Care of the Patient with Diabetes

Rosa Matonti RN, MSN, CDE, CNS
Inpatient Diabetes Educator
University of New Mexico Hospital
Pager 505-951-4352 Office 505-925-6100
rmatonti@salud.unm.edu
Sacred Coeur Hospital, Milot, Haiti
Objectives

At the end of the session the learner will be able to:

• Explain the role of counter regulatory hormones in maintaining glucose levels.
• Describe the importance of glucose control during illness and recovery
• Differentiate between type 1 and type 2 diabetes.
Prevalence of Diabetes

• National Statistics
  – Among people greater than 18 years of age in the US in 2007, 8% were diagnosed with diabetes.
  – In comparison, in Haiti diabetes affects 7.4% in men and 11.1% in women.
  – In the US diabetes is expected to increase 60% in the next 22 years


What is Diabetes?
Pathophysiology of Glucose Regulation

• Food eaten, carbohydrates converted into glucose
• Regulation of blood glucose depends on the liver
• 60% of glucose from food is converted to glycogen
• When liver cells are saturated additional glucose is converted to fat
• Peripheral muscle cells also store glucose
Insulin is secreted by the beta cells of the pancreas in response to glucose uptake by body tissues. Insulin promotes the uptake of glucose into muscle and other cells, where it can be used as an energy source or stored as glycogen. In the liver, glucose is converted to glycogen for storage or to other storage forms like fats and proteins.

When there is an increase in blood glucose levels, the pancreas responds by secreting more insulin. Conversely, a decrease in blood glucose levels leads to a reduction in insulin production. This regulation maintains homeostasis, keeping blood glucose levels within the normal range (below 110 mg per dl).

Glucagon, produced by the alpha cells of the pancreas, is secreted in response to low blood glucose levels. Glucagon promotes the breakdown of stored glycogen in the liver to release glucose into the bloodstream, helping to increase blood glucose levels when they are low. Glucagon also has a role in fat and protein metabolism.

The body maintains glucose homeostasis through the interplay of insulin and glucagon, as well as other hormones and metabolic processes, ensuring that blood glucose levels remain within a healthy range.
History of Insulin

- 1921 Nicolae Paulescu first to isolate insulin (pancrein)
- Spring 1921 Banting traveled to Toronto
- Banting and Best isolated beta cells from dogs, producing isletin (insulin).
- Took 6 weeks to extract isletin
- Went to using fetal calf pancreas
- Next Banting invited James Collip (biochemist) to purify the extract.
- January 11, 1922, Leonard Thompson was given first injection of insulin.
- Collip improved the extract and the second dose was given on January 23, 1922
- April 1922 Eli Lilly combined efforts with Banting
- Won Nobel Prize in 1923
Insulin…the impact

The Miracle of Insulin

Patient J.L., December 15, 1922

February 15, 1923
Important Functions of Insulin

- Insulin allows glucose into the cell
- Enhances uptake of glucose by the liver
- Prevents the breakdown of stored glycogen back to glucose.
Important Functions of Insulin

- Insulin secreted continuously is the **basal rate**.
- Insulin response after a meal is a **bolus**.
- Insulin affects protein and mineral metabolism
- Enhances fat storage and prevents fats from being used for energy
Physiological Insulin Secretion

Normal 24-Hour Profile

1. Nutritional Insulin

2. Basal Insulin: Suppresses Glucose Production Between Meals And Overnight
Insulin Requirements in Health and Illness

# Counterregulatory Hormones

<table>
<thead>
<tr>
<th>Raises Blood Sugar</th>
<th>Source</th>
<th>Action of Hormone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucagon</td>
<td>pancreas’ alpha cells</td>
<td>Stimulates glycogenolysis gluconeogenogenesis</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Adrenal gland’s medulla</td>
<td>Causes rapid rise in blood glucose in times of stress</td>
</tr>
<tr>
<td>Cortisol</td>
<td>Adrenal gland’s cortex</td>
<td>Maintains blood glucose levels during fasting and stress</td>
</tr>
<tr>
<td>Growth Hormone</td>
<td>Pituitary gland</td>
<td>Causes slow rise in blood glucose</td>
</tr>
</tbody>
</table>
To Review:

