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Obesity and Risk of Colorectal Adenomatous Polyps: a Case-Control Study in Hospital Kuala Lumpur

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ABSTRACT

Colorectal adenomas are precursor lesions of colorectal cancer. Several studies have proposed that obesity is a risk factor for colorectal adenoma. This case-control study examined the relationship between body mass index (BMI), waist circumference, waist-to-hip ratio (WHR), body fat percentage and colorectal adenomatous polyps (CRA) in patients who have had a colonoscopy at the Hospital Kuala Lumpur (HKL). Fifty-nine patients (42 males and 17 females) positively identified as having CRA and 59 polyp-free subjects were recruited as controls (33 males and 26 females). A pre-tested questionnaire was used to collect socio-economic information, while anthropometric measurements were determined directly by established methods. The mean BMI of female case subjects was significantly higher than control females (25.63 ± 4.87 kg/m^2 vs. 23.86 ± 3.70 kg/m^2, $p<0.05$) but the difference in BMI was not significant in men. The mean WHR of male subjects was significantly higher in the case group (0.92 ± 0.07 vs. 0.90 ± 0.06, $p<0.05$). After adjusting for confounders, waist circumference was the only indicator that was found to significantly increase the risk for CRA in women (OR = 6.349, 95% CI = 1.063 – 37.919). Higher BMI, WHR and body fat percentage showed a non-significant risk in female subjects. In men, none of the obesity indicators were found to be significant risk factors for CRA. These findings suggest that abdominal obesity may be a contributing factor to CRA risk particularly in women. A prospective study is needed to confirm the role of obesity in the development of CRA in Malaysians.

INTRODUCTION

Worldwide, more than ten million people are diagnosed with cancer and six million deaths occur yearly (World Health Organization, 2003). Cancer is now the third leading cause of death in Malaysia (National Cancer Registry, 2003).

Colorectal cancer (CRC) was estimated to be the third and fourth most commonly occurring cancer worldwide among men and women respectively in the year 2002 (International Association of Cancer Registries, 2002). CRC was estimated to contribute to 9.5% and 9.3% of total cancer cases among males and females respectively

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in the same year. Among Malaysians, colon cancer ranked third among all cancers reported, accounting for 7.6% and 6.0% of all cancer cases in males and females respectively in 2003 (National Cancer Registry, 2004). The age-standardised incidence rates per 100,000 of population for colon cancer in males and females were 10.9 and 10.0 respectively. Cancer of the rectum, on the other hand, ranked fifth among cancers reported in Malaysian males (6.6%) and females (4.1%) respectively.

Colorectal cancer develops over a period of several years, and nearly all arise from benign, neoplastic adenomatous polyps (Bond, 2000; Kahn *et al.*, 1998). The progression of adenoma to cancer may take five to ten years (Young, Rozen & Levin, 2002). These polyps are benign growths that protrude from the inner walls of the colon and rectum, and are relatively common in people over the age of 50. It is estimated that the average 60-year-old without special risk factors for polyps has a 25% chance of having a polyp (American Society for Gastrointestinal Endoscopy, 2006). Data on the incidence of colorectal adenoma (CRA) in the Malaysian population is yet to be reported. The closest available study which was done in Singapore showed that the prevalence of CRA amongst males was 20.4% for Chinese, 4.6% for Malays and 7.9% for Indians (Lee, 1987).

A diet high in fat, low in fibre, fruits and vegetables (Schatzkin *et al.*, 2000) as well as a sedentary lifestyle, tobacco smoking (Sandler, Pritchard & Bangdiwala, 1995) and alcohol consumption (Kahn *et al.*, 1998; Todoroki *et al.*, 1995; Otani *et al*; 2003) have been implicated in the development of sporadic adenomatous polyps while mutation in genes and DNA may also cause conditions known as familial adenomatous polyposis syndrome (FAP) or hereditary non-polyposis colorectal cancer (HNPCC) which leads to the development of multiple polyps (Burt, 2000).

