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Dyspepsia -A Gastrointestinal Problem: A Review

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ABSTRACT

Dyspepsia is a common disease and it is common in the community. The aim of this review is to know the information regarding dyspepsia, and to assess the frequency of various causes of dyspepsia. Dyspepsia is the commonest gastrointestinal problem affecting at least 25% of the population during a year. Its prevalence varies in different countries, depending upon the prevalence of Helicobacter pylori infection, obesity, drug - alcohol - tobacco intake and spices in diet. Dyspepsia is common in different surveys in western societies have recorded prevalence between 23 and 41%. For many people dyspeptic symptoms are an unavoidable part of living. A minority of those sufferers who do consult can become major consumers of resource. About 4% of general practice consultations are for dyspepsia and 2% of the entire adult population receive either an endoscopy or barium meal each year. Time lost from work and interference with quality of life are more difficult to measure but are likely to be considerable.

Keywords: Dyspepsia, Helicobacter pylori, endoscopy, peptic ulcer

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INTRODUCTION

Dyspepsia is a Greek word meaning “duis” (bad or difficult) and “peptin” (to digest), which is described by patients as indigestion; both these words are a poor expression, as dyspepsia has no relation to digestion of food. Dyspepsia refers to upper abdominal symptoms usually following intake of food, which appear to arise from an abnormality in the upper gastrointestinal tract. Dyspepsia is a symptom and not a diagnosis. Symptoms may last for decades (even lifetime) and remissions and relapses are common. It is one of the commonest gastrointestinal malady affecting at least 25% of the population during a year. Its prevalence varies in different countries, depending upon the prevalence of *Helicobacter pylori* (*H. pylori*) infection, obesity, drug - alcohol - tobacco intake and spices in diet[1].

Dyspepsia is a group of symptoms referable to upper gastrointestinal tract rather than diagnosis itself. It consists of variable combination of symptoms of upper gastrointestinal tract including abdominal discomfort or pain, postprandial fullness, abdominal bloating, early satiety, nausea, vomiting, heartburn and acid regurgitation. There is a group of patients who do not have definite structural or biochemical cause for their symptoms, are considered for suffering from functional dyspepsia (FD). There are several pathophysiological correlates have been identified for functional dyspepsia, including gastro intestinal motor abnormalities, altered visceral sensation, central nervous system- enteral nervous system (CNS-ENS) integration dysfunctions and psychological [2,3].

NSAIDs, particularly aspirin, have a very useful part to play in the treatment of a variety of non-gastrointestinal conditions (eg, arthritis, and prevention of ischemic heart disease and stroke). Individual evaluation of the risks and benefits is required, preferably by the medical practitioner responsible for that person’s care, although team consultation with other practitioners involved may be necessary. Risk factors for GI complications are defined, and potential adverse effects of NSAIDs are described. Where there is an increased risk of NSAID-induced GI complications, or any adverse effects, NSAID treatment may be able to be stopped. However, if he benefit of continued treatment outweighs the risks, treatment can be continued, providing appropriate steps are taken as described in this Guideline, to minimisethe degree of risk. An alternative medication could also be considered. While this Guidelineprovides evidence-based advice on best management, it cannot replace the art of medicine required in the care of individuals [4].

Classification of Dyspepsia

a. Organic dyspepsia: erosive oesophagitis, gastric erosions,acute or chronic gastritis, gastric ulcer, duodenal ulcer, duodenitis, malignancy (carcinoma, lymphoma). Evidence of an organic disease is observed on upper gastrointestinal endoscopy (and gastric biopsy), or barium meal. It is suspected in presence of alarm symptoms (weight loss, anaemia, bleeding or occult blood positive, and loss of appetite) or symptoms occurring at night.

b. Functional or non-ulcer dyspepsia: A patient with anxiety,worry over serious illness (cancer) and/or experiencing adverse events recently, is likely to suffer from dyspepsia.No organic lesion is detected on investigations.

c. Drug related: aspirin, non-steroidal anti-inflammatory drugs (NSAID), antibiotics, bisphosphonates (alendronate),oestrogens, steroids, digoxin, chloroquine, potassium supplements, iron, etc. Detailed history of drug intake (present and recent past) should be recorded and rechecked.

d. Extra intestinal systemic diseases such as diabetes mellitus, hypothyroid, hyper-parathyroid, Addison's disease, uremia. Symptoms of endocrine diseases, are looked for after organic dyspepsia is excluded [5,6].

