DISSERTATION ON

"A PROSPECTIVE CASE STUDY OF COLONIC MALIGNANCIES"

submitted in partial fulfillment

of requirements of

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BRANCH - I

GENERAL SURGERY

GOVERNMENT KILPAUK MEDICAL COLLEGE
THE TAMILNADU
DR.MGR MEDICAL UNIVERSITY
CHENNAI

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CERTIFICATE

This is to certify that this dissertation entitled **"A PROSPECTIVE CASE STUDY OF COLONIC MALIGNANCIES"** is the bonafide record work done by **Dr.KARTHIK.A** submitted as partial fulfillment for the requirements of **M**.S. Degree Examinations Branch I, General Surgery, April 2014.

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DECLARATION

I **Dr.KARTHIK.A,** solemnly declare that the dissertation submitted on the topic **"A PROSPECTIVE CASE STUDY OF COLONIC MALIGNANCIES"** is a bonafide work done by me from June 2011 to December 2013, towards partial fulfillment of the requirements of M.S Degree examinations, General Surgery, April 2014.

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CONTENTS

LIST OF FIGURES

LIST OF TABLES

INSTITUTIONAL ETHICAL COMMITTEE
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CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Prospective case study of colonic malignancies"– For Research Work submitted by Dr.A.Karthik, MS (GS), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

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ABSTRACT

It is the third most common cancer diagnosis in both men (behind prostate &lung)& women (behind breast & lung).

Second leading cause for death after lung cancer

Mean age at diagnosis is 5th decade

- Sporadic colon carcinoma accounts for 70%
- Genetic colon carcinoma accounts for 23%

A more thorough understanding of the molecular basis for this disease, coupled with the development of new therapeutic approaches, has dramatically altered the way in which patients are managed. New strategies for screening and for the detection of recurrent disease have improved the survival of the patients. This study demonstrates the importance of early diagnosis and the improved prognosis and survival in patients with prompt surgical management.

AIM OF THE STUDY:

- To analyze the clinical presentation, prognosis and outcome of prompt surgical management in colonic malignancies with respect to disease free survival rate, tumor recurrence and mortality.
- To compare the incidence of right and left sided colonic malignancies and to document their clinical and pathological differences.
- To elucidate the importance of CEA in determining prognosis and in follow-up.

METHODS:

This is a prospective case study of colonic malignancies with exclusion of genetic and familial cancers, patients with concomitant malignancies and severe cardiac ailments. This study was conducted with patients admitted to Government Royapettah hospital/Kilpauk Medical College, Chennai, located in the southern part of India. The study was conducted prospectively from the day of admission in 50 patients of colon cancers in various surgical units, between June 2011 and November 2013. All cases studied were subjected to detailed clinical examination with emphasis on the family history of colon cancers and duration of the presenting

complaints and the accurate chronology of symptoms complex associated with each of the colonic malignancies. Colonoscopy and CT findings were also documented. The surgeries performed for various colon cancers were also recorded with postoperative complications and its influence on the duration of stay at hospital. A detailed workup of comparison between the postoperative and preoperative CEA levels was done.

RESULT ANALYSIS AND CONCLUSION:

Of the 50 cases 36 patients (72%) presented with colon cancers in their fifth to sixth decade of life. The colonic malignancies were found to be more common in males (66%) when compared to females (34%). The right colon cancers presented with anemia (n=26), mass abdomen (n=22) and loss of weight (n=28). The left colon cancers presented predominantly with altered bowel symptoms either constipation or loose stools (n=25). 70% of the cases (n=35) were right-sided tumors (located in caecum, ascending colon, hepatic flexure or proximal transverse colon) whereas the left sided tumors that of splenic flexure, descending colon and sigmoid colon were seen only in 30% of cases (n=15). Of the 14 cases that showed evidence of lymph node in computerized tomography 9 were found to be poorly differentiated adenocarcinoma, 4 were moderately differentiated tumors and only one was well differentiated. It was found that all well differentiated tumors had CEA levels ranging

from 15 to 25ng/ml, in the range of 5 to 15ng/ml for moderately differentiated tumors and the poorly differentiated tumors had CEA levels in the range less than 5ng/ml. Of the 50 cases of carcinoma colon, 3 cases presented as emergencies (2 cases of obstruction and 1 was colo-vesical fistula). The common complications were paralytic ileus (n=20), wound infection (n=19), respiratory infection (n=17), sepsis (n=1) and anastomotic leak (n=1) in that order of frequency. Most of the cases (n=34) studied were of stage II disease and 10 cases were of stage III disease. Thus 72% of cases were of stage II and stage I. The average duration of postoperative stay was 11 days. However average stay in patients with postoperative wound infection was 15 days. The preoperative CEA levels were on an average above 5.5ng/ml in 84%(n=42) of cases. However the values returned to value below the baseline value of 5.5ng/ml in 74%(n=37) postoperatively signifying a tumor free state. Of the 50 cases operated (47 elective surgeries and 3 emergency surgeries), 3 cases of stage III/IV disease reported with recurrence in one year and there was 1 case of death due to septicemia in the case operated as an emergency.

Based on the statistical analysis of the study it was inferred that the incidence of colon cancers was common in 5^{th} to 6^{th} decade of life and right colon cancers where commoner than left colon cancers. Colon

cancers are more common in males than females. Results with respect to the symptom complex and duration of presenting complaints were confirming to the accepted knowledge about the right and left colon cancers. The lesions were predominantly ulcero-proliferative in right colon cancers but were predominantly circumferential in left colon cancers. It is found that there is a strong correlation between the lymph nodal involvement and tumor differentiation, poorly differentiated tumor has more propensities to cause lymph node spread early hence worse prognosis. The diffentiation of tumor also has inverse correlation with respect to CEA levels; higher the levels of CEA well differentiated will be the tumor grade. The common postoperative complications were paralytic ileus, wound infection and respiratory infection and the duration of postoperative stay was not related to the tumor grade or its stage but the presence or absence of wound infection. Postoperative CEA levels measured after 2 weeks is useful in assessing the surgical outcome as a successful surgery brings the value to baseline. Recurrent tumors had elevated CEA well above their immediate postoperative levels, thus implying the importance of postoperative follow-up of all cases operated for colon cancers. Lastly the mortality associated with tumor complicating emergency surgeries is high when compared to electively planned surgeries.

Early diagnosis of colon cancers can be curative as more than 90% of stage I/II tumors have longer disease free survival rate than the stage III/IV disease.

INTRODUCTION

Colorectal lesions may be classified as either benign, potentially malignant, or malignant based on their pathologic features. The overwhelming majorities of colorectal tumors are of epithelial origin and arise from the mucosal surface, where they become visible descriptively as a polyp. Benign polyps include non-neoplastic polyps (e.g., hyperplastic, hamartomatous, or inflammatory polyps); the potentially malignant group consists of adenomatous polyps. Once dysplastic cells in a polyp cross the boundaries of the mucosa (basement membrane) and start to invade the submucosa, a true cancer (carcinoma) with the potential to metastasize is established. Tumors of nonepithelial or mesenchymal origin are comparably rare and include, among others, lymphoma, carcinoid and sarcoma. Significant advances have been made in the study of colorectal cancer during last few decades. A more thorough understanding of the molecular basis for this disease, coupled with the development of new therapeutic approaches, has dramatically altered the way in which patients are managed. New strategies for screening and for the detection of recurrent disease have improved the survival of the patients. This study demonstrates the importance of early diagnosis and the improved prognosis and survival in patients with prompt surgical management.

AIMS AND OBJECTIVES

- To analyze the clinical presentation, prognosis and outcome of prompt surgical management in colonic malignancies with respect to disease free survival rate, tumor recurrence and mortality.

- To compare the incidence of right and left sided colonic malignancies and to document their clinical and pathological differences.

- To elucidate the importance of CEA in determining prognosis and in follow-up.

REVIEW OF LITERATURE

RISK FACTORS

There are various genetic and environmental risk factors associated with the colorectal cancers event though the specific cause of the cancer in sporadic cases is unknown. The majority of cases are however sporadic colon cancers that typically arise within a polyp, thus nutritional and environmental factors may play a key role in the adenoma to carcinoma progression.

Incidence of colonic malignancies is high in developed world than developing countries and there is a sharp rise in incidence after fifth decade. Obesity, sedentary life style, animal fat and low fiber diets are some of the risk factors associated with the tumourigenesis. More recent prospective trials, however, have questioned the benefit of dietary fiber supplementation in that they were at best inconclusive and did not reduce the incidence of colorectal cancer. Omega-3 fatty acids found in fish products have a proven beneficial effect. It therefore could be concluded that the total amount of fats or fibers is of lesser importance than their quality and origin. The protective effect of vegetables and fruits may come not only from their fiber content but also from the content of antioxidative and antiproliferative agents such as isothiocyanates in cruciferous vegetables which may enhance the expression of carcinogen-metabolizing enzymes and induce apoptosis in neoplastic cells.

The mechanisms by which calcium supplements are thought to reduce the risk of colon cancer are twofold. Calcium can bind with the bile products and thus protecting the colonic mucosa from its irritant effects, and second, it can interfere directly with the mucosal cells and decrease their proliferative potential on a cellular level.

Several vitamins were found to have a cancer-protective effect. Vitamins A, C, and E have been shown to have antioxidant activity.

Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) may interfere with the development of colorectal neoplasms by blocking the cyclooxygenase- dependent prostaglandin pathway. The targets are the constitutive COX-1, as well as the cytokine-inducible COX-2, which has been found at increased expression levels in both polyps and cancers. Several trials therefore have studied these agents (e.g., aspirin and sulindac) for the chemoprevention of colorectal cancer both in sporadic polyps and cancers and in familial adenomatous polyposis (FAP).

Evidence that bile acids may act as co-carcinogens or tumor promotors comes from both experimental and epidemiological studies. Bile acids can induce hyperproliferation of the intestinal mucosa via a number of intracellular mechanisms. Cholecystectomy, which alters the enterohepatic cycle of bile acids, has been implicated in cancers of proximal colon.

The risk of developing colon cancer is increased in smokers, alcoholics and in those with family history of colonic malignancies. A similar, even though proportionally lesser risk is observed for family members of individuals with colonic adenomatous polyps.

Familial adenomatous polyposis and HNPCC have 100% and 80% risk of developing colonic malignancies. Ulcerative colitis has 10 to 20% risk over a period of 20 years. Surgical procedures like uretero-sigmoidostomy have increased risk adjacent to the site of anastomosis.

PREVENTION AND SCREENING

Since symptoms are not reliable for early detection of colorectal cancer, risk-adjusted screening programs for asymptomatic individuals are important. Effective screening has to be based on an understanding of the adenoma-carcinoma sequence, which may take up to 5 to 10 years from the first molecular change to a clinically manifest cancer, and should reflect an individual's genetic and disease- or age-dependent risk for the development of colorectal cancer. Any prevention program has to be sensitive but also practical and cost-effective in order to achieve a broad screening of the population at risk. The term screening is applicable only to asymptomatic people; if symptoms are present, it is not screening but diagnostic tests that are initiated. Common tools for screening include fecal occult blood tests, flexible sigmoidoscopies or colonoscopies, and contrast enemas.

PATHOGENESIS OF COLONIC CANCER

Carcinogenesis in the colon is a complex multistep process in which a multitude of alterations have to coincide in order to transform a normal cell into a malignant cell. Several categories of genes are involved that normally are regulated in a sophisticated network to keep a tight balance between cell growth and turnover, cell death, DNA replication, and mismatch repair. Disruption of the fine balance between oncogenes, which promote cell proliferation, and tumor suppressor genes, which inhibit excessive growth, results malignant transformation.

COLON CANCER: A GENETIC DISEASE

Mutations in DNA can occur either as a germ-line mutation or as a somatic mutation. The former may be transmitted from one to the next generation as an inherited defect. Somatic mutation occurs in a non-germ cell confirming to the Knudson's two hit hypothesis. Genesis of a cancer therefore requires several independent hits to occur in one cell. One can assume that a normal cell will be able to detect damage to its own DNA and maintain an effective repair mechanism. However, if the cell is too severely damaged, it might rather initiate the inherent suicide program called apoptosis. When a cell fails to recognize or correct DNA damage and continues to replicate, accumulation of faulty gene products within the cell eventually may lead to a proliferative response. If that replication exceeds the growth potential of the

neighboring normal cells, the mutation provides a growth advantage that will increase the state of "genetic instability" and hence lead toward a malignant cell. Despite this potential, most mutations are silent or lethal to the cell rather than beneficial in terms of providing the cell a biologic advantage.

Two types of genetic instability may occur: at the chromosome level or at the DNA level. A loss of chromosomal material, i.e., a chromosomal instability (CIN), results when the chromosomes are not divided symmetrically during mitosis such that one daughter cell receives both copies and the other cell receives none. On an electrophoretic gel, this can be visualized as a loss of one or more bands, which is described as loss of heterozygosity (LOH), and has been associated with a worse prognosis of colorectal cancer. The second form of genetic instability, at the DNA level, occurs when replication errors in repetitive short polymorphisms lead to an additional band or bands. This phenomenon is described as microsatellite instability (MSI), and it has been a characteristic feature of hereditary nonpolyposis colon cancers (HNPCCs).

During the process of cell division, DNA is duplicated, with the original DNA serving as a template for the replicated copy. DNA polymerase serves as a "proof reader" that recognizes mismatched genes, halts the DNA synthesis, removes the defective sequence, and then resynthesizes the DNA. Failure of the DNA mismatch repair system predisposes to the development of mutations within daughter cells. Consistent with the Knudson two-hit model for tumor

suppressor genes, in all cases, inactivating mutations in both alleles are present in the cancers that arise in affected individuals.

ADENOMA-CARCINOMA MODEL

After identifying several genetic alterations in colorectal specimens at various stages of their neoplastic transformation and progression, Vogelstein and colleagues in 1988 pioneered a genetic model for colorectal tumorigenesis that since has been known as the adenoma-carcinoma sequence. This multistep model described the carcinogenesis as an accumulation of genetic events, uninhibited cell growth, and proliferation and clonal development. Gene mutations and chromosomal/gene losses that were observed in sporadic colon cancer include the APC gene (adenomatous polyposis coli), MMC gene (mutated in colon cancer), ras, and p53. Mutations of the APC gene, which is involved in intercellular adhesions and communications, are found in 60% of even small adenomatous polyps, as well as in carcinomas, and therefore are believed to occur as a very early event in carcinogenesis. Mutations of K-ras, which under normal function plays a role in intracellular signal transduction and stimulated cell division, occur in larger adenomas and carcinomas and are thought to stimulate cell growth. Deletion of the tumor suppressor gene DCC may be important in the progression from a benign polyp toward a malignant condition. Mutations of the p53 gene, which are among the most frequent gene mutations in human cancers, are also common in invasive colon cancers but rare

in adenomas, suggesting that p53 mutations occur as a late event in the development of the invasive phenotype. Thus there is a wide range of gene mutations, inactivations, and deletions have been associated with colonic carcinogenesis.

NONHEREDITARY COLON CANCER

SPORADIC COLON CANCER

Sporadic colon cancer, i.e., colon cancer arising in individuals without a family history or an inherited predisposition, accounts for approximately 60% of all colorectal cancers and affects patients commonly above the age of 50.

FAMILIAL COLON CANCER

Familial colon cancer is the second most common and at the same time least understood pattern of genetic colon cancer development. An association of familial colon cancer has been found with polymorphisms, which reflect subtle genetic changes in the form of variations in the nucleotide base sequences but which do not affect protein structure.

HEREDITARY COLON CANCER

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)

It is an autosomal dominant condition. The offspring of affected individuals thus have a 50% risk of inheriting FAP. However, up to 20% of

patients with FAP are new mutations without a family history. It results because of the truncation mutation of the APC gene in the chromosome 5q21. Variants of the polyposis syndrome are classified as Gardener's syndrome (i.e., osteomas, desmoid tumors, thyroid neoplasms, retinal pigment layer hypertrophy) and Turcot's syndrome, which is associated with cerebellar medulloblastomas.

The inherited syndrome of FAP and its variants accounts for less than 1% of all colon cancers. It is characterized by greater than 100 and often several thousand of adenomatous intestinal polyps that start to develop in the late teens and early twenties and turn into cancer by age. An attenuated variant of the disease is relatively rare and is characterized by a lower number and a later onset of both the polyps and the resulting cancer. Duodenal adenomas are common in FAP with adenocarcinoma developing in the periampullary region in 3 to 10% of patients. The main cause of death following colectomy is due to malignancies of duodenum and antrum. Nonadenomatous fundic polyps of stomach usually do not have any malignant potential. Ten percent of FAP patients develop desmoid tumors either intra-abdominally or on the abdominal wall, extremities, and trunk. Histologically, desmoids are fibromatous lesions consisting of large proliferation of myofibroblasts. Desmoids are lethal in 10% and are the third most frequent cause for mortality of FAP patients, mainly owing to the intra-abdominal variants, which cause small bowel and ureteral obstructions.

Nearly one third of the FAP patients have no demonstrable APC mutation and the lesser number of polyps, late clinical presentation and diagnosis, and decreased incidence of extra colonic manifestations as compared with classic FAP patients. This variant of FAP is known as *attenuated familial adenomatous polyposis* (AFAP).

HEREDITARY NONPOLYPOSIS COLON CANCERS (HNPCC)

Hereditary nonpolyposis colon cancer (HNPCC) is grouped as *Lynch I and II syndromes,* is an inherited autosomal dominant disease that accounts for 3to 5% all colorectal cancers.

It is characterized by an early onset of colorectal cancers predominantly but not exclusively on the right side of the colon with synchronous and metachronous cancers. Despite its name, these cancers typically arise from colonic polyps, but a diffuse polyposis is not present. There is 80 to 85% chance of developing colon cancers and 40 to 50% chance of developing endometrial cancers. Furthermore HNPCC patients are at increased risk of developing extracolonic malignancies like tumors of ileum, gastric malignancies and that of genito-urinary tract. The Lynch variants describe patients with predominantly colorectal cancer at a young age (Lynch I) and those with both colorectal and extra colonic cancers (Lynch II).

