Brief Communication

Correlation between liver fat content with dyslipidemia and Insulin resistance

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ABSTRACT

Total 33 obese patients were studied to determine correlation in between liver fat content with dyslipidemia and insulin resistance. Liver and spleen attenuation measurements were taken with three regions of interests (ROIs) from the liver and two ROIs from the spleen. Hepatic attenuation indices were measured as follows: (1) Hepatic parenchymal attenuation (CT_{LP}); (2) liver to spleen attenuation ratio (LS_{ratio}); and (3) difference between hepatic and splenic attenuation (LS_{dif}). Bivariate correlation analysis showed moderate but statistically significant negative correlation between CT_{LP}, LS_{ratio}, and LS_{dif} with body mass index, triglyceride, fasting plasma sugar, fasting plasma insulin, homeostasis model assessment-insulin resistance (HOMA IR), 2 h oral glucose tolerance test (OGTT), and statistically significant positive correlation with high density lipoprotein. Nonalcoholic fatty liver disease (NAFLD) is closely associated with features of the metabolic syndrome. The amount of intrahepatic fat closely correlates with the number of metabolic syndrome features. The values of CT_{LP}, LS_{ratio}, and LS_{dif} demonstrate strong inverse correlations with degree of steatosis.

Key words: Hepatic attenuation indices, homeostasis model of assessment insulin resistance, nonalcoholic fatty liver disease, waist-hip ratio

Introduction

An increase in fat accumulation in the liver and hepatic insulin resistance is independent of obesity and body fat distribution.[1] This increase in liver fat associated with insulin resistance is called nonalcoholic fatty liver disease (NAFLD). NAFLD is defined as excess fat in the liver (5-10% fat histologically) which is not brought about by excess alcohol use (over 20 g/day), effects of other toxins, autoimmune, viral, or other causes of steatosis.[1] NAFLD is common in the obese and is associated with hyperinsulinemia, dyslipidemia, and hypertension. Together, these abnormalities comprise insulin resistance syndrome (metabolic syndrome) and predicts the development of type 2 diabetes.[2] Although histologic confirmation is the gold standard for diagnosing fatty liver.[3] However, biopsies are invasive, induce pain, and require 6 h or more of bed rest. As a substitute for biopsy; imaging techniques including ultrasonography (US), computed tomography (CT), and magnetic resonance (MR) are now widely used. Of these, CT had been chosen as the method for this study.[4] CT attenuation values of the liver were strongly correlated with histological evidence of hepatic steatosis.[5] Hepatic attenuation was a reliable indicator of fatty liver if it was considerably lower than splenic attenuation.[6] Therefore, CT can be used as a noninvasive test to confirm the presence of hepatic steatosis.

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Objective

To determine correlation between liver fat content with dyslipidemia and insulin resistance

Materials and Methods

This was a prospective study. A total of 33 subjects were selected between February 2013 till June 2013 from

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Table 1: Correlation between hepatic attenuation indices and other features of metabolic syndrome

	BMI	WHR	TG	LDL	HDL	FBS	HOMA IR	2 h OGTT
CT_{LP}	r=−0.367*	r=−0.385*	r=−0.561**	r=−0.116	r=0.497**	r=−0.582**	r=−0.582*	r=−0.646**
	P=0.036	P=0.027	P=0.001	P=0.521	P=0.003	P=0.000	P=0.000	P=0.000
LSr_{atio}	r=−0.506**	r=−0.450*	r=−0.688**	r=−0.312	r=0.596**	r=−0.749**	r=−0.743*	r=−0.760**
	P=0.003	P=0.009	P=0.000	P=0.077	P=0.000	P=0.000	P=0.000	P=0.000
LS_{dif}	r=−0.538**	r=−0.502*	r=−0.688**	r=−0.331	r=0.616**	r=−0.788**	r=−0.767*	r=−0.807**
	P=0.001	P=0.003	P=0.000	P=0.060	P=0.000	P=0.000	P=0.000	P=0.000

