

Last Name _____ Ma _____ First Name _____ Yimeng _____

Diabetes Mellitus

DUE Wednesday 12/17/14 (by 4:00 pm in Meyer 3241)

Pt. Summary: Mr. B is a 48-yo Native American man admitted from the ER to the endocrinology service.

Hx:

Onset of disease: pt transported to ER when found ill in his house by his wife. During ER assessment, pt was noted to have a S. glucose of 610 mg/dL. Mr. B was diagnosed with T2DM one year ago and has been on metformin since that dx. He does not take the medication regularly as he felt it really wasn't necessary.

Medical hx: product of nl pregnancy and delivery; NKA

Surgical hx: none

Tobacco use: Smoked 1 ppd x 10 years (no longer smokes)

Alcohol use: occasional

FH: Father – MI; mother – ovarian CA, T2DM

Demographics:

Social hx: married, 3 children, works driving trucks

Years education: 12

Language: English & O'odham

Ethnicity: Pima Native American

Religious affiliation: Catholic

Admitting Hx/PE:

Chief complaint: Wife states that Mr. B had not been feeling well the previous day. He thought he was fighting off a virus. When he didn't answer the phone this morning, his wife went to check on him and found him groggy and almost unconscious at home. She called 911 and the pt was transported to University Hospital.

General appearance: Slim male, in obvious distress.

PE:

General:	WDWN 48 yo male; 5'10" 160#
Vitals:	T 99.6°F; P 100; RR 24; BP 100/78 mm Hg
Chest/Lungs:	Respirations are rapid – clear to percussion and auscultation
Heart:	Tachycardia
HEENT:	Head: WNL, Eyes: PERRLA, Ears: clear, Nose: clear, Throat: dry mucous membranes w/o exudates or lesions
Abdomen:	Active bowel sounds x4; tender, non-distended
Extremities:	+4 ROM; DTR 2+
Neurologic:	Lethargic but able to arouse. Follows commands appropriately. Glasgow Coma Scale: 13.
Skin:	Smooth, warm, dry, no edema
Peripheral Vascular:	Pulse 4+ bilaterally, warm, no edema
Genitalia:	Deferred

Admission Orders:

1. Regular insulin 1 unit/mL in NS 40mEq KCl/liter @ 300 mL/hr. Begin infusion at 0.1 unit/kg/hr = 3.7 units/hr and increase to 5 units/hr. Flush new IV tubing with 50mL of insulin drip solution prior to connecting to pt and starting insulin infusion.
2. Labs: stat
3. NPO except for ice chips and medications. After 12 hours, clear liquids when stable. Then advance to consistent CHO diet order: 70-80g breakfast and lunch; 85-95 g dinner; 30 g snack pm and HS.
4. Consult diabetes education team for self-management training to begin education after stabilized.

Nursing Assessment:

	12/9/14
Abdominal appearance (concave, flat, rounded, obese, distended)	Flat
Palpation of abdomen (soft, rigid, firm, masses, tense)	Tense w/ guarding
Bowel function (continent, incontinent, flatulence, no stool)	continent
Bowel sounds (P=present, AB=absent, hypo, hyper)	
RUQ	P
LUQ	P

RLQ	P
LLQ	P
Stool color	Light brown
Stool consistency	Soft
Tubes/ostomies	NA
Genitourinary	
Urinary continence	Catheter in place
Urine source	Clean specimen
Appearance (clear, cloudy, yellow, amber, fluorescent, hematuria, orange, blue, tea)	Cloudy, amber
Integumentary	
Skin color	Pale
Skin temperature (DI=diaphoretic, W=warm, dry, DL=cool, CLM=clammy, CD+=cold, M=moist, H=hot)	DI; CLM
Skin turgor (good, fair, poor, TENT=tenting)	Fair
Skin condition (intact, EC=ecchymosis, A=abrasions, P=petechiae, R=rash, W=weeping, S=sloughing, D=dryness, EX=excoriated, T=tears, SE=subcutaneous emphysema, B=blisters, V=vesicles, N=necrosis)	Intact
Mucous membranes (intact, EC=ecchymosis, A=abrasions, P=petechiae, R=rash, W=weeping, S=sloughing, D=dryness, EX=excoriated, T=tears, SE=subcutaneous emphysema, B=blisters, V=vesicles, N=necrosis)	Intact
Other components of Braden Scale: special bed, sensory pressure, moisture, activity, friction/shear (>18=no risk, 15-16=low risk, 13-14=moderate risk, <12=high risk)	20

