Metabolic Syndrome in Colorectal Cancer Patients

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Original Article

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ABSTRACT

Background: There is epidemiological evidence indicating the relation between metabolic syndrome (MetS) and colorectal cancer (CRC).

Objective: To detect the presence of MetS in newly diagnosed CRC patients and to assess the association of various components of MetS and CRC.

Methods: A cross-sectional study involved patients who had recently diagnosed to have CRC. Patient demographic data was gathered, and measurements were taken for the components of metabolic syndrome, including fasting plasma glucose (FPG), triglycerides (TG), high-density lipoprotein (HDL), blood pressure, and waist circumference (WC) for all participants.

Results: Seventy two participants who were recently diagnosed to have CRC, among them, 30 patients (40.67%) had MetS. The degree of differentiation was significantly different between patients with MetS and those without MetS; poor differentiation was significantly higher in patients with MetS compared to those without MetS (*p- value* = 0.001), while moderate differentiation was significantly lower in patients with MetS compared to those without MetS (*p-value* = 0.02). While, there was no significant difference between both groups regarding lymph node metastasis, distant metastasis and stage. The multivariate logistic regression analysis revealed that WC, total cholesterol, TG, HDL, FPG and HbA1c were associated significantly with CRC.

Conclusion: The occurrence of MetS among CRC patients is relatively high, which indicates a need for additional research to clarify the relationship between MetS and CRC development. If this link be validated, individuals with MetS have to be included in CRC screening initiatives.

Key Words: Association, colorectal cancer, metabolic syndrome.

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INTRODUCTION

Colorectal cancer (CRC) ranks as the third most frequently diagnosed cancer and is the second primary cause of cancer-related fatalities globally[1]. In Egypt, CRC is the seventh most prevalent cancer, accounting for 3.47% of cancers in males and 3% in females[2].

The incidence of colorectal cancer (CRC) has been steadily rising around the world, especially in developing countries that are adopting a Western lifestyle. Factors such as obesity, insufficient physical activity, intake of red meat, alcohol consumption, and tobacco smoking are seen as key contributors to the increasing number of CRC cases. Nonetheless, recent progress in early detection screenings and treatment methods has led to a decline in CRC-related deaths in developed countries, despite the rising incidence rates[3].

Metabolic syndrome (MetS), defined by a cluster of metabolic risk factors including obesity, dysglycemia, and dyslipidemia, is associated with an increased likelihood of developing colorectal cancer (CRC)[4].

The pathophysiological mechanisms connecting MetS and CRC primarily involve abdominal obesity and insulin resistance. Research from both population-based

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and experimental studies has shown that factors such as hyperinsulinemia, increased C-peptide levels, higher body mass index (BMI), elevated insulin-like growth factor-1(IGF-1), reduced insulin growth factor binding protein-3, elevated leptin concentrations, and low adiponectin levels all play a role in carcinogenesis[5].

Although there were a few studies previously conducted regarding the relation between MetS and CRC, there are no studies assessed the prevalence of MetS with CRC patients in Egypt,

AIM OF THE STUDY

So the current study aimed to detect the presence of MetS in newly diagnosed CRC patients and to assess the association of various components of MetS and CRC.

PATIENTS AND METHODS

Study design and population

A cross sectional analytical study that was conducted on adult individuals who were newly diagnosed with CRC at medical oncology department, Maadi Armed Forces Medical Complex, in the period from February to August 2024.

After a histopathology confirmed diagnosis of CRC, TNM staging (histopathological degree of differentiation, lymph node metastasis and distant metastasis) were determined. Patients who were diagnosed with double primary tumor and patients using hormonal treatments were excluded.

All included patients were subjected to medical history taking including age, sex, as well as any chronic illness (diabetes mellitus, hypertension, dyslipidemia and coronary vascular disease) and the current medications.

Clinical examination was performed including recording the anthropometric characteristics. Height was assessed without footwear to the closest centimeter; weight was recorded without shoes or bulky outer garments. The height and weight information were utilized to determine the body mass index (BMI), which is obtained by dividing the weight in kilograms by the square of height in meters[6]. Waist circumference (WC) was recorded to the nearest centimeter using a stretch-resistant tape, positioned midway between the lower ribs and the iliac crest at the conclusion of a normal exhalation, when the lungs are at their functional residual capacity, with the tape in a horizontal alignment, following the guidelines set by the World Health Organization[7]. Blood pressure measurements were taken using a sphygmomanometer, according to established protocols and techniques[8].

