A poorly controlled diabetic with elevated aminotransferases

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Overview of Initial Presentation

• 24 yo man with type 1 diabetes mellitus
• Presented to OSH ED on 12/26 with lower extremity edema
• Admitted due to hyperglycemia
• Transferred to MMC on 12/28 with rising transaminases
HPI at outside hospital

- 6 months of LE edema
- 1 week of oozing from LE wounds
- 1 year of diarrhea
- Intermittent RUQ pain
- Worked up over the last 6 months for liver disease
Past Medical History

- Diabetes mellitus, type 1
- Hypothyroidism
- Recently diagnosed liver disease
- MVA (femur frx, skin grafting) as teen
- Dirt bike accident (tib/fib frx with ankle surgeries) several years ago
Social History

- Lives with grandfather and aunt
- Toddler daughter
- Everyday smoker
- Rare alcohol use
- Completed 11th grade
- Not working
Physical Exam

**Vitals:** T 36.4, HR 96, BP 115/76, RR 18, 99% on RA

**GenApp:** Lanky chronically ill-appearing young man.

**HEENT:** No scleral icterus. Extremely poor dentition.

**Neck:** Supple, no LAD, no JVD.

**Card:** Regular rhythm, normal rate, no murmurs.

**Pulm:** No distress. CTAB.

**Abdomen:** Nondistended. + BS. Soft. Tender RUQ; cannot assess hepatomegaly. No shifting dullness.

**Extrem:** + Anasarca. Symm pitting edema. + distal pulses.

**Skin:** Superficial, tender, erythematous erosions on lower extremities, largest 8 cm on medial right shin. + tattoos. + skin grafts trunk.
OSH labs

12/26: CBC: WBC 5.9 / Hgb 11.9 (MCV 100.7) / plt 291
CMP: Bicarb 19/ AG 14/ BUN 19/ Cr 0.7/ Gluc 548/ Bili 0.1/ AST 118/ ALT 261/ AlkPh 197/ INR 1.0/ Alb 2.6
Trends in Hepatic Enzymes
### Hepatocellular Injury with Hepatomegaly

<table>
<thead>
<tr>
<th>Category</th>
<th>Conditions</th>
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</thead>
<tbody>
<tr>
<td>Hepatitis</td>
<td>Alcoholic hepatitis, Viral hepatitis, Autoimmune hepatitis, Drug or toxin</td>
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<tr>
<td>Infiltrative/Storage</td>
<td>Hemochromatosis, Alpha-1 antitrypsin defic., Wilsons, Amyloid</td>
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<tr>
<td>Impaired venous flow</td>
<td>Right heart failure, Budd-Chiari</td>
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Diagnosis of hepatocellular injury?

- Aminotransaminases ~ 50-100 x ULN
- Later-peak alkaline phosphatase
- Normal synthetic function
- Rapid resolution
- Most consistent with:
  - Ischemic hepatitis
Patient’s course

• Diabetes management.
• Diuresis.
• Wound care.
• Diagnosis of steatorrhea; management.
• Piercing headache → Head CT normal except sinusitis.
• Facial CT; followed by total teeth extraction.
Facial CT
Putting the pieces together

Cystic Fibrosis

- Diabetes mellitus
- Sinusitis
- Atrophic Pancreas
- Steatorrhea
- Liver Disease
- Malnutrition
Cystic Fibrosis

• Pathophysiology: defect in CFTR gene

• Definition:
  – One typical clinical feature
  – PLUS evidence of gene dysfunction or mutations on both chromosomes

From: www.hopkinscf.org
Cystic Fibrosis: Adults

- Spectrum of phenotypes
- Prevalence
- Importance of diagnosing
- How to identify these patients

Cystic Fibrosis: Our Patient

- Sweat chloride: Left 59 mEq/L; Right 53 mEq/L
- Gene N1303K
- Gene Mapping underway (JHU)

CF liver disease


Update on patient

- Followed at CF clinic.
- Improved steatorrhea on replacement.
- Continues to have very poorly controlled diabetes.
- Underwent TMA.
Learning Objectives

1. Review of pathophysiology and definition of cystic fibrosis.
2. Include cystic fibrosis in your differential diagnoses for adult patients.
References


Thank you

- Dr. Steve Hayes
- Drs. Ellis Johnson, Edmund Sears, Alan Kilby
- Warene Eldridge
- American College of Physicians Maine Chapter
- MMC Internal Medicine residency program