

The Rise of Early-Onset Colorectal Cancer: Clinical Characteristics and Outcomes in a Young Patient Population

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Background: The incidence of early-onset colorectal cancer (EOCRC), defined as diagnosis at age ≤ 50 years, is rising globally. Despite younger age and presumed clinical fitness, EOCRC often presents at advanced stages and displays distinct biologic and treatment profiles.

Methods: We conducted a retrospective cohort study of patients aged ≤ 50 years with histologically confirmed colorectal adenocarcinoma treated at a tertiary cancer center in Saudi Arabia from 2015 to 2021. Clinical, pathologic, molecular, and treatment data were extracted. Survival outcomes were analyzed using Kaplan–Meier methods, and prognostic factors were assessed via Cox regression models.

Results: Among 97 patients (mean age 43 ± 5 years; 56% male), 34% presented with metastatic disease and 75% had left-sided or rectal tumors. Obesity was prevalent in 24% of cases. Surgical resection was performed in 79% of patients, of whom 47% received adjuvant chemotherapy. First-line systemic therapy was administered in 39%, but attrition limited progression to subsequent lines. Median overall survival (OS) was 20 months (*interquartile range [IQR]*, 11–30). Progression-free survival (PFS) declined from 8 months in first-line to 4 months in third-line therapy. On multivariable analysis, obesity was independently associated with worse OS (hazard ratio [HR] 6.63, $p = 0.035$).

Conclusion: Despite favorable performance status, EOCRC frequently presents with advanced disease and limited systemic therapy durability. Obesity emerged as an independent adverse prognostic factor. These findings reinforce EOCRC as a biologically distinct entity, underscoring the need for tailored screening strategies, early intensification of therapy, and molecularly guided care.

Plain Language Summary:: Colorectal cancer used to be thought of as a disease affecting older adults, but more young people under the age of 50 are now being diagnosed. In this study, we looked at 97 patients treated at a cancer center to learn more about how the disease affects younger individuals. We found that many of them were diagnosed at a late stage, and that obesity and cancer spread at diagnosis were linked to shorter survival. Even though younger patients are often healthier, not all received the full recommended treatment. These results show the need to raise awareness, start screening earlier, and develop treatment plans tailored to younger patients. Our findings could help shape future research, policies, and clinical practices for this growing group of patients.

Keywords: early-onset colorectal cancer, survival outcomes, tumor biology, prognostic factors, treatment patterns

Introduction

Colorectal cancer (CRC) remains a major global health challenge, ranking as the third most commonly diagnosed cancer and the second leading cause of cancer-related mortality worldwide.^{1,2} While incidence in older adults has stabilized or declined in many high-income countries, largely due to screening and preventive strategies, there is growing concern about the rising incidence of CRC among individuals aged ≤ 50 years, a group classified as having EOCRC.^{3,4}

EOCRC often presents with distinct clinical and pathological features, including a higher likelihood of left-sided or rectal tumors, mucinous or signet ring histology, and more advanced stage at diagnosis.^{5,6} Despite emerging data, the etiologic drivers remain poorly understood. Lifestyle changes, increasing prevalence of obesity, and rising exposure to ultra-processed foods (UPFs) have been implicated, particularly in rapidly developing countries.^{7,8}

Prompted by these trends, multiple professional societies now recommend initiating CRC screening before age 50, with modeling studies supporting the cost-effectiveness and survival benefits of starting as early as age 40.^{9,10} However, most existing data originate from Western populations, and the presentation and outcomes of EOCRC in non-Western settings remain underexplored.

In Saudi Arabia, CRC is the most common malignancy in men and the second most common in women, with a steadily increasing incidence over recent decades.¹¹ National registry data indicate a rising incidence of EOCRC, particularly among individuals aged 40–49, with women demonstrating slightly higher rates than men.¹² Recent institutional findings further highlight a higher prevalence of aggressive histologic subtypes and adverse prognostic features among young Saudi patients.¹³

Given this growing burden, understanding the clinical behavior, treatment patterns, and survival outcomes of EOCRC in Saudi Arabia is critical. This study aims to describe the clinicopathologic characteristics and real-world outcomes of EOCRC patients treated at a tertiary cancer center in Saudi Arabia and to identify key prognostic factors influencing survival.