Several studies in Western populations have reported a relationship between obesity and CRA risk (Giovannuci *et al.*, 1995; Giovannuci *et al.*, 1996; Morimoto *et al.*, 2002; Almendingen, Hofstad & Vatn, 2001). The third National Health Morbidity Survey (NHMS, 2008) data revealed an increase in the prevalence of obesity in Malaysian adults by about 10% from 4.4% in 1996 to 14% in 2006. However, as it is not known whether obesity contributes to the risk of CRA among Malaysians, a case-control study was conducted in Hospital Kuala Lumpur to identify CRA risk associated with indicators of obesity.

METHODOLOGY

Selection of subjects

Men and women who were at least 30 years of age and had completed a colonoscopy between January 2005 and December 2005 were invited to participate in the study. Hospital Kuala Lumpur (HKL) served as the clinical centre and the source of participants for this study. Patients who were newly diagnosed with one or more histologically confirmed CRA removed through polypectomy, had no other types of polyps (hyperplastic polyps, FAP and HNPCC), and who were not involved in other studies were considered as eligible cases. The exclusion criteria were history of colorectal and / or any other cancers or, bowel resection, polyposis syndrome, inflammatory bowel disease, unsatisfactory colon preparation, incomplete colonoscopy, taking cholesterol-lowering drugs and chronic medical conditions such as HIV, diabetes mellitus, hypertension, chronic renal failure, cardiovascular diseases or dietary restrictions. The most common indication for the index colonoscopy is bleeding, large bowel complaints including constipation and routine screening for adenoma recurrence.

Three hundred and forty three patients (343) who fulfilled these criteria were selected by surgeons in the research team. Fifty-nine subjects who had at least one

histologically confirmed adenomatous polyp removed were recruited as study cases with written informed consent. Those patients who were suspected of having polyps, but upon colonoscopy were found to be negative and fulfilled the other criteria were recruited as control subjects (n=59) upon signing the consent form. The study protocol was reviewed and approved by the Clinical Research Centre, HKL and the Ethics Committee of the Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, prior to the commencement of the study.

Data collection

All data were collected during a face-to-face interview with the subjects at HKL. A pre-tested, structured questionnaire was used to record information on the socio-demographic background (age, ethnicity, marital status, educational status, occupation, and income), physical activity, smoking habits and alcohol consumption of respondents.

Anthropometric measurements were taken directly after the interview session. The subjects were barefooted and in light clothing when the measurements were taken. Height was measured to the nearest 0.1cm using a body meter (GIMA) and weight was measured to the nearest 0.1kg using an electronic weighing scale (TANITA). Body mass index (BMI) was calculated and was classified according to the recommendations of WHO (1995).

Waist and hip circumferences were measured using a non-elastic measuring tape and measured to the nearest 0.1cm. Waist-hip ratio (WHR) was then calculated, and both waist circumference and WHR were categorised according to the recommended classifications of WHO (1998).

The Omron body fat monitor HBF-302 (Omron Healthcare Co Ltd UK) was used to obtain subjects' body fat percentage and total body fat mass with accuracy up to 0.1% and 0.1kg respectively. The measurement of body fat using the Omron body fat monitor is based on the bioelectric impedance principle which is a non-invasive method based on the principle that resistance to an applied minor electric current is inversely related to the amount of fat-free mass within the body (Lukaski *et al.*, 1985). The cut-off points for percentage of body fat were those recommended by Omron HBF-302. The OMRON body fat monitor has been validated in previous studies (Martín-Moreno *et al.*, 2001; Lintsi, Kaarma & Kull, 2004).

Data analysis

All statistical analyses were performed using SPSS version 14.0. Descriptive statistics such as frequencies, means, and standard deviations were used to describe the data. Independent *t*-test was used to determine differences between case and control groups for continuous variables. The crude and adjusted odds ratios, and their corresponding χ^2 trends and 95% confidence intervals were determined using binary logistic regression analysis. The odds ratio was adjusted for variables such as age, ethnicity, physical activity, current smoking and alcohol consumption, based on previous studies (Bowers *et al.*, 2006; Otani *et al.*, 2006). 95% confidence intervals were determined. A *P*-value of <0.05 was considered as significant.

