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Food Intake And Visceral Fat Deposition Are Risk Factors Of Incidence And Severity Of Non-Alcoholic Fatty Liver Disease

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Abstract

Background: Non alcoholic fatty liver disease (NAFLD) is abnormalities of metabolism resulting in fat deposition in the hepatocyte occurred in people who do not consume alcohol. Carbohydrate and fat intake, also visceral fat deposition has been studied as a risk factor of NAFLD, however the results remain elusive.

Objective: To identify the nutritional and clinical risk factors of the incidence and severity of NAFLD.

Methods: This study was done from June to December 2014 in the Dr. Kariadi Hospital Semarang. A case-control group was established comprising 33 patients with NAFLD based on the ultrasonography (USG) criteria (case group) and 34 healthy subject (control group). Carbohydrate and fat intake was assessed by using the food frequency questionnaire (FFQ), visceral fat deposition was measured by body impedance analysis (BIA), and clinical markers were obtained from laboratory data.

Results: Carbohydrate intake, fat intake, and visceral fat deposition were risk factors of the incidence and severity of NAFLD ($OR=7.8$, $CI95\%$ 2.43-25.45; $OR=5.9$, $CI95\%$ 2.0-17.57; $OR=50.7$, $CI95\%$ 6.16-418.09) and ($OR=0.9$, $CI95\%$ 1.06-90.58; $OR=14.6$, $CI95\%$ 1.37-156.88; $OR=6.6$, $CI95\%$ 1.17-37.78). Multivariate regression showed that the most important risk factor of NAFLD for the incidence and severity were hypertriglyceridemia ($OR=8.7$, $CI95\%$ 2.20-34.44) and fat intake ($OR=48.4$, $CI95\%$ 2.78-844.1), respectively.

Conclusion: High carbohydrate intake, fat intake, and high visceral fat deposition are risk factors of the incidence and severity of NAFLD. Hypertriglyceridemia and fat intake are the most important risk factor of NAFLD incidence and severity, respectively.

Keywords: Non alcoholic fatty liver disease, carbohydrate intake, fat intake, visceral fat, risk factor

INTRODUCTION

Non Alcoholic Fatty Liver Disease (NAFLD) is a liver disorder that is histologically similar to Alcoholic Fatty Liver Disease (ALD), but occurs in patients who do not consume alcohol.

This disease is caused by abnormalities of metabolism resulting in fat deposition in the hepatocyte.^{1,2} NAFLD spectrum is very broad, ranging from simple fatty liver, fatty liver disease with NAFLD, Liver Cirrhosis and Hepatocellular Carcinoma.³ Approximately 37% of ALD patients on the simple fatty liver spectrum will develop into Fatty Liver Disease with NAFLD, 45% develop into cirrhosis within 7 years, and then 5-8% develop into hepatocellular carcinoma within 1 year.⁴

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In the last decade, the prevalence of NAFLD around the world has increased rapidly. World Gastroenterology Organization mentioned that NAFLD is the number one liver disease in western countries. An estimated 6 million Americans experience NAFLD. NAFLD incidence in some countries in Asia is reported to be about 5-40% depending on the population studied. NAFLD prevalence in the general population in Japan in 1988 was about 9-14% and there has been a significant increase in obese populations in the last two decades. NAFLD in India is estimated at 5-28%, and in China with liver ultrasound (ultrasound) examination, NAFLD prevalence is approximately 15%.⁵

Specific research on NAFLD has not been done in Indonesia. Several studies of NAFLD suggested that 30.6% NAFLD prevalence and important risk factors reported were metabolic syndrome, obesity, type 2 diabetes mellitus, insulin resistance (IR), and hypertriglyceridemia where caused by high food intake of carbohydrate and high fat intake is closely related to these risk factors.^{6,7}

The objective of this study was to identify the nutritional and clinical risk factors of the incidence and severity of NAFLD.

