*Dissertation*

# "A STUDY ON GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15-35 YEARS OF AGE) AND ITS ETIOLOGY"

**Dissertation submitted to**

**THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI**

**in partial fulfilment of the regulations for the Award of the degree of**

**M.S. (General Surgery)**

**Branch – I**

**THE TAMILNADU DR. MGR MEDICAL UNIVERSITY**

**CHENNAI**

**MAY 2019**

# CERTIFICATE

This is to certify that, the dissertation entitled **"A STUDY ON GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15-35 YEARS OF AGE) AND ITS ETIOLOGY"**

Is the bonafide work done by **DR. K. MADHANAGOPALAN** during his **M.S. (General Surgery)** course **2016-2019**, done under my supervision and is submitted in partial fulfilment of the requirement for the M.S.(BRANCH-I)-General Surgery of The Tamilnadu Dr.MGR Medical University, May 2019 examination.

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# DECLARATION

I, certainly declare that this dissertation titled **"A STUDY ON GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15-35 YEARS OF AGE) AND ITS ETIOLOGY"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The TamilNadu Dr. M.G.R Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Master of Surgery Degree Branch I (General Surgery).

DATE: DR K.MADHANAGOPALAN

PLACE: (POST GRADUATE)

# ACKNOWLEDGEMENT

I express my heartfelt gratitude to the Dean, **DR. R. JAYANTHI, M.D., FRCP,** Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for permitting me to do this study.

I am deeply indebted to **Prof. Dr.M.ALLI, M.S., DGO** Director& Professor, Institute of General Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital for his support and guidance.

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I would like to extend my sincere thanks to my Assistant Professors **Dr.JOYCE PRABHAKAR, M.S.,Dr.K.SENTHILKUMAR, M.S.,** and **Dr.M.SIVAN, M.S,** Madras Medical College & Rajiv Gandhi Government General Hospital who guided and trimmed my work throughout the period of my study.

I am extremely thankful to all the Members of the INSTITUTIONAL ETHICAL COMMITTEE for giving approval for my study. I also thank all the patients who were part of the study and my Professional colleagues, family and friends for their support and criticisms.

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This is to certify that this dissertation work titled **"A STUDY ON GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15-35 YEARS OF AGE) AND ITS ETIOLOGY"** of the candidate **Dr.K.MADHANAGOPALAN** with registration Number **221611004** for the award of **M.S degree** in the BRANCH -1 of **General Surgery**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **8%** percentage of plagiarism in the dissertation.

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# INSTITUTIONAL ETHICS COMMITTEE
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## CERTIFICATE OF APPROVAL

To

Dr.K.Madhanagopalan
I Year PG in MS General Surgery
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Dear Dr.K.Madhanagopalan,

The Institutional Ethics Committee has considered your request and approved your study titled **"GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15-35 YEARS OF AGE) AND ITS ETIOLOGY " - NO.07062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

| | |
|---|---|
| 1. Prof.Dr.C.Rajendran, MD., | :Chairperson |
| 2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3 | : Deputy Chairperson |
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| 8.Thiru S.Govindasamy, BA.,BL,High Court,Chennai | : Lawyer |
| 9.Tmt.Arnold Saulina, MA.,MSW., | :Social Scientist |
| 10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3 | : Lay Person |

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

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

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

# CONTENTS

# INTRODUCTION

Gastroduodenal perforation is one of the most serious and most overwhelming catastrophic event that is affecting human being.(Lord Moynihan)

Gastroduodenal perforation are third in frequency after acute appendicitis and acute intestinal obstruction among abdominal emergency.

Though a lot of work has been done on etiology of this condition, one specific etiology after cause be incriminated in causation of this particular disease especially in our part of country.

There is decline in incidence of duodenal and gastric ulcer and elective surgery, attributable to era of $H_2$ blocker and proton pump inhibitor which provides symptomatic relief to patient. But the percentage of patients with perforation has not declined probably due to inadvertent use of NSAIDS , corticosteroids, and because of irregular use of $H_2$ blocker.

Differences in the clinical presentation of gastroduodenal perforations vary from the typical severe acute abdominal pain at one end, to subtle or no symptoms in the hospitalized patients for unrelated illness at the other end. The various atypical presentations that mimic other abdominal conditions throw a real challenge over the diagnosis to the emergency surgeon.

A careful medical history, methodical clinical examination and radiological study play a major role in the early diagnosis of this acute abdominal emergency. There are multiple factors that influence the prognosis and outcome of the patient. Preoperative resuscitation, intravenous administration of broad-spectrum antibiotics

and good postoperative care are the mainstay in the management of Gastro duodenal Perforations. The operative management depends upon the cause of perforations.

The mortality has been reduced nowadays due to medical attention , quick diagnosis and prompt surgical management. But no significant method of treatment is appropriate for every patient with perforated gastroduodenal ulcer.

The study was conducted with aim of analysing various etiological factors.

# AIMS AND OBJECTIVES

To analyse and identify most common etiological and risk factors among patients with gastroduodenal perforations in age group of 15 to 35 years at our institution.

To help young people at risk from developing gastroduodenal perforations by bringing out essential life style modifications.

# REVIEW OF LITERATURE

## HISTORICAL REVIEW

Acute perforation of peptic ulcer is relatively a common complication. It was rarely reported 100 years ago. There is progressively an increase in its incidence during the last few decades in India. In the year 1944, Illingworth has shown from his 20 years study from 1924 to 1944, a fivefold increase in the incidence of gastrointestinal perforations.

Warren Cole assessed the occurrence of perforations in chronic duodenal ulcer and in chronic gastric ulcer was 20.5%. Rawlison was credited with the first published report in 1727 of a perforated gastric ulcer. The first published report of a perforated duodenal ulcer was by Hambergeiri in 1946.

Heusner was the first to close a perforated duodenal ulcer successfully. Simple closure of a perforated ulcer was done in 1892 by Kriege. Cellen Jones in 1929 described the most widely used method of closing a perforation with a live omental patch, often wrongly credited to Roscoe Graham. Moore and colleagues in 1950 found that recurrence of ulcer symptoms after repair of a perforation carried a bad prognosis in their 10 year follow up analysis of 1000 ulcer patients.

Collier and Pain in 1985 reported that 45% of the patients aged 15 years or more presenting with perforated ulcer had consumed NSAIDs. Watkini et al. in 1984 found that 25% of the patients in the Oxford area were consuming NSAIDs, and 4.8% were taking steroids at the time of perforation.[1]

Hamilton and Harbrecht in 1967 and Khan and Ralston in 1970 reported that operative mortality of truncal vagotomy with PGJ is about 1%. Jordan, De Bakey and Duncan in 1974 reported 535 emergency partial gastrectomies with an operative morality of 2.2%. J S Pierandozzi, B BHin Shaw and O E Stafford in 1960 treated perforated peptic ulcer by vagotomy and pyloroplasty. Laparoscopic treatment was reported in the year 1990.

Mouret et al. found that laparoscopic management is good because of avoiding large incision, decrease in the wound infection and good peritoneal lavage. He treated 4 out of 5 patients successfully. In 1997 John Wayman and Simon ARaimes found that simple closure treatement is safe and effective in long term, when combined with H.pylori eradication and pharmacological suppression.

## SURGICAL ANATOMY

Gastro Intestinal perforation is a complete perforation of the wall of the stomach, small intestine or large bowel, resulting in intestinal contents flowing into the abdominal cavity. Perforation of the intestine results in the potential for bacterial contamination of the abdominal cavity (a condition known as peritonitis). Perforation of the stomach can lead to chemical peritonitis due to leaked gastric acid. Perforation any where along the gastro intestinal tract is a surgical emergency.

## STOMACH

## ANATOMY OF STOMACH

The stomach, part of the gastrointestinal tract, is a digestive organ located between the esophagus and the duodenum.

It has a ‘J’ shape, and features a lesser and greater curvature. The anterior and posterior surfaces are smoothly rounded with a peritoneal covering.

### Anatomical Position

The stomach is located in the superior aspect of the abdomen. It lies in the epigastric and umbilical regions, mostly protected by the lower portion of the rib cage.

The exact size, shape and position of the stomach can vary from person to person. For example, in thin individuals, it is not uncommon for the stomach to extend into the pelvic region.

### Anatomical Structure

The stomach has four main regions; the cardia, fundus, body and pylorus:

Cardia – surrounds the superior opening of the stomach.

Fundus – the rounded portion superior to and left of the cardia.

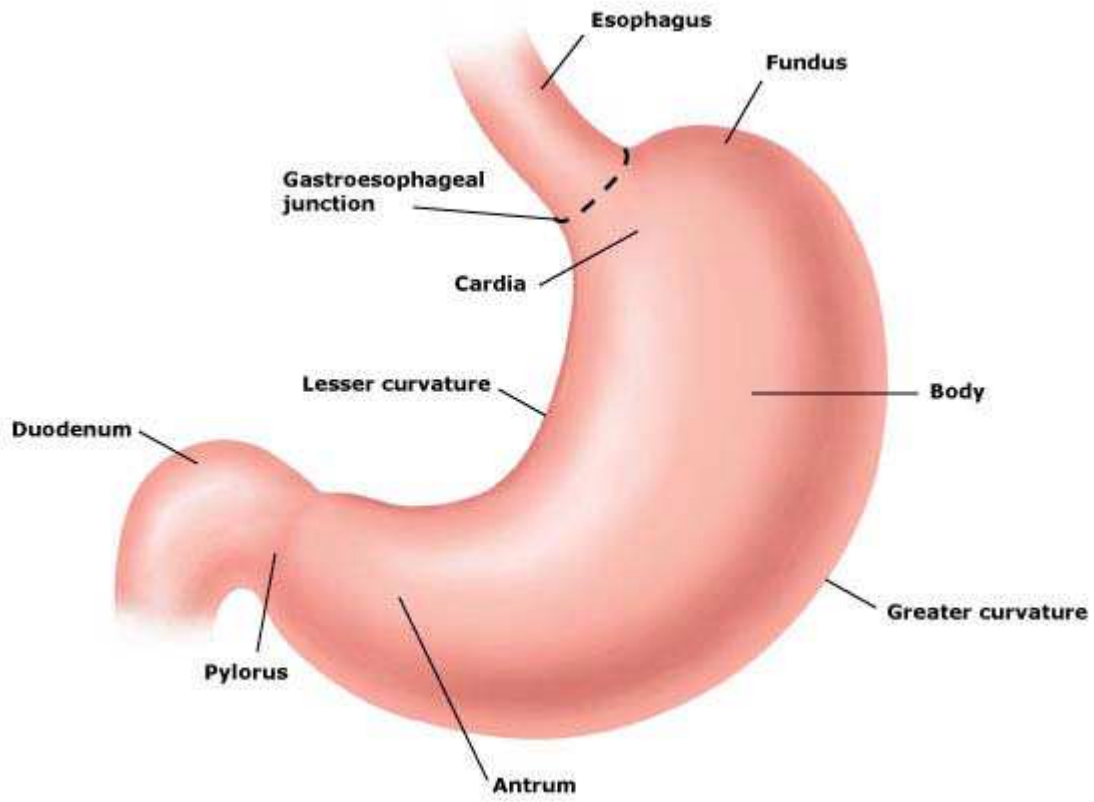
Body – the large central portion inferior to the fundus.

Pylorus – connects the stomach to the duodenum.

**Greater and Lesser Curvatures**

The medial and lateral borders of the stomach are curved, forming the lesser and greater curvatures.

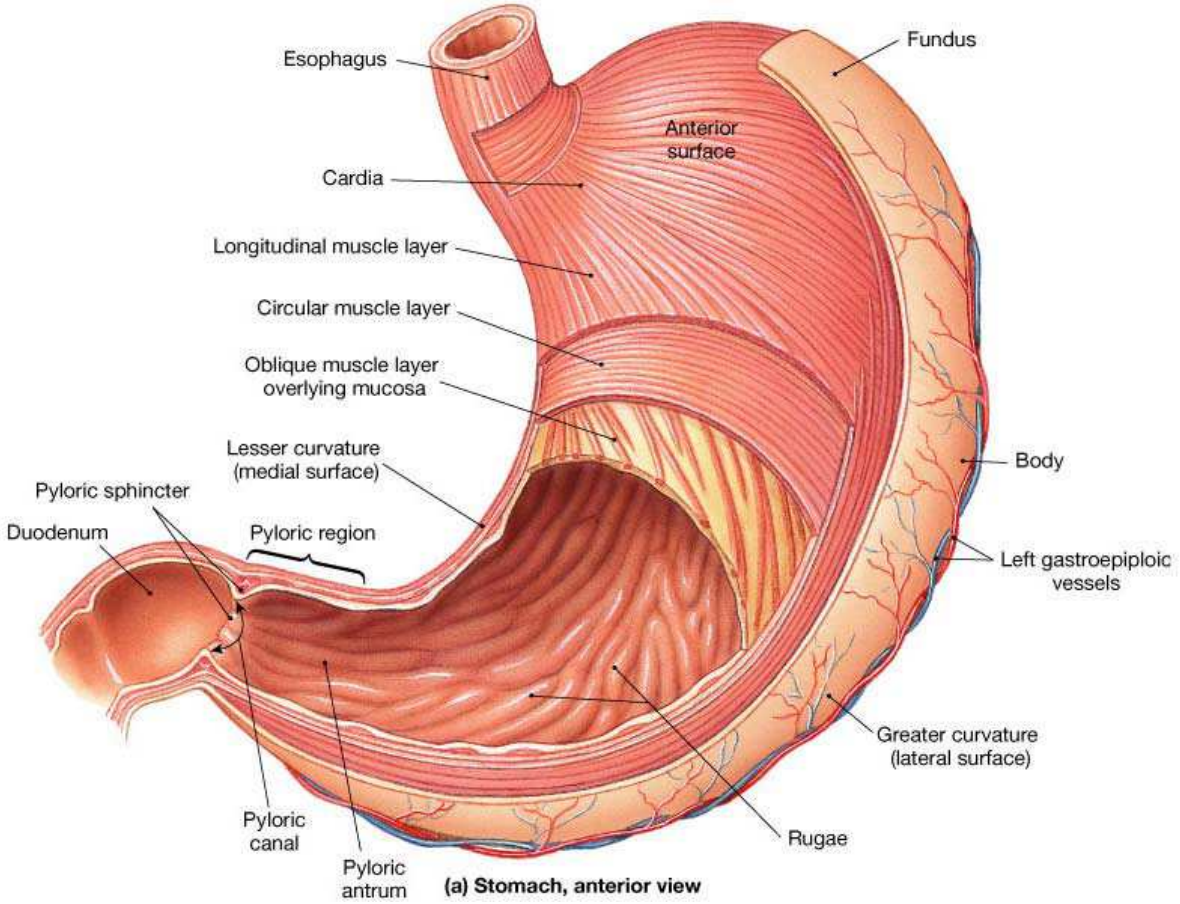
Greater curvature – forms the long, convex, lateral border of the stomach.




Arising at the cardiac orifice, it arches backwards and passes inferiorly to the left. It curves to the right as it continues medially to reach the pyloric antrum. The short gastric arteries and the right and left gastro-omental arteries supply branches to the greater curvature.

**Lesser curvature** – forms the shorter, concave, medial surface of the stomach. The most inferior part of the lesser curvature, the angular notch, indicates the junction of the body and pyloric region. The lesser curvature gives attachment to the

hepatogastric ligament and is supplied by the left gastric artery and right gastric branch of the hepatic artery.




**Neurovascular Supply**

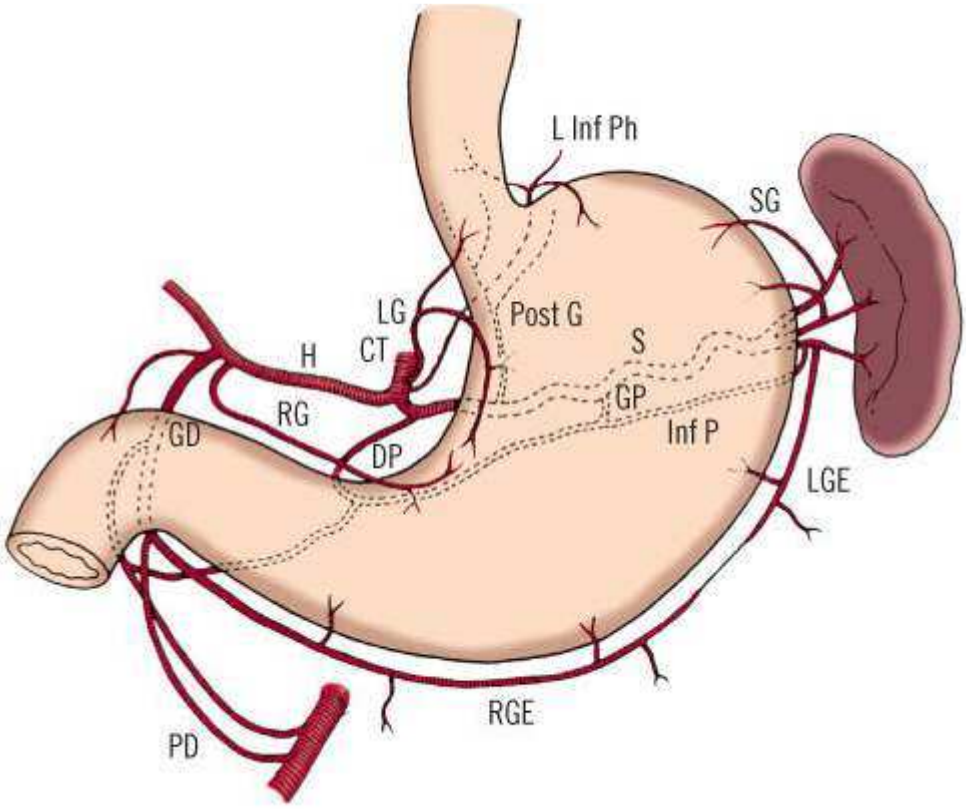
The arterial supply to the stomach comes from the coeliac trunk and its branches. Anastomoses form along the lesser curvature by the right and left gastric arteriesand along the greater curvature by the right and left gastro-omental arteries:

Right gastric – Branch of the common hepatic artery, which arises from the coeliac trunk.

Left gastric – Arises directly from the coeliac trunk.

Right gastro-omental – Terminal branch of the gastroduodenal artery, which arises from the common hepatic artery.

Left gastro-omental – Branch of the splenic artery, which arises from the coeliac trunk.



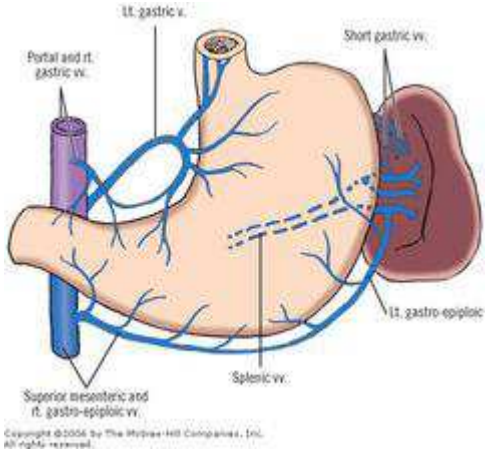

CT – COELIAC TRUNK H- HEPATIC

LG- LEFT GASTRIC GD- GASTRODUODENAL

RG- RIGHT GASTRIC DP- PANCREATICODUODENAL

S- SPLENIC RGE& LGE- RIGHT&LEFT EPIPLOIC

The veins of the stomach run parallel to the arteries. The right and left gastric veins drain into the hepatic portal vein. The short gastric vein, left and right gastro-omental veins ultimately drain into the superior mesenteric vein.

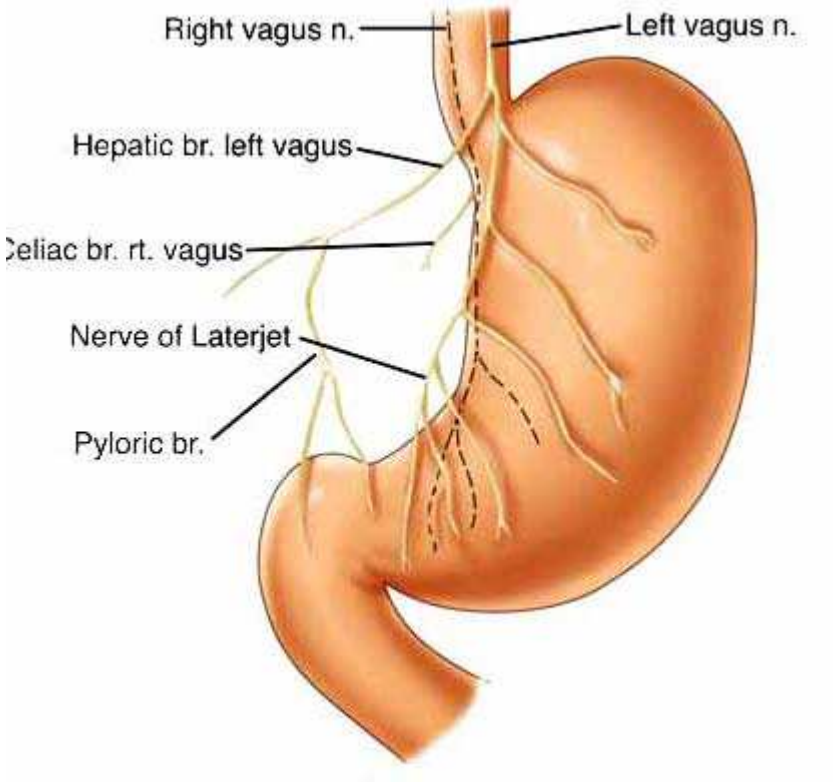



**Innervation**

The stomach receives innervation from the autonomic nervous system:

Parasympathetic nerve supply comes from the posterior vagal trunks, derived from the vagus nerve.

Sympathetic nerve supply from the T6-T9 spinal cord segments pass to the coeliac plexus. It also carries some pain transmitting fibres.



