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Early Detection of Colorectal Cancer is Key to a High Cure Rate

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1. Abstract

Colon cancer is a serious illness that begins in the colon's lining, frequently developing from polyps, particularly in individuals over 50 years old. Identifying it early through screening tests is essential for improving the chances of recovery. Some common signs are blood in the stool, changes in how often one uses the bathroom, discomfort, and unexplained weight loss.

2. Introduction

Colorectal cancer ranks as the third most frequently diagnosed cancer and the second leading cause of cancer-related deaths in the United Kingdom [1]. The likelihood of getting this cancer is closely tied to age, with nearly 85% of cases found in people aged 60 and older, although there has been a rise in cases among those under 50 since the 1990s. This cancer is primarily found in developed countries, with New Zealand, Canada, the United States, and the United Kingdom having the highest rates. There is no preference for either gender.

Colorectal cancer usually stems from the change of adenomatous polyps [2]. In 80% of situations, these cases appear randomly, while 20% have a genetic link. Factors that may increase risk include long-term ulcerative colitis and granulomatous colitis, and the risk of cancer grows with how long these conditions last. People at high risk often follow a diet low in fiber and high in animal proteins, fats, and processed carbs. Although carcinogens can enter the body through food, they are more often caused by bacteria interacting with nutrients or bile and intestinal fluids. The disease can spread directly through the intestinal wall, via the bloodstream, to local lymph nodes, around nerves, and within the lining of the intestine. This form of cancer leads to unchecked growth of abnormal cells in the colon or rectum. These cells can multiply rapidly, invading normal tissue or spreading to distant organs through blood or lymph. If caught and treated early, while the tumour remains confined to the digestive tract, the chances of curing the cancer are very high. However, once the cancer reaches the outer wall of the intestine and involves lymph nodes, the situation becomes much more difficult.

3. Types

Colorectal cancer is mainly seen as an environmental issue, with most cases linked to lifestyle choices and aging, while only a few arise from genetic factors [1].

Many of the lifestyle habits believed to increase the risk of developing colorectal adenocarcinoma are related to diet, with higher consumption of red meat, alcohol, and calories significantly contributing. Other contributing factors include smoking, lack of physical activity, and being overweight. This type of cancer is more prevalent in individuals with inflammatory bowel disease, and the risk escalates with the duration and severity of the condition.

About 5% of colorectal cancer cases are linked to genetic syndromes, with hereditary non-polyposis colorectal cancer (HNPCC or Lynch syndrome type I) being the most frequent. HNPCC is caused by germ-line mutations in several DNA mismatch repair genes and leads to a 40% lifetime chance of developing colorectal cancer. This condition is inherited in an autosomal dominant manner. Individuals with HNPCC usually develop colorectal cancer during their 30s or 40s. Other cancers including ovarian, endometrial, gastric, urinary, and hepatobiliary cancers can also occur, which are referred to as Lynch syndrome type II.

Familial adenomatous polyposis (FAP) is a less common hereditary form of colorectal cancer. It follows an autosomal dominant inheritance pattern and results from mutations in the APC tumour suppressor gene. Patients affected by this condition begin to develop numerous benign polyps in their colon from a young age, which typically turn into cancerous growths in their 30s or 40s. Therefore, a preventive colectomy is highly recommended.

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4. Subtypes

Recently, four consensus molecular subtypes (CMS) of colorectal cancer have been identified [3]. This classification is mainly used for research purposes rather than for everyday patient treatment. CMS 1 tumors (MSI immune, 14%) show signs of hypermutation, microsatellite instability (MSI), and significant immune activation. CMS2 (canonical, 37%) are epithelial tumours with chromosomal instability (CIN) and active WNT and MYC signalling. CMS3 (metabolic, 13%) are also epithelial but are marked by problems in metabolism. Lastly, CMS4 (mesenchymal, 23%) display strong activation of transforming growth factor-β (TGF-β), invasion of the surrounding tissues, and new blood vessel formation. The remaining 13% show mixed characteristics.