- Control of blood glucose depends on:
  - Insulin is secreted with **high blood glucose** and helps glucose enter the cells and inhibits the liver from converting glycogen back to glucose.
  - Counterregulatory hormones are stimulated by **low blood glucose** and act to raise blood glucose.
Physiology of the Stress Response

Stress is anything that activates the body’s mechanism’s to adapt

- Emotional stress
- Physical stress
  - Illness
  - Infection
  - Surgery
  - Trauma

Stress Response
- How bodies have adapted to help survive sudden danger.
- Increased secretion of counterregulatory hormones.
  - Increase oxygen availability and delivery.
  - Contribute to release of glucose from the liver
  - Oppose the action of insulin
Diagnosis of Diabetes
New ADA Diagnostic Criteria: 2010

• HgbA1c ≥ 6.5%
  • Not specified as the preferred test
  • Must use NGSP certified method
  • Fasting blood glucose of 126 mg/dl or higher
    • After 8 hr. fast
  • A 75 gm glucose tolerance test with a two hour glucose value ≥ 200mg/dl.
  • Random glucose ≥ 200 mg/dl with symptoms
Pathophysiology of Diabetes

Type 1 Diabetes

- 5-10% of population
- Beta cells are destroyed by autoimmune response
- Some genetic predisposition but low compared to type 2
- Usually those that develop are young peak age between 12 and 14, but…….
- S/S develop abruptly and are due to high blood glucose which leads to osmotic pressure.
Signs and Symptoms of Type 1

- Weight loss
- Polyphagia
- Polydipsia
- Polyuria
- Lack of energy and sleepiness
- Blurred vision
Pathophysiology of Diabetes

Type 2 Diabetes

- 90% of the population
- More common in those over 40 but…..
- Overweight or obese
- Sedentary
- Strong genetic predisposition
- Greater amongst certain ethnicities, i.e. African Americans, Native Americans, Latinos, and Pacific Islanders
- Women who have a Hx of Gestational Diabetes
Differences between Type 1 and 2

• Type 1 is an autoimmune response and a loss of beta cell function

• Type 2 is a dysfunction in glucose regulation, i.e.
  – Decreased insulin production
  – Increased insulin resistance
Two Theories on How Type 2 Develops

1. Defect in the beta cells causes the pancreas to secrete less insulin, resulting in hyperglycemia.

2. Initial problem is insulin resistance in muscle tissues, fat cells, and the liver. As a result the beta cells increase secretion of insulin to keep blood glucose in normal range.
Signs and Symptoms of Type 2

• Polyphagia
• Polydipsia
• Polyuria
• Blurred vision
• Fatigue
• Frequent infections
• Slow wound healing
Serious Complications of Diabetes
Serious Consequences of Type 1 Ketoacidosis

- hyperglycemia over 300 mg/dL
- low bicarbonate level (<15 mEq/L)
- acidosis (pH <7.30)
- ketonemia and ketonuria
- Nausea/ vomiting
- difficulty breathing (Kussmaul’s breathing)
- fruity odor on breath
- confusion
Serious Consequences of Type 2

Hyperosmolar Hyperglycemic state (HHS)

- Plasma glucose level of 600 mg/dL or greater
- Effective serum osmolality of 320 mOsm/kg or greater
- Profound dehydration (8-12 L) with elevated serum urea nitrogen (BUN)-to-creatinine ratio
- Small ketonuria and absent-to-low ketonemia
- Bicarbonate concentration greater than 15 mEq/L
- Some alteration in consciousness
## Comparison of DKA and HHS