RESULTS

The socio-economic and demographic characteristics of the study groups are described in Table 1. No significant differences were observed between groups. The majority of the study participants were Chinese and married. The majority of the male subjects were in the 60-69 years old age bracket, while female subjects were in the 50-59 years age group. The mean age of case subjects was 59.3 ± 9.3 years which was significantly higher than the mean age of controls (55.0 ± 11.2 years). A similar

Table 1. Socio-demographic characteristics of study groups

Variables	*Males*		*Females*		*All*	
	Case n (%)	*Control n (%)*	*Case n (%)*	*Control n (%)*	*Case n (%)*	*Control n (%)*
Age (years)						
30 – 39	1 (2.4)	1 (3.0)	0 (0.0)	3 (11.5)	1 (1.7)	4 (6.8)
40 – 49	6 (14.3)	7 (21.2)	2 (11.8)	8 (30.8)	8 (13.6)	15 (25.4)
50 – 59	10 (23.8)	11 (33.3)	7 (41.2)	9 (34.6)	17 (28.8)	20 (33.9)
60 – 69	18 (42.9)	11 (33.3)	7 (41.2)	4 (15.6)	25 (42.4)	15 (25.4)
>70	7 (16.7)	3 (9.1)	1 (5.9)	2 (7.7)	8 (13.6)	5 (8.5)
Mean ± SD	59.8 ± 9.8	57.4 ± 11.0	57.9 ± 8.3	52.2 ± 10.9	59.3 ± 9.3*	55.0 ± 11.2*
Ethnicity						
Malays	15 (35.7)	9 (27.3)	3 (17.6)	9 (34.6)	18 (30.5)	18 (30.5)
Chinese	17 (40.5)	13 (39.4)	10 (58.8)	8 (30.8)	27 (45.8)	21 (35.6)
Indians	10 (23.8)	10 (30.3)	3 (17.6)	9 (34.6)	13 (22.0)	19 (32.2)
Others	0 (0.0)	1 (3.0)	1 (5.9)	0 (0.0)	1(1.7)	1 (1.7)
Marital Status						
Single	2 (4.8)	4 (12.1)	3 (17.6)	1 (3.8)	5 (8.5)	5 (8.5)
Married	39 (92.9)	28 (84.9)	11 (64.7)	24 (92.4)	50 (84.7)	51 (86.4)
Widowed /Divorced	1 (2.4)	1 (3.0)	3 (17.7)	1 (3.8)	4 (6.8)	2 (5.1)
Education Status						
Primary	12 (28.6)	10 (30.3)	7 (41.2)	8 (30.8)	19 (32.2)	18 (30.5)
Secondary	16 (38.1)	13 (48.5)	8 (47.0)	10 (38.5)	24 (40.7)	26 (44.0)
Pre-U /Tertiary	14 (33.4)	10 (30.3)	2 (11.8)	8 (30.8)	16 (27.1)	18 (25.5)
Occupation						
Unemployed /retired	26 (63.7)	18 (54.6)	13 (76.4)	17 (65.4)	39 (66.1)	35 (59.3)
Blue collar	7 (16.7)	7 (21.2)	2 (11.8)	6 (23.1)	9 (15.3)	13 (22.0)
Businessmen	3 (7.1)	1 (3.0)	1 (5.9)	0 (0.0)	3 (5.1)	1 (1.7)
Government	1 (2.4)	2 (6.1)	0 (0.0)	1 (3.8)	2 (3.4)	4 (6.8)
Professionals	3 (7.1)	4 (12.1)	0 (0.0)	1 (3.8)	3 (5.1)	5 (8.5)
Others	1 (3.0)	1 (3.0)	1 (5.9)	0 (0.0)	3(5.1)	1 (1.7)
Personal income (RM)						
<500	16 (38.1)	15 (45.5)	12 (70.6)	16 (61.5)	28 (47.5)	31 (52.5)
500 – 999	5 (11.9)	2 (6.1)	4 (23.9)	3 (11.5)	9 (15.3)	5 (8.5)
1000 – 1999	13 (31.0)	9 (27.3)	0 (0.0)	4 (15.4)	13 (22.0)	13 (22.0)
2000 – 2999	2 (4.8)	3 (9.1)	0 (0.0)	3 (11.5)	2 (3.4)	6 (10.2)
>3000	6 (14.3)	4 (12.1)	1 (5.9)	0 (0.0)	7(11.9)	4 (6.8)
Mean ± SD	1721.6 ± 360.9	2936.1 ± 1045.1	770.1 ± 366.9	4786.9 ± 570.1	1113.0 ± 938.0	1214.2 ± 399.2
Physical activity						
Yes	26 (61.9)	14 (42.6)	12 (70.6)	10 (38.5)	38 (64.4)	24 (40.7)
No	16 (38.1)	19 (57.6)	5 (29.4)	16 (61.5)	21 (35.6)	35 (59.3)

