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BMI and the risk of colorectal adenoma in African Americans

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Abstract

Objectives—Obesity is associated with the activation of the molecular pathways that increase the risk of colorectal cancer. Increasing body mass index may accelerate the development of adenomatous polyps, the antecedent lesion of colorectal cancer. The aim of this study was to assess the BMI effect on the risk of colonic polyp and adenoma in African American.

Design and Methods—We examined the records of 923 patients who underwent colonoscopy. Demographic and clinical data were collected before colonoscopy. Polyp and adenoma diagnosis were confirmed by pathology examinations.

Results—Overall, 43% of the patients were male, median age was 57 years and 77% had BMI≥25.0 kg/m^2. The frequency of colorectal polyps and adenomas were 61% and 35%, respectively. BMI≥25.0 (OR=1.61, 95%CI=1.14-2.26), smoking (OR=1.61, 95%CI=1.15-2.26) and history of colon polyps (OR=1.64, 95%CI=1.09-2.47) were associated with higher risk of colon polyp. BMI≥25.0 (OR=1.81, 95%CI=1.24-2.62), age (OR=1.04, 95%CI=1.02-2.05 for each year), male gender (OR=1.38, 95%CI=1.02-1.86), and smoking (OR=1.73, 95%CI=1.23-2.42) were associated with higher risk of colon adenoma.

Conclusion—Male and overweight African Americans are at higher risk of colorectal adenoma. The findings of this study could be applied for risk stratification and modifying the colorectal cancer prevention including screening guideline in African Americans.

Keywords

Obesity; Colorectal polyp; African American

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Introduction

The global colorectal cancer (CRC) incidence is diverse and is affected by genetic and environmental factors (1). CRC is the second most common cancer diagnosed and the third leading cause of death among African American. Incidence rates for CRC are 23% higher in African American men and 22% higher in African American women when compared with white men and women, respectively (2). Racial difference in CRC incidence reflects differences in risk factors and screening. One of the major modifiable causes of CRC is obesity (3). Obesity is a universal problem with more than 1 billion overweight and more than 300 million obese people worldwide (4). Obesity has several definitions. The one that is universally available and easy to calculate is body mass index (BMI), which measures total adiposity (5). Higher BMI is directly associated with higher risk of CRC (6) and one of the goals of weight reduction program is to reduce the risk of colorectal cancer (7).

A large proportion of colorectal cancers arise from adenoma (8). Male gender, smoking and African American race are considered to be among the risk factors of colorectal adenoma (9, 10). Prevalence of polyps and adenomas is lower in people who exercised regularly (11).

Epidemiologic studies have shown that abdominal obesity was associated with increased risk of adenomatous colonic polyps (11) and may accelerate the development of adenomatous polyps (12). Certain polyps are triggered by the same pathway that is predominantly active in obesity including Insulin Growth Factor1, oxidative stress and inflammatory pathways. There is conflicting information about the effect of obesity on colorectal adenoma. Some studies indicated a higher risk of adenoma with obesity (13-19) whereas other did not find any significant relationship between adenoma and weight (1, 20, 21). The positive relation in some studies was limited to Caucasians(16) and/or distal colon adenoma (22). African American have higher rates of obesity (23) and there were a few studies assessed the relationship of colorectal adenoma and obesity in African American which had conflicting results (16, 24). In this study we investigated the effect of BMI on colonic polyp and adenoma formation among African-Americans.

Methods and Procedures

Patients

The patients recruited from colonoscopy unit at Howard University Hospital from January 1st, 2010 to December 30th, 2012. All subjects signed an approved consent form, and filled in one interviewer administered questionnaires at the time of colonoscopy. The institutional review board at Howard University approved the research procedure. Participants were ineligible for study if they had current or prior diagnosis of CRC, familial adenomatous polyposis, inflammatory bowel disease, or Lynch Syndrome. In addition, subjects with incomplete colonoscopy (non-visualized cecum and/or inadequate bowel preparation) were also excluded. We collected the information on demographic characteristics, smoking and alcohol consumption. Weight and height were measured by Registered Nurses at colonoscopy unit and we calculated the Body Mass Index (BMI) as weight in kilograms divided by height in meters squared. Pathology examinations confirmed polyp and adenoma diagnosis by an expert pathologist (E. Lee). Subjects were classified as overweight/obese

(BMI ≥25.0 kg/m^2) or normal weight (BMI <25.0 kg/m^2) by World Health Organization criteria. The location of the lesion is defined as “Right sided” if in ascending colon, hepatic flexure or transverse colon, and defined as “Left sided“ if located at splenic flexure, descending colon, sigmoid or rectum. Location was “Both sided” if there were polyp or adenoma at both right and left colons.

