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# **RESEARCH ARTICLE**

# **GASTROESOPHAGEAL REFLUX DISEASE: A MINI REVIEW**

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| ARTICLE INFO   | ABSTRACT   |
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| Article History:   | Gastroesophageal reflux disease is a disease experienced by adults at least once a week. The history of the patient and their response of empirical trial with the proton pump inhibitors, $H_2$ receptor antagonist and other antacids which are used to treat the Gastroesophageal Reflux Disease. Diagnosis of the disease can be done based on the severity of Gastroesophageal Reflux disease in primay care. The limited diagnostic information of the endoscopy provides that the majority of the patients will not have visible lesion on Gastroesophageal reflux disease. This Gastroesophageal reflux disease is caused by the passage of the gastric contents from the stomach into the esophagus and causes the symptoms and damages the mucosa. Obesity and smoking are the other genetic factors with which GERD occurs which might be important. The negative association of helicobacter pylori exists, but the eradication does not cause reflux disease. Gastroesophageal Reflux disease is a tipically chronic disease which is associated with the development of complications and increasing severity and significance of the disease. |
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### **INTRODUCTION**

Gastroesophageal reflux disease (GERD) is a chronic digestive disease. It occurs when stomach content i.e., stomach acid flows back into the food pipe (esophagus). The back wash (reflux) irritates the lining of the esophagus and causes Gastroesophageal Reflux Disease [1]. It is usually caused by changes between the barriers of the stomach and oesophagus, including abnormal relaxation of the lower esophageal sphincter which normally caused by holding the top of the stomach closed, the impaired expulsion of gastric reflux from the esophagus, or a hiatal hernia. These changes may be permanent [2] or temporary.



Treatment is typically via lifestyle changes and medication such as H2 receptor blockers or antacids and proton pump inhibitors, with or without alginic acid [3]. Surgery may be an option in those who do not improve.

#### Types of Gastroesophageal reflux disease

#### Non erosive

Non erosive reflux oesophagitis is a most common cause of GERD, in this non erosive oesophagitis the symptoms may present but when endoscopy was done it shows that no tissue damage may occur into the oesophagus. In this the patients with non erosive oesophagitis [3] do not respond to traditional treatment, researches believed that nonacid relux cause the non erosive reflux oesophagitis

#### Erosive

Erosive oesophagitis is considered as the gastroesophageal reflux disease which causes the erosions in the lining of the esophagus with increasing [4, 5] in acid production in the stomach. They present with breaks or tears on the lining of the oesophagus. The treatment for GERD are  $H_2$  receptor antagonist[6,7] which are used to inhibit the  $H^+/K^+ATP$ ase present at the secretory surface of the gastric cells and other PPIs which inhibits gastric acid secretion.

#### Barrett's Esophagus

Barrett's oesophagus is described as a condition of the oesophagus which appears with the lining if the intestine. The exact cause is of Barrett's oesophagus is unknown. This

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barrett's oesophagus occurs [3] three to five times in GERD patients. It alone does not cause any symptoms, but the patients with barrett's oesophagus have increased risk of developing oesophagus cancer. Therefore to avoid this doctors recommend the treating for GERD symptoms and stomach acid lowering.

### Epidemiology

The range of GERD was 18.1%-27.8% in North America, 8.8%-25.9% in Europe, 2.5%-7.8% in East Asia, 8.7%-33.1% in the Middle East, 11.6% in Australia and 23.0% in south<sup>12</sup> America. Incidence per 1000 person-year was approximately five in the overall UK and US populations, and 0.84 in pediatric patients aged 1-17 years in the UK. Evidence [3] suggests an increase in GERD prevalence since 1995(p<0.0001), particularly in North America and East Asia.

In the western world between 10 and 20% of the population is affected [3]. In India 7.6 % population reports weekly [1] with GERD.



Figure 2 Global variation in the prevalence of gastro-esophageal reflux disease, defined as at least weekly heartburn and/or acid regurgitation  $% \mathcal{A}$  .

#### Pathophysiology

The main cause of GERD is the periodic relaxation of the lower esophageal sphincter [8]. In this GERD occurs due to the expose of the damaged squamous mucosa of the Oesophagus to Acid, proteolytic enzymes (e.g. trypsin and pepsin) and other bile salts [9]. This exposure repeatedly to gastric reflux may cause oesophagitis which is visible on endoscopy in some patients, although it is not visible in some patients. In many people the GERD may result from the spaces between the epithelium of the mucosa, and causing excessive stimulation of nerve endings and peripheral sensitization [10].

