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Esophageal Reflux Disease

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Esophageal reflux, also referred to as gastroesophageal reflux, involves the regurgitation of gastric contents into the esophagus. The classic and predominant symptom of esophageal reflux is heartburn - a retrosternal burning and discomfort that worsens after eating. Sometimes regurgitation or excessive salivation may occur (9) along with rare respiratory problems due to aspirated refluxate. Another frightening symptom is crushing chest pain that is often indistinguishable from the angina associated with myocardial infarction. Studies show that esophageal reflux is quite common with around 44% of American adults experiencing heartburn at least once a month (11). Esophageal reflux actually occurs to a small degree in all people, but those who experience symptoms or tissue damage due to reflux are said to have gastroesophageal reflux disease (GERD). Many threatening complications of chronic reflux include esophagitis of varying degrees, hemorrhage, peptic stricture and Barrett's epithelium (5). The primary irritating constituents of the refluxate are gastric acid and pepsin, though pancreatic enzymes and bile acids cause deleterious effects as well.

Pathophysiology

Generally, two conditions must be met for a reflux to occur. The first condition requires that the gastrointestinal contents must be "prepared" to reflux, either because of large gastric volume, location of gastric contents near the gastroesophageal junction (due to bending or recumbent position), or increased gastric pressure (often due to obesity, pregnancy, ascots or tight clothing). The second condition involves compromise of the antireflux mechanism involving the lower esophageal sphincter located at the esophageal-gastric junction. Thus, even though the exact pathophysiology of esophageal reflux remains unclear (10), several of the mechanisms contributing to GERD have been elucidated. Abnormalities in one, and most likely more than one, of the following mechanisms plays a role in the development of GERD in a given patient: lower esophageal sphincter (LES) function, esophageal clearance and gastric emptying.

The esophagus is normally protected by excessive reflux by the contraction of the LES. Though everyone experiences transient LES relaxations, most patients with GERD demonstrate relaxations that are longer and more frequent than normal (9). Certain foods and activities reduce LES pressure (i.e. promotes LES relaxation) and therefore increase the likelihood of reflux. These factors include cigarette smoking, fatty foods, alcohol, chocolate, peppermint, garlic and onions (5,8,9,10). In addition, increased intra-abdominal pressure due to such factors as pregnancy, obesity, tight garments and recumbent body position may lower the strength of the LES (8,9).

Esophageal clearance involves the rapid restoration of normal pH after reflux has occurred. In normal individuals refluxate is returned to the stomach by a series of peristaltic movements initiated by the act of swallowing. In addition, bicarbonate in the saliva acts to neutralize residual acid. Impaired esophageal clearance mechanisms are now recognized as important factors in GERD and the early stages of esophagitis (9) due to the prolonged exposure time to refluxed acid [NOTE: It is crucial to understand the basic principle that an inverse correlation exists between the healing of esophagitis and the percentage of time that the esophagus is exposed to an abnormally low pH (3)]. Factors affecting proper esophageal clearance include impaired peristalsis, recumbent position, and sleep where esophageal peristalsis is naturally impaired (9).

The final main mechanism involved in GERD is gastric emptying delay. Such a situation lead to increased gastric volume which, as mentioned above, predisposes the patient to esophageal reflux.

Treatment: Nonpharmacological

The first treatment strategy should involve nonpharmacological lifestyle changes and diet modifications. As GERD is a chronic disease, the changes must be long-term to be fully effective. Now that we understand many of the mechanisms underlying esophageal reflux, we may understand the rationale behind the suggested changes:

1) To decrease intra-abdominal pressure, the patient should achieve desirable body weight and wear clothes that are not too tight (5,8,9,10).

2) To avoid reflux from increased gastric volume, frequent small meals rather than fewer large meals should be consumed (5,9,10)

3) The aforementioned dietary factors that cause decreased LES pressure should be avoided, along with smoking.

4) Meals within 2-3 hours before bedtime should be avoided due to the fact that lying down promotes reflux and peristalsis is impaired during sleep (5,8,9,)

5) Certain foods such as citrus fruit and juices, tomato products, carbonated beverages coffee (regular and decaffeinated), pepper, and very hot or very cold food have a direct irritating effect on inflamed esophageal mucosa, and, as such, should be avoided (5,8,9,)

6) Another common and long-standing strategy is to elevate the head of the bed about six inches in order to promote esophageal acid clearance, decrease the number of reflux episodes and decrease the quantity of refluxed material (5,9,10).

