RELATION BETWEEN CAMPYLOBACTER & LOWER END ESOPHAGITIS

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in General Medicine

by

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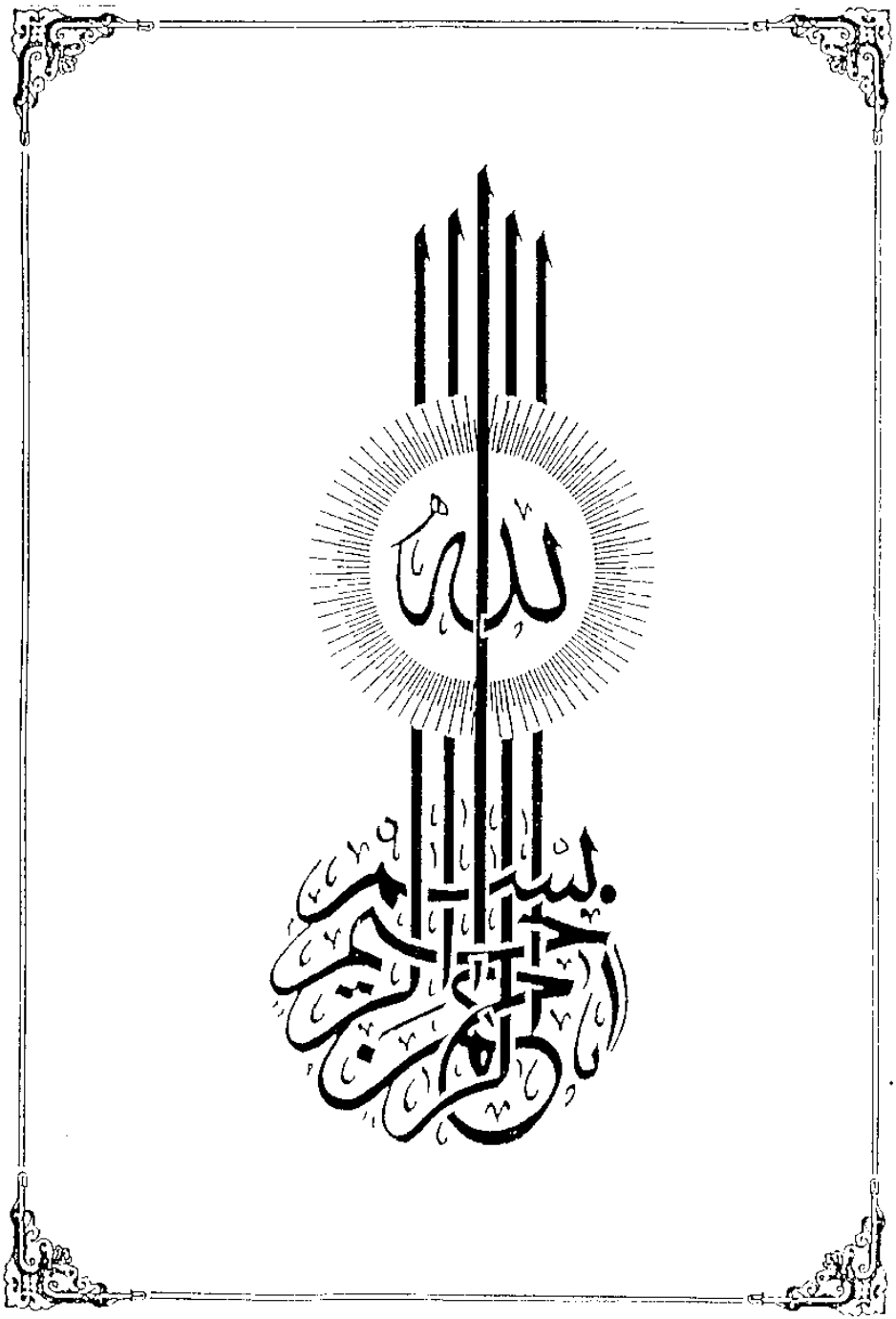

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Introduction
&
Aim of work

INTRODUCTION

Since the beginning of this century histopathologists have been describing the presence of spiral or curved bacteria in the stomach in a variety of animals including man. Hypotheses for their role have included the initiation of gastric carcinoma and non-neoplastic ulceration (Hart et al.,1989).