Methods

Study Design and Population

This retrospective cohort study included all patients aged 50 years or younger who were diagnosed with histologically confirmed colorectal adenocarcinoma at King Abdullah Medical City (KAMC), Makkah, Saudi Arabia, between January 2015 and December 2021. Eligible patients were identified from a prospectively maintained institutional oncology registry and supplemented by electronic medical record review.

Inclusion criteria were age ≤ 50 years at diagnosis, confirmed colorectal adenocarcinoma, including mucinous and signet ring subtypes and diagnosis and/or treatment at KAMC during the study period. Only patients with complete clinical, treatment, and survival data were included. Exclusion criteria included non-adenocarcinoma histologies (eg, neuroendocrine tumors, lymphomas), secondary colorectal involvement from non-colorectal primaries, or incomplete medical records precluding analysis of key outcomes.

Due to the retrospective design, no formal sample size calculation was performed. All eligible patients within the study period were included, yielding a total cohort of 97 patients.

Data Sources and Measurement

Clinical, pathological, and treatment-related data were extracted from the institutional electronic health record system and the KAMC oncology registry. Data were cross-validated using multiple sources, including pathology reports, chemotherapy order systems, and clinician documentation. Information bias was minimized by cross-referencing these data and performing manual chart reviews by senior oncology staff, with discrepancies resolved through consensus. Tumor staging was determined using the American Joint Committee on Cancer (AJCC) criteria at diagnosis. When available, molecular data, including microsatellite instability (MSI) status and rat sarcoma viral oncogene (RAS) and v-Raf murine sarcoma viral oncogene homolog B1 (BRAF) mutations, were obtained from institutional or outsourced panel testing. Comprehensive next-generation sequencing was not uniformly performed, and extended sequencing data were not included in the analysis. Germline testing, now guideline-recommended for EOCRC, was not systematically performed during the study period due to limited access to genetic counseling and testing infrastructure at our institution.

Data Collection and Variables

Collected variables included demographic information (age, sex, body mass index [BMI]), Eastern Cooperative Oncology Group (ECOG) performance status, carcinoembryonic antigen (CEA) level at diagnosis, tumor site, clinical stage, histologic subtype, molecular profile (MSI, RAS, BRAF), and metastatic distribution. Treatment variables included surgical intervention, surgical intent (curative vs palliative), receipt of adjuvant therapy, and systemic therapy regimens stratified by line and class (eg, oxaliplatin-based regimens such as capecitabine plus oxaliplatin [XELOX] or folinic acid, fluorouracil, and oxaliplatin [FOLFOX]; irinotecan-based regimens such as folinic acid, fluorouracil, and irinotecan [FOLFIRI] or capecitabine plus irinotecan [XELIRI]; capecitabine-based monotherapy; and late-line agents such as trifluridine/tipiracil [TAS-102] or regorafenib).

Tumor site classification followed anatomical groupings: right-sided colon cancer (cecum to transverse colon), left-sided colon cancer (splenic flexure to sigmoid colon), and rectal cancer. BMI was categorized as underweight (<18.5 kg/m²), normal (18.5–24.9), overweight (25.0–29.9), and obese (≥30). ECOG was grouped as ≤2 vs >2.

Outcomes

The primary outcome was OS, defined as the interval from diagnosis to death from any cause or last known follow-up. PFS was measured from the start of each systemic therapy line to documented clinical or radiologic progression or death. Treatment exposure and PFS were reported by line of therapy and regimen class.

This manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies.¹⁴

Statistical Analysis

All statistical analyses were performed using R software (version 4.3.2). Categorical variables were summarized as frequencies and percentages, while continuous variables were presented as means with standard deviations or medians with interquartile ranges, as appropriate.

Subgroup analyses were conducted for key clinical variables, including tumor stage, primary tumor site, ECOG, and treatment exposure. OS and PFS were estimated using the Kaplan–Meier method, and survival differences across subgroups were compared using the Log rank test.

