A HISTOMORPHOLOGICAL STUDY OF POLYPS AND POLYPOID LESIONS OF GASTROINTESTINAL TRACT WITH SPECIAL REFERENCE TO COLONIC NEOPLASTIC POLYPS

Dissertation submitted in partial fulfillment of
the requirements for the degree of

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THE TAMIL NADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

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CERTIFICATE

This is to certify that this Dissertation entitled **"A HISTOMORPHOLOGICAL STUDY OF POLYPS AND POLYPOID LESIONS OF GASTROINTESTINAL TRACT WITH SPECIAL REFERENCE TO COLONIC NEOPLASTIC POLYPS"** is the bonafide original work of DR.V.SHANTHI, in partial fulfillment of the requirement for M.D., (Branch III) in Pathology examination of the Tamilnadu Dr.M.G.R Medical University to be held in April 2013.

Prof.Dr.Geetha Devadas, M.D.
Professor of Pathology
Institute of Pathology
Madras Medical College
Chennai – 600 003.

Prof.Dr.P.Karkuzhali M.D.
Director
Institute of Pathology
Madras Medical College
Chennai – 600 003.

Prof.Dr.V.Kanagasabai, M.D.,
DEAN
Madras Medical College and
Rajiv Gandhi Government General Hospital
Chennai – 600 003.

DECLARATION

I Dr.V.Shanthi, solemnly declare that the dissertation titled **"A HISTOMORPHOLOGICAL STUDY OF POLYPS AND POLYPOID LESIONS OF GASTROINTESTINAL TRACT WITH SPECIAL REFERENCE TO COLONIC NEOPLASTIC POLYPS"** is the bonafide work done by me at Institute of Pathology, Madras Medical College under the expert guidance and supervision of Dr.Geetha Devadas M.D., Professor of Pathology, Institute of Pathology and Electron Microscopy, Madras Medical College. The dissertation is submitted to the Tamilnadu Dr.M.G.R Medical University towards partial fulfillment of requirement for the award of M.D., Degree (Branch III) in Pathology.

Place : Chennai

Date : **Dr. V.SHANTHI**

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. V. Shanthi
PG in MD Pathology
Madras Medical College , Ch-3

Dear Dr. V. Shanthi

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "A Histomorphological study of polyps and polypoid lesions of gastrointestinal tract with special reference to colonic neoplastic polyps" No. 11012011.

The following members of Ethics Committee were present in the meeting held on 28.01.2011 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan, MD	– Chairperson
2. Prof. A. Sundaram, MD Dean i/c , Madras Medical College, Chennai -3	– Member Secretary
3. Prof R. Sathianathan Director , Institute of Psychiatry, MMC,Ch-3	– Member
4. Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	– Member
5. Prof. Geetha Subramanian, MD,DM Prof. & Head , Dept. of Cardiology, MMC, Ch-3	– Member
6. Prof. Md. Ali, MD, DM Professor & Head ,,Dept. of MGE, MMC, Ch-3	– Member
7. Thiru. T.S. Bharathidasan Administrative Officer, MMC, Chennai -3	– Layperson
8. Thiru. S. Govindasamy . BA.BL	– Lawyer
9. Tmt. Arnold Soulina	– Social Scientist

We approve the Proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

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ABBREVIATIONS

GIT	:	Gastro-Intestinal tract
PJ polyp	:	Peutz-Jeghers polyp
H.pylori	:	Helicobacter pylori
MALT	:	Mucosa Associated Lymphoid Tissue
FAP	:	Familial Adenomatous Polyposis
MSI	:	Micro-Satellite Instability
HNPCC	:	Hereditary Non-Polyposis Colonic Cancer
OGJ	:	Oesophago-Gastric Junction
CRC	:	Colo-Rectal Cancer
NHL	:	Non-Hodgkin’s Lymphoma
GERD	:	Gastro-Esophageal Reflux Disease

CONTENTS

A HISTOMORPHOLOGICAL STUDY OF POLYPS AND POLYPOID LESIONS OF GASTROINTESTINAL TRACT WITH SPECIAL REFERENCE TO COLONIC NEOPLASTIC POLYPS

INTRODUCTION:

A polyp is a tumorous mass that projects above the surrounding mucosa and protrudes into the lumen of the gut. Polyps of Gastro-Intestinal tract (GIT) are seen in a wide age range and may arise as a result of inflammation, ectopia, hamartomatous or neoplastic proliferation. Majority of GIT polyps are sporadic in nature. Familial polyposis syndromes have also been described and show a high propensity for malignant transformation. Amongst the colonic polyps, adenomatous polyps are the commonest lesions. Various genes have been implicated in the progression of adenoma to carcinoma. Study of these genes can help in the development of tools for early detection of malignant transformation.

AIMS AD OBJECTIVES:

1. To study the incidence and morphology of (a) different types of polyps and polypoid lesions of gastrointestinal tract (b) malignancies associated with polyps.
2. To study nuclear p53 accumulation and bcl-2 gene mutations in colonic neoplastic polyps with and without coexisting carcinomas

MATERIALS AND METHODS:

The study sample consisted of 452 specimens covering a period of four and a half years. The study material included all lesions identified as polyps or polypoid lesions in the gastro-intestinal tract. The specimens were collected along with relevant clinical details and submitted

for histopathological examination. Cases with both colorectal adenomas and cancers present in the same patient were selected for Immunohistochemical study of p53 and bcl2 expression. The control group consisted of patients with isolated colorectal adenomas without any evidence of malignancy.

RESULTS:

- Polyps and polypoid lesions of GIT constitute 0.745% of all specimens submitted for histopathological examination at a tertiary care centre over four and a half year period.

- Majority of cases involved the large intestine (59.53%) followed by stomach (26.55%).The age group ranged from 13 yrs to 88 yrs and a male predominance was noted in all the sites except for stomach where females outnumbered males.

- The esophageal lesions were commonest at the lower end of esophagus and were predominantly changes of GERD.The stomachlesions were commonly hyperplastic polyps seated at the antrum.

- Duodenum was the predominant site of involvement in the small intestine (85.72%). Hyperplastic polyps were the commonest polyps seen.

- Colonic lesions were predominantly left sided (59.12%) with the rectum being the commonest site.Adenomatous polyps constituted the bulk of large bowel polyps (44.61%).

- 5 cases of adenomatous polyps showed malignant change (4.2%).5 cases showed multiple (more than 100) adenomatous polyps involving the large intestine. 16 cases of adenomatous polyps (13.33% of adenomatous polyps) were associated with non-contiguous colonic cancers which is much higher than other studies worldwide.

- P53 expression was found to be higher in polyps with contiguous or non-contiguous malignancies (61.9%) than in polyps without them (43.75%). They were much higher in the co-existing cancers (76.2%).Bcl2 expression was found to be variable in the three groups.

CONCLUSION:

Polyps and polypoid lesions in the gastrointestinal tract may vary from asymptomatic incidental findings and benign harmless lesions to invasive malignancies. A careful study of adenomatous polyps is needed in view of potential for malignant transformation. This study finds a high incidence of colonic adenomatous polyps with synchronous colonic malignancies which indicates the need for cost effective screening guidelines so as to detect the lesions earlier. Genetic studies are needed to establish predictive and prognostic markers for malignant transformation of adenomatous polyps.

A polyp is a tumorous mass that projects above the surrounding mucosa and protrudes into the lumen of the gut. Polyps of Gastro-Intestinal tract (GIT) are common in the 6th to 7th decade, though they may occur in any age. They may be seen in oesophagus, stomach and small intestine but are most commonly seen in the colon. They may arise as a result of inflammation, ectopia, hamartomatous or neoplastic proliferation. Polyps have to be differentiated from pseudopolyps, which represent areas of inflamed and regenerating mucosa that projects above the level of the surrounding frequently ulcerated mucosa. Pseudopolyps are commonly seen in localised or generalised inflammatory diseases of the GIT such as ulcerative colitis or Crohn's disease.

Majority of GIT polyps are sporadic in nature. Familial polyposis syndromes are rare autosomal dominant syndromes with a high propensity for malignant transformation. They usually present with multiple polyps and show early conversion to malignancy. Sporadic polyps may be asymptomatic, being discovered only in autopsy studies or symptomatic, sometimes with serious complications such as intussusception. They may also be manifestations of benign or malignant neoplasms, underscoring the significance of the lesion.

Various endoscopic techniques have helped the identification and sampling of polyps and polypoid lesions in various parts of

gastrointestinal tract. Clinical details and endoscopic findings are essential to interpret these biopsies correctly.

Polyps of gastrointestinal tract may be classified as non-neoplastic or neoplastic. The non-neoplastic polyps include inflammatory polyps, hyperplastic polyps, hamartomatous polyps and lymphoid polyps. Neoplastic polyps are broadly called as Adenomas. Though the non-neoplastic polyps are grouped so, they may be associated with malignancies elsewhere.

Many other lesions may mimic as polyps endoscopically, including Mesenchymal and lymphoid tumours. The commonest type of polyp varies in different parts of gastrointestinal tract, such as hyperplastic polyps in the stomach and adenomatous polyp in the large intestine. The natural course of the polyp has been difficult to study, as they are usually completely excised as a part of diagnostic procedure. However, various genetic studies and familial syndromes have helped understand the pathogenesis of polyps, especially in cases of colonic cancers arising from adenomatous polyps.

The incidence of neoplastic polyps and colorectal cancers is bound to increase in low prevalence countries like India due to westernization of

diet. Genetic studies are yet to provide definitive predictive markers of development of colonic cancer.

CLASSIFICATION OF POLYPS:

Non neoplastic polyps are classified as

1. Hyperplastic polyps
2. Hamartomatous polyps includes juvenile polyps, Peutz-Jeghers polyps, Cowden syndrome and Cronkite Canada syndrome
3. Inflammatory polyps
4. Lymphoid polyps

Neoplastic polyps include

1. Tubular adenomas
2. Villous adenoma
3. Tubulovillous adenoma

Certain diseases may present as **polypoidal lesions**. They are as follows :

In the oesophagus :

	Benign	1. Squamous papilloma 2. Gastric heterotopia 3. Glycogenic acanthosis

Epithelial lesions		4. Polypoid dysplasia in Barrett's oesophagus
	Malignant	1. Spindle cell carcinoma 2. Conventional squamous cell carcinoma(rare) 3. Conventional Adenocarcinoma (rare)
Mesenchymal lesions	Benign	1. Granular cell tumour 2. Leiomyoma 3. Fibrovascular polyps 4. Inflammatory myofibroblastic tumour
	Malignant	1. GIST 2. Leiomyosarcoma (rare)

In the stomach:

Presenting as hyperplastic polyps	1. Polypoid foveolar hyperplasia 2. Gastritis cystic polyposa / profunda 3. Menetrier's disease
Presenting as	Polypoid gastritis

inflammatory polyps	
Presenting as heterotopic polyps	1. Pancreatic heterotopias 2. Pancreatic acinar metaplasia 3. Brunner's gland hyperplasia
Others	1. Carcinoid tumour 2. GIST 3. Inflammatory myofibroblastic tumour

In the small intestine :

Endocrine tumour	1. Carcinoid tumour 2. Somatostatinoma 3. Gastrinoma
Mesenchymal tumours	1. GIST 2. Lipoma 3. Hemangioma 4. Neurofibroma
Lymphoid lesions	1. Nodular lymphoid hyperplasia 2. Diffuse large B cell lymphoma 3. Mantle cell lymphoma (lymphomatous polyp)

	4. Low grade B cell lymphoma 5. T cell lymphoma

In the large intestine

1. Ganglioneuroma
2. Neurofibroma
3. Granular cell tumour
4. GIST
5. Lipoma
6. Pneumatosis coli
7. Endometriosis
8. Mucosal pseudolipomatosis
9. Malakoplakia

1. To study the incidence and morphology of different types of polyps of gastrointestinal tract presenting at Madras Medical College.

2. To analyze the incidence and morphological features of malignancies associated with polyps.

3. To study nuclear p53 accumulation and bcl-2 gene mutations in colonic neoplastic polyps with and without coexisting carcinomas

POLYPS AND POLYPOID LESIONS OF ESOPHAGUS

- Epithelial tumours presenting clinically as polyps include adenomas, squamous cell carcinoma, sarcomatoid carcinoma, adenocarcinoma and rarely malignant melanoma. Adenomas of oesophagus are rare polypoid tumours that represent raised dysplasia in Barrett's oesophagus. They may be multiple and present with synchronous adenocarcinoma[1] .
- Superficial squamous cell carcinomas may be polypoidal or verrucous[2]. Well differentiated verrucous type presents as large warty lesion. Microscopically they present as broad papillae of well differentiated keratinising squamous epithelium with minimal cytological atypia; they invade underlying tissue as bulbous epithelial tongues.
- Sarcomatoid or spindle cell carcinomas otherwise called polypoid carcinomas occur in middle age or elderly men. They present as bulky polypoid masses reaching large sizes (upto 15 cm in length) in the mid or lower third of oesophagus.
- Adenocarcinoma of oesophagus show high grade dysplasia in a background of Barrett's type epithelium. Malignant melanoma presents as polypoid intraluminal growth; microscopically, they resemble the epitheloid and spindle cell types seen in cutaneous melanoma.

- Smooth muscle tumours most commonly arising in esophagus include Leiomyoma and Leiomyosarcoma. Leiomyoma presents as polypoidal growth covered by mucosa that may ulcerate. Cut surface appears bulging and grey whorled. Microscopically, the tumour cells are arranged in interlacing fascicles and have eosinophilic cytoplasm with blunt ended cigar shaped nuclei.

- Fibrovascular polyp presents at upper third of esophagus as solitary pedunculated polyps with core of edematous fibrous tissue. Large polyps may cause obstruction or asphyxia owing to compression of larynx or may regurgitate into posterior pharynx. Rare case of squamous cell carcinoma arising in a giant fibrovascular polyp has been reported by Cokelaere K et al.[3]

- Inflammatory fibroid polyps are rare lesions composed of fibrous and granulation tissue with spindle shaped fibroblasts arranged concentrically around blood vessels. LiVolsi VA et al[4] in his study has reported 4 cases of inflammatory fibroid polyp which may be mistaken for a leiomyoma both radiographically and on the operating table.

- Hyperplastic polyps at the gastro-esophageal junction are due to chronic mucosal injury such as erosive esophagitis and GERD. In a study of 30 hyperplastic polyps of esophagus by Abraham SC et al[5], 67% of

them were at the gastro-esophageal junction and were associated with ulcers or erosive esophagitis.

POLYPS AND POLYPOID LESIONS OF STOMACH

- Adenomas of stomach arise in context of atrophic gastritis with intestinal metaplasia[6,7]. They are usually solitary, sessile or pedunculated[8] and have potential for malignant transformation. In a study of 357 cases of gastric polyps by Laxen et al[9], 8% (27 cases) had gastric adenomas. In 14 cases carcinoma was found within polyp and in 13 cases carcinoma was found outside the polyp. Carcinoma was found in 38% of adenoma cases.
- Gastric adenomas may co-exist with carcinoma elsewhere in stomach especially in males[10]. Nakamura and Nakano[11] have studied 611 gastric polyps from 275 cases and classified them into 4 subtypes. In type 1, which comprise 60 to 80%, polyps were solitary, pedunculated and situated at the pyloric antrum. They are composed of irregularly proliferating non dysplastic glands and cysts. The glands are lined by high columnar epithelial cells without dysplasia. Type 2 polyps are multiple and located in the distal fundic mucosa of stomach. They have pink dimples which microscopically correspond to superficial erosion. The polyp is lined by hyperplastic regenerative epithelium at the center

and hyperplastic foveolae, fundic glands and cysts at the periphery. Type 3 polyps are sessile and located at the pyloric antrum. Histologically, they have a characteristic two layered structure-upper layer of densely arranged vertical glands lined by darkly staining columnar cells with hyperchromatic nuclei and stratification and lower layer is composed of hyperplastic pyloric glands, ducts and multiple cysts. Type 3 polyps are further subdivided into 3 groups based on the degree of proliferation of non-dysplastic glands in lower layer. Type 4 polyps resemble colonic polyps grossly. They are sessile or pedunculated, surface showing fine lobulations, with inter communicating clefts. Mild/moderate/severe dysplasia was noted. The lining epithelium is columnar which appears hyperchromatic with elongated oval or round nuclei. The polyps may show tubular/villous/tubulovillous architecture. Malignant change was absent in types 1 & 2, focal in type 3 and maximum in type 4.

- Hyperplastic polyps are the most common type of gastric polyps. They arise in association with H.pylori associated gastritis, pernicious anemia or ulcers/erosions in a background of atrophic gastritis with intestinal metaplasia. They may increase or regress with time. 2 to 3% of hyperplastic polyps of size more than 2 cm show dysplasia or intramucosal carcinoma and may be associated with synchronous adenocarcinoma of stomach. T Hattori[12] has studied the morphology of

67 hyperplastic polyps of which 18 showed intestinal metaplasia. 3 polyps showed intestinal type adenocarcinoma and 2 showed gastric type adenocarcinoma.

- Inflammatory fibroid polyps are seen in association with hypochlorhydria or achlorhydria and are seen at the antrum. Histologically they are similar to their esophageal counterparts.
- Gastritis cystica polyposa/profunda by definition is a hyperplastic polyp showing misplaced foveolar or glandular epithelium in the muscularis mucosae or submucosa or muscularis propria. When the gross appearance is that of an intraluminal polyp, the lesion is called polyposa, whereas profunda refers to the bulk of the lesion located in the wall of the stomach. These lesions are seen in patients with chronic bile reflux, usually in post-gastrectomy status. The pathogenesis is unclear; the lesion may be induced by the entrapped epithelium causing excess reactive tissue formation. These lesions are usually seen on the gastric side of gastro-enteric anastomoses. Histologically the entrapped epithelium may be mucinous or glandular and cystic, surrounded by lamina propria like stroma with inflammatory infiltrate. Dense collections of smooth muscle surround the cysts, the lining of which may show hyperplasia or metaplasia.

- Fundic gland polyps occur sporadically or as component of Familial Adenomatous Polyposis. They are usually multiple, glassy, transparent and sessile. Microscopically, the polyp shows cystically dilated glands lined by fundic epithelium admixed with normal glands.
- Peutz-Jeghers polyps are hamartomatous polyps which may show dysplasia or even frank carcinoma. Cochet[13] et al have described two cases of metastasizing gastrointestinal carcinomas arising from hamartomatous polyps and showed extensive metastasis. Both cases showed dysplastic areas within hamartomatous polyps.
- Juvenile polyps are rare in stomach. They may occur at any age and produce anemia and hypoproteinemia. Multiple juvenile polyps are seen in Juvenile Polyposis syndrome which is associated with an increased risk of carcinoma of colorectum and also the stomach. In a study by Hizawa[14] et al, 12 cases of juvenile polyposis involving stomach at initial presentation were compared with 29 cases of generalised juvenile gastrointestinal polyposis. They concluded that juvenile polyposis of stomach has malignant potential and is a separate entity from generalised gastrointestinal polyposis.
- Of the heterotopias, pancreatic heterotopias are the most common in the stomach; it occurs in the pylorus or antrum as conical or short cylindrical mass.

- Menetrier's disease presents grossly as hypertrophic rugae of 1 to 3 cm height or may resemble polyposis of body and fundus when uneven and nodular. It is said to be associated with an increased risk of gastric cancer and requires life-long surveillance due to its chronic nature[15].
- Gastric adenocarcinomas present as polypoid, fungating, ulcerating or diffusely infiltrating growth. Polypoid growths are more common in the corpus and greater curvature. M S Wu[16] has studied the expression of p53 in different subtypes of intestinal metaplasia and gastric cancer. 135 gastric cancer specimens were evaluated for p53 expression in cancerous epithelium and adjacent intestinal metaplastic epithelium. The study concluded that type 3 intestinal metaplasia is the most common lesion in dysplasia-carcinoma transition especially in intestinal type gastric cancer.
- Primary gastric B cell lymphomas occur in middle aged or elderly in a background of H.Pylori induced gastritis. They present as polypoid, fungating or ulcerating tumours at the antrum. 9 cases of synchronous adenocarcinoma and low grade B cell lymphoma of MALT type have been described by Wotherspoon et al[17].
- Malignant lymphomatous polyposis characterised by multiple gastric polyps or as a generalised thickening of rugal folds giving a cerebriform appearance. They are mostly Mantle cell lymphomas.

Microscopically, they show small lymphoid cells with folded nuclei forming monotonous infiltrates. Lymphoepithelial lesions are rare.

- Up to 30% of GI carcinoid tumors occur in the stomach. They occur most commonly in the body and fundus in middle aged individuals. They are usually asymptomatic and are associated with endocrine cell hyperplasia. WHO classifies gastric carcinoids into 4 types: Types 1, 2 and 4 are associated with hypergastrinemia whereas type 3 is sporadic. Microscopically gastric carcinoids are composed of small uniform polygonal or cuboidal cells with finely granular cytoplasm, regular round nuclei, stippled chromatin and scant mitosis. They are argyrophilic and show expression of chromogranin A, synaptophysin, PGP 9.5 and leu-7.
- The stomach is the commonest site for GISTs, constituting approximately 60% of all cases. Gastric GISTS predominantly show epithelioid features. The median age of occurrence is 60 yrs with fundus being the commonest site. Grossly, GISTs are solitary intramural tumours. However, predominantly extramural and dumbbell forms are also encountered. Malignant GISTs are predominantly expansile and rarely infiltrate adjacent tissues. External surface is granular and rubbery. On cut section, they are characteristically grey and lacking trabeculations or whorling pattern. The tumour cells of gastric GISTs are predominantly uniform spindle cells arranged in fascicles and showing oval blunt-ended

nuclei and abundant eosinophilic cytoplasm. Epithelioid GISTs are rare and show sheets of round cells with clear cytoplasm, cellular pleomorphism and bizarre nuclei. Both subtypes show hyalinization and myxoid degeneration. CD117(c-kit) and CD34 are the important markers for diagnosis.

POLYPS AND POLYPOID LESIONS OF SMALL INTESTINE

- Adenomas of small intestine are rare and are commonly seen in peri-ampullary region; they are usually multiple, sessile and villous. Perzin K H and Bridge M F[18] have studied 51 patients with small bowel adenomas. Of them, 18 had adenomas and 33 had tumours containing both adenomas and adenocarcinomas in the same lesion. Of the 33 cases, 5 were intramucosal carcinoma and 28 were invasive carcinomas. Their study concluded that adenomas of small intestine were probably pre-malignant lesions and many primary adenocarcinomas arise from these adenomas. According to their study, carcinomas tend to occur more frequently in villous adenomas, larger lesions and at the ampullary region.

- Adenocarcinomas are polypoid or ulcerating tumours most common at peri-ampullary region. Talbot I C[19] et al have studied 26 ampullary carcinoma resection specimens of which 25 were intestinal type adenocarcinomas and 1 case was papillary adenocarcinoma. Co-

existing adenomas were seen in 11 cases, over half of which were low grade carcinomas.

- Lymphoid polyps tend to occur in children and present with right iliac fossa pain, obstruction or intussusception.
- Other polyps and polypoidal lesions include Brunner gland hamartoma, Peutz-Jeghers polyp, juvenile polyps, pancreatic heterotopia and Inflammatory fibroid polyp.
- Peutz-Jeghers polyps arise most commonly in small intestine. They may be sporadic or a part of Peutz-Jeghers syndrome which included GI polyposis, oral pigmentation and an increased risk of cancer of GIT, pancreas, breast, ovary, uterus and lung. An autosomal dominant disease, Peutz-Jeghers syndrome is due to mutations in LKB1 gene on chromosome 19p. Grossly, PJ polyps may be sessile or pedunculated with a short broad stalk. Histologically, the polyps show branching core of smooth muscle arising from muscularis mucosa and covered by normal appearing epithelium and lamina propria. Dysplasia and malignancy are rarely seen.
- The small bowel GISTs constitute 25–30% of all GISTs. Though ileum is the commonest site, a substantial number of cases occur in duodenum also. The histological types seen are similar to those in stomach, but the epithelioid GISTs are uncommon in the small intestine.

Low grade small bowel GISTs are paucicellular spindle cell stromal tumours arranged in organoid pattern with extracellular skenoid fibres and show mitotic rate of <2/50 hpf. Whereas, aggressive tumours are characterized by high cellularity, less organoid morphology, necrosis and high mitotic rates. Size, cellularity and mitotic index are the most important prognostic factors in jejunal and ileal GISTs. The immunohistochemical markers are similar to those of GISTs elsewhere.

- Endocrine tumors of small bowel are commonest in the ileum and rare in the duodenum. Grossly, they present as smooth, tan-yellow, submucosal polypoid masses. Most serotonin-producing endocrine tumors are seen in the distal small intestine and are extremely rare in the duodenum. The serotonin produced by these tumours elicits peritumoral or mesenteric fibrosis resulting in symptoms of recurrent small bowel obstruction, vascular compression (mesenteric angiopathy), and protracted abdominal pain. Synchronous or metachronous malignancies have been reported including carcinomas of the GI, genitourinary, and gynecologic tracts. The tumor cells have abundant amphophilic or faintly eosinophilic cytoplasm, round nuclei, stippled chromatin, and inconspicuous nucleoli. Necrosis and atypia are absent, though perineural and lymphovascular invasion may be present.

