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Original Article

Association between Dyslipidemia and the Prevalence of Colon Polyps Based on a Health Evaluation of Subjects at a Hospital

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Background: Colonic neoplasm is associated with western diet intake and physical inactivity. These life styles are also risk factors for dyslipidemia and metabolic syndrome. The aim of this study was to evaluate the association between dyslipidemia and the prevalence of colon polyps including colon adenoma as a precancerous lesion of colonic neoplasms.

Methods: We selected subjects undergoing a colonoscopy for health screening at the Health Promotion Center of Eulji General Hospital from January 2006 to June 2010. Subjects with histories of cancers, dyslipidemia treatment, and other intestinal diseases like Crohn's disease and ulcerative colitis were excluded. The total numbers of subjects included in the study was 605. Chi-square test and t-test and were used for the analysis. Additionally we used multivariate logistic regression to adjust for sex, age, smoking, drinking, and other risk factors.

Results: The prevalence of colon polyps was 48.70% and 28.05% in males and females, respectively. When adjusting for variables that included age, body mass index, hypertension, diabetes mellitus, smoking, drinking, and exercise, dyslipidemia was not significantly associated with the prevalence of colon polyps. However upon analyzing adenomatous colon polyps in men, dyslipidemias due to triglycerides and high density lipoproteins were significant factors (odds ratio [OR], 2.13; confidence interval [CI], 1.14 to 3.98; OR, 2.24; CI, 1.15 to 4.34, respectively).

Conclusion: Dyslipidemia was not a significant factor in the prevalence of colon polyps. However it had a significant association with the prevalence of adenomatous colon polyps in men.

Keywords: Colonic Polyps; Serum Cholesterol; Dyslipidemias

INTRODUCTION

The incidence of colon cancer is related to the consumption of a meat-based diet and reduced levels of physical activity. The incidence of colon cancer has increased consequently to the westernization of South Korean dietary practices. Accordingly, in 2009 colon cancer was the third most common cancer in South Korea, and 47 of every 100,000 men and 25.6 of every 100,000 women developed this disease. These statistics demonstrate that the incidence of colon cancer increased significantly from the

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2007 incidence rates of 44 cases per 100,000 men and 24.8 cases per 100,000 women in Korea.[1)]

Colon cancer is known to result from genetic mutations in colon polyps; however, less than 1% of these adenomatous polyps have been known to progress to colon cancer. The risk of cancer progression increases along with larger polyp sizes, more villous matter, and higher grade dysplasia.[2)] Consequently, early detection and treatment of adenomatous polyps are crucial to reducing the incidence and morbidity of colon cancer.[3)] The prevalence of adenomatous colon polyps varies among nations; among symptomless patients, the probability of discovering an adenomatous polyp is approximately 10% via sigmoidoscopy and more than 25% via colonoscopy.[4)] However, the incidence of colon cancer is less than 1%.[4)]

Studies regarding colon cancer, colon polyps, and the associated risk factors are abundant. In a prospective cross-sectional study of 3,121 patients aged 50 to 75 years during the period of 2002 to 2003, histories of direct familial colon cancer, smoking, and above-moderate alcohol consumption were found to increase the risk of colon cancer. In contrast, consumption of fiber, vitamin D, and non-steroidal anti-inflammatory drugs reduced the risk of colon cancer.[5,6)] Obesity increased the risk of adenomatous colon polyps in young adults.[7)] In addition, the waist-to-hip ratios of both men and women were found to be significantly higher in patients with colon polyps. Furthermore, an increase in weekly exercise duration, frequency, and intensity was found to reduce the risk of colon polyps.[8)] In the European Prospective Investigation into Cancer and Nutrition study, the results determined from 984 patients demonstrated that the group with higher rates of obesity had a 55% increase in the risk of colon cancer.[9)]

Lifestyle factors such as obesity, increased fat intake, a lack of exercise, alcohol consumption, and smoking are all risk factors for metabolic syndrome. Some studies have shown that the incidence of adenomatous colon polyps is associated with obesity, hyperinsulinemia, and diabetic medication use.[10)] However, studies on the association between adenomatous colon polyps and other metabolic syndrome factors such as triglyceride and high density lipoprotein (HDL) cholesterol levels have produced conflicting results. The present study sought to examine the association between dyslipidemia and the prevalence of colon polyps, in order to provide evidence for the early detection of colon polyps and serum cholesterol regulation.