**Lymphatic drainage:**

Lymph nodes draining the stomach are numbered and divided into 4 levels, as follows:

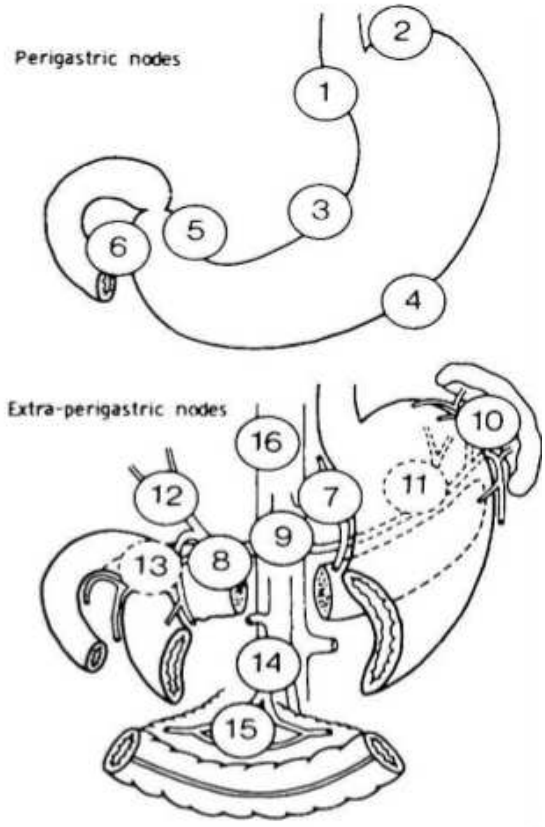
Level 1 - (perigastric lymph nodes) - Right paracardiac (1), left paracardiac (2), along lesser curvature (3) along greater curvature (4), suprapyloric (5), infrapyloric (6)

Level 2 - Along Left gastric artery (7), along Common hepatic artery (8), along celiac axis (9), at splenic hilum (10), along splenic artery (11)

Level 3 - In hepato-duodenal ligament (12), behind duodenum and pancreas head (13), at the root of small bowel mesentery (14)

Level 4 - Mesocolic (15), paraaortic (16)

| LN group | |
|---|---|
| 1 | R cardiac |
| 2 | L cardiac |
| 3 | Lesser curvature |
| 4 | Greater curvature |
| 5 | Suprapyloric |
| 6 | Infrapyloric |
| 7 | L gastric artery |
| 8 | Common hepatic artery |
| 9 | Celiac artery |
| 10 | Splenic hilar |
| 11 | Splenic artery |
| 12 | Hepatic pedicle |
| 13 | Retroduodenal |
| 14 | Mesenteric root |
| 15 | Middle colic artery |
| 16 | Paraaortic |
| 17 | Around lower oesophagus |
| 18 | Supradiaphragmatic |



## PHYSIOLOGY

The various functions of stomach are:

1. It begins the process of food breakdown exposing solid meal to proteolytic action of acid and pepsin.

2. It grinds and dilutes the mixture to form a more uniform consistent chyme.

3. It acts as a reservoir when food is stored for a period of approximately 4 hours.

## GASTRIC SECRETION[7]

The stomach secretes water and electrolytes, primarily in the form of acid and small amount of bicarbonates, enzymes such as pepsin, glycoprotein such as intrinsic factor and mucous. Gastric juice also contains small amounts of calcium, magnesium and trace amount of zinc, copper and iron.

## ACID SECRETION

Human stomach secretes about 2-5 mEq/hour of HCL in the fasting state, constituting basal acid secretion. After a mixed meal, the amount of acid secretion increases to 1 5-25mEq/hour. Acid is secreted by parietal cells situated in the glands of the fundus and body of the stomach. Regulation of acid secretion is a complex process involving endocrine, neural, paracrine and even autocrine mechanisms.

There are three phases in gastric secretions-

• Cephalic phase: Is stimulated by the sight of smell of chewing of food.

• Gastric phase: Is stimulated by the presence of food in the stomach

• Intestinal phase: Is stimulated by the presence of food in the small intestine.

**1. Cephalic phase:** Cephalic phase stimuli (sight or smell of food) presumably activate the vagal nuclei in the medulla. Impulses traverse the peripheral vagi and terminate in the gastric mucosa with the release of acetylcholine from vagal nerve endings. Release of acetylcholine in the fundic mucosa directly, stimulates and secretion by the parietal cell and release of pepsinogen by chief cells. Acetylcholine release in the antral mucosa may cause discharge of the antral hormone gastrin. Distension of stomach excites vaso-vagal reflex that also results in the release of acetylcholine in the fundic and antral mucosa.

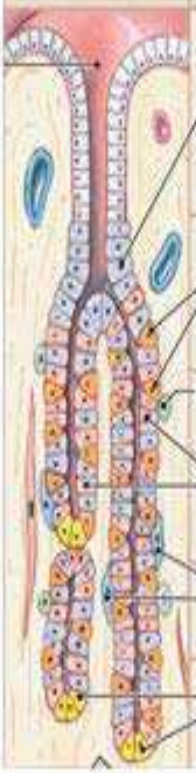
**2.Gastric phase:** This phase is initiated by the entry of food into the stomach. Food that enters the stomach buffers acid, raises pH and allows other stimuli to release acid. Through this gastrin is liberated from the gastric mucosa either due to antral distension and when the pH reaches 1.5, gastrin output is absolutely stopped. So this is a feedback mechanism in which production of gastrin is inhibited by the presence of acid in the antrum of stomach. The most remarkable action of gastrin is its ability to stimulate gastricacid secretion. It is 30 times more potent than histamine. Beside its action on acid secretion, it stimulates pepsin secretion and increases gastric mucosal blood flow. It also stimulates pancreatic enzyme secretion in man.

**3. Intestinal phase:** The intestinal phase of secretion begins as chyme begins to empty from the stomach into the duodenum. Distension of jejunum will also stimulate secretion. The cholecystokinin, the duodenal hormone which acts to stimulate secretion of pancreatic enzymes an stimulate contraction of gall bladder, also acts like gastrin.

## INHIBITION OF GASTRIC SECRETION[5]

Once cephalic stimulation is removed vagal activity is decreased. Most important is the secretion of acid itself blocks the further release of gastrin and to bring about active duodenal suppression of gastric secretion. Antral acidification has been clearly demonstrated to suppress the release of gastrin. Significant diminution in acid stimulation may occur with an antral pH as high as 5 and at pH 1.5 there is no release of gastrin.

Antral inhibition is apparently due to a passive removal of the gastricstimulus but there is clear evidence of active mechanisms of duodenal inhibition. Gastric secretion is inhibited by the presence of acid or fat or hypertonic solution in the duodenum. For a time gastric inhibitory polypeptide (GIP) appeared to be responsible for this enterogastrin like activity but recent evidence suggests that GIP is a weak inhibitor of gastric acid secretion in man and that its chief function is likely to be that of glucose dependent releaser of insulin. Whether nervous reflexes play a primary or a permissive role in duodenal inhibition has not been clarified. Acidification of duodenum inhibits gastric secretion. It also releases secretin and secretin is known to inhibit gastrin stimulated gastric secretion.

| GASTRIC MUCOSA | CELL TYPES | SUBSTANCE SECRETED | STIMULUS FOR RELEASE | FUNCTION OF SECRETION |
|---|---|---|---|---|
|  | Mucous neck cell | Mucus | Tonic secretion; with irritation of mucosa | Physical barrier between lumen and epithelium |
| | | Bicarbonate | Secreted with mucus | Buffers gastric acid to prevent damage to epithelium |
| | Parietal cells | Gastric acid (HCl) | Acetylcholine, gastrin, histamine | Activates pepsin; kills bacteria |
| | | Intrinsic factor | | Complexes with vitamin $B_{12}$ to permit absorption |
| | Enterochromaffin-like cell | Histamine | Acetylcholine, gastrin | Stimulates gastric acid secretion |
| | Chief cells | Pepsin(ogen) | Acetylcholine, acid secretion | Digests proteins |
| | | Gastric lipase | | Digests fats |
| | D cells | Somatostatin | Acid in the stomach | Inhibits gastric acid secretion |
| | G cells | Gastrin | Acetylcholine, peptides, and amino acids | Stimulates gastric acid secretion |

## DUODENUM

## ANATOMY OF DUODENUM[8]

## PARTS & RELATIONS OF DUODENUM

The duodenum is a C-shaped, first and shortest (about 10inches/25cms and most fixed part of the small intestine. It has no mesentery and thus is only partially covered with peritoneum. It extends from the pylorus to the duodenojejunal junction, making C-shaped curve, which is occupied by the head of pancreas and lies entirely above the level of umbilicus. Parts of duodenum: The duodenum is situated in the epigastric and umbilical regions and is divided into four parts.

**I. First/Superior part of the duodenum:** It is 2inches/5cms long, it begins at the pylorus and runs upwards and backwards on the right side of the first lumbar vertebra

towards liver and ends at the neck of gallbladder by bending sharply. It thus lies on the transpyloric plane. The first inch is covered with peritoneum on the front and back and can be moved with the stomach. The second inch is covered with peritoneum only above and in front.

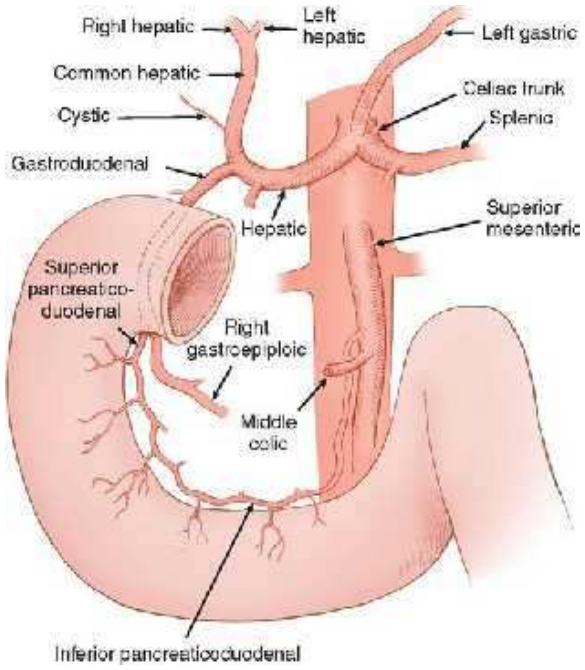
**II. Second/Descending part of the duodenum:** It is 3inches/8cms long. It runs vertically downwards in front of the hilum of the right kidney on the right side of the L2 and L3 vertebrae. It is crossed by the transverse colon. About halfway down on its medial border, the bile duct and the pancreatic duct unite to form a short dilated tube called hepatopancreatic ampulla, narrow distal end of this opens on the summit of the major duodenal papilla. The accessory pancreatic duct when present opens 2cms proximal to the major duodenal papilla as minor duodenal papilla.

**III. Third/Horizontal part of the duodenum:** It is 3inches / 8cms long. It runs horizontally and to the left on the subcostal plane and is crossed by the root of the mesentery.

**IV. Fourth/Ascending part of duodenum:** It is 2inches / 5cms long, shortest part of the duodenum. It runs upwards along the left side of the aorta on the left psoas muscle and ends about an inch to the left of the median plane at the level of the L1 vertebra. The duodenojejunal flexure is usually retroperitoneal, lies to the left of the disc of L1 & L2 vertebrae. It is fixed and held in position by the peritoneal fold called Ligament of Treitz, which is attached to the right crus of diaphragm.

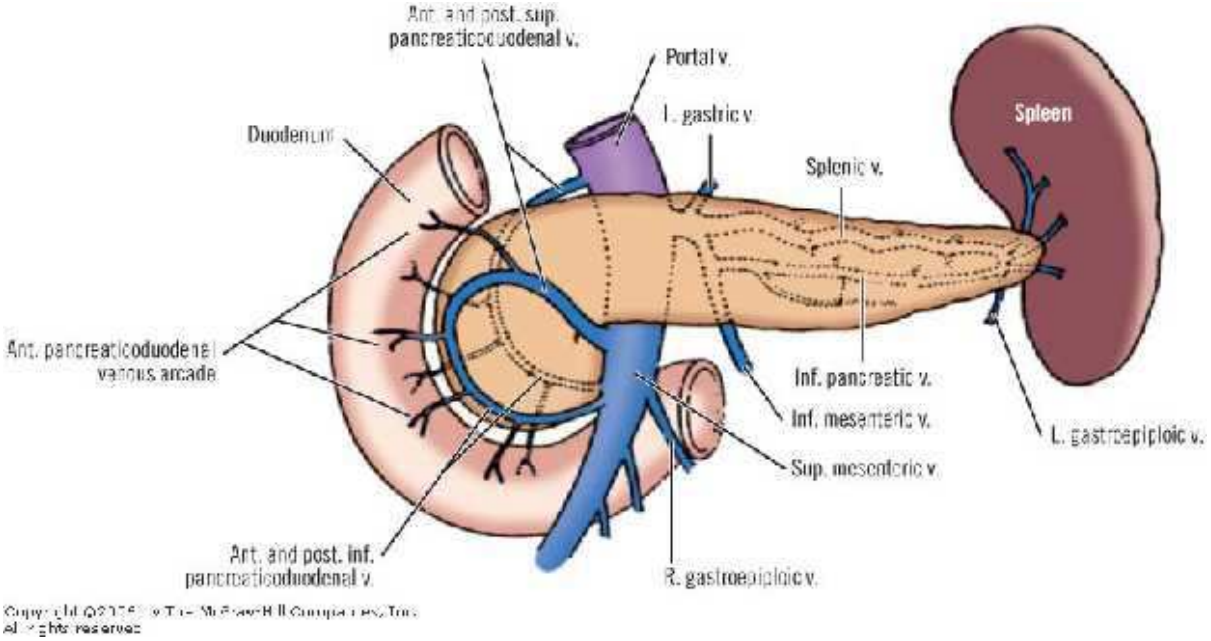
**Blood supply**

Arterial supply: The upper half of the duodenum is supplied by the superior pancreatoduodenal artery, a branch of gastroduodenal artery. The lower half is supplied by inferior pancreatoduodenal artery a branch of the superior mesenteric artery.




**Veins:** Drains to superior mesenteric and portal veins.

**Lymphatics:** The lymph vessels follow the artery and drains upward via pancreatoduodenal nodes to the gastroduodenal nodes and to the coeliac nodes: and downward via pancreatoduodenal nodes to superior mesenteric nodes.

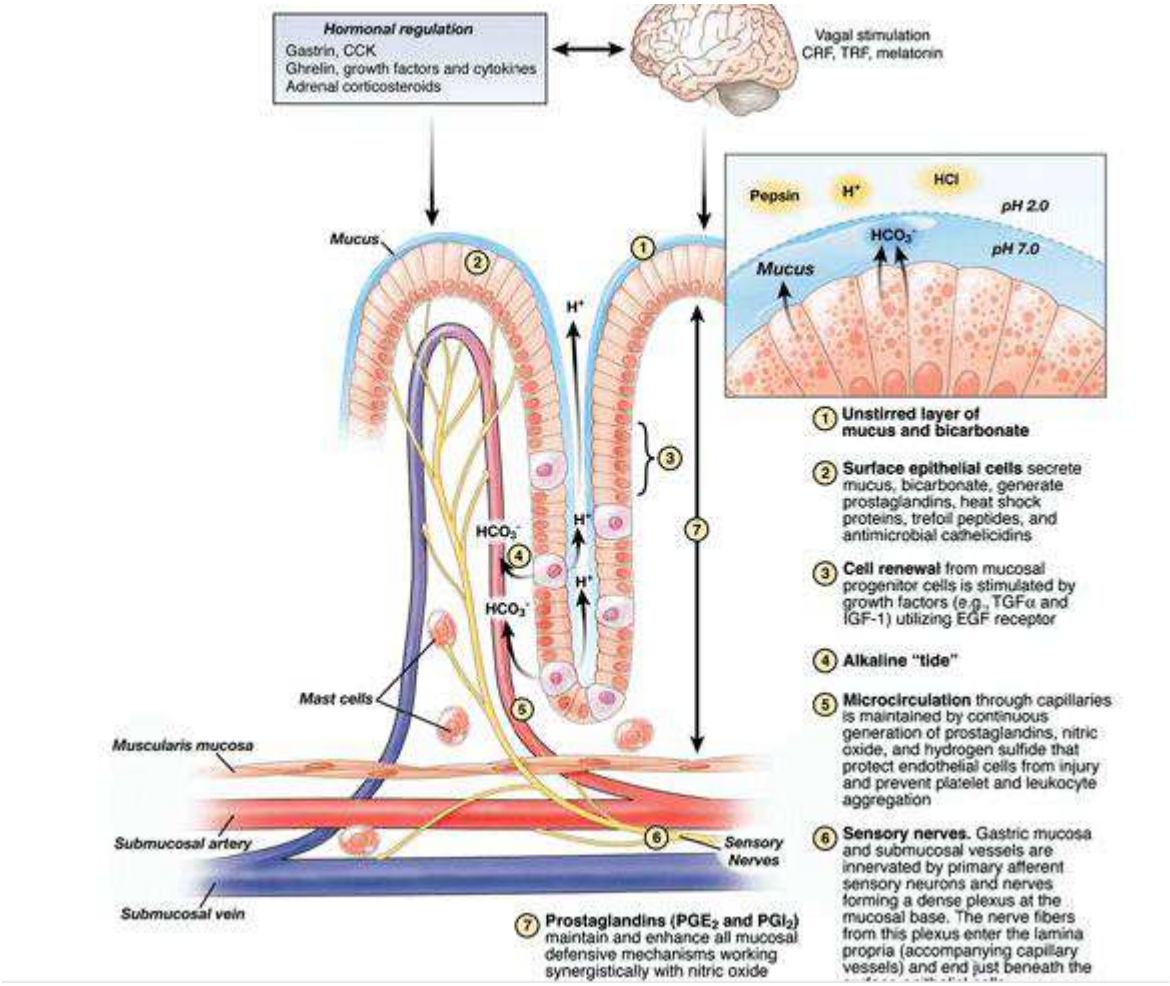



Nerve supply is derived from the sympathetic and parasympathetic (vagus) from the coeliac and superior mesenteric plexus.

**Mucosal defense system**

It is a three-level barrier system (Figure 3), composed of

- Pre epithelial,
- Epithelial and
- Sub epithelial elements




**Pre epithelial system**

It is a mucus-bicarbonate-phospholipid layer. It is the first line of defense in preventing ulcer formation. This is a physicochemical barrier to multiple molecules, including hydrogen ions protecting the mucosa. Mucus is secreted by gastroduodenal surface epithelial cells in a regulated fashion. Contents of mucus are of 95%water and a mixture of mucin a glycoprotein and phospholipids. This mucous gel acts as a nonstirred water layer which impedes diffusion of ions and molecules including

pepsin.

 Bicarbonate, secreted by surface epithelial cells of the gastroduodenal mucosa in a regulated manner. Bicarbonate is secreted into the mucous gel. Bicarbonate forms a

pH from 1 to 2 at the gastric luminal surface and reaching 6 to 7 along the epithelial cell surface.

**Epithelial barrier**

Surface epithelial cells provide the next line of defense in protecting the mucosa. They act by producing mucus, bicarbonate and epithelial cell ionic transporters and intracellular tight junctions. These ionic transporters maintain intracellular pH.

Surface epithelial cells generate heat shock proteins that prevent protein denaturation and protect cells from increased temperature, cytotoxic agents, or oxidative stress. Epithelial cells also generate trefoil factor family peptides and cathecidins, which also play a role in surface cell protection and regeneration Restitution: when the preepithelial barrier is breached, gastric epithelial cells along the site of mucosal injury can migrate and restore a damaged region.

This restitution process occurs independent of cell division. It requires

 Uninterrupted blood flow

 An alkaline pH in the surrounding environment.

 Several growth factors modulate restitution process which include epidermal growth factor (EGF), transforming growth factor (TGF), and basic fibroblast growth factor (FGF),

 Larger defects that are not effectively repaired by restitution require cell proliferation.

**Epithelial cell regeneration**

This is regulated by prostaglandins and growth factors. Growth factors are EGF and TGF-. During regeneration angiogenesis occurs within the injured micro vascular bed. Both FGF and vascular endothelial growth factor (VEGF) are important regulators of angiogenesis in the gastric mucosa.

**Sub epithelial system**

It is defense and repair system.

Key component - An elaborate microvascular system within the gastric Submucosa

**Functions**

- Provides bicarbonate to neutralize the acid generated by the parietal cell.
- Provides adequate supply of micronutrients and oxygen
- Removes toxic metabolic by products.

**Prostaglandins**

Prostaglandins play a central role in defense and repair. The gastric mucosa contains abundant levels of prostaglandins.

**Functions**

- Regulate the release of mucosal bicarbonate and mucus,
- Inhibit parietal cell secretion
- Maintains mucosal blood flow
- Epithelial cell restitution.

**Nitric oxide (NO)**

It maintains the gastric mucosal integrity. The key enzyme NO synthase is constitutively expressed in the mucosa which contributes to cytoprotection by

- Stimulating gastric mucus secretion,
- Increases mucosal blood flow and
- Maintains epithelial cell barrier function.

## GASTRO DUODENAL PERFORATIONS

### EPIDEMIOLOGY

Perforation occurs in 2-10% of patients with PUD and accounts for more than 70% of deaths associated with PUD. Often perforation is the first clinical presentation of PUD 24. The incidence of duodenal perforation is 7-10 cases/100.000 adults per year.

The perforation site usually involves anterior wall of the duodenum (60%), Antrum (20%) and Lesser-curvature gastric ulcers (20%) [12]. Gastric ulcers are associated with higher mortality and a greater morbidity than duodenal ulcers due to haemorrhage, perforation and obstruction[11] PPU used to be a disorder mainly of younger patients (predominantly males), but recently the age of PPU patients is increasing (predominantly females). Current peak age is 40-60 years[16].

The need for surgery for PPU has remained stable or even increased and the mortality following peptic ulcer perforation surgery have not decreased since the introduction of H2 receptor antagonists. The peptic ulcers are still responsible for

about 20.000-30.000 deaths per year in Europe. This may be due to an increase in use of aspirin and/ or NSAID'

**AETIOLOGY**

- Complications of peptic ulcer disease.

- Drug induced perforation

- Traumatic perforation

- Iatrogenic perforation

- Cushing ulcer perforation

- Curling's ulcer perforation

- Zollinger Ellison syndrome

- Malignant perforation: 10% of the perforations in the stomach are malignant.

**PEPTIC ULCER DISEASE**

**Background**

Peptic ulcer disease of the stomach and duodenum has undergone dramatic evolution of over the past 40 years. Overall morbidity, hospitalization and operations for peptic ulcer disease has decreased, thanks to the widespread use of gastric antisecretory agents and H.pylori eradiation.

There has been a relative increase in the incidence of peptic ulcer disease in the elderly, resulting in increased morbidity and hospitalization in that age group, the

elderly female has been the most profoundly affected largely because of use of NSAIDs in this population.

The changes in the Peptic ulcer diseases have not been confined to the west. Report from India and elsewhere support the global trend towards decreasing incidence of peptic ulcer disease.

But trends in complication of peptic ulcer disease however have not shown the same decline6. There has been no parallel decrease in cases of duodenal ulcer with complications (Perforation, Hemorrhage, obstruction) and hospitalization for complications for gastric ulcer are increasing.

