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EC MICROBIOLOGY
Review Article

Prevalence, Causes, Risk Factors and Updates of Management of GERD

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Received: January 09, 2020; **Published:** January 11, 2020

Abstract

Background: Gastroesophageal reflux disease (GERD) is a condition that develops when there is a retrograde flow of stomach contents causing symptoms or complications. GERD is an important health concern as it is associated with decreased quality of life and significant morbidity.

Aim: This review aims to highlight prevalence, causes, risk factors and updates of management of GERD.

Methodology: The review is comprehensive research of PUBMED since the year 1987 to 2019.

Conclusion: GERD is an extremely prevalent condition and became more common up until the turn of the last century. There are several important demographic factors associated with the risk of complications from GERD, but none are strongly associated with GERD symptoms. GERD management is a complex process involving multiple avenues of trial and fail from lifestyle modifications to medication therapy. The goal of treatment is decreasing symptoms, patient-physician communication is important. The paucity of literature on physician-patient communication in the treatment of GERD calls for more research in this area.

Keywords: *GERD; Management Of GERD; Prevalence of GERD*

Introduction

Gastroesophageal reflux disease (GERD) is a condition that develops when there is a retrograde flow of stomach contents causing symptoms or complications [1]. GERD is extremely common, with a prevalence of approximately 20% of adults in the western culture. Prevalence in North America is the highest ranging from 18.1% to 27.8% followed by 8.8% to 25.9% in Europe, followed by 2.5% to 7.8% in East Asia, 8.7% to 33.1% in the Middle East, followed by 11.6% in Australia, and 23.0% in South America [2].

Most adults with GERD have mild disease, but esophageal mucosa damage (reflux esophagitis) can develop in up to a third of the patients. GERD can present as Non-erosive reflux disease (NERD) when typical symptoms of GERD occur in the absence of visible mucosal injury during endoscopy or as erosive esophagitis (EE) when patients have histopathological changes in esophageal mucosa [3].

The typical symptom is heartburn. This most often occurs 30 min to 60 min after meals and upon reclining. Patients often report relief from antacids or baking soda. When patients present with this description of symptoms, the diagnosis can be established with a high degree of confidence [4]. It is important to distinguish between the underlying cause of the chest pain because of the potentially serious implications of cardiac chest pain and varied diagnostic and treatment algorithms based on etiology [5].

GERD is an important health concern as it is associated with decreased quality of life and significant morbidity. Diagnosis of GERD is typically based on classic symptoms and response to acid suppression after an empiric trial. A good clinical history may elicit GERD symptoms in patients with non-cardiac chest pain pointing to GERD as a potential etiology [6].

Medication therapy for GERD is targeted at symptom reduction and minimizing mucosal damage from acid reflux. While acid suppression is successful in the treatment of GERD, there does not appear to be a clear relationship between GERD severity and high gastric acid levels with the exception being Zollinger-Ellison syndrome [7].

Left untreated, GERD can result in several serious complications, including esophagitis and Barrett's esophagus. Esophagitis can vary widely in severity with severe cases resulting in extensive erosions, ulcerations and narrowing of the esophagus [8].

Epidemiology

GERD is one of the most commonly encountered conditions by both primary care physicians and gastroenterologists. A systematic review found the prevalence of GERD to be 10% - 20% in the Western world and 5% in Asia. There is a trend for higher prevalence in North America compared to Europe, and a trend for higher prevalence in Northern over Southern Europe [9].

Estimates of the prevalence of GERD in the Middle East therefore ranged from 8.7% to33.1% (sample size-weighted mean 14.4%) or 8.7% - 21.2% if the migrating nomads of Farsin Iran are excluded (sample size-weighted mean 13.4%) [10].

Few studies have reported GERD to be rare in Africa [11]. A recent study of Nigerian medical students reported a prevalence of 26.3% [12].

Pathogenesis

Reflux is a normal physiologic occurrence caused by transient relaxation of the lower esophageal sphincter [13]. GERD occurs when reflux of gastric contents causes troublesome symptoms or complications. In patients with GERD, transient relaxations occur more than normal. Diaphragm and gastric sling fibers provide structural support and contribute to LES pressure and competence [14,15].

