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Helicobacter Pylori Associated Gastritis: Case Series

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ABSTRACT

Helicobacter (H. Pylori) infection is a common habitual infection responsible for upper gastrointestinal symptoms such as indigestion, abdominal pain, spewing, heart burn, and abdominal wholeness along with nausea and vomiting [1]. H Pylori is a mortal pathogen. The primary complaint associated with this infection is habitual active gastritis. The inflammation can lead to peptic ulceration and atrophic gastritis in a considerable number of infected subjects. A minority eventually develops gastric cancer or tumour. The threat of similar complications depends upon the inflexibility of gastritis, which is determined by various host and bacteria related factors. Among bacterial factors, the common one is the cagA pathogenicity, the presence of which has been associated with more severe gastritis, peptic ulceration, atrophic gastritis and gastric cancer [2]. Among the host factors, most of the evidence focuses on acid production in response to H. Pylori infection. To confirm the pathogenic part of H. Pylori, both the existent's histological features of habitual gastritis and topographical patterns must be shown to be caused by the infection. Surface epithelial degeneration is a probable result of direct tissue injury by bacteria. Here are four intriguing cases of H. Pylori positive patients who had different admission presentations and were successfully treated with first line remedy and salvage remedy.

KEYWORDS: H. Pylori, PUD, gastritis, gastric cancer

INTRODUCTION

Helicobacter pylori (H. pylori) are a common gram negative bacterium that can colonise the human stomach and can cause chronic gastritis, Peptic Ulcer Disease (PUD), mucosa-associated lymphoid tissue lymphoma, and gastric cancer [3]. H. pylori are one of the common infections worldwide, with an estimated prevalence of 50% in developing countries and 10-20% in developed countries [4]. Neutrophil polymorph and chronic seditious cell infiltration are consequences of the mucosal immune response to bacterial antigens. Complement products and interleukin (IL)-8 are polymorph chemotaxins, and monocyte processing of antigens, followed by T helper cells and B lymphocyte responses, explain the presence of these cells in the mucosa. Atrophy may be a consequence of auto destructive products of neutrophil and monocyte activation, similar as reactive oxygen metabolites and proteases [5]. An unhealthy lifestyle and consumption of red meat and junk food are the common threats H. pylori infection. In addition, other threat

factors include poor hygiene, poor socio-economic status, poor sanitation, overcrowding, and consumption of defiled food and water, and bacterial infection within the menage that can get transferred between family members [6]. Here presents four cases of Rapid Urease Test (RUT) positive gastritis. These four cases are managed by first line PPI therapy and Salvage therapy.

CASE PRESENTATION

CASE 1

A 52 years old female, with a known case of Type II Diabetes Mellitus, Systemic hypertension, Hypothyroidism was presented with complaints of abdominal distension and discomfort for the past 2 days. She was diagnosed with COVID 19 four years back. She was taking T. GLIMEPIRIDE, T. LEVOTHYROXINE & T. TENELIGLIPTIN + METFORMIN as her own drugs. She was afebrile at the time of admission and was provisionally diagnosed as Gastritis. Admission examination showed: Palpitation rate 74 beats/min, Respiratory Rate 18/min, and Blood Pressure is 120/80 mmHg. On physical examination, she had mild wheeze. She was latterly diagnosed with H. Pylori gastritis via Upper GI Endoscopy with a positive value for Rapid Urease Test. Hence she was treated with IV Antibiotics (CEFOPERAZONE SULBACTAM 1.5gm BD), Proton pump inhibitors (PPIs – PANTOPRAZOLE 40mg BD), Pro-kinetic agents (METOCLOPRAMIDE 10mg OD), Anti-flatulence agents (SIMETHICONE 25mg HS). She became symptomatically better and was discharged after 2 days by giving H. Pylori kit (ESOMEPRAZOLE 40mg- AMOXICILLIN 750mg-CLARITHROMYCIN 500mg OD).

