49 yo Woman with Hypoglycemia

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Dr. Upala does not have any relevant financial relationships with any commercial interests
Learning Objectives

• Understand the importance of the critical sample
• Review hypoglycemia in non-DM
• Review cases series of hypoglycemia
HPI

• 49 yo non-diabetic female
• Had episodes of chest pain, fatigue, palpitation, diaphoresis, tingling sensation (happened 10 hours after her last meal with chicken salad and cocktail)
• Loss of consciousness at home
• EMS with BG 36-> given D50x1 amp
• Mental status improved with dextrose
• No history of previous episodes, LOC or seizure
• No history of diabetes, but mother has T2 diabetes (on insulin)
• No autoimmune disease in family
HPI

• At ED, she started to be confused, sweating and agitated. BG was 35 and started on D10W drip, repeat BG was 76
• Reports drinking alcohol occasionally but denies illicit drugs
• Denies any diarrhea, poor appetite, nausea, vomiting, diarrhea, polyuria, polydipsia, exogenous steroids, insulin or DM medication use
• Admitted to ICU
Other History

• Past Medical History
  – Inflammatory polyarthritis
  – Arthritis
  – Habitual alcohol use
  – HPV
  – Essential hypertension

• Past Surgical history
  – Cesarean section

• Family History
  – T2DM: mother

• Allergy
  – No Known Allergies

• Social History
  – Smoking 1ppd for 40 years
  – Alcohol 3 beers a day
  – Denies illicit drugs
  – Works as a social worker

• Medication
  – Humira subq every 14 days
  – Hydrocodone-acetaminophen (NORCO) 5-325 mg PO tab
Review of Systems

- Constitutional: **+Fatigue**. No fevers, night sweats, appetite change, unexpected weight change
- HEENT: No photophobia, blurred vision, pain, hearing loss, difficulty swallowing, thirst, hoarseness
- Resp: No cough, dyspnea, increased WOB
- CV: **+ CP, diaphoretic, palpitation**, No DOE, orthopnea, PND, palpitations, LE edema
- GI: No abdominal pain, nausea, vomiting, diarrhea, constipation
- GU: No dysuria, urgency, polyuria, hematuria
- MSK: **+ Myalgias, joint pain, back pain**
- Neuro: +Syncope, No numbness, paresthesias, headaches, tremors, memory problems, seizures
- Heme: No adenopathy or easy bruising/bleeding
- Endo: **+ Hypoglycemia**. No heat or cold intolerance, dry skin, dry hair, hair loss
- Derm: No rashes, ulcers, abdominal striae, hirsutism, acne
- Psych: No mood changes, sleep disturbance, anxiety, depression
Physical Exam
(when I saw her, with normal BG)

- Vitals: BP 144/95 | Pulse 76 | Temp 36.8 °C (98.2 °F) | Resp 15 | Ht 157.5 cm (5' 2") | Wt 59 kg (130 lb) | SpO2 100% | BMI 23.78 kg/m²

- General: No apparent distress. Appears stated age
- HEENT: No pharyngeal erythema. PERRL, EOMI
- Neck: No neck tenderness. No thyromegaly or thyroid nodules appreciated
- Cardiovascular: regular rate and rhythm. No peripheral edema
- Pulmonary/Chest: clear to auscultation bilaterally
- Gastrointestinal: soft, non-tender, non-distended. No rebound or guarding
- Musculoskeletal: normal range of motion of joints
- Neurological: Alert & oriented, no focal deficits
- Lymph: No cervical, supraclavicular, lymphadenopathy
- Skin: No apparent bald spots. No acanthosis nigricans. No illicit drug mark
- Psychiatric: normal mood, thought content, appropriate
Lab

- BG 35, Insulin 7.3 (2.6-24.9), Proinsulin 78 (3-20), C-peptide 0.63 (0.3-2.35), Cortisol 17.5
- A1c 4.9, Lactate 1.6, Beta OH butyrate 5.8 (<0.3)
- Positive alcohol and cocaine