7) Though certain foods decrease esophageal sphincter pressure, certain foods definitely do not. Thus the intake of these foods should be encouraged. They include low-fat carbohydrates and low-fat proteins (5,8).

8) As saliva helps neutralize the acid in the esophagus, lozenges to stimulate saliva secretion are suggested (9).

Pharmacological Therapy

The most common form of pharmacological therapy is the very prevalent use of antacids which serve to neutralize refluxed gastric acid. It has been estimated that 13% of the adult population takes at least two antacid tablets per week (7). For many with mild GERD, nonpharmacological therapy coupled with over-the-counter antacid usage is enough to hold symptoms in check. For others, however, a more aggressive treatment must be sought. Strategies involve using prokinetic drugs and/or inhibitors of gastric acid production.

As GERD is mainly a motility disorder, the use of prokinetic drugs somewhat improves the rate of gastric emptying, enhances esophageal clearance, and increases LES tone (4,9). Due to some of the side effects - abdominal discomfort, wheezing, bradycardia, nightmares, depression and sometimes neurologic symptoms resembling Parkinson's (9) - prokinetic agents are less than ideal. Due to these potential side effects and the recent discovery of the more efficient proton pump inhibitors (discussed below), use of prokinetic agents is declining. If administered, these agents are usually coupled with H2 receptor inhibitors (discussed below) to treat mild cases (4,9,10).

The most effective medication for long-term and short-term management of GERD are those drugs that reduce gastric acid secretion (10). If acid secretion is reduced, then less acid will be present within the refluxate, and the esophagus will not be prone to as much damage (also, as mentioned above, if esophagitis exists, reducing the amount of time that the esophagus is exposed to very low pH will promote healing). The two main classes of antisecretory drugs used are the H2 receptor antagonists and the proton pump inhibitors. Both drugs act by inhibiting different steps along the pathway that leads to HCl production by the parietal cells of the stomach. Normal secretion requires the activation of one or more receptors in the basolateral membrane (Histamine-2 (H2) receptor, Ach receptor, and gastrin receptor). Such activation ultimately leads to luminal HCl secretion by action of the H+/K+ ATPase in the apical membrane (1,10). The target of the H2 receptor antagonists is the histamine receptor in the basolateral membrane, while the proton pump inhibitors (PPIs) selectively block the proton pump in the apical membrane.

The H2 receptor antagonists (or HRAs) have been used since 1976 and had become the backbone of chronic reflux therapy. Healing rates for GERD with HRA treatment are from 50% to 60% (6). The newly available proton pump inhibitors (since 1989) have been shown to be much more effective in treating GERD patients due to the more efficient suppression of gastric acid secretion. This increased efficiency

may be primarily attributed to the fact that PPIs block the final step in parietal acid secretion. HRAs, on the other hand, block one of the initial steps - histamine receptor activation. The secretion pathway may still be activated by acetylcholine or gastrin even if HRA is present. Some other drawbacks to HRA usage are that patients may build up some degree of tolerance and the duration of the drugs effect is shorter than for PPIs (4). Some drawbacks for PPI usage involve the theoretical (but never proven) risk for carcinoma (9,10), lack of proof of safety for longer-term usage and increased cost.

There is no doubt that PPIs more effectively reduce acid secretion and promote healing in GERD than HRAs (1,2,4,10). In milder cases of reflux, however, where there is no mucosal inflammation, the abnormal exposure to high pH in the esophagus is minimal. Thus, a limited reduction in acid secretion may be enough to relieve symptoms. Therefore, the less inexpensive HRAs are still frequently used to treat milder cases of GERD (2,4,10), while the more expensive yet more effective PPIs are reserved for moderate to severe cases.

In conclusion, the treatment strategy for GERD should follow a logical progression (2,4,10). At the first onset of symptoms, antacid therapy for short-term relief and nonpharmacological lifestyle changes for long-term relief should be implemented. If symptoms persist, Histamine Receptor Antagonists should be used. If desirable results are not obtained, PPI therapy should be instigated. As esophageal reflux is a chronic disease, long-term maintenance therapy is usually called for. Since the safety of the proton pump inhibitors for long-term use is uncertain, the use of PPIs should be alternated with periodic "breaks" of HRA/promotility therapy (10). If none of the above methods work, surgical "fundoplication" - wrapping a part of the fundus of the stomach around the lower part of the esophagus in order to create a high pressure zone that prevents reflux - may be indicated (12).

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