Online Submissions: wjg.wjgnet.com
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doi:10.3748/wjg.15.5334

World J Gastroenterol 2009 November 14; 15(42): 5334-5339
World Journal of Gastroenterology ISSN 1007-9327

BRIEF ARTICLE

Synergistic effect of fatty liver and smoking on metabolic syndrome

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Received: August 20, 2009 Revised: October 2, 2009
Accepted: October 9, 2009
Published online: November 14, 2009

Abstract

AIM: To investigate the association of fatty liver and smoking on metabolic syndrome and its components.

METHODS: This cross-sectional study enrolled participants who attended annual health screening at Shin Kong Wu Ho-Su Memorial Hospital from January to December 2005. A total of 3455 (1981 men and 1474 women) subjects were included in final analyses. Fatty liver was diagnosed using abdominal ultrasonography by trained gastroenterologists. The modified National Cholesterol Education Program Adult Treatment Panel Ⅲ was used to define metabolic syndrome. The associations between smoking, fatty liver and metabolic syndrome were analyzed using multiple logistic regression.

RESULTS: Subjects with fatty liver, and who smoked tobacco, had the highest odds ratios (ORs) for high waist circumference [OR, 4.5 (95% CI: 3.3-6.1), $P < 0.05$], hypertriglyceridemia [OR, 8.1 (95% CI: 6.0-10.9), $P < 0.05$], low serum high-density lipoprotein cholesterol (HDL-C) [OR, 8.3 (95% CI: 6.1-11.3), $P < 0.05$], and metabolic syndrome [OR, 9.5 (95% CI: 6.7-13.4), $P < 0.05$] compared to subjects without fatty liver who did not smoke tobacco. We also found that the ORs for hypertriglyceridemia, low serum HDL-C, and metabolic syndrome for subjects with fatty liver who smoked tobacco had greater than the sum of the ORs for subjects with fatty liver who did not smoke plus those who did not have fatty liver and who did smoke.

CONCLUSION: Fatty liver and smoking had a synergistic effect on metabolic syndrome and its components, especially for hypertriglyceridemia and low serum HDL-C.

Key words: Smoking; Fatty liver; Synergistic effect; Metabolic syndrome

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Chiang PH, Chang TY, Chen JD. Synergistic effect of fatty liver and smoking on metabolic syndrome. *World J Gastroenterol* 2009; 15(42): 5334-5339 Available from: URL: http://www.wjgnet.com/1007-9327/15/5334.asp DOI: http://dx.doi.org/10.3748/wjg.15.5334

INTRODUCTION

Fatty liver is an increasingly recognized condition with growing prevalence in the modern world and is strongly associated with insulin resistance. The association between fatty liver and metabolic syndrome was broadly discussed in previous studies[1-9]. Fatty liver is often associated with central obesity, type 2 diabetes mellitus, dyslipidemia, and hypertension. Two studies based on Asian populations showed that Japanese subjects[1] with fatty liver had 5- to 9-fold risk of developing metabolic syndrome and Chinese subjects[6] had a 5.2-fold risk of developing metabolic syndrome. Lizardi-Cervera *et al*[3] found that the constellation of metabolic disturbances observed in subjects with fatty liver increased the risk of cardiovascular disease compared to those without fatty liver (OR, 4.7 *vs* 2.8). Fatty liver and metabolic syndrome are becoming important public health issues worldwide.

Smoking is a major risk factor for cardiovascular disease[10]. Previous studies have shown that smoking reduces insulin sensitivity or induces insulin resistance[10-15]

and enhances cardiovascular risk factors such as elevated plasma triglycerides (TG)[16-19], decreased high-density lipoprotein cholesterol (HDL-C)[16,20] and hyperglycemia[11]. Furthermore, several studies show that smoking is associated with metabolic abnormalities and increases the risk of metabolic syndrome[21-24]. Nakanishi *et al*[21] reported that subjects who habitually smoked tobacco had a 1.07- to 1.66-fold risk of developing metabolic syndrome compared to subjects who did not smoke, and the quantity of tobacco smoked had a dose-dependent relationship with the severity of metabolic syndrome.

Habitual smoking is a modifiable risk factor for metabolic syndrome and cardiovascular disease[10,19]. The most effective way for smokers to decrease the risk of metabolic syndrome and cardiovascular disease is to stop smoking[25]. However, Nakanishi *et al*[21] found that smoking cessation was also associated with a 1.3-fold risk of metabolic syndrome due to subsequent body weight gain. Wada *et al*[15] proposed that the effect of smoking on metabolic syndrome would remain over 20 years after smoking cessation.

