DISSERTATION ON

"STUDY ON COLONOSCOPIC FINDINDS IN POSITIVE FAECAL OCCULT BLOOD TESTING"

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations

for the award of the degree of

M.S. IN GENERAL SURGERY

BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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APRIL -201

CERTIFICATE

This is to certify that this dissertation entitled "STUDY ON COLONOSCOPIC FINDINGS IN POSITIVE FAECAL OCCULT BLOOD TESTING" is the bonafide work of **Dr.S.KAPIL RAJ** in partial fulfilment of the requirements for M.S Branch -I (General Surgery) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2015 under my guidance and supervision during the academic year january- 2013 to july - 2014.

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DECLARATION

I, **Dr.S.KAPIL RAJ**, solemnly declare that the dissertation titled **"STUDY ON COLONOSCOPIC FINDINGS IN POSITIVE FAECAL OCCULT BLOOD TESTING"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during January - 2013 to July - 2014 under the guidance and supervision of **Prof. Dr. R. YEGANATHAN M.S. D.A.,** Unit Chief S-III, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.S. degree (Branch -I) in General Surgery**.

Place: Thanjavur.

Date: **(Dr.S.KAPIL RAJ)**

ACKNOWLEDGEMENT

I gratefully acknowledge my sincere thanks to **Prof. Dr. K.Mahadevan M.S.,** Dean, Thanjavur Medical College, Thanjavur, for allowing me to do this dissertation and utilize the institutional facilities.

I am extremely grateful to **Prof. Dr. V. Balakrishnan M.S.,** Head of the Department, Department of General surgery, Thanjavur Medical College, for his full-fledged support throughout my study and for his valuable suggestions and guidance during my study and my post graduation period.

I am greatly indebted to **Prof. Dr. R. Yeganathan M.S. D.A.,** my Professor and Unit Chief, who is my guide in this study, for his timely suggestions, constant encouragement and scholarly guidance in my study and in my post graduation period.

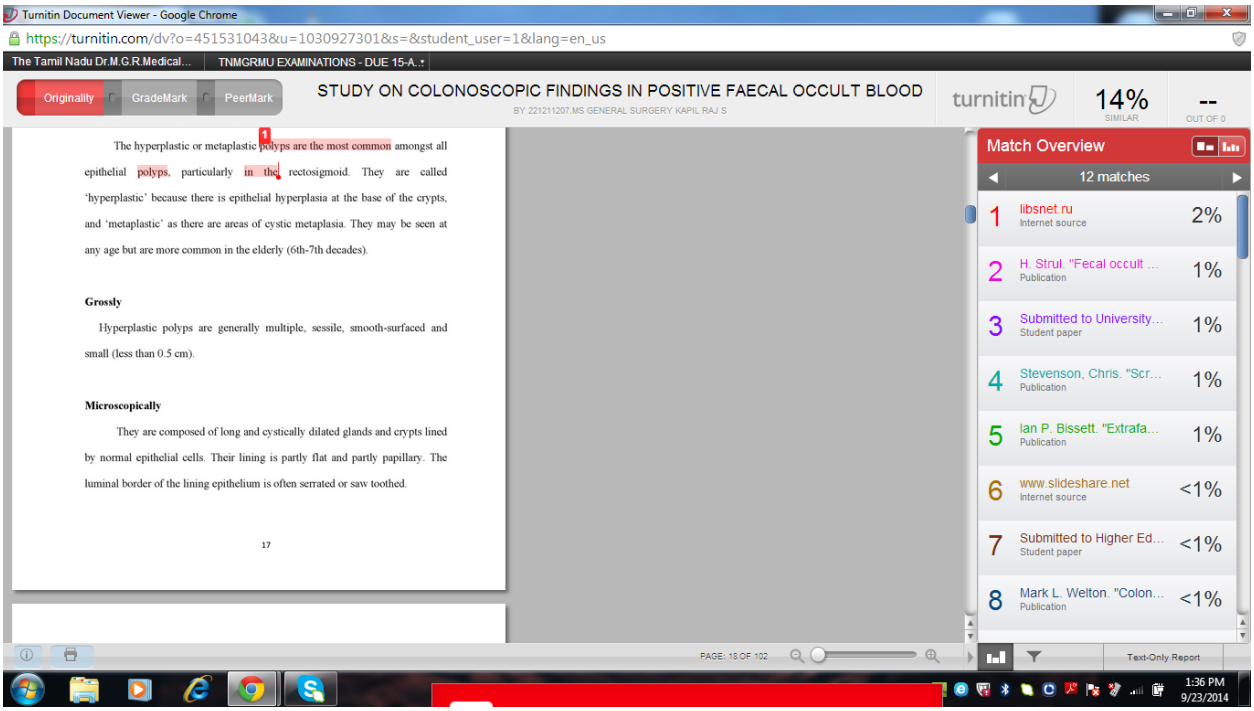
I profoundly thank my respected professors**, Prof. Dr. M.Elangovan MS., Prof. Dr.T.Karunaharan M.S., G.Rajendran MS.FICS.,** for their advice and valuable criticism which enabled me to do this work effectively.

A special mention of thanks to **Prof.Dr.U.Aravindhan MS.Mch.,** Head of department, surgical gastroenterology, **prof.Dr.R.Ganesan MD.DM.,** Head of department medical gastroenterology for their motivation, encouragement and support in completion of this study.

My sincere thanks to my assistant professors, **Dr.M.bala sundaram M.S.Mch,** and **Dr.S. Jeeva raman M.S.DLO., Dr.P.vanathi MS.,** for their motivation, encouragement and support.

A special thanks to all the patients who participated in this study for their kind cooperation.

I would like to thank my senior pgs and my colleagues and friends who have been a constant source of encouragement.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The hyperplastic or metaplastic polyps are the most common amongst all epithelial polyps, particularly in the rectosigmoid. They are called 'hyperplastic' because there is epithelial hyperplasia at the base of the crypts, and 'metaplastic' as there are areas of cystic metaplasia. They may be seen at any age but are more common in the elderly (6th-7th decades).
Grossly
Hyperplastic polyps are generally multiple, sessile, smooth-surfaced and small (less than 0.5 cm).
Microscopically
They are composed of long and cystically dilated glands and crypts lined by normal epithelial cells. Their lining is partly flat and partly papillary. The luminal border of the lining epithelium is often serrated or saw toothed.
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STUDY ON COLONOSCOPIC FINDINGS IN POSITIVE FAECAL OCCULT BLOOD TESTING

INTRODUCTION

Colorectal cancer is the third most common cancer in the world and a leading cause of cancer death in the Western world. There is an increase in incidence of colorectal carcinoma in India due to,economic shift from low income to middle income economy and increase in ageing population, life style and dietary factors.

AIMS OF STUDY

- To study colonoscopic findings in positive faecal occult blood test patients
- To study other screening modalities in colorectal carcinoma

MATERIALS AND METHODS

Patients admitted in various surgical units in thanjavur medical college hospital between January 2013 to july 2014 constitute the materials of this study. The exclusion criteria includes patients with complaints of bleeding PR, altered bowel habits , tenesmus , mucus discharge from PR, spurious diarrhea ,mass desending PR ,FOBT within last one year, sigmoidoscopy within last 3 to 5 years, colonoscopy within last 10 years.

The inclusion criteria includes patients with age of 50 and above, patients willing for further followup (invasive procedures like colonoscopy&UGI scopy)

A total of 200 patients were studied, patients with positive faecal occult blood test are included in this study, those who are negative for FOBT were adviced for follow up, one year later for another FOBT test.

RESULT

Only 52 patients were positive for FOBT out of 200 patients. In FOBT positive patients 12 patients are not willing for colonoscopy,40 patients went through colonoscopy .Of them, 6 had haemorrhoids, 1 had a polyp and a carcinoma each and one other patient had diverticulum rest of patients have normal colonoscopy.

CONCLUSION:

- Faecal occult blood test screening offers no benefit without appropriate follow up diagnostic tests and treatment.
- It is simple, safe and cost effective but is limited by lack of acceptability, compliance and adherence as well as poor sensitivity and specificity.

- In considering all the advantages and drawbacks of FOBT in colorectal cancer screening, we can conclude that this examination is certainly better than no testing at all.

INTRODUCTION

Colorectal cancer is the third most common cancer in the world and a leading cause of cancer death in the Western world. The life time risk of colorectal carcinoma was 6%-7%, incidence rate increases after the age of 50. .In India incidence rate in male-4.3/1,00,000 and female-3.4/1,00,000 ,Highest in korea-male-46/100000,female-25/100000.Datas from rural population based registries, shows incidence rate of colon cancer was very low in rural setting but incidence rate was disproportionately more in rural India .

Colorectal carcinogenesis is exceptionally suited for screening, since the adenoma–carcinoma sequence used in detection and removal of pre-cancerous lesions, and it is well established that patients who are maintained free of polypoidal adenoma by endoscopic polypectomy are generally kept cancer free. There is an increase in incidence of colorectal carcinoma in India due to,

- Economic shift from low income to middle income economy and increase in ageing population,
- life style and dietary factors,
- Increased use of screening modalities.

AIM OF STUDY

- To study colonoscopic findings in positive faecal occult blood test patients
- To study effectiveness of faecal occult blood test in colorectal carcinoma screening
- To study factors that affect low uptake of faecal occult blood test
- To study other screening modalities in colorectal carcinoma
- To study various treatment modalities and recent trends in colorectal carcinoma

REVIEW OF LITERATURE

EMBRYOLOGY

The caecum, ascending colon, and proximal third of the transverse colon are derived from the midgut. The rest of the large bowel, rectum, and upper part of the anal canal develops from the hindgut. The caecal swelling is the last part of the midgut to reenter the abdominal cavity. The caecal bud is originally present in the right upper quadrant, where it forms the caecum and the appendix and subsequently descends into the right iliac fossa, thereby forming the ascending colon and the hepatic flexure. Once the colon has reached its definitive position, the mesenteries of the ascending and descending colons fuse with the posterior abdominal wall, thus rendering them retroperitoneal structures. The transverse colon and the sigmoid colon retain their mesentery.

During the early developmental stage, the appendix is the caudal extension of the caecum and possesses the same caliber. The right wall of the caecum grows rapidly downward and begins to displace the appendix medially and closer to the ileocecal area.

The rectum and the proximal part of the anal canal (i.e., to the level of the pectinate line) are derived from the hindgut. The distal segment of the anal

canal develops from the proctoderm. The expanded lower part of the hindgut, known as the cloaca, is in direct contact with the surface ectoderm, the cloacal membrane. The allantois, which is a diverticulum of the yolk sac, opens on the ventral aspect of the cloaca. A sheet of mesenchymal tissue known as the urorectal septum grows caudally between the allantois and the cloaca and partitions the cloaca into an anterior portion, the primitive urogenital sinus, and a posterior part, the anorectal canal. The urorectal septum continues to grow caudally and fuses with the cloacal membrane, and this area of fusion represents the perineal body in adults. This fusion creates the anterior urogenital membrane and the posterior anorectal membrane. The blood supply of the anus can be explained by the different embryologic origins of its superior and inferior segments. The upper two thirds of the anal canal is derived from the endoderm and is therefore supplied by the artery of the hindgut, the inferior mesenteric artery, whereas the distal third of the anus is ectodermal in origin and is supplied by branches of the internal iliac artery.

ANATOMY

The large intestine extends from the ileocecal junction to the anal orifice and measures approximately 103 cm long. It can be distinguished from the small bowel by the following features:

(1) Presence of teniae, which are three bands of longitudinal muscle;

(2) The appendices epiploicae, which are fatty appendages projecting from the serosal surface of the bowel; and

(3) The haustra, which are sacculations caused by the longitudinal muscle's being shorter than the rest of the bowel wall.

The components of large intestine include the cecum, appendix, ascending colon, transverse colon, descending colon, sigmoid colon, rectum, and anal canal.

Cecum

The cecum is a dilated pouch measuring about 6 to 8 cm, below the level of the ileocecal valve. It is situated in the right lower quadrant lying on the iliacus and psoas muscles. It is completely invested with peritoneum, which allows it to be mobile. The three teniae of the cecum converge on the posteromedial aspect at the base of the appendix. The tip of the appendix is often lying in the pelvis but can also be found behind the cecum (retrocecal fossa). The ileocecal valve is an oval opening that is present on the medial aspect of the cecum. The circular muscle of the distal ileum acts as the sphincter. The arterial blood supply of the cecum is derived from anterior and

posterior cecal arteries, which are branches of the ileocolic artery. The venous drainage corresponds to the arterial supply and drains into the superior mesenteric vein. The lymphatic drainage is via nodes that follow the vessels.

Appendix

The appendix varies in length from 8 to 14 cm and arises from the posteromedial surface of the cecum, where all three taeniae coli meet. The position of the appendix can vary considerably among patients. It can be located in the pelvis, behind or along the lateral border of the cecum, or anterior or posterior to the distal ileum. The appendix possesses a complete peritoneal covering and has its own mesoappendix, which is attached to the mesentery of the distal ileum. Contained within the mesoappendix is the appendicular artery, which is a branch of the posterior cecal artery. Venous drainage of the appendix is via the appendicular vein, which drains into the posterior cecal vein. The nerve supply of the appendix is derived from both sympathetic and vagal fibers. Visceral pain from the appendix is conducted by the afferent sympathetic fibers that enter at the T10 spinal level.

Ascending Colon

The ascending colon (15 to 20 cm long) extends upward from the cecum and turns sharply to the left to form the hepatic flexure and then becomes continuous with the transverse colon. The peritoneum lines the anterior and lateral surfaces of the ascending colon, whereas the posterior surface lies against the posterior abdominal wall. Lateral to the ascending colon is the paracolic gutter, which leads to the subphrenic space superiorly. Posteriorly it lies on the iliacus, quadrates lumborum, and lower pole of the right kidney. It receives its arterial blood supply from the ileocolic and the right colic branches of the superior mesenteric artery. The veins correspond to the arteries and drain into the superior mesenteric vein. Lymphatic drainage is into nodes that lie along the above-named blood vessels

Transverse Colon

The transverse colon measures approximately 50 cm long and extends upward from the hepatic flexure to the left to form the splenic flexure, which is suspended from the diaphragm by the phrenicocolic ligament. It is attached by the transverse mesocolon to the anterior border of the pancreas on the posterior abdominal wall. The anterior leaf of the transverse mesocolon adheres to the greater omentum. Posteriorly the colon is related to the second part of

duodenum, head of the pancreas, loops of small bowel, and left kidney. Owing to the dual embryologic origin, the proximal two thirds of the transverse colon receives its blood supply from the middle colic artery, which runs within the transverse mesocolon and is the second branch of the superior mesenteric artery. The left colic artery, a branch of the inferior mesen-teric artery, supplies the distal one third of the transverse colon. The veins correspond to the arteries and drain into the superior and inferior mesenteric veins. The lymphatic drainage follows the colic blood supply.