Symptoms

There is convincing evidence that a patients symptoms cannot be used to identify structural disease in uninvestigated dyspepsia. Working teams have suggested subdividing dyspepsia into ulcer-like or dysmotility-like dyspepsia based on symptom patterns or predominance; it was postulated that symptom subgroups could identify more homogenous populations that would respond to targeted medical therapy . However, individual symptoms, symptom subgroups, and scoring systems have all failed to be useful in identifying underlying peptic ulcer disease, or distinguishing organic from functional dyspepsia. A study from Canada reported that the patient's dominant symptom (including heartburn) failed to predict endoscopic findings in a primary care population .It is thus controversial whether subdividing dyspepsia into symptom subgroups aids management in documented functional dyspepsia. [7,8,9].

Causes of dyspepsia/ indigestion

Indigestion is usually related to the lifestyle and what we eat and drink. It may also be caused by infection or some other digestive conditions.

Common cause of the indigestion are as follows:

- Eating too much,
- Eating too rapidly,

- Consuming fatty or greasy foods,
- Consuming spicy food,
- Consuming too much caffeine,
- Consuming too much alcohol,
- Emotional trauma,
- Gallstones,
- Gastritis (inflammation of the stomach)
- Infection, especially with bacteria known as the Helicobacter pylori,
- Peptic ulcer,
- Drugs (NASIDs) [10]

Epidemiology of dyspepsia:

In the United States, the point prevalence is approximately 25%, excluding those people who have typical GERD symptoms. The prevalence is lower if patients with any symptoms of heartburn and regurgitation are excluded. The incidence is more poorly documented. In the United States, approximately 9%of people who had no symptoms of dyspepsia annually in the prior year reported new symptoms on follow-up; however, those with a past history of dyspepsia or peptic ulcer were not excluded and hence the onset-rate may be exaggerated. In Scandinavia, an incidence rate of less than 1% over 3 months has been reported. Whatever the incidence, the number of subjects who develop dyspepsia is matched by a similar number of subjects who lose their symptoms [7,11].

The un-investigated dyspeptic patient in primary care

About 40 % of dyspeptic patientsare investigated by endoscopy, the remainder being treated on a symptomatic basis. Resourcesfor healthcare are always limited and it is tooexpensive to investigate every patient whopresents with dyspepsia. Strategies have thereforebeen proposed that will adequately determine which patients should receive what therapy, while reducing unnecessary endoscopy workload, These include:

- empirical anti-secretary therapy/treat-and-endoscope
- early endoscopy
- screening for H. pylori
- empirical H. pylori eradication therapy for all dyspeptic patients.

Empirical anti-secretary therapy/ treat and endoscope)

Treating young (under the age of 45 years) dyspeptic patients with antacids, histamine (H2)-receptor antagonists or proton pump inhibitors (PPIs) and only investigating those that fail to

respond has been a popular method of reducing endoscopy workload. This strategy reserves costly investigations for those patients who are consuming more medication and, hence, might recover the cost of investigation by decreased prescribing. The finding that patients are prescribed less anti-secretary medication and have reduced consultation rates after a negative endoscopy suggests that this assumption may be false. In addition, persistent symptoms might be positively associated with serious pathology, although there is little evidence to support this. A further criticism of this strategy is that patients with PUD may receive intermittent anti-secretary drugs, responding promptly at each recurrence, even though *H. pylori* eradication is now the treatment of choice for this group. Nevertheless, empirical anti-secretory therapy or early endoscopy is the usual approach taken by GPs when initiallyinvestigating patients with dyspepsia[12].

Early endoscopy

There is empiric evidence from a management trial of prompt endoscopy in older patients that this is the strategy of first choice. Delaney *et al.* evaluated the cost-effectiveness of an initial endoscopy compared with usual management in patients with dyspepsia over the age of 50 presenting in primary care . A total of 422 patients were randomly assigned to either usual care or initial endoscopy; the initial endoscopy arm showed significant improvement in symptom scores and quality of life as well as a 48% reduction in the use of PPIs. Hence, initial endoscopy in older patients with dyspepsia at least in U.K. study was potentially cost-effective provided the cost of EGD was low. The cost-effectiveness of endoscopy in older people in the U.S. setting needs investigation [13].