The effects of fatty liver and habitually smoking tobacco on metabolic syndrome have not been previously addressed. The purpose of the present study was to investigate the possible interactive effects of fatty liver and smoking on metabolic syndrome and its components.

MATERIALS AND METHODS

This retrospective cross-sectional study enrolled participants who voluntarily attended annual health screenings at the health center of the Shin Kong Wu Ho-Su Memorial Hospital from January to December 2005. Demographic data, medical histories, family histories regarding risk factors for metabolic syndrome, smoking habits, exercise habits, and alcohol consumption habits were collected through a self-administrated questionnaire. This study was approved by the Shin Kong Wu Ho-Su Memorial Hospital Institutional Review Board, and written informed consent was obtained from all participants. Hepatitis C (n = 109), alcohol consumption more than three times per week (n = 77), and subjects whose abdominal ultrasonographic findings revealed cirrhosis or other chronic liver parenchymal disease (n = 88) were excluded from this study. Thus, a total of 3455 (1981 men and 1474 women) subjects were included in the final analyses.

At the time of examination, sitting blood pressure (mmHg) was measured by an automated oscillometric BP recorder Dinamap DPC 100X-EN (GE Medical Systems, Milwaukee, Wisconsin, USA) applied to the patient's left arm following a sufficiently long period of rest. Height and body weight were determined with a foot-to-foot bioelectric impedance analyzer (BF-220, Tanita Corp., Tokyo, Japan); these measurements were made to the nearest 0.1 cm and 0.1 kg, respectively, with subjects wearing light clothing. Body mass index (BMI, kg/m^2) was calculated as body weight divided by the square of the individual's height. Waist circumference (WC, cm) was measured midway between the lowest border of the ribs and the iliac crest in the horizontal plane, and to the nearest 0.5 cm, by trained nurses using an anthropometric non-stretchable tape after normal expiration.

Fasting blood samples were collected from all subjects after at least an 8-h fast. Serum levels of total cholesterol (mg/dL), low-density lipoprotein cholesterol (LDL-C, mg/dL), HDL-C (mg/dL), serum triglycerides (TG, mg/dL), and fasting blood glucose (FPG, mg/dL) were measured by an automated Hitachi 7600 clinical analyzer (Hitachi, Ltd., Tokyo, Japan).

Abdominal ultrasonographic examinations were performed by trained gastroenterologists using a Philips EnVisor M2540R (Philips, Andover, Minnesota, USA) without reference to the participants' history of liver disease, biochemical data, or other clinical findings. Using sonography, fatty liver was diagnosed on the basis of abnormally intense, high-level echoes arising from the hepatic parenchyma, liver-kidney difference in echo amplitude, echo penetration into the deep portion of the liver, and clarity of liver blood-vessel structure.

Subjects were classified as current smokers or non-smokers. Alcohol consumption was quantified as the number of drinking times per week, and subjects who drank more than three times per week were excluded before analysis.

Definition of metabolic syndrome

Metabolic syndrome was defined as three or more risk factors as defined by the National Cholesterol Education Program Adult Treatment Panel Ⅲ Guideline criteria and modified by the International Diabetes Federation specifically for the Chinese population, including (1) fasting glucose level ⩾ 100 mg/dL, (2) serum triglyceride level ⩾ 150 mg/dL, (3) HDL-C level (men < 40 mg/dL, women < 50 mg/dL), (4) systolic blood pressure ⩾ 130 mmHg or diastolic blood pressure ⩾ 85 mmHg, and (5) WC (men ⩾ 90 cm, women ⩾ 80 cm). The participants were allocated to one of four study groups according to their smoking status and ultrasonographic liver findings. Group 1 was defined as subjects who did not smoke and did not have fatty liver [fatty liver (-)/smoking (-)]. Group 2 was defined as subjects who did not have fatty liver, but currently smoked [fatty liver (-)/smoking (+)]. Group 3 was defined as subjects who showed ultrasonographic evidence of fatty liver but did not smoke [fatty liver (+)/smoking (-)]. Group 4 was defined as subjects who had ultrasonographic evidence of fatty liver and smoked concurrently [fatty liver (+)/smoking (+)].