MATERIALS AND METHODS

This study was a case-control study.⁴⁷ The scope of this research was in the field of Clinical Nutrition Sciences and Internal Medicine Sub Section Gastroenterohepatology that conducted in Internal medicine clinic sub Gastroenterohepatology Dr. Kariadi Semarang Hospital between June and December 2014.

Study Subjects

The target population were patients in the Gastroenterohepatology clinic of Dr. Kariadi Hospital Semarang, with the reasonably affordable population for the case was patients who had been diagnosed with NAFLD, who had been screened early on and there was no change of diet and physical activity in the last 5 years. An population for control was a person who on an ultrasound examination that performed by a Gastroenterohepatology consultant, was not found NAFLD or other liver disease, and there was no history of taking medications that cause liver disorders.

Inclusion criteria of case subject were patients with: (a) Viewed from NAFLD diagnosed medical record, (b) Positive abdominal ultrasound for NAFLD (c) Not taking any medicines or herbs that interfere with the liver function or that cause fatty liver and (d) Not taking any medicines or herbs that inhibit fat absorption, fat burners, and weight loss (e) Not suffering from liver disease other than NAFLD (f) Passes screening on diet and living habits (see appendix) (g) Willing to be subject of the study. Exclusion criteria were having abnormalities and/or congenital defects that can interfere with research measurements. Inclusion criteria of control subject were patients (a) without history and symptoms of liver disease (b) who had not any liver abnormalities

on ultrasound examination. Exclusion criteria in the control group were the same as the case group.

Data processing

Data collection is done manually using the research form provided. Food intake assessment was done with semiquantitative FFQ, which then conducted data entry using intake analysis program with Nutrisurvey Program. Physical Activity Assessment based on interviews using International Physical Activity Questionnaire (IPAQ) was then calculated with an automatic calculator IPAQ score instrument.

Data analysis

Univariate analysis of variables analyzed data normality then data is presented in the form of distribution table and description. Bivariate analysis is an analysis to determine the relationship of each risk factor with NAFLD using test in accordance with the variables analyzed. Data from the variables corresponding to the p value <0.25 were included in a multivariate logistic regression analysis.

The study was approved by the University-Hospital's Ethics Committee (no. 128/EC/FK-RSDK/2014) and all patients provided written informed concept before participation.

RESULTS

Overview of Research Subject

NAFLD group obtained 33 subjects in which 15 men and 18 women, while healthy controls were 34 subjects with 12 males and 22 females. There were no difference of characteristics of subjects in this study in general. The age range was 28-68 years and the mean age was 43.39 years. While the control group obtained 34 subjects, with female gender more than men. Age range 28-55 years and average age 40.76 years. The mean of Waist circumference in the NAFLD group was higher than the control. Based on Waist circumference and sex by Asian classification, NAFLD subjects had more central obesity than those who did not, while control subjects who had fewer central obesity than those who did not. The mean of BMI in the NAFLD group was higher than the control, there was even a NAFLD subject with 43 kg / m² IMT (Table 1).

Most of the subjects from both groups are civil servants and all research subjects in both groups have middle to upper economic level where their income is above minimum wages rate for Semarang in 2014. Subjects in both groups have more activity than those with low activity. None of the subjects of the two groups had high activity. The NAFLD group in this study had a diverse NAFLD Fibrosis Score, but after classification according to its degree, the number of subjects was almost the same between groups. A total of 10 (30.3%) NAFLD subjects had a low NAFLD Fibrosis Score, 11 (33.3%) of NAFLD subjects had moderate Fibrosis Score NAFLD, and 12 (36.4%) NAFLD subjects had high NAFLD Fibrosis Score (Table 1).