Univariable and multivariable Cox proportional hazards regression models were used to identify predictors of OS. Variables with a p-value <0.10 in univariable analysis or deemed clinically relevant were included in the multivariable model. Results were reported as HRs with corresponding 95% confidence intervals (CIs). A two-sided p-value <0.05 was considered statistically significant.

Missing data were managed through case-wise deletion (complete-case analysis), with no imputation applied.

Results

Patient Characteristics

Ninety-seven patients with EOCRC were included (Table 1). The median age at diagnosis was 43 years, and 55.7% were male. Nearly half were overweight or obese, and most had favorable ECOG performance status. Tumors predominantly arose in the rectum and left colon, with one-third of patients presenting with metastatic disease. Mucinous histology was observed in 13.4%, and RAS/BRAF mutations were identified in 14.5%. The liver was the most frequent metastatic site.

Table 1 Summary of Baseline Demographic, Clinical, and Tumor Characteristics of Patients Aged ≤50 Years with CRC

Variable	n (%) or Mean ± SD
Age (years), mean (SD)	43 ± 5.30
Sex	
Male	54 (55.7%)
Female	43 (44.3%)
BMI	
Underweight (<18.5)	16 (16.5%)
Healthy (18.5–24.9)	34 (35.1%)

(Continued)

Table 1 (Continued).

Variable	n (%) or Mean $\pm$ SD
Overweight (25–29.9)	24 (24.7%)
Obese (≥ 30)	23 (23.7%)
ECOG	
ECOG ≥ 2	44 (45.4%)
ECOG < 2	53 (54.6%)
Stage Group	
Local (Stage I–III)	64 (66.0%)
Metastatic (Stage IV)	33 (34.0%)
Stage at diagnosis	
Stage I	6 (6.2%)
Stage II	23 (23.7%)
Stage III	35 (36.1%)
Stage IV	33 (34.0%)
Primary Tumor Site	
Left Side	45 (46.4%)
Right Side	22 (22.7%)
Rectum	28 (28.9%)
Site of Metastases	
Liver	21 (21.6%)
Lung	18 (18.6%)
Peritoneum	12 (12.4%)
Other	14 (14.4%)
Histologic Type	
Adenocarcinoma	81 (83.5%)
Mucinous	13 (13.4%)
Signet Ring	1 (1.0%)
Other	2 (2.1%)
Molecular Profile	
RAS Mutation	12 (12.4%)
BRAF Mutation	2 (2.1%)
CEA Level, Median (IQR)	2.48 (0.71–13.43)

Abbreviations: BMI, body mass index; ECOG, Eastern Cooperative Oncology Group; RAS, rat sarcoma viral oncogene; BRAF, v-Raf murine sarcoma viral oncogene homolog B1; CEA, carcinoembryonic antigen; IQR, interquartile range.

Treatment Patterns

Surgery was performed in 79.4% of patients, with curative intent in 71.1% (Table 2). Adjuvant chemotherapy was administered in less than half of eligible cases, most commonly with XELOX.

Systemic Therapy by Line and Regimen

Of the entire cohort, 39.2% received first-line systemic therapy, 27.8% proceeded to second-line, and 13.4% proceeded to third-line. (Table 3). Oxaliplatin-based regimens predominated in first line, while irinotecan-based regimens were more frequent in second line. Use of late-line agents (TAS-102 or regorafenib) was limited to 8.2%.

Survival Outcomes

Median overall survival (OS) was 20.2 months, while the 5-year OS probability was 55%, reflecting a skewed survival distribution driven by a subset of long-term survivors (Figure 1). Median progression-free survival (PFS) declined across successive lines (8.2, 6.4, and 4.1 months), underscoring the limited durability of current systemic therapy (Table 4).

Prognostic Factors

On univariable analysis, metastatic stage at diagnosis and lack of surgery or adjuvant therapy were significantly associated with poorer OS (Table 5). On multivariable analysis, obesity independently predicted worse OS (HR 6.63, 95% CI 1.14–38.50; $p = 0.035$), alongside metastatic stage at diagnosis (HR 10.92, 95% CI 1.42–83.70; $p = 0.021$). Other factors, including ECOG, primary tumor site, and adjuvant therapy, were not independently significant (Table 6). Non-significant p -values were reported to two decimals.