Descending Colon

This segment of the colon is the narrowest and measures approximately 30 cm. It extends downward in the left lumbar region from the splenic flexure to the pelvic brim, where it continues as the sigmoid colon. Similar to the ascending colon, the peritoneum covers the anterolateral wall and forms the left paracolic gutter. Thus, the posterior surface of the descending colon is a retroperitoneal structure. Posterior relations include the lower pole of the left kidney, diaphragm, quadrates lumborum, iliacus, and psoas muscles. The arterial blood supply is derived from the left colic branch of the inferior mesenteric artery. The vein corresponds to the artery and drains into the inferior mesenteric vein. The lymphatic drainage follows the colic blood vessels.

Sigmoid Colon

This segment of the colon measures approximately 40 cm and extends from the pelvic brim to the third sacral vertebra. It is suspended by a V-shaped sigmoid mesocolon, the apex of which is situated over the left ureter where it crosses the pelvic brim. The left limb of the V is attached along the medial aspect of the external iliac artery, and the right limb runs from the bifurcation of the left common iliac artery downward to the third sacral vertebra. The arterial blood supply of the sigmoid colon is derived from sigmoid branches of the inferior mesenteric artery, and the venous drainage is into the inferior mesenteric vein. Lymph drainage is into nodes along the inferior left colic artery and subsequently to the inferior mesenteric nodes.

Rectum

The rectum extends from the sigmoid colon to the anal canal. It measures approximately 12 to 13 cm and begins in front of the first sacral vertebra and follows the hollow of the sacrum to end at the level of the tip of the coccyx. When viewed in the coronal view, the rectum deviates initially to the left and then returns to the midline. In the sagittal plane the rectum can be seen following the concavity of the sacrum and inferiorly widens to form the rectal ampulla and then passes downward and posteriorly to join the anal canal. The

rectum does not have a mesentery, but the peritoneum covers the lateral and anterior surfaces of the upper third of the rectum; only the anterior surface of the middle third and the lower third has no peritoneal covering.

Several fasciae of the rectum are of surgical importance. The visceral pelvic fascia (also known as rectal fascia propria) surrounds the mesorectum posteriorly. This fascial envelope encloses fat, blood vessels, nerves, and lymphatics of the rectum. Between the rectal fascia propria and the parietal (presacral) fascia is loose areolar tissue that is the plane of dissection when performing total mesorectal excision. The parietal fascia, which lines the pelvic walls, is known as the presacral fascia in the region of the sacrum. The presacral fascia is tightly adherent to concavity of the sacral periosteum, especially in the midline and around the anterior sacral foramina. The presacral fascia protects the presacral veins and the hypogastric plexus lying beneath it. The fascia of Waldeyer is a more membranous portion of the pelvic fascia that extends from the sacrum to the rectal ampulla. Just above the pelvic floor there is condensation of areolar tissue around the middle rectal vessels known as the lateral ligament of the rectum. Anteriorly, the Denonvilliers' fascia is adherent to the fascia propria of the rectum.

The blood supply of the rectum is from

- The superior rectal artery, which is a terminal branch of the inferior mesenteric artery
- The middle rectal artery, which is a branch of the internal iliac artery
- The inferior rectal artery, which is a branch of the internal pudendal artery

The venous drainage of the rectum follows that of the arteries,

- superior rectal vein draining into the portal circulation
- the middle and inferior rectal veins drain into the systemic circulation.
- The free communications between the rectal veins form an important portosystemic anastomosis.

The lymphatic drainage of the rectum is via lymphatics that travel along with the superior, middle, and inferior rectal arteries

rectouterine or rectovesical pouch:

Inferiorly rectum and surrounding tissue are separated by denonviller's fascia in the anterior part.posterior rectum and mesorectum are covered by waldeyer'sfascia. Circumferential areolar tissue below the peritoneum

reflection which carries blood supply and lymphatic drainage is known as mesorectum.

lymphatic drainage:

Lymphatic drainage is extensive comprise of epicolic nodes adjacent to colon. paracolic nodes alone the marginal vessels which form tier 1 and 2 nodes, Intermediate nodes along larger arteries and fourth tier nodes along and superior and inferior mesenteric artery which form the principle nodes .when tier 4 nodes are involved the disease is incurable. Rectum majority drain along inferior mesenteric artery .Lower rectum drains laterally along the middle and inferior rectal artery to internal iliac nodes.

nerve supply:

Sympathetic- Hypogastric nerve from hypogastric plexus. Parasympathetic- nervi erigentes from pelvic plexus. Preservation of autonomic nerves important during surgery to prevent impotence.

histology

Wall of colon comprises

Mucosa –surface epithelim [columnar] Lamina propria Muscularis mucosa

Submucosa

Muscular layer

Sub serosa

Serosa

physiology of colon:

About 1000ml of ileal contents containing 90% water are discharged into the caecum of which only 100-200ml of water is excreted in faeces. Normal faeces are composed of 70% water and 30% solids are bacteria. Nutrient such as glucose, aminoacids, fatty acids and vitamins can be absorbed slowly through the colonic wall. Sodium absorption is very efficient. Pottasium is actively excreted frequency of bowel movements ranges from once in 8 hrs to once in 2-3 days.

Motility

Motor activity of the colon occurs in three patterns, and there is marked regional variation between the right and left colon. A pacemaker in the transverse colon has been postulated, perhaps pacing the proximal colon retrograde to facilitate storage and absorption while pacing the distal colon in the anterograde to favor propulsion. **Retrograde peristalsis (antiperistalsis)—annular contractions** moving orad—dominates in the right colon. This kind of activity churns the contents and tends to confine them to the cecum and ascending colon. As ileal effluent continually enters the cecum, some of the

column of liquid stool in the right colon is displaced and flows into the transverse colon. **Segmentation** is the most common type of motor activity in the transverse and descending colon. Annular contractions divide the lumen into uniform segments, propelling feces over short distances in both directions. **Mass movement** is a strong ring contraction moving aborad over long distances in the transverse and descending colon. It occurs infrequently—perhaps only a few times daily—most commonly after meals.

gastro colic reflex:

It refers to increased ileal emptying, increased mass movements and urge to defecate on eating. In general residue from meal reaches caecum after 4 hrs and rectosigmoid by 24 hrs.

microbiology of colon:

Microbes exist in symbiotic relationship with human bacteria degrades bile pigments, gives characteristic faecal odour, supply vitamin K to the host. Indicated in pathogenesis of carcinoma of large bowel.99% of bacterial flora is anaerobic. Bacterioides fragilis is most prevalent anaerobic bacteria. Aerobic bacteria are mainly Escheichia coli and streptococcus fecalis. Bacteria flora is readily altered by administration of oral neomycin.

LARGE INTESTINAL POLYPS AND TUMOURS

Large bowel is the most common site for a variety of benign and malignant tumours, majority of which are of epithelial origin. Most of the benign tumours present clinically as polyps.

COLORECTAL POLYPS

A polyp is defined as any growth or mass protruding from the mucous membrane into the lumen. Polyps are much more common in the large intestine than in the small intestine and are more common in the rectosigmoid colon than the proximal colon. Polyps are broadly classified into 2 groups— non-neoplastic and neoplastic. Non-neoplastic polyps have further subtypes indicating their mode of origin. Neoplastic polyps, on the other hand, include epithelial tumours, both benign and malignant.

I. COLORECTAL POLYPS

A. Non-neoplastic polyps

1. Hyperplastic (metaplastic) polyps

2. Hamartomatous polyps

(i) Peutz-Jeghers polyps and polyposis

(ii) Juvenile (Retention) polyps and polyposis

3. Inflammatory polyps (Pseudopolyps)

4. Lymphoid polyps

B. Neoplastic polyps (Adenomas)

1. Tubular adenoma (Adenomatous polyp)

2. Villous adenoma (Villous papilloma)

3. Tubulovillous adenoma (Papillary adenoma, villoglandular adenoma)

C. Familial polyposis syndromes

1. Familial polyposis coli (Adenomatosis)

2. Gardner's syndrome

3. Turcot's syndrome

4. Juvenile polyposis syndrome

II. OTHER BENIGN COLORECTAL TUMOURS

(Leiomyomas, leiomyoblastoma, neurilemmoma, lipoma and Vascular tumours)

III. MALIGNANT COLORECTAL TUMOURS

A. Carcinoma

1. Adenocarcinoma

2. Other carcinomas

(Mucinous adenocarcinoma, signet-ring cell carcinoma, adenosquamous carcinoma, undifferentiated carcinoma)

B. Other malignant tumours

(Leiomyosarcoma, malignant lymphoma, carcinoid tumours)

A. NON-NEOPLASTIC POLYPS

Non-neoplastic polyps are more common and include the following 4 subtypes:

Hyperplastic (Metaplastic) Polyps :

The hyperplastic or metaplastic polyps are the most common amongst all epithelial polyps, particularly in the rectosigmoid. They are called 'hyperplastic' because there is epithelial hyperplasia at the base of the crypts, and 'metaplastic' as there are areas of cystic metaplasia. They may be seen at any age but are more common in the elderly (6th-7th decades).

Grossly

Hyperplastic polyps are generally multiple, sessile, smooth-surfaced and small (less than 0.5 cm).

Microscopically

They are composed of long and cystically dilated glands and crypts lined by normal epithelial cells. Their lining is partly flat and partly papillary. The luminal border of the lining epithelium is often serrated or saw toothed.

Hyperplastic polyps are usually symptomless and have no malignant potential unless there is a coexistent adenoma.

Hamartomatous Polyps

These are tumour-like lesions composed of abnormal mixture of tissues indigenous to the part.

They are further of 2 types:

PEUTZ-JEGHERS POLYPS AND POLYPOSIS:

Peutz- Jeghers syndrome is autosomal dominant defect, characterized by hamartomatous intestinal polyposis and melanotic pigmentation of lips, mouth and genitalia. The polyps may be located in the stomach, small intestine or colon but are most common in the jejunum and ileum. The most common age is adolescence and early childhood.

Grossly

These polyps are of variable size but are often large, multiple and pedunculated and more commonly situated in the small intestine.

Microscopically:

The most characteristic feature is the treelike branching of muscularis mucosae. The lining epithelium is by normal-appearing epithelial cells. The glands may show hyperplasia and cystic change. Peutz-Jeghers polyps do not undergo malignant transformation unless a coexistent adenoma is present. However, patients with Peutz-Jeghers syndrome are more prone to certain other cancers such as of pancreas, lung, breast, ovary and uterus.

JUVENILE (RETENTION) POLYPS.

Juvenile or retention polyps, another form of hamartomatous polyps, occur more commonly in children below 5 years of age. Solitary juvenile polyps occur more often in the rectum, while juvenile polyposis may be present anywhere in the large bowel.

Grossly

Juvenile polyps are spherical, smooth-surfaced, about 2 cm in diameter and are often pedunculated.

Microscopically

The classical appearance is of cystically dilated glands containing mucus and lined by normal mucus-secreting epithelium. The stroma may show inflammatory cell infiltrate if there is chronic ulceration of the surface. Most

cases, on becoming symptomatic in the form of rectal bleeding, are removed. In common with other non neoplastic polyps, they are also not precancerous.

Inflammatory Polyps (Pseudopolyps)

Inflammatory polyps or pseudopolyps appear due to reepithelialisation of the undermined ulcers and overhanging margins in inflammatory bowel disease, most frequently in ulcerative colitis (colitis polyposa) and sometimes in Crohn's disease.

Grossly

They are usually multiple, cylindrical to rounded overgrowths of mucosa and may vary from minute nodules to several centimeters in size.

Microscopically

The centre of inflammatory polyp consists of connective tissue core that shows some inflammatory cell infiltrate and is covered superficially by regenerating epithelial cells and some cystically-dilated glands. These lesions have no malignant potential; carcinomas seen in long-standing cases of ulcerative colitis arise in the region of epithelial dysplasia and not from the polyps.

Lymphoid Polyps

Reactive hyperplasia of lymphoid tissue that is normally also more prominent in the rectum and terminal ileum, gives riseto localised or diffuse lymphoid polyps, also called rectal tonsils. Localised form occurs more often in the rectum in elderly, while diffuse form is seen at younger age and in children.

Grossly

They are solitary or multiple, tiny elevated lesions.

Microscopically

They are composed of prominent lymphoid follicles with germinal centres located in the submucosa and mucosa, and are covered by epithelium that may be inflamed. They are benign lesions and have to be distinguished from malignant lymphoma.

B. NEOPLASTIC POLYPS (ADENOMAS)

Neoplastic polyps are colorectal adenomas which have potential for malignant change while polypoid carcinoma is the term used for invasive epithelial tumours. Adenomas have 3 main varieties (tubular, villous and tubulovillous), each of which represents a difference in the growth pattern of the same neoplastic process and variable biological behaviour.

Tubular Adenoma (Adenomatous Polyp)

Tubular adenomas or adenomatous polyps are the most common neoplastic polyps (75%). They are common beyond 3rd decade of life and have slight male preponderance. They occur most often in the distal colon and rectum. They may be found singly as sporadic cases, or multiple tubular adenomas as part of familial polyposis syndrome with autosomal dominant inheritance pattern. Tubular adenomas may remain asymptomatic or may manifest by rectal bleeding.

Grossly

Adenomatous polyps may be single or multiple, sessile or pedunculated, vary in size from less than 1 cm to large, spherical masses with an irregular surface. Usually, the larger lesions have recognisable stalks.

Microscopically

The usual appearance is of benign tumour overlying muscularis mucosa and is composed of branching tubules which are embedded in the lamina propria. The lining epithelial cells are of large intestinal type with diminished mucus secreting capacity, large nuclei and increased mitotic activity. However, tubular adenomas may show variable degree of cytological atypia ranging from atypical epithelium restricted within the glandular basement membrane called

as ‘carcinoma in situ’ to invasion into the fibrovascular stromal core termed as frank adenocarcinoma. Malignant transformation is present in about 5% of tubular adenomas; the incidence being higher in larger adenomas.

Villous Adenoma (Villous Papilloma)

Villous adenomas or villous papillomas of the colon are much less common than tubular adenomas. The mean age at which they appear is 6th decade of life with approximatey equal sex incidence. They are seen most often in the distal colon and rectum, followed in decreasing frequency, by rest of the colon.

Grossly

Villous adenomas are round to oval exophytic masses, usually sessile, varying in size from 1 to 10 cm or more in diameter. Their surface may be haemorrhagic or ulcerated.