There is an established link between HNPCC and the DNA MMR system. In contrast to the gatekeeper concept applicable to the *APC* gene in FAP, the mutations involve mismatch in the gene repair system thus leading to genomic instability indirectly, thus cancer development.

In order to facilitate the clinical diagnosis off HNPCC, the Amsterdam Criteria was first proposed in1990. The two MMR genes *hMSH2* and *hMLH1* were later discovered in the families coming under Amsterdam I criteria. These genes accounted for 45 to 86% of all classic HNPCC families. There also was a higher risk for *hMSH2* mutation to develop extra colonic cancers like endometrial cancer when compared with those having *hMLH1* mutation. Several other MMR genes have been identified in conjunction with HNPCC and include *hPMS1*, *hPMS2,* and *hMSH6*. The ICG-HNPCC therefore revised the criteria (Amsterdam Criteria II), which now better weigh extra colonic manifestations (e.g., endometrial, breast, duodenum, ileum and genito-urinary tract cancers) as part of the family history. In addition, the less restrictive revised Bethesda Criteria were adopted to better serve patients, which carry *hMSH2* or *hMLH1* gene mutations but otherwise do not fulfill the Amsterdam Criteria. Testing for microsatellite instability (MSI) has become a valuable diagnostic tool to identify individuals with suspected HNPCC because 85 to 90% of HNPCC tumors have MSI as opposed to only 15-20% of sporadic colon cancers.

AMSTERDAM CRITERIA II

- At least three relatives with an HNPCC associated cancer (colorectal, endometrium, small bowel, ureter, or renal pelvis). One affected relative should be a first-degree relative of the other two.
- At least two successive generations should be affected.
- At least one relative should have been diagnosed before age 50 years.
- Familial adenomatous polyposis should be excluded

BETHESDA CRITERIA (FOR IDENTIFICATION OF PATIENTS WITH COLORECTAL TUMOR WHO SHOULD UNDERGO TESTING FOR MSI)

- Cancer in families that meet Amsterdam criteria
- Two HNPCC-related cancers, including colorectal or extra colonic
- Colorectal cancer and a first-degree relative with colorectal cancer and/or HNPCC-related
- Extra colonic cancer and/or colorectal adenoma: one cancer before age 45 and adenoma before age 40
- Colorectal cancer or endometrial cancer before age 45
- Right-sided colorectal cancer with an undifferentiated pattern on histopathology before age 45
- Signet-ring cell type colorectal cancer before age 45

Approximately 4% of colonic cancers are seen in the context of rare syndromes. Among these are inherited hamartomatous polyposis syndromes. Hamartomas result from a disordered differentiation during embryonic development and are characterized morphologically by disrupted representations of normal tissue components.

PEUTZ-JEGHERS SYNDROME

Peutz-Jeghers syndrome involves large but few colonic and small bowel polyps that can manifest by gastrointestinal (GI) bleeding or obstruction and an increased risk of colorectal cancer. The polyps are distinguished by a smooth muscle band in the submucosa. Hallmark clinical features on physical examination include freckles on the hands, around the lips, in the buccal mucosa and periorbital regions. Associated characteristics include sinus, bronchial, and bladder polyps, and about 5% to 10% of patients have sex cord tumors. Patients can also develop lung and pancreatic adenocarcinomas. The gene responsible for this syndrome is called *LKB1*, a serine threonine kinase. While a majority of these patients remain relatively asymptomatic, some may present with intestinal obstruction owing to an intussuscepted polyp and others with gastrointestinal bleeding. Patients with Peutz-Jeghers syndrome have a moderately increased risk in the range of 2 to 3% to develop gastrointestinal malignancies as well as extra intestinal malignancies.

JUVENILE POLYPOSIS SYNDROME

Juvenile polyposis has overlapping clinical manifestations with Peutz-Jeghers, but the polyps tend to be confined to the colon, although cases of gastric and small bowel polyps have been described and there is an increased risk of colorectal cancer. Extra colonic manifestations are not prevalent. This is a polygenic disease, involving germline mutations in *PTEN, SMAD4*, *BMPR1*, or other genes yet to be identified. In infancy, patients may present as a case of acute gastrointestinal bleeding, intussusception and malnutrition In adulthood, patients commonly present with gastrointestinal blood loss. The polyps are located most frequently in the recto sigmoid region.

A significant risk to develop colorectal cancers is associated with this condition, which should not be confused with isolated juvenile polyps because the latter have virtually no malignant potential.

COWDEN SYNDROME

Cowden's syndrome harbors hamartomatous polyps anywhere in the GI tract, and surprisingly, there is no increased risk of colorectal cancer. However, about 10% of patients will have thyroid tumors and nearly 50% of patients have breast tumors. Germ line *PTEN* mutations have been reported.

BANNAYAN-RILEY-RUVALCABA SYNDROME

Formerly known as its subentity, the Ruvalcaba-Myhre-Smith syndrome, this rare autosomal dominant condition is due to the mutation of *PTEN* gene on chromosome 10q23 and may be considered a variant of juvenile polyposis coli. Hamartomatous polyps of the gastrointestinal tract with involvement of central nervous system, thyroid and pigmentary cutaneous lesions of gentalia characterize it. There is no proven risk of colonic malignancies or development of extra intestinal malignancies in these patients.

CRONKITE-CANADA SYNDROME

Cronkite-Canada syndrome is characterized by diffuse polyposis and ectodermal abnormalities such as alopecia, onychodystrophy, and skin hyperpigmentation. The syndrome can present with diffuse polyposis of any part of gastro intestinal tract except the esophagus. There is a certain malignant potential associated with this condition.

PATHOLOGY AND STAGING

HYPERPLASTIC POLYPS

Most of the colorectal polyps, particularly those of size less than 5 mm are hyperplastic in nature and are not considered to be a precursor lesion to cancer development. Most of the polyps were found on retrospective autopsy analysis and they were commonly found in the recto-sigmoid region.

ADENOMATOUS POLYPS

The commonest epithelial neoplasm of colon is the adenomatous polyp, which occurs as tubulous, tubulovillous, and villous variants. It may either be pedunculated (with a stalk) or sessile (broad-based). By definition, the adenomatous polyps' neoplastic nature in itself is a manifestation of colonic dysplasia. However, different degrees of dysplasia are distinguished commonly that parallel the likelihood of cancerous transformation. The degree of dysplasia can be divided into three grades based on the histopathologic differentiation and the arrangement and architecture of the epithelial cells and the acinar complexes. Common terms for still benign polyps include low-grade dysplasia, intermediate-grade dysplasia, and high-grade dysplasia or in situ adenocarcinoma. The term invasive adenocarcinoma is appropriate for neoplastic lesions that have invaded through the basal membrane and the muscularis mucosae into the submucosa. The descriptive terms then switch to

well differentiated (grade I), moderately differentiated (grade II), or poorly differentiated (grade III) adenocarcinoma as additional criteria for staging.

Flat or depressed adenomas are a subtype of colonic adenomata with a propensity for high-grade dysplasia (carcinoma in situ) in 10 to 41% of affected patients regardless of the small size of these lesions. These lesions, which are flat or slightly raised to less than 2 mm and commonly less than 1 cm in size, may be overlooked easily on colonoscopy and turn into a cancer. Chromoendoscopy techniques, have confirmed that flat adenomas represent up to 25 to 36% of all polyps found in a random cohort. It is difficult to estimate the likelihood that a small adenoma will progress to a larger and more dysplastic adenoma and eventually into a cancer. A number of biologic and molecular markers have been analyzed at predictors of a malignant potential. Polyps may not only progress but also can regress.

Invasive carcinoma is present in 5% of all adenomas, but the incidence correlates with the size and type of the adenoma. The Haggitt classification, which defines four levels within the polyp, is useful in defining the degree of invasion into a pedunculated or sessile polyp adenoma. This classification forms the basis of the management of malignant polyps. In Haggitt levels 1, 2, and 3, the risk of lymph node metastasis is less than 1%, whereas a level 4 invasion of the stalk behaves like a sessile lesion and carries a much higher risk of 12 to 25% of having lymph node metastases.

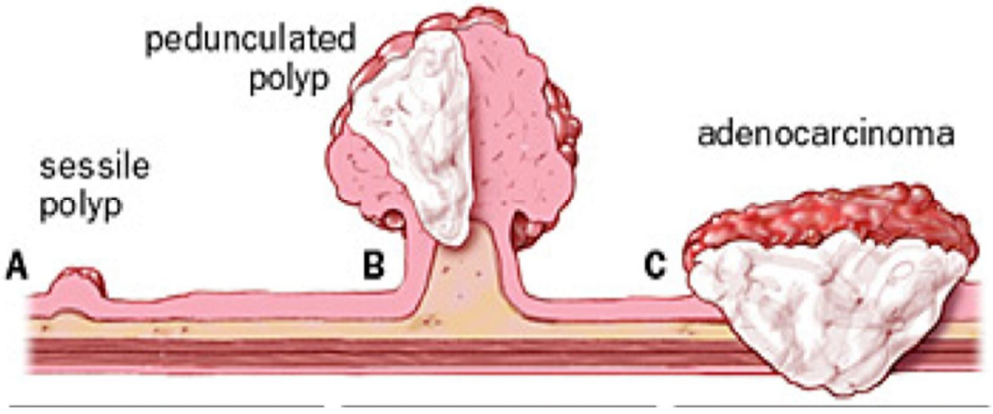


Figure 1: Showing the Appearance of Polyps

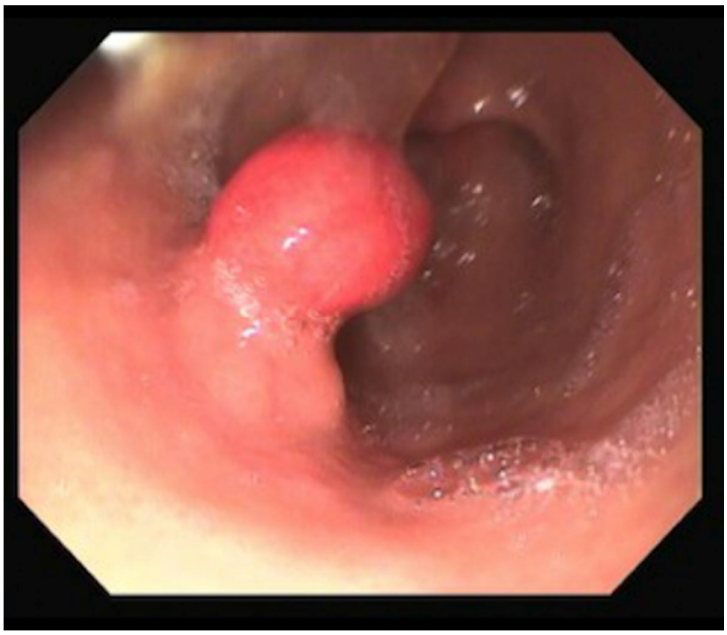


Figure 2: Showing the Colonoscopic Apprearance of a Pedunculated Polyp

When invasive cancer is found in a polyp, the management is based on the level of invasion and the completeness of polypectomy. Based on Haggitt's

observations, it has been suggested to treat pedunculated colonic cancers with Haggitt levels 1, 2, and 3 with complete endoscopic excision or snaring, whereas level 4 lesions should be treated as sessile T1 tumors. A sessile lesion, which can be snared adequately in one piece and on microscopic examination shows a clear margin of more than 2 mm, is considered adequately managed. If a sessile lesion is removed in piecemeal technique and demonstrates lymphovascular invasion, deep invasion into level Sm3, or has a microscopically clear margin of less than 2 mm, the patient should undergo colonic resection.

CARCINOMA OF THE COLON

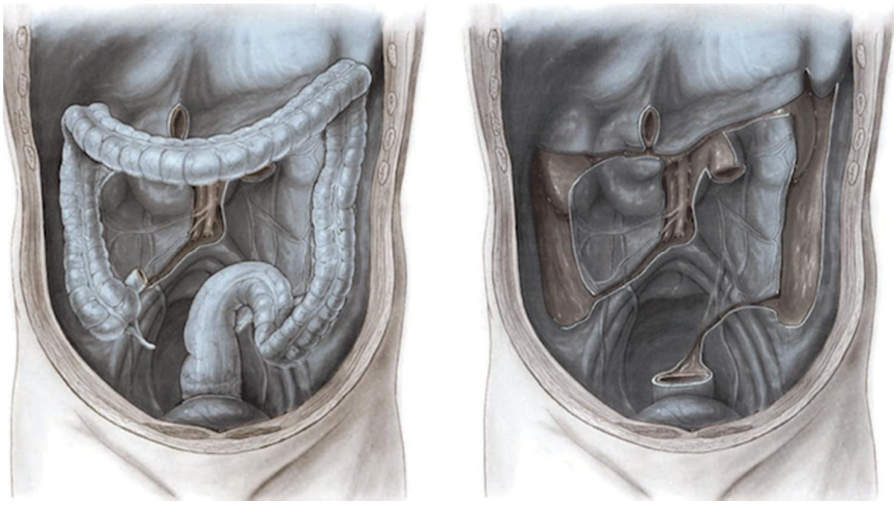


Figure 3: Showing the Topography of Colon

Adenocarcinoma accounts for 90 to 95% of all colorectal malignancies. Mucinous adenocarcinoma has incidence of 10% and has poor prognosis. Signet ring cell and small cell carcinomas have less than 1% incidence and

prognosis of very poor as almost all cases have liver and brain metastasis at the time of presentation. Other rare types include small cell adeno-squamous, squamous and undifferentiated medullary carcinomas.

The distribution of colorectal cancers among the various segments has seen a continued shift toward right-sided colon cancer. Around 45 to 55% of colon cancers are left sided whereas only 25 to 35% cancers occur on the right side of colon. The local growth pattern for colorectal cancer involves circumferential and transmural invasion of the tumor through the intestinal wall into the peritoneal cavity or surrounding organ structures. Tumor dissemination occurs primarily through access to the lymphatic vessels into the locoregional lymph nodes or through access to the bloodstream as hematogenous metastasis to distant organs. The most common site of blood borne spread is via the portal venous system to the liver; other secondary locations include the lung and, less frequently, kidneys, bone, etc. In addition, tumor dissemination can occur by trans peritoneal seeding and result in peritoneal carcinomatosis. Following gravity, peritoneal seeds may accumulate in the pelvic cul-de-sac or paracolic gutters, where they can grow to a considerable size (Blummer's shelf). Growth by perineural infiltration may be seen on microscopic examination and has a negative prognostic impact. About 20% of patients have evidence of distant metastases (stage IV disease) at the time of presentation.

TUMOR STAGING

The TNM system classifies colorectal tumors on the basis of the invasiveness (not size) of the primary (T stage), the number (not size or location) of local-regional lymph nodes containing metastatic cancer (N stage), and the presence or absence of distant metastatic disease (M stage).

T STAGE

I*n situ* adenocarcinoma (Tis) includes cancers confined to the glandular basement membrane or lamina propria. The terms *high-grade dysplasia* and *severe dysplasia* are synonymous with *in situ* carcinoma and are also classified as Tis. T1 tumors invade into but not through the submucosa. T2 tumors invade into but not through the muscularis propria, and T3 tumors invade through the muscularis propria into the subserosa or into nonperitonealized pericolic or perirectal tissue. T4 tumors invade other named organs or structures (T4a) or perforate the visceral peritoneum (T4b). Tumors invading other colorectal segments by way of the serosa (i.e., carcinoma of the cecum invading the sigmoid) are classified as T4a. A tumor that is adherent to other structures or organs macroscopically is classified clinically as T4a; however, if the microscopic examination of the adhesions is negative, then the pathologic classification is pT3.

N STAGE

Because of the prognostic significance associated with increased numbers of lymph nodes inspected (see later discussion) the current TNM classification scheme calls for at least 7 to 14 lymph nodes to be analyzed, and both the number of nodes that are positive for tumor and the total number of nodes inspected should be reported. A pN0 designation may be made even if fewer than the recommended numbers of nodes are present; however, the prognostic significance of this pN0 designation is weaker. N0 denotes that all nodes examined are negative. N1 includes tumors with metastasis in one to three regional lymph nodes (N1a:1 positive lymph node, N1b: 2-3 positive lymph nodes, N1c:extra nodal tumor deposits). N2 indicates metastasis in four or more regional lymph nodes (N2a: 4-6 positive nodes, N2b: >7 positive lymph nodes). Metastatic nodules or foci found in the pericolic, perirectal, or adjacent mesentery without evidence of residual lymph node tissue are regarded as being equivalent to a regional node metastasis and are counted accordingly.

M STAGE

Patients are designated M0 if no evidence of distant metastases is present. Identification of distant metastases denotes a classification of M1. Involvement of the external iliac, common iliac, para-aortic, supraclavicular, or other nonregional lymph nodes is classified as distant metastatic (M1) disease.

TNM CLASSIFICATION OF COLORECTAL CANCER

STAGE	T	N	M
0	Tis	N0	M0
I	T1, T2	N0	M0
IIA	T3	N0	M0
IIB	T4a	N0	M0
IIC	T4b	N0	M0
IIIA	T1-T2 T1	N1/N1c N2a	M0 M0
IIIB	T3-T4a T2-T3 T1-T2	N1/N1c N2a N2b	M0 M0 M0
IIIC	T4a T3-T4a T4b	N2a N2b N1-N2	M0 M0 M0
IVA	ANY T	ANY N	M1a
IVB	ANY T	ANY N	M1b

NONEPITHELIAL TUMORS OF COLON

CARCINOID OR NEUROENDOCRINE TUMORS

Carcinoid tumors may occur anywhere in the entire body. The gastrointestinal tract is affected in 55% of cases, with the most frequent location being the small intestine (44.7%), the rectum (19.6%), the appendix (16.7%), and the colon (10.6%), a finding that contrasts with traditional reports that the appendix is the most frequent site in the gastrointestinal tract. The annual incidences for the colon and rectum were reported to be 2 and 4.2 cases per 100,000 people per year, the risk of metastasis proportional to the size of the carcinoid. Carcinoids less than 1 cm in size are considered benign, lesions greater than 2 cm are likely malignant, and the gray zone in between remains undetermined or potentially malignant. Malignant carcinoids may spread loco-regionally into the lymph nodes or directly to the liver. An oncologic resection should be performed in all carcinoids larger than 2 cm, whereas the management of tumors measuring 1 to 2 cm remains controversial.