Statistical analysis

Continuous data are presented by median (interquartile range) while categorical variables with tables of frequency. We tested the distribution of continuous variables between subgroups of patients with Student's t-test after the best transformation to normal distribution and categorical variables with Chi^2 test. Multiple logistic regression models were built to assess the predictors of polyp and adenoma. In each model all predictors with p ≤ 0.20 in univariate analyses were entered and final models were selected by backward stepwise approach. In each model the statistical assumption including the collinearity and distribution of residual were tested. We also performed a subgroup analyses in patients who were referred for screening to limit the potential confounding effect of colorectal symptoms (including weight loss or change in life style). All p values ≤0.05 were considered significant in final analyses. Analyses were performed in STATA 12.0 (StataCorp, College Station, TX).

Results

Clinicopathological features of patients

There were 923 patients in the study with median age (interquartile range) of 57 (52-64) in which 43% were males. The median BMI was 28.8 and 77% had BMI≥25.0 kg/m^2. The reasons for colonoscopy were screening, follow up and diagnostic in 54%, 29% and 17%, respectively. Smokers composed 26% of participants and 45% of subjects were alcohol users. About 19% had a history of colon polyps and 18% were positive for family history of colon disease. The pathology result of colonoscopy biopsies was normal in 39% of patients, adenoma in 32%, hyperplastic polyp in 13%, benign polyp in 9%, colitis in 4% and serrated polyp in 3%. Overall, 61% of the patients had polyps and 35% of them were positive for adenoma. Among adenomas 88% were tubular, 5% were tubovillous and 7% sessile serrated polyps. The location of adenomas were right sided in 37% left sided in 33% and 30% were in both sides. The adenomas size was more than 1 cm in 26% of patients (Table 1).

Polyp formation and associated risk factors

There were 358 polyp negative (PN) patients and 565 polyp positive (PP). Age distribution was not different between these two groups (Table 2). Frequency of different gender was similar between PN and PP groups. The median BMI was almost similar for PP and PN (28.9 and 28.8 respectively, p=0.6). Frequency of overweight/obesity was higher in PP groups (79% of PP patients had BMI ≥ 25 kg/m^2 compared to 73% in PN, p=0.023). Chance of previous history of colon polyps was significantly higher in PP (22% and 13% for PP and PN respectively, p=0.002). Positive family history of colon disease was 20% and 14% for PP and PN (p=0.036). Frequency of smoking was higher in PP (29% versus 22% in PN,

p=0.016). There was also a positive relation between alcohol use and polyp formation (48% and 40% for PP and PN respectively, p=0.018).

Adenoma development and associated risk factors

There were 601 adenoma negative (AN) and 322 adenoma positive (AP) with median age 56 and 58 respectively (p=0.0001). Males were more represented in the AP group than the AN group (48% and 42% respectively, p=0.039; Table 3). The median BMI were 28.9 kg/m^2 and 28.5 kg/m^2 for AN and AP respectively (p=0.9). Overweight/obesity had higher frequency in adenoma group (81% of AP patients had BMI more than 25 kg/m^2 compared to 75% in AN patients, p=0.034). Previous history of polyp was seen in 22% of AP while it was 17% for AN (p=0.07). Smoking was higher in adenoma group, with 31% of AP vs. 24% of AN smokers (p=0.029). Alcohol use was not associated with risk of adenoma. In multiple logistic regression analysis (Table 4), history of colon polyp, BMI≥25 kg/m^2 and smoking were associated with higher risk of colon polyp while age, male gender, BMI≥25 kg/m^2 and smoking were associated with higher risk of colon adenoma.

Subgroup analysis in patients with screening colonoscopy

In a group of 499 patients who was referred for screening colonoscopy, BMI≥25 kg/m^2 was associated with higher risk of poly (OR=1.61, 95%CI: 1.01-2.55, P = 0.043) and adenoma (OR=1.82, 95%CI: 1.11-3.00, P = 0.019).