Laboratory investigations

Fasting venous blood samples were collected from the enrolled subjects, and serum was obtained after conventional centrifugation for testing fasting plasma glucose (FPG), glycosylated hemoglobin (HbA1c), total cholesterol, triglycerides (TG), high-density lipoproteins (HDL)-cholesterol and low-density lipoprotein (LDL)-cholesterol. Also, serum levels of cancer antigen 19-9 (CA 19-9) and carcinoembryonic antigen (CEA) were assessed.

Plasma glucose (GOD-POD method), HbA1c was measured using HPLC on a Biorad D-10 system. Serum total cholesterol (measured by the cholesterol oxidase-peroxidase method), serum triglycerides (measured using glycerophosphate oxidase-peroxidase), and HDL-C (assessed through a direct homogenous method) were analyzed on an automated Roche-Cobas 611 analyzer. CEA and CA 19-9 levels were determined on a Roche e411 using an electro-chemiluminescence approach. All procedures were performed in strict compliance with the provided instructions for the kits and instruments.

Metabolic Syndrome Criteria

Patients were classified as having MetS if they met a minimum of 3 out of the 5 specific criteria outlined by the Harmonized Criteria, which include: (i) central obesity (waist circumference (WC) ≥94 cm for men or ≥80 cm for women); (ii) raised triglycerides (>150 mg/dL) or current use of triglyceride-lowering medications; (iii) low HDL-c (<40 mg/dL for males and <50 mg/dL for females) or current use of HDL-c increasing medications; (iv) elevated blood pressure (systolic ≥130 and/or diastolic ≥85 mm Hg) or current use of antihypertensive medications; (v) fasting blood glucose levels ≥100 mg/dL or current treatment with glucose-lowering medications[9].

Statistical analysis

For statistical analyses SPSS v26 was used, which is developed by IBM and located in Armonk, NY, USA. The analysis of variance test was employed to compare the quantitative data, which include means and standard deviations (SD). Quantitative variables were compared in the two groups via independent t-test. Chi-square test was applied to compare the qualitative variables, which were presented as frequencies and percentages. If the *P-value* was less than 0.05, then statistical significance was established. Two quantitative variables were evaluated for their degree of association using spearman correlation. Results were presented as odds ratio (OR) with a 95% confidence interval (95% CI) to estimate the relationship between metabolic syndrome components and CRC, by multivariate logistic regression analysis.

ETHICAL APPROVAL

The Ethical Review Committee and Institutional Review Board of the Armed Forces College of Medicine (AFCM) gave their approval to the current study. Protocol number 450, date of signature; 17 February 2024.

Informed Consent

Prior to enrollment, all participants were asked to provide written informed consent. There were no violations of the biomedical ethics standards outlined in the Revised Helsinki Declaration in the execution of this study.

RESULTS

A total of 72 individuals were participated in this study who were recently diagnosed to have with CRC, among them, 30 patients (40.67%) had MetS and 42 patients (58.33%) did not have MetS. The clinical and biochemical characteristics of the study population are listed in (Table 1).

When comparing between patients with MetS and those without MetS, there was no significant difference regarding age and gender. While - as expected - BMI, WC, systolic and diastolic blood pressure, FPG, HbA1C, total cholesterol, TG, CA19-9 and CEA levels were significantly higher in patients with MetS compared to those without MetS (P<0.05). HDL was significantly lower in patients with MetS compared to those without MetS (P<0.05) (Table 1).

Table 1: Descriptive statistics for demographic characteristics and laboratory investigations of the study population, with comparison between patients with and without metabolic syndrome.