Continued on page 5

Table 1. continued

Smoking habit						
Yes	7 (15.9)	7 (21.2)	0 (0.0)	0 (0.0)	7 (11.9)	7 (11.9)
No	37 (84.1)	26 (78.8)	59 (100.0)	59 (100.0)	52 (88.1)	52 (88.1)
Alcohol consumption						
Yes	11 (26.2)	5 (15.2)	1 (5.9)	1 (3.8)	12 (20.3)	6 (10.2)
No	31 (73.8)	28 (84.8)	16 (94.1	25 (96.2)	47 (79.7)	53 (89.8)

*significant at $p<0.05$
The total percentages may not be exactly 100% as the individual percentages were rounded up to the nearest 0.1.

distribution of subjects in both groups was seen for educational status. The majority of the participants in the study groups were either unemployed or retired and came from low income households. More case subjects than controls were physically active. While none of the females smoked, an equal number of males in both groups were current smokers. The majority of the participants in both groups did not consume alcohol.

Table 2 describes the obesity characteristics of male patients in the study groups. More men in the case group were found to have either normal BMI (46.3%) or were overweight (48.8%) as compared to men in the control group (42.5% and 39.4% respectively). However, the majority of the women in both groups were in the normal BMI category (Table 3). Case group female patients had higher mean BMI than the control group (25.63 ± 4.87 kg / m^2 vs 23.86 ± 3.70 kg / m^2), and similar results were seen in men (25.10 ± 3.09 kg / m^2 vs 24.37 ± 3.79 kg / m^2). The adjusted odds ratio suggests higher risk with increasing BMI in both men and women, but the increase in risk was not significant (men: OR obese vs normal = 1.566, 95% CI = 0.049 – 5.203; women: OR = 1.349, 95% CI = 0.024 – 5.063).

The mean waist circumference of males in the case group was found to be higher than in the control group (90.29 ± 9.35 cm vs. 88.42 ± 10.12 cm). In female subjects, higher waist circumference ($\geq$88cm) was found to increase the risk for CRA by six-fold (OR = 6.349, 95% CI = 1.063 – 37.919). Although a similar trend was seen in the male subjects, the increase in the risk was not significant (OR = 1.721, 95% CI = 0.127 – 4.088). WHR was found to be significantly higher in male cases as compared to male controls (0.92 ± 0.07 vs 0.90 ± 0.06, $p<0.05$) but this was not observed in female subjects. WHR was not found to be a significant risk factor in both sexes (OR males = 1.008, 95% CI = 0.979 – 1.078; OR females = 2.849, 95%CI = 0.518 – 15.670).

For body fat percentage, no significant difference was found between study groups and between sexes, although the adjusted odds ratio suggested a non-significant three-fold increase in risk in females (OR = 3.938, 95% CI = 0.471 – 32.732) and by almost a two-fold increase in risk in males (OR = 1.596, 95% CI = 0.119 – 5.091) associated with the highest percentage of body fat.