## ASSOCIATION OF HELICOBACTER PYLORI AND PEPTIC ULCER

It is now found that H.pylori is present in 90% of patients with duodenal ulcer and 75% of patients with gastric ulcer. Infection appears to be acquired in the childhood and is inversely associated with socioeconomic status.

## MICROBIOLOGY

Helicobacter pylori is a gram -ve helical or curved bacillus. It is about 3 microns long and 0.5 microns in diameter. It is a fastidious, microaerophilic flagellate that has 4 to 6 lophotrichousflagellae which are composed of two types of Flagellins.2 It contains the enzyme ‘Hydrogenase’ which oxidizes Hydrogen molecules produced by intestinal bacteria.3 It is also capable of forming Biofilms.4 It also has the

capability to change into a non-culturable coccoid 5 form to offer survival advantage during adverse conditions. Helicobacter has the following culture needs

• Culture temperature of 37 degree Celsius

• Oxygen concentration of 5 to 10% ( Microaerophilic )

• Carbon di oxide concentration of 5 to 12% ( Capnophilic )

H.pylori has 5 major outer membrane proteins (OMP). The major one being the family of proteins called Adhesins. The remaining 4 OMP's include Porins, Iron transporters ,Flegellar proteins and some functionally unknown proteins.

Common to all Gram – veBacteria ,H.pylori also has a outer membrane bound Lipopolysaccharide (LPS). The O antigen on this membrane bound gycolipid can become fucosylated and resemble Lewis blood group antigen found in gastric mucosa.6 This may provide protection from immunological destruction.

The natural habitat of helicobacter pylori is human stomach. Any part of the stomach may become colonized but the mucus secreting epithelium of the antrum is the favoured site. Colonization of areas of gastric metaplasia or ectopic gastric mucosa in other parts of the gastrointestinal track. Helicobacter pylori has been detected in dental plaque by cultured. It has also been cultured from saliva of a patient with gastritis.

**Survival in gastric mucosa**

It lives beneath the mucus layer that covers the gastric mucosa. It lies deep inside the crypts of gastric glands. Spiral shape and motility makes it able to resist

peristalsis. Urease produces a protective alkaline environment around the organisms. This buffers the acid assault. Microaerophilic nature is suited for the environment and adhesions help in permanent residency. Protease produced by helicobacter pylori helps to establish itself in this stomach wall bypassing localized inflammation.

**MODE OF TRANSMISSION**

Potential mode of transmission is by three ways

**1. FAECO ORAL TRANSMISSION**

Water has been a source of Helicobacter pylori infection. The organism has been isolated from faeces. Polymerase chain reaction assays have demonstrated the presence of organism in food and drinking water.

**2. ORAL - ORAL TRANSMISSION**

Helicobacter pylori has been isolated from the oral cavity. There is evidence of transmission between spouses although it could be due to common source infection35. It is also possible that re- infection may occur by person to person transmission between spouses.

**3. GASTRO ORAL TRANSMISSION**

In children it may be due to reflux and vomiting. A physician become infected with Helicobacter pylori after he gave mouth to mouth resuscitation to a patients with positive Helicobacter pylori status who had recently vomited.

Another important source is iatrogenic transmission in individuals who have undergone endoscopy procedures with a contaminated pH electrode or biopsy forceps.

## FACTORS INFLUENCING TRANSMISSION

There are two major factors that influence the transmission of Helicobacter pylori. They are Socioeconomic status and Genetic predisposition. Low Socioeconomic status is strongly associated with infection with the bacteria. An interesting aspect is that the socioeconomic status of the individual during childhood has a strong bearing on the acquisition of infection.

The second factor influencing the transmission of bacteria is Genetic predisposition. High degree of concordance has been demonstrated in identical twins.[22]

## PATHOPHYSIOLOGY OF H.PYLORI INFECTION[23]

Helicobacter pylori is a bacteria with trophism towards the gastrointestinal tract, in particular, the stomach and the duodenum. Schwartz's dictum states "No acid-No ulcer". This epithet summarizes the thinking concerning the pathogenesis of peptic ulcer. However the recent dictum is "No H.pylori – No ulcer". 90% of duodenal ulcers and 70% of gastric ulcers are infected with Helicobacter pylori.90-100% of duodenal ulcers heal within 2 months of anti-secretory therapy. The damage to the stomach occurs due to a complex interaction between the organism and the host immune system.. It colonizes the mucosa and attaches to the epithelial surface. A myriad of mechanisms have been proposed as to how this ubiquitous bacilli cause the pathological changes with which they have been intimately linked to.

• Direct mucosal damage due to adherence of the bacteria to the epithelial surfaces

• Liberation of Vacuolating cytotoxin Vac A which causes vacuole formation within the epithelial cells , thereby leading to cellular damage

• Vac A causing a negative immunomodulatory effect causing suppression of local T cell induced immunological response leading to prolonged intense unopposed infection

• Direct stimulation of release of endogenous host inflammatory mediators such as IL-1, IL-6, IL-7, IL-12 and TNF Alpha from the mucosa

• Urease, produced by the bacteria , splits urea into ammonia in vivo. This ammonia confers local protection or so called buffer from the effects of gastric acids by causing alkalinization and also defers local attack by antibodies.

• Bacterial phospholipase caused degradation of membrane bound phospholipids leading to epithelial injury.

• Antral acidification causes stimulation of Gastrin secretion from antral G cells leading to hypergastrinemia and G cell proliferation.

The antrum is the predominant site of colonization in the stomach.[23] The pH on the surface of antral glands is well tolerated by the bacteria allowing survival and promoting growth. A subset of infected population develop “Antral-predominant gastritis” characterized by chronic inflammation of the pyloric antrum. These are the people prone to develop duodenal ulcers. With the administration of PPI’s , there is inhibition of H-K ATPase mechanism leading to decreased acidity of the antrum causing proximal migration of bacteria to corpus and fundus. This predisposes to

intestinal metaplasia of the fundic mucosa leading to increased incidence of Proximal gastric adenocarcinoma.

A second subset of individuals are prone to develop the so called "Corpuspredominant gastritis" the features of which overlap Type A Auto-Immune gastritis. It is these people with corpus predominant gastritis that are more prone to develop distal gastric adenocarcinoma. Chronic Helicobacter pylori infection has been linked to many other enteric infections such as cholera. It still remains under inquiry as to why a co-evolved bacteria would be pathogenic to humans. It is hypothesizes that an originally harmless commensal, has over time, acquired virulence genes as part of its own evolution from the host and environment. There seems to be an abundance of such laterally acquired genes that per say have no known function but can be linked to inflammatory responses within the host.

**PATHOPHYSIOLOGY OF NSAID ULCERS:[24]**

The pathophysiology of NSAID-induced injury can be grouped into two categories: those dependent on inhibition of the enzyme cyclooxygenase and those independent of cyclooxygenase inhibition. The later category comprises local mucosal toxic processes.

Topical effects of NSAIDs are likely the major mechanism responsible for the acute hemorrhages and erosions observed acutely after NSAID challenge. Within a few minutes of NSAID ingestion, denudation of surface epithelial cells and increased mucosal permeability occur. Most NSAIDs are weak organic acids that, in acidic gastric juice, are unionized and thus freely lipid soluble. The lipid-soluble, un-ionized

NSAIDs diffuse across gastric mucosal epithelial cell membranes into the cytoplasm, where they ionize at neutral pH and thus become “trapped” within the cells. The high intracellular concentrations of NSAIDs cause local toxic effects. One mechanism of these local effects is an uncoupling of oxidative phosphorylation, resulting in decreased mitochondrial energy production, a reduction in cellular integrity and increases in cellular permeability.

Another topical mechanism of NSAID injury is an attenuation of the phospholipid content and surface hydrophobicity of the gastric mucus gel layer. Some NSAID metabolites that are excreted in bile can also cause topical injury to the gastrointestinal mucosa.

The most important risk factor for an NSAID-induced complication is a history of prior peptic ulcer disease or a prior ulcer complication factors that increase the risk for NSAID-induced GI events by twofold to fourfold. Advanced age is also a substantial risk factor. Although there also appears to be a threshold age at which risk dramatically increases, the relative risk increases linearly at the rate of approximately 4% per year of advanced age. Data on the role that duration of NSAID exposure has in the risk for GI events have been conflicting.

Some case-control studies have suggested that the risk of NSAID-associated gastrointestinal complications is highest within the first 30 days of NSAID use. It has become clear from epidemiologic studies that as the dose of an NSAID increases, the risk of ulcer complications also increases in parallel fashion. Other risk factors are concomitant use of flucocorticoids or anticoagulants and comorbid conditions such as significant heart disease or rheumatoid arthritis.

NSAID use and H.pylori infection generally have been regarded as independent risk factors for peptic ulcer disease8. However, evidence is accumulating that H.Pylori infection and NSAID use may be more than just additional risk factors for ulcer disease. NSAID users infected with H.pylori have an almost twofold increased risk for developing bleeding peptic ulcers compared to that with uninfected NSAID users and low dose aspirin causes more gastric injury in H.pylori infected subjects than uninfected individuals.

**NSAIDS AND THE PERFORATION:**

The Non steroidal anti inflammatory drugs has been implicated as a treatment modality for patients of rheumatoid arthritis and osteoarthritis, which is considered as one of the important etiology for peptic ulcer and subsequently lead on to perforation. The incidence of NSAID induced perforation is more in gastric region than duodenum and the prevalence is around ten to 15% The cause of APD is increased thrice in patients who on NSAIDS than control whereas risk increases 5 fold in old aged patients of 60 years and above as the intake of drugs is more for pain and osteoarthritis. Consumption of steroidal anti inflammatory drugs have increased the incidence of perforation 6- 8 times and contribute towards a quarter of perforation patients.

Recent research has confirmed the association of NSAIDs as a cause of peptic ulcer disease, the reduction in the gastrointestinal side effect of NSAIDS can be controlled by limiting the intake of ulcerogenic drugs, counselling and prescription of anti ulcer medications (proton pump inhibitors and the use of H2 blockers), prostaglandins, and antisecretory medicines), and prescription of NSAIDs with

minimal gastrointestinal side effects to patients at risk of developing gastrointestinal complications A recent study of lumiracoxib 15 showed a three to four fold( 79 % ) reduction in ulcer complications compared with other NSAIDs in the treatment of patients with osteoarthritis.

But selective NSAIDs cost significantly more than nonselective agents. In the long term, refinement of NSAIDs and improved treatment protocols should further reduce the incidence of peptic ulcer disease and its complications. There is now more uniform agreement in recent reports concerning the incidence of nonsteroidal anti-inflammatory drugs (NSAIDs) used by patients presenting with perforated ulcers;

These vary from of 32% to 60% in those patients with perforated ulcer in whom NSAID usage was implicated as a major factor. So NSAIDS are accepted as iatrogenic cause of the peptic ulcer disease and for future perforation.

**V. CIGARETTE SMOKING :**[25]

Cigarette smoking has been mainly implicated and a strong independent risk factor in the pathogenesis of peptic ulcer disease and its complications.16 The complications implicated in cigarette smoking are due to

a) Decreases healing

b) Impairs response to healing

c) Increases complications as perforation.

But the exact mechanism is not known

Proposed mechanisms:

 Altered gastric emptying.

 Decreased bicarbonate production

 Increased H.pylori infection

 Noxious free radical production

Smokers have a three fold higher mortality from peptic ulcer than nonsmokers. The proposed mechanism in smokers is that smoking causes reduction in the blood supply to gastric mucosa due to vasoconstriction, leading on to ischemia and that ischaemia reduces mucosal resistance against, for instance, the action of acid and ulcerogenic contribute to ulcer perforation. Tobacco smoking is a well known risk factor for uncomplicated peptic ulcer. the risk of peptic ulcer progressively increased with increasing pack years cigarettes.

Silverstein[26] documented effects of the toxic constituents of cigarette smoke particularly nicotine, carbon monoxide, and hydrogen cyanide and suggested potential mechanisms by which smoking may undermine expeditious wound repair. Nicotine is a vasoconstrictor that reducesnutritional blood flow to the skin, resulting in tissue ischemia and impaired healing of injured tissue. Nicotine also increases platelet adhesiveness, raising the risk of thrombotic microvascular occlusion and tissue ischemia. In addition, proliferation of red blood cells, fibroblasts, and macrophages is reduced by nicotine. Carbon monoxide diminishes oxygen transport and metabolism, whereas hydrogen cyanide inhibits the enzyme systems necessary for oxidative metabolism and oxygen transport at the cellular level. This could also explain the toxic effects of cigarette smoking leading to perforation of gastroduodenal ulcer.

## ALCOHOL AND ULCER[27]

Alcohol contributes an important risk factor and independent risk factor for duodenal perforation. The current alcohol drinkers were at least three times increased risk of perforation as compared to nonalcoholics. Alcohol is known to impair wound healing through a variety of mechanisms: nutritional deficiencies leading to impaired wound healing and alcoholic disinhibition leads to increased risk behavior and more prone for duodenal perforation than non drinkers Chronic alcoholism is also associated with the presence of gastric metaplasia. both clinically and experimentally, alcohol had been shown to affect the mucosal barrier and histology and altering gastric mucosal defense Mechanisms. These Ulcerogenic Effects Play A Crucial Role in the study of perforations done in other parts of the world.

## EMOTIONAL STRESS:[27]

Since the recognition of the importance of H.pylori in the pathogenesis of peptic ulcer, however physician interest in the association between emotional stress and ulcer disease has wanted. Emotional stress alone does not appear to be sufficient to cause ulcers in most patients because eradication of H.pylori and elimination of NSAIDS generally prevents ulcer recurrence irrespective of emotional factors. Nevertheless, some modern studies still suggest that stress contributes to peptic ulcer disease[28].

Furthermore, it is not known why only a minority of individuals who take NSAIDS or who are infected with H.pylori develop peptic ulcers, and emotional stress and/or a genetic predisposition may well be risk factor is in these susceptible subjects

The mechanisms underlying stress as the risk factor for gastroduodenal ulcer perforation includes; Neuro-endocrine mechanism leading to a cascade of elevated levels of stress hormones, reduced inflammatory response and matrix degradation processes in early wound healing and increased vulnerability to risk behavior, hence more predisposed to peptic gastroduodenal ulcer perforation.

**DIET:**[29]

No study has established convincing link between diet and peptic ulcer disease. Ulcer patients often describe dyspepsia associated with ingestion of certain foods mostly spicy foods, but the evidence of such foods causing ulceration is virtually nonexistent, coffee, tea and colas are potent gastric acid secretagogues, but epidemiologic studies have not established an association between these beverages and peptic ulcer disease. Of note, both caffeinated and decaffeinated coffee appear to be equal in their ability to stimulate gastric acid secretion.

**Relation to Meals:**Jamioson (1944), Bean (1943), stated perforation is more 2-3hrs after meals, which may be due to over distension of stomach. Dr. S.S Hussain (1965) say perforation is more common immediately after food.

**Familial and genetic factors:** Duodenal ulcer is 2 to 3 times more prevalent in relatives of patients with ulcer, as supported by family studies and genetic marker investigations.

**Cultural and social factors:** Emotions, stress, cultural and social factors are involved in the course of peptic ulcer disease.

**Adrenocorticoids:** High dose adrenocorticoids have been implicated in some ulcerations of the mucosa of upper GIT by inhibiting regeneration of rapidly dividing cells of the gastrointestinal mucosa.

**Association with other disease:** There is thought to be an association between chronic lung disease, chronic renal failure and cirrhosis {these diseases often involve use of cigarettes, analgesics and alcohol}. Patients with rheumatoid arthritis probably have propensity for ulcer disease, which is independent of use of NSAIDs.

**Age:** Till 1940, 75% of perforations occurred in third to fifth decades. But since then there has been increasing percentage ofperforation in sixth to eighth decades. No age is exempted but rare in childhood. Perforation rarely occurs during neonatal period and early childhood. Ream (1963), reported a mortality of 57% in 39 neonatal perforations. Perforations were uncommon in adolescent. Mohammed and Mackey(1982), described 22 patients of peptic ulcer, out of which only 3 were perforations (13.6%). A study by J. Higham and colleague in Great Britain (1989-1999), shows perforations were highest in patients more than 65yrs of age**.**

**Sex:** Perforation more common in male than women. A study by J. Higham and colleagues from Great Britain (1989-1999), reported that perforation from gastric ulcer declined but perforation from duodenal increased among men at older age.Mc Kay and Mc kay (1976) reported the following male : female ratio 4.1:1

**Occupational Incidence:** The perforations are more likely to occur in those engaged in heavy manual work. Kozal and Mayer (1960) reported population incidence in 1904 perforations is as follows:

• Unskilled: 27.9%

• Semiskilled: 14.5%

• Skilled: 12.9%

• Dependents: 11.0%

Hence, perforation is highest in semiskilled or unskilled workers.

Daily Variation: Jamioson in 1944, Luer in 1949, Spence in 1950 found increased incidence of perforations in afternoons and evenings and less incidence in the nights. Hennessy in 1969 and Hendry and colleagues in 1984, suggest that there are two peaks of incidence, at the beginning of the day and in the evening, indicating that again periods of stress and strain are predisposing factors for perforations.

Pregnancy and Perforation: Wary (1945) noted increased incidence of perforation in pregnant women and presumed that hormonal basis for its cause.

**Other predisposing factors:** Upper respiratory tract infection, Fatigue, exposure to cold damp weather, worry and anxiety, alcoholism, heavy smoking and failure to maintain control of diet are some contributing factors during period of exacerbation.

## PATHOGENESIS OF PEPTIC ULCER[29]

All peptic ulceration probably arises because of an imbalance between the aggressive action of acid pepsin secretion and the normal defenses of the gastroduodenal mucosa. For duodenal ulcer, the major causal influence appears to be exposure of the duodenal mucosa to excess amount of acid and pepsin.

For gastric ulcer, the major causal influence appears to be some breakdown in the gastric mucosal defenses against acid and pepsin. The hypersecretion is related to an abnormally large total mass of parietal cells in the gastric mucosa, perhaps to either increasedresponsiveness of the parietal cells to secretory stimuli or lack of normal regulatory controls.

Increased levels of gastric or unusual sensitivity of the parietal cells to gastrin stimulation may also be involved. Individual with total achlorhydria never develops a duodenal ulcer. Defect in the defense mechanism includes deficiencies in mucosal cell removal, in mucous production in elaboration of bicarbonate and in production of prostaglandin. Irrespective of treatment, peptic ulcer takes one of the courses during the period of its progress:

• Healing

• Chronicity

• Complications

The complications of peptic ulcer are:

1. Haemorrhage

2. Perforation

3. Cicatrical contraction

4. Carcinomatous changes

**PATHOPHYSIOLOGY OF PERFORATION:**

A peptic ulcer is said to have perforated when it extends through the muscle wall and serosa of the gastro intestinal tract thereby establishing communication between the lumen and adjacent space or structure. The perforation occurs as a result of sudden sloughing of the ulcer due to impaired blood supply.

The site of pyloroduodenal perforation is usually the anterior wall and majority of the perforated gastric ulcers are located on the lesser curvature.11 Posterior perforated of a gastric ulcer may occur into the lesser sac.

Perforation leads to leakage of gastric or duodenal contents into the peritoneal cavity initiating an acute peritonitis. Although it is an initial an acute peritonitis. Although it is an initial chemical peritonitis, bacterial supervenes over the next few hours.

The presence of bacteria in the peritoneal cavity stimulates an inflow of acute inflammatory cells. The omentum and the viscera tend to localized the site of inflammation. This results in an area of localized hypoxia, which in turn facilitates growth of anaerobes and produce impairment of bactericidal activity of granulocytes. This leads to increased phagocytic activity of granulocyte, degradation of cells, hyper secretion of fluid forming the abscess, osmoticeffect, shift of more fluids into the abscess area and enlargement of the peritoneal exudates causing paralytic ileus.

Absorption of bacterial endotoxins through the inflamed peritoneal surface cause endotoxemia. The combination of fluid and electrolyte imbalance and

septicemia results in shock and multi organ failure, which is the cause of, increased mortality in untreated patients of perforative peritonitis.

## MICROBIOLGY

The microbiology of the Gastro intestinal tract changes from its proximal to its distal part. Few bacteria populate the proximal part of the bowel, where as the distal bowel contains aerobic organisms and higher percentage of anaerobic organisms. The common organisms are Escherichia coli and Bacterodies fragilis.

## Pathological course[30]

At the onset of perforation there is sudden spillage of the duodenal and gastric contents into the general peritoneal cavity and it results in chemical peritonitis.

The degree of involvement of the peritoneal cavity by bacteria is always uncertain. It is suggested that at first the visceral contents are sterile and the infective peritonitis in the early case is unlikely. However it depends on thegeneral condition of the patient and his resistence to infection.

Perforation of peptic ulcer may be classified as,

1. Acute perforation,

2. Sub acute perforation,

3. Chronic perforation,

4. Perforation associated with haemorrhage,

5. Pseudoperforation and rarely

6. Perforation of an intrathoracic gastric ulcer.

**A. Acute Perforation:** The ulcer perforatesand the general peritoneal cavity become flooded with gastric and duodenal contents, causing chemical peritonitis. The clinical features vary according to the stage of perforation, the course is divided in to3 stages of variable duration in to,

1. PRIMARY STAGE OR STAGE OF PERITONISM,

2. SECONDERY STAGE OR STAGE OFPERITONEAL REACTION and

3. TERTIARY STAGE OF BACTERIAL PERITONITIS.

**1. STAGE OF PERITONISM:** The clinical course if a perforation is generally unmistakable. At that moment the patient feels acute agoniging pain in the epigastrium or right hypochondrium which usually becomes rapidly generalized. Patient is plunged into a state of prostration and may be rendered immobile and helpless. The symptoms which arise with dramatic suddenness are due to intense irritation of peritoneum by the gastric and duodenal contents.

They produce neurogenic shock. In the early stagesnausea and vomiting are uncommon. Abdominal pain, pain referred to the both shoulders as a result of irritation of diaphragm, sub normal temperature, cold extremities, sweating, face will be pale, sweating and with expression of anxiety or fear. Patient lies almost still and rigid with his legs drawn up and his hands held tensely to his side. The temperature is sub normal and is as low as 95 – 96 degree to normal. Pulse rate is normal or raised to 90 or so. Respiration is shallow and increased, on inspection abdomen will be seen to be immobile with no movement with respiration,card board like rigidity will be

present, rigidity and tenderness are generalized. On auscultation bowel sounds will be absent and this stage lasts for 3 to 6 hrs.

Sometimes fluid creeping from the perforation may tickle down the paracolic gutter, producing signs suggestive of acute appendicitis with tenderness and rigidity limited to the right side of the abdomen.