Microscopically

The characteristic histologic feature is the presence of many slender, finger-like villi, which appear to arise directly from the area of muscularis mucosae. Each of the papillae has fibrovascular stromal core that is covered by epithelial cells varying from apparently benign to anaplastic cells. Excess

mucus secretion is sometimes seen. Villous adenomas are invariably symptomatic; rectal bleeding, diarrhoea and mucus being the common features. The presence of severe atypia, carcinoma in situ and invasive carcinoma are seen more frequently. Invasive carcinoma has been reported in 30% of villous adenomas.

Tubulovillous Adenoma

(Papillary Adenoma, Villoglandular Adenoma)

Tubulovillous adenoma is an intermediate form of pattern between tubular adenoma and villous adenoma. It is also known by other names like papillary adenoma and villoglandular adenoma. The distribution of these adenomas is the same as for tubular adenomas.

Grossly,

Tubulovillous adenomas may be sessile or pedunculated and range in size from 0.5-5 cm.

Microscopically

They show intermediate or mixed pattern, characteristic vertical villi and deeper part showing tubular pattern. The behaviour of tubulovillous adenoma is intermediate between tubular and villous adenomas. The contrasting features of non-neoplastic and neoplastic colorectal polyps are given in **.**

C. FAMILIAL POLYPOSIS SYNDROMES

Familial polyposis syndromes are a group of disorders with multiple polyposis of the colon with autosomal dominant inheritance pattern. Important conditions included in familial polyposis are:

1. Familial polyposis coli (adenomatosis)
2. Gardner's syndrome
3. Turcot's syndrome
4. Juvenile polyposis syndrome

Some other conditions in which multiple polyposis of colon occur but do not have familial basis are Peutz-Jeghers syndrome (hamartomatous), Cronkhite-Canada syndrome (inflammatory), and nodular lymphoid hyperplasia. The familial polyposis syndromes are as follows.

Familial Polyposis Coli (Adenomatosis)

This hereditary disease is defined as the presence of more than 100 neoplastic polyps (adenomas) on the mucosal surface of the colon; the average number is about 1000. Adenomatosis can be distinguished from multiple adenomas in which the number of adenomas is fewer, not exceeding 100. The condition has autosomal dominant transmission and is due to germline mutations in APC gene which results in occurrence of hundreds of adenomas which progress to

invasive cancer. The average age at diagnosis is 2nd and 3rd decades of life with equal incidence in both the sexes.

Grossly and microscopically

The commonest pattern is that of adenomatous polyps (tubular adenomas) discussed above. The malignant potential of familial polyposis coli is very high. Colorectal cancer develops virtually in 100% of cases by age of 50 years if not treated with colectomy.

Gardner's Syndrome

Gardner's syndrome is combination of familial polyposis coli and certain extra-colonic lesions such as multiple osteomas (particularly of the mandible and maxilla), sebaceous cysts and connective tissue tumours. The number of polyps in Gardner's syndrome is generally fewer than in the familial polyposis coli but their clinical behaviour is identical.

Turcot's Syndrome

Turcot's syndrome is combination of familial polyposis coli and malignant neoplasms of the central nervous system.

Juvenile Polyposis Syndrome

Juvenile polyposis is appearance of multiple juvenile polyps in the colon, stomach and small intestine but their number is not as high as in familial

polyposis coli. Family history in some cases may show autosomal dominant inheritance pattern, while it may be negative in others. They resemble the typical juvenile polyps as regards their age (under 5 years), sex distribution and morphology. They lack the malignant potential.

OTHER BENIGN TUMOURS

Some non-epithelial benign tumours that may rarely occur in large intestine are leiomyomas, leiomyoblastoma, neurilemmoma, lipoma and vascular tumours (haemangioma, lymphangioma).

MALIGNANT COLORECTAL TUMOURS

A. Colorectal Carcinoma

Colorectal cancer comprises 98% of all malignant tumours of the large intestine. It is the commonest form of visceral cancer accounting for deaths from cancer in the United States, next only to lung cancer. The incidence of carcinoma of the large intestine rises with age; average age of patients is about 60 years. Cancer in the rectum is more common in males than females in the ratio of 2:1, while at other locations in the large bowel the overall incidence is equal for both sexes.

ETIOLOGY.

As with most other cancers, etiology of colorectal carcinoma is not clear but a few etiological factors have been implicated:

1. Geographic variations.
2. The incidence of large bowel carcinoma shows wide variation throughout the world. It is much more common in North America, Northern Europe than in South America, Africa and Asia. Colorectal cancer is generally thought to be a disease of affluent societies because its incidence is directly correlated with the socioeconomic status of the countries. In Japan, however, colon cancer is much less common than in the US but the incidence of rectal cancer is similar.

2. Dietary factors.

Diet plays a significant part in the causation of colorectal cancer:

i) A low intake of vegetable fibre-diet leading to low stool bulk is associated with higher risk of colorectal cancer.

ii) Consumption of large amounts of fatty foods by populations results in excessive cholesterol and their metabolites which may be carcinogenic.

iii) Excessive consumption of refined carbohydrates that remain in contact with the colonic mucosa for prolonged duration changes the bacterial flora of the bowel, thus resulting in production of carcinogenic substances.

3. Adenoma-carcinoma sequence.

There is strong evidence to suggest that colonic adenocarcinoma evolves from preexisting adenomas, referred to as adenoma-carcinoma sequence . The following evidences are cited to support this hypothesis:

i) In a case with early invasive cancer, the surrounding tissue often shows preceding changes of evolution from adenoma → hyperplasia → dysplasia → carcinoma in situ → invasive carcinoma.

ii) Incidence of adenomas in a population is directly proportionate to the prevalence of colorectal cancer.

iii) The risk of adenocarcinoma colon declines with endoscopic removal of all identified adenomas.

iv) Peak incidence of adenomas generally preceded by some years to a few decades the peak incidence for colorectal cancer.

v) The risk of malignancy increases with the following

adenoma-related factors:

a) Number of adenomas: familial polyposis coli syndrome

almost certainly evolves into malignancy.

b) Size of adenomas: large size increases the risk.

c) Type of adenomas: greater villous component associated

with higher prevalence.

4. Hereditary non-polyposis colonic cancer (HNPCC or Lynch syndrome).

• No polyps. Autosomal dominant

• Three members of the family have colonic cancers

• Two first degree relatives will have same cancer

• Two consecutive generations observed

• One relative with less than 50 years age will have colonic cancer

• Lynch syndrome I is site specific - commonly right sided, occurs in early age group, 40% are metachronous

• Lynch syndrome II has other malignancy in, stomach, breast, ovary, endometrium and urinary bladder.

• Accounts for 3-5% of colonic cancers

• Amsterdam criteria I (1990); Amsterdam criteria II (1999) and revised Bethesda guidelines (2002) are used to diagnose HNCC

There are germline mutations in mismatch repair genes, human mutL homolog abbreviated as hMLH2 located on chromosome 2 and hMLH1 on chromosome 3 resulting in DNA instability(Microsatellite instability (MSI) at DNA level occurs)in HNCC.

colon cancer appears at a relatively younger age (<50 years), association with multiple primary cancers at different sites (e.g. endometrium, ovary), preferred location in proximal colon and better prognosis than other sporadic colon cancer cases.

5. Other factors.

Presence of certain pre-existing diseases such as inflammatory bowel disease (especially ulcerative colitis) and diverticular disease for long duration increase the risk of developing colorectal cancer subsequently. It may be recalled here that low fibre diet is implicated in the pathogenesis of diverticular disease as well. Besides, there is an etiologic role of tobacco smoking in development of colorectal cancer in younger patients.

GENETIC BASIS OF COLORECTAL CARCINOGENESIS.

Studies by molecular genetics have revealed that there are sequential multistep mutations in evolution of colorectal cancer from adenomas by one of the following two mechanisms:

1. APC mutation/ β-catenin mechanism.

This pathway of multiple mutations is generally associated with morphologically identifiable changes as described above in adenoma-carcinoma sequence. These changes are as under:

i) Loss of tumour suppressor APC (adenomatous polyposis coli) gene located on the long arm of chromosome 5 (5q) is present in 80% cases of sporadic colon cancer. Since the function ofAPC gene is linked to β-catenin, loss of APC gene results in translocation of β-catenin to the nucleus where it activates transcription of other genes, mainly MYC and cyclin D1, both of which stimulate cell proliferation.

ii) Point mutation in K-RAS gene follows loss of APC gene and is seen in 10 to 50% cases of adenoma-carcinoma.

iii) Deletion of DCC gene located on long arm of chromosome 18 i.e. 18q (DCC for deleted in colorectal cancer) in 60-70% cases of colon cancer.

iv) Loss of p53 tumour suppressor gene seen in 70-80% cases of colon cancer.

2. Microsatellite instability mechanism.

In this pathway also, there are multiple mutations but of different genes, and unlike APC mutation/β-catenin mechanism there are no morphologically identifiable changes. This pathway accounts for 10-15% cases of colon cancer. Basic mutation is loss of DNA repair gene. This results in a situation in which repetitive DNA sequences (i.e. microsatellites) become unstable during replication cycle, termed Microsatellite instability, which is the hallmark of this

pathway. The significant DNA repair genes which are mutated in colon cancer are as under:

i) TGF-β receptor gene which normally inhibits cell proliferation but in mutated form allows the uncontrolled proliferation of colonic epithelium in adenoma.

ii) BAX gene which normally causes apoptosis but a defect in

it results in loss of apoptosis and dysregulated growth.

MORPHOLOGIC FEATURES.

Distribution of the primary colorectal cancer reveals that about 60% of the cases occur in the rectum, followed in descending order, by sigmoid and descending colon (25%), caecum and ileocaecal valve (10%); ascending colon, hepatic and splenic flexures (5%); and quite uncommonly in the transverse colon .

Grossly

There are distinct differences between the growth on the right and left half of the colon

Right-sided colonic growths

Tend to be large, cauliflower- like, soft and friable masses projecting into the lumen(fungating polypoid carcinoma).

Left-sided colonic growths

On the other hand, have napkin-ring configuration i.e. they encircle the bowel wall circumferentially with increased fibrous tissue forming annular ring, and have central ulceration on the surface with slightly elevated margins (carcinomatous ulcers).

These differences in right and left colonic growths are probably due to the liquid nature of the contents in the ascending colon leaving space for luminal growth on right side, while the contents in left colon are more solid permitting the spread of growth into the bowel wall. However, early lesion in left as well as right colon are small, button-like areas of elevation.

Microscopically

The appearance of right and left-sided growths is similar. About 95% of colorectal carcinomas are adenocarcinomas of varying grades of differentiation, out of which approximately 10% are mucin-secreting colloid carcinomas. The remaining 5% tumours include uncommon microscopic patterns like undifferentiated carcinoma, signet-ring cell carcinoma, and adenosquamous carcinomas seen in more distal colon near the anus. The histologic grades indicating the degree of differentiation are: well-differentiated, moderately differentiated and poorly-differentiated.

SPREAD.

Carcinoma of the large intestine may spread by the following routes:

1. Direct spread.

The tumour spreads most commonly by direct extension in both ways—circumferentially into the bowel wall as well as directly into the depth of the bowel wall to the serosa, pericolic fat, and sometimes into peritoneal cavity. Locally,

- it can invade the bladder, obstruct ureter and so cause hydronephrosis.

• Can perforate and cause peritonitis/pericolic abscess/faecal fistula.

• Growth may get adherent to psoas muscle posteriorly.

• Carcinoma sigmoid colon can infiltrate and cause colovesical or colovaginal fistula. It can infiltrate ureter, ovary, uterus etc. It can cause pericolic abscess or abscess in lateral abdominal wall.

2. **Lymphatic spread**

Spread via lymphatics occurs rather commonly and involves, firstly the regional lymph nodes in the vicinity of the tumour, and then into other groups of lymph nodes like preaortic, internal iliac and the sacral lymph nodes.

• Growth through lymphatics spreads to pericolic, epicolic, intermediate and principal group of lymph nodes.

3. Haematogenous spread.

Blood spread of large bowel cancer occurs relatively late and involves the liver, lungs, brain, bones and ovary.

CLINICAL FEATURES

Clinical symptoms in colorectal cancer appear after considerable time. These are as follows:

i) Occult bleeding (melaena)

ii) Change in bowel habits, more often in left-sided growth

iii) Loss of weight (cachexia)

iv) Loss of appetite (anorexia)

v) Anaemia, weakness, malaise.

The most common complications are obstruction and haemorrhage; less often perforation and secondary infection may occur. Aside from the diagnostic methods like stool test for occult blood, PR examination, proctoscopy, radiographic contrast studies and CT scan, recently the role of tumour markers has been emphasized. Of particular importance is the estimation of carcino embryonic antigen (CEA) level which is elevated in 100% cases of metastatic colorectal cancers, while it is positive in 20-40% of early lesions, and 60-70% of advanced primary lesions. However, the test may have prognostic

significance only and is not diagnostic of colorectal cancer because it is positive in other cancers too. Such as of the lungs, breast, ovary, urinary bladder and prostate. CEA levels are elevated in some non-neoplastic conditions also like in ulcerative colitis, pancreatitis and alcoholic cirrhosis.

STAGING AND PROGNOSIS

The prognosis of colorectal cancer depends upon a few variables:

i) Extent of the bowel involvement

ii) Presence or absence of metastases

iii) Histologic grade of the tumour

iv) Location of the tumour

Three staging systems are in use:

1. Dukes' ABC staging (modified Duke's includes stage D as well).

2. Astler-Coller staging which is a further modification of Duke's staging and is most widely used.

3. TNM staging described by American Joint Committee is also used.

Histology (WHO)

• Adenocarcinoma – 90%.

• Mucinous adenocarcinoma – 5-10%.

• Signet ring cell carcinoma.

• Small cell/oat cell carcinoma – rare – extremely poorprognosis.

• Squamous cell carcinoma.

• Undifferentiated carcinoma.

Duke's histological grading of carcinoma colon (Nowmodified Morson-Dawson)

• Grade I- low grade.

• Grade II- average grade.

• Grade III- high grade.

• Grade IV- anaplastic.

Haggitt's Invasion of Malignant Polyp

In pedunculated polyp

Level 0 – non invasive carcinoma over the summit.

Level 1 – invasion to head

Level 2 – invasion to neck.

Level 3 – invasion to stalk.

Level 4 – invasion to base.

In sessile polyp – all lesions are level 4.

Sessile Malignant Polyp Invasion

Sm 1 – Submucosal invasion into upper 1/3 (superficial/inner).

Sm 2 – Submucosal invasion into middle 1/3 (inner 2/3).

Sm 3 – Submucosal invasion lower 1/3 (deep).