Statistical analysis

ANOVA was used for comparison of continuous variables. The χ^2 test was used for comparison of categorical variables. The association between smoking, fatty liver and metabolic syndrome components were analyzed using multiple logistic regression. We controlled confounding factors such as age, gender, drinking habit, and physical activity. A P-value less than 0.05 was defined as statistically significant. All statistical analyses

Table 1 Demographic data and lifestyle factors among study subjects (n = 3455), stratified by fatty liver and smoking habit (mean ± SD) n (%)

	Fatty liver (-)		Fatty liver (+)		*P*-value
	Non-smoker (n = 1702)	Smoker (n = 365)	Non-smoker (n = 1057)	Smoker (n = 331)	
Age (yr)	45.9 ± 11.9	42.6 ± 10.8	49.9 ± 11.0	44.9 ± 9.4	< 0.0001
BMI (kg/m^2)	22.0 ± 2.6	22.3 ± 2.7	25.7 ± 3.2	25.9 ± 3.0	< 0.0001
Male	681 (40)	290 (79.5)	695 (65.8)	315 (95.2)	< 0.0001
Drinking[1]	127 (7.5)	99 (27.1)	122 (11.5)	93 (28.1)	< 0.0001
Physical activity[2]	944 (55.5)	242 (66.5)	568 (53.8)	223 (67.4)	< 0.0001

[1]Alcohol consumption ⩾ 1 time and ⩽ 3 times per week (subjects who drank more than 3 times per week were excluded before analysis); [2]Exercise ⩾ 1 time per week. BMI: Body mass index.

Table 2 Clinical and biochemical characteristics of study subjects (n = 3455) classified by fatty liver and smoking habit (mean ± SD)

	Fatty liver (-)		Fatty liver (+)		*P*-value
	Non-smoker (n = 1702)	Smoker (n = 365)	Non-smoker (n = 1057)	Smoker (n = 331)	
WC (cm)	79.9 ± 8.3	80.8 ± 7.4	88.1 ± 8.5	88.8 ± 7.4	< 0.0001
FPG (mg/dL)	89.2 ± 19.3	89.8 ± 21.4	101.9 ± 33.4	99.7 ± 33.4	< 0.0001
SBP (mmHg)	113.9 ± 19.8	111.8 ± 16.8	125.9 ± 20.1	122.4 ± 18.8	< 0.0001
DBP (mmHg)	68.6 ± 11.3	69.7 ± 10.9	76.5 ± 11.4	78.1 ± 10.6	< 0.0001
TG (mg/dL)	97.7 ± 49.5	130.1 ± 74.3	170.6 ± 129.3	225.9 ± 238.5	< 0.0001
HDL-C (mg/dL)	60.1 ± 14.9	52.6 ± 13.8	48.9 ± 11.4	44.3 ± 12.6	< 0.0001

WC: Waist circumference; FPG: Fasting plasma glucose; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; TG: Triglyceride; HDL-C: High-density lipoprotein cholesterol.

Table 3 Prevalence of metabolic risk factors and metabolic syndrome among study subjects (n = 3455) classified by fatty liver and smoking habit n (%)

	Fatty liver (-)		Fatty liver (+)		*P*-value
	Non-smoker	Smoker	Non-smoker	Smoker	
Central obesity[1]	497 (29.2)	51 (14.0)	501 (47.4)	137 (41.4)	< 0.0001
FPG ⩾ 100 mg/dL	164 (9.6)	40 (11.0)	317 (30.0)	76 (23.0)	< 0.0001
High blood pressure[2]	356 (20.9)	63 (17.3)	477 (45.1)	119 (36.0)	< 0.0001
TG ⩾ 150 mg/dL	207 (12.2)	103 (28.2)	478 (45.2)	197 (59.5)	< 0.0001
Low HDL-C[3]	223 (13.1)	71 (19.5)	320 (30.6)	147 (44.4)	< 0.0001
Metabolic syndrome	123 (7.2)	33 (9.0)	364 (34.4)	121 (36.6)	< 0.0001

[1]Waist circumference in men ⩾ 90 cm or in women ⩾ 80 cm; [2]Systolic blood pressure ⩾ 130 mmHg or diastolic blood pressure ⩾ 85 mmHg; [3]High-density lipoprotein cholesterol in men < 40 mg/dL or in women < 50 mg/dL.

were performed using SAS statistical software, (SAS for Windows, version 8.02; SAS Institute Inc, Cary, NC, USA).

RESULTS

Table 1 summarized the demographic data and lifestyle factors among the study subjects stratified by fatty liver and smoking habit. The fatty liver group had more men, higher BMI, and more alcohol drinkers compared to subjects without fatty liver. The frequency of regular exercise was similar for subjects with and without fatty liver. Furthermore, subjects who smoked tobacco also drank alcohol with greater frequency and exercised more regularly compared to subjects who did not smoke.