Variable		Total population (n=72) Mean ± SD	With MetS n=30 (41.67%) Mean ± SD	Without MetS n=42 (58.33%) Mean ± SD	*P value*
Gender	Male n(%)	37 (51.4%)	17(56.67%)	20(47.62%)	0.449 #
	Female n(%)	35 (48.6%)	13(43.33%)	22(52.38%)	
Age (years)		5.38±68.64	5.29±70	5.29±67.7	0.069
BMI (Kg/m^2)		2.16±25.32	1.12±27.6	0.8±23.7	<0.001
WC (cm)	Male	4.13±89	92.76 ± 1.09	87.1± 0.71	<0.001
	Female	6.29±97	105.15± 1.63	82.63 ±1.17	<0.001
SBP (mmHg)		16.11±131.72	8.19±148.6	6.67±119.7	<0.001
DBP (mmHg)		12.1±78.89	4.62±91.7	5.62±69.7	<0.001
FPG (mg/dl)		36.26±123.6	24.36±159.4	18.38±98.6	<0.001
HbA1C (%)		1.02±5.4	0.67±6.4	0.45±4.7	<0.001
Total Cholesterol (mg/dL)		28±138.81	32.8±148	24.34±134.2	<0.001
TG (mg/dL)		54.46±161.28	47.79±212.9	13.31±124.4	<0.001
HDL (mg/dL)		7.31±49.64	9.5±46.3	3.88±52	<0.001
LDL (mg/dL)		14.42±145.72	16.35±147.7	12.89±144.3	0.374
CA199- (U/mL)		16.19±49.29	13.97±60.5	14.65±42.4	<0.001
CEA (ng/mL)		5.7±32.06	2.98±35.8	4.66±27.7	<0.001

BMI: body mass index; CA: cancer antigen; CEA for carcinoembryonic antigen; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL: high density lipoprotein; LDL: low density lipoprotein; SBP: systolic blood pressure; TG: triglycerides; WC: waist circumference.
chi-square test

The degree of differentiation was significantly different between both groups; poor differentiation was significantly higher in patients with MetS compared to those without MetS (*p- value* = 0.001), while moderate differentiation was significantly lower in patients with MetS compared to those without MetS (*p-value* = 0.02), with no significant difference between both groups regarding lymph node metastasis, distant metastasis and stage (Table 2).

Table 2: Tumor characteristics of the study population, with comparison between patients with and without metabolic syndrome (applied by chi-square test).

Variable		Total population (*n*=72)	With MetS (*n*=30)	Without MetS (*n*=42)	*P value*
Degree of differentiation	Well	8 (11.11%)	2 (6.67%)	6 (14.29%)	0.455
	Moderate	33 (45.83%)	8 (26.67%)	25 (59.52%)	0.021
	Poor	31 (43.06%)	20 (66.67%)	11 (26.19%)	0.001
Lymph node metastasis	N0	34 (27.2%)	10 (33.33%)	24 (57.14%)	0.200
	N1	28 (38.9%)	13 (43.33%)	15 (35.71%)	
	N2	10 (13.9%)	7 (23.33%)	3(7.14%)	
Distant metastasis	M0	56 (77.8%)	25 (83.33%)	31 (73.81%)	0.377
	M1	16 (22.2%)	5 (16.67%)	11 (26.19%)	
Stage	1	8 (11.11%)	2 (6.67%)	6 (14.29%)	0.254
	2	10 (13.89%)	3 (10%)	7 (16.67%)	
	3	38 (52.78%)	20 (66.67%)	18 (42.86%)	
	4	16 (22.2%)	5 (16.67%)	11 (26.19%)	

In patients with MetS, there was a significant positive correlation between the degree of differentiation (from well to poor differentiation) and BMI, SBP, DBP, total cholesterol, TG, FPG and HbA1c. While, there was a significant negative correlation with HDL levels (Table 3).

Table 3: Correlation between the degree of differentiation of CRC (from well to poor) and the components of metabolic syndrome in patients with and without metabolic syndrome

	Degree of differentiation			
	With metabolic syndrome		Without metabolic syndrome	
	r	*P*	r	*P*
Age (years)	0.213	0.072	0.103	0.518
Gender(male/female)	-0.041	0.735	0.031	0.844
BMI (Kg/m^2)	0.423	<0.001	-0.079	0.620
WC (cm)	0.052	0.663	-0.237	0.131
SBP (mmHg)	0.498	<0.001	0.096	0.544
DBP (mmHg)	0.451	<0.001	0.002	0.990
Total cholesterol (mg/dL)	0.197	0.015	0.101	0.523
TG (mg/dL)	0.466	<0.001	0.011	0.943
HDL (mg/dL)	-0.256	0.030	-0.003	0.985
LDL (mg/dL)	-0.015	0.900	-0.098	0.535
FPG (mg/dL)	0.512	<0.001	0.188	0.232
HbA1C (%)	0.456	<0.001	0.044	0.780

BMI: body mass index; CA: cancer antigen; CEA for carcinoembryonic antigen; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL: high density lipoprotein; LDL: low density lipoprotein; SBP: systolic blood pressure; TG: triglycerides; WC: waist circumference.