Microsatellite instability (MSI) colorectal cancers, a key part of the CMS1 category, are the first kind of colorectal cancers where immunotherapy has shown success. This type of cancer is known for very high mutation rates, mainly due to issues in the mismatch-repair genes (MMR). MMR helps cells fix their damaged DNA. Specifically, it identifies and repairs mistakes like insertions, deletions, and incorrect DNA base matches during DNA replication. When the MMR system fails, it leads to increased mutations. MSI is a sign of nonfunctioning mismatch-repair proteins in a tumour and was first used to find patients who might need germline testing for Lynch syndrome. However, it is crucial to realize that while 15% of colorectal cancers are classified as MSI-H, only about 3% of all colorectal cancers have a germline MMR mutation (indicating Lynch syndrome). Therefore, most MSI-H colorectal cancers are sporadic, resulting from acquired somatic defects in MMR gene function, commonly due to hypermethylation of the MLH1 promoter.

5. Polyps

The onset of colorectal cancer (CRC) generally starts with the benign expansion of mucosal epithelial cells, leading to the creation of formations called polyps [4]. These polyps can develop very gradually over a span of 10 to 20 years before they might turn cancerous. The most prevalent type of polyp is adenoma, which comes from gland cells that produce mucus to coat the large intestine. As these polyps increase in size, the likelihood of cancer also rises, with around 10% of adenomas advancing to invasive cancer. Those polyps that become invasive are termed "adenocarcinomas," making up 96% of all cases of CRC.

Colorectal cancer originates from the lining of the colon or rectum and can invade lymph vessels or blood vessels, allowing it to spread to far-off organs through nearby lymph nodes or the bloodstream. CRC staging includes cancers that have not broken through the wall of the colon or rectum, which are labelled in situ and are not counted as CRC. When cancers have breached the walls but remain contained, they are categorized as local cancers. Regional cancers have spread into adjacent lymph nodes or tissues. Distant cancers have moved through the bloodstream to faraway organs like the liver or lungs.

6. Risks

Unknown carcinogenic substances that are unintentionally consumed with food and drinks may directly affect the cells in the mucosa of the colon and rectum if they are not neutralized, absorbed, or processed in the stomach and small intestine [5]. Growing evidence from studies shows that specific eating habits, alcohol use, being overweight, and not exercising are common risk factors for colorectal cancer.

A group of global experts has determined that red meat, processed meats, and high alcohol intake significantly contribute to the risk of developing colorectal cancer, according to strong evidence from comprehensive studies. Foods that have dietary fibre, along with garlic, milk, and calcium, might help protect against this illness.

Fatty red meat items such as deli meats, sausages, and parts of cattle could elevate risk likely due to their high saturated fat levels. Diets that are high in fat, primarily those packed with cholesterol and saturated fats, may increase the chances of colon cancer due to their calorie richness. Alternatively, they could result in heightened levels of bile acids in the colon or an imbalance in the ratio of conjugated linoleic acid (CLA). Moreover, the intake of protein, iron, and harmful compounds from cooking has been implicated. Heterocyclic amines formed during the preparation of red meat are strong mutagens and carcinogens. The type of beef favoured by South American communities for grilling or cooking in iron pans tends to be fatty (30 to 33% of total fats). Therefore, poor quality cuts are exacerbated when risky cooking methods, like high temperatures and extended exposure to charcoal smoke, are used. These combinations likely increase the production of heterocyclic amines.

Since various types of meat share comparable protein levels, one can conclude that the primary differences lie in the quantity and quality of fat content. The fat percentage in meat varies from 4. 5% to at least 37% in fatty meats. Fats found in beef and dairy products have differing amounts of CLA, which is a potent anti-cancer agent. Notably, CLA exists within unseen fats that are evenly spread along muscle fibres. As a result, the positive effects of conjugated linoleic acid might be more pronounced in lean meats compared to fatty meats and their derivatives.

Research has shown that ethanol itself raises the levels of saturated fatty acids while lowering omega-6 and omega-3 essential fatty acids in rodents and normal as well as tumour cells in humans. This situation has been suggested to promote tumour formation. Overall, a high intake of alcohol, along with substantial consumption of fatty red meat, could work together to enhance the risk of colorectal cancer.

On the flip side, certain dietary aspects, like low folate consumption, are thought to increase the likelihood of colorectal cancer by 2 to 5 times, and alcohol disrupts folate metabolism. Therefore, drinking alcohol and having low folate intake may have a combined effect or alcohol might influence folate metabolism, raising colorectal cancer risk.

Diet significantly influences the development of colorectal cancer. Among its many components, alcoholic beverages have been identified as a strong contributor to this form of cancer in men and likely in women as well.

