SPECTRUM OF MANIFESTATIONS OF CARCINOMA STOMACH - AN INSTITUITIONAL EVALUATION

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SPECTRUM OF

MANIFESTATIONS OF

CARCINOMA STOMACH

- AN INSTITUTIONAL

EVALUATION

CERTIFICATE

This is to certify that this dissertation entitled "Spectrum of manifestations of cancer stomach " submitted by Dr.P.I.Rajanbabu, to the faculty of Medical Gastroenterology, The Tamilnadu Dr.MGR Medical University, Guindy, Chennai-600032, in partial fulfillment of the requirement for the award of DM., Degree Branch IV (Gastroenterology) is a bonafide work carried out by him under my direct supervision and guidance.

Prof.S.Jeevan Kumar,MD.,DM.,
(Gastroenterology)
Professor and HOD,
Department of Digestive Health and Diseases,
Government Peripheral Hospital, Anna Nagar,
Attached to Kilpauk Medical College,
Chennai-600010

Dr.Kanagasabai MD.,

Dean
Government Kilpauk Medical College, Kilpauk, Chennai-600010

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INTRODUCTION

INTRODUCTION

Despite a major decline in incidence and mortality over several decades, stomach cancer is still the fourth most common cancer and the second most common cause of cancer death in the world. There is a 10-fold variation in incidence between populations at the highest and lowest risk. The incidence is particularly high in East Asia, Eastern Europe, and parts of Central and South America, and it is about twice as high among men than among women. Prognosis is generally rather poor, with 5-year relative survival below 30% in most countries.

The best established risk factors for stomach cancer are *Helicobacter pylori* infection, the by far strongest established risk factor for distal stomach cancer, and male sex, a family history of stomach cancer, and smoking . While some factors related to diet and food preservation, such as high intake of salt-preserved foods and dietary nitrite or low intake of fruit and vegetables, are likely to increase the risk of stomach cancer, the quantitative impact of many dietary factors remains uncertain, partly due to limitations of exposure assessment and control for confounding factors.

REVIEW OF LITERATURE

Epidemiology of Stomach Cancer

Gastric cancer remains one of the most common forms of cancer worldwide with approximately 870,000 new cases and 650,000 deaths per year [1,2] accounting for about 9.9 percent of new cancers [3].

The worldwide incidence of gastric cancer has declined rapidly over the recent few decades. Part of the decline may be due to the recognition of certain risk factors such as H. pylori and other dietary and environmental risks. The decline first took place in countries with low gastric cancer incidence such as the United States, while the decline in countries with high incidence like Japan was slower.

Gastric cancer used to be the leading cause of cancer deaths in the world until the 1980s when it was overtaken by lung cancer [4]. According to data collected by the World Health Organization, the most common forms of cancer worldwide are lung (12.3 percent), breast (10.4 percent), and colorectal (9.4 percent) while the top three causes of death from cancer are lung (17.8 percent), gastric (10.4 percent), and liver (8.8 percent).

An interesting hypothesis is that the popularization of refrigerators marks a pivotal point for the decline [5]. Refrigerators improved the storage of food, thereby reducing salt-based preservation of food and preventing bacterial and fungal contamination. Refrigeration also allowed for fresh food and vegetables to be more readily available, which may be a valuable source of antioxidants important for cancer prevention.

Race

The rates of gastric cancer are higher in Asian and South American countries than in the United States. Japan, Chile, and Venezuela have developed a very rigorous early screening program that detects patients with early stage disease (ie, low tumor burden). These patients appear to do quite well. Some researchers suggest that this reflects a fundamental biologic difference in the disease as it manifests in Western countries.

In the United States, Asian and Pacific Islander males and females have the highest incidence of stomach cancer, followed by black, Hispanic, white, American Indian, and Inuit populations.

Sex

In the United States, gastric cancer affects slightly more men than women; the American Cancer Society estimated that in 2009, 12,820 new cases will

occur in men and 8,310 in women.[4]Worldwide, however, gastric cancer rates are about twice as high in men as in women.[1]

Age

Most patients are elderly at diagnosis. The median age for gastric cancer in the United States is 70 years for males and 74 years for females. The gastric cancers that occur in younger patients may represent a more aggressive variant or may suggest a genetic predisposition to development of the disease. It appears that higher geographic latitudes are associated with a higher gastric cancer risk. Global incidence of stomach cancer in men: the highest rates occur in Eastern Asia, South America and Eastern Europe.

Migration studies — Migration, and in particular, international migration, can lead to a change in risk, as the immigrants, especially second and third generations, adopt the lifestyle and consequently the local disease patterns. The risk of gastric cancer changes slowly in populations moving from high to low risk communities. Studies of Japanese migrants to the Unites States have confirmed that early exposure to environmental rather than genetic factors have a greater influence on mortality and incidence rates[6,7]. In the subsequent generations born in the United States, the mortality rate declined towards the lower rate of United States whites.

Change in histology pattern — It is more prevalent in high-risk areas and is likely linked to environmental factors. The diffuse type, or infiltrative type, is equally frequent in both sexes, is more common in younger age groups, and has a worse prognosis than the intestinal type. There has been a worldwide decline in the incidence of the intestinal type in recent few decades that parallels the overall decline in the incidence of gastric cancer. By contrast, the decline in the diffuse type has been more gradual.

Despite the decline in gastric cancer overall, there has been an explosive increase in incidence of cancer of the gastric cardia[8-10]. The shift from distal to proximal stomach may in part be due to the decrease in the distal cancers.

The proximal tumors share demographic and pathological features with Barrett's associated esophageal adenocarcinoma and are more likely to occur in men, which parallels the male predominance in the increasing incidence of carcinoma in the lower third of the esophagus. The proximal tumors also differ from distal tumors in that they are not associated with a severe form of gastritis characterized by atrophy and/or intestinal metaplasia. Furthermore, they tend to be more aggressive than those arising from distal sites. Environmental factors or chemical carcinogens (eg, cigarette and alcohol) may be more strongly associated with cardiac carcinomas compared with more distal carcinomas [11].

Epidemiology of Gastric cancer in India

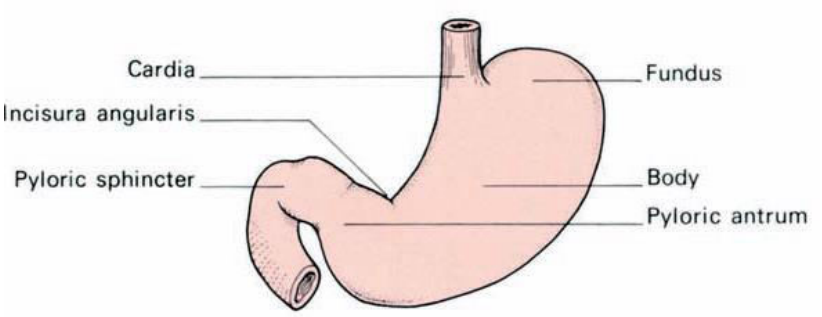
In India, across the various registries, there is a wide variation in the incidence of gastric carcinoma. The incidence rate of gastric cancer is four times higher in Southern India compared with Northern India. Among the six registries, the highest incidence in both sexes is reported from Chennai and the lowest from Barshi. The incidence varies among different religious groups also. In Kashmir, Muslims have a higher incidence compared with Hindus, whereas the reverse trend is seen in Mumbai[12].

Site of lesion of cancer stomach in India

Worldwide, the incidence of proximal gastric carcinoma is on the increase. In India also a trend towards an increase in the incidence of cardia tumors is seen. This is evident in the data from Mumbai, where the percentage of cardia and fundus tumors increased from 13% in 1941–1968 to 23% in 1987–1993 [13]. Nearly 95% of the tumors are adenocarcinomas. These may be further distinguished as intestinal and diffuse types. Intestinal type is seen more commonly than diffuse type in India. In India more than 90% of all gastric cancers are diagnosed in an advanced stage, and in those subjected to surgery more than 70% have serosal infiltration [13].

ANATOMY

The stomach is roughly J-shaped, although its size and shape vary considerably. The stomach has two surfaces—the anterior and posterior; two curvatures — the greater and lesser; and two orifices — the cardia and pylorus.



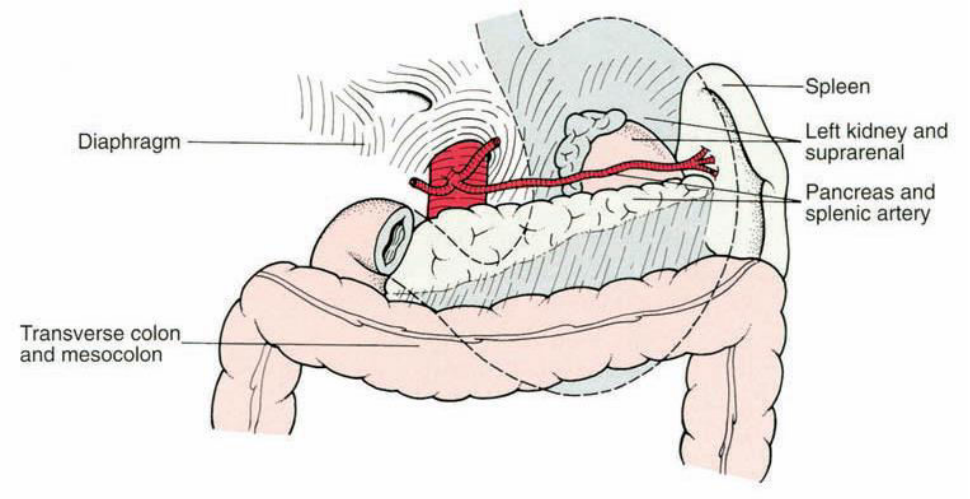
The stomach projects to the left, above the level of the cardia, to form the dome-like gastric fundus. Between the cardia and the pylorus lies the body of the stomach leading to a narrow portion, immediately preceding the pylorus, which is termed the pyloric antrum. The junction of the body with the pyloric antrum is marked by a distinct notch on the lesser curvature termed the incisura angularis. The junction of pylorus with duodenum is marked by a constriction externally and also by a constant vein (of Mayo) which crosses it at this level. The thickened pyloric sphincter is easily felt and surrounds the lumen of the pyloric canal. The pyloric sphincter is an anatomical structure as well as a physiological mechanism.

Relations of the stomach

•**Anteriorly** — the abdominal wall, the left costal margin, the diaphragm and the left lobe of the liver.

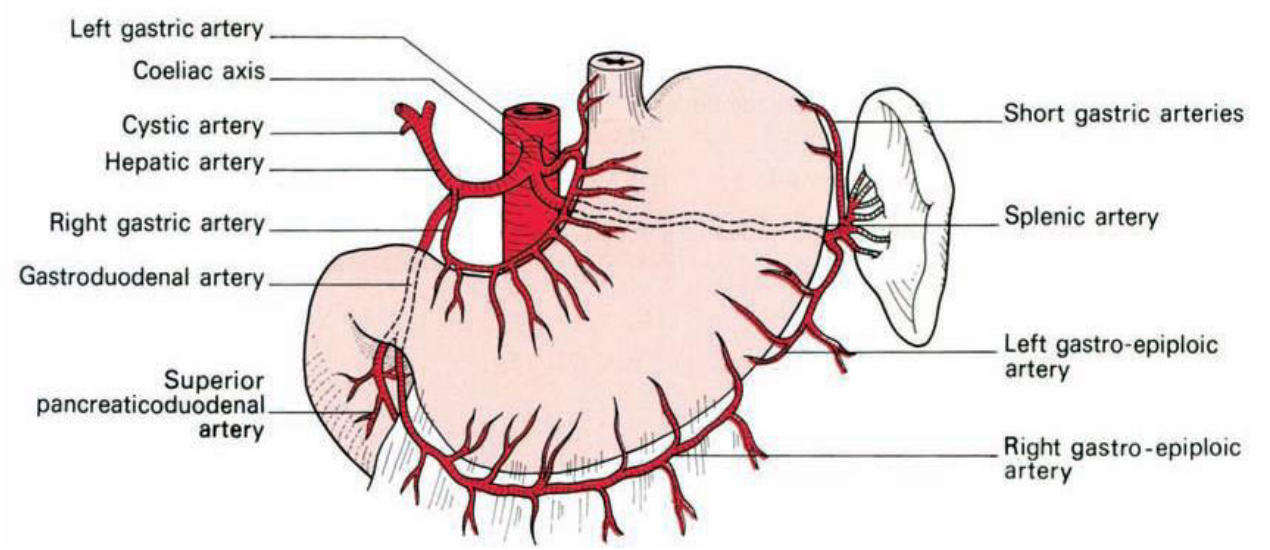
•**Posteriorly**—the lesser sac, which separates the stomach from the pancreas, transverse mesocolon, left kidney, left suprarenal, the spleen and the splenic artery.

•**Superiorly**—the left dome of the diaphragm. The lesser omentum is attached along the lesser curvature of the stomach, the greater omentum along the greater curvature. These omenta contain the vascular and lymphatic supply of the stomach.



Relations of the stomach

The blood supply to the stomach



The arterial supply of the stomach.

•The left gastric artery—from the coeliac axis;

•The right gastric artery—from the hepatic artery;

•The right gastro-epiploic artery—from the gastroduodenal branch of the hepatic artery;

•The left gastro-epiploic artery—from the splenic artery;

•The short gastric arteries—from the splenic artery.

The corresponding veins drain into the portal system.

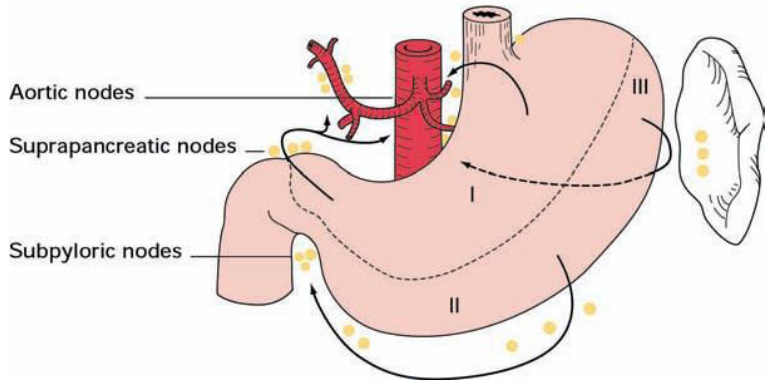
The lymphatic drainage of the stomach accompanies its blood vessels.

Drainage zones of the stomach

•Area I—the superior two-thirds of the stomach drain along the left and right gastric vessels to the aortic nodes.

•Area II—the right two-thirds of the inferior one-third of the stomach

•Area III — the left one-third of the greater curvature of the stomach drains along the short gastric and splenic vessels lying in the gastrosplenic and lienorenal ligaments, then, via the suprapancreatic nodes, to the aortic group.



The lymph drainage of the stomach & three drainage zones.

The lymph drainage of the stomach

•Area I drains along the right and left gastric vessels to the aortic nodes.

•Area II drains to the subpyloric and thence aortic nodes via lymphatics along the right gastro-epiploic vessels.

•Area III drains via lymphatics along the splenic vessels to the suprapancreatic nodes and thence to aortic nodes. drain along the right gastro-epiploic vessels to the subpyloric nodes and thence to the aortic nodes[14].

This extensive lymphatic drainage and the technical impossibility of its complete removal is one of the serious problems in dealing with stomach cancer. Involvement of the nodes along the splenic vessels can be dealt with by removing spleen, gastrosplenic and lienorenal ligaments and the body and tail of the pancreas. Lymph nodes among the gastro-epiploic vessels are removed by excising the greater omentum. However, involvement of the nodes around the aorta and the head of the pancreas may render the growth incurable.

Risk factors for gastric cancer

PRECURSOR LESIONS

Atrophic gastritis — Atrophic gastritis is characterized by progressive atrophy of the glandular epithelium with loss of parietal and chief cells. The loss of the normal exocrine glands of the gastric mucosa causes hypochlorhydria and a resultant increase in gastric pH. An abnormally high pH in the stomach permits microbial colonization, some of which possess nitrate reductase, allowing

nitrosation that is genotoxic. In addition, there is a loss of endocrine cells, which normally secrete epidermal and transforming growth factors, thereby aiding the stomach in regenerating damaged tissue. Populations with a high prevalence of atrophic gastritis have a high prevalence of gastric cancer, and vice-versa [15].

Intestinal metaplasia — Metaplasia is a potentially reversible change from a fully differentiated cell type to another cell type, a process in adaptation to environmental stimuli. The most common form of metaplasia in the stomach is the intestinal type. It occurs as a result of Helicobacter pylori infection, bile reflux, or can be induced experimentally by irradiation [16]. Intestinal metaplasia is more frequent in countries with a higher incidence of gastric carcinoma [17], and precedes gastric carcinoma [18].

Dysplasia — Most patients diagnosed with high-grade dysplasia of the gastric mucosa either already have or soon develop gastric cancer. In gastrectomy specimens for gastric cancer, 20 to 40 percent of patients had associated dysplasia [19]. Progression of dysplasia to gastric cancer has been estimated at 21 percent, 33 percent, and 57 percent of cases of mild, moderate, and severe dysplasia, respectively [20]. Epidemiological studies have shown that intestinal metaplasia and dysplasia in the stomach have a high cancer risk with high and low cancer risk [19]. The prevalence of intestinal metaplasia and dysplasia was much higher in areas with high risk for gastric cancer.

A widely accepted model of gastric cancer describes a progression from chronic gastritis to chronic atrophic gastritis, to intestinal metaplasia, dysplasia, and eventually to adenocarcinoma.

Similarly, gastric resection results in hypo- or achlorhydria, secondary hypergastrinemia, and bile reflux, especially after a Billroth II anastomosis. The increase in gastric pH would permit colonization of bacteria capable of converting dietary nitrates to potent mutagenic N-nitroso compounds. Chronic inflammation also results in epithelial cell damage with increased free radical generation, a further reduction in luminal ascorbic acid levels, and increased cell turnover.

This triad of events increased cell proliferation due to the promotional effects of hypergastrinemia and/or bile reflux, increased luminal levels of mutagens (eg, N-nitroso compounds and free radicals), and decreased luminal levels of protective factors (eg, vitamin C) provide an ideal milieu for enhanced carcinogenesis in susceptible hosts. The role of specific genetic alterations in this model remains unknown.

Environmental risk factors — Emigrants from high-incidence to low-incidence countries often experience a decreased risk of developing gastric carcinoma. Such findings strongly suggest that environmental factors have an

important role in the etiology of gastric cancer and that exposure to risk factors occurs early in life.

Diet — Large epidemiologic studies demonstrating the association between diet and gastric cancer were based mainly upon the amount of food imported and produced rather than the actual food consumption [21]. This takes no account of the losses during storage, distribution, and consumption of food or any ethnic dietary differences. Nevertheless, the information provides important insight into environmental causes of gastric cancer.

