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A Holistic Review of Colon Cancer: Causes, Diagnosis, and Current Therapies

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Abstract: Both men and women can develop colorectal cancer, which is the third most common and deadliest disease in the UK. Compared to colon cancer, the third most common cancer worldwide, lung cancer has a higher mortality rate. Based on incidence rates, colorectal cancer ranks as the third most common cancer in men and the second in women. Individuals with a family history of colorectal cancer are significantly more likely to develop the disease. As the colonic epithelium progresses from normal to dysplasia and eventually to cancer, genetic changes occur. Possible signs of colon cancer include rectal haemorrhage, an abdominal mass, abdominal pain, changes in bowel habits, unexplained weight loss, and iron-deficiency anaemia. For stage 0 cancer, a procedure called a colonoscopy is used to remove cancerous cells. Localised therapy is the primary treatment option if the cancer is small, early-stage, and has not spread to other organs. To fully remove the affected area, a surgical operation such as hemicolectomy, partial colectomy, or segmental resection may be performed, which can involve a colonoscopy or excising the section of colon containing the cancer.

Keywords: *Vascular Endothelial Growth Factor, Colorectal Cancer, Inflammatory Bowel Disease, Colonoscopy, and Bevacizumab.*

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I. INTRODUCTION

Both men and women, colorectal cancer is the third most common and deadliest kind of cancer in the US. Only lung cancer has a higher death rate than colon cancer, which is the third most prevalent disease globally. The frequency of new cases has decreased as a result of the widespread use of colonoscopy screening in Western nations. The disease is becoming more common in younger people. The unchecked growth of glandular epithelial cells in the colon causes colorectal cancer (CRC). The colon or rectum is the specific organ affected by this condition. Colorectal cancer falls into three primary categories: hereditary, sporadic, and colitis-associated. The number of colorectal cancer cases worldwide is increasing every day. Both environmental and genetic factors influence colorectal cancer susceptibility. Colorectal cancer is more likely to develop in older persons with Crohn's disease and ulcerative colitis. Numerous studies have demonstrated that dietary and lifestyle factors, genetic predisposition, and chronic inflammation are risk factors for colorectal cancer. Most incidences of colon cancer are sporadic, meaning they happen by accident. Genetic changes inherited from Lynch syndrome account for around 5% of colon cancers, primarily associated with hereditary nonpolyposis colon cancer (HNPCC) and familial adenomatous polyposis (FAP). The adenoma-carcinoma sequence, which is the progressive evolution of normal colon epithelium to invasive cancer over several years, is frequently characterised by the accumulation of genetic alterations, the development of adenomas, and subsequent carcinogenesis. However, certain tumours may take other paths, such as those linked to DNA mismatch repair (MMR) and the BRAF gene. Screening for colon cancer is recommended and may be done in a variety of ways. Policies regarding the start and ongoing implementation of screening processes vary throughout organisations.[1] A tissue sample, usually obtained during a colonoscopy, is commonly required for the diagnosis of colon cancer. A baseline carcinoembryonic antigen (CEA) test and a thorough colonoscopy are crucial for newly detected colon tumours. Additionally, these cancers should be checked for common genetic changes. For most patients with an aggressive cancer diagnosis, thoracic and abdominopelvic CT scans are essential. The main treatment for locally detected and early-stage colon cancer is surgical resection. The main factor influencing the prognosis is the stage of the condition that causes illness. Depending on the stage of the ailment, further treatment may be needed, such as immunotherapy, chemotherapy, or, in rare instances, radiation therapy. To detect local recurrence and metastatic illness, which may be responsive to multimodal therapy, post-treatment monitoring is crucial. Enhancing survival and quality of life for individuals with illnesses that are incurable or broadly metastatic is the goal of palliative systemic therapy. Screening is extremely important in this context since

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colorectal cancer is a common illness that often develops from adenoma to carcinoma. Although the precise time it takes for an early-stage adenoma to progress to a diagnosed colorectal cancer is unknown, recent studies suggest that it typically takes at least ten years. This offers sufficient opportunity for screening-based early detection and subsequent medical intervention. One preventative measure against colorectal cancer is the removal of colorectal adenomas.[2] The likelihood of dying from colorectal cancer decreases with an earlier diagnosis. Treatment outcomes are improved by interventions that target the adenoma-carcinoma pathway. Other successful approaches include identifying and monitoring high-risk groups, such as those with inflammatory bowel disease, families with hereditary colorectal cancer syndrome, those with a family history of a genetic susceptibility to colorectal cancer even in the absence of detectable genetic markers, and those with phenotypic characteristics suggestive of increased risk. The main metabolic pathways impacted by genetic variation were identified using the metabolome. The two main methods for screening for colorectal cancer are faecal occult blood tests (FOBTs) and lower endoscopy.[3]