Patients with a gastrointestinal carcinoid tumor may be either completely asymptomatic or present with intestinal obstruction, bleeding, carcinoid syndrome, or carcinoid heart disease (valvular right heart disease). Carcinoid syndrome is a bad prognostic sign because it is caused metastatic lesions in the liver that release vasoactive substances (e.g., serotonin and 5-hydroxyindolacetic acid) directly into the systemic circulation. Diagnosis may

be suspected clinically but is difficult to confirm histologically because the lesions are submucosal and not commonly reached with an endoscopic biopsy.

GASTROINTESTINAL STROMAL TUMORS (GISTS)

These tumors of mesenchymal origin arise from the interstitial cells of Cajal. Sixty percent of GISTs are found in the stomach; 29% in the small intestine; 2% in the colon, rectum, and rectovaginal septum; and 9% in the esophagus. Symptoms are nonspecific and include pain, obstruction, bleeding, and a mass. Distinction from other mesenchymal tumors (e.g., leiomyosarcoma) is important from a prognostic point of view. Tumor size and light microscopic determination of the mitotic rate. The diagnosis of GISTs is based on morphologic features and immunohistochemical demonstration of *c-Kit* (CD117) expression. This marker is seen in almost all GISTs and is regarded as one of the key diagnostic elements but a few otherwise characteristic tumors are found to be *c-Kit*-negative. Determination of CD117 expression is of practical importance because positivity correlates with a tumor response to imatinib.

NODULAR LYMPHOID HYPERPLASIA

Numerous protruding lymphoid nodules in the mucosa of gastro intestinal tract characterize this condition. Associated diseases are immune deficiencies of various origins (e.g., tumors, hematoproliferative disorders, immunoglobulin A deficiency, and HIV infection), in which case recurrent

infections (e.g., giardiasis) appear to promote the nodular lymphoid hyperplasia. Immunocompetent patients usually are asymptomatic. This condition is associated with the lymphoma of small bowel.

LYMPHOMA

Primary malignant lymphoma of the colon is uncommon and accounts for only 0.2 to 0.4% of all colonic malignancies and 10 to 15% of all primary lymphomas the gastrointestinal tract, which themselves account for about 30% of extra nodal lymphomas. The most frequent colonic location is the cecum (70%), follow by the rectum and ascending colon. The gross appearance may be a circumferential or polypoidal mass, ulceration, or a diffuse infiltration with stricturing and bowel wall thickening. Eighty-six percent of the lesions are solitary, but they can be multiple and diffuse in nature. The intestinal lymphomas may be subclassified into B-cell lymphomas (85%) and T-cell lymphomas (15%). Among these mantle cell lymphoma has worse prognosis, whereas mucosa-associated lymphoid tissue (MALT) lymphomas have a better prognosis. While surgical treatment may be indicated for some localized tumors, many authors consider medical management to be the primary treatment.

SMOOTH MUSCLE TUMORS

Smooth muscle tumors of the colon are rare and occur most commonly in

the form of a pedunculated leiomyoma of the muscularis mucosa. Leiomyosarcomas, which consist histologically of spindle cells that resemble smooth muscle cells, are even less frequent but are characterized by an extremely aggressive and rapid fatal growth pattern.

SURGICAL ANATOMY OF THE COLON

A fundamental understanding of the surgical anatomy is of utmost importance for an adequate surgical technique that aims at the best oncologic outcome with minimized morbidity. The large intestine starts at the ileocecal junction and extends to the anus. It is about 5 to 6 feet long and can be divided in the cecum with the appendix, the ascending colon, the transverse colon, the descending colon, the sigmoid colon, and the rectum. Definitions of where the sigmoid colon ends and the rectum begins have not always been uniform and included (1) a distance of 12 to 15 cm above the anal verge, (2) the level of the peritoneal reflection, and (3) the level of the sacral promontory. The most useful landmark from a functional as well as surgical viewpoint is the confluence of the teniae coli at the recto-sigmoid junction.as this important reference point cannot be seen endoscopically, the last 12 cm above anal verge is considered rectum. The endoscopic definition is necessary because the increasing trend toward neoadjuvant chemo radiation for rectal but not sigmoid cancer demands a determination whether a lesion is located in the rectum or in the sigmoid colon.

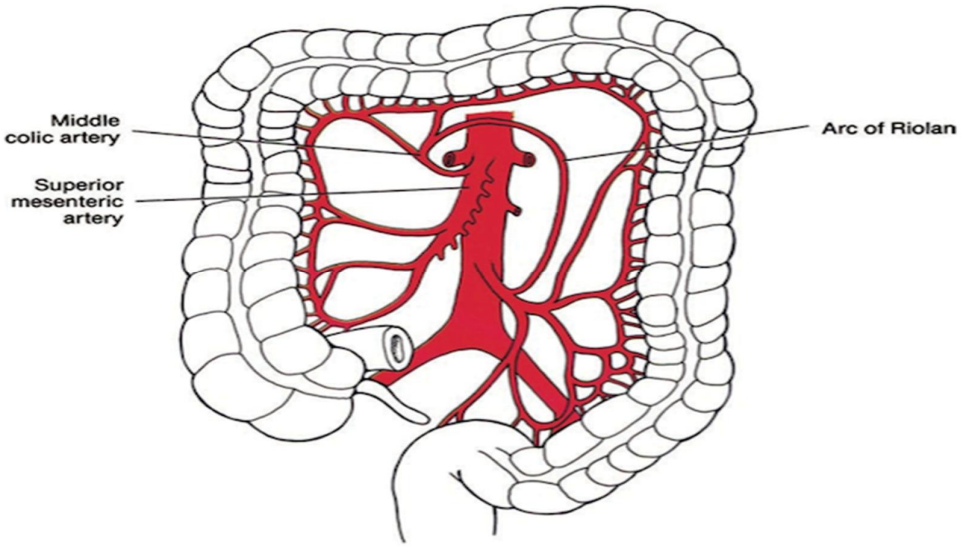


Figure 4: Shows the Arc of Riolan

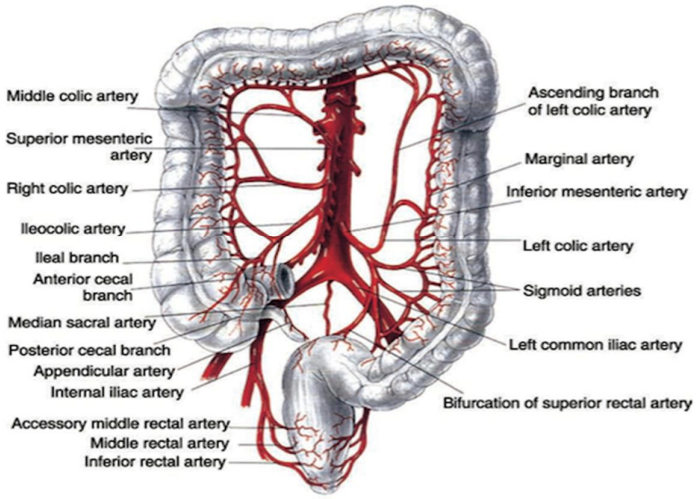


Figure 5: Shows the Arterial Blood supply of Colon

The arterial blood supply to the colon comes from the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA), which communicate in a watershed area in the splenic flexure (artery of Drummond). The rectum has additional branches from the internal iliac vessels. With a

significant degree of anatomic variation, the major vascular stalks to the colonic segments consist of the ileocecal and right colic artery (last branch of the SMA), the middle colic artery (second branch of the SMA), the left colic artery (first branch of the IMA), and the superior hemorrhoidal artery (distal branch of the IMA, feeding the sigmoid colon and upper rectum). The venous blood supply peripherally follows the arterial branches but more centrally divides into the superior mesenteric vein and the inferior mesenteric vein, which connect at separate levels to the portal system. The lymphatic drainage starts with lymphatic follicles in the colonic submucosa, drains through the colonic muscle wall into the epicolic nodes, and continues to the paracolic lymph nodes that follow the blood vessels to the bowel, along the major arteries to the principal lymph nodes at the level the arterial runoff from the aorta. These lymph node groups consist of the celiac, the superior mesenteric, and the inferior mesenteric group of lymph nodes.

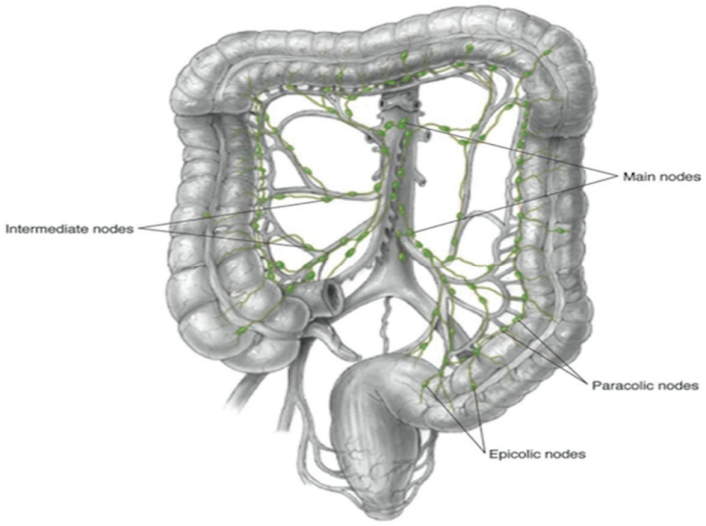


Figure 6: Shows the Lymphatic Drainage of Colon

CLINICAL PRESENTATION OF COLORECTAL CANCER

SYMPTOMS

Colorectal cancer does not have any early signs. In fact, symptoms often are absent until a tumor has grown to a significant size. Unless a patient presents with tumor complication (e.g., bowel obstruction, bleeding, perforation, or fistula formation), symptoms mostly are subtle or uncharacteristic and vague. They may consist of unexplained weight loss, anemia and weakness from chronic blood loss, flatulence, or episodes of colicky abdominal pain. If present, these symptoms therefore always should be suspicious for a locally relatively advanced tumor stage, which is also reflected by the fact that 20% of colorectal cancer patients at the time of first presentation have stage IV disease with distant metastasis. Proximal colon tumors may grow relatively large before they interfere with the passage of the still liquid or semisolid stool. The more distal a lesion is localized (e.g., left colon or rectum), the more likely changes in bowel habits occur. These include bloody or mucous discharge in or with the stool, sudden onset of constipation, alternating periods of diarrhea and constipation, or a decreasing diameter of the stool. Pelvic or anal pain is an ominous sign because it may occur with increasing size or sphincter invasion of a rectal cancer. Any large bowel obstruction, bleeding per rectum with or without peritoneal signs in an elderly should raise the index of suspicion for a colorectal malignancy until proven otherwise.

INVESTIGATIONS

Patients with symptoms suggestive of colorectal cancer should undergo a series of timely investigations with three goals: (1) to assess the large bowel for primary lesion, concomitant lesions, and a potential underlying colonic disease, (2) to determine whether the tumor has metastasized, and (3) to assess the patient's operability.

COLONOSCOPY

Colonoscopy clearly has evolved as the method of choice because of its high sensitivity in detecting tumors and its ability to take biopsies. It provides accurate information about the entire colonic mucosa (i.e., polyps, synchronous cancer, colitis and diverticula), and it may be used to remove synchronous neoplastic polyps. Apart from determining the circumferential and longitudinal extent of a colonic lesion, colonoscopy addresses functional aspects such as active bleeding or an imminent obstruction by cauterization, laser ablation, or placement of a self-expanding wall stents.

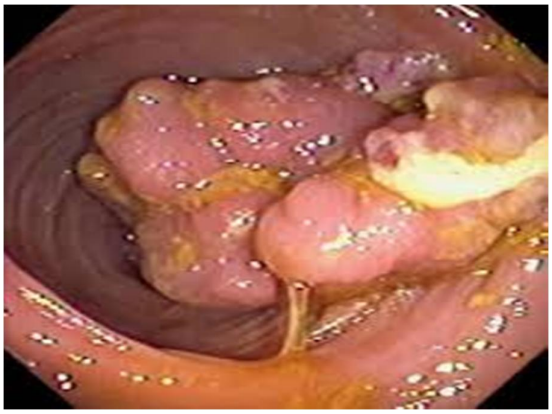


Figure 7: Shows the Colonoscopic Appearance of Adenocarcinoma Colon

While the overall risk of colonoscopy is very low, with a much less than 1% incidence of a bowel perforation, there are some limitations to the technique. There is a 25% risk of smaller lesions escaping detection and an estimated 10% incidence that the cecum may not be reached for technical reasons. In addition, the precise position of a lesion seen on colonoscopy may not be determined adequately because the only reliable landmarks are the dentate line and the terminal ileum, and the length from the anal verge may vary considerably.

CONTRAST ENEMA

Radiographic contrast enemas alternatively can be used for a colonic evaluation. They have the advantage of more accurately visualizing the anatomic position a colonic lesion. Most commonly, a barium-air double-contrast technique will be used; however, if there is suspicion of a colonic perforation, administration of barium is contraindicated (risk of barium peritonitis), and instead, a water-soluble contrast material (e.g., gastrografin) should be used. The typical aspect of a colon cancer is a fixed filling defect with destruction of the mucosal pattern in an annular configuration ("apple core"), as opposed to an intact mucosal pattern in a filling defect from an extra mucosal compression or from chronic diverticulitis. Although preoperative histologic confirmation of a colon cancer is preferable, an unequivocal and characteristic morphology on a barium enema or endoscopy is sufficient evidence to proceed to surgery. Contrast studies have the advantages of a better passage through even severely obstructing lesions and that they commonly

reach the cecum. In addition, they are superior in visualizing diverticula or a suspected fistula between the colorectal and other pelvic organs. The major disadvantage of contrast studies is the inability to take biopsies and to detect small lesions.

EVALUATION OF THE LOCAL TUMOR EXTENT AND OF METASTATIC DISSEMINATION

In contrast to rectal cancer, where endorectal ultrasound, computed tomographic (CT) scans, and magnetic resonance imaging (MRI) are used routinely in the preoperative work-up, a cancer of the colon does not necessarily require further imaging studies to determine the local extent of the tumor because in the majority of cases they do not change the local surgical approach. For a more general preoperative evaluation of the abdomen, however, abdominal sonography, CT scan, or MRI is indicated and equally accurate to check for the presence of liver metastases, ascites, hydronephrosis, or gross para-aortic lymph node involvement, a well as other concomitant diseases. CT scans are used most commonly and have a 90% and 95% sensitivity and specificity in detecting liver lesions greater than 1 cm. However, in some patients with liver metastasis or undetermined liver lesions, intraoperative liver ultrasound may be an extremely helpful tool because several studies now have shown it to be superior to preoperative radiologic examination and intraoperative clinical assessment.

In order to rule out extra hepatic metastases, a chest x-ray in two planes commonly is sufficient, although the yield of this test is relatively low. A CT scan of the chest may be necessary to substantiate a concern from conventional images. Only under special circumstances where the presence of previously unknown tumor manifestations (e.g., recurrence versus scar tissue, solitary versus multiple liver metastases, and presence of extra hepatic metastases) would have an impact on the treatment approach (e.g., operative versus non-operative) is a positron-emission tomographic (PET) scan indicated. Routine use of PET scanning in the primary management of colorectal cancer is not recommended.

LABORATORY AND PREOPERATIVE TESTS

Preoperative laboratory tests are aimed at providing evidence for pathophysiologic effects of the tumor and ruling out general health problems that could have an effect on the patients' general operability. A comprehensive work-up includes a complete blood count, electrolytes, creatinine/blood urea nitrogen (BUN), glucose, liver function tests (alkaline phosphatase, AST, ALT, bilirubin, total protein, albumin), and coagulation parameters (PT, PTT, INR).

Even though tumor markers such as carcinoembryonic antigen (CEA) are determined routinely, their role is limited because of the low sensitivity and specificity to colonic carcinoma and because the measured value virtually never changes the management. CEA can be elevated in proximal gastrointestinal

cancers, benign inflammatory conditions of the bowel, lung and breast cancer, and smoking.

> "*When there is return of an elevated preoperative CEA level to normalcy indicates a complete tumor resection or when a postoperatively elevated level may indicate residual recurrent disease.*"

Preoperative standard evaluation includes a chest x-ray in two planes for cardiopulmonary assessment and for detection of pulmonary metastases. Electrocardiogram (ECG) and pulmonary function tests (FVC, FEV_1, and RV diffusion capacity) are indicated in patients either older than 40 years of or with a respective personal history. Specialized tests such as cardiac stress tests, echocardiogram, perfusion scintigraphy, or interventional cardiologic studies depend on the individual patient's history and risk assessment.

SURGICAL PROCEDURES

GENERAL TECHNICAL PRINCIPLES

The objective of surgery for colonic cancer is to perform a curative resection by removing the cancerous segment of colon, the mesentery with the primary feeding vessel and the lymphatics, and any organ with direct tumor involvement. Since the lymphatics run with the arterial supply of the colon, the primary artery supplying the segment of the colon to be resected is divided at its origin. Ligation at the origin of the vessel ensures inclusion of apical nodes, which

may convey prognostic significance for the patient. The length of bowel and mesentery resected is dictated by tumor location and distribution of the primary artery. Nevertheless, with radical excision of a colonic tumor, at least a 5-cm distal and proximal clearance is required. There is no additional survival benefit with respect to extended resections however; tumors located in "border zones" should be resected with adjacent lymphatics to avoid possible bidirectional spread. When synchronous cancers are present in the colon, an extended resection or even total colectomy, with ideally only one anastomosis, should be performed. Occasionally, two separate resections (e.g., right hemi-colectomy and low anterior resection) with two anastomoses are preferable to preserve colon length and to avoid post colectomy diarrhea. Cancer on the basis of an underlying pan colonic disease (e.g., ulcerative colitis or FAP) requires a total procto-colectomy with either an ileo-anal pull-through procedure or an ileostomy.