**2. STAGE OF PERITONEAL REACTION:**

Transition from the primary stage to secondary stage takes 3 – 6 hrs which dependson the size and site of the perforation and amount of peritoneal contamination. During this stage spontaneous closure of the perforation may occur. If there is gross leakage of the gastric or duodenal contents, the patient may pass on to the stage of septic perforation. This stage rarely exceeds 6 -26 hrs. During this stage pain is reduced markedly, there would be improvement in the patients condition hence this stage sometimes called as stage of delusion.

The improvement in the wellbeing may cause the patient to delay calling medical attention and it is in this stage that most of the errors in diagnosis take place. On examination there will be varying amount of rigidity and tenderness of abdomen will be present, bowel sounds were infrequently heard or absent.

**3. STAGE OF BACTERIAL PERITONITIS:** This stage begins about 12 hrs after the perforation and lasts for about 24 hrs until it passes on to final stage of paralytic intestinal obstruction. Pathogenic organisms multiplies rapidly and peritoneal fluid becomes more purulent. Intestines slowly and pragmatically distend with gas and fluid, movements will be diminished and finally disappears with the onset of paralytic

ileus. Clinical features will be same as that occur in generalized peritonitis from any other cause. Pain is less severe, frequent vomiting and hiccoughs may trouble the patient. Sweating, vomiting and out pouring of the fluid in to the peritoneal cavity, distended paralyzed intestines, dehydration and electrolyte imbalance become more evident. Patient will complains of severe thirst, temperature will be raised, dryness of the tongue with thread pulse and shallow breathing will be present. Abdomen distended guarding and rigidity will still be present. On auscultation occasional tinkles heard. The typical Hippocratic facies denotes that end is not to for off. The face is ashen, body is cold and calamity. The patient drifts in to toxemia, dehydration and circulatory failure. Death usually takes place 4 – 5 days after perforation.

**B. SUB ACUTE PERFORATION:**

An ulcer may perforate and the perforation may rapidly before there is spillage of gastric and duodenal contents in top the peritoneal cavity. There is sudden onset of acute abdominal pain often more severe at the right upper quadrant. Respiration will be shallow and deep inspiration may be associated with an abrupt catch in the breath. On examination there is local tenderness and rigidity, but rest of the abdomen will be soft to palpate and non tender.

Unusually erect x-ray will reveal gas of small amount under diaphragm. After an hour or two, with bed rest pain will usually subside. Rarely tenderness and rigidity may extend and the signs of an acute perforation develops.

**C .CHRONIC PERFORATION:** When an ulcer perforates into the area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum

or into the omental sac, a chronic abscess may develop and will give rise to considerable confusion in diagnosis. As these patients do not present with signs and symptoms of peritonitis, they are seldom diagnosed as having perforated peptic ulcer.

Irregular temperature, rigors, leucocytosis, dullness at the base of the lung, consequent pleural effusion or basal congestion will lead to the diagnosis of subphrenic abscess. An erect x- ray abdomen may show subphrenic abscess containing gas and diaphragm is raised and fixed on the right side. USG of abdomen us the most reliable investigation on diagnosing intraperitoneal abscess.

**D. PERFORATION ASSOCIATED WITH HAEMORRHAGE:**

Perforation in association with massive haemorrhage is grave but fortunately its incidence is rare. It may present on one of the 3 ways,

¾ Haemorrhage and perforation occurring concomitantly.

¾ Haemorrhage following a recently sutured perforation.

¾ Perforation occurring during the medical treatment of haemorrhage.

The features are that of the acute perforation associated with signs of haemorrhage.

**E. PERFORATION OF AN INTRATHORACIC GASTRIC ULCER:**

This is a rare variety of perforation, where the ulcer is in hiatus hernia, which is fixed in the mediastinum. Unless existence of hiatus hernia is known it is extremely difficult to make a correct preoperative diagnosis. As the symptoms and signs point to some grave intrathoracic lesion such as coronary thrombosis, acute pericarditis and pulmonary embolism.

**F. RARE TYPE OF PERFORATEDPEPTIC ULCERS:** A peptic ulcer in a Meckel's diverticulum, in intestinal duplication which occasionally perforates. Multiple simultaneous perforations occurs in less than one percent of all cases.

**CLINICAL FEATURES**

Age: Duodenal perforation is rare before adolescence, common in 30-40 years age group.

Sex: More common in men than women.

History of Present illness :

• Time of onset: Very often the patient is able to exact the time of onset of perforation, common particularly after an exertion in the evening.

• Mode of onset: Sudden in onset, at times the patient may wake up from the sleep, due to onset of pain.

• Pain: Pain is tearing in the abdomen, intense in the epigastrium then spreads all over the abdomen.

• Shifting of pain: The pain shifts to right iliac fossa as the fluid flows along the right paracolic gutter to settle in right iliac fossa, thus mimicking appendicitis.

• Radiation of pain: Pain in peptic ulcer perforation is referred to the tip of the shoulder.

• Nausea: Present in some cases.

• Vomiting: Initially reflex vomiting occur due to irritation of nerves in the peritoneum and mesentery. In the later stages the vomiting is due to toxin action at the medullary centers and causing paralytic ileus. The vomiting then contains undigested food materials and occasionally blood when hemorrhage is present.

• Bowels: In the later stage, there may be desire to defecate due to irritation of retrovesical pouch by irritant fluid. Malaena occurs when the hemorrhage is associated with perforation.

• Micturition: Oliguria is present if patient is in shock.

**Past History**

In 80% of patients, there is a past history of dyspepsia of variable duration and in about 59% the perforation is recurrent. In the rest of the cases, the perforation may be the early clinical manifestation of a silent peptic ulcer.

**Physical Examination**

• General Appearance: In the initial shock of perforation, the face is pale livid with sweating.

• Decubitus: The patient lies in a characteristic posture of supine, rigid and immovable, refusing of any attempt to shift his postures.

• Pulse: Initially it is normal, rapid when peritonitis sets in and thready when the prognosis is grave.

• Respiration: Initially there is no change, becomes rapid and shallow when peritonitis sets in .

• Temperature: Initially normal, rises with the onset of peritonitis.

• Tongue: Usually moist, become dry and brown when the peritonitis sets in.

**Examination of Abdomen**

• Respiratory movements: Thoracic movement predominants over the abdominal movement with respiration.

• Rigidity of abdomen: Rigidity of abdomen is constant, continuous and characteristic. It is due to reflex contraction of the abdomen with predominance in the epigastrium and right hypochondrium. Rigidity is less in poor risk cases.

• Liver dullness: Obliteration of liver dullness elicited in front and in midaxillary line, is characteristic of this abdominal catastrophe in the second stage.

• Free fluid: Free fluid is present in variable degree on many acute abdominal conditions. When internal hemorrhage is excluded, fluid of appreciable amount points out the provisional diagnosis of perforation in acute abdomen.

• Rectal examination: There may be fullness in rectovesical or rectovaginal pouch

**INVESTIGATIONS**

**1. IMAGING STUDIES**

**a) X rays**

i) Erect radiographs of the chest and a plain upright radiograph of the abdomen are the most common first line of diagnostic imaging when a perforated peptic ulcer is considered.

As little as 1 ml of free air may be visualized. Free air is present in 6% to 80% of cases. In the upright view, curvilinear lucencies separate the most superior portion of the diaphragm from the liver on the right side and from the stomach and spleen on the left. An air – fluid level in the stomach should not be mistaken for free air. Usually the lateral margin of the air-fluid level can be seen extending to the lateral wall of the stomach, demarcated by serosal fat.

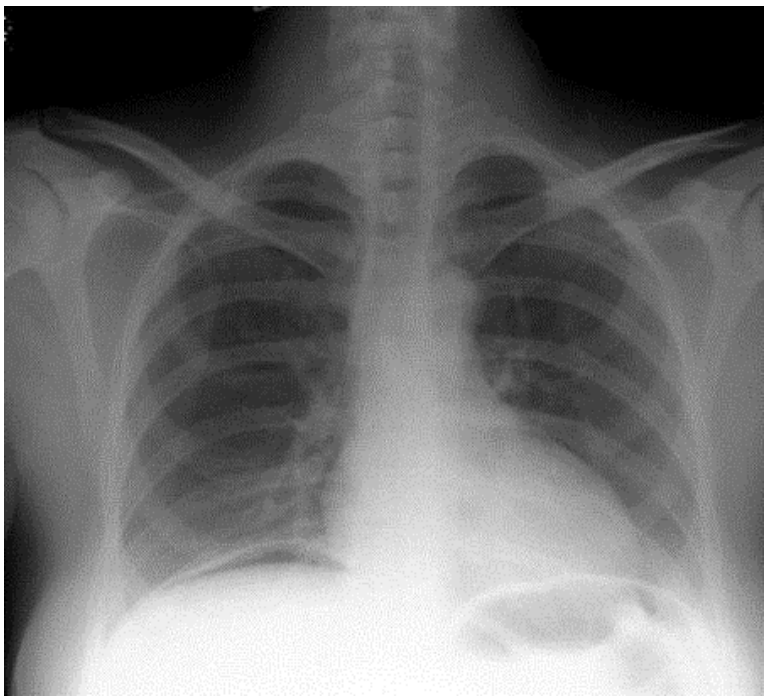
Causes of **pseudopneumoperitoneum** in a plain X- ray Abdomen are

- Chilladiti syndrome
- Sub diaphragmatic fat
- Curvilinear pulmonary collapse
- Omental fat
- Subphrenic abscess with gas forming organisms
- Subpulmonary pneumothorax
- Intramural gas in pneumatosis intestinals

ii) On the lateral decubitus view, the free air is usually best seen adjacent to the lateral margin of the liver, but in some patients the iliac portions of the peritoneum are more superior in location and free gas accumulates preferentially over the upper iliac bone.

iii) The supine view may occasionally be the only view ordered and available, especially if pneumoperitoneum is not suspected. Pneumoperitoneum can be detected in a supine view if free gas surrounds a gas-filed bowel loop. In this situation, the

inner and outer margins of bowel wall are clearly seen ( theRigler sign). Some fat may normally outline the serosal surface of bowel loops, but in the presence of pneumoperitoneum the outer surface of the bowel is sharply marginated and more distinct than fat-outlined bowel. Small amounts of air rise to the most superior portions of the abdomen and may be seen outlining the anterior margin of the liver, forming an oblique or triangular lucency superimposed over the lower portion of the liver. A linear lucency overlying the medial mid-liver may represent free air in the fissure for the ligamentum teres. If large amounts of free air are present, air may outline the falciform ligament anterior to the liver, producing the “football” sign, a large oval collection of air with a central soft tissue stripe produced by the falciform ligament outlined by surrounding gas. Air under the inferior abdominal wall may outline the umbilical fold the inverted – V sign. The Rigler sign and air collectionoverlying the liver are the most common signs of free air on a supine abdominal view.



AIR UNDER DIAPHRAGM

**B) Contrast Radiography**

i) Contrast radiography using water-soluble diatrizoate meglumine [Gastrograffin] is useful in doubtful cases. In free perforation there is leakage of contrast into the peritoneal cavity.

ii) Gastrograffin administered contrast is also useful in diagnosis of sealed perforation to plan a conservative management as in the case of formed frusta.

**C) Ultra Sonograms of the abdomen**

Localized gas collection related to bowel perforation may be detectable, particularly if it is associated with other sonographic abnormalities ( e.g. thickened bowel loop) The site of bowel perforation can be detected by sonography (e.g. gastric vs. duodenal perforation).

Ultra sonograms of the abdomen can also provide rapid evaluation of the liver, spleen, pancreas, kidneys, ovaries, adrenals and uterus, to rule out associated pathology.

**D) CT scans of the Abdomen:**

This modality can be valuable investigate tool, providing differential morphologic information not obtainable with plain radiography or ultrasonography. CT Scans may provide evidence of localized perforation (e.g., perforated duodenal ulcer) with leakage in the area of the gallbladder and right flank with or without free air being apparent.

**1. LAB STUDIES**

**Complete hemogram:**

 Parameters suggestive of infection (e.g. leukocytosis) : Leukocytosis may be absent in elderly patients.

 Elevated packed blood cell volume suggests a shift of intravascular fluid.

 Blood culture for aerobic and anaerobic organisms.

 Liver function and renal function: Findings may be within reference ranges, when no preexisting disorder is present.

**2. OTHERS TESTS**

Laparoscopy improves surgical decision making in patients with acute abdominal pain, particularly when the need for operation is uncertain.

**DIAGNOSIS OF H PYLORI:**

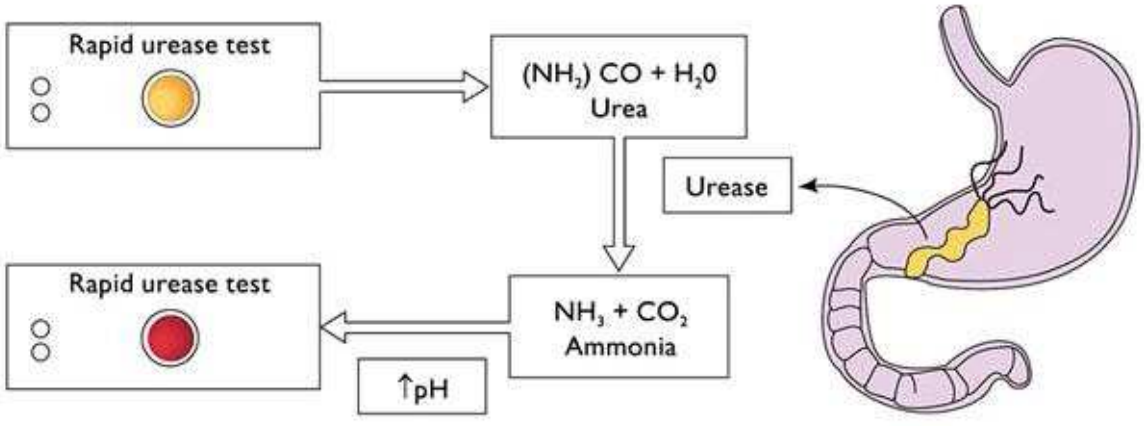
Detection of H pylori can be based on methods :

I (a) : Rapid urease test :

It is a rapid diagnostic test used for Helicobacter pylori. Mcnulty et al, first described the test. It is based on enzyme urease of Helicobacter pylori. An endoscopic biopsy is put into a solution having urea, (phenol red) a pH indicator and a gel contained bacteriostatic agent. If H. pylori are present, the bacterial urease hydrolyses the urea releases twomolecules of ammonia and one molecule of carbondioxide. It raises pH and alkalinise the medium changes yellow colour to red. The colour change

is assessed after 30 minutes and then after 2 hrs and categorized as strongly positive, moderately positive and negative.

The greatest advantage of this Rapid Urease test is, it can be done in the endoscopy room immediately after taking biopsy. Sensitivity is 90% and the specificity is 98 %.


The Rapid Urease Test. Done on biopsy samples to determine the presence of H.Pylori (Images.MD)

**1 (b) : HISTOLOGY:**

The Biopsies taken should be immediately treated in a fixative solution - Bouin's solution. 10% formaldehyde can also be used. The use of fixative is to prolong the delay before testing. By using a standard haematoxylin and eosin stain, Helicobacter pylori can be identified which appears rose colored (taylor and others – 1987) Warthin – Starry Stain, a silver stain helps in identifying small amount of bacteriapresent, but it doesn't work in case of histological tissue samples. Bacteria stains black on a yellow background.

Gentastain, has the advantage of both stains namely HE, a silver stain, and the other one is Alcian blue at pH 2.5. This stain identifies mucosal morphology, and also detects low density bacteriain specimen which has small biopsy with abundant debris

just like Warthin-Starry Stain.

This is cost effective & feasible just like all other silver stain with good detection rate. Sensitivity is > 99% vs 85% with H-E stain

**1(c) : CULTURE :**

H. pylori adheres to the gastric mucosa and is not recovered in stool or blood specimens. Bacteria can be isolated in culture if the specimen is inoculated onto enriched medium supplemented with blood, hemin or charcoal & incubated in a microaerophilic atmosphere for upto 2 wks. Viable bacteria are detected and antibiotic sensitivity can be obtained. Takes several days and results are dependent on the expertise of the operator and the lab. Expensive and unnecessary unless antibiotic sensitivity are required

**1 (d) : POLYMERASE CHAIN REACTION :**

This method is used to detect Helicobacter pylori in the fecal samples. PCR is more sensitive compared to all culture as it does not depend on bacterial viability. But the limiting factors are inhibitors of amplification reaction in feces.

**2 (a) : SEROLOGY :**

Antibody measurement of antibodies : Antibodies against Helicobacter pylori are present in infected patients and the detection of such antibodies found in blood, saliva, which are 95% sensitive and specific for H. pylori. It is also cost effective, feasible, and time limiting. They don't give false negative even in patients on Proton Pump Inhibitors, Bismuth and Antibiotics. (NIH Consensus Conference, 1994).

Helicobacter pylori has wide variations of Antigenic strains for antibody manufacture. IgG and IgA antibodies found in blood are Helicobacter pylori specific. Compared to other methods used for testing like histology and cultures, IgG and IgA assays have higher sensitivity and specificity and so these methods are widely

preferred.

The assays in practice are 1) Micro-titre plate assay 2) Near patient testing devices. These assays have a specific cut off value and a control sera to differentiate between infected and non infected. These two assays have high potential compared to the standard used techniques. Helicobacter pylori antibodies found in saliva and serum are equally effective. IgG immunoglobulin presents in the saliva is measured, rather than IgA which could not differentiate the infected from non infected.

Antibody tests are non invasive and cost effective. Near patient version can be performed in few minutes with the blood obtained from finger prick. Some researchers commented that serology testings are the gold standard techniques for detecting Helicobacter pylori infection (Blaser 1990).

Patient whose gastric biopsies positive for Helicobacter pyori, are found to be serology positive. Culture and histologic biopsy reveals infection in a particular inflammatory site of stomach, but serological assays covers the entire stomach.

**Quantity of antibody tests:**

Recently researchers (Nomura and colleagues), have diagnosed that raised level of circulating antibodies are present in Helicobacter pylori infected patients. The viability of a screening test is increased if these test can assess the circulating concentration of antibodies against Helicobacter pylori which could differentiate between peptic ulcer disease and the gastric cancer, thereby increasing the diagnostic significance.

**Antibody testing after H pylori eradication:**

After the eradication of Helicobacter pylori, the serum IgG and IgA levels falls very slowly. After 6 weeks of eradication, titres falls by 20-30% and after six months titres falls by 50% in 97% of patients. Since there is a slow fall in the antibody titre, it is not commonly used to assess the success of treatment. Thus the success of treatment is assessed by repeat endoscopy, histology and culture and urea breath test. One study showed that antibody titre in saliva specific to Helicobacter pylori were found to be reduced more fastly. More than 50% patient have shown 83% fall in antibody titre of

saliva with treatment after one month. Thus it is used as standard methods to monitoring Helicobacter pylori eradication. ELISA is the best method for serology because of its simplicity, reliability, and low cost. Seroconversion takes 22-23 days after the infection.

Sensitivity and Specificity of ELISA is over 90%. Immunoblotting is a qualitative serological test used to detect antibodies. This technique includes denaturing of bacterial antigen which were separated by electrophoresis and then put innitrocellulose membrane where it enables a contact with the serum which is going to be tested. This test concludes an immunological humoural response from an infected person rather to assess the variability of antigenic strain.

**2(b) : UREA BREATH TEST:**

C13 is the most commonly used isotope which occurs by nature and an isotope of non radioactive origin. Bacterial urease activity is the basis for breath tests. Urea along with enriched C 13, is then hydrolysed in the stomach by the enzyme urease secreted from H. pylori to produce two molecules of ammonia and one molecule of carbondioxide. As it comes to intestine the gas succumbed into the blood and is excreted via lungs as exhaled air. Breath samples are collected before the administration of C 13 CO2 is then calculated using an isotope ratio mass spectrometer [IRMS].

C 14 is also used but involves exposure to a small amount of radioactivity and must be used with caution in pregnant patients and children. C -14 is measured using a beta counter. The subjects are advised to be in a fasting state before conducting the

test. After a test meal, the subjects are instructed torinse their mouth and be seated for 30 minutes duration of the test. The breath collection is usually done by making the patient to blow into a tube with straw. The analysis is performed by mass spectrometry / beta counter.

## DIFFERENTIAL DIAGNOSIS

### Acute Medical Conditions

1. Pleurisy:

2. Acute Pericarditis.

3. Gastric crisis or Tabes dorsalis.

4. Lobar pneumonia.

5. Acute alcoholism

6. Coronary thrombosis

### Acute Surgical Conditions

1. Acute exacerbation of duodenal ulcer

2. Acute Pancreatitis.

3. Acute gastritis

4. Peritonitis from Acute appendicitis

5. Acute intestinal obstruction

6. Biliary colic in acute cholecystitis

7. Raptured aortic aneurysm

8. Raptured ectopic gestation

9. Perforated typhoid ulcer

**TREATMENT**

The perforation of gastroduodenal ulcer is common problem, the most important and immediate step in the management is adequate resuscitation of patient on admission.

Two methods of treatment are practiced:

1. Non-surgical or conservative management,

2. Surgical management.

**1. Open surgery**

A. Simple closure of perforation.

B. Closure of perforation with definitive surgery.

• Truncal vagotomy with gastrojejunostomy.

• Antrectomy with vagotomy.

• Pyloroplasty with vagotomy.

• Partial gastrectomy with vagotomy.

• Highly selective vagotomy.

**II. Laparoscopic surgery**

Immediate management (Resuscitation)

1. If patient is in shock, elevate foot end of the bed, start intravenous fluids immediately with Dextran or Saline/Plasma/expanders.

2. A nasogastric tube is passed and the contents of stomach aspirated and Repeated Every Half An Hour

To prevent further soiling of peritoneum

To prevent aspiration of gastric content into lungs

To decompress the stomach.

