“STUDY OF PEROPERATIVE MUCOSAL BIOPSY IN PEPTIC ULCER PERFORATION”

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the regulations for the award of the degree of

M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.PANDIYARAJ S** and submitted in partial fulfilment of the requirement for the Degree of M.S. General Surgery, Branch I of the TamilnaduDr. M.G.R. Medical University , Chennai.

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Period of Study	:	**1 year**
College	:	**Coimbatore Medical College & Hospital.**
Dissertation Topic	:	**Study of per operative mucosal biopsy in peptic ulcer perforation.**

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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DECLARATION

I solemnly declare that the dissertation titled "**STUDY OF PEROPERATIVE MUCOSAL BIOPSY IN PEPTIC ULCER PERFORATION"** was done by me from 2016 onwards under the guidance and supervision of **DR. V. LEKSHMINARAYANI , M.S , D.G.O.**

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

PLACE: **SIGNATURE OF THE CANDIDATE**

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DATE: **SIGNATURE OF THE CANDIDATE**

PLACE:

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ABSTRACT

BACKGROUND:

Previously the etiological factors of peptic ulcer is caused by stress, spicy foods before 1983 ,after that Warren and Marshall identified the relationship between the H. pylori infection and peptic ulcer.Now it recognized that H. pylori is one of the most common cause of peptic ulcer disease and the prevalence of H. pylori varies with age, socioeconomic status and other associated risk factors, so diagnosing and controlling the H.pylori infection will reduce the mortality and morbidity of the Patient.

AIM AND OBJECTIVES:

To study the association of H.pylori in patients with perforated peptic ulcer by RUT and HPE and to study the association between other risk factors and H.pylori infection.

MATERIALS AND METHODS :

About 50 cases of perforated peptic (gastroduodenal) ulcer evaluated and analysed in an emergency basis in general surgery department in coimbatore medical college and hospital for a period of between July 2017 to July 2018 are included in this study. Emergency exploratory laparotomy has been performed in all these cases due to peptic ulcer perforation. During

intraoperatively , two mucosal biopsies taken from the perforation site and sent for RUT and HPE to detect H.Pylori infection.

RESULT:

Out of 50 cases of peptic ulcer perforation, 39 cases (78%) turned out to be positive for H.pylori. Out of 50, H.pylori status positive for most of the middle aged persons (21-30 years) – 25.6 % and males were commonly affected 87.2 %. Acid peptic disease is most commonly asscociated with H.pylori positive patients around 84.6%. Post operatively H.pylori positive patients were put on anti H.pylori treatment.

CONCLUSION

There was a high prevalence of H.pylori infection in patients with perforated peptic ulcer. Patient with perforated peptic ulcer gave history of chronic dyspepsia, should be treated with anti H.pylori therapy post operatively to reduce the recurrence and reperforation.

INTRODUCTION

Peptic ulcer perforation is relatively common in our institute. Patients will present to the emergency department with features of Perforative peritonitis such as abdominal pain ,guarding, rigidity and some patients present late with features of shock and hypovolemia and needed aggressive management for survival of the patient.So peptic ulcer perforation is relatively causing economical burden on health care services, so that diagnosing the condition earlier will give good prognosis.

Previously the etiological factors of Peptic ulcer is caused by stress, spicy foods before 1983 ,after that Warren and Marshall identified the relationship between the H. pylori infection and peptic ulcer. Now it recognized that H. pylori is one the most common cause of peptic ulcer disease and the prevalence of H. pylori varies with age, socioeconomic status and other associated risk factors, so diagnosing and controlling the H.pylori infection will reduce the mortality and morbidity of the Patient.

The realization that H. Pylori may have a fundamental role in the etiology & the pathogenesis of peptic ulcer disease and its complications. So therapeutic strategies aimed at eradicating this bacterium and curing the disease.

Perforated peptic ulcer is most common in disease ranging approximately 7 to 10 cases per 100000 populations per year. Perforation is

one of the complication of peptic ulcer disease is present in about 7% of patients were admitted in hospital.

So that there is a necessity to study the association of perforation in peptic ulcer disease, but the correlation between the perforated peptic ulcer and H.pylori is not yet fully established if proved the control of H.pylori may reduce the economic burden on health services.

We are going to study the presence of H.pylori in all cases of perforated peptic ulcer by taking the peroperative mucosal biopsy and confirming it with RUT and HPE examination.

AIMS & OBJECTIVES

The aim of this study is know the presence of H.pylori in patients with perforated peptic ulcer by using rapid urease test and histopathological examination

Objectives of this studies are,

1. To study the various etiological factors in peptic ulcer perforation.
2. To study the cases of peptic ulcer perforation in relation to H. pylori infection

MATERIALS AND METHODS

About 50 cases of perforated peptic (gastroduodenal) ulcer evaluated and analysed in an emergency basis in General surgery department in Coimbatore medical college and hospital for a period of between July 2017 to July 2018 are included in this study. The details of 50 patients were arranged in the master chart.

INCLUSION CRITERIA:

1. Patients of age group between 13-80 years.
2. Patients undergoing emergency laparotomy with features of perforative peritonitis due to peptic ulcer perforation.

EXCLUSION CRITERIA:

1. Patients below the age group of 13 years and above the age group of 80 years
2. Pregnant patients ,
3. Patients presenting with perforative peritonitis due to trauma
4. Patients received Anti-H.pylori treatment.

DURATION OF STUDY :

12 months (July 2017 to July 2018)

STUDY DESIGN :

PROSPECTIVE COHORT STUDY

SAMPLE SIZE:

50

The study population contains of 50 patients included between the age group of 13-80 yrs. Emergency exploratory laparotomy has been performed in all these cases due to peptic ulcer perforation.

During intraoperatively , two mucosal biopsies taken from the perforation site, in that one tissue sample is used to perform rapid urease test(RUT) immediately and another tissue sample sent for histopathological examination to detect H.pylori by giemsa staining.

RAPID UREASE TEST:

- We have taken biopsy from the edge of ulcer in order to perform Rapid urease test
- Urea solution is prepared by adding 10gm of urea powder in 10ml of distilled water which gives 1% urea solution

- 1ml of this prepared urea solution is added to 2 sterile plain bulbs
- One of which is labelled as control and other is labelled as test
- 2-3 drops of Phenol red is added (indicator)
- Of 2-3mm thickness tissue sample taken from the edge of ulcer and added to this testing bulb

PRINCIPLE:

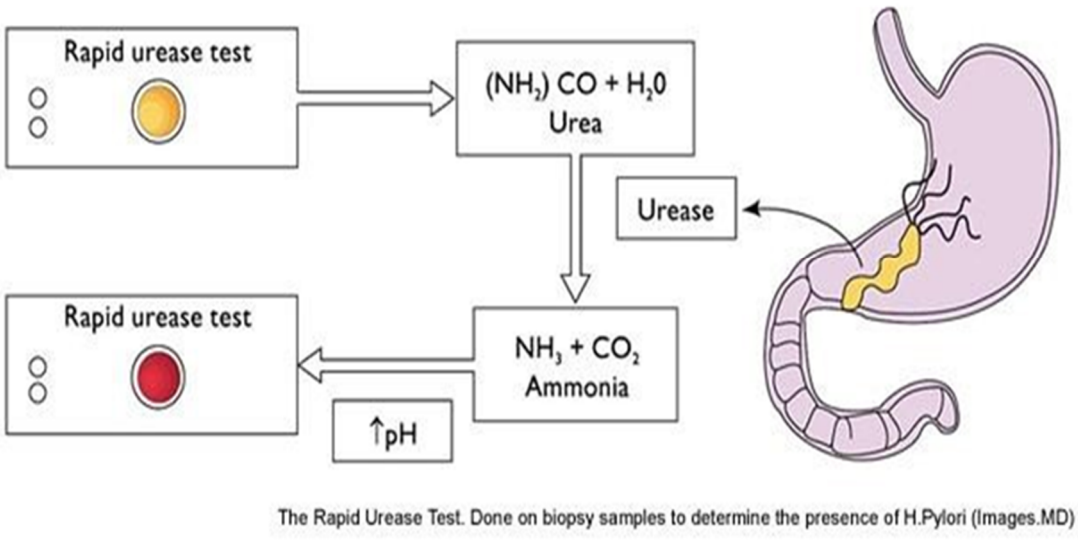
H.pylori secretes an enzyme-urease which catalyses conversion of urea to ammonia and bicarbonate there by raising the PH of medium which is interpreted by Colour change yellow to pink.

INTERPRETATION:

- If the colour changes from Yellow to Pink it is considered as Positive for H.pylori
- If the colour doesn't change from yellow to pink colour even after 24 hours it is considered as negative for H.pylori

RAPID UREASE TEST

FIGURE 1



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

FIGURE 2

STAINING TECHNIQUE:

The **Second tissue sample** is fixed in a 10% formalin solution further subjected to Giemsa staining in PATHOLOGY department demonstrating the histopathologic characteristics of the biopsy tissue while optimally demonstrating H. pylori.

FIXATION: 10% buffered neutral formalin solution

SECTION: 3 microns

PROCEDURE:

- Deparaffinize and hydrate to distilled water
- Place in methanol for 2 minutes
- Pre heat working giemsa solution in microwave on high for 45 seconds
- Filter the hot solution into a coplin jar containing the slides
- Stain for 2 minutes

(To prepare the working solution mix with 7ml of giemsa stock ,38ml of distilled water and 5ml of methanol)

- Rinse quickly in distilled water
- Dip one slide at a time in acetic acid1%

- Rinse quickly in distilled water
- Rinse in 3 changes of methanol
- Clear in xylene and mount

STAINING RESULTS:

- **H.Pylori** -Dark blue
- **Tissue Elements** – shades of blue and pink

GIEMSA STAINING – H.pylori

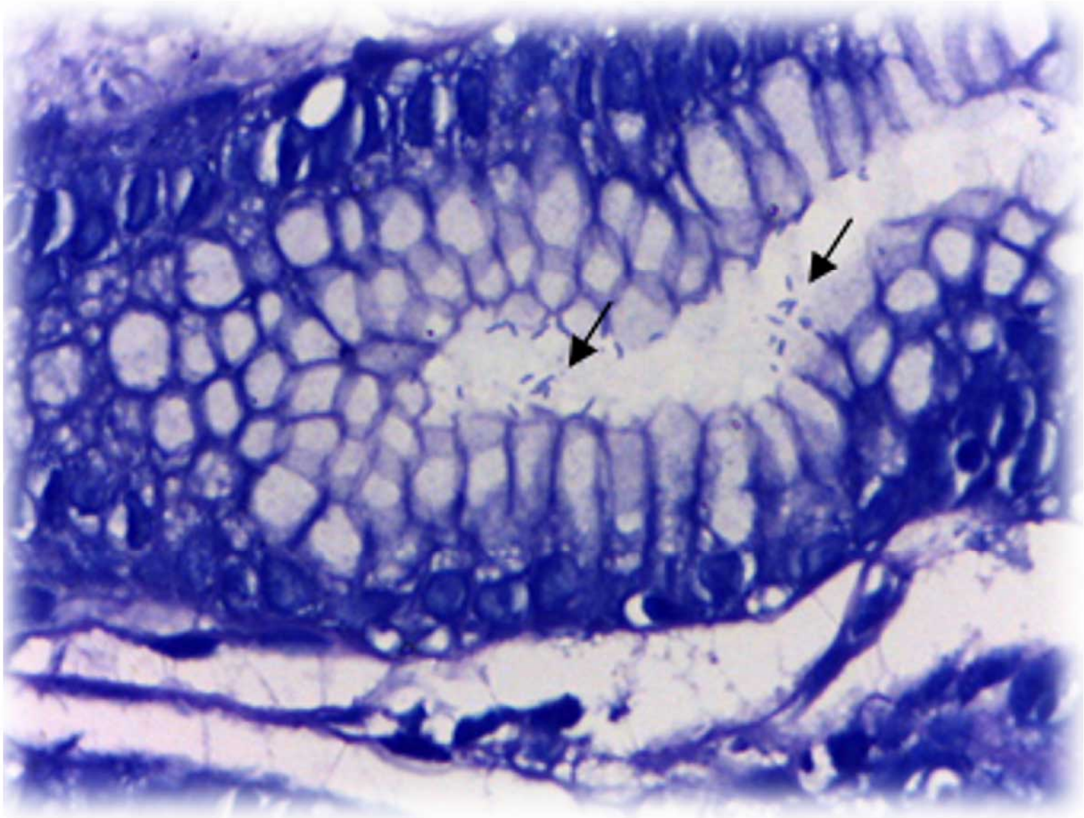


FIGURE 3

REVIEW OF LITERATURE

ANATOMY OF STOMACH

- Stomach is the most dilated part of the alimentary tract intervening between oesophagus and duodenum
- It occupies the epigastric,hypochondrial and umbilical region of the abdomen
- It is J-shaped when its empty and becomes pyriform shaped when partly distended
- Mean capacity is 30ml (birth),1000ml(puberty),1.5-2lit(adult

Stomach has two orifices (cardiac and pyloric), two curvatures(lesser and greater),two surfaces(anterior and posterior)

PARTS OF STOMACH:

- CARDIAC PART: Fundus & Body
- PYLORIC PART : Pyloric Antrum & Pyloric Canal

GROSS ANATOMY OF STOMACH

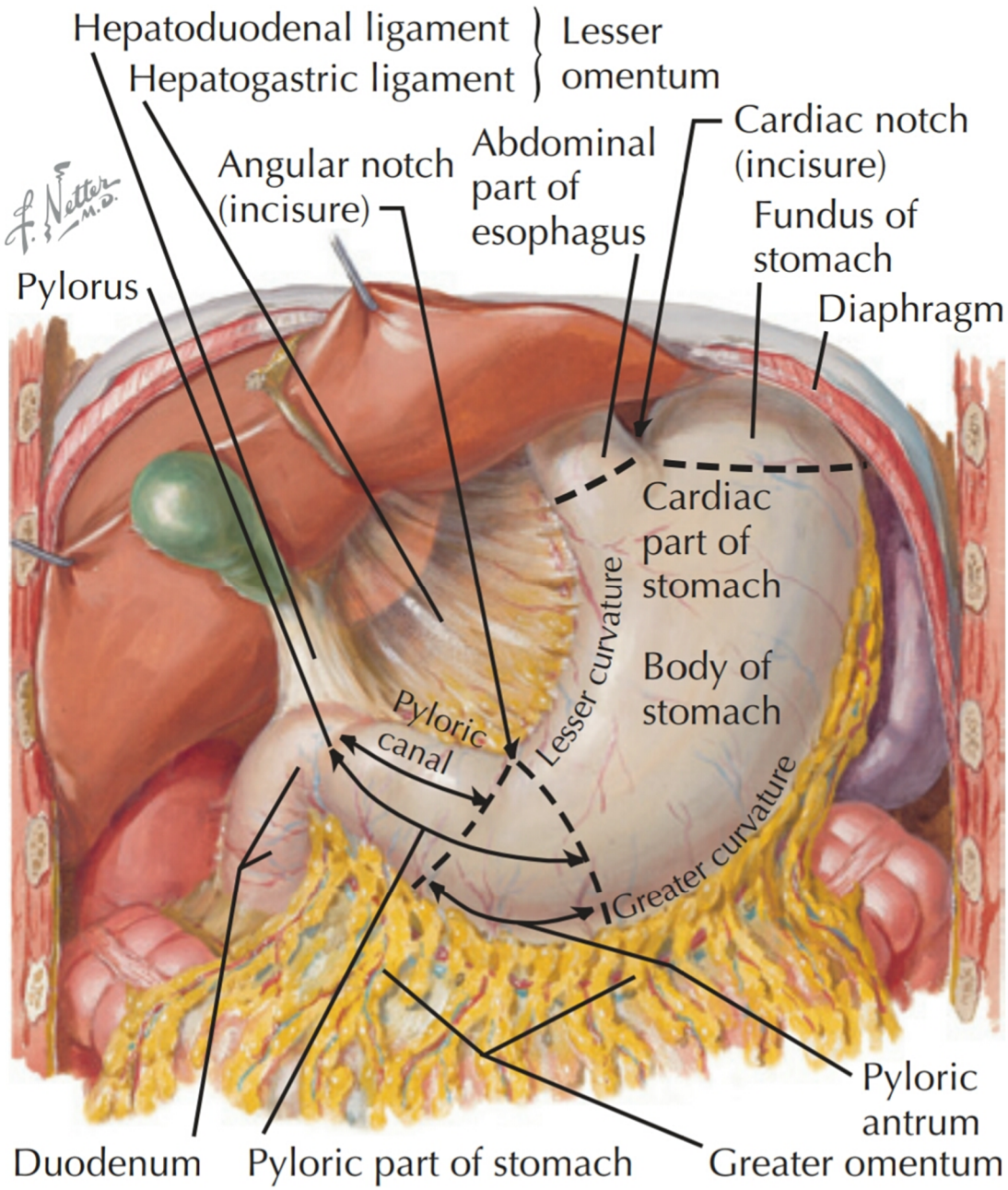


FIGURE 4

2 ORIFICES:

- **Proximal Cardiac Orifice** – located 2 to 3cm left of the midline in the left 7^{th} coastal cartilage
- **Distal Pyloric Orifice** – located in transpyloric line 1.2 cm right of midline

2 CURVATURES:

- **Lesser Curvature** – the most dependent part is marked by angular notch (incisura angularis)
- **Greater Curvature** – a line drawn downward and left from this notch and divides the stomach into 2 parts.

2 ANGLES

- Incisura Angularis
- Angle Of His : angle of entry of esophagus into the stomach which is 30 to 70 degree.
- Fundus is the part above the horizontal line drawn from the angle of his

POSTERIOR SURFACE RELATIONS:(STRUCTES FORMING STOMACH BED)

- Diaphragm
- Left Kidney
- Suprarenal Gland
- Pancreas Body
- Transverse Colon & Splenic Flexure Of Colon
- Splenic Artery

BLOOD SUPPLY TO STOMACH:

ARTERIAL SUPPLY:

The coeliac artery provides blood supply to the stomach

There are 4 main arteries

- **Along lesser curvature**
  - Left gastric artery from coeliac artery
  - Right gastric artery from common hepatic artery.
- **Along greater curvature**
  - Right gastro omental artery from the gastroduodenal artery.
  - Left gastro omental artery and short gastric arteries from the splenic artery .
- **Fundus** supplied by 5-7 short gastric arteries.

VENOUS DRAINAGE OF STOMACH

Veins of the stomach drain into **portal, superior mesenteric and splenic vein**

- Left gastric vein and right gastric vein drain to **portal vein**.
- Short gastric and left gastro epiploic vein drain into **splenic vein.**
- Right gastric epiploic vein drain into **superior mesenteric vein**.