STAGING OF CARCINOMA COLON

DUKE'S

A. Confined to bowel wall, mucosa and submucosa

B. Extends across the bowel wall to the muscularis propria with no lymph nodes involved

C. Lymph nodes are involved

D. wide spread metastasis

MODIFIED DUKE'S

A - Tumour confined to bowel mucosa

B1 -Tumour involves the muscle wall but not completely

B2 -Involves the serosa

C1 -Tumour involves the muscle wall but not completely

Local lymph nodes involved

C2 - Involves the serosa

Local lymph nodes involved

ASTLER-COLLER'S

A: Limited to mucosa

B1: Extending into muscularis propria but not penetrating through it; nodes not involved

B2: Penetrating through muscularis propria; nodes not involved

C1: Extending into muscularis propria but not penetrating through it. Nodes involved

C2: Penetrating through muscularis propria. Nodes involved

D: Distant metastatic spread

Groups of Lymph Nodes Draining Colon

- N1: Nodes immediately adjacent to bowel wall.
- N2: Nodes along ileo colic/right colic/middle colic/ left colic/sigmoid arteries.
- N3: Nodes near the origin of SMA and IMA.
- Nodal spread in carcinoma colon is sequential from N1 → N2 → N3.

Blood spread:

• 40% of carcinoma colon spreads to liver via portal veins.

• Secondaries may be either solitary or multiple, present as liver with hard, umbilicated nodules– Rarely it spreads to bone, lung, skin.

.

TNM Staging

T0-primary could not be assessed

Tis—Carcinoma in situ.

T1—Invasion into submucosa.

T2—Into muscularis propria

T3—Tumor invades through the muscularis propria into the subserosa, or into nonperitonealized pericolic or perirectal tissues

T4—Tumor directly invades other organs or structures and/or perforates visceral peritoneum

NX-Regional lymph nodes cannot be assessed

N0—No nodes.

N1—Up to 3 nodes.

N2—4 or more nodes.

M0—No metastasis.

M1—Metastasis present.

R0 – No residual tumour after resection;

R1 – Microscopic residual tumour after resection;

R2 – Macroscopic residual tumour.

B. Other Colorectal Malignant Tumours

Aside from colorectal carcinoma, other malignant tumours which are encountered sometimes in the large bowel are leiomyosarcoma and malignant lymphoma . Hindgut carcinoids may occur in the rectum and colon.

SCREENING

Screening Guidelines for Colorectal Cancer		
Population	**Initial Age**	**Recommended Screening Test**
Average risk	50 y	Annual FOBT or
		Flexible sigmoidoscopy every 5 y or
		Annual FOBT and flexible sigmoidoscopy every 5 y or

		Air-contrast barium enema every 5 y or
		Colonoscopy every 10 y
Adenomatous polyps	50 y	Colonoscopy at first detection; then colonoscopy in 3 y
		If no further polyps, colonoscopy every 5 y
		If polyps, colonoscopy every 3 y
		Annual colonoscopy for >5 adenomas
Colorectal cancer	At diagnosis	Pretreatment colonoscopy; then at 12 mo after curative resection; then colonoscopy after 3 y; then colonoscopy every 5 y, if no new lesions
Ulcerative colitis, Crohn's colitis	At diagnosis; then after 8 y for pancolitis, after 15	Colonoscopy with multiple biopsies every 1–2 y

	y for left-sided colitis	
FAP	10–12 y	Annual flexible sigmoidoscopy
		Upper endoscopy every 1–3 y after polyps appear
Attenuated FAP	20 y	Annual flexible sigmoidoscopy
		Upper endoscopy every 1–3 y after polyps appear
HNPCC	20–25 y	Colonoscopy every 1–2 y
		Endometrial aspiration biopsy every 1–2 y
Familial colorectal cancer first-degree relative	40 y or 10 y before the age of the youngest affected relative	Colonoscopy every 5 y
		Increase frequency if multiple family members are affected, especially before 50 y

FAECAL OCCULT BLOOD TESTING: (FOBT)

FOBT analysis is found to be one of the cost effective methods in finding out the patients in need of colonoscopy and so it forms an important part of the screening methods for colorectal carcinoma.

RATIONALE OF FOBT

Faecal occult blood test is based on the tendency of colorectal tumour and large polyps to bleed.

Single faecal occult blood test was not sufficient and has no clinical significance in screening. By one spot FOBT screening the detection rate of adenoma and carcinomas was 2/1000 and 0.6/1000 respectively.

FOBT screening has no benefit without appropriate followup and treatment. This is a screening test that shows a subgroup of average risk asymptomatic people likely to have serious colorectal carcinoma to justify more costly and invasive diagnostic tests. The FOBT itself neither rules out nor rules in colonic cancer.

Few drops of water before adding hydrogen peroxide gives more positive results thereby increasing sensitivity and decreasing specificity.

Some tests are unaffected by dietary peroxidases (ex) hemoquant test based on conversion of heme to porphyrin . it also has the advantage of estimating the amount of blood lost in stool, but this test was the most expensive than the conventional FOBT and therefore this test is unsuitable for screening programmes.

One of the major disadvantage of all faecal blood tests is that colorectal tumours and polyps bleed intermittently and distribution of blood is uneven in stools. Faecal occult blood test signifies the presence of possible cancers and polyps.

COMPLIANCE :

Stool collection for FOBT screening is an unpleasant work , this explains why even in RCT settings not more than 2/3 of people are compliant .outside the randomized control trials compliance is much lower.

ANNUAL FOBT vs BIENNIAL FOBT :

One of the largest randomized trial which last for 13 year follow up(Minnesota trial) clearly shows there is definitive 33% reduction of mortality by annual faecal occult blood test.

Testing every 2 years (biennial screening) resulted only 6% reduction of mortality. Thus annual FOBT is much more effective than biennial FOBT.

FAECAL MICRO RNA vs FOBT :

Cancer society of America guidelines includes faecal DNA test as recommended screening test for colorectal tumours. Upto this date faecal DNA test was the only test based on molecular biology techniques which is commercially available.

Faecal DNA test and cox 2 test for colorectal cancer screening was reported first in 2004.

Recently faecal miRNA (micro RNA) in faeces was used in CRC screening . microRNA was extracted from FOBT residuum. This is stable at 4 degree C than at room temperature. Faecal miRNA degraded at 37 degree C but PCR amplification is used under these situations.

DNA STOOL EXAMINATION :

Colorectal tumour can be diagnosed by extraction of altered DNA from stool. In one study it has a sensitivity of 90% and specificity of 80% for polyps with size 1cm or larger.

But these tests are highly expensive .Hopefully these expenses can be reduced in large scale screening tests. The reported sensitivity and sensitivity of stool DNA examination should be confirmed by large scale studies.

In future ,it would be ideal if DNA testing may be incorporated into the so called " SMART TOILET"(an intelligence toilet system designed by japan) – which can measure sugar levels in urine.

NEWER FOBTS – IMMUNOCHEMICAL FOBT:

These test detects intact human haemoglobin. Other advantages of immunochemical FOBT over guaiac based FOBT is that , iFOBT does not detect partly digested haemoglobin that comes from upper gastrointestinal tract and respiratory tract.

The initial study shows iFOBT have sensitivity and specificity compared with colonoscopy.

PRETREATMENT EVALUATION:

- Demographic data(age, sex, race,etc)
- Complete medical history
- Genetic assessment for those with family or personal history of colorectal ca
- General and systemic examination(DRE,PV, PROCTOSCOPY)
- Haemogram, biochemistry, etc
- Serum CEA
- Pretreatment imaging(DCBE, CT, EUS, MRI, PET)
- Colonoscopy,virtual colonoscopy(MR or CT)

• **CEA (Carcinoembryonic antigen):**

– It is a cell surface glycoprotein discovered by Gold and Freedman.

– It is normally produced by colonic epithelium.

– Its serum ½ life is up to 10 days and is cleared by liver through Kupffer cells.

So its ½ life prolongs in cholestasis and hepatocellular dysfunction.

– Normal level is < 2.5 ng/ml. Level > 5 ng/ml is

significant.

– Even though it is a widely used tumour marker,

it has got low sensitivity.

conditions like pancreatitis, hepatitis, obstructive jaundice, BPH.

– Uses in colorectal cancers are:

a. Preoperative levels >7.5 ng/ml signifies poor

prognosis.

b. If postoperative level does not fall, it indicates either incomplete resection, or occult metastasis elsewhere.

c. Increase CEA during follow-up indicates recurrence

orsecondaries.

– A slow rise indicates loco regional disease.

– A rapid rise signifies metastasis.

– It is not useful in assessing follow-up in poorly

differentiated adenocarcinoma as such tumour

will not produce CEA.

BARIUM ENEMA

The barium enema gives good anatomic and topographic information that not only may be sufficient to diagnose a polyp or carcinoma but also demonstrates the site and configuration of the lesion, and the presence or absence of diverticulosis. The anatomic position of a cancer is clearly of great importance to a surgeon planning an operation. Discrimination for small lesions and mucosal abnormalities is considerably enhanced by the double-contrast technique rather than the single-contrast enema. The air insufflated after the barium shows clearly the mucosal destruction from an ulcerated carcinoma or

the mucosal coating of both adenomatous polyps and polypoid carcinomas. Only double-contrast assessments are appropriate in the assessment of the cancer patient. In the presence of partial obstruction, a soluble-contrast evaluation with gastrografin may be more suitable

CT

CT scans of the chest, abdomen, and pelvis with oral, intravenous, rectum contrast are essential in patients with cancer of the colon. They are informative for identifying the primary tumor location, assessing extramural extension in patients with colon or rectal cancer and for detecting metastatic disease in distant organs or regional lymph node basin

ERUS (Endorectal Ultrasound) AND MRI

High resolution contrast MRI and EUS should be considered as the initial modalities to assess T stage.EUS is better in differentiating T1-T2 lesions while MRI is more appropriate for large T3-T4 lesions. However, both modalities have difficulty in determining the borderline T2,T3 due to presence of desmoplastic reation around tumour

T stage	Ultrasound Findings
uT1	Confined to the mucosa and submucosa
uT2	Invading into but not through the muscularis propria
uT3	Invading into the perirectal fat
uT4	Invading into an adjacent organ

COLONOSCOPY

Colonoscopy has now become the optimal diagnostic tool for most diseases of the large bowel and a major therapeutic modality as well. Certainly, small lesions (less than 1 cm), and mucosal as well as some submucosal conditions (colitis, angiodysplasia), are better demonstrated by colonoscopy. . While barium enema is only a diagnostic tool, with the colonoscope one can biopsy, remove, ablate, dilate, and even at times achieve hemostasis. Since in all colonoscopies there are potential blind spots (at flexures, for example) and occasionally the cecum cannot be intubated, there is still occasional need to complement colonoscopy with contrast radiography.

Contraindications:

Absolute contraindications: inadequate preparation, fulminant colitis, when perforation is suspected or peritonitis established, and in the presence of a recently created intestinal anastomosis.

Relative contraindications: acute inflammation (a limited, careful examination by an experience endoscopist may be vital to the diagnosis of ischemia or acute colitis

Indications:

For diagnostic colonoscopy,

the evaluation of signs, symptoms or history of colitis, resolution of abnormal findings from barium enema, evaluation of all categories of bleeding ,surveillance of high-risk patients for adenomas and cancer and intraoperative localization of non-palpable lesions (in open or laparoscopic procedures).

For therapeutic colonoscopy

1. hemostasis (vascular malformations, polypectomy site, malignant lesions);
2. resection and ablation (polyps, malignancy);
3. decompression and recanalization (volvulus, pseudo-obstruction, stricture malignancy, foreign body).

Equipment:

Colonoscopes are now available as fiberoptic or video instruments. . At least 160 cm of working length is desirable and an instrument channel of at least 3.7 mm in diameter. One needs an assortment of biopsy forceps, grasping forceps, cytology brushes, polypectomy snares, retrieval devices (baskets or graspers), injection needles, possibly laser fibers, and at least a monopolar cautery device.

Preparation and procedure:

Thorough mechanical bowel preparation is required before colonoscopy and is currently most efficiently obtained with rapid gut lavage by ingestion of a polyethylene glycol electrolyte lavage solution. Intravenous narcotics and sedatives are commonly administered, but many patients undergo the procedure without any medication. It is customary to monitor the patient during the procedure with pulse oximetry and sphygmomanometry. The endoscopist, be they gastroenterologist, surgeon or radiologist, should be qualified to perform the procedure and should have received training in the use of electrocautery as well as fluoroscopic equipment. Modern colonoscopes are constructed so that the instrument controls can be manipulated with the left hand; the right hand is

available for insertion of the instrument as well as twisting to the left or right (torquing).

The patient is customarily placed in the left lateral recumbent position to begin the procedure, unless a stoma is being intubated. The perianal area is examined, rectal examination performed, and the instrument is inserted. Once the rectum has been intubated, a small amount of air is inflated to visualize the lumen and the instrument is progressively advanced with the right hand, the tip being deflected with the controls of the left hand. The landmarks on reaching the cecum include visualization of the ileocecal valve and the appendiceal orifice. Precise localization may not be as necessary for a lesion seen anywhere in the right colon, since the surgical procedure will be usually the same. However, it is important to determine where a lesion is in the transverse or sigmoid colon, since different decisions may be made based on such localization. Obstacles to reaching the cecum include poor preparation, strictures, large tumors, and inability to straighten the sigmoid as from previous pelvic operations, radiation or sometimes from extensive diverticular disease.

Complications:

The most serious complications of colonoscopy are perforation and bleeding. Perforation occurs during diagnostic procedures in some 0.1 to 0.5 per

cent of examinations. Causes of perforation include longitudinal tears of the mesentery from excessive looping, especially in the sigmoid, distraction of the bowel wall from a similar maneuver, or impaction of the tip of the endoscope in a diverticulum. Manipulation of irradiated or inflamed bowel, of course, poses a greater risk.

This type of perforation at colonoscopy usually requires prompt surgical intervention and repair, often without the need for colostomy as the bowel will have been well prepared mechanically. Perforation following polypectomy, on the other hand, is usually from transmural thermal injury and may be delayed. Management will depend on the patient's status at the time perforation is recognized. Certainly the presence of signs of peritoneal irritation mandate intervention.

VIRTUAL COLONOSCOPY

Virtual colonoscopy is a relatively new and novel way to screen for colon polyps and cancers. new scanners, in combination with sophisticated three-dimensional (3D) software packages, now make virtual colonoscopy more accurate, affordable, and user friendly.