Those Needing Early Identifying Endoscopy

In patients whose dyspepsia is assessed as originating in the UGI tract, further differentiation is required to identify high risk patients who will need endoscopy from those who can be safely managed without early endoscopy. It is reasonable to discuss early endoscopy with patients over 50 years of age with new-onset dyspepsia particularly if the symptom is progressively worsening. I Most individuals with alarm features will not have a serious problem but the vast majority of patients with upper gastrointestinal malignancy will have alarm features when they present for investigation. Serious consideration should be given to arranging prompt endoscopy for patients who have alarm features. The British Society of Gastroenterology recommends that if endoscopy cannot be provided promptly, a 2 to 4 week treatment period before investigation may be acceptableThe American Gastroenterology Association suggests that referral for early, endoscopy is indicated in older patients presenting with new onset dyspepsia.This is because the incidence of gastric cancer increases with advancing age. The threshold of 50 years of age was selected because

gastric cancer is very uncommon before that age. The American Gastroenterology Association further recommends that patients whose symptoms have failed to respond to empiric therapeutic approaches should undergo endoscopy[14].

Those Not Needing Early Endoscopy

Selected dyspepsia patients can be managed without referral or early endoscopy. These selected patients are under age 50 years and have symptoms that have been determined to originate in the UGI tract and are not accompanied by alarm features. Once the dyspepsia patients using NSAIDs/ASA and those with reflux-like symptoms have been separated for management (see Guideline for Treatment of Gastroesophageal Reflux Disease) the remainder will be a mixed group of patients. It will include some who have peptic ulcers and others with non-ulcer dyspepsia (NUD).

According to the 2005 Canadian GERD Consensus Report, not all patients with dyspepsia require a diagnostic endoscopy. Patients whose symptoms respond to PPI treatment and are characteristic of GERD may not need endoscopy until the duration of symptoms approaches 10 years. At this point, endoscopy is reasonable to search for Barrett's esophagitis.

Young dyspepsia patients who fail to improve with appropriate treatment such as 4 - 8 weeks of PPI or H2RA should have a careful reassessment that often includes endoscopy.The optimal management of a patient who presents with dyspepsia remains controversial. For many persons, the symptoms of dyspepsia are short-lived or of mild severity and are therefore self managed.Only 3-5% patients with dyspepsia have peptic ulcers and only a fraction have cancer,and almost all of these had alarm features on initial presentation [8].

Screening for H.pylori

Test for *H. pylori* in those with past history of peptic ulcer, family history of gastric cancer, or where the prevalence of *H. pylori* is >30%. Urea breath test is recommended. Serology can be used wherethe prevalence of *H. pylori* is >30%. Faecal antigen test is also recommended, and is becoming increasingly available in New Zealand. If testing is positive, treat with triple therapy.

Check eradication of *H. pylori* in those with a peptic ulcer complication, important comorbidity,factors, symptom recurrence or those living in isolated areas. Re-treat if testing is positive [12].

Test-and-Treat H. pylori Versus Placebo in Dyspepsia in the Community

There are data indicating a small benefit for treating *H. pylori*empirically in those with the infection in the community(non-patients). In a U.K. community trial, 32,929 individualswere invited and 8,455 attended and were eligible; 2,329were positive for *H. pylori* andwere assigned

active treatmentor placebo, with 1,773 (76%) returning at 2 yr. There was an absolute risk reduction of 5% for upper GI symptomson active therapy *versus* placebo, although quality of life wasunchanged. Presumably much of this benefit is explained bythe treatment of undiagnosed peptic ulcer disease [15.]

Empirical H. pylori eradication therapy for all dyspeptic patients

The simplest *H. pylori* management strategy of all would be to prescribe empirical *H. pylori* eradication therapy to all young dyspeptic patients. This would avoid the inconvenience and cost of testing for *H. pylori*, and a published model hassuggested this may be the most cost-effective strategy for managing dyspepsia. Empirical treatment was only slightly cheaper than the screening and treatment strategy, and resulted in 50–70% of young dyspeptic patients who were *H. pylori*-negative receiving antibiotics unnecessarily. Whether the increase in antibiotic exposure is worth this small cost saving needs to be addressed by prospective studies. Too few data are currently available to justify a review in this area but a comparative model will still be applied to highlight the crucial uncertainties [16].

