GI café

SQUARE PHARMACEUTICALS LTD. BANGLADESH

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Guidelines for the Diagnosis and Treatment of Gastroesophageal Reflux Disease

INTRODUCTION AND PREAMBLE

Guidelines for the diagnosis and treatment of gastroesophageal reflux disease (GERD) were published by the American College of Gastroenterology in 1995 and updated in 1999. These and other guidelines undergo periodic review. Advances continue to be made in the area of GERD, leading us to review and revise our previous guidelines statements. These and the original guidelines are intended to apply to all health-care providers who address GERD and are intended to indicate the preferred, but not only, acceptable approach. Treatment should be based on the course best suited to the individual patients and the variables that exist at the moment of the decision. These guidelines are applicable to adult patients with the symptoms, tissue damage, or both that result from the reflux of gastric content into the esophagus. For the purpose of these guidelines, GERD is defined as *symptoms or mucosal damage produced by the abnormal reflux of gastric contents into the esophagus*.

These and the previous guidelines were developed under the auspices of the American College of Gastroenterology and its Practice Parameters Committee and approved by the Board of Trustees. The world literature was reviewed extensively for the original guidelines and again reviewed for each revision using the National Library of Medicine database. Appropriate studies were reviewed and any additional studies found in the reference list of these papers were obtained and reviewed. Evidence was evaluated along a hierarchy, with randomized, controlled trials given the greatest weight. Abstracts presented at national and international meetings were only used when unique data from ongoing trials were presented. When scientific data were lacking, recommendations were based on expert consensus obtained from both the literature and the experience of the authors and the Practice Parameters Committee.

DIAGNOSTIC GUIDELINE : EMPIRICAL THERAPY

If the patient's history is typical for uncomplicated GERD, an initial trial of empirical therapy (including lifestyle modification) is appropriate. Endoscopy at presentation should be considered in patients who have symptoms suggesting complicated disease, those at risk for Barrett's esophagus or when the patient and physician feel early endoscopy to be appropriate.

Level of Evidence

Symptoms which are highly specific for GERD include heartburn (pyrosis), regurgitation, or both, which often occur after meals (especially large or fatty meals). These symptoms are often aggravated by recumbency or bending over and are relieved by antacids. The combination of symptoms and endoscopic changes are highly specific (97%) for GERD (confirmed with pH testing). Expert opinion holds that it is appropriate to offer empirical therapy for GERD to patients with symptoms consistent with GERD. It is also reasonable to assume a diagnosis of GERD in patients who respond to appropriate therapy.

Table 1. Rating of Levels of Evidence Used for this Guideline

I. Strong evidence from at least one published systematic review of multiple well-designed randomized controlled trials

II. Strong evidence from at least one published properly designed randomized controlled trial of appropriate size and in an appropriate clinical setting

III. Evidence from published well-designed trials without randomization, single group prepost, cohort, time series or matched case-controlled studies

IV. Evidence from well-designed nonexperimental studies from more than one center or research group or opinion of respected authorities, based on clinical evidence, descriptive studies, or reports of expert consensus committees

Further diagnostic testing should be considered if the patient does not respond to therapy, when there are alarm symptoms suggesting complicated disease (dysphagia, odynophagia, bleeding, weight loss, or anemia) and when patients have a sufficient duration of symptoms to put them at risk for Barrett's esophagus. Patients who do not respond to therapy often have another cause for their symptoms, but this lack of response does not always exclude reflux as a possibility. Even when the most effective therapy for GERD is prescribed, some patients will continue to reflux acid. A short trial of a high dose proton pump inhibitor has 75% sensitivity, but only 55% specificity for reflux in heartburn patients using ambulatory pH testing as the "gold standard". These problems with the sensitivity and specificity of using a therapeutic trial as a test for GERD must be weighed against the ease of use and decreased cost (primarily related to decreased use of diagnostic testing of this approach). Finally, symptoms do not seem to predict the degree of esophagitis and are far from perfect in predicting complications of GERD including Barrett's esophagus.

DIAGNOSTIC GUIDELINE : USE OF ENDOSCOPY IN GERD

Endoscopy is the technique of choice used to identify suspected Barrett's esophagus and to diagnose complications of GERD. Biopsy must be added to confirm the presence of Barrett's epithelium and to evaluate for dysplasia.