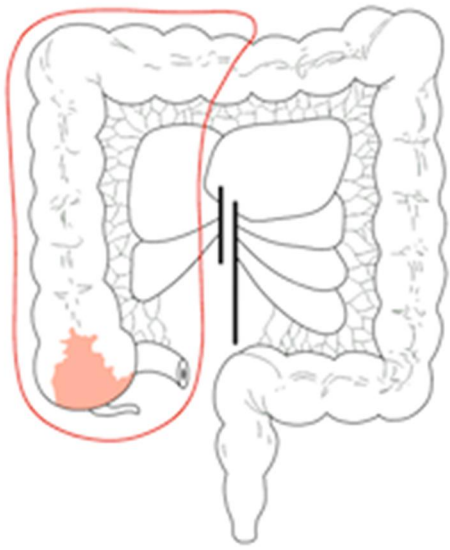


Figure 8: Shows the Technique of Right Hemicolectomy

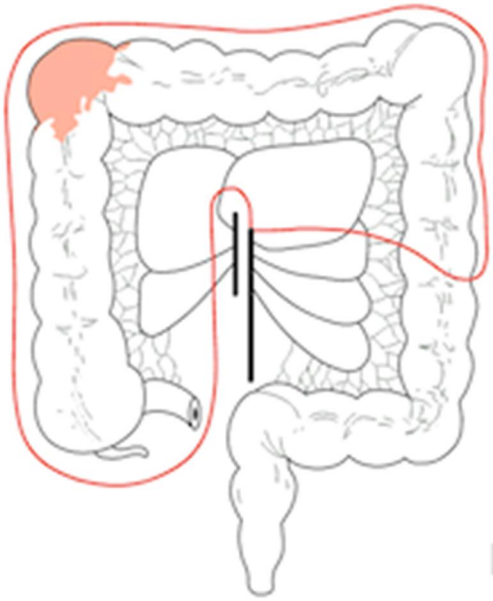


Figure 9: Shows the Technique of Right Extended Hemicolectomy

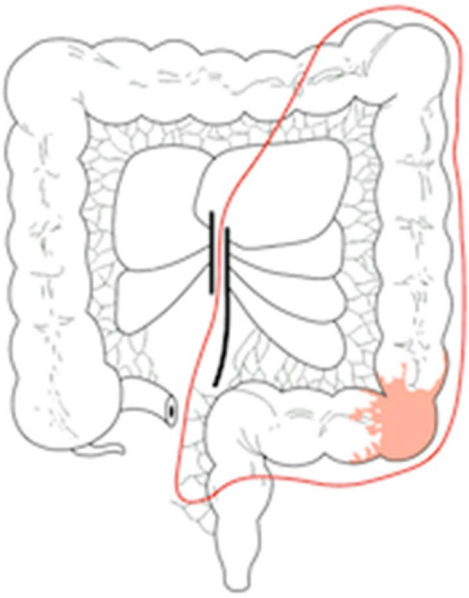


Figure 10: Shows the Technique of Left Hemicolectomy

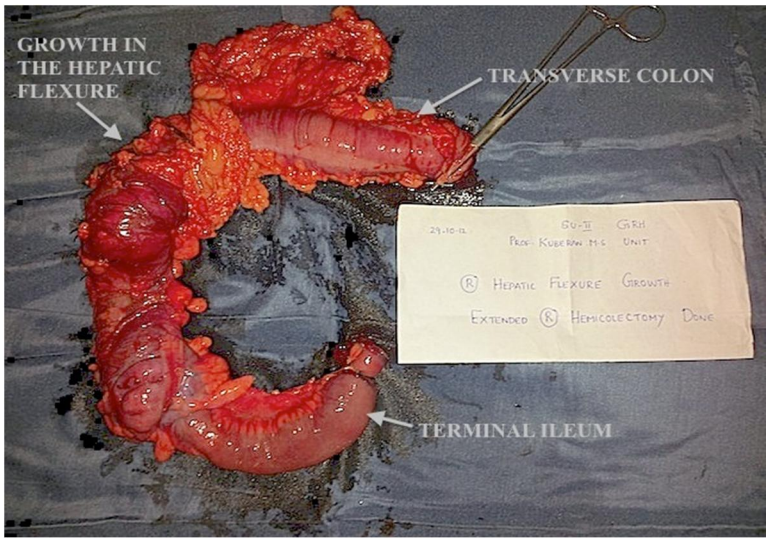


Figure 11: A Right Extended Hemicolectomy Specimen

Right hemi-colectomy is done for tumors involving caecum and ascending colon. Extended right hemi-colectomy is done for tumors involving hepatic flexure and transverse colon wherein terminal ileum to splenic flexure of the colon is resected. Extended left hemi-colectomy is done for splenic flexure growths. Left hemi-colectomy is done for tumors of descending colon.

SPECIAL CIRCUMSTANCES IN EMERGENCY SURGERY

Tumor-related complications (e.g., bowel obstruction, perforation, or massive bleeding) mandate emergency laparotomy, which accounts for around 20% of patients with colon cancers. Morbidity and mortality are significantly higher than under elective conditions. The risks for wound and intra-abdominal infections and anastomotic leakages are three to six times higher.

TUMOR OBSTRUCTION

Sixteen percent of patients with colon cancer present with a bowel obstruction and complain of colicky abdominal pain, abdominal distension, vomiting, constipation, and occasionally, paradoxical diarrhea. Imaging studies (abdominal x-ray or CT scan) characteristically demonstrate the features of a large or small bowel obstruction depending on how proximal in the colon the obstruction is located and whether the ileocecal valve is competent. There is risk of caecal perforation if the diameter reaches 12 cm or more. Urgent intervention is required in such circumstances to prevent caecal perforation. The most important differential diagnosis is pseudo-obstruction (Ogilvie's syndrome), which is seen as a result of various medical conditions and may mimic the features of bowel obstruction. Every patient therefore should have a rigid proctoscopy, followed by a water-soluble contrast enema, which should visualize only the colon up to the site of obstruction but not beyond the stenosis because the hyperosmolar nature of the contrast material can result in an increase in the intraluminal volume and trigger a perforation. If the level of obstruction in the colon is proximal enough, a resection with primary entero-colonic anastomosis, e.g., right hemi-colectomy, extended right hemi-colectomy, or subtotal colectomy, may be carried out. Surgeries for tumors involving left side of colon necessitates taking into consideration certain precautions because the stool load proximal to the obstruction is of concern for a colo-colonic anastomosis because that segment of

the colon could not be cleared before the operation. Synchronous lesions may occur in up 15% of tumors presenting as obstruction, may be missed and necessitate further intervention in the future. Obstructed left-sided tumors were treated with a three-stage approach starting with a defunctioning loop colostomy, followed by resection and anastomosis and last by closure of the defunctioning stoma. The Hartmann operation is a two-stage procedure, which consists of a recto-sigmoid resection and creation of a terminal end colostomy and rectal stump in the first stage, followed by re-anastomosis of the colon in a second sitting.

More recently colonoscopy guided insertion of a self- expanding metallic stents were used to relieve the acute obstruction at the tumor-bearing segment of the colon. Successful decompression of the prestenotic colon converts the emergency situation into an elective setting, allowing for stabilization the patient and performance of bowel preparation. The risk of a colonic perforation during stent placement is relatively low but acceptable because an emergency operation would be necessary anyway if the stent could not be placed successfully. Studies have demonstrated the safety of stent application in the cases of tumor complicating obstruction and were found to be highly successful. A proximal diversion may be performed with this procedure.

TUMOR-RELATED PERFORATION

Colonic perforation secondary to a tumor occurs in two different settings.

Either a transmural tumor perforates itself, or the proximal colon becomes over distended, particularly in the case of a competent ileocecal valve. Both conditions may result in diffuse fecal peritonitis with significant morbidity and mortality. In addition, the tumor perforation results in spillage of tumor cells and thus has to be considered a stage IV tumor. Surgical management is indicated every case and requires not only addressing the site of colonic perforation but also removing the tumor.

MASSIVE COLONIC BLEEDING

Massive bleeding from a colonic tumor is a relatively rare complication. Most commonly the bleeding site can be identified easily. If the patient remains unstable and requires repeated transfusions, surgical management is indicated.

MANAGEMENT OF ADVANCED DISEASE

LOCALLY ADVANCED DISEASE

It has been estimated that approximately 15% of colonic tumors will be adherent to adjacent organs. With locally advanced colon tumors, it is still possible to achieve cure if resection of the involved adjacent organs is done. Unfortunately it is difficult to distinguish between malignant adhesions from inflammatory one, but at least 40% of these adhesions harbor malignant cells. It is therefore imperative to perform an en-bloc resection to achieve a tumor-free margin.

OPERABLE METASTASES

Patients with stage IV disease at the time of presentation accounts for 20 percent of all cases. Distant metastasis, particularly liver and lung are a major cause of death in patients with colorectal carcinoma. However, patients with asymptomatic liver metastases may have a statistically natural life expectancy of several months up to almost 2 years without any treatment. Chemotherapy and surgical removal of metastasis in selected patients may improve disease-free and overall survival substantially, resulting in a cure rate of 30%. In the case of potentially resectable metastases, resection of the colonic primary tumor therefore should be performed with tumor-free margin status.

INOPERABLE DISSEMINATED DISEASE

In patients with unresectable metastatic disease, the surgical treatment goal is to provide palliation and to prevent complications. In contrast to the oncologically defined standard resections, a limited segmental wedge resection of the colon is acceptable in this setting. Particularly tumors located in the sigmoid colon or in the cecum and ascending colon are suitable for a laparoscopic or laparoscopically assisted resection because these segments can be mobilized easily a sufficient extent to ensure a safe anastomosis. If a tumor in a patient with metastatic disease with local spread (e.g., infiltration of other organs), palliation may be achieved by creating an internal bypass or a proximal diversion.

COMPLICATIONS OF SURGERY

The overall perioperative mortality within 30 days of colorectal resections is less than 2% after elective but up to 20% after emergency operations. Complications of surgery may be of a general or surgery-specific nature and can be classified with regard to the time of their occurrence either early (within the first 30 days) or late (after 30 days). Intraoperative complications such as injury to relevant anatomic structures such as ureters, spleen bowel, and duodenum are related to the surgical technique, to blurred anatomic landmarks and layers owing to the disease (e.g., peritonitis or massive adhesions), or to the patient's habitus (e.g., obesity). Early surgery-specific complications include bleeding, most frequently within the first few days of the resection, nonspecific infections, or infections related to an anastomotic dehiscence. Other more general complications in the early postoperative period (postoperative days 1 to 3) commonly are related to the cardiopulmonary system and include pulmonary problems (e.g., atelectasis, pneumonia, aspiration, and pulmonary embolism) and cardiac events (e.g., arrhythmia, myocardial ischemia, and dysfunction). Insufficient pain control has been recognized as an important factor promoting these conditions because it results in a poor respiratory effort by the patient and the inability to cough up sputum, leading to superficial respiration and suboptimal saturation. High fever in the 3 days therefore may be related to the development of an atelectasis rather than to an early infection.

Infectious complications usually occur after the third postoperative day and may be located either intra-abdominally, in the wound, in the urinary tract, or in the lungs. The primary work-up therefore includes bacteriologic cultures and stains, blood and urine analysis, and a chest x-ray.

Abdominal complications consist of delayed return of upper and lower gastrointestinal function (also referred to as *postoperative ileus*), fascial dehiscence, and anastomotic breakdown. An anastomotic leak may present with insidious symptoms such as fever, tachycardia, abdominal distension, ileus, feces draining through a drain or the wound, or local and generalized peritonitis. Occasionally, a leak may present with sudden deterioration, generalized peritonitis, and septic shock as the result of a significant and rapid contamination of the peritoneal cavity. Owing to the heterogeneous symptoms, a leak should be suspected in any patient who is not progressing to the expected degree. Blood parameters such as white blood cell counts and C-reactive protein may be elevated but are nonspecific and difficult to distinguish from a normal postoperative reaction. After an abdominal operation, normal free air should be resorbed within 7 to 10 days. The presence of substantial free sub diaphragmatic air later in the course therefore should raise the index of suspicion for an anastomotic leak.

Imaging studies to define the presence of an anastomotic leak include a water-soluble contrast enema to visualize extravasation of the contrast material and/or CT scan with oral, intravenous, and possibly rectal contrast material. Apart

from antibiotic treatment, the management of an anastomotic leak depends on its presumed extent and the clinical presentation. A patient with generalized peritonitis requires a re-laparotomy after appropriate resuscitation. Depending on its location, the anastomosis either should be taken down and the ends should be exteriorized or, in more favorable conditions, resected and a new anastomosis performed with healthy-looking bowel ends, either with or without proximal diversion. A local repair alone carries a high risk of failure but may succeed in combination with drain placement and a proximal diverting ostomy. A fecal fistula can be managed in a conservative manner if there is no evidence of generalized peritonitis or uncontrolled sepsis. Under favorable conditions, including good nutritional support and absence of a distal obstruction or disease of the involved bowel segment, the fistula may close spontaneously. The surrounding skin will need special care, and a stoma therapist will be helpful in this regard.

ADJUVANT CHEMOTHERAPY AND RADIOTHERAPY

The rationale for adjuvant chemotherapy is based on the fact that there is still recurrence following surgical curative resections even though the incidence is very low. 5 Fluorouracil was the first and most extensively evaluated drug for the treatment of colorectal cancer. Studies showed only 5% improvement in survival with adjuvant chemotherapy. However, when just those at high risk of recurrence are treated, the improvement in survival in this group is closer to 30%. High risk patients like those of stage III colon cancer are ideal candidates for FU/leucovorin

based adjuvant chemotherapy for 6 months after surgery and has proven to decrease recurrence and improve survival. The combination treatment of 5-FU/LV for 6 months was proven to be equivalent in efficacy to 12 months. The addition of levamisole to 5-FU/LV based regimens did not seem to add any benefit. Low-dose LV also was demonstrated to be equally efficacious as high-dose LV when used in combination with 5 FU.

Several new agents, e.g., irinotecan and oxaliplatin have demonstrated significantly superior activity in combination with 5 FU/LV in the metastatic setting. Irinotecan/5 FU/LV (FOLFIRI) and oxaliplatin/5 FU/LV (FOLFOX) prove that the new agents in association with 5 FU/LV were superior to 5 FU/LV alone in the treatment of stage III disease. The FOLFOX regime showed lesser side effects though sensory neuropathy and neutropenia were commonly associated with oxaliplatin based regimens.

Capecitabine is a oral drug that preferentially releases the active component (5-FU) to the tumor tissue. A randomized study comparing oral capecitabine versus intravenous 5-FU/LV concluded that capecitabine demonstrated a statistically significantly great response rate compared with 5-FU/LV and an equivalent time to progression and overall survival. This study demonstrated the capecitabine is a suitable alternative to IV 5-FU and perhaps a replacement in the future. There are currently phase II trails being conducted on capecitabine/oxaliplatin (CAPEOX) and capecitabine/irinotecan (CAPEIRI). The

epithelial growth factor receptor (EGFR) and vascular endothelial growth factor (VEGF) antagonists like cetuximab and bevacizumab respectively have shown proven benefit and can be effective as a first line drug.

Generally, radiotherapy does not play a primary role in the adjuvant treatment of colon cancer. However, it may be considered as a loco regional field radiation in selected locally advanced tumors.

OUTCOME AND PROGNOSIS

With the advent of better screening and diagnostic modalities there is better outcome and survival in patients diagnosed with colorectal cancer. This may be related to safer and more successful surgical treatment in combination with better non-operative and adjuvant treatments. The perioperative mortality within 30 days of elective colorectal resections is less than 2%. The 5 year survival is less than 10% for a stage 4 disease when compared to >90% for a stage 1 disease, thus implying the significance of screening and importance of early diagnosis of colonic malignancies.

MATERIALS AND METHODS

This is a prospective case study of colonic malignancies with exclusion of genetic and familial cancers, patients with concomitant malignancies and severe cardiac ailments. This study was conducted with patients admitted to Government Royapettah hospital/Kilpauk Medical College, Chennai, located in the southern part of India. The study was conducted prospectively from the day of admission in 50 patients of colon cancers in various surgical units, between June 2011 and November 2013. All cases studied were subjected to detailed clinical examination with emphasis on the family history of colon cancers and duration of the presenting complaints and the accurate chronology of symptoms complex associated with each of the colonic malignancies. A thorough physical examination was done with special emphasis on the abdomen and the relevant clinical findings like mass, hepatomegaly, ascites, left supraclavicular lymphadenopathy. After the clinical diagnosis of colon cancer the patients were subjected to colonoscopy to confirm the diagnosis and the documentation of the gross appearance of the tumor whether polypoidal, ulcerative, ulcero-proliferative or circumferential were recorded with the location of the tumor. Then tumor metastatic workup is done with ultrasound and computerized tomography of abdomen and the local nodal spread with hepatic metastasis were documented. The histopathology reports of the colonoscopy biopsy with respect to tumor differentiation (well differentiated, moderately differentiated or poorly

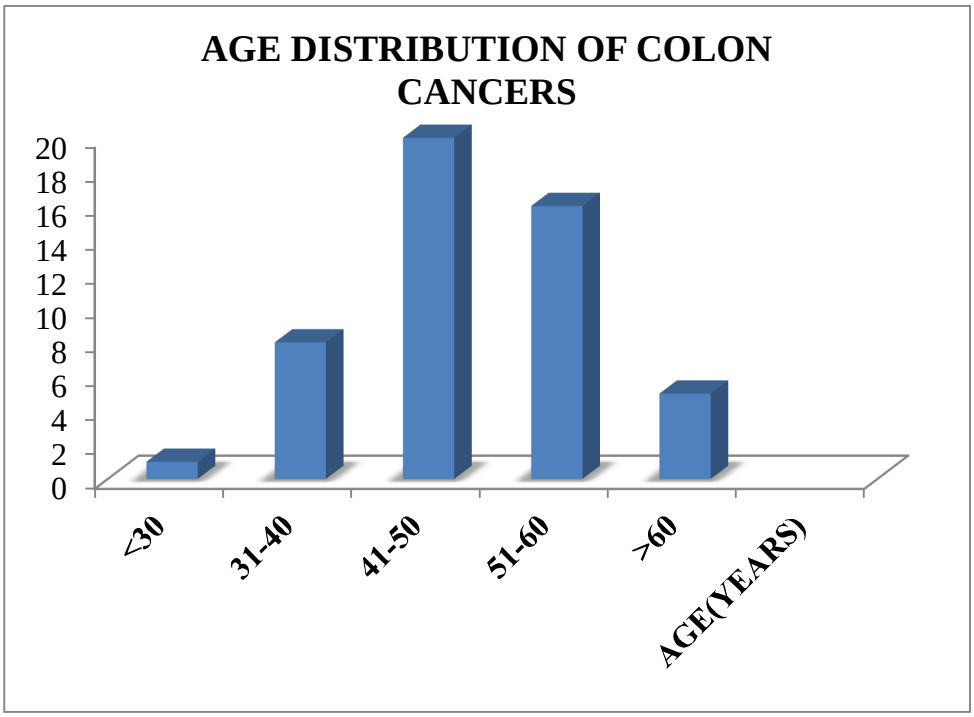
differentiated) were recorded. In addition to the routine blood investigations, CEA levels were recorded preoperatively. Then after the preoperative workup, the patients were taken up for surgery based on tumor operability and the general condition of the patient. The surgical procedures performed for various colonic malignancies based on their location were documented. Then the postoperative complications, which include wound infection, paralytic ileus, respiratory infection, and anastomotic leak, were studied and the duration of stay of the patients were also recorded. The histopathology report of the resected specimens with respect to the tumor penetration and number of nodal spread were also recorded and the stage of the disease was ascertained. All studied patients were evaluated for CEA levels at their 14^{th} postoperative day and those discharged prior were followed up accordingly. The patients irrespective of he tumor stage were treated with adjuvant chemotherapy with 5-FU based regimen. The discharged patients were asked to follow up with the CEA levels every 3 months or if there is any symptoms.