3. Bladder catheterization done in all patients to monitor urinary output.

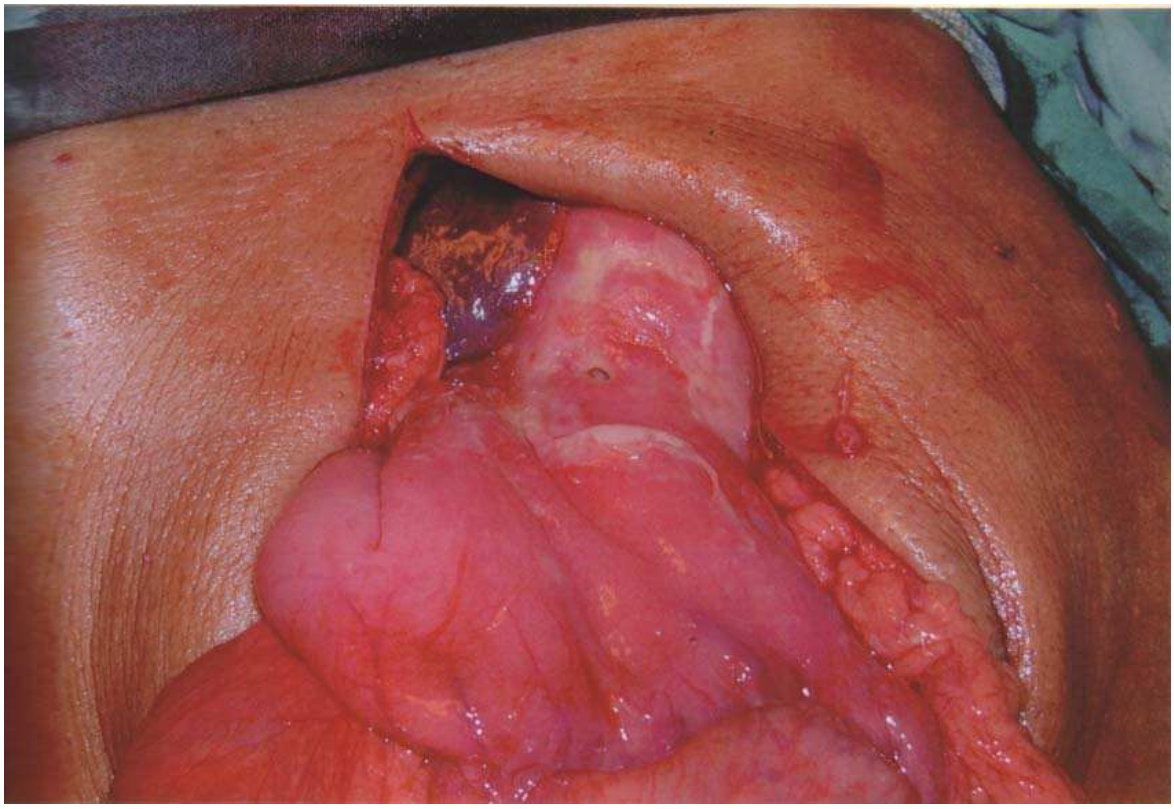
4. Blood sample collected for grouping and cross matching, complete haemogram, blood urea, serum creatinine, and serum electrolyte study.

5. Blood pressure and pulse rate and urinary output should be recorded at an half hourly intervals.

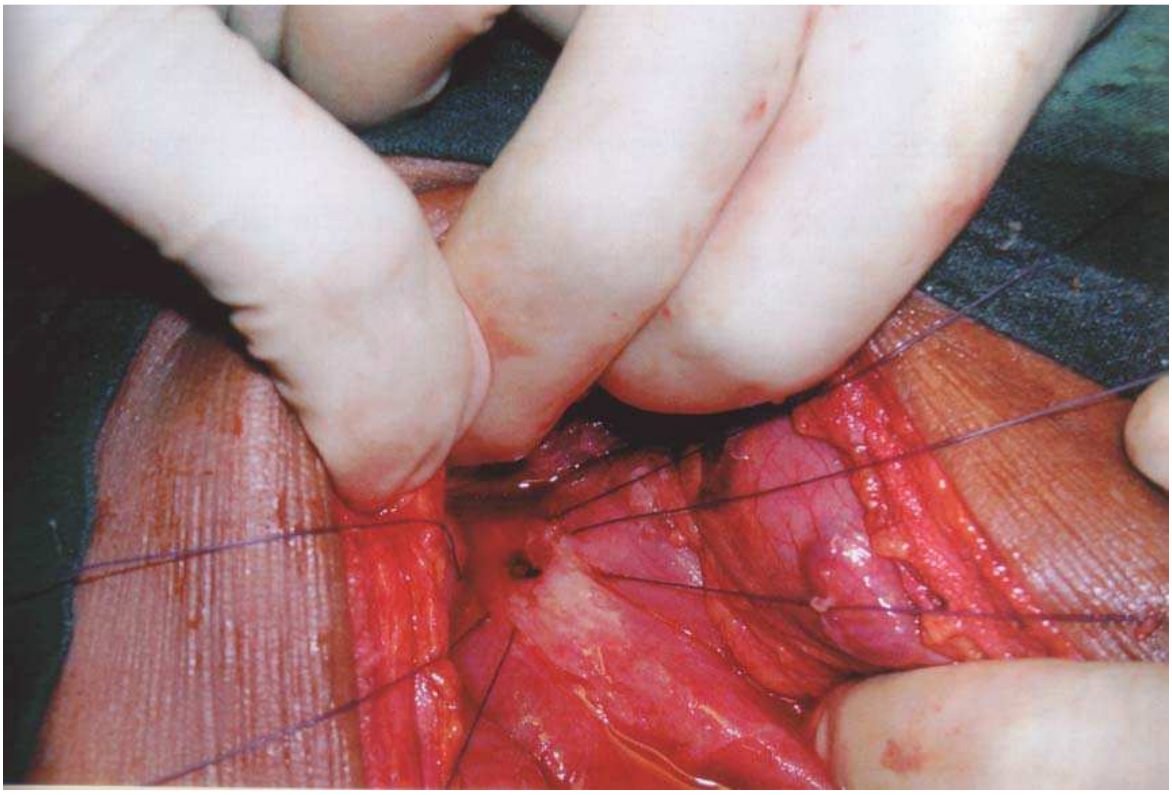
6. Appropriate antibiotics (broad spectrum) should be given. Third generation cephalosporin and metranidazole are preferred.

7. Preparation of abdomen to be done

**OPERATIVE MANAGEMENT**



GASTRIC PERFORATION



DUODENAL FIRST PART PERFORATION

Perforation of duodenal ulcer is usually treated by surgery.

**Simple closure**[31]

After adequate resuscitation patient can be posted for surgery. Anaesthesia: General anaesthesia, by IV thiopentone and Scolene, endotracheal intubation and maintenance by N2 and O2.

Procedure: Patient in supine position, abdomen opened with upper paramedian or upper midline incision. Bailey points out that in 10% of the cases, muffled pop of escaping gas can be heard on opening peritoneum. The free fluid is sucked and mopped with moist packs. The stomach is held near the greater curvature with a moist pack and search for perforation. The anterior part of the duodenum and distal stomach are inspected first, usually the perforation is found on the anterior surface of first part of duodenum. It is closed with interrupted suture using absorbable suture Material. After peritoneal toilet, two drains are placed, one in the right side near the perforation site and the other one in pelvis. The abdomen incision is then closed in layers.

**Methods of closure of perforation**

**1. Simple closure:**

• Indicated for small ulcers with little induration and healthy tissue around. - Converts ulcer into a linear scar

• More rapid healing.

• Early remission of symptoms.

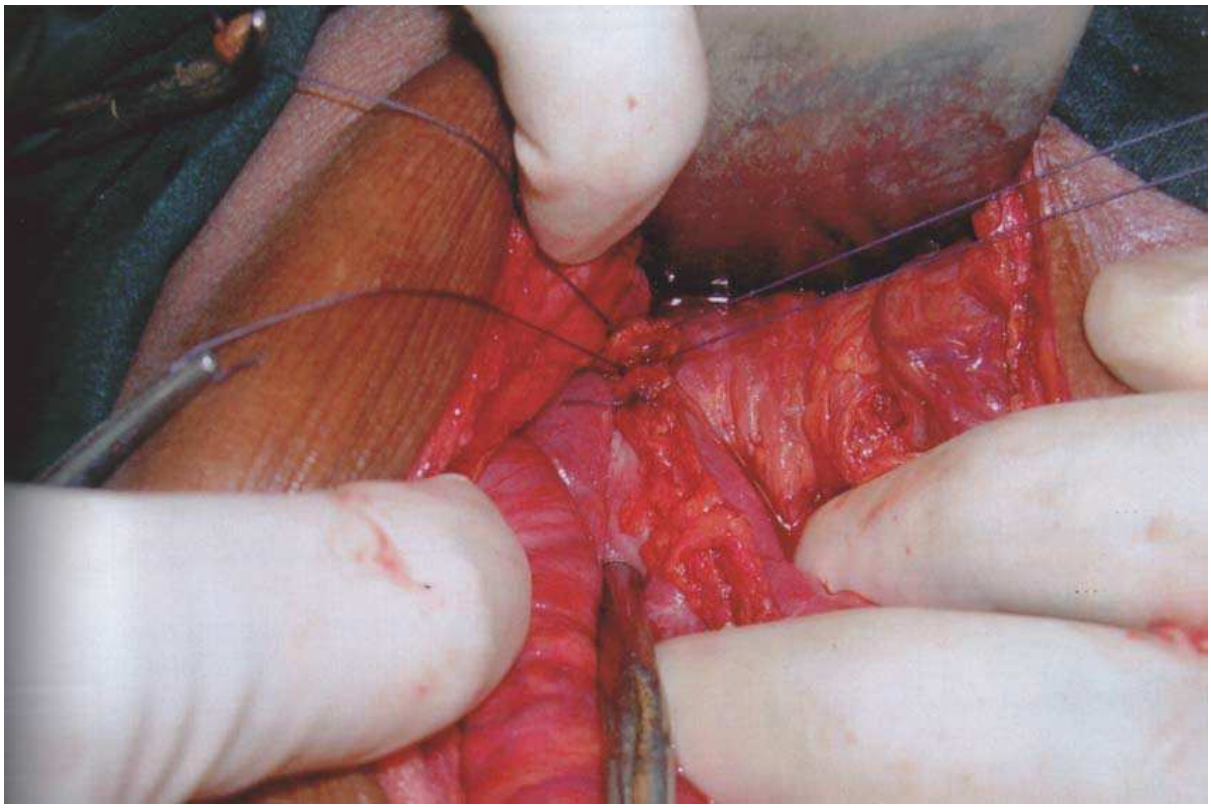
• Absorbable / delayed absorbable /- Purse string suture to be avoided.

• Inversion / eversion to be avoided.

• Suture should be applied in the long axis of the gut to avoid narrowing of lumen.

• Omentum is used for reinforcing the perforation.

2. Cellan Jones technique, Graham technique: Here free omental flap is used. Greater omentum is placed over the perforation and perforation is sealed by omentum.



LIVE OMENTAL PATCH CLOSURE

3. Dragging the omentum into perforation and plugging it into the Ryles tube.

4. Use of rectus muscle to seal the perforation.

## DEFINITIVE SURGERY[33]

This has been advocated as it has been found that patients treated with simple closure have a severe relapse of the disease in >50% of cases in 5 years follow-up.

Indications for definitive surgery:

**1. Definite indications:**

• Coexistent of perforation and obstruction.

• Previous operation for perforated duodenal ulcer.

• Perforated gastric ulcer with suspicion of malignancy.

• Coexistence of haemorrhage and perforation.

• Perforation of ulcer during medical treatment.

• Combined gastric and duodenal ulcer, one of which has perforated.

**2. Relative indications:**

• Young patient less than 45yrs.

• Smoker.

• Absence of purulent peritonitis.

• If patient has reported before 8hrs after perforation.

• Minimal peritoneal soiling.

**Contraindications for definitive surgery:**

• More than 24hrs of presentation.

• Poor risk patient.

• Concurrent medical illness

**Advantages of definitive surgery:**

• Reperforation is avoided.

• Second operation is avoided.

Gastric stasis after simple closure is avoided. – Postoperative pyloric stenosis of obstruction due to inflammation oedema is avoided. -

In haemorrhage with ulcer perforation, - haemorrhage is cured.

**Disadvantages of definitive surgery:**

• It is more operative trauma to patient.

• It may be unnecessary in 10-15% of patient.

**Types of definitive surgery**

1. Truncal vagotomy with gastrojejunostomy.

2. Antrectomy with vagotomy.

3. Pyloroplasty with vagotomy.

4. Partial gastrectomy with vagotomy.

5. Closure with highly selective vagotomy.

**1. Truncal vagotomy with gastrojejunostomy:**

In this, after simple closure of perforation, peritoneum over the oesophagus is opened and left lobe of liver is mobilized detaching its coronary ligament and bilateral truncal vagotomy is done. Truncal vagotomy is always performed with a drainage procedure, because of rise of gastric stasis and delayed gastric emptying. The posterior surface of stomach is brought into infracolic compartment by opening the transverse mesocolon. Short loop of jejunum from duodenojejunal flexure is taken and approximated with the stomach and a classical GJ is performed. Rent in the mesocolon closed in layers.

**2. Antrectomy with vagotomy:**

Here, the distal half of the stomach is resected with Billroth II anastamosis.

**3. Pyloroplasty with vagotomy:**

Here, after excising perforation, Heineke, Mikulicz or Finney type of phloroplasty is performed and with vagotomy.

**4. Partial gastrectomy with vagotomy:**

When perforation is in lower gastric area, this procedure is done, followed with Billroth I anastomosis and vagotomy.

**5. Highly selective vagotomy:**[34]

The objective of highly selective vagotomy is to denervate the parietal cell mass while preserving the vagal supply to the antrum, thereby avoiding drainage procedure. Preserving the anterior and posterior nerves of Laterjet does this, which

provide the motor innervation to antrum. This procedure is done in case of perforation associated with haemorrhage.

In case of difficult closure of duodenal stump, the duodenal stoma can be placed either laterally or terminally. Tube duodenostomy using Kehrs “T” tube or end duodenostomy using Foleys catheter.

## LAPROSCOPIC CLOSURE OF PERFORATION

Recent development in minimal invasive surgery now allows laparoscopic approach to the patient with perforated duodenal ulcer. The perforation can be approached using three additional ports. The perforation can be dealt by: - Fibrin glue for minute perforation. Simple closure with omental patch and copious irrigation of the abdominal cavity.

Automatic staples suture can be applied via laparoscope. A proximal gastric vagotomy or Taylor procedure (anterior seromyotomy and - truncal vagotomy) may be performed.

## POSTOPERATIVE MANAGEMENT[36]

• Nil by mouth till the intestinal sounds is regained.

• Nasogastric tube aspiration till it becomes less and bowel sounds - recover.

• Antibiotic covering anaerobic and aerobic organisms.

• Intravenous fluid therapy.

• H2 blockers.

• Chest care.

• Drainage tube care.

• Watch for any evidence of intra-abdominal collection in case of postoperative fever.

**POSTOPERATIVE COMPLICATIONS**[36]

• Pulmonary complications like atelectasis, pneumonia.

• Residual abscesses like subphrenic abscess, pelvic abscess.

• Peritonitis.

• Paralytic ileus.

• Early reperforation and leak, duodenal fistula.

• Deep vein thrombosis and pulmonary embolism.

• Renal failure.

• Mediastinitis.

**Management of early perforation and leak**[37]

• Priority towards fluid and electrolyte management. – Nasogastric aspiration.

• H2 blockers.

• Antibiotics.

• Nutrition of the patient, ideally total parental nutrition (TPN) or feeding jejunostomy.

Causes of leak are:

• Old patients.

• Large perforation.

• Inadequate closure.

• Difficulty closure with friable margin.

• Late presentation to hospital after perforation.

Leaks are usually seen from 2nd to 5th postoperative day presenting as bilious drain from the drain site.Fistula may be high or lowoutput. In case of high output fistula, where TPN facilities are available, patients can be managed conservatively, feeding jejunostomy can be added for enteral feeds. Trial can be given for 3weeks after which operative can be adopted. Where TPN facilities are not available, ideally after resuscitation patient can be taken up to surgery at an early stage to prevent further deterioration. In low output fistula, conservative management is usually adopted.

**Surgical management**

• Feeding jejunostomy.

• Use of serosal patch (Kobold &Thal) technique: The upper jejunum is serosa used as a loop or Roux en y loop to occlude the perforation.

• Partial gastrectomy: In large perforation with friable margins where repair cannot be done, it is ideal to proceed with partial gastrectomy with polya anastomosis.

• **Conservative or non-operative management:** By passing Foley s catheter into the drain wound and manipulating it into or near to the perforation can be converted into a controlled fistula.H2 Blocker antagonist: These drugs act by selectively blocking H2 receptors of parietal cells. They have a dose dependent antisecretory potency. Their simple dosage schedules and associated good therapeutic compliance.

**Drugs:** Cimetidine: No more used.

Ranitidine: 150mg Bid, after 4-6wks OD at night.

Famotidine: 20-40mg OD.

Roxatidine: 75mg OD.

Nizatidine: 20mg Bd.

**Proton pump inhibitors:**[40] These act by inhibiting the H+ / K+ ATP system on the luminal side of the parietal cells. Action is long lasting and dose dependent. In dose of 20-40mg OD, it achieves almost 100% inhibition of intragastric acidity throughout day and night. Dosage: 20-40mg OD for 4-6weeks,followed by 10-20mg OD.

**TREATMENT OF H.PYLORI**[38]

The aim of treatment is to eradicate Helicobacter pylori. Eradication is defined as negative tests for Helicobacter pylori atleast 28 days after the end of antimicrobial therapy. Ideal therapy for Helicobacter pylori should be

 Simple

 Safe

 free from side effects

 efficacy is 100%

 low cost

 acceptable to the patient

 available to the patient

Therapies available

**1. Dual drug therapy**

• Proton pump inhibitor + clarithromycin/ amoxycillin.

• Ranitidine + clarithromycin for 14 days. Not recommended .

**2. Triple drug therapy**

• Omeprazole 40mg OD + Clarithromycin 500mg BID +Metranidazole 400mg BID for7days.

• Omeprazole 40mg OD + Amoxicillin 500mgBID +Clarithromycin 500mg BID for 7 day .

• Omeprazole 40mg OD + Amoxicillin500mg BID +Metranidazole 400mg BID for7–10days.

• Colloidal Bismuth Subcitrate 125mg QID for 14 days +Amoxicillin500mg BID + metranidazole 400mg BID

**3. Quadruple drug therapy**

Omeprazole 40mg OD + Collidal Bismuth Subcitrate 125mg QID 4OD +Tetracycline 500mg TID + Metranidazole 400mg TID for 7days.

The Clarithromycin based regimens are much costlier than Amoxicillin based regimens.

**First line therapy:**

First-line therapy has been used for H. pylori eradication in many parts of the world. It consists of a triple therapy using a Proton pump inhibitor or ranitidine bismuth citrate, combined with clarithromycin and amoxicillin or metronidazole for those individuals with penicillin allergy. All are given twice daily. However, even with correct use of these combinations, infection is not eradicated in 10-23% of patients. The recommended duration of treatment range is between 7 and 14 days. The emergence of drug resistance and decreasing drug efficacy, has made the second-line therapy necessary.

**Second line therapy:**

H. pylori may acquire resistance by acquisition of mutations and recombination of genes from other bacteria. Chromosomic point mutations can also induce resistance. Metronidazole targets DNA and has a high mutation rate.. Clarithromycin and Metronidazole are two antibiotics noted for resistance and most of H. pylori isolates after two eradication failures are found to be resistant to the two drugs mentioned above. Subsequently, quadruple therapy which consists of PPI, bismuth, metronidazole and tetracycline is a recommended alternative to first-linetreatment,

which are used in areas of high antibiotic resistance. Where bismuth is not available, second-line therapy may be with PPI-based triple therapy.

**Third line therapy or salvage therapy:**

This is given after multiple (at least two) treatment failures with different regimens. Basically, it would be chosen based on the results of antimicrobial susceptibility testing. Often, a careful review of agents used previously will enable a regimen to be identified that will be successful. It was found that most of H. pylori isolates after two eradication failures are resistant to metronidazole and clarithromycin. Therefore, it is recommended that these two drugs should be removed from the third-line therapy. These third-line therapies are the new emerging therapies.

Levofloxacin based, Rifabutin and Rifampicin based, Furazolidone based, Doxycycline based and Lactoferrin based therapies are under study.

**NON-OPERATIVE OR CONSERVATIVE MANAGEMENT[38]**

In majority of patients surgery remains treatment of choice. In certain situations, conservative management should be considered.

**Indications** for conservative management:

1. Where general condition of the patient is bad, risk of general anaesthesia is considered too great.

2. Lack of surgical facilities.

3. Clinical signs suggesting only of minimal spillage with sealed perforation which has been shown by gastrograffin radiograph.

**Contraindications** for conservative management:

1. Perforation in presence of steroids, which would diminish the patients ability to heal the ulcer spontaneously.

2. Gastric ulcer.

3. Patients who have continued leakage on gastrograffin radiograph.

4. Patients who perforate while on active antacid therapy.

5. Uncertain diagnosis.

Conservative management consists of:

• Nil by mouth

• Continued nasogastric aspiration

• Intravenous fluids

• Intravenous H2 receptor antagonist

• Appropriate antibiotics

• Appropriate sedative.

If the distension of abdomen increases with the condition of deteriorating, then the flank drain (bilateral) may be placed under local anesthesia, to drain the fluid.

**Advantages** of conservative management:

• Operation can be avoided.

• A percentage of patients do not need any further definitive operation, in such patients, unnecessary operation can be avoided.

• In few patients, when perforation will get sealed off and such patients would be benefited.

**Disadvantages** of conservative management:

• The site of perforation usually remains in doubt.

• The nature of underlying condition (benign or malignant) remains uncertain.

• Recurrence of ulcer symptoms (Illingworth, 1994)

• Recurrence of perforation.

• Risk of detoriation.

**COMPLICATIONS:**[40]

**EARLY COMPLICATIONS:**

 Renal failure and fluid, electrolyte, and pH imbalance.

 Respiratory complications.

 Wound infection:

i. Wound infection rates correlate with the bacterial load in the peritoneal fluid.

ii. The judicious use of prophylactic antibiotics has been demonstrated to reduce the incidence of wound infection in contaminated and potentially contaminated wounds.

 Wound failure

 Wound failure (partial or total disruption of any or all layers of the operative wound) may occur early (i.e., wound dehiscence)

 The factors are associated with wound failure are malnutrition, sepsis, uremia, diabetes mellitus, corticosteroid therapy, obesity, heavy coughing, hematoma (with or without infection).

 Multiorgan failure and septic shock.

i. Septicemia is defined as proliferation of bacteria in the bloodstream resulting in systemic manifestations such as rigors, fever, hypothermia (in gram negative septicemia with end toxemia), leukocytosis or leucopenia (in profound septicemia), tachycardia and circulatory collapse.

ii. Septic shock is associated with loss of vasomotor tone, increased capillary permeability, myocardial depression, consumption of WBCs and platelets, dissemination of powerful vasoactive substances, such as histamine, serotonin, and prostaglandins, resulting in capillary permeability, complement activation and damage of capillary endothelium.

 Gram- negative infections are associated with a much worse prognosis than gram-positive infections, possibly because of associated endotoxemia.

 Endotoxemia.

 Localized abdominal abscess

 Enterocutaneous, fistula

 Deep vein thrombosis and pulmonary embolism.