ALARM FEATURES AND IDENTIFICATION OF STRUCTURAL

Disease in Uninvestigated Dyspepsia

The risk of malignancy increases with age and therefore empirical therapy is not currently recommended in individuals over 55 yr of age who develop new dyspeptic symptoms. Grade of evidence: C,New-onset dyspepsia in older age is an alarm feature or red flag. The American College of Physicians in 1985 published a guideline recommending that patients who were over theage of 45 deserved referral for prompt endoscopy to rule out underlying malignancy, as gastric cancer is very rare in the United States below the age of 45 yr although it increases thereafter. Some studies have reported that older age is an independent risk factor for identifying underlying structural [17].

Peptic ulcer

Gastric ulcer

By definition, a gastric ulcer occurs in the stomach and implies a break in the gastric lining that extends through the muscularis mucosa. The development of a gastric ulcer requiresthe presence of acid and pepsin, and in about 60 – 70% of cases, is also associated with the presence of the bacterium *H. pylori*. Most other gastric ulcers can be attributed to the ingestion of NSAIDs, including aspirin, Dyspepsia, nausea and vomiting are characteristic symptoms associated with gastric ulcer, particularly when dyspepsia occurs soon after the ingestion of meals. However, these symptoms have very poor sensitivity and specificity for gastroscopically confirmed gastric ulcer.

Complications (especially bleeding but also perforation and obstruction) can be life threatening, particularly in older people in whom comorbidity complicates medical management. Asymptomatic individuals (particularly those taking NSAIDs) who present for the first time with complications (eg, bleeding and perforations) may have peptic ulcers. Gastric ulcers must be differentiated from malignant ulcers with which they may occasionally coexist. Having a gastric ulcer increases the risk of gastric malignancy. Ordinary gastriculcers do not appear to progress into cancers. However, because gastric cancers can masquerade as, or coexist with, gastric ulcers, healing of gastric ulcers should be checked endoscopically and histologically. Most gastric ulcers can be treated medically, and surgery is usually reserved for a minority of those with complications. Duodenal ulcer ,these usually occur in the first 2 – 3 cm of the duodenum, just distal to the stomach, where the small intestine is exposed to gastric acid. The presence of acid is essential to the development of the ulcers, and about 95% of duodenal ulcers are also associated with the presence of *H. pylori*.In recent years, the proportion of *H. pylori*-positive duodenal ulcers appears to be declining in OECD countries. This has been associated with a decreased prevalence of *H. pylori* infection and possibly increased ingestion of NSAIDs. Duodenal ulcers can occur at any age, but are more frequent from the late 30s onwards, generally occurring two decades earlier than in people who have gastric ulcers. Individuals typically experience food-related dyspeptic symptoms (pain that occurs a few hours after a meal, is relieved by the ingestion of food or antacids, and/or may interrupt sleep). However, duodenal ulcers often occur in the absence of typical symptoms. The commonest complication is bleeding. Upper GI hemorrhage has an incidence of 100 per 100,000 in the USA and Britain; peptic ulcers are the commonest cause of this bleeding in both countries. A small proportion of duodenal ulcers may perforate, or heal with scarring (resulting in upper GI obstruction), although both of these complications are now extremely rare. Duodenal ulcers are not precancerous; indeed, gastric cancers are less common in people with duodenal ulcers. The vast majority of duodenal ulcers (over 95%) can be healed. with appropriate medical treatment, including acid inhibition &*H. pylori* eradication [18].

NSAID-Induced Ulcers

NSAID-induced ulcers may develop in otherwise histologically normal mucosa and are likely to be associated with other damage, such as erosions and mucosal haemorrhage. In contrast, *H. pylori*-related ulcers are associated with acute or chronic inflammation and the presence of *H. pylori* organisms. NSAID-induced ulcers are more likely to be gastric than duodenal and are often asymptomatic until complications occur**.** NSAID-related peptic ulcers and their complications are more common in people infected with *H. pylori* (see Chapter 4: *Helicobacter Pylori and Peptic*

Ulceration).The spectrum of NSAID-related gastroduodenal injury includes a combination of subepithelial hemorrhages, erosions and ulcerations, often referred to as NSAID gastropathy.