BLOOD SUPPLY OF STOMACH

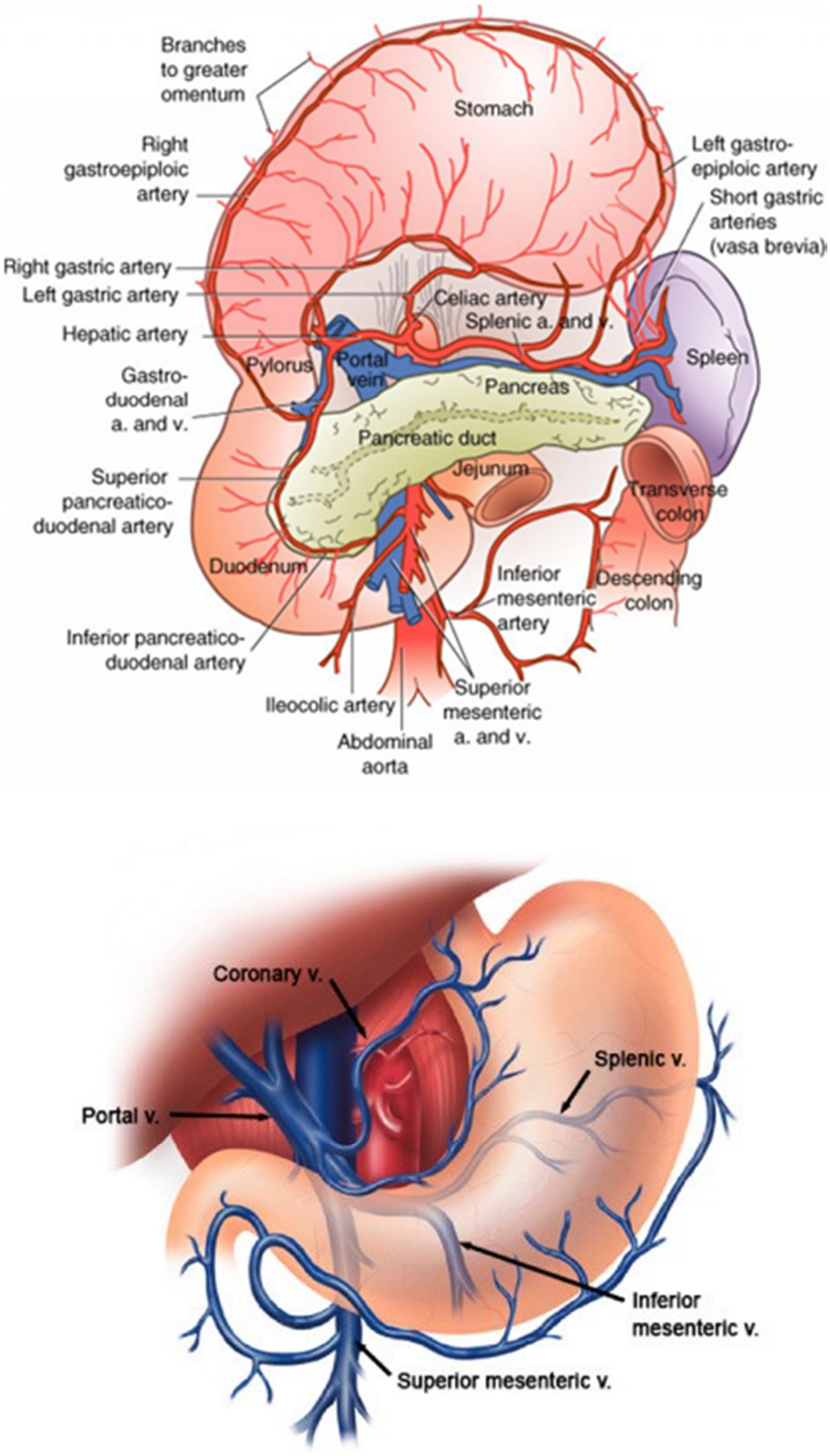


FIGURE 5

NERVE SUPPLY OF STOMACH

- **Intrinsic innervation** occur through myenteric auerbach plexus, submucosal plexus of meissener
- **Extrinsic innervation** occur by
    - Parasympathetic – vagus nerve (left anterior vagus and right posterior vagus. Criminal nerve of grassi is the first branch of Right vagus nerve- it is recognized as the potential cause of recurrent ulcer. Both anterior and posterior vagus gives off splanchnic branches, pyloric branches and nerve of later jet.
    - Sympathetic – coeliac ganglia (T6-T9 spinal nerve segment)

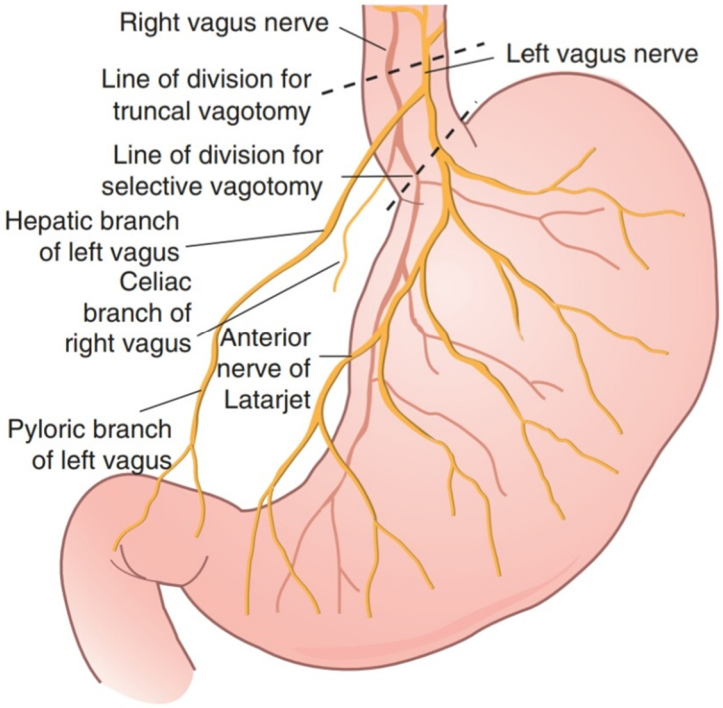


FIGURE 6

LYMPHATIC DRAINAGE :

Lymphatic drainage of stomach is divided into 4 zones

Superior Gastric group (left gastric, paracardial nodes- from upper lesser curvature)

Supra pyloric group (right suprapanceatic nodes- from the antral segments of lesser curvature)

Pancreaticolienal group (left gastroepiploic and splenic nodes-high on the greater curvature)

Inferior gastric subpyloric group (along the right gastro epiploic vascular pedicle)

Finally all lymphatics drains into coeliac group of lymph nodes

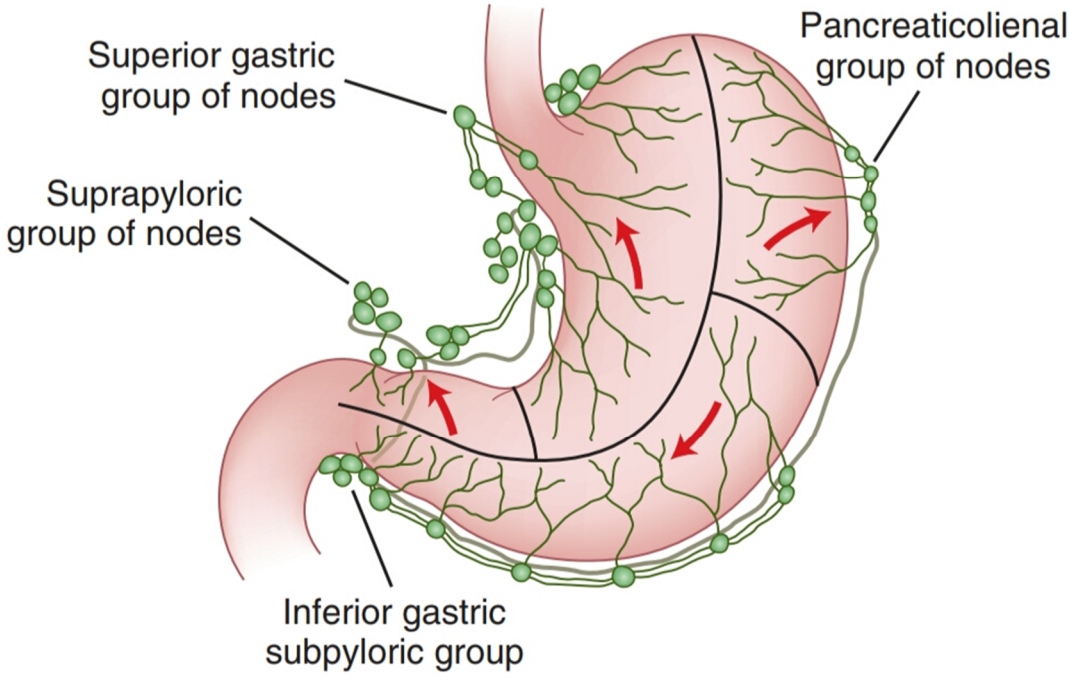


FIGURE 7

ANATOMY OF DUODENUM:

- Duodenum is the widest, shortest and fixed part of small intestine.
- Extending from pylorus to duodeno-jejunal flexure.
- Lies opposite to L2L3 vertebra above the umbilicus
- Curved around the head of pancreas forming a letter C shape

PARTS:

The duodenum is 25cm long and 2.5cm wide which are divided into four anatomic parts.

D1 - the cap / bulb (superior part-5cm)

Lies anterolateral to the L1 body and overlain by liver and gall bladder, ampulla (cap)-first 2cm- bears a mesentery, the hepatoduodenal ligament, part of the lesser omentum

Distal 3 cm retroperitoneal

D2 - descending portion (7.5cm)

Lies along the right side of L1-L3 bodies

Receives the outflow from bile and pancreatic ducts via the hepatopancreatic ampulla (vater) through the greater duodenal papilla (vater)

Receives outflow from accessory pancreatic duct through the lesser duodenal papilla.

D3 - transverse portion (10cm)

Crosses the L3 body and it lies posterior to the main trunk of superior mesenteric artery

D4 - ascending portion (2.5cm)

Lies left of the L3 to upper border of L2 and becomes continuous with D-J flexure.

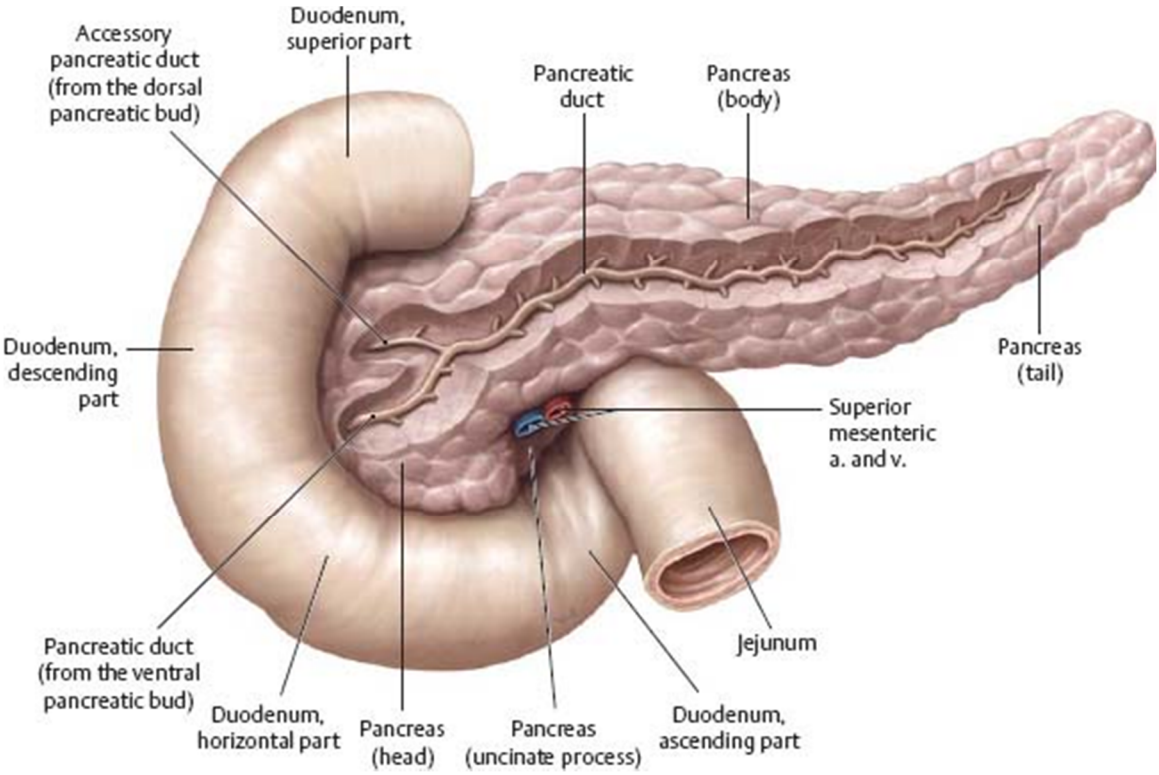


FIGURE 8

ARTERIAL SUPPLY:

The part of the duodenum before the opening of bile duct is supplied by coeliac trunk through superior pancreatico duodenal artery and distal to the opening of bile duct supplied by inferior pancreatico duodenal artery.

1st part of the duodenum receives additional supply from

1. right gastric artery
2. supraduodenal artery of wilke (branch of hepatic artery)

b. retroduodenal branches of gatroduodenal artery

VENOUS DRAINAGE:

Veins drain into splenic vein, superior mesenteric vein and portal vein.

LYMPHATIC DRAINAGE:

Flows through -pancreatic duodenal nodes, hepatic nodes, superior mesenteric nodes and ends up to intestinal lymph trunk- cisterna chyli.

NERVE SUPPLY:

Sympathetic nerve from Thoracic ninth to tenth spinal segments

Parasympathetic nerve-vagus through coeliac plexus to reach dudodenum along the arteries.

ARTERIAL SUPPLY:

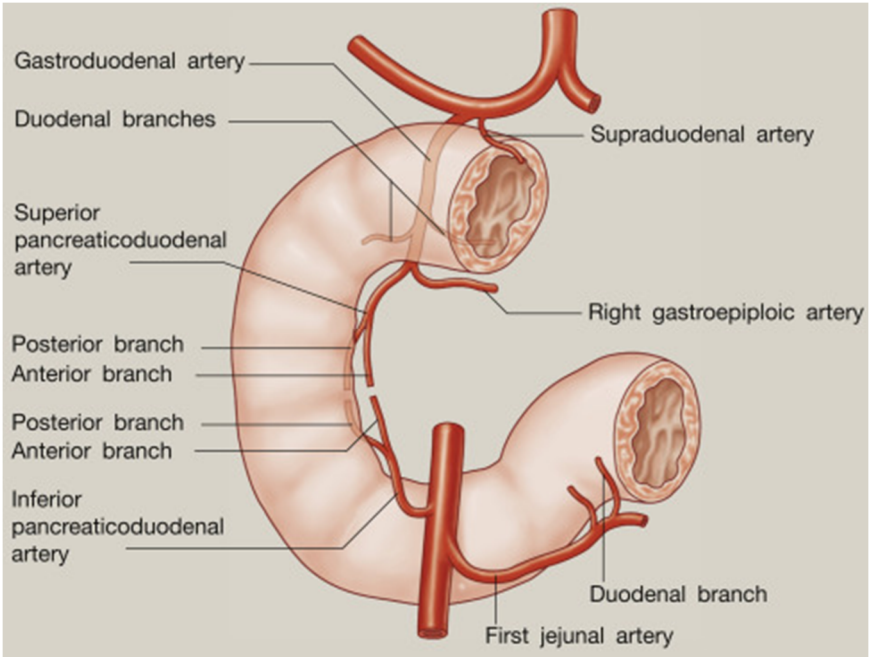


FIGURE 9

VENOUS DRAINAGE:

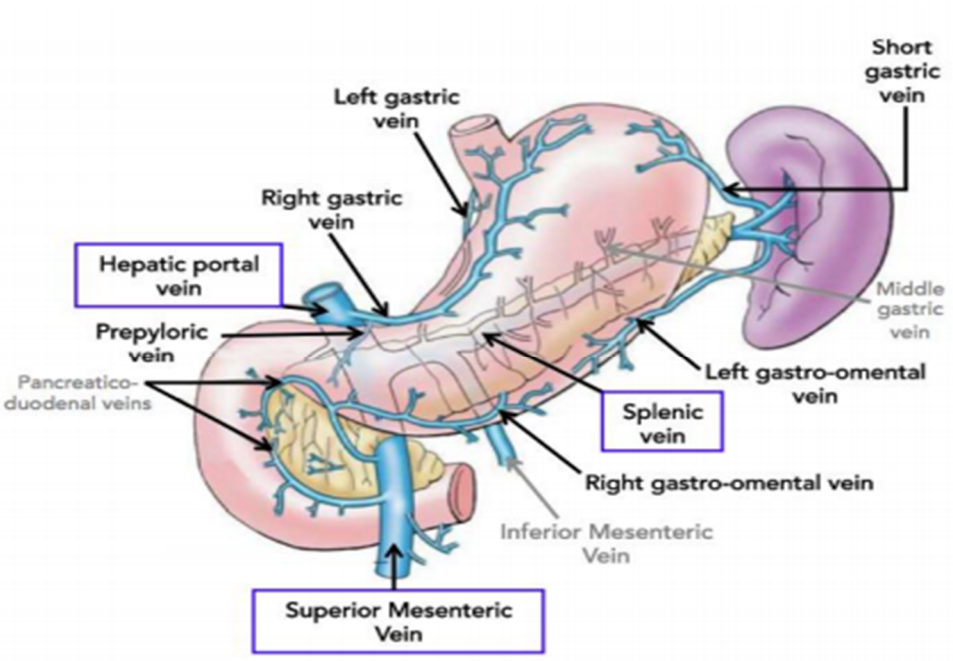


FIGURE 10

PEPTIC ULCER DISEASE:

These are multiple erosions which occurs in the stomach or duodenum due to disruption of mucosal barrier which extends to the muscularis mucosae. This can be divided in to two subtypes

- **gastric ulcer**
- **duodenal ulcer**

The most important etiological factor being commonly associated

1. **H.pylori**
2. Stress
3. drugs like analgesics
4. steroids
5. surgeries

HELICOBACTER PYLORI:

Marshall & Warren from Australia in 1982, cultivated a organism of spiral shaped similar to Campylobacter which colonizes the stomach of human is present in patients with type – B gastritis (chronic inflammation of stomach antrum).