## LATE COMPICATIONS:[41]

 Mechanical intestinal obstruction: Medical obstruction of the intestine is most often caused by postoperative adhesions.

 Incisional Hernia

## PROGNOSIS[42]

Age:Mortality increases with increasing age (Illingworth, 1994). General condition of patient: Poor general condition of the patient carries high mortality.

Presentation with shock: (Systolic BP <90 mmHg) Patients presenting with shock have a high mortality rate. Boey John et al revealed concurrent medical illness, preoperative shock and delayed operation (>48hours) as significant risk factors that increase mortality in patients with perforated duodenal ulcers (1982).

Presentation with renal failure: May be hypovolaemic/septicemic origin. Oliguria carries high mortality rate.

Ulcer history: Patient with long ulcer history have shown to carry high risk and concomitant bleeding and perforation also carried high mortality rate.

Concomitant medical illness: Concomitant cardiac, pulmonary, renal and other diseases in association with perforation carry high mortality rate. Duration of perforation: If the duration is longer, higher is the mortality. In the presence of gross contamination, late exploration (after 48 hours) carried a high mortality i.e. 50% (Arthue A. Cicola et al, 1999).

The importance of the peritoneal soilage and duration of perforation is mentioned as a risk in the outcome of the perforation of duodenal ulcer (Donaldson, 2000). Bharati C Ramesh et al reported that 12% of patients reached the hospital within 12 hours, 40% reached hospital within 25-48 hours and 24% after 48 hours (IJS, 2006). Brazynski M et al reported that 48.15% patients to hospital after 24 hours of perforation (1999).

Size of perforation: In duodenal ulcer, the size varies from 3mm to 1cm in diameter. Larger the perforation and older the patient, the higher the morbidity and mortality. Number of perforation: Perforation is always single, but there are reports of more than one perforation. More than one perforation, then higher the morbidity and mortality. Nature of gastric contents, kinds of microorganism which predominant:

**(Bacteriology)**

The peritonitis resulting from perforation of peptic ulcer is at first often non-infectious and is due to instant action of the gastric and duodenal ulcer contents and thus the infective peritonitis in early case is unlikely. However, it depends on the general condition of the patient and the resistance to infection. Grarco and Chawo in 1974 and Boey and colleagues in 1982 found that more than half the cultures of peritoneal fluid taken at the time of operation were sterile. The preoperative use of antibiotics may play a considerable part in these findings. It appears bacterial peritonitis is seen only in grossly neglected cases.

## OTHER GASTRO DUODENAL PERFORATIONS TRAUMATIC PERFORATION OF STOMACH

Penetrating injuries are more commonly associated with perforation of stomach than blunt injury24. They may be caused due to stab injury with sharp knife or high velocity bullets. In most of these cases, extent of damage is difficult to assess clinically due to alteration in the laminar layers of abdominal wall.Nasogastric aspiration may be frank blood, X-ray showing gas under diaphragm is not contributory. Immediate surgical exploration is the management. Simple closure or gastric resection should be done.

## TRAUMATIC PERFORATION OF DUODENUM

In blunt injury fixed pat of duodenum is affected more than the stomach, seat belt injuries are a well-recognized cause of duodenal rupture. In Retroperitoneal rupture, pain in the epigastrium and back, with vomiting, epigastric and flank tenderness are present. X-ray shows presence of air in the region of right kidney or Psoas muscle may be outlined by air. Mobilization of duodenum by kocherization and closure of the rent should be done. In Intraperitoneal rupture, there may be severe abdominal pain, Xray shows air under diaphragm. Simple closure with proximal diversion is ideal.

## IATROGENIC PERFORATION OF DUODENUM

Duodenal perforation is most common during ERCP with endoscopic sphincterotomy. This complication occurs in 0.3 to 2.1% of cases. Patients who have

undergone Billroth II gastrectomy are at increased risk; duodenal perforation complicates 1.5 to 5% of the ERCPs in these patients.

Perforations of the duodenum distal to its bulb, because of its retroperitoneal location, tend to be locally contained and can present insidiously.

Manifestations of contained duodenal perforation following ERCP can resemble those of ERCP – included pancreatitis, including hyperamylasemia25. Open transduodenal sphincteroplasty is complicated by duodenal perforation in 0.6% of cases26. Investigations include Plain Abdominal radiograph, contrast radiograph with Gastrograffin, CT scan. Iatrogenic perforation incurred during endoscopy, if immediately recognized, can be repaired using endoscopic techniques. Intraperitoneal duodenal perforations require surgical repair.

**CURLING ULCER:**

Curling first described this ulcer in patients with burns in 1842. Curling ulcer can occur both in stomach and duodenum, due to over activity of gastric glands and can be prevented by the use of H2 blockers. Perforation of ulcers in burns patients usually has fatal outcome. Perforation in burns patients tends to appear during convalescence and it is a different entity from acute stress ulcers.

**CUSHINGS ULCER:**

First described by Harvey Cushing in 1932. Ulcers that arises in the esophagus, stomach, duodenum usually after neuro surgical illness.

# MATERIALS AND METHODS

A prospective and retrospective study was conducted at our “Institute of General Surgery” – Rajiv Gandhi Govt General Hospital & Madras medical college, Chennai. Fifty cases of Gastroduodenal perforations were studied during the period of may 2017 to April 2018.

The diagnosis was established by the Emergency Surgeon provisionally, based on the clinical presentation and supporting radiological evidence, in the ward, and definitive diagnosis established at the time of operation.

**Inclusion criteria**

- Age 15 to 35 years
- Gastric or duodenal perforation

**Exclusion criteria**

- age <15 years
- age >35 years
- hollow viscus perforation other than gastroduodenal perforation

A proforma for study of etiological and risk factors for gastroduodenal perforations was used in patients in age group of 15 to 35 years diagnosed with gastroduodenal perforations.

Socioeconomic status was measured using modified kuppusamy scale.

Smoking habits were measured by categorizing patients as non smokers ,daily use of less than and more than 10 cigarettes per day.

Alcohol habits were measured by categorizing patients as non alcoholics ,alcoholics taking less than 21 units and more than 21 units per week.

H.pylori infectivity was diagnosed by taking intraop biopsies from ulcer edge and doing rapid urease test.

NSAIDS use was measured as no use or increased intake of nsaids for 6 months before diagnosing with perforation.

Psychological stress was measured using Cohen perceived stress scale with score of less than 13 categorised as negative for stress and more than 13 categorised as positive.

Irregular food habits were measured by categorizing as 1 to 2 meals per day and taking 3 or more meals per day.

# OBSERVATION AND RESULTS

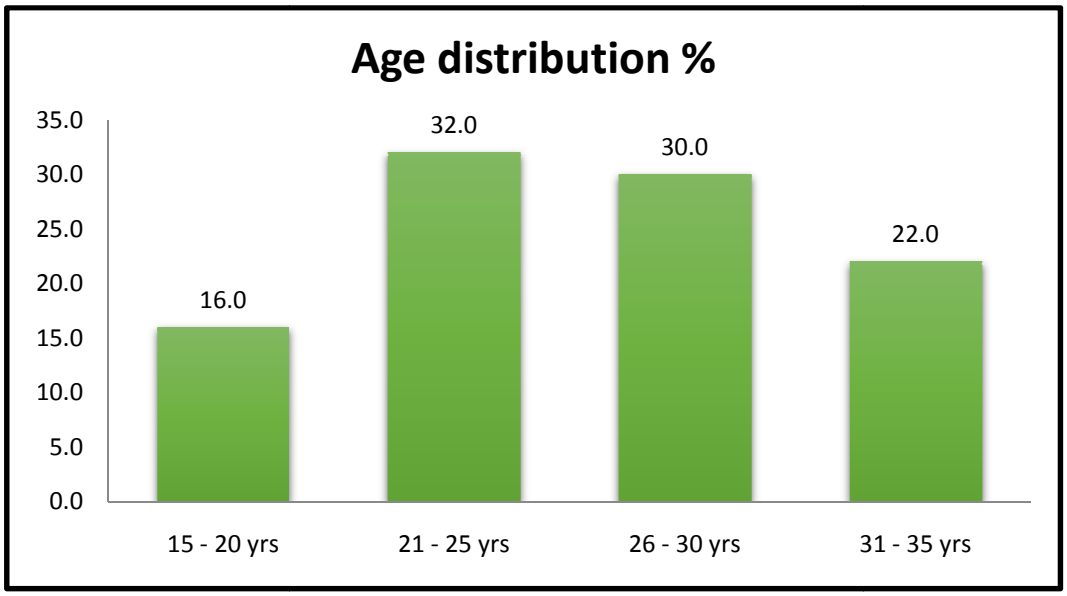
50 cases of gastroduodenal perforation patients were studied.

## AGE DISTRIBUTION:[44]

In my study of 15 to 35 age groups , gastroduodenal perforations were more common in 21 to 25 age groups followed closely by 26 to 30 yrs.

**AGE**

| | Frequency | Percent |
|---|---|---|
| 15 - 20 yrs | 8 | 16.0 |
| 21 - 25 yrs | 16 | 32.0 |
| 26 - 30 yrs | 15 | 30.0 |
| 31 - 35 yrs | 11 | 22.0 |
| Total | 50 | 100.0 |

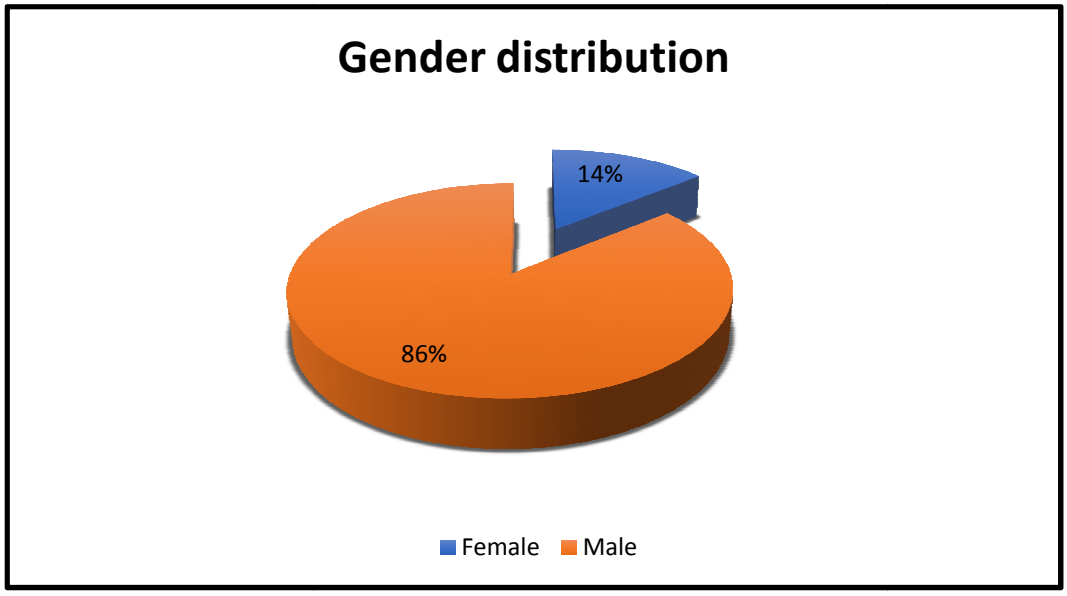


**SEX DISTRIBUTION:**

In my study, males were most commonly affected than females with a male to female ratio of 6:1 suggesting associated risk factors of smoking and alcohol among males.

**SEX**

| | Frequency | Percent |
|---|---|---|
| Female | 7 | 14.0 |
| Male | 43 | 86.0 |
| Total | 50 | 100.0 |

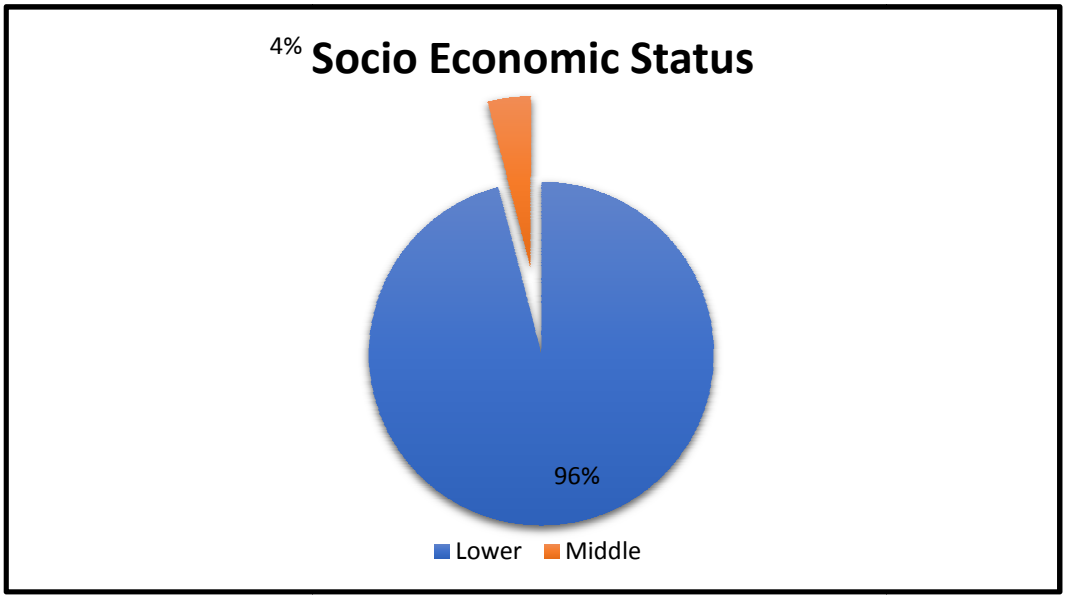


## SOCIOECONOMIC STATUS DISTRIBUTION:[45]

In my study, gastroduodenal perforations were more common among people in low socio economic status attributing upto 96 % of pts.

**SES**

| | Frequency | Percent |
|---|---|---|
| Lower | 48 | 96.0 |
| Middle | 2 | 4.0 |
| Total | 50 | 100.0 |

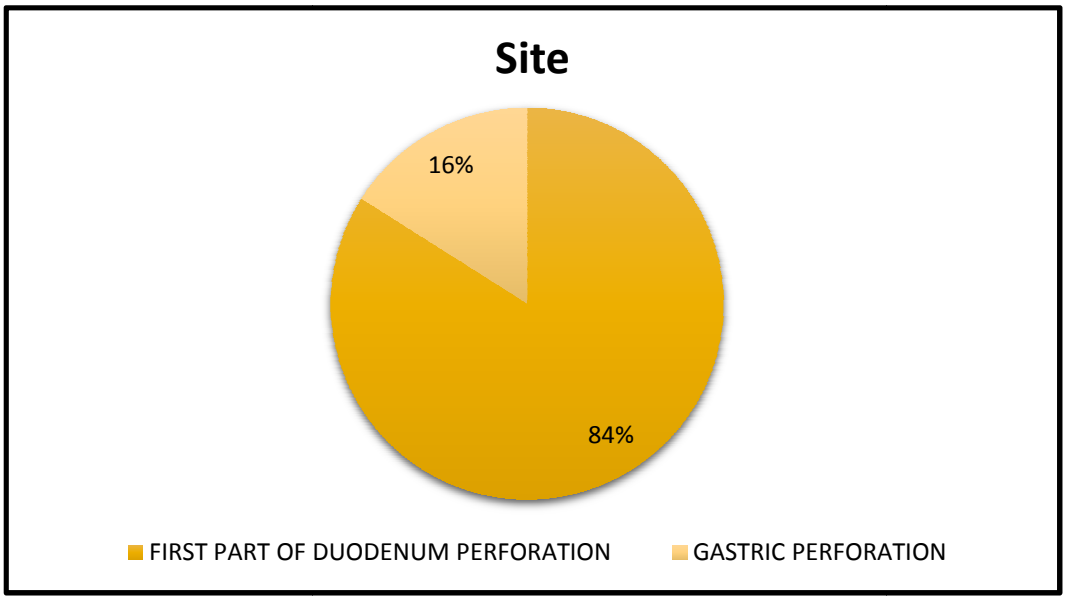


**SITE DISTRIBUTION:**

Duodenal perforations were more common than gastric perforations among patients in age group of 15 to 35 years, causing upto 42 out of 50 patients.

**SITE**

| | Frequency | Percent |
|---|---|---|
| FIRST PART OF DUODENUM PERFORATION | 42 | 84.0 |
| GASTRIC PERFORATION | 8 | 16.0 |
| Total | 50 | 100.0 |

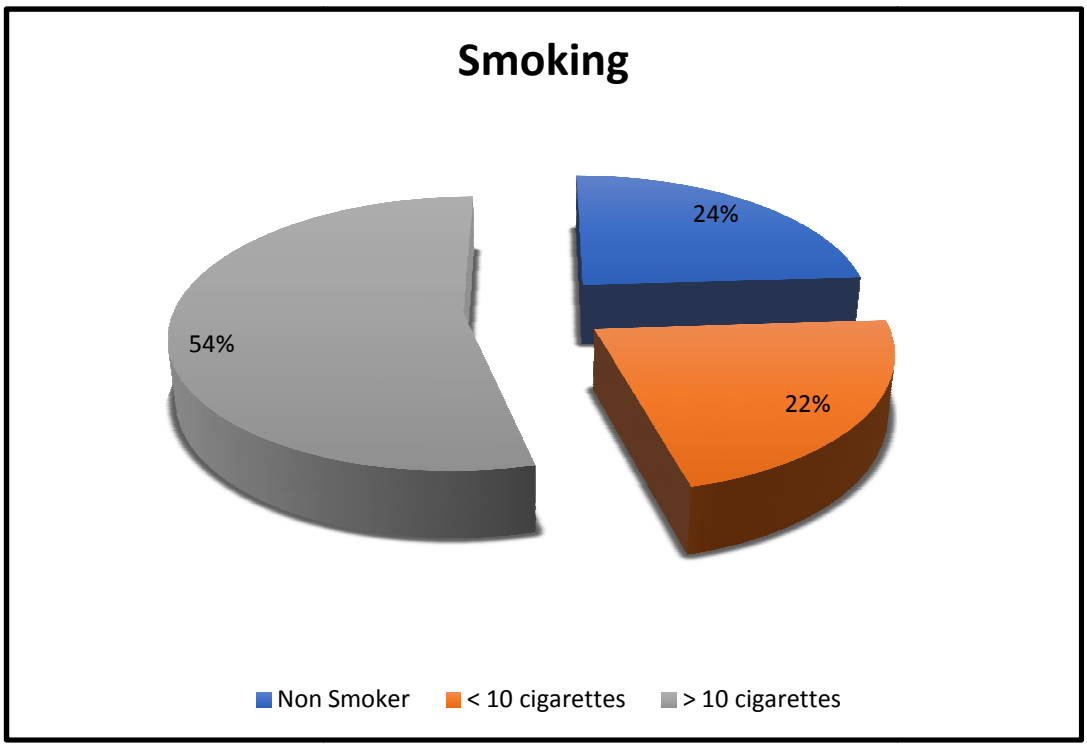


**RELATION TO SMOKING:**

In my study, 76 % of patients were smokers, out of which 54 % were chronic chain smokers smoking more than 10 cigarettes per day and 22 % smoked less than 10 cigarettes per day, suggesting high prevalence of gastroduodenal perforations among heavy smokers.

**SMOKING**

| | Frequency | Percent |
|---|---|---|
| Non Smoker | 12 | 24.0 |
| < 10 cigarettes | 11 | 22.0 |
| > 10 cigarettes | 27 | 54.0 |
| Total | 50 | 100.0 |

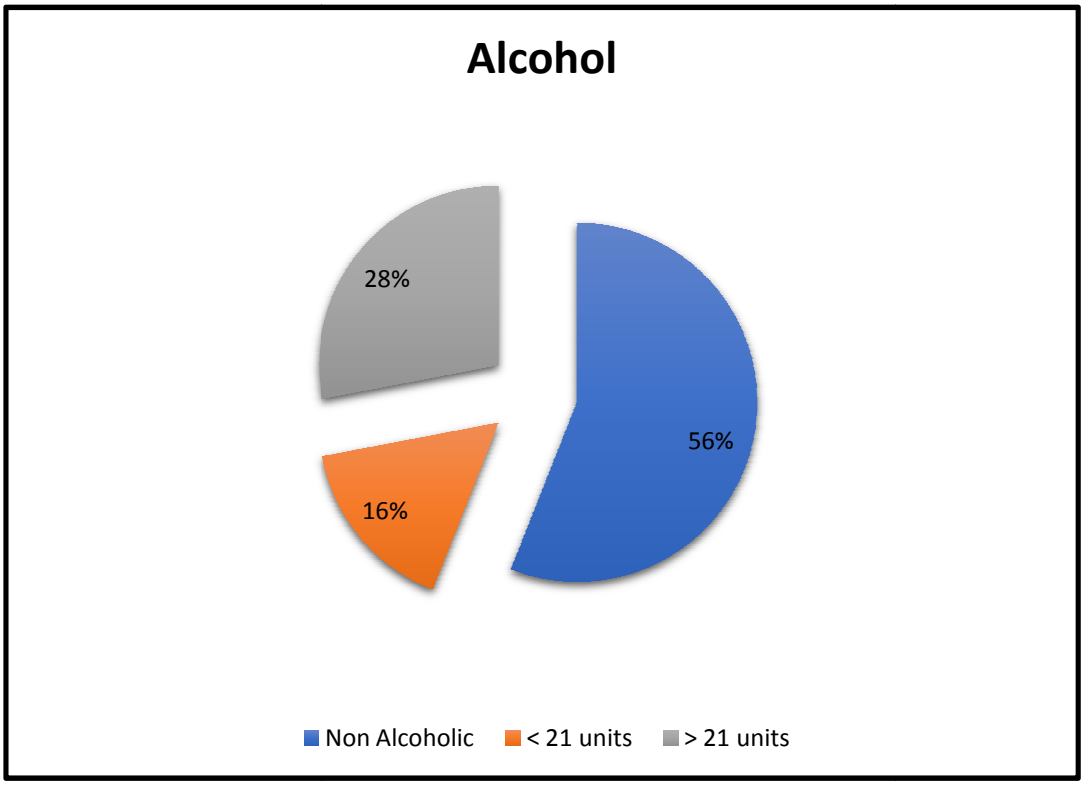


## RELATION WITH ALCOHOL:

In my study, 44 %of pts were alcoholics ,out of which 28 % were used to have more than 14 units of alcohol per week and 16 % were having less than 14 units of alcohol.56 % patients were nonalcoholic.