Table 1. Baseline patients' characteristics

No	Group	NAFLD (n=33)	Control (n=34)
1	Age (tahun)	43.4 (±9.0) 28-68	40.8 (±8.03) 28-55
2	Waist Circumference (cm)	99.6 (±11.2) 83-132	83 (±9s.7) 64-101
3	BMI (kg/m ²)	27.9 (±4.9) 22-43	22.8 (±3.3) 17-33
4	Occupation		
	Civil Servants	18 (26.8%)	22 (32.8%)
	Employees	12 (17.9%)	9 (13.4%)
	House wife	3 (4.4%)	3 (4.7%)
	Total	33 (49.3%)	34 (50.7%)
5	Income		
	Below Mean Income	0 (0%)	0 (0%)
	Upper Mean Income	33 (49.3%)	34 (50.7%)
6	Physical Activity		
	Mild	15 (22.4%)	9 (13.4%)
	Moderate	18 (26.9%)	25 (37.3%)
	Severe	33 (49.3%)	34 (50.7%)
7	Sex		
	Man	15 (22.4%)	12 (17.9%)
	Woman	18 (26.9%)	22 (32.8%)
	Total	33 (49.3%)	34 (50.7%)
8	NAFLD Fibrosis Score		
	Mild	10 (30.3%)	
	Moderate	11 (33.3%)	
	Severe	12 (36.4%)	
	Total	33 (100%)	

Carbohydrate and Fat intake, as well as Visceral Fat Deposition Score

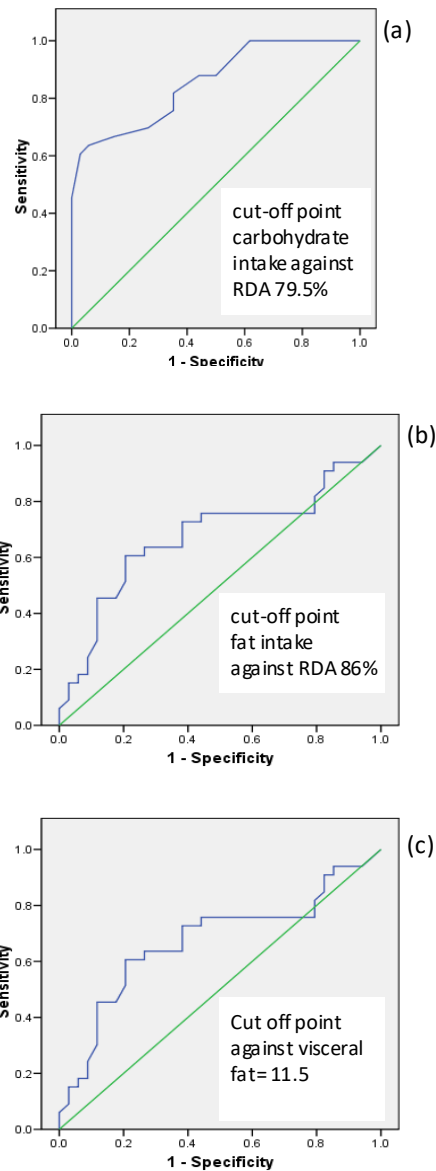
Carbohydrate and fat intake in NAFLD group was higher than control. The mean visceral fat deposition based on BIA scores in the NAFLD group was higher than in the control (Table 2).

Table 2. Description of Carbohydrate, Fat intake and Visceral Fat Deposition Score of Research subject

No	Variabel	NAFLD Mean ±SB (Min-Max)	Control Mean ±SB (Min-Max)
1	Carbohydrate Intake (gram)	315.8±128.4 (125.9-602.2)	216.2±65.2 (107.4-391.7)
2	Fat Intake (gram)	74.6 ±36.5 (17.8-204.5)	54.8±22.7 (17.9-115.4)
3	Visceral Fat Deposition Score	12.6 ±6.5 (4.0-30.0)	5.5 ±3.3 (1.0-12.0)

Risk Factors of NAFLD Incidents

In determining the risk factors for NAFLD events, the determination of the classification of carbohydrate and fat intake, as well as visceral fat classification was based on the results of Receiver Operating Characteristic (ROC) against Recommended Daily Allowance (RDA) analysis (Figures 1)

**Figure 1.** (a) Carbohydrate Intake against RDA. (b) Fat Intake against RDA. (c) Visceral Fat Deposition, on Case-Control Group.