Nutrition:

Meal type: NPO then progress to clear liquids and then consistent CHO-controlled diet

Fluid requirement: 2200mL

Hx: Does not follow traditional tribal eating pattern, with a few exceptions; likes fry bread and prepares wojapi seasonally. Pt does not tolerate milk and only eats cheese when obtained through government commodities. Fresh vegetables are grown at home: squash, peppers, beans, corn, and some greens. There is very little fruit in his diet and meat is only eaten at dinner.

Usual intake (for past several months):

AM:	toast, jelly, coffee, scrambled egg, juice
Lunch:	soup or stew or corn tortillas with cheese
Dinner:	Wife usually cooks rice or cornmeal, some type of meat (pork, beef, poultry, venison), vegetables, cornbread or fry bread.

MD Progress Note:

12/9/14 07:00

Subjective: Mr. B previous 24 hours reviewed. **Previously diagnosed with T2DM; treated with metformin but appears to not have taken it regularly.**

Vitals: Temp: 99.5, Pulse: 82, RR: 25, BP 101/78

Urine Output: 2660 mL (71.8mL/kg)

PE: General: Alert and oriented to person, place, time

HEENT: WNL

Neck: WNL

Heart: WNL

Lungs: Clear to auscultation

Abdomen: Active bowel sounds

Assessment: Results: + ICA, GADA, IAA consistent with T1DM. **Negative c-peptide.**

Dx: T1DM

Plan: Begin Novolog 0.5 u every 2 hour until glucose is 150-200 mg/dL. Tonight begin Glargine 15 u at 9 pm. Progress Novolog using ICR 1:15. Continue bedside glucose checks hourly. Notify MD if BG > 200 or < 80. RDN consult on SMBG.

C. Johnston, MD

Intake/Output

Date	12/9/14 0701 – 12/10/14 0700				
Time		07001-1500	1501-2300	2301-0700	Daily Total
IN	P.O.	NPO	NPO	720	720
	I.V. (mL/kg/hr)	2,400 (4)	2,400 (4)	2,400 (4)	7,200 (4)
	I.V. piggyback	0	0	0	0
	TPN	0	0	0	0
	Total intake (mL/kg)	2,400 (32)	2,400 (32)	3,120 (41.6)	7,920 (105.6)
OUT	Urine (mL/kg/h)	2,150 (3.58)	2,671 (4.45)	3,000 (5)	7,821 (4.34)
	Emesis output	150	0	0	150
	Other	0	0	0	0
	Stool	0	x 1	0	x 1
	Total output (mL/kg)	2,300 (30.7)	2,671 (35.6)	3,000 (40)	7,971 (106.3)
Net I/O		+ 100	-271	+120	-51
Net since admission (12/9)		+ 100	-271	+120	-51