Level of Evidence

Endoscopy allows direct visualization of the esophageal mucosa. This is the only reliable method for the diagnosis of Barrett's esophagus. A reticular pattern on barium esophagram is neither sensitive (26%) nor specific (50%) when compared to endoscopy with biopsy. Barium radiography is reasonably accurate in cases of severe esophagitis (80% or better), but is much less accurate with mild esophagitis (less than 25%). Finally, reflux of barium during radiographic evaluation is only positive in 25–75% of symptomatic patients and is falsely positive in up to 20% of normal controls. Patients with hiatal hernias or who reflux barium at fluoroscopy have more acid exposure by ambulatory pH testing, but these findings have poor specificity and sensitivity and should not be used as a screening test for GERD. These factors limit the usefulness of barium radiography in the routine diagnosis of GERD and it is not recommended.

Documentation of the presence or absence of esophagitis does not usually determine the initial approach to patients with GERD. Higher grades of esophagitis are more difficult to heal, but once healed can be maintained in remission with medical or surgical therapy. The main advantage of knowing a patient has (or had) esophagitis is to confirm the diagnosis of GERD prior to surgical or endoscopic therapy for GERD. Typical esophagitis is essentially diagnostic for GERD.

DIAGNOSTIC GUIDELINE : AMBULATORY REFLUX MONITORING

Ambulatory monitoring of the esophagus helps to confirm gastroesophageal reflux in patients with persistent symptoms (both typical and atypical) without evidence of mucosal damage, especially when a trial of acid suppression has failed. It may also be used to monitor the control of reflux in patients with continued symptoms on therapy.

Level of Evidence

While endoscopy allows for the evaluation of esophageal mucosa, the presence or absence of mucosal injury does not provide proof that the patient's symptoms are or are not related to GERD. Many patients with typical GERD symptoms and increased esophageal acid exposure do not have esophagitis. Patients with symptomatic, but not excessive gastroesophageal reflux have persistence of symptoms and requirements for therapy similar to patients with excessive reflux, but are less likely to have endoscopic findings. This "endoscopic negative" form of GERD produces symptoms and illness behavior identical to that of GERD with endoscopic findings. Ambulatory pH testing allows both the identification of patients with excess esophageal acid exposure and those with symptoms that correlate with esophageal acid (either with normal or abnormal total acid exposure). Good reproducibility (84–93%) and sensitivity and specificity (96%) have been reported in patients with erosive esophagitis.

Reasons for concern include the finding of normal acid exposure in up to 29% of patients with documented esophagitis and differences found in the simultaneous acid exposure recorded by two attached probes. A recent report repeated pH testing on patients who had an initial negative test. If the patient's symptoms had been typical or worse than typical during their first pH test, 22% of second tests were positive, while 55% of studies were abnormal if the patients said their day was "better than typical" during the first test. Despite these limitations, ambulatory pH testing remains the best

method to study the actual amount of reflux occurring in a given patient. Ambulatory pH testing while on reflux therapy may also be of benefit in the patient with refractory symptoms.

TREATMENT GUIDELINE : LIFESTYLE MODIFICATION

Lifestyle modification may benefit many patients with GERD, although these changes alone are unlikely to control symptoms in the majority of patients.

Level of Evidence

Education of the patient about factors that may precipitate reflux remains reasonable. Numerous studies have indicated that elevation of the head of the bed, decreased fat intake, cessation of smoking and avoiding recumbency for 3 h postprandially decreases distal esophageal acid exposure although data reflecting the true efficacy of these maneuvers in patients is almost completely lacking. Certain foods (chocolate, alcohol, peppermint, coffee and perhaps onions and garlic) have been noted to lower LES pressure, although randomized trials are also not available to test the efficacy of these maneuvers. Many authors assume the 20–30% placebo response rate seen in most randomized trials is due to lifestyle changes, but this has not been rigorously tested. The potential negative effect of lifestyle changes on a patient's quality of life has also not been examined.

