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Increased BMI, Sedentary Lifestyle & Metabolic Syndrome as Independent Risk Factors for Development of Colorectal Adenomas: A Prospective Study

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Abstract: *Background*: Several risk factors for development of colorectal adenomas has been studied over the years. But the effect of increased BMI, sedentary lifestyle and metabolic syndrome on the development of colorectal adenoma has been minimally studied. This study describes the influence of increased BMI, sedentary lifestyle & metabolic syndrome on the prevalence of colorectal adenoma. Secondary objective was to determine whether these factors influence the progression of benign adenomas into advanced adenomas or not. *Methods*: A total of 697 consecutive cases were included. Asymptomatic subjects aged more than 40 years who were undergoing their first colonoscopy were included in the study. Patients with a history of colonic neoplasia, IBD, colonoscopic polypectomy or colectomy were excluded from the study. Details of colonoscopy, polypectomy and histology were recorded. Data were analyzed by SPSS version 22.0. Chi-square (X^2) test was done to assess the association of different risk factors with the development of colonic adenomas. Odds ratio (OR) along with its 95% CI was calculated for every individual risk factor under evaluation. A p-value<0.05 was considered as significant. *Result*: Colonic polyps were detected in 153 (21.95%) subjects. Out of these 153 subjects 81 (11.62%) had histologically proved adenomas. Benign adenomas were detected in 63 (9.03%) cases, whereas advanced adenomas were detected in 18 (2.58%) cases. A BMI of≥30 showed an OR of 2.94 (1.64-4.24) for the development of colorectal adenomas. Presence of metabolic syndrome showed an OR of 1.74 (1.14-2.34) for the development of adenomas. Sedentary lifestyle showed an OR of 1.91 (1.31-2.51) for the development of colorectal adenomas. An age of≥50 years also showed significant effect (OR=1.39) on the development of colonic adenomas. Lastly the presence of increased BMI, sedentary lifestyle and metabolic syndrome was identified as individual risk factors for the progression of adenoma towards advanced adenomas with an OR of 1.67, 2.14 & 2.92 respectively. *Conclusion*: This study demonstrates that increased BMI, sedentary lifestyle and metabolic syndrome are associated with increased risk of development of colorectal adenomas. Moreover, increasing age (>50 years) also contribute to the increased risk of development of colorectal adenomas. And lastly it has been showed that increased BMI, sedentary lifestyle and metabolic syndrome individually contribute to the increased risk of progression of benign colonic adenomas to a more advanced adenoma.

Keywords: BMI, Sedentary Lifestyle, Metabolic Syndrome, Risk, Colorectal Adenomas, Colonoscopy

1. Introduction

Obesity is becoming more prevalent among general population a result of changes in their dietary pattern and changes in lifestyle. Obesity has been identified as a risk factor for several diseases, including diabetes mellitus, hypertension, dyslipidemia, ischemic heart disease and several other neoplastic diseases. Sedentary lifestyle is becoming more and more common in the urban population. The prevalence of metabolic syndrome has taken a sharp rise

over the past 20 years along with the increasing burden of obesity. The prevalence of colorectal adenomas rises with age. Almost half of the population over the age of 60 years have colorectal adenomas, and in half of these the polyps are multiple. They are more common in the rectum and left colon and are either pedunculated or sessile. Histologically, they are classified as either tubular, villous or tubulovillous, according to the glandular architecture. In most of the cases colorectal carcinoma develop on top of adenomatous polyps. [1]