POLYPS AND POLYPOID LESIONS OF LARGE INTESTINE

- These include :

1. Adenomatous polyps : Tubular, Villous, Tubulovillous
2. Hyperplastic polyps
3. Inflammatory polyps
4. Hamartomatous polyps : juvenile polyps
   Peutz-Jeghers polyps
   Polyps in Cronkite-Canada syndrome
   Polyps in Cowden syndrome
5. Lymphoid polyps and malignant lymphomatous polyposis
6. Colorectal carcinomas
7. Mesenchymal polyps

- **ADENOMA** :

➢ Adenoma is a circumscribed benign epithelial neoplasm with a potential for malignant change.

➢ Adenomas of large intestine are more commonly seen in populations at risk for colorectal carcinomas[20, 21] as demonstrated by 2 autopsy studies.

➢ They are more common in males in all regions of colon; but the risk of carcinoma in females is more at the rectum. This is because

adenomas in females tend to be larger and more dysplastic[22, 23, 25] with a higher conversion rate to carcinoma. Hoff G et al[22] examined 50 polyps obtained by endoscopic screening of rectosigmoid polyps. Of these 41 were adenomas. Adenomatous polyps were found in both proximal and distal colon in males, but greater extent of dysplasia was found in rectosigmoid adenomas in women.

- Adenomas are generally evenly distributed along the length of large intestine. However, carcinomas differ in site distribution as they are more common in rectum and distal colon. This is due to higher conversion rate of adenoma to carcinoma in the rectum[21, 24].
- Usually asymptomatic, adenomas may present with occult rectal bleeding, or water and electrolyte depletion.
- Adenomas may be inherited in an Autosomal Dominant mode as shown by Burt RW et al[26] and Aitken et al[27].
- The significance of adenomas lies in the fact that they serve as precursor lesions for colorectal carcinoma. However as the prevalence of adenomas is 30 times more than that of carcinoma, it is probable that only a small proportion of adenomas will turn malignant[28].
- Adenomas may be sessile or pedunculated; sessile ones resemble hyperplastic polyps and pedunculated polyps are cauliflower-like.

- Flat adenomas refer to adenomas without a polypoid component. As the name suggests, they are flat lesions with elevated edges. Cases of multiple hereditary flat adenomas reported in the past represent Attenuated FAP. The behaviour and prognosis of solitary flat adenomas are found to be similar to pedunculated adenomas.
- Histologically, adenomas are divided into tubular, villous and tubulovillous adenomas. By definition, all are lined by dysplastic epithelium which are immature cells with enlarged, hyperchromatic and stratified nuclei. Increased mitotic activity is seen in surface epithelium and upper crypts. Architectural abnormalities are noted.
- Tubular adenomas show branching tubules constituting atleast 80% of the neoplasm. They are usually pedunculated. Villous adenomas show atleast 80% of the tumour constituted by leaf-like or finger-like processes of lamina propria covered by dysplastic epithelium. They are usually sessile and large. Tubule-villous adenomas show both components neither more than 80%.
- Adenomas are classified based on degree of dysplasia into mild, moderate and severe dysplasia. Mild dysplasia is characterised by low nuclear cytoplasmic ratio with elongated, crowded and stratified nuclei and with preserved mucin secretion. Moderate dysplasia is characterised enlarged, hyperchromatic and pseudostratified nuclei with very few

goblet cells. Severe dysplasia is characterised by enlarged and hyperchromatic nuclei with prominent nucleoli and architectural abnormalities like crowded and back to back glands.

- The term carcinoma in situ is used when architecture is that of malignancy but the line of invasion has not crossed muscularis mucosa.
- Mixed polyp shows features of hyperplastic polyp and adenoma.
- Serrated adenomas show the features of adenomatous dysplasia and epithelial serration. They are related to hyperplastic polyps histogenetically. Both share certain phenotypic and genotypic alterations. Torlakovic E et al[29], in a study on morphological reappraisal of serrated colorectal polyps, identified a distinct group of serrated polyps with abnormal proliferation. These polyps show decreased expression of hMHL1 and hMSH2 compared to polyps with normal proliferation. They recommend evaluation of site, size and morphological features when serrated polyps are included in colorectal carcinogenesis research.
- Sessile serrated adenomas are more commonly located in proximal colon and relatively large. They show dilated crypts, exaggerated serrations but lack overt dysplasia. These hyperplastic like polyps are linked to subsets of colonic cancer via DNA methylation, BRAF mutation and DNA MSI pathway[30].

- Familial Adenomatous Polyposis (FAP) is an autosomal dominant disorder characterised by hundreds of adenomas throughout colon. The genetic mutation involves APC gene on chromosome 5q, a rare autosomal recessive type with mutation in gene MYH has been recognised[31]. Mutations in exon 3 and 4 of APC gene causes attenuated FAP with fewer numbers of adenomas that are right sided and flat. In FAP, adenomas are usually tubular and are also found at other sites namely duodenum, ileum and antrum of stomach. Non GI lesions have also been described.
- Gardner's syndrome in which APC gene mutation is at 3' end is characterised by multiple osteomas of skull and mandible and multiple epidermal cysts of skin[32]. Also seen are mesenteric and abdominal wall fibromatosis, soft tissue fibroids, dental cysts, retinal pigmentation, thyroid cancers and medulloblastoma.
- Turcot's syndrome is characterised by FAP and CNS tumours-glioblastoma and medulloblastoma. They are now considered as cases of either FAP or HNPCC[33].

- **COLORECTAL CARCINOMA**

- EPIDEMIOLOGY : Colorectal carcinoma is one of the commonest carcinoma in the west but of low incidence in India. In a study by Sinha[34]

et al, incidence in the United states was found to be 40.6(men) and 30.7(women) per lakh population whereas the incidence rate in India is 4.7(men) and 3.2(women) per lakh population. In another study by Mohandas K M and Desai D C[35], the incidence of colonic cancer in 8 population registries vary from0.7 to 3.7 (men) and 0.4 to 3(women) per lakh population. For rectal cancer, incidence rates range from 1.6 to 5.5(men) and 0 to 2.8(women) per lakh population. He also found that incidence rates for large bowel cancer in rural India is approximately half of that in urban India. Rectal cancer was also found to occur more commonly in young Indians.

- ETIOLOGY: This consists of both environmental and genetic factors, the latter being important. Environmental factors include diet especially increased meat consumption and alcohol. Vegetable fibre plays an important protective factor. Genetic factor plays an important role in adenoma formation.
- Carcinoma right colon is more common in females at all ages[36]. Carcinoma of left colon is more frequent in females under 50 yrs and in males over 70 yrs. The excess of carcinoma of right colon in individuals under 50 yrs is explained by the fact that HNPCC occurring in younger individuals involve the right colon. Isbister WH and Fraser J[37] studied the survival rate of young patients with CRC and concluded there was no

evidence to suggest that younger patients (younger than 40 yrs) with CRC had worse prognosis and did not survive as long as older patients(more than 40 yrs).

- The subset of CRCs associated with MSI and DNA methylation is associated with female gender, proximal location and age[38].
- Studies have shown that a large number of CRCs arise from pre-existing adenomas[39] and de novo carcinomas may arise from flat adenomas.
- HNPCC constitutes 1 to 5% of CRCs; it is due to defect in DNA mismatch repair gene MSH2, MSH6, MLH1 and PMS2[40]. In HNPCC, cancer tends to occur in right colon. Jass JR and Stewart SM[41] studied the prevalence of colorectal adenomas in 23 patients of HNPCC and compared it with age matched controls. Of the adenomas found in these patients, 9 were >1 cm and 6 of them were of the villous type. Contiguous adenocarcinomas were found in 6 adenomas. They concluded that adenomas in HNPCC were found in small numbers at a younger age and were of large size and villous type. Their conversion rate to malignancy was higher than that of sporadic cancer.
- Grossly, CRC may be ulcerating (most common), annular or protruberant types. Microscopically, 90% of CRCs are adenocarcinomas with tubular differentiation. Loy TS and Kaplan PA[42] reviewed 420 cases

of CRCs of which 95 tumours showed villous architecture. Those showing more than 50% villous architecture were designated as villous adenocarcinomas and were diagnosed by identifying epithelial islands in desmoplastic stroma. They found that villous adenocarcinoma carried a good prognosis

- Mucinous adenocarcinomas are diagnosed when more than 50% of tumour consists of mucus. Signet ring cell carcinoma in large bowel is rare (1% of large bowel carcinoma). More than 50% of tumour is constituted by cells containing intracytoplasmic mucin. Other types include squamous cell carcinoma, adenocarcinoma, small cell carcinoma and undifferentiated carcinoma.

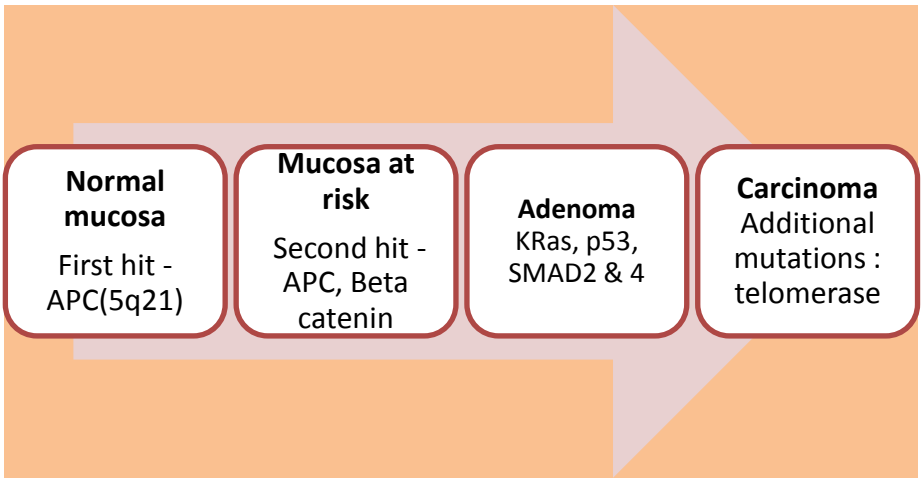
- **GENETICS OF COLORECTAL CARCINOMA :**

The syndromes of FAP and HNPCC are characterised by mutations in APC and DNA mismatch repair genes respectively. The evolution of adenoma to carcinoma and the genetic alterations in the evolution of CRC has been studied by Vogelstein et al[43]. 4 genetic alterations (K-ras, APC, Tp53 and del 18) were studied. Their study showed that alterations in APC, Kras and p53 were the principle events in the initiation, transformation and progression to adenocarcinoma. However this transformation does not involve a single linear pathway. In a study by

Gillian Smith et al[44] 38.7% of tumours had mutations in one of the 3 genes. The most common combination was APC and p53.

Various genes have been implicated at several stages in the progression of adenoma to carcinoma. The important initial event in this neoplastic process is the loss of both copies of APC gene at chromosome 5q21. This causes normal epithelium to transform into adenomatous epithelium. Homozygous loss of p53 is one of the events in the transformation of adenoma to carcinoma, others including LOH of SMAD 2 and 4 genes. This is followed by further additional mutations and chromosomal alterations. This classic model was proposed by Vogelstein and has been propagated for carcinogenesis at other sites also. However, these genetic changes are not always detectable in all the polyps and may not strictly follow the same sequence.

ADENOMA – CARCINOMA SEQUENCE :



- **ROLE OF P53 AND BCL2 IN NEOPLASIA :**

P53 is a tumour suppressor gene located at chromosome 17p13.1 . It acts as the molecular policeman by preventing propagation of genetically damaged cells[45]. P53 gene is the most common gene to be altered in human tumours. Mutations in both alleles of p53 lead to cancer. TP53 acts by causing cells with irreversible DNA damage to progress to apoptosis or senescence. The functions of p53 have been recently found to be regulated by a microRNA called mir34 which also acts on other genes such as cyclins and bcl2.

bcl2 was the first anti-apoptotic gene to be identified and is located at chr18q21. It is located on the outer leaflet of mitochondrial membrane, endoplasmic reticulum and nuclear membrane. Bcl2 family of proteins regulate apoptosis by activating the proteolytic enzymes (caspases). When factors favouring apoptosis act on the cell, bax gene (a member of bcl2 family) causes the exit of cytochrome C from the mitochondria into the cytoplasm. Bcl2 exerts its anti-apoptotic effect by blocking this action of bax. The released cytochrome C activates the caspases.

bcl2 expression in the colonic epithelium is normally restricted to the crypt base where stem cell proliferation takes place. Bcl2 expression is absent in the mature epithelial cells; hence persistent bcl2 expression prolongs life span of cells paving the way for tumorigenesis

INTERACTION BETWEEN P53 AND BCL2:

Wild type p53 causes its pro-apoptotic action by upregulating the transcription of bax gene which counteract the anti-apoptotic action of bcl2.

P53 AND BCL2 IN COLORECTAL NEOPLASMS :

Various studies have been done on the roles of p53 and bcl2, both individual and combined, in colorectal neoplasms.

1. In a study by Bosari et al[46], bcl2 expression was evaluated immunohistochemically in normal colonic mucosa, 24 hyperplastic polyps, 49 adenomas and 205 colorectal carcinomas. In normal colonic mucosa and hyperplastic polyps, bcl2 was positive in proliferating cells of colonic crypts only. Bcl2 positivity was noted in all adenomas irrespective of dysplasia. In CRC, bcl2 was not detected in 50% cases and focal in 38%. Remaining 12% showed diffuse bcl2 reactivity. Bcl2 did not have correlation with both clinic-pathologic parameters of CRC and p53 accumulation and had no prognostic significance. They concluded that bcl2 oncoprotein may play an early role in adenoma-carcinoma sequalae and in established tumors has no prognostic significance.

2. In a similar study by Flohil CC[47] et al of bcl2 expression in hyperplastic polyp, colonic adenomas and CRC, increased bcl2 expression was found in most adenomas (31/35) but not in most carcinomas (7/10), suggesting that the regulation of apoptosis in colorectal epithelia involves additional regulatory factors.

3. Diez et al[48] studied the influence of tumor localization on the prognostic value of p53 protein in colorectal adenocarcinomas and found that p53 overexpression was more frequent in distal than in proximal tumors. Their study showed that p53 positivity and distal tumour location were associated with a higher risk of recurrence.

4. Manne U et al[49] studied the significance of bcl-2 expression and p53 nuclear accumulation in 134 colorectal adenocarcinoma. Nuclear accumulation of p53 was detected in 44% of colorectal adenocarcinomas and was associated with decreased patient survival. . Tumors that did not express detectable levels of bcl-2 but exhibited nuclear accumulation of p53 were associated with the shortest patient survival. . Although a trend toward an inverse correlation between bcl-2 and p53 expression was observed, the prognostic value of bcl-2 expression was independent of p53 status. Thus, assessment of both Bcl-2 and p53 status may be valuable in predicting the prognosis of patients with colorectal adenocarcinomas.

5. Mosnier JF et al[50] studied of expression of bcl-2 and p53 oncogenes in 6 colorectal hyperplastic polyps, 33 adenomas, and 61 carcinomas. bcl-2 was expressed in 28 (85%) of the 33 adenomas, whereas p53 was expressed in only one adenoma, which had areas of in situ carcinoma. bcl-2 and p53 were each expressed in 43 (70.4%) of the 61 carcinomas. Thirty-one (50%) of the colorectal carcinomas coexpressed the two oncoproteins. There was no correlation between the number of cells expressing bcl-2 and the number expressing p53 in a given carcinoma. No correlation was observed between the expression of bcl-2 or p53 and the established prognostic factor. Abnormal bcl-2 oncoprotein expression appears earlier than p53 accumulation in colorectal carcinogenesis. This study suggests that there is more than one sequence and mechanism of bcl-2 and p53 gene deregulation in colorectal carcinomas.

6. Yao et al[51] studied the clinicopathological features and the expression of p53 and bcl-2 proteins in 50 villous tumors in the age group of 32 to 84 years. Their findings were : villous tumours of colon and rectum are more common in females and were common in sigmoid colon and rectum. 74% of villous adenoma showed high grade dysplasia and 34% showed adenocarcinoma with focal or diffuse mucin pools. p53 overexpression was seen in 12% of villous

adenomas and in 18% of invasive component of carcinoma in villous adenoma. bcl2 overexpression was seen in 57% of villous adenomas and 7% of invasive component of villous adenoma. Several characteristic features were recognized in villous tumors, which comprised: (i) a high frequency of coexistence of carcinoma; (ii) multiple foci of carcinomas arising in adenomatous tumors; (iii) a lower histological grade of carcinomas, often with mucin pools; (iv) the existence of extremely well-differentiated adenocarcinomas; and (v) less frequent expression of p53 protein in the carcinomatous components. According to these findings, the pathway of tumor progression in the villous tumors is possibly different from that of sporadic colorectal carcinomas. Because of the peculiarity of villous tumors, careful clinical management is required.

- OTHER POLYPS :

➢ HYPERPLASTIC POLYPS : usually asymptomatic, hyperplastic polyps are common in rectum and distal sigmoid colon. Hyperplastic polyps tend to show spatial clustering as was shown by Cappell MS and Forde KA[52]. In their study on relative polyp location in 426 patients with multiple colonic polyps, they found that hyperplastic polyps were spatially clustered among themselves and with neoplastic polyps

(adenomas). In a case report by Jeevarathnam et al[53], the subject had multiple large hyperplastic polyps and colorectal carcinoma with DNA replication errors. They concluded that giant hyperplastic polyposis is a new familial syndrome pre-disposing to CRC. Histologically, hyperplastic polyps show elongated crypts with serrated pattern. The cells are eosinophilic, columnar with apical mucin, round vesicular nuclei and prominent nucleoli. Lamina propria has increased number of smooth muscles and decreased number of lymphocytes and plasma cells. Multiple hyperplastic polyps have been described in young individuals in whom they are larger and occur throughout the large bowel[54]. Though hyperplastic polyps are generally considered benign, cases have been reported in the past where adenocarcinoma had developed from hyperplastic polyps with adenomatous changes[55].

- INFLAMMATORY POLYPS: These are seen as a complication of inflammatory bowel disease, uretero-sigmoidostomy or mucosal prolapse. The polyp is composed of branching dilated crypts with an oedematous stroma containing inflammatory cell infiltrate.
- JUVENILE POLYPS: Juvenile polyps are common in children and present with rectal bleeding. They are usually solitary but cases with multiple polyps have also been recorded. Juvenile polyps are pedunculated with a spherical head. Histologically, the characteristic

feature is presence of excess edematous lamina propria in the head of the polyp with cystically dilated glands lined by normal epithelium. Juvenile polyp is a pre-malignant condition, cancer risk being 20%.

- Lymphoid polyps are benign lesions occurring most common in rectum. They occur due to local inflammation and are seen in children subsequent to viral infection or due to immune deficiency in familial cases.
- MALIGNANT LYMPHOMATOUS POLYPOSIS/LYMPHOMA : High grade B cell lymphomas are rare in large bowel, most common site being caecum and rectum. Lymphomas of colon and rectum present as multiple polyps which histologically show nodular lymphoid infiltrate of mucosa and superficial submucosa. More than 90% cases show Cyclin D1 (bcl1) positivity.
- PJ polyps, hamartomatous polyps of Cowden's disease, Gastric heterotopias and Malakoplakia are the other polypoid lesions. Cowden syndrome presents with hamartomatous polyps of GIT, tumours of skin, oral mucosa, thyroid and breast. It is an autosomal dominant disease due to mutation of PTEN gene on chromosome 10q.
- MALAKOPLAKIA usually involves sigmoid colon and rectum. GIT is the second most common site for malakoplakia after urinary tract. It presents as polypoid or plaque like lesion showing large eosinophilic

histiocytes containing calcified Michaelis Guttmann bodies. It may be complicating suppurative carcinoma of large bowel.

Since polyps and polypoid lesions are of common occurrence at various sites in the gastrointestinal tract, this study aims at a comprehensive analysis of their features and their association with cancers particularly in the colon.

A total of 60,671 specimens were submitted to the study centre from January 2008 to July 2012. Of these, the study sample consisted of 452 specimens.

The study material included all lesions identified as polyps or polypoid lesions in the gastro-intestinal tract. The specimens included both endoscopic biopsies (polypectomies) and intestinal resection specimens showing such lesions.

Lesions described as nodules or ulcerated proliferating masses were excluded to avoid confusion with other lesions.

The specimens were collected along with relevant clinical details including age, sex, clinical presentation and family history of polyposis or GI cancers. The specimens were fixed using 10% Neutral Buffered Formalin and processed as for routine histopathological studies using H & E stain. Cases with both colorectal adenomas and cancers were present in the same patient were selected for Immunohistochemistry. A control group was selected by taking patients with isolated colorectal adenomas without any evidence of malignancy. The control population was matched with the case group for age, sex, site and type of adenomatous polyp. 21 cases and 16 matched controls were selected and Immunohistochemical studies were done on 5 micron sections of paraffin blocks in which

antigen retrieval was done using Microwave. p53 and bcl2 (Biogenex) antibodies were used and their expression identified using DAB chromogen.

Antigen	Vendor	Species(clone)	Dilution
P53	BIOGENEX	Mouse	Ready to use
Bcl2	BIOGENEX	Rabbit	Ready to use

p53 and bcl2 were considered positive if more than 10% of representative epithelium (adenomatous epithelium) showed positivity[56, 57]. The lymphocytes in lamina propria served as internal control for bcl2.

Statistical correlation was done using Epi info 7 software.

- Total number of polyps and polypoid lesions of Gastro-intestinal tract – 452
- Incidence – 0.745% of biopsies received in the same 4½ years period

TABLE 1 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN THE GASTRO-INTESTINAL TRACT

	ESOPHAGUS	STOMACH	SMALL INTESTINE	LARGE INTESTINE
No. of cases (%)	7(1.55%)	120(26.55%)	56(12.17%)	269(59.53%)
Age range	28 to 65 yrs	16 to 80 yrs	14 to 88 yrs	13 to 85 yrs
Mean age	47.6 yrs	51 yrs	47 yrs	47 yrs
No.of male patients	5	54	45	191
No.of female patients	2	66	11	78
Male:Female ratio	2.5:1	1:1.2	4.1:1	2.5:1
Commonest site And type	Lower end ; Hyperplastic squamous epithelium	Antrum ; Hyperplastic polyp	Duodenum ; Hyperplastic polyp	Left colon ; Adenomatous polyp

	with papillomatosis			

ESOPHAGUS:

TABLE 2 HISTOLOGICAL TYPES OF LESIONS OF ESOPHAGUS

TYPE	FREQUENCY(NUMBER OF CASES)
Hyperplastic squamous epithelium with papillomatosis	3
Barrett's esophagus	2
Fibroepithelial polyp	1
Non-specific inflammation	1
TOTAL	7

STOMACH:

TABLE 3 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN THE STOMACH BASED ON SITE

SITE	NUMBER OF CASES
OGJ & CARDIA	5
FUNDUS	17
BODY	40
ANTRUM	43
PYLORUS	15
TOTAL	120

TABLE 4 DISTRIBUTION OF POLYPS IN STOMACH BASED ON HISTOLOGICAL TYPE

POLYPS	NUMBER OF CASES
Hyperplastic polyps	46
Fundic polyps	2
Inflammatory polyps	7
Inflammatory fibroid polyp	1
Hamartomatous polyp	1
Adenomatous polyp	9

Tubular adenoma Tubulovillous adenoma Villous adenoma	4 4 1
TOTAL	66

TABLE 5 DISTRIBUTION OF POLYPOID LESIONS IN STOMACH BASED ON HISTOLOGICAL TYPE

POLYPOID LESION	NUMBER OF CASES
ADENOCARCINOMA	16
NON-SPECIFIC INFLAMMATION	16
CHRONIC LYMPHOCYTIC GASTRITIS	15
GIST	1
MULTIPLE LESIONS	6
TOTAL	54

TABLE 6 DISTRIBUTION OF HYPERPLASTIC POLYPS IN STOMACH BASED ON SITE

	Hyperplastic polyps Stomach
Antrum	21
Body including lesser curvature	16
Fundus	5
OG junction	1
Pylorus	3
TOTAL	46

- Hyperplastic polyps of stomach were totally 46 cases
- Age group ranged from 35 yrs to 80 yrs with mean age of 47 yrs
- More common in females (26 cases vs. 20 cases in males)
- Most common site – antrum
- 3 cases were pedunculated (6.5%); rest were sessile (93.5%).
- Two cases showed multiple polyps (4.3%); rest were single (95.7%).
- One male patient aged 50 yrs presented with clinical features of peptic ulcer disease and gastric outlet obstruction and was found to have a hyperplastic polyp measuring 1.5 cm diameter situated at the pylorus; another patient, a 70 yr female patient who had

cirrhosis with portal hypertension had a hyperplastic polyp at the fundus; rest of the patients had symptoms suggestive of peptic ulcer disease (epigastric pain, nausea, dyspepsia).

RARE LESIONS IN STOMACH:

1. GIST :

   59 yr/male, HIV positive, polypoidal growth in fundus of stomach with ulceration and GI bleeding; also had an adenomatous polyp of stomach in the past.