Table 2 summarized the clinical and biochemical characteristics of the study subjects. Participants with fatty liver had a significantly greater WC, FPG, blood pressure and TG, and lower serum HDL-C compared to participants without fatty liver. Higher TG levels and lower HDL-C levels were found in the smoking group compared to subjects who did not smoke. Participants with fatty liver who smoked had the highest triglyceride levels (225.9 mg/dL) and the lowest HDL-C levels (44.3 mg/dL) among the four study groups.

The prevalence of metabolic syndrome abnormalities was significantly higher in the fatty liver group compared to subjects without fatty liver (Table 3). Subjects with fatty liver also had a higher prevalence of metabolic syndrome compared to those without fatty liver. Subjects who smoked had a higher prevalence of metabolic syndrome and more metabolic syndrome abnormalities, especially for higher TGs and lower HDL-C compared to subjects who did not smoke.

Table 4 showed the associations among fatty liver, smoking, and metabolic syndrome using multiple

Table 4 ORs for metabolic risk factors and metabolic syndrome among subgroups of subjects, analyzed by multiple logistic regression, adjusted for age, gender, drinking alcohol, and physical activity (n = 3455)

	Fatty liver (-)				Fatty liver (+)			
	Non-smoker (n = 1702)		Smoker (n = 365)		Non-smoker (n = 1057)		Smoker (n = 331)	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Central obesity[1]	1 (reference)		0.7	0.5-1.1	3.5[a]	2.9-4.2	4.5[a]	3.3-6.1
FPG ≥ 100 mg/dL	1 (reference)		1.3	0.8-1.9	3.6[a]	2.9-4.6	2.8[a]	2.0-4.1
High blood pressure[2]	1 (reference)		0.7	0.5-1.0	2.7[a]	2.2-3.2	1.7[a]	1.3-2.3
TG ≥ 150 mg/dL	1 (reference)		2.4[a]	1.8-3.2	5.3[a]	4.4-6.5	8.1[a]	6.0-10.9
Low HDL-C[3]	1 (reference)		2.0[a]	1.4-2.7	3.4[a]	2.7-4.1	8.3[a]	6.1-11.3
Metabolic syndrome	1 (reference)		1.4	0.9-2.2	6.6[a]	5.2-8.4	9.5[a]	6.7-13.4

[a]P < 0.05. [1]Waist circumference in men ≥ 90 cm or in women ≥ 80 cm; [2]Systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg; [3]High-density lipoprotein cholesterol in men < 40 mg/dL or in women < 50 mg/dL. OR: Odds ratio; CI: Confidence interval.

Table 5 ORs for metabolic risk factors and metabolic syndrome among fatty liver groups, analyzed by multiple logistic regression, adjusted for age, gender, drinking alcohol, and physical activity (n = 1388)

	Fatty liver (+)			
	Non-smoker (n = 1057)		Smoker (n = 331)	
	OR	95% CI	OR	95% CI
Central obesity[1]	1 (reference)		1.2	0.9-1.6
FPG ≥ 100 mg/dL	1 (reference)		0.7	0.5-1.1
High blood pressure[2]	1 (reference)		0.6	0.5-0.9
TG ≥ 150 mg/dL	1 (reference)		1.5[a]	1.2-2.0
Low HDL-C[3]	1 (reference)		2.7[a]	2.0-3.7
Metabolic syndrome	1 (reference)		1.3[a]	1.0-1.8

[a]P < 0.05. [1]Waist circumference in men ≥ 90 cm or in women ≥ 80 cm; [2]Systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg; [3]High-density lipoprotein cholesterol in men < 40 mg/dL or in women < 50 mg/dL.

logistic regression. We found that subjects with fatty liver had significantly increased odds ratios (ORs) for developing metabolic syndrome and its components. Subjects who smoked had higher ORs for developing hypertriglyceridemia and low serum HDL-C, whether they had fatty liver or not. In addition, subjects with fatty liver who smoked had the highest ORs of high WC, hypertriglyceridemia, low HDL-C, and metabolic syndrome. We also found the ORs of hypertriglyceridemia, low HDL-C, and metabolic syndrome for subjects in group 4 [fatty liver (+)/smoking (+)] were greater than the sum of the ORs for subjects in group 3 [fatty liver (+)/smoking (-)] plus group 2 [fatty liver (-)/smoking (+)].