- Na 139, K 3.8, Cl 102, HCO3 10, BUN 6, Cr 0.6
- Ca 9, Alb 4.3
- CBC: Hct 36.4/WBC 6.7/Platelet 251
- AST 53, ALT 14, Alkphos 90, TB 0.3
- Negative tylenol, salicylate
Differential Diagnosis of Hypoglycemia

- Well appearance
  - Endogenous hyperinsulinism
    - Insulinoma
    - Functional B-cell disorder (nesidioblastosis, post RYBG)
    - Insulin antibody-mediated
    - Insulin secretagogue
  - Accidental/surreptitious

- Ill or medicated
  - Drugs-insulin, insulin secretagogues, ETOH
  - Critical illness-hepatic/renal/cardiac failure, sepsis
  - Hormone deficiency-cortisol, glucagon
  - Non islet cell tumor
Critical Sample

- Distinguish hypoglycemia caused by endogenous (or exogenous) insulin use from other mechanisms
- Plasma glucose, insulin c-peptide, proinsulin, B-hydroxybutyrate, cortisol, insulin antibodies, sulfonylurea screen
Algorithm Approach to Patient

Suspected/Documented Hypoglycemia

Diabetes
- Treated with
  - Insulin
  - Sulfonylurea
  - Other secretagogue
  - Adjust regimen
  - Document improvement and monitor

No diabetes
- Clinical clues
  - Drugs
  - Organ failure
  - Sepsis
  - Hormone deficiencies
  - Non-β-cell tumor
  - Previous gastric surgery
  - Provide adequate glucose, treat underlying cause

- Apparently healthy
  - Fasting glucose
    - <55 mg/dL
      - <55 mg/dL
        - History
          - Strong
            - Extended fast
            - Glucose
              - ↑ Insulin, Whipple’s triad
                - ↑ C-peptide
                  - Ab+ Insulinoma
                  - Autoimmune
                  - Sulfonylurea
                - Likely factitious
              - ↓ C-peptide
                - Exogenous insulin
                - Whipple’s triad
                - Reactive hypoglycemia
                - Hypoglycemia excluded
            - Mixed meal
          - Weak
        - ≥55 mg/dL
          - No Whipple’s triad
          - Hypoglycemia excluded
    - ≥55 mg/dL

Critical diagnostic findings when glucose < 55 mg/dL
- Insulin 3 U/mL (18 pmol/L)
- C-peptide 0.6 ng/mL
- Proinsulin 5 pmol/L
Critical sample

- BG 35
- Insulin 7.3 (2.6-24.9)
- Proinsulin 78 (3-20)
- C-peptide 0.63 (0.3-2.35)
- Cortisol 17.5
- A1c 4.9
- Lactate 1.6
- Beta OH butyrate 5.8 (<0.3)
- Sulfonylurea screen: + Glyburide
# Hypoglycemia: Interpretation of Laboratory Tests