**ALCOHOL**

| | Frequency | Percent |
|---|---|---|
| Non Alcoholic | 28 | 56.0 |
| < 21 units | 8 | 16.0 |
| > 21 units | 14 | 28.0 |
| Total | 50 | 100.0 |

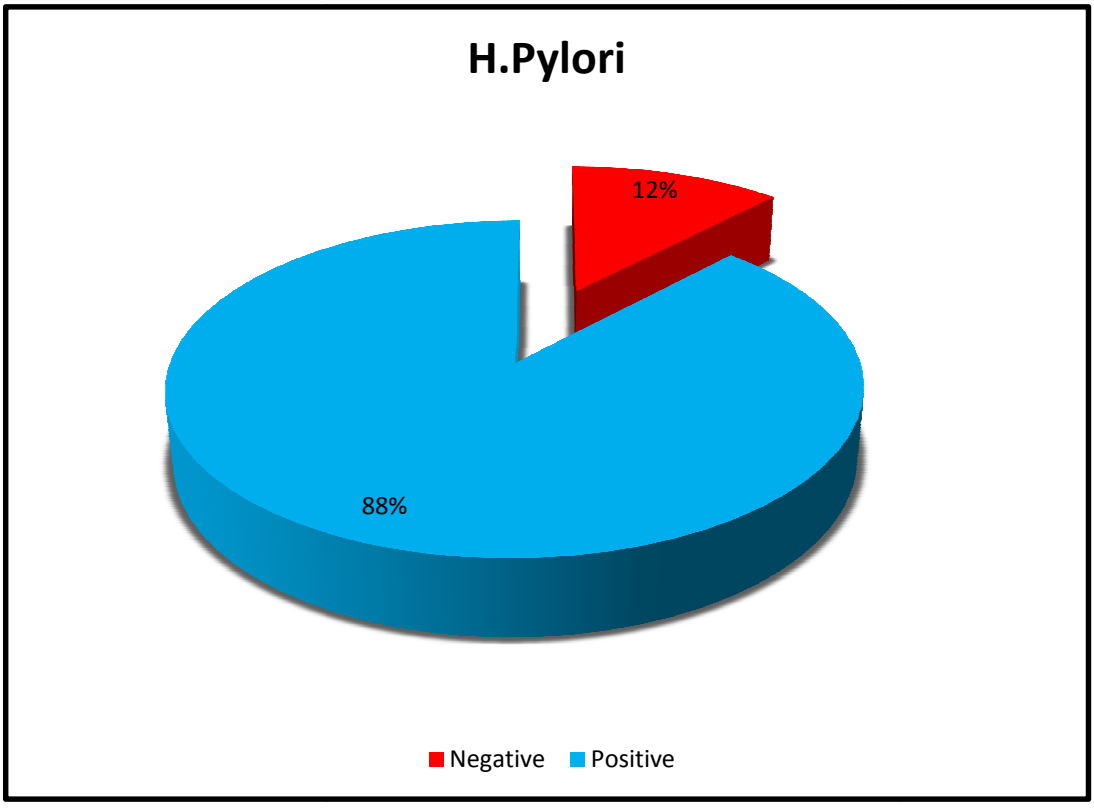


**RELATION WITH H.PYLORI:**

In my study, 88% patients were positive for h.pylori and only 12 % were negative suggesting high association of h.pylori with gastroduodenal perforations.

**H.PYLORI**

| | Frequency | Percent |
|---|---|---|
| Negative | 6 | 12.0 |
| Positive | 44 | 88.0 |
| Total | 50 | 100.0 |



## RELATION OF H.PYLORI WITH SITE OF PERFORATION:

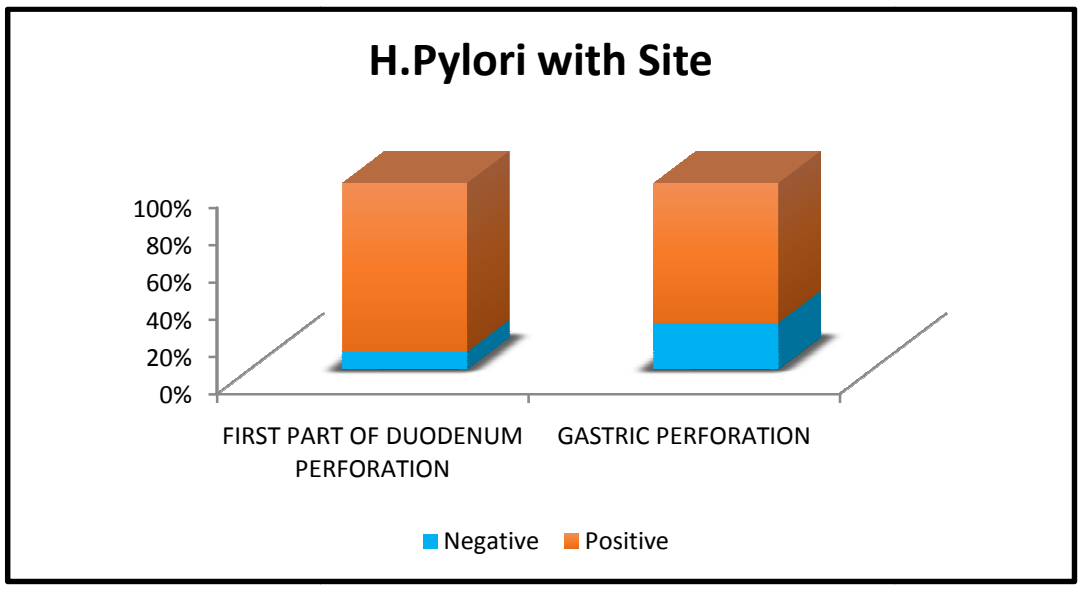
In my study, h.pylori was positive in 38 out of 44 patients with duodenal perforations, whereas it was positive in 4 out of 6 gastric perforations.

**Crosstabs**

**H.PYLORI * SITE Crosstabulation**

| | | | SITE | | |
|---|---|---|---|---|---|
| | | | D1 | G | Total |
| H.PYLORI | Negative | Count | 4 | 2 | 6 |
| | | % | 9.5% | 25.0% | 12.0% |
| | Positive | Count | 38 | 6 | 44 |
| | | % | 90.5% | 75.0% | 88.0% |
| Total | | Count | 42 | 8 | 50 |
| | | % within SITE | 100.0% | 100.0% | 100.0% |

| | FIRST PART OF DUODENUM PERFORATION | GASTRIC PERFORATION | |
|---|---|---|---|
| Negative | 9.5% | 25.0% | |
| Positive | 90.5% | 75.0% | |

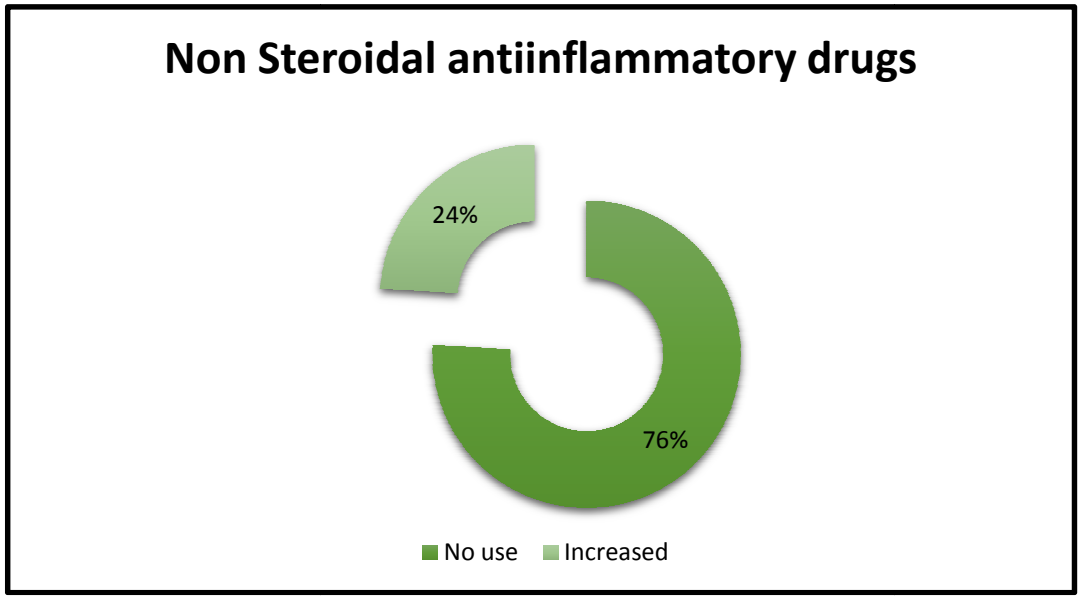


**RELATION WITH NSAIDS:**

In my study, only 24 % were using NSAIDS and remaining 76 % were not using nsaids.

**NSAIDS**

| | Frequency | Percent |
|---|---|---|
| No use | 38 | 76.0 |
| Increased | 12 | 24.0 |
| Total | 50 | 100.0 |

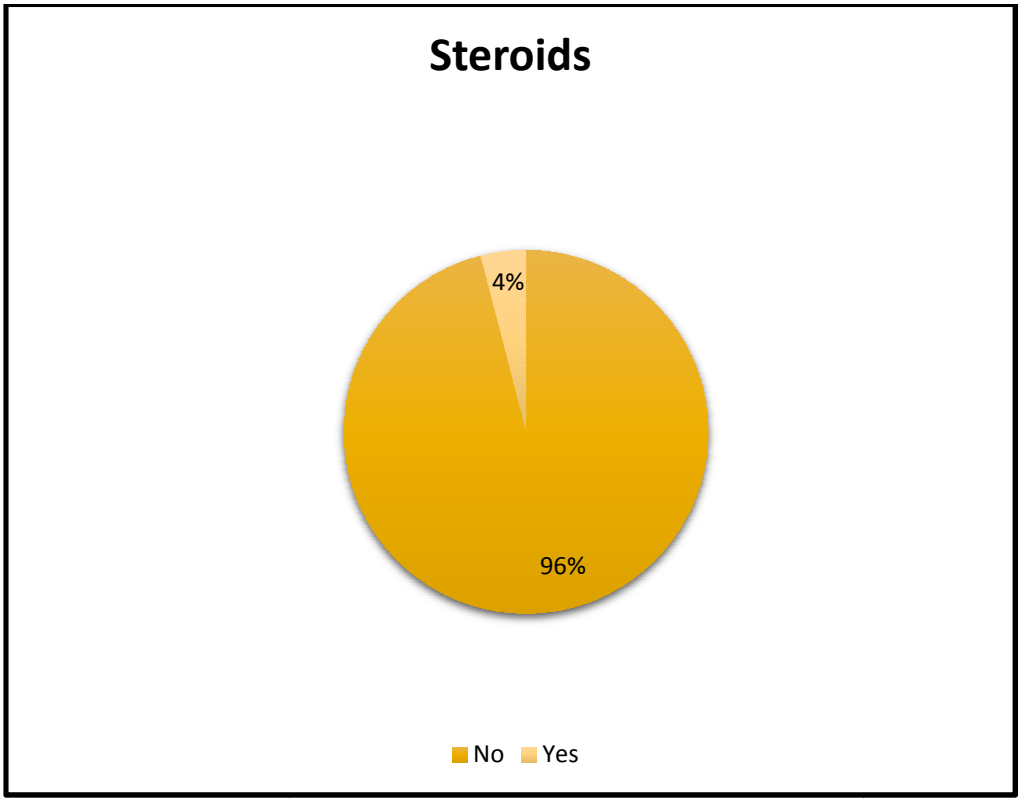


**RELATION WITH STEROIDS:**

In my study, only 4 percent of patients were using steroids regularly for their co morbidities.

**STEROIDS**

| | Frequency | Percent |
|---|---|---|
| No | 48 | 96.0 |
| Yes | 2 | 4.0 |
| Total | 50 | 100.0 |

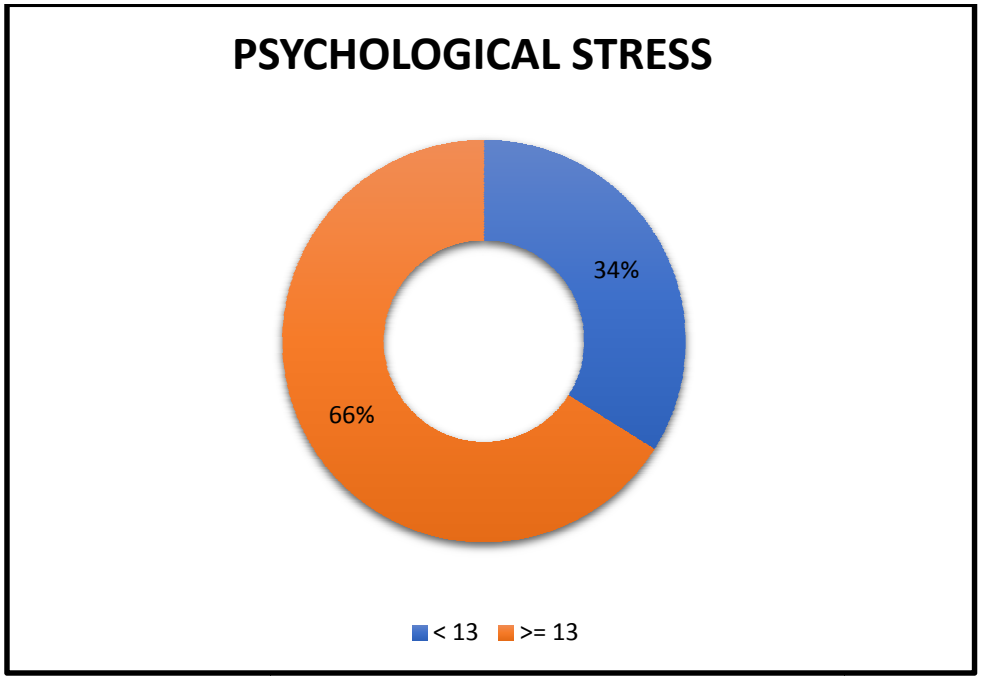


## RELATION WITH PYSCHOLOGICAL STRESS:

In my study, 66 % patients were subjected to physical or mental stresssuggesting significant risk for developing gastroduodenal perforations.

**PSY.STRESS**

| | Frequency | Percent |
|---|---|---|
| < 13 | 17 | 34.0 |
| >= 13 | 33 | 66.0 |
| Total | 50 | 100.0 |

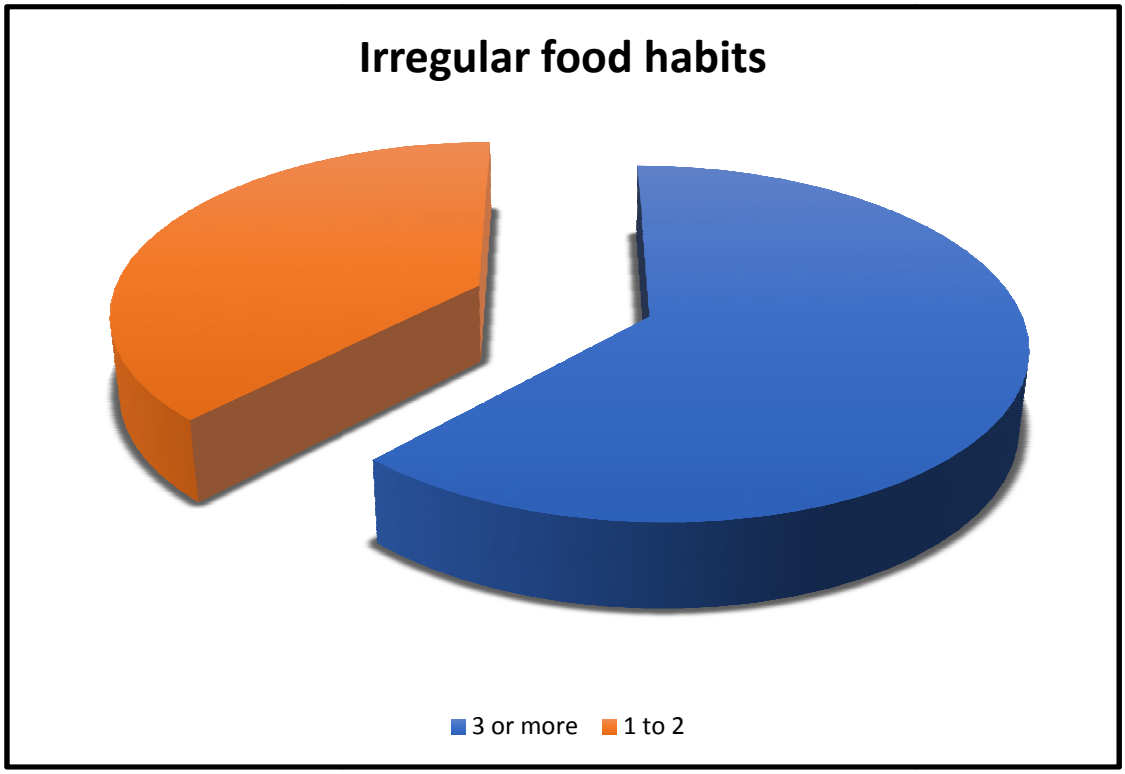


## RELATION WITH IRREGULAR FOOD HABITS:

In my study, 38 % of patients were having irregular food habits over a long period of time of having only 1 to 2 meals per day leading to risk of developing perforations.

**IRREGULAR FOOD HABITS**

| | Frequency | Percent |
|---|---|---|
| 3 or more | 31 | 62.0 |
| 1 to 2 | 19 | 38.0 |
| Total | 50 | 100.0 |



# DISCUSSION

Though recently perforations are on the rise among older people and particularly females who are abusing NSAIDS, in my study which was conducted in patients with gastroduodenal perforations in age group of 15 to 35 years, males were more commonly affected than females, the reason for which may be attributed to prevalence of risk factors like smoking and alcohol among males.

In my study, h.pylori was the predominant cause for gastroduodenal perforations. H.pylori affects nearly 50 % of world populations and most people acquire them in early childhood. H.pylori are associated with many diseases other than causing peptic ulcer perforations. H.pylori is common among people in low socioeconomic status and among people with poor sanitary hygiene.

Though not preventable, h.pylori can be treated and eradicated using proper mediacation savailable, awareness should be created among people regarding improved sanitary hygiene, patients with dyspeptic symptoms or peptic ulcer should undergo upper gi endoscopy to prove h.pylori or should be started on h.pylori eradication regimen.post surgical patients who underwent laparotomy for perforations also should be prescribed anti h.pylori regimen and should be followed up regularly with upper giscopy.

In my study, relation of h.pylori with gastroduodenal perforations was not statistically significant due to smaller sample of study this limitation can be overcome by selecting larger group of samples.

Gastroduodenal perforations were common among low socioeconomic groups with poor quality of life, and majority of them were unskilled labourers like construction workers who were immigrants from other states.

In my study, smoking was found to be one of the major risk factors for gastroduodenal perforations. this anti societal habit should be curbed among young adults for their own well being. Patients should be mentally prepared to quit smoking and with help of family, friends and self motivation.

Nicotine replacement therapies can be used to wean body off cigarettes with controlled dose of nicotine while sparing from exposure to other chemicals in cigarettes. commonly used therapies include skin patches, chewing gums, lozenges, nasalspray, inhaler.

Also FDA approved non nicotine drugs like bupropion and vareniciline can be used. heavy smokers should be counselled and advised socialization through joining in anti smoking groups. With the help of combination therapy and self motivation, smoking habit should be avoided to help them from developing complications like peptic ulceration and perforation.

Among young patients psychological stress is having a major impact in causing gastroduodenal perforations in my study. stress ranging from work stress,medical diseases to emotional stress like losing loved ones should be addressed to. Stressed ones should be counselled to do meditations , yoga or exercise. individual stress should be managed properly by assessing the cause for stress.

Nearly half of the young adults in my study were alcoholics and is also a risk factor for gastroduodenal perforation. alcoholics should be motivated from abstinence and should advised to join detoxification programmes in hospitals. Therapies include counselling and support group, cognitive behaviour altherapy, aversion therapy, family therapy, behaviortherapy, psychotherapy and group psychotherapy. drugs like naltrexone and acamprosate reduce alcohol cravings.disulfiram causes aversion to alcohol. topiramate and baclofen are also used.

Females should be advised of adverse effects of nsaids and their use should be regulated. young adults should be counselled regarding irregular food habits like avoiding spicy foods, carbonated drinks,coffee and outside foods. fasting and taking less than 2 meals per day should be avoided.

From my study it is obvious that along with h.pylori, smoking and stress in addition of alcohol and irregular food habits to a certain extent were major etiological risk factors in causing gastroduodenal perforations which will be reduced if adequate life style modifications are brought in form of anti smoking and anti alcoholmeasures. also the need to manage stress and having proper and regular food habits should be addressed to.

# CONCLUSION

Among young adults, gastroduodenal perforations were more common in 21 to 25 age groups.

More common among male patients with male to female ratio of 6:1.

Duodenal perforations were more common than gastric perforations.

In duodenum, perforation was more common in first part.

More common among lower socioeconomic groups.

H.pylori was the most common etiological risk factor for gastroduodenal perforations among 15 to 35 age groups, followed by smoking and psychological stress.

Role of alcohol, nsaids, steroids and irregular food habits as the causation of gastroduodenal perforations were little in my study.

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# DATA COLLECTION SHEET

**I.Patient particulars:**

| | | |
|---|---|---|
| Name | DOA | Case No. |
| Age | DOS | I.P. No. |
| Sex | DOD | Address |

Occupation:

**II.Diagnosis**

**III.ETIOLOGICAL RISK FACTORS**

| | |
|---|---|
| 1.SMOKING | YES / NO |
| 2.ALCOHOLISM | YES / NO |
| 3.NSAID ABUSE | YES / NO |
| 4.H.PYLORI INFECTION | YES / NO |
| 5.PSYCHOLOGICAL STRESS | YES / NO |
| 6.IRREGULAR FOOD HABITS | YES / NO |
| 7.STEROID USE | YES / NO |

**PERSONAL HISTORY**

**BLOOD GROUP**

**SOCIOECONOMIC STATUS**

**FAMILY HISTORY** **YES / NO**

# PATIENT CONSENT FORM

**STUDY TITLE:**

**" A CLINICAL STUDY ON GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15 -35 YEARS OF AGE)AND ITS ETIOLOGY"**

**STUDY CENTRE:**
Rajiv Gandhi Government General hospital and Madras Medical College.

**PARTICIPANT NAME:** **AGE:** **SEX:** **I.P. NO :**

I confirm that I have understood the purpose of interventional procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the interventional and interventional procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

**I hereby consent to participate in this study of the GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15 -35 YEARS OF AGE) AND ITS ETIOLOGY**

Date: Signature / thumb impression of the patient

Place:

Signature / thumb impression of the guardian

Patient's name:

Signature of the Investigator:

Name of the investigator : Dr.K.MADHANAGOPALAN

# INFORMATION SHEET

**We are conducting a study on ”GASTRODUODENAL PERFORATION IN YOUNG ADULTS (15 -35 YEARS OF AGE)AND ITS ETIOLOGY”** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your information is valuble to us.