Table 2 Summary of Treatment Patterns in Young CRC Patients ($n = 97$)

Treatment Variable	n (%)
Surgery	
Yes	77 (79.4%)
No	20 (20.6%)
Intent (Curative)	69 (71.1%)
Adjuvant treatment	
Yes	46(47.4%)
No	45 (46.4%)
Adjuvant Regimen	
XELOX	30 (30.9%)
Capecitabine	7 (7.2%)
FOLFOX	9 (9.3%)
Adjuvant Duration	
6 months	26 (26.8%)
3 months	20 (20.6%)

Abbreviations: XELOX, capecitabine plus oxaliplatin; FOLFOX, folinic acid, fluorouracil, and oxaliplatin; CRC, colorectal cancer; BMI, body mass index; ECOG, Eastern Cooperative Oncology Group; CEA, carcinoembryonic antigen; IQR, interquartile range.

Table 3 Systemic Therapy Regimen Categories by Line of Treatment (n = 97)

Regimen Category	1st Line 38 (39.2%)	2nd Line 27 (27.8%)	3rd Line 13 (13.4%)
Oxaliplatin-based ± Targeted	30 (30.9%)	1 (1.0%)	2 (2.1%)
Irinotecan-based ± Targeted	8 (8.2%)	21 (21.6%)	2 (2.1%)
Capecitabine ± Targeted	0 (0.0%)	3 (3.1%)	1 (1.0%)
TAS-102 or Regorafenib	0 (0.0%)	1 (1.0%)	8 (8.2%)

Abbreviations: XELOX, capecitabine plus oxaliplatin; FOLFOX, folinic acid, fluorouracil, and oxaliplatin; FOLFIRI, folinic acid, fluorouracil, and irinotecan; XELIRI, capecitabine plus irinotecan; TAS-102, trifluridine/tipiracil; CRC, colorectal cancer; BMI, body mass index; ECOG, Eastern Cooperative Oncology Group; CEA, carcinoembryonic antigen; IQR, interquartile range.

Discussion

This single-institution analysis provides novel insights into early-onset colorectal cancer (EOCRC) in a Middle Eastern cohort, an underrepresented population in the global EOCRC literature. We identified four major findings: (i) a substantial proportion of patients presented with advanced or metastatic disease, (ii) obesity emerged as an independent adverse prognostic factor for overall survival (OS), (iii) adjuvant therapy was underutilized despite curative resections, and (iv) systemic therapy demonstrated limited durability across successive lines. Together, these findings highlight the aggressive clinical behavior and unique care gaps in EOCRC within non-Western settings.

Over one-third of our patients presented with de novo metastatic disease, consistent with meta-analyses showing EOCRC is more often diagnosed at advanced stages than late-onset CRC.¹⁵ The liver was the predominant metastatic site, consistent with classical dissemination patterns and findings by Sharma et al.¹⁶ This figure is higher than typically

