Diabetes Management in the Inpatient Setting: Beyond Sliding Scale

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Disclosures:
• I have no financial relationships to disclose.
• I will not be discussing off-label/investigational uses.

Objectives:
• Describe the pathophysiology of diabetes and the impact of poor glycemic control on the hospitalized patient.
• Formulate a plan for managing glycemic control in the hospitalized patient, including complex cases.
• Correctly identify and treat diabetic emergencies.
**Physiology Review**

- **Insulin**: Produced by the beta cells of the pancreas, initially as preproinsulin (amino acid chain)
- Proteolysis of preproinsulin to proinsulin
- Proinsulin cleaved to produce C peptide plus A and B chains of insulin (21 amino acids and 30 amino acids, respectively) linked by two disulfide bonds.
  - C peptide can be used as a marker of insulin production
- Insulin needed to maintain glucose homeostasis (hepatic gluconeogenesis vs. peripheral glucose uptake and use)

(Powers, 2013)

**Type 1 Diabetes**

- Auto-immune destruction of insulin-producing beta cells in the pancreas, leading to lack of insulin
- Multiple genes involved including HLA region of chromosome 6 (DR3/DR4 haplotype)
- Possible environment factors [i.e. viruses] as triggers in those who are genetically susceptible?
- Also consider in those receiving immune-modulators

(Powers, 2013)

**Type 2 Diabetes**

- Insulin resistance and impaired insulin secretion
- Typically also with abnormal fat metabolism and increased hepatic glucose production
- Early increased insulin production to compensate for insulin resistance
- This is unsustainable long-term, leading to eventual decreased insulin secretion and finally, failure of beta cells to produce insulin

(Powers, 2013)
Other Classifications

- **Pancreatic insufficiency**: Chronic pancreatitis, pancreatectomy
  - Treat like type 1 diabetes
- **Steroid-induced**: May be temporary hyperglycemia (i.e. immediately following a transplant) or diabetes after long-term use of steroids or some other immunosuppressants (i.e. tacrolimus)
  - Tend to be insulin resistant, more like type 2 diabetes (Moghissi et al., 2009)
- **Stress-induced hyperglycemia**: Temporary hyperglycemia following trauma, significant surgery, need for continuous nutrition

Diabetes Specific H&P

- **Diabetes History**: Date of diagnosis, outpatient provider, meds, blood sugars, A1C
- **Complications?**
- **Lifestyle?**
- **Review of Systems**: Polyuria, polydipsia, blurry vision, unintentional weight loss, neuropathy
- **Past Medical History**: Pancreatitis, other auto-immune diseases, gestational diabetes, PCOS
- **Past Surgical History**: Eye surgeries from retinopathy, transplant, pancreatic surgeries
- **Family History**: Type 1 or type 2 diabetes, gestational diabetes, auto-immune diseases
- **Physical Exam**: Acanthosis nigricans, tactile sensation, foot exam
- **Labs**: Current A1C (accurate?), recent blood sugars, WBC, creatinine/GFR

Diabetes in the Hospital

- Increased rates of hospitalization and increased lengths of stay in those with diabetes vs. without
- Hyperglycemia following cardiac surgery linked to increased risk of infection
- Randomized, controlled trials show decreased morbidity and mortality with intensive insulin therapy in surgical ICU patients
- Hyperglycemia after subarachnoid hemorrhage associated with impaired cognition and neurological deficits at 3 months
- Hyperglycemia associated with acute graft vs. host disease, organ failure, and increased mortality following transplant
  (Moghissi et al., 2009)
At the Same Time...

• While intensive glucose control in a surgical ICU population decreased morbidity and mortality, the same protocol implemented in a medical ICU found a 6-fold increase in severe hypoglycemia (<40 mg/dL), which is an independent risk factor for mortality.
• Normoglycemia in Intensive Care Evaluation – Survival Using Glucose Algorithm Regulation (NICE-SUGAR): Multi-center randomized, controlled trial found increased 90-day mortality, increased mortality from cardiovascular events, and increased severe hypoglycemia from intensive glucose control in intensive care patients (Moghissi et al., 2009).

Where Do We Go From Here?

• American Association of Clinical Endocrinologists (AACE) and American Diabetes Association (ADA) release Consensus Statement on Inpatient Glycemic Control in 2009 (Moghissi et al., 2009).
• Chapter in the annual Diabetes Care supplement of Standards of Medical Care in Diabetes entitled “Diabetes Care in the Hospital” (Cefalu, 2016).