Nitroso compounds —One of the most consistent associations has been with dietary exposure to N-nitroso compounds[22]. N-nitroso compounds are generated after consumption of nitrates, which are natural components of foods like vegetables and potatoes and are used as a food additive in some cheeses and cured meats. Dietary nitrates are absorbed in the stomach and secreted in saliva in a concentrated form where they are reduced to nitrites by oral bacteria. Nitrites can react with nitrosatable compounds like amines, amides, and amino acids to form N-nitroso compounds. An increase in gastric nitrite has observed in patients with intestinal metaplasia, dysplasia, and gastric cancer.

The uses of nitrate-based fertilizers [23] and pickled foods that contain nitrosated products correlates with gastric cancer . Diets low in vegetables,

fruits, milk, and vitamin A and high in fried food, processed meat, and fish and alcohol have been associated with an increased risk of gastric carcinoma in several epidemiologic studies [24].

Diets low in citrus fruit show the strongest association with gastric carcinoma [25]. The protection afforded by vegetables and fruits is most likely related to their vitamin C content, which is thought to reduce the formation of carcinogenic N-nitroso compounds inside the stomach. Cooked vegetables do not show the same protective effect as uncooked vegetables.

Salt — High salt intake damages stomach mucosa and increases the susceptibility to carcinogenesis in rodents [26]. The risk of high salt intake was strongest in patients who had both H. pylori infection and atrophic gastritis [27].

Folate — A meta-analysis of epidemiology studies found an inconsistent association between dietary folate and the risk of gastric cancer [28].

Smoking — Several studies have examined the relationship between tobacco smoking and gastric cancer. A meta-analysis of 40 studies estimated that the risk was increased by approximately 1.5 to 1.60-fold and was higher in men[29]. A subsequent prospective study from Europe found a similar magnitude of risk, which diminished after 10 years of smoking cessation[30]. Approximately 18 percent of gastric cancer cases were attributed to smoking.

Alcohol — A consistent association between alcohol consumption and the risk of gastric cancer has not been demonstrated. A study from Europe suggested that daily intake of wine may be protective [31].

Socioeconomic status —The risk of distal gastric cancer is increased by approximately twofold in populations with low socioeconomic status[32]. By contrast, proximal gastric cancers have been associated with higher socioeconomic class [33].

Gastric surgery — There is an increased risk of gastric cancer after gastric surgery, with the risk being greatest 15 to 20 years after surgery and then increasing with time [34]. The Billroth II procedure (gastrojejunostomy) carries a higher risk than the Billroth I (gastroduodenostomy). Although the exact cause of the increased risk is unknown, it is thought to be due to regurgitation of alkaline bile and pancreatic juice.

Epstein-Barr virus — The Epstein-Barr virus (EBV) is associated with a number of malignancies, especially nasopharyngeal carcinoma. A possible role in gastric cancer was suggested in a study from Korea in which evidence of EBV was found in the tumor cells of 12 of 89 (13.5 percent) gastric carcinoma patients compared to none of 27 controls with a benign ulcer or any of the

benign tissues from the cases [35]. Some of the tumor cells had a histologic appearance similar to nasopharyngeal carcinoma.

Since then, it has been estimated that between 5 and 10 percent of gastric cancers worldwide are associated with EBV [36]. EBV-associated gastric cancers are characterized by DNA methylation of the promoter region of various cancer-associated genes, which silences the expression of these genes.

EBV-associated gastric cancers have distinct clinicopathologic characteristics, including male predominance, preferential location in the gastric cardia, a diffuse type of histology, and perhaps a more favorable prognosis.

Helicobacter pylori — The World Health Organization's International Agency for Research on Cancer classified Helicobacter pylori as a Group 1 or definite carcinogen [37]. As noted above, gastric carcinoma is believed to evolve as a progression from atrophy to metaplasia, dysplasia and then carcinoma. The most common cause of gastritis is Helicobacter pylori.

Three sources of evidence support the association of H. pylori infection and gastric cancer: epidemiologic studies comparing gastric cancer and H. pylori infection prevalence rates, cross-sectional studies evaluating H. pylori infection in cancer patients, and prospective studies associating H. pylori with gastric cancer. Histologic association of the bacteria with tumor can be difficult to

determine because H pylori has an affinity for normal gastric mucosa but not metaplastic, dysplastic, or malignant tissue [38].

Helicobacter pylori infection may trigger inflammation at the corpus mucosa that results in atrophy and intestinal metaplasia. H. pylori infection has been associated with an approximate 6-fold increase in the risk with adenocarcinomas distal to the cardia, including both intestinal and diffuse types.

A paradox in H. pylori infection is that divergent clinical outcomes occur: patients may develop duodenal ulcer or gastric cancer, while the majority develop no significant clinical symptoms. Bacterial virulence factors alone have not adequately explained why the ulcer or the gastric cancer phenotype develops.

HOST-RELATED FACTORS

Blood group — Individuals of blood group A have been known for decades to show an approximately 20 percent excess of gastric cancer than those of group O, B, or AB [40]. They also show a similar increase in the rate of pernicious anaemia. Some data suggest that group A may be particularly associated with the diffuse type of gastric cancer [39]. It is possible that the observed associations are not due to the blood group antigens themselves, but to the effects of genes closely associated with them.

Familial predisposition — A genetic predisposition to gastric cancer has been repeatedly confirmed [41]. A genetic predisposition for chronic atrophic gastritis, a precursor of gastric carcinoma, has been described and may account for at least some cases of familial gastric cancer [42]. Gastric cancer has been described in association with certain cancer syndromes (including hereditary nonpolyposis colorectal cancer, familial adenomatous polyposis, and Peutz Jeghers syndrome).

A germline mutation within the E-cadherin gene (CDH1) was identified in a New Zealand family with diffuse gastric cancer[43] and in a European family in 1998 [44]. Germ-line truncating mutations of the E-cadherin gene are detected in 50 percent of diffuse-type gastric cancers [45].

The disorder has an autosomal dominant pattern of inheritance. The lifetime cumulative risk for advanced gastric cancer has been estimated to be 67 percent in men and 83 percent in women. Affected patients generally develop gastric cancer at an average age of 38 years.

Genetic polymorphisms — The human interleukin 1 beta (IL-1B) gene is the most important candidate gene in the host that could affect the clinical outcome of H. pylori infection because it is upregulated by infection, is profoundly proinflammatory, and is the most powerful acid inhibitor known

Gastric polyps — Gastric polyps are present in less than 1% of the general population.[48] Up to 90% of these lesions are hyperplastic polyps, which usually remain small, rarely exceeding 1.5 cm. The rate of malignant transformation is generally quite low (<1%) and confined to polyps larger than 1 cm.[49] The rare hyperplastic polyps that do undergo malignant transformation often form well differentiated intestinal-type cancer. Less common are adenomas in the stomach, which constitute less than 10% of gastric polyps. However, gastric adenomas undergo malignant transformation at a high rate. Gastric adenomas can progress to dysplasia and then carcinoma in situ, which develops within 4 years of follow-up in approximately 11% of cases[50].

Hypertrophicgastropathy and immunodeficiencysyndromes — Hypertrophic gastropathy (including Menetrier's disease) and various immunodeficiency syndromes have been linked with gastric cancer. However, the strength of these associations remains undefined.

Gastric ulcer — Despite the known associations between H. pylori infection and peptic ulcer disease and H. pylori infection and gastric cancer, the association between benign gastric ulcer disease and gastric cancer remains controversial [46]. The risk of gastric cancer was increased among patients with benign gastric ulcers (incidence ratio 1.8), unchanged among patients with

prepyloric ulcers, and decreased among those with benign duodenal ulcers (incidence ratio 0.6).

Pernicious anemia — Pernicious anemia, a sequela of autoimmune chronic atrophic gastritis directed against hydrogen-potassium ATPase in the gastric parietal cells, is associated with an increased risk of intestinal-type gastric cancer. A two- to threefold excess risk has been reported [47] but, as with other predisposing conditions, the actual degree of risk varies with the duration of disease and geographic location.

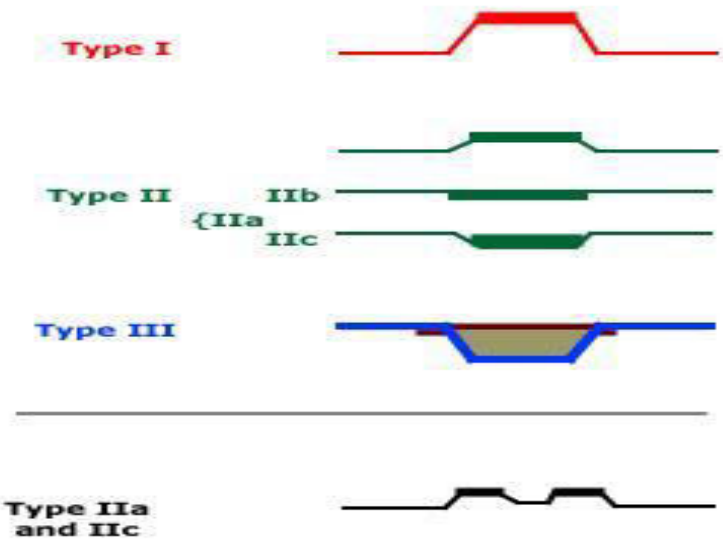
Pathology of gastric cancer

Early gastric cancer

Early gastric cancers, defined as tumors confined to the mucosa or submucosa regardless of lymph node status.

Classification of early gastric cancer

Type I - lesions protrude into the lumen and appear on endoscopy as a polyp on a short, broad-based stalk with an elevation greater than 5 mm. These protrusions are occasionally multiple but remain localized. They may be confused with thickened mucosal folds.



Type II - lesions are superficial with an uneven surface. These changes are subtler than type I lesions, and multiple biopsies are necessary for suspected lesions. Three subtypes are described:

A. Type IIa has a slightly elevated surface of less than 5 mm

B. Type IIb is characterized by a surface that is flat but irregular with no distinct elevations or depressions.

C. Type IIc is a localized area of depression of less than 1.5 cm in area without penetration of the muscularis mucosa.

Type III - is a localized shallow excavation or ulceration in the gastric lining.

Types II and III account over 60 percent of early gastric cancers.

Advanced gastric cancer

Advanced cancers commonly present as polypoid or fungating masses with superficial ulceration. Superficial spreading or infiltrating (linitis plastica) forms are less frequently seen.

Histology — The vast majority of malignant neoplasms of the stomach are adenocarcinomas.

Classifications of Advanced gastric cancer

The intestinal or expanding type	The diffuse or infiltrative type
Resembles colorectal cancer and is characterized by distinct glands comprised of well differentiated columnar epithelial cells with a well developed brush border.	Characterized by poorly organized clusters or solitary mucin-rich (signet ring) cells and a diffusely infiltrating growth pattern.
Predominate in high-risk populations	Predominates in women
Is more common in men and older patients	Younger patients
Is associated with a better prognosis	Carries a poorer prognosis
Is often preceded by a prolonged precancerous state.	Is not preceded by a known precancerous lesion.

- **Lauren's classification system**
  - intestinal
  - diffuse types [51]
- **Ming's classification system**
  - expanding and
  - infiltrating types[52].

CLINICAL FEATURES AND DIAGNOSIS OF GASTRIC CANCER

Clinical features — Weight loss and persistent abdominal pain are the most common symptoms at initial diagnosis [53].

Presenting symptoms of gastric cancer are

Symptom	Percent
Weight loss	62
Abdominal pain	52
Nausea	34
Dysphagia	26
Melena	20
Early satiety	18
Ulcer-type pain	17

Weight loss usually results from insufficient caloric intake rather than increased catabolism, and may be attributable to anorexia, nausea, abdominal pain, early satiety, and/or dysphagia.

The abdominal pain tends to be epigastric, vague and mild early in the disease, but more severe and constant as the disease progresses.

Dysphagia is a common presenting symptom in patients with cancers arising in the proximal stomach or at the esophagogastric junction.

Patients may also present with nausea or early satiety from the tumor mass, or in cases of an aggressive form of diffuse-type gastric cancer called linitis plastica, from poor distensibility of the stomach. They may also present with a gastric outlet obstruction from an advanced distal tumor.

Occult gastrointestinal bleeding with or without iron deficiency anemia is not uncommon, while overt bleeding (ie, melena or hematemesis) is seen in less than 20 percent of cases. The presence of a palpable abdominal mass is the most common physical finding and generally indicates long-standing, advanced disease [53].

Approximately 25 percent of patients have a history of gastric ulcer. All gastric ulcers should be followed to complete healing, and those that do not heal should undergo resection [53].

Signs of tumor extension

More unusual presentations, related to the propensity of gastric cancer to spread by direct extension through the gastric wall, can also alert the clinician to the diagnosis.

Signs of spread via lymphatics

- Virchow's node
  - Since gastric cancer can spread via lymphatics, the physical examination may reveal a left supraclavicular adenopathy which is the most common physical examination finding of metastatic disease,
- Sister Mary Joseph's node
  - A periumbilical nodule
- Irish node
  - A left axillary node.

Signs of Peritoneal spread

- Krukenberg's tumor
  - Peritoneal spread can present with an enlarged ovary

- Blumer's shelf
    - a mass in the cul-de-sac on rectal examination.
- Ascites
    - can be the first indication of peritoneal carcinomatosis.

A palpable liver mass can indicate metastases, although metastatic disease to the liver is often multifocal or diffuse.

Paraneoplastic manifestations —

Dermatologic manifestations

- sign of Leser-Trelat - the sudden appearance of diffuse seborrheic keratoses
- acanthosis nigricans- characterized by velvety and darkly pigmented patches on skin folds.

Neither finding is specific for gastric cancer.

Other paraneoplastic manifestations

- Microangiopathic hemolytic anemia
- Membranous nephropathy
- Hypercoagulable states (Trousseau's syndrome).

- ❖ Polyarteritis nodosa has been reported as the single manifestation of an early and surgically curable gastric cancer.

DIAGNOSIS

Endoscopy — Tissue diagnosis and anatomic localization of the primary tumor are best obtained by upper gastrointestinal endoscopy. Upper endoscopy is also more sensitive and specific for diagnosing a variety of gastric, esophageal and duodenal lesions than alternative diagnostic strategies . The early use of upper endoscopy in patients presenting with gastrointestinal complaints may be associated with a higher rate of detection of early gastric cancers.

Endoscopic techniques — During endoscopy, any suspicious-appearing gastric ulceration should be biopsied. A single biopsy has a 70 percent sensitivity for diagnosing an existing gastric cancer, while performing seven biopsies from the ulcer margin and base increases the sensitivity to greater than 98 percent [54].

The diagnosis of a particularly aggressive form of diffuse-type gastric cancer, so called "linitis plastica," can be difficult endoscopically. Because these tumors tend to infiltrate the submucosa, superficial mucosal biopsies may be falsely negative. For this reason, the combination of strip and bite biopsy techniques should be used when there is a suspicion of a diffuse type of gastric cancer [55].

Barium studies — Barium studies can identify both malignant gastric ulcers and infiltrating lesions. However, false-negative barium studies can occur in as many as 50 percent of cases [56].

Thus, in most settings, upper endoscopy is the preferred initial diagnostic test for patients in whom gastric cancer is suspected. The one scenario in which a barium study may be superior to upper endoscopy is in patients with linitis plastica. The decreased distensibility of the stiff, "leather-flask" appearing stomach is more obvious on the radiographic study, and the endoscopic appearance may be relatively normal.

STAGING AND PREOPERATIVE EVALUATION

Staging systems — There are two major classification systems currently in use for gastric cancer. The most elaborate, the Japanese classification, is based upon refined anatomic location, particularly of the lymph node stations [57]. The other staging system, developed jointly by the American Joint Committee on Cancer (AJCC) and the International Union Against Cancer (UICC) is the classification most often used in the Western hemisphere, and increasingly, in Asian countries as well.

TNM staging criteria — The staging schema of the AJCC/UICC is based on tumor (T), node (N), and metastasis (M) classifications[58].

American Joint Committee on Cancer staging for gastric cancer

Tumor (T) stage

TX Primary tumor cannot be assessed
T0 No evidence of primary tumor
T1s Carcinoma in situ: intra-epithelial tumor without invasion of the lamina propia
T1 Tumor invades lamina propria or submucosa
T2 Tumor invades muscularis propria or subserosa
T2a Tumor invades muscularis propria
T2b Tumor invades subserosa
T3 Tumor penetrates serosa (visceral peritoneum) without invasion of adjacent structures*
T4 Tumor invades adjacent structures*

Nodal (N) stage

NX Regional lymph node(s) cannot be assessed
N0 No regional lymph node metastasis
N1 Metastasis in 1 to 6 regional lymph nodes
N2 Metastasis in 7 to 15 regional lymph nodes
N3 Metastasis in more than 15 regional lymph nodes

Metastasis (M) stage

Mx Presence of distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis

Stage grouping

Stage 0	Tis N0 M0		
Stage 1A	T1 N0 M0		
Stage 1B	T1 N1 M0	T2a/b N0 M0	
Stage II	T1 N2 M0	T2a/b N1 M0	T3 N0 M0
Stage IIIA	T2a/b N2 M0	T3 N1 M0	T4 N0 M0
Stage IIIB	T3 N2 M0		
Stage IV	T1-3 N3 M0	T4 N1-3 M0	

- T stage is dependent on depth of tumor invasion and not size.
- Nodal stage is based upon the number of positive lymph nodes.
- Patients who have no obvious visceral metastases but who have 16 or more pathologically involved nodes are classified as having stage IV disease, which accurately reflects the poor prognosis for these patients.

The validity of the AJCC stage groupings is well established. Five-year survival rates range from 78 percent for patients with superficial T1 tumors and negative lymph nodes (stage IA disease) to 7 to 8 percent for patients with N3 nodes or any distant metastases [59].

Clinical staging and the selection of treatment — Although staging is most accurately determined through surgical pathology, clinical staging directs the initial approach to therapy: Patients who appear to have locoregional disease

(stage I to III) after preoperative testing are potentially curable and should be referred for multidisciplinary evaluation to identify the best treatment strategy. Patients who have T1 to T3, N1 or N2 tumors considered operable and resectable could be referred for initial surgery. However, neoadjuvant therapy, particularly on a clinical trial, is also a reasonable approach. Patients with advanced stage IV disease are usually referred for palliative therapy depending on their symptoms and functional status. Multiple studies indicate both longer survival and better quality of life with systemic treatment.

Abdominopelvic CT scan — Dynamic computerized tomography (CT) scan imaging is usually performed early in the preoperative evaluation after a diagnosis of gastric cancer is made. CT is widely available and noninvasive. It is best suited to evaluating widely metastatic disease, especially hepatic or adnexal metastases, ascites, or distant nodal spread. Patients who have CT-defined visceral metastatic disease can avoid unnecessary surgery, although biopsy confirmation is recommended because of the risk of false-positive findings.

Peritoneal metastases and hematogenous metastases smaller than 5 mm are frequently missed by CT, even using advanced techniques. In 20 to 30 percent of patients with a negative CT, intraperitoneal disease will be found at either staging laparoscopy or at open exploration.