BMI: Body mass index, WHR: Waist-hip ratio, TG: Triglyceride, LDL: Low density lipoprotein, HDL: High density lipoprotein, FBS: Fasting blood sugar, HOMA IR: Homeostasis model of assessment-insulin resistance, OGTT: Oral glucose tolerance test, CTLP: Hepatic parenchymal attenuation, LSrati: Liver to spleen attenuation ratio, LSdif: Difference between hepatic and splenic attenuation

obesity clinic who visited with self-perception of obesity. Information including age, sex, height, body weight (WT), body mass index (BMI), waist-hip ratio (WHR), history of alcohol intake, systolic and diastolic blood pressure, triglyceride level (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), fasting plasma sugar, fasting plasma insulin, and 2 h oral glucose tolerance test (OGTT) was collected for each patient. Any patient with a history of significant alcohol consumption, bile duct dilatation, hepatic mass, hepatitis, liver cirrhosis, or history of hepatic surgery was excluded. The attenuation of the liver and the spleen were measured using CT scans taken without intravenous contrast agent administration (GE Brightspeed Elite Select 16 Slice). The examination was done with a tube voltage of 120 kVp and a tube current of 50 mA. Five regions of interests (ROIs) were identified in the liver, avoiding vessels, bile ducts, calcifications, and artifacts; four ROIs were identified in the spleen in the same manner. The highest and lowest values were excluded when calculating the mean attenuation values of the liver and spleen. As such, three liver values and two spleen values were used to calculate the mean values. Liver and spleen attenuation measurements were taken with three ROIs from the liver and two ROIs from the spleen. Hepatic attenuation indices (HAIs) were measured as follows: (1) Hepatic parenchymal attenuation (CT_{LP}); (2) liver to spleen attenuation ratio (LS_{ratio}); and (3) difference between hepatic and splenic attenuation (LS_{dif}). Statistical analysis was performed with Statistical Packages for Social Sciences (SPSS) statistical software (version 17.0 for Windows). The alpha level was set at $P = 0.05$ for all tests. We used a bivariate correlation method to assess the relationship between the three HAIs and BMI, WHR, TG, LDL, HDL, fasting plasma sugar, homeostasis model of assessment-insulin resistance (HOMA IR), and 2 h OGTT.

Results

The mean ± standard deviation (SD) of CT_{LP}, LS_{ratio}, and the LS_{dif} value were 53.979 ± 14.452 (ranging 22.5-74.6), 0.998 ± 0.265 (ranging 0.527-1.549), and − 0.558 ± 13.742 (ranging − 28.6 to 24.3), respectively.

The mean values ± SD of BMI, WHR, TG, LDL, HDL, fasting plasma sugar, fasting plasma insulin, HOMA IR, and 2 hour OGTT were 27.697 ± 4.391 (ranging 22.290-37.756), 1.06 ± 0.11 (ranging 0.88-1.24), 173.73 ± 52.257 (ranging 58-342), 158.024 ± 42.125 (ranging 66.0-242.0), 47.13 ± 8.152 (ranging 32-65), 92.906 ± 19.874 (ranging 69.8-152.6), 102.34 ± 46.75 (ranging 28.80-186.30), 3.79 ± 2.23 (ranging 0.9-8.2), and 141.673 ± 34.723 (ranging 96.4-246.8), respectively.

As shown in [Table 1] there was statistically significant negative correlation between CT_{LP}, LS_{ratio}, and LS_{dif} with other features of metabolic syndrome

Discussion

NAFLD is closely associated with features of the metabolic syndrome.[7] The amount of intrahepatic fat closely correlates with the number of metabolic syndrome features.[1] A hepatic attenuation value (CT_{LP}) that is significantly lower than the splenic attenuation value is a reliable indicator for the presence of fatty liver.[5] CT_{LP} and LS ratio both demonstrate strong inverse correlations with degree of histologic steatosis.[4] In our study, we have observed that LS_{ratio} and LS_{dif} were more correlated to various features of metabolic syndrome than CT_{LP}.

Conclusion

As obesity is associated with fatty infiltration of liver, so patients with NAFLD need to be aware that NAFLD is associated with increased risk of developing type 2 DM and is associated with poor glycemic control, dyslipidemia, and increased risk of CVD.

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