Esophageal clearance limits the duration of contact between luminal contents and esophageal epithelium. Gravity and esophageal peristalsis remove volume from the esophageal lumen, while salivary and esophageal secretions neutralize acid. Researchers estimate that 50% of patients with GERD have some degree of decrease peristalsis. Also, conditions such as Sjogrën disease that affect the quality or quantity of the saliva, anticholinergic medications, and oral radiation can further worsen the natural protective mechanisms and lead to higher exposure of the esophageal mucosa to damage [16].

Increased intraabdominal pressure relative to LES resting pressure permits reflux of gastric contents into the distal esophagus. Increased intraabdominal pressure can be caused by medications, Valsalva maneuver, Trandelenberg position, or lifting [17].

Irritant effects of Refluxate: the gastric acid fluid (pH less than 4) is extremely caustic. Prolonged contact of gastric contents with esophageal mucosa leads to damage (esophagitis). In some patients, reflux of bile or alkaline pancreatic secretions can also lead to damage [1].

Risk factors

Risk factors for GERD include older age, excessive body mass index (BMI), smoking, anxiety/depression, and less physical activity at work. Eating habits may also contribute to GERD, including the acidity of food, as well as size and timing of meals, particularly with respect to sleep. Recreational physical activity appears to be protective, except when performed post-prandially [18].

Advancing age has been inconsistently associated with an increased risk for GERD symptoms. In North America and in Europe, there is no association between sex and symptoms of GERD, but in South America and in the Middle East, women are approximately 40% more likely to report GERD symptoms than men [19]. In a meta-analysis, the summary odds ratio for 50 years or more vs less than 50 years of age was 1.32, but with an I2 value of 91.5%, indicating substantial heterogeneity among study results [19].

Obese subjects are more likely to have GERD than those with normal body mass index [20]. Several researches reported relationship between body mass and GERD [21]. A meta-analysis found that obesity was associated with a statistically significant increase in the risk of GERD symptoms and erosive esophagitis [22].

Alcohol drinking and hiatus hernia are risk factors for GERD and esophagitis [23,24]. A previous study identified cigarette smoking and alcohol as risk factors for GERD [25]. A well-designed study did not find a positive association of current alcohol use with erosive esophagitis, and there is no positive association between alcohol use and Barrett's esophagus [26].

The presence of a hiatal hernia is associated with incompetent LES, defective peristalsis, more severe mucosal damage, and increased acid exposure [27]. Hiatal hernia is a significant factor as it disintegrates the gastro-esophageal sphincter, as the proximal stomach is dislocated into the chest and the crural diaphragm becomes separated from the LES. There is a linear correlation between hernia's size and the severity of reflux symptoms [28].

Increased consumption of cola and coffee by medical was associated with an increased prevalence of GERD [29]. A recent study [30] estimated the correlation between several categories of drugs and GERD. They reported that non-steroidal anti-inflammatory drugs, nitrates, tricyclic antidepressant medications, hypnotics and benzodiazepine, anticholinergic drugs and theophylline promote the onset of GERD.

Estimates of the proportion of phenotypic variance in GERD symptoms explained by genetic factors have ranged from 0 to 22% [31]. In a twin study, 13% of the variance in GERD symptoms was estimated to be due to genetic effects, but even that proportion appeared to be mediated by anxiety and depression [32]. The genetic risk for GERD is polygenic with no individual mutation found to be significantly associated with GERD in genome wide association studies, though larger studies might yet still be able to identify statistically significant individual mutations [33].

Diagnosis

Diagnosis is presumptive in most cases the. Diagnosis of GERD relies on questioning of the patient. Heartburn with or without regurgitation is typically sufficient to suspect GERD, particularly when these symptoms are worse post-prandially or when recumbent [34,35]. Practitioners should further investigate patients with "alarm features" such as troublesome dysphagia, odynophagia, weight loss, iron

deficiency anemia and in patients with troublesome symptoms that persist despite appropriate empiric proton pump inhibitor therapy. It must be remembered that diabetic patients may present with dyspeptic symptoms during a myocardial infarction. Thus, a high index of suspicion should be maintained in these patients in the acute setting [3].