OBSERVATION AND RESULTS

Based on data collected as per the proforma and the entries in the Master Chart the following observations were made.

Table 1: Age Distribution of Colon Cancers

AGE (YEARS)	INCIDENCE
<30	1
31-40	8
41-50	20
51-60	16
>60	5



Bar Chart showing the Incidence of Tumors during Various Decades of Life

Table 2: Sex Distribution of Colon Cancers

CASES	MALE	FEMALE
RIGHT COLON CANCERS	24	11
LEFT COLON CANCERS	9	6

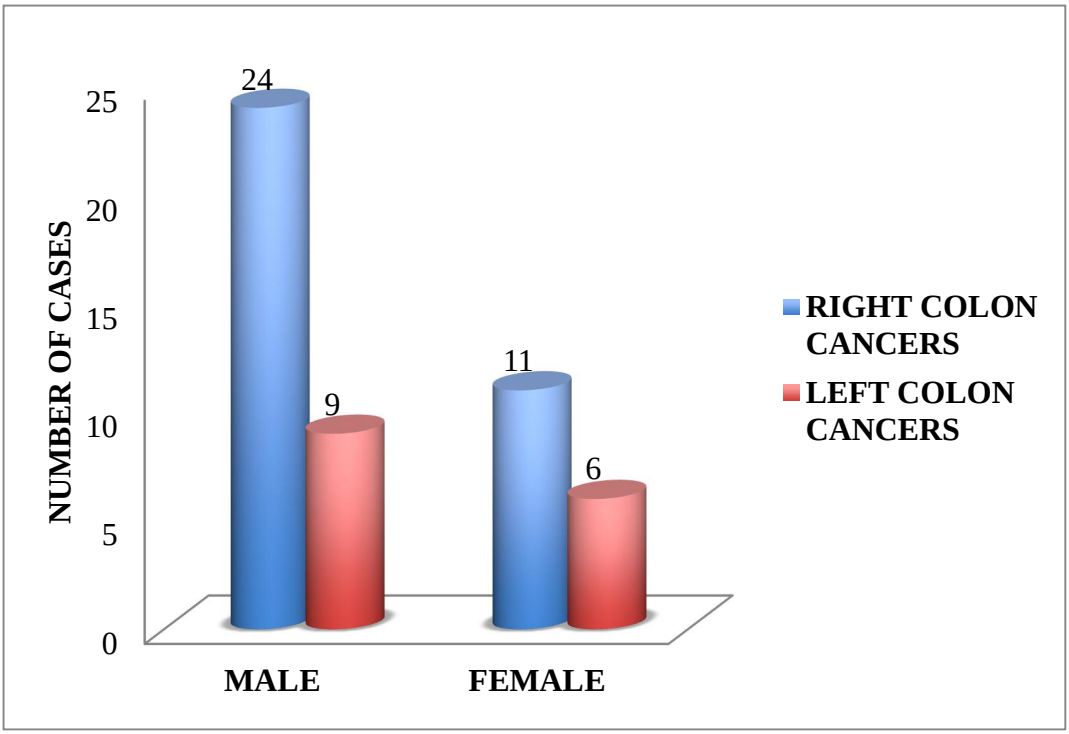


Table 3: Clinical Presentation of Colon Cancers

SYMPTOMS	RIGHT COLON CANCERS	LEFT COLON CANCERS
ANEMIA	26	3
MASS	22	2
PAIN	6	1
CONSTIPATION	5	12
LOOSE STOOLS	1	13
MALENA	11	5
BLEEDING P/R	5	9
LOSS OF WEIGHT	28	11
LOSS OF APPETITE	31	9

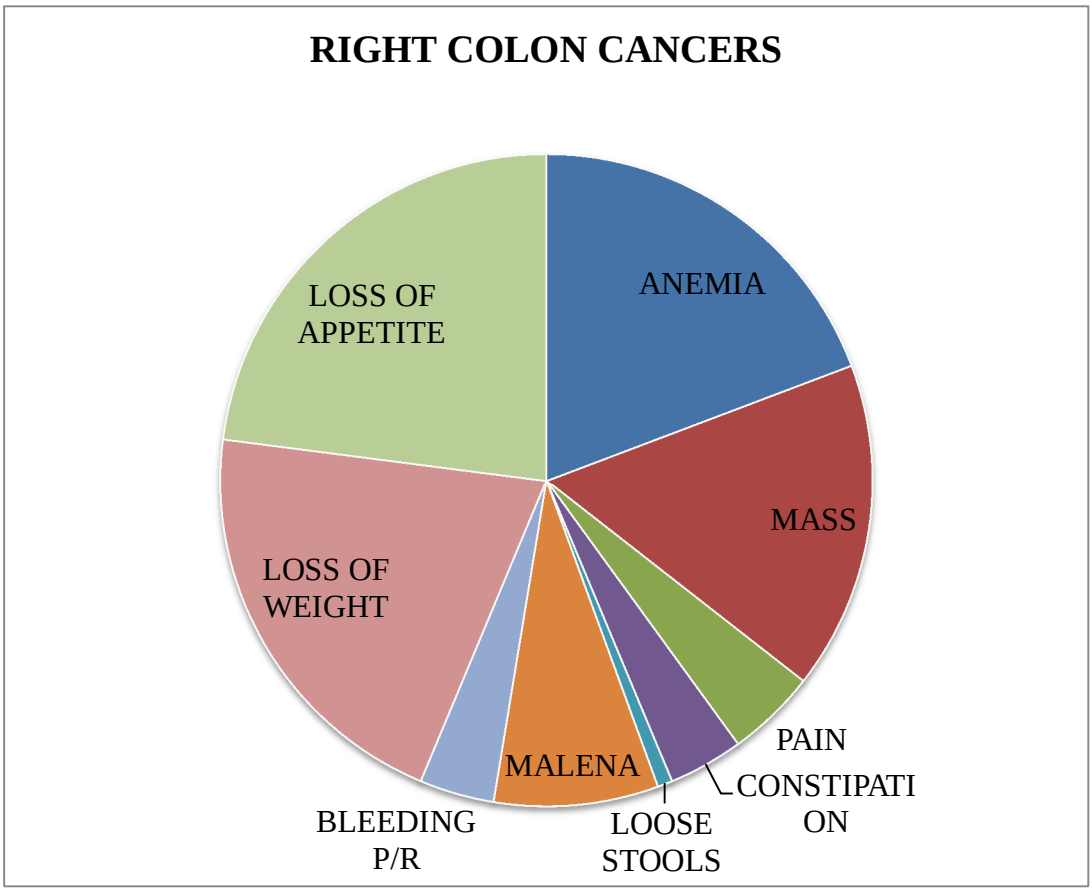


Chart depicting the symptom complex of right colon cancers

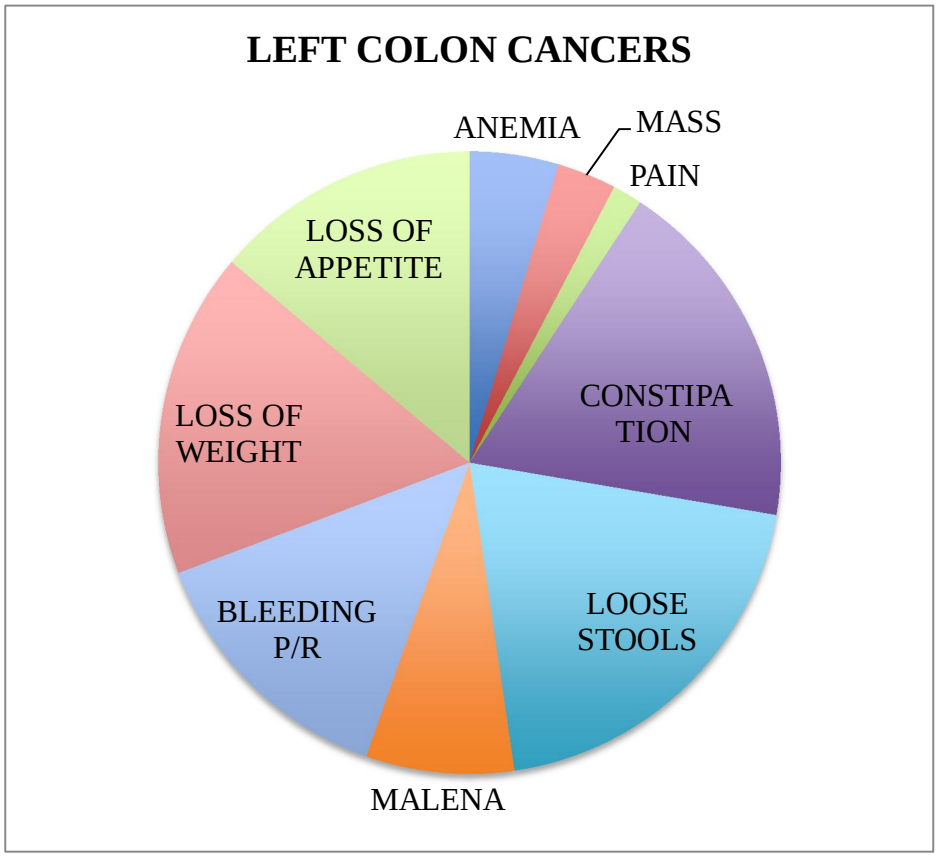


Chart depicting the symptom complex of left colon cancers

Table 4: Duration of Presenting Complaints

MONTHS	RIGHT COLON CANCERS	LEFT COLON CANCERS
1 TO 3	2	13
4 TO 6	19	2
7 TO 9	8	2
>9	4	0

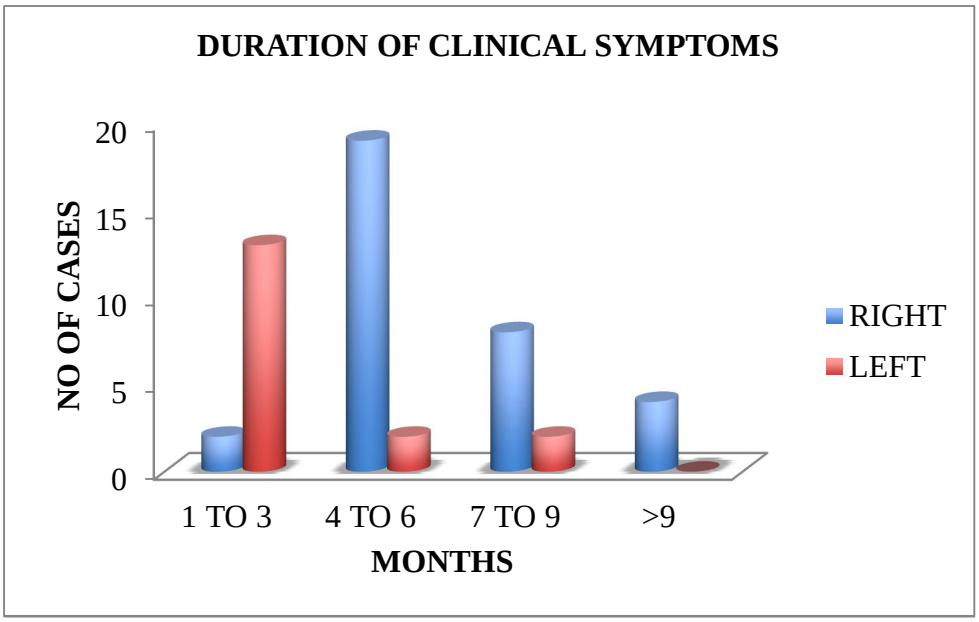


Table 5: Tumor Appearance in Colonoscopy

CASES	POLYPOIDAL	ULCERATIVE	ULCERO-PROLIFERATIVE	CIRCUMFERENTIAL
RIGHT COLONIC CANCERS	4	4	25	2
LEFT COLONIC CANCERS	0	0	6	9

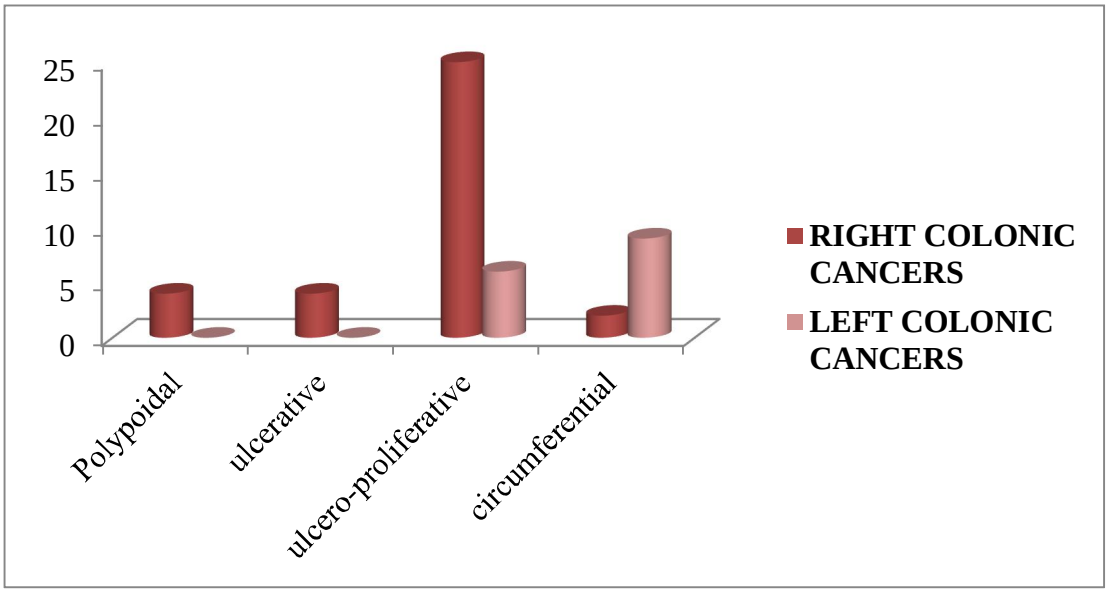


Table 6: Shows Tumor Location and its Incidence

TUMOUR LOCATION	INCIDENCE
CAECUM	3
ASCENDING COLON	16
HEPATIC FLEXURE	9
TRANSVERSE COLON	7
SPLENIC FLEXURE	4
DESCENDING COLON	6
SIGMOID COLON	5

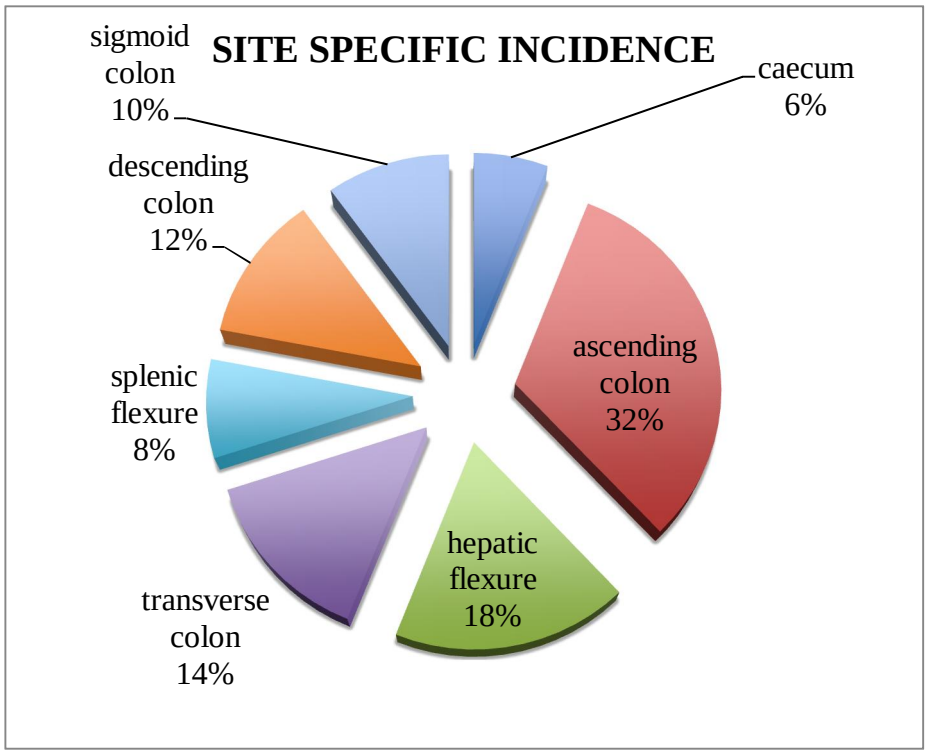


Chart depicting the site specific incidence of colonic malignancies

Table 7: Shows Correlation between the Tumor Differentiation and Nodal Disease in Computerised Tomography