BMI, SBP, DBP, TG, FPG and HbA1c were significantly higher in patients with poor differentiated tumor compared to patients with well and moderate differentiated tumor (P<0.05). While, HDL was significantly lower in patients with poor differentiated tumor compared to patients with well and moderate differentiated tumor (Table 4).

Table 4: Comparison between the study population regarding the degree of differentiation of CRC.

Well differentiated (*n*=8)		Degree of differentiation			*P value*
		Moderate differentiation (*n*=33)	Poor differentiation (*n*=31)		
Age (years)		67.0 ± 5.5	67.8 ± 5.2	69.93 ± 5.43	0.192
Gender	Male n(%)	6 (75.0%)	13 (39.4%)	18 (58.1%)	0.120#
	Female n(%)	2 (25%)	20 (60.6%)	13 (41.9%)	
BMI (Kg/m^2)		23.9 ± 0.79	24.6 ± 2.05	26.5 ± 1.99	<0.001
		P1=0.690, P2=0.005, P3=0.001 ^			
WC (cm)		90.9 ± 2.29	91.7 ± 6.40	93.4 ± 7.02	0.460
SBP (mmHg)		117.75 ± 7.55	127.1 ± 13.6	140.2 ± 15.9	<0.001
		P1=0.222, P2<0.001, P3=0.001 ^			
DBP (mmHg)		70.1 ± 6.01	75.1 ± 11.2	85.1 ± 11.3	<0.001
		P1=0.471, P2=0.002, P3=0.001 ^			
Total cholesterol (mg/dL)		145.0 ± 27.7	130.3 ± 26.8	149.0 ± 28.6	0.027
		P1=0.373, P2=0.930, P3=0.023 ^			
TG (mg/dL)		125.12 ± 16.37	143.1 ± 44.22	192.67 ± 59.7	<0.001
		P1=0.634, P2=0.003, P3<0.001 ^			
HDL (mg/dL)		51.7 ± 2.4	51.6 ± 6.3	46.9 ± 8.3	0.024
		P1=0.999, P2=0.206, P3=0.026 ^			
LDL (mg/dL)		145.12 ± 15.36	146.12 ± 14.1	145.4 ± 14.9	0.876
FPG (mg/dL)		92.0 ± 20.17	113.2 ± 32.5	143.5 ± 34.2	<0.001
		P1=0.224, P2<0.001, P3=0.001 ^			
HbA1C (%)		4.7 ± 0.45	5.05 ± 0.89	5.9 ± 1.0	<0.001
		P1=0.583, P2=0.003, P3=0.001 ^			

BMI: body mass index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL: high density lipoprotein; LDL: low density lipoprotein; SBP: systolic blood pressure; TG: triglycerides; WC: waist circumference.
chi-square test
^ independent t-test
P1: *p value* between well and moderate differentiated
P2: *p value* between well and poor differentiated
P3: *p value* between moderate and poor differentiated

In patients with MetS, there was a significant positive correlation between serum CA19-9 levels and BMI, WC, SBP, DBP, total cholesterol, TG, FPG and HbA1c. There was a significant negative correlation between CA19-9 levels and HDL. There was an insignificant correlation between CA19-9 and age, gender, and LDL. While, in patients without MetS, there was an insignificant correlation between serum CA19-9 levels and the demographics and laboratory criteria of the patients (Table 5).

In patients with MetS, there was a significant positive correlation between serum CEA levels and BMI, WC, SBP, DBP, total cholesterol, triglycerides, FPG and HbA1c. There was a significant negative correlation between CEA levels and HDL. There was an insignificant correlation between CEA and age, gender, and LDL. While, in patients without MetS, there was an insignificant correlation between serum CEA levels and the demographics and laboratory criteria of the patients (Table 5).

Table 5: Correlation between CA19-9 (U/mL) as well as CEA (ng/mL) and demographics and laboratory results in patients with and without metabolic syndrome.