METHODS

1. Subjects

The subjects were selected from a group of individuals who underwent health evaluations, including colonoscopies, from January 2006 to June 2010 at the Health Promotion Center of Eulji General Hospital. From these individuals, those who did not fully complete the questionnaire, and those who had received dyslipidemia treatments (n = 41), or cancer treatments (n = 11) were excluded. A total of 605 people were selected as study subjects.

2. Procedures

1) Selection and categorization of colon polyps

Colonoscopic examinations were conducted by gastroenterologists at our clinic. Diagnosis of colon polyps was made by the physician through visual inspection. Based on the biopsies, colon polyps were categorized as hyperplastic polyp, adenomatous polyp or advanced adenoma, etc. As descriptions of polyp size were inconsistent, polyp size data were not included in this study. Polyps were defined as advanced adenoma if the biopsy revealed high-grade villous matter or dysplasia.

2) Blood test and measurements of anthropometric parameters

The clinical evaluation recipients were instructed to fast for 8 hours prior to the examination. The subjects' heights and weights were evaluated with a digital body measurer (NeoGMTEC Co., Ltd., Seoul, Korea), which was provided by the clinic. The waist size was measured on a line that bisected the umbilicus and was parallel to the ground. The hip size was determined by measuring the location perceived to yield the greatest circumference at a significance of 0.1 cm. The body mass index was measured with a Bizmedic device (Bizmedic Co., Ltd., Seoul, Korea) and reported in units of kg/m^2. Blood pressure levels were measured with an automatic sphygmomanometer after the subjects had rested for a minimum of 5 minutes.

3) Survey

The health evaluation subjects completed a self-reported questionnaire after providing prior consent. The subjects provided information regarding their medical histories, current medication use, family histories, and alcohol consumption, smoking, exercise, physical activity, and dietary habits.

3. Research Methods

1) Explanation of the variables

The explanatory variables were the presence or absence of colon polyps. Adenomatous colon polyps were selected for a subgroup analysis. The independent variable was the serum cholesterol level or dyslipidemia according to the standards of the National Cholesterol Education Program's Adult Treatment Panel III. Dyslipidemia was defined as a triglyceride level ≥ 150 mg/dL, a total cholesterol level ≥ 200 mg/dL, a low density lipoprotein (LDL) cholesterol level ≥ 100 mg/dL, or a HDL cholesterol level < 40 mg/dL for men and < 50 mg/dL for women. Of the adjusted variables related to the prevalence of colon polyps, the subject' s age, sex, body mass index, diabetes status (fasting glucose level ≥ 126 mg/dL, hemoglobin A1c (HbA1C) ≥ 6.5%, or current use of a diabetes treatment), hypertension status (systolic blood pressure ≥ 140 mm Hg, diastolic blood pressure ≥ 90 mm Hg, or current use of a hypertension treatment), smoking history (non-smoker, ex-smoker, or current smoker), alcohol consumption history (none, ex, or current), and exercise habits (≤ 3 or > 3 times per week) were included in the analysis.

2) Statistical methods

We initially compared baseline characteristics by sexes. To investigate the differences in risk factors based on the presence or absence of colon polyps, the continuous and categorical variables were evaluated with t-test and chi-square tests, respectively. The result was considered statistically significant for $P < 0.05$. A multivariate logistic regression method was used to determine the risk values after adjusting for other factors that might influence the incidence of polyps. Statistical analysis was conducted with the SPSS ver. 12.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

A total of 384 men (63.5%) and 221 women (36.5%) were included as study subjects (Table 1). The mean age of the subjects was 48 years, and there was no significant difference between the sexes regarding this variable. The mean levels of body mass index, waist size, blood pressure, triglycerides, and LDL cholesterol were significantly higher in men than in women. With respect to lifestyle habits, smoking and alcohol consumption rates were 41.24% and 76.68% among the men and 7.46% and 44.61% among the women, respectively. The men were also found to have a significantly higher incidence of colon polyps (48.70%) than the women (28.05%).

A univariate analysis was conducted to analyze the colon polyp risk factors with respect to sex differences (Table 2). The mean age of the colon polyp group was older than that of the non-colon polyp group. Within the colon polyp group, 52.94% of the men and 56.45% of the women were ≥ 50 years of age. Among the men, the mean levels of systolic blood pressure, diastolic blood pressure, and hemoglobin were significantly higher in the colon polyp group; however, among the women, there were no significant differences in the levels of the above-mentioned variables between the groups with and without colon polyps. Furthermore, although the colon polyp group exhibited a higher percentage of dyslipidemia, including triglycerides, total cholesterol, HDL cholesterol, and LDL cholesterol levels, the differences in these variable values did not reach statistical significance. Univariate analysis did not reveal statistically significant results with respect to the relationships among the sex-differentiated smoking, alcohol consumption, and exercise rates by colon polyps. Among the women, however, menopause rates were significantly higher in the colon polyp group than in the non-polyp group.