A disadvantage of CT is its limited accuracy for assessing the depth of primary tumor invasion (particularly with small tumors) and the presence of lymph node involvement. CT accurately assesses the T stage of the primary tumor in only about 50 to 70 percent of cases [31]. Disease is more often understaged because the depth of invasion is underestimated; however, overstaging also occurs.

The accuracy of CT for determining regional lymph node involvement is also marginal. The classification of nodal status is usually based on lymph node size, and sensitivity is limited for involved nodes that are smaller than 0.8cm. Furthermore, false-positive findings may be attributed to inflammatory lymphadenopathy. Sensitivity and specificity rates for detection of regional nodal metastases are ranging from 65 to 97 [60].

Endoscopic ultrasonography — Endoscopic ultrasonography (EUS) is thought to be the most reliable nonsurgical method available for evaluating the depth of invasion of primary gastric cancers, particularly for early (T1) lesions[61]. The accuracy of EUS for differentiation of individual tumor stages (T1 to T4) ranges from 77 to 93 percent[62], with the experience of the operator markedly influencing these rates.

EUS-guided fine needle aspiration of suspicious nodes and regional areas adds to the accuracy of nodal staging [64]. Over staging can also occur that is attributed to inflammation around the tumor or in lymph nodes[63]. Distinguishing T2 from T3 lesions is especially difficult because of this problem.

EUS is not recommended for pretreatment evaluation of gastric cancer in guidelines from the National Comprehensive Cancer Network (NCCN) [65].

EUS staging is perhaps of greatest utility for patients with early gastric cancer because accurate assessment of submucosal invasion is essential before considering the option of endoscopic mucosal resection.

PET scan — The role of positron emission tomography (PET) using 18-fluorodeoxyglucose (FDG) in the preoperative staging of gastric adenocarcinoma is evolving. From the standpoint of locoregional staging, integrated PET/CT imaging can be useful to confirm malignant involvement of CT-detected lymphadenopathy[66]. However, this usually does not impact the decision to proceed to surgery. Furthermore, a negative PET is not helpful since even large tumors with a diameter of several centimeters can be falsely negative if the tumor cells have a fairly low metabolic activity. This scenario is fairly common in gastric cancer, particularly the diffuse type [67,68].

The main benefit of PET is that it is more sensitive than CT for the detection of distant metastases [68]. An important caveat is that the sensitivity of PET scanning for peritoneal carcinomatosis is only approximately 50 percent [69]. Thus, PET is not an adequate replacement for staging laparoscopy.

NCCN guidelines for preoperative evaluation of gastric cancer suggest integrated PET/CT [65].

Chest imaging — A preoperative chest x-ray is recommended in patients with gastric cancer [65]. However, the sensitivity for metastases is limited, and a chest CT scan is preferred (particularly for patients with a proximal gastric cancer) if the detection of intrathoracic disease would alter the treatment plan.

Serologic markers — Serum levels of carcinoembryonic antigen (CEA), the glycoprotein CA 125 antigen (CA 125), CA 19 9, and CA 72 4 may be elevated in patients with gastric cancer [70]. However, low rates of sensitivity and specificity prevent the use of any of these serologic markers as diagnostic tests for gastric cancer.

Recommendations for preoperative evaluation and staging of gastric cancer from the NCCN do not include assay of any tumor marker [68].

Staging laparoscopy — Laparoscopy, while more invasive than CT or EUS, has the advantage of directly visualizing the liver surface, the peritoneum, and

local lymph nodes. Between 20 and 30 percent of patients who have disease that is beyond T1 stage on EUS will be found to have peritoneal metastases despite having a negative CT scan [71]. As noted previously, the sensitivity of PET scans for the detection of peritoneal carcinomatosis is only about 50 percent.

Another advantage to laparoscopy is the opportunity to perform peritoneal cytology in patients who have no visible evidence of peritoneal spread. In most series this is a poor prognostic sign, even in the absence of overt peritoneal dissemination, and predicts for early peritoneal relapse. The majority of patients who are found to have peritoneal disease on laparoscopy will never require a laparotomy or resection.

Preoperative staging laparoscopy is advisable in any medically fit patient who appears to have more than a T1 lesion on EUS, no histologic confirmation of stage IV disease, and who would not otherwise require a palliative gastrectomy because of symptoms. Diagnostic laparoscopy should also be undertaken in patients who are being considered for neoadjuvant therapy trials.

MANAGEMENT OF GASTRIC CANCER

Surgical treatment for localized disease — Complete surgical eradication of a gastric tumor with resection of adjacent lymph nodes, represents the best chance for long-term survival. Since resection of the primary lesion also offers the most effective means of symptom palliation, abdominal exploration with curative intent should be undertaken unless there is unequivocal evidence of disseminated disease, a neoadjuvant approach is considered, or there are other contraindications to surgery.

The choice of operation for gastric cancer depends upon the location of the tumor within the stomach, the clinical stage, and the histologic type.

The major surgical considerations include the extent of luminal resection (total versus subtotal gastrectomy) and the extent of lymph node dissection.

Total versus subtotal gastrectomy — Gastrectomy is the most widely used approach for therapy of invasive gastric cancer. Total gastrectomy is usually performed for lesions in the proximal (upper third) of the stomach, while distal subtotal gastrectomy with resection of adjacent lymph nodes appears to be sufficient for lesions in the distal (lower two-thirds) of the stomach. However, patients with large midgastric lesions, or infiltrative disease (eg, linitis plastica) may require total gastrectomy.

Distal tumors — At least two trials show no added survival benefit for total compared to subtotal gastrectomy for patients with distal tumors [72,73]. The overall complication and perioperative mortality rates were 32 and 1.3 percent, respectively with total gastrectomy, and 34 and 3.2 percent with subtotal gastrectomy, respectively. Five-year survival was similar in both groups.

Proximal and GE junction tumors — Tumors of the proximal stomach that do not invade the GE junction can be approached by either a total gastrectomy. The Roux-en-Y reconstruction performed during total gastrectomy is associated with an extremely low incidence of reflux esophagitis compared to the roughly one-third of patients who develop reflux esophagitis after a proximal subtotal gastrectomy [74]. Proximal subtotal may fail to fully remove the lymph nodes along the lesser curvature. Thus, the most common site of nodal metastases may not be fully treated surgically.

Siewert classification of GE junction tumors [75]

- **Type I** — Carcinoma associated with Barrett's esophagus or true esophageal carcinoma growing down to the GE junction
- **Type II** — Tumors originating within 2 cm of the squamocolumnar junction
- **Type III** — Tumors of the subcardial region.

The origin of the tumor is sometimes unclear in patients who present with adenocarcinoma involving the GE junction. Patients with type I tumors are not appropriate candidates for a purely transabdominal approach. For type II or III tumors, a total gastrectomy may remove an adequate margin in the esophagus in patients with well- or moderately-differentiated histology.

The surgical options for type I include an abdominal transhiatal gastric pull-up to the neck or an Ivor-Lewis-type operation (combined transthoracic and transabdominal approach).

Linitis plastica — Linitis plastica has an extremely poor prognosis [76]. In one report, one-half of all patients had metastatic disease (mainly within the peritoneal cavity) at diagnosis [78]. Nodal involvement is frequent and extensive surgery may be required for complete excision [77,79]. One and seven year survival rates following gastrectomy were 50 and 8 percent, respectively [77]. Many surgeons consider the presence of linitis plastica to be a contraindication to potentially curative resection.

Extent of lymph node dissection — One of the most controversial areas in the surgical management of gastric cancer is the optimal extent of lymph node dissection. Japanese surgeons routinely perform extended lymphadenectomy, a practice that some suggest at least partially accounts for the better survival rates

in Asian as compared to Western series [80]. The term "extended lymphadenectomy" variably refers to either D2 or D3 lymph node dissection.

The draining lymph node basins for the stomach can be divided into 16 stations: stations 1 to 6 are perigastric, and the remaining 10 are located adjacent to major vessels, behind the pancreas, and along the aorta.

- **D1** lymphadenectomy refers to a limited dissection of only the perigastric lymph nodes.
- **D2** lymphadenectomy entails removal of nodes along the hepatic, left gastric, celiac and splenic arteries as well as those in the splenic hilum (stations 1-11).
- **D3** dissection includes nodes within the porta hepatis and periaortic regions (stations 1-16). Most Western surgeons classify disease in these regions as distant metastases, and do not routinely remove nodes in these areas during a potentially curative gastrectomy.

Nevertheless, these data as well as those from other groups [81,82] suggest that D2 dissection can be performed safely with a perioperative mortality rate that is under 2 percent.

The conclusion of the MRC trial and Dutch trial (and its accompanying editorial [83] was that D2 lymph node dissection did not confer a benefit compared to D1 dissection, and could not be routinely recommended.

Summary — Despite the results of randomized trials, major cancer centers frequently perform a D2 as compared to a D1 dissection, and treatment guidelines published by the National Comprehensive Cancer Network recommend that D2 lymph node dissection is preferred over a D1 dissection [65].

If there is a survival benefit to be gained by extended lymphadenectomy, it requires that there be no added operative mortality. A pancreas and spleen-preserving D2 lymphadenectomy provides superior staging information, and may provide a survival benefit while avoiding its excess morbidity. Splenectomy during gastric resection for tumors not adjacent to or invading the spleen increases morbidity and mortality without improving survival [84]. Thus it is not recommended unless there is direct tumor extension.

Adjuvant and neoadjuvant therapy — While complete resection provides the best chance for long-term survival, more than one-half of patients will have regional node involvement at the time of resection. Five-year survival rates are approximately 10 percent with N3 disease, 10 to 15 percent with N2 disease, and 50 percent with T3N0 disease[85]. These poor results with surgery alone,

especially in patients with nodal metastases, provide the rationale for adjuvant and neoadjuvant approaches using chemotherapy, radiation therapy (RT), or a combination of the two.

Adjuvant chemoradiotherapy — The benefit of postoperative adjuvant combined modality therapy using contemporary RT techniques and leucovorin-modulated 5-fluorouracil was shown in a United States Intergroup study (INT-0116). The trial included patients with GE junction adenocarcinomas that extended at least 2 cm into the stomach.

Three-year overall survival and disease-free survival were significantly better for patients receiving chemoradiotherapy (52 versus 41 percent, and 49 versus 32 percent, respectively). These results have been considered by many to have changed the standard of care in the United States.

Neoadjuvant chemotherapy and chemoradiotherapy — The goals of preoperative therapy are to increase the resectability rate, reduce the rate of local and distant recurrences, and ultimately improve survival. Data from several uncontrolled series suggest that some patients with initially locally advanced disease may respond to chemotherapy or chemoradiotherapy sufficiently that they are able to undergo potentially curative surgery. Clinicians who treat gastric cancer have a bias towards neoadjuvant therapy for two major reasons.

First, the ability to deliver adequate postoperative therapy may be compromised by complications of the surgery. Between one-third and one-half of patients do not recover quickly enough to tolerate adjuvant treatment within four to six weeks of surgery.

Second, some patients have aggressive disease and develop metastases within a short period of time, despite having an adequate operation. These patients do not benefit from surgery, and the delay in operative intervention with neoadjuvant therapy may have permitted their identification prior to exploratory laparotomy, thus sparing them unnecessary surgery.

If the posttherapy restaging evaluation demonstrates no evidence of metastatic disease, these patients are considered eligible for potentially curative surgery. When feasible, patients should be enrolled on therapeutic trials evaluating the benefit of neoadjuvant or adjuvant therapies.

Prognosis — The following five-year survival rates were reported in a series of 750 patients from MSKCC, in whom more than 15 lymph nodes were examined [17]: IA — 95 percent IB — 85 percent II — 54 percent IIIA — 37 percent IIIB — 11 percent IV — 7 percent

Somewhat lower five-year survival rates were reported in the National Cancer Data Base [66]: IA — 78 percent IB — 58 percent II — 34 percent IIIA — 20 percent IIIB — 8 percent IV — 7 percent

Palliative gastrectomy — In patients with locally advanced or metastatic disease, surgical intervention may provide effective palliation of symptoms such as pain, nausea, bleeding, or obstruction. Palliative gastrectomy can provide symptomatic relief, and a possible improvement in survival, although this is controversial. The criteria for selection of patients who may benefit from palliative gastrectomy are not firmly established.

AIM OF THE STUDY

1. To study the epidemiology of cancer stomach .

2. To assess the influence of risk factors in the causation of cancer stomach.

3. To correlate the clinical features of cancer stomach with the site of lesion.

4. To assess the commonest anatomical site of lesion in cancer stomach.

5. To assess the incidence of operability of tumour at the time of presentation.

6. To assess the role of CECT in staging the cancer stomach.

MATERIALS AND METHODS

Patients included in the study were recruited from the Department of Digestive Health and Diseases, Government Peripheral hospital, Anna Nagar, Chennai. The study period was from January 2008 to December 2009.

Consecutive patients diagnosed to have cancer stomach were included in the study group. Only biopsy proven adenocarcinomas, were included in the study. A detailed proforma was compiled for the patients with cancer stomach. A detailed history about dietary habits, social habits such as smoking, alcohol ,previous gastric surgery , previous history of gastric ulcer and family history of gastric cancer were recorded. Clinical history about dyspepsia, pain abdomen, anorexia and weight loss, vomiting, dysphagia and upper GI bleed were obtained and thorough clinical examination was done. Body mass index was calculated for all.

Appropriate investigations such as haemoglobin, erythrocyte sedimentation rate, X ray chest, U G I Scopy & biopsy and CECT scan of the upper abdomen were done. Histopathological grading was done by the pathologist. Treatment was individualized according to the stage of the disease during presentation.

The statistical analysis of the data was done using SSPS 11. ‘ p’ value of < 0.05 was considered to be statistically significant. The p value was calculated using chi square test. Percentage calculation and cross analysis were done to identify significant data.

RESULTS

During the study period of two years, a total number 12800 patients attended OPD. Endoscopy was performed for 5842 cases (31%). Among those, biopsy proven adenocarcinomas was 172. This accounts to 3% of total endoscopy and 1% of total OPD cases. Of these 172 cases, males were 72% and females 28% constituting a ratio of 3: 1.

The incidence of cancer stomach in patients below the age of 40 years was 13.95%. It slowly increased and reached a maximum in the 5^{th} decade and then slowly declined. The incidence was 13.3% in patients above the age of 70. The mean age of the patient was 55.76. The minimum age was 28yrs and the maximum age was 77years.

Most of them were from places in & around Chennai (88%). Many of them were farmers and labourers. About 80% of the female patients were housewives. Majority of the patients were Hindus (89.5%), followed by Muslims (6%) and Christians (4.5%).

RISK FACTORS

One patient had family history of cancer stomach. He had growth even before the age of 40years. No patient had previous documented history of gastric ulcer. Around 7% of them had previous history of gastric surgery. Alcoholics and smokers formed 46.5% & 61.6% respectively. 71.5%

had consumed high salt diet and 71.5% had not consumed fresh vegetables. Also these patients did not use refrigerators.

Abdominal pain was present in 61.% of patients but anorexia was only second to pain abdomen with 39-46%. Other common presentations were vomiting in 38.5%, indigestion in 40%, and early satiety in 18.6%. Even though ball rolling movement is a symptom of complicated peptic ulcer disease, 18% of cancer stomach patients had significance in curative lesions and early presentation to hospital. Less common symptoms like awareness of lump (12.8%), abdomen distension (8.7%), and GI bleed (6%) were also recorded.

Clinical findings - Body Mass Index was < 19 in 88.34% of patients . Only one patient had palpable supra clavicular lymph node. None had cutaneous markers of intra abdominal malignancy. 19 patients had epigastric lump, 14 had ascites & 11 had hepatomegaly. CVS & RS were normal in all cases.

Investigations

Haemoglobin was low in 65.7% of cases & 77.9% had raised ESR. 55.8% of cases had blood group "A".

EPIDEMIOLOGY		
	NO OF PATIENTS	**PERCENTAGE**
TOTAL	172	100
Age Group		
BELOW 40	24	13.95
41-50YRS	39	22.67
51-50urs	44	25.58
above 60yrs.	65	37.79
SEX		
MALE	123	71.5
FEMALE	49	28.5
RESIDENCE		
CHENNAI	152	88
OUTSIDE CHENNAI	20	12
RELIGION		
HINDU	154	89.5
CHRISTIAN	8	4.5
MUSLIM	10	6
SYMPTOM ANALYSIS		
	NO OF PATIENTS	**PERCENTAGE**
PAIN ABDOMEN	105	6100%
INDIGESTION	67	39
EARLY SATIETY	32	18.6
LOSS OF APPETITE	67	39
LOSS OF WEIGHT	79	46
NAUSEA/VOMITING	66	38.5
AWARENESS OF LUMP	19	11
BRM	31	18
ABDOMEN DISTENSION	15	8.7
DYSPHAGIA	22	12.8
JAUNDICE	1	0.6
UGI BLEED	13	7.6
SIGNS		
PALLOR	123	71.5
JAUNDICE	1	0.6
SUPRACLAVICULAR NODE	1	0.6
ABDOMINAL SCAR	12	7
EPIGASTRIC MASS	19	11
HEPATOMEGALY	11	6.4
ASCITES	14	8.1

RISK FACTOR ANALYSIS		
	NO OF PATIENTS	PERCENTAGE
PREVIOUS GASTRIC SURGERY	12	7
H/O GASTRIC ULCER	0	0
SMOKING	106	61.6
ALCOHOL	80	46.5
FAMILY H/O GASTRIC CANCER	1	0.6
HIGH SALT DIET	123	71.5
NO REFRIGERATOR/FRESH FRUITS	123	71.5
INVESTIGATIONS		
	NO. OF PATIENTS	**PERCENTAGE**
HEMOGLOBIN <9 (ANAEMIA)	113	65.7
BLOOD GROUP - 'A'	96	55.8
ESR >30	134	77.9
VOGD		
SITE OF LESION	**NO.OF PATIENTS**	**PERCENTAGE**
ANTRAL GROWTH	94	54.65
MID BODY GROWTH	38	22
OG JUNCTION GROWTH	26	15
DIFFUSE GASTRIC CANCER	14	8.35
GOO		
YES	56	
NO	40	
CT SCAN	**NO.OF PATIENTS**	**PERCENTAGE**
NORMAL	62	36.05
ASCITES	8	3.6
LYMPH NODES (N1+)	42	24.42
LIVER SECONDARIES	7	5.8
L.NODES +SEC/ASCIRES	7	4
T4 LESIONS	43	26.13
TREATMENT		
CURATIVE	62	36.05
PALLIATIVE	110	63.95

SITE OF LESION IN ENDOSCOPY

UGI Scopy was done for all cases and it was found that **antral growth** was present in 60%**(p<0.001**),** midbody growth in 20%, OGJunction growth in 12% and diffuse gastric cancer in 8%. 60% of antral growth presented with GOO. A point to be noted is that 50% of antral growth was found to be operable at the time of diagnosis **(p<0.001**)** due to its early presentation. Whereas most of the OGJunction growth(80.8%) and diffuse gastric cancer(92.9%) were inoperable at the time of presentation**(p<0.001**)**

Sit of Lesion	No. of patients	%	Curative (%)	Palliative	p value	Significance
ANTRAL	94	54.65	49.9	51.1		
MID	38	22	26.3	73.7		
OG	26	15	19.2	80.8		
DIFFUSE	14	8.35	7.1	92.9		
					<0.001**	Very significant

CT scan in staging

CT scan abdomen is the investigation to stage the cancer stomach used world wide until now. In this study CTscan staging showed 36% of cases in an operable stage and 67% in an inoperable stage **(p<0.001**)**, thus avoiding unnecessary laparotomy.