General Guidelines for Inpatient Glycemic Control

• Check an A1C if one not available within the past 3 months (C).
• Start insulin for blood sugars consistently > 180 mg/dL (A).
• Target blood sugars 140-180 mg/dL (may be 110-180 mg/dL if no hypoglycemia) (A if critical, C if not critical).
• Use IV regular insulin if needed based on written/computer protocols (E).
• Basal + bolus correction insulin in patient not eating (A).
• Basal + nutritional + correction insulin in patient who is eating (A).
• Strongly discourage use of sliding scale insulin alone (A).
• Review and adjust insulin doses as needed (C).
• Formulate individualized discharge plan (B). (Cefalu, 2016)
## Only insulin?

<table>
<thead>
<tr>
<th>Medication</th>
<th>Example</th>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin</td>
<td>N/A</td>
<td>Lactic acidosis</td>
</tr>
<tr>
<td>Sulfonylureas</td>
<td>Glyburide, glipizide</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td>Pioglitazone</td>
<td>Volume, heart failure</td>
</tr>
<tr>
<td>GLP-1 receptor agonists</td>
<td>Liraglutide, dulaglutide</td>
<td>Pen, titration, $$$</td>
</tr>
<tr>
<td>SGLT2 inhibitors</td>
<td>Dapagliflozin, canagliflozin</td>
<td>Dehydration, DKA</td>
</tr>
<tr>
<td>DPP-4 inhibitors</td>
<td>Sitagliptin, saxagliptin</td>
<td>Renal function, $$$</td>
</tr>
</tbody>
</table>

(Cefalu, 2016)

## Subcutaneous Insulin

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Onset</th>
<th>Peak (hours)</th>
<th>Duration of Action (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bolus—Rapid Acting</td>
<td>5-15 minutes</td>
<td>0.5-1.5</td>
<td>3-4</td>
</tr>
<tr>
<td>(aspart, glulisine, lispro)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bolus—Regular</td>
<td>30-60 minutes</td>
<td>2-3</td>
<td>4-6</td>
</tr>
<tr>
<td>Basal—NPH (isophane)</td>
<td>1-4 hours</td>
<td>6-10</td>
<td>10-16</td>
</tr>
<tr>
<td>Basal—Long-acting (detemir, glargine)</td>
<td>1-4 hours</td>
<td>No Peak</td>
<td>Up to 24 hours</td>
</tr>
</tbody>
</table>

(Powers, 2013)

## Other Insulin Options

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Onset</th>
<th>Peak (hours)</th>
<th>Duration of Action (hours)</th>
<th>Contra-indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular U-300</td>
<td>30 minutes</td>
<td>8</td>
<td>Up to 24</td>
<td>None, but use with caution</td>
</tr>
<tr>
<td>U-200 Lispro</td>
<td>15 minutes</td>
<td>0.5-1.5</td>
<td>3-5</td>
<td>Pen</td>
</tr>
<tr>
<td>U-300 Glargine</td>
<td>6 hours</td>
<td>No peak</td>
<td>36 hours</td>
<td>Pen, renal function</td>
</tr>
<tr>
<td>Degludec (U-100 and U-200)</td>
<td>1 hour</td>
<td>9 (Tmax) ½ life of 25 hours</td>
<td>Renal function, NPO, pen</td>
<td></td>
</tr>
<tr>
<td>Pre-mixed (70/30, 75/25, 50/50)</td>
<td>5-60 minutes</td>
<td>Varied, multiple</td>
<td>10-16 hours</td>
<td>Multiple peaks, Unpredictable</td>
</tr>
</tbody>
</table>

(ADA, 2015; FDA, 2015)
Beware of Home Insulin

- What is the patient actually taking at home? Home blood sugars? Diet at home?
- Case 1: 40 year old female admitted s/p ERCP for overnight observation, started on home dose of 25 units of glargine qHS.
  - Result: Blood glucose of 35 mg/dL the following morning. Was actually only taking 20 units of glargine at home, and not everyday due to frequent hypoglycemia.
- Case 2: 61 year old male with pancreatic mass, readmitted for leukocytosis and elevated creatinine (1.93 → 3.5), started on home dose of 56 units of glargine qHS.
  - Result: Blood glucose of 62 mg/dL the following morning. Blood sugars running 80s-low 100s at home.

