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Editorial

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A Report on Rectum and Colonal Cancers

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Editorial

Cancers of the colon and rectum (colorectal cancer) start when the process of the normal replacement of colon lining cells goes awry. Mistakes in cell division occur frequently. For reasons that are poorly understood, sometimes mistakes occur that escape our editing systems. When this occurs, these cells begin to divide independently of the normal checks and balances that control growth. As these abnormal cells grow and divide, they can lead to growths within the colon called polyps. Polyps vary in type, but many are precancerous tumors that grow slowly over the course of years and do not spread. As polyps grow, additional genetic mutations further destabilize the cells. When these precancerous tumors change direction (growing into the wall of the tube rather than into the space in the middle of it) and invade other layers of the large intestine (such as the sub mucosa or muscular layer), the precancerous polyp has become cancerous. In most cases this process is slow, taking at least eight to 10 years to develop from those early aberrant cells to a frank cancer. Colorectal cancer is typically an adenocarcinoma, a term that refers to a cancer that has formed in certain types of lining tissues in the body. Once a colorectal cancer forms, it begins to grow in two ways. First, the cancer can grow locally and extend through the wall of the intestine and invade adjacent structures, making the mass (called the primary tumor) more of a problem and harder to remove. Local extension can cause additional symptoms such as pain or fullness, perforation of the colon, or blockages of the colon or nearby structures. Second, as the cancer grows it begins the process of metastasis, shedding thousands of cells a day into the blood and lymphatic system that can cause cancers to form in distant locations. Colorectal cancers most commonly spread first to local lymph nodes before traveling to distant organs. Once local lymph nodes are involved, spread to the liver, the abdominal cavity, and the lung are the next most common destinations of metastatic spread. Colorectal cancer is the third most common cause of cancer in the U.S. in both men and women. It affects over 135,000 people annually, representing 8% of all cancers. About 4.3% of people will be diagnosed with colon or rectum cancer at some point in their lives.

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.

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. Both effects would help reduce the risk of cancer.

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. The risk of developing colon cancer in a patient with ulcerative colitis also is related to the location and the extent of his or her disease.

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 cancers occur sporadically in patients with no family history of colon cancer. Approximately 20% of cancers are associated with a family history of colon cancer.

Chromosomes contain genetic information, and chromosomal damage causes genetic defects that lead to the formation of colon polyps and later colon cancer. In sporadic polyps and cancers (polyps and cancers that develop in the absence of family history), the chromosome damages are acquired (develop in a cell during adult life). The damaged chromosomes can only be found in the polyps and the cancers that develop from that cell. But in hereditary colon cancer syndromes, the chromosomal defects are inherited at birth and are present in every cell in the body. Patients who have inherited the hereditary colon cancer syndrome genes are at risk of developing colon polyps, usually at young ages, and are at very high risk of developing colon cancer early in life; they also are at risk of developing cancers in other organs. Familial adenomatous polyposis (FAP) is one hereditary colorectal cancer syndrome where the affected family members will develop countless numbers (hundreds, sometimes thousands) of colon polyps starting during their teens. Unless the condition is detected and treated early (treatment involves removal of the colon), a person affected by FAP is almost sure to develop colon cancer from these polyps. Cancers almost certainly develop by the time a person is in their 40s. These patients are also at risk of developing other cancers such as cancers in the thyroid gland, stomach, and the ampulla (part of the bile duct where it drains into the small intestine from the liver) as well as benign tumors called desmoid tumors. FAP arises from a mutation in a specific gene called the APC gene. The specific mutation can be identified in most people with appropriate testing, and such testing is recommended for individuals diagnosed with FAP as well as their family members.

Attenuated familial adenomatous polyposis (AFAP) is a milder version of FAP. Affected members develop fewer than 100 colon polyps. Nevertheless, they are still at very high risk of developing colon cancers at a young age. They are also at risk of having gastric polyps and duodenal polyps. Hereditary non-polyposis colon cancer (also known as Lynch syndrome or HNPCC) is a hereditary colorectal cancer syndrome where affected family members can develop colon polyps and cancers, usually in the right colon, in their 30s to 40s. Patients with HNPCC are also at risk of developing uterine cancer, stomach cancer, ovarian cancer, and cancers of the ureters (the tubes that connect the kidneys to the bladder), and the bile ducts. Ironically, it appears that while colon cancer occurs more frequently in patients with HNPCC, these cancers may be more easily cured than "sporadic" colon cancers. The specific genetic abnormalities associated with HNPCC have been identified, and patients and family members can be tested to determine if HNPCC is present and if family members carry the abnormality and are likely to develop cancer.

MYH polyposis syndrome is a recently discovered hereditary colorectal cancer syndrome. Affected members typically develop 10 to 100 polyps at around 40 years of age and are at high risk of developing colon cancer. Here, too, the genetic abnormality has been identified.

It is important to remember that the overwhelming majority of colorectal cancers do not have a single, identifiable chromosomal abnormality that can be looked for in relatives in order to identify individuals at risk for colorectal cancer.

Survival rates for any cancer are often reported by stage, the extent of spread when the cancer is identified. For colon and rectum cancer, around 39% are diagnosed at the local stage, before the cancer has spread outside the local area. The five-year survival for these patients with localized colon and rectum cancer is around 90%. When the cancer has spread to the regional lymph nodes near the site of origin, the five-year survival rate is about 71%. When the cancer has metastasized to distant sites in the body (stage IV cancer), the five-year survival rate lowers to about 14%. The most effective prevention for colorectal cancer is early detection and removal of precancerous colorectal polyps before they turn cancerous. Even in cases where cancer has already developed, early detection still significantly improves the chances of a cure by surgically removing the cancer before the disease spreads to other organs. Regular physical activity is associated with lower risk of colon cancer. Aspirin use also appears to lower the risk of bowel cancer. The use of combined estrogen and progestin in hormone replacement therapy lowers the risk of colon cancer in postmenopausal women. Hormone replacement therapy has risks which must be weighed against this effect, and should be discussed with a doctor.