In bivariate analysis towards nutritional variables such as: high carbohydrate intake, high fat intake, and high deposition of visceral fat, were shown to be the risk factor for NAFLD incidence (Table 3). Clinical markers such as visceral obesity, insulin resistance, dyslipidemia, hyper triglyceridemia, Hipo-HDL, hypertension, and metabolic syndrome were also proved to be the risk factor for NAFLD incidence.

Multivariate analysis was performed on risk factors of NAFLD incidence, indicating that hypertriglyceridemia was the most influential factor (OR 8.7, CI95% 2.2-34.4) and visceral fat deposition (OR 5.8, CI95% 1.39-24.8) were the independent risk factor for incidence of NAFLD (Table 3 and 4).

Table 3. Risk Factors of High Carbohydrate, High Fat Intake and Clinical Markers on NAFLD Occurrences

No	Variabel	NAFLD N (%)	Control N (%)	OR (CI95%)	p
1. Carbohydrate Intake					
	High (>79.5%)	19 (28.4)	5 (7.5)	7.8 (2.43-25.45)	<0.001
	Low (≤79.5%)	14 (20.9)	29 (43.3)		
	Total	33 (49.3)	34 (50.7)		
2. Fat Intake					
	High (>86%)	20(29.9)	7 (10.4)	5.9 (2.00-17.57)	0.001
	Low (≤86%)	13 (19.4)	27 (40.3)		
	Total	33 (49.3)	34 (50.7)		
3. Visceral Fat Deposition Score					
	High (>11.5%)	20 (29.9)	1 (1.5)	50.7 (6.16-418.09)	<0.001
	Low (≤11.5%)	13 (19.4)	33 (49.3)		
	Total	33 (49.3)	34 (50.7)		
4. Central Obesity					
	Yes	23 (34.3)	5 (7.5)	13.3 (3.99-44.51)	<0.001
	No	10 (14.9)	29 (43.3)		
	Total	33 (49.3)	34 (50.7)		
5. Insulin Resistance					
	Yes	12 (17.9)	5 (7.5)	3.3 (1.01-10.83)	0.042
	No	21 (31.3)	29 (43.3)		
	Total	33 (49.3)	34 (50.7)		
6. Dyslipidemia					
	Yes	27 (40.3)	17 (25.4)	4.5 (1.48-13.66)	0.006
	No	6 (9.0)	17 (25.5)		
	Total	33 (49.3)	34 (50.7)		
7. Hypertriglyceridemia					
	Yes	25 (37.3)	5 (7.5)	26.1 (7.13-95.52)	<0.001
	No	8 (11.9)	29 (43.3)		
	Total	33 (49.3)	34 (50.7)		
8. Hipo-HDL					
	Yes	17 (25.4)	6 (9.0)	4.9 (1.62-15.12)	0.004
	No	16 (23.9)	28 (41.8)		
	Total	33 (49.3)	34 (50.7)		
9. Hypertension					
	Yes	15 (22.4)	4 (6.0)	6.2 (1.79-21.77)	0.002
	No	18(26.9)	30 (44.8)		
	Total	33 (49.3)	34 (50.7)		
10. Metabolic syndrome					
	Yes	25 (37.3)	4 (6)	23.4 (6.30-87.06)	<0.001
	No	8 (11.9)	30 (44.8)		
	Total	33 (49.2)	34 (50.8)		

Table 4. Logistic Regression Test Risk Factors Against NAFLD

No	Variables	B	SE	Sig	OR	CI95%
1.	Hipertriglyceridemia	2.16	0.70	0.002	8.7	2.20-34.44
2.	Visceral Fat Deposition	1.77	0.73	0.016	5.8	1.39-24.82

Carbohydrates and Fats, as well as visceral fat deposition According to NAFLD Degrees

Carbohydrate and fat intake (grams), as well as visceral fat deposition (score) increase with increasing degree of NAFLD (Figure 2).