Avoid Sliding Scale Alone

Weight-based Insulin Dosing:

- Type 1 Diabetes: 0.5 units/kg
- Type 2 Diabetes: 0.5 - 0.8 units/kg
- Elderly/ESRD: 0.3 units/kg
- Result = total daily dose of insulin
- Divide this evenly between 50% basal (long-acting) insulin and 50% bolus (short-acting) insulin
Example:

- 70 kg male on the floor (not in the ICU), admitted with an ischemic foot (Awake, alert, oriented, and eating)
- As patient is eating, time blood sugars checks and insulin with meals (ACHS)
- Type 1 Diabetes: 70 kg x 0.5 = 35 units of insulin total
  - 35 units/2 = 17.5 units of basal insulin and 17.5 units of bolus insulin
  - Patient is eating 3 meals/day, so 17.5 units/3 = 5.833 units basal insulin
  - So: 18 units of basal insulin (detemir/glargine) once a day and 6 units of rapid-acting insulin (aspart, glulisine, or lispro) with each meal

Example (Continued):

- Type 2 Diabetes: 70 kg x 0.8 = 56 units of insulin total
  - 56 units/2 = 28 units of basal insulin and 28 units of bolus insulin
  - Patient is eating 3 meals/day, so 28 units/3 = 9.333 units bolus per meal
  - So: 28 units of basal insulin (detemir/glargine) once a day and 9 units of rapid-acting insulin (aspart, glulisine, or lispro) with each meal
- ESRD/Elderly: 70 kg x 0.3 = 21 units of insulin total
  - 21 units/2 = 10.5 units of basal insulin and 10.5 units of bolus insulin
  - Patient is eating 3 meals/day, so 10.5 units/3 = 3.5 units bolus per meal
  - So: 10 units of basal insulin (detemir/glargine) once a day and 3 units of rapid-acting insulin (aspart, glulisine, or lispro) with each meal

Meal-Dependent Insulin

- Rapid-acting, bolus insulin (aspart, glulisine, or lispro)
- Dose rapid-acting insulin based on the amount eaten
- If patient or RN can count carbohydrates, 1 unit for every X grams of carbohydrates
- Otherwise, estimate:
  - Example: 18 units of basal insulin and 6 units of rapid-acting bolus insulin with meals
    - 6 units if eats > 50% of meal
    - 3 units if eats 25-50% of meal
    - 0 units if NPO or eating < 25%
  - Given short onset of action, can dose rapid-acting insulin with or immediately after the meal (ideal if not sure how much patient will eat) (Cefalu, 2016)
Sliding Scale!

- Sliding Scale = Correction Dose
  - Additional short/rapid-acting insulin given with scheduled doses to treat elevated blood sugars (Moghissi et al., 2009)
- Yes, you will have 2 orders for rapid-acting insulin (meal-dependent bolus and correction)
  - Unless in patients who are not eating or are just receiving clear liquids
- Serves as a guide for increasing insulin doses if continued hyperglycemia
- Beware of aggressive “high dose” sliding scales—risk of hypoglycemia, especially in the elderly, ESRD, or those with a low weight

Making Adjustments

- Look for a pattern—do not react to one high blood sugar
- Did anything change?: Steroids, nutrition, infection, renal function, IV fluids
- Talk to the patient and RN: compare food and insulin doses, snacking?
- Fix lows before fixing highs
- Decrease doses to correct low
- Increase everything if high across the board
- Adjust meal-dependent rapid-acting (bolus) insulin for high blood sugars during the day while eating
- Adjust basal, long-acting insulin to correct fasting blood sugars
Hypoglycemia

- Give 15 grams of carbohydrates (or D50/glucagon) and re-check blood sugar in 15 minutes
- Fix lows before fixing highs
- Decrease insulin doses by 20% to correct low
- Example: Blood sugar of 64 in a patient eating well, receiving 18 units of detemir (basal insulin) qAM and 6 units of rapid-acting (bolus) insulin with each meal.
  - Total of 36 units, decreased by 20% = 28.8 units total = 14 units of basal insulin qAM and 4-5 units of rapid-acting insulin bolus with meals
- Beware of holding all insulin for a low: Risk of DKA in Type 1 Diabetes
- Sulfonylurea?

