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**Moxifloxacin: A Unique Cause of Severe Hypoglycemia**

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**Introduction:**
As a class, quinolone antibiotics have been rarely associated with hypoglycemia in non-diabetic patients and rare reversible episodes of hypoglycemia documented in the literature typically occur within the duration of action of the medication. In this case, however, we believe the effect of the medication persisted much longer.

**Case Description:**
A thin 61-year-old man with a past medical history significant for schizoaffective disorder, bipolar disorder and recent episode of community acquired pneumonia (CAP) presented with shaking and the time of the fall was found to be 26 mg/dL. The patient was recently treated for CAP with sulfonylurea level and hypoglycemia documented in the literature typically occur within the duration of action of the medication. In this case, however, we believe the effect of the medication persisted much longer.

As a class, quinolone antibiotics have been rarely associated with hypoglycemia in non-diabetic patients treated with levofloxacin for CAP found only 2 suffered an adverse hypoglycemic event. Moxifloxacin is much less commonly cited as a cause of hypoglycemia among non-diabetic patients. Additionally, a perplexing aspect of this case was the duration of time from discontinuation of the offending medication to appearance of severe, symptomatic hypoglycemia. The patient reported decreased oral intake the morning preceding presentation, which may have worsened any persistent underlying hypoglycemia and resulted in altered mental status and fall. Advanced age and reduced renal function are thought to be risk factors for the development of hypoglycemia among non-diabetic patients treated with fluoroquinolones. This case adds to the base of literature regarding severe hypoglycemia resulting from moxifloxacin administration and brings awareness to the rare, but potentially fatal side effect of this commonly used class of medications.

**Blood Glucose Measurements**
- 6 a.m.: 26 mg/dL
- DSW stopped
- Blood glucose was 80 mg/dL before Lunch
- Oral glucose and food were given
- 8 a.m.: 110 mg/dL

**Blood Glucose Workup**
- **Whipple’s Triad Present?**
  - **YES**
  - **NO**
- **Mixed Meal Test**
  - **YES**
  - **NO**
- **Hypoglycemia Workup**
  - Symptoms + low blood glucose + half of symptoms with increased blood glucose
  - Observed 72 hour fact

**Proposed Mechanism of Fluoroquinolone-Induced Hypoglycemia:**
Like sulfonylureas, the medications disrupt the function of the ATP-sensitive K+ channel, resulting in increased insulin release by pancreatic beta cells.

**Hypoglycemia Workup**
1. Whipple’s Triad Present?
2. Only Postprandial Hypoglycemia
3. Order the Following as Indicated by Clinical Scenario:
   - Insulin
   - Proinsulin
   - Insulin-Antibody
   - Beta-hydroxybutyrate
   - C-peptide
   - Sulfonylurea & Meglitinide Level

**Results of these studies will help**
- Differentiate between factitious hypoglycemia, autoimmune hypoglycemia, insulinoma and other etiologies

**Comment:**
- Also consider checking TSH & cosyntropin stimulation test to rule out hypoglycemia resulting from hypothyroidism and adrenal insufficiency

**Discussion:**
Upon presentation, the origin of the patient’s hypoglycemia was unclear. He had no significant medical comorbidities, his psychiatric illness had been stable on the same treatment regimen for many years, and he was in an inpatient ward with a very low likelihood of having been given insulin or any other hypoglycemic agent. Omeprazole was the only other medication the patient was receiving associated with hypoglycemia and was discontinued on admission to the medical ward. This was thought not to be the cause of his acute hypoglycemia as he had been taking omeprazole for many years. Fluoroquinolones as a class have been shown to cause hypoglycemia by inappropriate activation of pancreatic beta cells resulting in increased insulin release. Considering the patient’s recent course of moxifloxacin this was thought to be the most likely culprit. Pharmacy and endocrinology services were consulted and both agreed that this was the most likely explanation for the patient’s presenting symptoms.

Among diabetic patients, especially those taking sulfonylureas, dysglycemia is a well-established side effect of fluoroquinolone antibiotics with moxifloxacin being most likely to cause hypoglycemia in these patients. Rarely fluoroquinolones have been shown to cause persistent hypoglycemia in non-diabetic patients. A handful of case reports have been published showing an association between levofloxacin and hypoglycemia. A phase IV study of 1701 patients treated with levofloxacin for CAP found only 2 suffered an adverse hypoglycemic event. Moxifloxacin is much less commonly cited as a cause of hypoglycemia among non-diabetic patients. Additionally, a perplexing aspect of this case was the duration of time from discontinuation of the offending medication to appearance of severe, symptomatic hypoglycemia. The patient reported decreased oral intake the morning preceding presentation, which may have worsened any persistent underlying hypoglycemia and resulted in altered mental status and fall. Advanced age and reduced renal function are thought to be risk factors for the development of hypoglycemia among non-diabetic patients treated with fluoroquinolones. This case adds to the base of literature regarding severe hypoglycemia resulting from moxifloxacin administration and brings awareness to the rare, but potentially fatal side effect of this commonly used class of medications.

**References:**

**Medications:**
- Acetaminophen 500 mg PO PRN
- Clonazepam 0.5 mg PO daily
- Clobazam 400 mg PO daily
- Docupate/Senna 50/8.6 mg PRN
- Finasteride 5 mg PO daily
- Gabapentin 1200 mg PO TID
- Lithium 600 mg PO BID
- Omeprazole 20 mg PO daily
- Tamsulosin 0.8 mg PO daily
- Trazodone 200 mg PO daily

**Hypoglycemia Workup**
- Results of these studies will help
  - Differentiate between factitious hypoglycemia, autoimmune hypoglycemia, insulinoma and other etiologies

**Proposed Mechanism of Fluoroquinolone-Induced Hypoglycemia:**
Like sulfonylureas, the medications disrupt the function of the ATP-sensitive K+ channel, resulting in increased insulin release by pancreatic beta cells.
Day 4
Patient on D5W Infusion 100 mL/hr

4 a.m.

Blood Glucose Measurements

Remaining Inpatient Days Without Hypoglycemic Episodes (Total stay 7 days)

D5W stopped

6 a.m.

D5W re-started

oral glucose & food given

before lunch

before lunch

D5W stopped

6 a.m.

Day 1
Day 2
Day 3
Day 4

Patient on D5W Infusion 100 mL/hr