7. Malignancy

More than 70% of colorectal cancers are adenocarcinomas that develop in the mucous layer from benign adenomatous polyps [1]. Adenomas usually grow slowly, with only a small fraction becoming cancerous if not surgically removed. The risk of turning malignant increases with the size of the adenoma and the duration it has existed. Once a tumour forms, it can invade the bowel wall layers and eventually spread to the liver, lungs, bones, brain, and skin. Several genetic changes have been linked to the transition from benign adenomas to invasive adenocarcinomas.

9. Symptoms

Colorectal cancer has a significant effect on healthcare systems globally [6]. Surgical intervention is the primary treatment for early-stage colorectal cancer. Neoadjuvant or adjuvant chemoradiotherapy may be administered depending on the cancer's stage and type. Surgical operations involve removing parts of the large intestine. Tumour resections can lead to damage to pelvic floor structures and issues with function.

Patients experiencing the disease at various stages often show symptoms related to the pelvic floor. The most common issues include bowel problems, such as urgent needs and a more frequent urge to defecate, as well as faecal incontinence, which can be either liquid or solid. Additional symptoms are gas incontinence, alterations in the nature of faces, embarrassment related to bowel issues, anxiety about toilet access, and lifestyle adjustments necessary to manage these new symptoms, including the use of incontinence pads or altering diets for bowel reasons. It has been observed that colorectal surgery can have negative effects on sexual activities, particularly in young men.

In the initial phases, colorectal cancer usually does not show symptoms, leading to 55% of patients being diagnosed with more advanced stages of the disease. Patients with right-sided cancer tend to show signs like a visible mass in the right lower abdomen, diarrhoea, weight loss, anaemia, and hidden bleeding in the gastrointestinal tract. The symptoms associated with left-sided cancer generally include a visible mass in the left lower abdomen, changes in bowel movements (most often constipation), a constant feeling of needing to have a bowel movement, rectal bleeding, and indications of bowel blockage. Those with left-sided cancer usually seek medical attention earlier compared to those with right-sided cancer.

10. Screening

The US Preventive Services Task Force advises that adults aged 45 to 75 undergo colorectal cancer screening and that those aged 76 to 85 be selectively screened based on their preferences, overall well-being, and previous screening experiences [7]. The type of screening test can vary according to what the patient prefers. One suggested method is to have a colonoscopy every 10 years. Screening with faecal occult blood tests once a year or every two years helps lower death rates from colorectal cancer and is also suggested, with the faecal immunochemical test (FIT) being the most commonly used. CT colonography, also known as virtual colonoscopy, is a non-invasive screening choice for colorectal cancer, demonstrating a strong safety record and performance on par with colonoscopy for detecting larger polyps and cancers.

The American Cancer Society recommends that individuals aged 25 to 65 get screened using primary HPV testing every 5 years, as studies indicate this method is more effective than others. The US Preventive Services Task Force advises that women aged 21 to 65 should have cervical cancer screenings using Papanicolaou tests (Pap tests) every 3 years. For women aged 30 to 65 who prefer less frequent screenings, both cytology and HPV testing every 5 years is an option, although this guideline is being reviewed. Additionally, the Task Force advises against screenings for women below 21 or average-risk women over 65 who have had adequate negative past screenings. Currently, HPV vaccination does not change screening frequencies, though it may have an impact as vaccination rates increase in the future.

The USPSTF advises that individuals who currently smoke and are between the ages of 50 and 80 should receive yearly lung cancer screenings using low-dose CT. This is also applicable to those who have a smoking history of at least 20 pack-years and to those who have quit smoking within the last 15 years. The latest guidelines from the American Cancer Society no longer limit screening based on how long it has been since a person stopped smoking. Screening should cease when a health issue arises that reduces life expectancy to under five years or if the individual is unable to safely receive treatment if lung cancer is found. Screening is intended to support, not replace, the effort to quit smoking.

11. DC Vaccine

Dendritic cells (DC) are essential components of the lamina propria and play a role in various local diseases [8]. Techniques involving mechanical breakdown and chemical digestion of intestinal samples from patients suffering from various colon conditions—such as colorectal cancer, Crohn's disease, ulcerative colitis, and benign, non-inflammatory issues—indicate that DC make up 2% of the cells collected from the lamina propria. Regarding their capacity to activate lymphocytes, suspensions rich in DC provoke a mixed lymphocyte response (MLR) by T cells. Nevertheless, DC that infiltrate tumours show limited ability to activate T lymphocytes during primary allogeneic cultures (MLR) and do not produce significant amounts of IL-2 or IFN-.