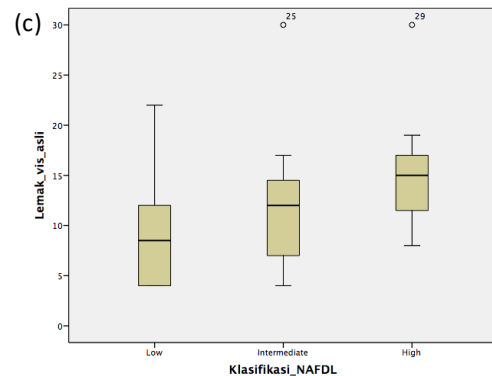
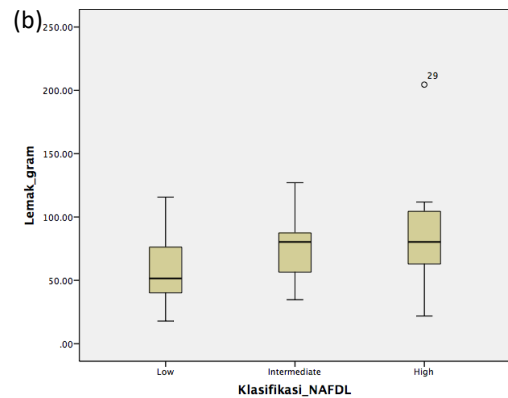
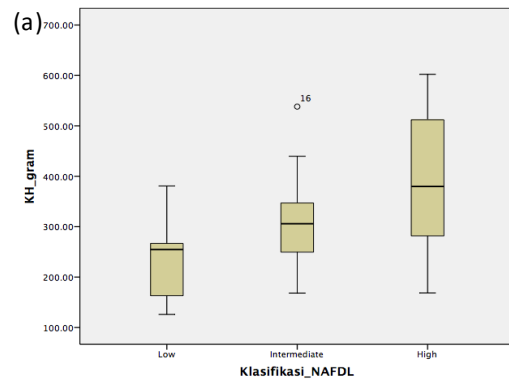


Figure 2. Correlation Between (a) Carbohydrate, (b) Fat Intake, and (c) Visceral Fat Deposition with NAFLD Degrees

Risk Factors of NAFLD Severity

The severity of NAFLD from the in this study is divided into 3 groups, but on the risk factor analysis for NAFLD severity we classify them into 2 groups, where the intermediate and high degree groups we make become the severity of the high degree NAFLD. While the low degree (low) we use for comparison.

In determining the severity risk factor of NAFLD, the determination of the classification of carbohydrate and fat intake, as well as fat classification is based on the results of Receiver Operating Characteristic (ROC) analysis (Figure 3).