Discussion

Numerous malignancies associate with obesity, however, its significance to initiate or accentuate colon neoplasia is still controversial (24). It is a potentially modifiable risk factor for colorectal cancer that can alter its incidence and outcome (25). Gender and racial differences were shown to affect this relationship (24). In our study we indicated that obesity is a risk factor of both polyp and adenoma formation. The current study is consistent with previous literature that indicate right sided colon adenoma are more frequent and adenoma is more frequent in male gender (26). Also the relationship between risk of adenoma and increasing age and smoking is consistent with former findings (27) but we were not able to confirm the association of alcohol consumption with adenoma. The health effects of obesity are assessed by using different anthropometric index including BMI. Most of our patients met the WHO definition of overweight (BMI≥25 kg/m^2). Our patient's mean BMI and the percentage of the patients with overweight or obesity were consistent the nationwide range for AAs (23). In these patients risk of colorectal adenoma (CRA) or colorectal polyp is increased. Although BMI cannot discriminate excess in body fat from increment in lean mass or fat distribution but it is the most common measurement used in studies worldwide (5). Because fat distribution location seems to be important in determining the risk of disease(3), complementary anthropometric methods such as waist circumference or waist-to-hip ratio or even waist-to-height ratio have now been used to depict adipose tissue distribution (14, 24), however none of them except visceral fat measurement (abdominal obesity) are precise in measuring fat distribution accurately (28). Visceral fat is associated with insulin resistance and higher levels of circulating IGF-1 (29, 30). To address this issue, Blake-Gumbs, et al.(31) studied weight changes in 516 cases, and 941 controls and

concluded that in postmenopausal women who were not on hormone replacement therapy, middle age weight gain increases colon cancer risk. The study concluded that it is the weight changes that has an effect on CRC not the BMI per se (31). Nimptsch et al (32) showed in 32,707 women from The Nurses' Health Study 2 that independent of adulthood BMI value, higher childhood height and weight increases the risk of left sided adenoma in adulthood (32). Ben et al.(25) conducted a meta-analysis that showed for each 5 unit higher BMI the risk of CRA increases around 20% (25). Leitzmann et al.(12) in a cross sectional study in women undergoing screening colonoscopy found positive relationship between risk of colorectal adenomas and higher BMI (12). Okabayashi et al. in a meta-analysis showed there is a consistent positive association between overweight and colorectal adenoma regardless of ethnicity (33). Nock and colleagues (16) found that in Caucasians the higher the BMI, the higher the risk of adenomas (16). Kim et al.(34) in a case-control study of colonoscopy patients found out that there is a positive link between BMI and CRA in Asians (34). We found that in African Americans, overweight or obesity increases the risk of colorectal polyp and adenoma to about two fold. The value of different obesity measurements on predicting risk of CRA is controversial. Some studies showed in AAs waist-to-hip ratio could predict the risk of CRA better than BMI (24) but our data showed a direct and significant relation between BMI and CRA in this population. The exact mechanisms by which obesity increase the risk of adenoma is not clear yet. Obese patients have elevated level of several inflammatory markers which could contribute to increased risk of colorectal carcinogenesis. Adipose tissue synthesizes and secrets pro-inflammatory mediators, including interleukin 6, tumor necrosis factor and interleukin 8 (35). The role Wnt pathway in association between obesity and colorectal carcinogens is more controversial. Despite the current evidences that support the role of Wnt and β-Catenin signaling in adipogenesis, obesity and metabolic disorders (36) a recent cohort study suggests that obesity exerts its effect in colorectal carcinogenesis pathway that is less likely dependent on Wnt activation (37). Lower vitamin D and lack of physical activity are the two important potential mechanisms (38). An inverse association between serum vitamin D3 and colon polyp in AAs have already been documented by our previous study (38) and improved vitamin D status would decrease colorectal adenoma or cancer frequency and death(39). IGF could also play important roles in this relationship (7, 13). Colorectal cancer is among the major sources of cancer disparity between African American and American White population in both sexes (2). Colorectal cancer screening which leads to substantial prevention from colorectal cancer remains lower in African American (40). In African American, targeted primary prevention and screening program in high risk population which includes obese people could substantially increase the efficiency of screening and prevention initiatives and reduces the disparity in colorectal cancer. The acceptance of BMI as a risk factor for CRA especially in the era of increasing obesity prevalence in the USA has many potential significances. For BMI there is a dose-response relationship due to positive association between obesity and CRA (33). Prevention of colon polyp and/or adenoma maybe possible by maintaining weight in the healthy range as defined by WHO and smoking cessation may substantially reduce the risk of colorectal polyps.

Our study was a retrospective study which could suffer from sampling and measurement error. Another weakness of our study is the lack of waist circumference measurement in

participants to compare with BMI. This would have allowed us to understand if BMI represents correctly the body fat composition. For future studies a non-invasive marker of visceral fat could precisely assess the relationship between obesity and risk of colon neoplastic lesion.

In conclusion, we confirmed that there were positive relation between colorectal polyps and obesity, positive family history of colon disease, alcohol, and smoking. There is stronger association if previous history of colon polyps exists. The frequency of colon adenoma was more in men, it was right sided, related to BMI more than 25 kg/m^2 and smoking (14, 16, 18, 26, 27). Our data also found that in African Americans there is strong relation between overweight/obesity and colorectal polyp and adenoma, with almost two folds more chance of adenoma in overweight/obese AA patients. Obesity may represent a risk that validates earlier age screening in order to reduce evolution of colorectal polyps to cancer. The results of this study could be significant in reducing colorectal cancer related health disparity in AAs by an implementation of more meaningful and targeted CRC screening policy.

