Katie Davis & Liz DeJulius

KNH 411

Case Study Presentation

Type 1 Diabetes Mellitus in the Adult

Armando Gutierrez is a 32-year-old male admitted from the ER to the endocrinology service with acute uncontrolled hyperglycemia.

I. Understanding the Diagnosis and Pathophysiology

1. What are the differences among TIDM, T2DM, and LADA?

T1DM results from a cellular-mediated autoimmune destruction of B-cells of the pancreas causing cessation of insulin production which leads to fasting hyperglycemia and ketoacidosis. The body is unable to use glucose for energy (Nelms, 482-483). This form is often caused by hereditary factors.

T2DM can result from obesity, physical inactivity, impaired glucose metabolism, and other factors. In this form of DM, individuals B-cells in the pancreas produce insulin, but their tissues are insulin resistant causing the increased need for insulin, which increased insulin production by the B-cells, which eventually leads to the pancreas losing its ability to produce insulin (Nelms 498-499)

LADA (latent autoimmune diabetes of adulthood) is also called T1.5DM, is a slowly progressive form of T1DM; individuals are often diagnosed as T2DM due to the presence of insulin because they may still have some beta-cells. Individuals with T1.5DM have positive pancreatic inslet antibodies, especially to glutamic acid decarboxylase (GADA) indicating the progressive destruction of beta cells (Nelms, 485)

2. What are standard diagnostic criteria for each of these diagnoses?

T1DM: Symptoms of diabetes (polyuria, polydipsia, and unexplained weight loss) plus casual (any time of day without regard to time since last meal) plasma glucose concentration greater than or equal to 200 mg/dL **OR** fasting glucose greater than or equal to 126 mg/dL **OR** 2-hour post-prandial glucose of greater than or equal to 200 mg/dL during an oral glucose tolerance test (Nelms, 485).

T2DM: For individuals 45 years or older, they should be tested every three years if they have a BMI or 25 or higher. For all other individuals with a BMI of 25 or over, the diagnostic criteria includes being habitually physically inactive, have first-degree relative with diabetes, are members of high-risk ethnic populations, delivered a baby weighing over nine pounds or have been diagnosed with GDM, hypertensive, have HDL cholesterol below 135 mg/dL or triglycerides greater than 250 mg/dL, have postcyctic ovarian syndrome, had IGT or IFG on previous testing, have other clinical condition associated with insulin resistance, or have history of vascular disease (Nelms, 500).

LADA: Presence of diabetes-related autoantibodies such as insulin autoantibodies (IAA), islet cell cytoplasmic autoantibodies (ICA), glutamic acid decarboxylase autoantibodies (GADA), and insulinoma-associated-2 autoantibodies (IS-2A) (Nelms, 485).

3. Why do you think he was originally diagnosed with T2DM? Why does the MD now suspect he may actually have T1DM or LADA?

He may have been originally diagnosed with T2DM because he has a family history of T2DM and no family history of T1DM. Additionally, his body may have still been producing some insulin at the time of the diagnosis, which indicates T2DM. Sometimes, residual B-cell function that is sufficient to prevent ketoacidosis may be preserved in adults diagnosed with T1DM (Nelms 483). The presence of certain autoantibodies serve as indicators for the body's destructive immune response against its own B-cells, which indicates T1DM. The fact that Armando has elevated ICA, GADA, and IAA is reason enough for the MD to suspect T1DM of the progressive type (LADA) as opposed to T2DM (Nelms, 485).

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

Armando's potential T1DM causes his body to be deficient of insulin due to the destruction of B-cells, resulting in the inability of his cells to take up glucose and use it for energy. This causes plasma glucose levels to rise (hyperglycemia) and cells are starved of energy, potentially causing Armando's fatigue and grogginess due to the fact

that the brain's main source of energy is glucose. To make up for the high levels of glucose in the blood, the excess glucose is lost in the urine resulting in frequent urination (polyuria). This loss of fluid due to frequent urination leads to polydipsia, or excessive thirst (Nelms, 483).

5. Describe the metabolic events that result in the signs and symptoms associated with DKA. Was Armando in this state when he was admitted? What precipitating factors may lead to DKA?