The studies are of limited use in the management of GERD due to poor sensitivity in milder forms of GERD, but they can detect moderate to severe esophagitis, strictures, hiatal hernia, and tumors, most commonly used are the barium swallow, which only examines the esophagus, and the upper gastrointestinal series, which examines the esophagus, stomach, and small intestines [36].

Additional testing may be necessary, however, for those who do not respond to acid suppression, those who have alarm symptoms (e.g., dysphagia, odynophagia, iron deficiency anemia, weight loss, etc.) and those who have suffered from the disease for an extended period of time due to concern for Barrett's esophagus. The rationale for pursuing additional testing includes confirmation of GERD as well as evaluation of GERD associated complications or alternate diagnoses [37].

Management and treatment

Symptoms relief, healing of esophagitis, prevention of recurrence, and prevention of complications are the main aims of treatment. Treatment includes life style modifications and drugs or surgical treatment with corrective anti-reflux surgery [38].

Non-pharmacological management

Initial treatment of GERD is lifestyle modifications. Education about the factors precipitating physiological and pathological reflux is also required [39]. Advice on diet, alcohol and tobacco use, sleep position and weight loss needs to be modified. Stopping smoking, elevating the bed headrest and avoiding late evening meals can reduce symptoms [40]. Management should start with parental support [41].

No evidence that reduced feeding volume or extensively hydrolyzed or amino-acid based formula is effective for the treatment of infants presenting with GER symptoms. There is a consensus that thickened formula reduces regurgitation [42].

Supine sleeping is recommended as the safest position to prevent the risk of SIDS [43]. Patients should improve their sleep hygiene. Physicians should recommend additional lifestyle modifications [44].

Pharmacological Management: Patients who continue to have GERD symptoms despite lifestyle modifications, medical therapy is commonly offered or used. Gaviscon, histamine 2 receptor antagonists (H2RAs), PPIs, Cara-fate, TLESR reducer, and pro-kinetics are recommended.

Antacids and alginates neutralize acid and contain aluminum, magnesium, or calcium salts or sodium/potassium bicarbonate. Alginates reduce reflux symptoms and the number of episodes of regurgitation and vomiting. Alginates are recommended as an alternative treatment to feed thickening agents in breastfed infants or as a trial in infants whose symptoms persist despite conservative measures [45].

PPIs are the most effective medical therapy for GERD. They have profound and consistent acid suppression. PPIs are safe and demonstrate different levels of satisfaction as compared with other anti-reflux medications. Several studies have shown that PPI treatment is superior to H2RA treatment for the symptomatic relief of both EE and NERD patients [46,47]. The efficacy of different PPIs has been evaluated although no study compared different PPIs [48]. In summary, PPIs can provide symptom relief in approximately 57% to 80% of patients with EE and about 50% of the patients with NERD. In addition, healing of EE can be obtained in greater than 85% of GERD patients undergoing treatment with PPI [49].

Parietal cells secrete acid in response to three stimuli: histamine at the H2 histamine receptor, acetylcholine, and gastrin. H2RAs suppress gastric acid secretion by competitively inhibiting histamine at the parietal cell's H2receptor. In adequate doses, H2RAs are effective

in the treatment of peptic disease [50] and healing erosive esophagitis compared to placebo [51]. However, patients requiring more than occasional use can develop to rapid tachyphylaxis [52]. Dosage requirements vary by age, but children require a relative higher dose than adults [53].

Surgical Management: Majority of patients do not require surgery. Surgical therapy is preferred for patients who do not respond well to acid suppression medication, patients who prefer surgical approach, and patients who present with complications due to GERD. Complications of surgical procedures include bloating and gas-bloat syndrome, dysphagia, diarrhea, recurrent heartburn and recurrent atypical symptoms [34,54].

Conclusion

GERD is an extremely prevalent condition and became more common up until the turn of the last century. There are several important demographic factors associated with the risk of complications from GERD, but none are strongly associated with GERD symptoms. GERD management is a complex process involving multiple avenues of trial and fail from lifestyle modifications to medication therapy. The goal of treatment is decreasing symptoms, patient-physician communication is important. The paucity of literature on physician-patient communication in the treatment of GERD calls for more research in this area.

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Volume 16 Issue 2 February 2020