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A STUDY OF INCIDENCE OF HELICOBACTER PYLORI IN CASES OF DUODENAL ULCER PERFORATION IN PREGNANCY.

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ABSTRACT

Introduction: Most of the patients having chronic peptic ulcer disease are usually found to be infected with helicobacter pylori infection. They are more prone if pregnant. 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 in pregnancy and the incidence of H. pylori was found from the biopsy taken from ulcer edge, using Rapid urease test & HPE. **Results:** Among thirty patients in pregnancy, samples of 16patients (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 in pregnancy, 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 **Aim Of Study** To study the incidence of Helicobacter pylori in a case of duodenal ulcer perforation in pregnancy.

KEYWORDS :

INTRODUCTION

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.

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.

Clinical Manifestations:

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

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 thedegree 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.

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 blockersfollowed 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.

Helicobacter Pylori:
In 1982, Marshall & Warren from Australia cultivated a spiral organism similar to Campylobacter colonizing the stomach ofhuman 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.

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.

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

Diagnosis Of H Pylori In Pregnancy :
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.

MATERIALS AND METHODS
Thirty patients who underwent surgery for perforated duodenal ulcer on emergency basis in the department of Obs and Gynec in IPGMER and SSKM Hospital, Kolkatawere included in this study.

Inclusion criteria
1) Patients between 20-50 years of age in pregnancy.
2) Patients having perforated duodenal ulcers in pregnancy.

Exclusion criteria
1) Patients below 20 years and above 50 years of age.
2) Patients on NSAID's for more than one month duration.
3) Patients who have received Anti-Helicobacter pylori treatment.
4) Patientswith 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.pyloridetection 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 pinkcolor develops within 30mins the test is taken as strongly positive.
2. Ifapinkcolorisnotdeveloped within half an hour b u t 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.

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.

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 significicant.

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.

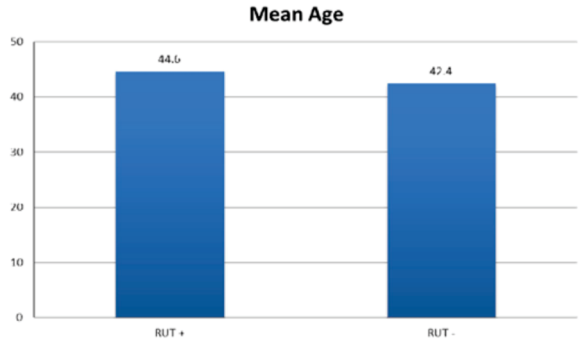
(53.3 %) 16 of 30 patients whose mucosal biopsy was subjected to rapid urease test (RUT) tested positive for urease. (10 strongly positive and 6 moderately positive)

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

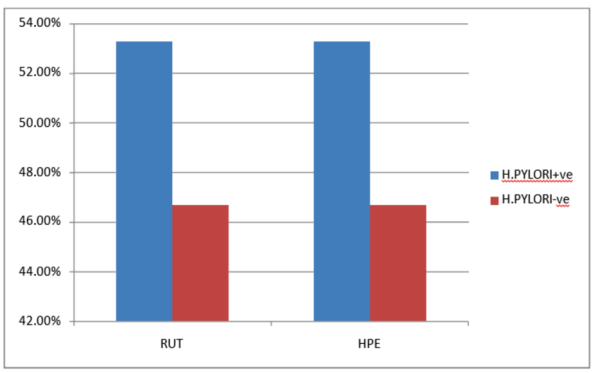
MEAN AGE	RUT +	RUT -
	44.6	42.4



Out of 30 patients in total:

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

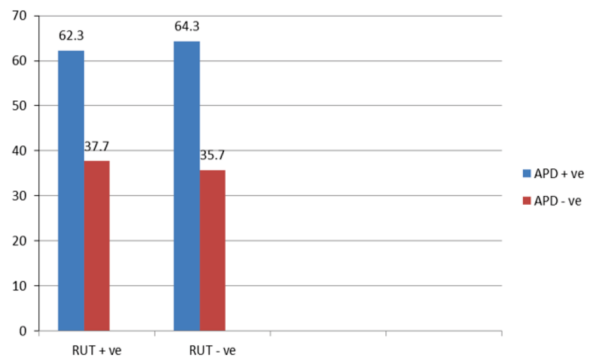
Chi square - 0.5 df - 1P < 0.05



Distribution of APD:

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

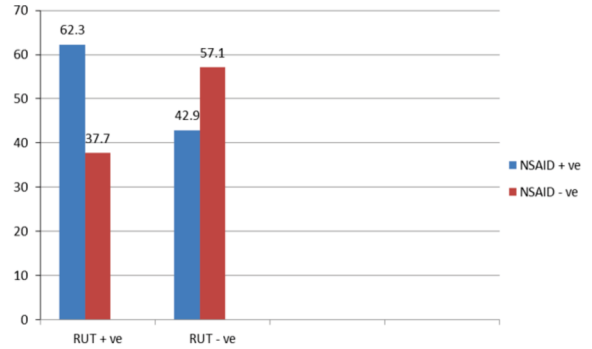
Chi square value	df	P
0.07	1	0.9(not significant)



Distribution of NSAIDs:

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

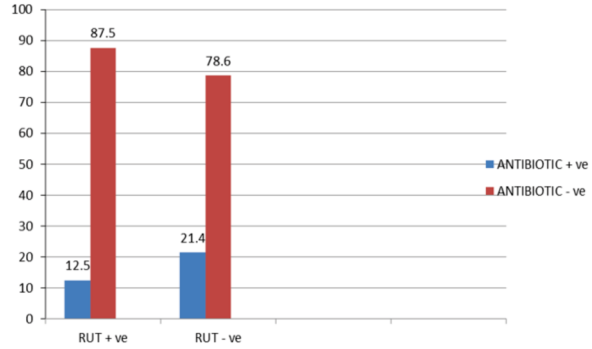
Chi square value	df	P
1.2	1	0.3



Distribution in Antibiotic users:

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

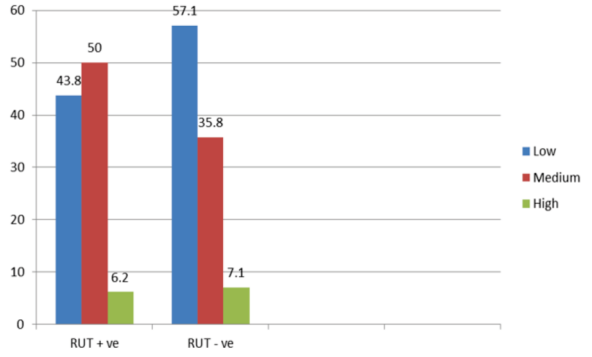
Chi square value	df	P
0.4	1	0.5



Distribution 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



DISCUSSION

This is a prospective study of thirty cases of duodenal ulcer perforation in pregnancy admitted to IPGMER and SSKM Hospital, Kolkata 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. 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) 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.

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