To investigate the association between the prevalence of colon polyps and dyslipidemia, multivariate logistic regression was used to adjust for the variables of age, body mass index, hypertension, diabetes mellitus, smoking, drinking, and exercise (Table 3). There were no significant associations between the prevalence of colon polyps and dyslipidemia indicators such as triglycerides, HDL cholesterol, LDL cholesterol, and total cholesterol levels. Subsequently, the hyperplastic polyp group was assimilated into the non-polyp group (474 people) to define

Table 1. Baseline characteristics of subjects

Variable	Men (n = 384)	Women (n = 221)	P-value*
Age (y)	48.28 ± 9.68	48.54 ± 10.92	0.76
Body mass index (kg/m^2)	24.47 ± 2.95	22.90 ± 3.03	<0.001
Waist (cm)	86.58 ± 7.96	78.78 ± 8.31	<0.001
Systolic blood pressure (mm Hg)	123.66 ± 12.40	118.04 ± 14.00	<0.001
Diastolic blood pressure (mm Hg)	74.31 ± 9.01	70.73 ± 10.30	<0.001
Total cholesterol (mg/dL)	192.05 ± 32.76	190.86 ± 34.51	0.68
Triglyceride (mg/dL)	143.67 ± 100.49	91.77 ± 57.48	<0.001
Low density lipoprotein cholesterol (mg/dL)	120.59 ± 30.21	116.75 ± 32.58	<0.001
High density lipoprotein cholesterol (mg/dL)	47.68 ± 10.99	56.68 ± 13.12	0.15
Fasting glucose (mg/dL)	97.93 ± 20.87	95.72 ± 27.03	0.30
Hemoglobin A1c (%)	5.73 ± 0.80	5.69 ± 1.01	0.64
Education			<0.001
≤Primary school	19 (5.32)	30 (15.08)	
≤Middle school	26 (7.28)	22 (11.06)	
≤High school	110 (30.81)	74 (37.19)	
≥College graduate	202 (56.58)	73 (36.68)	
Income (million won)			0.03
≤300	103 (30.93)	66 (38.82)	
≤600	167 (50.15)	64 (37.65)	
>600	63 (18.92)	40 (23.53)	
Marital status			0.01
With spouse	338 (91.60)	171 (84.24)	
Without spouse	31 (8.40)	32 (15.76)	
Hypertension treatment			0.55
No	335 (87.24)	189 (84.24)	
Yes	49 (12.76)	32 (15.76)	
Diabetes mellitus treatment			0.96
No	368 (95.83)	212 (95.93)	
Yes	16 (4.17)	9 (4.07)	
Family cancer history			0.63
Colon	17 (18.89)	7 (15.56)	
Non-colon	73 (81.11)	38 (84.44)	
Smoking			<0.001
Non	145 (39.08)	183 (91.04)	
Ex	73 (19.68)	3 (1.49)	
Current	153 (41.24)	15 (7.46)	
Alcohol			<0.001
Non	88 (23.22)	113 (55.39)	
Current	291 (76.68)	91 (44.61)	
Exercise (d)			0.004
≤3	297 (88.39)	157 (84.86)	
>3	39 (11.61)	28 (15.14)	
Physical activity			0.038
Mild	217 (73.81)	123 (69.10)	
Moderate	50 (17.01)	47 (26.40)	
Severe	27 (9.18)	8 (4.49)	
Polyp			<0.001
No	197 (51.30)	159 (71.95)	
Yes	187 (48.70)	62 (28.05)	
Hyperplastic	83 (44.39)	35 (56.45)	
Adenomatous	104 (55.61)	27 (43.55)	

Values are presented as mean ± SD or number (%).

*Chi-square test was used for categorical variables and t-test was used for continuous variables.