II. EPIDEMIOLOGY

Based on incidence rates, colorectal cancer is the third most common cancer in males and the second most common in women. More than 1.9 million new cases were recorded in 2020. About 935,000 people die from colorectal cancer each year, making it the second most common cause of cancer-related deaths. It is one of the cancers with an increasing incidence and makes up 11% of all cancer diagnoses worldwide. The incidence and mortality rates of colorectal cancer vary significantly among countries globally, according to GLOBOCAN 2020 data. The incidence and death rates of colorectal cancer are rising significantly in nations with moderate to high human development indices (HDI) that are adopting a "Western" lifestyle. In industrialised nations, colon cancer is more common. Red meat eating, obesity, a sedentary lifestyle, alcohol drinking, and tobacco use are the main drivers of the growth in colorectal cancer. The majority of colorectal cancer cases occur in affluent nations with Westernised lifestyles.[4] Determinants that have a substantial impact on life expectancy, such as socioeconomic factors like education, income, and government health spending, as well as health-related behaviours like smoking, obesity, and physical activity, have a considerable impact on the onset of cancer. Life expectancy levels must be taken into consideration while developing cancer preventive and treatment plans. Strong facts about the incidence of colon cancer were shown in a review of 36 Asian and European nations. Ten of these countries had an increase in colon cancer cases between 2007 and 2016. The country with the most increase was India, followed by Poland. Depending on the statistics available, the growth period varied between 2006 and 2015 or between 2005 and 2014.[5] These 10 nations have ratings ranging from moderate to high on the Human Development Index. The United States and Israel had the largest decrease in the incidence of colon cancer among the six countries with the highest Human Development Index ratings. The proportion of persons 50 and older declined in seven North American countries. Eight countries, including the UK and India, have seen a rise in the prevalence of colon cancer in persons under 50. People under 50 were more common than people over 50 in Germany, Australia, the United States, Sweden, Canada, and the United Kingdom. Among persons over 50, the incidence rate either stayed constant or showed a downward trend. In Italy, the incidence of colorectal cancer decreased only among those under 50. The incidence of colon cancer in women increased in 12 of the 36 countries (all from Asia and Europe), whereas it decreased in 7. The two countries with the biggest increases were Slovenia and India. Several studies have shown that the stage of colorectal cancer diagnosis affects the prognosis, with worse survival rates for those with advanced stages of the disease. Colorectal cancers found early have a 90% five-year survival rate, but those detected later have a 13% five-year survival rate. For women and men, the risk of dying from colon cancer between the ages of 0 and 74 is 0.45% and 0.65%, respectively. For both sexes, the age-standardised global colorectal cancer death rate is 8.9 per 100,000 cases.[6]

It is projected that colorectal cancer will cause 1.1 million deaths and around 2.2 million new diagnoses globally by 2030, representing a significant 60% rise over previous years. Significant growth will result from the process of economic development, which is marked by the transition of low-to-medium Human Development Index countries and the changing demographics of wealthy countries. A sedentary lifestyle, abnormal bone density, consumption of highly processed foods, alcohol, and red meat, as well as a general rise in life expectancy, are some of the environmental factors that have been linked to this increase in several studies. Developing plans for the disease's prevention and treatment requires a current examination of the patterns and temporal trends of colorectal cancer from a global perspective. The most recent scientific knowledge must be taken into account in this examination.[7]

III. RISK FACTORS OF COLON CANCER

- *Family History and Genetics*

People who have a family history of colorectal cancer are much more likely to get the disease. Both behavioural and genetic factors have an impact on this phenomenon. In predicting the future occurrence of colorectal cancer, important variables to take into account are: (i) the distance between at-risk individuals and their family members; (ii) the age at which first-degree relatives were diagnosed with colorectal cancer; (iii) the number of family members with the disease; (iv) the presence of other neoplasms, such as endometrial, ovarian, and urinary tract cancers, in the family; and (v) the individual's cancer history. According to earlier studies, those who had one first-degree relative (parents, siblings, or children) with colorectal cancer were twice as likely to have the disease as people without a family history. A family member has a high chance of developing colorectal cancer themselves if they were diagnosed with the disease before the age of 60. The greater the number of affected relatives, including both near relatives and distant cousins, the greater the chance of contracting the disease. [8]

