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WORLDWIDE OF COLORECTAL CANCER: A REVIEW

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ABSTRACT

Colorectal cancer is the third most frequently diagnosed cancer and the fourth leading cause of cancer mortality globally, making it a significant public health issue. Due to varying exposure to risk factors, screening introduction and uptake, and availability to appropriate treatment options, there is significant variance over time across various geographic regions. Indeed, socioeconomic position accounts for a significant part of the differences. Although colorectal cancer is still mostly a disease of the developed world, it is becoming more common in emerging nations. Furthermore, the worldwide burden is projected to rise owing to population expansion and aging, as well as the adoption of westernized habits and lifestyles. Colorectal cancer screening has been shown to significantly lower death rates, which have decreased in many industrialized and developing nations. Statistics on colorectal cancer incidence are necessary for developing focused measures to reduce the disease's impact. The purpose of this article is to give an overview of colorectal cancer incidence, mortality, and survival rates, as well as regional differences and historical trends.

KEYWORDS: *Colorectal Cancer, Diagnosed Cancer, Epidemiology, Health Issue, Incidence Mortality.*

1. INTRODUCTION

Colorectal cancer is one of the main causes of death and morbidity all over the globe, making it a significant public health issue. It is the third most prevalent cancer in the world (after lung and breast cancers) and the fourth leading cause of oncological mortality. Due to varying exposure to risk factors, the introduction of preventative measures, and the development of therapies, epidemiological data varies throughout time across various geographic regions. Accurate data on cancer incidence and outcome are critical for research as well as the design and assessment of cancer control initiatives. The purpose of this article is to provide a thorough review of CRC incidence, mortality, and survival rates, as well as regional variations and temporal trends.

Incidence CRC is the third most prevalent cancer in men and the second most common cancer in women globally. It is responsible for more than 9% of all cancer cases, with an estimated 1.4 million cases in 2012. The frequency of the disease varies greatly throughout the globe, with almost 55 percent of cases happening in more developed nations. Different food and environmental exposures may be placed on a backdrop of genetically determined sensitivity, resulting in regional variations. Australia, New Zealand, Europe, and Northern America have the greatest rates of infection(1–4).

In Africa, South-Central Asia, and Central America, on the other hand, incidence rates are low. CRC was the second most prevalent cancer after breast cancer, according to statistics from the Italian Association of Cancer Registries from 2007 to 2011. CRC is now the most common cancer in the Italian population, with almost 52,000 new cases predicted in 2015. It is the third most prevalent cancer in males, behind prostate and lung cancers, accounting for 14% of all cancer cases. CRCs account for 13% of all new malignancies in women, and they are the second most common malignancy after breast cancer(5). Incidence rates vary tenfold across the world, with the greatest rates estimated in Australia/New Zealand and the lowest in Western Africa. Both sexes have comparable geographic trends.

CRC rates, on the other hand, have been increasing in emerging nations. Western Asia (Kuwait and Israel) and Eastern Europe have seen the most significant rises (Czech Republic, Slovakia and Slovenia). This rise may be due to an increase in the prevalence of CRC risk factors linked to westernization, such as poor nutrition, obesity, and smoking(6–10). Furthermore, owing to population expansion and aging, the worldwide burden of CRC is projected to rise much more. Low socioeconomic status has been linked to a higher risk of CRC. Even after controlling for other risk factors, a study of 7676 patients diagnosed with primary CRC among 506,488 participants found that people with low educational levels or who lived in low-socioeconomic status neighborhoods had a significantly higher incidence rate than those with higher educational levels or who lived in high-socioeconomic status neighborhoods. This disparity may be explained by a greater prevalence of theoretically modifiable risk factors (physical inactivity, poor diet, smoking, obesity) as well as lower rates of smoking(11–14).

In most areas of the globe, males have much greater incidence of cancer than women. The causes for this variation are unknown, although they are likely to represent complicated interactions between sex-specific risk factor exposure and protective effects of both endogenous and exogenous hormones, as well as gender-specific screening methods. The age gap between men and women varies. For example, the male-to-female incidence ratio in the United States was found to be 1.1 from birth to 49 years, 1.4 from 50 to 79 years, and 1.2 from 80 years and older. The risk of CRC increases as one gets older. CRC is rare in individuals under the age of 40; however, between the ages of 40 and 50, the incidence of CRC starts to rise substantially, and age-specific incidence rates continue to rise in each subsequent decade. However, several cancer registries showed an increase in the incidence of large bowel cancer, especially rectal cancer, among young people under the age of 40(15). A change in the colonic site localization of CRC has been observed in many investigations, with a greater percentage of tumors developing in the right colon. Females have a higher percentage of proximal tumors than males, and older individuals have a higher proportion of proximal tumors than younger ones. Although biologic changes may have happened, enhanced screening, which is more successful in identifying and preventing left-sided CRCs than right-sided CRCs, may have contributed to this shift in the anatomic location of CRC.

