

A 40-year-old man with ileocolonic Crohn's disease complains of worsening diarrhea, vague abdominal pain, and fatigue. He receives infliximab 5mg/kg every 8 weeks and azathioprine (2.0 mg/kg). He notes improvement of his symptoms following his infliximab infusion lasting 5-6 weeks. His last infusion was 7 weeks ago. An infliximab trough concentration is measured and is undetectable. Antibodies to infliximab are low (ATI <8 µg/mL).

What is the next best step in management?

- A. Discontinue infliximab and start adalimumab
- B. Discontinue infliximab and start vedolizumab
- C. Increase infliximab dose to 10 mg/kg
- D. Start prednisone taper
- E. Discontinue azathioprine and start methotrexate

The correct answer is C.

This patient's infliximab trough level and antibody level are low. The low antibody level indicates the low trough level is not a consequence of immunogenicity. Increasing the infliximab dose is appropriate. While treatment with vedolizumab or adalimumab might be effective in this patient, it is preferred to optimize his current therapy before switching to another agent. While prednisone is an option to manage this patient's symptoms, there are significant side effects associated with corticosteroid treatment and increasing his infliximab dose might be sufficient to relieve his symptoms. There is no evidence that switching immunomodulators (azathioprine to methotrexate) would increase infliximab trough levels.

Vande Casteele N, Ferrante M, et al. Trough concentrations of infliximab guide dosing for patients with inflammatory bowel disease. *Gastroenterology* 2015 Feb 24. pii: S0016-5085(15)00253-X. doi: 10.1053/j.gastro.2015.02.031. [Epub ahead of print]

34-year-old woman ten days post-partum presents with 6 days of abdominal distension. She had been previously well except for a history of asymptomatic Crohn's disease.

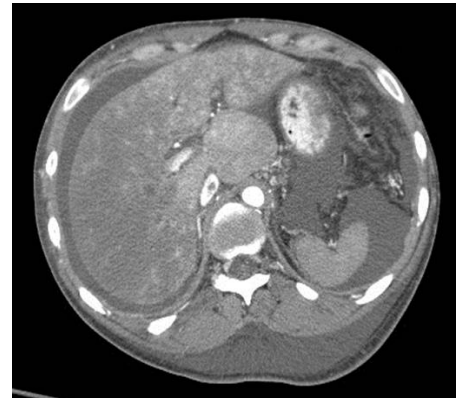
Exam: Mild jaundice and abdominal distention

Lab: AST 280, ALT 300, bilirubin 3.0, albumin 3.4, INR 1.1

CT is as shown to the right

What is the most likely diagnosis?

- A. Primary sclerosing cholangitis
- B. Portal vein thrombosis
- C. Acute pericarditis
- D. Budd-Chiari syndrome
- E. Acute fatty liver of pregnancy



The correct answer is D.

Budd-Chiari syndrome usually presents with sudden onset of ascites, especially in young women and patients with hypercoagulable states. When associated with pregnancy it most commonly occurs in the post-partum state. The CT reveals findings of hepatic congestion, ascites and caudate lobe hypertrophy which would all support Budd-Chiari syndrome.

Primary sclerosing cholangitis is a chronic progressive cholestatic disorder that would not manifest suddenly in the post-partum setting. Acute pericarditis would be associated with chest pain (while constrictive pericarditis is a cause of ascites this would not typically to cause acute ascites in the post-partum setting). Acute fatty liver of pregnancy (AFLP) typically occurs in the 3rd trimester of pregnancy and is associated with severe liver dysfunction. Treatment of AFLP is delivery of the fetus so it would not present in the post-partum setting. Portal vein thrombosis can present with relatively sudden ascites although it would not be associated with hepatic congestion or caudate hypertrophy and would be associated with splenomegaly which is not present.

Bissonnette J, et al. Pregnancy and vascular liver disease *J Clin Exp Hepatol.* 2015 Mar;5(1) 41-50.

Menon KV, Shah V, Kamath PS. The Budd-Chiari syndrome. *N Engl J Med* 2004 Feb 5;350(6)578-85.

A 38-year-old woman with a long history of ileocolonic Crohn's disease, short-bowel syndrome s/p multiple bowel resections, PSC, and home TPN for several years, is seen by you with a resting tremor, muscle rigidity and abnormal gait.

Which likely should be done?

- A. Add oral zinc
- B. Remove copper in TPN
- C. Begin oral rifaximin
- D. Decrease nitrogen content in TPN
- E. Remove manganese in TPN

The correct answer is E.

Manganese Excess:

- Can occur in people on chronic TPN
 - Those with cholestatic diseases are at risk
- Deposition in basal ganglia
 - May be seen on MRI imaging
- Neurotoxicity (movement disorders, gait)
 - Parkinson-like