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➢ *Inflammatory Bowel Disease (Crohn's Disease; Ulcerative Colitis)*

Inflammatory bowel disease (IBD) is the third most important risk factor for the development of colorectal cancer, behind FAP and HNPCC. IBD is a group of long-term, incurable conditions that affect the gastrointestinal tract's immune system and cause uncontrollable inflammation. Crohn's disease and ulcerative colitis are the two main types of IBD. Although the exact cause of IBD is unknown, it is believed to be the consequence of a combination of environmental, genetic, and immunological factors. Compared to healthy people, those who have inflammatory bowel disease have a much higher chance of developing colorectal cancer, around 2.6 to 6 times higher. This increased risk results from chronic inflammation, which speeds up cancer growth and development. The chance of developing colorectal cancer is increased by the duration, scope, and severity of IBD.[9]

➢ *Diabetes Mellitus*

Persistent hyperglycemia is a metabolic disorder caused by abnormalities in insulin synthesis and/or activity that characterises diabetes mellitus. There are an estimated 460 million people with diabetes worldwide, and the number is expected to increase. Diabetes is a substantial risk factor for several gastrointestinal cancers, including colorectal cancer, according to epidemiological research. People with type 2 diabetes are about twice as likely to develop colorectal cancer as people without the disease. It is believed that elevated insulin levels and an inflammatory state are related to the development of colorectal cancer. By increasing insulin-like growth factor 1 (IGF-1) levels and encouraging colonic cell proliferation, hyperinsulinemia may hasten the development of colorectal cancer. IGF-1 is a growth factor that inhibits apoptosis and encourages cellular division. Furthermore, in the context of chronic inflammation associated with diabetes, proinflammatory cytokines, such as interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), promote the development of distant metastases, the transformation of normal cells into malignant cells, tumour proliferation and dissemination, and invasion of neighbouring tissues.[10]

➢ *Colon Polyps*

A colon polyp is categorised as a precancerous neoplastic lesion and is an abnormal tissue growth that emerges from the colon's mucosal layer. These polyps may be classified histologically into two main categories: neoplastic and non-neoplastic. The latter group includes inflammatory, hyperplastic, and hamartomatous polyps. The potential for adenomatous polyps to become malignant tumours makes them extremely significant. Adenomatous polyps are thought to be the cause of more than 95% of instances of colorectal cancer. Although adenomas are thought to be the cause of almost all colorectal malignancies, only around 5% of polyps are thought to develop into cancer. Over five to fifteen years, adenomatous polyps may develop into invasive adenocarcinomas. The size of the polyp, the degree of dysplasia, and the age of the person all increase the chance that this may turn into cancer. Indicators of a poor prognosis include advancing age, severe dysplasia, and polyps larger than 1-2 cm. Since 40% of people over 50 have adenomatous polyps, it is important to detect and remove these lesions as soon as possible to prevent the development of cancer.[11]

➢ *Cholecystectomy*

It is yet unclear how cholecystectomy, the surgical removal of the gallbladder, and the subsequent development of colorectal cancer are related. While some studies showed no increased risk, others suggested that having a cholecystectomy increased the chance of developing colorectal cancer. Changes in bile acid secretion and content are thought to be associated with a higher risk of colorectal cancer after cholecystectomy. Under typical biological circumstances, bile acids are regularly produced in response to food consumption. The continuous flow of bile to the stomach in the absence of a gallbladder increases the bacterial conversion of bile acids into secondary bile acids. By generating reactive oxygen and nitrogen species, weakening the cell membrane, damaging DNA, and causing apoptosis in colonic mucosal cells, secondary bile acids can increase the risk of colon carcinomas. [12]