TREATMENT GUIDELINE : ACID SUPPRESSION

Acid suppression is the mainstay of therapy for GERD. Proton pump inhibitors provide the most rapid symptomatic relief and heal esophagitis in the highest percentage of patients. Although less effective than PPIs, histamine2-receptor blockers given in divided doses may be effective in some patients with less severe GERD.

Level of Evidence

In the original guideline statement, the results of 33 randomized trials including over 3,000 patients with erosive esophagitis are presented. Symptomatic relief can be expected in 27% of placebo treated, 60% of H2RA treated, and 83% of PPI treated patients. Esophagitis healed in 24% of placebo treated, 50% of H2RA treated, and 78% of PPI treated patients. We will not readdress those studies here, but it is clear that while some patients may have relief of symptoms and improvement or healing of esophagitis on H2RAs, PPIs eliminate symptoms and heal esophagitis more frequently and more rapidly than the other agents. Both higher doses and more frequent dosing of H2RAs appear to improve results in the treatment of reflux, but are still inferior to PPIs. In addition to controlling symptoms and esophagitis, PPI therapy has been shown to normalize the impaired quality of life caused by GERD.

TREATMENT GUIDELINE : SURGERY

Antireflux surgery, performed by an experienced surgeon, is a maintenance option for the patient with well-documented GERD.

Level of Evidence

Considerable controversy exists over the long-term effectiveness of surgical intervention in GERD and whether it is equal or superior to chronic medical therapy. In the early-published trials of medical versus surgical therapy, surgery was shown to be more effective, although both trials used medical therapy that would be considered ineffective today. The initial comparison favored surgical over a rather modest medical therapy (essentially antacids and lifestyle changes) over a 36-month period. A comparison of surgery versus ranitidine and metoclopramide indicated superiority for the surgical approach. The long-term outcome of many of these patients reported that after 10 yr, 92% of the patients randomized to medication were still on medications and 62% of those who were initially treated with surgery were now back on reflux medication. A trial that randomized 310 patients between surgery and PPIs found surgery to be slightly superior to omeprazole 20 mg per day at the end of 5 yr, but if dose titration up to 40–60 mg per day of omeprazole were used, the two treatments were equal. Proper selection and preoperative evaluation of patients is very important. In a study of 100 patients, the best predictors of a good outcome were; age <50 yr and typical reflux symptoms that had ccompletely resolved on medical therapy. It is also clear that these typical reflux symptoms are more likely to resolve after surgery than the other atypical and supraesophageal symptoms.

AREAS IN NEED OF ADDITIONAL STUDY

GERD has been extensively studied and we continue to see a steady improvement in our understanding of the condition. Despite this, many questions remain to be answered, including:

Will impedance monitoring and "tubeless" pH monitoring change our approach to subsets of GERD patients?

(i) Will esophageal manometry prior to antireflux surgery be abandoned or perhaps be replaced by impedance testing? If motility testing is abandoned, will a partial or complete fundoplication become the operation of choice?

(ii) How will the availability of OTC and generic PPIs change the face of GERD for both primary care and gastroenterology?

(iii) Will new promotility agents be developed to address the underlying physiological defect in GERD?

(iv) Will the results from endoscopic therapy of GERD improve and become more attractive options?

(vi) There are many questions related to Barrett's esophagus covered extensively in other guidelines, but some of these include:

(a) Is there an appropriate public health benefit for Barrett's screening and surveillance?

(b) Do patients who have their GERD diagnosed and controlled with medication still eventually need a "once in a lifetime" endoscopy to exclude Barrett's esophagus?

(c) Will less invasive (small caliber, unsedated) endoscopy allow for more cost-effective screening for Barrett's esophagus in GERD patients?

Ref : 1. DeVault KR, Castell DO. Guidelines for the diagnosis and treatment of gastroesophageal reflux disease. Arch Intern Med 1995;155:2165–73.

Proton Pump Inhibitors and *Helicobacter pylori* Gastritis: Friends or Foes?

