**INTRODUCTION**

Dyspepsia is defined as chronic or recurrent central upper abdominal pain or discomfort which is referable to the upper gastrointestinal tract \(^1\,^2\). It is usually associated with intake of food or hunger. Discomfort according to the Rome Working Teams refers to a subjective, negative feeling that does not reach the level of pain according to the patient. This can incorporate a variety of symptoms such as upper abdominal fullness, early satiety, bloating, belching or nausea \(^1\,^2\).

Dyspepsia is a common presentation in clinical practice worldwide \(^1\,^2\). It has a prevalence of between 20% and 40% in the adult population \(^3\,^4\). In a study carried out among the British population it was found to be 38%\(^5\). It is estimated to account for 2% to 5% of primary care office visits and 30% of consultations by Gastroenterologists\(^6\,^7\). A prevalence of 26% to 45% was found in some parts of Nigeria \(^8\,^9\). Dyspepsia has a significant impact on quality of life \(^10\), and results in enormous societal costs, either due to direct medical costs for physician visits, diagnostic tests, medications, or indirect costs from absenteeism or reduced productivity at work. It is therefore important to explore the management options available, especially in a resource poor setting like Nigeria, in the light of the foregoing.

**CLASSIFICATION OF DYSPEPSIA**

Dyspepsia can be broadly classified into two major groups. These include organic dyspepsia and functional dyspepsia.
**Organic dyspepsia:** This is dyspepsia that results from a structural or anatomical lesion. These structural lesions include chronic gastritis, duodenitis, gastric and duodenal erosions, gastric and duodenal ulcers, gastric adenocarcinoma and mucosal associated lymphoid tissue (MALT) lymphoma.\textsuperscript{13, 14} *Helicobacter pylori* infection has been noted to be associated with most of the disease entities presenting as dyspepsia.\textsuperscript{13, 14} The particular end result of *H. pylori* infection is determined by a complex interaction between bacterial, host and other environmental factors.\textsuperscript{13} A detailed description of this interaction is beyond the scope of this review.

**Functional dyspepsia:** This is dyspepsia in which there is no evidence of organic disease that can adequately explain the symptoms. It is also known as idiopathic or non-ulcer dyspepsia, and is often a diagnosis of exclusion. Many patients with functional dyspepsia (FD) have multiple somatic complaints, as well as symptoms of anxiety and depression.\textsuperscript{15} It is further subdivided clinically into ulcer-like, reflux-like, dysmotility-like, and non-specific dyspepsia.\textsuperscript{16} This sub-grouping, however, has not been found to be of much practical value in identifying the underlying cause of dyspepsia as the symptoms overlap considerably.

The pathophysiology of functional dyspepsia is poorly understood. There is symptom overlap with those of other functional gastrointestinal disorders, such as functional heartburn, irritable bowel syndrome (IBS), and non-cardiac chest pain.\textsuperscript{17} Like other functional gastrointestinal disorders, FD is best understood in the context of the bio-psychosocial model of illness in which symptoms arise out of a complex interaction between abnormal gastrointestinal physiology and psychosocial factors that affect how a person perceives, interprets, and responds to the altered gastrointestinal physiology.\textsuperscript{18} Several pathophysiological mechanisms that have been suggested as playing a part in its development include delayed gastric emptying,\textsuperscript{18, 19} impaired gastric accommodation,\textsuperscript{20, 21} myoelectric abnormalities,\textsuperscript{22, 23} altered antro-duodenal motility,\textsuperscript{24} visceral hypersensitivity,\textsuperscript{25} altered vagal function,\textsuperscript{26} altered duodenal sensitivity to lipids or acid,\textsuperscript{27, 28} and psychological disorders.\textsuperscript{29, 30}

**MANAGEMENT**

Several approaches that have been proposed for the management of a newly diagnosed patient with dyspepsia include:\textsuperscript{1, 31}

1. Empirical trial of acid suppression with antisecretory drugs like proton pump inhibitor (PPI) or Histamine 2 receptor blocker for 4-8 weeks
2. The “test and treat” approach for *H. pylori* infection using a validated non-invasive test and a trial of gastric acid suppression if eradication is successful but symptoms do not resolve, and
3. Initial upper gastrointestinal endoscopy (UGE) to determine the nature of the disease.

**Empirical trial of acid suppression**

This approach is recommended in populations with low prevalence of *H. Pylori* infection (<10%).\textsuperscript{1, 3} It is done using antisecretory drugs like proton pump inhibitor (PPI) or Histamine 2 receptor blocker for 4-8 weeks. If there is no amelioration of symptoms within 2-4 weeks of commencement of treatment, it is recommended that drug class be changed. Generally, PPIs have been found to be more effective than the H2RBs.

Although this approach is cheap, a major drawback to its use is the generally high prevalence of *H. pylori* infection in regions of the world with poor socio-economic condition.