Table 2. Risk factors of colon polyps

Variable	Men			Women		
	Polyp (+)	Polyp (-)	P-value*	Polyp (+)	Polyp (-)	P-value*
Age (y)	51.46 ± 9.85	45.26 ± 8.50	<0.001	52.40 ± 9.83	47.04 ± 10.98	<0.001
<50%	88 (47.06)	145 (73.60)		27 (43.55)	90 (56.60)	
≥50%	99 (52.94)	52 (26.40)	<0.001	35 (56.45)	69 (43.40)	0.08
Body mass index (kg/m^2)	24.63 ± 3.14	24.32 ± 2.76	0.30	23.30 ± 3.11	22.74 ± 2.99	0.22
Waist (cm)	87.13 ± 8.44	86.06 ± 7.45	0.19	80.11 ± 8.53	78.27 ± 8.19	0.15
Systolic blood pressure (mm Hg)	125.29 ± 12.04	122.13 ± 12.56	0.01	119.16 ± 12.89	117.59 ± 14.43	0.43
Diastolic blood pressure (mm Hg)	75.28 ± 8.65	73.41 ± 9.27	0.04	71.30 ± 9.42	70.52 ± 10.64	0.60
Hemoglobin A1c (%)	5.86 ± 0.89	5.60 ± 0.68	<0.001	5.70 ± 0.57	5.68 ± 1.14	0.86
Total cholesterol (mg/dL)			0.91			0.09
<200	111 (59.36)	118 (59.90)		33 (53.23)	104 (65.41)	
≥200	76 (40.64)	79 (40.10)		29 (46.77)	55 (34.59)	
Triglyceride (mg/dL)			0.86			0.52
<150	124 (66.31)	129 (65.48)		53 (85.48)	141 (88.68)	
≥150	63 (65.48)	68 (34.52)		9 (14.52)	18 (11.32)	
Low density lipoprotein cholesterol (mg/dL)			0.57			0.12
<100	44 (23.53)	41 (21.13)		15 (24.19)	56 (35.22)	
≥100	143 (76.47)	153 (78.87)		47 (75.81)	103 (64.78)	
High density lipoprotein cholesterol (mg/dL)			0.17			0.51
Low†	51 (27.27)	42 (21.32)		22 (35.48)	49 (30.82)	
High‡	136 (72.73)	155 (78.68)		40 (64.52)	110 (69.18)	
Smoking			0.51			0.62
Non	67 (37.22)	78 (40.84)		53 (94.64)	130 (89.66)	
Ex	32 (17.78)	41 (21.46)		1 (1.79)	2 (1.38)	
Current	81 (45)	72 (37.70)		2 (3.57)	13 (8.97)	
Alcohol			0.43			0.06
Non	44 (23.91)	36 (18.46)		37 (67.27)	72 (48.32)	
Ex	5 (2.72)	3 (1.54)		1 (1.82)	3 (2.01)	
Current	135 (73.37)	156 (80)		17 (30.91)	74 (49.67)	
Excercise			0.48			0.07
≤3/wk	140 (86.42)	157 (90.23)		35 (76.09)	122 (87.77)	
>3/wk	22 (13.58)	17 (9.77)		11 (23.91)	17 (12.23)	
Physical activity			0.28			0.14
Mild	104 (72.73)	113 (74.83)		29 (69.05)	94 (69.11)	
Moderate	25 (17.48)	25 (16.56)		12 (28.57)	35 (25.74)	
Severe	14 (9.79)	13 (8.61)		1 (2.38)	7 (5.15)	
Menopause						0.04
No				25 (22.52)	32 (35.56)	
Yes				86 (77.48)	58 (64.44)	

Values are presented as mean ± SD or number (%).

*Chi-square test was used for categorical variables and t-test was used for continuous variables. †<40 for men, <50 for women. ‡≥40 for men, ≥50 for women.

Table 3. The associations between dyslipidemia and colon polyps

Variable (mg/dL)	Men			Women		
	aOR	95% CI	P-value	aOR	95% CI	P-value
Triglycerides						
<150	1.00	Reference		1.00	Reference	
≥150	1.21	0.69–2.13	0.50	1.03	0.30–3.53	0.96
High density lipoprotein cholesterol						
High*	1.00	Reference		1.00	Reference	
Low†	1.52	0.82–2.80	0.19	0.84	0.36–1.93	0.68
Low density lipoprotein cholesterol						
<100	1.00	Reference		1.00	Reference	
≥100	0.96	0.53–1.74	0.89	1.02	0.44–2.41	0.96
Total cholesterol						
<200	1.00	Reference		1.00	Reference	
≥200	1.24	0.74–2.07	0.42	0.99	0.45–2.22	0.99

Dyslipidemia was defined by National Cholesterol Education Program's Adult Treatment Panel III criteria. Adjusted variables include age, body mass index, hypertension, diabetes mellitus, alcohol, smoking, and exercise. Multiple logistic regression was used.
aOR: adjusted odds ratio, CI: confidence interval.
*≥40 for men, ≥50 for women. †<40 for men, <50 for women.