Laboratory Results

	Ref. Range	12/9/14 1780
Chemistry		
Sodium (mEq/L)	136-145	130 !↓
Potassium (mEq/L)	3.5-5.5	3.6
Chloride (mEq/L)	95/105	101
Carbon dioxide (CO ₂ , mEq/L)	23-30	31 !↑
BUN (mg/dL)	8-18	18
Creatinine serum (mg/dL)	0.6-1.2	1.1
Glucose (mg/dL)	70-110	683 !↑
Phosphate, inorganic (mg/dL)	2.3-4.7	2.1 !↓
Magnesium (mg/dL)	1.8-3	1.9
Calcium (mg/dL)	9-11	10
Osmolality (mmol/kg/H ₂ O)	285-295	306 !↑
Bilirubin total (mg/dL)	≤1.5	0.2
Bilirubin, direct (mg/dL)	<0.3	0.01
Protein, total (g/dL)	6-8	6.9
Albumin (g/dL)	3.5-5	4.4
Prealbumin (mg/dL)	16-35	32
Ammonia (NH ₃ , umol/L)	9-33	9
Alkaline phosphatae (U/L)	30-120	110
ALT (U/L)	4-36	6.2
AST (U/L)	0-35	21
CPK (U/L)	30-135 F 55-170 M	61
Lactate dehydrogenase (U/L)	208-378	229
Cholesterol (mg/dL)	120-199	210 !↑
Triglycerides (mg/dL)	35-135 F 40-160 M	175 !↑
T ₄ (ug/dL)	4-12	8
T ₃ (ug/dL)	75-98	81
HbA _{1C} (%)	3.9-5.2	12.5 !↑
C-peptide (ng/mL)	0.51-2.72	0.09 !↓
ICA	-	+ !↑
GADA	-	+ !↑
IA-2A	-	-
IAA	-	+ !↑
tTG	-	-
Hematology		

WBC ($\times 10^3/\text{mm}^3$)	4.8-11.8	10.6
RBC ($\times 10^6/\text{mm}^3$)	4.2-5.4 F 4.5-6.2 M	5.8
Urinalysis		
Collection method	-	Catheter
Color	-	Yellow
Appearance	-	clear
Specific Gravity	1.003-1.030	1.008
pH	5-7	4.9 ! ↓
Protein (mg/dL)	Neg	+1 ! ↑
Glucose (mg/dL)	Neg	+3 ! ↑
Ketones	Neg	+ 4 ! ↑
Blood	Neg	Neg
Bilirubin	Neg	Neg
Nitrites	Neg	Neg
Urobilinogen (EU/dL)	<1.1	Neg
Leukocyte esterase	Neg	Neg
Protein check	Neg	tr ! ↑
WBCs (/HPF)	0-5	0
RBCs (/HPF)	0-5	0
Bacteria	0	0
Mucus	0	0
Crys	0	0
Casts (/LPF)	0	0
Yeast	0	0
Arterial Blood Gases (ABGs)		
pH	7.35-7.45	7.31 ! ↓
pCO ₂ (mm Hg)	35-45	35
SO ₂ (%)	≥95	97
CO ₂ content (mmol/L)	25-30	28
O ₂ content (%)	15-22	21
pO ₂ (mm Hg)	≥80	89
Base excess (mEq/L)	>3	-
Base deficit (mEq/L)	<3	-
HCO ₃ ⁻ (mEq/L)	24-28	22 ! ↓
COHb (%)	<2	1.1

1. What are the differences between T1DM and T2DM? Explain the pathophysiology, dx, and treatment of each. (4 pts)

Type 1 diabetes:

T1DM is characterized by an absolute deficiency of insulin due to destruction of pancreatic beta cells, resulting in inability of cells to use glucose for energy. usually diagnosis in childhood. rapid onset Diagnosis can be made on the basis of a casual plasma glucose ≥ 200 mg/dl in addition to certain symptoms (unexplained weight loss, polydipsia, polyuria, vomiting ketoacidosis have ketone smell), or fasting plasma glucose ≥ 126 mg/dl or 2 hour post prandial glucose ≥ 200 mg/dl (NTP, p485) treatment included tight glycemic control, insulin therapy, evaluate serum lipid levels, monitor blood glucose level. initiate self-management training for patient on (insulin administration, nutrition prescription, meal planning, signs/symptoms and Tx, hypo-hyperglycemia monitoring instructions (SBGM, urine ketones, and use of record system, exercise), baseline visual examination, contraception education (lecture 15, slide 90).