➢ *Developmental Stages of CRC*

The American Joint Committee on Cancer subsequently adopted the three-part colon cancer staging system created by the Union for International Cancer Control. These stages are indicated by the TNM system. An illustration of a primary tumour shows its size, growing area, and extent of infiltration into neighbouring layers. An alphanumeric code, such as M2, which provides further information on the stage of the malignancy, is sometimes included in the classification. The amount indicated indicates the degree of cancer invasion in the organs or walls. The initial stage, known as stage 0, indicates that the mucosa, or innermost layer of the colon, has not grown.[13] The tumour invades the submucosa in stage 1 but does not spread to the lymph nodes. The following stage, known as stage 2, is characterised by tumour growth that does not affect lymph nodes. There are three subcategories of the stage: stage 2A, where the tumour penetrates the colon's outer layers but does not spread to the visceral peritoneum; stage 2B, where the tumour has spread to the visceral peritoneum; and stage 2C, where the cancer cells have spread to nearby organs or structures. There are three components to Stage 3. Stage 3B is divided into two groups according to the number of lymph nodes involved: either three or four or more lymph nodes. The tumour spreads to neighbouring lymph nodes in stage 3A. When a cancer spreads beyond the layers of muscle and is found in four or more adjacent lymph nodes within the afflicted region, it is said to be at stage 3C. There are three additional levels in level 4, which is the last level. Stage 4A indicates that the cancer has migrated to a single, remote site, such as the liver, lungs, or lymph nodes. While stage 4C, the last stage, indicates that the cancer has spread across the peritoneum, stage 4B indicates that the disease has spread to two or more distant locations. Colorectal cancer is ranked from 1 to 4 according to how closely malignant and healthy cells resemble each other when observed under a microscope. Higher grades show faster growth rates, whereas lower grades more closely match normal cells. Furthermore, strong academic success is frequently a sign of rapid expansion.[14]

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➢ *Pathophysiology*

As the colonic epithelium progresses from normal to dysplasia and finally to cancer, genetic changes take place. Three main genetic mechanisms can contribute to the development of colon cancer: chromosomal instability (CIN), CpG island methylator phenotype (CIMP), and mismatch repair (MMR). Despite their significant overlap, these routes do not conflict with one another. A mutation gain that upsets the balance between tumour suppressors and oncogenes characterises the CIN pathway. This process was initially discovered to be the conventional adenoma-carcinoma sequence. Mutations were shown to be significantly prevalent in TP53, KRAS, and APC. APC mutations, which frequently encourage the formation of cancer, are implicated in around 60% of colon malignancies. Normal APC regulates the signalling pathway by interacting with β-catenin. APC mutations cause anomalies in this system by compromising the regulation of cell differentiation, apoptosis, and proliferation. BRAF and KRAS mutations are also found in CIN tumours, albeit these changes are not exclusive to this pathway.[15]

Mutations in the MSH1, MSH2, MSH6, or PMS2 genes cause replication mistakes to accumulate in DNA, which is what defines MMR. Microsatellite instability caused by defects in mismatch repair (MMR) may be seen in tissue samples. MMR gene germline mutations are unmistakably indicative of hereditary nonpolyposis colorectal cancer (HNPCC). However, most tumours that exhibit considerable microsatellite instability (MSI) are random. The histology of lesions with high microsatellite instability is often mucinous or poorly differentiated. Their response to 5-fluorouracil (5-FU) treatment is rather low, and they are primarily located on the right side. These tumours respond better to immunotherapy than do microsatellite-stable cancers. The CIMP pathway, which is marked by hypermethylation of CpG islands, is connected to about 15% of colorectal tumours. Unlike normal adenomas, CIMP-related cancers are often associated with BRAF and KRAS mutations and mostly occur in serrated polyps. The prognosis of cancers with microsatellite instability is significantly better than that of tumours with hypermethylation but no MSI.

Through the study and classification of several gene-expression-based categorisation techniques, researchers have discovered four main molecular categories. These subtypes, which make up 14% of cases, are known as CMS1 (MSI-immune) and are characterised by dMMR, a hypermutated load, microsatellite instability, and strong immune system activation. The malignancy exhibits the CMS2 subtype, which is distinguished by substantial WNT and MYC signalling increase, considerable chromosomal instability, and 37% canonical pathway activation. In addition to CpG hypermethylation, the patient has a KRAS mutation, namely CMS4 (mesenchymal, 23%). Angiogenesis, stromal invasion, and transforming growth factor-β are all markedly activated. Additionally, the patient has CMS3 (metabolic, 13%), which is characterised by significant metabolic abnormalities and epithelial involvement.[16]