	CA199- (U/mL)				CEA (ng/mL)			
	With metabolic syndrome		Without metabolic syndrome		With metabolic syndrome		Without metabolic syndrome	
	r	*P*	r	*P*	r	*P*	r	*P*
Age (years)	-0.032	0.788	-0.217	0.167	0.020	0.869	-0.238	0.130
Gender(male/female)	-0.016	0.893	0.000	1.00	0.029	0.810	0.061	0.700
BMI (Kg/m^2)	0.537	<0.001	0.160	0.310	0.667	<0.001	0.133	0.400
WC (cm)	0.313	0.007	-0.159	0.316	0.363	0.002	-0.022	0.888
SBP (mmHg)	0.572	<0.001	0.234	0.136	0.584	<0.001	-0.134	0.398
DBP (mmHg)	0.584	<0.001	0.218	0.166	0.677	<0.001	0.035	0.827
Total cholesterol (mg/dL)	0.234	0.048	0.367	0.170	0.265	0.025	-0.042	0.792
TG (mg/dL)	0.546	<0.001	0.086	0.589	0.638	<0.001	0.006	0.969
HDL (mg/dL)	-0.180	0.031	-0.041	-0.798	-0.204	0.014	0.027	0.863
LDL (mg/dL)	0.017	0.891	-0.040	0.799	0.081	0.501	-0.170	0.281
FPG (mg/dL)	0.446	<0.001	-0.103	0.517	0.604	<0.001	-0.172	0.277
HbA1C (%)	0.520	<0.001	-0.002	0.990	0.631	<0.001	-0.048	0.762

BMI: body mass index; CA: cancer antigen; CEA for carcinoembryonic antigen; DBP: diastolic blood pressure; FBS: fasting plasma glucose; HDL: high density lipoprotein; LDL: low density lipoprotein; SBP: systolic blood pressure; TG: triglycerides; WC: waist circumference.

The multivariate logistic regression analysis revealed that WC, total cholesterol, TG, HDL, FPG and HbA1c were associated significantly with CRC (Table 6).

Table 6: Multivariate logistic regression analysis for the association between metabolic syndrome components and colorectal cancer.

	Odds ratio	95% CI	*P value*
Age (years)	17.8299	14.3474 to 18.4467	1.000
Gender	2.5620	23.5975 to 29.009	1.000
BMI (Kg/m^2)	4.9078	0.7956 to 1.5698	0.998
WC (cm)	1.2812	1.1082 to 1.4811	0.001
SBP (mmHg)	2.7798	4.9784 to 5.7329	0.999
DBP (mmHg)	25.9348	0.1351 to 1.7947	0.999
Total cholesterol (mg/dL)	1.0176	1.0000 to 1.0355	0.049
TG (mg/dL)	1.7576	1.9796 to 3.1532	0.050
HDL (mg/dL)	0.8832	0.8129 to 0.9596	0.003
LDL (mg/dL)	1.0176	0.9818 to 1.0547	0.339
FPG (mg/dL)	1.4431	1.0282 to 2.0252	0.034
HBA1C (%)	1.521	1.1297 to 2.134	0.045
CA19-9 (U/mL)	1.0421	1.0074 to 1.0780	0.017
CEA (ng/mL)	1.3592	1.1535 to 1.6017	<0.001

BMI: body mass index; CA: cancer antigen; CEA for carcinoembryonic antigen; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL: high density lipoprotein; LDL: low density lipoprotein; SBP: systolic blood pressure; TG: triglycerides; WC: waist circumference.

DISCUSSION

CRC has been on the rise over the past decade, becoming the third most prevalent cancer overall with a 10.0% incidence and the second leading cancer killer with a 9.4% fatality rate. Also, the number of colon cancer cases recorded has been on the rise among people aged 20–49 years, and experts predict that this trend will persist until 2030[10].

Changes in the epidemiology and incidence of illness symptoms have been noted throughout the Arab world, and rising cancer rates, particularly among younger people, are reported in many parts of the world, including the Middle East. The frequency of CRC has increased and impacted younger generations in Arab countries as a result of Western lifestyle influences[11].

The risk of CRC emerging at an earlier age was inversely proportional to the number of MetS components. While inactivity, Western eating habits, and alcohol use are all known to increase the likelihood of CRC, other risk factors, such as obesity and MetS, may also play a role (1). The aim of this study was to determine the presence of MetS in newly diagnosed CRC patients and to assess the relation between various components of MetS and CRC. A total of 72 individuals were participated in this study who were diagnosed to have with CRC, among them, 30 patients (40.67%) had MetS and 42 patients (58.33%) did not have MetS.

