 15 g of carbohydrates for every hour of moderate physical activity and 30 g for strenuous physical activity. Since she mostly likely engages in physical activity at least every other day, she will probably not have to decrease her insulin levels before exercising because her body would have already adjusted and her insulin dosage levels will be lower in general. She should not exercise if her blood sugar is over 250 mg/dL prior to exercise. She should always carry a carbohydrate snack just in case.

**9. 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?**

The dawn phenomenon is a brief increase in insulin requirements resulting from the lack of insulin injections while the diabetic individual is sleeping. Specifically with Type 1 diabetes, if the requirements after 4 hours is not met, hyperglycemia develops. Rachel is experiencing this because her blood glucose levels have been consistently high in the morning. This could be controlled by using a conventional therapy insulin regimen. This particular regimen used to control the dawn phenomenon is composed of a combination of short and intermediate-acting insulin injections before breakfast, short-acting insulin before evening meals, and intermediate-acting insulin at bedtime (489). Methods of prevention is to eat dinner earlier at night, participate in physical activity after dinner, avoid carbohydrates at bedtime, adjust dosage of medication or insulin, or switch to a different medication.

Bolli, G.B., Brunetti, P., De Feo, P., Fanelli, C., Perriello, G., Santeusanio, F., Torlone, E. (1991). The Dawn Phenomenon in Type 1 (insulin-dependent) diabetes mellitus: magnitude, frequency, variability, and dependency on glucose counterregulation and insulin sensitivity. *Diabetologia.* 34: 21-28

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

The ADA deems monitoring carbohydrates a significant tactic in realizing glycemic control. The basis of this rationale is based on research demonstrating that consistent intake of a wide variety of carbohydrates results in similar postprandial (after meal) glucose responses (493). Rachel should be cognoscente of the amount of carbohydrates that she is taking in to determine the appropriate insulin dosage.

11. **Outline the basic principles for Rachel’s nutrition therapy to assist in control of her T1DM.**

There are four specific goals for nutrition therapy for anyone with T1DM. The first goal is to attain and maintain optimal metabolic outcomes. These are evidenced in the lab values including keeping glucose levels in normal range to prevent or reduce risk of complications, lipid or lipoprotein profile that reduces risk of macrovascular disease, and blood pressure levels that reduce risk for vascular disease (489). A second nutritional goal is to prevent and treat risk for chronic complications associated with T1DM such as obesity, dyslipidemia, cardiovascular disease, hypertension, and nephropathy through diet and lifestyle modification. A third goal is to improve health through involvement in physical activity and appropriate dietary choices for their disease state. A fourth goal is to address individual nutritional needs with consideration of lifestyle and preferences (491). Appropriate macronutrient distribution for this disease state can be achieved by methods such as carbohydrate counting or exchange lists.

12**. Assess Rachel’s ht/age; wt/age; ht/ wt; and BMI. What is her desirable weight?**

Based on CDC growth charts, her stature-for-age percentile is in the 50th. Her weight-for-age percentile is just about the 25th. Her BMI was calculated with the following equation:

BMI= kg/M^2. 60”\*2.54=152.4 cm or 1.524 M. 82/ 2.2= 37.3 kg.

37.3 kg/ (1.524)^2=16

∴Her BMI is 16 kg/M^2. Based on BMI percentiles she is just under the 25th percentile. Acute malnutrition occurs below the 5th percentile, therefore this is not evident in this case study because she is above the 5th.

Her desirable weight for height can be found by finding the BMI at the 50th percentile on the growth charts for 12 year olds. This BMI is 17.4

17.4=kg/(1.524)^2

kg= 40.4 kg, therefore her desirable weight is 40.4 kg. Note that this is for the 50th percentile, however, and children with a BMI over the 5th percentile and under the 85th percentile are still within normal range.