A decade ago, the role of *Helicobacter pylori* in chronic gastritis and peptic ulcer disease had become recognized and further research had revealed that chronic *H. pylori* gastritis predisposed to atrophic gastritis and gastric cancer. This led to the recognition by the WHO that *H. pylori* was a class I carcinogen, i.e. a carcinogen beyond doubt (International Agency for Research on Cancer 1994). This classification strongly stimulated further research into the interaction between *H. pylori* and its host, among others into factors which modulate the severity of *H. pylori* gastritis and the risk of long-term complications. This research focused in subsequent years on three topics, respectively related to bacterial virulence factors, host genetics, and gastric acid secretion. The message for all three factors was similar, i.e. patients with more severe gastritis had a higher risk of developing long-term complications. This message was well accepted in relation to bacterial virulence factors and host genetics, but it evoked much controversy and debate in relation to acid secretion. The main reason for this was that gastric acid secretion can in contrast to host genetics and bacterial virulence be influenced and thus the discussion involved acid suppressive therapy. A reduction of gastric acid secretion was shown to change the pattern and severity of *H. pylori* gastritis, which opened a discussion on longterm consequences of profound acid suppressive therapy in *H. pylori*-positives. To provide more insight into this important clinical issue, this paper will review the issue of *H. pylori*, chronic gastritis and profound acid suppressive therapy.

Knowledge from the pre-Helicobacter era

The discussion on chronic gastritis and acid suppression had a long prelude. From the 1950s onward, various cohort studies in the pre-Helicobacter era, some with up to 25 years follow-up, had shown that chronic active gastritis was a very common condition, which predisposed to gland loss, or scarring of the mucosa, leading to atrophic gastritis (Siurala *et al*. 1968; Ihamäki *et al*. 1978; Kuipers 1998). The cause of chronic gastritis in most of these patients was at the time unknown. Other cohort studies showed that the condition of atrophic gastritis considerably increased the risk for gastric cancer (Hitchcock *et al*. 1955; Zauicheck *et al*. 1955; Siurala et al. 1966; Sipponen et al. 1985; Kato *et al*. 1992). These studies also showed that the severity and distribution of gastritis would largely vary between individuals, but that the intraindividual pattern would usually remain very stable over time. The pattern differed in particular between patients with duodenal ulcer and those with gastric ulcer disease, the former having an antral-predominant pattern with little inflammation in the corpus and the latter having a corpus-predominant pangastritis pattern (Thomas *et al*. 1972). It was also recognized that the latter pattern was associated with a more rapid development of atrophic gastritis (Maaroos *et al*. 1985), and gastric ulcer patients were thought to have a higher gastric cancer risk than duodenal ulcer patients, a thought that was later confirmed in a large cohort follow-up study (Hansson *et al*. 1996).

***H. pylori*, acid and atrophy, initial studies**

Persistent *H. pylori* gastritis may lead to a destruction of gastric glands with replacement by fibrosis, a condition of atrophic gastritis (Dixon *et al*. 1996). The presence of atrophic gastritis facilitates the development of intestinal metaplasia, dysplasia and gastric adenocarcinoma.

For an *H. pylori*-positive individual, the chance of developing atrophic gastritis depends on the severity and distribution of gastric mucosal inflammation (Kuipers *et al*. 1995a & b; Uemura *et al*. 2001). *H. pylori*-positive patients with low acid production and a corpus-predominant pangastritis thus appeared at increased risk for development of atrophic gastritis compared to *H. pylori*-positive patients with unimpaired acid output. This was first observed from the 1970's onwards in duodenal ulcer patients undergoing vagotomy (Meikle *et al*. 1976; Peetsalu *et al*. 1991).

The *H. pylori* status of these patients was unrecognized, but we can retrospectively assume that most were *H. pylori*-positive. Initial studies on the efficacy of proton pump inhibitors reported that the use of these drugs also was associated with aggravation of corpus gastritis and a progression towards atrophy similar to that reported after vagotomy (Solcia et al. 1989; Lamberts *et al*. 1993; Klinkenberg-Knol *et al*. 1994). Again, the *H. pylori* status of these patients was not reported, but aggravation of gastritis and progression towards atrophy only occurred in those with antral gastritis before the start of proton pump inhibitor therapy, once more suggesting the association with *H. pylori*. We therefore performed a follow-up study in two populations of GERD patients (Kuipers et al. 1996). One group was treated with omeprazole, the other group with a fundoplication without further acid suppression. It appeared that in both cohorts, development of atrophic gastritis was very rare in *H. pylori*-negatives. In *H. pylori*-positives however, progression towards atrophic gastritis of the corpus mucosa occurred significantly faster in those who were treated with omeprazole than in those treated with a fundoplication (Kuipers *et al*. 1996).