CROSS ANALYSIS OF PRESENTING SYMPTOM Vs TYPE OF GROWTH (SUB GROUP ANALYSIS)

	ANTRAL GROWTH	MIDBODY GROWTH	OGJ	DIFFUSE	P VALUE	SIGNIFICANCE
SYMPTOMS VS SITE OF LESION						
PAIN ABDOMEN(%)	69	50	50	57	0.113	NOT SIGNIFICANT
INDIGESTION	45.7	15.8	15.4	100	<0.001**	VERY SIGNIFICANT
EARLY SATIETY	24.5	13.2	7.7	14.3	0.164	NS
LOSS OF APPETITE	39.4	39.5	46.2	21.4	0.496	NS
LOSS OF WEIGHT	47.9	47.4	42.3	35.7	0.825	NS
NAUSEA/VOMITING	44.7	21.1	34.6	50	0.061	NS
AWARENESS OF LUMP	11.7	13.2	7.7	7.1	0.866	NS
BRM	24.5	7.9	7.7	21.4	0.063	NS
ABDOMEN DISTENSION	9.6	2.6	3.8	28.6	0.022*	SIGNIFICANT
DYSPHAGIA	2.1	5.3	61.5	14.3	<0.001**	VERY SIGNIFICANT
JAUNDICE	1.1	-	-	-	0.841	NS
UGI BLEED	9.6	5.3	3.8	7.1	0.717	NS
RISK FACTOR VS SITE OF LESION						
PREVIOUS GASTRIC SURGERY	9.6	2.6	3.8	7.1	0.479	NS
H/O GASTRIC ULCER	0	0	0	0	-	NS
SMOKING	63.8	60.5	61.5	50	0.798	NS
ALCOHOL	48.9	42.1	46.2	42.9	0.897	NS
HIGH SALT DIET	70.2	73.7	73.1	71.4	0.978	NS
NO REFRIGERATOR/FRESH FRUITS	70.2	73.7	73.1	71.4	0.978	NS
CL.FEATURES VS SITE OF LESION						
PALLOR	70.2	73.7	73.1	71.4	0.978	NS
JAUNDICE	1.1	-	-	-	0.841	NS
ABDOMINAL SCAR	9.6	2.6	3.8	7.1	0.479	NS
EPIGASTRIC MASS	11.7	13.2	7.7	7.1	0.866	NS
HEPATOMEGALY	6.4	-	19.2	-	0.013*	SIGNIFICANT
ASCITES	8.5	2.6	3.8	28.6	0.018*	SIGNIFICANT
INV VS SITE OF LESION						
BLOOD GP 'A'	38.3	55.3	96.2	100	<0.001**	VERY SIGNIFICANT
ESR >30	77.7	76.3	80.8	78.6	0.979	NS
HB <9	64.9	68.4	69.2	57.1	0.861	NS
OPERABILITY VS VOGD						
CURATIVE (%)	48.9	26.3	19.2	7.1		
PALLIATIVE	51.1	73.7	80.8	92.9	<0.001**	VERY SIGNIFICANT

- In this study dysphagia was predominantly present in OGJunction growth($p<0.001$) which is very significant.
- In the cross analysis indigestion was present in 45% of antral growth and in all cases of diffuse gastric cancer which is also statistically very significant ($p<0.001$).
- Ascites was a presenting feature in 30% of diffuse gastric cancer in cross analysis, which was also statistically significant($p=0.022$*).

In this study **Blood group A** was present in all cases of diffuse gastric cancer and 96% of OGJunction cancers in cross analysis, which was also statistically very significant**(p=0.001*).**

In this study two third of all types of gastric cancers were inoperable in the pre-operative CT evaluation, which was statistically significant($p<0.001$).

In the sub group 93%, 81% and 70% of diffuse gastric cancer, OGJ growth and mid body growth respectively, in descending order, were inoperable whereas antral growth was operable in 50%. This is statistically very significant($p<0.001$**).

DISCUSSION

Cancer stomach is one of the commonest digestive tract cancers in and around Chennai. **Malhotra et al** reported that The incidence rate of gastric cancer is four times higher in Southern India compared with Northern India[90]. Among the six registries, the highest incidence in both sexes is reported from Chennai and the lowest from Barshi.

The mean age of carcinoma stomach in this study was 55.8 years. **Jayanthi V et al** from Chennai reported similar age group in her study in 2007[88]. Ferlay[1],Parkin[3] and Henderson[86] et al in their report stated that the age at initial presentation was 70 years which was similar to this present study. Kurihara et al reported that in countries with high incidence of gastric cancer, the age at diagnosis tends to be a decade earlier.**[87]**

The male female ratio in this study was 3:1similar to that quoted by Jayanthi V[88] et al & Ferlay[1] et al. **Jayanthi.V[88]et al** reported that gastric cancer predominantly affected male with an overall ratio of 3(p=0.001) which was very significant as in this study. **Ferlay[1],et al** reported that Worldwide gastric cancer rates are about twice as high in men as in women.

In the present study, gastric cancer was more common among the Hindus, followed by Muslims and then Christians. The Mumbai cancer registry had reported a preponderance among Hindus & Muslims with a low incidence

among Christians. **Siddiqi et al** in his study showed significant differences in Hindus, Muslims and Sikh population. Muslims had highest incidence in his study. [91]

Risk factors analysis

In western countries, a causal relationship has been established with the consumption of alcohol and smoking. In this study 61.6% of the cases had smoking habit which was a significant risk factor in males. Similar reports were showen by **Gajalakshmi C.K.et al** in the year 2001.In a case–control study from Chennai,smokers had a twofold increased risk of gastric cancer compared to nonsmokers, and the risk seen among current smokers was significantly higher than that among ex-smokers. The risk among those who smoke "bidi" (a type of local cigarette made from suncured tobacco that is rolled in a rectangular piece of dried leaf of Diospyros melanoxylon) was thrice as that among cigarette smokers. [92].

Sumathi B.et al showed pickled food consumption as an independent risk factor for the development of gastric cancer, while consumption of pulses was protective[93]. **Tatematsu et al** showed that high salt intake damages stomach mucosa and increases the susceptibility to carcinogenesis in rodents [26]. Shikata, K et al showed that the risk of high salt intake was strongest in patients who had both H. pylori infection and atrophic gastritis [27]. In this study around 71.5% had

consumed high salt diet and 71.5% had not consumed fresh vegetables. Also these patients had no refrigerators which was also an important risk factor both in males and females.

Zhao et al showed a genetic predisposition to gastric cancer has been repeatedly confirmed[41]. Bonney A genetic predisposition for chronic atrophic gastritis, a precursor of gastric carcinoma, has been described and may account for at least some cases of familial gastric cancer [42]. But in this study only one patient had family history of gastric cancer which was diffuse gastric cancer. He had growth even before the age of 40years. Role of genetics in cancer stomach needs further evaluation in our country.

Hansson, et al, concluded that despite the known associations between H. pylori infection and peptic ulcer disease and H. pylori infection and gastric cancer, the association between benign gastric ulcer disease and gastric cancer remains controversial [46].In this study none of our patients had previous history of gastric ulcer. **Nomura et al** assessed there is an increased risk of gastric cancer after gastric surgery, with the risk being greatest 15 to 20 years after surgery and then increasing with time [34]. In this study around 7% of them had previous history of gastric surgery. It shows that previous gastric surgery is still a continuing risk factor.

Even though there were many risk factors in a large number of cases, nothing is statistically significant in the cross analysis .

Symptom analysis

Wanebo et al and **Sleisenger Text Book** of Gastrointestinal and Liver Disease clearly states that **Weight loss (62%) and persistent abdominal pain (52%)** are the most common symptoms during initial diagnosis [53]. Contradictory to that in this study **Abdomen pain was present in 61.%** of patients but anorexia and **weight loss** was only second to pain abdomen with **39-46%.** Other common presentations were vomiting in 38.5%, indigestion in 40%, and early satiety in 18.6%. Contradictory to the belief that ball rolling movement is a symptom of complicated peptic ulcer disease, this was seen in 18% of growth stomach in the current study. But it had a significance in curative lesions and early presentation to hospital. Rare and late presentations like awareness of lump in 12.8%, abdomen distension in 8.7%, and GI bleed in 6% were also recorded.

According to **Tucker et al** tumors affecting the cardia (OGJunction) can cause dysphagia.[98] In our study **dysphagia** predominantly was found in OGJunction growth **with p<0.001** which is **very significant**.

In cross analysis **indigestion** was present in45% of antral growth and in all cases of diffuse gastric cancer acquiring a **very significant p value of <0.001.**

Clinical findings

Pallor presented in two third of cases, yet not significant. This may be because of nutritional deficiency due to decreased intake and the bleeding from tumor.

Supraclavicular node and jaundice were present only in one case each. Also cutaneous markers of intra abdominal malignancy were not noted. There are only a few case reports regarding this in literature. Larger study is needed.

Secondaries liver presenting as **hepatomegaly** was more common with OGJunction cancers with a value of around 20% which was statistically significant**(p=0.018*).**

Ascites a presenting feature in 30% of diffuse gastric cancer was also found to be statistically significant**(p=0.022*)** in cross analysis,

Arid **et al** showed individuals of blood group A have been known for decades to show an approximately 20 percent higher risk of gastric cancer than those of groups O, B, or AB [40]. Langman **et al** suggest that group A may be particularly associated with the diffuse type of gastric cancer [39]. In this study **Blood group A** was present in almost all cases of diffuse gastric cancer and 96% of OG Junction cancers with a statistically very significant value **p=0.001*.**

Kampschoer et al and Powell et al demonstrated in their analysis that despite the decline in gastric cancer, overall there has been an explosive increase

in incidence of cancer of the gastric cardia[8-10]. **Correa P et al** showed that in the United States, the distribution of gastric cancer within the stomach is **39% in the proximal third**, 17% in the middle third, **32% in the distal third**, and 12% involving the entire stomach [95]. The decline in gastric cancer rates reflects a drop in the rate of distal gastric cancers.

In India also a trend towards an increase in the incidence of cardia tumors is seen. This is evident in the data from Mumbai, where the percentage of cardia and fundus tumors increased from 13% in 1941–1968 to 23% in 1987–1993 shown by **Mohandas KM et al** [95].

In this study **antral growth was present in 60%** ($p<0.001$)**,** midbody growth in 20%, **OGJunction growth in 12%** and diffuse gastric cancer in 8%. On endoscopy around 60% of patients had antral growth which is contradictory to western data where OGJunction growth is more common i.e., around 39%[95]. This study is supported by **Jayanthi V.et al** who showed similar incidence of **67% antral growth**, mid body 23%, **cardia&OG Junction growth 10%**[88].

This may be because of a large low socioeconomic group in India, a developing country. Moreover food habits here differ from western people. Lastly the most important factor, H.Pylori is 100% prevalent in developing countries among the age group of 20yrs.

Around 60% of antral growth presented with GOO. Another point to be noted is that around 50% of antral growth was found to be operable at the time of admission **(p<0.001**)** due to its early presentation. On the contrary most of the OG Junction growth (80.8%) and diffuse gastric cancer (92.9%) were inoperable during that time.

Haemoglobin was low in 65.5% and ESR raised in 77% of cases which were not statistically significant.

CT scan in staging

Grote R et al showed that CT abdomen has a sensitivity of 65% to 90% for advanced gastric cancer[97]. The accuracy rate was approximately 60% to 70% for T staging and between 40% and 70% for N staging. CT scan abdomen is the investigation to stage the cancer stomach used world wide until now. In this study CTscan staging showed 36% of cases in operable and 67% in inoperable stages. Thus unnecessary laprotomy can be avoided.

TREATMENT

Mohandas et al showed that in India more than 90% of all gastric cancers are being diagnosed in an advanced stage, and in those subjected to surgery more than 70% have serosal infiltration[96]. In this study two third of all types of gastric cancers were inoperable in the pre-operative CT evaluation and this has a very statistical significance. **(p<0.001**).**

CONCLUSION

- Mean age of the patients with cancer stomach is 56 years.
- Males outnumbered female cases probably due to increased smoking and alcohol consumption.
- People with Low intake of vegetables and fruits and those who had no refrigerators lead to a higher risk of developing cancer stomach in both sexes.
- Past history of gastric surgery still continues to be a risk factor. And gastric ulcer is not a significant risk factor.
- **Pain abdomen is the commonest presenting symptom.**
- **Significant number of patients with Ball Rolling Movement were found to be operable in the pre-operative evaluation.**
- Dysphagia and vomiting immediately after taking food along with loss of appetite commonly present as OG Junction growth.
- **Cutaneous markers of intra abdominal malignancy are extremely rare.**
- **Antral growth is more common than OG Junction growth.**
- Antral growth presents earlier and more amenable to surgery compared to OGJunction and diffuse gastric cancer.

- **Blood group "A" is a significant risk factor for cancer stomach particularly for diffuse gastric cancer and OGJunction growth.**

- **CT Scan is a valuable modality of investigation preoperative evaluation.**

- Two thirds of patients were found to be inoperable during the time of diagnosis and hence underwent only palliative treatment.

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MASTER CHART

SNO	DDHO	NAME	AGE	SEX	Abd.pain	indig	E..Sati	lOA	LOW	vomit	lump	BRM	Abd.dis	dysph	Jaun	bleed	Pallor	scar	mass	live	ascites	HB	BL.GP	ESR	VOGD	CT_SCAN	TREAT
1	47/08	KPK MENON	75	1	2	2	2	1	1	1	2	2	2	1	2	2	2	2	2	1	2	2	1	2	3	A	1
2	73/08	EKAMBARAM	63	1	1	1	1	1	1	1	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	E	2
3	188/08	VEDIAPPAN	65	1	1	1	1	1	1	1	2	2	1	2	2	2	1	2	2	2	1	1	1	1	1	E	2
4	202/08	EZHUMALAI	63	1	1	2	2	1	1	2	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
5	250/08	RENGANAYAKI	53	2	1	2	2	1	1	2	1	2	2	2	2	2	1	2	1	2	2	1	2	1	1	A	2
6	2012/07	MOORTHI	42	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2	2	1	1	1	2	F	2
7	348/08	PERUMAL	64	1	2	1	2	2	2	2	2	2	1	2	2	2	1	1	2	2	1	1	1	1	4	B	2
8	426/08	MANICKAM	65	1	1	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
9	433/08	SUNDARAMBAL	75	2	2	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	1	1	1	C	2
10	4879/08	BALAKRISHNAN	62	1	1	1	1	1	1	2	2	2	2	2	1	2	1	1	2	1	2	1	2	1	1	A	1
11	535/08	YUVARAJ	53	1	2	2	2	1	1	1	1	2	2	2	2	2	1	2	1	2	2	1	1	1	3	A	1
12	644/08	HYAD BASHA	42	1	1	1	2	2	2	1	2	2	2	2	2	1	1	2	2	1	2	1	1	1	1	C	2
13	775/08	RAJENDRAN	52	1	2	2	2	1	1	2	2	2	2	1	2	2	1	2	2	1	2	1	1	1	3	D	2
14	727/08	MANICKAM	65	1	2	1	2	1	1	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	1	A	1
15	857/08	CHINNAPAN	46	1	2	2	2	1	2	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
16	858/08	PALANIVEL	48	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	A	1
17	419/08	NANDAGOPAL	64	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	4	C	2
18	1162/08	DEIVANAI	55	2	1	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
19	1261/08	GANESAN	50	1	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	C	1
20	1524/08	KANNIAMMAL	60	2	1	2	2	2	1	2	2	2	2	1	2	2	2	2	2	2	2	2	1	2	3	C	2
21	1695/08	ABDHUL KADHEER	65	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	3	F	2
22	1670/08	PADMANABAN	61	1	1	2	2	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	1	F	2
23	1791/08	MURUGESAN	35	1	1	1	2	2	1	1	2	2	1	2	2	2	1	2	2	2	1	2	1	1	4	B	2
24	1814/08	MAHALINGAM	50	1	1	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	3	A	1
25	1857/08	RADHAKRISHNAN	47	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1
26	1873/08	SUNDARRAJAN	37	1	2	2	2	1	1	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	C	2
27	1722/08	DASS	62	1	1	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	1	1	A	1

28	1899/08	KUPPAMMAL	56	2	1	2	2	1	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	C	2
29	1875/08	JAYARAMAN	48	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
30	1749/08	VEERAMMAL	45	2	1	1	1	1	2	2	2	1	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
31	2172/08	SIVALINGAM	40	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	1	F	2
32	2206/08	PATCHIAPPAN	45	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	1	2	3	F	2
33	2304/08	VENKATESH	68	1	1	1	2	1	2	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	C	2
34	2447/08	MUNIAMMAL	50	2	1	1	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	A	1
35	2510/08	DEIVASIGAMANI	45	1	2	1	2	1	1	1	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	C	2
36	2449/08	KUMARI	37	2	1	1	2	2	2	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	1	A	1
37	2539/08	MURUGAN	40	1	2	1	1	2	1	1	2	1	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1
38	2549/08	ELUMALAI	57	1	1	2	2	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	1	A	1
39	2739/08	RAMANATHAN	61	1	1	2	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	C	2
40	2799/08	PATHARUNISHA	40	2	1	1	2	2	2	2	2	1	2	2	2	2	1	2	2	2	2	1	1	1	4	F	2
41	3013/08	KOLLAPURI	74	1	1	1	1	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	A	1
42	2950/08	VELU	68	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
43	3165/08	PALANI	50	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	1	A	1
44	3177/08	NARAYANAN	61	1	2	1	1	2	2	2	2	2	1	2	2	2	1	2	2	1	1	1	2	1	1	E	2
45	3250/08	MARIAMMAL	46	2	2	2	2	1	2	2	2	2	1	1	2	2	2	2	2	2	1	2	2	2	2	B	2
46	3326/08	GEETA	40	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	3	F	2
47	3345/08	VARADHAN	70	1	1	2	2	2	1	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
48	3621/08	SELVARAJ	38	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2	1	C	2
49	3379/08	AUBRAJ	48	1	1	2	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2	2	1	1	1	1	C	2
50	3560/08	SEENIVASAN	48	1	1	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
51	3729/08	VAITHIYANATHAN	61	1	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	2	2	1	2	A	1
52	4113/08	GANDHIMATHI	59	1	1	2	2	2	1	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	F	2
53	4162/08	DEVENDRAN	59	1	1	1	1	1	1	2	2	2	1	1	2	2	1	2	2	2	1	1	2	1	1	C	2
54	4182/08	MANICKAM	50	1	1	2	2	2	1	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
55	4312/08	SARAVANAN	71	1	1	1	2	1	1	1	1	1	2	2	2	2	1	2	1	2	2	1	1	1	1	C	2
56	4303/08	MOHANDOSS	40	1	1	2	2	2	1	2	1	2	2	2	2	2	1	2	1	2	2	1	1	1	2	F	2