CASE 2

A 62 year old male patient came to the hospital with complaints of epigastric pain and left sided chest pain for the past 3 days. Moreover, he had a habit of eating junk foods from outside. Clinical examination revealed that he had distended abdomen and had heavy wheezing. He had a history of Type II Diabetes mellitus for the past 4 years and was taking T. METFORMIN 500mg. His blood routines were within normal range and USG of the abdomen and pelvis revealed internal echoes in gall bladder & mildly increased (L) renal parenchymal echoes. His sodium levels (124mmol/L) were depleted and he had similar incidence earlier. Depleted serum sodium levels were corrected by infusing 3% Normal Saline. Upper GI Endoscopic findings confirmed positive H. Pylori associated gastritis. He was treated with PPIs (T. PANTOPRAZOLE 40mg BD). H. Pylori kit (ESOMEPRAZOLE 40mg-AMOXICILLIN 750mg-CLARITHROMYCIN 500mg OD) was given in discharge. On follow up visit, his RUT turned negative.

CASE 3

A 23 year old female patient with the complaints of nausea and vomiting for 2 days was admitted to the hospital. She also reported the complaints of abdominal pain and heart burn. She had a past history of tension type headache and was taking T. NAPROXEN – DOMPERIDONE 500mg-10mg & FLUPENTHIXOL 0.5mg. She had asthma for the past 8 months, but was not on regular treatment. Her BP was found to be declined at the time of admission (100/70 mmHg). Moreover, she had been apprehensive and had tenderness on the epigastric region. All the parameters except Blood urea were found to be normal. Urea level was declined to 12 mg/dL. Gram staining study of CSF fluid was carried out and it was normal. Upper GI Endoscopy showed Prolapse gastropathy, Fundal diverticulum and H. Pylori Gastritis. No significant abnormalities detected on USG report. She was treated with PPIs (T. PANTOPRAZOLE 40mg), systemic anti-emetics (ONDANSETRON 4mg Q8H), GI Protectants (SYP SUCRALFATE 10ml TDS) and Antihistaminics (T. FLUNARIZINE 5mg HS). She became

stable and was discharged after 2 days by giving H. Pylori kit (ESOMEPRAZOLE 40mg-AMOXICILLIN 750mg-CLARITHROMYCIN 500mg OD). She was discharged after 12 days of hospitalization.

CASE 4

A 24 years old male, with a known case of liver cirrhosis presented with complaints of multiple episodes of vomiting and abdominal pain for 10 days. He also experienced nausea on the day of admission. His vomitus contains food particles, sometimes it was bilious but not blood stained. He had taken H. Pylori treatment on childhood. He was a non-alcoholic and non-smoker. He worked in a remote area and ate from local restaurants and consumed meat most of the time. Upper GI Endoscopy revealed Pangastritis, H. Pylori infection, GOO (Gastric Outlet obstruction). Magnetic Resonance Cholangio pancreatography (MRCP) and MRI of abdomen with Magnetic Resonance-generated synthetic Computed Tonography (MRCT) was taken it shows- short segment pyloric stenosis with fluid stasis in stomach and dilated main pancreatic duct respectively. Biopsy of Pylorus reveals chronic active gastritis with ulceration, and the presence of H. Pylori organisms. No malignancy detected from the biopsy sample. He was treated with oral PPIs (PANTOPRAZOLE 40mg BD), Prokinetic agent (T. METOCLOPRAMIDE 10mg OD), Multivitamins and H2 receptor blockers (T. RABEPRAZOLE 20mg OD) & T. SOMPRAZ 40mg BD & H. Pylori kit (ESOMEPRAZOLE 40mg-AMOXICILLIN 750mg-CLARITHROMYCIN 500mg OD) was prescribed on discharge. He was symptomatically relieved and was discharged after 6 days of hospitalization.

TABLE 1: LABORATORY VALUES OF FOUR CASES

BLOOD TESTS	CASE 1	CASE 2	CASE 3	CASE 4
HEMOGLOBIN	13.3	15.6	12.5	11.8
WBC	9540	7070	9430	5500
CRP	13	2.4	37	2.9
ESR	10	06	18	30
RUT	+	+	+	+
SODIUM	139	124	138	136
POTASSIUM	4.1	3.8	3.8	3.6
UREA	23	28	12	12
CREATININE	0.6	0.7	0.7	0.7
INR	1.03	1.01	1.08	1.33
IRON	65	89	48	20

TABLE 2: SCAN REPORTS AND ENDOSCOPIC FINDINGS

TESTS	CASE 1	CASE 2	CASE 3	CASE 4
Upper GI Endoscopy	RUT +	RUT +	RUT +	RUT +
USG Abdomen & Pelvis	No abnormality	Internal echoes on Gall bladder	No abnormality	No abnormality
Others	CECT Abdomen-Umbilical Hernia	NIL	NIL	MRCP-Pyloric Stenosis Biopsy of Pylorus-chronic active gastritis with ulcers

