CLINICAL VIGNETTE

Nonalcoholic Fatty Liver Disease

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Case Report

A 33-year-old woman presented for evaluation of abnormal liver tests. She was noted to have liver test abnormalities on labs performed for a recent physical exam. She had no history of liver disease, ascites, peripheral edema, or gastrointestinal bleeding. She recalls having "hepatitis" as a child but she does not recall any sequelae.

Her past medical history is significant for obesity, and she is status post cholecystectomy. She takes no medications and has no known drug allergies. She does not smoke or drink and has never used injection drugs. She specifically denied abdominal pain, jaundice, and pruritus. Physical exam was remarkable for a morbidly obese female who was 5'5" and 250 pounds with BMI of 40. Blood pressure was 140/90 with pulse of 102. Abdomen was soft without hepatosplenomegaly.

Chemistries included albumin 4.5, total bilirubin 0.4, alkaline phosphatase 85, AST 39, ALT 90, cholesterol 220, triglycerides 175 and LDL 145. Abdominal ultrasound shows a normal sized post cholecystectomy liver with evidence of diffuse fatty infiltration. The spleen was normal.

Nonalcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease[1, 2, 3]. NAFLD can be secondary to numerous causes but a great majority of cases occur in patients who are obese or have other components of metabolic syndrome (abdominal obesity, hypertension, dyslipidemia, insulin resistance). NAFLD is considered the hepatic manifestation of the metabolic syndrome[1]. 90% of patients with NAFLD have one of the four characteristic features of the metabolic syndrome and 33% have all four features[1].

Nonalcoholic fatty liver disease (NAFLD) spans a spectrum from simple steatosis to nonalcoholic steatohepatitis (NASH), advanced fibrosis, and cirrhosis[1]. NAFLD is estimated to affect over 90 million persons in the United States[2, 3]. Of these persons approximately 20% are non obese and 75% to 100% are obese or morbidly obese[1]. NAFLD is usually asymptomatic and commonly considered in overweight and obese persons with elevations in liver enzymes[3]. NAFLD remains a diagnosis of exclusion of other liver diseases. ALT levels are typically greater than AST levels[1]. The "two-hit hypothesis" suggests that there is a progression from simple steatosis to NASH[1]. Once steatosis is present inflammation and oxidative stress associated with the metabolic syndrome may promote progress ion to NASH and fibrosis[1]. Simple steatosis generally has a benign prognosis but if patients progress to NASH, approximately 9% develop cirrhosis[3].

Liver biopsy is the current diagnostic standard to measure hepatic fat. No imaging modalities are capable of distinguishing NASH from simple steatosis[2]. Ultrasonographic elastrography (FibroScan) and serum biomarkers, including markers of fibrosis (eg, FibroSURE) are promising non-invasive means of detecting liver stiffness and fibrosis but need further validation[3]; indications and timing of liver biopsy remain controversial[1]. Unfortunately, no histological gold standard is available for non-alcoholic steatohepatitis (NASH) and there is still a significant diversity among pathologists concerning the minimal requirements for NASH.

Management of NAFLD patients should be directed at metabolic risk factors such as visceral obesity, hyperglycemia, type II diabetes mellitus, and hypertriglyceridemia. Recommendations include weight reduction through both diet and physical activity, and weight-loss surgery for extreme obesity. Most medical regimens target components of the metabolic syndrome or oxidative stress associated with the pathogenesis of NASH. These include antiobesity regimens, insulin sensitizers such as metformin, statins, and antioxidants[3]. Bariatric surgery is effective for achieving and main-taining weight loss and reversing the complications of metabolic syndrome. However, we await randomized control trials that assess the efficacy of anti-obesity regimens on histologic and long-term outcomes of NAFLD[2].

REFERENCES

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Submitted on November 8, 2011