<table>
<thead>
<tr>
<th>Symptoms, signs, or both</th>
<th>Glucose (mg/dL)/(mmol/L)</th>
<th>Insulin (microU/mL)/(pmol/L)</th>
<th>C-peptide (nmol/L)/(ng/mL)</th>
<th>Proinsulin (pmol/L)</th>
<th>Beta-hydroxybutyrate (mmol/L)</th>
<th>Glucose increase after glucagon (mg/dL)/(mmol/L)</th>
<th>Circulating oral hypoglycemic agent</th>
<th>Antibody to insulin</th>
<th>Diagnostic interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>&lt;55/3</td>
<td>&lt;3/20.8</td>
<td>&lt;0.2/0.6</td>
<td>&lt;5</td>
<td>&gt;2.7</td>
<td>&lt;25/1.4</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>&gt;&gt;3</td>
<td>&lt;0.2</td>
<td>&lt;5</td>
<td>≤2.7</td>
<td>&gt;25</td>
<td>No</td>
<td>Neg (Pos)</td>
<td>Exogenous insulin</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>≥3</td>
<td>≥0.2</td>
<td>≥5</td>
<td>≤2.7</td>
<td>≤25</td>
<td>No</td>
<td>Neg</td>
<td>Insulinoma, NIPHS, PGBH</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>≥3</td>
<td>≥0.2</td>
<td>≥5</td>
<td>≤2.7</td>
<td>≤25</td>
<td>Yes</td>
<td>Neg</td>
<td>Oral hypoglycemic agent</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>&gt;&gt;3</td>
<td>&gt;&gt;0.2^§</td>
<td>&gt;&gt;5^†</td>
<td>≤2.7</td>
<td>&gt;25</td>
<td>No</td>
<td>Pos</td>
<td>Insulin autoimmune</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>&lt;3</td>
<td>&lt;0.2</td>
<td>≤5</td>
<td>≥2.7</td>
<td>≥25</td>
<td>No</td>
<td>Neg</td>
<td>IGF^§</td>
</tr>
<tr>
<td>Yes</td>
<td>&lt;55</td>
<td>&lt;3</td>
<td>&lt;0.2</td>
<td>&lt;5</td>
<td>&gt;2.7</td>
<td>≤25</td>
<td>No</td>
<td>Neg</td>
<td>Not insulin (or IGF)-mediated</td>
</tr>
</tbody>
</table>

Neg: negative; Pos: positive; NIPHS: non-insulinoma pancreaticogenic hypoglycemia syndrome; PGBH: post-gastric bypass hypoglycemia; IGF: insulin-like growth factor.

* Patterns of findings during fasting or after a mixed meal in normal individuals with no symptoms or signs despite relatively low plasma glucose concentrations (i.e., Whipple’s triad not documented) and in individuals with hyperinsulinemic (or IGF-mediated) hypoglycemia or hypoglycemia caused by other mechanisms.

^§ Free C-peptide and proinsulin concentrations are low.

† Increased pro-IGF-2, free IGF-2, IGF-2/IGF-1 ratio.
Whipple’s triad

- Signs & Symptoms of Hypoglycemia
- Resolution of Symptoms once Glucose Level Rises
- Low Plasma Glucose Level
  - Non-diabetic < 54mg/dL (3mmol/L)
  - Diabetic < 63mg/dL (3.5mmol/L)

Hypoglycemia

Health Hype
www.health hype.com
Sulfonylurea

• The first reported case of factitious hypoglycemia: chlorpropamide by a non-dm patient
• Hypoglycemia in an older person: mix-up in medication
• All these agents peak up to 8 h and may last >24 h and hypoglycemia is seen many hours after the dose.
### Table 5.1 Characteristics of sulfonylureas and meglitinides

