IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 19, Issue 1 Ser.12 (January. 2020), PP 19-21
www.iosrjournals.org

Prevalance of H.Pylori in Dudodenal Ulcer Perforation by Peroperative Mucosal Biopsy-A Prospective Study in CMCH (A Tertiary Care Hospital)

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Abstract: *Introduction : Duodenal ulcer perforation is a common surgical emergency in our institution. It is associated with significant morbidity and mortality as many patients present in a later stages with shock and hypovolemia which needs aggressive resuscitation and management .99% of dudodenal ulcers have H.Pylori infection. Hence control of H.Pylori may reduce the catastrophe and significant burden.*
Aims and objectives : The aim of this study is know the presence of H.pylori in patients with perforated duodenal ulcer by using Rapid Urease Test.
Materials and methods : The study included 30 patients who underwent Emergency laparotomy for perforation peritonitis under general anaesthesia. Per operative mucosal biopsy taken at perforation edge and the specimen is added into H.pylori detection kit for rapid urease test (RUT),which showed the urease production by the bacteria interpretted by colour change of the medium within a time .
Results : In our study, we found the presence of H.Pylori in 60% of cases of duodenal ulcer perforation . Incidence of H.Pylori was 53% by Rapid Urease Test.Triple Drug Regimen (H.Pylori Kit) used for treatment of patients was very effective in reduction in recurrence of symptoms
Key words: *H.Pylori, Rapid Urease Test, Duodenal perforation.*

Date of Submission: 07-01-2020 Date of Acceptance: 23-01-2020

I. Introduction

Duodenal ulcer perforation is among common surgical emergency. It is associated with significant morbidity and mortality as most of the patients present late with shock and hypovolemia which needs aggressive resuscitation and management. Hence duodenal ulcer perforation is causing economical burden on health services.

The demonstration of infective aetiology of peptic ulcer was made in 1990, the decade of H.Pylori for gastroenterologist. After the original description by Marshal and co workers in 1947 there has been a transformation zone in management of peptic ulcer disease.

There is necessity to study the association of H.Pylori infection in perforated duodenal ulcer. If proved control of H.Pylori may reduce this catastrophe and burden of society. We are going to study presence of H.pylori in all cases of perforated duodenal ulcer in our hospital

H.pylori is a gram negative flagellate, spiral bacilli that colonizes crypts of gastric mucosa. It weakens the protective mucosal coating of stomach and duodenum and allows the acid to get through this sensitive lining. Both acid and bacilli irritate the gastric lining and causes mucosal ulcer.

There are various methods to diagnose H.pylori infection like endoscopy, Urea breathe test, Stool and blood antigen test, blood antibody test and tissue test (Rapid Urease Test, Histopathological Examination and culture).

We selected Rapid urease test for detection of H.Pylori as it is simple, economical and not time consuming and highly specific and sensitive.

Aim

The aim of the study is to establish the prevalence of H.Pylori infection in duodenal ulcer perforation by per operative mucosal biopsy using rapid urease test.

Primary Objective
To study the cases of duodenal ulcer perforation in relation to H.pylori using rapid urease test.
Secondary Objective
To study the different etiological factors in cases of perforated duodenal ulcer.

II. Materials And Methods

About thirty patients of perforated peptic (duodenal) ulcer evaluated and analysed in an emergency basis in General surgery department in Coimbatore medical college and hospital for a period of between July 2018 to July 2019 are included in this study.

Inclusion Criteria:
1) Patients age group between 15 – 80 years
2) Patients with perforated duodenal ulcer
Exclusion Criteria:
1) Age less than 15 years and above the age of 80 years
2) All cases of prepyloric and Antral perforation

After proper clinical assessment the patients were actively resuscitated with intravenous fluids, nasogastric aspiration and antibiotics. The bladder was catheterized to monitor the urine output.

After stabilizing the general condition, the patients were taken up for surgery. Postoperatively nasogastric aspiration was continued, nutrition and electrolyte balance were maintained with intravenous fluids.