Diabetic ketoacidosis (DKA) is an acid-base imbalance caused by an increase in concentration of ketones in the blood. As previously described, persistent insulin deficiency causes the production of additional hormones, which leads to lipolysis. This is a state where the body breaks down the fat stored in adipose tissue and the resulting fatty acids are transformed into keto acids in the liver. This causes the pH to fall and keto bodies to be secreted in the urine. Over time, bicarbonate concentration in the blood is reduced resulting in ketoacidosis. The body can try to offset metabolic acidosis through heavy breathing, N/V, stomach pain, acetone breath, and changed mental status. Armando may have been in this state when admitted because he exhibited many of these symptoms such as viral infection –like symptoms and a groggy and almost unconscious state. His charts also indicated rapid respirations. Lack of blood glucose self-monitoring, severe illness or infection, insulin omitted, increased insulin needs with growth spurts, or inappropriately stored insulin are precipitating factors that may lead to DKA (Nelms 483,486)

6. Armando will be started on a combination of Novolog 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.

Novolog (or insulin aspart) has an onset of 5 to 15 minutes, a peak at 30 to 90 hours, a duration of 3 to 5 hours, and may be used in pump therapy. Insulin glargine has an onset of 2 to 4 hours, no peak of action, and a duration of 20 to 40 hours, and may not be mixed with other insulins (Nelms, 488).

7. Using his current weight of 165 lbs., determine the discharge dose of glargine as well as an appropriate ICR for Armando to start with.

Discharge dose = 0.6 units/kg (75 kg) = 45 units of glargine daily (Nelms, 488). Insulinto-Carbohydrate Ratio (ICR) = 500 / (45 units) = 11 g of carbohydrates per 1 unit of insulin (Nelms, 493).

8. Intensive insulin therapy requires frequent blood glucose self-monitoring. What are some of the barriers to success for patients who begin this type of therapy? Give suggestions on how you might work with Armando to support his compliance.

In William Polonsky's book *Diabetes Burnout*, he lists the top ten reasons people do not monitor their blood glucose:

- Their meter makes them feel bad about themselves.
- Monitoring seems pointless because they believe there is nothing they can really do about their blood glucose results anyways.
- Checking their blood glucose reminds them that they have diabetes, which is something they would rather not think about too much.
- Their meter seems to control their life—telling them what they can and cannot do.
- Monitoring serves as an opportunity for your friends and family to bother them.
- None of their health care providers ever do anything with the results anyways.
- Checking blood glucose hurts sometimes.
- Monitoring can be inconvenient.
- Monitoring can be expensive.
- Life is too busy and demanding to take the time for regular monitoring (*Mayo Clinic,* "Diabetes").

In order to ensure success and Armando's compliance with SMBG, it will be important to provide him with counseling that properly educates him on how to monitor his blood glucose, how to incorporate it into his schedule, why it is important and complications that may result if he does not self-monitor, what he can do to lower high readings and increase low readings with diet, exercise, and medications, and hold him accountable for his monitoring by having him record readings in a journal and collecting it and asking him questions about it at each counseling session. It is also important to make sure that the entire healthcare team collaborates to hold him accountable and encourage him in SMBG. It may also be helpful enrolling him in a support group or group counseling.

9. Armando tells you that he is very frightened of having his blood sugar drop too low. What is hypoglycemia? What are the symptoms? What information would you give to Armando to make sure he is well prepared to prevent or treat hypoglycemia?

Hypoglycemia is an abnormally low blood glucose level that occurs when glucose is utilized too rapidly, glucose release rate falls behind tissue demands, or excess insulin enters the bloodstream (Nelms 507). Symptoms may include confusion, abnormal behavior or both, such as the inability to complete routine tasks, visual disturbances, such as double vision and blurred vision, seizures, though uncommon, loss of consciousness, heart palpitations, shakiness, anxiety, sweating, hunger, and tingling sensation around the mouth. In order to prevent hypoglycemia, it is important for Armando to carefully follow the diabetes management plan that his healthcare team develops. Treatment of hypoglycemia involves immediate initial treatment to raise his blood sugar level such as consuming sugar (i.e. candy, drinking fruit juice, taking glucose tablets), consuming small and frequent meals, or injection of glucose using a kit provided by a doctor. It may also be effective to treat the underlying cause of his hypoglycemia (*Mayo Clinic*, "Hypoglycemia").

10. Armando's mother has T2DM. She is currently having problems with vision and burning in her feet. What is she most likely experiencing? Describe the pathophysiology of these complications. You can tell that he is worried not only about his mother but also about his own health. Explain, using the foundation research of the Diabetes Control and Complication Trial (DCCT) as well as any other pertinent research data, how he can prevent these complications.

