"A STUDY IN THE INCIDENCE OF HELICOBACTER PYLORI IN CASES OF DUODENAL ULCER PERFORATION IN GOVERNMENT VELLORE MEDICAL COLLEGE"

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

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

M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

GOVERNMENT VELLORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

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This is to certify that the dissertation titled **"A STUDY IN THE INCIDENCE OF HELICOBACTER PYLORI IN CASES OF DUODENAL ULCER PERFORATION IN GOVERNMENT VELLORE MEDICAL COLLEGE & HOSPITAL"** is a genuine work done by ***Dr.G.AMARNATH*** Post Graduate student (2013–2016) in the Department of General Surgery, Government Vellore Medical College, Vellore under the guidance of **Prof. Dr. R. Rajavelu M.S., FRCS.,**

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Date: **Prof.Dr.G.Selvarajan, M.S.,DLO.,**
The Dean,
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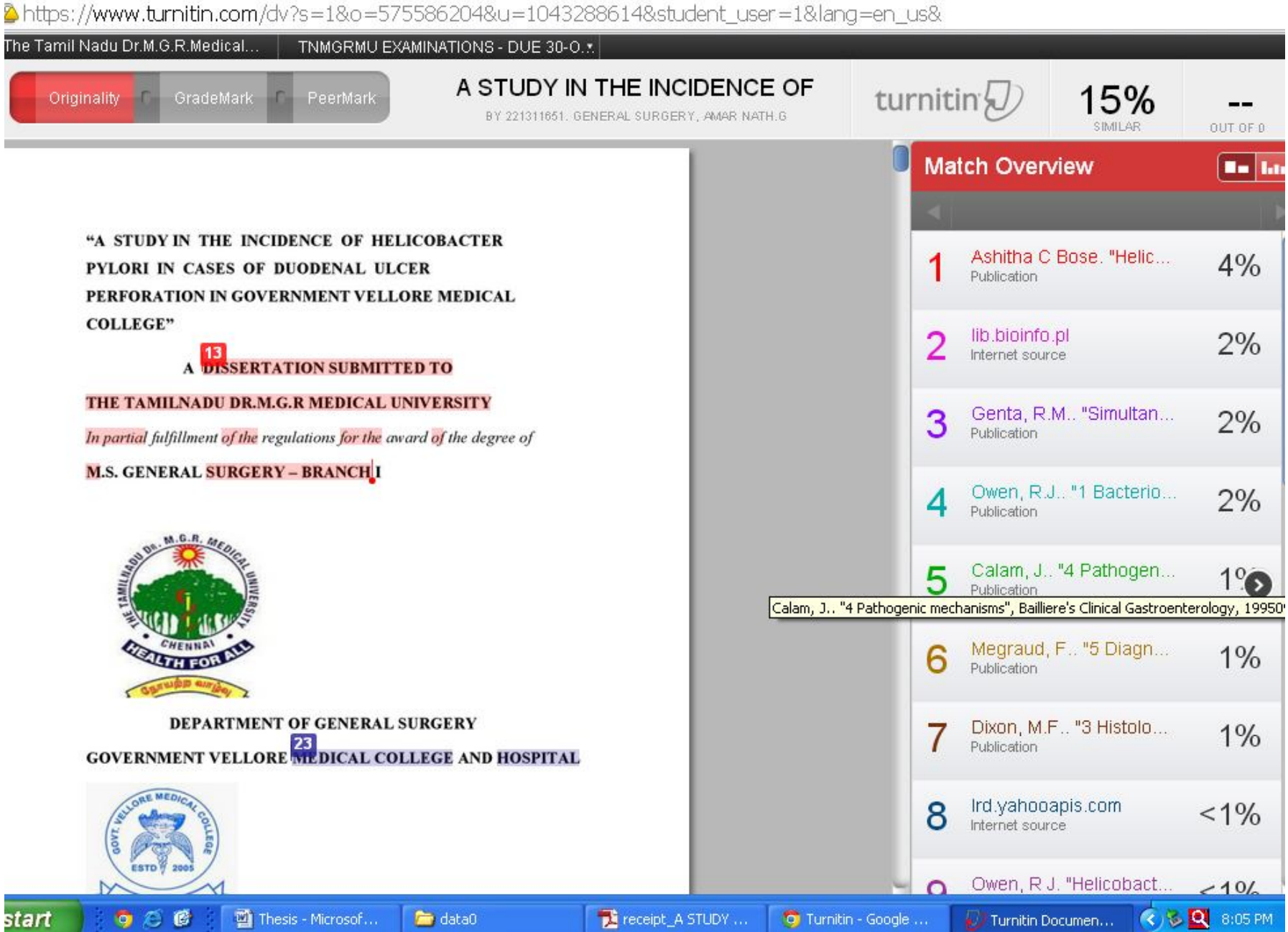
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This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of M.S.,Degree in General Surgery (Branch – I) ,Examination to be held in April 2016

Place : Vellore **DR. G.AMARNATH**

Date :

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I am extremely thankful to my patients who consented and participated to make this study possible.

LIST OF ABBREVIATIONS:

CAG –A	**-**	**Cytotoxin associated gene A**
CCK-PZ	**-**	**Cholecystokinin pancreozymin**
CO2	**-**	**Carbondioxide**
CNS	**-**	**Central nervous system**
DU	**-**	**Duodenal ulcer**
ELISA	**-**	**Enzyme linked immune sorbent assay**
G.I	**-**	**Gastrointestinal**
GU	**-**	**Gastric Ulcer**
H/O	**-**	**History of**
H. pylori	**-**	**Helicobacter pylori**
H & E	**-**	**Haematoxylin & eosin**
MALT	**-**	**Mucosa associated lymphoid tissue**
NSAIDs	**-**	**Non steroidal anti inflammatory drugs**
PUD	**-**	**Peptic Ulcer**
PMN	**-**	**Polymorphonuclear neutrophil**
TNF	**-**	**Tissue necrosis factor**
VAC – A	**-**	**Vacuolating cytotoxin A**

ABSTRACT

BACKGROUND:

Most of the patients having chronic peptic ulcer disease are usually found to be infected with helicobacter pylori infection. Previously, when the ulcer goes for perforation, immediate acid-reduction surgery was being done, as there was a high incidence of relapse of ulcers after a simple closure. But, since most of the ulcers are caused by H. pylori, eradication of these organisms reduces the recurrence of ulcers . The aim of our study was to study the incidence of Helicobacter pylori in cases of duodenal ulcer perforation.

METHODS:

Our study included 30 operated patients of duodenal ulcer perforation and the incidence of H. pylori was found from the biopsy taken from ulcer edge, using Rapid urease test & HPE.

RESULTS:

Among thirty patients, samples of 16 patients (53.3%) showed Positive for Helicobacter pylori in Rapid urease test (CLO TEST) and HPE. The p value calculated is < 0.05 which is found to be significant by chi square test.

CONCLUSION:

As Helicobacter pylori is the most common cause for duodenal ulcer perforation, anti-helicobacter pylori eradication regimen can be used in all cases of perforated duodenal ulcer to prevent the ulcer recurrence and to prevent acid reducing surgeries

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INTRODUCTION

Previously, the researches done on 'Duodenal ulcer' mainly concentrated on the abnormalities in the secretion of acids. Most of the researches found that all duodenal ulcer patients have raised maximal acid output (MAO), in response to histamine or pentagastrin, showing a greater parietal cell mass (Blair and others 1987). Also, the acid secretion physiology is abnormal. Basal acid is raised to greater extend, which is identified from the increase in MAO.

Researches done on hormonal control mainly concentrated on Gastrin, an antral acid stimulating hormone. Research showed that gastric acid inhibition by intra gastric acid is decreased in the duodenal ulcer patients (Calam & Moss 1992). An important mediator named somatostatin inhibits gastric acid, also inhibits immune reactive somatostatin cells. In patients with duodenal ulcer, amount of somatostatin peptide present in gastric antral mucosa are reduced (Mc Henry Jr et al 1993).

Normally, the gastrin release is inhibited by intragastric acid. This mechanism is altered in patients infected with Helicobacter pylori causing Duodenal ulcer.

Eradication of H.pylori from duodenal ulcer patients increases somatostatin RNA by about two folds in antral and duodenal mucosa, but not in corpus biopsies, this reflects typical distribution of involvement of mucosa in patients with duodenal ulcer (Sipponen 1992).[8]

The realization that Helicobacter Pylori may have a fundamental role in the etiology & the pathogenesis of peptic ulcer has lead to therapeautic strategies aimed at eradicating this bacterium and curing the disease.

Perforated peptic ulcer is approximately 7 to 10 cases per 100000 population per year. Perforation is present in about 7% of patients were admitted in hospital for peptic ulcer disease. Also it is the first manifestation of the disease in about 2% of duodenal ulcer patients. Whether, the eradication of H. pylori will reduce this complication has lead many investigators to find out the presence of Helicobacter pylori in duodenal ulcer perforation.

[12]The present study is done to find out the incidence of Helicobacter pylori in duodenal ulcer perforation, making use of RUT and Geimsa staining as the previous studies quoted did not have the advantage of a definite and sensitive staining method to detect H. pylori.

AIM OF STUDY

To study the incidence of Helicobacter pylori in a case of duodenal ulcer perforation in Government Vellore medical college & hospital

REVIEW OF LITERATURE

DUODENAL ULCER:

The duodenum extends from the pylorus about 20 – 30 cms and end at the ligament of Treitz. The duodenum is divided into four anatomic parts.

D1 - the cap / bulb

D2 - descending portion

D3 - transverse portion

D4 - ascending portion

90% of ulcers usually occurs in the duodenal cap region

The central issue that likes all theories for peptic ulcer disease is acid. Acid must be present for non-malignant ulceration of the upper gut to occur. The degree of acid secretion varies with the individual disease state ranging from the extreme hyper acidity of Zollinger – Ellision syndrome to the hypoacidity present with type I and IV gastric - ulcer.[38]

Epidemiological studies showed a strong association mainly between Helicobacter pylori infection & both gastric & duodenal ulcer disease. Treatment of the infection showed greater results in long – term cure of peptic ulcers. [5]The cause of peptic ulcer is complex multifactorial, as they result from interplay of effects of gastric acid and pepsin & the gastric mucosal barrier. Any entity that either increases acid & pepsin secretion or weakens gastric mucosal barrier can result in ulcers.

Also Gastric stasis causes peptic ulceration by inadequate clearance of normal amounts of acid & NSAIDS disrupt the prostaglandin – driven support of the mucosal barrier.

Recently works have shown 100% association of Helicobacter pylori in patients with duodenal ulcer. Evidence suggests that eradication of the H. pylori bacteria has led to a decreased recurrence rate and greatly supports the hypothesis that H-pylori is an etiologic factor.

Aetiology of Duodenal ulcer:

- Most commonly it is present O +ve in blood group

- Among all the most common etiological factor is Helicobacter pylori infection & it carries about 90%

- NSAIDs, Steroids.

- Alcohol, Smoking and also vitamin deficiencies can cause

- Zollinger – Ellison syndrome, MEN, Hyperparathyroidism are all some endocrine causes

- Even stress & anxiety can cause duodenal ulcer

PATHOLOGY:

Ulcers often occurs in duodenum first part, mainly in the first inch of it. Involves mainly the muscular layer of the duodenum. Ulcer causes cicatrisation leading to pyloric stenosis.

[37]Serosa lying over the duodenal ulcer often shows petechial haemorrhages with speckled red dots, giving appearance of sprinkled cayenne pepper.

When microscopically seen, the ulcers are found with chronic inflammation, granulation tissue, gastric metaplasia of duodenal mucosa, and also with endarteritis obliterans.

Kissing ulcers are two ulcers lying opposite involving anterior and the posterior surface of duodenum. Anterior ulcers often goes for perforation & the posterior ulcer goes for bleeding.

CLINICAL MANIFESTATIONS:

In our country, ratio of the ratio of duodenal ulcer to gastric ulcer is 30 : 1.

Commonly it involves all socio economic groups, more commonly involving stressed professionals. (Type A personality)

- usually presents with "hunger pain" (pain more before food, in early morning & decreases after taking food) Night pains are also more common.
- It commonly involves males
- Periodicity is more common when compared to gastric ulcer
- Vomiting, heart burn, and also water – brash can occur
- Hematemesis and malena can occur
- Increase in appetite and increase in weight gain
- Loss of weight & appetite occurs once the ulcer going for stenosis

- Patients often eats more frequently with out any restrictions
- Chronic duodenal ulcer can be in either uncomplicated or complicated forms

COMPLICATIONS OF DUODENAL ULCER:

Perforation

Bleeding

Obstruction (pyloric stenosis: mainly due to scarring and cicatrisation of duodenal first part due to chronic ulcer)

PERFORATION:

There will be a severe dyspepsia for few days prior to the perforation mainly in chronic peptic ulcer cases. Also there will be no premonitory symptoms when an acute ulcer perforates in a younger patients.

A sudden excruciating epigastric pain will be present at the time of perforation. Subsequent symptoms mainly depends on the

degree of peritoneal soiling & if the perforation is sealed by greater omentum. Also the pain becomes generalized later. Patient may also have an referred shoulder pain mainly due to diaphragmatic irritation. The contents from gastro duodenum may spill and spread along the right paracolic gutter and become localized in right iliac fossa and mimic acute appendicitis. Vomiting is usually uncommon, till the paralytic ileus is established.

[41]In elderly and seriously ill patients, perforation usually develops very gradually with generalized peritonitis with fewer intense symptoms and signs. Depending on the degree and the rate of peritoneal soiling, the physical signs varies.

Abdominal tenderness with guarding may be localized to upper quadrant to generalized. If the contamination spreads and involves whole of the abdomen, then a marked board like rigidity, rebound tenderness and a silent abdomen will be present. After the onset of paralytic ileus, abdominal distension occurs because of the subsidence of the musculature in the anterior abdominal wall. Also a variable degree of failure in the peripheral circulation will occur, which may present as tachycardia, hypotension, cold peripheries,

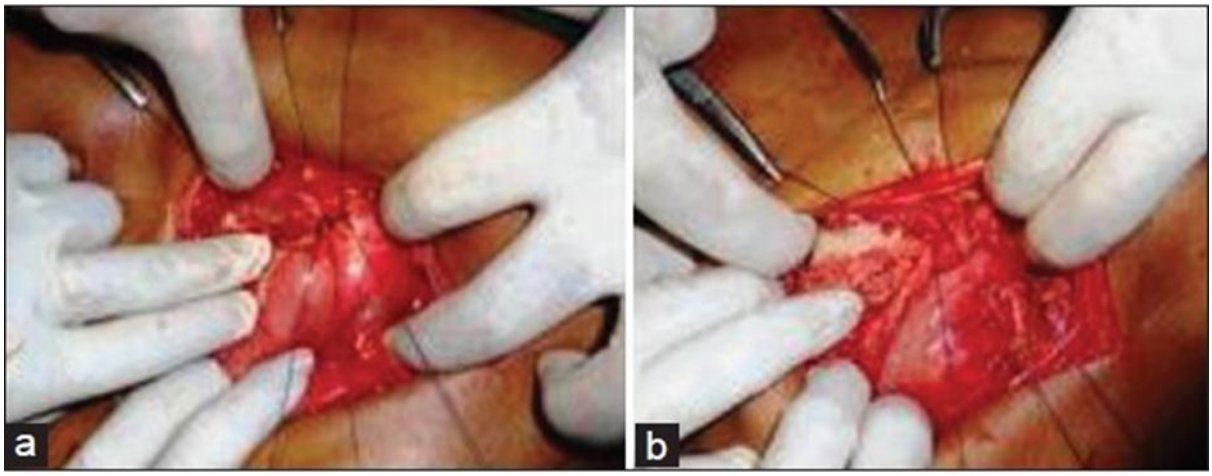
reduced urine output. The respiration will be very shallow and grunting.

Diagnosis is done mainly based on the symptoms, signs and the plain abdomen & chest radiograph taken in an erect posture. Lateral decubitus position can be used in a very ill patients. Sub-diaphragmatic air on the right side is pathognomonic of gastro duodenal perforation. Paralytic ileus may be present in more advanced cases. [36]If pneumoperitoneum is not seen radiologically, then differentiation between sealed perforation and acute pancreatitis has to be made. If doubt persists, then a diagnostic peritoneal lavage or tap has to be done.

Management : first step in treatment should be to correct hypovolemia and electrolyte imbalance. Proper peripheral circulation and proper urine output should be present for an operative management. Colloids are used for resuscitation measures. Oxygen support given. Pain should be relieved before physical examination. Intramuscular pethidine is usually very effective. Nasogastric aspiration to be done. Broad spectrum antibiotics given.

Some patients can be treated conservatively with nasogastric tube aspiration, iv fluids, antibiotics, analgesics, PPI or H2 blockers

followed by upper gastrointestinal scopy after the acute illness get settled. Main problem with conservative treatment is patient can develop residual abscess in subphrenic region which requires drainage.



[37]Open surgical technique consisting perforation of duodenum using Graham's Omental patch is the most commonly performed technique. Closure followed by thorough peritoneal lavage using 3 litres of warm normal saline should be given. Insertion of abdominal drains done.