DISCUSSION

Several studies have been conducted on the association between colon cancer and obesity (Loos *et al*., 2007). However, studies conducted on measures of obesity as risk factors of colonic adenoma are limited and the association remains controversial.

There was an increasing but insignificant trend in the risk of CRA with increasing BMI regardless of sex. The reason for the lack of a significant finding could be attributed to the small sample size. A study using a large number of subjects found that men in the upper quartiles of BMI had a higher risk of recurrent adenomas, but a similar finding was not observed in women

Table 2. Indicators of obesity in male patients

Variables	*Males*		*Crude OR*	*95% CI*	*Adjusted OR*[φ]	*95% CI*
	Case n (%)	*Control n (%)*				
BMI (kg / m^2)						
Normal (18.5 – 24.9)	19 (46.3)	15 (42.5)	1.000		1.000	
Underweight (<18.5)	0 (0.0)	3 (9.1)	1.002	0.952 – 1.010	1.003	0.871 – 1.201
Overweight (25.0 – 29.9)	20 (48.8)	13 (39.4)	1.215	0.459 – 3.212	1.267	0.440 – 3.652
Obese (≥30.0)	2 (4.9)	2 (6.1)	1.789	0.099 – 6.279	1.566	0.049 – 5.203
Mean ± SD	25.10 ± 3.09	24.37 ± 3.79				
Waist circumference (cm)						
Normal (<102)	38 (90.5)	30 (90.9)	1.000		1.000	
High risk (≥102)	4 (9.5)	3 (9.1)	1.053	0.219 – 5.068	1.721	0.127 – 4.088
Mean ± SD	90.29 ± 9.35	88.42 ± 10.12				
WHR						
Normal (≤1.0)	39 (92.9)	33 (100.0)	1.000		1.000	
High risk (>1.0)	3 (7.1)	0 (0.0)	1.009	0.012 – 1.921	1.008	0.979 – 1.078
Mean ± SD*	0.92 ± 0.07	0.90 ± 0.06				
Body fat percentage (%)						
Low / Normal (<20)	6 (14.3)	3 (9.1)	1.000		1.000	
Moderate (20 – 25)	4 (9.5)	6 (18.2)	1.333	0.051 – 3.177	1.341	0.043 – 2.692
High (>25)	32 (76.2)	24 (72.7)	1.667	0.151 – 3.939	1.596	0.119 – 5.091
Mean ± SD	27.12 ± 5.42	27.43 ± 6.50				

[φ] adjusted for age, ethnicity, physical activity current smoking and drinking habit.
The total percentages may not be exactly 100% as the individual percentages were rounded up to the nearest 0.1.

(Davidow *et al.*, 1996). A higher BMI was also found to be strongly associated with increased risk of advanced adenoma (OR = 10.8, 95% CI = 4.6–25.3) (Chung *et al.*, 2006). These researchers reported a mean BMI of 25.6 ± 2.7kg / m^2 in the case group and 23.2 ± 2.1 kg / m^2 for the control group, which are almost similar to the means obtained in this study. Obesity together with physical inactivity were suggested to be inversely related to the risk for advanced neoplastic polyps (Wallace *et al.*, 2005). However, this study did not find any significant association between physical activity and study groups, though Guilera *et al.* (2005) have suggested that physical activity may modify the association between obesity and colorectal adenoma, the risk being less in physically active subjects.

Waist circumference and waist-hip ratio, both of which are surrogate markers for abdominal obesity have been strongly associated with CRA risk (Moore *et al.*, 2004). In this study, higher waist circumference (≥88cm) in female subjects was found to increase the risk for CRA significantly but not in men. Similar findings have been reported elsewhere (Giovannucci *et al.*, 1996; Morimoto *et al.*, 2002). A recent prospective study demonstrated that visceral abdominal fat (VAT) as measured by CT scan is significantly higher in those with colorectal neoplasm (Oh *et al.*, 2008).