2. INFLAMMATORY FIBROID POLYP :

   45 yr/male, polypoid lesion in fundus of stomach; CT scan – suggestive of leiomyoma. Histopathology showed features of Inflammatory Fibroid Polyp.

MULTIPLE LESIONS IN STOMACH:

1. Two cases of adenomatous polyps with adenocarcinoma stomach:
   a) 50 yr female with 1 cm villous adenoma in body of stomach with infiltrating moderately differentiated adenocarcinoma
   b) 60 yr female with 1 cm tubulovillous adenoma of antrum with infiltrating moderately differentiated adenocarcinoma.
2. One case of multifocal adenocarcinoma

3. one case of inflammatory polyp and adenocarcinoma stomach
4. two cases of hyperplastic polyp and inflammatory polyp

SMALL INTESTINE:

TABLE 7 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN SMALL INTESTINE BASED ON SITE

	SITE DISTRIBUTION
DUODENUM	48 (85.72%)
JEJUNUM	4 (7.14%)
ILEUM	4 (7.14%)
TOTAL	56

TABLE 8 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN SMALL INTESTINE BASED ON HISTOLOGICAL TYPE

POLYPS	NUMBER OF CASES
Hyperplastic polyp	15
Inflammatory polyp	2
Inflammatory fibroid polyp	1
Peutz-Jeghers polyp	2

Adenomatous polyp Tubular adenoma Tubulovillous adenoma	4 2 2

POLYPOID LESIONS	NUMBER OF CASES
Adenocarcinoma	2
Brunner gland hyperplasia	3
Chronic duodenitis	3
Non-specific inflammation	13
GIST	1
Pseudopolyp	2
Carcinoid	1
Multiple lesions	7
TOTAL(polyps & polypoid lesions)	56

RARE LESIONS IN SMALLL INTESTINE:

1. GIST: 40 yr/female, polypoid lesion in duodenum; grossly, pedunculated mass measuring 6 cm diameter with 1 cm stalk; cut surface – encapsulated, solid, grey white. Microscopy showed features of GIST intermediate grade.

2. PEUTZ-JEGHER'S POLYP: 2 cases: 30 yr/female, ileal polyp presented with intussusception ; 35 yr/male, presented with melena; found to have jejunal polyp.
3. PSEUDOPOLYPS: 2 cases: 65 yr male and 65 yr female patients; both had duodenal polyps. Microscopic examination showed inflammatory pseudopolyps (no past history of Inflammatory Bowel Disease).
4. CARCINOID: 65 yr/ male; carcinoid tumour presented as sessile polyp in first part of duodenum (measuring 1cm x 0.5cm x 0.5cm).
5. INFLAMMATORY FIBROID POLYP: 65 yr/male; ileoileal intussusception with ileal polyp (pedunculated,3 cm diameter, with stalk 1 cm length); polyp appeared gangrenous; Histopathological examination showed features of inflammatory fibroid polyp with torsion.

MULTIPLE LESIONS:

- Inflammatory polyps duodenum occurring along with 1.Adenocarcinoma stomach 2.Squamous cell carcinoma esophagus.
- hyperplastic polyp with adenocarcinoma stomach.
- adenomatous polyp duodenum with adenocarcinoma stomach.

LARGE INTESTINE:

TABLE 9 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN THE LARGE INTESTINE BASED ON SITE

SITE	NUMBER OF CASES
CAECUM	24
COLON	
1. Ascending, Transverse, Descending, Sigmoid	94
2. Rectosigmoid	21
3. Multifocal	13
4. Unspecified(Inadequate Data)	18
RECTUM	70
ANAL CANAL	29
TOTAL	269

TABLE 10 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN THE LARGE INTESTINE BASED ON SITE (RIGHT VS LEFT COLON)

RIGHT COLON		LEFT COLON	
CAECUM	24	SPLENIC FLEXURE	4
ASCENDING COLON	13	DESCENDING COLON	26
HEPATIC FLEXURE	3	SIGMOID COLON	34
TRANSVERSE COLON	14	RECTUM RECTOSIGMOID	70 21
TOTAL	54 (20.07%)	TOTAL	159(59.12%)

ANAL CANAL	29
BOTH RIGHT AND LEFT COLON (MULTIFOCAL)	9
DATA INADEQUATE	18

LEFT COLON + ANAL LESIONS : 188 (69.89%)

TABLE 11 DISTRIBUTION OF POLYPS AND POLYPOID LESIONS IN THE LARGE INTESTINE BASED ON HISTOLOGICAL TYPE

Polyp/polypoid lesion	Number	Percentage
Hyperplastic polyps	42	15.61
Inflammatory polyps	34	12.63
Hamartomatous polyps a. Peutz jeghers polyp b. Juvenile polyp c. Retention polyp	13 2 5 6	4.83
Adenomatous polyps	120	44.61
Mesenchymal polyps a. Lipomatous polyps b. Fibroepithelial polyp c. Angiomatous polyp	20 4 15 1	7.43
Malignancies: a. Adenocarcinoma b. NHL c. Malignant melanoma	27 25 1 1	10.04
Pseudopolyps	11	4.09

a. Inflammatory b. Ulcerative colitis	7 4	
Others -Crohn's disease	2	0.74
Total	269	

TABLE 12 COMPARISON OF MAJOR TYPES OF POLYPS OF LARGE INTESTINE (OTHER THAN ADENOMATOUS POLYPS)

	HYPERPLASTIC POLYP	INFLAMMATORY POLYP	JUVENILE POLYP
TOTAL (%)	42(15.6%)	34(12.6%)	11(4.1%)
MEAN AGE	51 yrs	46.5 yrs	33.7 yrs
MALE/FEMALE	6:1	2.8:1	10:1
LOCATION	Caecum – 5 Right colon – 5 Left colon – 13 Rectum – 15 Anal canal - 4	Caecum – 7 Right colon-3 Left colon – 6 Rectum – 10 Anal canal-1 Data inadequate-7	Rectum -7 Sigmoid colon-1 Anal -1 Hepatic flexure-1 multifocal-

			1
MULTIPLE POLYPS	-	1	1 case
ASSOCIATED CANCERS	5 1-anal squamous cell carcinoma 4-adenocarcinomas (3-rectal 1-transverse colon)	2 Both adenocarcinomas colon	None

SYNCHRONOUS LESIONS OF COLON AND STOMACH– 2 CASES:

28 yr/ male, Hyperplastic polyp stomach & Tubulovillous adenoma colon; 61 yr/male, GIST stomach & Adenocarcinoma Colon

SYNCHRONOUS LESIONS OF COLON AND SMALL INTESTINE – 2 CASES:

19 yr/male, Inflammatory polyps of small and large intestine; 68 yr/male, chronic lymphocytic duodenitis & hyperplastic polyp of transverse colon

TABLE 13 ADENOMATOUS POLYPS OF LARGE INTESTINE

Polyp	No Malignancy	Malignant Features Seen	With Non-Contiguous Cancer	Associated With Other Lesions	Total (Percent)
TA	54	1	5	4 3-inflammation 1-TB	64 (53.33%)
TVA	25	3	8	2 1-Hyperplastic polyp 1-ulcerative colitis	38 (31.67%)
VA	11	1	3	1-inflammation	16 (13.33%)
FA	2				2(1.67%)
	92(76.7%)	5(4.2%)	16(13.3%)	7(5.8%)	120

TA – Tubular Adenoma; TVA – Tubulovillous Adenoma

VA – Villous Adenoma; FA – Flat Adenoma

Totally 5 cases of multiple (more than 100 polyps) adenomatous polyps in colon were noted (Table 14). Two of these patients showed malignant transformation. No definite positive family history was obtained in these patients.

TABLE 14 ADENOMATOUS POLYPOSIS COLI

	Age	Sex	Predominant Type of polyp	Malignant change
1.	30	Female	Villous adenomas	Present
2.	35	Female	Tubulovillous adenomas	Present
3.	39	Male	Tubular adenoma	Absent
4.	42	Male	Tubular adenoma	Absent
5.	65	Male	Tubular adenoma	Absent

TABLE 15 POLYPS SHOWING MALIGNANT CHANGE

AGE/SEX	ADENOMA	CARCINOMA
54/F	Tubular	Poorly differentiated signet ring cell carcinoma
79/M	Tubulovillous	Well differentiated adenocarcinoma
70/M	Tubulovillous	Well differentiated adenocarcinoma

56/F	Tubulovillous	Moderately differentiated adenocarcinoma
78/F	Villous	Well differentiated adenocarcinoma

TABLE 16 DETAILS ABOUT POLYPS WITH CO-EXISTING COLONIC CANCERS

ADENOMA	ADENOCARCINOMA			TOTAL
	Well Differentiated	Moderately Differentiated	Poorly Differentiated	
Tubular	2	3	0	5
Tubulovillous	3	4	1	8
Villous	1	1	1	3
	6(37.5%)	8(50%)	2(12.5%)	16

IMMUNOHISTOCHEMISTRY STUDIES:

TABLE 17 P53 EXPRESSION IN ADENOMATOUS POLYPS, CO-EXISTING ADENOCARCINOMAS AND CONTROL GROUP

	P53 positive	P53 negative	Total
COMPARATIVE POLYPS	7 (43.75%)	9 (56.25%)	16
POLYPS WITH NEARBY CANCER	13 (61.9%)	8 (38.1%)	21
CANCERS	16 (76.2%)	5 (23.8%)	21
TOTAL	36	22	

TABLE 18 CHI SQUARE TEST FOR P53 (COMPARING POLYPS WITH AND WITHOUT SYNCHRONOUS CANCERS)

	CHI-SQUARE VALUE	2 TAILED P
UNCORRECTED	1.2052	0.2722944784
MANTEL-HAENSZEL	1.1726	0.2788717700
CORRECTED	**0.5850**	**0.4443568801**

Chi square value is 0.5850 (p=0.4443), the association between p53 expression and risk of malignancy in adenomatous polyp is not statistically significant.

TABLE 19 ODD BASED PARAMETERS FOR P53

	ESTIMATE	LOWER	UPPER
ODDS RATIO	2.0893	0.5562	7.8476
MLE ODDS RATIO(MID P)	2.0470	0.5362	8.1491
FISCHER-EXACT		0.4622	9.6047

TABLE 20 RISK BASED PARAMETERS FOR P53

	ESTIMATE	LOWER	UPPER
RISK RATIO	1.3813	0.7595	2.5119
RISK DIFFERENCE	17.9412	-13.6811	49.5635

TABLE 21 BCL2 EXPRESSION IN ADENOMATOUS POLYPS, CO-EXISTING ADENOCARCINOMAS AND CONTROL GROUP

	bcl2 positive	bcl2 negative	Total
COMPARATIVE POLYPS	10 (62.5%)	6(37.5%)	16
POLYPS WITH NEARBY CANCER	15 (71.4%)	6 (28.6%)	21
CANCERS	6 (28.6%)	19 (71.4%)	21
TOTAL	31	31	

TABLE 22 CHI SQUARE TEST FOR BCL2 (COMPARING POLYPS WITH AND WITHOUT SYNCHRONOUS CANCERS)

	CHI-SQUARE VALUE	2 TAILED P
UNCORRECTED	0.3304	0.5654493732
MANTEL-HAENSZEL	0.3214	0.5707508900
CORRECTED	**0.0485**	**0.8256164151**

Chi square value is 0.0485(p=0.8256) hence bcl2 expression is not correlated with risk of malignancy in adenomatous polyps.

TABLE 23 ODD BASED PARAMETERS FOR BCL2

	ESTIMATE	LOWER	UPPER
ODDS RATIO	1.5000	0.3751	5.9978
MLE ODDS RATIO(MID P)	1.4834	0.3548	6.2657
FISCHER-EXACT		0.2995	7.4483

TABLE 24 RISK BASED PARAMETERS FOR BCL2

	ESTIMATE	LOWER	UPPER
RISK RATIO	1.2000	0.6264	2.2988
RISK DIFFERENCE	10.0000	-24.1925	44.1925

INCIDENCE:

Polyps of gastrointestinal tract have varying incidence depending on the site of the lesion. Esophageal polyps are very rare whereas polyps in the stomach and colon are relatively common. This study is hospital based and does not reflect the true incidence of gastrointestinal polyps. Gastrointestinal polyps and polypoid lesions constituted 0.745% of biopsies received at the tertiary care center. The majority of lesions are situated in the large intestine, constituting 59.53% of cases followed by the stomach which constituted 26.55% of cases. The incidence of gastric polyps is reported as 0.4% of autopsy cases[58] and 5% of endoscopic studies[59, 9]. Small intestinal lesions, especially ileal polyps are being increasingly detected in the early stages due to the advent of new endoscopic techniques such as small bowel push endoscopy and video capsule endoscopy. Large intestinal lesions have remained the predominant population with most of them being adenomatous polyps. True incidence of these lesions is lacking. Table 25 shows a comparison of autopsy based studies of large intestinal polyps in various countries.

TABLE 25 AUTOPSY BASES STUDIES OF LARGE INTESTINAL POLYPS

Study	Place	Total no.of cases	Predominant polyp	n(%) of total
Jass JR et al[60]	New Zealand	495	Hyperplastic polyps	252(50.9%)
Williams et al[61]	Liverpool	843	Hyperplastic polyps	574(68.1%)
Rickert RR[62]	New Jersey	1023	Adenomatous polyp	658(64.32%)
Vatn MH[63]	Oslo	445	Adenomatous polyp	329(73.93%)
Lee YS[64]	Singapore	1,014	Adenomatous polyp	170 (16.8%)
Tony et al[65]	India	124	Adenomatous polyp	99(79.8%)

As can be noted, the incidence and type of colorectal polyp varies in different populations across the world. In our study, adenomatous polyps were commonest, almost 2.7 times commoner than hyperplastic polyps.

PATHOGENESIS :

The pathogenesis of polyps is reflected in the classification of polyps. Hyperplastic polyps arise in a background of chronic inflammation, hence they are common in the stomach in the setting of chronic atrophic gastritis. Hamartomatous polyps are composed of mixture of mature tissues at their indigenous site. Inflammatory polyps arise in a setting of chronic cycles of injury and healing, such as in solitary rectal ulcer syndrome. Neoplastic polyps may arise de novo or may be a component of Familial Adenomatous Polyposis syndromes.

LESIONS OF ESOPHAGUS :

Polyps and polypoid lesions of esophagus are uncommon though malignancies are associated with polypoid presentation. In our study, three cases showed hyperplastic squamous epithelium and two cases with features of Barrett's esophagus. No malignant polypoid lesions were seen in our study. Squamous hyperplasia is considered as early histologic manifestation of Gastro-esophageal Reflux disease, even in the absence of macroscopic esophagitis[66]. The interpretation of early changes is subject to inter and intra-observer variation and also depends on number and topography of biopsies and technical aspects.

Barrett's esophagus is defined as endoscopically recognized columnar metaplasia of esophageal mucosa which is confirmed pathologically by the presence of goblet cells[67]. It is more common in males; in our study of two cases, equal incidence was seen and both presented with epigastric pain. Endoscopically, the lesion is identified by salmon coloured mucosa with focal ulceration or erosion. Dysplasia in Barrett's esophagus presents as flat, nodular or polypoid lesions[68] and are called as adenomas. In our study, both cases showed intestinal metaplasia and no evidence of dysplasia was seen.

LESIONS OF STOMACH :

Polyps and polypoid lesions of stomach constitute 26.55% of total cases in our study. The age group ranged from 16 yrs to 80 yrs with a mean age of 51 yrs. In our study, female patients outnumbered males in the ratio 1.2:1. The commonest site of involvement was antrum and the commonest lesion was hyperplastic polyp.

The predominant polyp in our study was hyperplastic polyp. Hyperplastic polyps, according to literature, are commonly seen in the antrum and reflected in our study also. They present as single or multiple lesions- solitary lesions were common in our study. Grossly they appear as oval elevations of mucosa with smooth contours. They may be sessile

or pedunculated, sessile lesions constituting 93.5% of cases in our study. One polyp of size 1.5 cm was associated with gastric outlet obstruction. Rare cases of hyperplastic polyp simulating hypertrophic pyloric stenosis in pediatric age group[69] and causing gastric outlet obstruction in an adult female patient[70] have been reported. Histologically, hyperplastic polyps showed hyperplastic and elongated foveolae with corkscrew appearance, lined by columnar epithelium and cystically dilated glands with mild to moderate inflammation in the stroma. Some polyps also showed intestinal metaplasia and duct-like structures with cuboidal epithelium and eosinophilic granular cytoplasm. Two cases of hyperplastic polyps co-existing with inflammatory polyps were seen in our study. The incidence of malignancy in hyperplastic polyps is reported to range from 1.5% to 4.5%[71,72]. No cases of hyperplastic polyposis or hyperplastic polyps co-existing with carcinoma stomach were seen in our study, showing that malignant transformation of hyperplastic polyps are very rare. The significance of identifying hyperplastic polyps lies in identifying associated gastric pathology.

Inflammatory polyps constituted 10.6% of gastric polyps. Fundic gland polyps and inflammatory fibroid polyps were seen in less numbers. Adenomatous polyps constituted 13.6% with 2 of them in female patients associated with infiltrating adenocarcinoma of stomach.

LESIONS OF SMALL INTESTINE:

Small intestinal polyps are being increasingly detected using various techniques such as capsule endoscopy and single/double balloon endoscopy. Various studies have shown different distribution of polyps. Most of the previous studies of small intestinal polyps are confined to the duodenum or the distal ileum due to lack of investigative modalities to access the entire small intestine. The predominant site of involvement in this study was the duodenum, constituting 85.72% of cases of small intestine. Hyperplastic polyps were the most common, followed by inflammatory polyps. 2 cases of Peutz-Jeghers polyps were recorded. Hyperplastic polyps were all seen in the duodenum. A large Japanese study[73] showed that 15 out of 25 lesions seen in duodenum were Hyperplastic polyps. In our study, all the patients with hyperplastic polyps were males, with one female patient in whom, it was seen as a verrucous polypoid lesion. The youngest age of presentation was 17 yr male patient who had presented with acute small bowel obstruction and incidentally, a hyperplastic polyp was found in the resected gangrenous segment of bowel. This study shows a possible younger age of onset of hyperplastic polyps and predominance in males.

Peutz-Jeghers polyps may be sporadic or syndromic, with sporadic cases reported in various sites such as duodenum[74], stomach[75], jejunum[76] and rectum[77]. Peutz Jeghers syndrome is defined by the following criteria: (a) three or more histologically confirmed Peutz-Jeghers polyps (b) any number of Peutz-Jeghers polyps with a family history (c) characteristic mucocutaneous pigmentation with a family history or (d) any number of Peutz-Jeghers polyps and characteristic mucocutaneous pigment. PJ polyps are the commonest hamartomatous polyps of the small intestine, with majority in the jejunum, followed by ileum and duodenum. Solitary or multiple, they present with GI bleed, anemia, abdominal pain and recurrent intussusception. In our study, PJ polyps of small intestine were rare, only two cases were seen : a 30 yr old female patient who had ileal PJ polyp and presented with intussusception and a 35 yr male patient with jejunal PJ polyp who had melena. As noted, both were in 4th decade and symptomatic. No features of Peutz Jeghers syndrome were seen. The existence of sporadic PJ polyps has been questioned as they have been associated with development of carcinoma at other sites during follow up studies[78].

LESIONS OF LARGE INTESTINE:

The predominant population of colonic polyps in this study was formed by adenomatous polyps(44.61% of colonic polyps). Of these, 53.3%(64) were tubular adenomas which are the commonest type of colonic polyps as seen in other studies also (TABLE 26).

TABLE 26 VARIOUS STUDIES OF ADENOMAS OF LARGE INTESTINE

AUTHOR LOCATION	Tubular adenoma	Tubulo- villous adenoma	Villous adenoma	(A)	(B)	(C)
Rahat N et al[79] KARACHI	10 (55.55%)	3 (16.67%)	5 (27.78%)	1 (5.6%)	-	18
Ali Zare Mirzaie et al[80] IRAN	121 (69.94%)	24 (13.87%)	28 (16.18%)	-	-	173
Nouraie et al[81] AFRICAN-AMERICAN	3641 (86.57%)	414 (9.84%)	151 (3.59%)	-	-	4206

Tony J et al[65] INDIA	67 (68%)	24 (24%)	14 (14%)	6 (6.06%)	1 (1.01%)	99
A Khan et al[82] CANADA	566 (65%)	225 (25.8%)	63 (7.2%)	12 (1.4%)		871
Present study INDIA	64 (53.33%)	38 (31.67%)	16 (13.33%)	5 (4.2%)	16 (13.33%)	120

(A) – polyps showing malignancy (invasion across muscularis mucosa);

(B) – polyps associated with synchronous non-contiguous malignancy;

(C) – total cases studied

The mean age of colonic adenomatous polyps (49 yrs) is lesser than that of another South Indian study in which the same is 58.1 yrs. The male female ratio and type distribution are similar. The type distribution is similar to other studies worldwide as shown in table 26 , tubular adenomas being the commonest followed by tubulovillous adenomas with the exception of two studies(Karachi and Iran) in which villous adenomas are more common than tubulovillous adenomas. The size of the polyps were not available for all cases in the present study and hence was not

analysed. 2 cases of Flat adenomas which endoscopically showed raised mucosa resembling tiny polypoid lesions were noted. Microscopically only adenomatous changes with no infiltration were seen.

5 cases of adenomatous polyposis coli were noted in which entire colon was studded with more than 100 polyps. These patients belonged to a wide age range from 30 yrs to 65 yrs. Notably, all were in late adulthood and no positive family history was obtained. Two of these patients showed malignant transformation. Familial adenomatous polyposis presents in childhood or adolescent or early adulthood with thousands of polyps in colon as well as in other parts of gastrointestinal tract. By definition more than 100 polyps should be present. Genetic studies in FAP show loss of both copies of APC gene on chromosome 5q. Proctocolectomy was done in these 5 cases.

ADENOMATOUS POLYPS WITH MALIGNANT CHANGES AND NON-CONTIGUOUS MALIGNANCIES:

A malignant adenomatous polyp by definition shows malignant epithelium invading across muscularis mucosa[83,84,85]. The incidence of malignant change in adenomatous polyps varies from 1.4% in Canada to 6.06% in South India. The proportion of such polyps in our study (4.2%) is similar to Tony et al and is higher than those of Western studies.

16 cases (13.33%) showed presence of adenomatous polyps and non-contiguous colonic cancers. The age range is variable from 22 yrs to 70 yrs; males were more commonly affected. The predominant lesions were left sided. All the three types of adenomatous polyps are found to be associated with cancers. All three grades of Cancer were also noted to be associated with polyps.

This study shows a higher percentage of adenomatous polyps co-existing with colonic cancers when compared to other Indian as well as Western studies. The incidence of adenomatous polyps parallels the incidence of Colonic cancers. Adenomas are considered as epidemiological indicators of increased risk of cancers. Western studies show a high colonic cancer incidence(United States – male : 40.6 females : 30.7 per 1,00,000 population) when compared to India(males : 4.7 females : 3.2 per 1,00,000 population) as per GLOBOCAN 2000 data[34]. The current study has covered the period of 2008 to 2012, which indicates that incidence of adenomatous polyps in this part of India is increasing and also suggests an increasing trend in the incidence of colonic cancer, especially left sided colonic cancers.

The second major population in this study was hyperplastic polyps which constituted 15.6% cases. The average age is 51 yrs similar to that

in literature. Hyperplastic polyps of large intestine are uncommon before the fifth decade[60] and are seen more commonly in the rectum and sigmoid colon. Left sided lesions were more common in this study (69.89%) when compared to right sided lesions (20.07%). A male predominance was also seen.

According to various studies, hyperplastic polyps are

1) possibly neoplastic lesions due to demonstration of k-ras mutations and clonal changes involving chromosome 1 which is also seen in adenomas

2) more commonly associated with adenomas

3) presence of distal hyperplastic polyp indicates risk for proximal adenoma (debated)

4) share the same risk factors as for adenomas and carcinomas.

However, in this study,

1) adenomas were three times commoner than hyperplastic polyps

2) no cases of both types of colonic polyps were recorded in the same patient

3) 5 cases(11.9%) out of 42 cases co-existed with large intestinal cancers.

These results indicate that hyperplastic polyps are less common when compared to adenomatous polyps in this part of the country and do not serve as indicator for presence of adenomas. The definite role of hyperplastic polyps in colonic cancers requires molecular studies even though 11.9% of hyperplastic polyps showed association with cancer.

12.6% of colonic polyps were inflammatory polyps. 11 cases (4.09%) were pseudopolyps of which 7 were non-specific inflammatory origin and 4 were associated with Inflammatory Bowel Disease.

Hamartomatous polyps constituted 4.83% (11 cases) of large intestinal lesions in this study. Of these, 2 cases were Peutz-Jeghers polyps while remaining 11 cases were juvenile polyps. Juvenile polyps were solitary sporadic lesions in our study except for one case in which it was multiple. The remaining 10 patients showed predominant involvement of left colon, especially rectum. Juvenile polyps are commonly seen in the paediatric age group, but also reported in adults, with the incidence beyond 20 yrs reported between 0 and 40%[86]. They present with usually with melena. Similar to other studies, there were no colonic malignancies associated with juvenile polyps in this study.