Type 2 diabetes:

Whereas T1DM results from lack of insulin caused by destruction of beta cells, patients with T2DM produce insulin, but their tissue are insulin resistant. This causes increased need for insulin, so the pancreas increases production. Eventually the pancreas loses its ability to produce insulin. Usually metabolic syndrome is related to insulin resistance. Typically diagnosis in adulthood. Medical diagnosis is the same as T1DM but patient usually has no symptoms like ketoacidosis, sudden weight loss, absence of autoantibodies of T1DM (NTP p499). T2DM is associated usually with overweight & obesity, has genetic contribution. can be controlled with diet, exercise, medications. Symptoms included polyuria, polydipsia, polyphagia, blurry vision, feeling tired, slow healing of cuts and wounds. Treatment included weight management, carbohydrate counting, frequent blood glucose monitoring.

2. Why do you think Mr. B was originally diagnosed with T2DM? Why does the MD now state that he has T1DM? (2 pts)

He was probably originally diagnosed with T2DM because of his age, T1DM usually diagnosis in childhood, and T2DM is usually diagnosis in adults, and the prevalence increases with age (NTP p498). And he has family history of T2DM (his mother), but no history of T1DM. Also, his body might still have been producing some insulin at the time of diagnosis, which might be an indicator of DM2. Rate of beta cell destruction is variable, fast primarily in infant and children, slow in adults. And residual beta cell function that is sufficient to prevent ketoacidosis may be preserved in adults diagnosis with T1DM. (NTP P483). Furthermore, his blood glucose level and A1c is consistent with diabetic state. Those reasons might lead the doctor to diagnose him with T2DM.

The MD now suspects that he has T1DM because his lab assessment was positive with presence of islet cell autoantibody (ICA), GADA, and IAA. Those autoantibodies are indicators for the body's destructive immune response against its own beta cells, which indicate T1DM.

3. Describe the metabolic events that led to Mr. B's symptoms and subsequent admission to the ER (polyuria, polydipsia, polyphagia, fatigue, and weight loss), integrating the pathophysiology of T1DM into your discussion. (2 pts)

T1DM will cause an increased sugar content in blood due to absence of insulin that would allow glucose into cells. Mr. B experiences polyuria because the kidney senses the abnormal blood glucose level, and trying so hard to get it back to maintain homeostasis through urination. Since this is a chronic situation, the kidney needs to work continuously, producing extra urine to get rid of extra glucose. Mr. B felt polydipsia because the extra urine the kidney produced in order to get rid of extra glucose is the fluid drawn from the rest of body tissues. So the person will feel thirsty because the brain senses that and wants to get more fluid to prevent dehydration. Mr. B experiences polyphagia because cells are starving without glucose as an energy source, so signals are being sent to the brain, and the person will want to consume more food. Mr. B felt fatigue because the glucose in blood can't enter cells due to deficiency of insulin, so energy is not as readily available for the cells, so they have to seek other sources such as stored fat for energy, which takes longer so the person feels fatigued. Mr. B experienced weight loss because his body is not able to use glucose as an energy source, so it has to hydrolyze triglycerides from its fat stores to get energy. This will reduce the

sizes of fat cells and cause the person to lose weight. Also because the kidney worked so hard to urinate in order to get rid of extra glucose, so water is eliminated along with it. This results in dehydration and lowered body weight. Although glucose is not able to be utilized by cells, the body still thinks cells have a need for carbohydrate, so the lean body tissue is broken down for making glucose (gluconeogenesis) in order to provide the body with some carbohydrate source. This results in tissue loss and further weight loss.

4. Describe the metabolic events that result in the signs and symptoms associated with DKA. Was Mr. B in this state when he was admitted? What precipitating factors may lead to DKA? (2 pts)

When the body has to use fat stores for the production of energy and functioning, it hydrolyzes fat into fatty acid chains and glycerol for energy, and the fatty acids go through beta oxidation, and ketone bodies are produced. Then the body gets energy by converting ketone bodies into acetyl-coA molecules that can go through the citric acid cycle and be oxidized to produce energy. However, if there is an overwhelming quantity of those ketones in the blood, the pH can drop severely due to the acidic nature of the molecules and ketone bodies are secreted in the urine. Metabolic acidosis develops as bicarbonate concentration is reduced. This can lead to a state of overly acidic blood known as diabetic ketoacidosis, which is characterized by deep, labored respirations (Kussmaul respirations), acetone breath, impaired brain function, vomiting, or in severe cases, coma and death. (NTP, P483)

In addition to ketogenesis, the breakdown of lean muscle tissue for gluconeogenesis, which will further raise blood glucose, we see the persistence of hyperglycemia, polyuria, polydipsia, and the reliance on other energy stores to provide energy for the body. This in turn leads to the continued pathogenesis that causes DKA.