Hyperglycemia

- Increase total daily insulin requirements by ~10-20%
- Example: Patient receiving 18 units of detemir (basal) insulin qHS and 6 units of rapid-acting (bolus) insulin with each meal.
- Blood sugars all > 180 mg/dL

<table>
<thead>
<tr>
<th>Time</th>
<th>Scheduled Insulin</th>
<th>Correction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>6 units rapid-acting</td>
<td>4 units rapid-acting</td>
</tr>
<tr>
<td>Lunch</td>
<td>6 units rapid-acting</td>
<td>2 units rapid-acting</td>
</tr>
<tr>
<td>Dinner</td>
<td>6 units rapid-acting</td>
<td>4 units rapid-acting</td>
</tr>
<tr>
<td>Bedtime</td>
<td>18 units basal</td>
<td>2 units rapid-acting</td>
</tr>
</tbody>
</table>

Hyperglycemia (continued)

- Patient has received a total of 48 units of insulin over the past 24 hours
- Increase 48 units by 20%
  - 48 units x 1.2 = 57.6 units total (new total daily dose)
  - 57.6/2 = 28.8 units basal and 28.8 units total bolus
  - 28.8 units total bolus/3 meals = 9.6 units bolus per meal
- New insulin regimen: 28 units of basal long-acting insulin (detemir or glargine) and 9-10 units of bolus, rapid-acting insulin (aspart, glulisine, or lispro) with each meal.
Steroids

- Often need to increase scheduled insulin doses by 20-30%, especially bolus doses

Graph courtesy of Dr. Kelli Dunn, Carolinas Medical Center, 2015

Continuous Nutrition

- Tube Feeds or TPN
- Carbohydrate content of tube feeds/TPN?
- Still use basal, bolus, and correction insulin
- As patient is not eating, blood sugar checks q6 hours
- Basal: 1-2x daily detemir or glargine vs. smaller, more frequent dosing of NPH
- Bolus: Rapid-acting (3-4 hour duration) vs. regular insulin (4-6 hour duration)
- Sliding Scale: Same insulin as bolus
- If high risk for changes to nutrition, consider smaller, more frequent doses using insulin of shorter durations
- 2/3 NPH and 1/3 regular insulin q6-8 hours
- Can put regular insulin in TPN bag
- Caveat: If you over do it, may need to temporarily stop TPN

(Cataldo, 2016)

Continuous Nutrition

Ideal  Options:

Graphs courtesy of Dr. Kelli Dunn, Carolinas Medical Center, 2015
Continuous Nutrition

• Still use weight-based dosing
• Example: 70 kg female with no prior history of diabetes, now s/p total pancreatectomy on continuous tube feeds
  - 70 kg x 0.5 units/kg = 35 units total
  - Insulin q6 hours = 35 units/4 doses = 8.75 units every 6 hours
  - 2/3 NPH and 1/3 Regular insulin q6 hours = 6 units of NPH and 3 units of regular insulin every 6 hours
• To allow for nutritional changes/prevent lows:
  - Hold Regular insulin for BG < 120 mg/dL
  - Hold both insulin doses if tube feeds stopped
  - Consider D10 IV fluids if received insulin before nutrition stopped (D10 at 100 ml/hr = 240 grams of dextrose)

Adjusting Insulin for Nutritional Changes:

• Proportional changes: Double the carbs, double the insulin
  - Total insulin requirement of 50 units for 100 grams of carbohydrates?
  - Total insulin requirement of 100 units for 200 grams of carbohydrates
• Nocturnal tube feeds:
  - Use total insulin needs for continuous tube feeds as a guide
  - ≤ 12-14 hours of tube feeds: 1 dose of 2/3 NPH and 1/3 regular insulin at start of tube feeds
  - ≥ 14-16 hours of tube feeds: Dose of NPH and regular insulin at start of tube feeds and again in the middle of tube feeds

Critically Ill

• IV regular insulin is best in the critical care setting
• Use validated protocols (written vs. computer) to adjust insulin (Cefalu, 2016)
• IV insulin infusion rates (units/hr) allow for good estimate of subcutaneous insulin doses when patient is stable enough to be transitioned off IV insulin
• When transitioning from IV regular insulin to subcutaneous insulin, be sure to overlap subcutaneous basal insulin and IV regular insulin by at least 1 hour (Cefalu, 2016; Powers, 2013)
  - IV regular insulin has a short half-life while basal (long-acting) insulins generally take at least 1 hour to start working
Other Considerations:

• **Insulin pumps:**
  - As long as patient is alert, oriented, and using pump safely, allow patient to use their pump (Moghissi, 2009)
  - Discontinue all subcutaneous insulin injections while patient is using pump
  - Document settings and boluses
  - If at all possible, keep insulin pump on in the OR (or have an alternate plan in place for insulin administration)

• **U-500 Concentrated Regular Insulin:**
  - Obtain detailed information of how much patient uses at home (mL vs. units, type of syringe used)
  - May be able to use more conventional basal/bolus insulin regimen in the hospital, particularly if patient will be NPO
  - Cut home dose in half if it must be used in the hospital
  - If possible, consider endocrinology consult for insulin pumps and U-500

Discharge Recommendations

• Detailed, structured, and individualized discharge plan for outpatient diabetes management can reduce re-admission rates
• If good A1C on oral medications at home, can generally resume all oral medications at discharge
• High A1C on only oral medications may need insulin upon discharge
• In patients already on insulin at home, discharge on inpatient insulin doses
• Outpatient follow-up recommended within 1 month, sooner if on insulin or safety concerns

(Cefalu, 2016)

Diabetic Emergencies
**Diabetic Ketoacidosis (DKA)**

- Typically type 1, but may see ketosis-prone type 2 diabetes
- Lack of insulin = inability to use carbohydrates for energy, increased hepatic metabolism of fatty acids, resulting in ketones
- **Need all 3:** Diabetes/hyperglycemia (may be normal, near-normal in pregnancy), serum ketones, and metabolic acidosis
- **Risk factors:** New diagnosis of diabetes, lack of basal (long-acting) insulin, relative lack of insulin (i.e., infection, illness, steroids), insulin pump malfunction, cocaine use
- **Presentation:** Hyperglycemia, polypnea, polydipsia, lethargy, obtundation, nausea, vomiting, abdominal pain
- **Exam:** Poor skin turgor, tachycardia, dry oral mucosa, fruity odor (exhaled acetone), Kussmaul respirations
- **Labs:** Hyperglycemia, serum ketones present, elevated anion gap, low CO₂, metabolic acidosis, hyponatremia, elevated serum potassium, elevated BUN and creatinine

(Kitabchi, Hirsch, & Emmett, 2014; Powers, 2013)

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**DKA Treatment**

- **Treatment goals:** Correct volume, hyperglycemia, and electrolytes/ketosis
- **IV fluids** (usually start with NS for first 1-3 hours, then may need to change based on hydration status, electrolytes, and urine output)
  - Add dextrose to fluids once BG ~200-250 mg/dL
- **IV regular insulin** (caution: If potassium < 3.3 mEq/L, may need to correct potassium before starting insulin)
- Risk of cerebral edema from rapid correction of blood sugar and electrolytes almost exclusively seen in children
- **Bicarb use controversial** (UpToDate: consider if arterial pH < 6.9)
- **Treat/fix underlying cause**
- **Resolution based on labs/anion gap closing rather than blood sugar**
- Transition to subcutaneous basal insulin (risk of going back into DKA if not overlapping IV and subcutaneous basal insulin—be sure basal insulin ordered at proper time)

(Kitabchi, Hirsch, & Emmett, 2015)

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**Hyperosmolar Hyperglycemic State**

- Similar to DKA, but typically seen in type 2 diabetes
- No/minimal serum ketosis, no metabolic acidosis
- Blood glucose often > 1,000 mg/dL
- Plasma osmolality > 320 mosm/kg
- Often with neurologic complications, including coma
- **Treatment:** Aggressive fluid replacement and IV regular insulin
- Resolution once patient is alert and plasma osmolality < 315 mosm/kg
- Transition to subcutaneous insulin with overlap of IV insulin

(Kitabchi et al., 2014; 2015)

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In Conclusion:

- Don't be afraid to use insulin
- But, when using insulin, use more than just sliding scale
- Avoid oral medications
- Target 140-180
- Fix lows first, then highs
- Overlap IV insulin and basal insulin by 1 hour
- Diabetic Emergencies (DKA, Hyperosmolar) = IV fluids and insulin

References


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Questions?

Thank You