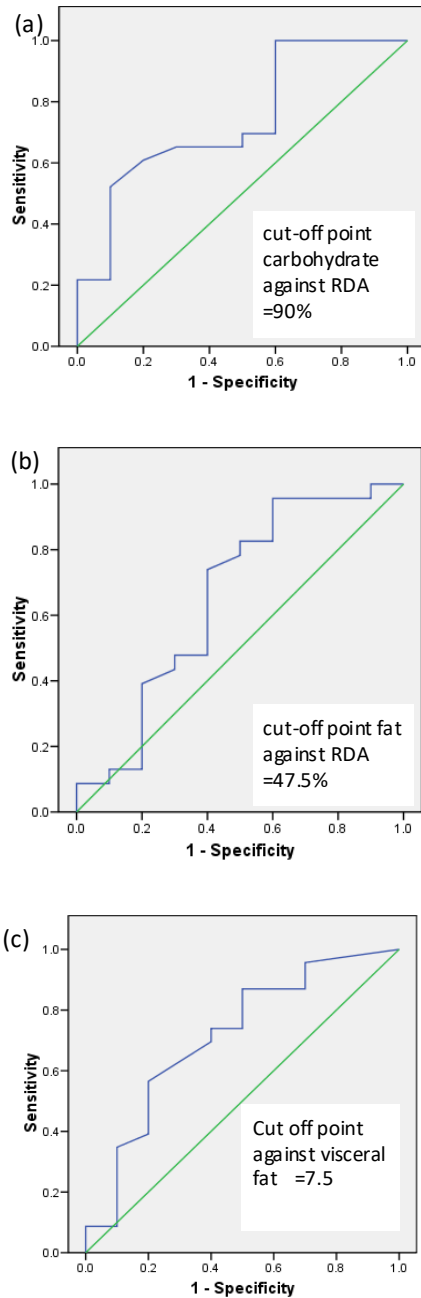


Figure 3. (a) Carbohydrate Intake against RDA. (b) Fat Intake against RDA. (c) Visceral Fat Deposition, on Case Group

Based on bivariate analysis showed high carbohydrate (>90% RDA) and fat intake (>47,5% RDA), visceral fat deposition score >7,5% were proved as risk factors of NASH or severity of NAFLD (table 5).

However, in multivariate analysis, only high fat intake (>47,5%) proved as a risk factor for NAFLD severity or incidence of NASH (p= 0.008).

Table 5. Risk Factors Carbohydrate and Fat intake, Visceral Fat Deposition Score Against the severity of NAFLD

No	Variabel	NAFLD Severe degree N (%)	NAFLD Mild degree N (%)	OR (CI 95%)	p
1. Carbohydrate intake					
	High (>90%)	12 (36.4)	1 (3.0)	9.8 (1.06-90.58)	0.023
	Low (≤90%)	11 (33.3)	9 (27.3)		
	Total	23 (69.7)	10 (30.3)		
2. Fat Intake					
	High (>47.5%)	22 (66.7)	6 (18.2)	14.6 (1.37-156.88)	0.009
	Low (≤47.5%)	1 (3.0)	4 (12.1)		
	Total	23 (69.7)	10 (30.3)		
3. Risk Factor of Visceral Fat Deposition Score					
	High (>7.5%)	20 (60.6)	5 (15.2)	6.6 (1.17-37.78)	0.023
	Low (≤7.5%)	3 (9.1)	5 (15.2)		
	Total	23 (69.7)	34 (30.3)		

On bivariate analysis for clinical markers on NAFLD severity showed that visceral obesity and metabolic syndrome were as risk factors for NAFLD severity consecutively (OR 7.1 (1.35-37.55) and OR 6.6 (1.17-37.7)) (table 6).

Table 6. Risk Factor of Clinical Markers on NAFLD Severity

No	Variabel	NAFLD Severe degree N (%)	NAFLD Severe degree N (%)	OR (CI 95%)	p
1. Central Obesity					
	Yes	19 (57.6)	4 (12.1)	7.1 (1.35-37.55)	0.014
	No	4 (17.4)	6 (18.2)		
	Total	23 (69.7)	10 (30.3)		
2. Insulin Resistance					
	Yes	10 (30.3)	2 (6.1)	3.0 (0.53-17.7)	0.198
	No	13 (39.4)	8 (24.2)		
	Total	23 (69.7)	10 (30.3)		
3. Dyslipidemia					
	Yes	18 (54.5)	9 (27.2)	0.4 (0.4-3.9)	0.422
	No	5 (15.1)	1 (3.2)		
	Total	23 (69.7)	10 (30.3)		
4. Hypertriglyceridemia					
	Yes	18 (54.5)	9 (27.2)	0.4 (0.4-3.9)	0.422
	No	5 (15.1)	1 (3.2)		
	Total	23 (69.7)	10 (30.3)		
5. Hipo-HDL					
	Yes	12 (36.4)	5 (15.2)	1.09 (0.24-4.81)	0.909
	No	11 (33.3)	5 (15.2)		
	Total	23 (69.7)	34 (30.3)		
6. Hypertension					
	Yes	12 (36.4)	3 (9.1)	2.5 (0.52-12.36)	0.240
	No	11 (33.3)	7 (21.2)		
	Total	23 (69.7)	34 (30.3)		
7. Metabolic Syndrome					
	Yes	20 (60.6)	5 (15.2)	6.6 (1.17-37.7)	0.023
	No	3 (9.1)	5 (15.2)		
	Total	23 (69.7)	34 (30.3)		

DISCUSSION

The result of statistical test of the characteristics between two groups (age, sex, occupation, income, and physical activity) showed no difference, this indicated the initial condition in both groups is alike except in the variable of central obesity, insulin resistance, dyslipidemia, hypertriglyceridemia, hypo-HDL, hypertension, and metabolic syndrome.