TUMOUR DIFFERENTIATION	TUMOURS WITH NODAL DISEASE IN CT
WELL DIFFERENTIATED	1
MODERATELY DIFFERENTIATED	4
POORLY DIFFERENTIATED	9

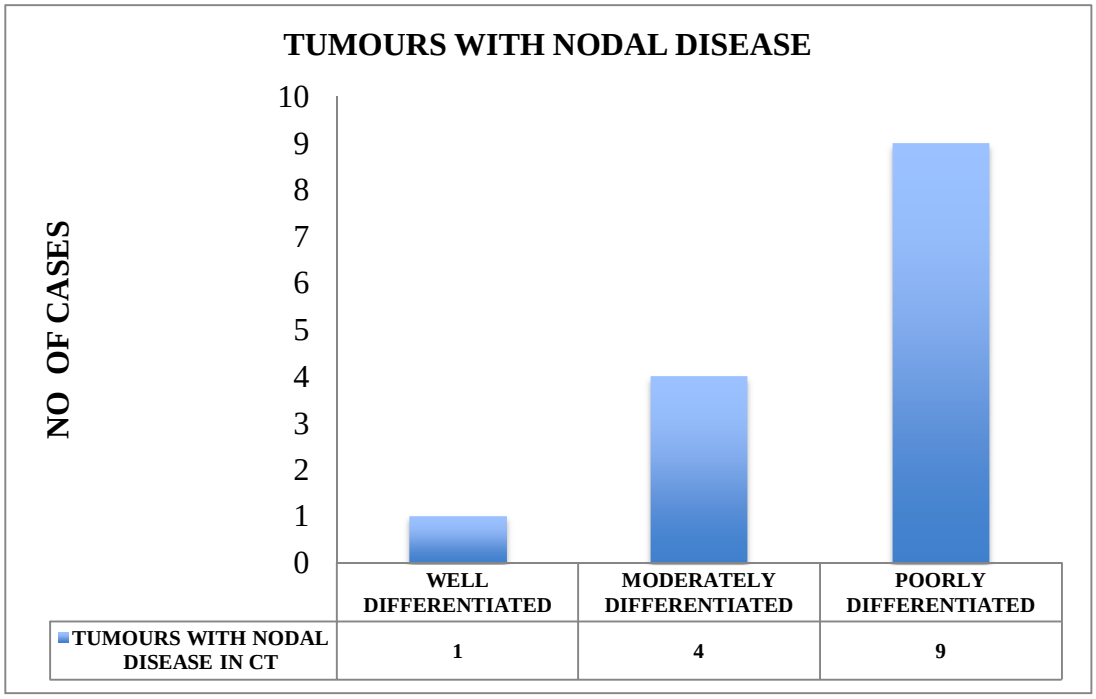
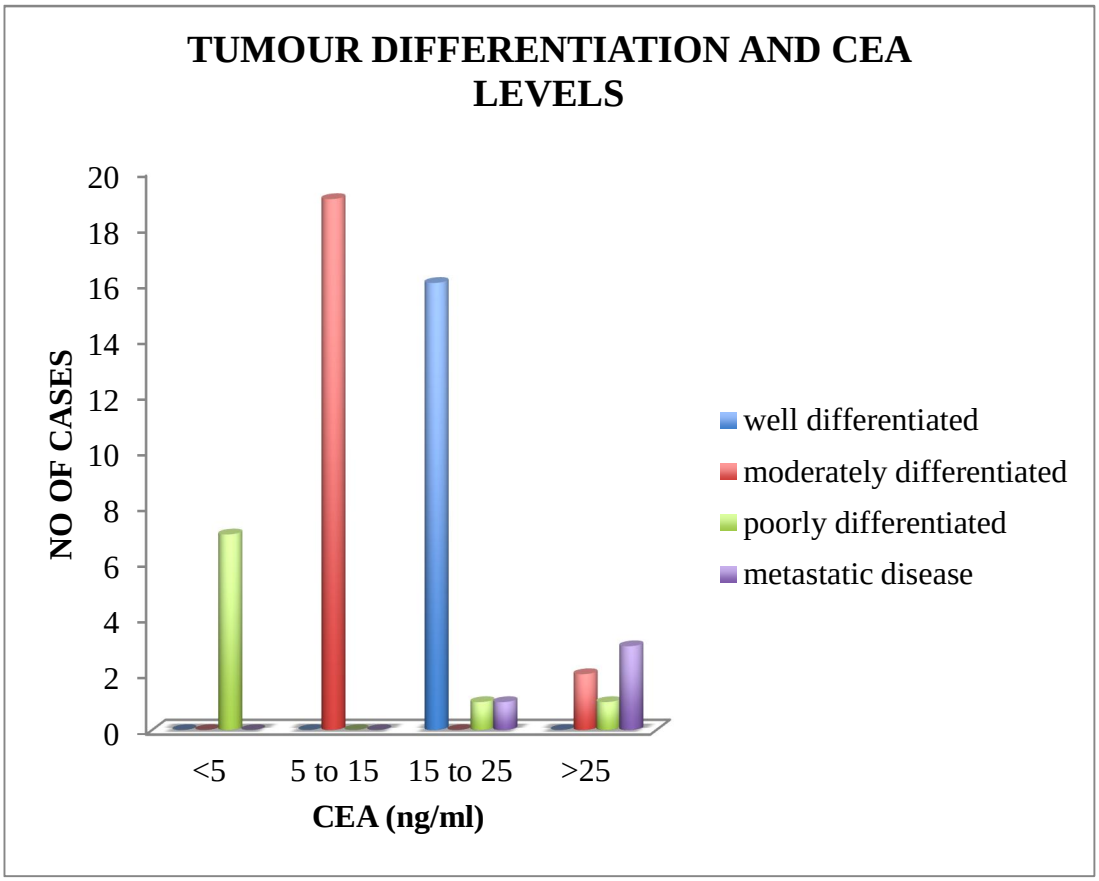


Table 8: Shows correlation between Tumor Differentiation and CEA Levels

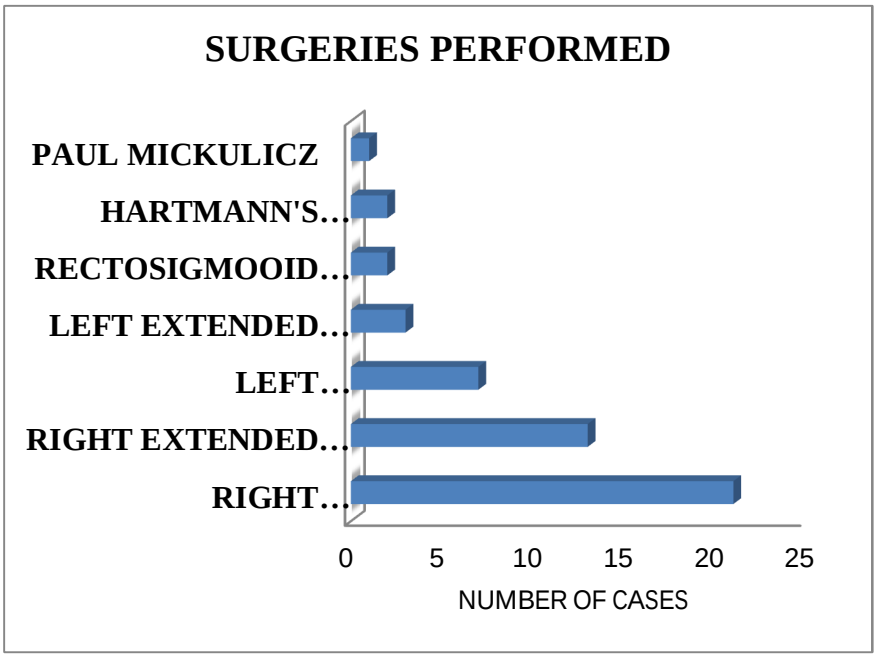
TUMOUR DIFFERENTIATION	CEA LEVELS (ng/ml)			
	<5	5 to 15	15 to 25	>25
WELL DIFFERENTIATED	0	0	16	0
MODERATELY DIFFERENTATED	0	19	0	2
POORLY DIFFERENTATED	7	0	1	1
METASTATIC DISEASE	0	0	1	3



Bar Chart Showing the CEA levels with respect to grade of tumor differentiation

Table 9: Shows the Surgical Procedures Performed

SURGERIES PERFORMED	NUMBER OF CASES
RIGHT HEMICOLECTOMY	22
RIGHT EXTENDED HEMICOLECTOMY	13
LEFT HEMICOLECTOMY	7
LEFT EXTENDED HEMICOLECTOMY	3
RECTOSIGMOOID RESECTION	2
HARTMANN'S PROCEDURE	2
PAUL MICKULICZ	1



Bar chart shows the number of surgical procedures performed in the cases studied

Table 10: Shows the Number of Emergency Surgeries Performed

SURGERIES PERFORMED	NUMBER OF CASES
ELECTIVE	47
EMERGENCY	**3**

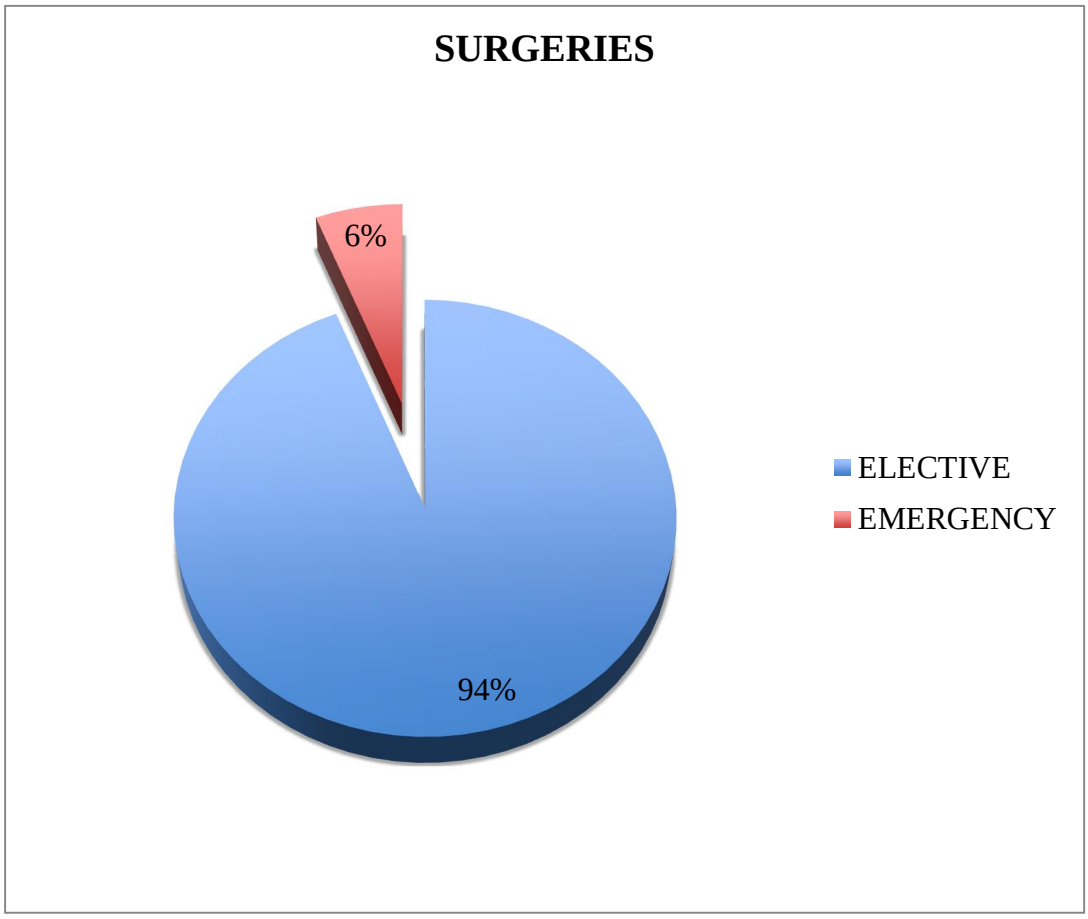
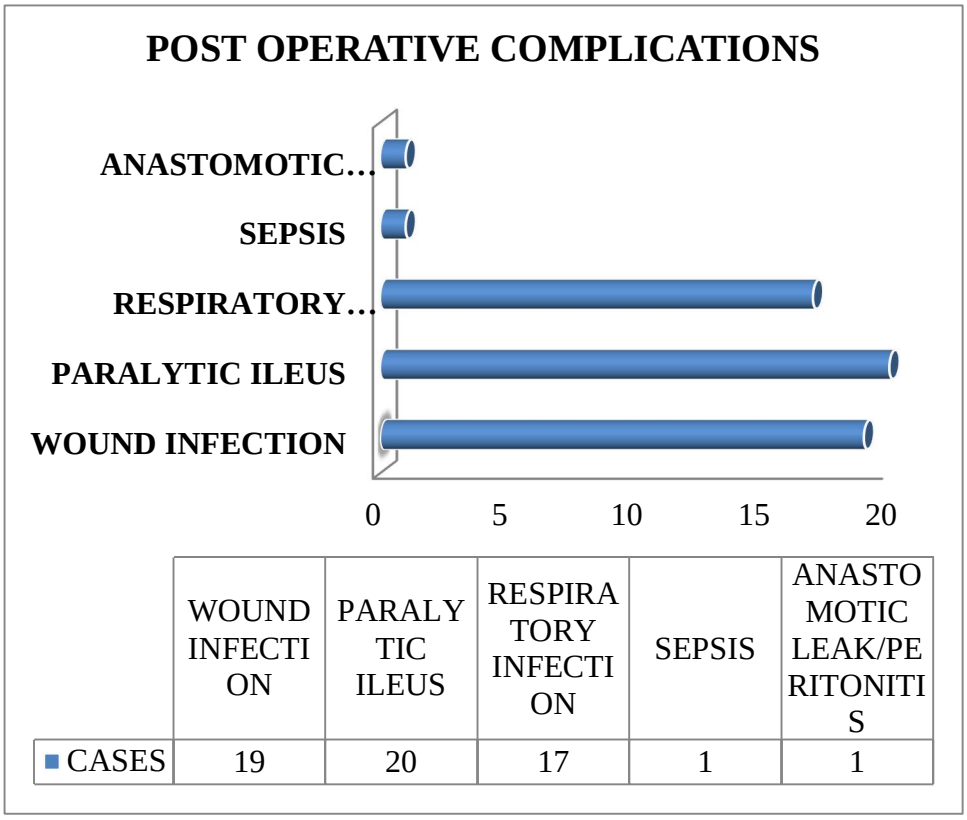


Table 11: Shows the Incidence of Postoperative Complications

POSTOPERATIVE COMPLICATIONS	NUMBER OF CASES
WOUND INFECTION	19
PARALYTIC ILEUS	20
RESPIRATORY INFECTION	17
SEPSIS	1
ANASTOMOTIC LEAK/PERITONITIS	1



Bar Diagram showing the common postoperative complications encountered in the cases studied

