Ultrasound in Med. & Biol., Vol. 31, No. 3, pp. 293–299, 2005

0301-5629/05/$–see front matter

doi:10.1016/j.ultrasmedbio.2004.12.015

● *Original Contribution*

LIVER ECHOGENICITY: RELATION TO SYSTEMIC BLOOD PRESSURE AND OTHER COMPONENTS OF THE METABOLIC SYNDROME

TAPIO VEHMAS,* ARI KAUKIAINEN,* PIRJO IMMONEN-RÄIHÄ,† MARTINA LOHMAN,‡ and KATARIINA LUOMA§

*Finnish Institute of Occupational Health, Helsinki, Finland; †Department of Internal Medicine, Turku University Hospital, Raisio, Finland; ‡Orton Hospital, Helsinki, Finland and §Helsinki University Central Hospital, Peijas Hospital, Vantaa, Finland

(*Received* 3 *September* 2004; *revised* 29 *November* 2004; *in final form* 17 *December* 2004)

Abstract—We studied the impact of liver echogenicity among other potential predictors of systemic blood pressure (BP) and the metabolic syndrome. 38 persons (32 males, six females, aged 29 to 66) had their liver echogenicities scored, BPs measured and standard serum laboratory tests studied. There was a significant correlation between both systolic ($r = 0.438$, $p = 0.006$) and diastolic ($r = 0.498$, $p = 0.001$) BP and liver echogenicity. Liver echogenicity was the strongest predictor for systolic BP and the second strongest (after body mass index, BMI) for diastolic BP. Body height may modify the relation between liver echogenicity and systolic BP. Liver echogenicity also correlated significantly with BMI ($r = 0.527$, $p = 0.001$), serum triglycerides ($r = 0.472$, $p = 0.003$) and, to a lesser degree, with serum total cholesterol ($r = 0.305$, $p = 0.066$). Incidentally found increased liver echogenicity should alert the US performer and the clinician reading the report on the possibility of elevated BP or other features of the metabolic syndrome. (E-mail: tapio.vehmas@ttl.fi) © 2005 World Federation for Ultrasound in Medicine & Biology.

Key Words: **Hypertension, Liver, fatty, Metabolism, Obesity, Syndrome X, Ultrasound (US).**

INTRODUCTION

Upper abdominal ultrasonography is a widely used standard procedure. The prevalence of bright liver (liver with increased echogenicity) is 13 to 14 % in the general population (Nomura et al. 1988; Shen et al. 2003) and 20 % in nonselected patients studied (Lonardo et al. 1997). The increase of liver echogenicity is caused mainly by fatty degeneration, cirrhosis being a minor contributor (Layer et al. 1991; Suzuki et al. 1992). Besides being noninvasive and easily repeatable, ultrasound gives a high degree of certainty of the diagnosis, depending on the prevalence of fatty liver in the population being studied (Joy et al. 2003).

Nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis are the two most common chronic liver diseases in USA general population, with a prevalence of 20% and 3%, respectively (Yu and Keeffe 2002). Hepatic steatosis is frequently associated with obesity, type II diabetes and hyperlipidaemia with insulin resistance as a key pathogenic factor. The most common predictors of bright liver are obesity, age, elevated levels of serum lipids, alcohol consumption and male sex (Lonardo et al. 1997; Nomura et al. 1988; Shen et al. 2003).

NAFLD is defined as fatty infiltration of the liver exceeding 5% to 10% by weight. It is a spectrum of disorders ranging from simple fatty liver (steatosis without liver injury) and nonalcoholic steatohepatitis (steatosis with inflammation) to fibrosis/cirrhosis, that resembles alcohol-induced liver disease, but which develops in individuals who are not heavy drinkers. NAFLD is likely the most common cause of chronic liver disease in many countries. NAFLD is being increasingly recognized as a common liver disorder that represents the hepatic manifestation of the metabolic syndrome, a variably-defined aggregate of disorders related to obesity, insulin resistance, type II diabetes, hypertension and hyperlipidaemia.