➢ *Clinical Manifestation*

When certain signs of lower gastrointestinal diseases are observed, suspicions of colorectal cancer may arise. Guidelines for healthcare professionals to identify individuals with a high risk of colorectal cancer have been created by the National Institute for Health and Care Excellence. Signs of possible colorectal cancer include iron-deficiency anaemia, abdominal mass, stomach pain, changes in bowel habits, unexplained weight loss, and rectal haemorrhage. These conditions call for further testing. Certain symptoms, such as deep vein thrombosis and unexplained anorexia, must be recognised since they are not exclusive to one location. Taking these symptoms into account, screening for other symptoms, indications, or results may help determine which type of cancer is most likely to require more research. This might lead to an immediate examination or a referral for a suspected cancer route.[15,16]

Several investigations have evaluated the significance of symptoms in detecting colorectal cancer. For colorectal cancer, their distinct presentations have limited diagnostic value (specificity and sensitivity). Moreover, both positive and negative likelihood ratios (PLR and NLR) demonstrate that the presence or lack of symptoms has no bearing on the probability of identifying colorectal cancer. However, according to several guidelines, anyone exhibiting gastrointestinal symptoms and indicators suggestive of likely colorectal cancer should have a colonoscopy. However, some research suggests that the presence of specific symptoms at the same time, such as a palpable abdominal mass during examination and the presence of dark red rectal bleeding or rectal bleeding combined with weight loss and changes in bowel habits, may increase the accuracy of the colorectal cancer diagnosis.[17]

Patients whose disease was detected early and who were diagnosed before symptoms appeared or at the first indicators had a significantly better prognosis for colorectal cancer treatment. Therefore, if the patient has any alarming symptoms that might indicate colorectal cancer, they should schedule a visit with a doctor right away and have colon diagnostic tests done.[16,17]

➢ *Diagnostic Methods*

The current screening guidelines for moderate-risk persons aged 50 and older include the high-sensitivity faecal occult blood test (TSOH), the Guaiac or immunologic test (TSOHi) once a year, sigmoidoscopy every five years with TSOH every three years, and colonoscopy every ten years. The majority of diagnostic techniques rely on the patient's life expectancy. However, when combined with a diet high in fruits, vegetables, and red meat that contains peroxidase, some techniques, such as faecal occult blood tests, may provide false-positive results. In contrast, those without a bleeding disorder could consume vitamin C. However, advances in technology have made it possible to develop techniques such as PCR, which allow for the focused detection of biological genetic markers. Finding changes in specific genes connected to colorectal cancer is the goal. These days, colon and colorectal cancer may be identified using the homeobox duodenal pancreatic transcription factor

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(PDX-1). It plays a major role in the growth and spread of the pancreas, and its levels are higher in pancreatic cancer, breast, colon, prostate, kidney, and metastases than in original tumours or healthy colon, the latter two being absent. It is therefore considered a colorectal cancer biomarker.[14,18]

Such as sigmoidoscopy, are constantly developing and can now identify 40-65% of colorectal malignancies and 65-75% of adenomatous polyps. However, around 50% of proximal colon cancer and advanced adenomas (>1 cm) are missed by this method. One reliable and important prognostic indicator of unfavourable outcomes is hypoalbuminemia. Despite being the only procedure that undergoes thorough inspection, the diagnostic and therapeutic treatment of the colon involves more expenses, dangers, and patient discomfort when compared to other examinations. Genetic factors and overly synthesised proteins are currently thought to be indicators of a bad prognosis for patients. Tumour size, histological tumour grade, clinical TNM stage, and distant metastases are all correlated with elevated levels of mRNA and paxillin in colorectal cancer. Studies have shown that those with higher levels of paxillin had fewer positive outcomes than those with lower levels. In addition, a lot of people had hypoalbuminemia before taking the medication.[18, 19]