It is named as **campylobacter pyloridi are** and then changed to the campylobacter pylori. Since the organism was supposed to be considered of taxonomical variant from other campylobacter species, due to the presence of

sheathed flagella, a different respiratory quinines, a unique fatty acid profile, and a different 16s RNA sequence, the they created a new **genus Helicobacter**.

It is more common in the lower socioeconomic group.

H. pylori is associated mainly with peptic ulcer disease, type – B gastritis, gastric associated lymphoid tissue (MALT) B- cell lymphomas, gastric adeno carcinoma.

Duodenal ulcer - 95%

Gastric ulcer - 70%

Gastritis - 70 – 90%

MECHANISM RESPONSIBLE FOR GI INJURY:

H. pylori releases **UREASE** the enzyme which hydrolyses urea & releases ammonia which via negative feedback mechanism there by it increases gastrin release from G – cells.

It also secretes enzyme **DEHYDROGENASE** (which converts alcohol to aldehyde a toxic metabolite to the mucosa), **ENDOPEPTIDASE** (which intervene the mucosal barrier).

Urease the enzyme released will create alkaline environment around it in mucus layer of gastric epithelium. It will survive only in gastric epithelium /gastric metaplasia in the duodenum/Barrett's oesophagus/ and in heterotrophic

gastric mucosa in meckel's diverticulum/rectum since the receptors available only in gastric mucosa for these organism to get adhered.

H.pylori affects the mucosal healing and degranulates eosinophils. It secretes cytotoxins such as (cag A and vac A) causing inflammatory reaction or malignancy. . It releases proteases & lipases which breaks mucus & the barrier.

HABITAT :

In stomach, present only in the mucous layer of gastric epithelium mostly involving the antrum (humans)

BLOOD :

H. pylori also identified from the patient having malignant lymphoma of the upper GIT including stomach via blood culture.

FAECES:

Polymerase chain reaction are done to identify the H.pylori in faeces.

ORAL CAVITY:

H. pylori occasionally were isolated from the saliva and also from dental plaques.

ANIMAL SOURCES: Rhesus monkey, baboon, pigs& domestic cats

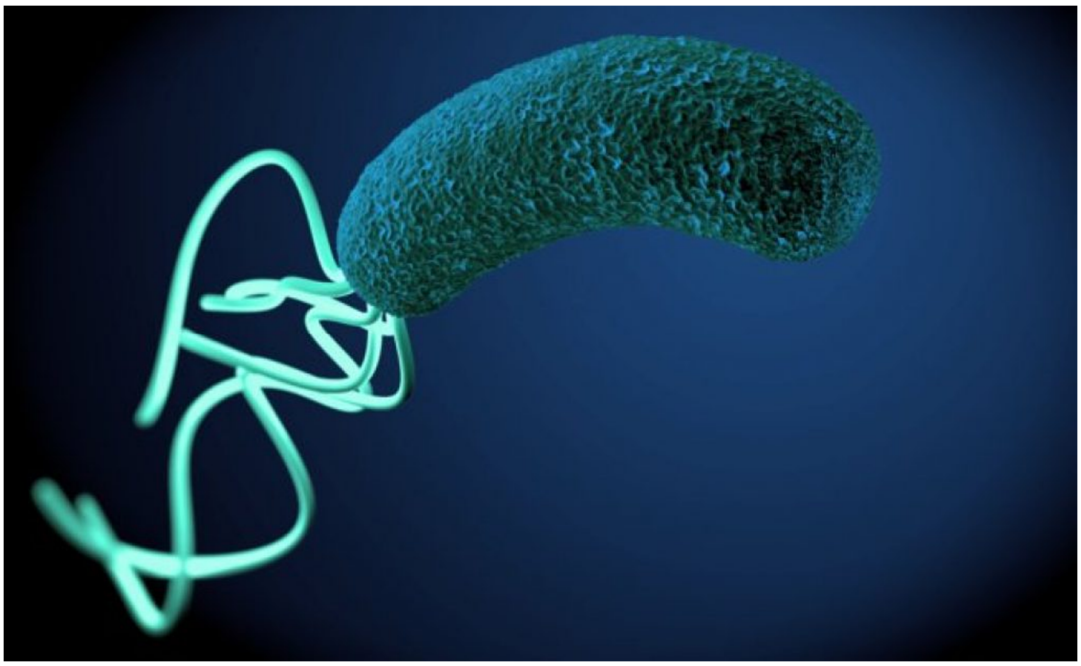


FIGURE 11

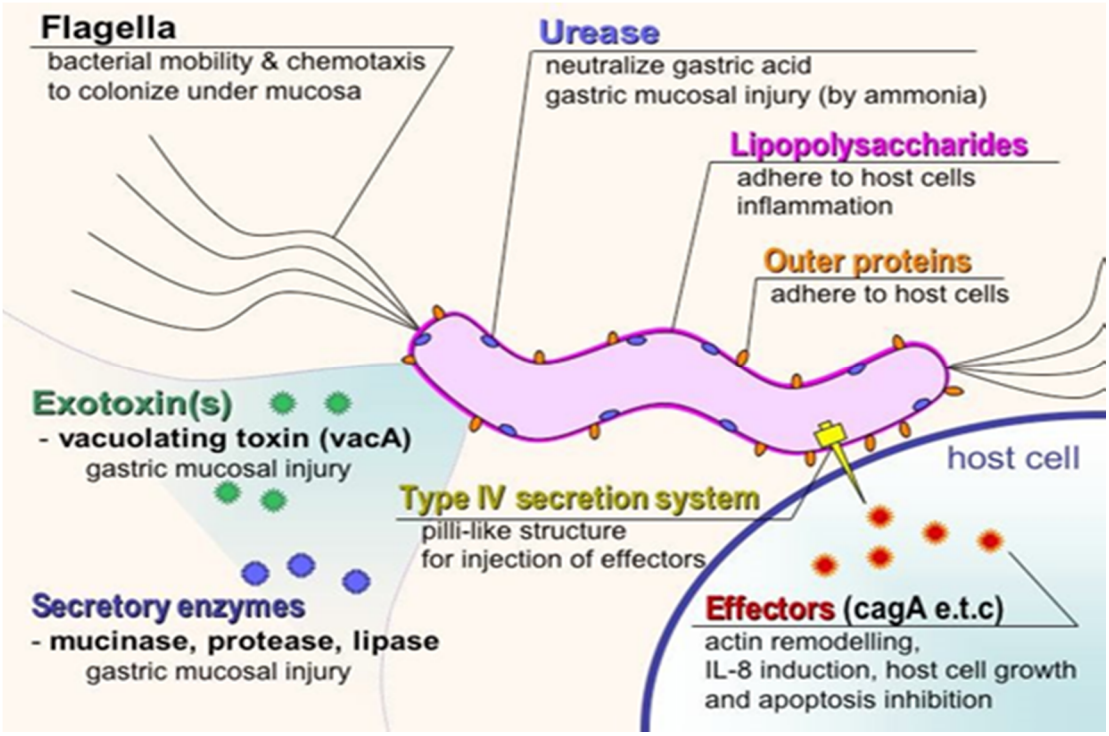


FIGURE 12

FEATURES :(microbiological)

Cellular features :

- H. pylori is a gram negative rod shaped flagellated organism of (0.5 - 0.9 micro meters wide and 2-4micro meters long).
- Has 1 to 3 turns when observed in vivo.
- In light microscopy, Helicobacter cells have about 5-6 polar (lophotrichous) flagella.
- Motile via polar flagella and produce large amount of urease

PHYSIOLOGICAL FEATURES :

- H. pylori are microaerophilic organisms
- It grows well in an atmosphere (5% O2 with 5 - 10% CO2), and on blood containing media like “brain heart infusion agar” (BHI).
- BHI has 5% horse blood agar with 1% Isovitalex.
- Temperature range of 33 to 40 degree Celsius.
- pH 5.5 – 8.5.

BIOCEMICAL FEATURES :

Positive Features	**Negative Features**
Urease Production	Nitrate reduction
Cytochrome Oxidase	Hippurate hydrolysis
Catalase production	Carbohydrate Oxidation / fermentation

VIRULENCE FACTORS:

Heat shock protein

Cag A and Vac A proteins

Leukocyte recruitment and activating factors

Lipopolysaccharide (LPS)

UREASE SPECIFIC FEATURE:

Urease contains nickel which will digests urea, there by bypasses freely from plasma to the gastric juice producing AMMONIA.

$NH_2\text{-}CO\text{-}NH_2 + H_2O \rightarrow CO_2 + 2NH_3$

At neutral pH

$NH_3 + H_2O \rightarrow NH_4OH \rightarrow NH_4^+ + OH^-$

H. pylori infected patients, has less urea and more ammonia than the normal patients in gastric juice.

Ammonia released by H. pylori due to the Urease activity it buffers the bacterial hydrogen ions which is present in gastric acid juice, so that providing a source of nitrogen for H.pylori.

WAYS OF SPREAD:

- Faeco-Oral route- it is seen in subjects consuming uncooked vegetables

- Oro-Oral route-Rate of infection in children affected from H.pylori positive parents due to presence of the similar strain in the family member which suggest, close contact is one of the important modality for the spread of infection
- Gastro-Oral route: aspirated gastric juice, this route may account for endoscopist ,nurses and theatre staff

MANAGEMENT OF H.PYLORI INFECTION:

INVESTIGATIONS:

INVASIVE METHODS:

- Rapid urease test (CLO / Campylobacter like organism Test)
- Histology
- Culture

NON INVASIVE METHODS:

- Serology
- Urea breath test

RAPID UREASE TEST:

- It is a rapid diagnostic test which is used for H.pylori which is based on the enzyme urease
- Tissue sample is put into a solution containing urea, with (phenol red) as a pH indicator and a gel which contains bacteriostatic agent

- Result -If H. pylori are present, urease will hydrolyses the urea and releases two molecules of ammonia and one molecule of carbon dioxide there by raising the pH and alkalinising the medium which is appreciated by colour change (yellow colour to pink)

- The test has High specificity (95-100%) and good sensitivity (90-100%)

- False negative test seen in atrophic gastritis with achlorhydria

HISTOLOGY :

- Giemsa staining
- Standard haematoxylin and eosin stain
- Warthin – Starry Stain
- Gretna stain
- Alcian blue

H.PYLORI

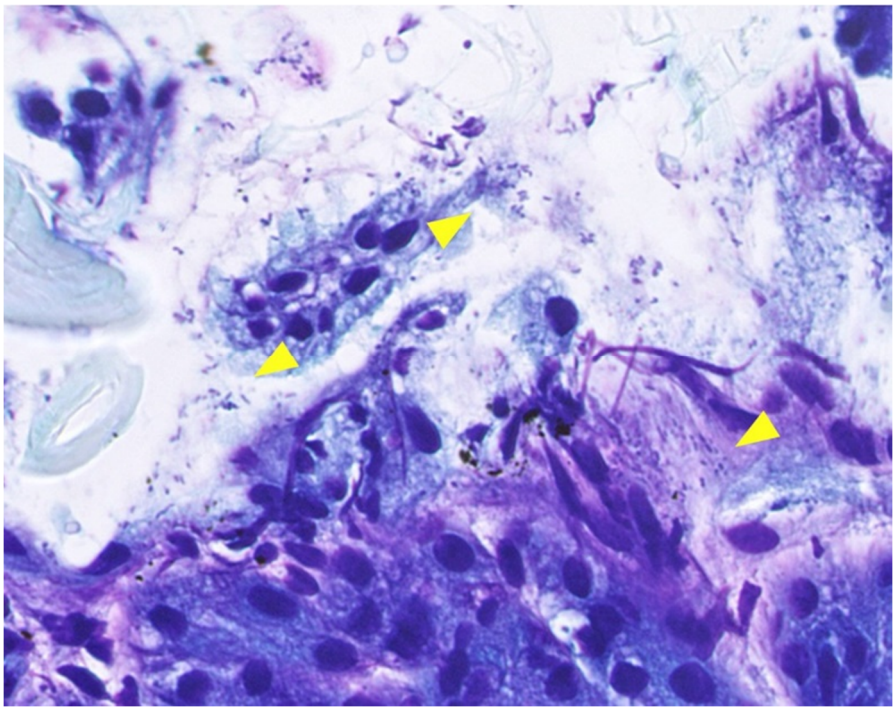


FIGURE 13

CULTURE:

It can be isolated in culture if the specimen is inoculated into an enriched medium which is supplemented with blood, hemin or charcoal and further incubated in a microaerophilic atmosphere for upto 2 weeks.

POLYMERASE CHAIN REACTION :

- It is used to detect H. pylori in the fecal samples. PCR is more sensitive on comparing all other culture methods since it does not depend on bacterial viability
- Limiting factors are inhibitors of amplification reaction in feces.

SEROLOGY / ELISA:

Serological test can be done for detection of IgG,IgM and IgA antibodies. The systemic IgG response is most commonly used parameter to detect the H.pylori infection.The advantages of serological test are non invasive ,inexpensive, easy technique and not affected by prior antibiotics and PPI and it has high sensitivity and specificity compared to invasive test and urea breath test

UREA BREATH TEST:

- Urea along with C 13 which is a non-radioisotope , is hydrolysed in the stomach by the enzyme urease secreted from H. pylori to produce two molecules of ammonia and one molecule of carbon dioxide which 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].

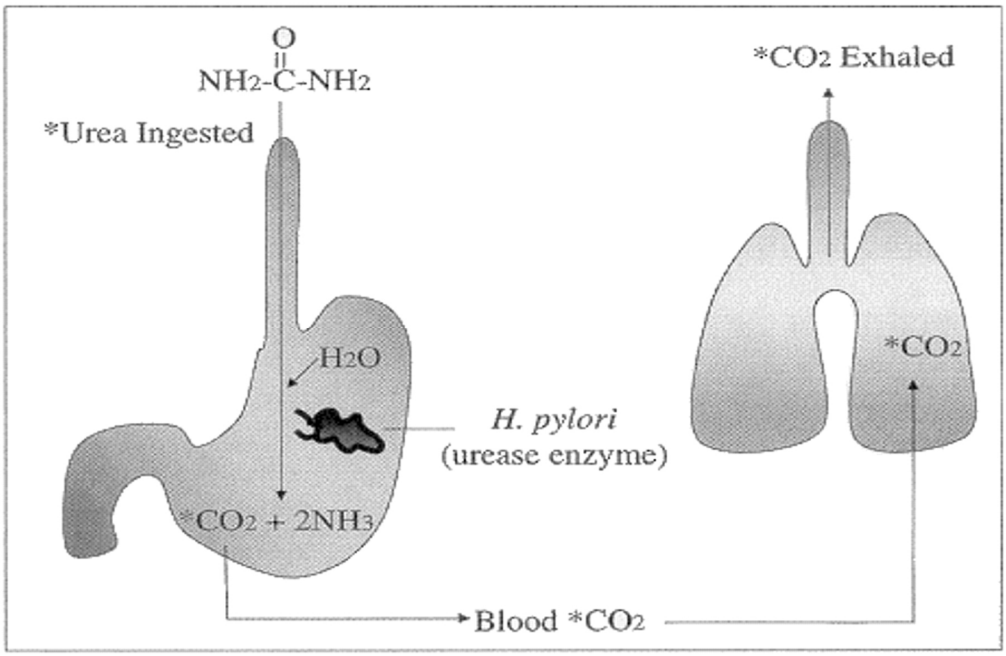


FIGURE 14

TABLE 1 : SENSITIVITY, SPECIFICITY AND COST OF DIAGNOSTIC TESTS FOR H.PYLORi

Methods	**Sensitivity (%)**	**Specificity (%)**	**cost**	**invasive**
Culture	98	100	++++	Yes
Histology	98	99	++++	yes
CLO	90	100	+++	Yes
Breath	100	100	++	No
Serology	98	96	+	No

TREATMENT OF H.PYLORI:

ERADICATION REGIMEN:

Triple therapy

2 antibiotics and one proton pump inhibitor for 7-14 days.

- PPI (omeprazole 20 mg bid or rabeprazole 20mg bid or lansoprazole30 mg bid or pantoprazole 40mg bid)
- amoxicillin 1 g bid
- clarithromycin 500mg bid
- metronidazole 500mg bid (can be used instead of amoxicillin)

Quadraple therapy : includes PPI plus bismuth subsalicylate with tetracycline and metronidazole incoporated

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%.

PEPTIC ULCER DISEASE:

Peptic ulcer disease is caused by increased aggressive factors and decreased defensive factors or both, this in turn leads to mucosal damage and ulceration. Most majority of peptic ulcer disease is caused by H.pylori infections or NSAIDS

Peptic ulcer disease can be gastric or duodenal ulcer

GASTRIC ULCER:

Gastric ulcers usually present on the lesser curvature near the Incisura about 60 % and these ulcers(type 1 ulcer) are not associated with excessive acid secretion with low to normal acid output. In contrast, type 2 gastric ulcers(15%) are located in the body of the stomach in association with duodenal ulcer. Type 3 ulcers are prepyloric ulcers and account for about 20% .Type 2 and 3 ulcers are associated with excessive acid secretions. Type 4

ulcers occur near the OG junction and account less than 4 % and associated with normal acid level

Gastric ulcers occur in age between 55-65 years ,low socioeconomic class and female to male ratio of 2:1. Presence of acid is essential to produce gastric ulcers ,so rapid healing occurs following an effective Antacids therapy

NSAIDS induced ulcers are most often found in the stomach ,so ingestion of NSAIDS remains as the important factor in ulcer pathogenesis by breaking the mucosal barrier and the risk of bleeding and ulceration is directly proportional to the dosage of NSAIDS especially in the older age group(more than 60) and patient with concurrent use of steroids or anti-coagulant. NSAIDS users have a two to ten fold increased risk for GI complications

ETIOLOGY:

1. **H.pylori(75%)**
2. atrophic gastritis
3. gastric stasis
4. duodenogastric bile reflux
5. abnormalities in acid and pepsin secretion
6. smoking
7. alcohol
8. NSAIDS

9. Steroids

10. Low socioeconomic group

FACTORS RESPOSIBLE OF GASTRIC ULCER:

- Due to gastric stasis- acid stagnant leading to mucosal eruption
- Duodenogastric reflux- bile and lysolecithin breaks the mucosa barrier
- Type 2 and 3 gastric ulcers leads to hypersrection of acids

PATHOPHYSIOLOGIC ABNORMALITIES IN GASTRIC ULCER:

- Increased serum levels of pepsinogen2
- Increased duodenogastric reflux
- Decreased gastric parietal cell mass
- Decreased maximal acid output

MORPHOLOGY:

GROSS MORPHOLOGY:

Margins of benign gastric ulcers are clear(in lesser curvature) ,edges are not everted with gastric mucosal folds which converges to the base of ulcer

HISTOLOGICAL MORPHOLOGY:

Shows ulcer crater containing inflammatory cells and granulation tissue and epithelial proliferation and end arteritis obliterans.