P53 AND BCL2 IN COLORECTAL ADENOMATOUS POLYPS AND THEIR RELATION TO CANCER:

Various individual studies of p53 and bcl2 expression in colorectal polyps and cancers have been done. Results have been consistent with p53 and more variable with bcl2. Shanmugam et al[56] have studied the expression of p53 and bcl2 in normal colonic epithelium, contiguous colorectal adenomas and cancers and found that expression of p53 and Bcl-2 progressively increased from normal-appearing epithelium to adenomas to carcinomas. They also concluded that the presence of p53 in the adenomatous epithelium is an indicator of aggressive behavior of colonic lesions, and that these patients are more likely to develop aggressive invasive cancer.

In the current study, the expression of p53 also shows similar progressive increase from adenomatous polyps without synchronous colonic malignancy to those with synchronous malignancies to frank colonic malignancies. However, the difference between p53 expression in adenomatous polyps with and without synchronous malignancies has not been statistically significant. This could be possibly due to the small sample size in both the groups. A higher proportion of adenomatous polyps with synchronous colonic malignancies have shown p53

expression than those without synchronous malignancies(61.9% vs. 43.75%). The progressive increase in p53 expression has been noted in other human studies and has been proved in animal models but may be difficult to prove directly in humans as most polyps detected during endoscopy are removed by polypectomy. However, the increased expression of p53 can serve as an indication for placing such patients under high risk group with need for surveillance. Considering these findings, the time span for progression of an adenoma to carcinoma and the use of p53 as a predictive marker for malignancy in an adenomatous polyp requires more extensive research.

Bosari et al[46] found diffuse bcl2 positivity in all colorectal adenomas and negative in 50% colorectal cancers. He also found that bcl2 expression was not correlated with p53 expression and had no prognostic significance. The expression of bcl2 has been variable in this study, with maximum expression in polyps associated with cancers than in cancers themselves or the comparative group of polyps. Mutations in p53 and expression of bcl2 in mature epithelium (which is abnormal) both favour the abnormal cells to survive. The different expressions of bcl2 in the three groups in this study indicate possible interactions between bcl2 and other genes involved in carcinogenesis. Table 27 shows the various results of bcl2 expression in various studies.

TABLE 27 BCL2 EXPRESSION IN ADENOMATOUS POLYPS AND COLORECTAL CARCINOMAS IN VARIOUS STUDIES

Author	Bcl2 in adenomatous polyps – proportion of cases positive	Bcl2 in cancers - proportion of cases positive
Bosari et al[46]	100%	50%
Nakamura et al[87]	80%	30%
Husain A Saleh et al[88]	51.9%	78.6%
Nigel Scott et al[89]	95%	35%

Studies show bcl-2 expression is seen in various pre-malignant and malignant lesions. Hence, it is suggested that bcl-2 genetic alterations are seen early in the pathway of carcinogenesis. bcl 2 expression is said to have a favorable prognosis in breast and lung carcinomas and unfavorable in prostate carcinoma[90]. bcl-2 expression in colorectal carcinomas is found to be associated with a better clinical course especially in the absence of p53 expression indicating that neoplasia caused by inhibition of apoptosis may cause less aggressive malignancies than those caused by

other oncogenes like p53 and K-ras[46,91,92]. An inverse relationship has been noted between bcl-2 and p53 expression in many malignancies, suggesting that these proteins may interact through opposite mechanisms: inhibition of apoptosis (bcl-2), and promotion of apoptosis (p53)[92,93].

STRENGTHS AND LIMITATIONS OF THE STUDY:

STRENGTHS :

1. The study analyses a large number of cases received at a tertiary care centre in the country.
2. All types of polyps and polypoidal lesions of gastro-intestinal tract are analyzed.
3. Important genetic markers have been studied in both pre-malignant and malignant lesions.
4. The genetic markers studied include bcl2 and p53, whose roles in adenoma-carcinoma sequence are respectively in early and late stages. Hence both early and late changes are studied with respect to the two genes.

LIMITATIONS :

1. The study is hospital based; so true incidence and prevalence in the community is not known.
2. Clinical details namely symptoms and size of polyp were not available in all cases and not analyzed.
3. Follow-up of patients is not analyzed.

- Polyps and polypoid lesions of gastro-intestinal tract constitute 0.745% of all specimens submitted for histopathological examination at a tertiary care centre over four and a half year period.
- Majority of cases involved the large intestine constituting 59.53% of total.
- The second commonest site of involvement was the stomach (26.55%).
- The age group ranged from 13 yrs to 88 yrs.
- A male predominance was noted in all the sites except for stomach where females outnumbered males.
- The esophageal lesions were commonest at the lower end of esophagus and were predominantly changes of GERD; Barrett's esophagus was also seen.
- The stomach lesions were commonly hyperplastic polyps seated at the antrum. Rarer polypoid lesions encountered in the stomach include GIST and inflammatory fibroid polyp.
- Duodenum was the predominant site of involvement in the small intestine (85.72%). Hyperplastic polyps were the commonest polyps seen.
- Other less frequent lesions seen in the small intestine were Gastrointestinal stromal tumour, pseudopolyps, carcinoid, inflammatory

fibroid polyp (presented with torsion and intussusception) and Peutz-Jeghers polyp (presented with intussusception).

- Maximum number of lesions were seen in the colon, especially left colon (59.12%) with the rectum being the commonest site.
- Adenomatous polyps constituted the bulk of large bowel polyps (44.61%).
- Hyperplastic polyps were the second commonest (15.71%).
- Among the adenomatous polyps, tubular adenomas constituted 53.33%, tubulovillous adenomas – 31.67%, villous adenomas – 13.33%.
- Adenomatous polyps were seen in a wide age group of 22 yrs to 70 yrs.
- 5 cases of adenomatous polyps showed malignant change (4.2%).
- 5 cases showed multiple (more than 100) adenomatous polyps involving the large intestine. They were in the 4^{th} to 7^{th} decade; 2 of them showed malignant transformation.
- 16 cases of adenomatous polyps (13.33% of adenomatous polyps) were associated with non-contiguous colonic cancers which is much higher than other studies worldwide.
- P53 expression was found to be higher in polyps with contiguous or non-contiguous malignancies (61.9%) than in polyps without them (43.75%). They were much higher in the co-existing cancers (76.2%).

- However, P53 expression was not significantly correlated with risk of malignancy in adenomatous polyps (p=0.5850).
- Bcl2 expression was found to be variable in the three groups and was not significantly correlated with adenomatous polyps with co-existent cancers(p=0.825).

Polyps and polypoid lesions in the gastrointestinal tract may vary from asymptomatic incidental findings and benign harmless lesions to invasive malignancies. Various investigatory modalities are being developed and available in developing countries for screening and diagnosis of these lesions. The morphology of the polyps are well defined to delineate them from one another. A careful study of adenomatous polyps is needed in view of potential for malignant transformation. This study finds a high incidence of colonic adenomatous polyps with synchronous colonic malignancies. The study is hospital-based and may not represent the true incidence of the disease in the community. Hence, community based studies are essential to assess the prevalence and risk factors for adenomatous polyps. Though surveillance programs have been framed, it is imperative to establish cost effective screening guidelines so as to detect the lesions earlier. As most of the lesions are left-sided they would be more accessible with colonoscopy techniques. Genetic studies are needed to establish predictive and prognostic markers for malignant transformation of adenomatous polyps. Future studies should be community-based with assessment of possible etiological and/or risk factors.

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CHARTS

CHART 1. DISTRIBUTION OF POLYPS IN STOMACH BASED ON SITE

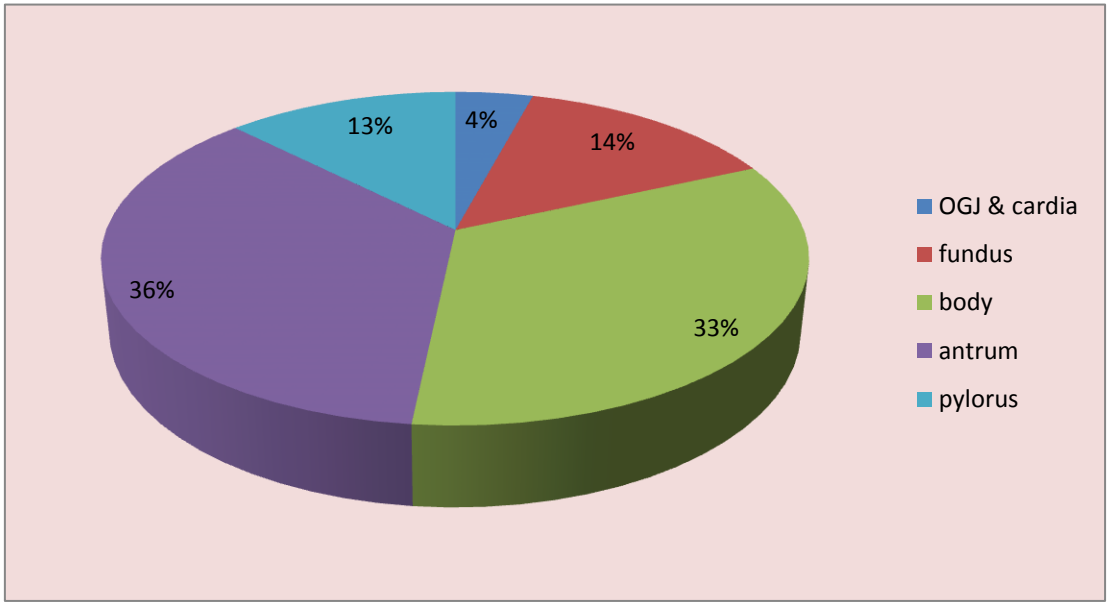


CHART 2. DISTRIBUTION OF HYPERPLASTIC POLYPS OF STOMACH

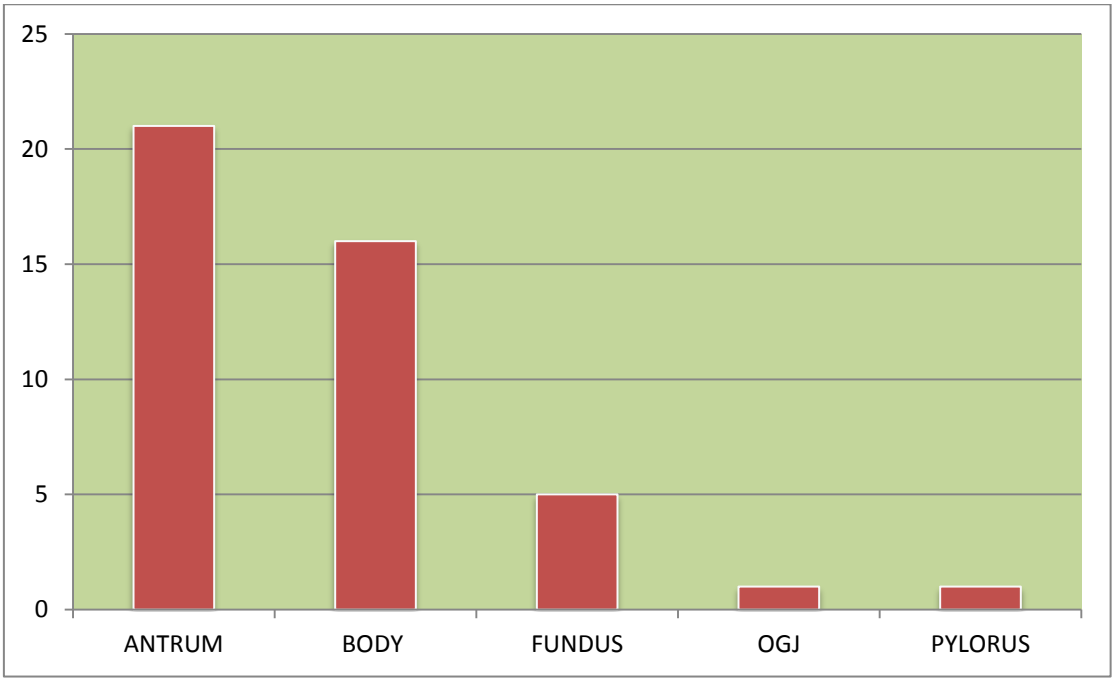


CHART 3. DISTRIBUTION OF POLYPS OF SMALL INTESTINE BASED ON SITE

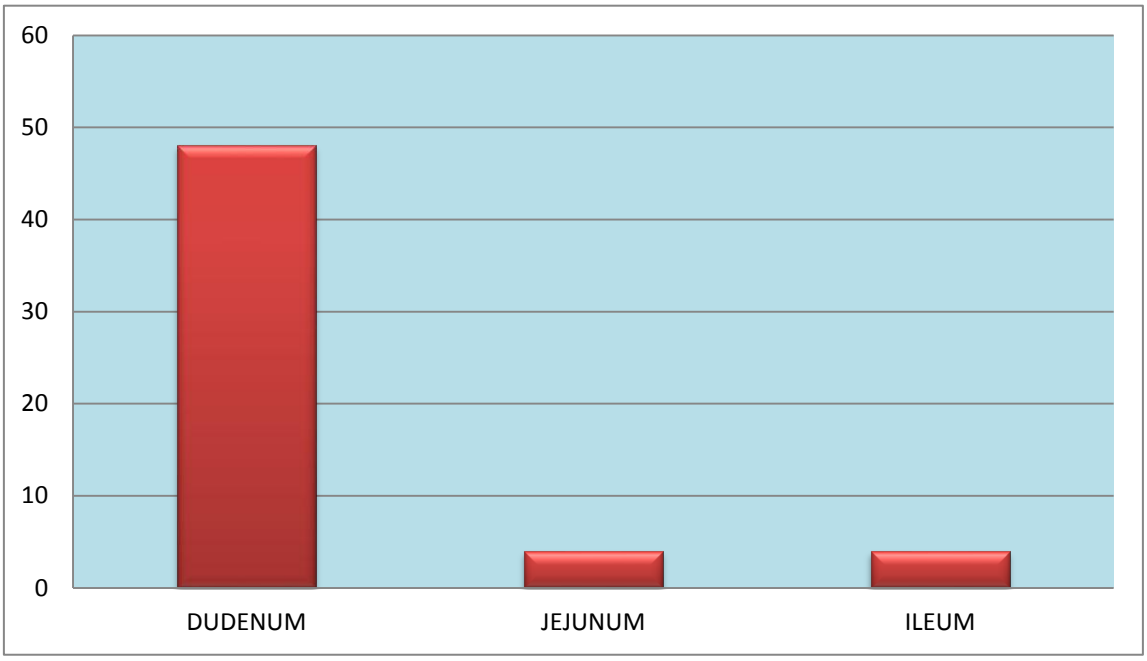


CHART 4. DISTRIBUTION OF POLYPS OF SMALL INTESTINE BASED ON TYPE

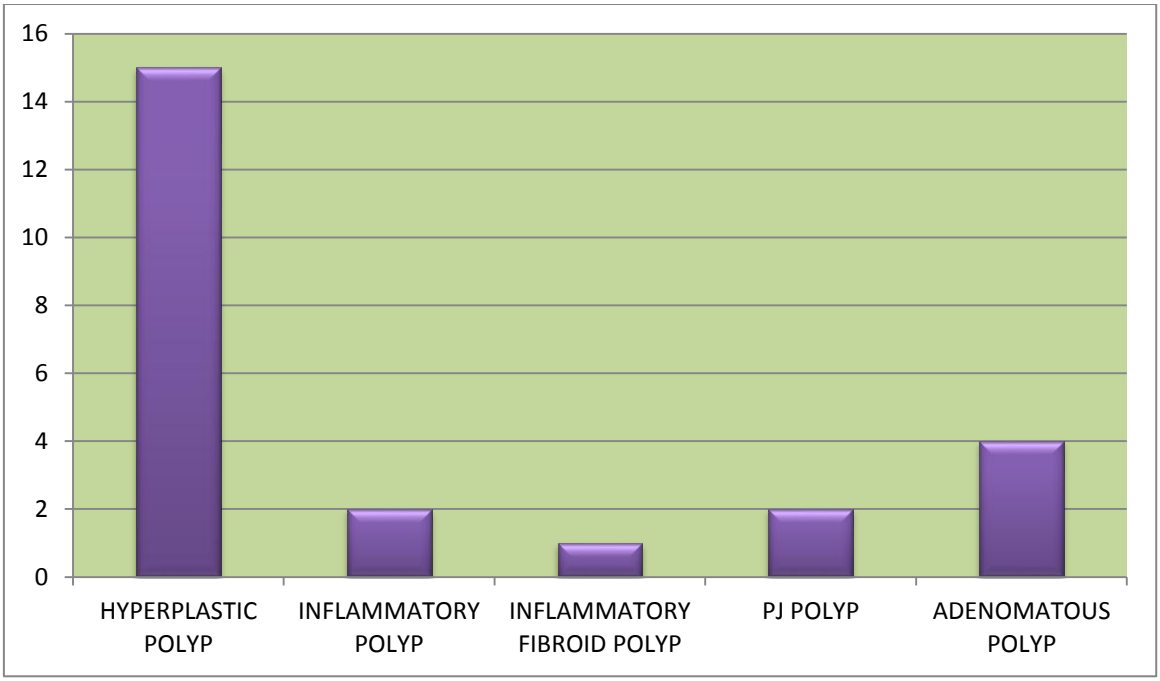


CHART 5. DISTRIBUTION OF POLYPS OF LARGE INTESTINE BASED ON SITE

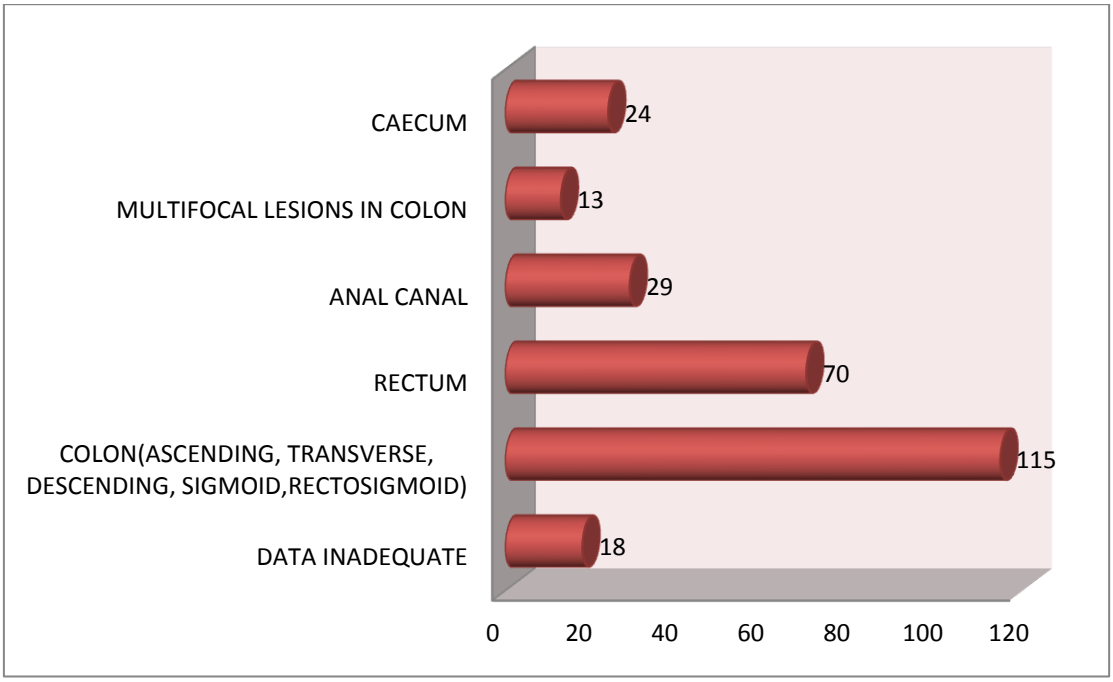
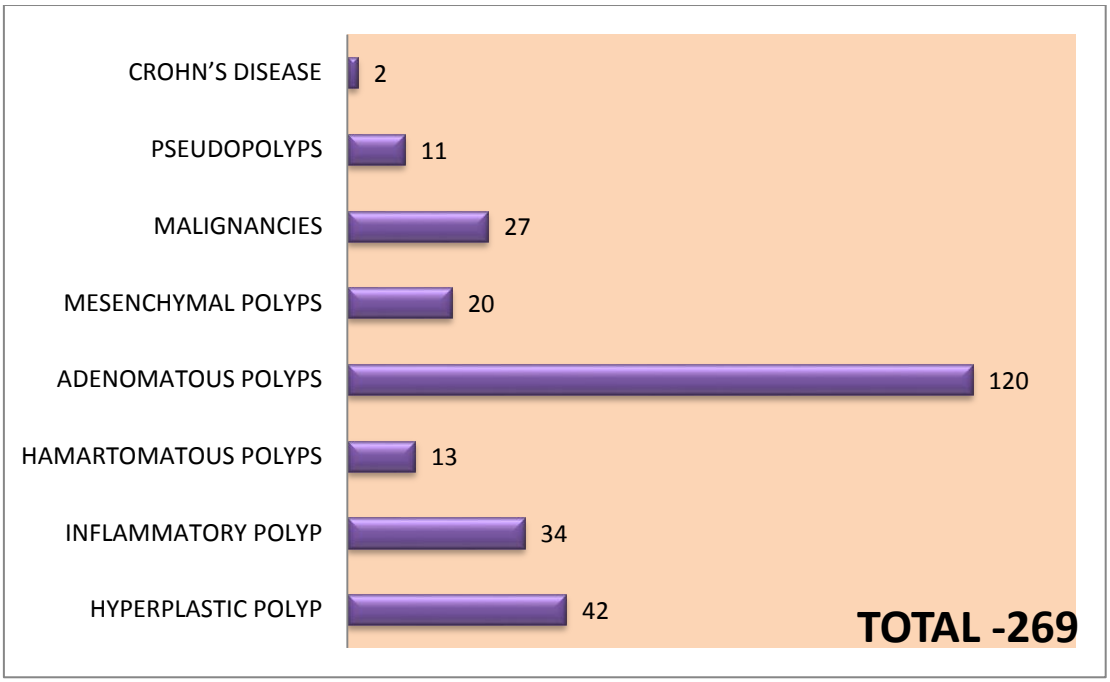


CHART 6. DISTRIBUTION OF POLYPS OF LARGE INTESTINE BASED ON TYPE



COMPARISON OF P53 (CHART 7) AND BCL2 (CHART 8) EXPRESSION IN CANCERS, ADENOMATOUS POLYPS WITH AND WITHOUT SYNCHRONOUS COLONIC MALIGNANCIES