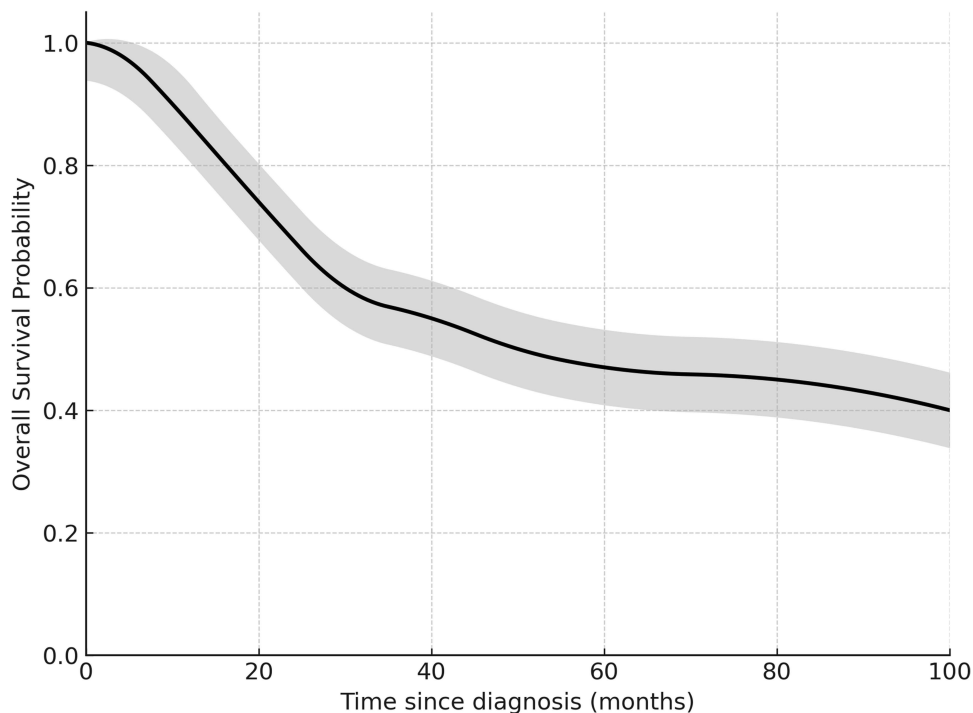


Figure 1 Kaplan–Meier overall survival (OS) curve for the entire cohort (n = 97). Shaded area represents the 95% confidence interval. The survival curve illustrates a skewed distribution, with a subset of long-term survivors accounting for the observed 5-year OS of ~55% despite the earlier median of 20.2 months.

**Table 4** Survival and Progression-Free Outcomes

Metric	Group	Median (Months)	IQR (Months)	P value ¹
Overall Survival (OS)	All Patients	20.2	11.0–30.0	–
Overall Survival (OS)	Localized (Stage I–III)	20.3	11.0–32.1	< 0.001
Overall Survival (OS)	Metastatic (Stage IV)	18.4	11.8–25.3	
Progression-Free Survival (PFS)	1st-Line Therapy	8.2	4.1–13.2	0.045
Progression-Free Survival (PFS)	2nd-Line Therapy	6.4	2.6–10.5	< 0.001
Progression-Free Survival (PFS)	3rd-Line Therapy	4.1	1.5–6.8	< 0.001

Notes: ¹P values calculated using the Log rank test.

Abbreviations: OS, Overall Survival; PFS, Progression-Free Survival; IQR, Interquartile Range.

Table 5 Univariable Cox Proportional Hazards Regression for Overall Survival

Variable		Hazard Ratio	95% CI	P value
Stage group at Diagnosis	Localized (I–III)	Reference group		
	Metastatic (IV)	11.87	(3.41–41.36)	0.001
Primary Tumor Site	Left-sided	Reference group		
	Right-sided	1.41	(0.28–2.99)	0.88
	Rectum	0.91	(0.46–4.31)	0.55
ECOG	ECOG < 2	Reference group		
	ECOG ≥ 2	1.22	(0.48–3.09)	0.68
BMI Category	Normal	Reference group		
	Obese	2.68	(0.77–9.21)	0.12
	Overweight	0.23	(0.03–2.10)	0.19
	Underweight	2.64	(0.74–9.41)	0.13
Surgery	Not Performed	Reference group		
	Performed	0.095	(0.03–0.26)	p < 0.001
Adjuvant Chemotherapy	Not Received	Reference group		
	Received	0.26	(0.072–0.93)	0.040
Gender	Female	Reference group		
	Male	1.45	(0.56–3.76)	0.44

Abbreviations: HR, hazard ratio; CI, confidence interval; ECOG, Eastern Cooperative Oncology Group; BMI, body mass index.

observed in late-onset CRC but consistent with EOCRC cohorts such as Saraste et al,¹⁷ and Georgiou et al.¹⁸ Such patterns emphasize persistent diagnostic delays and low clinical suspicion in this age group, reducing opportunities for curative treatment.¹⁹

Tumor sidedness in our cohort favored left-sided and rectal primaries, while right-sided tumors, typically linked with adverse biology were less common, consistent with Angelakas et al and Baran et al^{20,21} RAS and BRAF mutations were detected at lower frequencies than in older-onset CRC, possibly reflecting distinct EOCRC biology or limited testing