Table 5 showed the ORs for metabolic syndrome components among fatty liver groups. In comparison with subjects with fatty liver who did not smoke, subjects with fatty liver who smoked had significantly increased ORs for hypertriglyceridemia [OR, 1.5 (95% CI: 1.2-2.0), P < 0.05], low HDL-C [OR, 2.7 (95% CI: 2.0-3.7), P < 0.05] and metabolic syndrome [OR, 1.3 (95% CI: 1.0-1.8), P < 0.05].

DISCUSSION

In this study, we found that fatty liver was associated with an increasing OR for metabolic syndrome. The OR for developing metabolic syndrome increased significantly among subjects with fatty liver compared to those without fatty liver. We also found that smoking was associated with high TG levels and low HDL-C levels. Subjects who smoked had increased ORs for hypertriglyceridemia and low HDL-C, whether they had fatty liver or not. Moreover, fatty liver and smoking seemed to have a synergistic effect on metabolic syndrome. The ORs for hypertriglyceridemia, low HDL-C, and metabolic syndrome for subjects in group 4 [fatty liver (+)/smoking (+)] had greater than the sum of the ORs for subjects in group 3 [fatty liver (+)/smoking (-)] group plus group 2 [fatty liver (-)/smoking (+)]. In fatty liver groups, subjects who smoked had a significantly increased OR for hypertriglyceridemia, low HDL-C and metabolic syndrome compared to subjects who did not smoke.

Metabolic syndrome and fatty liver were well discussed in previous studies. Insulin resistance played a pivotal role in the pathophysiology of both fatty liver and metabolic syndrome[26,27]. Furthermore, several studies showed that smoking induced insulin resistance or hyperinsulinemia and led to metabolic syndrome[13,23,28]. The associations between fatty liver, smoking, and metabolic syndrome were discussed in many studies individually, but the effect of interaction of fatty liver and smoking on metabolic syndrome was not investigated. In our study, subjects with fatty liver who smoked had a significantly increased risk of metabolic syndrome.

The effects of cigarette smoking inducing high plasma TGs and low HDL-C levels were shown in several studies[14,16-20,23,24,29,30]. While these studies provided evidence that smoking increased plasma TG and decreased HDL-C levels, most of these studies did not control potential confounding factors. Fatty liver also increased the risk of dyslipidemia such as hypertriglyceridemia and decreased HDL-C[3]. In our study, after controlling for confounding factors such as age, gender, drinking habit, and physical activity, the ORs for hypertriglyceridemia in subjects remained high: group 2 [fatty liver (-)/smoking (+)] 2.4 (95% CI: 1.8-3.2), group 3 [fatty liver (+)/smoking (-)] 5.3 (95% CI: 4.4-6.5), and group 4 [fatty liver (+)/smoking (+)] 8.1 (95% CI: 6.0-10.9) compared to subjects without fatty liver who did not smoke. Additionally, the ORs for low HDL-C were 2.0 (95% CI: 1.4-2.7) for group 2 [fatty liver (-)/smoking (+)], 3.4 (95% CI: 2.7-4.1) for group 3 [fatty liver (+)/smoking (-)], and 8.3

(95% CI: 6.1-11.3) for group 4 [fatty liver (+)/smoking (+)], compared to subjects without fatty liver who did not smoke. Thus, our study provided evidence that fatty liver and smoking increased the risk of hypertriglyceridemia and low HDL-C.

Cigarette smoking decreased insulin sensitivity through increasing circulating levels of insulin-antagonistic hormones (i.e. catecholamines, cortisol, and growth hormone) and increasing lipolysis, resulting in high circulating levels of free fatty acid[21]. Nicotine, carbon monoxide, and other metabolites from smoking also played important roles in insulin resistance. Furthermore, several mechanisms by which cigarette smoking promoted dyslipidemia were proposed, including decreased lipoprotein lipase activity, increased 3-hydroxy-3-methylglutaryl-CoA reductase activity, increased glucose-6-phosphatase dehydrogenase activity and increased central obesity[16]. Thus, both fatty liver and smoking increased plasma TG and decreased HDL-C by increasing insulin resistance and/or decreasing insulin sensitivity.

While there was no significant difference in WC in subjects without fatty liver, regardless of whether or not subjects smoked, the OR for central obesity was greater among smokers than non-smokers for subjects with fatty liver. These findings were consistent with a previous study[31]. Mizuno *et al*[31] proposed that WC showed no difference between non-obese subjects with or without a smoking habit. Nonetheless, WC was significantly higher in obese subjects who smoked than those who did not. Smoking seemed to accelerate visceral fat accumulation and promote obesity-related disorders.