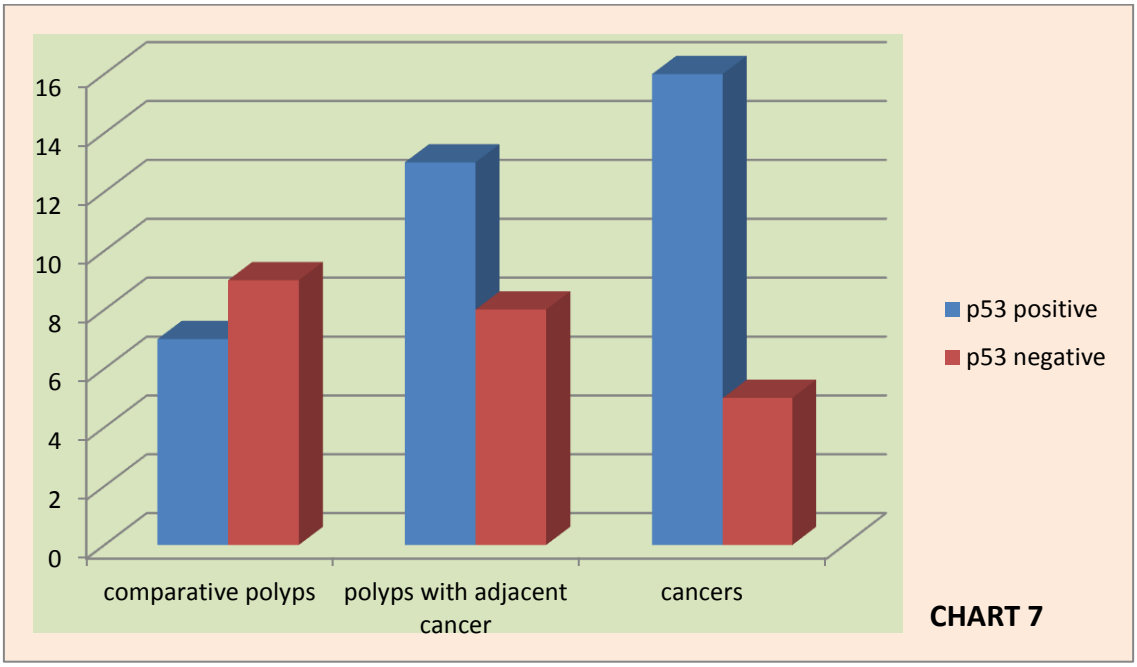


CHART 7

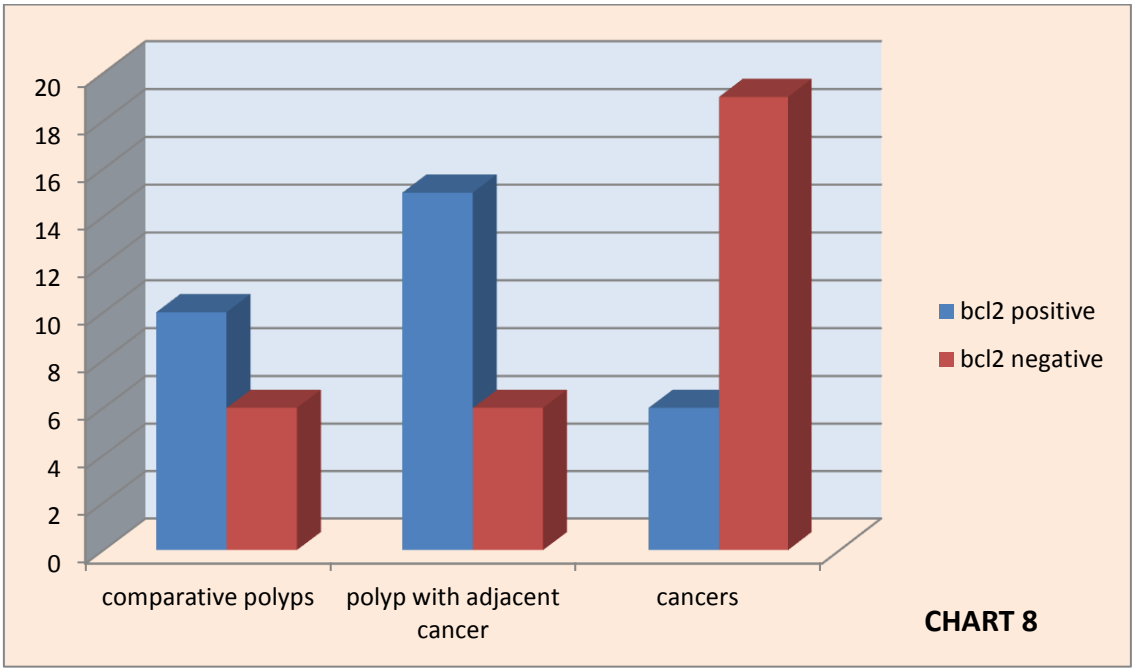
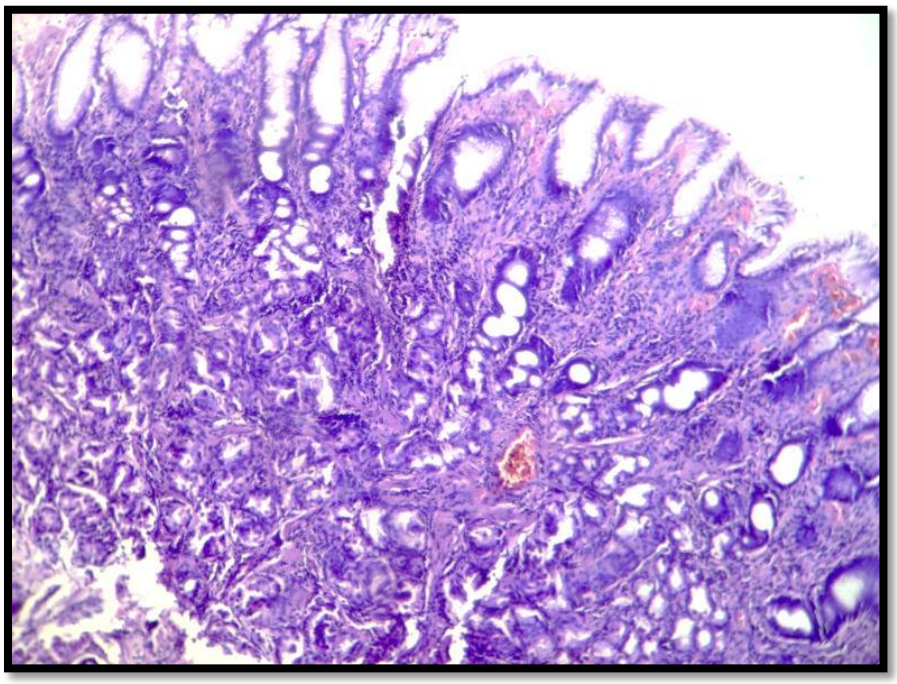


CHART 8

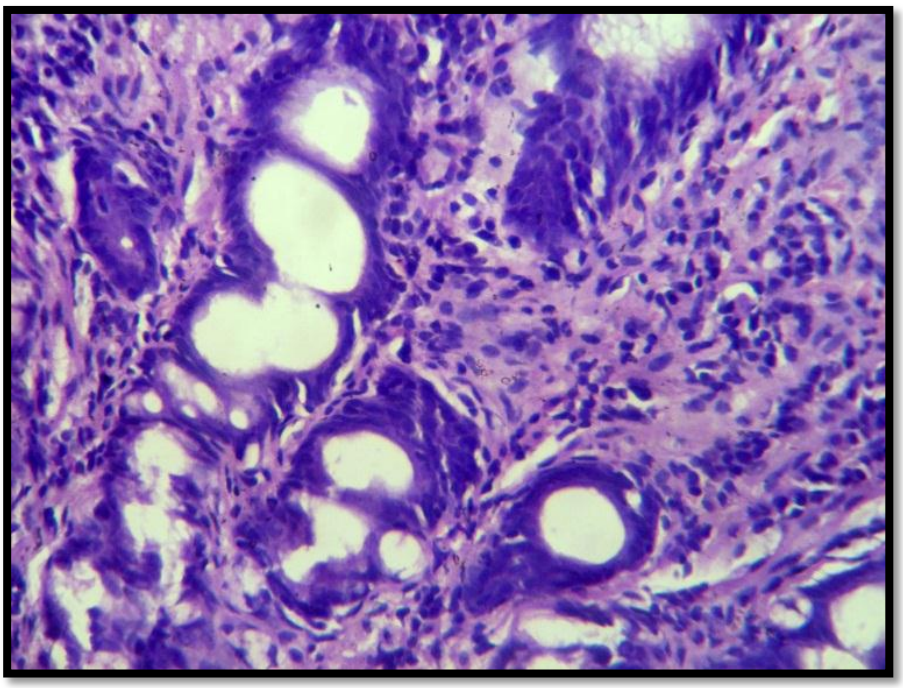
HYPERPLASTIC POLYP

Fig.1



Elongated dilated and serrated crypt architecture. H & E Fig 1. x10. Fig 2. X40

Fig. 2



INFLAMMATORY FIBROID POLYP

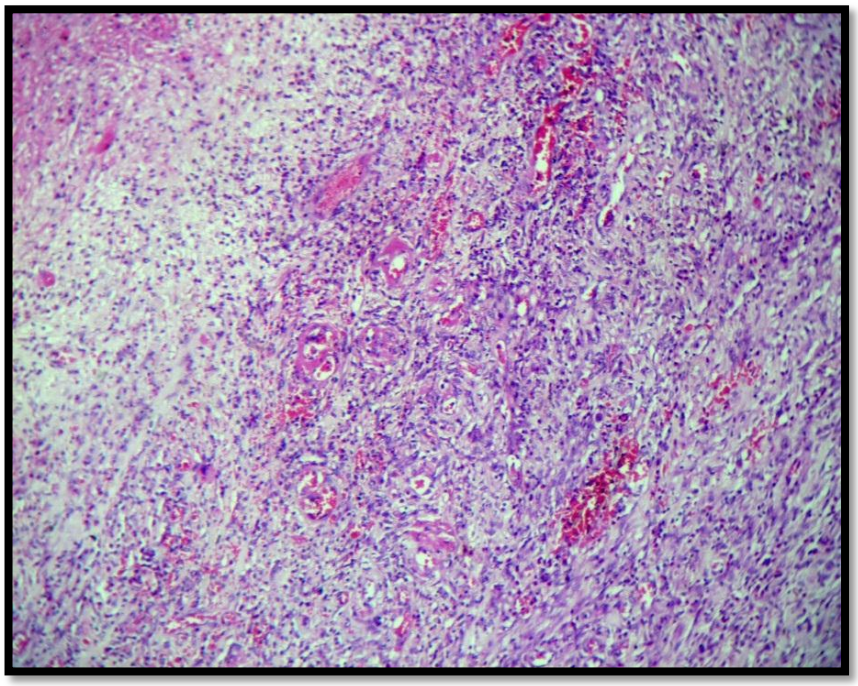


Fig. 3 Loose edematous lamina propria showing blood vessels and inflammatory cells. H & E x10

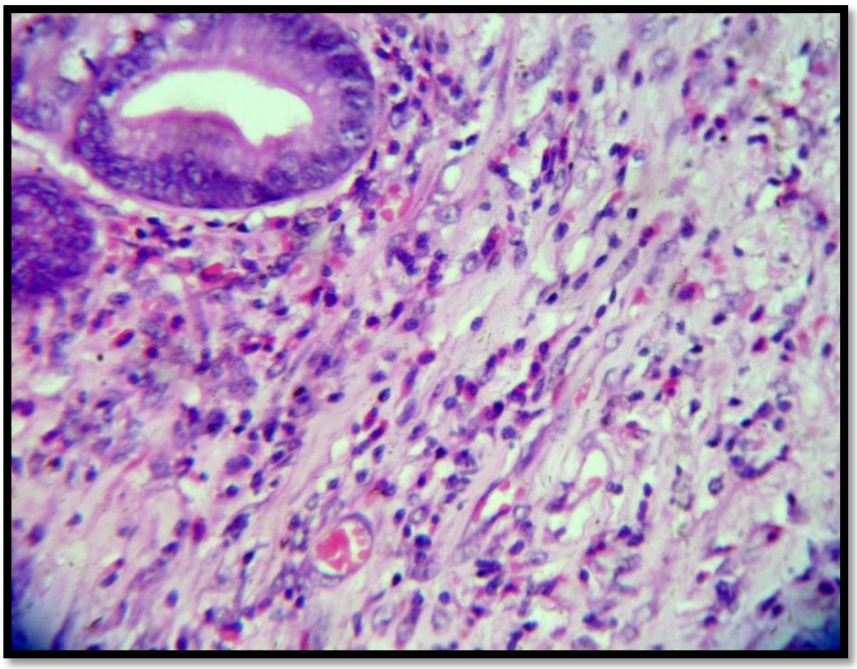


Fig. 4 Glands surrounded by myxoid lamina propria containing capillaries admixed with eosinophils and lymphocytes. H & E x40

JUVENILE POLYP

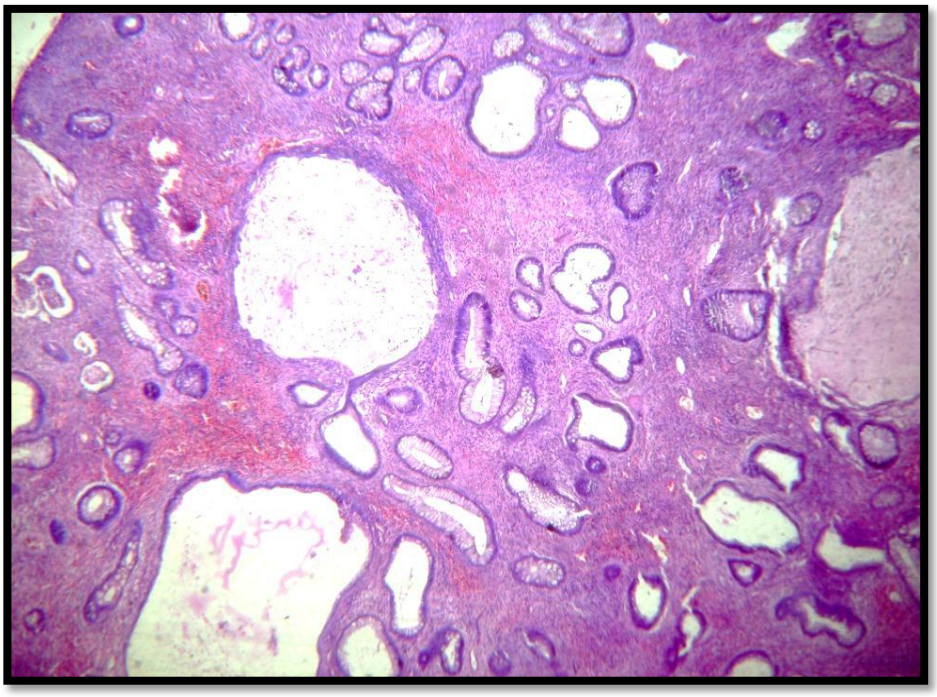


Fig. 5 Large dilated cysts, some filled with mucin. H & E x10

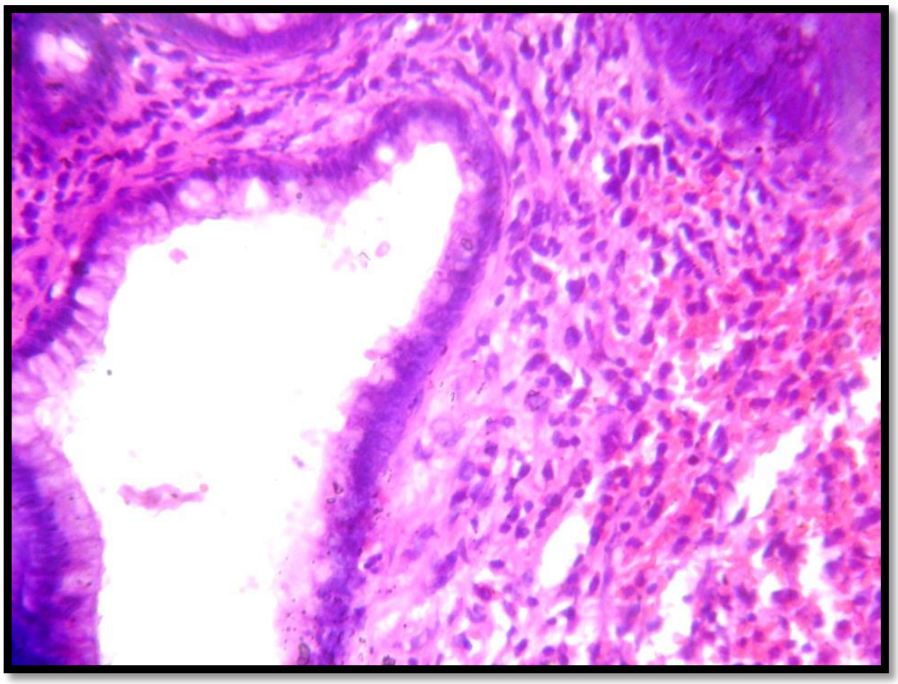


Fig. 6 Dilated crypts separated by an expanded lamina propria showing edema, hemorrhage and inflammatory cells. H & E x40

PEUTZ-JEGHERS POLYP

Fig. 7

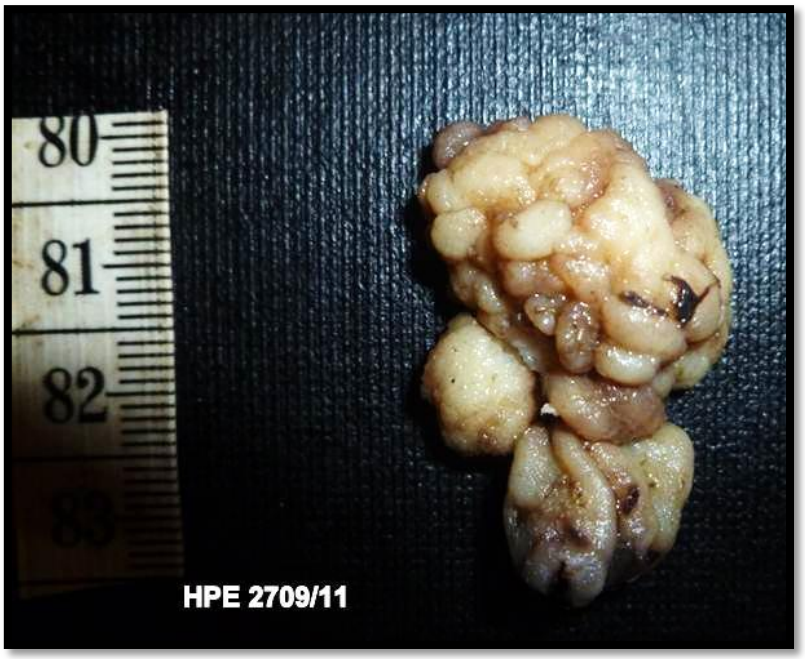


Fig. 8

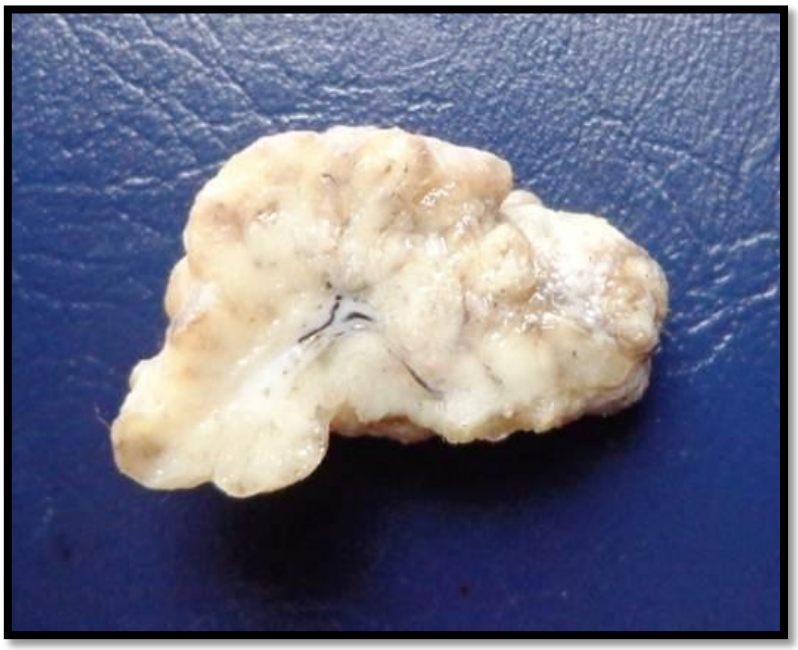


Fig 7 & 8. PJ polyp. Externally (Fig 7) lobulated grey-white polyp. Cut surface (Fig 8) shows splaying of muscular layer.

PEUTZ-JEGHERS POLYP

Fig. 9

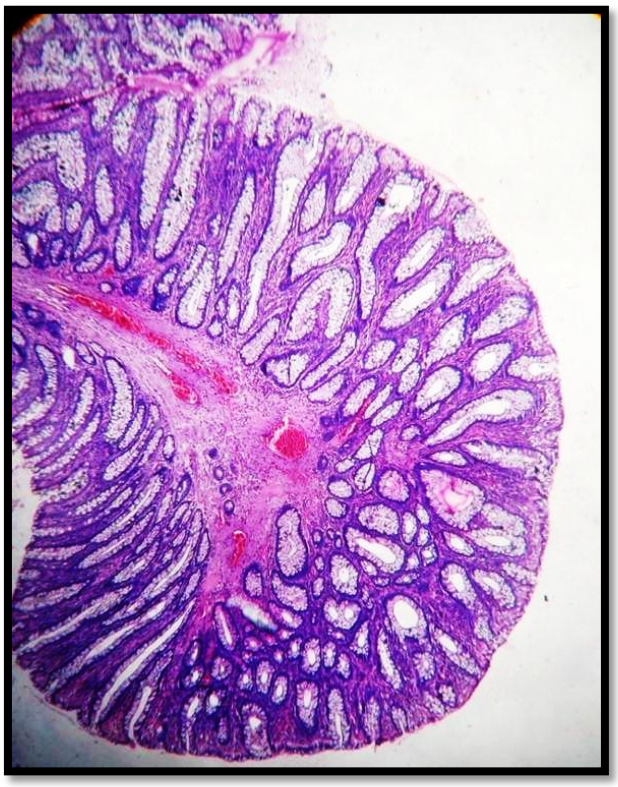


Fig. 10

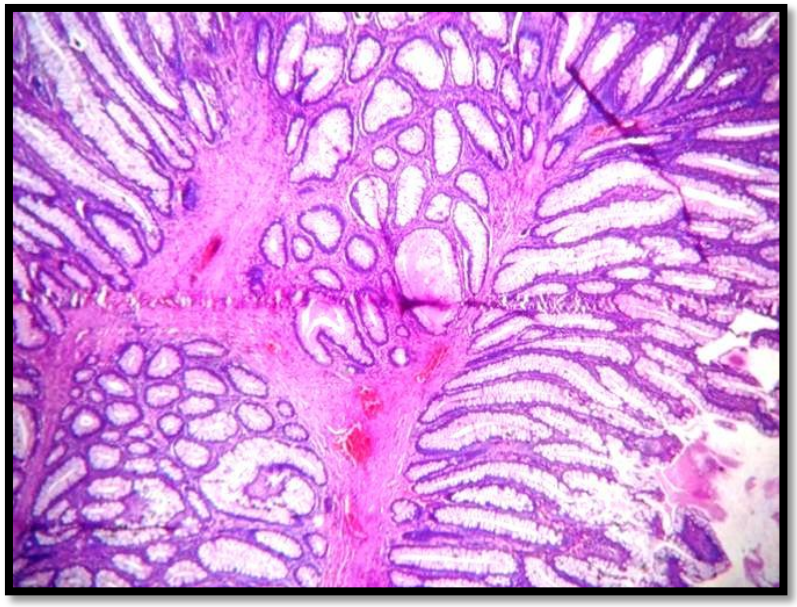


Fig.9 & 10 PJ polyp. Shows splayed muscularis mucosae covered by normal-appearing epithelium. H & E x10

TUBULAR ADENOMA

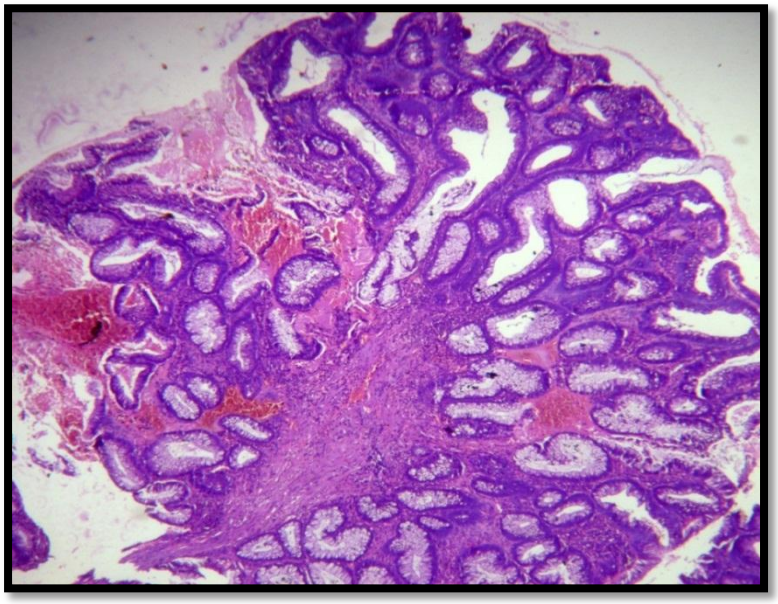


Fig. 11 More than 80% of mucosa showing tubular glands lined by dysplastic epithelium. H & E x10

VILLOUS ADENOMA

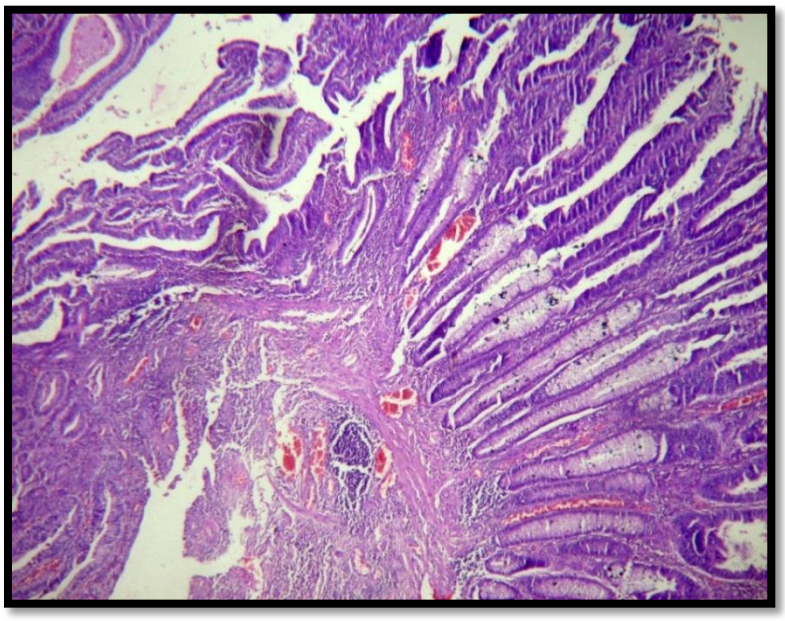


Fig. 12 Mucosa showing finger-like processes of glands lined by dysplastic epithelium. H & E x10