Magnesium citrate, phosphor soda, and polyethylene glycol solutions used in colon preparation, , elderly patients or patients with renal or cardiac issues cannot take phosphor soda, and therefore we use polyethylene glycol for these patients. Stool tagging is a method by which residual stool in the colon is "tagged" with a high-density contrast agent so that it can be easily identified on CT . If stool tagging is not used, it is often difficult or impossible to distinguish small stool particles from polyps. Stool tagging consists of both solid and liquid stool tagging. Solid stool tagging is accomplished with diluted barium agents. ,Iodinated oral contrast is administrated the day before the CT colonoscopy in liquid tagging.This is a liquid water-soluble iodine-based contrast agent that increases the density of any residual fluid. , it is essential that polyps greater than 5 mm (especially those ≥1 cm) be detected with high sensitivity and specificity. Virtual colonoscopy has limited sensitivity and specificity for lesions smaller than 5 mm. However, because polyps of less than 5 mm are almost never malignant, the detection of these small lesions is not crucial, especially if the patient will be screened at regular 5- to 10-year intervals

Limitations

1, radiation issue

2, Residual stool(can mask or simulate polyps. However, as described earlier, the use of the fecal solid and liquid tagging agents greatly reduces this difficulty.)

3, cost.

4, current lack of sufficient radiologist training.

FDG-PET

^{18}F-Fluorodeoxyglucose positron emission tomography imaging has been successfully used in the diagnosis, staging, and treatment monitoring for a number f cancers, including ovarian, breast, lung, and colorectal.

Basic Theory :

^{18}F-FDG is preferentially taken up by metabolically active cells with up regulated cell surface glucose transporters and increased rate of glycolysis. Images can be displayed in a coronal, sagittal, and transverse manner. Normal PET scans demonstrate physiologic ^{18}F-FDG uptake in the brain, heart, colon, bladder, stomach, kidney, spleen, liver, and bone marrow. the resolution of PET images is currently limited in lesions below 1 cm

Screening and Staging of Colorectal Cancer :

detect both malignant and premalignant colorectal lesions. However, the sensitivity is dependent on the size and the grade of dysplasia. For example, it has been reported that ^{18}F-FDG-PET detects 100% of colorectal cancers 2 cm or larger but only 17% of cancers smaller than 2 cm, including premalignant adenomas

In terms of staging, limitation is due to resolution of PET scanners and signal overlap with the primary tumor. Given the higher sensitivity of magnetic resonance imaging (MRI) and endorectal ultrasound (EUS), ^{18}F-FDG-PET in its present form does not appear to provide any further reliable information on regional lymph node staging in patients with rectal cancer. Therefore the potential role of ^{18}F-FDG-PET in the initial staging of colorectal cancer is the detection of distant disease.

Assessment of Response to Neoadjuvant Therapy in Rectal Cancer :

CT, MRI, and EUS appear unable to assess response because they cannot accurately differentiate scar, fibrosis, and viable tumor. Although the sensitivity of ^{18}F-FDG-PET may be reduced after recent chemotherapy, because it has been suggested that in patients with a great response to neoadjuvant therapy local surgical approaches could be an alternative to radical surgery, assessment

of response by ^{18}F-FDG-PET may have the potential to identify these patients preoperatively

Follow-up and Management of Recurrent Colorectal Cancer :

First, if used routinely, ^{18}F-FDG-PET may detect and localize unsuspected locally recurrent and distant metastatic colorectal lesions. Second, ^{18}F-FDG-PET may help differentiate among scar, fibrosis, and viable tumor in cases in which recurrent colorectal cancer is suspected or other imaging modalities show no evidence of disease despite an elevated carcinoembryonic antigen (CEA). Third, ^{18}F-FDG-PET may help to determine resectability in patients with diagnosed recurrent colorectal cancer at high risk for unresectable disease.

^{18}F-FDG-PET is a valuable tool in patients with a rising CEA and can detect recurrent disease with a positive predictive value of 89% and a negative predictive value of 100% in patients in whom conventional imaging techniques show no evidence for recurrent local or distant disease.

For recurrent colorectal cancer, ^{18}F-FDG-PET shows promise and may currently be used to (1) verify nonspecific findings detected on conventional imaging modalities, (2) evaluate an unexplained CEA elevation, and (3) identify occult unresectable disease preoperatively.

Predicting Resectability:

^{18}F-FDG-PET as a whole-body imaging technique is the most accurate imaging modality for detecting extrahepatic disease and that it can predict the patients who are most likely to benefit from a second surgical procedure.

PET-CT:

PET/CT is significantly more accurate than PET or CT alone for staging and restaging colorectal cancer

Summary

Its role in the screening for and the initial local staging of colorectal cancer may be limited, in patients with suspected distant disease ^{18}F-FDG-PET can help to determine operability. In terms of rectal cancer response to neoadjuvant therapy, ^{18}F-FDG-PET has clear advantages compared with other imaging modalities and may be able to predict response early during the course of treatment. The use of ^{18}F-FDG-PET during follow-up of colorectal cancer patients has yet to be defined. However, for patients with suspected recurrent disease, ^{18}F-FDG-PET can be used to determine unresectable disease and therefore identify patients unlikely to benefit from an exploratory laparotomy

Imaging techniques and their specific role in staging

- primary tumour: MRI&EUS
- CRM assessment: MRI
- Nodal staging: MRI&EUS
- Metastatic work up: CECT abdomen and chest
- Post neoadjuvant therapy: MRI, PET-CT

TREATMENT:

Aims of treatment:

Relieve symptoms and prolong survival

Prevent or minimize the locoregional recurrence and distant metastasis

Preserve urinary and sexual function

Preserve sphincter function whenever possible

Surgical treatment of colon cancer

Right colon cancers

Right hemicolectomy is the standard operation for cancers of the cecum and ascending colon, removing as little of the terminal ileum The 'space of Treves' is medial to the ileocolic artery, with an absence of lymphatics. Hence, taking more than 10 to 15 cm of ileum will not add to the nodal clearance. The

right colic artery is also ligated at its origin. It is a variable vessel, branching from the ileocolic, superior mesenteric artery directly, or from the right branch of the middle colic. Some patients do not have any definable 'right colic' artery. The distal extent of colon removed depends upon the site of the tumor. The resection may need to extend to the mid-transverse colon for cancers near the hepatic flexure, which always requires entering the lesser sac with clearance along the right gastroepiploic vessels and head of the pancreas. The terminal ileum is then anastomosed to the transverse colon in one or two layers with sutures or staples. The anastomosis may be end to end, end-to-side, or side-to side.

In younger and slender patients, lymphadenectomy may be carried out along the superior mesenteric vein to the uncinate process of the pancreas. It is important to remember that the superior mesenteric vein is to the right of the superior mesenteric artery, and may be damaged during aggressive mobilization and dissection. It is a thin-walled structure, not easily repaired. Bleeding at the root of the mesentery should not be dealt with by deep sutures placed 'blindly'. If bleeding occurs, the vessels should be dissected and repaired under direct vision. A thrombosed or occluded superior mesenteric vein at this location will lead to infarction of the entire small bowel. The duodenum and pancreas are also at risk during mobilization of the hepatic flexure.

Transverse colectomy

The lymphatic drainage for cancers of the transverse colon is along the middle colic vessels to the root of the superior mesenteric artery. Resection therefore removes most of the colon supplied by the middle colic artery and in particular the splenic flexure, where thepericolic vascular arcade may be incomplete (The ascending colon is often resected to facilitate reconstruction and avoid a mesenteric 'trap'. The omentum is removed en bloc with the tumor, leaving the gastroepiploic vessels. The most common procedure involves a right and transverse colectomy, with ileal reconstruction to the descending colon.

Left hemicolectomy

For cancers at the splenic flexure, descending colon, or proximal sigmoid colon, a left hemicolectomy is performed .Nodal drainage from the splenic flexure is more common toward the left colic than the middle colic. Generous exposure is necessary to provide good access to the splenic flexure, which is often high and contiguous with the spleen. The lesser sac must be opened The proximal resection should include the splenic flexure because the ascending left colic artery is ligated at its origin and the pericolic anastomosis is often insufficient to ensure a good blood supply to the anastomosis. The transverse colon is mobilized sufficiently to perform a tension-free anastomosis. The

inferior mesenteric artery is seldom ligated at its origin, but should it be necessary to take this vessel because of nodal metastases, the distal resection should be extended to the proximal rectum, which is adequately supplied from the middle rectal artery.

Sigmoid and rectosigmoid colectomy

Cancers of the mid or low sigmoid colon are resected with either a sigmoid or full left hemicolectomy. In either case, resection of the superior hemorrhoidal (rectal) artery is essential. Resection of the inferior mesenteric artery with full mobilization of the splenic flexure and left colectomy is based on the size and exact location of the patient's tumor, the redundancy of the sigmoid, nodal findings, age, general medical condition, and body habitus.

Management of rectal cancer

Combined modality treatment:

All locally advanced rectal cancer require multimodality treatment which includes surgery,radiotherapy and chemotherapy.Correct sequencing of these has been evaluated and debated for last several decades.

PREOPERATIVE THERAPY;

The era of preoperative therapy started as short course radiotherapy[without concurrent chemotherapy]in 1970's.In the 1980's,postoperative chemotherapy showed survival benefit in carcinoma colon .Preoperative chemoradiation thus become the standard of care for patients with cT3 and /or node positive tumors.

PREOPERATIVE CHEMORADIATION:

ADVANTAGES:

1. Biologic: Decreased tumor seeding at the time of surgery and increased responsiveness to radiotherapy as tumor cells are better oxygenated.

2. Physical: No post surgical bowel fixation in pelvis and hence reduced dose to bowel.

3.Functional:Increased resectability in previously unresectable locally advanced disease and better chances of sphincter preservation even in low lying tumors.

DISADVANTAGES:

1. over treatment of patients due to overstaging of T1-2N0 tumors

2. Delay in surgical treatment

3. Uncertainity in deciding post-operative adjuvant treatment in absence of initial pathological staging

POSTOPERATIVE CHEMORADIATION

ADVANTAGES:

1. Precise knowledge of tumor stage available.

2. Accurate definition of tumor bed available by surgical clips

3. avoidance of over treatment

DISADVANTAGES:

1. Small bowel in radiation fields leading to higher rate of complications

2. Potentially hypoxic tumor bed

3. potentially compromised margins for bulky tumors fixed to surrounding structures or pelvic side wall

4.If APR is done radiation fields have to be extended to cover scar

Approaches in preoperative treatment:

There are two main approaches of preoperative therapy for resectable rectal cancer

1.Short course preoperative radiotherapy(SCRT)

2.Long course preoperative radiotherapy with concurrent chemotherapy

The incidence of positive CRM was lower following CTRT compared with SCRT.

Time interval between completion of CTRTand surgery:

Traditionally, radical surgery is recommended 6 weeks after completion of CTRT but retrospective data indicate that surgery performed more than 7 to 8

weeks after completion of CTRT results in higher rates of ypCR and improved outcome.

	Short course(SCRT)	Long course(CTRT)
Eligible clinical stage	cT1-3/n0	cT3-4 and / or N+
Treatment schedule	only RT 5Gy/fraction x 5 days[25 Gy]	RT 1.8 Gy/fraction over 5-6 weeks[45-50.4GY]with concurrent 5-FU based chemo
Interval between RT and surgery	5 days	4-8 weeks
Concurrent chemotherapy	No	yes
Sphincter preservation	No	yes
Improved local control	yes	yes

SURGERY:

Surgery remains the only curative modality of treatment for rectal cancer and the most important determinant of long-term oncologic results

SURGICAL PROCEDURES:

A. Sphincter saving procedure

-Anterior resection

-Low anterior resection

-Intersphincteric resection

-Local excision

-Transanal excision [transanal]

-TEM [Transanal endoscopic microsurgery]

B. Abdominoperineal resection [APR]

- When the sphincter is involved by disease

-Negative margins cannot be achieved by sphincter saving procedures.

-elderly and frail patients where sphincter is already compromised [incontinent patient]

FACTORS DECIDING THE CHOICE OF PROCEDURES:

-Patient factors-age, comorbidities ,status of anal sphincter and pelvic floor ,and patient's wish.

-Tumor location-extraperitoneally located tumors are more suited for local excision[i.e.below the middle rectal valve].Similarly, distance from anal verge is an important criteria in addition to sphincter involvement to decide between anterior dissection[AR] and abdominoperineal resection[APR]

-Tumor stage-depth of invasion of rectal wall, lymph node involvement, size of tumor and rectal circumference involved ,histological features[grade ,mucinous or non mucinous, depth of penetration, lymphovascular and perineural invasion].

PREOPERATIVE COUNSELLING:

A detailed discussion about the disease status and treatment option with their merits and demerits with the patient and family helps in informed decision making ultimately culminating into a properly taken informed consent .

A trained person should counsel all those likely to have temporary or permanent stoma care ,preferably several days before the surgery. stoma marking is done after inspecting the contours of abdominal wall in lying down, sitting and standing position.

TECHNIQUE OF TOTAL MESORECTAL EXCISION:

BASIC PRINCIPLE OF TOTAL MESORECTAL EXCISION:

I. Sharp dissection either with electrocautery or scissors under good lighting and direct vision.
II. The plane should be opened gently by continuous traction and counter traction avoiding tearing or riping apart of tissues.
III. Posteriorly,the avascular plane [holy plane]lies in the loose areolar tissue which lies between the visceral and parietal layers of endopelvic fascia.
IV. Circumferential excision of mesorectum.
V. Laterally inside the plane of pelvic plexus.

EXTENT OF MESORECTAL EXCISION:

The tumor usually spreads into the lymphatics of mesorectum approximately 2 to 5cm distal to the rectal lesion, hence following is the recommendation for the mesorectal excision.

Upper rectal growth-5cm of mesorectal excision distal to the tumor

Mid and lower rectum-total mesorectal excision

DISTAL RESECTION MARGIN:

Intramural microscopic tumor extension is usually restricted to within 2cm from grossly visible distal margin of the tumor with more than 2cm distal spread found only in approximately in 2.5% of patients.

-1 to 2cm distal mural margin is adequate for majority

-8 to 10mm margin is adequate in patients who have had excellent response to chemoradiation

-2cm distal margin is suggested for poorly differentiated carcinoma

An intraoperative frozen section control may further help in confirming negative distal margin.

TECHNIQUE OF TOTAL MESORECTAL EXCISION:

Position: Lithotomy, trendelenburg or Lloyd-davies position.

Anaesthesia: Generalanaesthesia with endotracheal intubation and Foleys catheter.

Incision: Long midline incision[or laproscopic ports for lap TME]

Dissection is divided into six zones

- Above the pelvic brim:

-Divide the peritoneum to sigmoid colon near it base after it pulling the sigmoid anteromedially

-Identify the loose areolar tissue at the back of mesosigmoid.