CLINICAL FEATURES:

1. Equal in both sex, being more commonly associated with females
2. Above the age of 40yrs
3. **EPIGASTRIC PAIN AFTER TAKING FOOD** which last upto 2 hours and **relieved by vomiting**
4. Has symptom free interval of 2-3months
5. Haemetemesis is more common
6. Loss of weight
7. Good appetite but once full blown complication occurs appetite will decrease

COMPLICATIONS:

- **PERFORATION** is the most common complication of gastric ulcer(into the lesser sac)
- Hour glass contracture(due to cicatrisation of lesser curvature)
- Tea pot deformity(due to cicatrisation and shortening of lesser curvature)
- Bleeding (due to involvement of LGA)
- Penetration(posterior to pancreas & anteriorly to liver)
- Malignant transformation- ADENOCARCINOMA (5-10%)

DUODENAL ULCERS:

Duodenal ulcers occurs most commonly in the duodenal cap region and acid should be there for non-malignant ulceration involving the upper GIT. There is a large overlap of acid levels between the ulcer patients and normal subjects. Almost 70% of duodenal ulcer patients have a normal acid output.NSAID induced ulcers are mostly found in the stomach on comparing to H.pylori induced ulcers are most commonly found in the duodenum.

ETIOLOGY:

- **H.pylori (95%)** is the important etiological factor
- Stress
- Anxiety (hurry,worry,curry)
- NSAIDS
- Steroids
- Smoking
- Alcohol
- Vitamin deficiency
- Endocrinological factors: Zollinger elison syndrome, MEN 1 ,Cushing syndrome, hyperparathyroidism
- Chronic pancreatitis
- Cirrhosis

PATHOPHYSIOLOGICAL ABNORMALITIES IN DUODENAL ULCER:

- Decreased duodenal HCO3 secretion (most common)
- Increased nocturnal acid secretion
- Increased duodenal acid loss
- Increased day time acid secretion
- Increased gastric emptying
- Increased pentagastrin -stimulated Mean acid output
- Decreased PH inhibition on acid release

MORPHOLOGY:

GROSS MORPHOLOGY: The serosa of the involved duodenal ulcer part shows petechial haemorrhages along with speckled red dots looking like a sprinkled -**CAYENNE PEPPER**

HISTOLOGICAL MORPHOLOGY:

- Ulcer along with chronic inflammatory cells with granulation tissue
- Gastric metaplasia of duodenal mucosa
- End arteritis obiters

CLINICAL FEATURES:

1. DUODENAL ULCER incidence is more comparing to gastric ulcers in INDIA
2. More common in TYPE A personality people
3. More common in males
4. **PAIN produced in the early morning, decreased after food intake (occurring before food (HUNGER PAIN)**
5. Malena is more common
6. Periodicity with seasonal variation occurs
7. Heart burn, water brash and vomiting is present
8. Appetite is good- eats frequently and gains more weight

COMPLICATIONS OF DUODENAL ULCER:

1. Intractable duodenal ulcer
2. Bleeding
3. Perforation
4. Obstruction (Rule out malignancy/ pyloric stenosis)
5. Residual abscess
6. Penetration into pancreas

PEPTIC ULCER PERFORATION:

It may occur in both Duodenal and Gastric ulcer

Due to perforation, the gastroduodenal contents will spill into the peritoneal cavity which will result to gross chemical peritonitis. In the early stage visceral contents are sterile ,so infective peritonitis is very rare ,but the features and prognosis depends on the general condition of the patient.

CLASSIFICATION OF PERFORATION:

- Acute perforation
- Subacute perforation
- Chronic perforation
- Perforation associated with haemorrhage
- Perforation of intrathoracic gastric ulceration
- Pseudoperforation

1. ACUTE PERFORATION:

Here the ulcer perforate and occurs the spillage of gastric and duodenal contents into the peritoneal cavity there by resulting to Chemical Peritonitis

Clinical features depends on the stage of perforation.

Three stages of perforation, each of variable duration are as follows

a. Primary stage or stage of peritonism

b. Secondary stage or stage of peritoneal reaction

c. Tertiary stage or stage of bacterial peritonitis

1. Primary stage:

The clinical course in perforation is classical. Vigorous agonizing pain starts in the epigastrium or right hypochondrium first and later turns into a generalized pain.

The patient then becomes prostrated. Symptoms are due to irritation of the peritoneum by gastroduodenal contents spillage.

Pain shock may occur. Abnormal temperature, cold peripheries, sweating, palpitation, radiation of abdominal pain to both the shoulders as the diaphragm gets irritated, pale face.

Patient will assume a classical rigid posture by lying with legs updrawn and hands will be held tensely by his side. Temperature will be subnormal, as low as 95 – 96 degree or normal. Pulse rate may be normal or raised above 90 per minute. Shallow Respiration with increased respiratory rate may be there.

Per abdomen examination reveals, restricted or no movements of abdomen with respiration with prominent rectus muscle. On palpation warmth and diffuse tenderness is noted. Guarding and rigidity will be present. Bowel sounds will be absent on auscultation

The stage of which lasts for 3 – 6 hours.

The Perforated fluid may leak into Right Para colic gutter which will cause inflammation and pain, there by mimicking the features of acute appendicitis.

2. Secondary stage:

Transition time from the primary stage to secondary stage will be taking around 3-6 hours which will be depending on the size and site of perforation and amount of peritoneal soiling.

During this stage the spontaneous sealing of perforation may occur. Gross leakage of gastroduodenal contents, will lead the patient to stage of septic peritonitis which will rarely exceeds 6 hours.

Here the pain is lessened markedly with general condition improvement. Because of this stage of reaction it has sometime called **stage of delusion** and it is in this stage most of error in diagnosis takes place.

On examination varying amount of rigidity of abdomen and tenderness will be present with Bowel sounds are infrequently heard or absent

3. Tertiary Stage :

Stage of diffuse peritonitis which begins about 12 hours of perforation and lasts for about 24 hours until it passes Into final stages of paralytic intestinal obstruction. Gross contamination by Pathogenic organisms is likely here.

Peritoneal fluid will become purulent with bowel loop distension with fluid and gas. Intestinal movements diminish and finally disappear with onset of paralytic ileus.

The clinical features are same as those of generalized peritonitis from any other cause with less Pain ,frequent vomitus, hiccoughs which may further depress the patient.

Sweating, vomiting, outpouring of fluid into peritoneal cavity, distended paralysed intestine, dehydration and electrolyte imbalance becomes more severe.

Patient will complaints of intense thirst, elevated temperature, dry & coated tongue, rapid thread pulse, shallow and rapid respiration.

Abdomen is distended, guarding still persists. On auscultation occasional tinkles heard. The typical Hippocratic facies denote that end is not far off

Patient drifts into toxaemic stage, dehydration and circulatory failure. Patient will die usually 4-5 days after perforation

2. SUBACUTE PERFORATION :

The ulcer may perforate but the peritoneum will seal rapidly before there is spillage of duodenal contents, into general peritoneal cavity. There will be sudden onset of acute abdominal pain more to the right upper quadrant. Respiration will be shallow and on deep inspiration it may be associated with an abrupt catch in the breath

On examination, there is localised tenderness and rigidity, leaving behind the rest of the abdomen soft on palpation and non-tender– ray film usually reveals only a small amount of gas under diaphragm. Patient's symptoms will usually subside in half an hour to 2 hours . Rarely it extend and the signs of an acute perforation develop

3. Chronic perforation :

It occurs when an ulcer perforated into an area which is walled off by adhesions or by adjacent viscera like liver, colon or greater omentum or it may also occurs when gastric ulcer perforates into omental sac there by leading to a chronic abscess there by making the diagnosis confusable.

Since these patients doesn't present with classical signs and symptoms of peritonitis, they are seldom diagnosed as peptic ulcer perforation. Irregular temperatures, rigors, leucocytosis, dullness at the base of the lung, Consequent pleural effusion or basal congestion will lead to the diagnosis of sub phrenic abscess, containing gas and diaphragm is raised and fixed on right side.

USG of abdomen -most reliable investigation of choice for intraperitoneal abscess

4. Perforation associated with haemorrhage:

Perforation with massive haemorrhage is grave but fortunately a very rare complication. It will occur in one of the three ways

a. Haemorrhage and perforation occurring concomitantly

b. Haemorrhage following a recently sutured perforation

c. Perforation occurring during the medical treatment of haemorrhage

The clinical features will be similar to acute perforated peptic ulcer with signs of haemorrhage

5. Perforation of an intra-thoracic gastric ulcers:

An extremely rare variety of perforation. Here the ulcer will be in hiatus hernia, which will be fixed in the mediastinum. Unless we identify the hiatus hernia, it will be extremely difficult to make a proper pre – operative diagnosis.

Since the symptoms and signs will be pointing an intra thoracic lesions such as coronary thrombosis, acute pericarditis, pulmonary embolism.

Rare type Perforated peptic ulcer :

Peptic ulcer in meckel's diverticulum in intestinal duplication may occasionally perforates. Simultaneously multiple perforations occur in less than one percent of all cases.

CLINICAL FEATURES OF PEPTIC ULCER PERFORATION:

AGE :

Duodenal ulcer perforation is rare before adolescence, common in 30-40 years age group and it is the most common type.

Gastric ulcer Perforation the mortality is very high

SEX:

More common in men than in women

HISTORY OF PRESENT ILLNESS :

Time on onset : the patient is able to exact the time of onset of perforation, most commonly after an exertion

Mode of onset : sudden

Pain : Pain is extremely vigorous in the abdomen ,more in the epigastrium and then spreads all over the abdomen

Shifting of pain : shift to right iliac fossa since the fluid flows threw the right paracolic gutter in order to settle in the right iliac fossa ; thus mimicking features of acute appendicitis

Radiation of pain : pain in peptic ulcer perforation is referred to the tip of right shoulder

Nausea : may be present

Vomiting : early stage -reflex vomiting will be there since because of irritation of nerves in peritoneum and mesentery. Later stage- vomiting occurs because of toxin acting at the level of medullary centres and because of this paralytic ileus occurs. The vomitus contains undigested food materials & blood occasionally when haemorrhage is seen.

Abdominal distension:patient may present with significant abdominal distension in late stage of perforative peritonitis.

Micturition: oliguria will be seen in shock patients.

PAST HISTORY :

Dyspepsia will be seen in 80% of cases. Rest of all the cases, perforation will be the very first clinical manifestation of a silent duodenal ulcer

PHYSICAL EXAMINATION:

General appearance : Initially during the shock of perforation, patient will present with pale face with sweating.

Decubitus : supine, rigid and immovable, staying in the similar posture refusing any movements.

Pulse : normal at early perforation (beginning), rapid and thready when full blown peritonitis sets in.

Respiration : early stage no change occurs,it becomes rapid & shallow when full blown peritonitis sets in or it may also seen in perforation with haemorrhage .

Temperature : initially normal, rises with onset of peritonitis.

Tongue : moist initially and becomes dry and brown when peritonitis sets in.

EXAMINATION OF ABDOMEN :

Respiratory movements : thoracic movement dominates over the abdominal movements along with respiratory effort

Rigidity of abdomen : constant, continuous rigid abdomen is characteristic which occurs due to reflex contraction of the abdominal muscles and it will be predominantly seen in the epigastrium and right hypochondrium.

Liver dullness : liver dullness obliteration is characteristic of this abdominal condition usually evident in the second stage.

Free fluid : free fluid is present .

Rectal examination :

Fullness noted in rectovesical or rectovaginal pouch in late stage.

INVESTIGATIONS :

1. **X-ray abdomen erect:**

   Plain X – ray erect of the abdomen demonstrates free air under the diaphragm.

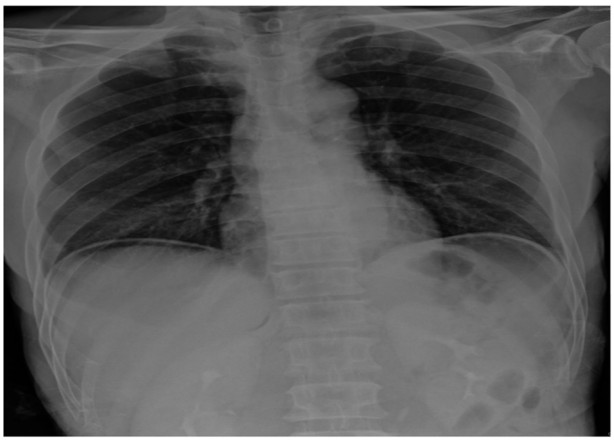


FIGURE 15

X-RAY SHOWING AIR UNDER DIAPHRAGM

2. **Gastroduodenogram:**

Following injection of 60ml of gastrograffin via the nasogastric tube X-ray abdomen erect taken.

INFERRENCE: dye escapes via the perforation, thereby enabling to demonstrate the site and size of perforation, chronicity, associated gastric ulcer and other second ulcer.

3. **Ultrasound examination:**

Performed with multi frequency probe (3.5 – 5 MHz)

Intra peritoneal free fluid along with decreased intestinal peristalsis is considered be a finding of perforation indirectly.

4. **Computerized tomographic examination :**

CT – scanning is rarely required for diagnosis as clinically it is easily diagnosed. However some patients with perforated duodenal ulcer on steroid therapy or hospitalized bed ridden patients for other abnormalities will develop occult causes of abdominal pain and sepsis. At that time Gatrograffin swallow and / or CT scanning may be required to determine the cause on occult abdominal sepsis

5. **Serum amylase :**

Normal value of serum amylase is 80 – 180 somogyi units. Above 200 units is considered pathological. Mortality is high when it is seen in peptic ulcer perforation.

Helicobacter Pylori infection diagnosis :

The diagnosis of helicobacter pylori infections is done by following different methods

i. Non – invasive : a. serology – ELISA

b. urea breath test

ii. Invasive test : a. rapid urease test

b. histology

c. culture

6. **Abdominal paracentesis :**

Four quadrant paracentesis to be done.

DIFFERENTIAL DIAGNOSIS:

- acute medical conditions
- acute surgical conditions

ACUTE MEDICAL CONDITIONS :

- Acute pericarditis
- Coronary thrombosis
- Lobar pneumonia
- Pleurisy
- Gastric crisis or tabes dorsalis
- Acute alcoholism

ACUTE SURGICAL CONDITIONS :

- Acute pancreatitis
- Ruptured ectopic gestation
- Acute gastritis
- Perforated typhoid ulcer
- Acute exacerbation of duodenal ulcer
- Peritonitis from acute appendicitis
- Biliary colic in acute cholecystitis
- Acute intestinal obstruction
- Ruptured aortic aneurysm

SURGICAL TREATMENT :

OPEN PROCEDURES :

1. **SIMPLE CLOSURE :**

   - Most commonly performed surgery for peptic ulcer perforation is simple closure with an omental onlay patch closure
   - Surgery is described by Graham
   - It is performed through upper midline Incision
   - After lavaging the peritoneal cavity of purulent or bilious fluid. Perforated ulcer site must be visualized and three commonly or may be occasionally four-zero (3-0 / 4-0) silks sutures should be placed in the ulcer edge, passing through the wall of duodenum and it should be 0.5 – 1 cm from edge of perforation
   - Omentum will be laid over the simple closure and secured.
   - Suture must be applied long the long axis of the gut in order to avoid luminal narrowing
   - Additional sutures can be placed as a plication for omental catch

2. **Cellan Jones technique, Graham technique :** free omental flap is used here. Greater omentum is used as a sealer for perforation closure
3. **Omental drag into perforation site and plugging it into the Ryle's tube**
4. Rectus muscle use in order to seal perforation