TUBULOVILLOUS ADENOMA

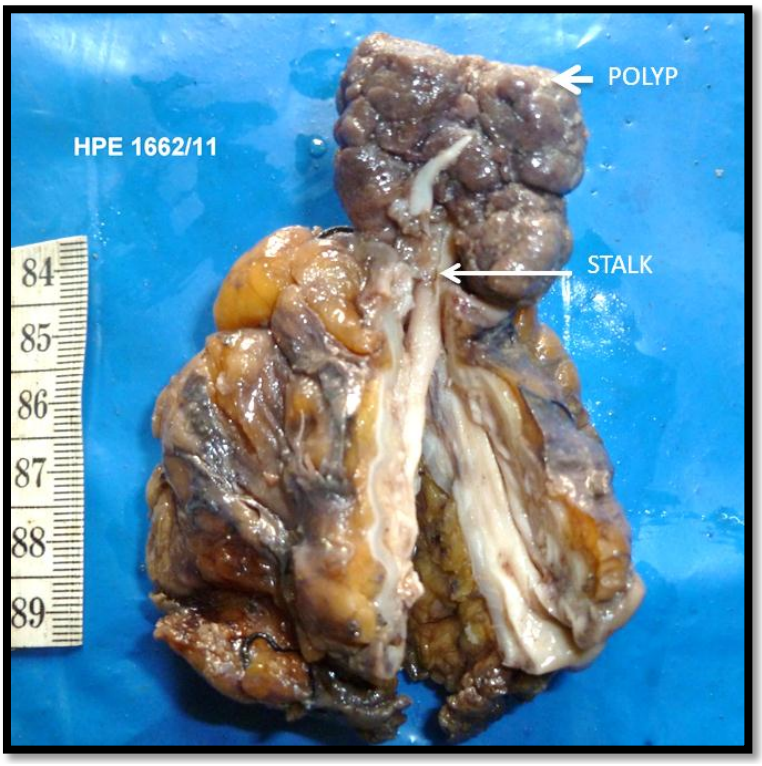


Fig. 13 Resected portion of large intestine with a pedunculated polyp arising from the mucosa

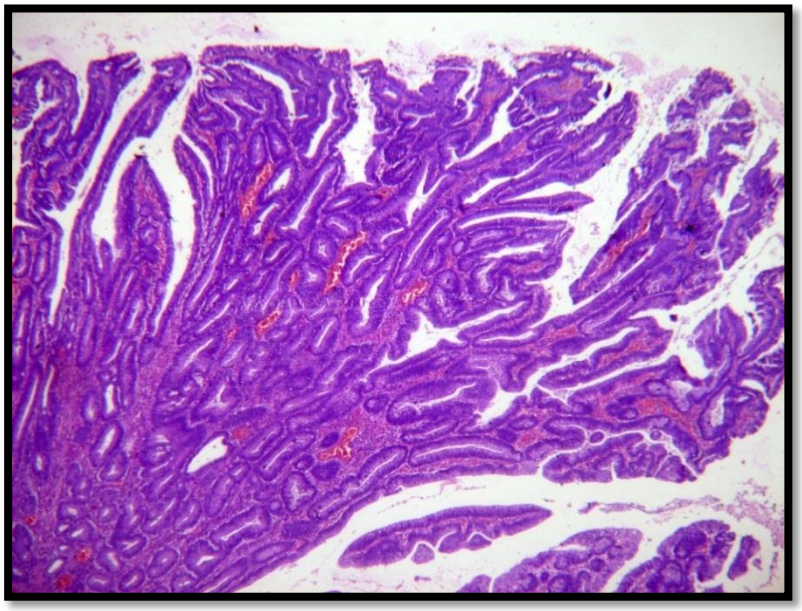


Fig. 14 Mucosa showing glands in villous and tubular pattern lined by dysplastic epithelium. H & E x10

DYSPLASIA

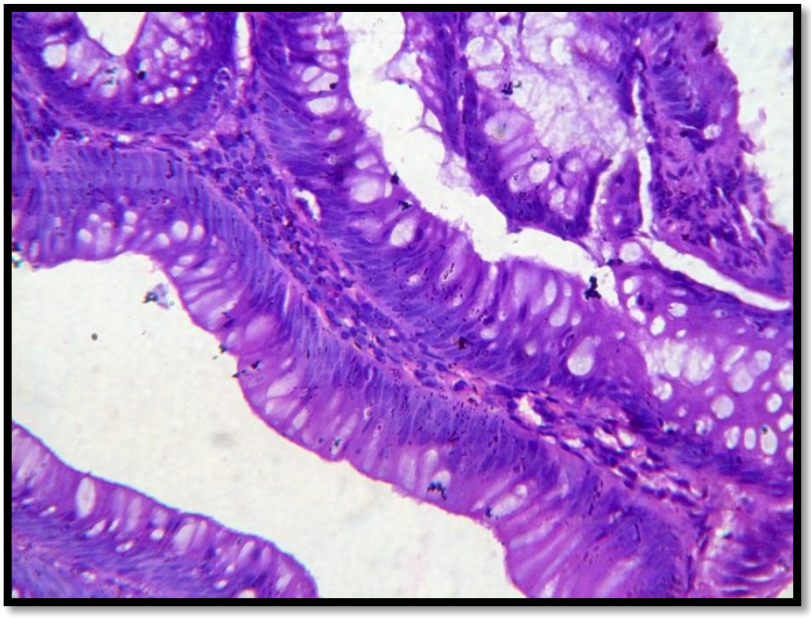


Fig. 15 Adenoma with low grade dysplasia – elongated crowded hyperchromatic nuclei with preserved polarity and mucin secretion. H & E x40

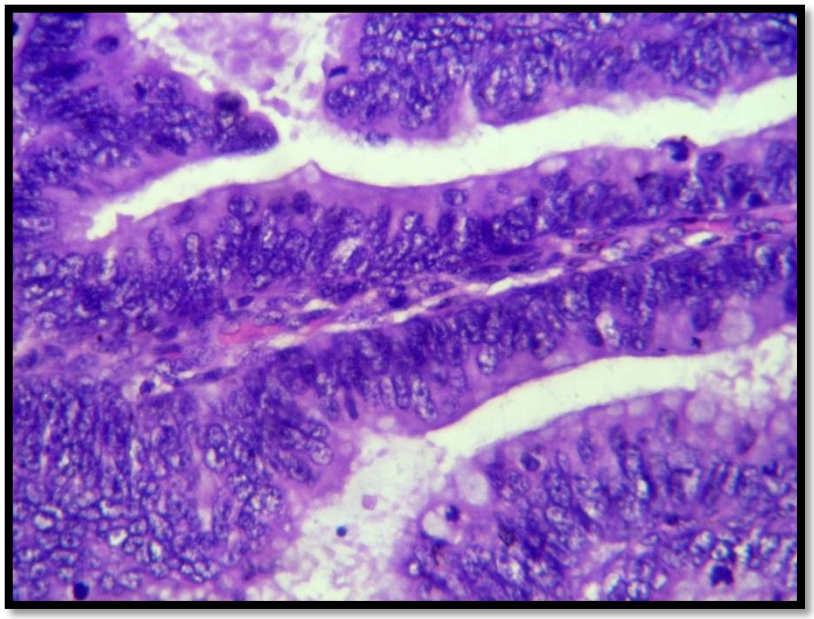


Fig. 16 Adenoma with high grade dysplasia–irregular glands with nuclei showing loss of polarity, prominent nucleoli, mitosis and loss of mucin secretion. H & E x40

P53 EXPRESSION IN ADENOMATOUS POLYP

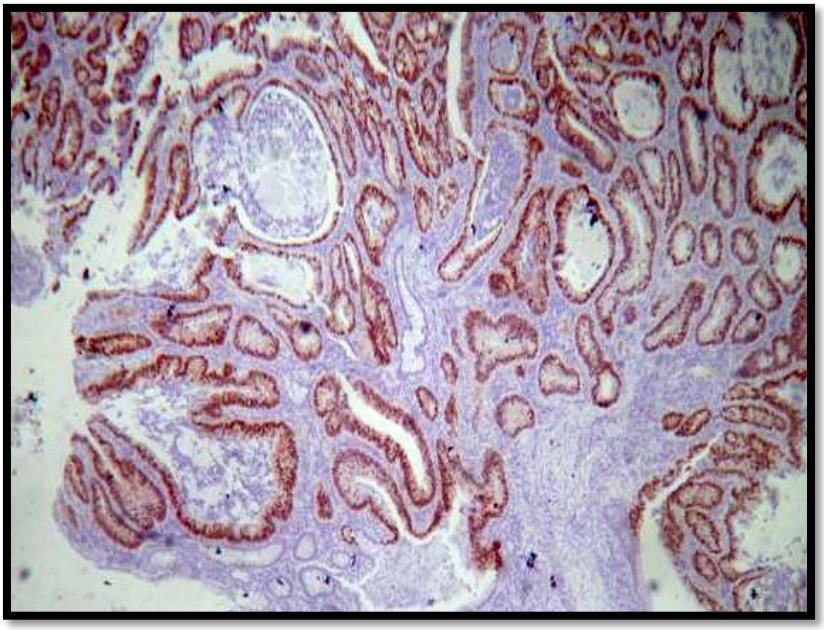


Fig. 17 Adenomatous polyp showing strong positivity for p53. x10

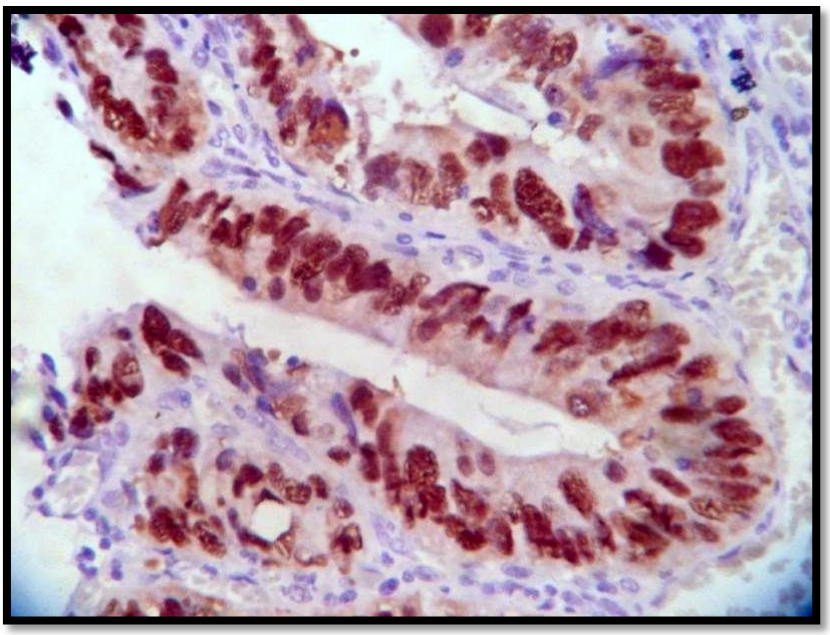


Fig. 18 p53 nuclear staining - strong positivity. x40

BCL 2 EXPRESSION IN ADENOMATOUS POLYP

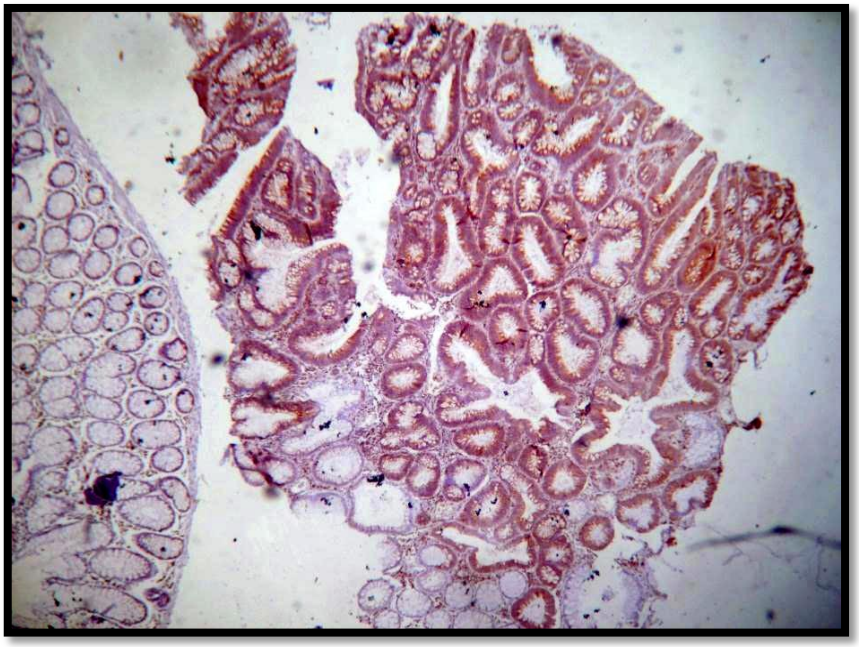


Fig. 19 bcl2 staining – diffuse strong positivity in adenomatous epithelium x10

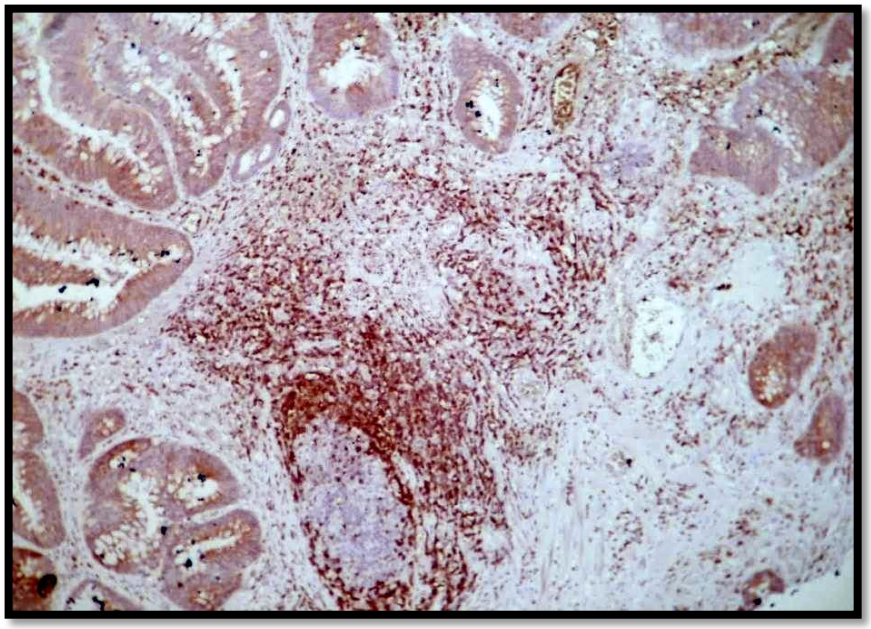


Fig. 20 bcl2 cytoplasmic staining – internal control (mantle zone lymphocytes) and adenomatous epithelium show strong positivity. x10

ADENOMATOUS POLYPOSIS COLI WITH SYNCHRONOUS MALIGNANCY

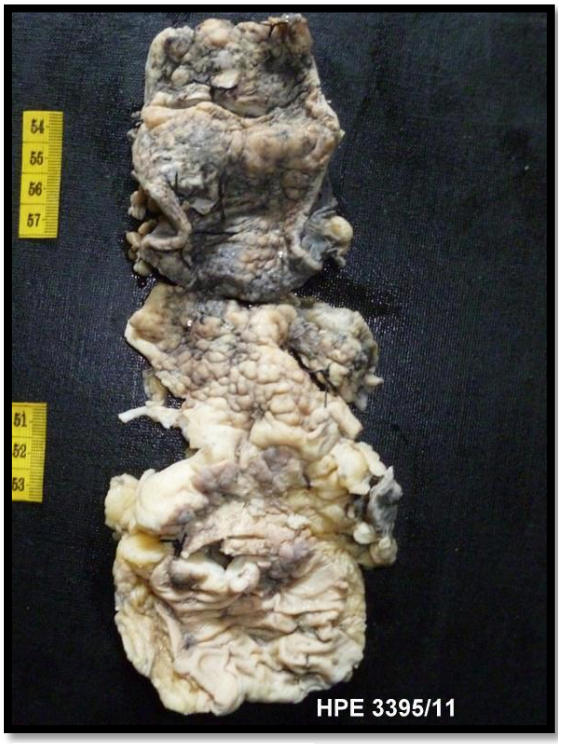


Fig. 21

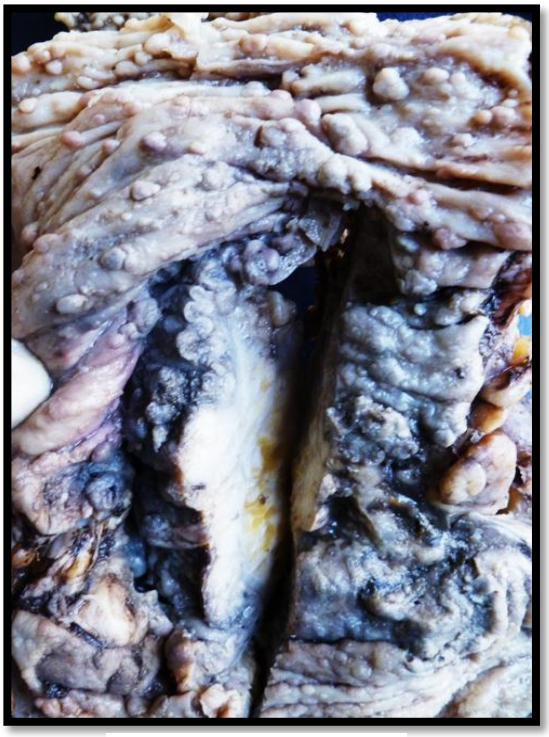


Fig. 22

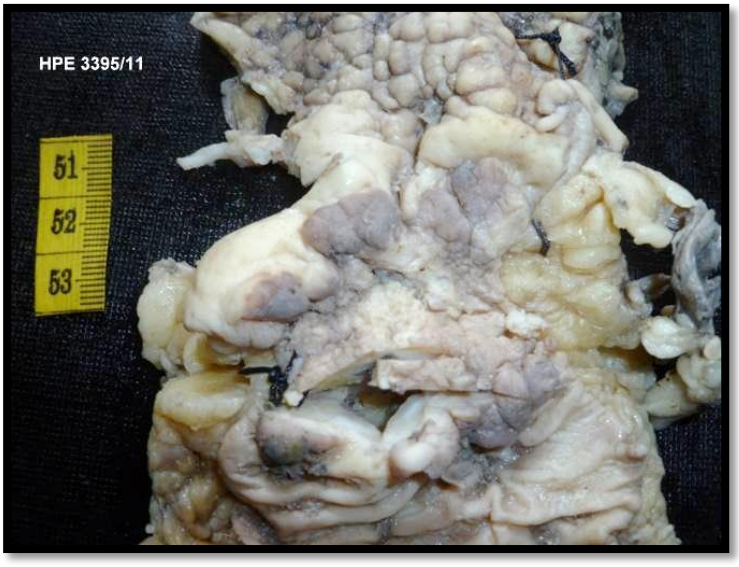


Fig. 23

Fig. 21, 22, 23 Multiple Colonic Adenomatous Polyps with ulcerated solid infiltrating growth of colon.

ADENOMATOUS POLYP WITH INVASION INTO MUSCULARIS MUCOSA

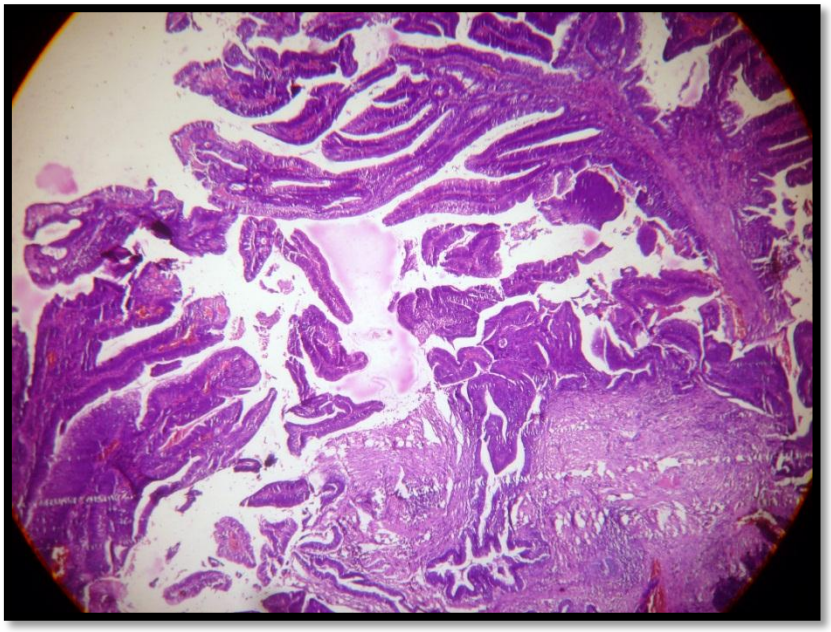


Fig. 24 Villous adenoma with invasion into muscularis mucosa (malignant transformation) H & E x10

ANNEXURE I

PROFORMA

CASE NUMBER : HPE NUMBER :

NAME : OP/IP NUMBER :

AGE :

SEX :

CLINICAL DIAGNOSIS :

SYMPTOMS :

ENDOSCOPY FINDINGS :

SESSILE/PEDUNCULATED :

NUMBER OF LESIONS :

SITE:

SIZE:

GROSS FEATURE :

MICROSCOPY (TYPE) :

ASSOCIATED CANCERS IF ANY : SITE :

TYPE:

ANNEXURE II

IMMUNOHISTOCHEMISTRY PROCEDURE

PREPARATION OF SLIDES:

1. Sections of 4 micron thickness were cut from formalin fixed paraffin embedded tissue specimens and transferred to gelatin-chrome alum coated slides.
2. The slides were incubated overnight at 58ºC.
3. The sections were then deparaffinized in xylene for 15 minutes x 2 changes.
4. Dehydration of sections was done with absolute alcohol for 5 minutes x 2 changes.
5. The sections were then washed in tap water for 10 minutes.
6. The slides were immersed in distilled water for 5 minutes.

ANTIGEN RETRIEVAL:

7. Heat induced antigen retrieval was done with microwave oven in appropriate temperature with Citrate buffer for 20 minutes.
8. The slides were then cooled to room temperature and washed in running tap water for 5 minutes.
9. The slides were then rinsed in distilled water for 5 minutes.
10. Wash with appropriate wash buffer (phosphate buffer) for 5 minutes x 2 changes.

BLOCKING STEPS:

11. Apply peroxidase block over the sections for 10-12 minutes.
12. Wash the slides in phosphate buffer for 5 minutes x 2 changes.
13. Cover the sections with power block for 15 minutes.

APPLICATION OF ANTIBODY AND LABELS:

14. The sections were drained (without washing) and appropriate primary antibody was applied over the sections and incubated for 45 minutes.
15. The slides were washed in phosphate buffer for 5 minutes x 2 changes.
16. The slides were covered with SuperEnhancer for 30 minutes.
17. The slides were washed in phosphate buffer for 5 minutes x 2 changes.
18. The slides were covered with SS Label for 30 minutes.
19. Wash in phosphate buffer for 5 minutes x 2 changes.

APPLICATION OF CHROMOGEN AND COUNTERSTAIN:

20. DAB substrate was prepared by diluting 1 drop of DAB chromogen to 1 ml of DAB buffer.
21. DAB substrate solution was applied on the sections for 8 minutes.
22. Wash with phosphate buffer solution for 5 minutes x 2 changes.
23. The slides are washed well in running tap water for 5 minutes.
24. The sections were counterstained with Hematoxylin stain for 2 seconds (1 dip).
25. The slides are washed in running tap water for 3 minutes.
26. The slides are air dried, cleared with xylene and mounted with DPX.

