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Not All That “Glisson” Is Fat

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A 14-year-old Inuit female presented with a history of persistent Epstein Barr (EBV) viremia and EBV hepatitis. She had intermittent fevers with abdominal discomfort. She reported a diet rich in seal meat and significant recent weight gain. Her BMI was 29 and abdominal examination revealed tenderness in the right hypochondrium with hepatomegaly.

Laboratory exam was notable for Alkaline Phosphatase 178U/L (50–162 U/L), Alanine Aminotransferase 98U/L (5–30U/L), Aspartate Aminotransferase 95 U/L (0–40U/L), bilirubin total <0.2 mg/dl (0–1.2mg/dl), albumin 3.9 (3.5–5g/dl), platelet 209 K/uL (173–369 K/uL), Total Cholesterol 141 mg/dl (0–169 mg/dl), Triglycerides 145 mg/dl (0–149mg/dl), Low Density Lipoprotein 65 mg/dl, Glucose 105 mg/dl (74–106mg/dl), Cytomegalovirus negative, EBV 6.13 log₁₀ IU/ml, Hepatitis B surface antigen negative, Hepatitis C virus antibody negative.

An abdominal ultrasound was obtained (figure 1).

What is the etiology of the increase in liver echogenicity?

Answer: Diffuse increase in liver echogenicity suggests hepatic steatosis. However, the MRI showed minimal signal dropout in the liver on the in and out of phase T1 sequence (Figure 2) suggesting minimal steatosis.

A liver biopsy demonstrated accumulation of glycogen in hepatocytes in addition to EBV hepatitis (Figure 3).

Therefore, the increase in hepatic echogenicity on ultrasound was due to glycogen, not fat.

Sonographic findings suggestive of hepatic glycogenosis include an increase in echogenicity, which could be easily misinterpreted as hepatic steatosis. Increased echogenicity due to glycogenosis has been shown in poorly controlled diabetes mellitus, glycogen storage diseases, and those receiving high dose steroids. It is important to differentiate steatosis from glycogenosis because management and natural history may be different.

Given our patient’s clinical presentation, misinterpretation of the ultrasound, as diagnostic of steatosis is understandable. However, careful review of the MRI and liver biopsy led to the correct diagnosis. Physicians utilizing ultrasound of the liver should be aware that not all

increased hepatic echogenicity equates to steatosis and should consider the differential diagnosis.

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Figure 1.
Hepatic ultrasound showed increased echogenicity of the liver

Figure 2:
In and out of phase T1 sequence showed minimal signal drop out.

Figure 3:
10X and 40X views showed hepatocytes with cytoplasmic clearing due to glycogen.