-Plane between the back of mesosigmoid and parietal fascia is entered ,ureter and superior hypogastric nerves are identified.

-Divide inferior mesenteric artery distal to left colic artery or at the site of division of sigmoid colon. However, lymph nodes from the root of inferior mesenteric artery should be included in the specimen.

- At sacral promontory and posterior dissection:

-make a plane between superior rectal artery and superior hypogastric plexus.

-enter the retrorctal space between the mesorectum and the parietal layer of the endopelvic fascia [loose shiny areolar tissue]

-continue dissection up to the sacrorectal ligament [S4 level],a dense connective tissue layer.

-divide the ligament sharply to reach the pelvic floor.

Along the pelvic side wall [lateral dissection]

-Identify the presacral superior hypogastric plexus [sympathetic nerves]and dissect the left hypogastric nerves away from the visceral fascia.

-Similar plane is entered on the right side

The lateral ligament containing the nerves of the rectum from the pelvic plexus are situated 2 to 3 cm below the peritoneum, lateral to rectum. This hardly contains any significant vessels and can be easily divided with cautery/scissor.

No clamping should be done to avoid nerve injury. The precise dissection plane here is most difficult to identify.

- Anterior dissection:

-Division of peritoneum 1cm above the rectovesicle reflection behind the seminal vesicles and posterior border of prostate.

-plane lies anterior to denonvilliers fascia, which is divided transversely at the base of the prostate [or vaginal vault].Further dissection is continued distally upto pelvic floor posterior to the fascia except in anterior transmural tumors when this fascia included in this level also.In females the vaginal wall and rectum should be dissected carefully an sufficiently to avoid vaginal injury during anastomosis which could result in recto vaginal fistula.

- Extreme distal dissection:

After the mesorectum has been dissected down to the pelvic floor ,3 to 4 cm of rectum is cleared of mesorectal fat for anastomosis.

RECONSTRUCTION AFTER SPHINCTER SAVING RESECTION:

Several techniques of reconstruction have been described with studies reporting superiority of one over the other in terms of functional outcome. However long-term does not indicate better quality of life with anyone technique over the other.

-Straight colorectal/coloanal anastomosis

-Side to end colorectal/coloanal anastomosis

-Colonic pouch anal anastomosis

-Coloplasty

PROXIMAL DIVERSION [EITHER ILEOSTOMY OR TRANSVERSE COLOSTOMY]

Diversion is not required routinely,but it is should be judiciously used in select group of patients,some of which are as follows.

-Anastomosis <7cm from anal verge

-TME

-Male gender

-obese patient

-Straight coloanal anastomosis

-Technical mishap during surgery

-Preoperative CTRT

-Significant intraoperative bleeding and hypotension.

Local excision:

Local excision [LE] is an acceptable alternative to radical surgery in patients unfit for major surgery although at a cost of poorer oncologic outcome. LE has been assessed as an alternative to TME in selected only stage rectal

cancer s because it is less invasive,avoids colostomy and is associated with lesser morbidity and mortality.

Criteria:

1. T1, well to moderately differentiated lesion

2. sizeupto 3cm involving less than a third of rectal circumference

3. No lymphovascular [LVI] or perineural[PNI] invasion or mucinous feature on histopathology.

4. T1N0 tumors on EUS and MRI

5. Clear margins of resection

Even in T1 tumors with no adverse features [as mentioned above],there is a 10 to 15% incidence of lymph node metastasis which cannot be addressed by LE.

All T2 tumors and T1 tumors with high –risk features on histology [poor differentiation ,presence of LVI or PNI ,lymph node positivity]must be subjected to radical resection unless contraindicated in which as adjuvant CTRT is adviced to improved local recurrence and survival.

There may also be a role of LE in selected patients with T2 and T3 tumours having excellent response to CTRT and who are unfit for or refuse more radical surgery. The available data suggests local recurrence and survival data comparable to TME but requires further validation.

Transanal excision and Transanal endoscopic microsurgery:

The conventional transanal excision[TAE] is appropriate for lower lesions while Transanal endoscopic microsurgery[TEM] offers better visualization and access for mid and upper rectal lesion.TEM also follows dissection to be more precise, makes resection of large tumor easier and improves lymph node harvest .Although TEM is superior to TAE in terms of achieving negative margins and lower local recurrence ,it does not translate into significant survival advantage.TEM also requires expensive equipment and has a steep learning curve

ABDOMINOPERINEAL RESECTION:

The preoperative preparation and the abdominal portion of the procedure are similar to the anterior resection of the rectum; therefore, the perineal resection is described here.

Perineal resection

the key anatomic landmarks: the perineal body, right and left ischialtuberosities, and coccyx. An elliptic incision extending from the perineal body to the coccyx is drawn with an indelible pen . The skin is incised with scalpel deepened down through the subcutaneous tissue. The perianal skin ellipse is grasped with three Allis clamps, and the subcutaneous tissue is further divided with electrocautery.

Dissection proceeds directly down toward the coccyx. A moon-shaped incision is made at the tip of the coccyx, and the anococcygeal raphe is identified. The anococcygeal raphe is sharply divided with electrocautery. The Waldeyer fascia is encountered next and sharply divided to enter the presacral space. Laterally, the superficial fascia is divided to enter the ischiorectal fossa on both sides. The fat within the ischiorectal fascia is divided along the same furrow created on the lateral aspect. It is prudent to conceptualize the boundaries of the ischiorectal fossa:

- Laterally, the ischial tuberosity and the obturator fascia overlying the extrapelvic portion of the obturatorinternus
- Medially, the infra-anal fascia covering the levatorani and the sphincter ani muscles
- Ventrally, the transverse perineal superficialis and profundus muscles
- Dorsally, the fascia of the gluteus maximus muscle

In the upper part of the ischiorectal fossa, the inferior hemorrhoidal vessels are clamped, divided, and ligated with 2-0 absorbable sutures. Electrocoagulating these vessels must be avoided because they tend to retract, causing bleeding at a later point postoperatively. Once the ischiorectal fossa has been opened

bilaterally, a self-retaining St. Mark's retractor is placed to facilitate further exposure. Posterior dissection is continued by elevating the anal canal with a malleable retractor and dividing the areolar tissue between the presacral fascia and the rectal fascia with electrocautery. The anal canal and then the rectum are progressively elevated from the presacral fascia, avoiding dissection too close to the sacrum because this can lead to troublesome bleeding from the presacral veins. Conversely, dissection too far anteriorly should be avoided to prevent risk of entering the rectum. If necessary, the abdominal operator should be allowed to direct the perineal dissector into the proper plane. In addition, it is important to keep a mental note of the curves of the rectum as the posterior dissection proceeds.

The index and middle fingers are passed beneath the iliococcygeus muscle on the right and then the left side, and the iliococcygeus muscles are divided sequentially with electrocautery or Metzenbaum scissors .The lateral dissection is continued ventrally by dividing the pubococcygeal muscle. The rectum still remains suspended by the puborectalis sling muscle. This muscle is placed under tension with the use of two malleable retractors in the lateral space, one laterally to retract the ischiorectal fat and the other medially over the rectum. The puborectalis muscles are divided in a longitudinal fashion on each side, thus allowing the rectum to fall dorsally. It is important to avoid drifting too far

ventrally, because this may result in injury to the urethra. It is prudent to feel for the catheter within the urethra intermittently during this dissection. After the posterior dissection has been completed, the clamped sigmoid end of the operative specimen is drawn out through the posterior end of the perineal wound until the sigmoid and the upper two thirds of the rectum have been delivered out of the pelvic cavity.

The anterior dissection is now started. It is the most difficult portion of the perineal dissection because the rectum/anal sphincters are closely attached to membranous urethra, prostate, and seminal vesicles. The anterior skin is retracted while downward traction on the anal skin ellipse is applied to reveal the plane of dissection. The posterior border of the transverse perineal muscle is exposed. The rectourethralis is divided in the midline, and the plane between the rectum and the prostate is entered. The remaining anterior attachments are thinned by progressively dividing tissues on the sides. Again, it is important to palpate the Foley catheter and the prostate intermittently to avoid injury to these structures. Near the final stages of the dissection, it is important to support the rectum because it can be easily avulsed off the prostate or the urethra, which can result in inadvertent injury and troublesome bleeding.

The empty pelvic cavity is irrigated, and bleeding points are secured with a combination of electrocautery and suture ligatures using 2-0 absorbable sutures. If there is concern regarding hemostasis within the pelvis, it should be packed. Otherwise, two closed suction drains are placed in the pelvis and secured with 3-0 nonabsorbable sutures.

Closure:

The skin is approximated with interrupted mattress sutures using 3-0 monofilament nonabsorbable sutures

ADJUVANT THERAPY:

Role of postoperative adjuvant chemotherapy:

Indications for chemotherapy

• Positive nodes.

• T4 lesions.

• Venous (microscopic) spread.

• Signet cell type.

• Poorly differentiated tumour/aneuploidy.

• Changes in CEA level.

Postoperative chemotherapy is used commonly.

Occasionally also given preoperatively.

Regimes are -

• 5 Fluorouracil (5 FU) with folinic acid (leucoverin/ LV) is the most commonly used regime for 6 months as monthly cycles. Folinic acid potentiates the action of 5 FU.

• Levamisole 150 mg/day for 3 days given once in 15 days for one year with intravenous 5 FU monthly for one year.

• Irinotecan/5 FU/LV – IFL regime is also used.

• Folinic acid (LV)/5 FU/oxaliplatin – FOLFOX regime is also used. It is becoming treatment of choice.

• Irinotecan/oxaliplatin – IROX regime is used in previously untreated metastatic colonic cancer.

• Capecitabine (xeloda) an oral drug which generates 5 FU at tumour tissue, shows significantly greater response than 5 FU/LV regime.

• Phase II trials are going on for capecitabine/oxaliplatin and capecitabine/irinotecan combination regimes.

• 5 FU infusions into the portal vein during and immediately after surgery have shown benefits in terms of outcome and recurrence.

EGFR and VEGF blockers (EGFR is epithelial growth factor receptor; VEGF is vascular endothelial growth factor)—

• They are used as single agent and also in combination with chemotherapy drugs in phase II and III trial.

• Drugs are monoclonal antibody, cetuximab which blocks EGFR, bevacizumab which binds VEGF.

The routine practice is to add four to six months of postoperative adjuvant chemotherapy using 5FU,leuocovorin and oxaliplatin [folfox] in patients who have received preoperative radiation or chemoradiation.

Post operative radiotheraphy

Patients with pT3N0 tumours with either adverse pathological features,resected without TME or in whom less than 12 nodes are examined should receive post operative CTRT.

RECENT ADVANCES

1.induction chemotheraphy and use of additional chemotheraphy cycles during the resting period after completion of radiotheraphy

2.capicitabine oral prodrug of 5-fluoro uracil, widely used due to ease of administration and decreased adverse effects

3.Complete clinical response of pre operative CTRT:

A significant proportion of cases with complete clinical response who undergo radical surgery have residual tumour of histopathology which has led to the use of radical surgery in all patients. Local recurrence was higher in the group managed non operatively. However this approach needs very close surveillance to detect and treat recurrence early.

4. Response to CTRT-The biomarkers

Molecular biology information to identify patients who are likely to have greater or complete response to non operative therapy. DNA microarray other biomarkers are also being evaluated to predict response to therapy like epidermal growth factor receptor (EGFR),P53,Ki-67,P21,Bcl2/Bax,etc..

5. Enhanced recovery after surgery protocol

ERAS pathway is to attenuate stress response to surgery and facilitate rapid recovery, reduce the length of hospital stay and post-opertaive complication.

ERAS pathway includes avoidance of preoperative polyetheleneglycol bowel preparation, consuming of carbohydrate rich solution 3 hours before to surgery. Use of total intravenous anesthesia, early mobilization in post operative field, avoidance of NG tube and abdominal drains, intake of liquids and solid foods in early post operative field, minimising opiates for pain control and use of bowel stimulating drugs .

6.Extralevator technique in abdomino-perinealReseach

The traditional APR involves taking a cuff of levator muscle around the rectum, increases CRM positivity is likely to be due to tapering and ultimately absent, mesorectum at the level of levatorani that result in coning in towards rectum at this level, while following the plane of TME from abdominal side. In addition, access and vision often poor at the level of pelvic floor leading to higher specimen perforation and CRM involvement

To circumvent this problem, more radical cylindrical – type APR (ExtralevatorAPR). The abdominal dissection stops at the level levators, abdominal is closed. And patient turned to prone jack knife position perineal dissection takes place in extralevator plane cutting the levator at their attachment to pelvic side wall providing a very wide cuff of levator around the rectum. In anteriorly located tumor apart of prostate or vaginal wall will be resected EN-BLOC. Perineal defect may require with Mesh and / flap to avoid wound related morbidity.

7. Recent advances in in RT

Précise localisation of target and delivery of treatment by 3D conformal therapy (SDCRT) and IMRT (Intensity Modulated Radio Therapy) and IGRT (Image Guided Radio Therapy).

Guidelines for Surveillance after Surgery and Adjuvant Therapy for Colorectal Cancer

History and Physical Examination	Every 3 Months for 2 Years and Then Every 6 Months for a Total of 5 Years
CEA	Every 3 months for 2 years and then every 6 months for 5 years for lesions ≥T2
Colonoscopy	Perform in 1 year. If abnormal, repeat in 1 year. If no polyps found, repeat every 2–3 years. If a preoperative colonoscopy could not be performed because of obstruction, must be done in 3–6 months. For patients with rectal cancer who did not receive pelvic radiation, alternatives may include a flexible sigmoidoscopy every 6 months for 5 years.
CT scans	May be considered annually for people at high risk of recurrence as defined by poorly differentiated histologic grade and tumors with perineural or venous invasion. If the patient is postmetastectomy for synchronous liver disease, the recommendation for CT scans may be increased to every 3–6 months.

MATERIALS AND METHODS:

Patients admitted in various surgical units in thanjavur medical college hospital between January 2013 to july 2014 constitute the materials of this study.

Patients admitted with complaints of abdominal pain , dyspepsia and other non specific compliants not related to colorectal symptoms were included in this study.

The exclusion criteria includes patients with complaints of bleeding PR, altered bowel habits , tenesmus , mucus discharge from PR, spurious diarrhea ,mass desending PR ,FOBT within last one year, sigmoidoscopy within last 3 to 5 years, colonoscopy within last 10 years.

The inclusion criteria includes patients with age of 50 and above, patients willing for further followup (invasive procedures like colonoscopy&UGI scopy)

All patients subjected to basic blood,urine and biochemical evaluation including liver function tests ,USG abdomen ,UGI scopy and CT abdomen in selected cases.

