Blurry Vision: Is it an Ophthalmic, Neurologic, or Endocrine Problem?

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Introduction

Statistics

- Diabetes is an epidemic that has impacted over 38 million people in America in 2021
- Of those 38 million, 2 million have type 1 diabetes
- 304,000 type 1 diabetics are children and adolescents
- 1 in 5 Americans has diabetes and doesn't know it

Healthy vs. Type I Diabetes

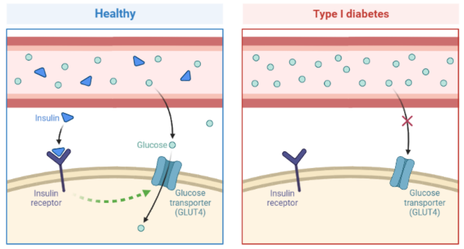

Figure 1: Diabetic Pathophysiology *Obtained from BioRender*

Pathophysiology

Diabetes Mellitus Type 1 (T1DM) is an autoimmune disease. The body identifies insulin-producing pancreatic beta cells as a threat and destroys them. Without insulin, the body is unable to metabolize glucose for energy and glucose stays in the vessels. This buildup of glucose causes hyperglycemia which subsequently injures major systems of the body, including the eyes.

Diabetic Eye Diseases

Diabetic retinopathy, diabetic macular edema, glaucoma, and early cataracts can result from poorly controlled diabetes. Of these diseases, diabetic retinopathy is the most common complication and contributes to blindness.

Diabetic Retinopathy

- Risk factors: chronic uncontrolled hyperglycemia, hypertension, hyperlipidemia
- Chronic excess glucose in systemic vessels → decreased blood flow → small vessels leak in retinas of eyes → retinal tissue swells → cloudy/blurred vision → blindness

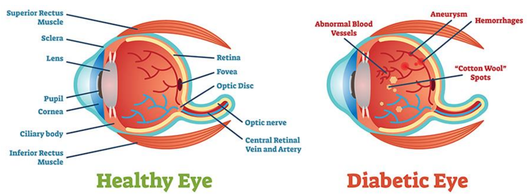

Figure 2: Diabetic Eye *Obtained from American Optometric Association*

Case Description

History of Present Illness

- 35-year-old white female presenting to ED
- **CC: "blurry vision for the past 3 days"**
- Pt reports she can see well up close, but her distance vision is now blurry.
- Previously 20/20 uncorrected.
- Received a massage over the weekend and instructed to increase water intake; now reports increased thirst and urination for 3 days
- ROS: no headache, dizziness, numbness, tingling, nausea or vomiting.

Medical History – depression
Medications – fluoxetine 20mg
Allergies – NKDA
Family History – diabetes in father, maternal and paternal grandparents (type 1/2 unknown).

Physical Exam

T: 98.7 F HR: 106 BP: 182/117 RR: 16
SpO2: 96% BMI: 34.27 kg/m^2

- **Eyes**: No scleral icterus, EOMs intact, conjunctivae normal, PERRLA.
- **Neurologic**: A&O x3. Clear speech. No facial droop. 5/5 upper extremity strength, grip strength, and lower extremity strength bilaterally. Finger-nose-finger intact. Heel-to-shin appropriate bilaterally. CN II-XII grossly intact.

Initial workup: CBC, CMP, CTA of head and neck

Results

CMP			
Sodium	127 (L)	Anion Gap	12 (H)
Potassium	4.4	Albumin	4.3
Chloride	94 (L)	Bilirubin, Total	0.6
CO_2	21	Alk Phos	72
BUN	13	ALT	23
Creatinine	0.82	AST	17
Glucose, Random	681 (HH)	Protein, Total	7.3
Calcium, Total	8.8	Albumin/Globulin	1.4
Osmolality	288		

Table 1: Initial lab results

CBC non-actionable.
CTA cancelled after identifying hyperglycemia.
Phosphorous: 3.7 mg/dL
Magnesium: 1.7 mg/dL

Additional Labs

Beta hydroxybutyrate: **21.1 mg/dL**
Hgb A_{1C}: **9.4%**
Urinalysis: **3+ glucose, 2+ ketones**

Lab	Result	Reference Range
ZNT8 Antibodies	371 U/mL	<15 Negative
IA2 Autoantibodies	<7.5 U/mL	<7.5 Negative
GAD-65	521.0 U/mL	0.0-5.0 U/mL

Table 2: Autoimmune lab results

Diagnosis

Diabetic ketoacidosis, undiagnosed T1DM.

Management

1. Volume Repletion
   - Normal saline, lactated ringers, or other crystalloid is acceptable.
   - Administer 2L bolus over 2 hrs.
   - Hyponatremic: normal saline at 250-500 mL/hr
   - Eunatremic/hypernatremic: 0.45% saline 250-500mL/hr
2. Potassium Correction
   - 3.3<K+<5.2: supplement 20-30 mEq/hr to maintain K+ at 4-5 mEq/L.
   - K+<3.3: hold insulin, give 20-30mEq/hr until >3.5.
   - K+>5.2: obtain ECG, fluids and insulin will typically correct hyperkalemia.
3. Insulin
   - Administer insulin at 0.1-0.14 unit/kg/hr once hypokalemia has been alleviated.
   - When serum glucose <250 mg/dL, add dextrose to fluids and reduce insulin to 0.02-0.05 unit/kg/hr.

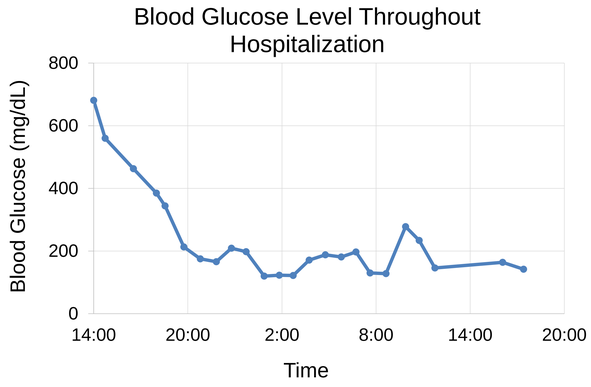

Figure 3: Blood glucose levels throughout hospitalization

Patient Course

- DKA resolved following treatment
- Endocrinologist was consulted and recommended discharge once blood sugars could be controlled with subcutaneous insulin.
- Pt was provided education on managing diabetes and using insulin.
- Pt was discharged with follow up appointment with endocrinology.

Discussion

- Diabetes can be diagnosed by having a random glucose >200 or an A1C >6.5%
- Due to T1DM being a result of autoimmune dysfunction, 96% of patients will test positive for islet cell autoantibodies such as ZNT8, IA2, and GAD-65.
- Diabetic Ketoacidosis (DKA) is a life-threatening complication of T1DM and can be diagnosed by identifying the triad:
  1. Hyperglycemia: glucose >250
  2. Acidosis: elevated anion gap
  3. Ketones: present in urine, elevated beta hydroxybutyrate
- The goal of DKA treatment is to close the anion gap.
- Management of T1DM requires insulin therapy due to the destruction of beta cells.
- It is important to keep a broad differential in a single symptom complaint.
- Remember – T1DM can develop at any age.

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