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Rectal and Colon Cancer: Not just a Different Anatomic Site

Lee Le Ye [1]

[1]*Senior Consultant, Department of Neonatology, Khoo Teck Puat - National University Children's Medical Institute, National University Hospital, National University of Singapore, Sengkang, Singapore*

[*]**Corresponding author:** Dr. Rosivall Laszlo, Senior Consultant, Department of Neonatology, Khoo Teck Puat - National University Children's Medical Institute, National University Hospital, National University of Singapore, Singapore, Tel: +65 6772 5075; E-Mail: paeangj@nus.edu.sg

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Introduction

Due to differences in anatomy, primary rectal and colon cancer require different staging procedures, different neo-adjuvant treatment and different surgical approaches. For example, neoadjuvant radiotherapy or chemo radio therapy is administered solely for rectal cancer. Neodjuvant therapy and total mesorectal excision for rectal cancer might be responsible in part for the differing effect of adjuvant systemic treatment on overall survival, which is more evident in colon cancer than in rectal cancer. Apart from anatomic divergences, rectal and colon cancer also differ in their embryological origin and metastatic patterns. Moreover, they harbor a different composition of drug targets, such as v-raf murine sarcoma viral oncogene homolog B (BRAF), which is preferentially mutated in proximal colon cancers, and the Epidermal Growth Factor Receptor (EGFR), which is prevalently amplified or overexpressed in distal colorectal cancers. Despite their differences in metastatic pattern, composition of drug targets and earlier local treatment, metastatic rectal and colon cancer are, however, commonly regarded as one entity and are treated alike. In this review, we focused on rectal cancer and its biological and clinical differences and similarities relative to colon cancer. These aspects are crucial because they influence the current staging and treatment of these cancers, and might influence the design of future trials with targeted drugs. Health care professionals are certain that colorectal cancer is not contagious (a person cannot catch the disease from a cancer patient). Some people are more likely to develop colorectal cancer than others. Factors that increase a person's risk of colorectal cancer include increasing age, African-American race, high fat intake, a family history of colorectal cancer and polyps, and the presence of polyps in the large intestine, and inflammatory bowel diseases, primarily chronic ulcerative colitis. Age: Increasing age is the main risk factor for colorectal cancer. Around 90% of colorectal cancers are diagnosed after age 50. Race: African Americans have a higher incidence of colorectal cancer than people of other races.

Research has shown that most colorectal cancers develop in colorectal polyps. Therefore, removing benign (but precancerous) colorectal polyps can prevent colorectal cancer. Precancerous colorectal polyps are most commonly called adenomatous polyps. They develop when chromosomal damage occurs in cells of the inner lining of the colon. The damage produces abnormal cells, but the cells have not yet developed the ability to spread, the hallmark of cancer. Instead, the growing tissue remains localized within the polyp. When chromosomal damage increases further within the polyp, cell growth becomes uncontrolled, and the cells begin to spread, that is, they become cancer. Thus, colon polyps which are initially benign acquire additional chromosome damage to become cancerous. Chronic ulcerative colitis causes inflammation of the inner lining of the colon. Bowel cancer is a recognized complication of chronic ulcerative colitis. The risk for cancer begins to increase after eight to 10 years of colitis. Patients at higher risk of cancer are those with a family history of colon cancer, a long duration of ulcerative colitis, extensive colon involvement with ulcerative colitis, and those with ulcerative colitis-associated liver disease, sclerosing cholangitis. Since the cancers associated with ulcerative colitis have a more favorable outcome when caught at an earlier stage, yearly examinations of the colon often are recommended after eight years of known extensive disease. During these examinations, samples of tissue (biopsies) are taken to search for precancerous changes in the cells lining the colon. When precancerous changes are found, removal of the entire colon may be necessary to prevent colon cancer. A person's genetic background is an important factor in colon cancer risk. Having a first-degree relative with colorectal cancer, especially if the cancer was diagnosed before the age of 55 years, roughly doubles the risk of developing the condition.

Even though a family history of colon cancer is an important risk factor, a majority (80%) of colon cancer occurs sporadically inpatients with no family history of coloncancer.Approximately20% of cancers are associated with a family history of colon cancer. Diets high in fat have been shown in numerous research studies to predispose people to colorectal cancer. In countries with high colorectal cancer rates, the fat intake by the population is much higher than in countries with low cancer rates. It is believed that the digestion of fat that occurs in the small intestine and the colon leads to the formation of cancer-causing chemicals (carcinogens). Likewise, research studies also reveal that diets high in vegetables and high- fiber foods such as whole-grain breads and cereals contain less fat that produces these carcinogens and may counter the effects of the carcinogens.