1.1 Mortality CRC:

Mortality CRC is the fourth most common cause of cancer death in men and the third in woman worldwide. Almost 693,900 deaths from CRC are estimated to have occurred in 2012, accounting for approximately 8 % of all cancer deaths. There is less variability in mortality rates worldwide (sixfold in men, fivefold in women), with the highest mortality rates in both sexes

estimated in Central and Eastern Europe (20.3 per 100,000 for males, 12.1 per 100,000 for females), and the lowest in Middle Africa. Conducted a study about mortality trends for 29 selected countries. The authors found that CRC mortality rates have declined in many longstanding as well as newly economically developed countries such as the United States, Australia, New Zealand, the majority of Western Europe (Austria, France, Germany, Spain, Ireland, and the United Kingdom), some Asian (Japan) and Eastern European countries (Czech Republic, Latvia, Slovakia), and South Africa. These improvements in mortality rates are thought to be a result of CRC prevention and earlier diagnosis through screening as well as reduced prevalence of risk factors, and/or availability of improved treatment regimens. In Italy, CRC was the second most common cause of cancer death (preceded by lung cancer in men and breast cancer in woman), with 19.202 cases occurred in 2012. CRC mortality rates have been decreasing since 1999 of 0.6 % per year in men and 1.2 % per year in women. However, increases in mortality rates are still occurring among both males and females in some low-resource countries, including Mexico, Brazil, Chile and Ecuador in Southcentral America and Romania and Russia in Eastern Europe. Increasing mortality rates may reflect increasing CRC incidence trends as well as a lack of prevention measures(16–19).

1.2 Natural history of colorectal cancer:

i. Key stages and features:

The natural history of CRC can be divided into four major stages: initiation, promotion, progression and metastasis. Initiation involves irreversible genetic damage that predisposes the affected cells to subsequent neoplastic transformation. In the promotion stage, the initiated cells proliferate, inducing abnormal growth. In the subsequent progression phase, by undergoing further genetic and epigenetic alterations that could confer a selective growth advantage to cells, benign tumor cells transform into malignant cancer cells and acquire aggressive characteristics and metastatic potential(20). Metastasis is marked by the spread of cancer cells from the primary organ to other organs or tissues through the bloodstream or the lymphatic system. The duration of each phase is difficult to accurately estimate and has wide ranges; these processes generally take a long time and decades are required for all stages to be completed in CRC30. For hereditary CRCs, progression through some of the stages can be more rapid. Notably, there has been an increasing consideration of the presence of cancer stem cells, which could have a role in colorectal carcinogenesis by dividing rapidly and continuously into cancer cells or identical daughter cells and thus forming a proliferative cancer cell population.

ii. Carcinogenic pathways:

CRCs arise through distinct carcinogenic pathways: adenoma–carcinoma sequence, serrated pathway and inflammatory pathway. The adenoma–carcinoma sequence, wherein adenoma serves as the precursor to CRC, is the classic pathway that explains the majority of CRCs. In this model, gradual stepwise accumulation of genetic and epigenetic alterations drives the transformation of normal cells to small adenoma, to large adenoma and, finally, to cancer. Inactivating mutations in APC, a tumor suppressor gene regarded as the gatekeeper against colorectal neoplasms, result in over activation of the Wnt/β-catenin signaling pathway, triggering dysregulated cell proliferation and adenoma development. Subsequent mutations of the oncogene KRAS promote adenoma growth and ensuing inactivation of TP53 tumor suppressor gene contributes to the progression to CRC47. The adenoma– carcinoma pathway is predominantly

associated with the development of the CIN-positive CRC subtype, although it remains unclear whether CIN underlies the accumulation of mutations in the critical tumors suppressor genes and oncogenes, or vice versa48. Nevertheless, in a mouse model with established colorectal tumors induced by Apc knockout, restoration of APC function led to tumor cell differentiation and sustained regression regardless of Kras and Tp53 mutation status in lesion.

iii. Subtypes of colorectal cancer:

Although CRC develops in a single organ, namely the large intestine, it is a highly heterogeneous disease consisting of subtypes with variant etiology and clinical outcomes. Traditionally, subtypes of CRC have been defined by tumor anatomical site in three segments of the colorectal: proximal colon (caecum, ascending colon, hepatic flexure and transverse colon), distal colon (splenic flexure, descending colon and sigmoid colon) and rectum. Studies have shown that CRCs at different anatomical subsides have distinct risk factors (for example, smoking was associated with increased risk of proximal colon cancer and rectal cancer but not with distal colon cancer). Etiological heterogeneity of CRC across the tumor locations might, in part, relate to variations in microbial and host characteristics of the large intestine(21). Along the colorectal axis from proximal colon to rectum, there is a progressive increase in pH, microbial loads and short-chain fatty acid abundance, which could have divergent implications for colorectal carcinogenesis.