HELICOBACTER PYLORI :

In 1982, Marshall & Warren from Australia cultivated a spiral organism similar to Campylobacter colonizing the stomach of

human were present in patients with type – B gastritis (chronic inflammation of stomach antrum) It was then named as campylobacter pyloridis which was then changed to the term campylobacter pylori. [12]But the organism was considered varying taxonomically from other campylobacter species, because of the presence of sheathed flagella, a unique fatty acid profile, a different respiratory quinines and a different 16s RNA sequence, and as a result they created a new genus Helicobacter. During the past five years the greater expansion occurred and now it includes 12 species, among those most are of non human origin and are not pathogenically significant.It is more common in the lower socioeconomic group.

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

Duodenal ulcer - 95%

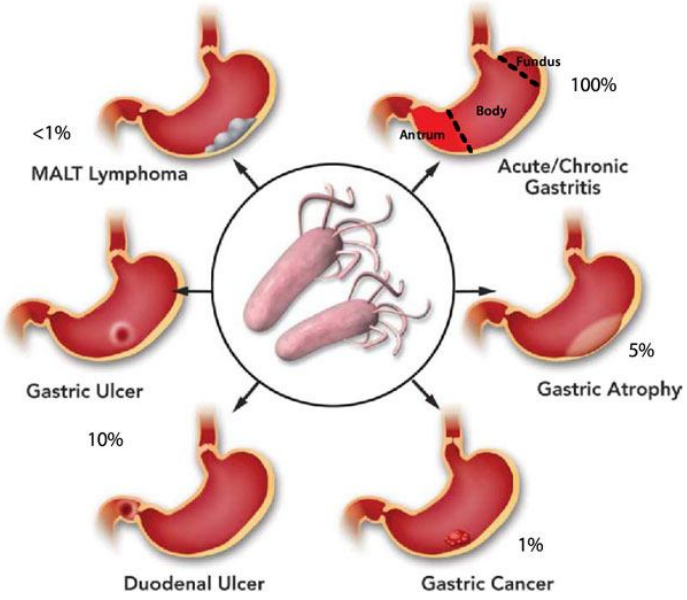
Gastric ulcer - 70%

Gastritis - 70 – 90%

H. pylori releases enzymes mainly like UREASE that hydrolyzes urea & releases ammonia which through negative feedback mechanism increases gastrin release from G – cells.

Also secretes dehydrogenase (that converts alcohol to aldehyde which is very toxic to the mucosa), endopeptidase (disrupts mucosal barrier).

[12]Urease creates an alkaline environment around it in mucus layer of gastric epithelium. It can survive only in gastric epithelium or gastric metaplasia in the duodenum or barett's esophagus or in heterotropic gastric mucosa in meckel's diverticulum or rectum. Because, receptors for these organisms to adhere into mucosa are available only in gastric mucosa.



Helicobacter pylori impairs mucosal healing and also causes degranulation of eosinophils. It also releases many number of proteases & lipases which breaks mucus & the barrier. It also secretes cytotoxins such as (cag A and vac A). These causes inflammatory reaction or malignancy.

Normal duodenum harbor the bacteria H. pylori, duodenum having gastric metaplasia can be infected by the organism. This tells, why the Helicobacter pylori are involved in causing the duodenal ulcer.

HABITAT :

In human stomach, it is mainly present in the mucous layer of gastric epithelium mostly involving the antrum. Also it is documented that theseorganisms can also be cultured from metaplastic gastric epithelium in the duodenum.

BLOOD : Recently, Helicobacter pylori were also recovered from the blood culture of a patient having malignant lymphoma of the upper GIT including stomach.

ORAL CAVITY:

H pylori occasionally was isolated from the saliva and from dental plaques. Recovery from the Oral cavity is very difficult.

FAECES ; Polymerase chain reaction are successfully done to identify the Helicobacter pylori in faeces at a greater frequency.

ANIMAL SOURCES :

H pylori are isolated from various animal species.

Including rhesus monkey, baboon, pigs& domestic cats.

Rhesus monkey is the only natural reservoir.

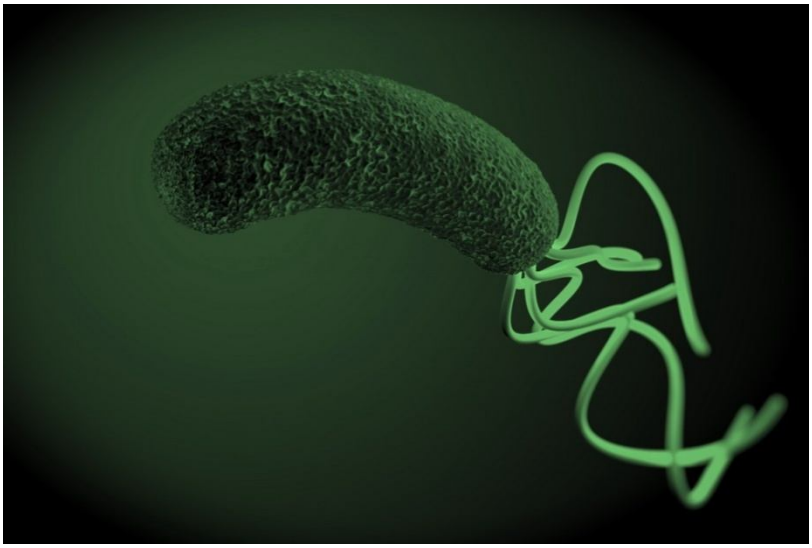
ENVIRONMENTAL SOURCES :

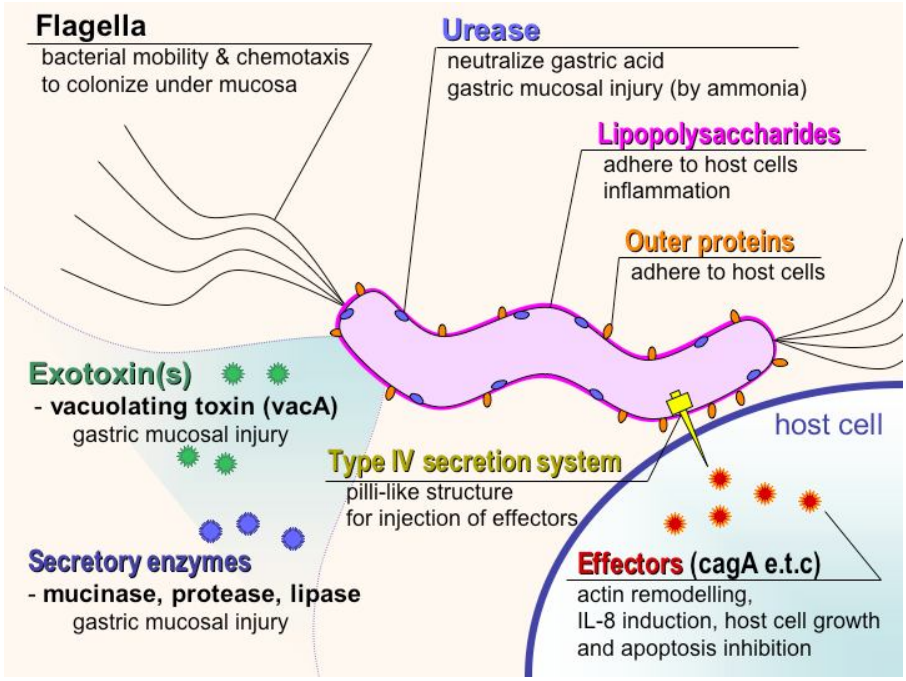
No free Helicobacter pylori are been isolated from soil, water or other environmental sources.

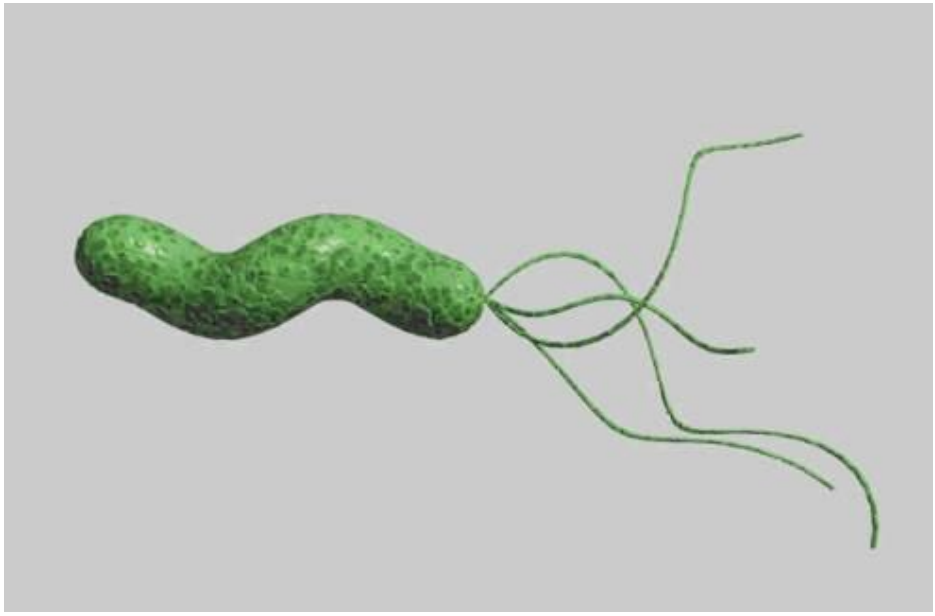
MICROBIOLOGICAL FEATURES :

Cellular morphology : [2]Helicobacter pylori is a gram negative, curved rod or S shaped (0.5 - 0.9 micro meters wide and 2-4 micro meters long). It has 1 to 3 turns when observed in vivo. In blood agar cultures (in vitro) no spores were formed. Using

light microscopy, Helicobacter cells found to have about 5-6 polar (lophotricate) flagella filaments at 2 days of growth. They are highly motile via polar flagella & produce an abundance of urease.







Colonial Morphology : Colonies from primary culture on supplementation with blood agar at 37^0C takes around 3 – 5 days to

appear & are circular (1-2 mm). they appear convex and translucent.

Coccoid Bodies : Older cultureshows a morphological change from bacillary to coccoid forms along with a loss in culturability. These forms can be found in faeces and could also be responsible in oro-faecal route transmission between humans

Physiological Properties : Helicobacter pylori are microaerophilic and grows well in an atmosphere of 5% O2 with 5 - 10% CO2, on blood containing media like " brain heart infusion agar "(BHI). BHI contains 5% horse blood agar mainly supplemented with 1% Isovitalex. All strains grow over a relative narrow temperature range of 33 to 40 degree Celsius. Grow over a wide range of pH 5.5 – 8.5.

Biochemical Properties :

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

Virulence Factors :

Promote Colonization	**Induce Tissue Injury**
Motility	Lipopolysaccharide (LPS)
Induction of hypochlorhydria	Leukocyte recruitment and Activating factors
Urease Production	Cag A and Vac A proteins
Adherence	Heat shock protein
Ptype ATPase	

Urease : The enzyme urease has nickel, that digests urea, which passes freely from plasma into the gastric juice. Finally, alkali is produced

$NH_2\text{-}CO\text{-}NH_2 + H_2O \rightarrow CO_2 + 2NH_3$ Then spontaneously at neutral pH

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

[2]In Helicobacter pylori infected patients, the gastric juice contains less urea and more ammonium than the normal. Ammonia

generated by H. pylori Urease activity buffers the bacterial hydrogen ions in gastric acid juice, and provides a source of nitrogen for H pylori. Besides its protective role, ammonia may also alter the permeability of gastric epithelium, and mucous ionic integrity and also it diffuses hydrogen ion back towards the gastric mucosa, resulting in mucosal injury.

MODE OF SPREAD :

The route of transmission is still unknown. The high rate of infection in children from H pylori positive parents and the presence of the same strain within members of the same family suggest that close contact is very important for the spread of infection. Animal studies revealed oral-oral transmission.In 80 – 90 % of population, infection is feco-oral route.

Iatrogenic person to person transmission via endoscopes has been reported, and the high prevalence of infection among endoscopists those who do not use gloves, suggest that transmission occurs through instruments contaminated with gastric secretions.

PATHOGENESIS & IMMUNITY :

Is a remarkable bacterium in its ability to establish life - long colonization in the stomach of untreated humans. Multiple factors contribute to the gastric colonization, inflammation, alteration of gastric acid production, & tissue destructionthat are characteristic of H. pylori disease. Initial colonization is facilitated by a) bacterial acid inhibitory protein which blocks acid production & b) bacterial urease activity produces ammonia that neutralizes gastric acid.

[7]The actively motile H. pylori then passes via the gastric mucus and then adheres to the gastric epithelial cellsby multiple surface adhesion proteins. Surface proteins can also bind host proteins & help the bacteria evade immune detection. Urease byproducts, mucinase, phospholipases can cause localized tissue damage and the activity of the vacuolating cytotoxin A (Vac A), a protein that after endocytosis by epithelial cells, producing vacuoles and damages the cells.

One more factor is cytotoxin – associated gene (cag A) that resides on a pathogenicity island containing approximately 30 genes. These genes encode a structure that acts like a syringe to inject the Cag A protein into the host epithelial cells, which

interferes with the normal cytoskeletal structure of the epithelial cells.

The cag phosphoribosylanthranilateisomerase (PAI) genes also stimulates interleukin – 8 (IL-8) production, which attracts neutrophils. Neutrophils releases Reactive Oxygen Molecules and proteases that causes gastritis and gastric ulcers.

H PYLORI AND DUODENAL ULCER :

More than 50 % of people world wide harbor H.pylori infection but less than 10 % of those infected with PUD.

Number of factors determine whether H.pylori infection causes disease:

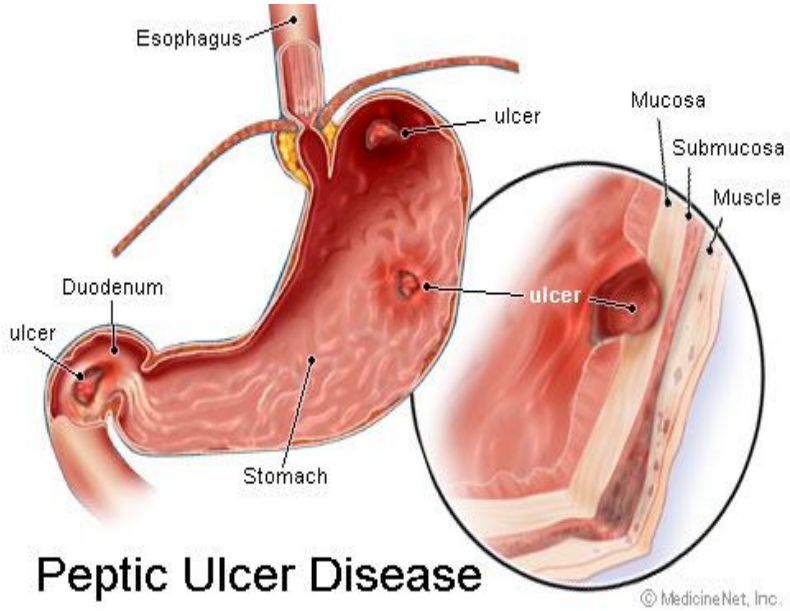
- Histologic gastritis induced pattern
- Alteration in homeostasis of gastrin & acid secretion
- Gastric metaplasia acquiring in the duodenum
- H.pylori interaction with the mucosal barrier
- Strain of H.pylori present

In patients with duodenal ulcer, inflammation, severity, infection, density are greatest mainly in the distal antral region. The

acid secreting body mucosa is spared. In response to stimulation with gastrin, duodenal ulcer patients with H.pylori produce more acid than infected patients without ulcers. This may result from an impaired acid secreting ability of the nonulcer - H.pylori infected patient's more diseased acid secreting fundus mucosa. Increased gastric acid can lead to the development of gastric metaplasia in duodenal bulb. This is a necessary forerunner to colonization of the duodenal epithelium with H. pylori. [7]The metaplastic H.pylori colonized, duodenal epitheliumthen becomes more susceptible to acid & pepsin effects and ulceration. After the eradication of H. pylori infection, gastric metaplasia in the duodenum does not revert to normal, but with elimination of infection, the risk of ulcer recurrence is eliminated.

[16]H. pylori infection alters the gastrin release negative feedback somatostatin secreted by antral D cells. Somatostatin causes inhibition of gastrin by paracrine effect. The bacteria produces alkaline ammonia on both the surface epithelium & in the antral glands prevents the D cells from properly interpreting the level of acid present. This leads to low levels of somatostatin, thus loss of gastric inhibition. Chronic hypergastrinemia caused by H. pylori

exerts a trophic effect & hyperplasia of the acid secreting parietal cells.