57	4383/08	SAROJA	65	2	1	1	1	2	2	2	2	1	1	2	2	2	1	2	2	2	1	2	2	1	1	A	1
58	6345/08	ARUMUGAM	73	1	2	2	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	A	1
59	4400/08	SUBBRAMANI	77	1	1	1	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2	1	C	2
60	4427/08	AJMUNISHA	70	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	F	2
61	4305/08	SELVAMANI	36	1	1	1	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	F	2
62	4439/08	ALLIMUTHU	30	1	2	1	2	2	1	2	2	2	1	2	2	1	2	2	2	2	1	2	1	2	4	F	2
63	4618/08	MARY	47	2	1	2	2	1	1	1	2	2	2	1	2	2	2	2	2	1	2	2	1	2	3	F	2
64	4690/08	PARVATHI	45	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	D	2
65	4860/08	RATHINAMALA	65	2	2	1	2	1	2	1	2	2	1	1	2	2	1	2	2	2	1	1	1	1	4	B	2
66	4636/08	GOVINDAN	53	1	1	1	1	2	2	1	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
67	4728/08	MARIAPPAN	40	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	F	2
68	4853/08	SANKARI	50	2	1	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	4	F	2
69	4790/08	MARUDHARAJ	60	1	1	1	1	1	1	2	1	1	2	2	2	1	2	2	1	2	2	2	2	2	1	A	1
70	4852/08	BALAKRISHNAN	61	1	1	1	1	1	1	1	2	1	2	2	2	2	2	2	2	2	2	2	1	1	1	A	1
71	4728/08	MARIAPPAN	40	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	A	1
72	4940/08	SUBBRAMANI	57	1	1	1	1	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
73	5034/08	KAMALA	70	2	1	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	F	2
74	2569/08	PATCHIAMMAL	34	2	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
75	5130/08	RAVANAMMA	35	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
76	5214/08	AIYASAMY	60	1	1	1	1	1	1	1	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	A	1
77	5189/08	RADHAKRISHNAN	40	1	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2	1	2	1	1	A	1
78	5749/08	RAZIYA BEGAM	68	2	2	2	2	1	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2	1	C	2
79	5113/08	RUCKMANI	65	2	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	C	2
80	5324/09	VARADHARAJAN	41	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	F	2
81	4690/08	PARVATHI	40	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
82	5852/08	THATHIYAN	74	1	2	1	1	2	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	4	A	1
83	5394/08	CHINNAPAIYAN	66	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
84	5561/08	DEVAKIRUBAI	45	2	1	1	2	2	2	1	1	1	2	2	2	2	1	2	1	2	2	1	1	1	4	D	2
85	5738/08	SANKARLAL	60	1	2	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1

86	5749/08	KANNIAMMAL	60	2	1	2	2	1	1	1	1	2	2	2	2	2	1	2	1	2	2	1	2	1	2	A	1
87	6047/08	DHANALAKSHMI	47	2	2	1	1	2	2	1	2	1	2	2	2	2	1	2	2	2	2	2	1	1	4	F	2
88	6143/08	RAMAREDDY	65	1	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1
89	6215/08	GANESAN	64	1	1	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
90	6370/08	LEENA	50	2	1	1	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
91	6412/08	ELUMALAI	58	1	1	1	1	2	1	2	2	1	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1
92	6547/08	JAYARAMAN	70	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
93	6551/08	KRISHNAN	58	1	1	1	2	2	2	2	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
94	6253/08	ARUMUGAM	46	1	1	1	1	2	1	2	1	1	2	2	2	2	1	2	1	2	2	2	1	1	3	C	2
95	6294/08	PANNER SELVAM	40	1	2	1	1	2	2	2	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
96	4117/08	RAMADOSS	53	1	1	1	2	2	1	2	2	2	2	2	2	2	1	1	2	2	2	1	1	1	1	C	2
97	2232/08	RADHA	47	2	2	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
98	6388/08	GANESAN	70	1	1	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	F	2
99	108/09	CHELLAMUTHU	49	1	1	2	1	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
100	312/09	MOHAN	56	1	1	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	A	1
101	339/09	MURUGAN	65	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
102	428/09	VEERAMMAL	65	2	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
103	485/09	AMIRTHAMMAL	70	2	1	1	2	1	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	F	2
104	63/09	KESAVAN	75	1	1	2	2	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	2	1	1	C	2
105	61/09	SEETHALAKSHMI	28	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
106	86/09	SENTHILVELAN	38	1	2	1	1	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	A	1
107	242/09	BAASHA	48	1	1	2	2	2	2	2	2	2	2	1	2	2	1	2	2	2	2	1	2	1	3	F	2
108	179/09	MUNISAMY	54	1	1	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
109	358/09	GANGAN	55	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	A	1
110	656/09	MUNUSAMY	65	1	1	2	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	3	C	2
111	435/09	VARADHAN	70	1	2	2	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	C	2
112	711/09	SUMATHI	34	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	F	2
113	861/09	PRABAKARAN	53	1	1	1	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
114	990/09	KUPPAMMAL	66	2	1	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	C	2

115	1134/09	SUDALAIMANI	75	1	2	2	2	1	1	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	2	A	1
116	1133/09	GOVINDARAJ	65	1	1	2	2	1	1	1	2	1	2	2	2	1	1	2	2	2	2	2	2	1	1	A	1
117	1156/09	MANI	55	1	1	1	1	1	1	2	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
118	747/09	RAJENDRAN	47	1	1	2	2	1	1	2	1	2	2	2	2	2	1	2	1	2	2	1	1	1	2	F	2
119	912/09	SARADHA	48	2	1	2	1	2	2	2	1	1	2	2	2	2	1	2	1	2	2	1	2	1	1	A	1
120	1154/09	DURAI	57	1	1	2	1	2	1	1	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
121	1433/09	EASAKIYAL	60	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
122	1537/09	CHOKKAMMAL	49	2	1	2	2	1	1	2	2	1	2	2	2	2	1	1	2	2	2	1	2	1	1	A	1
123	1541/09	ANWAR BASHA	63	1	1	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
124	1693/09	RAJI	47	1	2	2	2	2	1	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	F	2
125	1749/09	PEETHAMBARAM	58	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	3	D	2
126	1428/09	POONKODI	36	2	1	2	2	1	1	2	1	2	2	2	2	2	1	2	1	2	2	1	2	1	1	C	2
127	1677/09	GOVINDAMMAL	60	2	2	1	2	1	1	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	4	E	2
128	1924/09	PONNUSAMY	55	1	1	1	2	1	1	1	1	2	2	2	2	2	1	2	1	2	2	1	2	1	1	C	2
129	2242/09	MADHAN	49	1	1	1	2	2	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	4	F	2
130	2236/09	MUTHUKUMAR	60	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	F	2
131	2225/09	SAVITHRI	45	2	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	A	1
132	561/09	PRABHAKARAN	53	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	A	1
133	2213/09	MUTHU	70	1	1	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	C	2
134	2209/09	PATCHIAPPAN	60	1	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2	1	B	2
135	2257/09	MAGIMAIRAJ	74	1	2	2	2	1	1	2	2	2	2	1	2	1	1	2	2	1	2	1	1	1	3	E	2
136	2525/09	PAULRAJ	72	1	1	1	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	A	1
137	2532/09	BALARAMAN	65	1	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	1	2	C	2
138	2699/09	KANDAMMAL	55	2	1	2	2	2	2	2	2	2	2	1	2	2	1	2	2	1	2	1	1	1	3	D	2
139	4613/09	BABY	57	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	2	1	1	1	1	3	B	2
140	4660/09	GEETHA	39	2	1	1	1	2	2	1	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
141	4976/09	VARADHARAJAN	63	1	1	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	A	1
142	4934/09	CHENNAMMAL	55	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	C	2
143	5095/09	KRISHNAN	76	1	2	2	2	1	1	1	2	2	1	2	2	2	1	2	2	1	1	1	2	1	1	E	2

144	5079/09	NARAYANAN	64	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	2	F	2
145	5168/09	RAJAMANI	68	1	2	2	2	2	2	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	D	2
146	5258/09	PANCHATCHARAM	70	1	2	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	C	2
147	5168/09	RAJAMANI	68	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	3	D	2
148	5509/09	SUBRAMANI	43	1	2	2	2	2	2	1	2	2	2	2	2	1	1	2	2	2	2	1	2	1	1	A	1
149	5598/09	VELU	66	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	2	1	A	1
150	5666/09	BASKAR	46	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	4	D	2
151	5798/09	ELUMALAI	57	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
152	5904/09	GANESAN	65	1	2	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	C	2
153	5894/09	VARADHAN	58	1	1	1	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	A	1
154	6151/09	NAGAMMAL	55	2	1	1	2	1	1	1	2	1	2	2	2	1	1	2	2	1	2	1	1	1	1	D	2
155	6212/09	RAMALINGAM	61	1	1	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	1	1	1	F	2
156	6268/09	KUMUDHAVALLI	67	2	1	1	1	1	1	1	2	1	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
157	6994/09	SOORAN	55	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	F	2
158	861/09	PRABAKARAN	53	1	1	1	1	2	2	2	2	2	2	2	2	2	1	1	2	2	2	1	1	1	1	C	2
159	5491/09	ARUMUGAM	72	1	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	4	D	2
160	5653/09	GANESAN	65	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	A	1
161	5737/09	RANI	50	2	2	2	2	1	1	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	F	2
162	5868/09	SABIYABEE	65	2	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	C	2
163	6036/09	AMBIKA	55	2	2	2	2	1	1	2	2	2	2	2	2	2	1	2	2	2	2	1	1	1	2	A	1
164	5989/09	KANNIAMMAL	76	2	1	1	2	2	2	1	2	1	1	2	2	2	1	2	2	2	2	1	1	1	1	A	1
165	6154/09	NATARAJ	47	1	1	2	2	1	1	1	2	2	2	2	2	1	1	2	2	2	2	1	2	1	2	F	2
166	6799/09	BAKTHAN	60	1	1	2	2	1	2	2	2	2	2	1	2	2	1	2	2	2	2	1	1	1	3	A	1
167	6884/09	SUBRAMANI	60	1	1	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	1	1	2	F	2
168	6857/09	BALARAMAN	61	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	1	1	1	2	1	1	E	2
169	6901/09	RAJARAM	57	1	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2	1	2	1	1	C	2
170	7110/09	SANKAR	40	1	1	2	2	2	1	1	2	1	2	2	2	2	2	2	2	2	2	2	1	1	1	A	1
171	7227/09	ARUMUGAM	58	1	1	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	2	1	2	1	1	A	1
172	7237/09	AYYANAR	41	1	1	2	2	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2	1	1	1	F	2

PROFORMA

DDHD NO. OGD NO. DATE

NAME/ADDRESS AGE SEX RELIGION

SYMPTOMS

- PAIN ABDOMEN
- INDIGESTION
- EARLY SATITY
- LOSS OF APPETITE
- LOSS OF WEIGHT
- NAUSEA/VOMITING
- AWARNES OF LUMP
- BRM
- ABD. DISTENSION
- DYSPHAGIA
- JAUNDICE
- UGI BLEED
- OTHERS

PAST HISTORY

- GASTRIC SURGERY
- COMORBID ILLNESS
- GASTRIC ULCER
- SMOKING
- ALCOHOL
- FAMILY H/O GASTRIC CANCER

HIGH SALT DIET/ DRY FISH

REFRIGERATOR/ FRESH FRUITS

ON EXAMINATION

HEIGHT WEIGHT BMI

PALLOR

JAUNDICE

SUPRA CLAVICULAR NODE

SCAR

VGP

EPIGASTRIC MASS

LIVER

ASCITES

OTHER SYSTEMS

INVESTIGATION

HB%

BLOOD GROUP

ESR

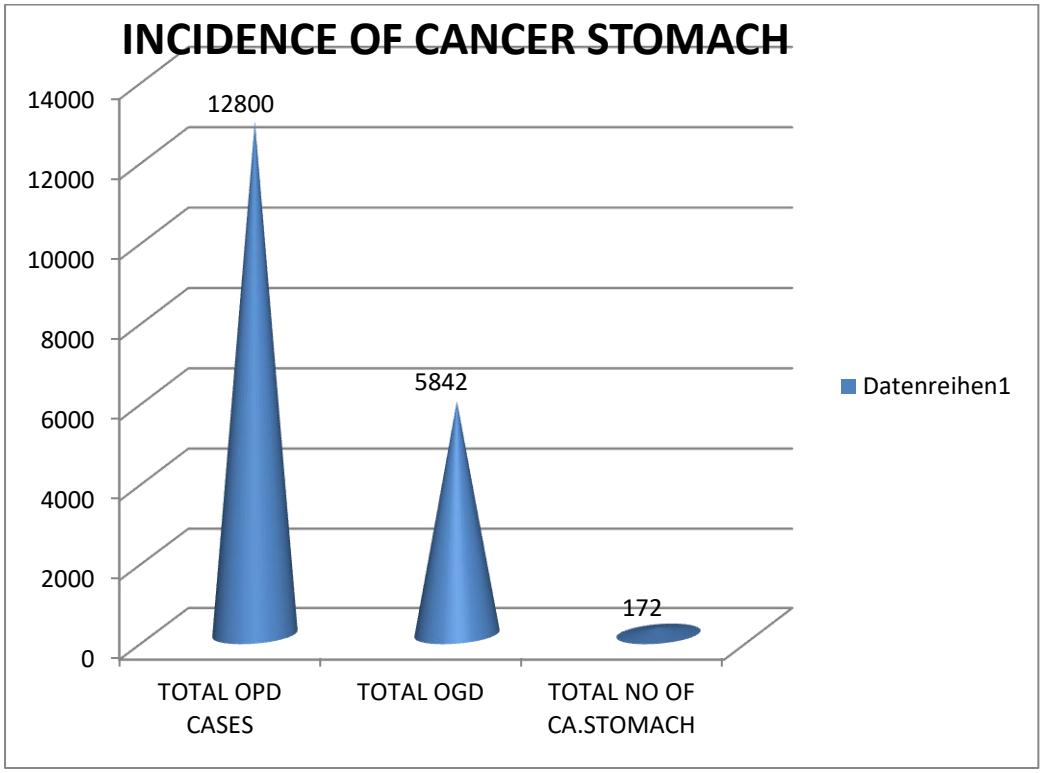
USG (ABD)