iv. Genetic risk factors:

The cumulative risk of developing CRC before age 75 years is estimated to be 5% in the general population from a high-incidence country. The lifetime risk of CRC is considerably increased when individuals have a family history of CRC or hereditary cancer syndromes. For individuals with a family history of CRC, a meta- analysis of observational studies found the risk of CRC increased by fold for those with at least one affected first-degree relative for those with at least two affected first-degree relatives(22). The associations became stronger when the relatives were diagnosed with CRC before age 50 years. Except for rare hereditary cancer syndromes, most of the known inherited mutations, albeit genetically predisposing to CRC, are of low penetrance. Thus, a substantial proportion of CRCs clustered in families are not inherited, but occur through acquired genomic aberrations, which points to the importance of environmental risk factors in modulating CRC risk.

v. Lifestyle and nutritional factors:

Genetics contribute to individual risk2 , but CRC incidence in a population are largely affected by modifiable diet and lifestyle factors because rates can change dramatically over short courses of time and migrants from countries with low CRC rates rapidly take on the high rates of their adoptive country15,99. The 2017 extensive summary report by the World Cancer Research Fund and American Institute of Cancer Research, based on a systematic review of studies available globally, concluded that obesity, low physical activity, poor diets and alcohol increase CRC risk. By population-attributable fraction, a measure of public health impact of exposure to a risk factor on a disease outcome in a population100, 47% of CRC cases in the USA and 45% in the UK were estimated to be attributable to the aforementioned modifiable risk factors. In addition, smoking increases CRC risk101. This constellation of factors is probably the driverof increasing CRC incidence in populations undergoing economic transition.

vi. Obesity:

Excess adiposity is an established risk factor for CRC, which is consistently supported by epidemiological studies using diverse anthropometric measures97,98. The two most commonly used measures are BMI, which represents overall body fatness, and waist circumference, which largely reflects abdominal fatness. Some evidence suggests that WC is a stronger risk factor for CRC than BMI102,103. For instance, when BMI and WC were simultaneously included in the statistical model for colon cancer risk, the relative risk for BMI diminished but not the relative risk for WC.

Abdominal fat is further categorized into two distinct compartments: visceral adipose tissue and subcutaneous adipose tissue. Compared with SAT, VAT secretes more pro-inflammatory adipocytes and less adiponect in, and is more heavily infiltrated with immune cells. All of these traits contribute to the development of chronic low-grade systemic inflammation and insulin resistance106. As exemplified by colitis-induced CRC, inflammatory conditions in the tumor microenvironment promote tumor growth and progression(23).

2. DISCUSSION

The optimal reduction of CRC incidence and mortality will require concerted efforts to reduce modifiable risk factors, to leverage chemoprevention research and to promote population-wide and targeted screening. The effort devoted to each approach needs to be balanced with the overall health priorities of the specific population, taking economic resources and health-care infrastructure into account. The current study was limited by data availability, because incidence data are not available for all countries and in most instances are only region-specific. Although mortality data are more complete, it is possible that the increasing mortality trends noted in some countries could be the result of improvements in death certification systems or data abstraction. screening and/or improved treatment, have been observed in a large number of countries examined; however, decreasing colorectal cancer mortality rates, most likely due to colorectal cancer increases in mortality rates are still occurring in countries that may have more limited resources, including Mexico and Brazil in South America and Romania and Russia in Eastern Europe, compared with longstanding, economically developed countries

3. CONCLUSION

Worldwide, colorectal cancer incidence rates are highest in the registries of newly economically developed countries such as the Czech Republic and Slovakia in Eastern Europe, and also remain high in longstanding, economically developed countries such as Japan and Australia as well as the majority of registries in Western Europe and North America. CRC constitutes an enormous burden worldwide that is expected to increase due to the growth and aging of the population, and because of the adoption of at-risk behaviors and lifestyle, especially in economically less developed countries. Low socioeconomic status may be an important underlying factor accounting for disparities in incidence, mortality and survival rates for CRC. Screening has been proven to greatly reduce mortality and may prevent the onset of the disease. Greater international efforts are needed to put into practice targeted prevention strategies that could alleviate the burden of CRC worldwide. In addition, as people continue to live longer, colorectal cancer will become an even greater public health problem worldwide. Colorectal cancer screening has been proven to greatly reduce mortality and in some instances may prevent the onset of disease

through the removal of precancerous polyps. The variety of existing screening tests makes colorectal cancer screening accessible for most countries, and therefore, greater international consideration of targeted screening programs and/or screening recommendations could help to alleviate the burden of colorectal cancer worldwide.

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