Insulin resistance secondary to cigarette smoking decreased insulin-mediated glucose uptake and also resulted in hyperglycemia[11,32]. In present study, the ORs for hyperglycemia were 1.3 (95% CI: 0.8-1.9) for group 2 [fatty liver (-)/smoking (+)], 3.6 (95% CI: 2.9-4.6) for group 3 [fatty liver (+)/smoking (-)], and 2.8 (95% CI: 2.0-4.1) for group 4 [fatty liver (+)/smoking (+)], compared to subjects without fatty liver who did not smoke. Thus, fatty liver increased the risk of hyperglycemia, but the effect of smoking on blood sugar was not obvious.

The effect of smoking on blood pressure is controversial. Frati *et al*[10] showed that the acute effect of cigarette smoking increased blood pressure and heart rate, but this effect was not seen after the first hour. Geslain-Biquez *et al*[23] proposed that the frequencies of increasing blood pressures did not differ between smokers and non-smokers. Moreover, Weitzman *et al*[24] found that active smoking was associated with decreasing blood pressure. We found lower ORs for high blood pressure in smoking subjects with or without fatty liver. Fatty liver had a positive effect on elevating blood pressure, but smoking seemed to have a negative effect on raising blood pressure in present study.

There are several limitations in this study. First, since abdominal ultrasonography can only detect fatty liver when steatosis affects more than 33% of the liver parenchyma[33,34], subjects in this study might be affected by fatty liver but not be detected. The impacting effects of fatty liver on metabolic syndrome and its components might be underestimated. Second, the information of alcohol consumption was collected by the drinking times per week. We did not count the amount of alcohol units and the impacting effects of alcohol on metabolic syndrome components might be diluted. Besides, the smoking groups tended to have a higher prevalence of drinking, even though we excluded subjects who drank more than three times per week and using multiple logistic regression to adjust the impacting effects of alcohol on metabolic syndrome components, the effects of alcohol on metabolic syndrome components can not be totally ruled out. Third, the smoking data was collected by binary criteria in this study so the effects of smoking on metabolic syndrome components might be underestimated[35].

In summary, fatty liver and smoking were closely related to insulin resistance[1-9,11-14,16,21-23,25,32], and our study provided evidence that fatty liver and smoking had a synergistic effect on metabolic syndrome and its components, especially for triglyceride and HDL-C levels. We suggested that smoking cessation would have the great benefit of reducing the risk of metabolic syndrome, especially for subjects with fatty liver.

ACKNOWLEDGMENTS

The authors thank all the staff at the health check-up center of Shin Kong Wu Ho-Su Memorial Hospital for their assistance.

COMMENTS

Background

Metabolic syndrome is a frequent metabolic abnormality affecting approximately 20% of the nondiabetic population worldwide and increasing risks of cardiovascular diseases and mortality. Fatty liver and smoking increase the risk of developing metabolic syndrome through the pathogenetic factor of insulin resistance and were well discussed in previous studies separately. We aimed to investigate possible interactive effects of fatty liver and smoking on metabolic syndrome.

Research frontiers

It was recognized that fatty liver, smoking and metabolic syndrome associated with insulin resistance would increase the risks of serious complications such as cirrhosis, hepatocellular carcinoma and cardiovascular diseases. We want to identify the interactive effect of fatty liver and smoking on metabolic syndrome and provide evidence for lifestyle modification and appropriate management.

Innovations and breakthroughs

The authors provided evidence that fatty liver and smoking had a synergistic effect on metabolic syndrome and its components, especially for triglyceride and high-density lipoprotein cholesterol (HDL-C) levels.

Applications

After identifying the synergistic effect of fatty liver and smoking on metabolic syndrome, the authors suggested that smoking cessation would have the great benefit of reducing the risk of metabolic syndrome, especially for subjects with fatty liver.

Terminology

Metabolic syndrome: Metabolic syndrome is a cluster of metabolic abnormalities and is associated with waist circumference, serum triglycerides level, serum HDL-C level, blood pressure and fasting blood glucose (FPG) level. Fatty liver: Fatty liver is a clinicopathologic condition characterized by significant lipid deposition in the liver parenchyma.

Peer review

This retrospective study investigated the possible interactive effect of fatty liver and smoking on metabolic syndrome. This study was based on a large scale population base (n = 3455) and the results are interesting. Study results

suggested that smoking cessation would reduce the risk of metabolic syndrome, especially for people with fatty liver.

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