Armando's mother is most likely experiencing both retinopathy and diabetic neuropathy. Retinopathy is likely caused by hyperglycemic damage to the blood vessels by the accumulation of sorbitol. Similarly, damage to the cells and normal nervous system pathways in or related to her feet is caused by the continued presence of hyperglycemia that causes the accumulation of abnormal substances such as sorbitol and glycated proteins. Armando can prevent retinopathy by maintaining glycemic control and lowering his blood pressure. He can prevent damage to his nervous system by again maintaining proper blood glucose levels, having a consistent carbohydrate intake, having adequate fiber intake, and properly self-managing his diabetes (Nelms 497-48). According to the DCCT, Armando should use intensive control by keep his hemoglobin A1C levels as close as possible to the normal value of 6 percent or less. A1c reflects a person's average blood glucose over the last 2-3 months (*National Diabetes* *Information Clearinghouse (NDIC), "DCCT* and EDIC: The Diabetes Control Complication Trial and Follow-up Study").

II. Understanding the Nutrition Therapy

11. Outline the basic principles for Armando's nutrition therapy to assist in control of his DM.

It is important to consider that the impact of dietary modifications on overall health, metabolic control, and treatment for acute and chronic complications is substantial. The main principles for Armando's therapy include:

- Attaining and maintaining optimal metabolic outcomes such as keeping glucose levels in the normal range, maintaining a lipid or lipoprotein profile that reduces the risk for macrovascular disease, and maintaining blood pressure levels that reduce risk for vascular disease.
- Treating chronic complications by modifying nutrient intake and lifestyle as appropriate for prevention and treatment of obesity, dyslipidemia, cardiovascular disease, hypertension, and nephropathy.
- Enhancing health through food choices and physical activity.
- Addressing individual nutritional needs with regard to personal and cultural preferences and lifestyles while respecting the individual's wishes and willingness to change.

(Nelms, 489-490)

III. Nutrition Assessment

12. Assess Armando's height and weight. Calculate his BMI.

Armando's BMI is 23 based on his height and weight, which is considered to be normal for his current height and weight.

13. Identify any abnormal laboratory values measured upon his administration. Explain how they may be related to his newly diagnosed DM.

	Ref. Range	Armando	
Chemistry			
Sodium (mEq/L)	136-145	130	Indicates loss of total body water due to body's response to hyperglycemia (frequent urination).
Carbon Dioxide (CO2 mEq/L)	23-30	31	Indication of an acidotic state/ketoacidosis.
Glucose (mg/dL)	70-110	683	Indicates fasting hyperglycemia and improper use of blood glucose due to insulin resistance or lack of insulin production.
Phosphate, inorganic (mg/dL)	2.3-4.7	2.1	Indicates loss of total body water due to body's response to hyperglycemia (frequent urination).
Osmolality (mmol/kg/H2O)	285-295	306	This is a result of the fluid loss Armando is experiencing.
Triglycerides (mg/dL)	140-160 M	175	Indicates insulin deficiency which causes increased hormones to break down adipose tissue and release fatty acids in the blood.

Cholesterol (mg/dL)	120-199	210	Relates to dietary intake and/or family history.	
HbAıc(%)	3.9-5.2	12.5	Measurement of blood glucose concentration from previous 2-3 months; high value indicates long- term increased blood glucose	
C-peptide (ng/mL)	0.51-2.72	0.09	Indication of the presence of insulin in the blood, which in turn indicates slowly progressing beta-cell destruction. Normally absent in T1DM.	
ICA	-	+	Autoantibody indicating attack of B- cells and thus a marker for T1DM.	
GADA	-	+	Autoantibody indicating attack of B- cells and thus a marker for T1DM (and LADA).	
IAA	-	+	Autoantibody indicating ongoing destruction of B-cells and thus a marker for developing T1DM (and LADA)	
Urinalysis				
рН	5-7	4.9	Evidence of ketoacidosis, which is the first sign of T1DM. Insulin deficiency is causing breakdown of adipose tissue, which leads to the transformation of fatty acids into ketones in the liver, which are	

			excreted in the urine causing pH to drop.	
Protein (mg/dL)	Neg	+1	Indicator of decreased fluid volume, which is the body's response to hyperglycemia (frequent urination).	
Glucose (mg/dL)	Neg	+3	To compensate for hyperglycemia, excess glucose is lost in the urine.	
Ketones	Neg	+4	Evidence of ketoacidosis (see pH above) which is also an indication of insulin deficiency. Insulin deficiency causes the release of hormones that release fatty acids in the blood that are converted to ketone bodies, which are secreted in the urine (ketoacidosis).	
Prot chk	Neg	Tr	This indicates spilling of protein into the urine and that the body is no longer digesting or absorbing protein.	
Arterial Blood Gases				
рН	7.35-7.45	7.31	Evidence of ketoacidosis (see pH above).	

