Type 1 Diabetes Mellitus in the Adult

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KNH 411: Medical Nutrition Therapy I
Diabetes Mellitus: Type I

- Genetic factor
- Sudden onset
- Majority children and adolescents with an increasing incidence in adults
- Autoimmune destruction of B-cells of pancreas resulting in insulin deficiency
- Cell become in able to use glucose causing hyperglycemia & cells starve
- Excess sugar cannot be processed by kidneys and is removed from urine causing polyuria and glycosuria
Type I Diabetes

1. Stomach converts food to glucose
2. Glucose enters bloodstream
3. Pancreas produces little or no insulin
4. Glucose unable to enter body effectively
5. Glucose levels increase
Diabetes Mellitus: Type II

- Most frequently diagnosed in adults with increasing rates in children and adolescents
- Progressive onset
- Increased prevalence with age and race
- Obesity, heredity, physical inactivity contribute
- Produce insulin but have insulin resistant tissues
- Pancreas increased production with increased need
- Pancreas eventually loses ability to produce insulin
  - Insulin resistance and relative insulin deficiency
Type II Diabetes

1. Stomach converts food to glucose

2. Glucose enters bloodstream

3. Pancreas produces sufficient insulin but the body is resistant to its effective use

4. Glucose unable to enter body effectively

5. Glucose levels increase
LADA

• “Latent autoimmune diabetes of adulthood”
• Form of T1DM
  o Sometimes called T1.5DM
  o Slowly progressive form
• Often diagnosed with T2DM first but have positive PIA, GADA, and low levels of C-peptide during progression
• Slow destruction of B-cells, decrease in insulin production by pancreas over time
Patient Summary

Armando Gutierrez
- 32-year-old hispanic male
- **Weight:** 165 lbs  **Height:** 5’11”  **BMI:** 23 kg/m²
- **IBW:** 172 lbs  **ABW:** 170 lbs
- Family hx of MI, T2DM, ovarian cancer
Patient Summary (cont.)

- Consumes **alcohol** daily, **smokes** 1 ppd X 10 yrs
- **Usual diet:** high carbs, high fat, low fruit & vegetable intake, eats out 3-4 x per week
- **Sedentary job** (computer software engineer)
Dx with T2DM one year ago, Rx for metformin, but does not take regularly

Transported to ER by a friend
  ○ Found groggy and almost unconscious
  ○ Serum glucose of 610 mg/dL

MD suspects T1DM or T1.5DM
Diabetic Ketoacidosis

- Armando in ketoacidosis upon admission
  - Insulin deficiency causes production of hormones leading to lypolysis
  - Fatty acids in blood transformed to keto acids in liver
  - pH falls (acid-base imbalance), keto bodies excreted in urine
  - Symptoms: heavy breathing, N/V, stomach pain, acetone breath, and changed mental status.
Etiology

- T1.5DM results from the cellular-mediated autoimmune progressive destruction of beta-cells of the pancreas, which rises plasma glucose levels and starves the body’s cells.
## Laboratory Data - blood