Infection with H. pylori also interferes with the neural connections b/n antrum & fundus that down regulates acid production. This impaired neural control, coupled with hypergastrinemia leads to further increases in acid production. With H. pylori eradication, hypergastrinemia rapidly resolves. There is a great deal of variations in the virulence of different strains H. pylori. More virulent strains have increased production of toxic enzymes & tend to be more adherent to the mucosa. They appear

to produce more urease that catalyzes the production of ammonia, further enhancing their potential for harm. Some genotypes of H. pylori appear to be particularly toxic & are more common in patients with peptic ulcers. These are vac A -positive & cag A-positive. There is also a genetic predisposition to acquire H. pylori infection. This has been demonstrated in monozygotic & dizygotic twins with the increased risk in monozygotic twins.

[22]Wyatt et al (1987) first emphasized, the relation between H pylori gastritis, gastric metaplasia and active chronic duodenitis . A sequence of events was made that showed metaplasia (acid–induced), infection that spread from the stomach and following colonization of the gastric epithelium, the development of an active chronic inflammatory cell response in the duodenum.

Thus H pylori infection and gastric metaplasia(acid induced) are essential prerequisites for the development of duodenal ulceration and active chronic duodenitis.

The appearance of patches of gastric mucus types of cells among the absorptive cells over the duodenal villi is called gastric metaplasia. It is restricted mainly to the epithelial surfacealthough the parietal cells acquisition occurring in chronic duodenitis might

show a parallel glandular metaplasia which could conceivably contribute to the local acid production.

DIAGNOSIS OF H PYLORI :

Detection of H pylori can be based on methods :

1. **Invasive :** based on biopsy samples and needs endoscopy.
   - **a)** Rapid urease test (CLO / Campylobacter like organism Test)
   - **b)** Histology
   - **c)** Culture
   - **d)** Polymerase chain reaction (PCR)

2. **Non Invasive :**
   - **(a)** Serology
   - **(b)** Urea breath test

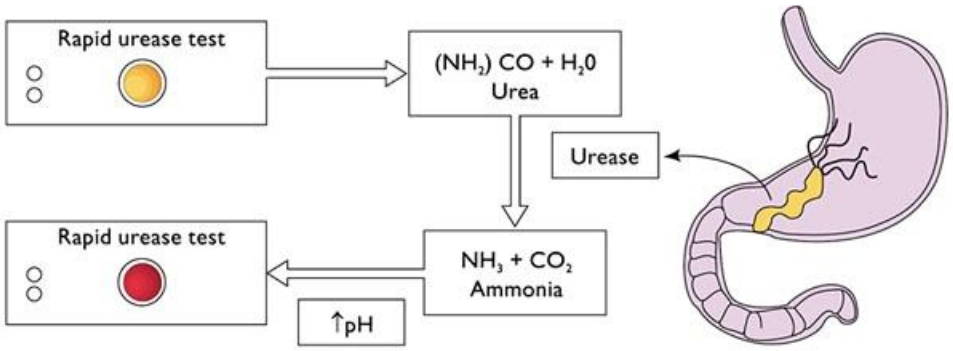
I (a) : Rapid urease test :

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.

Genta stain, 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).

[12]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.

[12]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.

TABLE : **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	88	+	no

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.[12] 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 in

nitrocellulose 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 to

rinse 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.

HELICOBACTER PYLORI ERADICATION REGIMEN:

[29]Triple therapy includes 2 antibiotics and one proton pump inhibitor for 7-14 days. 2-week therapy with omeprazole, amoxicillin, and clarithromycin achieved a significantly higher eradication rate than 1- or 2-week regimens with metronidazole.

- 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)

Four quadrant regimen includes triple regimen plus bismuth subsalicylate.

A longer duration of treatment (7 vs 14 days) may be more effective in curing infection but this remains controversial. A meta-

analysis suggested that extension of PPI-based triple therapy from 7 to 14 days was associated with a 5 percent increase in eradication rates. Most studies included were based upon amoxicillin-based triple therapy.

REVIEW OF LITERATURE:(Previous Studies)

Edward C.H.Chung Surgical unit, United Christian Hospital, Kwnutong, Hong Kong.

Using an urease indicator (CLO test) the Helicobacter pylori prevalence among peptic ulcer perforation was studied prospectively.

Of 59 subjects, 44 PDU (84% positive for CLO)

15 PGU (67% positive for CLO)

Subjects who have perforation were initially documented to have peptic ulcers, were predominantly CLO test + ve, then concluding that ulcer relapse is because of Helicobacter pylori which also got supporting evidence by a latest H. pylori eradication studies done on non-perforated ulcers.

All adult patients diagnosed as, and operated for , perforated peptic (gastric and duodenal) ulcer within the 12 month period of march 1991 to February 1992 were prospectively entered into this

study. Cases were excluded subsequently if the perforation was due to Causes other than peptic ulceration, e.g., tumours, foreign bodies. Details of previous ulcer disease were specifically sought and the use of antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), Steroids, medications for peptic ulcer by subjects within the preceding one week were also ascertained.

Discussion

H. Pylori infection is frequently seen among peptic ulcer patients in Hongkong. The prevalence rates reported in two recent local studies are 67% for gastric ulcer and 84% for duodenal ulcer. ”the corresponding figures for PGU and PDU of 53.3% and 86.4% derived from this survey are comparable. Detection of H.pylori within the ulcer especially duodenal ulcer is generally difficult because an ulcer crater represents an inhospitable place for this fastidious microbe.

Conclusion:

This study has demonstrated that H. Pylori is present both at the ulcer edge and antrum in a large percentage of perforated ulcers and ulcer relapse in the form of perforation is probably correlated with pre-existing H. pylori infection. What remains to be evaluated

is the relationship between the presence/absence of infection and future relapse, and perhaps also H. pylori eradication and relapse, in PPU patients treated with simple repair only.

	ANTRUM	ULCER EDGE	NO: OF SUBJECTS
PDU	+	+	18
	+	+	15
	-	+	5
	-	-	6 (Total -44)
PGU	+	+	4
	+	-	3
	-	+	1
	-	-	7 Total – 15)

Percentage with atleast one site positive: PDU-86.4% & PGU – 53.3%

Professor Vikram Kate, professor of Surgery, Jipmer, Pondicherry.

Reports on association between H. pylori infection and perforated duodenal ulcer are conflicting and few.

Following a simple closure after perforation, there was a high prevalence of H. pylori infection leading to relapse of duodenal ulcer (Sebastian et al) A report from Hongkong showed 80% association of H. pylori infection in duodenal ulcer perforation.A

recent North India report also showed a similar association. 17 patients who were diagnosed to have perforated duodenal ulcer were found to be positive for Helicobacter pylori infection and these patients were followed up for a period of 10.9 weeks . 13 among them had active duodenal ulcer while among the non infected patients ulcer prevalence was found to be nil. Kate et al, reported that Helicobacter pylori associated with duodenal ulcer perforation does not vary much that of control, but at every follow up he found that infection rate was assessed to be high among patients who had persistent or residual ulcer following a simple closure done for a perforation duodenal ulcer.

[31]Reinbach et al. reported that the prevalence of H. Pylori infection in patients with perforated duodenal ulcer was 47%, which was similar to the 51% seen in controls. Other reports also did not find an association between perforated duodenal ulcer and H. pylori infection. In view of these conflicting reports there is a need for a prospective study after simple closure of a perforated duodenal ulcer to determine whether eradication of H. pylori reduces the incidence of recurrent/residual ulcer after simple closure. As the association of H. pylori occurrence and ulcer recurrence has already

been shown by our earlier study, randomization to eradication therapy and no treatment arms was not considered necessary.

Our previous study using the standard triple drug regimen with metronidazole as one of the drugs failed to clarify the role of eradication rate with the regimen was only 52% at 12 months follow up. Hence, a four-drug regime with a high reported eradication rate was chosen now for this study.

As the association between H. pylori and ulcer recurrence was demonstrated in our previous study, attention was focused mainly on the effect of eradication on H. pylori on the persistence/recurrence after simple closure. The results of the present study showed that in the eradicated group, the ulcer persistence or relapse rate was significantly lower at 18.6% compared to the 70% seen in the non- eradicated group.

In conclusion, we can state that H. pylori infection does play an important role in duodenal ulcer relapse following simple closure of perforated duodenal ulcer. Eradication of H. pylori infection significantly reduces the relapse of duodenal ulcer. Combining acid reducing surgery with Omental patch closure is, therefore, not necessary for ensuring cure. Hence, anti H.pylori therapy using

regimens with high eradication rates should be prescribed for all patients with perforated duodenal ulcer who are positive for H.pylori infection after simple closure.

Racial differences in Helicobacter pylori seroprevalence in Singapore:

[33]Correlation with difference in peptic ulcer frequency. Helicobacter Pylori Journal of gastroenterology & hepatology. 12 (9/10) : 655-659 October 1997.

The aim of this study was to determine, whether racial differences exist in the seroprevalence of Helicobacter pylori infection in Singapore, and whether these difference correlate with racial differences in peptic ulcer frequency. A commercial serological test for immunoglobulin (IgG) antibody to H. pylori which was 90% sensitive and 83% specific in our population was used to screen 403 adult blood donors of Chinese, malays and Indian origin, aged between 15 – 60 years. Serum specimens from 84 paediatric patients admitted to the paediatrics Department, national university of Singapore, with non-gastroenterological illnesses were also tested. In all three races, seroprevalence of H. pylori increased

with age. Indians have the highest prevalence of infection followed by Chinese and Malays. Peptic ulcer prevalences are known to be highest in Chinese, followed by Indians and Malays. [33]The Malays have the lower prevalence of H.pylori and peptic ulcer among the three races in Singapore. Indians have a higher prevalence of H. pylori antibodies but a lower frequency of peptic ulcer than the Chinese. Racial differences in peptic ulcer frequency between Chinese and Indians are not explained by the prevalence of H. PYLORI

MATERIALS AND METHODS

Thirty patients who underwent surgery for perforated duodenal ulcer on emergency basis in the department of general surgery in Government vellore medical college between August 2014 to July 2015 were included in this study.

Inclusion criteria

1) Patients between 18-70 years of age.
2) Patients having perforated duodenal ulcers.

Exclusion criteria

1) Patients below 18 years and above 70 years of age.
2) Patients on NSAID's for more than one month duration.
3) Patients who have received Anti-Helicobacter pylori treatment.
4) Patients with gastric ulcers or ulcero proliferative growth.

The study population consisted of 30 patients between age group of 18 – 55 yrs. Exploratory laparotomy was performed in all cases. Two mucosal biopsies were taken through the perforation site. [40]One specimen is immediately put into a preformed H.pylori

detection kit for rapid urease test (RUT), which shows the presence of urease producing bacteria by a change in the colour of the medium within a time frame which is read as follows:

1. If a pink color develops within 30mins the test is taken as strongly positive.

2. If a pink color is not developed within half an hour but develops within two hours then the test is moderately positive.

3. If a pink color is not developed within two hours but develops within 24 hours then the test is is weakly positive.

4. If a pink color is not develop at the end of 24 hours then the test is negative for H pylori.

The **Second biopsy specimen** is fixed in 10% formalin solution and subjected to genta staining in the department of department of pathology. This is a novel staining procedure that allows the unencumbered observation of the histopathologic characteristics of the tissue while optimally demonstrating H. pylori.

Formalin fixed duodenal specimen is processed, cut into 4 micrometer sections and placed on regular microscopic slides. To

melt the paraffin, slides are dried in an oven at 60 degree centigrade for 15mins followed by microwaving for 3mins. Slides are then stained according to the following procedure:

1. Deparaffinize the sections and rehydrate the sections in distilled water.

2. Sensitization of sections is done by placing them in 1% aqueous uranyl nitrate at room temperature for three minutes, followed by transferring them to distilled water.

3. Rinse Slides in distilled water thoroughly as there is chance for cross contamination.

4. Keep the section in 1% AgNO3 (silver nitrate)at room temperature and then heat them together.

5. The microwave oven should be setup to heat below boiling point.

6. Rinse slides for three times in distilled water until the chance of contamination is eliminated.

7. Rinse it two times in 95% alcohol.

8. Rinse it two times in 100% alcohol.

9. Keep the slides in 2.5% gum acacia for 5 mins.

10. Air drying of sections is done for 1min.

11. Rinse it two times in distilled water.

12. Reduce it in the reducing solution, in a 45 degree centigrade water bath for 10 – 15 min or until the sections have developed a dark brown H. pylori and a light yellow.

13. Rinse the sections in water to stop reduction .

14. Stain in alcian blue with pH 2.5 for 10 mins.

15. Rinse the slide in running tap water.

16. Stain the sections in hematoxylin for 5 mins.

17. Rinse it in running tap water.

18. Quickly Dip the slide in acid alcohol.

19. Rinse it in ammonia water 0.25% until it turns blue.

20. Stain the sections in eosin for 5 Mins.

21. Quickly rinse in tap water.

22. Rinse the slide twice in 95% alcohol.

23. Rinse it three times in absolute alcohol.

24. Rinse it three times in xylene and place the coverslip and examine under microscope.

Reducing solution: 10 ml of 2.5 % gum acacia, 25 ml of 2% hydroquinone, 5ml of absolute alcohol and 2.5ml of 0.04% silver nitrate, unfiltered.

STATISTICAL ANALYSIS :

Descriptive results were expressed as mean, SD and percentages, qualitative variables were assessed using chi square test. Probability value (p value) was used to determine the level of significance p value < 0.05 was considered as significant, p value < 0.01 was considered as highly significant.

PATIENT DATA

CASE STUDY NO : 1

A 25 yrs old Dhineshkumar, engineering student from vellore IP No : 3214, presented to me with pain abdomen of 2 days duration, h/o vomiting& fever and passing scanty urine 2 days and constipation of 2 – days Pt is a Known alcoholic, smoker, and a known case of acid peptic disorder - proved to have duodenal ulcer not on proton pump inhibitors, not a known asthmatic,DM, HTN.

Palpation findings per abdomen: rigidity +, diffuse tenderness all over the abdomen, more in the right hypochondrium and epigastrium, guarding +, rebound tenderness +,

Investigations: complete blood count, Rbs, Rft, blood urea, sr. creatinine, Serum electrolytes, HIV, HbsAg, Xray erect abdomen, CXR- PA view, USG abdomen, ECG, Blood grouping, rh typing

Hb – 13gms,

Blood grouping – B positive,

RBS : 112 mgs

B/U :26 mgs

S/C :0.8 mgs %

Na+ : 140mEq

K+ : 4.5 mEq

Cl : 102mEq

HIV : Negative

HBs Ag :Non reactive

X-ray erect abdomen - Showed air under the diaphragm, suggestive of pneumoperitoneum

USG abdomen: moderate free fluid in abdomen and pelvis-

Patient data : PR : 102/min, BP : 118 / 68 mm Hg

Cvs : S1 + S2 Normal, RS : B/L air entry +,

UOP : 1200 ml

Patient was diagnosed with pneumoperitoneum -? hollow viscus perforation, resuscitated, prognosis explained, High Risk consent taken, and planned for Emergency Laparotomy

Under Epidural anaesthesia, patient put in supine position , parts painted & drapped, Upper midline incision taken, abdomen

opened in layers , peritoneum opened, bile stained peritoneal fluid about 2 litres sucked out , about 1cm x 1 cm perforation noted in 1st part of duodenum -2 bits of duodenal mucosal biopsy taken from perforation site, perforation closed with no 2 - 0 vicryl with onlay omental patch, peritoneal toilet given using warm saline and bilateral drains fashioned across both the flanks - left in the pelvis and right in the right sub hepatic area, after counting instruments & pads and checking for haemostasis , abdomen closed enmass using No : 1 prolene, skin sutured with No :1 mersilk, aseptic dressing applied.