Mr. B was in an early state of DKA when he was admitted, as he was found groggy and nearly unconscious in his home. His state could have advanced to coma if he had not been brought to the hospital when he was. The precipitating factors that may lead to DKA are the new onset of T1DM, lack of blood glucose self-monitoring, severe illness or infection, insulin omitted, increased insulin needs with growth spurts, inappropriately stored insulin. (NTP, P486)

5. What is the relationship of HgbA1c values to the micro- and macro-vascular complications of diabetes? List 3 micro-vascular complications of DM. (2 points)

HgbA1c values above the normal range (4-6%) create an increased risk of complications of diabetes. These long-term complications of insulin deficiency along with hyperglycemia can be either macrovascular and microvascular. The microvascular complications include nephropathy (end-stage renal disease), retinopathy (blindness), and neuropathy (peripheral neuropathy).

6. Mr. B will be started on a combination of Glargine given in the pm with Novolog prior to meals and snacks. Describe the onset, peak, and duration for each of these types of insulin. (2 pts)

Glargine:

Glargine is a long-acting insulin analog taken once daily. Glargine's onset time is 2-4 hours, it is considered peakless because it is a steady-release, and its duration is 20-24 hours so it provides basal insulin throughout the day. (NTP, P488)

Novolog:

Novolog is a fast-acting insulin analog taken with meals. Novolog's onset time is 5-15 minutes, peak is 30-90 minutes, and its duration is 3-5 hours. (NTP, P488)

7. Identify any abnormal laboratory values measured upon Mr. B's admission. Explain how they may be related to his newly diagnosed T1DM. Discuss only relevant labs. (2 pts)

His sodium level is below normal range, sodium retain water ,it is a indicator of loss of total body water due to body's response to hyperglycemia (frequent urination)

His carbon dioxide is above normal, it is a indicator of increased CO₂ retention, which is common with DKA
DKA is one of common symptoms of uncontrolled T1DM.

His glucose level is way out of normal range, indicating fasting hyperglycemia due to lack of insulin production of T1DM

His inorganic phosphate level is low, usually phosphate depleted due to urine loss.It indicates loss of total body water might due to body's response to hyperglycemia caused by T1DM(frequent urination)

His osmolality is quiet high , it indicate imbalance of body water, low sodium or water loss ,which may be associated with T1DM(frequent urination)

His C peptide level is low ,it indicates the presense of insulin in the blood,which in turn indicates slowly progressing beta-cell destruction

His cholesterol and triglyceride are above normal range,this may suggest hyperlipidemia or uncontrolled hyperglycemia caused by T1DM,or family history.Espeically triglyceride ,might indicate insulin deficiency which cause increased hormones to break down adipose tissue and release fatty acids in blood.

His high HBA1C levels is way high above normal in blood, Hba1c is a measurement of blood glucose concentration from previous 3 months, high value indicates long term increased blood glucose

Additionally, the presence of ICA (Islet Cell Autoantibody) GADA (Glutamic Acid Decarboxylase Autoantibody), and IAA (Islet Autoantibody) in the blood indicating autoantibody attacking of B cells and ongoing destruction of B cells, thus those are biomarkers for T1DM

urinalysis results

The low pH is evidence of ketoacidosis, which is symptoms of T2DM.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.

His urine has a high protein level. Along with carbohydrate metabolism, protein metabolism is affected by insulin deficiency. In a normal individual, insulin lowers blood amino acid and glucose levels, aid in amino acid incorporation into tissue protein, and decreases gluconeogenesis. With insulin deficiency, gluconeogenesis increases and amino acid release (proteolysis) occurs in muscle, therefore increasing protein levels.

There is a high level of glucose in urine, which indicates the excretion of excess glucose from the blood. Due to this hyperglycemic condition.