SL. NO	BIOPSY NO	AGE	SEX	S/P	SIZE	SITE	SUB-SITE	HPE	CLINICAL DETAILS
1	303/08	43	M			Duodenum		Hyperplastic polyp	
2	387/08	53	M			Colon		Tubular adenoma	
3	536/08	43	M			Duodenum	D1 D2	Brunner gland hyperplasia	
4	876/08	55	M			Rectum		Inflammatory pseudopolyp	
5	977/08	26	M	S		Duodenum	Bulb	Hyperplastic polyp	
6	952/08	53	M		1x0.5x0.5cm	Colon		Mucinous adenoca ,tubulovillous adenoma	
7	1021/08	30	M			Colon	Transverse	Pseudopolyp with non-spec ulcer	Ulcerated circumferential stricture with polyp
8	1444/08	22	M		2.5x2x1 cm	Rectum		Tubular adenoma	
9	1511/08	25	F			Colon	Sigmoid	Tubular adenoma	
10	1616/08	51	F			Rectum		Tubular adenoma	
11	1618/08	61	M			Rectum		Tubular adenoma	
12	1685/08	33	M		Multiple	Duodenum	D2	Non spec inflammation	Multiple nodular polypoidal lesions in d2,dj flexure
13	1686/08	25	M	S	1 cm	Rectum		Tubular adenoma	
14	1831/08	25	F			Colon	Sigmoid	Tubular adenoma	
15	2251/08	60	F			Rectum		Non-spec inflammation	
16	2332/08	35	M	S		Stomach	Antrum	Hyperplastic polyp	
17	2601/08	60	M			Duodenum		Hyperplastic polyp	
18	2699/08	40	F			Duodenum		GIST-intermed grade	
19	2735/08	40	F			Colon	Transverse	Tubulovillous adenoma	
20	2820/08	65	M			Rectum		Villous adenoma	
21	2913/08	60	M			Stomach	Body	Hyperplastic polyp	
22	2985/08	39	F			Stomach	Lesser curvature	Hyperplastic polyp	
23	3047/08	19	M			Duodenum		Hyperplastic polyp	
24	3513/08	14	M			Rectum		Inflammatory polyp	
25	3541/08	55	M		0.2 - 2 cm	Colon r+l	Throughout	CA rectum,adenoca well diff papillary, tubulovillous adenoma	
26	3603/08	21	M			Rectum		Juvenile polyp	
27	3615/08	71	M			Caecum		Hyperplastic polyp	Intestinal obstruction
28	3924/08	30	F	S		Stomach	Antrum	Adenoca infiltr well diff intestinal type	
29	3925/08	42	M			Duodenum	Bulb	Hyperplastic polyp	
30	4272/08	50	F			Duodenum	D2	Hyperplastic polyp	Verrucous polypoidal lesion
31	4374/08	48	M	S		Colon	Mid descending colon	Tubular adenoma	
32	4377/08	13	M			Rectum		Juvenile polyp	
32	4375/08	35	F	S		Stomach	Antrum	Tubulovillous adenoma	
34	4450/08	50	F			Duodenum		Tubulovillous adenoma	
35	4740/08	65	M			Duodenum	D2	Inflammatory pseudopolyp	
36	4742/08	38	M	P	1 cm	Rectum		Juvenile polyp	

37	4858/08	43	F			Stomach	Antrum	Adenoca infiltr well diff	
38	4876/08	40	F	S		Stomach	Body	Non spec inflammation	
39	4878/08	58	M			Rectum		Hyperplastic polyp	
40	5080/08	60	F	P		Anal	Anal verge	Villous adenoma	
41	5107/08	30	F			Stomach	Body	Chronic gastritis	
42	5227/08	37	F		5 mm	Rectum	Lower rectum	Hyperplastic polyp	Bleeding pr
43	5240/08	38	F			Stomach	Antrum	Villous adenoma	
44	5322/08	59	M		2.5 cm	Stomach	Lc	Tubulovillous adenoma	
45	5550/08	35	M			Duodenum	D2 & d3	Hyperplastic polyp	
46	5741/08	35	M			Anal		Fibroepithelial polyp	
47	5754/08	65	M			Stomach	Fundus	Hyperplastic polyp	
48	5755/08	59	M			Stomach	Body	Hyperplastic polyp	
49	5850/08	56	F			Stomach	Lc	Chronic non specific gastritis	
50	5874/08	31	M			Stomach	Antrum	Chronic lymphocytic gastritis	
51	5876/08	47	M			Stomach	Pylorus	Chronic lymphocytic gastritis	
52	5879/08	35	F			Stomach	Antrum	Hyperplastic polyp	
53	5880/08	55	F			Stomach	Antrum	Hyperplastic polyp	
54	5882/08	58	F	P		Stomach	Pylorus A. Pyloric growth	Tubular adenoma	
55	6053/08	50	F			Stomach	b.polypoidal gr incisura	(a)infiltr adenoca mod diff (b)well diff adenoca papillary type	
56	6062/08	88	M	S		A.growth ogj b.polyp duodenum	Bulb	(a) adenoca well diff (b)chronic lymphocytic duodenitis with superficial ulceration	
57	6150/08 6227/08 cr	43	M			Rectum		Hyperplastic polyp Chronic lymphocytic gastritis &	
58	4858/08	43	F			Stomach	Antrum	few dysplastic glands- s/o early gastritic ca	
59	6301/08	40	M			Colon	Transverse	Hyperplastic polyp	
60	6302/08	65	M			Colon	Recto sigmoid	Non spec inflammation	
61	6307/08	70	M			Stomach	Pylorus	Inflammatory polyp	
62	6387/08	40	M			Stomach	Antrum	Hyperplastic polyp	
63	6739/08	47	M			Colon	Descending	Tubular adenoma	
64	6742/08	45	M	S		Stomach	Pylorus	Chronic lymphocytic gastritis	
65	6751/08	55	F	P		Stomach	Antrum	Hyperplastic polyp	
66	6787/08	38	M	S		Duodenum	Bulb	Chronic lymphocytic duodenitis	
67	6959/08	50	M	S, p	2x2x2, <0.5 cm	Colon	Desc & sigmoid	Tubulovillous adenoma c cystic dilatation of glands	Multiple colonic polyps
68	6964/08 cr 6053/08	50	F			Stomach	A.ulcer 3*2cm b.polyp 1cm	A.infiltr adenoca intestinal type mod diff. B.villous adenoma c dysplastic changes	Chronic lymphocyti c gastritis
69	6984/08	32	F			Stomach	Antrum	Gastritis c dysplastic glands	
70	6997/08	60	M			A)duodenal polyp b)growth stomach		A-inflammed hyperplastic polyp b-infiltr adenoca well diff	
71	7104/08	65	F			Stomach	Fundus	Fundic gastric polyp	
72	7226/08	48	M			Stomach	Fundus	Fundic gastric polyp	
73	7471/08	74	F	S		Duodenum		Chronic lymphocytic duodenitis	
74	7480/08	50	M	S		Colon	Sigmoid	Hyperplastic polyp	
75	7609/08	60	F			Stomach	Fundus	Chronic lymphocytic gastritis	

76	7893/08	25	F			Duodenum	D2,D3	Chronic lymphocytic duodenitis	
77	8229/08	28	M			Rectum		Inflammatory polyp	
78	8255/08	55	M	S	6x4cm	Rectum		Infltr adenoca well diff	
79	8307/08	45	F			Stomach	Antrum	Hyperplastic polyp with ulcer	
80	8478/08	16	F		3-5 mm	Stomach	Antrum	Tubular adenoma	
81	8483/08	34	F			Colon		Tubulovillous adenomas	
82	8486/08	50	M			Colon	Sigmoid	Tubular adenoma	
83	8548/08	55	M			Colon	Sigmoid	Tubulovillous adenomas	
84	8550/08	48	M			Stomach	Body	Chronic lymphocytic gastritis	
85	8609/08	38	M			Rectum		Pseudopolyp	
	8667/08 cr								Polyp presenting with intussusception
86	8486/08	56	M			Rectum		Lipomatous polyp	
87	8833/08	42	M			Stomach	Antrum	Infiltr adenoca well diff intestinal type	
88	8888/08	30	M			Rectum		Adenoca infilt well diff	
89	8892/08	30	F	S		Stomach	GC	Chr lympho gastritis	K/c men 2b with multiple polyps hypophary nx,stomach
90	8948/08	44	F			Colon r+l	A.caecum, right colon b. Left colon	A)non spec inflammation b)infiltr adenoca well diff	
91	9014/08	26	M			Colon	Splenic flexure	Hyperplastic polyp	C/o constipation
92	9022/08	60	M	S		Colon r+l	A.asc colon b.sigmoid colon polyp	A.infiltr adenoca well diff b.infiltr adenoca papillary type	
93	9023/08	42	F	S		Colon	Transverse	Tubular adenoma c mild dysplasia	
94	27/2009	56	M			Rectum		Hyperplastic polyp c foci of adenomatous change	
95	30/2009	65	F	S		Stomach	A.growth stomach b. Polyp antrum	A-adenoca mod diff b-adenoma	
	149/09 cr						A.growth sigmoid c b.4 pedunc ,4 sessile polyps	A.infiltr mod diff adenoca b.pedunc-tubular adenoma	Multiple polyps throughout colon
96	8948/08	44	F			Colon+rectum	rectosigmoid c	sessile-hyperplastic polyp	
97	186/09	55	M			Duodenum	D2	Inflammatory polyp	
98	187/09	61	M			Duodenum	D2	Hyperplastic polyp	
99	222/09	60	M			Stomach	Antrum	Hyperplastic p	
100	441/09	19	M			Duodenum	D2,D3	Chronic duodenitis	
101	517/09	44	M			Rectum	A.bx prox rectum b.large polypoidal lesion distal rectum	A)mod diff adeno ca b)hyperplastic polyp	
102	555/09	30	M	P		Colon	Sigmoid	Lipomatous polyp	
103	579/09	58	F			Stomach	Fundus	Chronic gastritis	
104	599/09	56	M	P		Stomach	Body	Adenoca infilter mod diff	
105	604/09	53	F	S		Duodenum	D2	Chronic inflammation	
106	655/09	62	N	S	5 cm	Rectum		Inflammatory polyp	

107	661/09	70	M			A.duodenal polyp,b.growth esophagus		A-inflamm polyp b-mod diff scc
108	662/09	55	M			Colon		Villous adenoma c low grade dysplasia
109	710/09	45	M			Caecum+colon	A-unhealthy mucosa in desc colon b-polyp in cecum & asc colon	A)adenomatous changes b)tubulovillous adenoma c low grade dysplasia
110	830/09	70	M			Rectum		Tubular adenoma c low grade dysplasia
111	1010/09	53	M			Anal polyp		Inflammatory polyp
112	1011/09	47	M			Stomach	GC	Inflammatory polyp
113	1140/09	39	M			Duodenum	D2	Inflammatory polyp
114	1211/09	46	M			Colon	Transverse	Inflammatory polyp
115	1257/09	51	F			Stomach	Fundus	Inflammatory polyp
116	1268/09	58	M			Stomach	Fundus	Inflammatory polyp
117	1386/09	40	M	S		Anal canal		Inflammatory polyp
118	1390/09	30	F			Colon		Adenomatoup polyp with malignant transformation
119	1392/09	80	F	P		Stomach	LC	Hyperplastic polyp
120	1425/09	48	F			Stomach	Antrum	Adenoca mod diff papillary
121	1450/09	60	F			Stomach	Body	Inflammatory polyp
122	1479/09	38	F			Stomach	Body	Hyperplastic polyp
123	1584/09	67	M			Colon	Asc colon & hepatic flexure	Adenoca infiltr mod diff
124	1656/09	75	M			Caecum		Tubular adenoma
125	1715/09	53	M			Duodenum		Adenoca well differentiated papillary
126	1720/09	28	M			Rectum		Inflammatory polyp
127	1828/09 cr	50	M			Stomach	Antrum	Adenoca
128	1898/09	19	M			Colon+si	Ileum.caecum.rectum	Inflammatory polyps
129	1919/09	48	M			Rectum	A-rectosigmoid growth b-rectal polyp	A)mod-poorly diff adenoca b)hyperplastic polyp
130	2001/09	63	F	S		Colon		Adenoca infiltr mucin secreting , hyperplastic villi c adenomatous changes
131	2031/09	45	M			Caecum	Caecum	Inflammatory polyp
132	2214/09	40	M	S		Stomach	Antrum	Hyperplastic polyp
133	2227/09	29	M	P		Rectum		Hyperplastic polyp
134	2229/09	47	M			Colon	A-growth asc colon b-splenic flexure polyp	A)poorly diff ca b)villous adenoma
135	2280/09	42	M	S		Rectum		Hyperplastic polyp
136	2547/09	71	M	S	5 mm	Colon	Descending	Hyperplastic polyp
137	2990/09	54	M			A-polypoidal lesion d2 b-growth esophagus		A)inflammatory polyp b-scc
138	3010/09	30	F	S		Colon		Infiltr mod-poorly diff adenoca ; polyps- villous adenoma, some

								with high grade dysplasia	
139	3035/09	44	M			Rectum		Hyperplastic polyp	
140	3075/09	60	M			Colon	A-colonic polyp b-growth sigmoid colon	A)tubulovillous adenoma c low grade dysplasia b)infiltr mod diff pap adenoca	
141	3110/09	40	F			Anal polyp		Fibroepithelial polyp	
142	3364/09	64	F			A-duodenum b-antral polyp	Antrum	A)chr non spec duodenitis b-antral gastritis	
143	3518/09	65	F			Stomach	Pylorus	Non spec inflammation	
144	3684/09	65	F			Stomach	Body	Hyperplastic polyp	
145	3711/09	60	M	S		Colon	Transverse	Hyperplastic polyp,ulcer- infiltr mod diff adenoca	Polyp,ulcer
146	3876/09	65	F	S		Colon+rectum	A-desc colon b-rectum	A) non spec inflamm b)villous adenoma	
147	4041/09	34	F			Colon	A-growth rectum b-5 polyps in colon	Mucinous adenoca,inflammatory polyp	
148	4198/09	14	F	S		Colon+rectum	Sigmoid,rectum ,asc colon	Inflammatory polyps	
149	4219/10 cr-3876/09	65	F			Rectum		Villous adenoma c low grade dysplasia c focus showing ca in situ	
150	4643/09	42	M			Colon		Tubular adenoma	Polyposis coli
151	4649/09	45	F	P		Caecum	Caecum	TVA	
152	4741/09	19	M	S, p	1.5*1*1, 0.5*0.5	Colon+rectum	Asc c, hepatic fl,sigm,rectum	Juvenile polyps c focal adenomatous change	Fap-total proctocolectomy-4826/09-juv poly c focal adenomatous change
153	4852/09	42	M			A-D2 polyp b-stomach body	Body	A) non spec infl b)hyperplastic p	
154	4858/09	50	M	P		Colon	40cm from a.v	Tubular adenoma	
155	5149/09	52	M		1*1*0.5	Colon	Sigmoid	Hyperplastic polyp	
156	5235/09	32	F	P	1.5*1*1	Anal polyp		Fibro epithelial polyp	
157	5629/09	65	M			Stomach	Junction of fundus & body	Chronic non specific gastritis	
158	5635/09	55	F			Caecum	Caecum	Non specific colitis	
159	5642/09	17	M			Duodenum		Hyperplastic polyp	Acute small bowel obstruction
160	5682/09	47	M			Stomach	Body	Lymphoma/poorly diff ca	
161	5749/09	60	F			Stomach	LC	Hyperplastic polyp	
162	5750/09	78	M			Stomach	Antrum	Hyperplastic polyp c mild mod dysplasia	C/o dyspepsia
163	5755/09	35	M	S		Stomach	Distal body	Hyperplastic polyp	
164	5952/09	68	M			Colon		Tubular adenoma	
165	6142/09	50	M	S		Stomach	Antrum	Chronic non specific gastritis	
166	6284/09	55	M	S		Colon	Descending	TVA	
167	6413/09	62	F			Rectum		Adenoca infiltrating well diff	
168	6421/09	22	M			Stomach	Pyloro-duodenal	Tubular adenoma	

							opening		
169	6586/09	32	F			Colon	Sigmoid	Villous adenoma	
170	6692/09	65	M	S		Colon	Sigmoid	Adenoca mucinous type	
171	6758/09	14	M	S	0.5 to 0.75 cm	Rectum		Retention polyp	
172	6791/09	28	F	S		Esophagus		S/s hyperplastic squamous epithelium with papillomatosis	
173	6802/09	74	M			Colon	Sigmoid	Tubulovillous adenoma + infiltr well diff adenoca	
174	6907/09	32	F			Colon	Rectosigmoid	Adenoca well differentiated	
175	6936/09	14	F			Rectum	10 cm from av	Tubular adenoma with high grade dysplasia	
176	6987/09	56	M			Colon	Desc	Tubular adenoma	
177	7085/09	50	M	S		Stomach	Fundus	Adenoca poorly diff c neuroendocrine diff/neuroendocrine ca	
178	7164/09	56	M			Duodenum		Poorly diff adeno ca	
179	7169/09	75	M			Caecum		Adenoca mucinous	Multiple polyps throughout colon
180	7202/09	79	M			Colon	Sigmoid	Tubulovillous adenoma c well diff adenoca	
181	7376/09	58	F			Rectum		Villous adenoma c low grade dysplasia	
182	7547/09	65	M			Esophagus-lower		Barrett's esophagitis	
183	7507/09	55	M			Rectum		Retention polyp	
184	8011/09	60	F			Stomach	OGJ,cardia	Adenoca well differentiated	
185	8515/09	74	M			Rectum		Tubular adenoma	
186	8565/09	70	M			Rectum		Non specific colitis	
187	8517/09	30	M	S	5.5*5*3cm	Colon	Sigmoid	Lipomatous polyp	
188	8766/09	59	M			Stomach	Antrum	Adenoca infiltrating well diff	
189	8569/09	32	F			Anal polyp		Fibroepithelial polyp	
190	8726/09 cr 7085/09, 8010/09	50	M	S	Multiple -largest 1.7 cm smallest 0.2 cm	Stomach	Cardia,body	Hyperplastic polyps	
191	380/10	47	M			Colon	Sigmoid	Retention polyp	
192	573/10	35	F			Caecum		Tubular adenoma	
193	1161/10	65	F	S		Stomach	LC	Hyperplastic polyp	
194	1309/10	75	F			Colon	Sigmoid	Adenoca well diff	
195	1659/10	75	F			Stomach	Antrum	Hyperplastic polyp	
196	1663/10	80	M			Colon	Sigmoid	Tubular adenoma	
197	1792/10	60	F	S		Stomach	Pylorus	Adenoca poorly diff intestinal type	
198	2136/10	40	M			Rectum		Villous adenoma	
199	2307/10	40	F			Colon	Sigmoid	Adenoca infiltr well diff	

200	2319/10	50	M		Colon	60 cm from anal verge	Tubular adenoma	
201	2370/10	70	M	S	Rectum	A-ulceronodular growth 10-15cm from anal verge b-polyp 5cm from anal verge	A)infilt mod diff adenoca b)tubular adenoma	
202	2438/10	40	M	S, P	Rectum	Multiple polyps 5 cm from anal verge	Tubulovillous adenoma	
203	2580/10	35	M		Anal polyp		Tubular adenoma	
204	2729/10	60	M		Rectum		Tubular adenoma	
205	3347/10	78	M		Colon	Transverse	Tubular adenoma	Abdominal pain
206	3523/10	40	M		Stomach	Antrum	Hyperplastic polyp	Fundal gastritis
207	3554/10	56	M		Duodenum	D1,d2	Hyperplastic polyp	
208	3592/10	15	F		Caecum	Polypoidal mass in appendicular opening of caecum	Non spec inflammation	
209	4031/10	68	F		Stomach	Junction of fundus & body	Hyperplastic polyp	
210	4156/10	70	M		Colon	Rectosigmoid,9 to 13 cm from anal verge	Tubular adenomas	
211	4217/10	31	M		Colon	Descending	Tubular adenoma	
212	4546/10	73	M	S	Stomach	Distal antrum	Hyperplastic polyp	
213	4838/10	51	M		Colon	Sigmoid	Adenoca well diff pap	Secondaries liver
214	4842/10	70	M	S	Rectum+sigmoid	Sessile at 7-12 cm from AV;pedunculated-18cm from AV	Hyperplastic polyps	
215	4996/10	28	M		Rectum		Hyperplastic polyp	
216	5015/10	48	M		Stomach	Entire stomach	Non spec inflammation	Hypokalemic periodic paralysis;rec vomiting
217	5111/10	22	F		Colon	Asc colon	Villous adenoma	
218	5121/10	45	M		Duodenum	D1,D2	Non spec duodenitis	
219	5248/10	80	M		Duodenum		Chr non spec duodenitis	
220	5294/10	61	M		Rectum		Angiomatous polyp	
221	5420/10	41	M		Duodenum		Brunner gland hyperplasia	
222	5470/10	81	F		Anal		Fibro epthelial polyp	
223	5530/10	40	M		Stomach	Antrum	Hyperplastic polyp	
224	5554/10	65	M		Anal canal		Adenoca infiltrating well diff	
225	5969/10	46	M	S	Stomach	Entire stomach	Non spec inflammation	Pheochromocytoma,pancreatic abscess,multiple polyps stomach
226	6263/10	14	M		Anal canal		Hyperplastic polyp	

227	6382/10	50	M			Rectum		Non spec inflammation	
228	6475/10	45	M			Colon	Hepatic flexure	Adenoca infiltr mod diff with extensive ulcer	
229	6477/10	60	M			Stomach	OGJ	Adenoca infiltr poorly diff	
230	6479/10	65	M			Colon	Rectosigmoid,35 cm from anal verge	Hyperplastic polyp with focal adenomatous change	Ileoileal intususception
231	6560/10	65	M			Ileum		Inflammatory fibroid polyp c torsion	
232	6585/10	36	M			Colon	Descending	Hyperplastic polyp	
233	6656/10	45	M			Caecum	Caecum	Adenoca infiltr mod diff	
234	6743/10	37	F			Anal		Fibroepithelial polyp	
235	6745/10	30	F			Duodenum	D1.D2	Non spec inflammation	
236	6849/10	61	M			Anal canal		Hyperplastic polyp	Post renal transplant
237	6877/10	39	M		3x4 mm	Esophagus	Just above OGJ	Non spec inflammation	
238	6936/10	30	F			Anal polyp		Fibrepithelial polyp	
239	6962/10	46	M			Stomach	Pyloro duodenal junction	Non spec inflammation	
240	7148/10	62	M			Stomach	Fundus	Non specific gastritis	
241	7193/10	65	M			Rectum	A-rectal polyp 5cm from anal verge b-multiple friable sessile polypoidal lesions in distal desc colon	A)tubular adenoma B)well diff infiltr adeno ca	
242	7194/10	74	M	S		Anal		Chronic non-spec inflammation	Ca trans colon;post hemi colectomy
243	7259/10	55	M	S		Rectum		Inflammatory polyp	
244	7313/10	59	M			Caecum	Caecum	Flat adenoma	
245	7336/10	41	M			Caecum	Caecum	Non spec inflammation	
246	7411/10	22	M	P		Anal		Fibroepithelial polyp	Post GJ status
247	7790/10	41	M			Caecum	Caecum	Hyperplastic polyp	
248	7797/10	26	M			Anal		Fibroepithelial polyp	
249	7846/10	61	M			Anal canal		Hyperplastic polyp c focal adenomatous changes	
250	7854/10	54	M	S		Caecum	Caecum	Crohn's disease	
251	7855/10	44	M	P		Colon	Descending	Tubular adenoma	
252	7862/10	23	F			Anal polyp		Fibroepithelial polyp	
253	8293/10	32	M	S		Ileum		Non specific inflammation	
254	8328/10	52	F			Anal		Fibroepithelial polyp	
255	8426/10	53	M	S		Caecum	Caecum	Hyperplastic polyp	
256	8720/10	42	M	S		Colon	A-rectal growth b-multiple polyps colon	B-tubulovillous adenoma c high gr dysplasia b-mucin secreting infiltr adenoca (mod diff stage b2)	
257	8628/10	40	F	S		Stomach	Body	Chronic non specific gastritis	