14. Determine Armando's energy and protein requirements. Be sure to explain what standards you used to make this estimation. Would you recommend that he gain or lose weight in the future?

Armando's weight is in the normal range so there is no need for him to lose weight, and he needs to work on maintaining his weight.

Used the Mifflin-St. Joer to estimate his energy needs.

REE = (10 x 75kg) + (6.25 x 180cm) - (5 x 32yrs) + 5 = 1720 kcal x 1.1 (PAL) = 1890 kcal

Armando need 0.8 g of protein per kg of body weight. Therefore, he needs **60g of protein** every day.

IV. Nutrition Diagnosis

15. Prioritize two nutrition problems and complete the PES statement for each.

Self-monitoring deficit R/T noncompliance to taking medication regularly and SMBG AEB serum fasting glucose of 610 mg/dL.

High fasting glucose R/T insulin deficiency, pancreatic beta-cell destruction, and ketoacidosis AEB positive ICA, positive GADA, positive IAA, c-peptide of 0.09 ng/mL, A1c of 12.5%, ketones of +4 in urinanalysis, glucose of +3 mg/dL in urinanalysis, and urine pH of 7.31.

High fasting glucose R/T excessive carbohydrate intake, low fiber intake, and high fat intake AEB dietary recall.

V. Nutrition Intervention

16. Determine Armando's initial CHO prescription using his diet record from home as a guideline, as well as your assessment of his energy requirements. What nutrition education material would you use to teach Armando CHO counting?

Armando's should consume 55% of his daily kcal (2750 kcal) from CHO, which is 1510 kcal <u>or</u> 378g of CHO <u>or</u> 25 15 g CHO choices or servings. Armando will consume 7 CHO choices at breakfast, lunch, and dinner and 2 CHO choices at 2 snacks during the day (between meals). It would be important to education Armando on how to self-

monitor his blood glucose and how to fine-tune the amount of insulin required for each carbohydrate choice by understanding ICRs and correction factors to return glucose levels to normal. It would be helpful to provide him with an exchange list so he can more easily count his CHO servings while allowing for flexibility in the diet (Nelms 493).

17. Armando's usual breakfast consists of 2 slices of toast, butter, 2 tbsp jelly, 2 scrambled eggs and orange juice (~1 c). Using the ICR that you calculated in question #7, how much Novolog should he take to cover the carbohydrate in his meal.

ICR calculated in question 7 was 11 g of carbohydrates per 1 unit of insulin. Armando's breakfast contains approximately 60g of CHO; therefore, he should consume 5.5 units of Novolog.

18. Using the ADA guidelines, what would be appropriate fasting and postpandal target glucose levels for Armando?

Fasting/before a meal: 70-130 mg/dL

Postprandial/1-2 hours after beginning of a meal: less than 180 mg/dL

(American Diabetes Association, "Checking Your Blood Glucose")

VI. Nutrition Monitoring and Evaluation

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

Assessment	FH: Estimated usual intake of 1500 kcal per day, low fruit and vegetable intake, appears to have not taken metformin regularly		
	AD: 71 inches tall, 165 lbs, BMI 23.1		
	BD: fasting glucose 683 mg/dL, low sodium and phosphate, high CO ₂ , high cholesterol and triglycerides, A1c 12.5, presence of ICA, GADA, and IAA, negative c-peptide, low blood and urine pH, high glucose and ketones in urine, REE of 1890 kcal		

	PD: alert, oriented
	CH: smokes 1 ppd x 10 years, father had MI, mother has ovarian cancer and T2DM, Hispanic and catholic background, 16 years of education, computer software engineer
Diagnosis	Self-monitoring deficit R/T noncompliance to taking medication regularly and SMBG AEB serum fasting glucose of 610 mg/dL.
	High fasting glucose R/T insulin deficiency, pancreatic beta-cell destruction, and ketoacidosis AEB positive ICA, positive GADA, positive IAA, c-peptide of 0.09 ng/mL, A1c of 12.5%, ketones of +4 in urinalysis, glucose of +3 mg/dL in urinalysis, and urine pH of 7.31.
	High fasting glucose R/T excessive carbohydrate intake, low fiber intake, and high fat intake AEB dietary recall.
Intervention	Counseling session to quit smoking, incorporate physical activity into his lifestyle, increase fruit, vegetable, and whole grain consumption, and incorporate a self-management training regimen on carbohydrate counting/exchange system and taking his medications (Novolog and glargine) throughout the day.
Monitoring & Evaluation	Armando will record blood sugar from self-monitoring of blood glucose before each meal and 1-2 hours after each meal, dietary intake, and medications taken in a journal or on a blood sugar log sheet. All findings will be reported in 2 weeks to medical team so any adjustments can be made and he can ask questions. He will receive urine testing for glucose and ketones, blood testing for glucose and lipids, and a blood pressure test. He will continue to receive education and complete a less detailed log as he establishes a routine to self- manage his condition into his daily routine. These same tests will be done in successive appointments as well as A1c starting after 2 months of diagnosis.