Table 12: Shows Stage Specific Incidence of Colon Cancers

TUMOUR STAGE	NUMBER OF CASES
I	2
II	34
III	10
IV	4

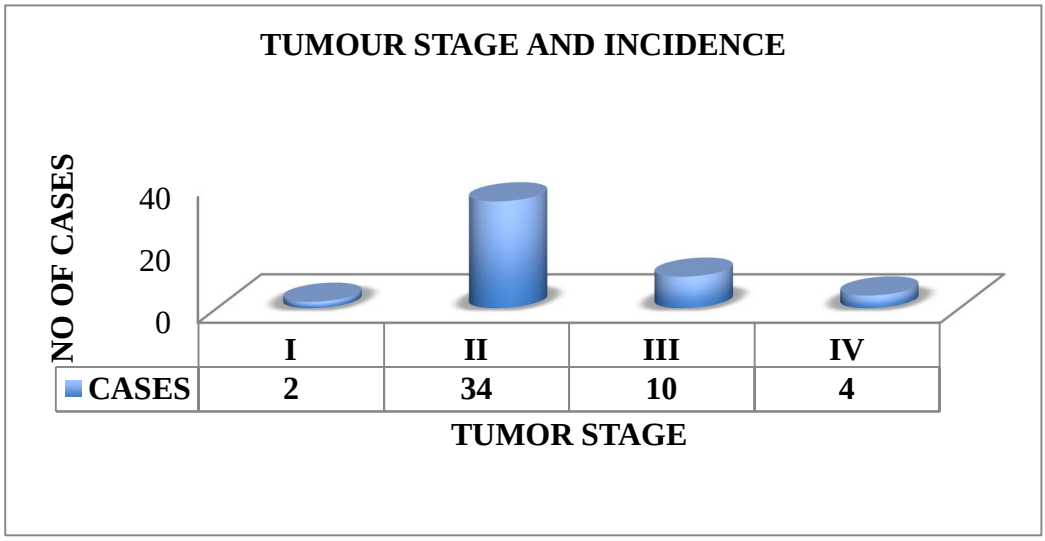


Table 13: Duration of Postoperative Stay

POSTOPERATIVE DURATION OF STAY (DAYS)	NUMBER OF CASES
<10	31
10 TO 15	15
>15	4

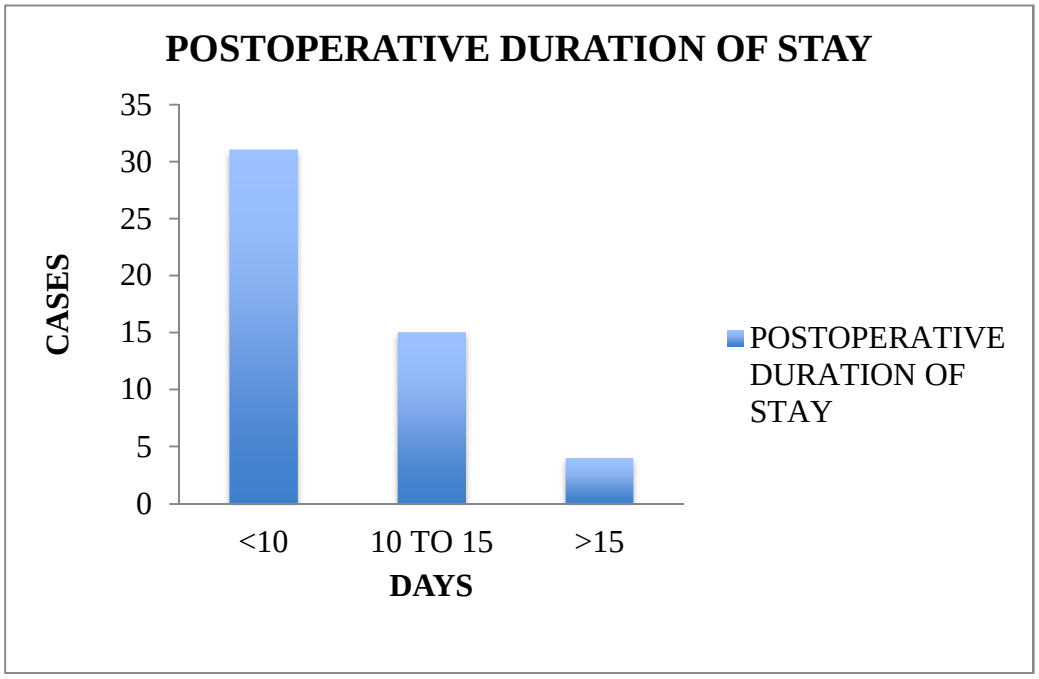
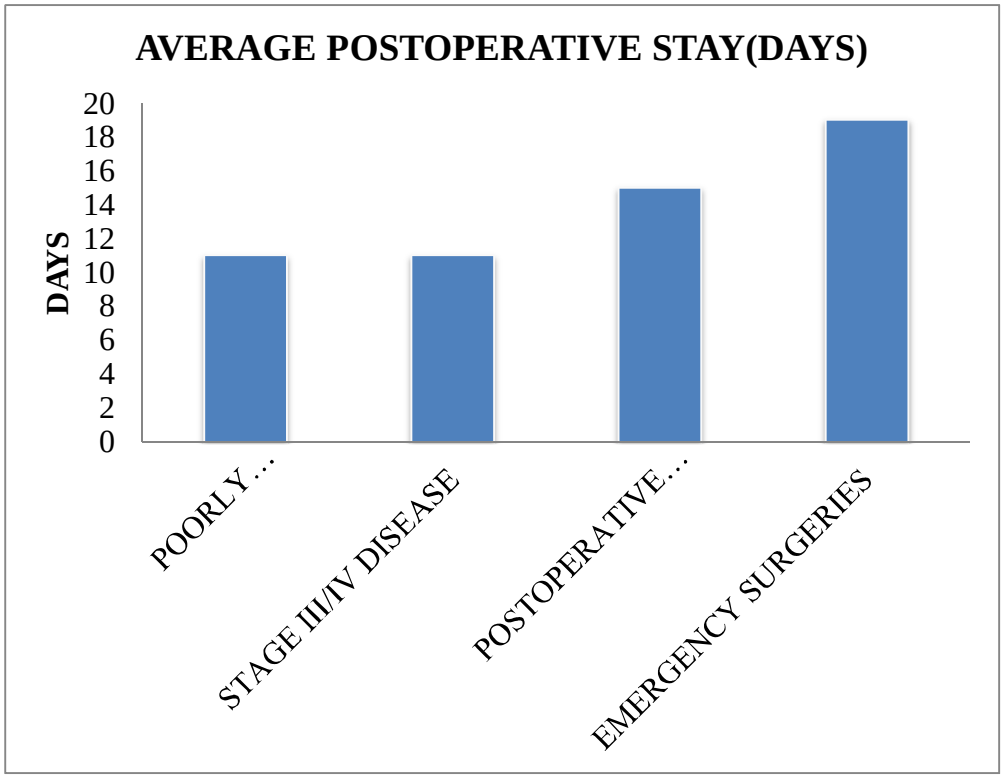


Table 14: Shows the Factors Affecting Postoperative Stay

CASES AND CONSIDERATIONS	AVERAGE POSTOPERATIVE STAY (DAYS)
POORLY DIFFERENTIATED TUMORS	11
STAGE III/IV DISEASE	11
POSTOPERATIVE WOUND INFECTIONS	15
EMERGENCY SURGERIES	19



Bar Chart shows various factors affecting the duration of postoperative stay

Table 15: Shows Preoperative CEA Levels

CEA LEVELS (ng/ml)	PRE-OPERATIVE CASES	POST-OPERATIVE CASES
<2.5	3	9
2.5 TO 5.5	5	28
5.5 to 7.5	13	11
>7.5	29	0

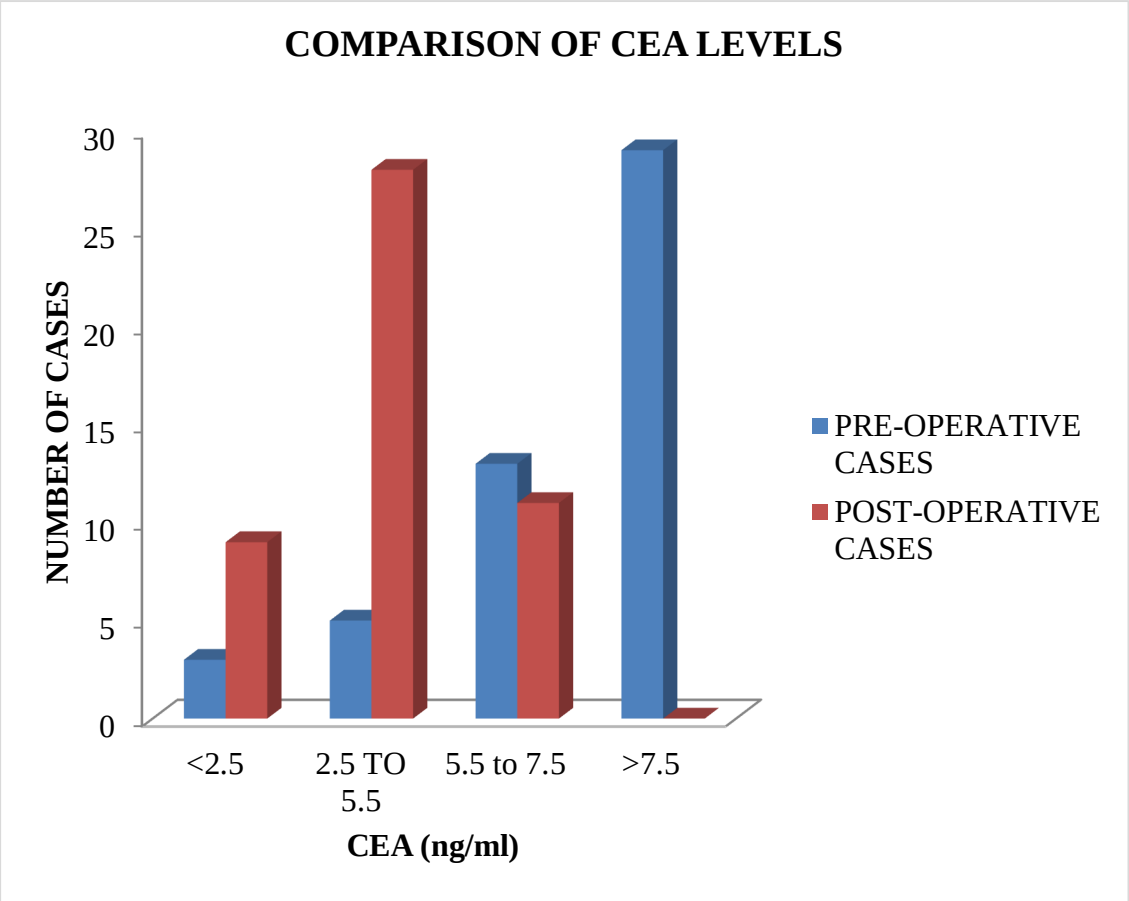


Table 16: Shows Stage Specific Recurrence in Advanced Tumors

TUMOR STAGE	RECURRENCE
STAGE I	0
STAGE II	0
STAGE III	2
STAGE IV	1

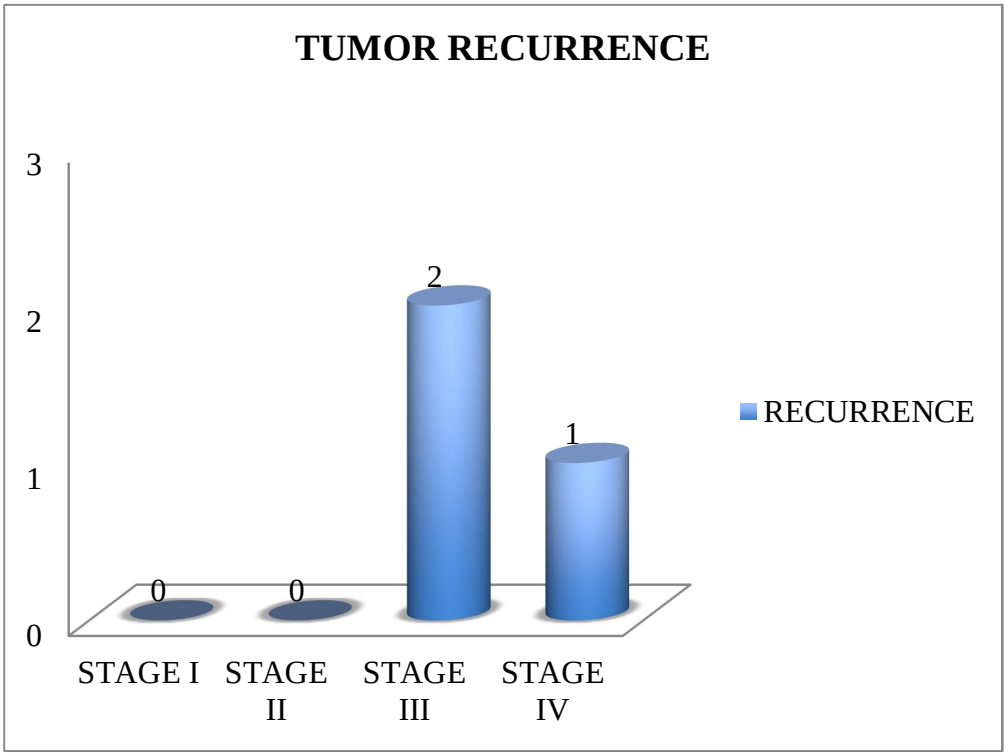


Table 17: Shows Comparison of CEA Levels in Recurrent Cases

	PREOPERATIVE	POSTOPERATIVE	3RD MONTH	6TH MONTH
CASE 1	2.7	2.5	6.7	18.8
CASE 2	2.8	2.6	8.6	24.8
CASE 3	26.3	4.8	9.7	20.6

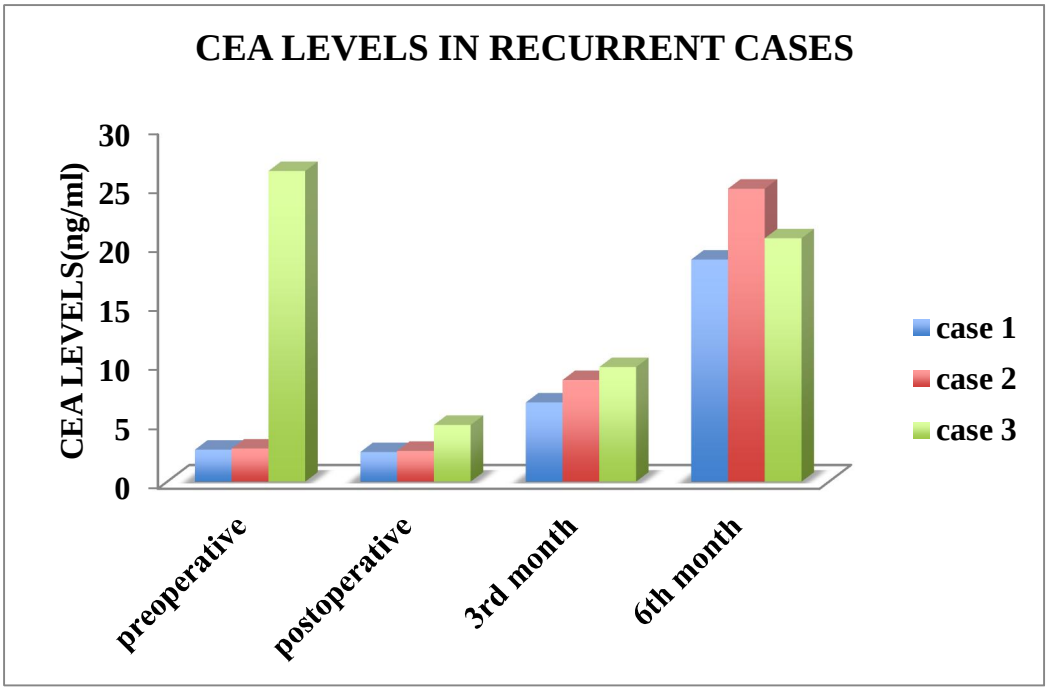
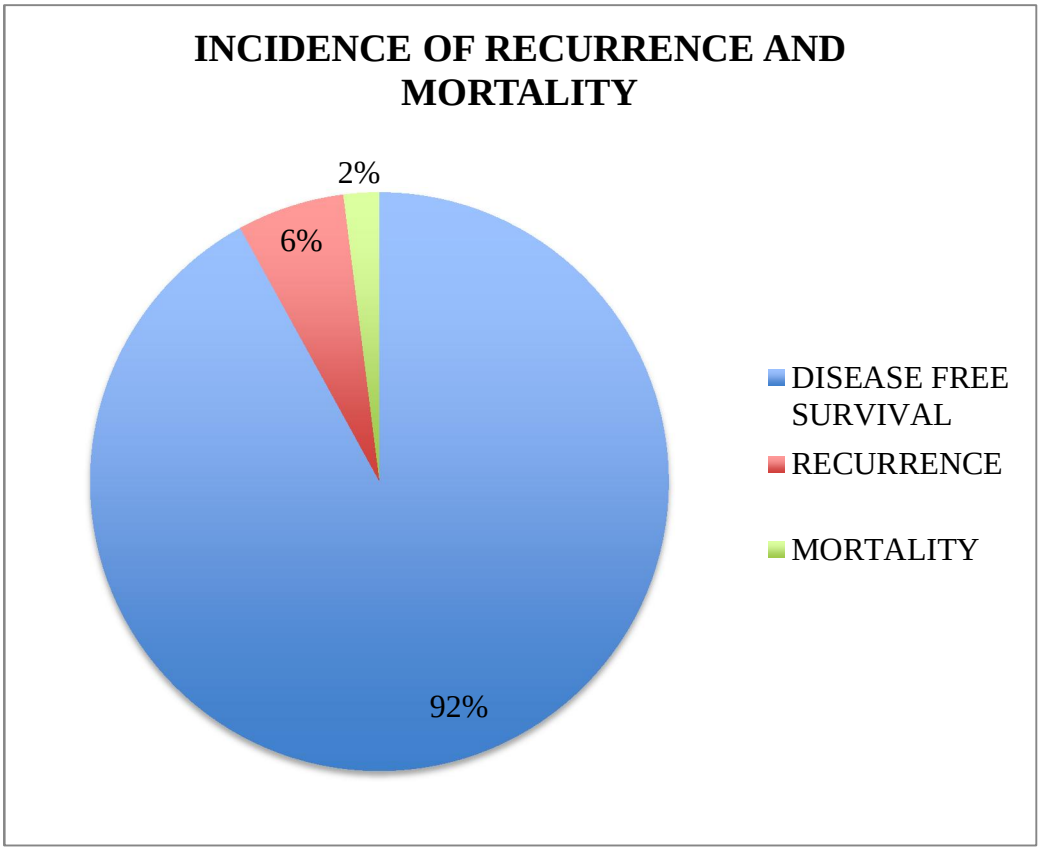


Table 18: Shows the Incidence of Recurrence and Mortality

DISEASE FREE SURVIVAL	RECURRENCE	MORTALITY
46	3	1



DISCUSSION

After analyzing the statistical data of the study, we could arrive at the following valuable inferences with respect to the colonic malignancies in our hospital setup.

AGE DISTRIBUTION

Majority of the cases happened to fall in the 5^{th} and 6^{th} decade group. Of the 50 cases 36 patients (72%) presented with colon cancers in their fifth to sixth decade of life, this is in conformity to the global incidence of tumor in this age group.

SEX DISTRIBUTION

The incidence of right colonic malignancies (70%) was more than that of the left colonic malignancies (30%) in both sexes. The colonic malignancies were found to be more common in males (66%) when compared to females (34%).

PRESENTING COMPLAINTS

The right colon cancers presented with anemia (n=26), mass abdomen (n=22) and loss of weight (n=28). The left colon cancers presented predominantly with altered bowel symptoms either constipation or loose stools (n=25).

DURATION OF PRESENTING COMPLAINTS

It is a known fact that the presentation of the right colonic malignancies is delayed when compared to left colonic tumors due to the gross pathology of the tumor being predominantly ulcero-proliferative on the right side whereas it is

predominantly circumferential in the left side, also being compounded by the stool consistency. My study revealed that the average duration of complaints for the right colonic malignancies is 5 months whereas it is 2 months for left colonic malignancies.