Patient withstood the procedure well,

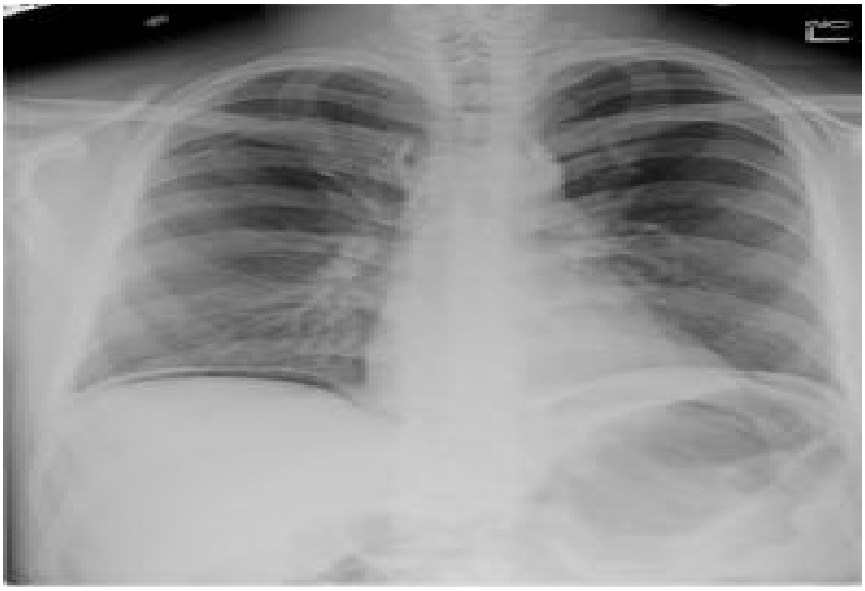
Immediate post operative period uneventful,

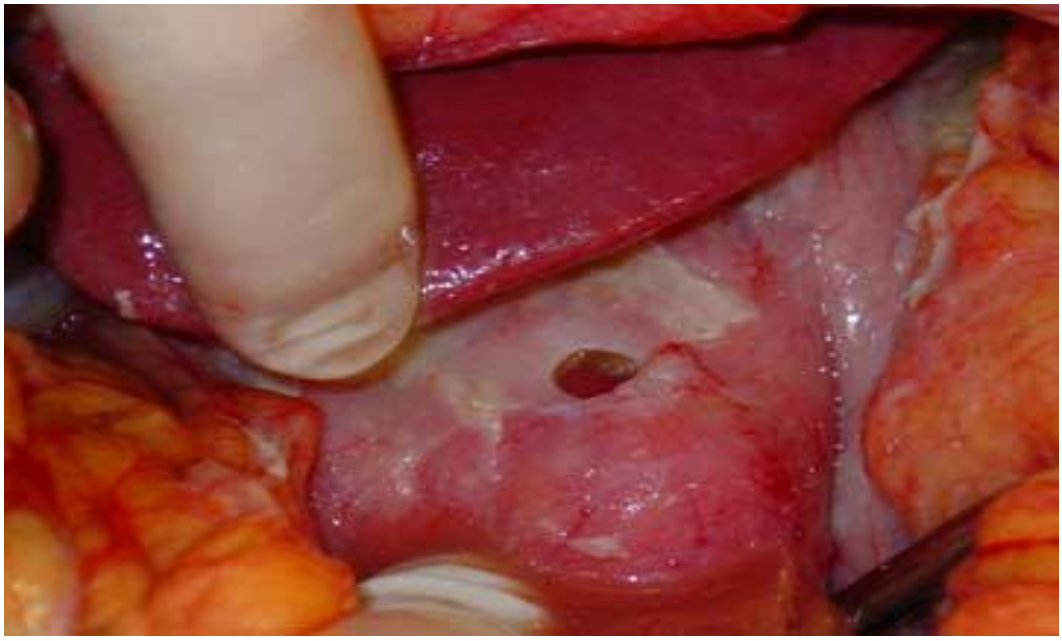
The biopsy specimens taken were subjected one for RUT test another for histopathology to pathology laboratory.

Patient treated with NPO / RTA, IV FLUIDS – 2500 ML / inj. piperecillin +tazobactum 4.5 gmsBD / Metrogyl500mg TDS / amikacin 500mg BD / pantaprazole 40mg / tramadol 100 mg / TPR / BP- 2nd hrly / I / O chart. Patient extubated after 2 days .Orals were started on day-5. Patient drains were removed on day -7, suture removal done on

day - 9, patient was advised H.Pylori kit, and advised for follow up with upper GI endoscopy.

Chest xray showing Air under diaphragm:





Duodenal ulcer perforation

CASE STUDY NO : 2

A 40yrs old Sekar , from Ambur , IP No : 3044 , presented with pain abdomen of 2 – days duration, distention of abdomen, vomiting 4-5 episodes / day, fever - 2 days and constipation of 2 – days

Pt is a Known alcoholic, smoker, not a known DM, HTN, TB, Asthmatic

Pt k/c/o APD takes PPI occasionally.

Palpation findings per abdomen: distention & diffuse tenderness all over the abdomen, epigastrium,Right hypochondrium guarding +, rigidity +,

P/R - no fecal staining,

Investigations: complete blood count, Rbs,Rft, S/C, B/u, Serum electrolytes, HIV, HbsAg,Xray erect abdomen, CXR- PA view, USG abdomen, ECG, Blood grouping, rh typing

Hp – 12.6gms,

Blood grouping – A - positive,

RBS : 120 mg/dl

B/U :30 mg/dl

S/C :1.0mg/dl

Na+ : 138mEq/l

K+ :3.8 mEq/l

Cl : 112mEq/l

HIV :Negative

HBs Ag :Non reactive

X-ray erect abdomen - Showed air under the diaphragm, S/O hollow viscus perforation

USG abdomen: minimal interbowelfree fluid , absence of peristaltic movements in the bowel - suggestive of hollow viscus perforation

Patient data : PR : 94/min, BP : 108/74 mm of Hg

Cvs : Normal, RS : Bilateral air entry +

UOP :750 ml, on catheterization.

Patient was diagnosed with ? hollow viscus perforation, patient resuscitated, prognosis explained, High Risk consent taken, and planned for Emergency Laparotomy

Under GA, Patientput in supine position, parts painted &drapped, premedicated , antibiotics given,

Midline incision made , abdomen opened in layers, peritoneum opened, bile stained peritoneal fluid about 1.5 litres sucked out, 0.5 cm x 0.5 cm perforation noted in 1st part of duodenum -2 bits of duodenal mucosal biopsy taken from perforation site, perforation closed with no 2 - 0 vicryl with onlayomental patch, peritoneal toilet given and bilateral drains fashioned across both the flanks - left in the pelvis and right in the right sub hepatic area, after counting instruments & pads and checking for haemostasis abdomen closed using No : 1 prolene, skin sutured with No :1 mersilk, aseptic dressing applied.

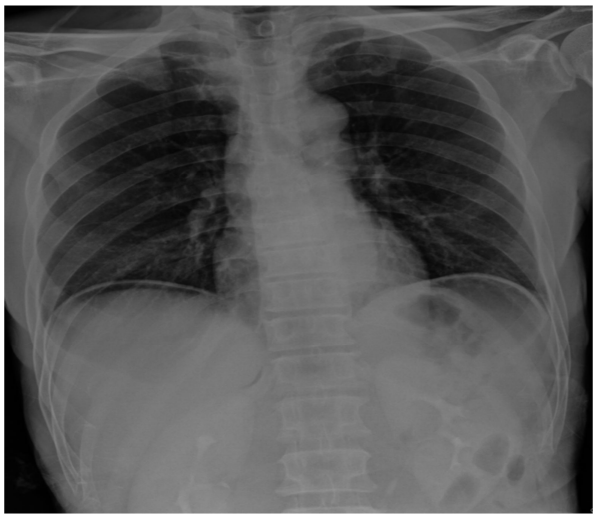
Patient withstood the procedure well,

Immediate post operative period uneventful,

The biopsy specimens taken were subjected one for RUT test another for histopathology to pathology laboratory.

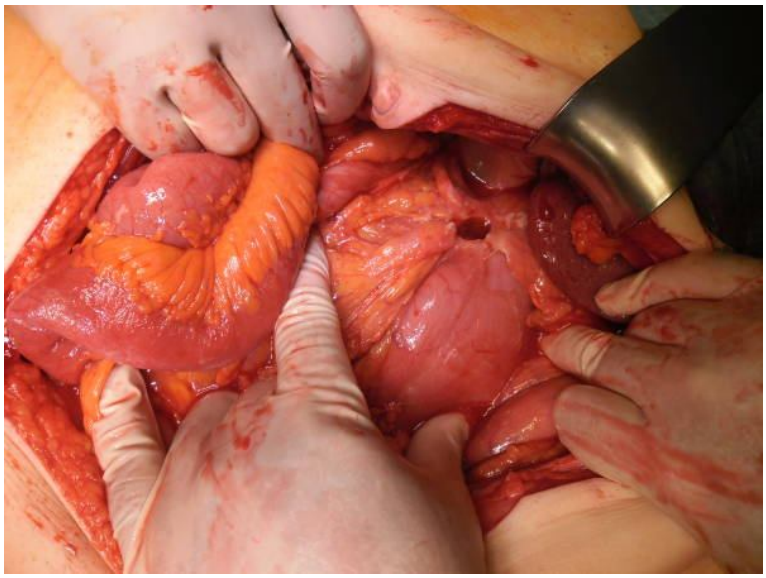
Patient treated with NPO / RTA, IV FLUIDS – 2500 ML / inj. Ceftriaxone +sublbactum 1.5 gms / metrogyl500mg BD / amikacin 500mg / pantaprazole 40mg / tramadol 100 mg / TPR / BB- 2nd hrly / I / O chart.

Orals were started on day- 5, Patient drains were removed on day -6, suture removal done on day - 10, patient was advised H.Pylori kit, and advised for follow up.



Xray abdomen erect showing air under diaphragm:

DUODENAL ULCER PERFORATION



CASE STUDY NO : 3

A 42yrs old male patient, Chidambaram , agricultural labourer, from gudiyatham, IP No : 3954, presented with pain abdomen for 3 days with distention of abdomen, vomiting8 episodes / day, fever and constipation of 2 – days

Patient is a Known alcoholic, smoker, known patient of acid peptic disorder, treated with Antacids not a known DM, HTN.

Palpation findings per abdomen: distention & diffuse tenderness all over the abdomen, diffuse guarding +, board like rigidity +, .

Investigations: Complete blood count, Rbs, Rft, S/C, B/U, Serum electrolytes, HIV, HbsAg, Xray erect abdomen, CXR- PA view, ECG, Blood grouping, rh typing

Hp – 11.5gms,

Blood grouping – A+ positive,

RBS : 96 mg/dl

B/U : 40 mg/dl

S/C : 1.1 mg/dl

Na+ : 138mEq/l

K+ : 3.9 mEq/l

Cl : 112 mEq/l

HIV : Negative

HBs Ag :Non reactive

X-ray erect abdomen - Showed air under the diaphragm, S/O hollow viscus perforation

Patient data : PR : 112 /min, BP : 104 /64 mm of Hg

CVS : Normal,

RS : Bilateral air entry +

UOP : 450 ml

Patient was diagnosed with ? hollow viscus perforation, resuscitated, prognosis explained, High Risk consent taken, and planned for Emergency Laparotomy

Under Spinal Anaesthesia, Patient put in supine position, parts painted & drapped premedicated, antibiotics given, Midline incision made, abdomen opened in layers bile stained peritoneal fluid sucked out. About 1 cm x 0.5 cm perforation noted in 1st part of duodenum -2 bits of duodenal mucosal biopsy taken from perforation site, perforation closed with no 2 - 0 vicryl with onlay omental patch, peritoneal toilet given and bilateral drains fashioned across both the flanks - left in the pelvis and right in the right sub hepatic area, after counting instruments & pads and checking for haemostasis, abdomen closed using No : 1 prolene, skin sutured with No :1 mersilk, aseptic dressing applied.

Patient withstood the procedure well,

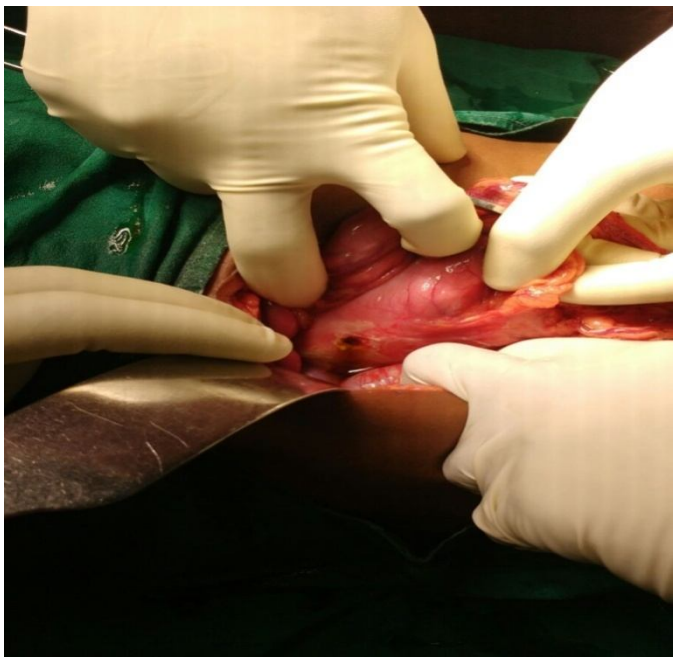
Immediate post operative period uneventful,

The biopsy specimens taken were subjected one for RUT test another for histopathology to pathology laboratory.

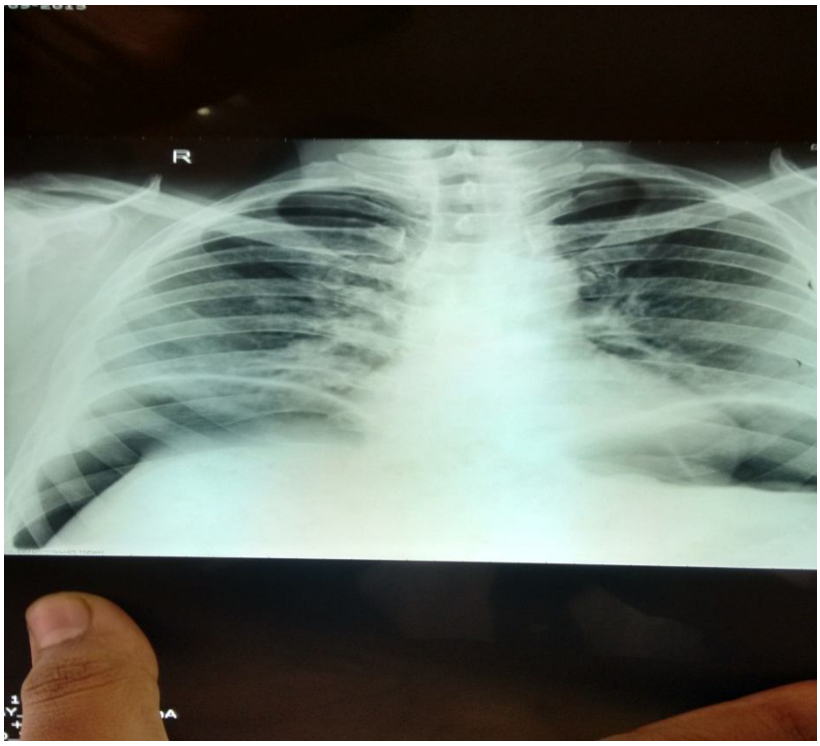
Patient treated with NPO / RTA, IV FLUIDS – 2500 ML / inj. Ceftriaxone + sulbactum 1.5 gms / metrogyl500mg TDS / amikacin 500mg / pantaprazole 40mg / tramadol 100 mg / TPR / BB- 2ndhrly / I / O chart.

Orals started on day- 5, Patient drains were removed on day - 6, suture removal done on day - 10 , patient was advised H.Pylori kit, and advised for follow up.

DUODENAL ULCER PERFORATION



XRAY SHOWING AIR UNDER DIAPHRAGM



CASE STUDY NO : 4

A 44 yrs old Srinivasan IP: 6827 , agricultural labourer from ambur Presented with c/o fever, vomiting, pain abdomen and increasing distention of abdomen and constipation of 3 – days

Patient is a known smoker, alcoholic, taking steroids , not a known DM, HTN, TB, Asthma

Palpation findings per abdomen: distention & diffuse tenderness all over the abdomen, guarding +, rigidity +.

Investigations: Complete blood profile, Rbs,Rft, S/C, B/U, Serum electrolytes, HIV, HbsAg, Xray erect abdomen, CXR-PA view, USG abdomen, ECG, Blood grouping, rh typing

Hb	:	14gms,
Blood grouping	:	A- negative,
RBS	:	110 mg/dl
B/U	:	80 mg/dl
S/C	:	2.2 mg/dl
Na+	:	135 mEq/l
K+	:	2.9mEq/l
Cl	:	96 mEq/l
HIV	:	Negative
HBs Ag	:	Non reactive

X-ray erect abdomen - Showed air under the diaphragm, S/O hollow viscus perforation

USG abdomen: free fluid in abdomen and pelvis, - suggestive of hollow viscus perforation

Patient data : PR : 114 / min, BP : 94 / ?mm of Hg

CVS : Normal, RS : Bilateral air entry +

UOP : nil

Patient was diagnosed with ? hollow viscus perforation, resuscitated, prognosis explained, High Risk consent taken, and planned for Emergency Laparotomy

Under GA, Patient put in supine, premedicated , parts painted & drapped Midline incision made, abdomen opened in layers, bile stained peritoneal fluid about 2 litres sucked out. About 1cm x 1 cm perforation noted in 1st part of duodenum -2 bits of duodenal mucosal biopsy taken from perforation site, perforation closed with no 2 - 0 vicryl with onlay Omental patch, peritoneal toilet given and bilateral drains fashioned across both the flanks - left in the pelvis and right in the right sub hepatic area, after counting pads

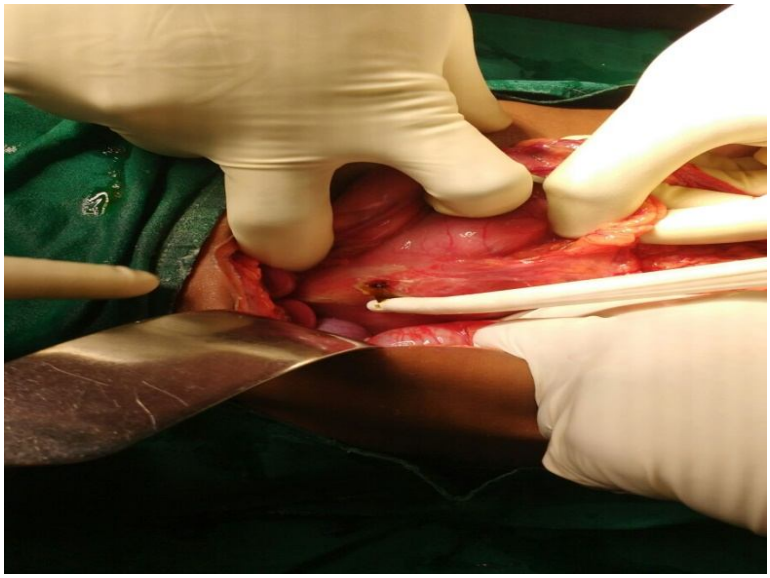
& instruments and checking for haemostasis abdomen closed enmass using No : 1 prolene, skin sutured with No :1 mersilk, aseptic dressing applied.