Among several modifiable factors that has been suspected to increase susceptibility to CRC, increased weight and smoking have been strongly implicated. [2, 3] Several studies have described an increased prevalence of both nonadvanced and advanced adenomas with increasing BMI [4, 5], suggesting that obesity promotes neoplastic change at an early stage by increasing adenoma formation. But the association between obesity and rise of developing colonic adenoma has been minimally studied. The mechanisms whereby obesity might promote colonic carcinogenesis are complex. It involves insulin resistance, hyperinsulinemia, insulin-like growth factor, adipokines, and inflammation. [6] Adipose tissue produces a variety of proteins, hormones and cytokines that are referred to collectively as adipokines. [7] These adipokines possess wide spectrum of biological activities, including homeostatic and pathologic functions. Visceral adipose tissue in particular is thought to play an important role in systemic inflammation. [8] As visceral adiposity increases so does the release of proinflammatory adipokines including IL-6, leptin and TNF-a. And at the same time there is decreased release of anti-inflammatory adipokines, including adiponectin. [8] It is thought that elevated inflammatory adipokines contribute to the process of carcinogenesis. [9]

Earlier studies indicate an increased risk for CRC in persons with Metabolic syndrome [10] as well as an incrementally increased risk for colorectal malignancy and colorectal adenomas with the number of elements of Metabolic syndrome present [11, 12] suggesting that Metabolic syndrome might be included in the list of risk factors for colorectal malignancy. Sedentary lifestyle has also been suggested to be associated with the development of colorectal adenomas in several studies. This study was designed to examine the relationship of increased BMI, sedentary lifestyle & metabolic syndrome with the occurrence of colorectal adenomas. Secondary objective was to determine whether these factors (increased BMI, sedentary lifestyle & metabolic syndrome) influence the progression of benign adenomas into advanced adenomas or not.

2. Methods

This prospective and observational study was done in the department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders (GHPD), Square Hospitals Ltd, Dhaka, Bangladesh from August, 2017 to June, 2019. Consecutive type of non-probability sampling technique was applied to enroll the patients. Prior to the commencement of this study, the research protocol was approved by the Ethical Review Committee (ERC) of the institution. The aims and objective of the study along with its procedure, alternative diagnostic methods, risk and benefits were explained to the patients in easily understandable local language and then informed consent was taken from each patient. Patients unwilling to give voluntary consent to participate in the study were also excluded. A predesigned structured questionnaire was used for recording all the data.

Asymptomatic subjects aged more than 40 years who were undergoing their first colonoscopy were included in the study. Patients with a history of colonic neoplasia, IBD, colonoscopic polypectomy or colectomy were excluded from the study. A total of 697 consecutive cases were included. The following data were noted at the time of enrollment: age, sex, BMI, waist circumference, level of physical activity, family H/O colonic neoplasia in a first degree relative at young age, smoking status etc. Presence of comorbid diseases like diabetes mellitus, hypertension, dyslipidemia, ischemic heart disease was identified at enrollment and was noted down. Laboratory data like fasting lipid profile, serum uric acid level, vitamin D level were recorded. Weight and height were measured at the time of enrolment. BMI was calculated as kg/m^2. Normal weight was defined as BMI<25.1, overweight 25.1 to 30, and obesity>30. Metabolic syndrome was defined according to the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) definition. Metabolic syndrome was labelled to be present if three or more of the following five criteria were met: waist circumference over 40 inches (men) or 35 inches (women), blood pressure over 130/85 mmHg, fasting triglyceride (TG) level over 150 mg/dl, fasting high-density lipoprotein (HDL) cholesterol level less than 40 mg/dl (men) or 50 mg/dl (women) and fasting blood sugar over 100 mg/dl.

Colonoscopy was done in all cases and data regarding bowel preparation, polyp location, size & number of polyps, morphology of polyps was recorded (whenever a polyp was identified). Resected polyps were sent for histopathology. Subjects were categorized by the most advanced lesion identified. The histologic diagnosis of the reporting pathologist was accepted as accurate. Polyps were classified as adenoma (A) or advanced adenoma (ADV). Hyperplastic and inflammatory polyps were considered non-neoplastic. ADV was defined as any adenoma 10 mm or larger, or with greater than 25% villous component or high-grade dysplasia or cancer. Serrated adenomas were considered A if less than 10 mm and ADV if 10 mm or larger in size. Data were analyzed by computer analysis method using SPSS version 22.0.