This result is close to what was found in the study by ***Forootan et al.***, which indicated that approximately 36% of CRC patients had MetS[12]. Similarly, ***Chiu et al.*** noted that 150 of 418 CRC patients were diagnosed with MetS[13]. On the other hand, a separate study conducted in Korea reported a MetS prevalence of 17% among CRC patients[14]. Variations in the reported rates may stem from differing demographic characteristics of the populations involved or the various laboratory techniques used to assess the components of MetS.

The results showed that patients with MetS and those without MetS differed significantly in the degree of differentiation CRC. Patients with MetS showed much higher rates of poor differentiation compared to those without the condition, and significantly lower rates of moderate differentiation. There was no statistically significant difference between both groups regarding lymph node count, distant metastases, or stage.

In line with these results, ***Li et al.*** investigated the influnce of MetS in people with CRC and found that people with MetS had a much lower overall survival compared to people without MetS. Poorly differentiated tumors and other aggressive tumor features were associated with MetS. Specifically, the overexpression of certain metabolic genes associated with MetS was correlated with increased malignancy in CRC, suggesting that metabolic disturbances contribute to poor differentiation[15].

Similarily, ***Lu et al.*** compared CRC patients with MetS to those without MetS, the risk of CRC-specific death was 2.122 times greater in the former group. In support of the idea that MetS significantly influences tumor features, they demonstrated that the likelihood of poor tumor differentiation and poor prognosis was proportionate to the number of metabolic risk variables[16].

And, according to ***Cai et al.*** the likelihood of acquiring CRC increases with a higher BMI. Patients who were overweight were more likely to have tumors that were poorly differentiated than those who were of normal weight. This suggests that metabolic factors, including BMI, may influence tumor biology and differentiation in CRC patients[17].

Furthermore, ***Tao et al.*** studied the correlation between blood lipids and CRC development and discovered genes involved in cholesterol metabolism that are overexpressed in CRC. Serum HDL levels were shown to be significantly correlated with tumor size and differentiation in patients with CRC. Patients with tumors bigger than 5 cm had decreased HDL levels, suggesting a link between lipid metabolic disorders and insufficient tumor differentiation[18].

In a previous study by ***Forootan et al.***, they did not find any statistically significant difference in the rates of lymph node metastasis or organ metastasis between individuals with MetS and those without MetS[12]. And, ***Silva et al.*** investigated how MetS affects colon cancer; they found that the components of MetS did not adversely affect the progression or outlook of the disease. They suggested that a significant number of patients with MetS were receiving treatment with blood pressure and cholesterol-lowering medications, implying that the possible protective effects of these drugs should not be excluded[19].

The findings from this study indicated that in MetS patients, CA19-9 and CEA were positively correlated with BMI, WC, systolic and diastolic blood pressure, total cholesterol, TG, FPG, and HbA1c, while negatively correlated with HDL. The statistical significance of the correlations with age, sex, and LDL was not proven.

Consistent with these results, ***Single et al.*** found that in diabetic individuals the serum levels of CEA exhibited a notable positive correlation with fasting plasma glucose (FPG) and HbA1c, while showing a significant negative correlation with HDL-C levels. And, serum CA 19-9 demonstrated a strong positive correlation with FPG[20]. Additionally, research by ***Haoyong et al.*** indicated that serum CA 19-9 had a positive correlation with total cholesterol. This association may be attributed to the lipotoxic impact of intracellular cholesterol on the functionality of pancreatic beta cells[21]. Moreover, ***Kim***

et al. discovered that an increasing aggregate of MetS components were significantly linked to a linear rise in CEA levels[22].

A potential explanation for this is that insulin promotes the proliferation of both normal and cancerous epithelial cells and exhibits mitogenic effects in laboratory settings, either directly or via IGF-1. Therefore, insulin resistance in diabetes may be related to elevated levels of CEA[23]. Similar findings were reported by ***Lee et al.*** among females in North Korea[24].

Regarding the multivariate logistic regression analysis the results revealed that WC, total cholesterol, TG, HDL, FPG and HbA1c were the significantly associated components of MetS in CRC patients.

