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EDITORIAL COMMENTARY

THE ALARMING SURGE OF EARLY ONSET COLORECTAL CANCER: A CALL FOR EARLY DETECTION AND PREVENTATIVE MEASURES

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The past decade has witnessed a concerning rise in colorectal cancer (CRC) among individuals under 50 years of age, a group now categorized as having early-onset colorectal cancer (EO-CRC) (1). Projections suggest that by 2030, EO-CRC will account for 11% of all colon cancer cases and 23% of rectal cancer cases (1). While approximately 20% of EO-CRC cases can be attributed to hereditary cancer syndromes, the majority occur in individuals without a known genetic predisposition (2). Factors such as increased antibiotic use, decreased physical activity, rising obesity rates, and lifestyle changes that have accelerated since the 1950s are believed to influence the gut microbiome and may be contributing to this alarming trend (3, 4). Unfortunately, due to the absence of routine screening for younger populations, many EO-CRC cases are diagnosed at advanced stages, showing the urgent need for comprehensive, long-term studies that start in childhood to elucidate the environmental and biological factors driving this disease.

Globally, CRC is named the third most common cancer, along with being one of the leading causes of cancer-related mortality (5). In high-income countries, effective screening programs have led to a reduction in CRC incidence among the population 50-75 years of age. However, these successes are overshadowed by the disturbing rise in CRC among those under 50 years (6-10). The most pronounced increase in EO-CRC cases is seen in the 20-39 year age group (7, 11), prompting reconsideration of the current age thresholds for CRC screening. Although the exact causes of this surge are not fully understood, they appear to be linked to birth cohort effects and lifestyle changes after the year 1950, including shifts in diet, physical activity, and antibiotic use (7, 12, 13).

While a small proportion of EO-CRC cases are linked to hereditary colon cancer syndromes, the majority occur in individuals without a familial history of the disease (2). The first significant report of rising CRC rates among young adults in the United States (US) was declared in 2003 with the through Surveillance, Epidemiology, and End Results (SEER) program data, covering 1973-1999 (14). During this period, CRC rates stabilized among older adults but increased by 17% for colon cancer and a staggering 75% for rectal cancer among younger adults, who often present tumors in more advanced stages, further highlighting the severity of the trend (14).

Subsequent analysis by the American Cancer Society in 2009, utilizing SEER data from 1992-2005, reinforced this troubling pattern (15). Research has shown a decline in CRC risk for individuals born before 1950 but a significant increase for those born between 1950 and 1990 (16). Notably, individuals born around 1990 have an increased risk of colon cancer by two folds, and the rectal cancer by four folds in comparison to the ones born around 1950 (16).

This phenomenon is not limited to the United States; similar increases in EO-CRC incidence have been observed in other high-income countries (17). By 2030, CRC cases among 20-34-year-olds in the US are expected to surge by 90% for colon cancer and 124.2% for rectal cancers (1). While CRC incidence is

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declining among those over 65 years, the annual increase in colon and rectal tumors among younger individuals in the US is approximately 2% (18, 19).

Numerous risk factors are thought to take part in EO-CRC formation, including type 2 diabetes mellitus, obesity, Western dietary patterns, sedentary lifestyles, antibiotic use, alcohol consumption, and smoking (20-25). Collectively, these factors play a role in the growing incidence of EO-CRC. Additionally, EO-CRC often presents distinct histopathological and molecular characteristics compared to sporadic CRC, including higher rates of perineural or venous invasion, poor differentiation, mucinous or signet-ring cell histology, with generally poorer prognoses (26). At the molecular level, EO-CRCs are more likely to be microsatellite-stable, with frequent long interspersed nuclear element-1 (LINE-1) hypomethylation and TP53 mutations, and less frequent alterations in Kirsten rat sarcoma viral oncogene homologue (KRAS), v-raf murine sarcoma viral oncogene homolog B1 (BRAF), and adenomatous polyposis coli (APC) genes (27).

In conclusion, the aggressive nature of EO-CRC and its frequent diagnosis at advanced stages significantly worsen patient outcomes. Given the increasing incidence and unique characteristics of EO-CRC, it is imperative that we enhance our understanding of its underlying causes and reconsider screening practices to improve early detection and outcomes in this vulnerable population (28).

Ethics

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Footnotes

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