258	8844/10	24	M		1.5cm	Colon	Rectosigmoid	Tubulovillous adenoma	
259	8920/10	65	M			Colon	Rectosigmoid	Tubulovillous adenoma c low gr dysplasia & focal areas showing high grade dysplasia	Dyspepsia/ GERD
260	8962/10	70	M	S		Duodenum		Hyperplastic polyp	
261	8972/10	39	F			Anal polyp		Fibroepithelial polyp	
262	9026/10	36	F	S		Caecum	Caecum	Non specific inflammatory pathology	
263	9027/10	63	F	P		Colon	Descending	TVA	
264	9159/10	65	M	S		Duodenum	D1	Carcinoid	
265	9188/10	61	M			Stomach	Antrum	Hyperplastic polyp	
266	9230/10	45	F			Colon	Asc	Adenoca infiltr well diff	
267	9500/10	40	F	S		Stomach	Junction of body & antrum	Tubulovillous adenoma	
268	47/11	55	F	P	0.5 cm	Colon	Sigmoid	Tubular adenoma	Markedly inflamed colonic mucosa
269	151/11	25	F			Colon		Pseudopolyp-ulcerative colitis	
270	155/11	70	M			A-ulcer lc stomach b-duodenal bulbar polyp		A)infiltr adenoca mod diff (b) ulcerated hyperplastic polyp	
271	393/11	70	M		2x1x1cm	Rectum		TVA with malignant transformation into well diff adenoca	
272	562/11	48	M	S		Colon	Asc	Tubular adenoma	Cirrhosis,
273	609/11	70	F			Stomach	Mutiple-antrum,fundus	Hyperplastic polyp c mild inflammation	
274	618/11	58	M	S		Colon		Tubular adenoma	
275	619/11	50	M	S		Colon	A-anal ulcer b-caecal polyp	A-infiltr SCC well diff b-hyperplastic polyp	
276	647/11	30	F	P		Stomach	LC	Chronic lymphocytic gastritis	Growth rectum-post APR
277	659/11	70	M	S		Colon	Sigmoid	Hyperplastic polyp	Intussusception
278	726/11	30	F	P	3x2x2cm	Ileum		Peutz jeghers polyp	
279	790/11	54	F			Colon	Descending	Lipomatous polyp	
280	800/11	62	M	S		Colon	Ascending	Hyperplastic polyp	
281	1184/11	56	F			Colon	Descending	Pseudopolyp inflammatory	
282	1231/11	30	F			Colon	Rectosigmoid	Villous adenoma	
283	1248/11	63	M		0.5x0.5cm	Esophagus	At 27cm	Hyperplastic squamous epithelium	
284	1343/11	61	M			Colon	Splenic flexure	Hyperplastic polyp	
285	1387/11	56	M	S		Colon		Non specific inflammation	
286	1513/11	68	M			Stomach	OGJ	Hyperplastic polyp	
287	1571/11	70	M			Stomach	Body	Lymphocytic gastritis	
288	1606/11	62	M	S		Colon	Asc	Hyperplastic polyp	
289	1623/11	21	F			Stomach	Pre pyloric	Chronic lymphocytic gastritis	
290	1644/11	28	F			Anal		Fibroepithelial polyp	Tubo-ovarian mass-paraovarian

									simple serous cyst
29	1662/11 cr-								
1	1231/11	30	F			Colon	Sigmoid	Tubulo villous adenoma	
29 2	1963/11	65	M			Colon	Rectosigmoid	Tubular adenoma	Multiple poyps throughout colon,lips pigmentation+?PJ syndrome
29 3	2028/11	20	M			Duodenum	D2	Hyperplastic polyps	
29 4	2037/11	55	F			Stomach	Antrum	Hyperplastic polyp	
29 5	2190/11	44	F			Stomach	A-ulceroproliferative growth distal body,antrum b-pyloric polyp	A)infiltr adeno ca signet ring cell type b-)chronic lymphocytic gastritis	dysphagia
29 6	2378/11	45	F			Stomach	Pylorus	Inflammatory polyp	
29 7	2380/11	66	M			Colon	Ascending	Adeno ca well diff papillary type	
29 8	2381/11	54	M	P		Colon	Sigmoid,30cm from AV	Tubulovillous adenoma	
29 9	2424/11	50	M	P		Esophagus	At 35cm	Hyperplasia of squamous epithelium & papillomatosis	
30 0	2515/11	66	M	S, P		Rectum		Tubular adenoma & hyperplastic polyp with focal adenomatous change	
30 1	1972/11	23	F			Anal		Fibroepithelial polyp	
30 2	3355/11 cr- 2960/11	23	M	S, P		Colon		Tubulovillous adenoma	
30 3	3776/11	53	M			Anal		Retention polyp	
30 4	4708/11	35	F			Colon	Transverse	Adenoca well diff	
30 5	5063/11	60	F	S		Stomach	Body	Hyperplastic polyp with severe inflammation	
30 6	6109/11	40	F			Anal		Fibroepithelial polyp	bleeding PR
30 7	7630/11	55	F			Colon	Sigmoid & rectum	Pseudopolyp	
30 8	7912/11	49	M			Colon	Hepatic flexure	Retention polyp	
30 9	8197/11	54	F			Stomach	Antrum	Adenoca mod diff	
31 0	8496/11	56	M	S		Rectum		Inflammatory polyp	
31 1	8839/11	21	M			Duodenum	D1	Hyperplastic polyp	
31 2	2595/11	50	F			Rectum		Hyperplastic polyp	
31 3	2709/11	35	M			Jejunum		Peutz jeghers polyp	
31 4	2960/11	30	M			Colon		Tubulovillous adenoma	
31 5	2963/11	40	M	S	3 mm	Esophagus		Fibroepithelial polyp	
31 6	3041/11 cr- 2028/11	20	M			Colon	Rectosigmoid	Tubular adenoma	
31 7	3156/11	23	M	P	2x2cm	Rectum		Tubulovillous adenoma with focal moderate dysplastic changes	
31 8	3252/11	65	M	P		Duodenum	D1,d2	Brunner gland hyperplasia	
31 9	3395/11	35	F			Rectum		Adenoca infiltrating , TVA	

320	3622/11	31	M			Stomach	Antrum	Hamartomatous polyp	
321	3849/11	32	M			A-stomach b-multiple polyps d1,d2		A-mod diff adenoca b-non spec infl	
322	3904/11	45	M	S	0.5cm	Stomach	Fundus	Nonspec inflammation	
323	3907/11	32	M	S, P		Colon		Tubular adenoma	
324	3924/11 cr-3077/11	66	M			Colon	Ascending	Villous adenoma	
325	3972/11	28	F	S		Stomach	Antrum	Non spec inflammation	
326	3975/11	58	M	S		Colon	Transverse	Nonspec inflammation	
327	4102/11	45	M			Rectum		Adenoca papillary with areas of extracellular mucin	
328	4203/11	30	M			Colon	Descanding ,splenic flexure,distal colon	Flat adenoma	
329	4357/11	49	M			Colon		Nonspec inflammation	
330	4427/11	61	M	P		A-duodenum b-stomach	A-D2 b-prominent ampulla c-ulceroproliferative lesion cardia & fundus	A-adenomatous polyp b-ulcer c-adenoca well diff	
331	4821/11	41	M			Stomach	Body LC	Adenoca poorly diff	
332	4938/11	23	M	S		Stomach	OGJ	Inflammatory polyp	Ct abdomen-?Leiomyoma
333	4974/11	45	M			Stomach	Fundus	Inflammatory fibroid polyp	
334	5213/11 cr-3881/11	22	M			Colon	Rectosigmoid	Infiltr well diff adenoca+tva with high grade dysplasia	
335	5219/11	36	F			Stomach	Antrum	Chronic non-spec gastritis	Gastric outlet obstruction
336	5230/11	70	F			Stomach	Antrum	Chronic non-spec gastritis	Polypoidal growth in fundus with ulcer with bleed; HIV+; previous biopsy - adenomatous polyp
337	5438/11	59	M			Stomach	Fundus	GIST	
338	5476/11	27	M	P		Caecum	Caecum	Tubular adenoma	
339	5487/11	61	M	S	0.5cm	Colon	A-growth descending colon b-polyps colon	Adenoca infiltr well diff	Post GJ status
340	5555/11	61	M			Jejunum	Peristomal	Ulcer	
341	5601/11	39	F			Duodenum		Chronic non-spec duodenitis	Rectal mucosa inflammed; multiple polypoidal lesions from sigmoid c upto

									caecum
342	5630/11 5749/11	29	F			Colon+rectum	A-right colon b-transverse colon; c-left colon; d-rectum	Nhl diffuse type	
343	cr-5487/11, 4427/11	61	M			Stomach,colon	A-gastrectomy;b-right hemicolectomy	Stomach-GIST ; colon-infiltr adenoca	Anemia,dyspepsia
344	5753/11	53	M			Stomach	Antrum	Adenoca infiltr well diff	polypoid lesion 5cm above anal verge
345	5755/11	30	F			Rectum	A-multiple bx rectum b-polyp rectum	A,b-ulcerative colitis active phase	
346	5824/11	50	M			Caecum	Caecum	Chronic non-spec colitis	Multiple polyps throughout colon
347	5838/11	24	M			Colon	20 cm from anal verge	Peutz jeghers polyp	Chronic kidney disease
348	5840/11	37	M			Duodenum	Bulb	Hyperplastic polyp	Peptic ulcer disease/gastric outet obstruction
349	5925/11	70	M			Stomach	Pylorus	Hyperplastic polyp with ulcer	Bleeding per-rectum
350	6032/11	36	F			Rectum		Juvenile polyp	
351	6711/11	70	M	S, P	A-0.75cc;b-1cc	Colon+rectum	Descending colon, rectum	Tubular adenomas with mild atypia	
352	6712/11	45	M			Colon+rectum	A-desc c;b-rectum(15 cm from anal verge)	A-tva mild dysplasia;b-tva severe dysplasia	Ca rectum with liver sec;post ct/rt;ulcero prolif lesion from 3cm to 7cm of anal verge
353	6878/11	54	F			Colon	Rectum	Adenoca poorly diff signet ring cell with adenomatous polyp	
354	6879/11	32	M		2-3mm	Rectum		Tubular adenoma	
355	7144/11	54	M	S		Colon	Caecum	Non-spec inflammation	Ibs;old hyperplastic polyp
356	7145/11	69	M	S		Colon	A-descending colon; b-polyp sigmoid 25cm from AV	Non spec inflammation	
357	7146/11	51	M			Stomach	Antrum	Hyperplastic polyp with ulcer	
358	7568/11	49	F			Stomach	Antrum	Hyperplastic polyp	
359	7615/11	28	M			Colon+stomach	A-gastric mucosa; b-colonic polyps	A-hyperplastic polyp ; b-TVA	
360	8003/11	75	F	P		Stomach	Lesser curvature	Tubulovillous adenoma	
361	8006/11	56	F			Stomach	Antrum	Hyperplastic polyp	
362	8076/11 8118/11	85	M	S		Colon	Splenic flexure	Non-spec inflammation	
363	cr-7048/11	53	M			Colon	Ascending	Adenoca infiltr mod diff	

364	8239/11	48	F			Esophagus	Just above OGJ	Barrett's esophagitis	
365	8561/11	37	F			Rectum		Non-spec inflammation	
366	8610/11	50	M	S		Duodenum	D1	Non-spec duodenitis	
367	8766/11	55	F	P		Colon	Descending	Tubular adenoma	
368	8809/11	40	M			Colon	Ascending	Tubular adenoma	
369	8881/11	55	M	S		Colon	Ascending	Adenoca infiltr well diff	
370	8882/11	57	M	S		Colon	Descending	TVA	Ca rectum ; APR done
371	8932/11	75	M	S		Caecum	On ileocaecal valve	Adenoca well diff	
372	8954/11	25	F			Rectum+caecum	A-polypoidal lesion rectosigmoid; b- biopsy from ileocaecal valve	A- Tubular adenoma B- tuberculosis	
373	8958/11	59	M			Duodenum	D2,D3	TVA	
374	8995/11	42	M			Colon	Descending	Tubular adenoma	
375	9157/11	41	M			Colon	Caecum	Tubular adenoma	
376	9350/11	67	F			Stomach	Pylorus	Hyperplastic polyp	
377	9389/11	39	M			Duodenum	D2	Non-spec duodenitis	
378	9461/11	26	M	P	1.5x1cm	Rectum	A-rectosigmoid b-rectal p 5cm from anal verge	A-ulcerative colitis b- TVA	SOL liver, grade 1 esophageal varices
379	9589/11	75	M	S		Duodenum		Non-spec duodenitis	
380	9770/11	60	M			Rectum		TVA	
381	9789/11	48	M			Colon	A-descending ; b-transverse colon	Crohn's disease	
382	10000/11	60	M			Anal polyp;rectal growth	A-polyp 1.5x2cm;b-noduloprolif growth from 4 to 10 cm of av	A-TVA; B- infiltr well diff adenoca	
383	10003/11	18	M			Jejunum		Tubular adenoma	
384	10008/11	60	M	P		Colon	Sigmoid	TVA	Nodular lesion distal ileum, chronic diarrhoea
385	10103/11	14	M			Ileum		Non spec inflammation	
386	10240/11	26	M			Rectum		Retention polyp	
387	10297/11	50	F			Colon	40cm from AV	Tubular adenomatous polyp with no dysplasia	
388	10303/11	65	F			Stomach	Fundus	Hyperplastic polyp	
389	10312/11	55	F			Stomach	Antrum	Hyperplastic polyp	
390	10339/11	38	M			Colon+rectum	A-colon;b-rectal	Tubular adenomas	Antral gastritis
391	10344/11	66	F			Stomach	Fundus	Hyperplastic polyp	
392	10352/11	61	M			Stomach	Body lc	Adenoca infiltr mucinous type	
393	201/12	55	M			Colon	A-polypoidal lesion asc colon;b-	A-infiltr mod diff adenoca; b-TVA	

							multiple colonic polyps	
394	245/12	34	M			Colon	A-growth asc colon;b-diminutive polyp	Inflammatory polyps
395	255/12	57	M			Colon	Hemicolectomy	TVA;infiltr well diff adenoca
396	400/12	50	M			Stomach	Pylorus	Adenoca infiltr mod diff papillary intestinal type
397	425/12	30	M	S		Colon	A-mid asc colon polypoid growth b-descending colon polyp	A-infiltr mod diff adenoca; b-tubular adenoma
398	493/12	40	F	S		Stomach	Body	Hyperplastic polyp
399	502/12	55	M	S		Colon	Transverse	Tubulovillous adenoma
400	589/12	50	M			Colon	A-multiple polyps b-polypoid growth in descending colon	A-nonspecific ulcer b-tubular adenoma
401	592/12	40	F			Anal		Inflammatory pseudopolyp
402	651/12	66	M			Rectum	A-small polyp at 17cm from AV(rectum) b-growth anorectum (from above anal verge to upto 8cm)	A-hyperplastic polyp; B-adenocarcinoma papillary type
403	656/12 761/12	29	F			Stomach	Polypoidal lesion at OGJ & cardia	Chronic lymphocytic gastritis
404	cr-9788/11	30	F			Jejunum		Tubular adenoma
405	785/12	66	M		<0.5cm	Rectum		Inflammatory pseudopolyp
406	975/12	70	M			Colon	A-polypoidal lesion ileocaecal region b-<0.5cm polyp in desc colon	A-nonspec inflammation b-tubular adenoma
407	1060/12	57	M		<0.5 cm	Rectum		Tubular adenoma with superficial ulceration
408	1298/12	60	F			Rectum		Hyperplastic polyp
409	1407/12	51	M			Colon	Rectosigmoid	Tubular adenoma
410	1442/12	42	F		0.5x0.5cm	Colon	A-hyperemic area in caecum b-pedunculated polyp sigmoid colon	A-chronic non-specific colitis b-hyperplastic polyp
411	1510/12	50	F	S		Colon	Descending	Hyperplastic polyp
412	1511/12	65	M			Colon	Ascending & transverse	Tubular adenoma
413	1542/12	36	M			Duodenum		Hyperplastic polyp
414	1941/12	65	M			Colon	Descending	Hyperplastic polyp
415	1958/12	70	F			Rectum		Tubular adenoma
416	2032/12	37	M	P		Anal		Malignant melanoma

417	2070/12	57	F			Colon r+l	A-polypoidal lesion above ileocaecal valve b-polypoidal lesion in descending colon	A-inflammatory polyp b-tubular adenoma	
418	2071/12	58	M	P		Stomach	Antrum	Hyperplastic polyp	
419	2190/12	60	M			Anal		Hyperplastic polyp	
420	2270/12	65	F	S		Duodenum	D1	Inflammatory pseudopolyp -? H.pylori duodenitis	
421	2312/12	65	M			Colon		Tubular adenoma	C/o constipation
422	2325/12	76	M			Colon	Descending	Tubular adenoma	
423	2350/12	38	M			Colon	Descending	Tubular adenoma with mild dysplasia	
424	2521/12	21	F	S		Caecum	Caecum	Hyperplastic polyp	
425	2528/12	55	M	S		Colon	Hepatic flexure	TVA	
426	2538/12	53	M	S		Colon	Transverse	Well diff adenoca, tubular adenomas	
427	2598/12	45	M			Rectum	10 cm from AV	Tubular adenoma	?Inflammatory Bowel Disease
428	2804/12	40	F	S		Colon	Sigmoid	Non-specific inflammation	
429	2994/12	55	F	S		Stomach	Fundus	Tubular adenoma	
430	2997/12	50	M			Stomach	Fundus	Hyperplastic polyp	
431	3003/12	45	F	S		Stomach	Prepyloric	Hyperplastic polyp	
432	3386/12	45	F			Rectum		TVA	
433	3444/12	50	F			Stomach	Pylorus	Hyperplastic polyps	
434	3800/12	20	F	P		Colon	Descending	TVA	
435	4110/12	78	F	P	3cmx2cm	Colon	Sigmoid	Villous adenoma with mod to severe dysplastic changes	
436	4233/12 cr-4110/12	78	F	P	2cmx1cm	Colon	Sigmoid & ascending	Villous adenoma with severe dysplasia with e/o intramucosal carcinoma	
437	4313/12	33	M	S		Colon	Sigmoid	Hyperplastic polyp	
438	4466/12	68	M	S		Colon+small intestine	A-d3 b-mid transverse colon	A-chronic lymphocytic duodenitis b-hyperplastic polyp	
439	4468/12	39	M			Stomach	Lesser curvature	Hyperplastic polyp/non spec inflammation	
440	4668/12	39	F			Stomach	Body	Hyperplastic polyp	
441	4675/12	49	M	P		Colon	Sigmoid	Inflammatory pseudopolyp	Bleeding PR
442	4744/12	56	M		Diminutive	Rectum		Hyperplastic polyp	
443	4746/12	70	F			Stomach	Beyond OGJ	Non spec inflammation	Ulcerative colitis
444	4960/12	52	M			Caecum	Caecum	Inflammatory pseudopolyp	Bleeding PR; mass descending PR
445	4993/12	56	F	S	4cmx3cmx1cm	Rectum		TVA with malignant transformation	
446	5001/12	60	M			Rectum	6 cm from AV	Well diff adenoca	
447	5051/12	45	F			Stomach	Proximal body	Non specific erosive gastritis	

448	5052/12	60	F			Stomach	Antrum	Hyperplastic polyp	Two polyps
449	5107/12	20	F	P	2cmx2cmx1cm	Colon	Sigmoid	Peutz jeghers polyp(a&b)	
450	5395/12	34	F			Colon	Transverse	Tubular adenoma	
451	5477/12	64	M	S		Colon		Hyperplastic polyp	Multiple polyps throughout colon
452	5520/12	39	M			Colon	Descending, transverse	Tubular adenoma	

IMMUNOHISTOCHEMISTRY :

SL.NO	BX.NO	AGE	SEX	CARCINOMA	p53	bcl2	POLYP	p53	bcl2	COMPARATIVE POLYP	BX.NO	AGE/SEX	p53	bcl2
1	425/12	30	M	MOD DIFF INFILTR ADENOCA	P	P	TA	P	P	TA	3907/11	32/M	N	P
2	5213/11	22	M	WELL DIFF ADENOCA	N	N	TA	N	P	TA	1444/08	22/M	P	N
3	2370/10	70	M	MOD DIFF ADENOCA	P	N	TA	P	P	TA	2729/10	60/M	N	P
4	149/09	44	F	MOD DIFF INFILTR ADENOCA	P	N	TA	N	P	TA	1616/08	51/F	N	N
5	7193/10	65	M	WELL DIFF ADENOCA	P	N	TA	N	P	TA	6711/11	70/M	P	P
9	3541/08	55	M	WELL DIFF PAPILLARY ADENOCA	N	N	TVA	P	P	TVA	2528/12	55/M	N	P
7	201/12	55	M	MOD DIFF INFILTR ADENOCA	P	P	TVA	P	P	TVA	10008/11	60/M	P	P
8	10000/11	60	M	WELL DIFF INFILTR ADENOCA	P	N	TVA	N	P	TVA	8882/11	57/M	P	P
6	3395/11	35	F	MOD DIFF INFILTR ADENOCA	N	N	TVA	P	P	TVA	1662/11	30/F	P	P
10	8720/10	42	M	MOD DIFF MUCIN SECRETING INFILTRATING ADENOCA	P	P	TVA	P	P	TVA	6712/11B	45/M	P	P
11	3075/09	60	M	MOD DIFF INFILTR PAP CA	P	P	TVA	N	P	TVA	502/12	55/M	N	P
12	952/08	53	M	POORLY DIFF MUCINOUS ADENOCA	N	N	TVA	N	P	TVA	8548/08	55/M	N	N
13	255/12	57	M	WELL DIFF INFILTR ADENOCA	P	N	TVA	P	P	TVA	8920/10	65/M	N	N
14	3010/09	30	F	MOD DIFF INFILTR ADENOCA	P	P	VA	P	P	VA	6586/09	32/F	N	N
15	2229/09	47	M	POORLY DIFF ADENOCA	P	P	VA	P	N	VA	662/09	55/M	N	N
16	6907/09	32	F	WELL DIFF ADENOCA	N	N	VA	N	N	VA	1231/11	30/F	P	P

SL.NO	BX.NO	AGE/SEX	CARCINOMA	p53	bcl2	POLYP	p53	bcl2
17	6878/11	54/F	POORLY DIFF SIGNET RING CELL CARCINOMA	P	P	TA	N	P
18	7202/09	79/M	WELL DIFF ADENOCA	P	N	TVA	P	N
19	393/11	70/M	WELL DIFF ADENOCA	P	N	TVA	P	N
20	4993/12	56/F	MOD DIFF ADENOCA	P	N	TVA	P	N
21	4233/12	78/F	WELL DIFF ADENOCA	P	N	VA	P	N

KEY TO MASTERCHART:

TA – tubular adenoma

TVA – tubulovillous adenoma

VA – villous adenoma

MOD DIFF – moderately differentiated

ADENOCA or AC – adenocarcinoma

INFILTR – Infiltrating

P – positive

N – negative

M-male

F-female

AV-anal verge

NON-SPEC INFLAM – non-specific inflammation

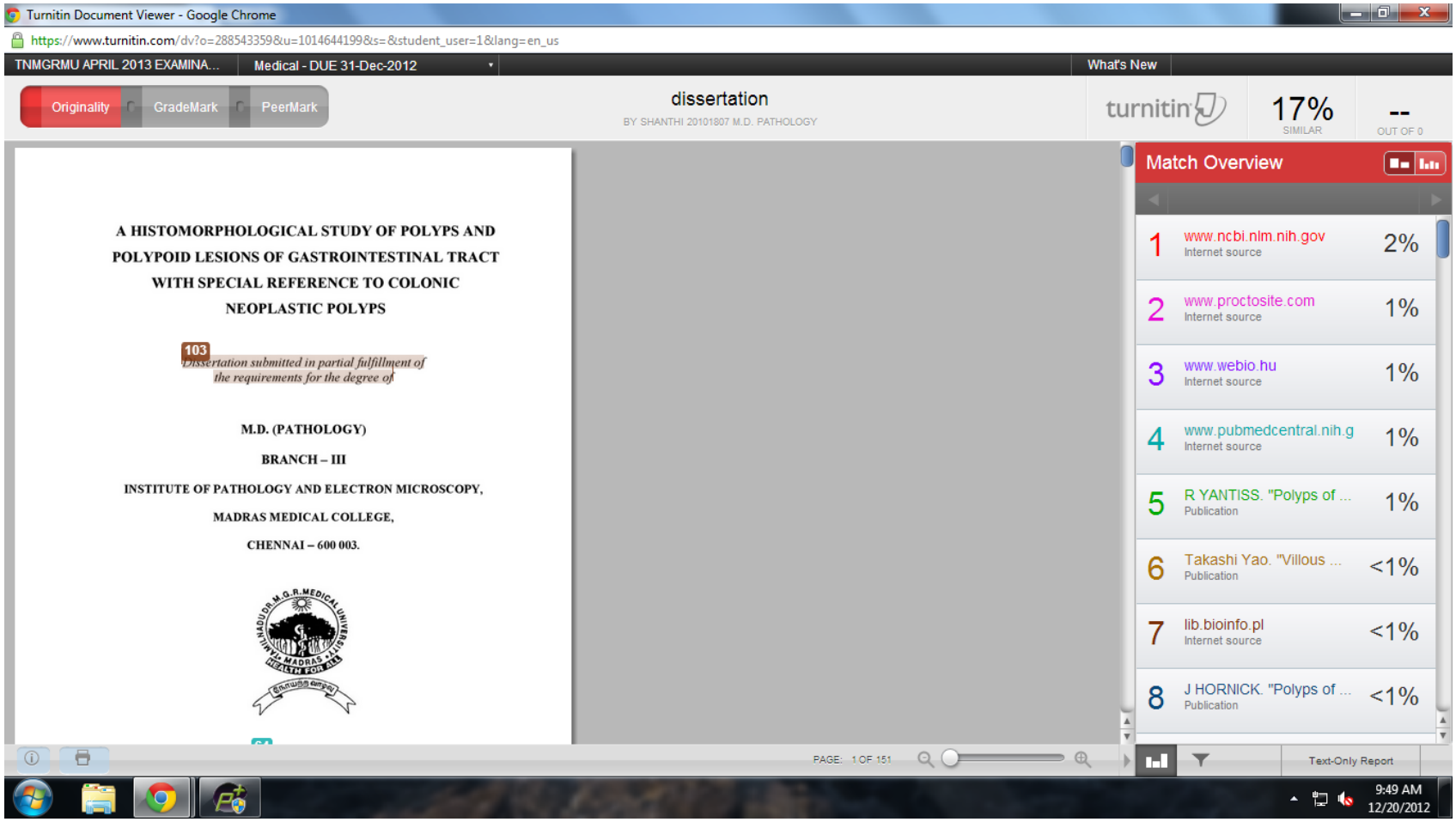
S/P – solitary/pedunculated

LC-lesser curvature of stomach

GC- greater curvature of stomach

Asc colon – ascending colon

SCC – Squamous Cell Carcinoma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A HISTOMORPHOLOGICAL STUDY OF POLYPS AND POLYPOID LESIONS OF GASTROINTESTINAL TRACT WITH SPECIAL REFERENCE TO COLONIC NEOPLASTIC POLYPS
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Dissertation submitted in partial fulfillment of the requirements for the degree of
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BRANCH – III
INSTITUTE OF PATHOLOGY AND ELECTRON MICROSCOPY,
MADRAS MEDICAL COLLEGE,
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