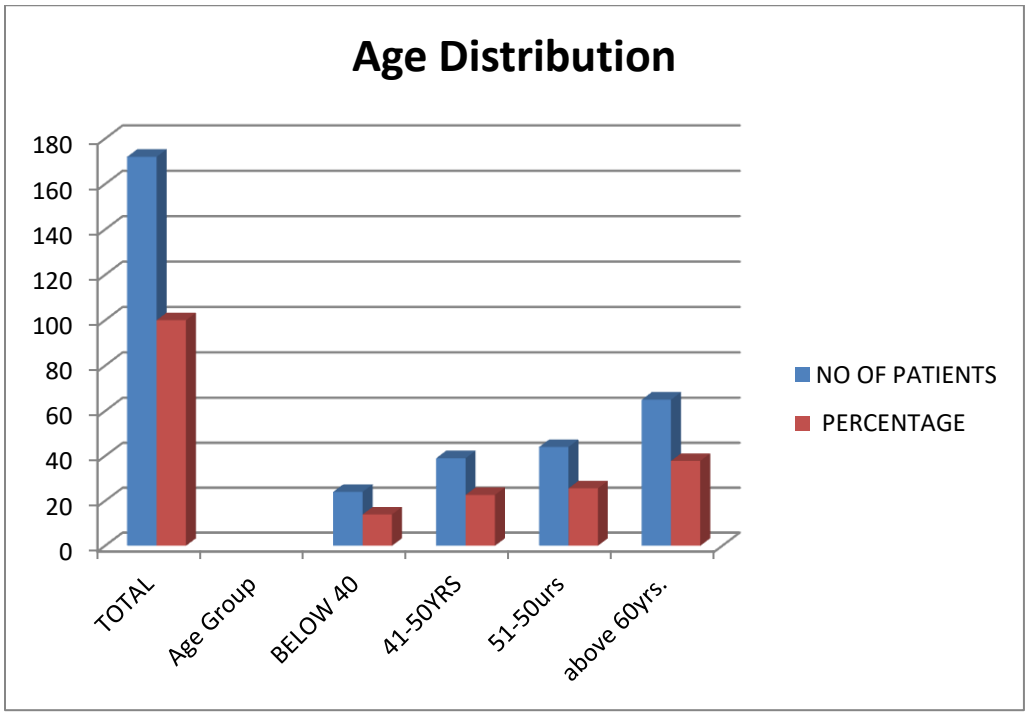
VOGD

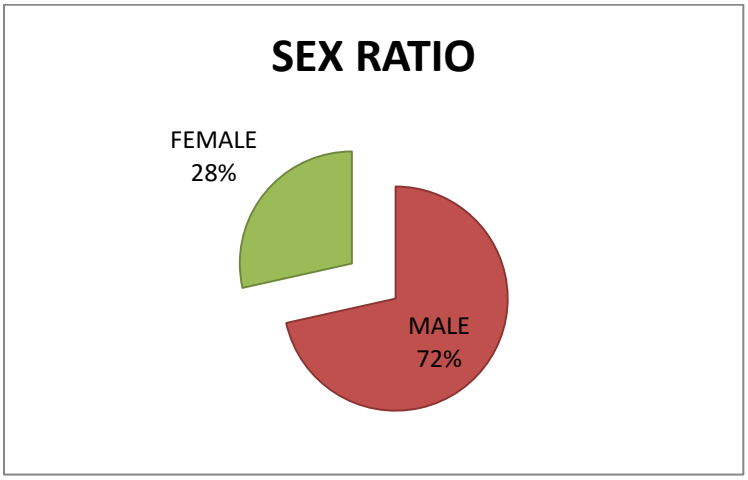
GOO IN OGD

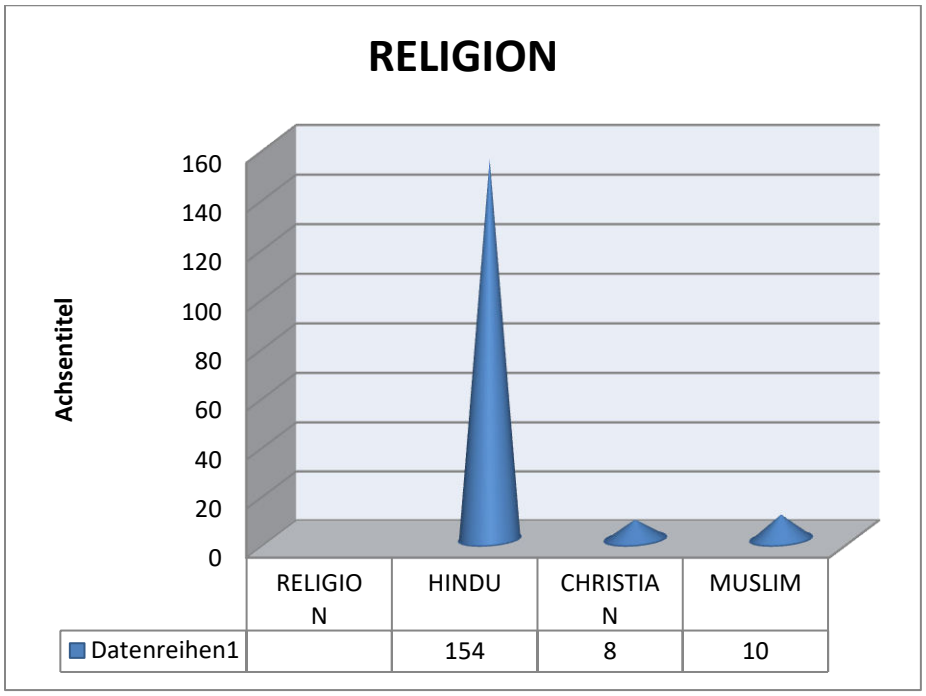
CT SCAN

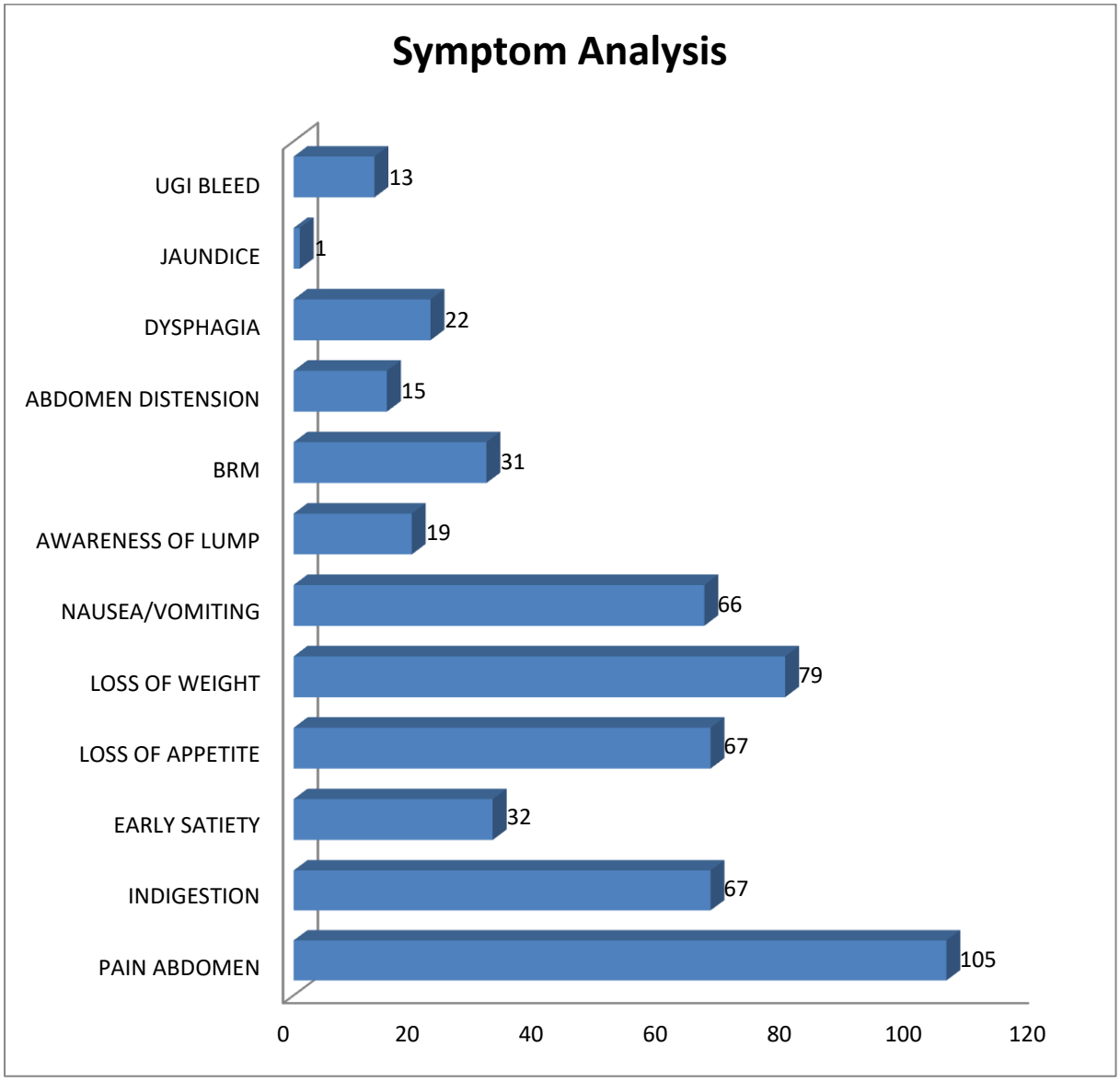
TREATMENT & FOLLOW UP

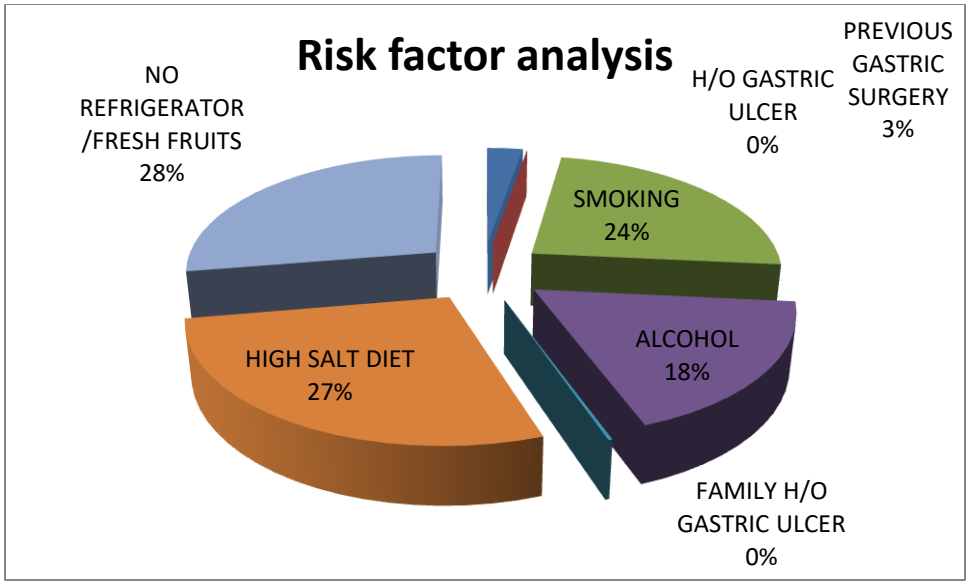

INCIDENCE OF CANCER STOMACH
14000
12000
10000
8000
6000
4000
2000
0
12800
5842
172
TOTAL OPD CASES
TOTAL OGD
TOTAL NO OF CA.STOMACH
Datenreihen1

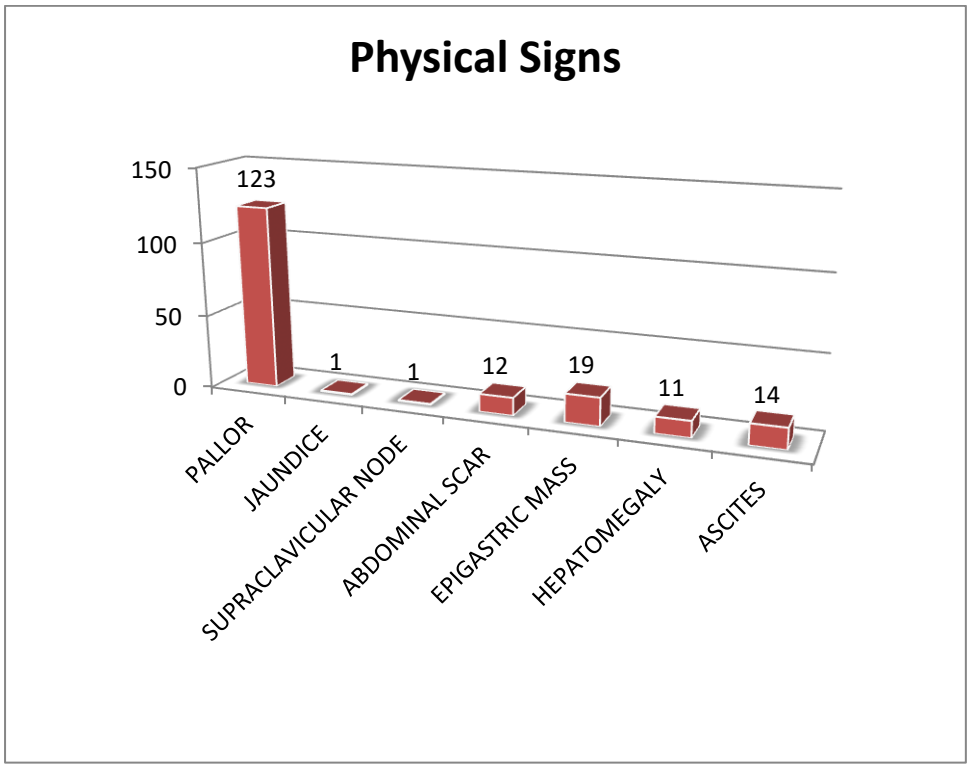

Age Distribution
180
160
140
120
100
80
60
40
20
0
TOTAL
Age Group
BELOW 40
41-50YRS
51-50urs
above 60yrs.
NO OF PATIENTS
PERCENTAGE

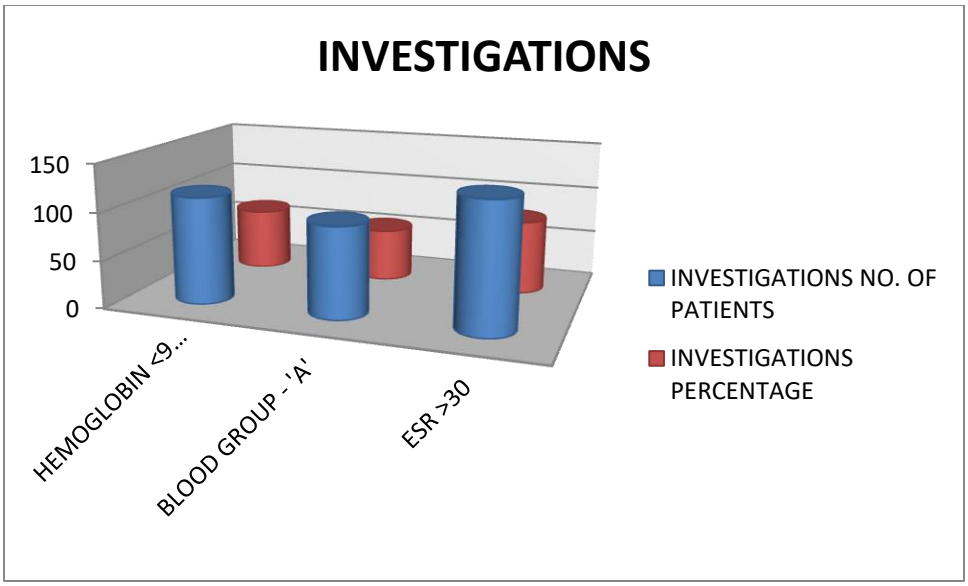

SEX RATIO
FEMALE
28%
MALE
72%

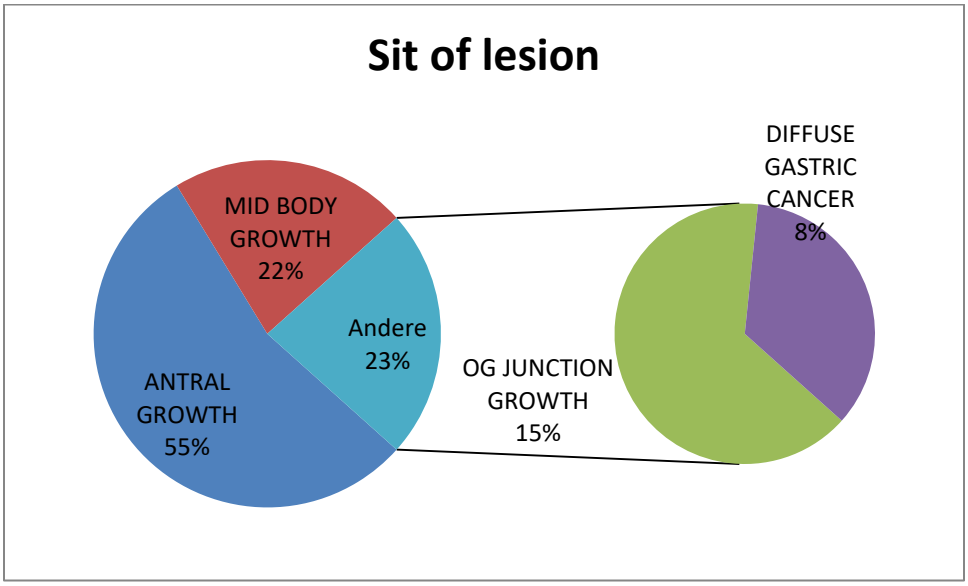

RELIGION
Achsentitel
160
140
120
100
80
60
40
20
0
RELIGION
HINDU
CHRISTIAN
MUSLIM
Datenreihen1
154
8
10

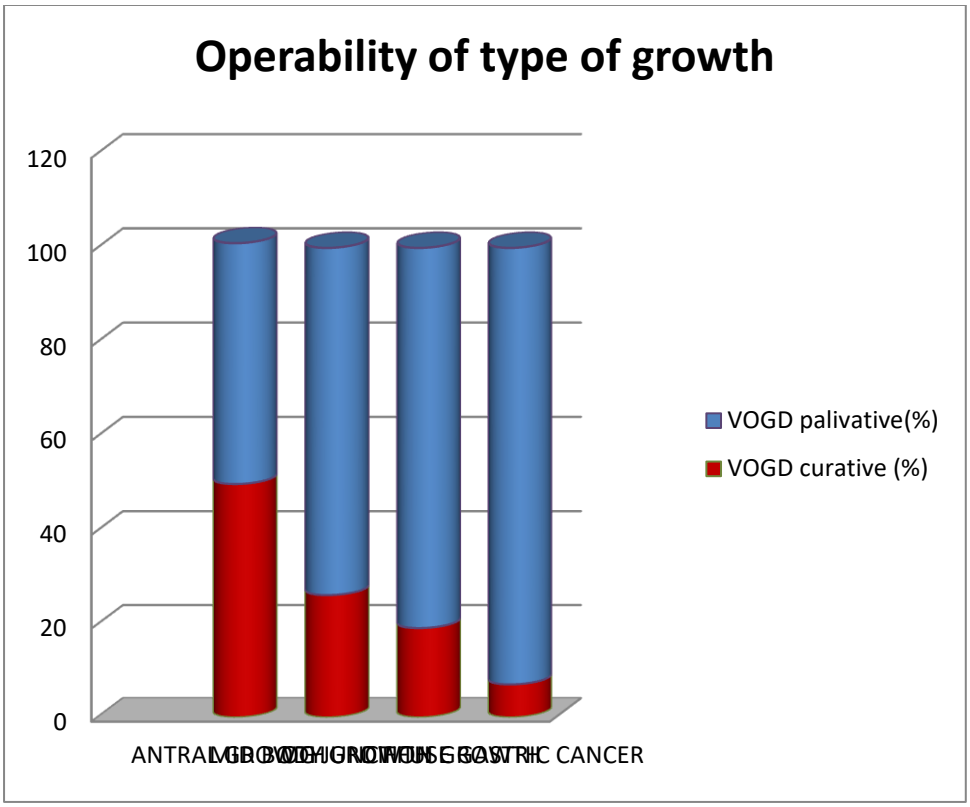

Symptom Analysis
UGI BLEED
13
JAUNDICE
1
DYSPHAGIA
22
ABDOMEN DISTENSION
15
BRM
31
AWARENESS OF LUMP
19
NAUSEA/VOMITING
66
LOSS OF WEIGHT
79
LOSS OF APPETITE
67
EARLY SATIETY
32
INDIGESTION
67
PAIN ABDOMEN
105
0
20
40
60
80
100
120

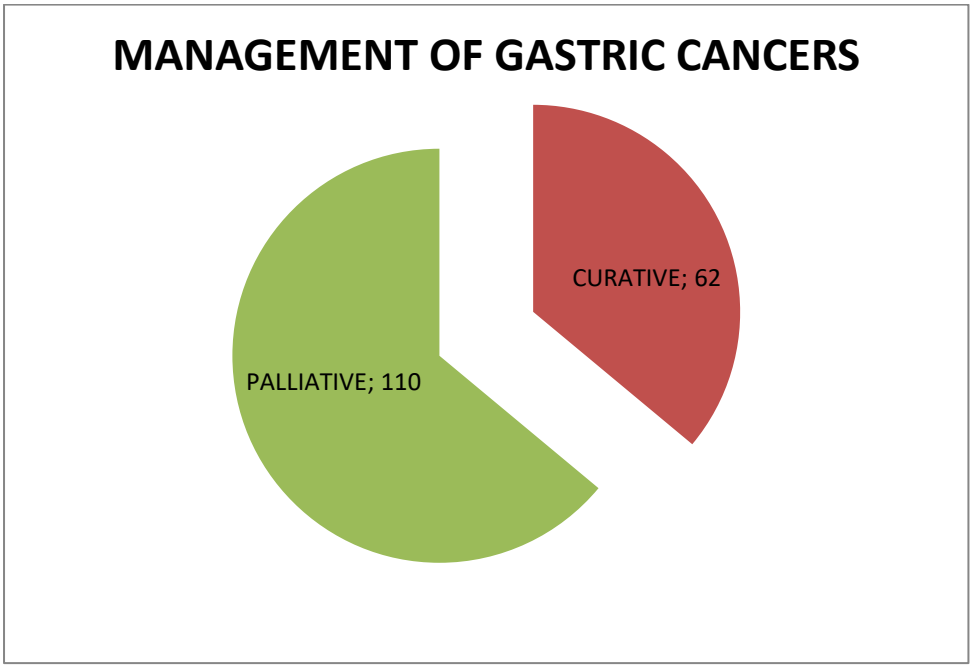

Risk factor analysis
NO REFRIGERATOR /FRESH FRUITS 28%
H/O GASTRIC ULCER 0%
PREVIOUS GASTRIC SURGERY 3%
SMOKING 24%
ALCOHOL 18%
HIGH SALT DIET 27%
FAMILY H/O GASTRIC ULCER 0%