The distinction between erosions and ulcerations depends on pathological definitions: ulcersare defined as lesions that penetrate to the level of the submucosa, and erosions as lesions confined to the mucosa. For practical purposes, an OGD definition that is based on a subjective assessment of size, shape and depth of the lesion is used. Erosions are likely to be small and superficial, while ulcers tend to be larger (>5 mm in diameter) anddeeper.9The clinical significance of OGD findings remains unclear. It has been suggested that an ulcer complication cannot occur without the presence of an ulcer. However, in up to 50% of serious GI haemorrhage cases, an active ulcer cannot be seen with OGD. endoscopically verified ulcers can be documented in up to 40% of chronic NSAID users,but it is estimated that as many as 85% of these never become clinically apparent.Conversely, common side effects, such as nausea and dyspepsia [19].

RECOMMENDED MEDICATION FOR PROTON PUMP INHIBITORS

Table 1: Medicine and dose to treat dyspepsia/ indigestion 12

	Half dose	**Standard dose**	**Double dose**
Lansoprazole	15 mg/day	30 mg/day	60 mg/day OR 30 mg BD
Omeprazole	10 mg/day	20 mg/day	40 mg/day OR 20 mg BD
Pantoprazole	20 mg/day	40 mg/day	80 mg/day OR 40 mg BD

H2-receptor antagonists

Table 2: Medicine& dose to treat from peptic ulcer and GORD[12].

	PEPTIC ULCER		GORD	
	Treatment	Maintenance	Treatment	Maintenance
Famotidine	40 mg nocte OR 20 mg BD	20 mg BD	40 mg BD	20 mg BD
Ranitidine	300 mg nocte OR 150 mg BD	150 mg BD	150-300mg BD	150 mg BD

Diagnosis of dyspepsia

- **Physical examination**

1. General appearance: Fat in gall-bladderdyspepsia: nervous dyspeptic usually thin: polar in pernicious anemia,

2. Dental caries: conditions of tongue,

3. Abdomen: peristalsis, localized tenderness, palpable mass, enlargement of liver,etc.

4. Examination of heart and lungs if cough or dyspnoea.

5. Wasting in anorexia nervosa, addison's disease, tuberculosis,

- **Radiological Examination**

Diagnosis of peptic ulcer, carcinoma stomach, achlasia chronic appendicitis, regional ileities, hiatus hernia, gall stone,

- **Laboratory investigation**

a) Gastric analysis,
b) Stool examination for cyst, ova, incomplete digestion, occult blood .
c) Urine examination and renal function tests,
d) Red cell count, haemoglobin and haematocritestimation.
e) Absorption studies of small-bowel function.
f) Liver function tests
g) Needle biopsy of the liver,
h) Pancreatic function tests
i) Test of endocrine disorder- such as diabetes mellitus; addison's disease, hyperthyroidism or hyperparathyrodism.

- **Investigation by instruments**

  Endoscopy, colonoscopy, ultrasound, and laparoscopy.

- **Exploratory laparotomy**

  If a definite diagnosis is not possible after all other investigations are carried out.

- **Blood test-** If patient has any symptoms of anaemia the doctor may order a blood test
- **Test of Helicobacter pylori infection**

  This may include a urine breath test,a stool antigen test, and a blood test.peptic ulcer are often caused by this bacterium.

- **Liver function tests**

  if the doctor thinks the patient may have a biliary condition, which affects the bile ducts in the liver. This involves a blood test that determine how the liver is working.

- **X-ray**

  Usually an upper gastrointestinal and small bowel series. X-ray are taken of the esophagus, stomach and small intestine.

- **Abdominal ultrasound**

High-frequency sound waves makes images that show movement, structure and blood flow. A gel is applied to the patients abdomen and a hand –held device is pressed against the skin , this device emits sound waves and the doctor can see the inside of the abdomen in detail on a monitor [10].

Treatment for dyspepsia/ Indigestion

- ✓ **Diet and life style changes-**
- ✓ if symptoms are mild and your indigestion is not occurring often,some lifestyle changes will probably ease symptoms,This usually involves consuming less fatty foods less caffeine alcohol.
- ✓ **H2-receptor antagonists**

The development of H2-receptor antagonists inthe 1970s revolutionized the treatment. These drugs reduce pentagastrin-stimulated acidoutput by 30–50%. They are particularly effective at reducing nocturnal acid secretion, althoughtheir effect on meal-stimulated acid output is more modest. RCTs have shown that H2-receptor antagonists are significantly superior to placebo in treating duodenal ulcer disease, with 80% of lesions healed at 4 weeks. H2-receptor antagonists are less effective in patients with gastrooesophageal reflux disease (GORD) but are still superior to placebo. Cimetidine was the first H2-receptor antagonist to be developed and is the cheapest drug in this class. The main disadvantage of cimetidine is that it competitively displaces dihydrotestosterone from androgen binding sites, occasionally leading to gynaecomastia in men.The newer H2-receptor antagonists, ranitidine, nizatidine and famotidine, are more potent inhibitors of acid secretion ona weight basis and do not have anti-androgenic side-effects.41 They are, however, moreexpensive than cimetidine 10.