Table 6 Multivariable Cox Proportional Hazards Regression for Overall Survival

Variable		Hazard Ratio	95% CI	P value
Stage at Diagnosis	Localized (I–III)	Reference group		
	Metastatic (IV)	10.92	(1.42–83.70)	0.021
Primary Tumor Site	Left-sided	Reference group		
	Right-sided	0.95	(0.16–5.44)	0.96
	Rectum	0.52	(0.07–3.68)	0.51
ECOG	ECOG < 2	Reference group		
	ECOG ≥ 2	2.86	(0.63–12.94)	0.17
BMI Category	Normal	Reference group		
	Obese	6.63	(1.14–38.50)	0.035
	Overweight	0.41	(0.02–5.99)	0.51
	Underweight	2.90	(0.55–15.29)	0.21
Surgery	Not Performed	Reference group		
	Performed	0.25	(0.04–1.45)	0.12
Adjuvant Chemotherapy	Not Received	Reference group		
	Received	1.81	(0.28–11.52)	0.53
Gender	Female	Reference group		
	Male	3.77	(0.76–18.65)	0.10

Abbreviations: HR, hazard ratio; CI, confidence interval; ECOG, Eastern Cooperative Oncology Group; BMI, body mass index.

access. Akkus et al,²² linked higher RAS mutation rates to with recurrence risk, underscoring the value of routine molecular profiling in younger patients. By contrast, Cercek et al,²³ reported no major genomic differences between EOCRC and older-onset CRC in microsatellite-stable tumors, suggesting that treatment should be guided by molecular features rather than age.

Surgery was undertaken in 79% of patients, with curative intent in 71%. Nevertheless, fewer than half of eligible patients received adjuvant chemotherapy, most often XELOX. In contrast, Western cohorts report adjuvant uptake rates exceeding 85%.¹⁷ This underutilization may reflect patient-level factors (toxicity concerns, treatment acceptance), physician decision-making, or system-level barriers to access. Given the high relapse risk in EOCRC despite aggressive therapy, suboptimal use of adjuvant chemotherapy represents a modifiable gap in care.

The sharp decline in PFS across successive lines underscores the limited long-term effectiveness of standard regimens in EOCRC, despite patients' young age and preserved fitness. The minimal use of later-line agents (≤8%) highlights restricted therapeutic options and reflects either tolerance issues or access barriers. These findings mirror attrition patterns in other EOCRC cohort reported by Georgiou et al,¹⁸ and emphasize the urgent need for novel strategies such as biomarker-guided intensification, immunotherapy combinations, and broader clinical trial participation to extend survival beyond early treatment lines.

Despite their younger age and presumed better fitness, patients in our cohort experienced poor survival, with only a modest difference between localized and metastatic disease. This narrow separation underscores the biological aggressiveness of EOCRC and mirrors similarly unfavorable outcomes reported by Georgiou et al.¹⁸ By contrast, studies showing longer survival in resected, left-sided, or rectal primaries highlight the prognostic importance of tumor site and surgical resectability.²⁴

Our analysis identified obesity as an independent adverse prognostic factor, emerging alongside advanced stage as a key driver of poor outcomes. This observation challenges the so-called “obesity paradox” described in older CRC populations, where excess body weight has sometimes been associated with improved outcomes. Instead, our findings align with growing evidence that metabolic dysregulation accelerates EOCRC risk and fuels its biological aggressiveness. The high prevalence of overweight and obese individuals in our cohort reflects regional lifestyle patterns and underscores the importance of metabolic health in shaping disease trajectory. Conventional markers including performance status, tumor location, and adjuvant therapy, did not retain significance after adjustment, highlighting obesity as a biologically distinct determinant of prognosis. Taken together, these findings reinforce EOCRC as a unique clinical and biologic entity, echoing Xu et al,²⁵ who emphasized the need for integrative models that combine clinical, genomic, and metabolic dimensions. They also support Chen et al,²⁶ who linked metabolic dysregulation to increased EOCRC risk, underscoring obesity not merely as a comorbidity but as a central factor in the pathogenesis and outcomes of this rising disease.