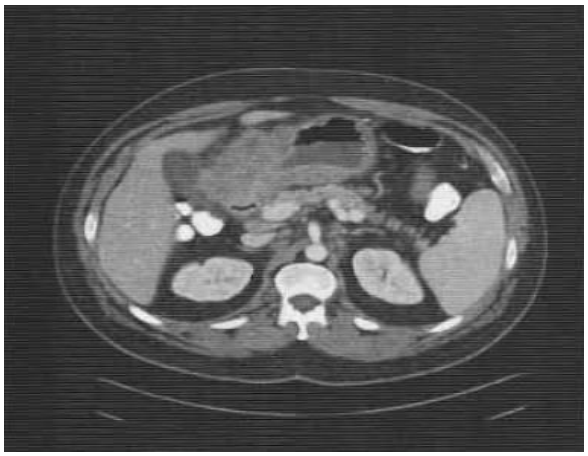

Physical Signs
150
100
50
0
123
1
1
12
19
11
14
PALLOR
JAUNDICE
SUPRACLAVICULAR NODE
ABDOMINAL SCAR
EPIGASTRIC MASS
HEPATOMEGALY
ASCITES

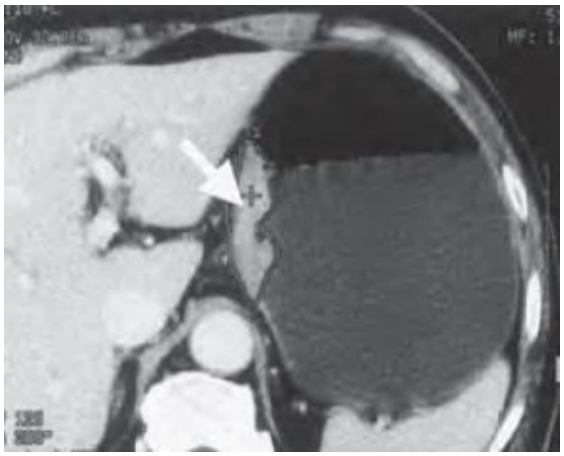

INVESTIGATIONS
150
100
50
0
HEMOGLOBIN <9...
BLOOD GROUP - 'A'
ESR >30
INVESTIGATIONS NO. OF PATIENTS
INVESTIGATIONS PERCENTAGE


Sit of lesion
MID BODY GROWTH 22%
Andere 23%
ANTRAL GROWTH 55%
DIFFUSE GASTRIC CANCER 8%
OG JUNCTION GROWTH 15%

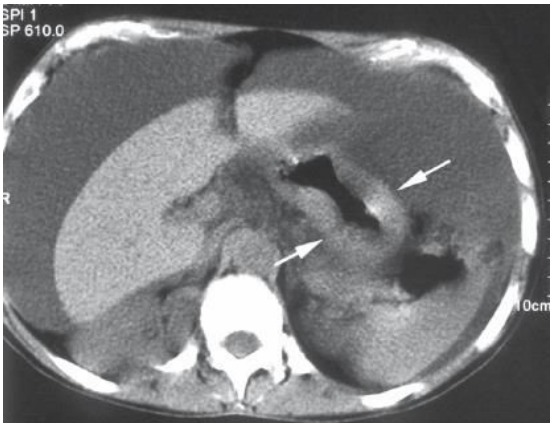

Operability of type of growth
120
100
80
60
40
20
0
VOGD palivative(%)
VOGD curative (%)


MANAGEMENT OF GASTRIC CANCERS
CURATIVE; 62
PALLIATIVE; 110

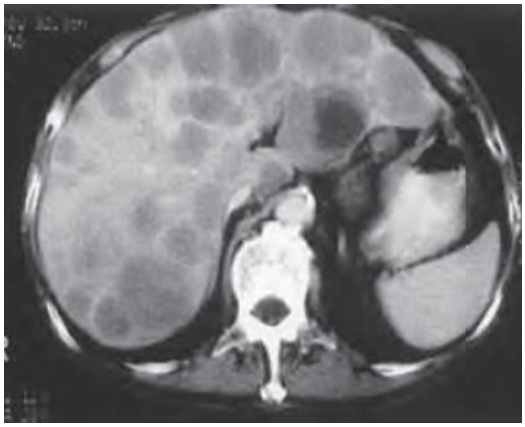




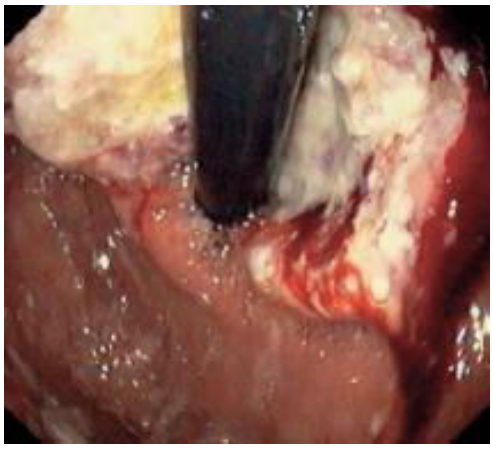
CT scan abdomen – Growth stomach with gastric wall thickening



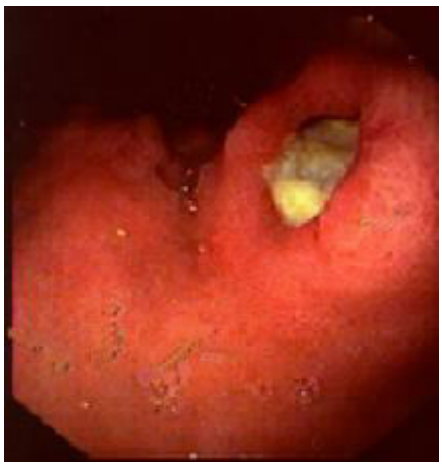
Antral wall thickening with ascites



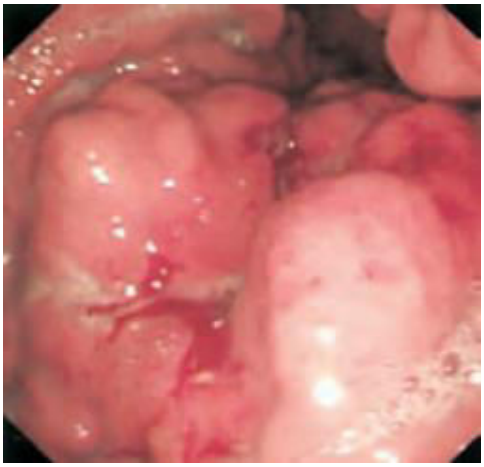
Growth stomach with multiple liver secondaries



OG Junction Growth



Malignant Gastric Ulcer



Antral growth

	Frequency	Percentage
Age Group		
Below 40	24	13.95
41-50	39	22.67
51-60	44	25.58
Above 60	65	37.79
Total	172	100.00

	Frequency	Percentage
Sex		
Male	123	71.51
Female	49	28.49
Total	172	100.00

	Yes		No		Total	
	Count	%	Count	%	Count	%
PC1	105	61.05	67	38.95	172	100.0
PC2	67	38.95	105	61.05	172	100.0
PC3	32	18.60	140	81.40	172	100.0
PC4	67	38.95	105	61.05	172	100.0
PC5	79	45.93	93	54.07	172	100.0
PC6	66	38.37	106	61.63	172	100.0
PC7	19	11.05	153	88.95	172	100.0
PC8	31	18.02	141	81.98	172	100.0
PC9	15	8.72	157	91.28	172	100.0
PC10	22	12.79	150	87.21	172	100.0
PC11	1	.58	171	99.42	172	100.0
PC12	13	7.56	159	92.44	172	100.0
PC13						

	Yes		No		Total	
	Count	%	Count	%	Count	%

RF1	12	6.98	160	93.02	172	100.0
RF2						
RF3						
RF4	106	61.63	66	38.37	172	100.0
RF5	80	46.51	92	53.49	172	100.0
RF6						
RF7	123	71.51	49	28.49	172	100.0
RF8	123	71.51	49	28.49	172	100.0

	Yes		No		Total	
	Count	%	Count	%	Count	%
PF1	123	71.51	49	28.49	172	100.0
PF2	1	.58	171	99.42	172	100.0
PF3			172	100.0	172	100.0
PF4						
PF5	12	6.98	160	93.02	172	100.0
PF6						
PF7	19	11.05	153	88.95	172	100.0
PF8	11	6.40	161	93.60	172	100.0
PF9	14	8.14	158	91.86	172	100.0
PF10						

	Frequency	Percentage
HB		
<= 9	113	65.70
> 9	59	34.30
Total	172	100.00
Blood Group		
A	96	55.81
Others	76	44.19
Total	172	100.00

	Frequency	Percentage
ESR		
>= 30	134	77.91

< 30	38	22.09
Total	172	100.00

	Frequency	Percentage
VOGD		
Antral growth	94	54.65
Mid Body growth	38	22.09
OG Junction growth	26	15.12
Diffuse gastric growth	14	8.14
Total	172	100.00
GOO		
Yes	56	58.33
No	40	41.67
Total	96	100.00

	Frequency	Percentage
CT scan		
Normal	62	36.05
Ascites	6	3.49
Nodes	42	24.42
Liver Secondary	10	5.81
Nodes + Secondary/Ascites	7	4.07
T4 lesion	45	26.16
Total	172	100.00
Treatment		
Curative	62	36.05
Paliative	110	63.95
Total	172	100.00

Variable	Mean	Std Dev	Minimum	Maximum	Valid N	Label
AGE	55.76	11.34	28	77	172	

PC1 by VOGD VOGD

VOGD Page 1 of 1

Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
	1	2	3	4	
PC1					
1 Yes	65	19	13	8	105
	61.9	18.1	12.4	7.6	61.0
	69.1	50.0	50.0	57.1	
2 No	29	19	13	6	67
	43.3	28.4	19.4	9.0	39.0
	30.9	50.0	50.0	42.9	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	5.96894	3	.11313
Likelihood Ratio	5.96743	3	.11321
Mantel-Haenszel test for linear association	3.36397	1	.06664

Minimum Expected Frequency - 5.453

Number of Missing Observations: 0

PC2 by VOGD VOGD

VOGD Page 1 of 1

Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
	1	2	3	4	
PC2					
1 Yes	43	6	4	14	67
	64.2	9.0	6.0	20.9	39.0
	45.7	15.8	15.4	100.0	
2 No	51	32	22		105
	48.6	30.5	21.0		61.0
	54.3	84.2	84.6		
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance

Pearson	38.41138	3	.00000
Likelihood Ratio	44.87449	3	.00000
Mantel-Haenszel test for linear association	.52641	1	.46812

Minimum Expected Frequency - 5.453

Number of Missing Observations: 0

PC3 by VOGD VOGD

VOGD Page 1 of 1

PC3	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
1 Yes		23	5	2	2	32
		71.9	15.6	6.3	6.3	18.6
		24.5	13.2	7.7	14.3	
2 No		71	33	24	12	140
		50.7	23.6	17.1	8.6	81.4
		75.5	86.8	92.3	85.7	
Column Total		94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	5.09549	3	.16494
Likelihood Ratio	5.48721	3	.13941
Mantel-Haenszel test for linear association	3.59802	1	.05785

Minimum Expected Frequency - 2.605
Cells with Expected Frequency < 5 - 2 OF 8 (25.0%)

Number of Missing Observations: 0

PC4 by VOGD VOGD

VOGD Page 1 of 1

PC4	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
1 Yes		37	15	12	3	67
		55.2	22.4	17.9	4.5	39.0

	39.4	39.5	46.2	21.4	
2	57	23	14	11	105
No	54.3	21.9	13.3	10.5	61.0
	60.6	60.5	53.8	78.6	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	2.38591	3	.49626
Likelihood Ratio	2.53377	3	.46922
Mantel-Haenszel test for linear association	.29311	1	.58824

Minimum Expected Frequency - 5.453

Number of Missing Observations: 0

PC5 by VOGD VOGD

PC5 / Count / Row Pct / Col Pct	VOGD 1 Antral growth	2 Mid Body growth	3 OG Junct ion grow	4 Diffuse gastric	Row Total
1 Yes	45	18	11	5	79
	57.0	22.8	13.9	6.3	45.9
	47.9	47.4	42.3	35.7	
2 No	49	20	15	9	93
	52.7	21.5	16.1	9.7	54.1
	52.1	52.6	57.7	64.3	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.90015	3	.82539
Likelihood Ratio	.91155	3	.82264
Mantel-Haenszel test for linear association	.76064	1	.38313

Minimum Expected Frequency - 6.430

Number of Missing Observations: 0

PC6 by VOGD VOGD

VOGD Page 1 of 1

Count / Row Pct / Col Pct	Antral growth 1	Mid Body growth 2	OG Junction growth 3	Diffuse gastric 4	Row Total
PC6					
1 Yes	42	8	9	7	66
	63.6	12.1	13.6	10.6	38.4
	44.7	21.1	34.6	50.0	
2 No	52	30	17	7	106
	49.1	28.3	16.0	6.6	61.6
	55.3	78.9	65.4	50.0	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	7.35782	3	.06133
Likelihood Ratio	7.74528	3	.05158
Mantel-Haenszel test for linear association	.33621	1	.56203

Minimum Expected Frequency - 5.372

Number of Missing Observations: 0

PC7 by VOGD VOGD

VOGD Page 1 of 1

Count / Row Pct / Col Pct	Antral growth 1	Mid Body growth 2	OG Junction growth 3	Diffuse gastric 4	Row Total
PC7					
1 Yes	11	5	2	1	19
	57.9	26.3	10.5	5.3	11.0
	11.7	13.2	7.7	7.1	
2 No	83	33	24	13	153
	54.2	21.6	15.7	8.5	89.0
	88.3	86.8	92.3	92.9	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance

Pearson	.72832	3	.86652
Likelihood Ratio	.77779	3	.85477
Mantel-Haenszel test for linear association	.40444	1	.52480

Minimum Expected Frequency - 1.547
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

PC8 by VOGD VOGD

	VOGD				Page 1 of 1
Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junct ion grow	Diffuse gastric	Row Total
	1	2	3	4	
PC8					
1 Yes	23	3	2	3	31
	74.2	9.7	6.5	9.7	18.0
	24.5	7.9	7.7	21.4	
2 No	71	35	24	11	141
	50.4	24.8	17.0	7.8	82.0
	75.5	92.1	92.3	78.6	
Column Total	94	38	26	14	172
	54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	7.26907	3	.06380
Likelihood Ratio	8.03387	3	.04532
Mantel-Haenszel test for linear association	2.45000	1	.11752

Minimum Expected Frequency - 2.523
Cells with Expected Frequency < 5 - 2 OF 8 (25.0%)

Number of Missing Observations: 0

PC9 by VOGD VOGD

	VOGD				Page 1 of 1
Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junct ion grow	Diffuse gastric	Row Total
	1	2	3	4	
PC9					
1	9	1	1	4	15

Yes	60.0	6.7	6.7	26.7	8.7
	9.6	2.6	3.8	28.6	
2	85	37	25	10	157
No	54.1	23.6	15.9	6.4	91.3
	90.4	97.4	96.2	71.4	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	9.56232	3	.02268
Likelihood Ratio	8.01935	3	.04561
Mantel-Haenszel test for linear association	.91170	1	.33967

Minimum Expected Frequency - 1.221
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

PC10 by VOGD VOGD

PC10	VOGD 1 Antral growth	2 Mid Body growth	3 OG Junct ion grow	4 Diffuse gastric	Row Total
1	2	2	16	2	22
Yes	9.1	9.1	72.7	9.1	12.8
	2.1	5.3	61.5	14.3	
2	92	36	10	12	150
No	61.3	24.0	6.7	8.0	87.2
	97.9	94.7	38.5	85.7	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	66.92915	3	.00000
Likelihood Ratio	50.38358	3	.00000
Mantel-Haenszel test for linear association	28.57044	1	.00000

Minimum Expected Frequency - 1.791
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

PC11 by VOGD VOGD

VOGD Page 1 of 1

PC11	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
1 Yes		1				1
		100.0				.6
		1.1				
2 No		93	38	26	14	171
		54.4	22.2	15.2	8.2	99.4
		98.9	100.0	100.0	100.0	
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.83464	3	.84117
Likelihood Ratio	1.21325	3	.74983
Mantel-Haenszel test for linear association	.60770	1	.43566

Minimum Expected Frequency - .081
Cells with Expected Frequency < 5 - 4 OF 8 (50.0%)

Number of Missing Observations: 0

PC12 by VOGD VOGD

VOGD Page 1 of 1

PC12	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
1 Yes		9	2	1	1	13
		69.2	15.4	7.7	7.7	7.6
		9.6	5.3	3.8	7.1	
2 No		85	36	25	13	159
		53.5	22.6	15.7	8.2	92.4
		90.4	94.7	96.2	92.9	

Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	1.34963	3	.71738

RF1 by VOGD VOGD

RF1	VOGD Count / Row Pct / Col Pct	Antral growth 1	Mid Body growth 2	OG Junct ion grow 3	Diffuse gastric 4	Row Total
1 Yes		9 75.0 9.6	1 8.3 2.6	1 8.3 3.8	1 8.3 7.1	12 7.0
2 No		85 53.1 90.4	37 23.1 97.4	25 15.6 96.2	13 8.1 92.9	160 93.0
Column Total		94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	2.47610	3	.47963
Likelihood Ratio	2.77528	3	.42758
Mantel-Haenszel test for linear association	.94649	1	.33062

Minimum Expected Frequency - .977
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>RF2 by VOGD

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>RF3 by VOGD

RF4 by VOGD VOGD

	VOGD				Page 1 of 1
Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
	1	2	3	4	
RF4					
1 Yes	60	23	16	7	106
	56.6	21.7	15.1	6.6	61.6
	63.8	60.5	61.5	50.0	
2 No	34	15	10	7	66
	51.5	22.7	15.2	10.6	38.4
	36.2	39.5	38.5	50.0	
Column Total	94	38	26	14	172
	54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	1.01276	3	.79816
Likelihood Ratio	.99212	3	.80316
Mantel-Haenszel test for linear association	.72155	1	.39564

Minimum Expected Frequency - 5.372

Number of Missing Observations: 0

RF5 by VOGD VOGD

	VOGD				Page 1 of 1
Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
	1	2	3	4	
RF5					
1 Yes	46	16	12	6	80
	57.5	20.0	15.0	7.5	46.5
	48.9	42.1	46.2	42.9	
2 No	48	22	14	8	92
	52.2	23.9	15.2	8.7	53.5
	51.1	57.9	53.8	57.1	
Column Total	94	38	26	14	172
	54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.59517	3	.89754