<table>
<thead>
<tr>
<th>Lab Results</th>
<th>Ref. Range</th>
<th>Patient Results</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mEq/L)</td>
<td>136-145</td>
<td>130</td>
<td>Indicator of fluid loss &amp; frequent urination caused by overworking of kidneys to filter high levels of blood glucose.</td>
</tr>
<tr>
<td>Carbon Dioxide (CO₂, mEq/L)</td>
<td>23-30</td>
<td>31</td>
<td>Indication of an acidotic state/ketoacidosis.</td>
</tr>
<tr>
<td>Phosphate, inorganic (mg/dL)</td>
<td>2.3-4.7</td>
<td>2.1</td>
<td>Indicator of fluid loss &amp; frequent urination caused by overworking of kidneys to filter high levels of blood glucose.</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>70 - 110</td>
<td>683</td>
<td>Indicates fasting hyperglycemia and improper use of blood glucose due to insulin resistance or lack of insulin production.</td>
</tr>
<tr>
<td>Osmolality (mmol/kg/H₂O)</td>
<td>285-295</td>
<td>306</td>
<td>Indicator of fluid loss &amp; frequent urination caused by overworking of kidneys to filter high levels of blood glucose.</td>
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<td>-----------------------</td>
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<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>120 - 199</td>
<td>210</td>
<td>Related to family Hx, current diet high in fat, CHO, eating out and low in fruits and vegetables, and/or diabetes, which is a risk factor for dyslipidemia.</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>140 - 160 M</td>
<td>175</td>
<td>Related to family Hx, current diet high in fat, CHO, eating out and low in fruits and vegetables, and/or diabetes, which is a risk factor for dyslipidemia.</td>
</tr>
<tr>
<td>HbA₁C (%)</td>
<td>3.9 - 5.2</td>
<td>12.5</td>
<td>Measurement of blood glucose concentration from previous 2-3 months; high value indicates long-term increased blood glucose and poor DM management</td>
</tr>
<tr>
<td>C-peptide (ng/mL)</td>
<td>0.51-2.72</td>
<td>0.09</td>
<td>Indication of the presence of insulin in the blood, which in turn indicates slowly progressing beta-cell destruction. Normally absent in T1DM.</td>
</tr>
<tr>
<td>ICA</td>
<td>-</td>
<td>+</td>
<td>Autoantibody indicating attack of B-cells and thus a marker for T1DM</td>
</tr>
<tr>
<td>GADA</td>
<td>-</td>
<td>+</td>
<td>Autoantibody indicating attack of B-cells and thus a marker for T1DM (and LADA).</td>
</tr>
<tr>
<td>IAA</td>
<td>-</td>
<td>+</td>
<td>Autoantibody indicating ongoing destruction of B-cells and thus a marker for developing T1DM (and LADA).</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>Ref. Range</td>
<td>Patient Results</td>
<td>Explanation</td>
</tr>
<tr>
<td>------------------</td>
<td>------------</td>
<td>----------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>pH</td>
<td>5 - 7</td>
<td>4.9</td>
<td>Indication of an acidotic state/ketoacidosis.</td>
</tr>
<tr>
<td>Protein (mg/dL)</td>
<td>-</td>
<td>+1</td>
<td>Indication of protein loss into the urine due to overworked kidney filtration system and subsequent leakage as a result of high blood sugar.</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>-</td>
<td>+3</td>
<td>To compensate for hyperglycemia, excess glucose is lost in the urine.</td>
</tr>
<tr>
<td>Ketones</td>
<td>-</td>
<td>+4</td>
<td>Indication of an acidotic state/ketoacidosis.</td>
</tr>
<tr>
<td>Prot chk</td>
<td>Neg</td>
<td>Tr</td>
<td>Indication of protein loss into the urine due to overworked kidney filtration system and subsequent leakage as a result of high blood sugar.</td>
</tr>
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</table>
Standard Diagnostic Criteria

• **T1DM**
  - Symptoms of diabetes (i.e. polyuria, polydipsia, unexplained weight loss) plus plasma glucose 200mg/dL or
  - Fasting glucose >126mg/dL or
  - 2 hour post-prandial glucose >200mg/dL

• **LADA**
  - Presence of diabetes-related autoantibodies (i.e. IAA, ICA, GADA, IS-2A)
Medications

- MD prescribed Novolog and glargine
- **Novolog**
  - Rapid acting insulin analog
  - Onset of action **5-15 min**, peak of action **30-90 min**, duration of action **3-5 hrs**
  - can be used in pump therapy
- **Glargine**
  - Extended long-acting analog
  - Onset of action **2-4 hrs**, peakless, duration of action **20-24 hrs**
  - Cannot be mixed with other insulins
Medications (cont.)

• Armando’s discharge dose glargine = 45 units of glargine daily

• Insulin-to-Carbohydrate Ratio (ICR) = 11g CHO per 1 unit of glargine (insulin)
Medical Nutrition Therapy - Basic principles

- Attain and maintain metabolic outcomes (i.e. plasma glucose, lipid profile, B.P.)
- Enhance health through food choices and physical activity
- Address individual needs (i.e. personal preferences, cultural preferences, individual’s wishes and willingness to change)
Nutrition 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 urinanalysis, glucose of +3 mg/dL in urinanalysis, protein of +1 and low pH of urine and blood.

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

- Self-manage carbohydrate counting
  - Record blood glucose at least 3X/day
  - Educate on target ranges
    - <100 mg/dL = fasting glucose
    - <180 mg/dL = postprandial
- Counseling session to quit smoking
- Incorporate physical activity
- Take medication daily
MNT - Monitoring & Evaluation

- Record SMBG before & after each meal
- Record dietary intake & medications
  - Report back to medical team in 2 weeks
- Test urine for glucose and ketones
- Test blood for glucose and lipids
- Test blood pressure
- Education sessions progress with routine
Prognosis

- Lifelong disease
- Control blood glucose to prevent complications
  - Complications can arise despite control
- Get to a point of management
Questions? Comments?
References