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What is already known about this subject

- Obesity increases the risk of colorectal cancer
- African Americans have higher rate of colorectal cancer
- Obesity is highly frequent in African American

What this study add

- Risk of colorectal polyp and adenoma is higher in obese African American
- Male, overweight and smoking could define a group of African American with higher risk of colorectal adenoma.

Table 1

Clinicopathological features of patients.

Variable	N	Results
Age*	923	57 (52-64)
Male, n (%)	923	399 (43)
BMI*	856	28.8 (25.2-33.5)
Reason for colonoscopy, no (%)	923	
Screening		499 (54)
Follow up		266 (29)
Diagnosis		158(17)
Positive Hx. of colon disease, no (%)	443	8 (2)
Positive Hx. of colon polyp, no (%)	806	150 (19)
Positive family Hx. Of colon disease, no (%)	776	137 (18)
Positive Hx. of smoking, no (%)	815	214 (26)
Positive Hx. of alcohol no (%)	804	362 (45)
Results of pathology/colonoscopy, no (%)	923	
Normal Colonoscopy		358 (39)
Adenoma		298 (32)
Hyperplastic Polyp		124 (13)
Benign polyp		82 (9)
Colitis		37 (4)
Sessile Serrated Polyp		24 (3)
Positive Polyp, no (%)	923	565 (61)
Positive adenoma, no (%)	923	322 (35)
Type of adenoma**, no (%)	294	
Tubular		282 (88)
Tubulovillous		16 (5)
Sessile Serrated Polyp		24 (7)
Adenoma location**, no (%)	322	
Right		121 (37)
Left		105 (33)
Right and left		96 (30)
Adenoma size≥1 cm**, no (%)	322	83 (26)

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Variable	N	Results
High grade dysplasia**, no (%)	322	89 (28)

* Median and Interquartile range

** In patients who have adenoma.

Table 2

Association of potential risk factors and colon polyps

	Polyp Negative		Polyp Positive		P value
	N	Results	N	Results	
Age*	358	57 (52-64)	565	57 (52-64)	**0.6**
Male, n (%)	358	145 (41)	565	254 (45)	**0.18**
BMI*	326	28.8 (24.6-33.8)	530	28.9 (25.7-33.3)	**0.6**
BMI≥25, no (%)	326	237 (73)	530	421 (79)	**0.023**
Positive Hx. of colon disease, no (%)	177	1 (1)	266	7 (3%)	**0.11**
Positive Hx. of colon polyp, no (%)	320	43 (13)	486	107 (22)	**0.002**
Positive family Hx. of colon disease, no (%)	311	44 (14)	465	93 (20)	**0.036**
Current or past Hx. Of smoking, no (%)	323	70 (22)	492	144 (29)	**0.016**
Current or past Hx. Of alcohol no (%)	323	129 (40)	481	233 (48)	**0.018**

*Median and Interquartile range

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Table 3

Association of potential risk factors and colon adenoma

	Adenoma Negative		Adenoma Positive		P value
	N	Results	N	Results	
Age*	601	56 (52-63)	322	58 (53-65)	<0.0001
Male, n (%)	601	245 (42)	322	154 (48)	0.039
BMI*	542	28.9 (24.9)	314	28.5 (25.8-33.4)	0.9
BMI≥25, n (%)	542	404 (75)	314	254 (81)	0.034
Positive Hx. Of colon disease, no (%)	294	7 (2)	149	1 (1)	0.2
Positive Hx. Of colon polyp, no (%)	530	89 (17)	276	61 (22)	0.07
Positive family Hx. Of colon disease, no (%)	510	85 (17)	266	52 (20)	0.3
Current or past Hx. Of smoking, no (%)	537	128 (24)	278	86 (31)	0.029
Current or past Hx. Of alcohol no (%)	530	246 (46)	274	116 (42)	0.3

*Median and Interquartile range

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Table 4

Significant association of potential factors with polyps and adenomas in multiple logistic regression analyses

		OR	95% CI	P value
Polyp				
	Hx of Colon Polyp	1.64	1.09-2.74	0.017
	BMI≥25 kg/m^2	1.61	1.14-2.26	0.006
	Smoking	1.61	1.15-2.26	0.006
Adenoma				
	Age*	1.04	1.02-2.05	<0.0001
	Male gender	1.38	1.02-1.86	0.035
	BMI≥25 kg/m^2	1.81	1.24-2.62	0.002
	Smoking	1.73	1.23-2.42	0.002

*For each year increment