DUODENAL PERFORATION

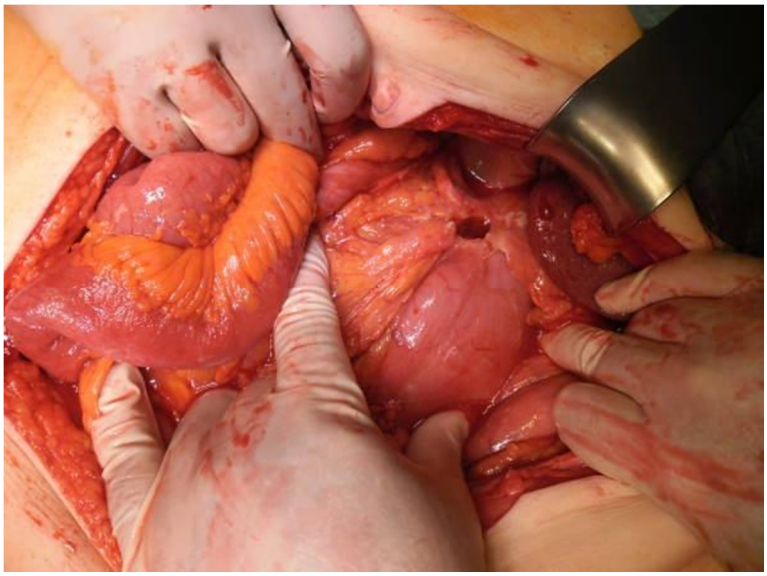


FIGURE 16

GASTRIC PERFORATION

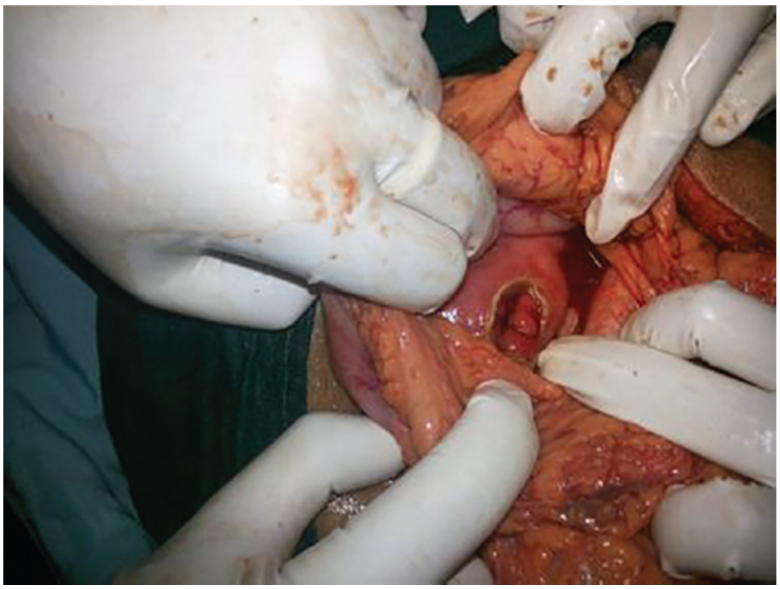


FIGURE 17

PERFOATION CLOSURE METHOD

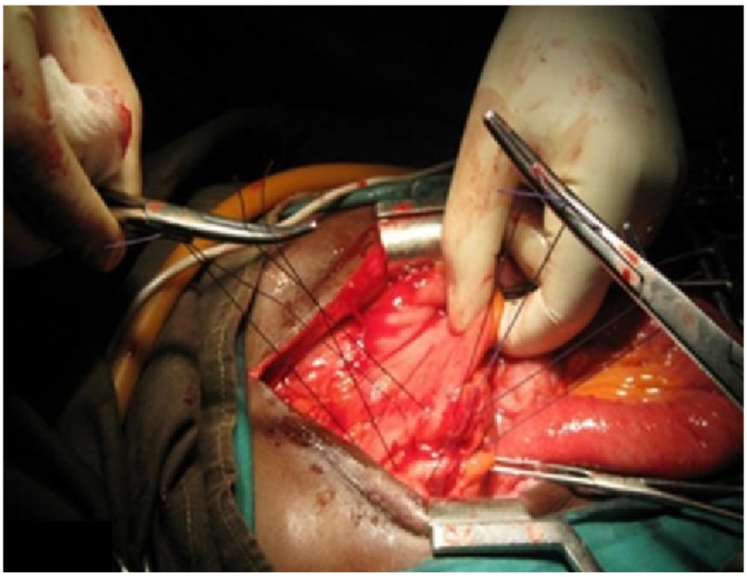


FIGURE 18

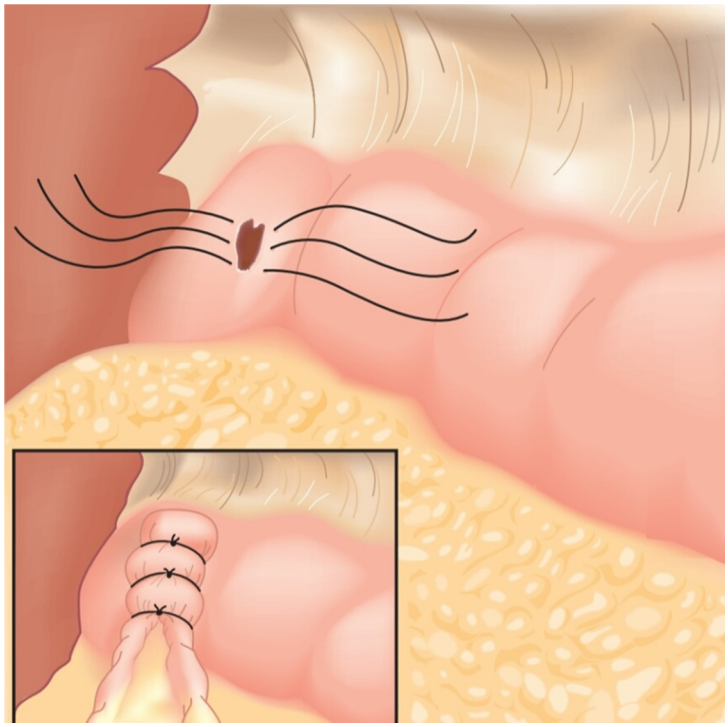


FIGURE 19

After combining with post-operative H. Pylori eradication the morbidity and mortality and ulcer recurrence rate after primary closure and omental onlay technique, have been shown acceptably low.

Since no significant alteration in gastrointestinal anatomy with ulcer closure -patient doesn't suffer from post Vagotomy or post gastrectomy side effects.

DEFINITIVE SURGERY:

Patients with peptic ulcer perforation usually presents with unstable Conditions. So we do damage control surgery. But in various conditions we recommend definitive surgery if patients presented with stable vitals and coexistence of bleeding or obstruction and with initial presentation of less than 8hrs duration

- In case of duodenal perforations , primary closure of perforation with omental patching done + definitive procedures (truncal vagotomy + pyloroplasty)
- In case of gastric perforation primary closure of perforation with omental patching done+ definitive procedures
    - Type 1 - distal gastrectomy with bilroth I or II reconstruction
    - Type 2 and 3 – truncal vagotomy plus antrectomy

TRUNCAL VAGOTOMY

It is performed by the division of right and left vagus nerve above hepatic and celiac branches just above gastroesophageal junction. It is usually done along with a drainage procedure like heineke-mikulicz pyloroplasty or finney pyloroplasty. It is usually associated with low reoccurrence rate and high incidence rate of post vagotomy syndromes.it is most commonly done for duodenal ulcers.

HIGHLY SELECTIVE VAGOTOMY

This highly selective vagotomy is also called parietal cell vagotomy or proximal gastric vagotomy.this procedure divides only the vagus nerves supplying the acid producing portion of the stomach within the corpus and fundus. This procedure also preserves the vagal innervations of gastric antrum so that there is no need for rout ne drainage procedures. This is usually associated with high recurrence rate and low incidence of post vagotomy complications.

TRUNCAL VAGOTOMY AND ANTRECTOMY:

The main goal of this procedure is removing vagal stimulation along with removal of gastrin driven secretion by performing an antrectomy.it is more effective in reducing acid secretion and reccurrence than truncal vagotomy in combination with a drainage procedure or a highly selective

vagotomy.the recurrence rate of ulceration after truncal vagotomy with antrectomy is low about 0-2%.however they are associated with high rates of post vagotomy and post gastrectomy syndromes of around 20%.

POST OPERATIVE COMPLICATIONS:

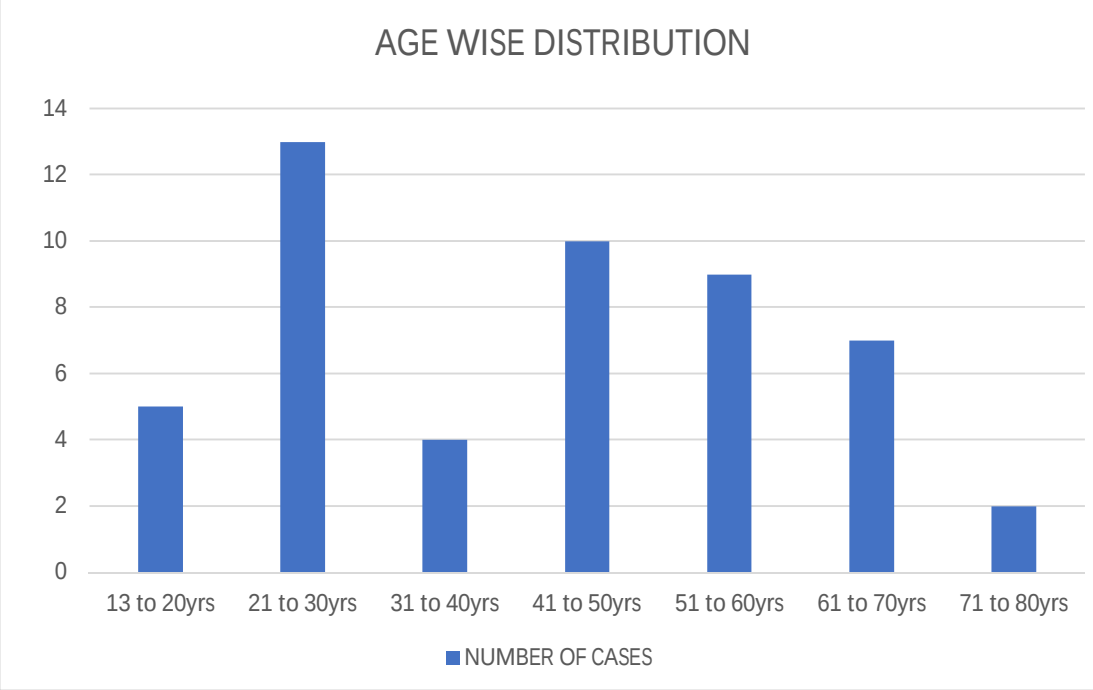
- Pulmonary complications like- atelectasis and pneumonia
- Peritonitis
- Residual abscess (pelvic and subphrenic abscess)
- Paralytic ileus
- DVT
- Early perforation and leak and fistula
- Renal failure
- Mediastinitis

OBSERVATION AND ANALYSIS

TABLE 2 : AGE WISE DISTRIBUTION OF CASES IN PEPTIC ULCER PERFORATION

AGE GROUP (AGE IN YEARS)	NUMBER OF CASES	PERCENTAGE
13 to 20yrs	5	**10.00%**
21 to 30yrs	13	**26.00%**
31 to 40yrs	4	**8.00%**
41 to 50yrs	10	**20.00%**
51 to 60yrs	9	**18.00%**
61 to 70yrs	7	**14.00%**
71 to 80yrs	2	**4.00%**
Total	**50**	100.00%
Mean: 43.16 / Median: 45.00 / S.D.: 17.117 / Min.: 15 / Max.: 77		

FIG 20 : BAR DIAGRAM SHOWING AGE WISE DISTRIBUTION OF CASES IN PEPTIC ULCER PERFORATION

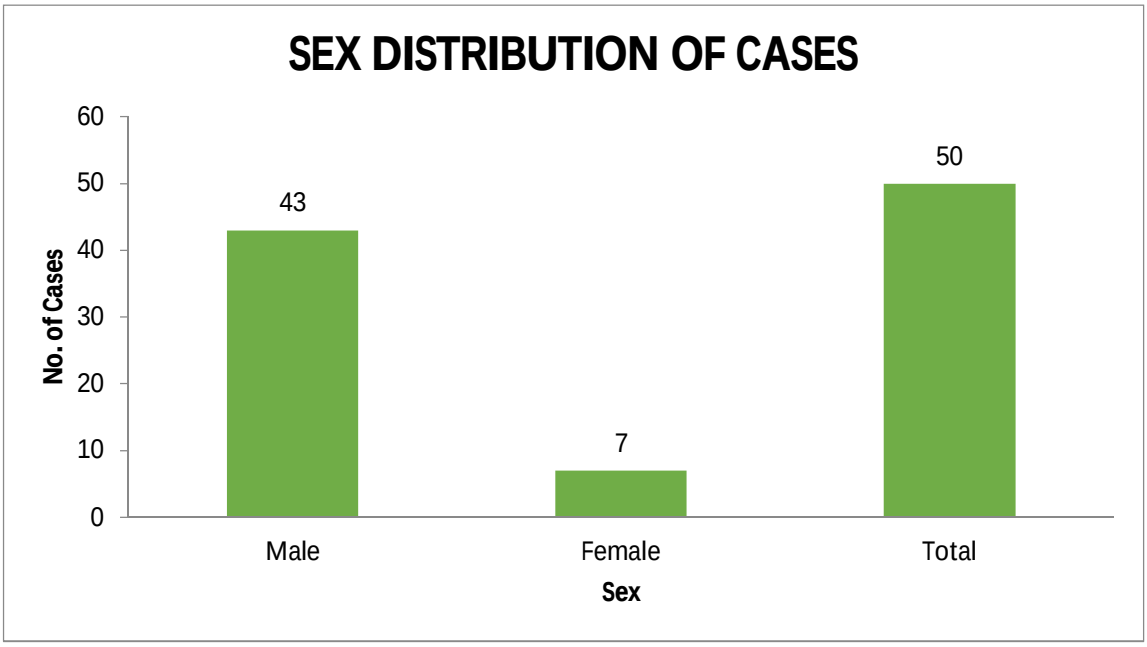


- Maximum(26%) number of patients belong to the age group of 21-30 years.
- Minimum(4%) number of patients belong to the age group of 71-80 years.

TABLE 3 - SEX WISE DISTRIBUTION OF CASES IN PEPTIC ULCER PERFORATION

SEX	NUMBER OF CASES	PERCENTAGE
Male	43	**86.00%**
Female	7	**14.00%**
Total	50	100.00%

FIG 21 ; BAR DIAGRAM SHOWING SEX WISE DISTRIBUTION OF CASES IN PEPTIC ULCER PERFORATION

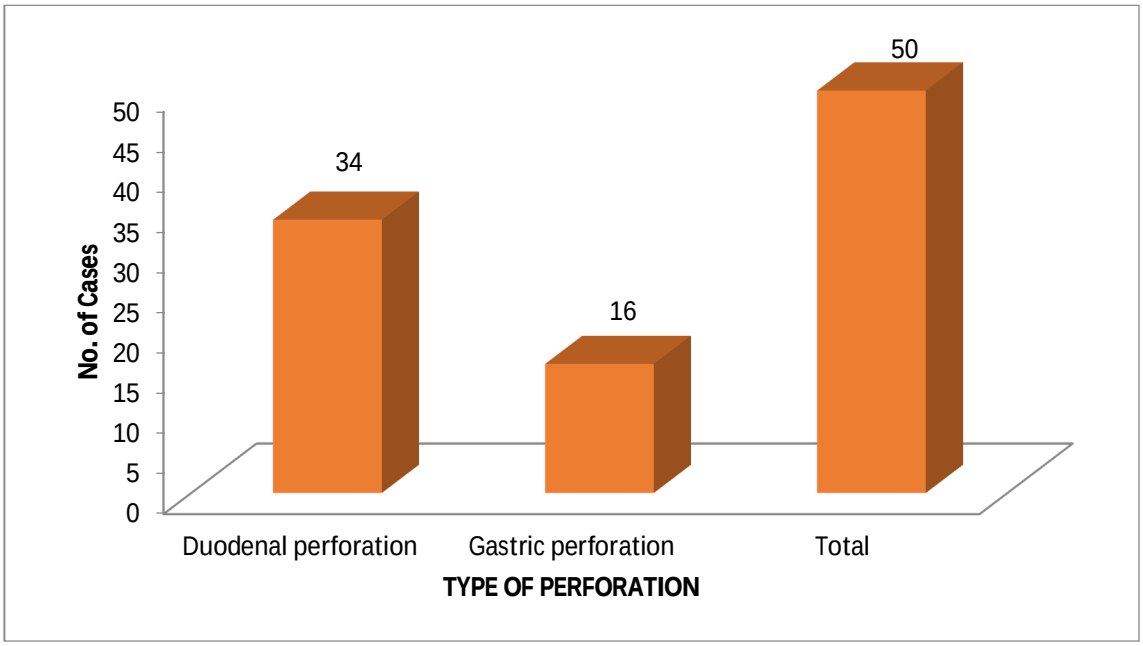


In our study 86% of patients were found be MALE and 14% patients were found to be FEMALE .

TABLE 4 - TYPE OF PERFORATION IN TOTAL NUMBER OF CASES

TYPE OF PEFORATION	NUMBER OF CASES	PERCENTAGE
Duodenal perforation	34	**68.00%**
Gastric perforation	16	**32.00%**
Total	50	100.00%

FIG 22: BAR DIAGRAM SHOWING TYPE OF PERFORATION IN ALL CASES OF PEPTIC ULCER PERFORATION

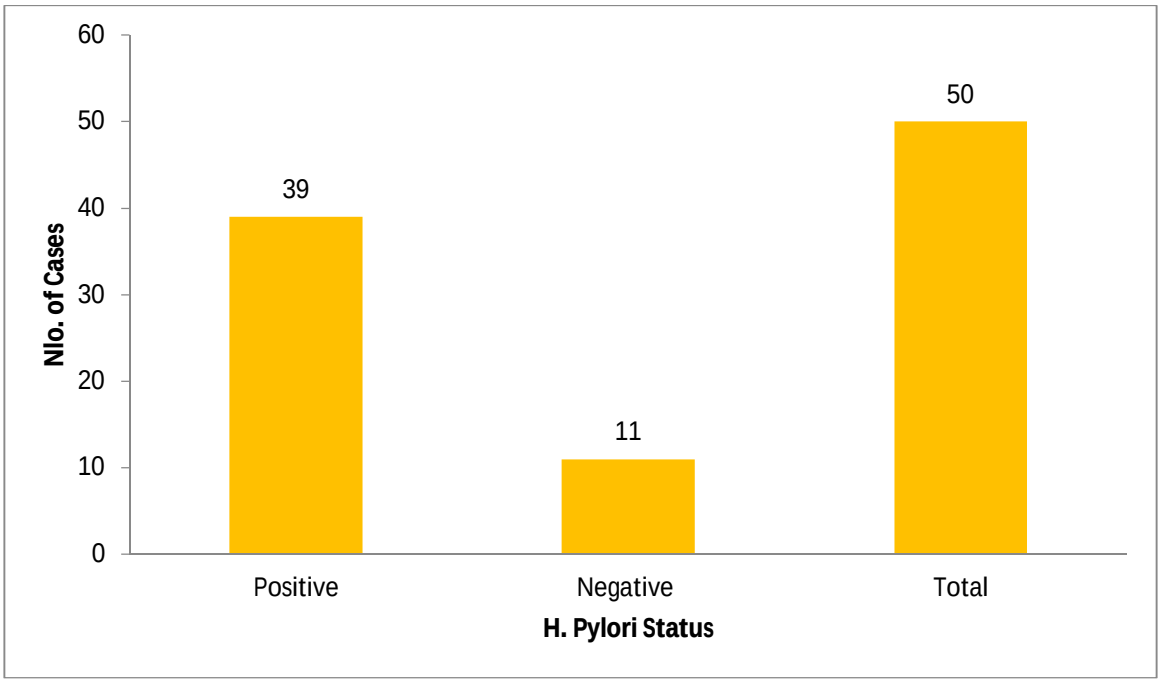


In our study duodenal perforation were found to be 68% and gastric perforation were found to be 32%

TABLE 5 : H.PYLORI POSITIVE CASES IN PEPTIC ULCER PERFORATION

H.PYLORI STATUS	NO OF CASES	PERCENTAGE
Positive	39	**78.0**
Negative	11	**22.0**
Total	50	100.0