The mean of age on the NAFLD group was 43.3 years, while the control was 40.8 years. Age is one of

the predisposing factors for NAFLD. The aging process will increase the oxidative stress that plays a role in the process of NAFLD

Insulin resistance is a risk factor for the incidence of NAFLD (OR 3.3, CI95% 1.01-10.83). It may cause metabolic disorders of some macro nutrients due to hepatic insulin resistance resulting in increased plasma glucose concentration and increased VLDL production and cause hypertriglyceridemia. Triglycerides will accumulate in the hepatocyte so that NAFLD will occur. This result is consistent with Cortez et al's 2006 study which suggests that there is a significant difference ($p < 0.01$) between NAFLD patients in the Fatty Liver Disease with NAFLD spectrum compared with healthy control.⁴⁸ Studies by Hannah et al, 2014 suggest that there was a significant difference ($p < 0.05$) between NAFLD patients on simple steatosis spectrum and NAFLD compared with healthy control.⁴⁹ This result is directly proportional to Lei et al's 2012 study, which states that RI is one of the risk factors for NAFLD with 4.96 times compared with healthy control (95% CI 1.10-22.266).⁵⁰

Each of the metabolic syndrome components such as central obesity, dyslipidemia, hipertrigliceridemia, hipo-HDL, insulin resistance and hypertension as well as stand-alone variables as the variables studied are risk factors for NAFLD events. Metabolic syndrome is the risk factor for the incidence (OR 23.4, CI95% 6.30-87.06) and severity of NAFLD (OR 6.6, CI95% 1.17-37.7), as well as central obesity (CI95% 1.35-37.55).

Multivariate analysis showed that hypertriglyceridemia and fat intake were independent risk factor for NAFLD incidence. These results fit the theoretical basis that dyslipidemia, in particular hypertriglyceridemia, can result from a high fat intake and may increase the risk of incidence and severity of NAFLD.^{31,32} The results of this study are consistent with Lei et al's 2012 study of multivariate tests suggesting that dyslipidemia increased the risk of incidence of NAFLD 4.405 times compared with healthy control ($p = 0.004$).⁵⁰ Similarly, Hannah et al's study, 2014, stated that there was a significant difference in the presence of dyslipidemia ($p = 0.05$) between NAFLD patients with healthy control.⁴⁹ There was a 4-11 times individual risk with metabolic syndrome to suffer NAFLD than healthy individuals.³³

Descriptive analysis (box plot) showed that average carbohydrate intake in NAFLD group was higher than control. Average fat intake in NAFLD group was also higher than control. The results were consistent with Cortez's 2012 study suggesting that fat and carbohydrate intake in NAFLD patients was higher than in controls and significantly different.⁴⁸ The Israeli study by Sagi et al, 2007 showed different results, whereas fat intake in NAFLD and healthy control, showed no difference ($p = 0.76$) similar with carbohydrate intake also no difference ($p = 0.6$). Although the intake of both groups was not significantly different, the multivariate analysis showed that carbohydrate intake (from soft drinks)

was a risk factor for NAFLD (OR 1.45, CI95% 1.13-1.85).⁷ After classification based on ROC curve cut-off, intake of carbohydrate and fat to RDA according to individuals, showed a relationship of fat and carbohydrate intake with the incidence and severity of NAFLD. Interveriable test showed that high carbohydrate and fat intake increased risk of NAFLD incidence (OR = 7.8, 95% CI 2.43-25.45), (OR = 5.9, CI95% 2.00-17.57). High carbohydrate and fat intakes increase the risk of NAFLD severity (OR = 9.8, 95% CI 1.06-90.58), (OR = 14.6, CI95% 1.37-156.88).