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Total daily dose range (mg)</th>
<th>Frequency of administration</th>
<th>Half-life (h)</th>
<th>Duration of action (h)</th>
<th>Metabolism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First generation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolbutamide</td>
<td>500–2000</td>
<td>3 times daily</td>
<td>4.5–6.5</td>
<td>6–12</td>
<td>Mainly hepatic, &lt;20% excreted unchanged in urine</td>
</tr>
<tr>
<td>Chlorpropamide</td>
<td>125–500</td>
<td>Once daily</td>
<td>36</td>
<td>60</td>
<td>Mainly renal, ~20% hepatic</td>
</tr>
<tr>
<td>Tolazamide</td>
<td>100–1000</td>
<td>Twice daily</td>
<td>7</td>
<td>12–24</td>
<td>Renal, hepatic</td>
</tr>
<tr>
<td><strong>Second generation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glibenclamide (glyburide)</td>
<td>1.25–20*</td>
<td>2 or 3 times daily</td>
<td>Bliphasic (4 and 10 h)</td>
<td>12–24</td>
<td>Hepatic (~100%); metabolites excreted in bile (50%) and urine (50%)</td>
</tr>
<tr>
<td>Gilipizide</td>
<td>2.5–40</td>
<td>Twice daily</td>
<td>2–4</td>
<td>12–24</td>
<td>Hepatic; &lt;10% excreted unchanged in urine</td>
</tr>
<tr>
<td>Gilipizide extended release</td>
<td>2.5–20</td>
<td>Once daily</td>
<td>&gt;12</td>
<td>24</td>
<td>Hepatic; &lt;10% excreted unchanged in urine</td>
</tr>
<tr>
<td>Gliclazide</td>
<td>40–320</td>
<td>Twice daily</td>
<td>10–12</td>
<td>12–16</td>
<td>Hepatic; &lt;5% excreted unchanged in urine</td>
</tr>
<tr>
<td>Gliclazide modified release (MR)</td>
<td>30–120</td>
<td>Once daily</td>
<td>12–20</td>
<td>24</td>
<td>Hepatic; &lt;5% excreted unchanged in urine</td>
</tr>
<tr>
<td><strong>Third generation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glimipiride</td>
<td>1–8*</td>
<td>Once daily</td>
<td>5–8</td>
<td>16–24</td>
<td>Hepatic; inactive metabolites excreted in urine (60%) and feces (40%)</td>
</tr>
<tr>
<td><strong>Meglitinides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Repaglinide</td>
<td>0.5–16</td>
<td>3–4 times daily</td>
<td>1</td>
<td>2–6</td>
<td>Hepatic</td>
</tr>
<tr>
<td>Nateglinide</td>
<td>60–540</td>
<td>3 times daily</td>
<td>0.5–1.9</td>
<td>2–6</td>
<td>Hepatic; excreted in urine</td>
</tr>
</tbody>
</table>

*In Europe the maximum recommended daily dose of glibenclamide (glyburide) is 15mg and of glibencliride 6mg.
Mechanism of action
Sulfonylurea

• Herbal products contaminated with sulfonylureas
• According to WHO estimates up to 1% of drugs in the industrialized world are counterfeit. Globally it is an even bigger problem - up to 10% of drugs worldwide are thought to be counterfeit

Power 1
Walnut

- 150 nondiabetic patients
- 10 died (men aged 35 to 84)

Power 1 Walnut

Contamination of Sexual-Enhancement Drugs. High-performance liquid chromatograms of samples of a drug manufactured in January 2008 (Panel A) and before January 2008 (Panel B) contaminated with glyburide from 13 to 100 mg per tablet.

Nangen Zengzhangsu

One capsule of “Nangen Zengzhangsu” contained 7.7 mg of sildenafil citrate and 122.7 mg of glibenclamide

144 patients, 3 died, 1 vegetative state, 1 cognitive impairment
Cialis

One tablet of counterfeit Cialis contained 152.8 mg of glibenclamide and 0.5 mg of sildenafil

Chaubey et al, Medical Journal of Australia, 2010
Clinical Question

• No sulfonylurea drugs were detected when the patient’s blood was tested. What should we do?
An Unusual Cause of Hyperinsulinemic Hypoglycemia Syndrome

• 47-year-old woman was admitted comatose in the overnight fasted state
• No significant past medical history, no medication
• She was a trained physiotherapist and married to a local consultant psychiatrist in New Zealand
• Denied any access to hypoglycemic medication
An Unusual Cause of Hyperinsulinemic Hypoglycemia Syndrome

**Table 1: Chronology of events**

<table>
<thead>
<tr>
<th>Day</th>
<th>Event</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>First admission—hypoglycemic coma</td>
<td>Plasma venous glucose 1.9 mmol/l</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Plasma insulin 105 pmol/l³</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Plasma C-peptide 1060 pmol/l²</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Plasma sulphonylurea screen negative</td>
</tr>
<tr>
<td>5</td>
<td>Discharged to care of spouse</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Second admission—hypoglycaemic coma</td>
<td>Insulinoma suspected</td>
</tr>
<tr>
<td>16</td>
<td>Calcium stimulation test (see Fig. 1)</td>
<td></td>
</tr>
</tbody>
</table>

Plasma insulin > 36 pmol/l and C-Peptide > 200 pmol/l in the presence of hypoglycaemia (plasma glucose < 2.5 mmol/l) is indicative of endogenous hyperinsulinemia.