- ✓ **Drug interaction**

Warfarin ,theophylin ,phenytoin,lindocaine,quinidine,metoprolo,tricyclic antidepressants,

Syrup used in dyspepsia

- ***Aluminium hydoxide and magnesium hydoxide***

  Which contain antacid for preventing ulcer,heart burn relief,and stomach upset these are the syrup neutralizes the acid in the stomach.

  Trade name: Aludrox(200ml),

- ***Calcium carbonate***

  This is a dietarysupplement prescribed for calcium deficiency state which may occur in disease such as decreased level of parathyroid osteomalacia.also used as an antacid.

- ***Omeprazole and domeperidone*****:** This drugs contain the proton pump inhibitor (PPI), anti-dopaminergic agent, prescribed for the ulcers , indigestion, stomach acid.

Complication on indigestion:

In the vast majority of cases indigestion is mild and does not happen frequently. Severe indigestion can occasionally cause the following complication:

- ***Esophageal stricture***:

  If the indigestion is caused by acid reflux ,when the stomach acid leak back up into the esophagus and irritate the mucosa, the esophagus can become scarred, the esophagus can eventually become narrow and constricted. Patient may suffer from swallowing difficulties; food can get stuck in the throat,causing chest pain.

- ***Pyloric stenosis***:

  This is caused by long term of the lining of the digestive system for stomach acid,the pylorus-the passage between the stomach and the small intestine become scarred and narrow,food is not properly digest.

- ***Peritonitis*** :

  Inflammation of the peritoneum (the tissue layer of cell lining the inner wall of the abdomen and pelvis) 10.

Dyspepsia sub-groups

Reflux- like Heartburn - acid regurgitation

Ulcer- like Upper abdominal pain predominant with three or more of:

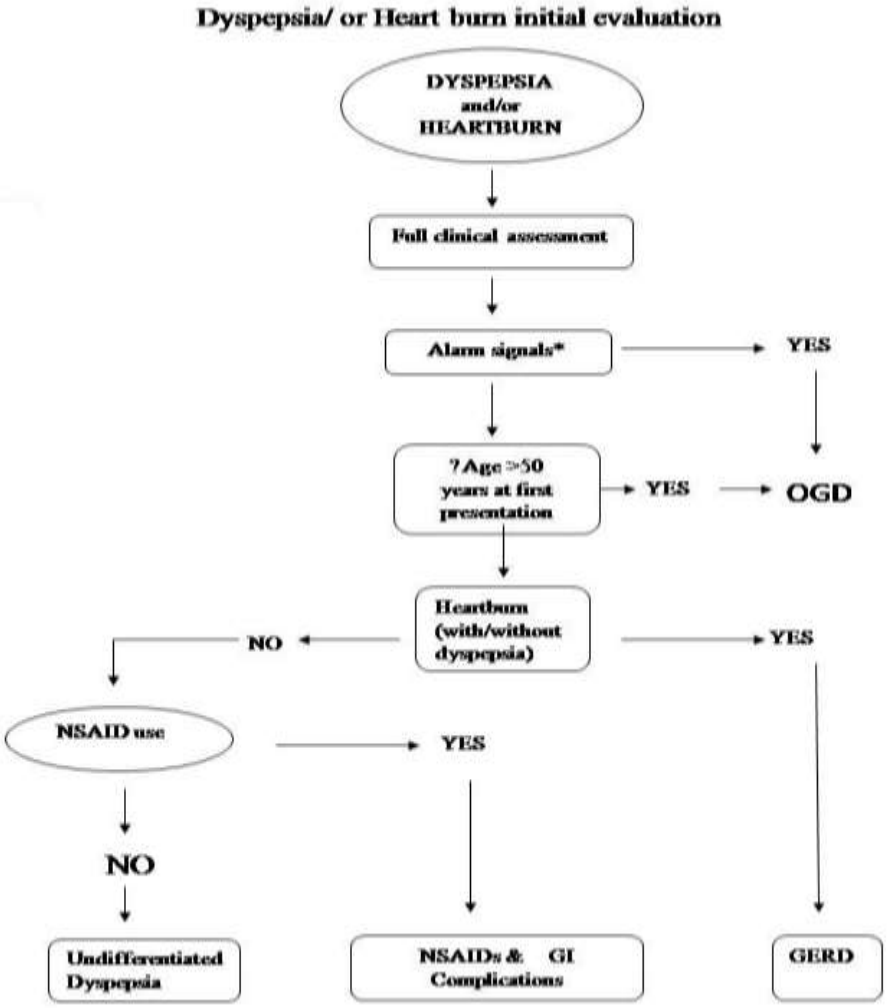
- epigastria pain or discomfort
- pain relieved by food
- pain relieved by antacids or ulcer-reducing drugs
- pain occurring before meals or when hungry
- pain that at times wakens the person from sleep
- periodic pain with remission and relapse.