Statistics

Statistical analyses were carried out by using the Statistical Package for Social Sciences version 22.0 for Windows (SPSS Inc., Chicago, Illinois, USA). Continuous variables were expressed as mean, standard deviation, and categorical

variables as frequencies and percentages. Chi-square (X^2) test was done to assess the association of different risk factors with the development of colonic adenomas. Odds ratio (OR) along with its 95% CI was calculated for every individual risk factor under evaluation. A p-value<0.05 was considered as significant.

3. Result

A total of 697 asymptomatic patients who fulfilled the inclusion criteria were included in the study. Colonoscopy was complete in all the cases examined. Terminal ileal intubation was done in 98.1% cases. Preparation was considered to be satisfactory in 96.3% cases. Colonic polyps were detected in 153 (21.95%) subjects. Out of these 153 subjects only 81 (11.62%) had histologically proved adenomas or advanced adenomas. Adenomas were detected in 63 (9.03%) cases, whereas advanced adenomas were detected in 18 (2.58%) cases. 223 (32%) cases had normal BMI; 300 (43%) cases were overweight & 174 (25.1%) cases were obese. Sedentary lifestyle was detected in 411 (58.96%) cases. 207 (29.69%) cases fulfilled the case definition of metabolic syndrome.

More than one polyp was detected in 37% cases with polyps. 27% polyps were located in the right colon, 61% were in the left colon & the rest (12%) was in the rectum. A positive family H/O colorectal neoplasia in a young age was detected in only 26 (3.73%) cases. Table 2 shows the influence of BMI on prevalence of adenoma & advanced adenomas, which reveals an OR of 2.94 (1.64-4.24) (p<0.001) for a BMI of more than 30. Presence of metabolic syndrome showed an OR of 1.74 (1.14-2.34) (p<0.005) for the development of adenoma & advanced adenomas (Table 3). Presence of sedentary lifestyle showed an OR of 1.91 (1.31-2.51) (p<0.001) for the development of adenoma & advanced adenomas (Table 5).

An age of more than 50 years also showed significant effect (OR=1.39) on the development of adenoma & advanced adenomas. Lastly the presence of increased BMI, sedentary lifestyle and metabolic syndrome was identified as individual risk factors for the progression of adenoma towards advanced adenomas with an OR of 1.67, 2.14 & 2.92 respectively (Tables 6, 7 & 8). The result of the study is presented in following tables.

Table 1. *Demographic, clinical and biochemical characteristics of the study population (n=697).*

Parameters	Result
Age (years)	53.21±15
Sex (Male)	388 (55.67)
Smoker	101 (14.5)
BMI	27.36±5.1
Overweight	300 (43.0)
Obese	174 (25.1)
DM	187 (26.83)
HTN	36 (30.8)
Dyslipidemia	117 (16.78)
IHD	87 (12.48)
Hyperuricemia	53 (7.6)
Metabolic syndrome	207 (29.69)
Family history of CRC	26 (3.73)
Sedentary lifestyle	411 (58.96)
Vitamin D level (ng/mL)	12.31±4.35

Values are expressed as mean±SD. Values within the bracket are expressed as percentage.

Table 2. *Influence of BMI on prevalence of adenoma & advanced adenomas (n=697).*

BMI	Adenoma/advanced adenoma		Total	p-value
	Present	Absent		
BMI ≥30	37 (45.68)	137 (22.24)	174 (18.8)	<0.001
BMI<30	44 (54.32)	479 (77.76)	523 (81.2)	
Total	81 (100.0)	616 (100.0)	697 (100.0)	

Chi-square (X^2) test was done to measure the level of significance.

Table 3. *Influence of metabolic syndrome on prevalence of adenoma & advanced adenomas (n=697).*

Metabolic syndrome	Adenoma/advanced adenoma		Total	p-value
	Present	Absent		
Present	33 (40.74)	174 (28.25)	207 (29.69)	<0.005
Absent	48 (59.26)	442 (71.75)	490 (70.30)	
Total	81 (100.0)	616 (100.0)	697 (100.0)	

Chi-square (X^2) test was done to measure the level of significance.