General condition of the patient is evaluated , presense of features like anaemia, lymphadenopathy, pallor, jaundice are noted.

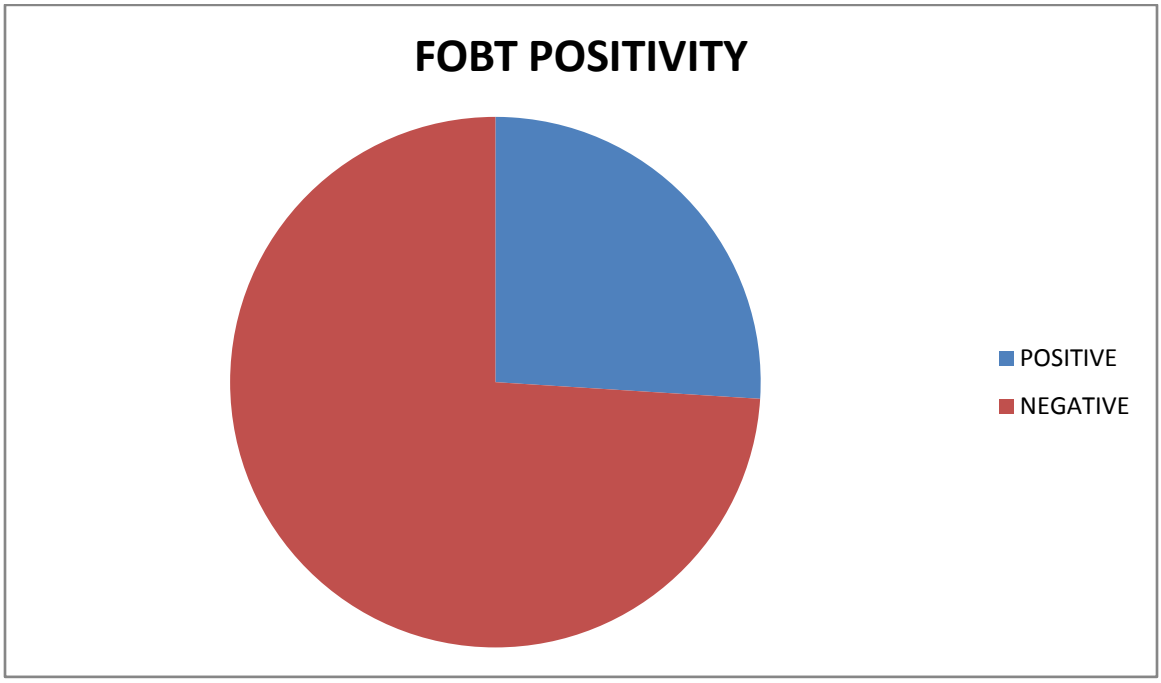
Examination of cardiovascular, respiratory and skeletal system was done. Thorough examination of abdomen (looking for abdominal pain, any mass per abdomen , distention , ascites) was done. Per rectal examination was done in all cases.

A total of 200 patients were studied, patients with positive faecal occult blood test are included in this study, those who are negative for FOBT were adviced for follow up, one year later for another FOBT test.