- **1st admission 5 days:** intravenous 10% dextrose
  - Sulfonylurea screen was reported as negative and drug-induced hypoglycemia was felt to have been excluded
- **2nd admission 29 days:** prolonged intravenous dextrose, normal growth hormone and cortisol
  - Normal CT scan of the pancreas
  - An insulinoma was suspected as the most likely diagnosis and, as hypoglycemia had been documented on two separate occasions, a formal fast was deemed unnecessary
  - Endocrine consult

An Unusual Cause of Hyperinsulinemic Hypoglycemia Syndrome

- Selective arterial calcium stimulation test
- Positive result (a doubling of hepatic venous insulin concentration in response to calcium) was detected in all arterial territories

- Generalized pattern of response with calcium stimulation, plan for distal pancreatectomy if no insulinoma was identified
- Intra-operative ultrasound, no insulinoma was detected, and a two-thirds distal pancreatectomy was performed

### An Unusual Cause of Hyperinsulinemic Hypoglycemia Syndrome

<table>
<thead>
<tr>
<th>No.</th>
<th>Event Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>Calcium stimulation test (see Fig. 1)</td>
</tr>
<tr>
<td>28</td>
<td>Insulinoma suspected</td>
</tr>
<tr>
<td>35</td>
<td>Subtotal pancreatectomy</td>
</tr>
<tr>
<td>36</td>
<td>Normal histology (no insulinoma)</td>
</tr>
<tr>
<td>37</td>
<td>Capillary BSL 2.1 mmol/l</td>
</tr>
<tr>
<td>39</td>
<td>36-h supervised fast</td>
</tr>
<tr>
<td>43</td>
<td>Discharged to care of spouse</td>
</tr>
<tr>
<td>46</td>
<td>Euglycaemic throughout</td>
</tr>
<tr>
<td>48</td>
<td>Plasma venous glucose 1.9 mmol/l</td>
</tr>
<tr>
<td>49</td>
<td>Glibenclamide and Glipizide present in same sample</td>
</tr>
<tr>
<td>50</td>
<td>Patient found dead 06.15 h</td>
</tr>
<tr>
<td>51</td>
<td>Autopsy toxicology positive for Glibenclamide, Glipizide and Metformin</td>
</tr>
</tbody>
</table>

- POD #9 had hypoglycemia. Despite patient reluctance, a formal supervised fast was commenced but was discontinued at 36 h because of co-morbidity associated with recent abdominal surgery.
- Euglycemia until discharge on POD#12.
- Multiple hypoglycemia at home, patient passed away 12 days after discharge.

Plasma insulin $> 36$ pmol/l and C-Peptide $> 200$ pmol/l in the presence of hypoglycaemia (plasma glucose $< 2.5$ mmol/l) is indicative of endogenous hyperinsulinemia.

*Plasma venous glucose 1.9 mmol/l, Glibenclamide and Glipizide present in same sample.*

*Autopsy toxicology positive for Glibenclamide, Glipizide and Metformin.*

An Unusual Cause of Hyperinsulinemic Hypoglycemia Syndrome

### Table 1: Chronology of events

<table>
<thead>
<tr>
<th>Day</th>
<th>Event</th>
<th>False prescriptions*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>First admission—hypoglycemic coma</td>
<td>Glibenclamide 225 mg</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Discharged to care of spouse</td>
<td>Glibenclamide 450 mg</td>
</tr>
<tr>
<td>14</td>
<td>Second admission—hypoglycaemic coma</td>
<td>Glibenclamide 600 mg</td>
</tr>
<tr>
<td>16</td>
<td>Calcium stimulation test (see Fig. 1)</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Subtotal pancreatectomy</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>Capillary BSL 2.1 mmol/l</td>
<td></td>
</tr>
<tr>
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<td>36-h supervised fast</td>
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<tr>
<td>39</td>
<td>Discharged to care of spouse</td>
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<tr>
<td>43</td>
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<td>45</td>
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<td></td>
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<tr>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>51</td>
<td>Patient found dead 06.15 h</td>
<td></td>
</tr>
</tbody>
</table>