- *Treatment*

When treating stage 0 cancer, a medical procedure called a colonoscopy is used to remove cancerous cells. Stage I, II, and III cancers are managed surgically by excising the affected portion of the colon with at least a 5-cm margin, removing the nearby lymph nodes (at least 12), and biopsying any suspect lymph nodes outside the excised area. According to research, the laparoscopic approach to treating colorectal cancer is just as safe as traditional open surgery. Adjuvant chemotherapy is given to patients with stage III cancer for six to eight months after surgery. The main aims of this treatment are to alleviate symptoms and extend the survival time of patients with stage IV cancer. Patients' lives were prolonged when irinotecan was combined with leucovorin and fluorouracil, which was recognised as the new primary treatment approach for this condition. The survival rates of patients treated for colon cancer who received 5-fluorouracil, or approached the therapeutic threshold, were higher. It remains the cytostatic drug most frequently used. Bevacizumab (Avastin), an antibody variant approved by the US Food and Drug Administration (FDA), is currently under investigation. Vascular endothelial growth factor (VEGF), the primary regulator of angiogenesis produced by both healthy and cancerous cells, is reduced in concentration as a result of this modification. Human monoclonal antibodies targeting VEGF have been shown in preclinical research to effectively inhibit the growth of human cancer xenografts.[20]

Its primary antiangiogenic property, bevacizumab, may enhance direct antiangiogenic effects and improve the results of chemotherapy by modifying tumour vasculature and reducing interstitial pressure within tumours. By inhibiting tumour growth, the addition of bevacizumab to IFL improved overall survival. Instead of solely relying on cytoreduction, the IFL regimen with bevacizumab increased progression-free survival from a median of 6.2 months to 10.6 months. The average response duration rose from 7.1 months to 10.4 months, while the total response rate increased from 34.8% to 44.8%. However, regardless of whether bevacizumab is administered or not, the use of IFL is associated with a higher risk of thrombosis, haemorrhage, proteinuria, and hypertension. The standard treatment for stage III rectal cancer involves a combination of chemotherapy and radiation therapy when tumour invasion or adhesion to the retroperitoneal space or other organs prevents surgical removal. Patients with stage IV colon cancer that has metastasised to the liver should carefully evaluate all available therapy options tailored for the affected organs. These may include ablation, radiation, chemotherapy, surgery, cryotherapy, or other suitable treatments. Active KRAS oncogenes in metastatic patients confer resistance to therapy with anti-EGFR antibodies. Therefore, it is crucial to determine whether a patient's KRAS gene is mutated before initiating anti-EGFR therapy. It is estimated that 30-50% of individuals with colorectal cancer have a KRAS mutation, suggesting that anti-EGFR drugs could benefit over 50% of patients. However, only 40-60% of these patients are expected to respond favourably. Additionally, compared to patients with wild-type KRAS, who do not exhibit carcinogenic activity, those with mutant KRAS have a lower likelihood of survival.[21]

- *Colon Surgery*

Localised therapy is the patient's main choice if the cancer is small, still in its early stages, and has not spread to other organs. For colon cancer in stages 0 and I, surgery is the primary treatment used. To completely remove the affected area, a medical operation called a hemicolectomy, partial colectomy, or segmental resection may involve performing a colonoscopy or excising the section of the colon that contains cancer cells. Sometimes, especially after a complete colectomy, the surgeon will decide to reattach the healthy portion of the colon to the rectum. A colostomy, a technique that involves making an incision in the abdominal wall, is required to divert waste. On the other hand, high-resolution, three-dimensional images of the surgical field are made possible by advanced robotic procedures and enhanced visualisation technologies, which enable surgeons to more clearly identify even the smallest features. Increased visual acuity makes it easier to spot important anatomical features, allows precise incisions, and reduces damage to healthy tissues.[22]

IV. CONCLUSION

Colorectal cancer is a serious health concern that is becoming more commonplace worldwide. To improve outcomes and reduce mortality from this disorder, screening, early detection, and targeted treatments are crucial. Early diagnosis through screening and preventative measures can significantly impact treatment outcomes and reduce mortality. Colon polyps, diabetes mellitus, inflammatory bowel disease, cholecystectomy, and genetic susceptibility and family history are risk factors for colon cancer. Understanding these risk factors and their implications can

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help predict and prevent colorectal cancer. While surgical intervention is necessary for stages I, II, and III of cancer, colonoscopy is a crucial method for controlling stage 0. Irinotecan is the main therapeutic medication used in post-surgery chemotherapy, which aims to reduce symptoms and increase survival. Although there are certain negative effects, bevacizumab has shown promise in reducing tumour growth and improving overall survival rates. Individualised treatment plans should be considered for patients with metastatic colon cancer, and before starting anti-EGFR medication, it is essential to determine whether the KRAS gene is mutated. In the early stages of colon cancer, surgical techniques including hemicolectomy and robotic surgeries are effective local therapies.

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