FIG 23 : BAR DIAGRAM SHOWING H.PYLORI POSITIVE CASES IN PEPTIC ULCER PERFORATION

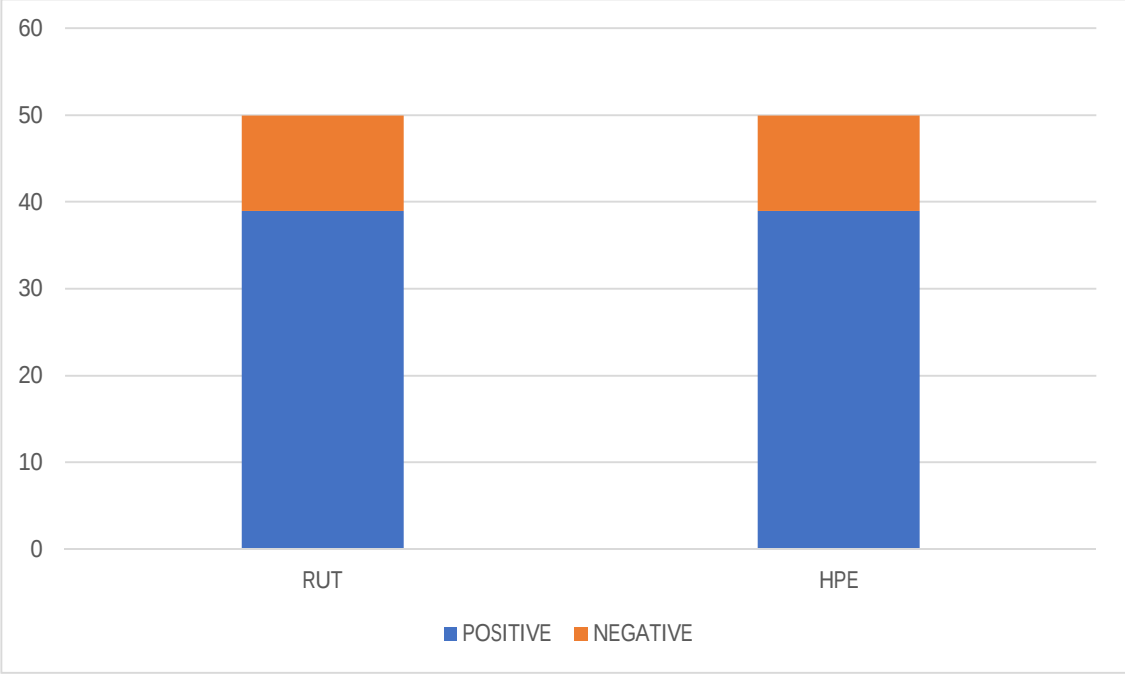


In Our study showed that 78 % of patients were found to be Positive for H.PYLORI and 22% were found to be Negative

TABLE 6 : COMPARISON OF RAPID UREASE TEST AND HPE FOR DETECTION OF H.PYlORI

H.PYLORI	POSITIVE		NEGATIVE	
	N	**%**	**n**	%
RUT	39	78 %	11	**22 %**
HPE	**39**	**78 %**	**11**	**22 %**

FIG 24 : BAR DIAGRAM SHOWING COMPARISON OF RUT AND HPE IN DETECTION OF H.PYLORI

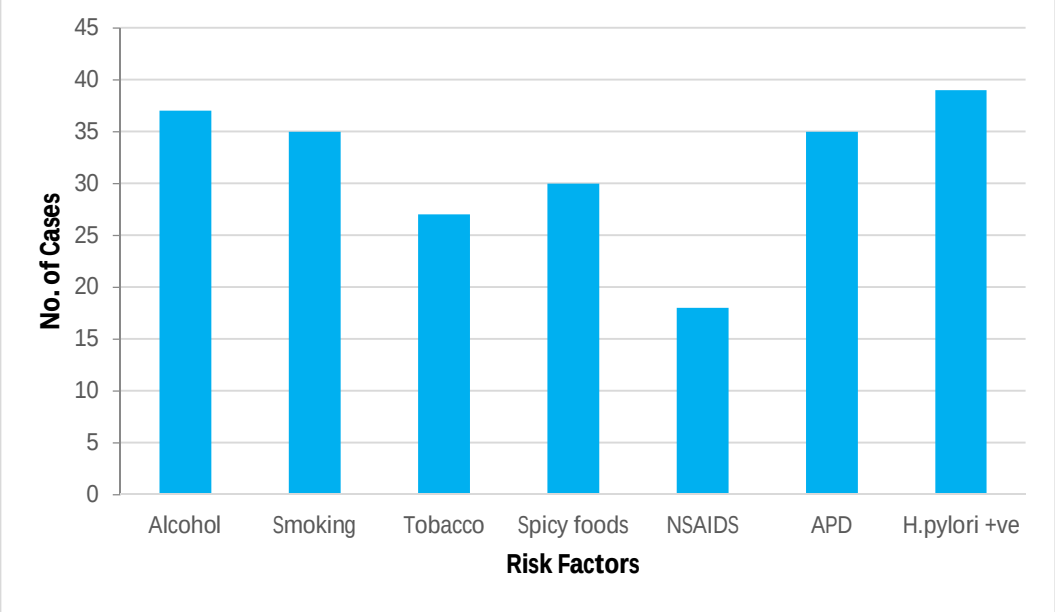


In Our study showed that both RUT and HPE has same sensitivity in detection of H.pylori

TABLE 7 : RELATION OF RISK FACTOS WITH PERPTIC ULCER PERFORATION:

RISK FACTORS	NO OF CASES	PERCENTAGE
Alcohol	37	**74%**
Smoking	35	**70%**
Tobacco	27	**54%**
Spicy foods	30	**60%**
NSAIDS	18	**36%**
APD	35	**70%**
H.pylori +ve	**39**	**78%**

FIG 25 : BAR DIAGRAM SHOWING RELATION OF RISK FACTORS WITH PEPTIC ULCER PERFORATION

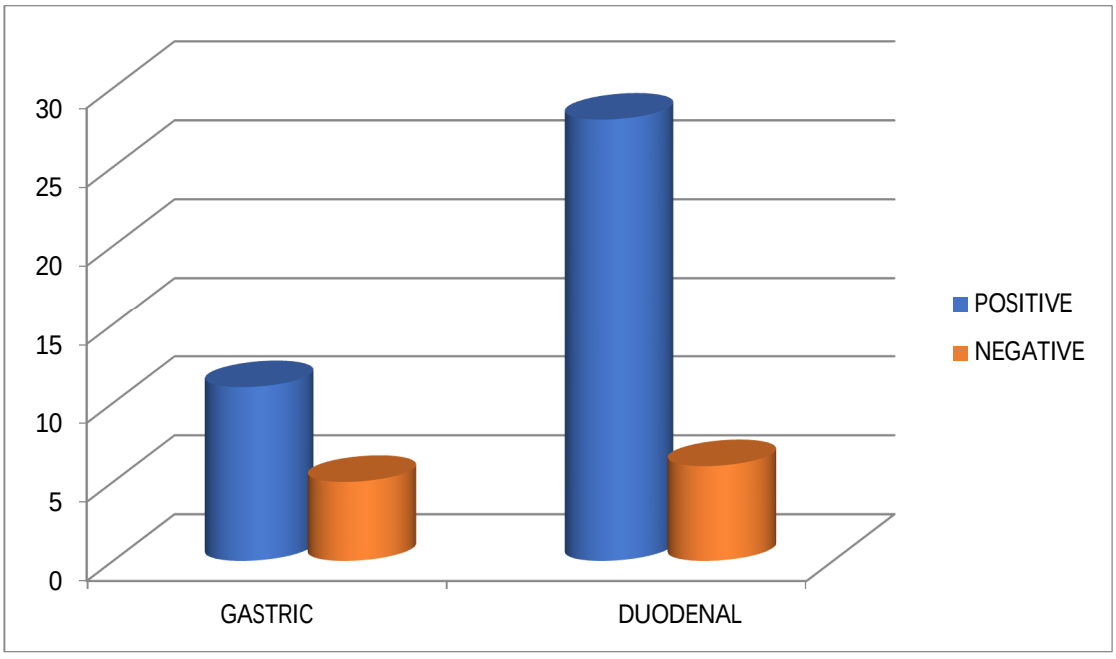


In our study we found that maximum number of the patients (39) i.e. 78% shows H.pylori as a common risk factor & Alcohol (74%) is the 2nd most common risk factor ,followed by smoking and Dyspepsia (70%) contributes for peptic ulcer perforation.

TABLE 8 : RELATIONSHIP BETWEEN THE TYPE OF PERFORATION AND H.PYLORI STATUS:

TYPE OF PERFORATION	H.PYLORI					
	POSITIVE		**NEGATIVE**		TOTAL	
	N	***%***	***N***	***%***	***n***	*%*
GASTRIC	11	28.20%	5	45.45%	16	**36.0%**
DUODENAL	28	71.80%	6	54.54%	34	**64.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 26 : BAR DIAGRAM SHOWING RELATION BETWEEN TYPE OF PERFORATION AND H.PYLORI STATUS IN PEPTIC ULCER PERFORATION

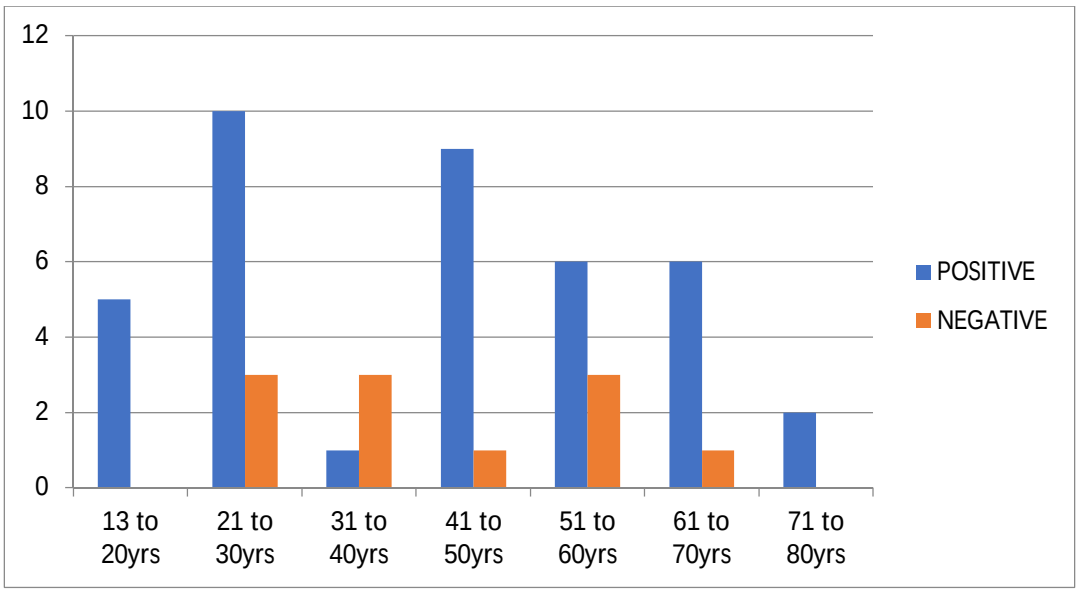


In our study duodenal perforation(28 patients (71.80%))Were found to be positive for H.pylori Gastric perforation only 11 patients were H.pylori positive.

TABLE 9 : AGE WISE DISTRIBUTION OF H.PYLORI POSITIVE CASES IN PERFORATED PEPTIC ULCER

AGE	H.PYLORI STATUS					
	POSITIVE		**NEGATIVE**		Total	
	n	***%***	***n***	***%***	***n***	*%*
13 to 20yrs	5	12.8%	0	.0%	5	**10.0%**
21 to 30yrs	10	25.6%	3	27.3%	13	**26.0%**
31 to 40yrs	1	2.6%	3	27.3%	4	**8.0%**
41 to 50yrs	9	23.1%	1	9.1%	10	**20.0%**
51 to 60yrs	6	15.4%	3	27.3%	9	**18.0%**
61 to 70yrs	6	15.4%	1	9.1%	7	**14.0%**
71 to 80yrs	2	5.1%	0	.0%	2	**4.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 27 : BAR DIAGRAM SHOWING AGE WISE DISTRIBUTION OF H.PYLORI POSITIVE CASES IN PEPTIC ULCER PERFORATION



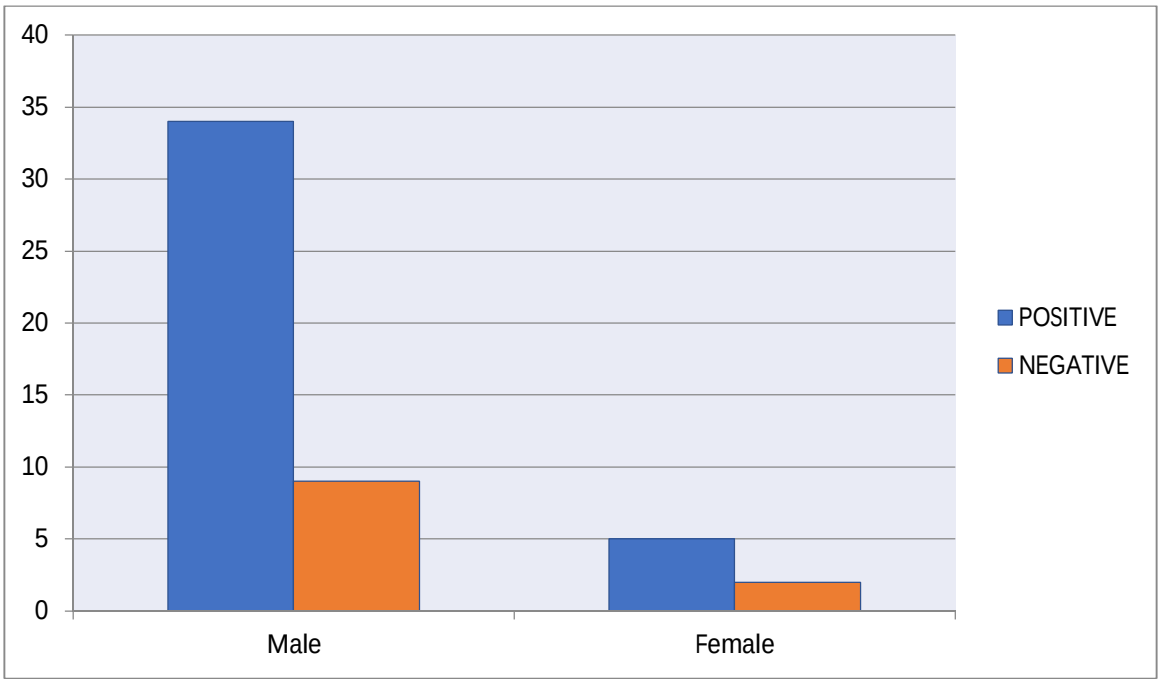
Chi square – 0.113	**df – 6**	**P > 0.05**

Maximum number of H.PYLORI positive cases(25.6%) belongs to the age group of 21-30 years and maximum number of H.PLYORI negative cases belongs to the age group of 21-40 years (27.3%)

TABLE 10 : SEX WISE DISTRIBUTION OF H.PYLORI POSITIVE CASES IN PERPTIC ULCER PRFORATION

SEX	**H.PYLORI**					
	POSITIVE		**NEGATIVE**		**Total**	
	n	***%***	***n***	***%***	***n***	***%***
Male	34	87.2%	9	81.8%	43	86.0%
Female	5	12.8%	2	18.2%	7	14.0%
Total	39	100.0%	11	100.0%	50	100.0%

FIG 28 : BAR DIAGRAM SHOWING SEX WISE DISTRIBUTION OF H.PYLORI POSITIVE CASES IN PEPTIC ULCER PERFORATION



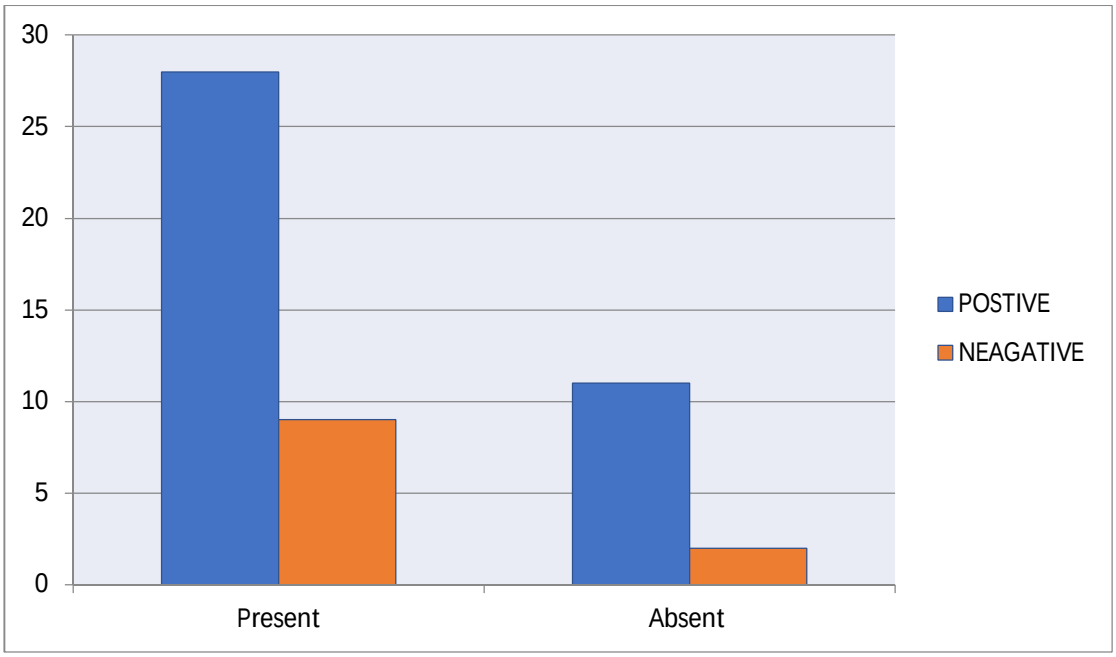
Chi square – 0.651	**df – 1**	**P > 0.05**

In our study incidence of HYPLORI in males were found to be 87.2% and in females were found to be 12.8%

TABLE – 11 : COMPARISON OF ALCOHOLISM AND H.PYLORI STATUS

ALCOHOL	H.PYLORI					
	POSTIVE		**NEAGATIVE**		TOTAL	
	n	***%***	***n***	***%***	***N***	*%*
Present	28	71.8%	9	81.8%	37	**74.0%**
Absent	11	28.2%	2	18.2%	13	**26.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 29 : BAR DIAGRAM SHOWING COMPARISON OF ALCOHOLISM AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