The presence of ketones in urine is evidence of ketoacidosis,which is also an indication of insulin deficiney. Insulin deficiency cause the release of hormones that release fatty acids in the blood that are converted to ketone bodies, which are secreted in the urine.

The presence of protein in urine might indicate spilling of protein into the urine , kidney dysfunction (complications of DM1).

Arterial blood gases

Ph is low, evidence of ketoacidosis

HCO₃ level is low, bicarbonate abnormal level is suggesting body is having trouble of maintaining acid –base balanced ,might be the evidence of diabetic ketoacidosis. kidney disease caused by DM1

8. You meet with Mr. B before d/c and review SMBG. Based on the information above, write your initial nutrition assessment ADIME note for Mr. B, including 2 PES statements (include calculations & references on an attached sheet). (12 points)

A:

Patient is 48 year old Native American male Anthropometric data Ht= 1.78m Wt =72.7kg BMI=22.9 IBM=75.45kg %IBW=96.4%

Biochemical data: low sodium level of 130 mg/dL, low phosphate level of 2.1 mg/dL high carbon dioxide level 31mEq/L, high osmolality level 306 mmol/kg/h₂O Elevated blood glucose level of 683 mg/dL and elevated HbA1c level at 12.5% ,elevated cholesterol level of 210 mg/dL, elevated triglycerides level of 175 mg/dL, and low C-peptide level of 0.09 ng/dL urinalysis;low ph 4.9,presense of protein+1, presence of glucose+3,presense of ketone +4, ABGs; low ph 7.31,low Hco₃ 22mEq/L urine appearance; cloudy amber

NPO then progress to clear liquids and then consistent CHO-controlled diet. does not follow traditional tribal eating pattern,with a few exceptons like fry bread and prepare wojapi seasonally.Pt does not tolerate milk and only eat cheese when obtained through government commodities.Fresh vegetables are grown at home ,squash ,peppers beans corn and some greens.There is very little fruit in his diet and meat is only eaten at dinner

Admission order ; 1. Regular insulin 1 unit/mL in NS 40mEq KCl/liter @ 300 mL/hr. Begin infusion at 0.1 unit/kg/hr = 3.7 units/hr and increase to 5 units/hr. Flush new IV tubing with 50mL of insulin drip solution prior to connecting to pt and starting insulin infusion.

2. NPO except for ice chips and medications. After 12 hours, clear liquids when stable. Then advance to consistent CHO diet order: 70-80g breakfast and lunch; 85-95 g dinner; 30 g snack pm and HS

3 Consult diabetes education team for self-management training to begin education after stabilized.

Protein Requirement

= **58 – 73 g protein/day**

Fluid Requirement =**2181 ml.**

Carbohydrate recommend intake =**1277 kcal**

Fat recommend intake =**697 kcal**

energy requirement

EER=TEE=2322 kcal

Patient diagnosed with T2DM one year ago, and has been on metformin since that dx.He does not take the medication regularly as he felt it really wasn't necessary.Mother has history of T2DM.smoked 1 ppx 10 years (no longer smokes) usual lunck intake for past several months ,AM ;toast jelly coffee ,scrambled,egg juice.Lunch;soup,or stew or corn tortillas with cheese.Dinner wife usually cooks rice or cornmeal ,some type of meat (pork,beef ,poultry ,venison),vegetables ,cornbread or fry bread.

D:

Food and nutrition-related knowledge deficit (NB-1.1) related to lack of education regarding newly diagnosed T1DM diagnosis as evidenced by patient self report of not thinking necessary to take medication regularly.

Altered nutrition-related laboratory values(NC-2.2) related to uncontrolled DM1 as evidence by serum glucose level of 610 mg/dl and HBA1C level of 12.5%

I:

Follow consistent and controlled diet so that nutrition-related laboratory values will normalize.