This real-world analysis provides a comprehensive dataset encompassing clinical, molecular, and treatment-related variables across the EOCRC continuum. The inclusion of data on multi-line systemic therapy, adjuvant utilization, and survival outcomes enhances the clinical applicability of our findings.

Nevertheless, the retrospective design introduces important limitations, including documentation inconsistencies and potential selection bias. Details of surgery and radiotherapy, particularly for rectal cancer, were not systematically captured, limiting assessment of local control strategies. Similarly, treatment adherence, toxicity, and reasons for adjuvant omission were unavailable, constraining insight into therapeutic decision-making. The absence of extended molecular profiling (eg, MSI, tumor mutational burden, germline testing) and family history data restricts biologic interpretation. Germline testing, now guideline-recommended for all EOCRC patients,²⁷ was not uniformly available during our study period, limiting our ability to assess hereditary contributions. Its systematic integration is critical for identifying hereditary syndromes, refining risk stratification, and guiding management in this young population.²⁸ Moreover, the lack of a late-onset comparator arm precluded direct age-based comparisons. Finally, the relatively small sample size, interdependence of variables, and small subgroup numbers increase the risk of chance findings, limiting the robustness of multivariable Cox regression results. These prognostic associations should therefore be interpreted with caution.

Our results are consistent with global observations of late-stage EOCRC presentation, including among individuals without overt risk factors, reinforcing the call to revisit screening approaches in high-incidence populations.²⁹ Patterns of adjuvant therapy underutilization and treatment sequencing, even among patients with favorable performance status, highlight an opportunity to optimize care delivery. The modest prevalence of RAS/BRAF mutations and predominance of distal tumors further underscore the need for subtype-specific research. Early therapeutic intensification and personalized care pathways may help improve outcomes in this distinct patient group.

Future multicenter studies with larger sample sizes, integrated molecular profiling, and age-matched comparators are essential to unravel the heterogeneity of EOCRC and refine age-specific treatment strategies. Expanding biomarker-driven and immunotherapy research in this population will guide the development of novel therapeutic approaches. Beyond treatment, research must address the psychosocial and economic challenges unique to younger patients. Embedding survivorship care, fertility preservation, and quality-of-life measures into clinical pathways is critical. Given its rising incidence, EOCRC warrants urgent integration into clinical guidelines, research priorities, and public health strategies.

In conclusion, this study underscores the aggressive clinical behavior and unique management challenges of EOCRC. Despite their younger age and favorable fitness, many patients presented with advanced-stage disease, adjuvant therapy was underutilized, and systemic regimens showed limited durability. Obesity, alongside metastatic presentation, emerged as a distinct adverse prognostic factor, reinforcing the need to integrate metabolic health into risk stratification. Together, these findings highlight EOCRC as a biologically aggressive and clinically unique entity that requires earlier detection strategies, optimized treatment pathways, and molecularly informed precision care. As incidence continues to rise, EOCRC warrants prioritization in clinical guidelines, research agendas, and public health policies.

Data Access and Responsibility

Emad Tashkandi had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Abbreviations

AJCC, American Joint Committee on Cancer; BMI, body mass index; CEA, carcinoembryonic antigen; CI, confidence interval; CRC, colorectal cancer; ECOG, Eastern Cooperative Oncology Group; EOCRC, early-onset colorectal cancer; HR, hazard ratio; IQR, interquartile range; IRB, Institutional Review Board; KAMC, King Abdullah Medical City; MSI, microsatellite instability; OS, overall survival; PFS, progression-free survival; RAS, rat sarcoma viral oncogene; BRAF, v-raf murine sarcoma viral oncogene homolog B1; TMB, tumor mutational burden; UPF, ultra-processed foods; XELOX, capecitabine plus oxaliplatin; FOLFOX, folinic acid, fluorouracil, and oxaliplatin; FOLFIRI, folinic acid, fluorouracil, and irinotecan; TAS-102, trifluridine/tipiracil.

Data Sharing Statement

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Statement

The study protocol was approved by the IRB of KAMC, Makkah, Saudi Arabia (IRB no. 22-965). The need for informed consent was waived because de-identified data was used. All procedures were performed in accordance with the principles outlined in the Declaration of Helsinki.

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Disclosure

The authors declare no conflicts of interest.

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