Dysmotility-like Upper abdominal discomfort characterized by three or more of:

- early satiety
- post-prandial fullness
- nausea
- retching and/or vomiting
- bloating in upper abdomen not accompanied by visible distension
- upper abdominal discomfort often aggravated by food.

Unspecified

(non-specific)-Dyspepsia that cannot be classified into the other groups 4.



CONCLUSION

This review article will aware us about the dyspepsia. Once a diagnosis of functional dyspepsia is confirmed by a negative endoscopy, an empiric trial of therapy is commonly prescribed. However, the benefits of all therapies in this condition have been questioned. Many patients do not require medication for dyspepsia after they have had reassurance and education. It is therefore important for the clinician to explain the meaning of the symptoms and their benign nature. Ascertaining why a patient with long-standing symptoms has presented on this occasion for care can be helpful, as this may identify those who have fears of an underlying serious disease or specific psychological distress that can be addressed. Potential precipitating factors in dyspepsia remain poorly defined. High-fat meals should be avoided; eating frequent and smaller meals throughout the day can sometimes be helpful. Specific foods that precipitate symptoms can be avoided. Food intolerance is uncommon, however, and food allergy very rare. Follow-up of the patient helps determine the natural history and allows further correction of faulty ideas and provides reassurance that can be very helpful in long-term management. Antacids and sucralfate were not superior to placebo in

functional dyspepsia based on a Cochrane review. However,a recent trial of simethicone has suggested potential benefit compared with placebo, and in another study equivalence with cisaprid. In contrast, another recent trial reported significant benefit with lansoprazole in a U.S. population. *H. pylori* status is unlikely to affect the therapeutic outcome of acid suppression therapy in functional dyspepsia. Large trials have failed to identify any difference in therapeutic outcome in *H. pylori*-positive *versus* negative patients, although. Patients with functional dyspepsia should be reassured that they do not have serious illness after minimum investigations. Drugs, excessive tea, alcohol, tobacco ingestion, smoking, causing gastritis are omitted. Dietetic instructions such as small frequent meal, less water with meals, omission of chillies, will help. A physician-patient relationship determines the success rate. Besides the severity and persistence of dyspepsia, many other factors (including psychosocial issues) are important in determining who seeks medical consultation, although these factors are not well understood. Studies such as a postal survey conducted by Jones *et al*,1 have shown that only about 25% of people with dyspepsia actually seek medical advice. Nevertheless, because dyspepsia symptoms are so common, this accounts for between 3and 10% of the adult population and for between 2 and 7% of visits to general practitioners. In general, people with functional dyspepsia score higher on measures of anxiety, neuroticism, depression and hypochondriasis compared with healthy controls. These personality profiles are similar to people with other pain syndromes, both organic and functional in nature. Incompared 100 people with functional dyspepsia,100 with duodenal ulcer, and 100 controls, and found that those with functional dyspepsia had more anxiety and depression, and a lower general level of functioning than people in the other two groups. In addition, those with functional dyspepsia had more frequent dyspepsia symptoms and longer duration of symptoms than those with duodenal ulcer. However, it was more difficult to recruit people with functional dyspepsia, which may have biased this group towards those with more severe symptoms. Interestingly, quality-of-life scores (as judged by total symptoms) improve with improvement of dyspepsia, suggesting that anxiety and stress-related symptoms may be the result of dyspepsia rather than its cause. The quality of life of people with GORD has been shown to be similar to that of people with severe angina pectoris.

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