The dynamics of *H. pylori* gastritis in proton pump inhibitor users

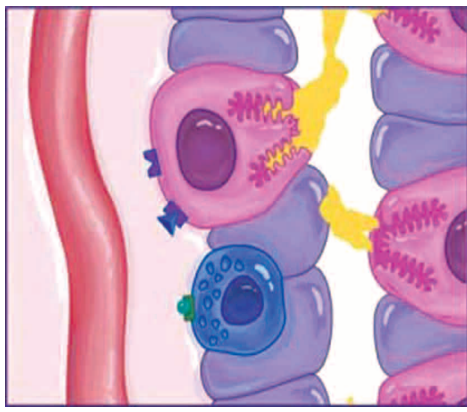
After this publication, various other studies addressed the effects of proton pump inhibitor therapy on *H. pylori* gastritis. Without any exception, they first of all confirmed that most *H. pylori*-positive GERD patients before the start of acid suppressive therapy have an antral-predominant gastritis consistent with intact acid secretion as expected in patients with a condition of acid reflux. The start of proton pump inhibitors was then consistently reported to induce a corpus predominant pangastritis in the large majority of *H. pylori*-positive patients taking these drugs. This condition develops within weeks after start of treatment and persists for the duration of therapy. A number of studies with a follow-up of 6 months or longer further addressed the rate of development of atrophic gastritis of the corpus mucosa in *H. pylori*-positives taking proton pump inhibitor maintenance treatment. Most of these studies were uncontrolled.

Most other uncontrolled data however supported the concept that 25 to 40% of *H. pylori*-positive individuals have signs of atrophic gastritis after 4–7 years of proton pump inhibitor maintenance treatment (Eissele et al. 1997; Klinkenberg-Knol *et al*. 2000; Schenk *et al*. 2000; Geboes *et al*. 2001; Lamberts et al. 2001; Rindi *et al*. 2005). Apart from our own data (Kuipers *et al*. 1996), there was unfortunately only one other controlled study into this phenomenon, which was outstanding for being the only study with a randomized design (Lundell *et al*. 1999). It reported no significant differences with respect to the incidence of atrophic gastritis in GERD patients randomized to either omeprazole or to a fundoplication procedure during a follow-up of three years. The authors concluded that profound acid suppression does not accelerate the development of atrophic gastritis in *H. pylori*-positive GERD patients.

The distinction between inflammation and gland loss

The question was raised whether the observed development of atrophic gastritis in these studies truly reflected a situation of loss of glands or prominent inflammation falsely suggesting loss of gland by preventing glands to abut to each other (Genta 1996). The latter was a logical possibility, but it was refuted by several observations. First, it appeared that the influx of inflammatory cells into the corpus mucosa following the start of proton pump inhibitor therapy in *H. pylori*-positives would occur within days to weeks, while atrophic gastritis developed much slower, confirming that these are separate phenomena (Klinkenberg-Knol *et al*. 2000). Second, quantitative morphometric histological studies showed that the actual volume proportion of the gastric corpus mucosa occupied by inflammatory cells is much smaller than the volume proportion that consists of glands or the loss of glands and the development of fibrosis in case of atrophic gastritis (van Grieken *et al*. 2001). These morphometric results, correlated well with the standard histological evaluation by experienced pathologists using the updated Sydney classification. Third, the development of moderate to severe corpus atrophic gastritis in *H. pylori*-positive proton pump inhibitor users was associated with functional changes, in particular an increase in serum gastrin levels (Schenk *et al*. 1998), and a decrease in serum vitamin B12 levels, which decrease did not occur in *H. pylori*- positive proton pump inhibitor users who did not develop atrophic gastritis (Schenk et al. 1999a & b).

In summary, various experimental and clinical studies show that acid suppression affects the pattern and distribution of *H. pylori* gastritis. This can be explained by bacterial physiology. The chance for destruction of gastric glands or development of atrophic gastritis increases with the severity of chronic gastritis.