Table 4. *Influence of sedentary lifestyle on prevalence of adenoma & advanced adenomas (n=697).*

Sedentary lifestyle	Adenoma/advanced adenoma		Total	p-value
	Present	Absent		
Present	63 (77.78)	348 (56.49)	411 (58.96)	<0.05
Absent	18 (22.22)	268 (43.51)	286 (41.04)	
Total	81 (100.0)	616 (100.0)	697 (100.0)	

Chi-square (X^2) test was done to measure the level of significance.

Table 5. *Odds ratio (OR) of different risk factors under evaluation for the development of adenoma/advanced adenomas.*

Factors	OR (95% CI)	p value
BMI ≥30	2.94 (1.64-4.24)	<0.001
Metabolic syndrome	1.74 (1.14-2.34)	<0.005
Sedentary lifestyle	1.91 (1.31-2.51)	<0.001
Hyperuricemia	1.64 (1.12-2.17)	<0.05
Age>50 years	1.39 (1.06-1.72)	<0.05
DM	1.97 (1.37-2.57)	<0.001
HTN	1.37 (1.09-1.63)	0.002
Dyslipidemia	2.04 (1.13-2.95)	<0.05
Hypovitaminosis D	2.53 (1.79-3.11)	<0.05

Table 6. *Influence of BMI on progression of adenoma towards advanced adenomas.*

BMI	Adenoma or advanced adenoma		Total	p-value
	Advanced adenoma	Adenoma		
BMI ≥30	10 (55.56)	27 (42.86)	37 (45.68)	<0.05
BMI<30	8 (44.44)	36 (57.14)	44 (54.32)	
Total	18 (100.0)	63 (100.0)	81 (100.0)	

Chi-square (X^2) test was done to measure the level of significance. (OR=1.67).

Table 7. *Influence of metabolic syndrome on progression of adenoma towards advanced adenomas.*

Metabolic syndrome	Adenoma or advanced adenoma		Total	p-value
	Advanced adenoma	Adenoma		
Present	11 (61.11)	22 (34.92)	33 (40.74)	<0.001
Absent	7 (38.89)	41 (65.08)	48 (59.26)	
Total	18 (100.0)	63 (100.0)	81 (100.0)	

Chi-square (X^2) test was done to measure the level of significance. (OR=2.92).

Table 8. *Influence of Sedentary lifestyle on progression of adenoma towards advanced adenomas.*

Sedentary lifestyle	Adenoma or advanced adenoma		Total	p-value
	Advanced adenoma	Adenoma		
Present	13 (72.22)	50 (79.36)	63 (77.78)	<0.05
Absent	5 (27.78)	13 (20.63)	18 (22.22)	
Total	18 (100.0)	63 (100.0)	81 (100.0)	

Chi-square (X^2) test was done to measure the level of significance. (OR=2.14).

4. Discussion

Although the actual relationship between increased BMI and the increased risk of colorectal adenomas are not clear, several mechanisms have been hypothesized. A well-established hypothesis is that insulin resistance and consequent hyperinsulinemia might lead to direct mitogenic and antiapoptotic signaling by insulin/insulin-like growth factor axis. [13] The involvement of insulin and insulin-like growth factor 1 in colorectal carcinogenesis has been supported by various studies. [14] In addition, weight gain can be considered as a state of chronic low-grade inflammation, as evidenced by elevated levels of proinflammatory cytokines, such as tumor necrosis factor and interleukin-6. Interleukin-6 and tumor necrosis factor are synthesized and released by adipocytes and have direct tumorigenic affect on the gastrointestinal tract. [15, 16] Adipokines, such as leptin and adiponectin can also be involved in colorectal carcinogenesis. A recent meta-analysis demonstrated that levels of adiponectin were significantly decreased in colorectal carcinomas (CRC) and colorectal adenomas (CRA) compared with controls, which suggested that adiponectin might be involved in colorectal carcinogenesis. [17]

As the prevalence of obesity continues to increase worldwide, the contribution of increased BMI to the development of CRA and CRC increases. A recent study concluded that it is possible for obese individuals to reduce their CRC risk by maintaining an ideal body weight. [18]

This study was designed to examine the relationship of increased BMI, sedentary lifestyle & metabolic syndrome with the occurrence of colonic adenoma. Secondary objective was to determine whether these factors (increased BMI, sedentary lifestyle & metabolic syndrome) influence the progression of benign adenomas into advanced adenomas or not.

