Study suggests obesity accelerates progression of cirrhosis

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Researchers from the United States and Europe involved in an NIH-funded multicenter study have determined that increased body mass index (BMI) is an independent predictor of clinical decompensation in patients with compensated cirrhosis, independent of portal pressure and liver function. The findings suggest obesity accelerates cirrhosis progression and measures to reduce BMI could improve the prognosis for patients with advanced liver disease. Study details are available in the August issue of *Hepatology*.

Obesity is a global health epidemic and according to a 2008 report by the World Health Organization (WHO), 1.5 billion adults, age 20 and older, were overweight and worldwide obesity more than doubled since 1980. Of those in the overweight population, WHO estimates more than 200 million men and close to 300 million women were obese. Prior studies have shown that obesity is a frequent cause of chronic liver disease that can progress to cirrhosis, and one study estimated that 17% of liver cirrhosis is attributable to excess body weight. Further studies found lower survival rates among patients with cirrhosis caused by obesity-related liver disease than from viral cirrhosis.

"Given the prior evidence of the detrimental effects of obesity on chronic liver disease, we hypothesized that increased BMI may increase the risk of transition from compensated to decompensated cirrhosis," said Dr. Guadalupe Garcia-Tsao, Professor of Medicine at Yale University School of Medicine in Connecticut. The research team recruited 161 patients with compensated cirrhosis from a trial of

betablockers used for varices prevention. Participants were followed until clinical decompensation (ascites, hepatic encephalopathy or variceal hemorrhage) occurred, or until September 2002. Laboratory tests and portal pressure, assessed by the hepatic venous pressure gradient, were performed.

BMI analysis showed that 29% of participants were in normal range, 40% were overweight and 30% were obese. Study subjects were followed for a median of 59 months, with clinical decompensation occurring in 30% of patients. Decompensation of cirrhosis (that is, development of ascites, variceal hemorrhage or hepatic encephalopathy) was observed at higher rates in patients at the upper end of BMI—31% of overweight and 43% of obese patients—compared to only 15% of patients with normal BMI. Researchers noted the probability of developing clinical decompensation was significantly higher in patients with abnormal BMI.

"Patients who are overweight or obese are at greater risk of accelerating the progression of cirrhosis," concluded Dr. Garcia-Tsao. "Weight reduction may improve patient outcomes in this high-risk population and studies addressing this specific issue are warranted."

More information: "Obesity is an Independent Risk Factor for Clinical Decompensation in Patients with Cirrhosis." Annalisa Berzigotti, Guadalupe Garcia-Tsao, Jaime Bosch, Norman Grace, Andrew Burroughs, Rosa Morillas, Angels Escorsell, Juan Carlos Garcia-Pagan, David Patch, Daniel S. Matloff, Roberto J. Groszmann and the Portal Hypertension Collaborative Group. *Hepatology*; Published Online: June 26, 2011 (DOI: 10.1002/hep.24418); Print Issue Date: August 2011.

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