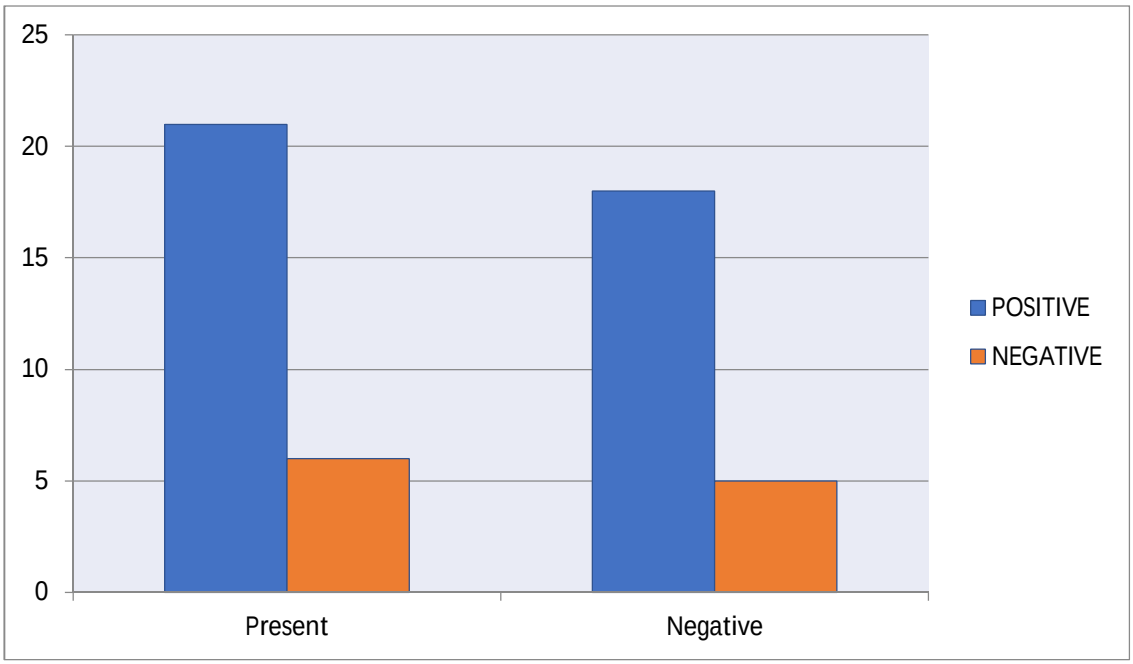
Chi square – 0.503	**df – 1**	**P > 0.05**

Interpretation: Observed difference in alcohol and the result of HPYLORI status is not significant (P>0.05)

TABLE 12 : COMPARISON OF TOBACCO AND H.PYLORI STATUS

TOBACCO	H.PYLORI					
	POSTIVE		**NEAGATIVE**		TOTAL	
	n	***%***	***n***	***%***	***N***	*%*
Present	21	53.8%	6	54.5%	27	**54.0%**
Negative	18	46.2%	5	45.5%	23	**46.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 30 : BAR DIAGRAM SHOWING COMPARISON OF TOBACCO AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



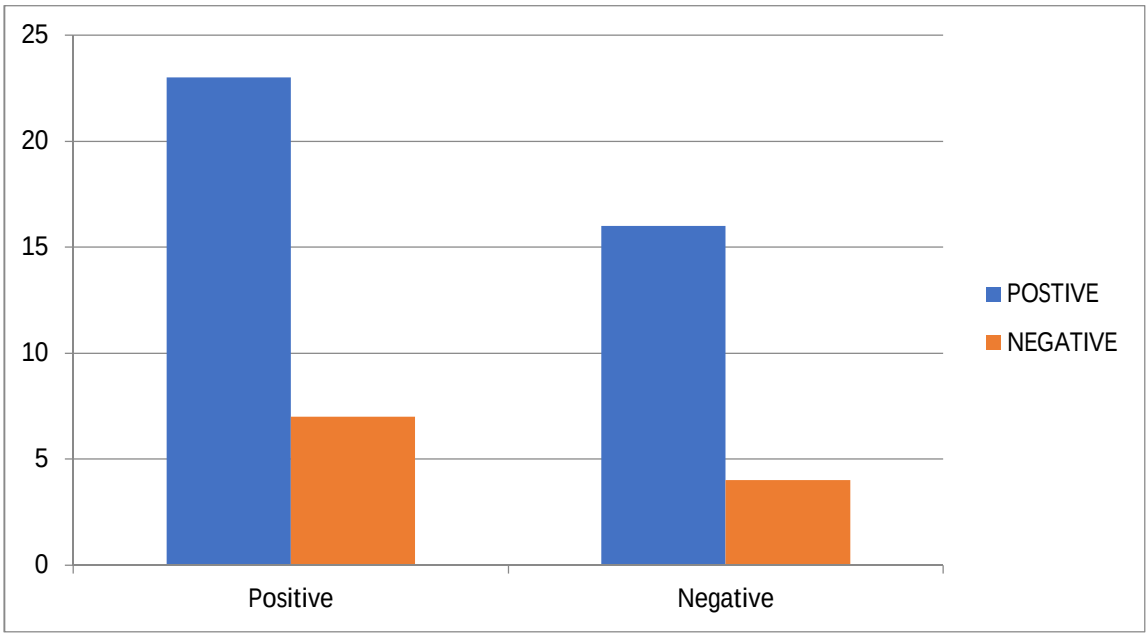
Chi square – 0.967	**df – 1**	**P > 0.05**

Interpretation: Observed difference in tobacco and the result of HPYLORI status is not significant (P>0.05)

TABLE 13: COMPARISON OF SPICY FOODS AND H.PYLORI STATUS

SPICY FOODS	H.PYLORI					
	POSTIVE		**NEGATIVE**		**TOTAL**	
	n	***%***	***n***	***%***	***n***	***%***
Positive	23	59.0%	7	63.6%	30	**60.0%**
Negative	16	41.0%	4	36.4%	20	**40.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 31: BAR DIAGRAM SHOWING COMPARISON OF SPICY FOODS AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



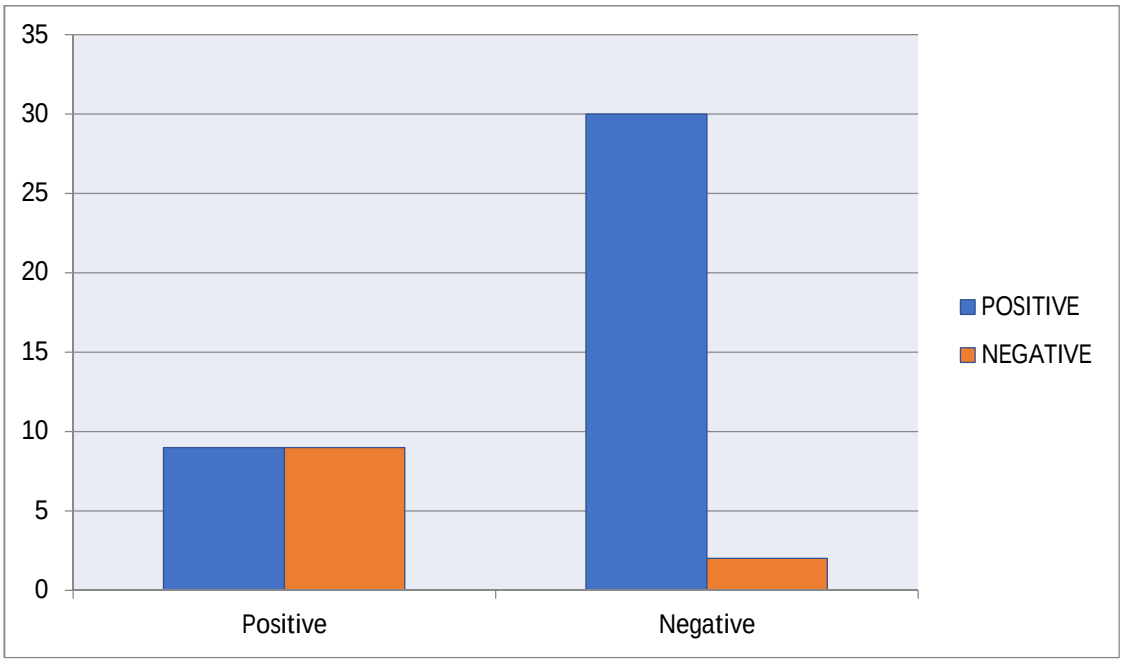
Chi square – 0.780	**df – 1**	**P > 0.05**

Interpretation: Observed difference in spicy foods and the result of HPYLORI status is not significant (P>0.05)

TABLE 14: COMPARISON OF NSAIDS AND H.PYLORI STATUS

NSAIDS	H.PYLORI					
	POSITIVE		**NEGATIVE**		TOTAL	
	n	***%***	***n***	***%***	***n***	*%*
Positive	9	23.1%	9	81.8%	18	**36.0%**
Negative	30	76.9%	2	18.2%	32	**64.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 32: BAR DIAGRAM SHOWING COMPARISON OF NSAIDS AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



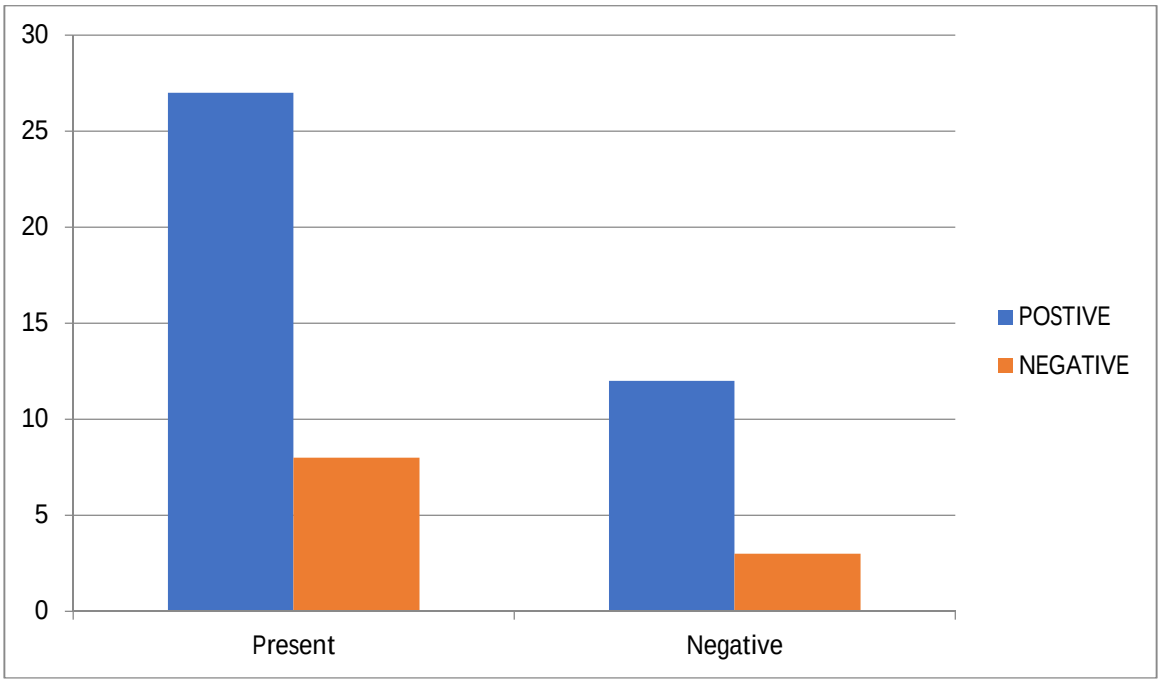
Chi square – 0.000	**df – 1**	**P < 0.05**

Interpretation: Observed difference in NSAIDS and the result of H. PYLORI status is SIGNIFICANT (P<0.05)

TABLE 15: COMPARISON OF SMOKING AND H.PYLORI STATUS

SMOKING	H.PYLORI					
	POSTIVE		**NEGATIVE**		TOTAL	
	n	***%***	***n***	***%***	***n***	*%*
Present	27	69.2%	8	72.7%	35	**70.0%**
Negative	12	30.8%	3	27.3%	15	**30.0%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 33: BAR DIAGRAM SHOWING COMPARISON OF SMOKING AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



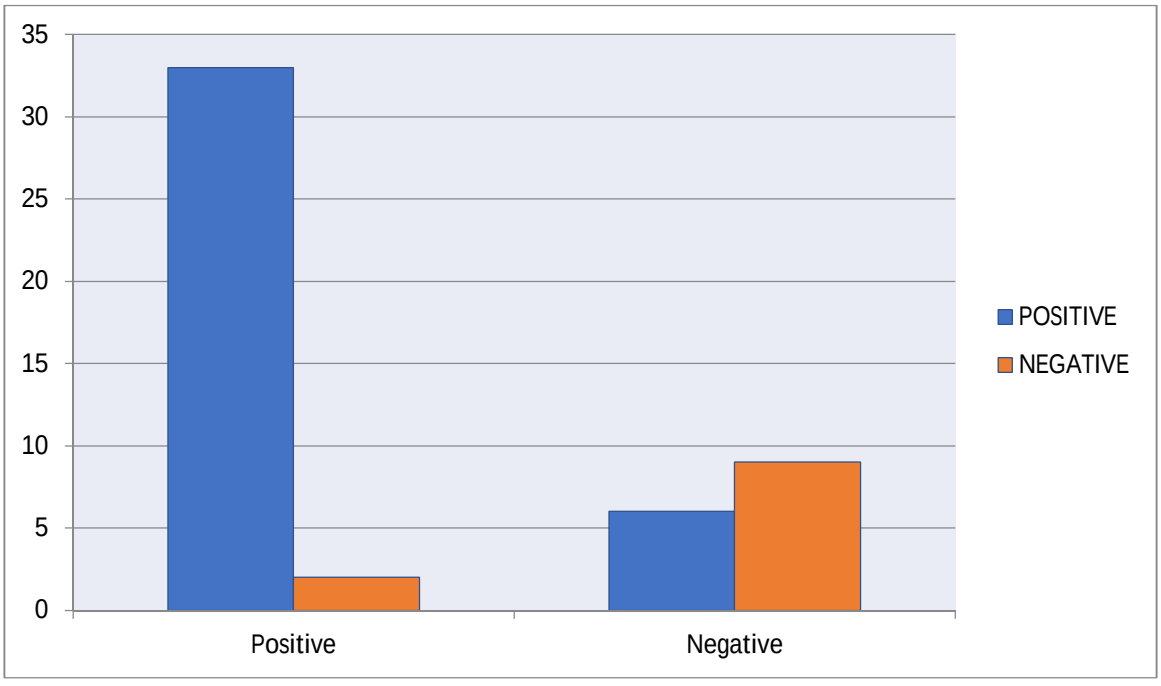
Chi square – 0.823	**df – 1**	**P > 0.05**

Interpretation: Observed difference in smoking and the result of HPYLORI status is not significant (P>0.05)

TABLE 16: COMPARISON OF APD AND H.PYLORI STATUS

APD	H.PYLORI					
	POSITIVE		**NEGATIVE**		TOTAL	
	n	***%***	***n***	***%***	***n***	*%*
Positive	33	84.6	2	18.2	35	**70.00%**
Negative	6	15.4	9	81.8	15	**30.00%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 34: BAR DIAGRAM SHOWING COMPARISON OF APD AND H. PYLORI STATUS IN PEPTIC ULCER PERFORATION



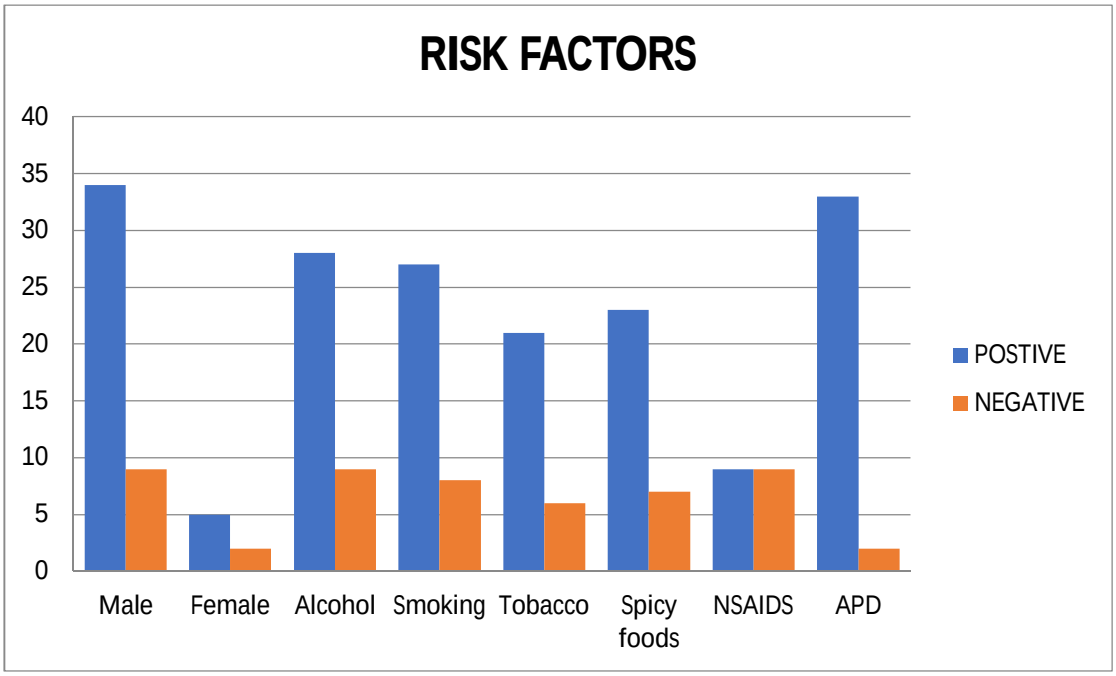
Chi square – 0.000	**df – 1**	**P < 0.05**

Interpretation: Observed difference in APD and the result of HPYLORI status is SIGNIFICANT (P<0.05)

TABLE 17: RELATIONSHIP BETWEEN RISK FACTORS OF PEPTIC ULCER PERFORATION AND H.PYLORI STATUS

RISK FACORS	H.PYLORI					
	POSTIVE		**NEGATIVE**		TOTAL	
	n	***%***	***N***	***%***	***n***	*%*
Male	34	87.2%	9	81.8%	43	**86.0%**
Female	5	12.8%	2	18.2%	7	**14%**
Alcohol	28	71.8%	9	81.8%	37	**74%**
Smoking	27	69.2%	8	72.7%	35	**70%**
Tobacco	21	53.8%	6	54.5%	27	**54.0%**
Spicy foods	23	59%	7	63.6%	30	**60%**
NSAIDS	9	23.1%	9	81.8%	18	**36.0%**
APD	**33**	**84.6%**	**2**	**18.2%**	**35**	**70%**