A total of 697 asymptomatic patients who fulfilled the inclusion criteria were included in the study. Colonoscopy was complete in all the cases examined. Terminal ileal intubation was done in 98.1% cases. Preparation was considered to be satisfactory in 96.3% cases. Colonic polyps were detected in 153 (21.95%) subjects. Out of these 153 subjects only 81 (11.62%) had histologically proved adenomas or advanced adenomas. Adenomas were detected in 63 (9.03%) cases, whereas advanced adenomas were detected in 18 (2.58%) cases.

Theodore et al. [19] in their study showed an overall adenoma detection rate of 23.7% where adenoma was detected in 18.8% cases & advanced adenoma was detected in 4.9% cases, which is much higher than our findings. And it can easily be explained by increased prevalence of colorectal adenomas in western population when compared to our Asian subcontinent. 223 (32%) cases had normal BMI; 300 (43%) cases were overweight & 174 (25.1%) cases were obese. 207 (29.69%) cases fulfilled the case definition of metabolic syndrome. More than one polyp was detected in 37% cases with polyps. 27% polyps were located in the right colon, 61% were in the left colon & the rest (12%) was in the rectum. A positive family H/O colorectal neoplasia in a young age was detected in only 26 (3.73%) cases.

Table 2 shows the influence of BMI on prevalence of adenoma & advanced adenomas, which reveals an OR of 2.94 (1.64-4.24) ($p<0.001$) for a BMI of more than 30. Suminori et al. [20] in their study found that a BMI of ≥26.90 had an OR of 2.4 (1.1-5.1) for the development of colonic adenomas. Presence of metabolic syndrome showed an OR of 1.74 (1.14-2.34) ($p<0.005$) for the development of adenoma & advanced adenomas (Table 3). Sedentary lifestyle showed an OR of 1.91 (1.31-2.51) for the development of colorectal adenomas.

Presence of diabetes mellitus, hypertension & dyslipidemia showed an OR of 1.97, 1.37 & 2.04 respectively for the development of adenoma & advanced adenomas (Table 5). Theodore et al. [19] in their study found an OR of 1.40 (0.98-1.98), 1.39 (1.08-1.79) & 1.30 (0.99-1.69) for diabetes mellitus, hypertension & dyslipidemia respectively for the development of adenoma & advanced adenomas.

An age of more than 50 years also showed significant effect (OR=1.39) on the development of adenoma & advanced adenomas (Table 5). Kim et al. [21] in their study found a

significant increase in the prevalence of colonic adenomas & advanced polyps to occur with increasing age. Moreover, he found a positive association between increased BMI & the prevalence of colorectal adenomas. Lastly both the presence of increased BMI, sedentary lifestyle and metabolic syndrome was identified as individual risk factors for the progression of adenoma towards advanced adenomas with an OR of 1.67, 2.14 & 2.92 respectively (Tables 6, 7 & 8).

5. Conclusion

This study demonstrates that increased BMI, sedentary lifestyle and metabolic syndrome are associated with increased risk of development of colorectal adenomas. Moreover, increasing age (>50 years) and some comorbid illnesses like DM & dyslipidemia contributes to the increased risk of development of colorectal adenomas. And lastly it has been showed that increased BMI, sedentary lifestyle and metabolic syndrome individually contribute to the increased risk of progression of benign colonic adenomas to a more advanced adenoma and thereby increasing the risk of colorectal malignancy. Thus, weight reduction, lifestyle modification & correction of the components of metabolic syndrome can reduce the risk of development of colorectal adenomas and subsequently can reduce an overall risk of colorectal malignancy.

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