The biopsy specimens taken were subjected one for RUT test another for histopathology to pathology laboratory.

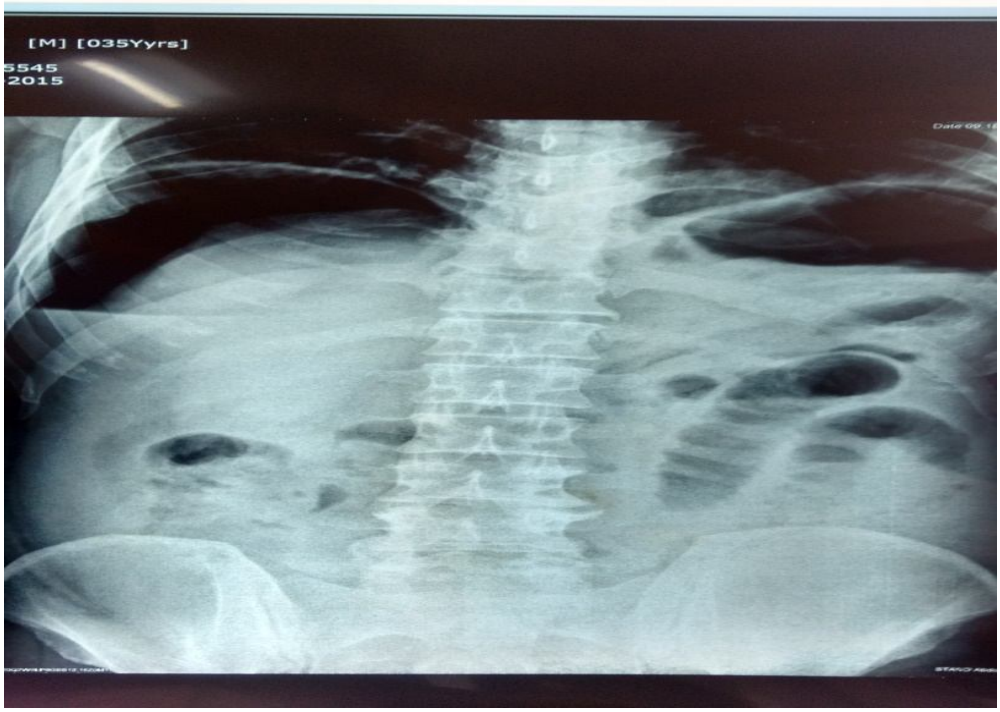
Patient treated with NPO / RTA, IV FLUIDS – 2500 ML / inj. Ceftriaxone + sulbactum 1.5 gms / metrogyl 500mg tds / amikacin 500mg / pantaprazole 40mg / tramadol 100 mg / TPR / BP- 2ndhrly / I / O chart.

Orals started on day – 4 . Patient drains were removed on day -6, suture removal done on day - 9, patient was advised H.Pylori kit, and advised for follow up.

DUODENAL ULCER PERFORATION



XRAY ABDOMEN ERECT SHOWING AIR UNDER DIAPHRAGM



CASE STUDY NO : 5

A 62yrs old Elumazhai, IP: 8782, farmer from katpadi, Presented with c/o pain abdomen of 4- days duration, Vomiting> 15 episodes, fever > 3 days, constipation of 4 – days

Patient is a known alcoholic for 20 yrs, not a smoker. known DM under treatment.Not a known HT / Asthmatic / TB

Palpation findings per abdomen: diffuse tenderness all over the abdomen, more in the rthypochondrium, epigastrium guarding +, board like rigidity +, obliteration of liver dullness +,

Investigations: Complete blood picture, Rft, Rbs, S/C, B/U, Serum electrolytes, HIV, HbsAg, Xray erect abdomen, Cxr- PA view, USG abdomen, ECG, Blood grouping, rh typing, Serum Amylase, Widal test.

Hb	:	9.8gms,
Blood grouping	:	O+ positive,
RBS	:	85 mgs
B/U	:	52 mgs
S/C	:	1.5 mgs
Na+	:	136mEq/L
K+	:	3.0 mEq/L
Cl	:	110 mEq/L
HIV	:	Negative

HBs Ag : Non - reactive

Widal Test : Negative,

X – ray abd – showed air under right dome of diaphragm

USG Abdomen: Bilateral minimal pleural Effusion, decreased / absent peristaltic movements of bowel loops, loculated free fluid in the abdomen.

Patient data

PR : 98 / min

BP : 90 / 70 mm of Hg

CVS : S1 + S2 – Normal,

RS : Bilateral air entry +, bilateral ronchi

UOP : 850 ml

Patient was diagnosed with hollow viscus perforation features of peritonitis, resuscitated, prognosis explained, High Risk consent taken, and planned for Emergency Laparotomy

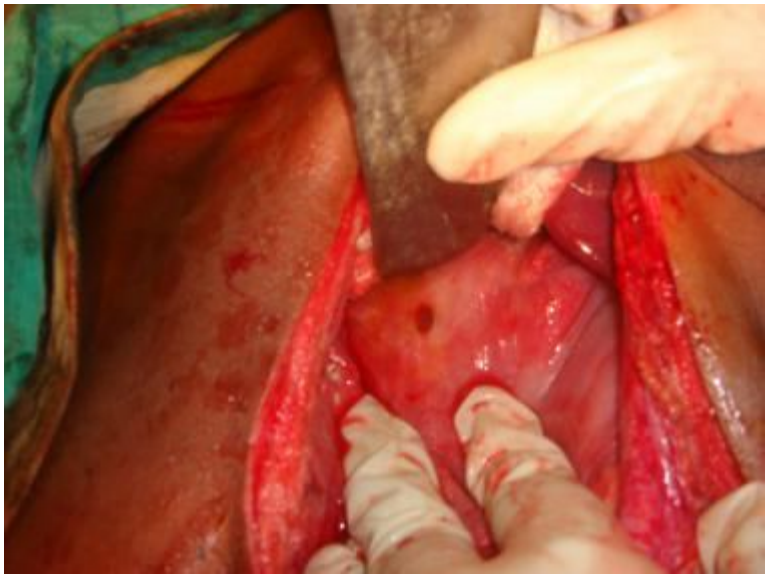
Under GA, patient put in supine position, parts painted & drapped Midline incision made, abdomen opened in layers,

peritoneum opened. Bile stained toxic fluid about 2 litres drained. About 1 * 0.5 cm perforation noted in the first part of the duodenum. Two biopsy specimen taken. The biopsy specimens taken were subjected one for RUT test another for histopathology to pathology laboratory.

Patient treated with NPO / RTA, IV FLUIDS – 2500 ML / inj. Ceftriaxone + sublbactum 1.5 gms / metrogyl 500 mg iv tds / amikacin 500mg / pantaprazole 40mg / tramadol 100 mg / TPR / BB- 2ndhrly / I / O chart / duolin nebulisation.

Day #5 pt shifted from ICU to Male Post operative ward & orals were started. Patient Drains were removed on day - 8, suture removal done on day -10th , patient was advised H.Pylori kit, and advised for follow up.

DUODENAL ulcer perforation



Xray abdomen showing air under diaphragm

RESULTS AND OBSERVATIONS

A prospective study for determining the incidence of H pylori in a perforated duodenal ulcer is carried out. The following observations are made:

The mean age of the patient is 44.6 yrs. All the patients are males. 83 % are smokers (25 of 30 patients).

5 (16.6%) were addicted to pan chewing and 3 (10%) were in the habit of gutkha chewing. 17 (56.6%) of 30 patients had past history of pain abdomen more in the epigastrium relived by taking antacids / H2 blockers. 16 (53.3%) of 30 patients had history of NSAIDS abuse. Two patients (6.6%) were on steroids.

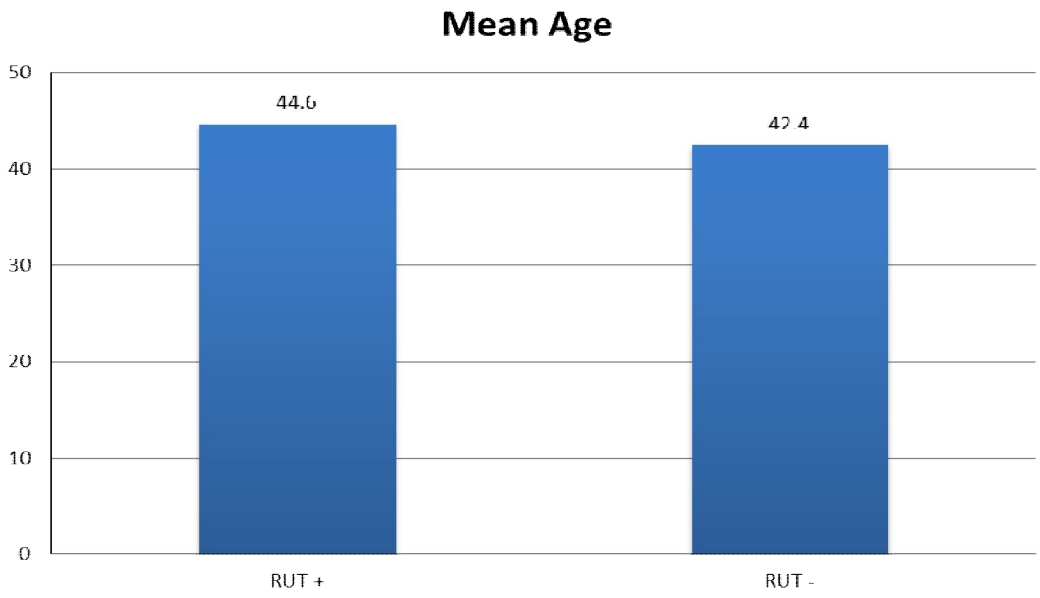
(53.3 %) 16 of 30 patients whose mucosal biopsy was subjected to rapid urease test (RUT) tested positive for urease. (10 strongly positive and 6 moderately positive)

14 (46.7%) of tested negative for RUT.

16 of the 30 patients (53.3 %) whose mucosal biopsy was specially investigated for H pylori by genta tested positive for the bacteria.

CHARTS

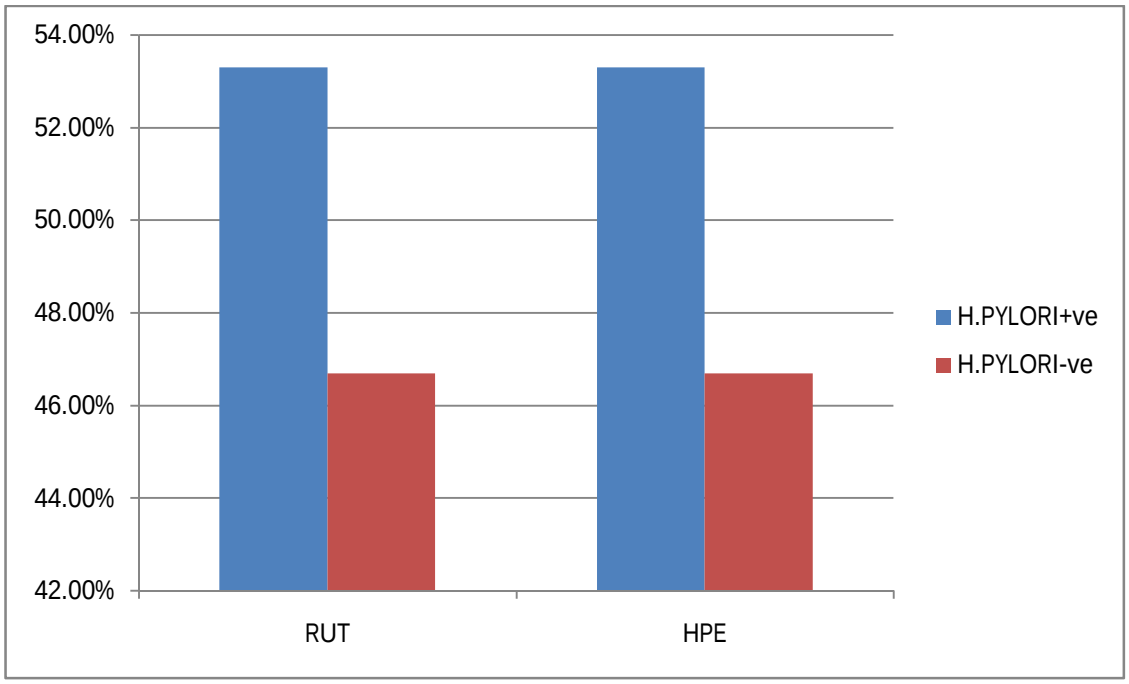
MEAN AGE	RUT +	RUT -
	44.6	42.4



Out of 30 patients in total:

	H. PYLORI +ve	H.PYLORI –ve
Rapid Urease Test	16	14
HPE	16	14

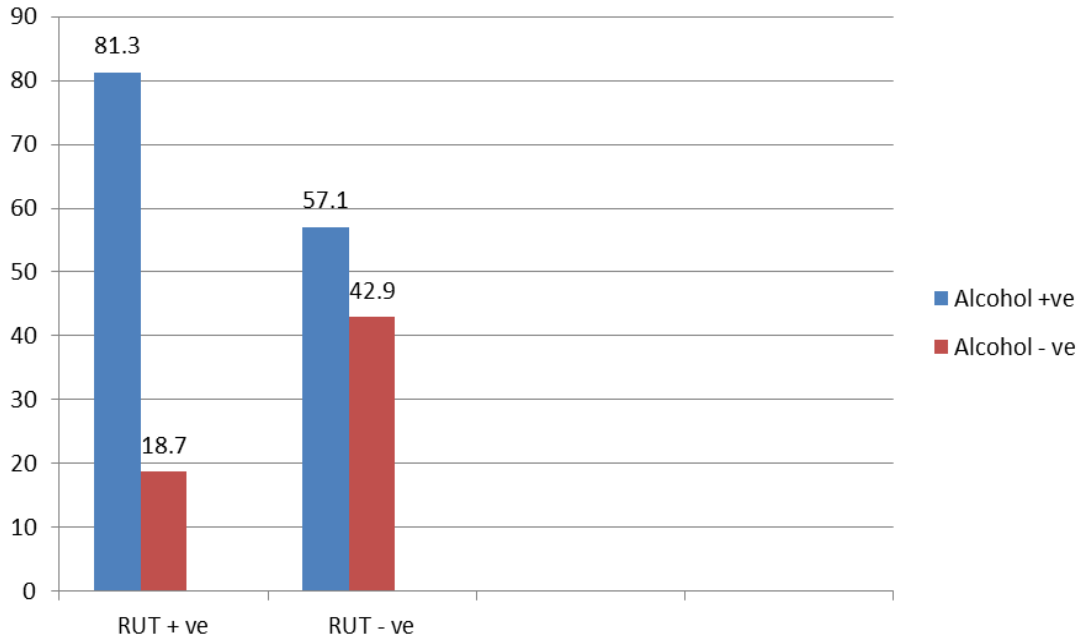
Chi square - 0.5 **df - 1** **P < 0.05**



Distribution in Alcoholics:

Alcohol	RUT +	RUT -
+ ve	81.3	57.1
-ve	18.7	42.9

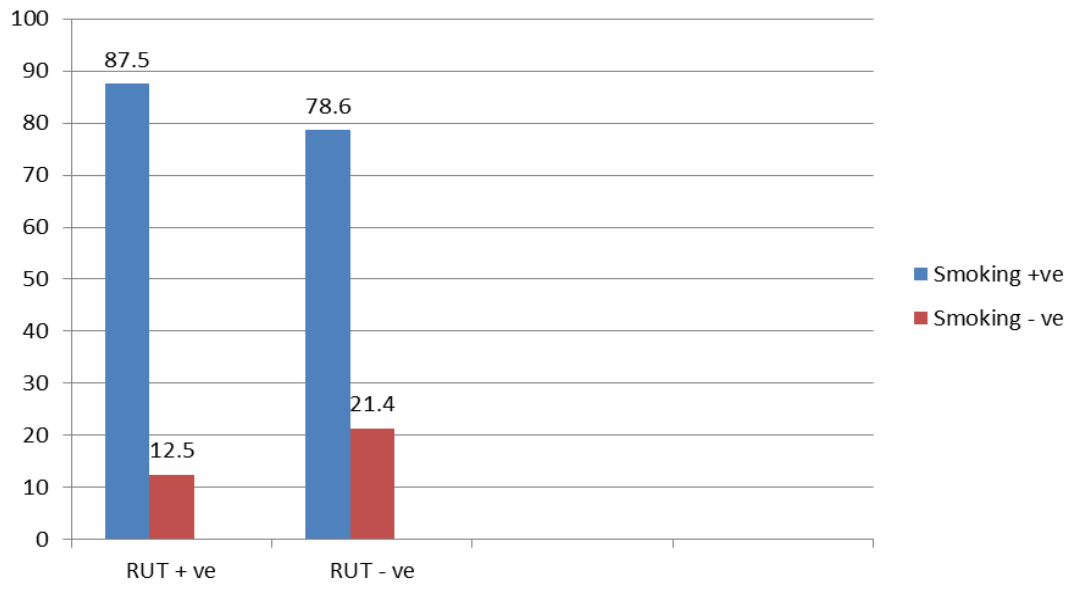
Chi square value	df	P
2.1	1	0.2(not significant)



Distribution in Smokers:

Smoking	RUT +	RUT -
+ ve	87.5	78.6
-ve	12.5	21.4

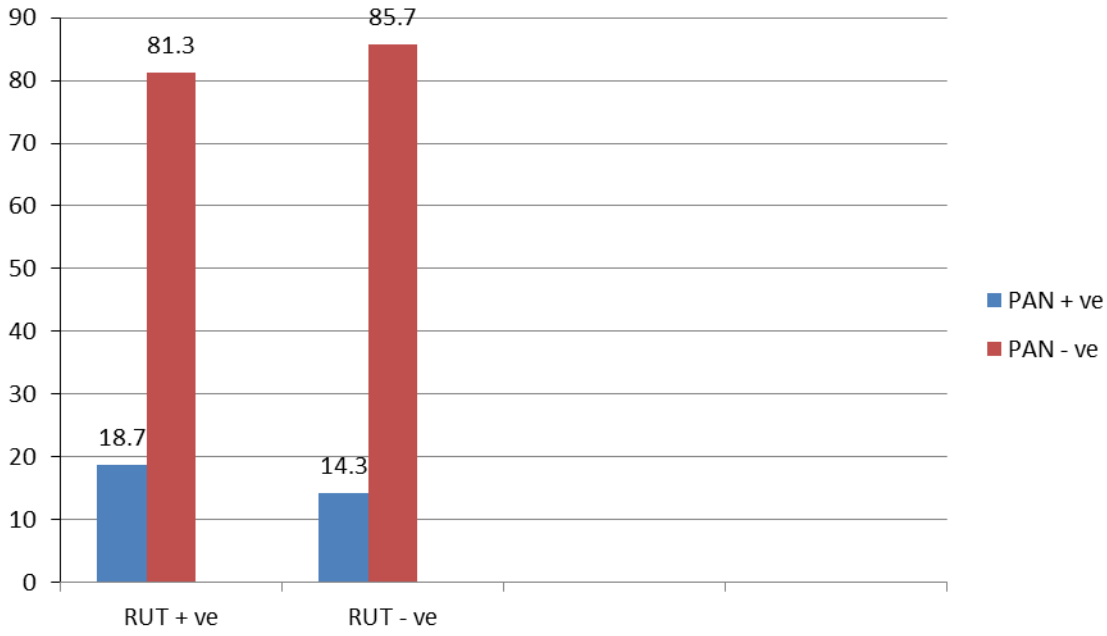
Chi square value	df	P
0.4	1	0.5(not significant)



Distribution among PAN chewers:

PAN	RUT +	RUT -
+ ve	18.7	14.3
-ve	81.3	85.7

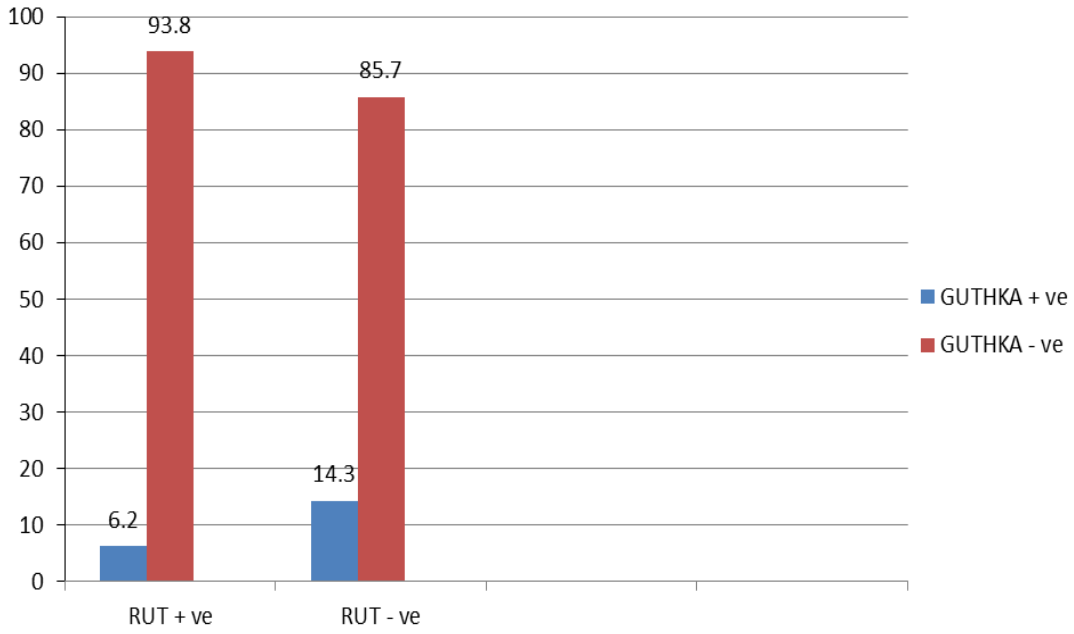
Chi square value	df	P
0.4	1	0.7(not significant)



Distribution in Ghutkha chewers:

Guthka	RUT +	RUT -
+ ve	6.2	14.3
-ve	93.8	85.7

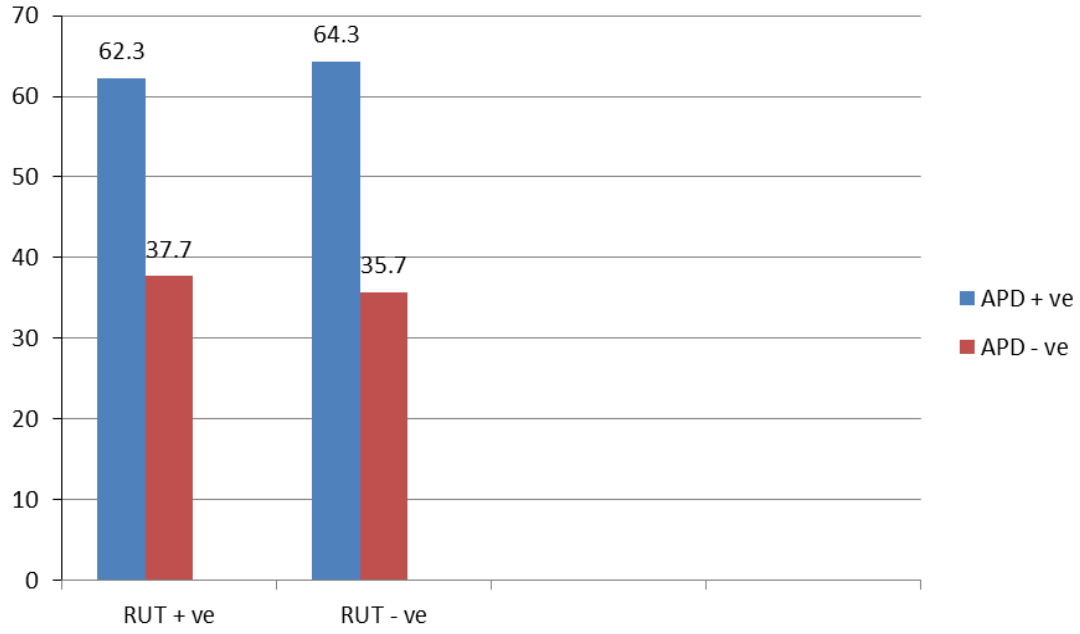
Chi square value	df	P
0.5	1	0.4



Distribution of APD:

APD	RUT +	RUT -
+ ve	62.3	64.3
-ve	37.7	35.7

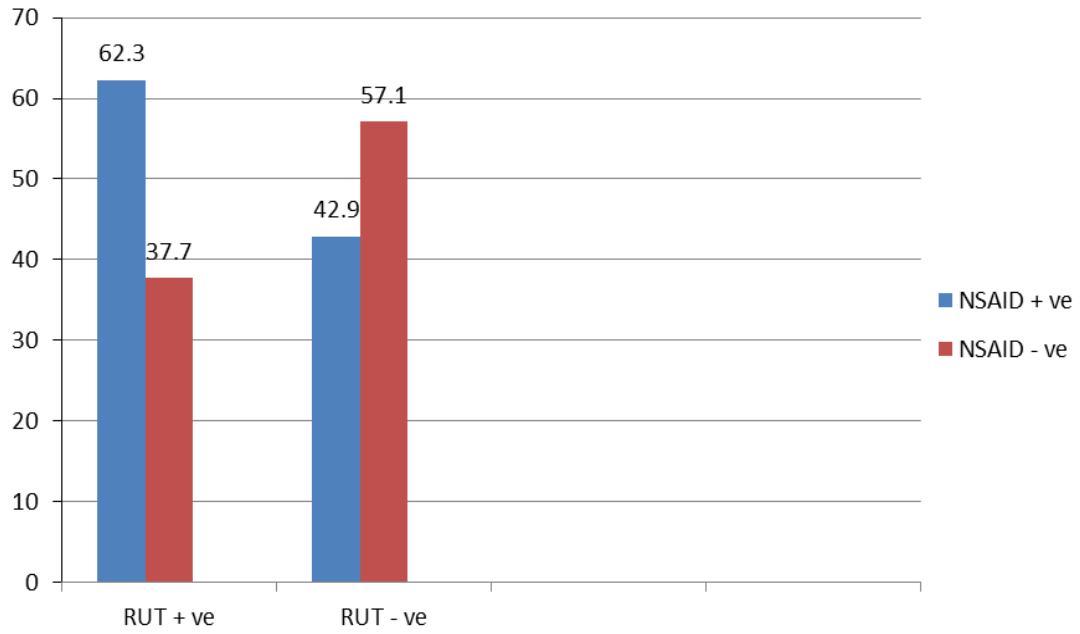
Chi square value	df	P
0.07	1	0.9(not significant)



Distribution of NSAIDs:

NSAID	RUT +	RUT -
+ ve	62.3	42.9
-ve	37.7	57.1

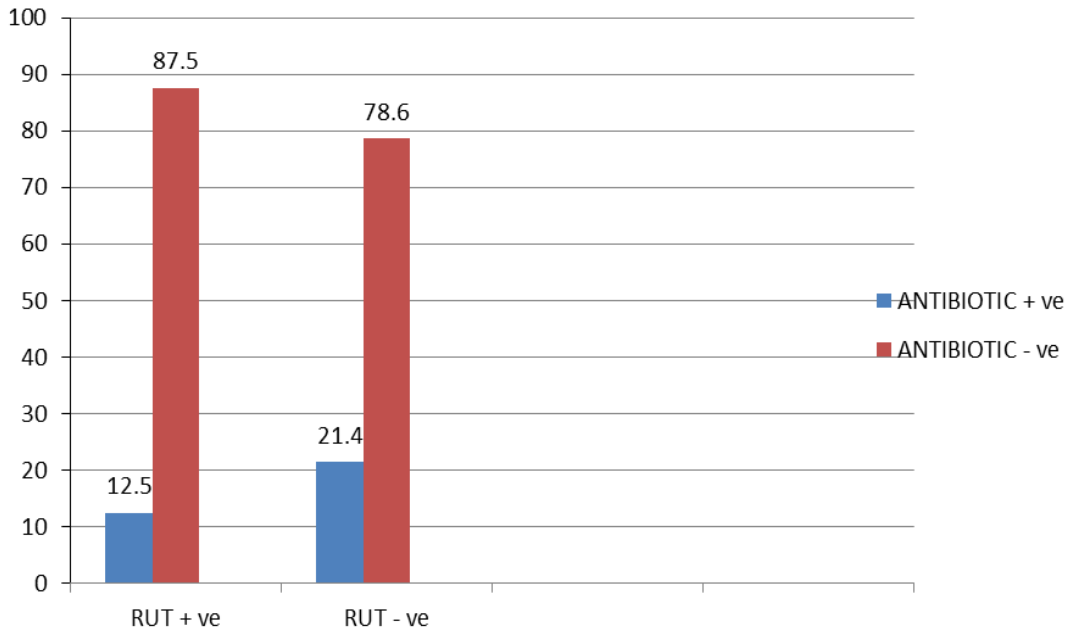
Chi square value	df	P
1.2	**1**	**0.3**



Distribution in Antibiotic users:

ANTIBIOTIC	RUT +	RUT -
+ ve	12.5	21.4
-ve	87.5	78.6

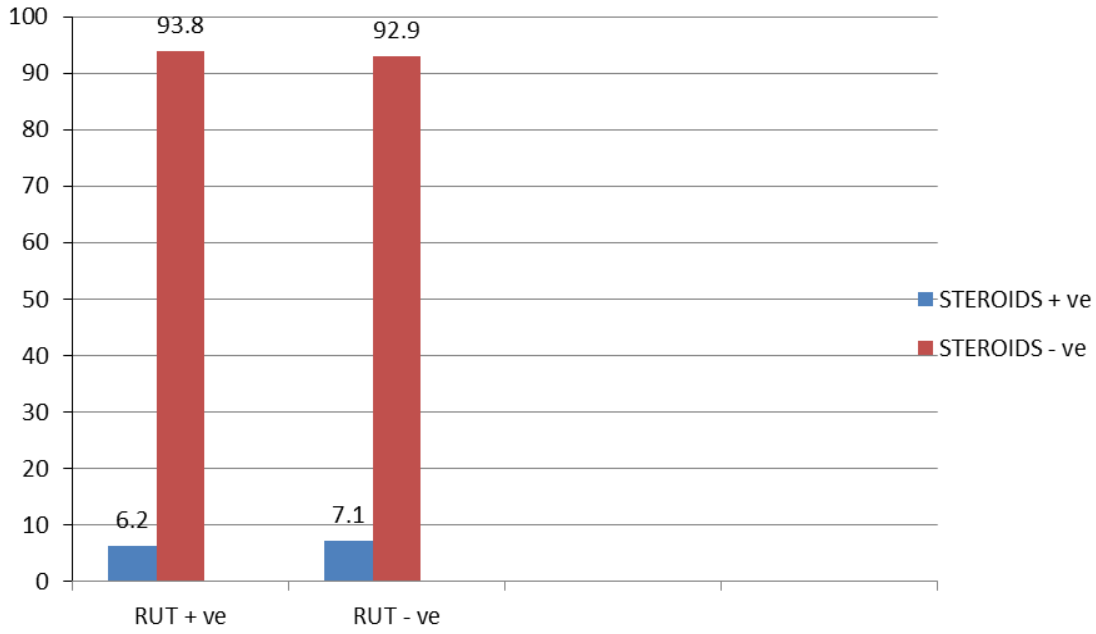
Chi square value	df	P
0.4	1	0.5



Distribution in Steroid Users:

STEROIDS	RUT +	RUT -
+ ve	6.2	7.1
-ve	93.8	92.9

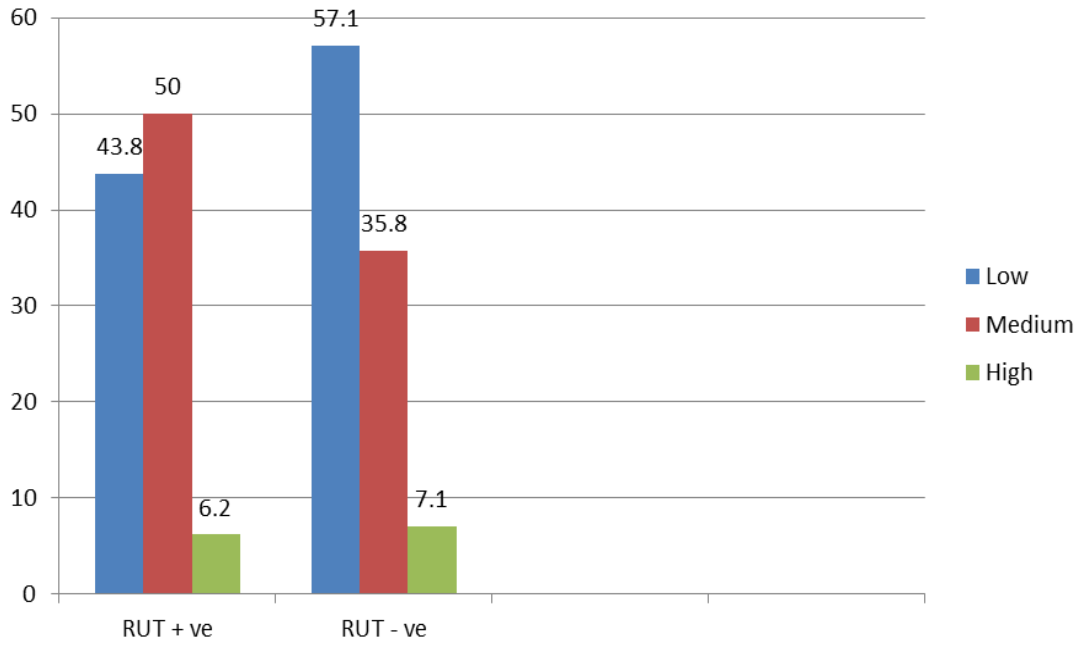
Chi square value	df	P
0.009	1	0.9

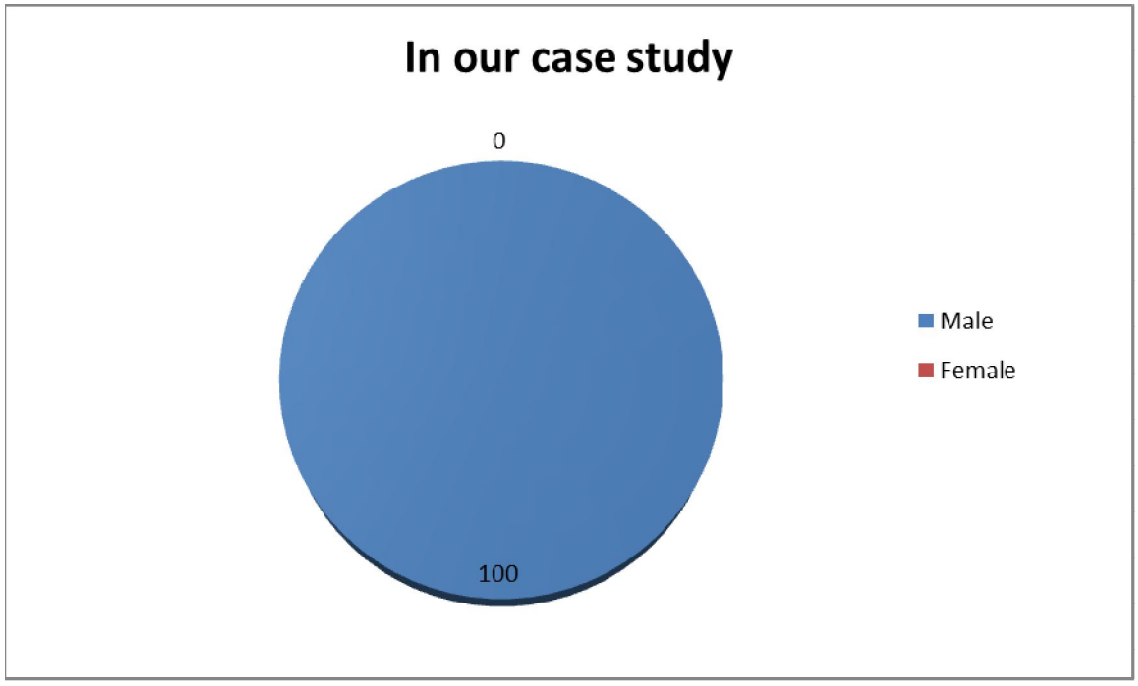


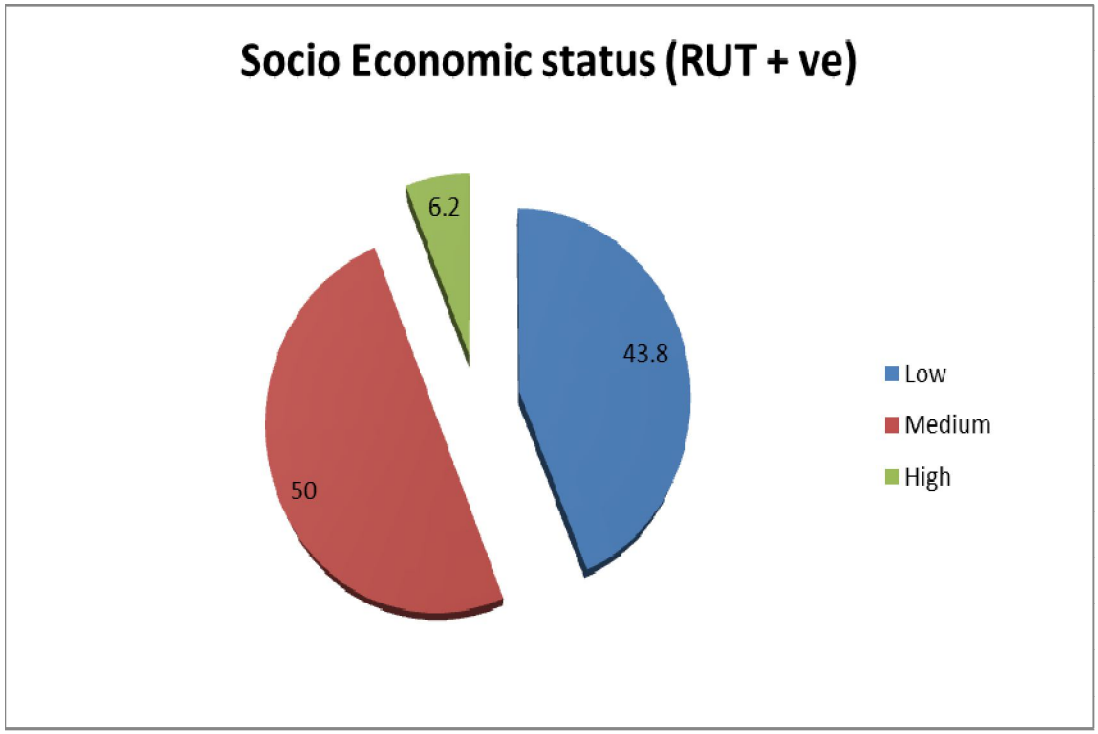
Distribution of Socioeconomic status:

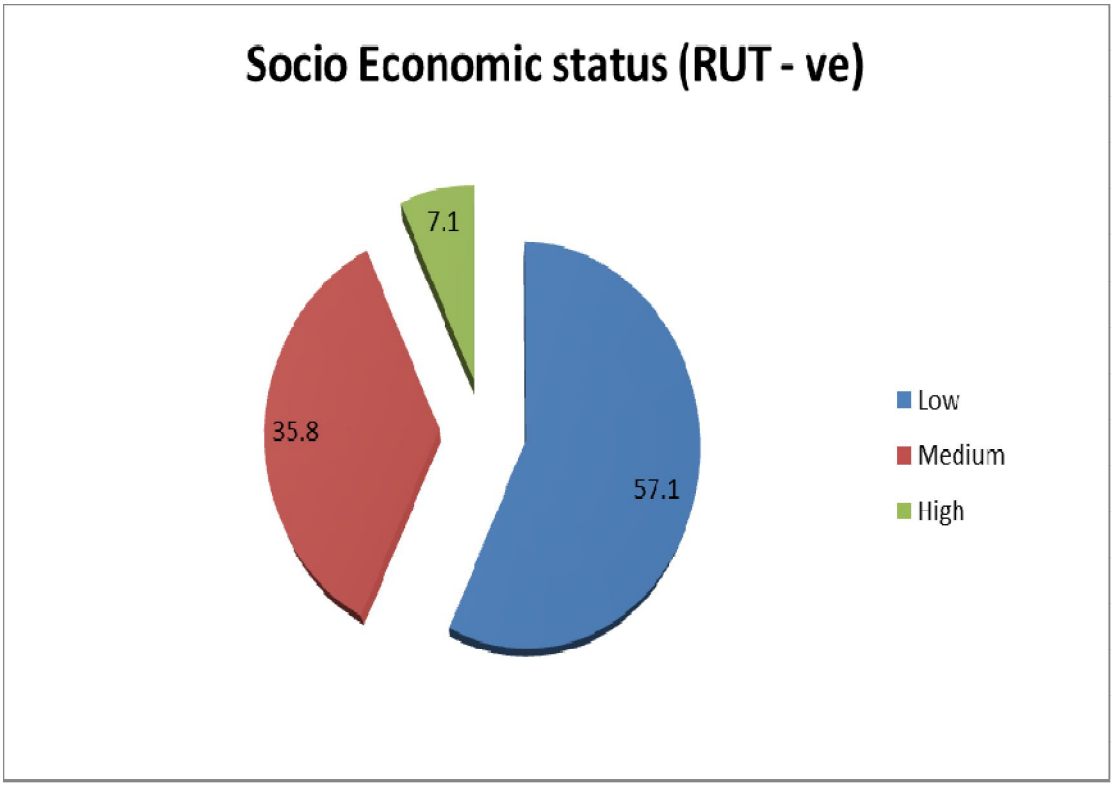
SOCIO ECONOMIC STATUS	RUT +	RUT -
L	43.8	57.1
M	50	35.8
H	6.2	7.1

Chi square value	df	P
0.5	1	0.4




In our case study
0
100
Male
Female


Socio Economic status (RUT + ve)
6.2
43.8
50
Low
Medium
High


Socio Economic status (RUT - ve)
7.1
35.8
57.1
Low
Medium
High

DISCUSSION

This is a prospective study of thirty cases of duodenal ulcer perforation admitted to Government vellore medical college & Hospital during the period August 2014 to July – 2015 to find out association of H pylori in the case of duodenal ulcer perforation.

The aim of the study was to evaluate the incidence of helicobacter pylori infection in perforated duodenal ulcer. The pylori infection was found to be significantly higher in the younger age group with male preponderance the present finding correlates with the findings of M.Sebastin, V.P.Permchand etal.

This prospective study examined a pathological etiological agent namely Helicobacter pylori in perforated duodenal ulcer. The high incidence of H. pylori infection suggests antibiotic therapy to eradicate the microorganism should be given to all patients with persisting duodenal ulcer.

This study indicates that patients with perforated ulcer were infected with H. pylori more severly. A close relationship was observed between the perforated ulcer and density of H pylori our study correlates with the findings of yukihkotokunaga etal.

Similar studies were conducted by

1.Minmamli N Isforaand et al, department of surgery and pathology, Sislietfal training hospital, Istanbul Turkey.

In the study conducted by dept of surgery, Sisli et al Hospital, Istanbul, Turkey, patients who underwent Surgery for a perforated duodenal ulcer in between January 94 to July 96 were studied. The study population consisted of 18 patients with a mean age of 32.7 years. (21 to 48 years). Ulcer was excised with the pylori ring. All the patients were treated by bilateral truncal vagotomy and Weinberg pyloroplasty. Two biopsies were taken from the antral mucosa by endoscopic biopsy forceps. The cut was then extended by about 2 cm on both the gastric and duodenal bulb. The defect was closed transversely.

The ulcer specimen and the antral biopsies were fixed separately in 10 % formalin solution and sent for histopathology. Specimens were stained with H – E stain and examined for H – pylori.

H – pylori was found in the antral biopsies of 16 patients (88.8 %) and in (7) of the ulcer specimens (38.8 %). H – pylori was

found in the mucosa and also extended through the wall of the ulcer. H pylori was present at a high ratio in the antral biopsies of patients with duodenal ulcer perforation.

In our series a combination of RUT positivity and presence of organism on Genta staining was taken as positive for H pylori.

53.3 % of the patients tested positive for RUT (10 of strongly positive, 6 moderately positive)

46.7 % (14 of 30 patients) tested negative for RUT.

The Istanbul study was based on the presence of H.pylori in biopsy specimens stained by H – E stain.

In our study all the cases are subjected to a preliminary RUT, whose sensitivity and specificity is of the order of 90 – 95 %. Later all the biopsy specimens were subjected to Genta staining, which is a much more better staining procedure than H – E staining, used in Istanbul study, and differs from H –E staining, in showing the mucosal morphology, but also stains bacteria which are in low density, in a small biopsy specimen, and in presence of abundant debris or mucus on the duodenal surface

CONCLUSION

Duodenal ulcer perforation was seen in the age group of 25 to 60 years of age (mean 44.6 years)

All the patients are males.

Eighty three percent (83%) are smokers.

Seventy threepercent (73.3%) are chronic alcoholics.

Sixteen (16%) percent are addicted to pan chewing.

Ten percent (10%) are addicted to gutkha chewing.

Sixty three percent (63.3%) had suffered with previous history of APD.

Fifty three percent(53.3%) had the history of NSAID abuse.

sixteen percent(16.6%) were under Antibiotic cover.

Socio enocomic status of the patients were considered with High, Middle, low.

High group - 6.2% RUT positive,

Middle group – 50% RUT positive

Low group - 43.8% RUT positive

Forty five percent of patients were in treatment for APD.

Most of the patients positive on rapid urease test positivity were also found positive for genta staining.

There being paucity of previous pertaining to the role of H.Pylori in perforated duodenal ulcers and the other studies like Istanbul study showing as association of H.Pylori in perforated duodenal ulcer.

However our study showed @ 53.3% association of H. Pylori with duodenal ulcer perforation and the extensive usage of Antibiotics and liberal utility of proton pump with or with out combination of H2 receptor blockers shows 53.3% positivity and 46.7% negativity with the incidence of H. pylori in both RUT and HPE.

The incidence of Helicobacter pylori in our case study is about 53.3%. Event though, the total number of cases are less, H. pylori correlates well with the cause for duodenal ulcer & it can be considered as commonest cause. Hence, Helicobacter pylori should be eradicated using anti- H. pylori regimen in all cases of perforated duodenal ulcer.

The number of cases studied and the various entities considered still requires further studies on these lines.

BIBILIOGRAPHY

1. Marshall BJ, Warren JR. Unidentified curved bacilli, Word J gastroenterol October 28,2006 Volume 12 Number 40 stomach of patients with gastritis and peptic ulceration. Lancet 1984; 1: 1311 – 1315.

2. Tovey FI, Hobsley M, Holton J. Helicobacter pylori virulence factors in duodenal ulceration: A primary cause or a secondary infection causing chronicity. World JGastroenterol 2006; 12: 6-9

3. Segal I, Ally R, Sitas F, Walker AR. Co-Screening for primary billary cirrhosis and celiac disease. Helicobacter pylori: the African enigma. Gut 1998 ; 43: 300 – 301.

4. Boulos PB, Botha A, hobsley M, Holten J, Osjowo AO, Tovey FI. Possible absence of Helicobacter pylori in the early stages of duodenal ulceration.QJM 2002: 95: 749-752.

5. Pest P, Zarate J, Varsky C, Man F, Schraier M. Helicobacter pylori in recently-diagnosed versus chronic duodenal ulcer. ActaGastroeenterolLatinoam 1996; 26: 273-276

6. Bytzer P, Teglhjaerg PS. Helicobacter pylori-negative duodenal ulcers:pervalaence, clinical characteristics, and prohnosis results from a randomized trila with 2 – years follow-up. Am Jgastroenterol 2001; 96: 1409 – 1416.

7. Gdalevich M, Cohen D, Ashkenzi I, Mimouni D, Shpilberg O, kart JD. Helicobacter pylori infection and subsequent peptic duodenal disease among young adults. Int JEpidemiol 2000; 29: 592 – 595.

8. Nomura A, Stemmermann GN, Chyou PH, Perez-Perez GI, Blaser MJ. Helicobacter pylori infection and the risk for duodenal and gastric ulceration. Ann intern Med 1994; 120: 977-81.

9. Schwarz K. Ueberpenetrierendemegen- und jejuna gashwure. Betirklinchir 1910; 67: 96-128.

10. Hobsley M, WhitfieldPF.The likelihood of a disease in realition to the magnitude of a risk factor. The example of duodenal ulcer. Theoretical Surgery 1987: 2: 6 – 9.

11. Holcombe C, Helicobacter pylori: the Africa enigma. Gut 1992; 33: 429 – 431

12. Holcombe C omatara BA, Eldridge J, Jones Dm. Helicobacter pylori, the most common bacterial infection in Africa. A random serological study random serological study.Am J gastroenterol 1992; 87: 28-30.

13. Holcombe C, Omatara BA, Padonu MKO, Bassi AP. The prevalence of symptoms of dyspepsia in north eastern Nigeria: a random community based study. Trop Geog Med 1991; 43 209 – 214.

14. Segal l, Ally R, Mitchell H. Helicobacter pylori – an African perspective. QJM 2001; 94: 561 – 565.

15. ToveyFl, Hobsley M, Segal l, Jayaraj AP. Duodenal ulcer in South Africa : home - produced versus milled maize. J GastroenterolHepatol 2005; 20: 1008 – 1011.

16. Tovey Fl, Hobsley M, Kaushik SP, Panday R, Kurian G, Singh K, Sood A, Jehangir E, Duodenal gastric metaplasia and Helicobacter pylori infection in high and low duodenal ulcer – prevalent areas in india. J Gastroenterol Hepatol 2005; 19: 497 –505.