Increased intake of carbohydrate and fat is closely related to elevated blood glucose, FFA, insulin concentration. A person with a higher fat intake (>37%), would increase the risk of NAFLD by 2.51 times compared with healthy control.⁵¹ High fat intake along with IR through LpL and the genetic changes of the fat metabolism pathway will increase the blood FFA so that the concentration of TG and CE in the liver and fat in the muscle increases and accumulates. Excessive carbohydrate intake combined with IR will also increase the stimulation of *de novo lipogenesis* so that glucose in the liver increases. Glucose will be converted to glycogen and FFA. Glycogen will increase FC and increase CE, whereas FFA will increase TG and CE and eventually lead to excessive accumulation of TG in the liver and infiltrate hepatocytes and then form NAFLD.¹² FFA also increase DAG (diacylglycerol). Accumulation of DAG interferes with insulin signaling in the liver by activation of protein kinase-C. Ceramide on the spingolipid membrane will also increase FFA in the liver and contribute to insulin resistance through activation of proinflammatory cytokines, cell apoptosis and increased endoplasmic reticulum stress.⁵²

High visceral fat deposition is a risk factor for the incidence and severity of NAFLD (OR 50.7, 95% CI 6.16-418.09), (OR 6.6, 95% CI 1.17-37.78). The visceral fat deposition in a person with obesity and overweight will increase adipose tissue, where adipose tissue is the site of macrophage accumulation which is the main source of TNF- α expression. TNF- α secretion then stimulates MCP-1 by preadipocytes. TNF- α , interleukin-6, interleukin -1 β are secreted by activated macrophages.¹² These proinflammatory cytokines will lead to stem cell activation and cause fibrosis, in addition the kupfer cells will also be activated to produce proteins acute phase such as CRP and protrombosis molecules all of which will result in liver tissue damage (Hit 2).¹² Increased visceral fat deposition boosting proinflammatory cytokines will also increase ROS, affect lipid peroxidation and decrease adiponectin. Adiponectin is an anti-inflammatory adipocytokine that modulates the effects of insulin. Adiponectin levels are negatively correlated with fasting sugar levels, and insulin resistance and TG levels. Decreased adiponectin will promote more intensive necroinflammation in NAFLD. High necroinflammation will increase ROS and lead to increased peroxidation of further pro-inflammatory lipid and

cytokine products leading to cell death, inflammation, and fibrosis.⁵³

The result of multivariate test stated that hypertriglyceridemia was the independent risk factor for the incidence of NAFLD. Hypertriglyceride is the beginning of triglyceride buildup in the liver which will damage the structure and function of the liver so that it develops into various diseases like IR, DM, dyslipidemia, and metabolic syndrome which is a pathogenesis that can not be separated with NAFLD disease. Nevertheless, the above risk factors after the regression test contributed 55.3%, and as much as 44.7% may be influenced by other factors not investigated but related to NAFLD.

The overall risk factors above must be addressed comprehensively. Handling either medically or non-medically as nutrition therapy as indicated or with lifestyle changes, and good dietary regulation, and education to the patient as a follow-up to prevent NAFLD should be done.

The limitations of this study are: data was taken from a single measurement, so that can not always describe a relationship in the longer time. FFQ method, has weakness, including recall bias, and underestimate or even overestimate the size of household food subject intake. FFQ can not provide information about the intake of simple carbohydrates, fructose, or certain antioxidant intake that may be related to the process of NAFLD.

CONCLUSION

High carbohydrate intake, fat intake, and high visceral fat deposition are risk factors of the incidence and severity of NAFLD. Hypertriglyceridemia and fat intake are the most important risk factor of NAFLD incidence and severity, respectively.

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