FH = Food/Nutrition related history, AD = Anthropometric measurements, BD = Biochemical data, medical tests, and procedures, PD = Nutrition-focused findings, CH = Client history

20. Armando comes back to the clinic 2 weeks after his diagnosis. List the important questions you will ask him to order to plan the next steps for providing the additional education that he might need.

- How often are you monitoring your blood sugar? Have you had trouble finding time to take check your blood sugar?
- Have you been able to keep consistent blood glucose readings through carb counting and paying attention to your ICR?
- How often are you smoking?
- Is your new physical activity regimen working for you?
- How often, if ever, do you feel sick?
- Have your family/friends been supportive of your lifestyle changes?
- Have you made any changes in your diet to increase fruit, vegetable, and whole grain intake?

21. Armando states that he would like to start exercising again as he is feeling better. He is used to playing tennis several times per week as well as cycling at least 2 days per week for over 20 minutes each time. Again, he expresses his concern regarding low blood sugar. How would you counsel Armando regarding physical activity, his diet, and his blood glucose monitoring.

I would tell him that he is right in being concerned about low blood sugar because hypoglycemia is an acute risk of exercise lasting longer than one hour. This condition can last for up to 24 hours after unusually strenuous, prolonged, or sporadic exercise. Blood glucose levels should be monitored both before and after exercise to understand how his diabetes is affecting glycemic control and to determine appropriate adjustments. I would assure him that this is rarely necessary for moderate exercise lasting less than 30 minutes and to follow the rule of thumb that an additional 15g carbohydrate snack would be necessary and adequate for one hour of moderate physical activity and additional 30g carbohydrate snack would be necessary and adequate for one hour of strenuous exercise. He may not need to make adjustments in his insulin intake, but if he begins exercising more often or takes up more strenuous activities, he may need counseling on how much to decrease his intake (Nelms 495).

22. Armando states that one of his friends has talked about using the glycemic index as a way to manage his diabetes. He says that he has also seen some nutrition programs advertise their food products as being "low glycemic index" on TV. Explain glycemic index, glycemic load, and how he might use this information within his nutrition therapy plans.

The glycemic index is used to rank carbohydrate sources by their ability to raise blood glucose levels as compared to some reference food such as white bread and glucose. The glycemic load of a food is the glycemic index multiplied by the number of available carbohydrates in the food (i.e. non-fiber sources of carbohydrates) in grams. It could be important for Armando to consume a moderate glycemic index diet, but it still remains unknown whether further lowering of dietary glycemic index is beneficial. However, foods with a low glycemic index have been shown to have beneficial effects of blood glucose control in diabetic patients (Mahan 39, 685).

References

Checking Your Blood Glucose. American Diabetes Association. (2013). Retrieved from

http://www.diabetes.org/living-with-diabetes/treatment-and-care/blood-glucose-control/checking-your-blood-glucose.html

DCCT and EDIC: The Diabetes Control Complication Trial and Follow-up Study. *National Diabetes Information Clearinghouse (NDIC).* (2013). Retrieved from

http://diabetes.niddk.nih.gov/dm/pubs/control/

Diabetes. *Mayo Clinic*. (2013). Retrieved from http://www.mayoclinic.com/health/blood-glucose/MY00558

Hypoglycemia. *Mayo Clinic.* (2013). Retrieved from http://www.mayoclinic.com/health/hypoglycemia/DS00198/DSECTION=treatme nts-and-drugs

- Mahan, L.K., Escott-Stump, S., and Raymond, J.L. (2012). *Krause's Food & The Nutrition Care Process, Thirteenth Edition.* St. Louis, MO: Elsevier Saunders.
- Nelms, M., Sucher, K. P., Lacey, K., and Roth, S. L. (2011). *Nutrition Therapy & Pathophysiology, Second Edition*. Belmont, CA: Brooks/Cole Cengage Learning.