Likelihood Ratio	.59670	3	.89719
Mantel-Haenszel test for linear association	.27637	1	.59909

Minimum Expected Frequency - 6.512

Number of Missing Observations: 0

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>RF6 by VOGD

RF7 by VOGD VOGD

VOGD Page 1 of 1

RF7	Count / Row Pct / Col Pct	Antral growth 1	Mid Body growth 2	OG Junction growth 3	Diffuse gastric 4	Row Total
Yes	1	66	28	19	10	123
		53.7	22.8	15.4	8.1	71.5
		70.2	73.7	73.1	71.4	
No	2	28	10	7	4	49
		57.1	20.4	14.3	8.2	28.5
		29.8	26.3	26.9	28.6	
	Column Total	94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.19720	3	.97804
Likelihood Ratio	.19828	3	.97787
Mantel-Haenszel test for linear association	.07538	1	.78366

Minimum Expected Frequency - 3.988
Cells with Expected Frequency < 5 - 1 OF 8 (12.5%)

Number of Missing Observations: 0

RF8 by VOGD VOGD

VOGD Page 1 of 1

Count / Row Pct	Antral g	Mid Body	OG Junct	Diffuse

RF8	Col Pct	rowth 1	growth 2	ion grow 3	gastric 4	Row Total
1 Yes		66	28	19	10	123
		53.7	22.8	15.4	8.1	71.5
		70.2	73.7	73.1	71.4	
2 No		28	10	7	4	49
		57.1	20.4	14.3	8.2	28.5
		29.8	26.3	26.9	28.6	
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.19720	3	.97804
Likelihood Ratio	.19828	3	.97787
Mantel-Haenszel test for linear association	.07538	1	.78366

Minimum Expected Frequency - 3.988
Cells with Expected Frequency < 5 - 1 OF 8 (12.5%)

Number of Missing Observations: 0

PF1 by VOGD VOGD

PF1	Count / Row Pct / Col Pct	VOGD: Antral g rowth 1	Mid Body growth 2	OG Junct ion grow 3	Diffuse gastric 4	Row Total
1 Yes		66	28	19	10	123
		53.7	22.8	15.4	8.1	71.5
		70.2	73.7	73.1	71.4	
2 No		28	10	7	4	49
		57.1	20.4	14.3	8.2	28.5
		29.8	26.3	26.9	28.6	
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.19720	3	.97804
Likelihood Ratio	.19828	3	.97787
Mantel-Haenszel test for linear association	.07538	1	.78366

Minimum Expected Frequency - 3.988
Cells with Expected Frequency < 5 - 1 OF 8 (12.5%)

Number of Missing Observations: 0

PF2 by VOGD VOGD

VOGD Page 1 of 1

PF2	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
1 Yes		1				1
		100.0				.6
		1.1				
2 No		93	38	26	14	171
		54.4	22.2	15.2	8.2	99.4
		98.9	100.0	100.0	100.0	
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.83464	3	.84117
Likelihood Ratio	1.21325	3	.74983
Mantel-Haenszel test for linear association	.60770	1	.43566

Minimum Expected Frequency - .081
Cells with Expected Frequency < 5 - 4 OF 8 (50.0%)

Number of Missing Observations: 0

PF3 by VOGD VOGD

VOGD Page 1 of 1

PF3	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
		1	2	3	4	
2 No		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0
		100.0	100.0	100.0	100.0	

Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

>Warning # 10307
>Statistics cannot be computed when the number of non-empty rows or columns
>is one.

Number of Missing Observations: 0

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>PF4 by VOGD

PF5 by VOGD VOGD

PF5	VOGD Count / Row Pct / Col Pct	1 Antral growth	2 Mid Body growth	3 OG Junction growth	4 Diffuse gastric	Row Total
Yes	1	9 75.0 9.6	1 8.3 2.6	1 8.3 3.8	1 8.3 7.1	12 7.0
No	2	85 53.1 90.4	37 23.1 97.4	25 15.6 96.2	13 8.1 92.9	160 93.0
	Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	2.47610	3	.47963
Likelihood Ratio	2.77528	3	.42758
Mantel-Haenszel test for linear association	.94649	1	.33062

Minimum Expected Frequency - .977
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>PF6 by VOGD

PF7 by VOGD VOGD

VOGD Page 1 of 1

PF7	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
Yes	1	11	5	2	1	19
		57.9	26.3	10.5	5.3	11.0
		11.7	13.2	7.7	7.1	
No	2	83	33	24	13	153
		54.2	21.6	15.7	8.5	89.0
		88.3	86.8	92.3	92.9	
	Column Total	94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.72832	3	.86652
Likelihood Ratio	.77779	3	.85477
Mantel-Haenszel test for linear association	.40444	1	.52480

Minimum Expected Frequency - 1.547
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

PF8 by VOGD VOGD

VOGD Page 1 of 1

PF8	Count / Row Pct / Col Pct	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
Yes	1	6		5		11
		54.5		45.5		6.4
		6.4		19.2		
No	2	88	38	21	14	161
		54.7	23.6	13.0	8.7	93.6
		93.6	100.0	80.8	100.0	
	Column Total	94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance

Pearson	10.70819	3	.01341
Likelihood Ratio	11.68847	3	.00853
Mantel-Haenszel test for linear association	.24187	1	.62286

Minimum Expected Frequency - .895
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

PF9 by VOGD VOGD

PF9	VOGD: 1 Antral growth	2 Mid Body growth	3 OG Junct ion grow	4 Diffuse gastric	Row Total
1 Yes	8	1	1	4	14
	57.1	7.1	7.1	28.6	8.1
	8.5	2.6	3.8	28.6	
2 No	86	37	25	10	158
	54.4	23.4	15.8	6.3	91.9
	91.5	97.4	96.2	71.4	
Column Total	94	38	26	14	172
	54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	10.01671	3	.01842
Likelihood Ratio	7.86645	3	.04885
Mantel-Haenszel test for linear association	1.44469	1	.22938

Minimum Expected Frequency - 1.140
Cells with Expected Frequency < 5 - 3 OF 8 (37.5%)

Number of Missing Observations: 0

>Warning # 10370
>The crosstabulation table is empty.
>It is a 2-way table for the variables:
>PF10 by VOGD

BLO_GRP Blood Group by VOGD VOGD

BLO_GRP	VOGD	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
A	1	36	21	25	14	96
		37.5	21.9	26.0	14.6	55.8
		38.3	55.3	96.2	100.0	
Others	2	58	17	1		76
		76.3	22.4	1.3		44.2
		61.7	44.7	3.8		
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	39.93825	3	.00000
Likelihood Ratio	50.26253	3	.00000
Mantel-Haenszel test for linear association	37.39904	1	.00000

Minimum Expected Frequency - 6.186

Number of Missing Observations: 0

ESR ESR by VOGD VOGD

ESR	VOGD	Antral growth	Mid Body growth	OG Junction growth	Diffuse gastric	Row Total
>= 30	1	73	29	21	11	134
		54.5	21.6	15.7	8.2	77.9
		77.7	76.3	80.8	78.6	
< 30	2	21	9	5	3	38
		55.3	23.7	13.2	7.9	22.1
		22.3	23.7	19.2	21.4	
Column Total		94	38	26	14	172
		54.7	22.1	15.1	8.1	100.0

Chi-Square	Value	DF	Significance
Pearson	.18659	3	.97973

Likelihood Ratio	.18981	3	.97922
Mantel-Haenszel test for linear association	.04685	1	.82864

Minimum Expected Frequency - 3.093
Cells with Expected Frequency < 5 - 1 OF 8 (12.5%)

Number of Missing Observations: 0

HB HB by VOGD VOGD

HB	VOGD 1 Antral growth	2 Mid Body growth	3 OG Junct ion grow	4 Diffuse gastric	Row Total
1 <= 9	61	26	18	8	113
	54.0	23.0	15.9	7.1	65.7
	64.9	68.4	69.2	57.1	
2 > 9	33	12	8	6	59
	55.9	20.3	13.6	10.2	34.3
	35.1	31.6	30.8	42.9	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.75069	3	.86122

TREAT Treatment by VOGD VOGD

TREAT	VOGD 1 Antral growth	2 Mid Body growth	3 OG Junct ion grow	4 Diffuse gastric	Row Total
1 Curative	46	10	5	1	62
	74.2	16.1	8.1	1.6	36.0
	48.9	26.3	19.2	7.1	
2 Paliative	48	28	21	13	110
	43.6	25.5	19.1	11.8	64.0
	51.1	73.7	80.8	92.9	
Column Total	94 54.7	38 22.1	26 15.1	14 8.1	172 100.0

Chi-Square	Value	DF	Significance
Pearson	16.59804	3	.00086

CT_SCAN CT scan by TREAT Treatment

CT_SCAN	Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total
1 Normal	Count	61	1	62
	Row Pct	98.4	1.6	36.0
	Col Pct	98.4	.9	
2 Ascites	Count		6	6
	Row Pct		100.0	3.5
	Col Pct		5.5	
3 Nodes	Count	1	41	42
	Row Pct	2.4	97.6	24.4
	Col Pct	1.6	37.3	
4 Liver Secondary	Count		10	10
	Row Pct		100.0	5.8
	Col Pct		9.1	
5 Nodes + Secondar	Count		7	7
	Row Pct		100.0	4.1
	Col Pct		6.4	
6 T4 lesion	Count		45	45
	Row Pct		100.0	26.2
	Col Pct		40.9	
Column Total		62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	163.49759	5	.00000

PC1 by TREAT Treatment

PC1	Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total

1	39	66	105
Yes	37.1	62.9	61.0
	62.9	60.0	
2	23	44	67
No	34.3	65.7	39.0
	37.1	40.0	
Column Total	62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.14054	1	.70774
Continuity Correction	.04497	1	.83206
Likelihood Ratio	.14093	1	.70736
Mantel-Haenszel test for linear association	.13973	1	.70855

Minimum Expected Frequency - 24.151

Number of Missing Observations: 0

PC2 by TREAT Treatment

PC2	TREAT: Curative (1)	TREAT: Paliative (2)	Row Total
1 Yes	22	45	67
	32.8	67.2	39.0
	35.5	40.9	
2 No	40	65	105
	38.1	61.9	61.0
	64.5	59.1	
Column Total	62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.49078	1	.48358
Continuity Correction	.28915	1	.59077
Likelihood Ratio	.49346	1	.48239
Mantel-Haenszel test for linear association	.48792	1	.48486

Minimum Expected Frequency - 24.151

Number of Missing Observations: 0

PC3 by TREAT Treatment

	TREAT	Page 1 of 1	
Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total
PC3			
1 Yes	20 / 62.5 / 32.3	12 / 37.5 / 10.9	32 / 18.6
2 No	42 / 30.0 / 67.7	98 / 70.0 / 89.1	140 / 81.4
Column Total	62 / 36.0	110 / 64.0	172 / 100.0

Chi-Square	Value	DF	Significance
Pearson	11.93408	1	.00055
Continuity Correction	10.56592	1	.00115
Likelihood Ratio	11.48570	1	.00070
Mantel-Haenszel test for linear association	11.86469	1	.00057

Minimum Expected Frequency - 11.535

Number of Missing Observations: 0

PC4 by TREAT Treatment

	TREAT	Page 1 of 1	
Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total
PC4			
1 Yes	26 / 38.8 / 41.9	41 / 61.2 / 37.3	67 / 39.0
2 No	36 / 34.3 / 58.1	69 / 65.7 / 62.7	105 / 61.0

Column Total	62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.36252	1	.54711
Continuity Correction	.19295	1	.66047
Likelihood Ratio	.36126	1	.54781
Mantel-Haenszel test for linear association	.36041	1	.54828

Minimum Expected Frequency - 24.151

Number of Missing Observations: 0

PC5 by TREAT Treatment

TREAT Page 1 of 1

PC5	Count / Row Pct / Col Pct	Curative (1)	Paliative (2)	Row Total
1 Yes		30 38.0 48.4	49 62.0 44.5	79 45.9
2 No		32 34.4 51.6	61 65.6 55.5	93 54.1
Column Total		62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	.23563	1	.62738
Continuity Correction	.10633	1	.74436
Likelihood Ratio	.23543	1	.62753
Mantel-Haenszel test for linear association	.23426	1	.62838

Minimum Expected Frequency - 28.477

Number of Missing Observations: 0

PC6 by TREAT Treatment

PC6	TREAT	Curative (1)	Paliative (2)	Row Total
Yes	1	30	36	66
	Row Pct	45.5	54.5	38.4
	Col Pct	48.4	32.7	
No	2	32	74	106
	Row Pct	30.2	69.8	61.6
	Col Pct	51.6	67.3	
	Column Total	62	110	172
	Column Total	36.0	64.0	100.0

Chi-Square	Value	DF	Significance
Pearson	4.11185	1	.04258
Continuity Correction	3.47630	1	.06225
Likelihood Ratio	4.07818	1	.04344
Mantel-Haenszel test for linear association	4.08794	1	.04319

Minimum Expected Frequency - 23.791

Number of Missing Observations: 0

PC7 by TREAT Treatment

PC7	TREAT	Curative (1)	Paliative (2)	Row Total
Yes	1	6	13	19
	Row Pct	31.6	68.4	11.0
	Col Pct	9.7	11.8	
No	2	56	97	153
	Row Pct	36.6	63.4	89.0
	Col Pct	90.3	88.2	
	Column Total	62	110	172
	Column Total	36.0	64.0	100.0

Chi-Square	Value	DF	Significance

Pearson	.18493	1	.66717
Continuity Correction	.03123	1	.85972
Likelihood Ratio	.18819	1	.66443
Mantel-Haenszel test for linear association	.18385	1	.66808

Minimum Expected Frequency - 6.849

Number of Missing Observations: 0

PC8 by TREAT Treatment

PC8	TREAT 1	TREAT 2	Row Total
1 Yes	22	9	31
Row Pct	71.0	29.0	18.0
Col Pct	35.5	8.2	
2 No	40	101	141
Row Pct	28.4	71.6	82.0
Col Pct	64.5	91.8	
Column Total	62	110	172
	36.0	64.0	100.0

Chi-Square	Value	DF	Significance
Pearson	20.00427	1	.00001
Continuity Correction	18.19907	1	.00002
Likelihood Ratio	19.33087	1	.00001
Mantel-Haenszel test for linear association	19.88796	1	.00001

Minimum Expected Frequency - 11.174

Number of Missing Observations: 0

PC9 by TREAT Treatment

PC9	TREAT 1	TREAT 2	Row Total
1	2	13	15

Yes	1	13.3	86.7	8.7
		3.2	11.8	
No	2	60	97	157
		38.2	61.8	91.3
		96.8	88.2	
Column Total		62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	3.67746	1	.05515
Continuity Correction	2.67728	1	.10179
Likelihood Ratio	4.24162	1	.03944
Mantel-Haenszel test for linear association	3.65608	1	.05587

Minimum Expected Frequency - 5.407

Number of Missing Observations: 0

PC10 by TREAT Treatment

PC10	Count / Row Pct / Col Pct	TREAT Curative (1)	TREAT Paliative (2)	Row Total
Yes	1	5	17	22
		22.7	77.3	12.8
		8.1	15.5	
No	2	57	93	150
		38.0	62.0	87.2
		91.9	84.5	
Column Total		62 36.0	110 64.0	172 100.0

Chi-Square	Value	DF	Significance
Pearson	1.94129	1	.16353
Continuity Correction	1.33531	1	.24786
Likelihood Ratio	2.06628	1	.15059
Mantel-Haenszel test for linear association	1.93001	1	.16476

Minimum Expected Frequency - 7.930

Number of Missing Observations: 0

PC11 by TREAT Treatment

PC11	TREAT: Curative (1)	TREAT: Paliative (2)	Row Total
1 Yes	1		1
	100.0		.6
	1.6		
2 No	61	110	171
	35.7	64.3	99.4
	98.4	100.0	
Column Total	62	110	172
	36.0	64.0	100.0

Chi-Square	Value	DF	Significance
Pearson	1.78457	1	.18159
Continuity Correction	.08495	1	.77070
Likelihood Ratio	2.05111	1	.15209
Mantel-Haenszel test for linear association	1.77419	1	.18286
Fisher's Exact Test:			
One-Tail			.36047
Two-Tail			.36047

Minimum Expected Frequency - .360
Cells with Expected Frequency < 5 - 2 OF 4 (50.0%)

Number of Missing Observations: 0

PC12 by TREAT Treatment

PC12	TREAT: Curative (1)	TREAT: Paliative (2)	Row Total
1 Yes	5	8	13
	38.5	61.5	7.6
	8.1	7.3	
2 No	57	102	159
	35.8	64.2	92.4

	91.9	92.7
Column	62	110	172
Total	36.0	64.0	100.0

Chi-Square	Value	DF	Significance
Pearson	.03558	1	.85039

VOGD VOGD by TREAT Treatment
Controlling for..
PC8 Value = 1 Yes

TREAT Page 1 of 1

VOGD	Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total
1 Antral growth		20	3	23
		87.0	13.0	74.2
		90.9	33.3	
2 Mid Body growth		2	1	3
		66.7	33.3	9.7
		9.1	11.1	
3 OG Junction grow			2	2
			100.0	6.5
			22.2	
4 Diffuse gastric			3	3
			100.0	9.7
			33.3	
Column		22	9	31
Total		71.0	29.0	100.0

Chi-Square	Value	DF	Significance
Pearson	15.10291	3	.00173

GOO by TREAT Treatment

TREAT Page 1 of 1

GOO	Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total

1	24	32	56
Yes	42.9	57.1	58.3
	52.2	64.0	
2	22	18	40
No	55.0	45.0	41.7
	47.8	36.0	
Column Total	46 47.9	50 52.1	96 100.0

Chi-Square	Value	DF	Significance
Pearson	1.37858	1	.24034

- - - - - Chi-Square Test

AGE_G Age Group

Category	Cases Observed	Expected	Residual
Below 40	1	24	43.00	-19.00
41-50	2	39	43.00	-4.00
51-60	3	44	43.00	1.00
Above 60	4	65	43.00	22.00
Total		172	

Chi-Square	D.F.	Significance
20.0465	3	.0002

VOGD VOGD by TREAT Treatment
Controlling for..
PC10 Value = 1 Yes

TREAT Page 1 of 1

VOGD	Count / Row Pct / Col Pct	Curative 1	Paliative 2	Row Total
1 Antral growth		1 50.0 20.0	1 50.0 5.9	2 9.1
2 Mid Body growth		1 50.0 20.0	1 50.0 5.9	2 9.1
3 OG Junction grow		3 18.8	13 81.3	16 72.7

	60.0	76.5	
4		2	2
Diffuse gastric		100.0	9.1
		11.8	
Column	5	17	22
Total	22.7	77.3	100.0

Chi-Square	Value	DF	Significance
Pearson	2.42647	3	.48873