*Only hypoglycaemic drugs are listed. Numerous other false scripts were written (commencing day −48) for a variety of sedatives, e.g. clonazepam, throughout the illness.
Clinical Question

- Sulfonylurea may have been undetectable for the following reasons:
  - Not sensitive
  - Hypoglycemic effects of sulfonylureas may persist after the elimination of the drug
  - Blood sample may have been sent after the initial presentation
Starvation Ketosis

**Starvation State**

- Low glucose levels in blood
- Glucagon ↑
- Insulin ↓

**Fatty Acid Oxidation**

- (Occurs in liver)
- Fatty Acids → Acetyl CoA → Kreb’s Cycle

- 1-2 days of fasting!
- ATP ← ETC

**Starvation Ketoacidosis**

- Blood glucose level is decreased
- \([\text{INSULIN}] / [\text{GLUCAGON}] \downarrow\)

- Excess mobilization of fatty acids

- Hyperglucagonemia alters hepatic metabolism to favour ketone body formation, through activation of the enzyme carnitine palmitoyltransferase I.

- Excess β-oxidation of fatty acids in the hepatocytes

- Excess ketone body formation
Alcohol Induced Hypoglycemia

- Ethanol intake
- NAD
- NADH
- Acetate

NADH >> NAD
- Gluconeogenesis is inhibited
- Decreased released of alanine from muscle
- Decrease uptake of gluconeogenic substrate by the liver

Hypoglycemia
Drug induced Hypoglycemia

<table>
<thead>
<tr>
<th>Moderate quality of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cibenzoline</td>
</tr>
<tr>
<td>Gatifloxacin</td>
</tr>
<tr>
<td>Pentamidine</td>
</tr>
<tr>
<td>Quinine</td>
</tr>
<tr>
<td>Indomethacin</td>
</tr>
<tr>
<td>Glucagon (during endoscopy)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Low quality of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroquine oxalate sulfonamide</td>
</tr>
<tr>
<td>Artesunate/arterisin/artemether</td>
</tr>
<tr>
<td>IGF-1</td>
</tr>
<tr>
<td>Lithium</td>
</tr>
<tr>
<td>Propoxyphene/dextropropoxyphene</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Very low quality of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs with &gt;25 cases of hypoglycemia identified</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
</tr>
<tr>
<td>Angiotensin receptor antagonists</td>
</tr>
<tr>
<td>Beta-adrenergic receptor antagonists</td>
</tr>
<tr>
<td>Levofoxacin</td>
</tr>
<tr>
<td>Mifepristone</td>
</tr>
<tr>
<td>Disopramide</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
</tr>
<tr>
<td>Heparin</td>
</tr>
<tr>
<td>6-mercaptopurine</td>
</tr>
</tbody>
</table>

IGF-1: insulin-like growth factor 1.
Assessment and Plan

• Improved after dextrose solution, no octreotide or glucagon
• Diagnosis: hypoglycemia induced by glyburide, worsening with alcohol intake with possible starvation ketosis
Take Home Messages

• Patients with insulinoma and insulin secretagogue-induced hypoglycemia can have plasma insulin, C-peptide, and proinsulin values above or within the normal overnight fasting range with low plasma glucose

• Many techniques for detection of first-generation sulfonylureas do not detect second-generation sulfonylureas or meglitinides
References

49 yo Woman with Hypoglycemia

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Dr. Upala does not have any relevant financial relationships with any commercial interests