Modify composition of eating bouts, specifically a carbohydrate-modified diet ,in order to control the amount of carbohydrate consumed and ensure the adequacy of prescribed insulin therapy. This includes following a consistent carbohydrate-controlled diet, consuming breakfast and lunch with 70-80g carbohydrate each, dinner with 85-95g carbohydrate, and an afternoon and bedtime snack each with 30g carbohydrate. Total daily carbohydrate intake should be between 232 and 336 grams.

Provide nutritional education regarding new T1DM diagnosis so as to enhance patient ability to consume a controlled yet adequate diet necessary for improvement of current condition.

Provide nutrition-related education to the patient regarding priority modifications to the diet

Continue to provide application-centered education, in which result interpretation and skill development are assessed to ensure the patient is fully competent in managing his T1DM. This includes ensuring patient is capable of calculating carbohydrate content of foods, understanding glycemic index, making healthful dietary choices in order to meet carbohydrate goals, and staying within daily recommendations for carbohydrate intake. Also that the patient understands physical activity interaction with blood glucose levels,how to calculate the amount of insulin required for each carbohydrate choice by understanding ICRs and correction factors to return glucose level to normal.Providing him with an exchange list so he can more easily count his CHO serving while allowing for flexibility in the diet.

M/E:

Monitor serum glucose and Hg-A1c levels over time compared to normal ranges, to evaluate dietary control of
Continue nutrition education sessions to monitor
patient'understanding of T1DM until knowledge proficiency is evident and patient is able to take necessary steps in controlling his disease on his own.

Monitor for perceived importance of compliance to medications

Monitor client-reported dietary intake to ensure adherence to carbohydrate intake recommendations for T1DM

Monitor weight to ensure body weight is being maintained

9. **Mr. B comes back to clinic 2 weeks after his new diagnosis. List the important questions you will ask him in order to plan the next steps for providing the additional education that he might need. (2 pts)**

It is important to ask him questions about his daily regimen, including how often he is checking his blood

glucose, his eating habits, exercise, how he has been feeling, what his blood sugar range has been, and any concerns or questions he has. If there are any signs that he isn't following his insulin regimen or he is having hypoglycemic events despite treatment, if his family or friends been supportive of his lifestyle life change ,if he made any changed in his diet to increase fruit ,vegetables and whole grain intake

10. Mr. B's usual breakfast consists of 2 slices of toast, butter, 2 T jelly, 2 scrambled eggs, and orange juice (~1 cup). Using the ICR 1:15, how much Novolog should he take to cover the carbohydrate in this meal? (1 pt)

2 slices of toast: 30 g/carb (15g per slice or 2 carbohydrate exchanges) butter: 2 tbsp jelly: 30 g/carb (15g per tbsp or 2 carbohydrate exchanges) 2 scrambled eggs: 0g carb 1 cup orange juice: 30g/carb (15g per ½ cup or 2 carbohydrate exchanges)

Total carbs: 90g or 6 carbohydrate exchanges

Because 1 unit of Novolog covers 15 grams of carbs (or 1 carbohydrate exchange,) he would need about 6 units to cover his breakfast meal containing 90 grams of carbs (or 6 carbohydrate exchanges).

11. You determine that Mr. B needs 2322 kcals/day based on EER calculations. You want to follow his normal eating pattern as much as possible while still meeting his protein requirements and keeping the kcal from fat at 30% or less of total kcals. Using the Diabetes Exchange/Food List and the worksheet below, develop a "pattern" for Mr. B's diet. (15 points)

Food group	Exchanges	CHO grams	Protein grams	Fat grams
Breakfast 116				
Starch	2	30	6	2
Fruit	1	15	0	0
Milk (circle: whole, 2%, 1%, or NF)				
Meat (circle: lean , med or high fat)	3	0	28	8
Non-starchy vegetables	2	10	4	0
Fat	3	0	0	15
Morning Snack (list food groups)				
starch	2	30	6	2
fruit	1	15	0	0
Lunch				
Starch	2	30	6	2
Fruit	1	15	0	0
Milk (circle: whole, 2%, 1%, or NF)				
Non-starchy vegetables	3	15	6	0
Meat (circle: lean , med or high fat)	2	0	14	4
Fat	2	0	0	10
Afternoon Snack (list food groups) 48				
fat	3.5	0	0	17.5
starch	2	30	6	2
Dinner 6				
Starch	2	30	6	2