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

 Abnormal lab values:

|  |  |  |  |
| --- | --- | --- | --- |
| **Laboratory Results** | **Ref. Range**  | **5/4 1780** | **5/5 1522** |
| Sodium (mEq/L) | 136-145 | 126 | 131 |
| Glucose (mg/dL) | 70-110 | 683 | 250 |
| Phosphate (mg/dL) | 2.3-4.7 | 1.9 | 2.1 |
| Osmolality (mmol/kg/H20) | 285-295 | 295.3 | 304 |
| HbA1C (%) | 3.9-5.2 | 14.6 |  |
| C-peptide (ng/mL) | 0.51-2.72 | 0.10 |  |

She had low sodium laboratory values. Upon hospital admittance, her values were 126 mEq/L and the normal range is 136-145 mEq/L. This is indication of an electrolyte imbalance, which is most likely attributed to sodium excretion as a result of her polyuria. She has high blood glucose levels. Upon hospital admittance, she had blood glucose levels of 683 mg/dL, which are well above the normal range of 70-110 mg/dL. This hyperglycemic state is a result of reduced insulin production as a result of autoimmune destruction of pancreatic β cells as discussed in the etiology. She also has low inorganic phosphate levels. Upon admittance they were 1.9 mg/dL, which is lower than the range of 2.3-4.7 mg/dL. This is due to the increased secretion of phosphate through frequent urination. Her laboratory values report high osmolality. This was 295.3 mmol/kg/H20, which is below the normal range of 285-295 mmol/kg/H20. This could be a result of her state of dehydration. Her HbA1c % levels were low, which is indicative of consistently high blood glucose levels. This was 14% as opposed to the normal range of 3.9-5.2%. Her C-peptide levels are low, which means she most likely has low insulin levels in her body. This is because insulin is secreted as two polypeptide chains joined by a disulfide bond. C-peptide is released upon separation of the chains, therefore low C-peptide levels indicate low insulin secretion. Her blood work indicates the presence of glutamic acid decarboxylase autoantibodies, insulin autoantibodies, and islet cell cytoplasmic autoantibodies, all indicative of the autoimmune response associated with T1DM. The urinalysis indicates that Rachel has abnormally high specific gravity of urine. It is 1,035, which is above the normal range of 1,003-1,030. This means that abnormally high amounts of glucose and electrolytes are excreted in her urine. Her urinalysis also reports a low pH. The pH of her urine is 4.9, which is lower than the normal range of 5-7. This could be a result of an increased production of keto acids related to diabetic ketoacidosis. There is protein, glucose, and ketones present in the urine due to hyperglycemia and ketoacidosis.

14. **Determine Rachel’s energy and protein requirements. Be sure to explain what standards you used to make this estimation.**

I used the EER for females 9 through 18 years. I will incorporate a physical activity coefficient of 1.31 because she is an active individual.

EER= 135.3- 30.8\*age+PA\*(10.0\*weight+934\*height)+25

135.3-30.8\*12+1.31\*(10.0\*38.6 kg+934\*1.524)+25= 2485 kcal

Her protein needs should come from 15-20% of her total kcal.

This can be calculated by 2485 kcal .15= 372.8 and 2485 kcal\*.2=497

Therefore the range of calories from protein is 372.8-497 kcals and the range of grams of protein is 93-124 grams.

15. **Prioritize two nutrition problems and complete PES statements for each.**

PES Statement 1:Altered nutrition-related lab values (NC 2.2) related to Type 1 Diabetes Mellitus as evidenced by a blood glucose concentration of greater than 110 mg/dL.

PES Statement 2: Unintended weight loss (3.2) related to inadequate insulin due to Type 1 Diabetes as evidenced by a reported weight loss of 8 lbs.

16. **Determine Rachel’s initial nutrition prescription using her diet record from home as a guideline, as well as your assessment of her energy requirements.**

Rachel should have 2400-2500 kcals per day. She should have about 15-20% of her kcals coming from protein, which is about 350-500 kcals from protein and 90-125 grams. 25-35% of her total calories should come from fat. This would equate to 620-870 kcals from fat or 70-90 grams of fat per day. The remainder of her calories should be coming from carbohydrates. This would be about 50-60% of her total calories. This equates to 1225- 1500 kcals or 300-370 grams of carbs. Based on her age, she should have 70-90 g of carbohydrates per meal and 30-45 g per snack. She again should take insulin injections accordingly.

17. **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. of skim milk, how much Apidra should she take to cover the carbohydrate in this meal?**

An ICR is a calculated ratio of the amount of insulin needed to limit glycemic excursions after meals based on their intake of carbohydrates.They are important of any intensive diabetes management program. The total amount of carbohydrates in her breakfast is 82 grams. Since her ICR is 1:15, she should take 6 units of insulin to cover the carbohydrate in her meal.