This gastric reflux without any reflux of gastric fluid may cause heartburn. In some people who do not respond to PPI treatment is possible that gas reflux may be causing distension with mechanoceptors in the esophageal wall. Acid production is high when the stomach is empty, but patients may experience GERD after consumption of meal, when [11] acid causes the lowest production [12]. This is caused because after eating an unbuffered volume of food, acid is formed in the proximal region of the stomach reffered to as the acid pocket.



Figure 3 Pathophysiology of GERD

#### The symptoms of GERD

*Heart burn:* It is the characteristic symptom. It occurs after meals and characteristically is brought on by bending and by lifting [13] or straining due to an increase in the pressure of intra-abdominal region. This is sometimes precipitated immediately by acid food or drink- tomatoes, orange juices, especially fortified wines, spirits and by cola and alcohol.

**Regurgitation:** regurgitation of gastric content into the mouth may occur during bending, after large meals or at night. Regurgitation may contribute to bronchitis and asthma [14]. Sore throat, globus sensation and hoarseness are other consequences.

*Chest pain:* oesophagitis may cause chest pain [15], similar to cardiac pain which occurs after meal by bending.

*Esophageal strictures:* persistent narrowing of the [1] esophagus caused by reflux induced inflammation.

*Esophageal ulcers:* it may occur, usually in the columnar mucosa close to the junction between squamous and columnar mucosa [1].

#### **Causes of GERD**

*Obesity:* increasing body weight is associated with more severe GERD. It has been shown that 13 % of changes in esophageal acid exposure are attributable to change in body [16] weight.

*Hypercalcemia*: Hypercalcemia increases the production [17] of gastrin which may lead to acidity.

*Medications:* medication such as prednisolone, antibiotics (tetracyclines), and pain relievers such as aspirin and ibuprofen [2]

#### Diagnosis

The diagnosis of GERD can be done by based on the symptoms of complicated and uncomplicated symptoms. The uncomplicated presentation [18] is heart-burn, regurgitation, or both, which occurs after by consuming food and aggravated when lying down or bending over, which can get relief by the use of single and multiple dose of antacids and other doses [19] of drugs doses.

It can be diagnosed by using endoscopy, oesophageal manometry, and x-rays etc.,[20]

The drugs which are used for this treatment is antacids, proton pump inhibitors, H2 receptor blockers.[3]



Figure 4 Endoscopic Pictures of a Reflux Oesophagitis (Red Streaks are erosions in the oesophagus

#### Treatment

Antisecretory agents:

H<sub>2</sub> receptor antagonist

Eg: Cimetidine[21], Ranitidine, Famotidine, Nizatidine Proton pump inhibitors:

Lansoprazole, Pantoprazole, , Esomeprazole, Omeprazole, Gastric antacids:

- 1. Systemic or absorbable antacids:
- Eg: Sodium bicarbonate
- 2. Non systemic or non absorbable antacids.
- 3. Buffer type antacids
- Eg:Aluminium hydroxide gel
- 4. Non buffer antacids
- Eg: Calcium carbonate
- 5. Miscellaneous agents
- Eg: Simethicone
- 6. Mucosal protective drugs
- Eg: Sucralfate, colloidal bismuth compounds
- 7. Ulcer healing drugs
- Eg: Carbenoxolone sodium

# CONCLUSION

Gastroesophageal reflux disease can be managed by the use of PPIs and other  $H_2$  receptor antagonist and some antacids. These medicatons are used in treating non-erosive gastroesophageal reflux disease. The over use of PPIs may be a problem. If a paient favorably responds to PPI, it is pre-sumed as GERD which has been addressed effectively. GERD is a simplistic model in which the acid exposure does not bear the literature which is equal to the degree of erosion. The main pathophysiology is with the increase in the acid production

with the proteolytic enzymes (pepsin and trysin) which causes the damage to the squamus mucosa. The acid production is more when the stomach is empty, also it is caused by eating the unbuffered volume of food. The diagnosis is done by endoscopy, x-rays, and esophageal manometry. Most of the drugs in GERD has shown the effect on combination therapy. Evidence suggest that most of the benefits can obtained by consuming a low carbohydrate diet. It also suggest that changes can be produced with the benefit of weight loss in GERD.

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