Fruit	2	30	0	0
Milk (circle: whole, 2%, 1%, or NF)				
Non-starchy vegetables	6	30	12	0
Meat (circle: lean , med or high fat)	2	0	14	4
Fat				
HS Snack (list food groups)				
Other carbohydrates	1	15	0	5
Total grams:		295	108	73.5
		x4	X4	X9
kcal from each macronutrient:		1180	432	661.5
% kcal from each macronutrient:				
% kcal GOAL:		51.9%	19%	29.1%
TOTAL KCAL:		2273 kcal		

12. You review Mr. B's diet, insulin injections, SMBG, and other self-care issues. He continues on injections of Glargine and Novolog. You reinforce teaching Mr. B about carbohydrate counting. How many CHO "points" or servings are in his daily diet from question 11? (1 point)

Total carb/15=295g /15=20 serving
There are 20 CHO serving in his daily diet from question 11

13. If Mr. B's pre-prandial BG was measured at 200 mg/dL and he plans to eat a lunch consisting of a cup of vegetable bean soup, a piece of fry bread, a piece of fruit and a diet soda, how much insulin should he take to cover the meal, and how should it be adjusted to compensate for the BG level? Assume that the correction dose of 1 unit of insulin decreases blood glucose by 50 mg/dl, correct to 150 mg/dl, and an ICR of 1:15. (2 points)

1 cup of vegetable bean soup; 15 g /carb (15 g per cup or 1 carbohydrate exchange)
a piece of fry bread; 15 g/carb (15g per slice or 1 carbohydrate exchange)
a piece of fruit 15g/carb(1 carbohydrate exchange)
a diet soda; 0g/carb
correction dose 200 mg/dl-150 mg/dl=50 mg/dl
50 mg/dl/50 mg/dl./unit insulin =1 unit insulin

3 unit insulin he should take to cover meal.1 unit insulin should be adjusted to compensate for the BG level

14. Describe the Native American foods, fry bread and wojapi. These would be categorized as what type of exchange? Include the reference used. (1 point)

Fry bread and wojapi is a traditional native American berry dish. Fry bread is often made of coconut milk, white flour and baking powder, and need oil for frying the bread. Wojapi is a thick berry sauce.If your berries are ripe and tasty,there is no need to add additional sweeteners. Traditionally, Wojapi is not made with cornstarch, flour and sugar.These would be categorized as sweets, desserts, and other carbohydrate.

Reference; <http://recipes.sparkpeople.com/recipe-detail.asp?recipe=1785669>

CALCULATIONS:

- Height: 5'10" (70" x 2.54 cm = 177.8cm = **1.78 m**)

- Weight: 160# (160# / 2.2 kg = **72.7 kg**)

- BMI (NTP p. 247): 72.7 kg / (1.78)² = **22.9**

Normal Weight stage (NTP p. 247): BMI = 18.5 - 25.0 kg/m²

IBW: Hamwi equation (NTP p. 48) = 106 lbs for 5' + 6 lbs for every inch above 5'
: 5'10" = 106 lbs + (6 lbs x 10 in) = **166 lbs ± 10%**

166 lbs / 2.2 = **75.45 kg**

- % IBW: 160# / 166# = 1.04 → patient is **96.4% IBW**

- energy requirement (NTP p242)

EER=TEE

EER=662-9.53x age+PA x(15.91xweight+539.6 x height)

=662-9.53x48+1x(15.91x72.7+539.6x 1.78)

=204.56+1156.657+960.488

=2322 kcal

Protein Requirement (NTP p. 61): 0.8 – 1.0 g/kg/day

72.7 kg x (0.8 – 1.0 g/kg/day) = **58 – 73 g protein/day**

Fluid Requirement =Wt(kg)x30ml

=72.7 kgx30ml=2181 ml.

Carbohydrate recommend intake 55% of EER

2322 X 55 %=1277 kcal

Fat recommend intake 30 % of EER

2322 x30%=697 kcal