COLONOSCOPIC APPEARANCE OF TUMOR AND LOCATION

The right colon tumors were predominantly ulcero-proliferative whereas the left colon tumors mostly presented as a circumferential growth, thus explaining the longer duration of symptomatology as evidenced above. 70% of the cases (n=35) were right-sided tumors (located in caecum, ascending colon, hepatic flexure or proximal transverse colon) whereas the left sided tumors that of splenic flexure, descending colon and sigmoid colon were seen only in 30% of cases (n=15).

TUMOR DIFFERENTIATION AND LYMPHNODE STATUS

Of the 14 cases that showed evidence of lymph node in computerized tomography 9 were found to be poorly differentiated adenocarcinoma, 4 were moderately differentiated tumors and only one was well differentiated. Thus implying the important association of lymph node spread with respect to tumor differentiation, poorer the tumor differentiation more is the chance of lymphatic spread and hence worse prognosis.

CORRELATION BETWEEN CEA AND TUMOR DIFFERENTIATION

All cases had were evaluated for CEA (carcino-embryonic antigen) preoperatively and compared with diffentiation of tumor following the

histopathological report of colonoscopic biopsy. It was found that all well differentiated tumors had CEA levels ranging from 15 to 25ng/ml, in the range of 5 to 15ng/ml for moderately differentiated tumors and the poorly differentiated tumors had CEA levels in the range less than 5ng/ml. Thus we infer that the CEA levels are inversely related to the differentiation of the tumor. One other important finding is that all the tumors with liver secondaries had high levels of CEA (>25ng/ml).

SURGERIES PERFORMED

The incidence of right colon cancers being more, the surgical procedures like right hemi-colectomy and exented right hemi-colectomy were predominantly done when compared to left hemi-colectomy, left extended hemi-colectomy or recto-sigmoid resection.

Of the 50 cases of carcinoma colon, 3 cases presented as emergencies (2 cases of obstruction and 1 was colo-vesical fistula). Incidentally all cases that presented with obstruction were sigmoid colonic malignancies. Hartmann's procedure was done for cases of obstruction and modified supralevator pelvic exenteration with end colostomy was done for colo-vesical fistula.

POSTOPERATIVE COMPLICATIONS

The common complications were paralytic ileus (n=20), wound infection (n=19), respiratory infection (n=17), sepsis (n=1) and anastomotic leak (n=1) in that order of frequency. Although respiratory and wound infections have other variables (comorbid illness like diabetes, cancer cachexia, postoperative wound

management) to be factored in, around 40% of patients with wound infection had longer postoperative stay.

TUMOR STAGE

After the pathologic analysis of the resected specimen accurate details with respect to depth of tumor spread and the node positivity, stage of the disease was ascertained. Most of the cases (n=34) studied were of stage II disease and 10 cases were of stage III disease. Thus 72% of cases were of stage II and stage I.

DURATION OF POSTOPERATIVE STAY

The average duration of postoperative stay was 11 days. However average stay in patients with postoperative wound infection was 15 days. Both the tumor stage and differentiation had no bearing on the duration of postoperative stay.

PREOPERATIVE VS POSTOPERATIVE CEA LEVELS

The CEA levels were measured on the 14^{th} postoperative day and compared with the preoperative levels. The preoperative CEA levels were on an average above 5.5ng/ml in 84%(n=42) of cases. However the values returned to value below the baseline value of 5.5ng/ml in 74%(n=37) postoperatively signifying a tumor free state. This fall in CEA levels postoperatively is an important predictor of the surgical outcome.

RECURRENCE AND MORTALITY

Of the 50 cases operated (47 elective surgeries and 3 emergency surgeries), 3 cases of stage III/IV disease reported with recurrence in one year and there was 1 case of death due to septicemia in the case operated as an emergency. The CEA

levels in the recurrent cases were found elevated during follow-up. Thus it could be inferred that the mortality rate following a tumor complicating emergency surgery is high (30%) when compared to elective surgeries. Also the elevation of CEA in tumor recurrence signifies the importance of regular follow up of all cases postoperatively with serial monitoring of CEA levels (once in every 3 months for one year followed by every 6 months).

CONCLUSION

Based on the statistical analysis of the study it was inferred that the incidence of colon cancers was common in 5^{th} to 6^{th} decade of life and right colon cancers where commoner than left colon cancers. Colon cancers are more common in males than females. Results with respect to the symptom complex and duration of presenting complaints were confirming to the accepted knowledge about the right and left colon cancers. The lesions were predominantly ulcero-proliferative in right colon cancers but were predominantly circumferential in left colon cancers. It is found that there is a strong correlation between the lymph nodal involvement and tumor differentiation, poorly differentiated tumor has more propensity to cause lymph node spread early hence worse prognosis. The diffentiation of tumor also has inverse correlation with respect to CEA levels; higher the levels of CEA well differentiated will be the tumor grade. The common postoperative complications were paralytic ileus, wound infection and respiratory infection and the duration of postoperative stay was not related to the tumor grade or its stage but the presence or absence of wound infection. Postoperative CEA levels measured after 2 weeks is useful in assessing the surgical outcome as a successful surgery brings the value to baseline. Recurrent tumors had elevated CEA well above their immediate postoperative levels, thus implying the importance of postoperative follow-up of all cases operated for colon cancers.

Lastly the mortality associated with tumor complicating emergency surgeries is high when compared to electively planned surgeries.

Early diagnosis of colon cancers can be curative as more than 90% of stage I/II tumors have longer disease free survival rate than the stage III/IV disease.

PROFORMA

NAME: **AGE:** **SEX:**

ADDRESS: **UNIT:**

I.P NO:

OCCUPATION:

D.O.A: **D.O.S** **D.O.D:**

PRESENTING COMPLAINTS:

DURATION OF COMPLAINTS:

PAST HISTORY:

PERSONAL HISTORY:

PHYSICAL EXAMINATION:

Consciousness
Orientation
Hydration status:
Anemia:
Jaundice:
Vitals:
CVS:
RS:
Abdomen:
CNS:
P/R
External genitalia:

INVESTIGATIONS:
Colonoscopy
Computerized tomography

Blood parameters:
Renal function test
Serum electrolytes

Complete haemogram
Liver function tests
CEA levels
Stool for occult blood
X RAY chest

OPERATIVE PROCEDURE:

TUMOR STAGE:

POST OPERATIVE PERIOD:
Fever
Wound infection
Wound dehiscence
Respiratory tract infection
Paralytic ileus
Sepsis
Anastomotic leakage/peritonitis

DURATION OF STAY IN HOSPITAL:

POSTOPERATIVE CEA (POD 14)

ABBREVIATIONS

M	-	MALE
F	-	FEMALE
A	-	ANEMIA
M	-	MASS
P	-	PAIN
D	-	DURATION OF PRESENTING COMPLAINTS IN MONTHS
P	-	POLYPOIDAL
C	-	CIRCUMFERNTIAL
U	-	ULCERATIVE
N	-	NO
R	-	RECURRENCE
D	-	DEATH
LS	-	LOOSE STOOLS
UP	-	ULCERO PROLIFERATIVE
AC	-	ASCENDING COLON
HF	-	HEPATIC FLEXURE
TC	-	TRANSVERSE COLON
SF	-	SPLENIC FLEXURE
DC	-	DESCENDING COLON
SC	-	SIGMOID COLON
LN	-	LYMPH NODE
WD	-	WELL DIFFERENTIATED
MD	-	MODERATELY DIFFERENTIATED
PD	-	POORLY DIFFERENTIATED
RH	-	RIGHT HEMICOLECTOMY
LH	-	LEFT HEMICOLECTOMY
LR	-	LIVER RESECTION
PI	-	PARALYTIC ILEUS
WI	-	WOUND INFECTIO
RI	-	RESPIRATORY INFECTION
MLN	-	MALENA
BPR	-	BLEEDING PER RECTUM
LOW	-	LOSS OF WEIGHT
LOA	-	LOSS OF APPETITE
REH	-	RIGHT EXTENDED HEMICOLECTOMY
LEH	-	LEFT EXTENDED HEMICOLECTOMY
RSR	-	RECTOSIGMOID RESECTION
POC	-	POST OPERATIVE COMPLICATIONS
CVF	-	COLOVESICAL FISTULA
CONST	-	CONSTIPATION
METS	-	METASTASIS TO LIVER
OBST	-	OBSTRUCTION
PODD	-	POST OPERATIVE DAY OF DISCHARGE.

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MASTER CHART

NO	NAME	AGE	SEX	IP NO	COMPLAINTS	D	TYPE/ SITE	LN/METS	GRADE	EMERGENCY	SURGERY	TUMOR STAGE	POC	POD D	CEA (ng/ml)				R / D
															Pre	Post	3m	6m	
1	JEYAVEL	60	M	101990	A, LOW/LOA	4	UP/HF	NIL	MD	N	REH	II	NIL	9	5.6	2.1	2.0	2.3	N
2	MOTHILAKSHMI	49	F	119517	A, M, P, LOW/LOA	6	UP/AC	NIL	WD	N	RH	II	NIL	9	16.5	2.3	2.5	2.4	N
3	RAJESH	28	M	21395	MLN, BPR, P, LOW/LOA	3	UP/TC	LN+	PD	N	RH	III	PI, WI	15	2.4	2.0	2.2	2.1	N
4	GOVINDA RAJ	48	M	111399	A, M, CONST, LOW/LOA	8	UP/TC	NL	MD	N	REH	II	NIL	9	7.5	4.2	3.1	2.2	N
5	RAVISHANKAR	47	M	112143	A, BPR, CONST, LS	2	C/DC	LN+/ M+	MD	N	LH&LR	IV	PI, WI, RI	14	31.3	5.6	4.7	4.1	N
6	ANTONY PRAKASH	44	M	109328	A, BPR, CONST, LS, LOW/LOA	5	C/SC	NIL	WD	N	RSR	II	WI	11	16.5	4.6	3.5	2.8	N
7	RAVI	51	M	107559	A, M, MLN, LOW	12	UP/AC	NIL	MD	N	RH	II	NIL	9	8.5	4.8	4.4	3.2	N
8	SASIKUMAR	38	M	103891	A, LOW/LOA	3	UP/AC	LN+/ M+	PD	N	RH&LR	IV	PI, RI	13	22.3	2.1	2.2	2.0	N
9	JAYAPANDIAN	64	M	102779	A, M, LOW/LOA	6	UP/HF	NIL	MD	N	REH	II	NIL	9	8.6	5.8	3.8	1.9	N
10	KANAGAVALLI	46	F	996917	M, P, LOW/LOA	8	UP/AC	NIL	WD	N	RH	II	PI	9	18.9	5.6	4.6	2.7	N
11	VAITHIYALINGAM	40	M	996213	MLN, LS, LOW/LOA	5	UP/SF	NIL	MD	N	LEH	II	RI, WI	18	9.6	6.8	4.3	3.1	N
12	SAYED ABBAS	64	M	994163	A, P, LOW/LOA	12	UP/HF	LN+	PD	N	REH	III	PI	9	2.4	2.2	2.1	1.8	N
13	JAMAL	40	M	989468	M, LOA/LOW	9	U/CAECUM	NIL	WD	N	RH	II	RI	9	16.6	4.2	3.5	2.7	N
14	VELU	60	M	989250	A, CONST, BPR, LOW/LOA	4	UP/TC	NIL	MD	N	REH	II	WI, PI	12	7.2	5.5	4.0	2.6	N
15	VISALAKSHI	64	F	995366	A, M, MLN, LOA	6	U/AC	NIL	WD	N	RH	II	RI	9	19.3	4.8	3.5	2.6	N

16	BABU	50	M	981524	A, M, P, LOW/LOA	4	UP/AC	NIL	MD	N	RH	II	PI	9	7.2	4.8	3.2	2.1	N
17	KARUNA KARAN	59	M	983432	M, CONST, LOW/LOA	5	UP/AC	LN+	PD	N	RH	III	WI, RI	15	2.7	2.5	6.7	18.8	R
18	BABU	56	M	986415	M, LOW/LOA	4	P/HF	NIL	MD	N	REH	I	PI	9	6.2	2.8	2.5	1.8	N
19	KRISHNAN	70	M	977546	M, MLN, LOA	6	UP/HF	NIL	WD	N	RH	II	WI, RI	14	17.7	5.3	4.1	3.6	N
20	SRINIVASAN	42	M	972841	A, M, LOA	5	UP/AC	NIL	MD	N	RH	II	PI	9	6.3	4.7	3.2	2.5	N
21	RAJA	53	M	113267	M, P, MLN, LOA	6	P/AC	NIL	MD	N	RH	II	WI, RI	12	7.8	3.5	2.7	2.1	N
22	MUNUSAMY	48	M	11495	P, BPR, LS, LOW/LOA	2	UP/SC	NIL	MD	CVF	HP	II	WI, P	26	8.9	4.4	3.5	2.5	N
23	KALAISELVI	58	F	12610	A, M, CONST, BPR, LOW/LOA	3	UP/SF	LN+	PD	N	LEH	III	PI	9	2.8	2.6	8.6	24.8	R
24	SHANTHI	50	F	15058	CONST, LS, LOW/LOA	1	C/DC	LN+/ M+	MD	N	LH&LR	IV	WI	16	26.3	4.8	9.7	20.6	R
25	ALLIMUTHU	50	M	17179	A, M, LOW/LOA	6	UP/HF	NIL	MD	N	REH	II	PI, RI	9	5.9	4.3	3.5	1.8	N
26	CHANDRA	43	F	17327	CONST, LS, BPR, LOA	2	C/SC	LN+/ M+	PD	OBST	HP	IV	WI,RI, SEPSIS	10	32.8				D
27	SAKUNTALA	55	F	17620	A, LOW/LOA	8	UP/AC	NIL	MD	N	RH	II	PI	9	6.5	4.9	3.5	2.1	N
28	GOVINDASAMY	65	M	21748	MLN, CONST, LS, LOW/LOA	2	C/SC	LN+	PD	N	RSR	III	NIL	9	2.8	2.1	2.2	1.8	N
29	MARIMUTHU	55	M	110163	A, MLN, LOW/LOA	4	UP/HF	NIL	MD	N	REH	II	WI	12	7.2	4.7	4.1	2.6	N
30	RAJA	53	M	113267	A, LOW	8	P/AC	NIL	MD	N	RH	II	NIL	9	8.6	5.1	3.5	2.2	N
31	DURAIRAJ	40	M	105746	A, M, MLN, LOW/LOA	7	UP/AC	NIL	WD	N	RH	II	PI, RI	9	16.2	4.8	3.1	1.8	N
32	RAJESHWARI	35	F	109662	A, M, LOW/LOA	9	UP/HF	LN+	MD	N	REH	III	WI	12	8.8	4.7	2.7	1.7	N
33	SHANTHI	45	F	111381	M, LS, CONST, BPR, LOW	3	UP/SF	NIL	WD	N	LEH	II	PI	9	16.8	5.2	3.6	2.1	N

34	VENKAT	44	M	114742	LS, BPR, CONST	2	C/SC	NIL	WD	OBST	PMP	II	PI, WI, RI	20	19.8	5.8	3.7	2.5	N
35	DEVI	35	F	113915	A, MLN, BPR, LOW/LOA	5	UP/TC	NIL	MD	N	REH	II	NIL	9	6.2	4.3	2.8	2.1	N
36	RAGHINI	51	F	991978	A, M, LOW/LOA	9	UP/CAECUM	NIL	WD	N	RH	II	PI	9	15.5	5.0	3.2	1.7	N
37	THIMMA RAYAN	52	M	12609	CONST, LS, BPR, LOW/LOA	2	C/DC	LN+	MD	N	LH	III	WI, RI	14	8.2	5.2	3.8	2.2	N
38	SHANKAR	38	M	114078	A, M, CONST, BPR, MLN	4	C/TC	LN+	WD	N	REH	III	NIL	9	18.3	4.8	4.4	2.8	N
39	MANI	48	M	113875	A, LOW/LOA	6	UP/HF	NIL	MD	N	REH	II	WI	12	6.3	4.2	4.1	2.7	N
40	FATHIMA	40	F	112140	A, CONST, LOW/LOA	3	C/DC	LN+	PD	N	LH	III	NIL	9	2.2	2.0	2.1	1.8	N
41	SUGUNA	58	F	112876	LS, MLN, LOW	3	UP/SF	NIL	MD	N	LEH	II	PI, RI	9	8.3	4.7	3.5	2.8	N
42	SUMATHI	50	F	168738	A, M, LOA	6	UP/AC	NIL	WD	N	RH	II	NIL	9	16.5	4.8	4.1	3.2	N
43	VASANTH	47	M	106376	A, M, CONST, LOW/LOA	4	C/TC	NIL	WD	N	REH	II	WI, RI	14	18.8	5.2	4.5	2.8	N
44	ELUMALAI	43	M	105783	LS, CONST, BPR, MLN, LOW	2	UP/DC	NIL	MD	N	LH	II	PI	9	5.8	4.2	3.6	1.7	N
45	MEGHALA	60	F	104378	A, MLN, LOW/LOA	8	U/AC	NIL	PD	N	RH	II	NIL	9	2.8	2.2	2.3	1.8	N
46	LALITHA	46	F	103876	M, CONST, MLN, LOW/LOA	7	UP/TC	NIL	WD	N	RH	II	PI	9	21.1	4.9	3.1	2.9	N
47	MOHAN	59	M	137865	A, M, LOW/LOA	11	U/CAECUM	NIL	WD	N	RH	II	NIL	9	18.6	5.0	4.2	2.5	N
48	MURUGESAN	55	M	137811	MLN.LOW/LOA	10	P/AC	NIL	WD	N	RH	I	WI, RI	12	16.8	4.8	3.5	1.5	N
49	LOGAIYAN	50	M	123521	LS, CONST, LOW/LOA	3	C/DC	NIL	MD	N	LH	II	PI, WI, RI	14	7.2	4.1	2.8	2.1	N
50	MUNIYAMMA	46	F	107368	A, M, LS, LOW/LOA	5	UP/AC	LN+	PD	N	RH	III	NIL	9	2.6	2.2	2.1	1.7	N