**‘Test and treat’ method**

With the burden of evidence implicating *H. pylori* in the aetiology of different diseases manifesting clinically as dyspepsia, it will be appropriate for all patients with dyspepsia who are positive for *H. pylori* to undergo *H. pylori* eradication therapy.\textsuperscript{15, 3} More so, that *H. pylori* eradication has been associated with significant reduction in rate of recurrence of peptic ulcer disease and cure of MALT.\textsuperscript{13} When there are no ‘alarm symptoms’ the ‘test and treat’ method using multidrug therapy for *H. pylori* eradication is a rational approach, especially in populations with a moderate to high prevalence of *H. pylori* infection (≥10%) followed by a course of empirical antisecretory therapy in patients who fail to respond or relapse rapidly on stopping *H. pylori* eradication therapy.\textsuperscript{1, 33, 34}

The urea breath test and the stool antigen test are the recommended tests in non-invasive diagnosis of *H. Pylori* because of their high diagnostic accuracy.\textsuperscript{35, 36} Serological tests are not recommended because of their low discriminatory power between old and current infections. They cannot also be used to ascertain cure of infection.

One major drawback to this approach in developing countries like Nigeria is the rarity of these non-invasive diagnostic tests of choice.

**Initial upper gastrointestinal endoscopy**

There is controversy as to when UGE should be done considering the cost and the risks involved. Nevertheless, UGE is clearly indicated when a patient with dyspepsia presents with any of the following features: Presentation with a first episode of dyspepsia.
at >45 years of age (because of risk of malignancy), failure to respond to empirical anti-secretory therapy, and presence of alarm symptoms. 1, 32

Alarm symptoms include anorexia, weight loss, odynophagia, dysphagia, persistent vomiting, haematemesis, melaena, anaemia, unexplained weight loss (>10% body weight), a family history of gastrointestinal cancer, previous esophagogastric malignancy, lymphadenopathy, or an abdominal mass. 1

A careful history-taking, thorough physical examination and investigations such as abdominal ultrasound scan, barium studies, computer tomography and magnetic resonance imaging may be required for further characterization of disease in those who have alarm symptoms.

For younger patients who do not have alarm symptoms further diagnostic investigations are not usually required since upper gastrointestinal malignancy is rarely present in them, although the positive predictive value of alarm features remains very poor. 37

Endoscopy may also be required to reassure patients who are worried that a malignant condition may be responsible for their symptoms. 1 However, repeat endoscopy is not recommended once a diagnosis of non-ulcer dyspepsia has been clearly established in such patients, unless a completely new set symptoms or alarm features develop.

**Treatment of endoscopy-negative dyspepsia (functional dyspepsia)**

Endoscopy-proven functional dyspepsia is also treated with initial antisecretory therapy and *H. pylori* eradication just as organic dyspepsia. Management challenge arises when these measures fail because no other measure has been found to have optimal efficacy. 1

Simple reassurance as regards the benign nature of the illness may go a long way to ameliorate patients’ anxiety. Occasionally, a reconsideration of diagnosis may be needed in order not to miss other conditions that may mimic dyspepsia. Dietary therapy may help some individuals although it has no established efficacy.
Simethicone, low-dose tricyclic antidepressants and antispasmodics have all been used but there are very limited data supporting their efficacy.  

Limited studies support psychotherapy, hypnotherapy, and cognitive-behavioral therapy but they cannot be generally recommended for now.  

**Helicobacter pylori eradication therapy without initial diagnostic test**

This approach is usually the last result in resource poor regions of the word where diagnostic tests for *H. pylori* are not readily available or diagnostic tests for the infection are not cost-effective.  

The decision to treat is based on the assumption that *H. pylori* infection is present in patients with symptoms of dyspepsia since the prevalence of *H. pylori* is generally high in such settings.  

It is therefore better to treat the infection empirically than to do nothing because of patients’ inability to afford the cost of investigation, considering the immense benefits accruing to the dyspeptic patient following eradication of the organism.

The drawback to this approach, however, is that one cannot say with all certainty that the organism has been eradicated after treatment.

**CONCLUSION**

Considering the high cost of UGE and the high prevalence of *H. pylori* infection in developing countries like Nigeria, it seems reasonable that the ‘test and treat’ method using recommended non-invasive tests will be of immense usefulness in population sub-group who are less than 45 years of age without alarm symptoms, while those with alarm symptoms irrespective of age and those with onset of symptoms after 45 years will require initial upper gastrointestinal endoscopy.

It is highly desirable that the recommended non-invasive diagnostic tests for *H. pylori*, in addition to the existing gastrointestinal endoscopy facilities, are made available by policy makers. This will go a long way to improve the quality of care of patients, save cost of care and reduce the burden on the already overburdened Endoscopists and facilities for UGE in such populations.

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