

Visceral adiposity index directly correlated to viral load in genotype 1 chronic hepatitis C

October 19 2010

Researchers at the University of Palermo in Italy provide the evidence that a higher visceral adiposity index score—a new index of adipose dysfunction—has a direct correlation with viral load and is independently associated with both steatosis and necroinflammatory activity in patients with genotype 1 chronic hepatitis C (G1 CHC). Details of this study are available in the November issue of *Hepatology*, a journal published by Wiley-Blackwell on behalf of the American Association for the Study of Liver Diseases (AASLD).

According to public health surveillance data gathered by the Centers for Disease Control and Prevention, nearly 75% of people with [chronic hepatitis](#) C virus infection in the U.S. have genotype 1, the hardest type to treat. Steatosis (fatty [liver](#)) and insulin resistance (IR), are frequent findings in patients with G1 CHC. Prior studies indicate that these metabolic features not only are independently associated with the severity of liver damage, but also are negative predictors of sustained virologic response (SVR) after standard antiviral therapy.

Visceral adipose tissue is believed to secrete a variety of substances that regulate metabolism, inflammation, and immunity, participating in the pathogenesis of cardiovascular disease, IR and diabetes. To determine whether visceral adiposity is directly associated with the metabolic factors that cause liver damage, a recent study introduced the visceral adiposity index (VAI) score, which uses both anthropometric (body mass index (BMI) and waist circumference) and metabolic (triglyceride and HDL) parameters to measure visceral fat mass, a key factor in metabolic

alteration development, and the effect of visceral obesity on the histological features of liver disease. The VAI assesses fat distribution and function as well as cardiometabolic risk, and has been proposed as a surrogate marker of adipose tissue dysfunction.

In the current study, Salvatore Petta, M.D., and colleagues tested the VAI and its association with histological features and SVR in 236 patients with G1 CHC. Participants were evaluated by liver biopsy and anthropometric and metabolic measurements, including IR, the homeostasis model assessment (HOMA), and VAI by using waist circumference, BMI, triglycerides and HDL. All biopsies were scored for staging and grading, and graded for steatosis, which was considered moderate-severe if $\geq 30\%$. Patients were treated with the standard antiviral therapy of pegylated interferon plus ribavirin. The majority of study participants were in the overweight to obesity range, and nearly 25% were hypertensive. Diabetes was present in 11% of patients, and IR in 42.8%. Metabolic syndrome was diagnosed in 14.9% of patients. One patient in five had fibrosis ≥ 3 by Scheuer score, with a high prevalence of moderate/severe necroinflammation (grading 2-3). Half of the cases had histological evidence of steatosis, though of moderate/severe grade in only 40 cases (16.9%).

One hundred sixty-two patients completed the antiviral treatment program. SVR was achieved in 77 patients (47.5%). VAI score had a direct correlation with HCV viral load and was independently associated with higher HOMA score, higher HCV RNA levels, necroinflammatory activity, and steatosis, by multiple linear regression analysis. IR, higher VAI score, and fibrosis were linked to steatosis, suggesting adipose tissue may interfere with liver fatty accumulation not only by IR promotion, but also by exercising its well-known function as an endocrine organ able to modulate metabolic functions, including steatogenesis. Older age, high VAI score, and fibrosis were independently associated with moderate-severe necroinflammatory

activity by logistic regression analysis.

"Our study found that moderate-severe necroinflammatory activity is independently associated not only with older age but also with VAI score," commented Dr. Petta. "To the best of our knowledge, we have provided the first evidence of an independent link between adipose dysfunction and liver inflammation in CHC." The authors indicate that the index may be able to reflect the ability of adipose tissue to generate proinflammatory mediators capable of participating in liver inflammatory response during HCV infection.

Dr. Petta concluded, "We suggest using VAI as an indicator of adipose-related [liver damage](#), as predictor of liver disease progression, and as a new therapeutic outcome measure in the management of G1 CHC patients."

More information: "Visceral Adiposity Index is Associated with Histological Findings and with High Viral Load in Patients with Chronic Hepatitis C Due to Genotype 1." Salvatore Petta, Marco Amato, Daniela Cabibi, Calogero Cammà, Vito Di Marco, Carla Giordano, Aldo Galluzzo, Antonio Craxì. *Hepatology*; Published Online: JUL 29, 2010 ([DOI:10.1002/hep.23859](https://doi.org/10.1002/hep.23859)); Print Issue Date: November 2010.

Provided by Wiley

Citation: Visceral adiposity index directly correlated to viral load in genotype 1 chronic hepatitis C (2010, October 19) retrieved 4 May 2024 from <https://medicalxpress.com/news/2010-10-visceral-adiposity-index-viral-genotype.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.