The purpose of this study is to identify and analyse more common etiological risk factors among young patients with gastroduodenal perforation at RGGGH, Chennai.

We are selecting certain cases and if you are found eligible, we may be using your information which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Participant/Guardian

Signature of the Investigator

Date

Place

# ஆராய்ச்சியில் பங்கேற்பவர்கான தகவல் அறிக்கை

**ஆராய்ச்சி தலைப்பு**

**இளம்பெரியவர்களில் ஏற்படும் இரைப்பை மற்றும் முன்சிறுகுடல் துளைகளுக்கான காரண காரணிகளை கண்டறியும் ஓர் ஆய்வு**

பங்கு கொள்பவரின் பெயர் :

ஆராய்ச்சி செய்பவரின் பெயர் : மரு.கு.மதனகோபாலன்

இடம் : இராஜீவ்காந்தி அரசு பொது மருத்துவமனை,
சென்னை-3.

இந்த ஆராய்ச்சி/ஆய்வு/ செய்முறை/சோதனையில் தாங்கள் பங்கேற்க அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தாங்கள் இந்த ஆராய்ச்சியில் பங்கேற்கலாமா வேண்டாமா என்பதை முடிவு செய்ய உதவியாக இருக்கும். இந்த படிவத்தில் உள்ள தகவல்கள் பற்றி உள்ள சந்தேகங்களை நீங்கள் தயங்காமல் கேட்கலாம்.

**இந்த ஆய்வின் நோக்கம் என்ன?**

இளம்பெரியவர்களில் ஏற்படும் இரைப்பை மற்றும் முன்சிறுகுடல் துளைகளுக்கான காரண காரணிகளை பற்றி ஆய்வு மேற்கொள்ளப்படும்.

**ஆய்வு முறைகள்**

இளம்பெரியவர்களில் இரைப்பை மற்றும் முன்சிறுகுடல் துளை ஏற்பட்ட நோயாளிகளிடம் அதற்கு சம்பந்தப்பட்ட தகவல்கள் மற்றும் வழக்கமான இரத்தப் பரிசோதனை மற்றும் பல்வேறு ஸ்கேன்கள் எடுக்கப்படும்.

**ஆய்வினால் நோயாளிக்கு ஏற்படும் நன்மைகள்**

இந்த ஆய்வின் முடிவில் கிடைக்கும் தகவல்கள் சமுதாயத்திற்கு பயனுள்ளதாகவும், எதிர்காலத்தில் நோயாளிகளுக்கு மருத்துவ தீர்வாகவும் அமையும்

**ஆய்வினால் மருத்துவருக்கு ஏற்படும் நன்மைகள்**

மருத்துவர் நோயின் தன்மையை தேர்வு செய்யவும் அதன் பயனை நோயாளிக்கு எடுத்து உறைக்கவும் பயன்படும்.

**தங்களிடமிருந்து பெறப்படும் தகவல்களின் நம்பகத்தன்மை**

தங்களிடமிருந்து பெறப்படும் தகவல்கள் பாதுகாக்கப்படுவதற்கான முழு உரிமையும் தங்களுக்கு உண்டு.

இந்த படிவத்தில் கையொப்பமிடுவதன் மூலம், தாங்கள் தங்களை பற்றிய விவரங்களையும், ஆய்வு விவரங்களையும் ஆராய்சியாளர், ஆய்வு நடத்தும் ஏனையோர் வரைமுறை ஒழுங்கு குழுவினர் மற்றும் சட்டத்திற்கு உட்பட்ட மருந்து கட்டுப்பாடு இயக்குநர் ஆகியோர் பார்வையிட அனுமதிக்கின்றீர்கள்.

இந்த ஆய்வில் காட்டப்படும் தகவல்கள் அறிவியல் நாளேடுகளிலோ அறிவியல் கூட்டங்களிலோ சமர்ப்பிக்கப்படும் பட்சத்தில் தங்களது அடையாளம் வெளிப்படுத்தப்படமாட்டாது.

**இந்த ஆய்வில் பங்கேற்காமல் இருப்பதனால் ஏற்படும் பாதிப்பு**

இந்த ஆய்வில் தாங்கள் பங்கேற்க விருப்பம் தெரிவிக்காத நிலையில் தங்களின் மருத்துவர் மற்றும் மருத்துவமனையில் தங்களுக்கு உள்ள உறவில் எந்த பாதிப்பும் ஏற்படாது. தாங்கள் சிறப்பாக கவனிக்கப்படுவீர்கள். மேலும் இதனால் தங்களுக்கு இழப்பு ஏதும் ஏற்படாது.

**ஆய்வின் நடுவில் அதிலிருந்து விலகிகி கொள்ள நினைத்தால்**

இந்த ஆய்வில் பங்கேற்பது தங்களின் சொந்த விருப்பமே. மேலும் ஆய்வின் நடுவில் எந்த நேரத்திலும், எக்காரணமும் கூறாமல் விலகிக் கொள்ள தங்களுக்கு முழு உரிமை உண்டு. இருப்பினும் ஆய்விலிருந்து விலகுவதற்கு முன் ஆராய்ச்சி குழுவுடன் கலந்து ஆலோசிப்பது உகந்தது என பரிந்துரைக்கப்படுகிறது.

**ஆராய்ச்சியாளர் கையொப்பம்** **பங்கேற்பாளர் கையொப்பம்**

தேதி: தேதி :

# ஆராய்ச்சி ஒப்புதல் படிவம்

**ஆராய்ச்சியின் தலைப்பு:**

**இளம்பெரியவர்களில் ஏற்படும் இரைப்பை மற்றும் முன்சிறுகுடல் துளைகளுக்கான காரண காரணிகளை கண்டறியும் ஓர் ஆய்வு**

ஆராய்ச்சி செய்பவரின் பெயர் : மரு.கு.மதனகோபாலன்

ஆராய்ச்சி மையம் : ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை-600 003.

..... எனும் நான், எனக்கு கொடுத்துள்ள தகவல் தாளை படித்து புரிந்து கொண்டேன். நான் பதினெட்டு வயதை கடந்துள்ளதால், என்னுடைய சுய நினைவுடனும், முழு சுதந்திரத்துடனும், இந்த ஆராய்ச்சியில் என்னை சேர்த்துக கொள்ள சம்மதிக்கிறேன்.

1) நான் எனக்கு அளிக்கப்பட்ட ஒப்புதல் படிவத்தையும் தகவல்களையும் படித்து புரிந்துகொண்டேன்.

2) ஒப்புதல் படிவத்தில் உள்ள தகவல்கள் எனக்கு விளக்கிக் கூறப்பட்டன

3) ஆய்வின் தன்மை பற்றி எனக்கு விளக்கப்பட்டது.

4) என்னுடைய உரிமைகளையும், பொறுப்புகளையும் ஆராய்ச்சியாளர் விளக்கிக் கூறினார்.

5) நான் ஆராய்ச்சியாளருடன் ஒத்துழைப்பேன் என்றும் எனக்கு ஏற்படக்கூடிய அசாதாரணமான நிகழ்வுகள் பற்றியும் உடனடியாக ஆராய்ச்சியாளரிடம் தெரிவிப்பேன் என்று உறுதி கூறுகிறேன்.

6) நான் கடந்த ..... மாதங்களாக வேறு எந்தவிதமான ஆய்வுகளிலும் பங்கேற்கவில்லை.

7) எனக்கு செய்யப்படும் அனைத்து பரிசோதனைகளும் (உதாரணம்: இரத்தம் எடுத்தல்) என நோயின் தன்மையை அறிவதற்காக செய்யப்படுபவை என்பதை அறிகிறேன்

8) இந்த ஆய்விலிருந்து எப்போது வேண்டுமானாலும் எக்காரணமும் கூறாமல் என்னை விடுவித்துக் கொள்ளலாம் என்பதை அறிவேன். மற்றும் இதனால் எனக்குத் தரப்படும் சிகிச்சைக்கு எந்த பாதிப்பும் வராது என்பதை அறிவேன்.

9) ஆராய்ச்சியாளர்கள் இந்த ஆய்வில் எனது பங்களிப்பை எந்த நேரத்திலும், எக்காரணமும் கூறாமல் என் சம்மதம் இல்லாமலும் என்னை விலக்கிவிட முடியும் என்பதை அறிவேன்.

10) என்னிடம் இருந்து பெறப்படும் தகவல்களை அரசு, வரைமுறை அதிகாரிகள் ஆகியோர்களுடன் பகிர்ந்து கொள்ள ஆராய்ச்சியாளர்களுக்கு அனுமதி அளிக்கிறேன். என்னுடைய தஸ்தாவேஜ்க்களை பார்வையிட அவர்களுக்கு உரிமை உண்டு.

11) என்னிடம் பெறப்படும் தகவல்களை பொதுவாக பிரசுரிக்கப்பட்டால், என்னுடைய அடையாளம் இரகசியமாக வைக்கப்படும் என்பதை அறிவேன்.

12) இந்த ஆராய்ச்சியில் பங்கேற்க தன்னிச்சையாக முழு மனதுடன் நான் சம்மதிக்கிறேன்.

இந்த ஆய்வின் போது எனக்கு என்ன சந்தேகம் ஏற்பட்டாலும் ஆராய்ச்சியாளரை தொடர்பு கொள்ளலாம் என்பதை அறிவேன். இந்த ஒப்புதல் படிவத்தில் கையெழுத்திடுவது மூலம் இங்கு தரப்பட்டிருக்கும் அனைத்து தகவல்களும் தெளிவாக கூறப்பட்டு என்னால் முபமையாக புரிந்து கொள்ளப்பட்டது என்பதை சான்றளிக்கிறேன். இந்த ஒப்புதல் படிவத்தின் நகல் என்னால் பெற்றுக் கொள்ளப்பட்டது.

பங்கேற்பவரின் கையொப்பம்: இடம்:

கட்டை விரல் ரேகை: தேதி:

பங்கேற்பவரின் பெயர்:

ஆய்வாளரின் பெயர்:

இடம்:
தேதி:

## MASTER CHART

| S.NO | NAME | AGE | SEX | IP NO | SES | SITE | SMOKING | ALCOHOL | H.PYLORI | NSAIDS | STEROIDS | PSY.STRESS | IRREGULAR FOOD HABITS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Natesan | 25 | m | 68412 | L | D1 | S2 | A2 | 1 | 0 | 0 | 0 | 0 |
| 2 | Babu | 22 | m | 60115 | L | D1 | S2 | 0 | 1 | 0 | 0 | 0 | 0 |
| 3 | Suresh | 19 | m | 98554 | L | D1 | 0 | 0 | 1 | 0 | 0 | 1 | 1 |
| 4 | muniyan | 34 | m | 57112 | L | G | S1 | A1 | 1 | 1 | 0 | 1 | 0 |
| 5 | rajesh | 24 | m | 58664 | L | D1 | S2 | A2 | 1 | 0 | 0 | 1 | 0 |
| 6 | saroja | 23 | f | 80457 | L | D1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 7 | venkatesan | 33 | m | 65874 | L | D1 | S1 | A2 | 1 | 0 | 0 | 1 | 0 |
| 8 | kumar | 21 | m | 54865 | L | D1 | S2 | 0 | 1 | 0 | 0 | 1 | 0 |
| 9 | azhagan | 31 | m | 66675 | L | D1 | S2 | A2 | 1 | 1 | 0 | 1 | 0 |
| 10 | kuppan | 22 | m | 74635 | L | D1 | S2 | 0 | 0 | 0 | 0 | 1 | 1 |
| 11 | pichaimuthu | 30 | m | 88452 | L | D1 | S2 | A1 | 1 | 0 | 0 | 1 | 0 |
| 12 | veni | 23 | f | 89845 | L | D1 | 0 | 0 | 1 | 0 | 0 | 1 | 1 |
| 13 | mohan | 20 | m | 11245 | L | D1 | S1 | A2 | 1 | 0 | 0 | 0 | 0 |
| 14 | vinoth | 25 | m | 15825 | L | D1 | S2 | 0 | 1 | 0 | 0 | 1 | 0 |
| 15 | vasanth | 24 | m | 78546 | L | D1 | S2 | 0 | 0 | 0 | 0 | 1 | 1 |
| 16 | rajan | 27 | m | 22548 | L | D1 | S2 | A2 | 1 | 1 | 0 | 1 | 0 |
| 17 | karthik | 17 | m | 84569 | L | D1 | S1 | A1 | 1 | 0 | 0 | 0 | 0 |
| 18 | rangan | 28 | m | 95456 | L | D1 | S2 | A2 | 1 | 0 | 0 | 1 | 0 |
| 19 | sulochana | 34 | f | 92456 | L | G | 0 | 0 | 1 | 1 | 1 | 0 | 1 |
| 20 | paneer | 35 | m | 12874 | L | D1 | S2 | A2 | 0 | 0 | 1 | 1 | 0 |
| 21 | selvam | 23 | m | 16649 | L | D1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 22 | boopathi | 30 | m | 85344 | L | D1 | S2 | A2 | 1 | 0 | 0 | 1 | 1 |
| 23 | vimal | 24 | m | 76552 | L | D1 | S1 | 0 | 1 | 0 | 0 | 1 | 0 |
| 24 | ragu | 27 | m | 64492 | L | D1 | S2 | A2 | 1 | 0 | 0 | 1 | 1 |
| 25 | poorna | 22 | f | 35499 | L | D1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |

| S.NO | NAME | AGE | SEX | IP NO | SES | SITE | SMOKING | ALCOHOL | H.PYLORI | NSAIDS | STEROIDS | PSY.STRESS | IRREGULAR FOOD HABITS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | ashokan | 31 | m | 31665 | L | G | S2 | A2 | 0 | 1 | 0 | 1 | 0 |
| 27 | balan | 28 | m | 66595 | L | D1 | S2 | 0 | 1 | 0 | 0 | 0 | 0 |
| 28 | ganesan | 26 | m | 97856 | L | D1 | 0 | A1 | 1 | 0 | 0 | 0 | 1 |
| 29 | arivu | 32 | m | 32489 | L | D1 | S2 | A2 | 1 | 1 | 0 | 1 | 0 |
| 30 | muthu | 18 | m | 35548 | L | D1 | S1 | A0 | 1 | 0 | 0 | 1 | 1 |
| 31 | sekar | 29 | m | 48645 | M | D1 | S2 | 0 | 1 | 1 | 0 | 1 | 0 |
| 32 | sanjeev | 34 | m | 69945 | L | G | S2 | A2 | 0 | 0 | 0 | 0 | 0 |
| 33 | seetha | 18 | f | 48955 | L | D1 | 0 | 0 | 1 | 0 | 0 | 1 | 1 |
| 34 | subash | 28 | m | 95946 | L | D1 | S1 | 0 | 1 | 0 | 0 | 1 | 0 |
| 35 | praveen | 27 | m | 49959 | L | G | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 36 | murugan | 20 | m | 45894 | L | D1 | S2 | 0 | 1 | 0 | 0 | 1 | 0 |
| 37 | palanisamy | 30 | m | 49564 | L | D1 | S2 | 0 | 1 | 1 | 0 | 1 | 0 |
| 38 | srinivasan | 27 | m | 48496 | L | D1 | S1 | A1 | 0 | 0 | 0 | 0 | 1 |
| 39 | arumugam | 33 | m | 49596 | L | G | S2 | 0 | 1 | 1 | 0 | 1 | 0 |
| 40 | sandeep | 22 | m | 49567 | L | D1 | S2 | 0 | 1 | 0 | 0 | 1 | 0 |
| 41 | saratha | 22 | f | 49567 | L | D1 | 0 | 0 | 1 | 0 | 0 | 1 | 1 |
| 42 | mannan | 28 | m | 56594 | L | D1 | S1 | A2 | 1 | 0 | 0 | 0 | 0 |
| 43 | arjunan | 34 | m | 49495 | L | G | S2 | 0 | 1 | 1 | 0 | 1 | 1 |
| 44 | naveen | 26 | m | 87548 | M | D1 | S2 | A1 | 1 | 0 | 0 | 1 | 0 |
| 45 | rajiv | 25 | m | 99794 | L | D1 | S2 | 0 | 1 | 0 | 0 | 0 | 1 |
| 46 | pushpa | 25 | f | 99569 | L | D1 | 0 | 0 | 1 | 1 | 0 | 1 | 0 |
| 47 | joseph | 28 | m | 13265 | L | G | S1 | 0 | 1 | 0 | 0 | 1 | 0 |
| 48 | ibrahim | 19 | m | 46949 | L | D1 | 0 | A1 | 1 | 0 | 0 | 0 | 1 |
| 49 | michael | 31 | m | 49496 | L | D1 | S1 | 0 | 1 | 1 | 0 | 1 | 0 |
| 50 | vivek | 16 | m | 49647 | L | D1 | S2 | 0 | 1 | 0 | 0 | 0 | 1 |

# MASTER CHART

KEY TO MASTER CHART:

SES : SOCIOECONOMIC STATUS

L:LOWER SES

M:MIDDLE SES

SITE : D1:FIRST PART OF DUODENUM PERFORATION

G:GASTRIC PERFORATION

SMOKING: 0 – NON SMOKER

S1 – LESS THAN 10 CIGARETTES PER DAY

S2 – MORE THAN 10 CIGARETTES PER DAY

ALCOHOL : 0 – NON ALCOHOLIC

A1 – ALCOHOLIC WITH INTAKE OF LESS THAN 21 UNITS OF ALCOHOL PER WEEK

A2 – ALCOHOLIC WITH INTAKE OF MORE THAN 21 UNITS OF ALCOHOL PER WEEK

H.PYLORI : HELICOBACTER PYLORI

1 – INFECTION POSITIVE

0 – INFECTION NEGATIVE

NSAIDS : NON STEROIDAL ANTI INFLAMMATORY DRUGS

0 – NO USE OF NASIDS

1 – INCREASED NSAIDS USE IN 6 MONTHS BEFORE PERFORATION

PSY.STRESS : PSYCHOLOGICAL STRESS

0 – SCORE OF LESS THAN 13 IN COHEN PERCEIVED STRESS SCALE

1 – SCORE MORE THAN 14 IN COHEN PERCEIVED STRESS SCALE

IRREGULAR FOOD HABITS

0 – 3 OR MORE MEALS PER DAY

1 – 1 TO 2 MEALS PER DAY.

# MODIFIED KUPPUSAMY SCALE: 2018

**Table 1: Modified Kuppuswamy Socioeconomic scale updated for January 2018.**

**(a) Occupation of the Head of the Family: -**

| Sr. No. | Occupation of the Head | Score |
|---|---|---|
| 1 | Legislators, Senior Officials & Managers | 10 |
| 2 | Professionals | 9 |
| 3 | Technicians and Associate Professionals | 8 |
| 4 | Clerks | 7 |
| 5 | Skilled Workers and Shop & Market Sales Workers | 6 |
| 6 | Skilled Agricultural & Fishery Workers | 5 |
| 7 | Craft & Related Trade Workers | 4 |
| 8 | Plant & Machine Operators and Assemblers | 3 |
| 9 | Elementary Occupation | 2 |
| 10 | Unemployed | 1 |

**(b) Education of the Head of the Family:-**

| Sr. No. | Education of the Head | Score |
|---|---|---|
| 1 | Profession or Honours | 7 |
| 2 | Graduate | 6 |
| 3 | Intermediate or diploma | 5 |
| 4 | High school certificate | 4 |
| 5 | Middle school certificate | 3 |
| 6 | Primary school certificate | 2 |
| 7 | Illiterate | 1 |

**(c) Total Monthly Income of the Family: -**

| Sr. No. | Updated Monthly Family Income in Rs. (2012) | Updated Monthly Family Income in Rs. (2016) | Updated Monthly Family Income in Rs. (2018) | Score |
|---|---|---|---|---|
| 1 | >30375 | ≥ 40,430 | >126,360 | 12 |
| 2 | 15188-30374 | 20,210–40,429 | 63,182-126,356 | 10 |
| 3 | 11362-15187 | 15,160–20,209 | 47,266-63178 | 6 |
| 4 | 7594-11361 | 10,110–15,159 | 31,591-47262 | 4 |
| 5 | 4556-7593 | 6060–10,109 | 18,953-31589 | 3 |
| 6 | 1521-4555 | 2021–6059 | 6327-18949 | 2 |
| 7 | ≤1520 | ≤ 2020 | ≤6323 | 1 |

**(d) Kuppuswamy's Socio-Economic Status Scale 2018:-**

| Sr. No. | Score | Socioeconomic Class |
|---|---|---|
| 1 | 26–29 | Upper (I) |
| 2 | 16–25 | Upper Middle (II) |
| 3 | 11–15 | Lower Middle (III) |
| 4 | 5–10 | Upper Lower (IV) |
| 5 | < 5 | Lower (V) |

# COHEN PERCEIVED STRESS SCALE:

## PERCEIVED STRESS SCALE

**The questions in this scale ask you about your feelings and thoughts** during the last month**. In each case, you will be asked to indicate by circling *how often* you felt or thought a certain way.**

Name _____ Date _____

Age _____ Gender (*Circle*): **M F** Other _____

**0 = Never 1 = Almost Never 2 = Sometimes 3 = Fairly Often 4 = Very Often**

| | | | | | |
|---|---|---|---|---|---|
| 1. In the last month, how often have you been upset because of something that happened unexpectedly? | 0 | 1 | 2 | 3 | 4 |
| 2. In the last month, how often have you felt that you were unable to control the important things in your life? | 0 | 1 | 2 | 3 | 4 |
| 3. In the last month, how often have you felt nervous and "stressed"? | 0 | 1 | 2 | 3 | 4 |
| 4. In the last month, how often have you felt confident about your ability to handle your personal problems? | 0 | 1 | 2 | 3 | 4 |
| 5. In the last month, how often have you felt that things were going your way? | 0 | 1 | 2 | 3 | 4 |
| 6. In the last month, how often have you found that you could not cope with all the things that you had to do? | 0 | 1 | 2 | 3 | 4 |
| 7. In the last month, how often have you been able to control irritations in your life? | 0 | 1 | 2 | 3 | 4 |
| 8. In the last month, how often have you felt that you were on top of things? | 0 | 1 | 2 | 3 | 4 |
| 9. In the last month, how often have you been angered because of things that were outside of your control? | 0 | 1 | 2 | 3 | 4 |
| 10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them? | 0 | 1 | 2 | 3 | 4 |