In line with these results, ***Milano et al.*** revealed that individuals diagnosed with MetS exhibit a significantly greater risk of developing colonic and rectal adenomas during endoscopic examinations[25]. The potential implication of MetS components in relation to CRC have been largely investigated[5,26,27]. Some studies have indicated a positive relationship between CRC and obesity[27]. Others demonstrated that the distribution of body fat is more closely linked to the risk of colorectal carcinoma or adenoma than BMI[14,28].

Meanwhile, glucose intolerance and diabetes mellitus have been linked to the increased risk of colon cancer[29] or adenoma[30]. Conversely, elevated arterial blood pressure[31], hypertriglyceridemia and hypercholesterolemia[32,33] have also been associated with CRC.

The specific mechanisms behind these associations remain partially unclear; however, insulin resistance has been suggested as a crucial factor that may foster cancer through an imbalance of adipokines, the generation of reactive oxygen species, and the production of inflammatory cytokines like tumor necrosis factor-alpha and interleukin-6, which act as ancillary pathways in this context[26].

Given the significance of comprehending the pathological mechanisms linking CRC to MetS[5], further studies are essential to explore strategies for preventing CRC as MetS advances[34] and to determine the potential usefulness of MetS as a marker for increasing risk of colonic neoplasia, along with assessing its practical implications for CRC screening programs and prevention efforts.

CONCLUSION

Our results showed that the occurrence of MetS among CRC patients is relatively high. Consequently, additional analytical and multicenter studies are necessary to establish a deeper insight into how MetS contributes to the onset of CRC. If future researches validate this correlation, individuals with MetS have to be included in CRC screening programs.

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CONFLICT OF INTERESTS

There is no conflicts of interest.

ETHICAL STATEMENT

This manuscript has been read and approved by all the authors, and authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. We confirm that this work is original and has not been published elsewhere nor is it currently under consideration for publication elsewhere.

AUTHORS' CONTRIBUTION

Nesma A. Ibrahim: Data analysis, Manuscript preparation and editing.

Fatma M. Abouelkasem: Design, Supervision and Validation.

Yousry W. Nada: Methodology and Data Acquisition.

Abdallah R.Elkordy: Literature search and Statistical analysis.

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متلازمة الأيض في مرضى سرطان القولون والمستقيم

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المقدمة: هناك أدلة وبائية تشير إلى العلاقة بين متلازمة الأيض (MetS) وسرطان القولون والمستقيم .(CRC)

الهدف: قياس انتشار MetS في مرضى CRC المشخصين حديثا وتقييم ارتباط مكونات مختلفة من MetS و. CRC

الأساليب: هذه الدراسة الشاملة تضمنت المرضى الذين تم تشخيصهم حديا بسرطان القولون والمستقيمCRC . تم جمع البيانات و اخذ التاريخ المرضى و الفحص الطبى للمرضى ، وتم اتخاذ قياسات لمكونات متلازمة الأيض ، بما في ذلك مستوى السكر الصائم (FPG) ، والدهون الثلاثية (TG) ، وبروتين الدهون عالي الكثافة (HDL) ، وضغط الدم ، ومحيط الخصر (WC) لجميع المشاركين.

النتائج: اثنان وسبعون مريضا تم تشخيصهم حديثا بسرطان القولون والمستقيم CRC، من بينهم ٣٠ مريضاً (٤٠,٦٧٪) لديهم . MetS كانت درجة التمييز لخلايا الورم مختلفة بشكل كبير بين المرضى الذين يعانون من MetS وأولئك الذين لا يعانون منه. في حين أنه لم يكن هناك فرق كبير بين المجموعتين فيما يتعلق بالعقدة الليمفاوية، والانتشار البعيد والمرحلة. كشف تحليل الانحدار اللوجستي المتعدد المتغيرات أن WC، الكوليسترول الكلي، FPG ،HDL ،TG و HbA١c ارتبطت بشكل كبير مع. CRC

الاستنتاج: حدوث MetS بين مرضى CRC مهم جدا ، مما يدل على الحاجة إلى إجراء أبحاث إضافية لتوضيح العلاقة بين MetS و CRC ،إذا تم التحقق من صحة هذا الرابط ، يجب تضمين الأفراد الذين لديهم MetS في مبادرات فحص. CRC