We previously investigated two methods to estimate liver echogenicity with ultrasound (US) (Vehmas et al. 2004) and laboratory test results in solvent-exposed workers and controls (Kaukiainen et al. 2004). Now this

Address correspondence to: Tapio Vehmas, M.D., Ph.D., Department of Radiology, Finnish Institute of Occupational Health, Topeliuksenkatu 41 a A, FIN-00250 Helsinki, Finland. E-mail: tapio.vehmas@ttl.fi

material is used to study liver echogenicity as a potential predictor for hypertension and other components of the metabolic syndrome. The relative contribution of bright liver and some other potential predictors to high BP is also studied.

MATERIALS AND METHODS

Subjects

We studied 19 male and three female industrial workers exposed to various occupational solvents, as well as 13 male and three female workers of our occupational health institute without such exposure. The industrial workers were referred to the institute due to a suspicion of solvent-induced neurotoxicity. Exclusion criteria included systemic diseases or medication with known hepatic effects, positive hepatitis serology and current pregnancy. Demographic data were obtained with a questionnaire. There were 14 current smokers, 14 ex-smokers and nine never smokers (one with missing data). Two subjects used beta-blockers as a medication (one for paroxysmal atrial fibrillation and the other for essential tremor and hypertension). No other antihypertensive drugs were used. Blood samples were taken after 12 h fasting before the US examination. Standard laboratory methods were used for blood testing. The alcohol consumption index was calculated as the average number of drinks per one month. One drink contains about 13 g of ethanol. This is equal to a bottle of beer (330 mL at 5% ethanol), glass of wine (140 mL at 12% ethanol) or a shot of spirits (40 mL at 40% ethanol). The assessment of solvent exposure has previously been described in detail (Kaukiainen et al. 2004). Blood pressures were measured with the standard manual technique. Descriptive data on the study population are given in Table 1. The study was approved by the local ethical committee and the study subjects gave their written informed consent.

Sonography

Liver was scanned with the SonoAce 8800 MT scanner (Medison, Seoul, South Korea) with a 3.5 to 5 MHz convex probe by an experienced radiologist (TV). A slice showing a representative area of liver parenchyma next to the right kidney was frozen and images printed with a Mitsubishi P91E thermic printer (Mitsubishi, Kyoto, Japan). Liver echogenicity was assessed separately and blinded from the printed images by two experienced radiologists (ML, KL) visually by comparing the echogenicity of liver parenchyma with that of the neighboring kidney cortex. Previously published reference images (1 = normal, 2 = slightly abnormal, 3 = severely abnormal; Brodkin et al. 1995) were used as an aid and decimals were used when the actual image could not be matched with any of the reference images. The intraclass correlation (weighted kappa) between the radiologists' readings was 0.68. The radiologists' average echogenicity estimation was used for the calculations. Examples of echogenicity estimations are given in Fig.1.

Statistical methods

Bivariate Pearson's correlations were computed between systolic/diastolic BP and its potential predictors. The same procedure was used to test associations between liver echogenicity and its potential predictors. One-way analysis of variance was used to compare liver echogenicity between non-, ex- and current smokers. Linear regression analysis was used to study the relation between liver echogenicity and systolic/diastolic BP. Furthermore, general linear model was used to adjust the relation between liver echogenicity and BP with covariants (height, BMI and occupational solvent exposure) correlating strongly with BP. Smoking status (never smoker, ex smoker or current smoker) was also included in the preliminary models as a categorical covariant.

Table 1. Descriptive data on the study population.

Variable	Min.	Max.	Mean	SD (±)
Liver echogenicity	1.00	3.25	1.82	0.67
Age (y)	29	66	49	8.1
Body mass index (kg/m^2)	18	34	26	3.7
Height (cm)	155	191	176	7.9
Mean alcohol consumption (drinks per month)	0	100	24	26.7
Solvent exposure (OELY†)	0	17	5	5.3
Serum AST (U/l)	8	55	30	10.2
Serum ALT (U/l)	6	118	36	24.4
Serum gamma GT (U/l)	13	158	43	33.7
Serum total cholesterol (mmol/l)	4.0	8.8	5.7	1.0
Serum triglycerides (mmol/l)	0.46	7.0	1.4	1.1
Blood glucose (mmol/l)	4.0	6.2	5.0	0.47
RRsyst (mmHg)	102	169	131	17.3
RRdiast (mmHg)	65	108	85	9.1

† Occupational exposure limit years (Kaukiainen et al. 2004).