

# HBV ECHO Case Recommendations



Session 10: October 31, 2022

**Case Recommendations and Considerations: 62 y/o Samoan woman with untreated HBV, obesity and low ceruloplasmin**

CATEGORY	RECOMMENDATIONS	Relevant Presentation Question or Concern	REFERENCES/ RESOURCE LINKS
History	•		
Physical Exam	•		
Diagnostic evaluation	<ol style="list-style-type: none"> <li>1. Hemochromatosis &amp; Wilson disease. Complete the workup to rule them out.               <ol style="list-style-type: none"> <li>a. Overall, we aren't very concerned either of these are the cause of this patient's liver disease. Wilson disease diagnosis at her age (60s) is rare but can happen. Low serum ceruloplasmin is seen with Wilson. Sometimes with liver synthetic deficiency, low ceruloplasmin can be seen as well. However, her high albumin points against this. Rule out Wilson disease with a 24h urine copper and consider adding a slit lamp exam to look for Kayser-Fleischer rings.</li> <li>b. Hemochromatosis, though more common, is unlikely in this case as well. High ferritin levels are nonspecific indicators of inflammation. Consider obtaining another ferritin level to confirm that it was not transiently elevated at the time due to something else. Concurrent NASH can cause elevated ferritin. Also, continue with</li> </ol> </li> </ol>	<ol style="list-style-type: none"> <li>1. What should we make of the low ceruloplasmin and high ferritin levels?</li> <li>2. Might the iron issues resolve if therapy against HBV is initiated</li> <li>3. Can low ceruloplasmin synthesis be caused by active hepatic inflammation from HBV and NAFLD?</li> <li>4. How much can we trust this patient's ultrasound, which was unremarkable in terms of radiographic evidence of portal hypertension?</li> <li>5. Does AST/ALT ratio &gt;1 indicate active NASH?</li> </ol>	

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	<p>obtaining a full iron profile including transferrin saturation levels, as this can help to determine iron overload vs. reactively high ferritin. High transferrin saturation would suggest the former. An HFE gene mutation can rule this out (identifies about 90% of genetic hemochromatosis) Consider also causes of iron overload such as iron supplementation and contaminated utensils at home.</p> <ol style="list-style-type: none"><li>2. Active liver inflammation can raise ferritin, so treatment of the HBV may have some effect at reducing ferritin levels. However, we suspect given her BMI that NASH is a large component of her LFT elevation and liver inflammation due to viral loads only in the 1000 range. Even if HBV may not be the primary disease causing liver fibrosis, treatment should be started due the presence of advanced fibrosis.</li><li>3. No, they are not signaling a hepatic crash but they should be ruled out to provide diagnostic clarity and guide focused treatment of the obesity and HBV.</li><li>4. Ultrasonography is operator-dependent and we shouldn't completely rely on the interpretation of findings to make diagnoses. We should always take the entire context of the patient into consideration. Thus, although her ultrasound findings are rather benign we are still strongly concerned for cirrhosis and portal hypertension.<ol style="list-style-type: none"><li>a. May see splenomegaly, varices, recanalization of the umbilical vein on ultrasound</li></ol></li></ol>		
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	<p>b. Her platelet count of 80 is highly concerning for cirrhosis and portal hypertension. Platelet counts &lt;130 and splenomegaly are indicators of portal hypertension and indicate advanced fibrosis or cirrhosis</p> <p>5. Yes, the AST/ALT ratio &gt;1 may indicate NASH, although we suspect she is becoming cirrhotic due to the initial ALT &gt; AST, which subsequently became AST &gt; ALT after a number of years.</p>		
<p>Medication Therapy &amp; Adjustments</p>	<ol style="list-style-type: none"> <li>1. Based on her metavir score of F3, she at least has advanced fibrosis, if not cirrhosis. She should be treated for hepatitis B due to her advanced fibrosis. Patients with HBV and detectable HBV DNA with advanced fibrosis or cirrhosis should be started on antiviral therapy regardless of the ALT level.</li> <li>2. Patient could be started on either tenofovir or entecavir since her kidney function is okay. This will likely depend on what her insurance will cover.</li> <li>3. Genotyping should be performed prior to the initiation of treatment. This will tell us if there are any mutations present.</li> <li>4. Address her morbid obesity and risk factors that come with the Metabolic Syndrome, as her NAFLD and NASH are likely contributing to hepatic inflammation</li> <li>5. Consider adjusting her other medications. Assuming her metoprolol and verapamil are both for hypertension, there may be more suitable agents available with less negative inotropic effects and more cardio-protection.</li> </ol>	<ol style="list-style-type: none"> <li>1. Is treatment with entecavir appropriate in this patient?</li> <li>2. Are there any preferences for tenofovir or entecavir in this patient?</li> <li>3. When should genotyping be performed?</li> <li>4. NAFLD/NASH</li> </ol>	
<p>Vaccination</p>	<ul style="list-style-type: none"> <li>•</li> </ul>		

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Social Determinants of Health (SDOH)	•		
Behavioral Health	•		
Screening	•		
Risk Reduction	<ol style="list-style-type: none"> <li>1. An EGD should be performed to evaluate for varices due to strong suspicion of portal hypertension. If varices are present she may be considered for further therapy such as beta blockers or variceal banding. A platelet count &lt;100 in the setting of known liver disease is an indication for upper endoscopy and as she likely has advanced fibrosis or cirrhosis.</li> </ol>		
Other	•		

***PLEASE NOTE that case consultations and recommendations for the HBV ECHO do not create or otherwise establish a provider-patient relationship between any participant, Hawaii Learning Groups, and/or any other clinician on the HBV ECHO faculty.***