RESULTS

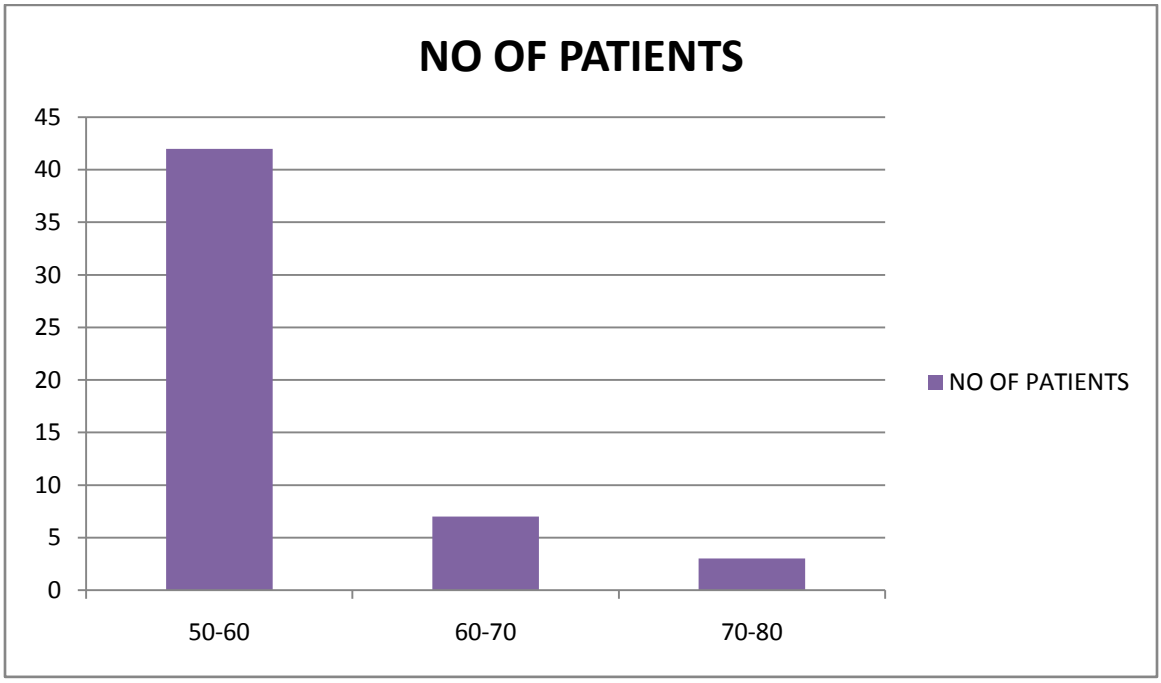
1. FOBT POSITIVITY

NO. OF PATIENTS	FOBT +VE	FOBT –VE
200	52	148



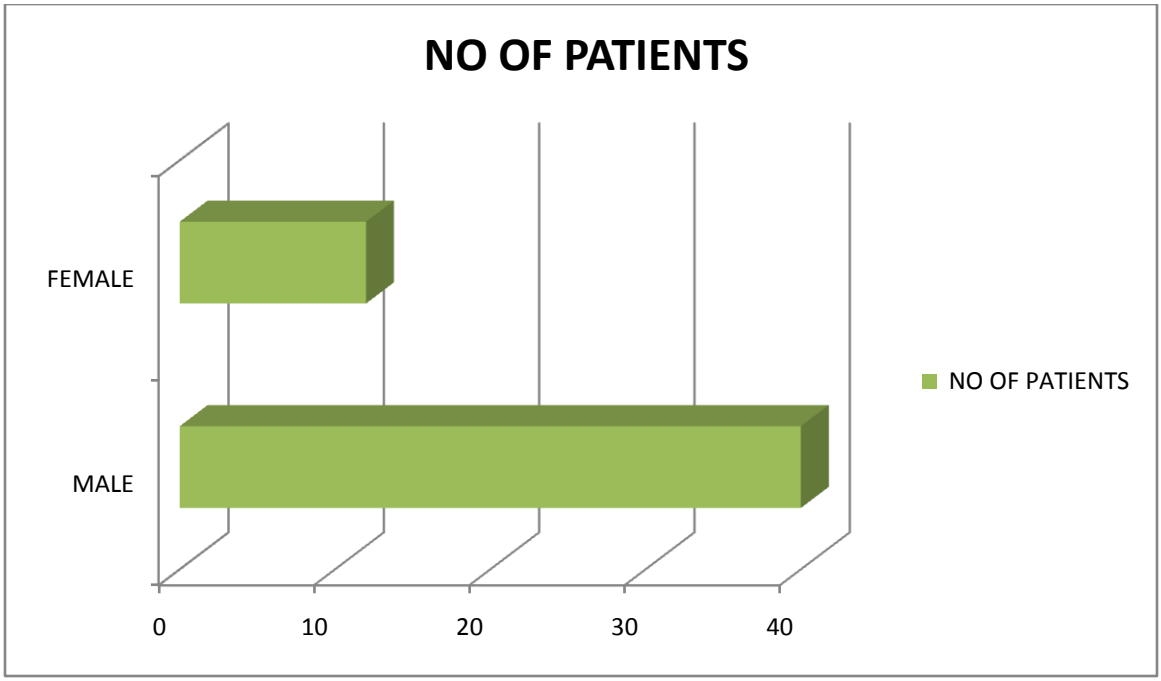
2. AGE DISTRIBUTION OF FOBT POSITIVITY

AGE IN YEARS	50-60	60-70	70-80
NO OF PATIENTS	42	7	3



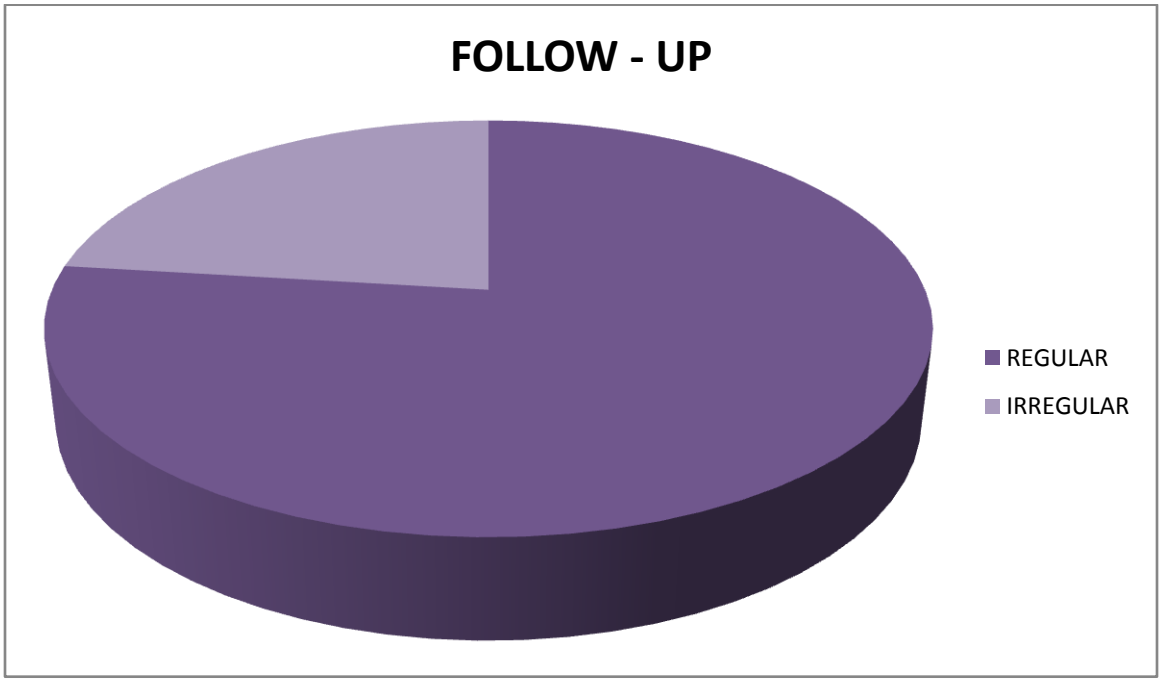
3. SEX DISTRIBUTION OF FOBT POSITIVITY

SEX	MALE	FEMALE
NO OF PATIENTS	40	12



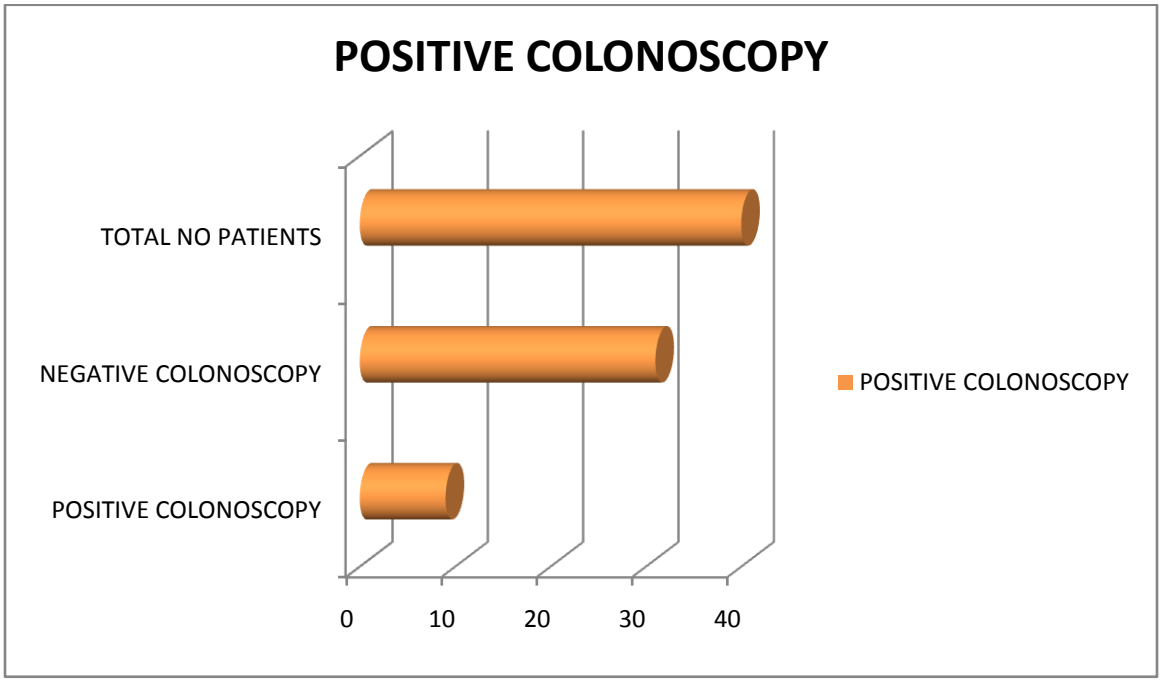
4. FOLLOW – UP STATUS

FOLLOW – UP	REGULAR	IRREGULAR
NO OF PATIENTS	40	12



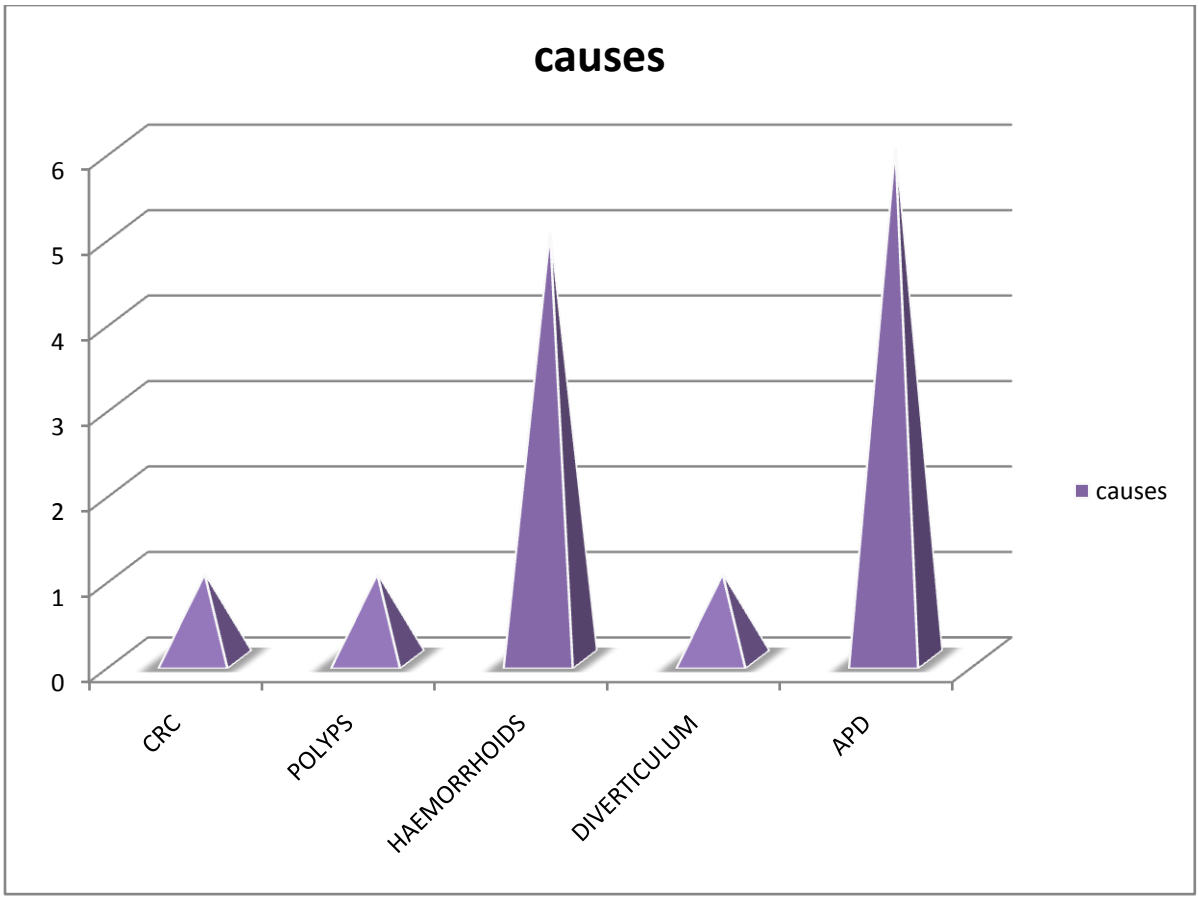
4. NUMBER OF PATIENTS WITH POSITIVE COLONOSCOPY

NO OF PATIENTS	POSITIVE COLONOSCOPY	NEGATIVE COLONOSCOPY
40	9	31



6. CAUSES OF FOBT POSITIVITY

CAUSES	CRC	POLYPS	HAEMORRHOIDS	DIVERTICULUM	APD
NO OF PATIENTS	1	1	6	1	6



DISCUSSION

DEFINITION OF SCREENING

By examining asymptomatic people in order to classify them as likely or unlikely to have the disease.

People who are positive in screening test are likely to go through further investigations to arrive at a final diagnosis. The motive of screening test is early detection of disease, so early treatment is much more easy and effective than later management.

The results of screening test designated either positive if patient is identified to have disease or negative if they are not. Mostly all screening tests have their own negative points in the form of test itself or by its interpretation. These drawbacks are designated as false positive (in this the patient without disease will have positive screening result) and false negative (in this patient with disease will have negative screening result) results.

The sensitivity of the test is defined as “the probability that a person with disease has a positive test result”.

The specificity of test is test is defined as “the probability that a person without disease has a negative test result”.

Screening programmes are mainly assessed by comparing disease mortality in the presence or absence of screening. Evaluation of screening

programmes are done either by comparing screening groups control group as in randomized control trials (or) by comparing mortality before and after the introduction of screening test.

COLORECTAL CANCER SCREENING

All colorectal tumours pass through a long detectable precancerous phase as a polyp in bowel lumen . By early detection and removal of polyp these type of cancers are easily prevented. Even though carcinomas are detected in established early stage it has a better prognosis than its detection in the later stage of tumour progression. These features make colorectal carcinoma as an ideal disease for screening. A lot of screening tests are available for colorectal cancer ranging from low cost and moderately effective occult blood tests to highly expensive and more effective colonoscopy.

Four major randomized control trials clearly shows there was an overall reduction in mortality(26%) by faecal occult blood test (mandel et al ,hard castle et.al, kewenter et.al, kronborg et al).

Greegor (1961) first reported the effectiveness of faecal occult blood test by detecting asymptomatic colorectal cancer patients by using guaiac impregnated test cards.

Though many different chemicals are used to detect faecal blood the guaiac test, haemoccult-ii continue to be one of the most commonly recommended tests because of high specificity , low cost and simplicity.

This test is done for three consecutive bowel movements . this test is done by taking stool samples from different sites using some applicator stick and smeared into two windows in one slide.

FOBT based on colour change is mainly due to pseudoperoxidase activity of haemoglobin. positive test doesn't tell about the amount of blood being lost, so this test was not specific for tumours because non cancerous lesions such as gum bleeding, gastric ulcer, peptic ulcer disease and haemorrhoids will also give positive result because bleeding will also be present in these conditions.

The test is not specific for blood itself because other substances with peroxidase and pseudoperoxidase activity (meat , some fruits, vegetables bacteria) also give false positive result ,if these substances are present in stool sample. because of these drawbacks screening subjects are adviced to follow diet restriction before going to test.

Some substances block pseudoperoxidase activity and give false positive results (ascorbicacid) .Positive test may revert to negative if the slides are stored in lab for more than few days prior to screening.rehydration of test slides by adding patients with positive screening results must undergo definitive diagnostic evaluation by double contrast barium enema with or without flexible sigmoidoscope and colonoscopy.

FOBT in Thanjavur medical college:

Benzidine hydrochloride , barium peroxide , 5 ml of glacial acetic acid all these mixed in test tube. a clean class stick dipped in faecal matter is smeared in white paper a little benzidine solution poured over that white paper.

Deep blue colour within 15 sec indicates strong positive, Greenish blue within 30 sec indicate weakly positive, No colour within 30 sec indicates negative test.

INFLUENCEOF DIET IN FOBT

Experiments in food stuffs indicates uncooked and untreated vegetable or animal tissue gives a positive result which was reduced or nullified by cooking. beef and mutton are exceptions .In fish kingdom sardines and salmon retain their positivity even after cooking. **In our study,** 20% of the study population was found to be vegetarians. So, the dietary predisposition

toward non-vegetarian diet seems to be the contributory factor towards the incidence of colorectal carcinoma and thus subsequently FOBT positivity.

INSTRUCTION ON DIET CONTROL BEFORE TEST:

Foods that must be avoided are,

1) Salmon and sardines
2) Mutton, beef & soup and sausage
3) Rabbit, pigeon
4) Turnip
5) Unboiled vegetables .

Collect first three motions available from the third day onwards.

ADHERENCE TO SCREENING

The advantage of screening occurs only if eligible population are accurately screened .But problems with screening adherence is mainly due to low income, un education and unawareness of population .

Recent study shows people may adhere more with faecal test than colonoscopy.

Is our study, only 77 percentages of people who were found to be having FOBT positivity attended futher colonoscopic evaluation.

SENSITIVITY :

Bleeding from CRC will not be present in all cases of CRC , this explains the low sensitivity of FOBT in CRC screening. The actual sensitivity will not be more than 55% , even though the sensitivity for polyp is much Lower than carcinoma But FOBT undoubtedly detects some polyps, explaining the long term follow up period and annual testing for reduction in mortality of CRC.

SPECIFICITY:

Specificity of this test is around 95% , but FOBT is used as a screening procedure for the general population-say persons above 50 years- the positive predictive value of less than 6-10 %.

In our study, patients with negative FOBT not subjected to colonoscopy, so it was not possible to calculate sensitivity (false positive) and specificity (true negative)

COST :

Guaiac faecal occult blood test itself is very cheap but positive screening test requires additional expensive tests, which may be negative at the end. **In our study,** this being a government instituition, the test was done free of cost to all the patients. The government has to spend around thirty rupees for each analysis.

CAUSES OF FOBT POSITIVITY:

In our study, only 9 patients had treatable diseases among those with FOBT positivity. Of them, 6 had haemorrhoids, 1 had a polyp and a carcinoma each and one other patient had diverticulum. Eventhough the numbers are statistically negligible, the interventions done in these patients in the form of polypectomy and haemorrhoidectomy according to the associated pathologies helped in improving the quality of the precious life of those patients.

A special mention has to be made about the patient who was identified to have the carcinoma. The disease was diagnosed at an earlier stage in this patient and the patient subsequently underwent curative surgery. Thus even as the number is statistically insignificant, our screening with FOBT has helped in saving the life of that patient.

CONCLUSION:

- Faecal occult blood test screening offers no benefit without appropriate follow up diagnostic tests and treatment.
- Single occult blood test examination has no clinical significance in colorectal cancer screening.
- Colonoscopy is more superior to all other screening tests in colorectal cancer screening.
- Colonoscopy offers both diagnostic and therapeutic options.
- It is simple, safe and cost effective but is limited by lack of acceptability, compliance and adherence as well as poor sensitivity and specificity.
- In India, where there is scarcity of resources, in order to have population impact FOBT is the most affordable test.
- In considering all the advantages and drawbacks of FOBT in colorectal cancer screening, we can conclude that this examination is certainly better than no testing at all.

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PROFORMA

NAME :

AGE/SEX :

I.P. NO. :

UNIT :

OCCUPATION :

ADDRESS :

DATE OF ADMISSION :

DATE OF DISCHARGE :

PRESENTING COMPLAINTS :

HISTORY OF PRESENTING ILLNESS :

Abdominal pain

Dyspepsia

Constipation

vomiting

Bleeding per rectum

altered bowel habits

tenesmus

haematemesis/malena

mucous discharge

Loss of wt/appetite

PAST HISTORY :

h/o previous colonoscopy

h/o previous gi surgeries

h/o NSAID intake

h/o previous FOBT

h/o DM/HT/BA/TB

PERSONAL HISTORY :

diet

appetite

smoking

alcohol

GENERAL EXAMINATION :

Anaemia:

icterus:

BP :

pulse :

SYSTEMIC EXAMINATION:

CVS :

RS :

P/A :

P/R :

FOBT :

COLONOSCOPY:

HISTOPATHOLOGICAL REPORT OF BIOPSY :

S.NO	NAME	AGE	SEX	IP.NO	CLI NICAL FEATURES				DIET	DRE	C
					ABDOMINAL PAIN	CONSTIPATION	DYSPEPSIA	ANAEMIA			
1	asokan	57	M	47823	+	_	_	+	NV	N	N
2	Saroja	61	F	56023	+	_	_	+	NV	N	di
3	Charles	58	M	43461	+	_	_	_	VEG	N	N
4	ravi	51	M	41209	_	_	_	+	NV	N	ha
5	Saraswathi	63	F	40127	+	_	_	+	NV	N	N
6	Manivel	50	M	57012	_	_	_	+	NV	N	N
7	kamalraj	55	M	44301	+	_	_	_	NV	N	N
8	Umarani	52	F	45834	_	_	+	_	NV	N	N
9	Balasundar	56	M	47812	_	+	_	_	NV	N	Pc
10	Balraj	51	M	48126	+	_	_	_	VEG	N	N

S.NO	NAME	AGE	SEX	IP.NO	CLI NICAL FEATURES				DIET	DRE
					ABDOMINAL PAIN	CONSTIPATION	DYSPEPSIA	ANAEMIA		

1 1	natarajan	52	M	48201	+	_	_	+	NV	N	
12	Selvaraj	54	M	55123	+	_	_	_	NV	N	
13	Ramani	62	F	48567	+	_	_	_	NV	N	
14	Ganapathy	56	M	48923	_	+	+	_	VEG	N	
15	Veerammal	53	F	67690	_	_	_	+	NV	N	
16	Thasthageer	64	M	45670	+	_	_	_	NV	N	
17	Subramanian	55	M	48452	+	_	_	+	NV	N	
18	Thiyagarajan	61	M	74623	_	_	_	+	VEG	N	
19	Karupaiya	51	M	51236	_	_	+	_	NV	N	
20	Muthulakshmi	57	F	44562	+	_	_	+	NV	N	

S.NO	NAME	AGE	SEX	IP.NO	CLI NICAL FEATURES				DIET	DRE	C
					ABDOMINAL PAIN	CONSTIPATION	DYSPEPSIA	ANAEMIA			
21	Srinivasan	53	M	43276	_	+	_	_	VEG	N	ha
22	magesh	52	M	58445	+	_	+	_	NV	N	N
23	Tamilarasi	54	F	49551	_	_	+	_	NV	N	N
24	Arumugam	63	M	60112	+	_	_	_	NV	N	N
25	Sankar	51	M	58342	_	_	_	+	NV	N	N
26	vasanthi	54	F	47901	+	+	_	_	NV	N	N
27	Ganesan	52	M	38987	+	_	_	_	VEG	N	ha
28	Mani	58	F	47564	_	_	+	_	NV	N	N
29	Karikalan	54	M	39033	+	_	_	_	NV	N	N
30	vijayan	55	M	58965	+	_	_	_	NV	N	N

S.NO	NAME	AGE	SEX	IP.NO	CLI NICAL FEATURES				DIET	DRE	C
					ABDOMINAL PAIN	CONSTIPATION	DYSPEPSIA	ANAEMIA			
31	Gandhi	50	M	43271	+	-	+	-	NV	N	N
32	Siluvainathn	52	M	53462	-	-	-	+	NV	N	N
33	Bakya	55	F	35621	-	-	+	-	VEG	N	N
34	Sundharam	62	M	56342	+	+	-	-	NV	N	N
35	tamilarasan	51	M	23451	+	-	-	+	NV	N	h
36	Raevathi	52	F	36542	+	-	+	-	NV	N	N
37	Kabala	56	M	65473	+	-	+	-	NV	N	N
38	Pitchai	50	M	56342	+	-	-	+	NV	N	N
39	sugaanya	52	F	55435	-	-	+	-	NV	N	h
40	Rengarai	53	M	56321	_	+	-	-	VEG	N	N

DRE-digital rectal examination, APD-acid peptic disease, VUJ-vesicoureteral junction,RPD-renal parenchymal disease .