17. Wong Bc, Ching CK, Lam SK, Li ZL, Chen BW, Li YN, Liu HJ, Liu JB, Wang BE, Yuan SZ, Xu CP, Hou XH, Zhang AT, Zheng AT, Zheng ZT, Differential north to south gastric cancer - duodenal ulcer gradient in China. China Ulcer study Group. J GastroenterolHepatol 1998; 13: 1050 – 1057.

18. Ching CK, Lam SK. Helicobacter pylori epidemiology in relation to peptic ulcer and gastric cancer in south and north China. J GastroenterolHepatol 1994; 9 Suppl 1: S4 – 7.

19. Jayaraj AP, ToveyFl, Clark CG. Possible dietary protective factors in relation to the distribution of duodenal ulcer in India and Bangladesh. Gut 1980; 21: 1068 – 1076.

20. Jayaraj AP, ToveyFl, Lewin MR, Clark CG. Duodenal ulcer prevalence: experimental evidence for the possible role of dietary lipids. J GastroenterolHepatol 2000; 15: 610 –616.

21. ToveyF.Peptic ulcer in India and Bangladesh. Gut 1979; 20: 329 – 347.

22.ToveyFl, Tunstall M. Deodenal ulcer in black populations in Africa South of the Sahara. Gut 1975; 16: 564 – 576

23.ToveyFl, Deodenal ulcer in China. J GastroenterolHepatol 1992; 7: 427 – 431.

24.Jayaraj AP, ToveyFl, Clark CG, Hobsley M. Dietary factors in relation to the distribution of duodenal ulcer in India as assessed by studies in rats. J GastroenterolHepatol 2001; 16: 501 – 505.

25.Jayaraj AP, ToveyFl, Lewin MR, Clark CG,Deodenal ulcer prevalence: experimental evidence for the possible role of dietary lipids. J GastroenterolHepatol 2000; 15: 610 –616.

26.Jayaraj AP, Rees KR, ToveyFl, White JS. A molecular basis of peptic ulceration due to diet. Br JExpPathol 1986; 67: 149 – 155.

27.Jayaraj AP, ToveyFl, Clark CG, Rees KR, White JS, Lewin MR. The ulcerogenic and protective action of rice and rice fractions in experimental peptic ulceration. ClinSci (Lond) 1987; 72: 463 – 466.

28.PaulJayaraj A, ToveyFl, Hobsely M. Duodenal ulcer prevalence: research into the nature of possible protective dietary lipids. Phytother Res 2003; 17: 391 – 398.

29.Jyotheeswaran S, Shah AN, Jin Ho, Potter GD, Ona FV, Chey WY. Prevalence Helicobacter pylori in peptic ulcer patients in greater Rochester, NY : is empirical triple therapy justify ed: Am J Gastroenterol 1998; 93: 574 –578.

30.Borody TJ, George LL, Brandl S, Andrews P, Ostapowicz N, Hyland L, Devine M. Helicobacter pylori - negative duodenal ulcer. Am JGastroenterol 1991; 86: 1154 – 1157.

31.Dwyer B, Sun NX, Kaldor J, Tee W, Lambert J, Luppino M, Flannery G. Antibody resopnce to Campylobacter pylori in ethnic group lacking peptic ulceration. Scand J Infect Dis 1988; 20 : 63 - 68.

32.Jones DM, Eldridge J, Fox AJ, Sethi P, Whorwell PJ. Antibody to the gastric campylobacter - like organism ("Campylobacter pyloridis") - clinical correlations and distribution in the normal population. J Med Microbiol 1986; 22 : 57 – 62.

33.Kang JY, Wee A, Math MV, Guan R, Tay HH, Yap I, Sutherland IH. Helicobacter pylori and gastritis in patients with peptic ulcer and non – ulcer dyspepsia: ethnic differences in Singapore. Gut 1990; 31: 850 – 853.

34.Kochhar R, Siddeshi ER, Ayyagiri A, Bhasin DK, Metha SH. Campylobacter pylori in dyspeptic patients: a report from North India. Trans RoySoc Trop Med Hygiene 1989; 83: 135.

35.Lahaie RG, Lahaie M, Boivin M, Gagnon M Lemoyne M Nguyen B, Plourde V, Poitras S, Sahai A. Changing prevalence of Helicobacter pylori infection in endoscopically demonstrated duodenal ulcer. GUT 2000; 47 Suppl 1: A77 - A78.

36.Sabiston 19th edition

37.Schwartz principle of surgery 10th edition

38.Bailey and love 26th edition

39.panikers textbook of microbiology 9th edition

40.Manual of clinical microbiology - Patrick 2nd edition

41.**Shackelford's Surgery** of the **Alimentary Tract** (Shackelfords Surgery of the **Alimentary Tract**) **eBook**: Charles J. Yeo, David W McFadden,

42.**Maingot's Abdominal Operations**, 12th Edition (Zinner, **Maingot's Abdominal Operations**):

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: Serum creatinine: BT: CT:

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:

Anesthesia: Position:

Exploratory laprotomy + 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 _____

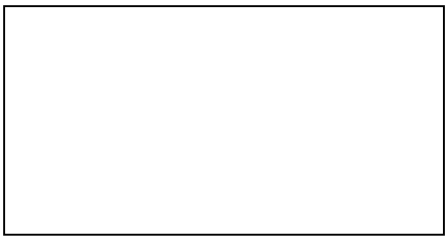
I have accurately read or witnessed the accurate reading of the consent from to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of participant _____

Signature of Participant _____

Date _____

Thumb print of participant



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

பங்கேற்பவர்களுக்கு ஆய்வின் விவரம்

செய்முறை விளக்கம் :

இந்த ஆய்வில் பங்கேற்பவர்களுக்கு அறுவை சிகிச்சை செய்து கொண்ட பின்பு, அந்த தசையை மருத்துவர் பரிசோதனை செய்து கொண்டு அதன் விவரம் தெரிந்து கொள்ள முழு மனதுடன் சம்மதிக்கிறேன்.

ஆராய்ச்சி நிலையம் :

பொது அறுவை சிகிச்சை துறை

அரசு வேலூர் மருத்துவ கல்லூரி மற்றும் மருத்துவமனை

வேலூர்

ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் .....

நான் .....

கட்டை விரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....

நாள் .....

ஆய்வாளரின் பெயர் .....

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

பங்கேற்பவரின் கையொப்பம் ..... இடம் .....

நாள் .....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....

நாள் .....

ஆய்வாளரின் பெயர் .....

KEY TO MASTER CHARTS:

PA	–	PAIN ABDOMEN
FVCO	–	FEVER VOMITING, CONSTIPATION / OLIGURIA
ALCO	–	ALCOHOL
SMO	–	SMOKING
PAN	–	PAN CHEWER
GUTK	–	GUTKA CHEWER
T	–	TEMPERATURE
PR	–	PULSE RATE
UOP	–	URINE OUTPUT
SES	–	SOCIO ECONOMIC STATUS
H / M / L	–	HIGH / MIDDLE / LOW
RR	–	RESPIRATORY RATE
RUT	–	RAPID UREASE TEST
HPE	–	HISTOPATHOLOGICAL EXAMINATION
APD	–	ACID PEPTIC DISORDER
NSAID	–	NONSTEROIDAL ANTI INFLAMMATORY DRUGS
ABIO	–	ANTIBIOTICS
STE	–	STEROIDS
PPI	–	PROTON PUMP INHIBITORS
H2	–	H2 RECEPTOR ANTAGONISTS
Hb %	–	HAEMOGLOBIN
RBS	–	RANDOM BLOOD SUGAR
BU	–	BLOOD UREA

SC	–	SERUM CREATININE
SAMY	–	SERUM AMYLASE
Na +	–	SODIUM
K +	–	POTASSIUM
CL	–	CHLORIDE
XREA	–	X – RAY ERECT ABDOMEN
USG	–	ULTRASONOGRAPHY
UGIE	–	UPPER GASTOINTESTINAL ENDOSCOPY
EL OPC	_	EMERGENCY LAPROTOMY & LIVE OMENTAL PATCH CLOSURE

S.No.	NAME	AGE / SEX	IP	COMPLAINTS		RISK FACTORS					VITALS			ASSOCIATED FACTORS							INVESTIGATIONS											PROC	POST OP
				Abd pain	fv	co	alc	smo	pan	gutka	T	PR	BP	RR	UO	APD	NSAID	Ab	Ster	PPI	H2b	Hb	RBS	BU	SC	SAM	Na	K	cl	XRA	SES	RUT	HPE		
1	DINESH KUMAR	25/M	3214	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	102	118/68	30	1200	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	(+VE)	13	112	26	0.8	70	140	4.5	102	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
2	SEKAR	40/M	3044	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	94	108/74	29	750	(+VE)	(-VE)	(+VE)	(-VE)	(+VE)	(+VE)	12.6	120	30	1	48	138	3.8	112	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
3	VINOD KUMAR	30/M	3912	(+ve)	fv	c	(-ve)	(+ve)	(-ve)	(-ve)	high	96	120/74	25	900	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	(-VE)	14	135	28	0.9	89	142	4.3	104	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
4	CHINNAPAIYAN	67/M	3721	(+ve)	fv	co	(+ve)	(-ve)	(-ve)	(-ve)	high	87	98/68	28	600	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	10.8	80	48	1.4	145	130	2.8	94	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
5	CHIDAMBARAM	42/M	3954	(+ve)	v	c	(+ve)	(+ve)	(-ve)	(+ve)	norm	112	104/64	20	450	(+VE)	(-VE)	(-VE)	(+VE)	(-VE)	(+VE)	11.5	96	40	1.1	125	138	3.9	112	(+VE)	L	(+VE)	(+VE)	EL OPC	UNEVENT
6	CHINNAKANNAN	60/M	4394	(+ve)	fv	c	(+ve)	(+ve)	(+ve)	(-ve)	high	104	110/74	22	1300	(-VE)	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	11	180	90	2.1	75	135	3	102	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
7	MANI	34/M	5664	(+ve)	fv	c	(-ve)	(+ve)	(-ve)	(-ve)	high	98	100/60	28	450	(+VE)	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	13.5	118	30	0.8	160	145	4.5	116	(+VE)	M	(-VE)	(-VE)	EL OPC	UNEVENT
8	KUMAR	44/M	6478	(+ve)	fv	co	(-ve)	(+ve)	(-ve)	(-ve)	high	122	90/?	45	NIL	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	(+VE)	12.5	75	60	1.9	49	125	2.9	96	(+VE)	L	(+VE)	(+VE)	EL OPC	UNEVENT
9	KRISHNAN	50/M	6770	(+ve)	v	c	(-ve)	(-ve)	(-ve)	(-ve)	norm	96	104/74	32	850	(+VE)	(+VE)	(-VE)	(-VE)	(+VE)	(-VE)	11.8	130	50	1.4	68	135	3.8	102	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
10	SRINIVASAN	38/,M	6827	(+ve)	fv	co	(+ve)	(+ve)	(+ve)	(+ve)	high	114	94/?	46	NIL	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	(-VE)	14	110	80	2.2	102	135	2.9	96	(+VE)	L	(+VE)	(+VE)	EL OPC	UNEVENT
11	KUMARESAN	35/M	6976	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	80	120/84	30	1500	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	(+VE)	10.2	98	36	1.1	-	140	3.9	114	(+VE)	M	(-VE)	(-VE)	EL OPC	UNEVENT
12	ELUMAZHAI	62/M	8782	(+ve)	v	(+ve)	(+ve)	(+ve)	(-ve)	(-ve)	norm	98	90/70	24	850	(+VE)	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	9.8	85	52	1.5	60	136	3	110	(+VE)	L	(+VE)	(+VE)	EL OPC	EXP
13	AKBAR SHARIF	27/M	9201	(+ve)	v	c	(-ve)	(+ve)	(-ve)	(-ve)	norm	94	98/70	28	350	(+VE)	(-VE)	(+VE)	(-VE)	(+VE)	(-VE)	14.2	128	28	0.9	170	142	4.3	108	(+VE)	H	(-VE)	(-VE)	EL OPC	UNEVENT
14	SELVAM	55/M	9500	(+ve)	fv	co	(+ve)	(-ve)	(-ve)	(-ve)	high	106	90/70	32	300	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	11.8	95	52	1.4	50	135	3.2	102	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
15	SIVALINGAM	40/M	9681	(+ve)	fv	nil	(+ve)	(+ve)	(-ve)	(-ve)	high	116	102/64	28	150	(-VE)	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	12	110	40	1.2	89	138	3.9	104	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
16	UMAPATHY	63/M	10342	(+ve)	fv	co	(+ve)	(+ve)	(+ve)	(-ve)	high	110	90/70	24	600	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	9.6	110	90	2	61	130	2.8	96	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
17	MAHESH	22/M	11460	(+ve)	fv	c	(-ve)	(+ve)	(-ve)	(-ve)	high	126	90/60	28	500	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	(+VE)	14.2	126	28	1	132	142	4.5	102	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
18	PALANI	32/M	13247	(+ve)	fv	c	(-ve)	(-ve)	(-ve)	(-ve)	high	106	100/60	22	800	(+VE)	(-VE)	(+VE)	(-VE)	(+VE)	(+VE)	13	130	30	0.7	118	145	3.9	112	(+VE)	L	(+VE)	(+VE)	EL OPC	UNEVENT
19	VENKATESAN	45/M	15734	(+ve)	v	c	(+ve)	(+ve)	(-ve)	(-ve)	norm	85	110/72	20	1000	(+VE)	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	11.8	180	32	0.9	-	150	4.5	108	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
20	PITCHANDI	43/M	17034	(+ve)	v	c	(+ve)	(+ve)	(+ve)	(-ve)	high	95	120/76	35	1250	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	12.5	108	50	1.3	98	136	4.1	112	(+VE)	L	(+VE)	(+VE)	EL OPC	EXP
21	SARAVANAN	24/M	19359	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	98	94/74	40	750	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	(-VE)	13.8	95	48	1.4	74	135	3.1	98	(+VE)	H	(-VE)	(-VE)	EL OPC	UNEVENT
22	GUNASEKARAN	47/M	19806	(+ve)	fv	nil	(+ve)	(+ve)	(-ve)	(-ve)	high	105	90/60	36	600	(+VE)	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	12	86	50	1.8	80	130	2.6	94	(+VE)	M	(+VE)	(+VE)	EL OPC	UNEVENT
23	DHANAJEYAN	40/M	26171	(+ve)	fv	co	(+ve)	(+ve)	(-ve)	(-ve)	high	90	108/78	30	1300	(+VE)	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	11.2	130	30	1	-	140	4.2	100	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
24	MUNUSAMY	47/M	27179	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	98	120/76	26	1100	(+VE)	(+VE)	(-VE)	(-VE)	(-VE)	(-VE)	13.2	125	20	0.5	152	138	4.5	116	(+VE)	M	(+VE)	(+VE)	EL OPO	UNEVENT
25	SUNDAR	28/M	27735	(+ve)	fv	c	(-ve)	(-ve)	(-ve)	(-ve)	high	104	100/60	36	500	(-VE)	(-VE)	(+VE)	(-VE)	(+VE)	(+VE)	14.8	112	48	1.4	139	128	3	98	(+VE)	L	(-VE)	(-VE)	EL OPC	UNEVENT
26	RAMARAJ	39/M	28441	(+ve)	v	c	(+ve)	(+ve)	(+ve)	(-ve)	norm	84	130/80	20	1200	(-VE)	(+VE)	(-VE)	(-VE)	(+VE)	(-VE)	11.5	118	28	0.9	79	138	4.2	108	(+VE)	H	(+VE)	(+VE)	EL OPC	UNEVENT
27	PARASURAMAN	53/M	31176	(+ve)	fv	co	(+ve)	(+ve)	(-ve)	(-ve)	high	102	104/68	32	800	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	12.8	130	35	0.7	90	145	4	112	(+VE)	M	(-VE)	(-VE)	EL OPC	UNEVENT
28	GOPAL	65/M	31644	(+ve)	fv	co	(+ve)	(+ve)	(-ve)	(-ve)	high	110	94/74	38	700	(-VE)	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	10.8	108	40	0.9	94	135	3.9	114	(+VE)	L	(-VE)	(-VE)	EL OPC	EXP
29	SUBRAMANI	46/M	32982	(+ve)	fv	c	(+ve)	(+ve)	(-ve)	(-ve)	high	98	110/74	30	600	(+VE)	(-VE)	(-VE)	(-VE)	(+VE)	(-VE)	14.2	140	50	1.4	88	130	2.8	94	(+VE)	M	(-VE)	(-VE)	EL OPC	UNEVENT
30	DHARMAN	65/M	3933	(+ve)	FV	CO	(+ve)	(+ve)	(-ve)	(-ve)	norm	88	110/70	35	800	(-VE)	(+VE)	(-VE)	(-VE)	(+VE)	(+VE)	11.2	150	48	1.3	70	130	3	94	(+VE)	L	(+VE)	(+VE)	EL OPC	UNEVENT