



Hepatomegaly, Hepatitis, and Neutropenia in a T1DM patient

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Introduction

Hepatomegaly and hepatitis have a wide differential, but it can be challenging to identify a cause when there are few symptoms.

Case Description

History of Present Illness:

A 20-year-old male with type 1 diabetes mellitus, hemoglobin A1C=12, presented to the emergency department with abdominal pain of one day. Initially the pain was located in the epigastrium but then localized to the right upper quadrant. His only medication was insulin. He had no diarrhea, constipation, melena, hematochezia, nausea, vomiting, jaundice, fevers, chills, weight loss, or night sweats. He denied recent travel, tick exposure, food indigestion, recent illness, sick contacts, alcohol use, and recreational drug use. Family history was non-contributory.

Physical Exam:

BP 126/80, Pulse 79, Temp 36.9 °C, Resp 16, SpO2 97%, BMI 23.43 kg/m²

General: AOx4. Well-appearing.

Eyes: No scleral icterus.

HENT: Normocephalic, atraumatic. No oral lesions.

CV: Regular rhythm, normal rate. No murmurs, clicks, or rubs. 2+ pulses. No JVD.

Pulm: Clear to auscultation.

GI: Non-distended. Normoactive bowel sounds. Soft. Tender to palpation on right quadrant. Non-tympanic.

Hepatomegaly. No splenomegaly.

MSK: Full painless range of motion. No joint swelling.

Neuro: Normal.

Skin: No rashes or lesions. No peripheral edema. Skin tone is pale. No bronzing.

Psych: Attention normal, thoughts organized, mood euthymic with appropriate range in affect.

Lymph: No adenopathy.

Laboratory results

MCV: 94.7
12.8
2.2 187
37.5

138 | 94 | 9
3.9 | 31 | 0.6

AST: 1923
ALT: 603
Alk Phos: 138
INR: 0.9
Lactate 3.5
Albumin: 3.6
Bili: 1.1

Differential:
Neutrophil: 0.75
Atypical lymphocytes: 0.07

LDH: 886
Haptoglobin: 121
Hemoglobin A1C: 12.1
Acetaminophen level: <5.0

Fe Studies:
Fe 45
Fe binding capacity: 293
%saturation: 15
Ferritin 553

Virology:
Hepatitis A: (-)
Hepatitis B: Surface Ab (+)
Hepatitis C: (-)
HIV: (-)
EBV: (-)
HSV: (-)
CMV: IgG and IgM (+)

Microbiology:
Blood cultures: No growth
Urine culture: No growth
Babesia: (-)

Immunology:
ANA: < 1:80
Smooth muscle ab: <20

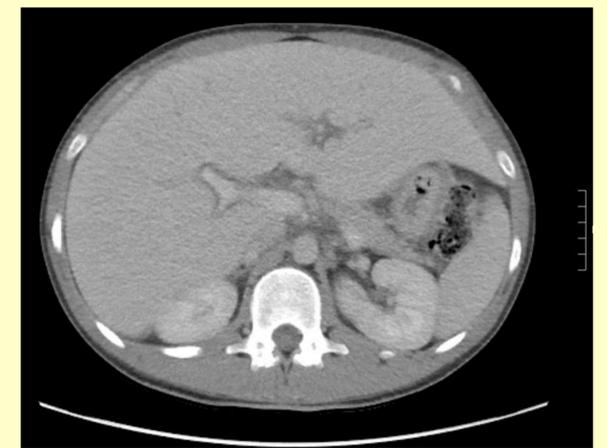
Hospital Course

- Imaging compared to abdominal US from 5 months prior showing hepatomegaly was new.
- Hematology consulted for neutropenia and atypical lymphocytes. Felt atypical lymphocytes represented reactive lymphocytes and neutropenia was secondary to infection.
- LFTs downtrended.
- Neutropenia resolved.
- Patient was discharged on day 3.
- CVM IgG and IgM resulted after discharge.
- Patient missed primary care follow-up but was admitted 1 month after initial hospitalization for DKA. LFTs were normal at that time.

References

1. Epidemiology, clinical manifestations, and treatment of cytomegalovirus infection in immunocompetent adults. Uptodate. Accessed September 2018.
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3. Susceptibility to infections in persons with diabetes mellitus. Uptodate. Accessed September 2018.
4. Functional heterogeneity and high frequency of CMV-specific CD8+ T lymphocytes in healthy seropositive donors. J Virol. 200; 74(17):8140.
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6. CMV. Principles and Practice of Infections Disease. Eight Edition

Imaging



CT Abdomen and pelvis with contrast:

Impression:

Hepatomegaly with hepatic periportal edema. Distended gallbladder with prominent wall. This is a nonspecific finding in patients with underlying liver pathology.

Discussion

- CMV is member of the herpesvirus family and typically establishes a latent infection after resolution of primary infection.
- Secondary symptomatic disease represents either reactivation of latent CMV or reinfection with a novel strain. Reactivation can occur at any time but is more likely in the setting of immunosuppression.
- The most common presentation of symptomatic CMV infection is mononucleosis.
- Abnormal liver function is commonly encountered in acute CMV infection, but does not typically cause significant hepatomegaly in immunocompetent individuals.
- In immunocompromised individuals, hepatomegaly and neutropenia are more commonly encountered.
- Hyperglycemia leads to impaired immune response through impairment in neutrophil activity and cell-mediated immunity.
- CD4+ and CD8+ T cell response is important in controlling CMV replication and disease.
- This patient's poor glucose control likely contributed to his presentation through impaired immune response. His prior hemoglobin A1C had been above 14 for over 4 years.