Effect of *H. pylori* and *H. pylori* eradication on reflux disease

H. pylori colonization may to some extent protect against GERD and complications of GERD such as Barrett's oesophagus and oesophageal adenocarcinoma (O'Connor 1999). In addition, *H. pylori* gastritis may augment the acid suppressive effects of proton pump inhibitors (Verdu *et al*. 1995). These two factors raised the concern that *H. pylori* eradication in patients with GERD may worsen reflux and impair symptom control by proton pump inhibitor therapy. This concern however was refuted by a number of studies. *H. pylori* may improve healing of oesophagitis in the first weeks of proton pump inhibitor treatment, but this effect is very limited (Holtmann et al. 1999). During proton pump inhibitor maintenance treatment, *H. pylori* has no measurable effect on GERD control. This conclusion was based on a number of case-control studies which showed similar symptom scores, omeprazole maintenance doses, relapse rates, and endoscopy and 24 hr oesophageal pH measurement results in *H. pylori*-positive and -negative GERD patients treated with proton pump inhibitors (Carlsson et al. 1997; Peters et al. 1999; Schenk et al. 1999a & b; Klinkenberg-Knol et al. 2000). Two further prospective randomized studies suggested that *H. pylori* eradication in GERD patients is not associated with an increased disease relapse rate when initial proton pump inhibitor therapy is withdrawn (Moayyedi et al. 2001; Schwizer et al. 2001). A third randomized study showed that *H. pylori* eradication during proton pump inhibitor maintenance therapy also had no effect on symptom control and did not necessitate increase of the dose of omeprazole (Kuipers. 2004). An exception is likely to be found in Asian populations, in whom the acid-suppressive effects of *H. pylori* gastritis may be more clinically pronounced. In these populations, eradication of *H. pylori* has been claimed to potentially aggravate GERD symptoms (Wu et al. 2004). In summary, *H. pylori* may have some effect on the prevention of GERD, but H. pylori eradication in most populations does not increase the severity or relapse rate of GERD, nor does it impair the efficacy of proton pump inhibitor treatment for this disease.

Conclusions

H. pylori gastritis and gastric acid closely interact. In *H. pylori*-positive patients, profound acid suppressive therapy induces a corpus-predominant pangastritis, which is associated with accelerated gland loss and development of atrophic gastritis. Both corpus-predominant and atrophic gastritis have in other patient categories been associated with a considerably increased risk for development of gastric cancer. However, when these patients are treated with *H. pylori* eradication, the gastritis completely resolves and pre-existent gland loss may to some extent be repaired. This is associated with a rise in ascorbic acid secretion into the gastric juice, and with a reduction of cell turnover and reactive oxygen radical formation. *H. pylori* eradication does not aggravate GERD nor does it impair the efficacy of proton pump inhibitor maintenance therapy for this condition. This is the background for the advice within the European guidelines for the management of *H. pylori* infection to offer an *H. pylori* test and treat policy to patients who require proton pump inhibitor maintenance therapy for GERD (Malfertheiner et al. 2006). As such a policy fully reverses *H. pylori* pangastritis even in patients who have been treated for years with proton pump inhibitors, there is no need to eradicate *H. pylori* before the start of proton pump inhibitors. In fact, the somewhat slower initial response of *H. pylori*-negative GERD patients to proton pump inhibitor therapy and the fact that many GERD patients will only require short-term therapy suggests to first start the proton pump inhibitor and only test and treat when maintenance therapy needs to be prescribed.

Ref : 1. DeVault KR, Castell DO. Guidelines for the diagnosis and treatment of gastroesophageal reflux disease. Arch Intern Med 1995;155:2165–73.

Congratulations!

The Winners of GI café Quiz Competition

(November 2012 Volume : 5 Issue : 2)

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Editorial Note:

Dear Doctor, It's our immense pleasure to inform you that we have published the first issue, 2013 of GI Café. In this issue we try to focus on Guidelines for the Diagnosis and Treatment of Gastroesophageal Reflux Disease and Proton Pump Inhibitors and *Helicobacter pylori* Gastritis: Friends or Foes?. Your comments and suggestions will enrich our upcoming issues. Please participate in quiz competition and win prizes.