In (1983) Warren and Marshall described the presence of curved bacillus on gastric epithelium taken from the patients with active chronic gastritis and gave more information about the nature of the organism, and its morphology, and named it as Campylobacter pylori recently called Helicobacter pylori.

Since then, evidence has continued to be gathered linking this bacterium, Helicobacter pylori, with gastroduodenal diseases (Hart et al.,1989).

Infection of esophagus may be bacterial, fungal or viral.

Several questions were asked about Coexisting relation between Helicobacter pylori and esophagitis, and if Helicobacter pylori has a role in esophageal infection or not.

AIM OF THE WORK

To study the presence of Camplyobacter pylori recently called Helicobacter pylori in esophagitis either lower end esophagitis or refulx esophagitis.

Review of literature

GASTRO-ESOPHAGEAL REFLUX DISEASE

Definition:

Gastro-esophageal reflux disease (GERD) refers to the varied clinical manifestation of reflux of stomach and duodenum contents into esophagus. It is preferable to the term "reflux esophagitis" because the latter expression tends to mean different things to the clinician, the endoscopist and pathologist. Although it may be associated with a sliding hiatus hernia, symptomatic hiatus hernia is the term that tends to put the emphasis on wrong anatomic entity and pathophysiology (Pope,1988).

GERD can be characterized by any combination of symptoms, and radiologic, endoscopic or pathologic changes in its milder manifestation. It is a common disease whereas its most florid state is uncommon but may be life threatening (Pope,1988).

Pathophysiology:

The modern concept of esophageal inflammation due to acid and pepsin was first described by Winkelstein,(1935).

Hiatal hernia was initially thought to be essential for development of reflux esophagitis (Allison,1951), but it become clear that it was possible to have esophageal injury without hiatal hernia and that hiatal hernia could be present with no obvious abnormality of the esophageal mucosa (Kramer,1969).

Richter and Castell,(1982) proposed the concept of balance between defense mechanisms and aggressive factors in pathogenesis of GERD.

Aggressive factors:

* Acid and pepsin.

* Bile acids.

* Pancreatic enzymes and prostaglandins in some instances.

* Defence mechanisms:

1- Competent anti-reflux barrier primarily determined by lower esophageal sphincter pressure (LESP).

2- Intra-abdominal segment of esophagus.

3- Esophageal mucosal resistance.

4- Esophageal peristalsis and esophageal clearing.

5-- Acid clearance stimulated by saliva.

6- Gastric emptying and reflux.

7- Angle of His (acute angle between the esophagus entering the cardia and adjacent gastric fundus) and mucosal folds.

Aggressive factors in GERD

Clearly the type, volume and frequency of reflux are important.

Pepsin is required to produce experimental esophagitis but acid alone can injure the epithelium by protein denaturation (Goldberg et al.,1969).

Although hypersecretion of acid has been associated with esophagitis, most patients with reflux do not have gastric acid hypersecretion. Enterogastric reflux occurs in some patients. The permeability of the esophageal epithelium to hydrogen ions is increased by bile acids and activated pancreatic enzymes may contribute to tissue injury. Enterogastric reflux may be partially important in the elderly who have hypersecretion of acid (Kaye and Showalter,1974).

Because indomethazin protects against radiation induced esophagitis, prostaglandins have been incriminated as aggressive agents (Northway et al.,1980).

Castell,(1978) found that some prostaglandins e.g. PGE1, PGE2 and PGA2 increase GERD through decrease LESP.