FIG 35: BAR DIAGRAM SHOWING RELATION BETWEEN RISK FACTORS OF PEPTIC ULCER PERFORATION AND H.PYLORI STATUS



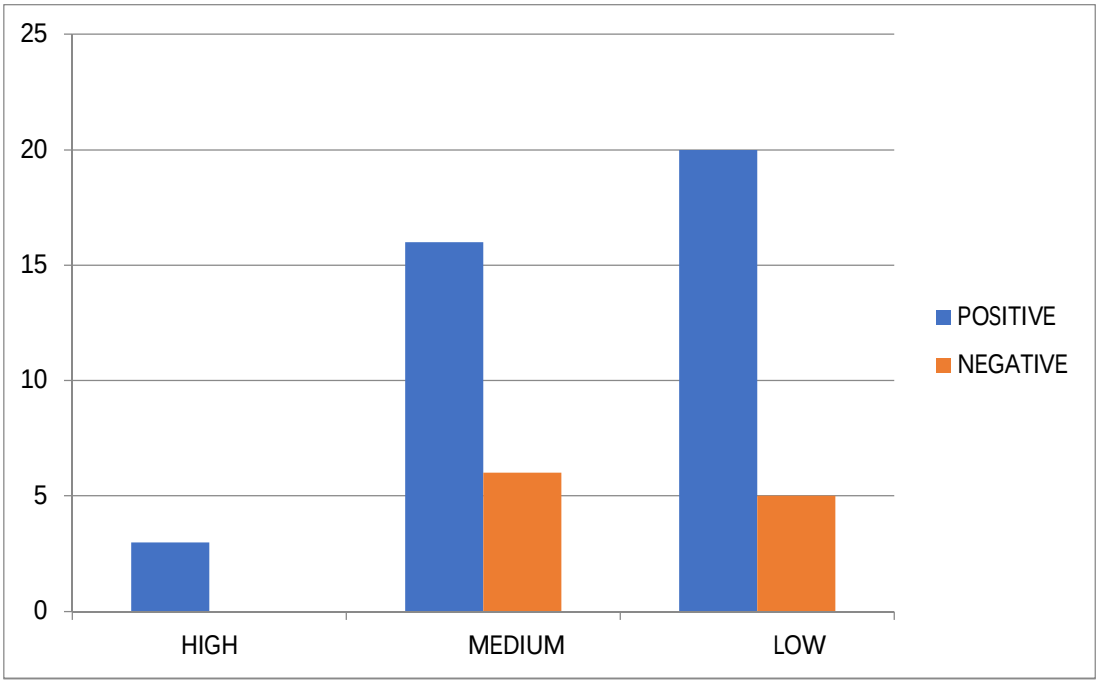
In our study we found that APD is most commonly associated with H.pylori positive patients (33) i.e. 84.6% and followed by alcohol (71.8%) and smoking (69.2%).

Patient with NSAIDS intake had low prevalence of H.pylori positivity i.e. (23.1%).

TABLE 18 - RELATIONSHIP BETWEEN H.PYLORI AND SOCIOECONOMIC STATUS

SOCIOECONOMIC STATUS	H.PYLORI					
	POSITIVE		NEGATIVE		TOTAL	
	N	*%*	*N*	*%*	*n*	*%*
HIGH	3	7.69%	0	0.00%	3	**6%**
MEDIUM	16	41.02%	6	54.54%	22	**44%**
LOW	20	51.28%	5	45.45%	25	**50%%**
Total	39	100.0%	11	100.0%	50	100.0%

FIG 36: BAR DIAGRAM SHOWING RELATION BETWEEN SOCIOECONOMIC STATUS AND H.PYLORI STATUS



In our study we found that H.pylori positive cases were more in LOW SOCIOECONOMIC STATUS patients about 51.28% and followed by MEDIUM SOCIOECONOMIC STATUS patients i.e. 41.02%

DISCUSSION

In this study, most of the patients were middle aged (21 – 30 years) out of 50 patients, 43 were males and 7 were females in the ratio of 4: 1

In our study 74% of patients had history of alcohol, 70% of patients had history of smoking and acid peptic disease, only 36% of patients had history of NSAID's use.

Out of 50 cases studied, 34 patients (68.00%) were found to have duodenal perforation and 16 patients (32.00%) were found to have gastric perforation

In our study, prevalence of H.pylori infection in peptic ulcer perforation was 78% (39 cases out of 50). This result was in accordance with study by **Sebastian et al.** In their study, they reported infection rate of 83 %.

But some studies reported the prevalence of H.pylori infection was in the range of 50 – 90 %. This wide variation could be due to different population group, routine use of proton pump inhibitors for treatment of upper gastrointestinal symptoms. According to study by **Connor et al** using PPI in peptic ulcer disease shows false negative results with rapid urease test. In our study 39 (78%) patients were positive for H.pylori out of which 28 patients were of duodenal perforation and only 11 patients were having gastric perforation.

In our study, the association of H.pylori infection with duodenal perforation was more. i.e., 28 out of 34 duodenal ulcer perforation cases found to H.pylori positive (82%). This was corresponding to **Ng et al.** in their study; they found 70 % prevalence of H.pylori infection with duodenal perforation.

In this study, rapid urease test and Giemsa stain had same validity while detection of H.pylori. But in **Kumar et al study**, found H.pylori infection state of 57% by means of rapid urease test, histopathological examination, and culture method and reported H.pylori infection rate of 70% by rapid urease test method.

In the study, 50 cases of peptic ulcer perforation, H.pylori status positive for most of the middle aged persons (21-30 years) – 25.6 %.

Out of 39 cases of H.pylori positive patients, 34 were males and 5 were females in ratio of 7 : 1. But in **A Shin et al and Maria Rodrigues** found that there was no difference in prevalence of H.pylori infection in males and females.

In our study, we compared various risk factors with H.pylori infection. Among them, patient with history of acid peptic disease had high incidence of H.pylori infection. i.e., 84.6% which was significant in our study, followed by alcohol (71.8%) and smoking which were not significant in our study. But patients with NSAID’s had low H.pylori infection rate 23.1%. This was similar

to the study of **Asud ullah et al**. In their study history of dyspepsia had 87% of H.pylori infection and NSAID's has 9% of H.pylori infection.

In our study, out of 50 patients 39 (93.0%) were positive for H.pylori. They were treated with anti H.pylori regimen by administration of standard triple therapy for 14 days. The remaining 11 patients were given only Cap.omeprazole 20mg bd for 14 days.

In our study most of the patients had no dyspepsia except only one patient who had dyspepsia inspite of anti H.pylori treatment. We gave a repeated course of same drug and had no dyspepsia.

CONCLUSION

In our study we found that H.pylori infection was the most common risk factor for peptic ulcer perforation and the patients of peptic ulcer perforation with dyspepsia had a high prevalence rate of H.pylori infection. Therefore if we treat these patients with anti H.pylori triple therapy postoperatively we can reduce the recurrence of ulcer and reperforation . Early diagnosis and controlling of the H.pylori infection in dyspepsia patients may reduce the incidence of peptic ulcer perforation and can prevent the morbidity and mortality of the patient.

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PROFORMA

Name: IP No:

Age: SL No:

Sex: Date of admission:

Occupation: Date of surgery:

Religion: Date of discharge:

PRESENTING COMPLAINTS:

Pain Abdomen:

Fever:

Vomiting:

Distension of Abdomen:

Constipation:

PAST HISTORY:

Surgeries:

Medical conditions: Diabetes / Hypertension / Tuberculosis / Asthma / Epilepsy

FAMILY HISTORY:

PERSONAL HISTORY:

Diet: Sleep:

Bowel / Bladder: Smoker / Alcoholic:

EXAMINATION GPE:

Pallor: Icterus:

Cyanosis: Clubbing:

Lymphadenopathy:

Vitals: Pulse rate: Blood Pressure: RR:

SYSTEMIC EXAMINATION:

Per Abdomen:

Cardiovascular System:

Respiratory System:

Central Nervous System:

DIAGNOSIS:

INVESTIGATIONS:

Hb %: TC: DC: ESR:

RBS:

Blood urea: BT: CT:

Serum creatinine:

X – ray erect abdomen: ECG:

USG abdomen:

HIV: HBsAg:

PREOPERATIVE PREPARATION:

NPO, RTA, Injection TT, Injection Xylo test dose, IV Antibiotics, Preparing of relevant parts; Informed High risk consent

PROCEDURE:

Anaesthesia: Position:

Exploratory laparotomy + perforation closure + grahams patch:

Postop: Antibiotic: Analgesic:

COMPLICATIONS:

FOLLOWUP:

CERTIFICATE OF CONSENT

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research and understand that I have the right to withdraw from the research at any time without in any way affecting my medical care.

Name of participant _____

Signature of Participant _____

Date _____

If illiterate

A illiterate witness must sign (if possible, this person should be selected by the participant and should have no connection to the research team).

I have witnessed the accurate reading of the consent from to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of participant _____

Signature of Participant _____

Date _____

I have accurately read or witnessed the accurate reading of the consent from to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of participant _____

Signature of Participant _____

Date _____

Thumb print of participant

A copy of this Informed Consent From has been provided to participant _____ (initialled by the researcher / assistant).

பங்கேற்பவர்களுக்கு ஆய்வின் விபரம்
செய்முறை விளக்கம்

இந்த ஆய்வில் பங்கேற்பவர்களுக்கு அறுவை சிகிச்சை செய்து கொண்ட பின்பு, அந்த தசையை மருத்துவ பரிசோதனை செய்து கொண்டு அதன் விபரம் தெரிந்து கொள்ள முழு மனதுடன் சம்மதிக்கிறேன்

ஆராய்ச்சி நிலையம்
பொது அறுவை சிகிச்சை துறை
அரசு மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை
கோயம்புத்தூர்

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன் . எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன்

பங்கேற்பவரின் கையொப்பம் .....

இடம் .....
நாள் .....

கட்டை விரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்
.....
.....

ஆய்வாளரின் கையொப்பம் .....
இடம் : .....
நாள் : .....
ஆய்வாளரின் பெயர் : .....

KEY TO MASTER CHART

S.food	–	Spicy Food
NSAID	–	Non-Steroidal Ani Inflammatory Drugs
APD	-	Acid Peptic Disease
RUT	–	Rapid Urease Test
HPE	–	Histopathological Examination
HP kit	–	Anti H.pylori kit
OT kit	–	Omeprazole Therapy
1	–	positive
2	–	negative
AUD	–	Air Under Diaphragm
H	–	anti H.pylori therapy
O	–	Omeprazole therapy
ND	–	No dyspepsia
D	–	Dyspepsia

MASTER CHART

S.NO	NAME	AGE	SEX	IPNO	SES	ALCOHOL	SOMKING	TOBACCO	S.FOOD	NSAIDS	APD	PAIN	VOMITING	FEVER	HYPOTENSION	TACHYCARDIA	XRAY	RUT	HPE	DIAGNOSIS	HPKIT/OT	FOLLOW UP
1	BHARATHAN	15	M	108946	M	2	2	2	1	2	1	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
2	KUMAR	30	M	1511	M	1	1	2	1	2	1	1	2	1	2	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
3	BIRENDRA	19	M	3203	M	2	2	2	1	2	1	1	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
4	SATISH	30	M	12606	M	1	1	1	1	2	1	1	2	1	2	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
5	PAPPU	28	M	178852	L	1	1	1	1	2	2	1	1	1	2	1	AUD+	2	2	DUODENAL PERFORATION	O	ND
6	KANNISAMY	47	M	197687	H	1	1	1	1	2	1	1	1	1	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
7	MUTHUKRISHNA	65	M	192974	L	1	1	1	2	2	1	1	2	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
8	MANI	65	M	192991	M	1	1	2	1	2	1	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
9	BALASUBRAMANI	39	M	129702	M	1	1	2	2	1	2	1	1	1	2	1	AUD+	2	2	GASTRIC PERFORATION	O	ND
10	BATHIRAPAN	61	M	841	L	1	1	1	2	1	1	1	1	1	2	2	AUD+	1	1	DUODENAL PERFORATION	H	ND

S.NO	NAME	AGE	SEX	IPNO	SES	ALCOHOL	SOMKING	TOBACCO	S.FOOD	NSAIDS	APD	PAIN	VOMITING	FEVER	HYPOTENSION	TACHYCARDIA	XRAY	RUT	HPE	DIAGNOSIS	HPKIT/OT	FOLLOW UP
11	RAMALINGAM	60	M	925	L	1	1	2	2	1	2	1	1	1	1	1	AUD+	2	2	GASTRIC PERFORATION	O	ND
12	JAYAMMA	65	F	10864	L	2	2	1	2	1	1	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
13	BANNARI	40	M	190665	L	1	1	1	2	2	2	1	1	1	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
14	GANESH	29	M	38818	M	1	1	1	1	1	2	1	1	1	2	1	AUD+	2	2	DUODENAL PERFORATION	O	D
15	BAGADUR	58	M	46113	M	1	1	2	1	1	2	1	1	1	1	1	AUD+	2	2	DUODENAL PERFORATION	O	ND
16	UDAIDULA	46	M	48494	M	1	1	2	1	2	2	1	1	1	1	2	AUD+	1	1	GASTRIC PERFORATION	H	ND
17	RAVIKUMAR	45	M	97298	M	1	1	1	2	2	1	2	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
18	NAGARAJ	17	M	101780	L	2	2	2	1	2	1	1	2	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	D
19	RAJASEKAR	30	M	106307	M	1	1	1	2	2	1	2	1	1	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
20	MALLIAG ARJUNAN	47	M	110833	L	1	2	1	1	2	1	1	1	1	1	2	AUD+	1	1	GASTRIC PERFORATION	H	ND
21	RAJESH	24	M	130547	L	1	1	1	1	2	1	1	1	1	2	1	AUD+	1	1	DUODENAL PERFORATION	H	ND

S.NO	NAME	AGE	SEX	IPNO	SES	ALCOHOL	SOMKING	TOBACCO	S.FOOD	NSAIDS	APD	PAIN	VOMITING	FEVER	HYPOTENSION	TACHYCARDIA	XRAY	RUT	HPE	DIAGNOSIS	HPKIT/OT	FOLLOW UP
22	VIJAYAKUMAR	38	M	176476	L	1	2	1	2	1	2	2	1	1	1	2	AUD+	2	2	DUODENAL PERFORATION	O	ND
23	ESWARI	30	F	52743	L	2	2	2	1	1	2	1	1	1	2	1	AUD+	2	2	DUODENAL PERFORATION	O	ND
24	RENUGA	19	F	130960	L	2	2	2	1	1	1	2	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
25	ARIYAMMAL	49	F	62321	M	2	2	1	2	1	1	2	1	1	2	1	AUD+	2	2	GASTRIC PERFORATION	O	ND
26	SIVAGAMI	48	F	38323	M	2	2	1	2	1	2	2	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
27	MARIMUTHU	65	M	83764	L	1	1	1	2	1	1	2	1	2	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
28	PERIYASAMY	57	M	9057	L	1	1	2	1	2	2	1	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
29	PANDIYAN	47	M	9057	L	1	1	2	1	2	2	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
30	NANJAN	60	M	23369	M	1	1	1	2	1	1	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
31	MYILSAMY	65	M	27570	M	1	1	2	1	1	2	1	1	1	1	1	AUD+	2	2	DUODENAL PERFORATION	O	ND
32	BHARATHAN	52	M	36282	M	1	1	2	1	2	1	2	1	1	2	1	AUD+	1	1	GASTRIC PERFORATION	H	ND

S.NO	NAME	AGE	SEX	IPNO	SES	ALCOHOL	SOMKING	TOBACCO	S.FOOD	NSAIDS	APD	PAIN	VOMITING	FEVER	HYPOTENSION	TACHYCARDIA	XRAY	RUT	HPE	DIAGNOSIS	HPKIT/OT	FOLLOW UP
33	SARASWATHY	75	F	23698	L	2	2	1	2	1	1	1	1	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
34	RAJENDRAN	56	M	31045	L	1	1	2	1	2	1	2	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
35	ANU	15	F	31252	H	2	2	2	1	1	1	1	1	1	2	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
36	SURY	25	M	82082	L	1	1	2	2	2	1	1	2	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
37	PONNUSAMY	55	M	83896	L	2	1	1	1	2	1	1	2	1	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND
38	PALRAJ	45	M	87506	M	1	1	1	2	2	1	2	1	1	2	2	AUD+	1	1	DUODENAL PERFORATION	H	ND
39	SALEEM	25	M	4889	M	1	1	2	1	2	1	1	2	1	1	2	AUD+	1	1	DUODENAL PERFORATION	H	ND
40	RANGAN	55	M	16627	L	1	1	1	1	2	1	1	1	1	1	1	AUD+	2	2	GASTRIC PERFORATION	O	ND
41	KARUPPUSAMY	65	M	3053	L	1	2	2	1	2	1	1	1	2	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
42	SASIL	23	M	51670	H	1	1	2	2	2	2	1	1	1	1	2	AUD+	1	1	DUODENAL PERFORATION	H	ND
43	RAMARATHIANAM	56	M	83686	M	2	1	1	2	2	1	2	1	1	2	2	AUD+	1	1	DUODENAL PERFORATION	H	ND

S.NO	NAME	AGE	SEX	IPNO	SES	ALCOHOL	SOMKING	TOBACCO	S.FOOD	NSAIDS	APD	PAIN	VOMITING	FEVER	HYPOTENSION	TACHYCARDIA	XRAY	RUT	HPE	DIAGNOSIS	HPKIT/OT	FOLLOW UP
44	ANBARASAN	33	M	13630	M	1	1	1	1	1	2	2	2	1	2	1	AUD+	2	2	GASTRIC PERFORATION	O	ND
45	SUBBU	77	M	22887	L	1	1	1	1	1	1	1	2	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
46	MUMMORTHY	27	M	946911	L	1	1	2	2	2	1	2	1	1	1	2	AUD+	1	1	DUODENAL PERFORATION	H	ND
47	KUMAR	22	M	105531	L	2	1	1	1	2	1	1	2	1	1	2	AUD+	1	1	GASTRIC PERFORATION	H	ND
48	JANAGAR	45	M	117415	L	1	1	2	1	2	1	1	2	1	1	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
49	BIRASH	24	M	133152	M	1	2	1	1	2	1	1	2	2	2	1	AUD+	1	1	DUODENAL PERFORATION	H	ND
50	THIRUMURGAN	45	M	131968	M	1	2	1	2	2	1	1	1	2	1	1	AUD+	1	1	GASTRIC PERFORATION	H	ND