Table 4. The associations between dyslipidemia and adenomatous colon polyps

Variable (mg/dL)	Men			Women		
	aOR	95% CI	P-value	aOR	95% CI	P-value
Triglycerides						
<150	1.00	Reference		1.00	Reference	
≥150	2.13	1.14–3.98	0.02	1.03	0.17–6.24	0.97
High density lipoprotein cholesterol						
High*	1.00	Reference		1.00	Reference	
Low†	2.24	1.15–4.34	0.02	0.99	0.33–3.05	0.99
Low density lipoprotein cholesterol						
<100	1.00	Reference		1.00	Reference	
≥100	0.96	0.49–1.86	0.90	1.06	0.33–3.41	0.92
Total cholesterol						
<200	1.00	Reference		1.00	Reference	
≥200	1.07	0.60–1.91	0.82	0.99	0.33–2.93	0.99

Dyslipidemia was defined by National Cholesterol Education Program's Adult Treatment Panel III criteria. Adjusted variables include age, body mass index, hypertension, diabetes mellitus, alcohol, smoking, and exercise. Multiple logistic regression was used.
aOR: adjusted odds ratio, CI: confidence interval.
*≥40 for men, ≥50 for women. †<40 for men, <50 for women.

the adenomatous polyp group (131 people) as a precancerous lesion of the colon. The analysis was conducted using the same method that had been used previously to analyze the association between adenomatous colon polyps and dyslipidemia (Table 4). Among the men, the risk of adenomatous colon polyp prevalence significantly increased by twofold (odds ratio [OR], 2.13; 95% confidence interval [CI], 1.14 to 3.98; OR, 2.24; 95% CI, 1.15 to 4.34) when triglyceride levels were ≥ 150 mg/dL or HDL cholesterol levels were < 40 mg/dL. In contrast, no significant association was found between the prevalence of adenomatous colon polyps and dyslipidemia in the women.

DISCUSSION

The present study aimed to investigate the association between the prevalence of colon polyps and dyslipidemia. A case-control study by Lee et al.[11] demonstrated that patient groups with and those without colorectal adenoma exhibited no significant differences in average serum cholesterol levels. Furthermore, the risk of colorectal adenoma occurrence increased as body mass index increased. According to a case-control study of young Korean adult men who visited a particular health screening and promotion center in South Korea, men with adenomatous colon polyps were comparatively older, of lower economic and educational status, and exhibited higher smoking rates, body mass index, waist-to-hip ratio, and serum triglyceride levels. In addition, the HDL cholesterol levels of the men with adenomatous colon polyps were lower than those of normal men.[12] Our study showed that dyslipidemia, which was defined by triglyceride levels ≥ 150 mg/dL or HDL cholesterol levels < 40 mg/dL in men, increased the risk of adenomatous colon polyps, thus being consistent with the results of previous studies.

Smoking and alcohol consumption have been reported to increase the incidence of colon polyps.[13] According to a 2002 study by Kim et al.,[6] the risk of colon polyps increased by 2.5-fold in subjects who smoked ≥ 20 pack-years relative to non-smokers. Similarly, the risk of colon polyps increased by 2.8-fold in subjects that consumed alcohol ≥ 4 times per week relative to non-drinkers. The present study corroborated these earlier results by demonstrating via multivariate logistic regression that smokers were twofold more likely to develop colon polyps relative to non-smokers (data not shown). As a result, smoking is clearly an important risk factor with respect to incidence of colon polyps.

Among the women, those in the colon polyp group were more likely to have entered menopause, compared with the control group (P = 0.04); LDL cholesterol levels were also significantly higher in the colon polyp group. In an analysis of dyslipidemia and mean serum cholesterol levels according to menopause presence or absence, the menopausal group had significantly higher dyslipidemia prevalence and mean levels of body mass index, blood pressure, waist size, and total cholesterol, triglycerides, LDL cholesterol, and HbA1C levels. In addition, HDL cholesterol levels were significantly lower in the menopausal women than in the premenopausal women. Consequently, a multivariate logistic regression analysis was conducted to investigate the association among menopause, colon polyp prevalence, and dyslipidemia. After adjusting for age, menopause and dyslipidemia were not found to increase the risk of colon polyp prevalence. As a result, the fact that the women with colon polyps had higher LDL cholesterol levels than did the women without colon polyps might be regarded as an effect of the increased age in the former group.