The C-type lectin known as DC-SIGN (DC-specific intercellular adhesion molecule-3-grabbing non-integrin) plays a role in recognizing colorectal cancer cells through DC. In tumours within the colon, immature DC that express DC-SIGN-unlike mature DC-bind to tumour cells via the Lewisx and Lewisy carbohydrates present on CEA found in those cells. Notably, DC-SIGN does not interact with CEA from normal colon cells because those cells have low levels of Lewis epitopes. As a result, DC interact with SW1116 colon tumour cells that display abnormally glycosylated

Lewis epitopes (Lea/Leb) on CEA and CEACAM1, leading to the release of immunosuppressive cytokines such as IL-6 and IL-10.

Analysis using immunohistochemistry has shown that mature CD83+ DC are present in nearly all primary colon cancer samples and in some metastases. The patterns of infiltration vary, showing everything from scattered cells to groups of DC that often gather around blood vessels and the edges of lymphoid clusters. Reports about the maturation markers on DC infiltrating primary tumours are inconsistent. Some researchers found that about 90% of CD83+ cells were double-stained with anti-CD40 or anti-CD86 antibodies, suggesting their activation in vivo, while others noted that 64-97% of cells did not show B-7 molecules, even after being stimulated with TNF-, IL-4, and GM-CSF. The density of DC at tumour sites was increased in patients who had a higher ratio of activation markers (CD86 and CD40), indicating that mature DC might actively migrate to or become activated within the tumour microenvironment when exposed to tumour antigens.

Vaccinating patients with a DC vaccine in early clinical trials revealed that the vaccine worked for 16. 7% of participants in the phase I trial and for 23% of those in the phase II trial. The messenger RNA from the TAT protein transduction domain and calreticulin boosts the immune response to CEA and enhances the effectiveness of mRNA-stimulated human DCs. It is notable that introducing DCs with calreticulin mRNA appears to promote the activation of CD4+ T cells, while TAT protein mRNA mainly triggers CD8+ cells. Since mRNA constitutes only about 5% of total cellular RNA, amplifying mRNA in vitro has been shown to effectively generate immunogenically active mRNA that encodes CEA.

12. Management

All individuals suspected of having colorectal cancer should undergo a rectal examination, a complete blood count, and an evaluation of kidney and liver functions [1]. A colonoscopy is recommended; however, if a patient cannot have this procedure, options such as flexible sigmoidoscopy or a double-contrast barium enema may be used. CT scans of the chest, abdomen, and pelvis are important for understanding how far the disease has spread and can help determine if surgery is possible. Carcinoembryonic antigen (CEA) levels may be high and can assist in monitoring treatment effectiveness. Staging is determined by how deeply the cancer has penetrated the bowel wall, and at least twelve lymph nodes should be examined during surgery to evaluate involvement, as these aspects are closely related to prognosis.

Most patients should think about surgery to obtain and remove the tumour along with nearby lymph nodes. If there are liver metastases, they may also be removed. Eighty percent of tumours are amenable to surgical removal, commonly through a hemicolectomy. Rectal surgeries tend to be more difficult, but improvements in total mesorecta excision techniques have enhanced survival rates.

Radiotherapy has minimal effect on survival rates for rectal cancer; however, it can serve as a neoadjuvant treatment to reduce tumour size prior to surgery and decrease the chances of recurrence in the pelvic area. It can also help alleviate local symptoms, including pain or bleeding.

Approximately half of the patients who undergo curative surgeries are likely to experience a relapse within two years. The use of adjuvant chemotherapy involving combinations of fluorouracil, capecitabine, and oxaliplatin has resulted in better survival rates for both colon and rectal cancers.

Biological treatments targeting the epidermal growth factor receptor, such as Cetuximab, have significantly improved outcomes for patients with advanced or metastatic cancers without the KRAS mutation, which affects around 30-40% of cases.

13. Conclusion

Colon cancer presents a significant health issue in Western countries. It ranks as the third most common cause of cancer deaths among both men and women. Due to varying medical histories, colon cancer is treated and documented separately from rectal cancer. Colon cancer occurs more than 2. 5 times as often as rectal cancer. Rectal cancer is identified as cancer that develops 12-15 cm away from the anal edge.

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