Defensive mechanisms in GERD

1) Lower esophageal sphincter (LES):

The lower esophageal sphincter is not a distinct muscle but rather an area of increased intraluminal pressure about 3 cm long. The pressure in this area normally ranges from 12-30 mmHg (Behar et al.,1967). It appears that increased pressure is an inherent property of the circular smooth muscle in this region and is not dependent upon innervation or the effect of circulating hormones (Pope,1976). The lower esophageal sphincter normally

relaxes with stimulation which appears to be mediated by the non cholinergic, non adrenergic inhibitory nerve fibers (Pope,1976). Its tone can be modulated by the gastrointestinal mucosal peptides, by a variety of medications and perhaps by local vagal reflexes that increase tone (Pope,1976) Table (A).

Table (A): the effect of medication and hormones on lower esophageal sphincter pressure (Nelson,1984).

Raised LESP*	Reduced LESP
- Gastric alkalinization	- Gastric acidification.
- Protein meal.	- Fatty meal, peppermint, spearmint, chocolate, and caffeine.
- Cholinergic agonist.	- Cholinergic antagonist.
- Prostaglandin F2	- Prostaglandin E1,E2,A2.
- α-adrenergic agonist.	- α-adrenergic antagonist β-adrenergic agonist.
- Gastrin.	- Secretin.
- Motilin.	- Cholecystokinin.
- Substance P.	- Gastric inhibitory polypeptide.
- Pancreatic polypeptide.	- Vasoactive intestinal peptide
- Histamine	- glucagon.
- Indomethacin.	- Progesterone.
- Metaclorpromide.	- Theophylline.
	- Smoking.
	- Ethanol.
	- Calcium channel blockers.
	- Diazepam.
	- Meperidine, morphine.

* LESP: Lower esophageal sphincter pressure.

It was found that LES tone is an important component of the anti-reflux mechanism (Winans and Haris,1967; Dodds et al.,1982).

Indeed a minimal resting LESP in the range of 5-10 mmHg generally prevents reflux, even during transient increase in the intra-abdominal pressure, generally LESP must approach zero as an essential precondition for GER, although transient change in intra-abdominal pressure may intermittently overcome a hypotensive sphincter (Dodds et al.,1982).

Studies by Baldi et al.,(1985) and supported the general concept that reflux occurs when sphincter is relaxed mainly during brief periods of lower esophageal sphincter relaxation rather than because of continuous weakness of lower esophageal sphincter contraction. These workers concluded that a majority of these recorded sphincter relaxations were produced by swallowing, technical limitations of their methods probably do not justify this conclusion.

Studies on mechanisms of reflux by Mittale et al., (1987a) on healthy subjects and by Dent et al., (1988) on large group of patients with reflux disease, show the major importance of swallow-independent transient lower esophageal sphincter relaxation. The observation of Holloway et al., (1985) and Dent et al., (1988) suggested that transient lower esophageal sphincter relaxation is stimulated by gastric distension and that it is an inhibitory neural response which also affects the esophageal

body. In dog model of transient lower esophageal sphincter relaxation, Patrikios et al.,(1986) and Martin et al.,(1986) have shown that transient lower esophageal sphincter relaxation triggered by gastric distension depends entirely on vagal pathway. The better definition of these relaxations holds promise of better targeted pharmacological and surgical treatment of pathological reflux.

2- Intra-abdominal segment of esophagus:

The importance of abdominal segment of esophagus in controlling reflux is evident in many studies. Studies of the abdominal and thoracic portions of the HPZ (high pressure zone) in patients during respiratory maneuvers employed in Standard Acid Reflux Test (SART) demonstrated the importance of abdominal segment (Pelleergrini et al.,1976).

A correlation between the abdominal length of high pressure zone (HPZ) and control of reflux is observed, it is of the same statistical significance as the correlation between HPZ pressure and reflux and is variable from the amplitude of HPZ (O'Sullivan et al.,1982).

Normally inspiration causes increase pressure within the abdomen while decrease pressure within the thorax. The point at which respiratory excursion causes a pressure reversal in esophagus is called respiratory pressure inversion point (RIP). RIP is normally located within 3-4 cm segment of distal esophagus of high pressure zone (HPZ). Normally two or more centimeter of HPZ lies below RIP (Skinner,1985).