<table>
<thead>
<tr>
<th></th>
<th>DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild</td>
<td>Moderate</td>
</tr>
<tr>
<td>Plasma Glucose (mg/dL)</td>
<td>&gt;250</td>
<td>&gt;250</td>
</tr>
<tr>
<td>Arterial ph</td>
<td>7.25-7.30</td>
<td>7.00-&lt;7/24</td>
</tr>
<tr>
<td>Serum bicarbonate (mEq/L)</td>
<td>15-18</td>
<td>10-&lt;15</td>
</tr>
<tr>
<td>Urine Ketones</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Serum Ketones</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Effective Serum Osmolality</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Anion Gap</td>
<td>&gt;10</td>
<td>&gt;12</td>
</tr>
<tr>
<td>Alteration in Sensorium or mental obdundation</td>
<td>Alert</td>
<td>Alert/drowsy</td>
</tr>
</tbody>
</table>

### Criteria for Resolution of DKA and HHS

<table>
<thead>
<tr>
<th>DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>BG &lt; 200 mg/dL</td>
<td>BG &lt; 300 mg/dL</td>
</tr>
<tr>
<td>Serum bicarb ≥ 18 mEq/L</td>
<td>Improvement in mental status</td>
</tr>
<tr>
<td>Venous pH &gt; 7.3</td>
<td>Serum osmolality &lt; 320 mOso/kg</td>
</tr>
<tr>
<td>Anion gap ≤ 12 mEq/L</td>
<td></td>
</tr>
</tbody>
</table>
Effects of Hypoglycemia

- Early phases alpha cells release glucagon
- Glucagon stimulates hepatocytes
- Glycogen to glucose
- Hepatic gluconeogenesis
- Lead to a rise in blood glucose

Signs and Symptoms of Hypoglycemia

Can vary from patient to patient

- At first patient may feel
  - Nervous
  - Sweaty
  - Shaky or
  - Dizzy
- Later
  - Angry or confused
  - Feel off balance
  - Have difficulty talking
  - Loss of consciousness
Treatment Options for Hypoglycemia

• Rule of 15 s
  – 15 grams of carbohydrate
  – Will raise blood glucose 15 mg/dl
  – In about 15 minutes

• Examples of 15 grams of oral carbohydrate
  – 4 ounces of regular juice or soda
  – 3 to 4 hard candies
  – box of raisins
  – 3-4 teaspoons of sugar
  – 1 teaspoon of jelly
Treatment Options for Hypoglycemia

- If patient unable to swallow and IV present
  - IV 50% dextrose bolus

- If unable to swallow and no IV
  - Inject 1 mg of Glucagon
Prevention of Hypoglycemia

- Insulin or medication dosages
- Blood glucose targets
- Blood glucose monitoring frequency
How do we care for people diagnosed with Diabetes?
# Inpatient Glycemic Goals

<table>
<thead>
<tr>
<th></th>
<th>ICU</th>
<th>Non-ICU Preprandial</th>
<th>Non-ICU Maximal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AACE/ADA</strong></td>
<td>140 mg/dL-180 mg/dL</td>
<td>&lt; 140 mg/dL</td>
<td>&lt; 180 mg/dL</td>
</tr>
</tbody>
</table>

Outpatient Goals of Treatment*

- Blood pressure ≤ 130/80
- LDL < 100 mg/dl (<70 if pre-existing cardiac dx)
- HDL >40 mg/dl in men and > 50 mg/dl in women
- Triglycerides < 150 mg/dl
- HgA1c < 7%
Goals of Treatment
Self Blood Glucose Monitoring
(For Healthy Non-Pregnant Adults)

ADA

- Premeal blood glucose: 90 – 130 mg/dl
- Peak post meal blood glucose: <180 mg/dl
- HbA1c <7%

AACE

- Premeal blood glucose: <110 mg/dl
- Peak 2 hour post meal blood glucose: <140 mg/dl
- HbA1c ≤6.5%

Diabetes Care 2010;33 (supplement): S11
Goals of Treatment SBGM*

Higher target goals for those with:
• Advanced complications
• Life-limiting comorbid illness
• Cognitive or functional impairments
• Hypoglycemic unawareness
• Young children
• Lower goals for pregnant women
Take away from the presentation

• Counterregulatory hormones and the autonomic system affect blood glucose levels.
• Differences in type 1 versus type 2 diabetes.
• Importance of adhering to blood glucose goals to decrease morbidity and mortality.
Thank you
Questions