Although the adjusted odds ratio suggested increased risk with increasing percentage of body fat, the trend was not significant in both genders. In a previous study, tricep skinfold thickness (TSF),

Table 3. Indicators of obesity in female patients

Variables	*Females*		*Crude OR*	*95% CI*	*Adjusted OR*$^{\phi}$	*95% CI*
	Case (n=26)	*Control (n=17)*				
BMI (kg / m^2)						
Normal (18.5 – 24.9)	9 (56.3)	12 (46.2)	1.000		1.000	
Underweight (<18.5)	1 (6.3)	0 (0.0)	1.009	0.004 – 1.610	1.509	0.056 – 2.214
Overweight (25.0 – 29.9)	5 (31.3)	7 (26.9)	0.952	0.226 – 4.006	1.002	0.191 – 5.249
Obese (≥30.0)	1 (6.3)	7 (26.9)	1.190	0.020 – 1.837	1.349	0.024 – 5.063
Mean ± SD	25.63 ± 4.87	23.86 ± 3.70				
Waist circumference (cm)						
Normal (<88)	8 (47.2)	19 (73.1)	1.000		1.000	
High risk (≥88)	9 (52.9)	7 (26.9)	3.054	0.843 – 11.067	6.349**	1.063 – 37.919
Mean ± SD	84.28 ± 9.34	82.47 ± 12.92				
WHR						
Normal (≤0.85)	7 (41.2)	15 (57.7)	1.000		1.000	
High risk (>0.85)	10 (58.8)	11 (42.3)	1.948	0.564 – 6.733	2.849	0.518 – 15.670
Mean ± SD	0.85 ± 0.07	0.84 ± 0.09				
Body fat percentage (%)						
Low / Normal (<30)	5 (29.4)	9 (34.6)	1.000		1.000	
Moderate (30 – 35)	6 (35.3)	8 (30.8)	1.350	0.295 – 6.183	1.483	0.219 – 10.024
High (>35)	6 (35.3)	9 (34.6)	1.200	0.267 – 5.400	3.928	0.471 – 32.732
Mean ± SD	33.54 ± 5.20	33.02 ± 5.63				

$^{\phi}$ adjusted for age, ethnicity, physical activity current smoking and drinking habit. **significant based on 95% confidence interval

The total percentages may not be exactly 100% as the individual percentages were rounded up to the nearest 0.1.

percentage of body fat and BMI did not differ between patients with polyps and healthy controls (Almendingen *et al.*, 2001). However, adenoma growth was highly associated with all these factors. A 10-year cohort study found that adiposity was associated with CRA risk (OR = 2.16, 95% CI = 1.13 – 4.14) and the effects were stronger in women (OR = 4.42, 95% CI = 1.53 – 12.78) (Sedjo *et al.*, 2007).

Obesity has been suggested to reflect the metabolic and hormonal changes due to increased body fat, and this might be true especially for premenopausal women (Neugut *et al.*, 1991). Abdominal obesity along with low physical activity has been associated with insulin resistance risk and hyperinsulinemia which may mediate the effect of low physical activity on CRC risk as insulin is an important growth factor for colonic mucosa cells and colonic carcinoma cells in vitro. Thus obesity may also serve as a marker of physical activity for CRA (Giovannuci *et al.*, 1995)

This study is not without its limitations. The study population was relatively small and therefore confirmation of these results by studies with a larger sample size is necessary. The use of the body fat monitor may also be a limitation as the handheld monitor is more accurate in estimating only the upper body fat, and not the entire body. The possibility that the associations may be confounded or modified by other genetic or dietary factors cannot be excluded. However, the controls were recruited from the same

population as the adenoma cases. Further, our controls were screened and found to be polyp-free by colonoscopy and the risk of any of them having colon cancer at the time of inclusion is not likely.

CONCLUSION

Higher waist circumference was found to increase the risk for CRA in female subjects, but not in males. This finding contributes to the existing evidence supporting a positive relationship between abdominal obesity and CRA risk. Though an increase in the risk with increasing BMI and body fatness was observed, the trends were not significant. Thus a prospective study on patients recruited from various hospitals in Malaysia is needed to confirm the relationship between obesity and CRA risk in Malaysians.

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