Daneman, D. (2006). Type 1 diabetes. *The Lancet*, *367*(9513), 847-858.

18. **Dr. Cho set Rachel’s 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?**

High blood sugar correction dose= difference between actual blood sugar and target blood sugar/ correction factor.

Correction factor= 1500/ 33= 45mg/dL

240-90/45=3 units of insulin

(2013). *Calculating Insulin Dose.* Retrieved November 17th, 2013 from University of California Diabetes Education Online website: http://dtc.ucsf.edu/types-of-diabetes/type2/treatment-of-type-2-diabetes/medications-and-therapies/type-2-insulin-rx/calculating-insulin-dose/

**19. Write an ADIME note for your initial nutrition assessment.**

|  |  |
| --- | --- |
| **A (assessment)** | **82 lbs., 5’ tall, BMI of 16. Low lab values include sodium, inorganic phosphate, c-peptide, and a low pH of urine. High lab values include blood glucose, osmolality, HbA1C, specific gravity of urine, protein in urine, glucose in urine, ketones in urine, and prot chk in urine. Lab values indicate presence of autoantibodies ICA, GADA, and IAA. Slim, healthy-appearing 12 year old female. Experiencing polyuria, polydipsia, and unintentional weight loss. She has an active lifestyle and a fairly balanced diet.** |
| **D****(Diagnosis)** | **Altered nutrition-related lab values (NC 2.2) related to Type 1 Diabetes Mellitus as evidenced by a blood glucose concentration of greater than 110 mg/dL. Unintended weight loss (NC 3.2) related to inadequate insulin due to Type 1 Diabetes as evidenced by a reported weight loss of 8 lbs.** |
| **I****(Intervention)** | **Educate patient and parents on carbohydrate counting and how she should take insulin accordingly. Educate patient on appropriate macronutrient ratios and how her physically active lifestyle is affected by her disease state. Set glycemic goals.** |
| **M/E****(Monitoring/ Evaluation)** | **Monitor/measure glucose daily at home. A1C should be tested at least twice a year. Laboratory tests should be taken in follow-up sessions to assess glycemic control. Adherence to glycemic goals should be analyzed. Meet biweekly initially to assess labs and food logs. She can have less frequent follow-up appointments once she has gotten used to her disease state and new lifestyle and blood glucose levels have normalized.**  |

**20. When Rachel comes back to the clinic, she brings the following food and blood glucose records 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.**

**c. Calculate a correction dose for her to use.**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Time** | **Diet** | **Grams of CHO** | **Exercise** | **BG****(mg/dL** | **Insulin dosage: what patient took** | **Insulin Dosage:****What you would recommend** |
| **7:30 a.m.** | **2 pop tarts****1 banana 16 oz. skim milk with Ovaltine (2 tbsp.)** | **115 g** |  | **(pre) 150** | **5 u Apidra** | **8 u Apidra** |
| **10:30 am** |  |  |  |  |  |  |
| **12 noon** | **2 slices of pepperoni pizza** **2 chocolate chip cookies****water** | **60 grams** |  | **(pre) 180** | **6 u Apidra** | **4 u Apidra** |
| **2 pm** | **Granola bar** | **23 g**  | **PE class- 30 minutes** |  |  | **1 u Apidra** |
| **4:30 pm** | **Apple** **6 saltines with 2 tbsp peanut butter** | **30 g** |  | **(Pre) 110** |  | **2 u Apidra** |
| **5-6:30** | **16 oz. Gatorade**  |  | **Soccer practice- 1.5 hours** | **(Pre) 140** |  |  |
| **6:30 pm** | **Chicken with broccoli stir-fry (1 c fried rice, 2 oz chicken, .5 c broccoli)****Egg roll-1** **2 c skim milk** | **95 g** |  | **(pre) 80** | **5 u Apidra** | **7 u Apidra** |
| **8:30 pm** | **2 c ice cream with 2 tbsp. peanuts**  | **60 g** |  | **(pre) 150** | **4 u Apidra** | **4 u Apidra** |
| **10:30 pm** | **bed** |  |  |  |  |  |