Per operative mucosal biopsy taken at perforation edge and the specimen is added into H.pylori detection kit for rapid urease test (RUT), which showed the urease production by the bacteria interpreted by colour change of the medium within a time. All positive cases were given triple drug regimen (H. Pylori kit) for a period of 14 days and negative cases were given omeprazole therapy for the same period.

III. Results

This prospective study was conducted among 30 high risk patients who underwent emergency laparotomy for duodenal ulcer perforation in the general surgery department CMCH, Coimbatore. The study was carried out to establish the prevalence of H.Pylori infection in duodenal ulcer perforation by per operative mucosal biopsy and to study the different etiological factors in cases of perforated duodenal ulcer and reduce the catastrophe and significant economical burden in the society

In our study we found H.Pylori in 60% cases (18) of perforated duodenal ulcer. 40% cases (12) of duodenal ulcer perforation were due to other etiological factors like NSAIDS, alcohol, smoking and spicy foods.

Maximum number of patients of duodenal ulcer perforation belonged to age group of 41-50 years with peaking at age of 50 years and decline. Incidence of duodenal ulcer perforation was more in male population. Incidence of H.pylori infection was 60%. Among various etiological factors, H.Pylori infection was found to have maximum association with duodenal ulcer perforation. Alcohol was found to be the second most common etiological factor. The most common presenting symptom was pain in epigastria. Triple drug regimen (H.Pylori kit) used for treatment of patients was very effective in reduction in recurrence of symptoms. Hence this study advocates early diagnosis and treatment of H.Pylori in all high risk patients. Early intervention with simple closure of duodenal ulcer with eradication of H.Pylori can reduce the morbidity and mortality.

IV. Discussion

H.pylori is a gram negative flagellate, spiral bacilli that colonizes crypts of gastric mucosa. It is more common in the lower socioeconomic group. H. pylori is associated mainly with peptic ulcer disease, type – B gastritis, gastric associated lymphoid tissue (MALT) B- cell lymphomas, gastric adeno carcinoma.

H. pylori releases UREASE the enzyme which hydrolyzes urea & releases ammonia which via negative feedback mechanism there by it increases gastrin release from G – cells. It also secretes enzyme DEHYDROGENASE (which converts alcohol to aldehyde a toxic metabolite to the mucosa), ENDOPEPTIDASE (which intervene the mucosal barrier). Urease the enzyme relased will create alkaline environment around it in mucus layer of gastric epithelium. It will survive only in gastric epithelium /gastric metaplasia in the duodenum / barett'sesophagus / and in heterotropic gastric mucosa in meckel's diverticulum / rectum since the receptors available only in gastric mucosa for these organism to get adhered.

Investigations:
Invasive Methods:

- Rapid urease test Culture
- PCR (polymerase chain reaction)

- Histology

Non Invasive Methods:

- Serology
- Urea breath test

Rapid Urease Test :

- It is a rapid diagnostic test which is used for H.pylori which is based on the enzyme urease
- biopsy taken from endoscopically is put into a solution having urea, with (phenol red) as a pH indicator and a gel which contains bacteriostatic agent
- Result -If H. pylori are present, urease will hydrolyses the urea and releases two molecules of ammonia and one molecule of carbon dioxide there by raising the pH and alkalinising the medium which is appreciated by colour change (yellow colour to red)
- Usually assessed after 30 minutes and after 2 hrs which further categorized as strongly positive, moderately positive and negative.
- Advantage - can be done in the endoscopy room immediately after taking biopsy.
- Sensitivity is 90% and the specificity is 98 %.

Treatment Of H.Pylori:

Triple Therapy

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

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

Quadraple Therapy : includes triple regimen plus bismuth subsalicylate.

V. Conclusion

Study concludes that H.Pylori infection was found to have maximum association with duodenal ulcer perforation. Triple drug regimen (H.Pylori kit) used for treatment of patients was very effective in reduction in recurrence of symptoms.

References:

[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]. 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.

Dr.R.Jayakumar Ms,et.al. "Prevalance of H.Pylori in Dudodenal Ulcer Perforation by Peroperative Mucosal Biopsy-A Prospective Study in CMCH (A Tertiary Care Hospital)". *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(1), 2020, pp. 19-21.