Bird et al.[14] reported a case-control study of serum cholesterol levels and the incidence of left colon and rectal adenomatous polyps; in this study, the patient group exhibited higher triglyceride levels than did the control group. However, there were no significant differences between the groups with regard to levels of other forms of cholesterol, and no correlation was found between serum cholesterol levels and adenomatous polyp locations. The above-mentioned study also reported that triglyceride levels were significantly lower in the group that participated in high levels of exercise. Although there were no significant differences, triglyceride levels were found to be higher in the smoker group than in the non-smoker group. According to a case-control study by Yamada et al.[15] regarding colorectal carcinoma in situ, after adjusting for age and sex, the total cholesterol levels and the triglyceride levels were significantly higher in the patient group than in the control group; the difference was more with regard to triglyceride levels. Moreover the risk of colorectal carcinoma *in situ* increased about threefold when triglyceride levels were more than 150 mg/dL. The study by Yamada et al.[15] is meaningful because it is the first to investigate the relationship between hypertriglyceridemia and colon cancer.

In addition, this study proposes that an increase in bile release in the colon, which was related to an increase in serum triglyceride levels, was among the factors responsible for the incidence of colorectal carcinoma *in situ*. However, hyperinsulinemia and insulin resistance mechanisms were suggested to be more meaningful.

In a German study conducted in 1993, high levels of very LDL were found to increase the risk of adenomatous colon polyps by twofold or threefold.[16] A 2005 study by Wang, which incorporated sigmoidoscopy, also demonstrated that patients with adenomatous polyps had higher triglyceride and lower HDL cholesterol levels relative to the control group.[17] Additional studies have shown that a reduction in HDL cholesterol levels, a factor of metabolic syndrome, slightly increases risk of adenomatous colon polyps and, consequently, of colon cancer.[10] We also confirmed that after isolating the adenomatous colon polyp group, the prevalence of adenomatous colon polyps was found to increase by twofold when HDL cholesterol levels were < 40 mg/dL. In the 2010 case-control study by Tsilidis et al.,[18] the colorectal adenoma patient group exhibited a significantly higher proportion of patients who were also receiving treatment for diabetes. However, colorectal adenoma was not significantly associated with triglyceride and cholesterol levels.[18] In a cross-sectional study of 731 subjects with adenomatous colon polyps and 1,800 control subjects, metabolic syndrome significantly increased the risk of colorectal adenoma (OR, 1.51; 95% CI, 1.18 to 1.93).[19] In the present study, we also demonstrated that metabolic syndrome increased the prevalence of colon polyps, although this increase was not statistically significant (data not shown).

It has been suggested that serum cholesterol may stimulate carcinogenesis.[20] Our data did not show a significant association between the prevalence of colon polyps and serum cholesterol levels. However, a study that compared subjects with adenomatous polyps and those with no polyps or hyperplastic polyps demonstrated that dyslipidemia of an increase in triglyceride levels or a decrease in HDL cholesterol levels was significantly associated with prevalence of adenomatous colon polyps. Therefore, we expected that hypertriglyceridemia-affected neoplastic cell growth would be more pronounced in the presence of adenomatous polyps. In addition, we concluded that the association between lower HDL cholesterol levels and adenomatous polyps resulted from interactions with triglycerides and HDL cholesterol.

As the present study is a cross-sectional study, the degree of causality between polyp incidence and the various risk factors is difficult to judge. In addition, information bias might have been present because gastroenterologists who conducted colonoscopy were not consistent. Because the study design has not been previously formulated, incomplete information exists with regard to colonoscopy data, including the numbers and sizes of the polyps discovered during colonoscopy. Therefore, analysis of variables could not be performed. Furthermore, lifestyle data was collected via self-reported inventories, a practice that can introduce information bias due to omissions and inaccurate data records. Finally, dietary practices such as increased fat intake could not be examined as potential risk factors for colon polyps. In the future, we need to compensate for these deficiencies.

Many studies have investigated the risk factors for colon cancer or colon polyps. One prospective study reported that among the variables of alcohol consumption, smoking, body mass index, and serum triglyceride levels, all increased the risk of colon cancer, except serum triglyceride levels, which showed no association at all.[21] It is hoped that prospective studies will be conducted to facilitate better understanding of the relationship between adenomatous colon polyps, which are precancerous lesions of colon cancer, and dyslipidemia.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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