TABLE 3: SYMPTOM COMPARISON AMONG FOUR CASES

CASE NUMBERS	SYMPTOMS ON ADMISSSION
CASE 1	Abdominal distension & Abdominal discomfort * 2 days
CASE 2	Epigastric pain and (L) chest pain * 3 days
CASE 3	Nausea & Vomiting * 2 days, Abdominal pain, Heart burn(+)
CASE 4	Multiple episodes of vomiting. Vomitus contains food particles and sometimes it was bilious, Abdominal distension * 2 days and Abdominal pain * 10 days

TABLE 4: TREATMENT COMPARISON AMONG FOUR CASES

CASES	TREATMENT REGIMEN
CASE 1	PANTOPRAZOLE 40mg BD, METOCLOPRAMIDE 10mg OD, SIMETHICONE 25mg HS, AMOXICILLIN-ESOMEPRAZOLE-CLARITHROMYCIN (750mg-40mg-500mg) OD
CASE 2	PANTOPRAZOLE 40mg BD, AMOXICILLIN-ESOMEPRAZOLE-CLARITHROMYCIN (750mg-40mg-500mg) OD
CASE 3	PANTOPRAZOLE 40mg BD, ONDANSETRON 4mg Q8H, AMOXICILLIN-ESOMEPRAZOLE-CLARITHROMYCIN (750mg-40mg-500mg) OD
CASE 4	METOCLOPRAMIDE 10mg OD, PANTOPRAZOLE 40mg BD, RABEPRAZOLE 80mg OD, AMOXICILLIN-ESOMEPRAZOLE-CLARITHROMYCIN (750mg-40mg-500mg) OD

DISCUSSION

Helicobacter Pylori (H. Pylori) is a gram negative bacterium inhabiting the luminal surface of the gastric epithelium, first insulated in 1982, and is allowed to have infected humans for years ago. Ongoing H. Pylori infection has been linked to an increased threat of gastric cancer, though the data are clashing on whether the treatment of H. Pylori infection prevents gastric cancer [7]. There are two types of pyloric gland – suchlike metaplasia in the corpus of stomach: pyloric and pseudo pyloric metaplasis. They show the original morphology as the original pyloric glands in Hematoxylin & Eosin (H&E) staining. Pseudo pyloric metaplasia is positive for pepsinogen (PG) immunohistochemically (IHC), whereas pyloric metaplasia is negative [8]. All peoples infected with H. Pylori develop active habitual gastritis, but there is a relationship between the presence of H. Pylori gastritis and symptoms. Therefore, gastritis is a pathological rather than a clinical diagnosis [9].

The usual first line test for H. Pyloric discovery is the Rapid Urease Test, while others are faecal antigen testing, serology, or invasive tests grounded on endoscopic biopsy. Prompt Endoscopy is needed if the case has red flag symptoms similar as – weight loss, epigastric pain, anemia, melena or heart burn. Antibiotics should be halted for at least four weeks; PPIs should be withheld for at least one week before a Urea breath or faecal antigen test for H. Pylori to minimize the chance of false-negative results.

An important consideration for treatment is that primary CLARITHROMYCIN resistance is common in numerous groups of cases. The primary treatment can be with either standard triple therapy (CLARITHROMYCIN AMOXICILLIN & ESOMEPRAZOLE) or bismuth-containing quadruple therapy. Treatment for 10 to 14 days is more likely to annihilate the pathogen than treatment for 7 days [10]. When H. Pylori infection is originally diagnosed in a case over age 50, gastritis stratification should be performed by means of endoscopic biopsy and histologic examination.

Here four cases with variable symptoms and medical histories are bandied and are treated according to the standard therapeutic guideline.

CONCLUSION

H. Pylori infection remains a significant cause of morbidity worldwide. The presented four cases were having variable symptoms, but having nearly analogous life pattern. All of them were consuming more junk foods, consumed plenitude of meat, spicy and oily food particulars. They all led a sedentary life, did not engage in physical exercise. Two cases reported belong to the age group of early 20s and the other two were at their 50s. This shows that irrespective of the age, everyone can get H. Pylori infection in common. Early

intervention with triple therapy along with salutary changes is the cornerstone of the treatment of this condition. Thus we conclude that, as per the standard treatment guidelines, all the four cases were treated and discharged after their characteristic relief.

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