UNIT 1 DISORDERS OF UPPER GASTROINTESTINAL TRACT

Structure

1.0 OBJECTIVES

After reading this unit, you should be able to:

- describe the common upper gastrointestinal disorders in the elderly;
- distinguish clinically between these common upper gastrointestinal disorders;
- treat the clinical problems; and
- institute preventive measures wherever possible.

1.1 INTRODUCTION

You are aware that these days very advanced investigative techniques are available for correct diagnosis of upper GIT symptoms commonly seen in old age. Yet, you know that a good clinical history always has an important place in the diagnosis. Symptoms which you cannot explain by structural or bio-chemical abnormalities, are labelled, as functional. You must

have seen these in your subjects, but you have to be careful in applying the results of these in older subjects in whom disease presentation is less specific. You should be careful and avoid tendencies to attribute symptoms to old age.

In this unit, you will learn about common upper gastrointestinal disorders, how to diagnose and treat them. This unit will also give you information regarding the pathogenesis of these disorders, thus helping you to know what preventive measures can be instituted or complications that arise from these disorders.

1.2 NORMAL AGE RELATED CHANGES IN UPPER GIT

You have already learnt from Unit-1, Block-2 of Course 1 about the age related changes in gastrointestinal tract in brief. In this unit, let us revise important process involved in the highly coordinated event of swallowing. Swallowing is transecting of ingested food from mouth to stomach after mixing it with saliva from the mouth. For the purpose of learning, swallowing can be divided into four consecutive steps to understand the programmed sequence of muscular events of which upper oesophageal sphincter (U.S.) relaxation is only one step.

Preparation: During this stage food bolus is in oral cavity where chewing and mixing with saliva changes it physically and chemically. These changes are preparatory for further transit.

Oral phase: During this phase, food is propelled by sequential squeezing of the tongue against hard and soft palates. This starts a peristalic pressure wave that propels the food bolus into the pharynx from the oral cavity.

Pharyngeal phase: During this phase, nasopharynx constricts, the soft palate is elevated. The thyroid bone and larynx are elevated protecting the laryngeal orifice. Out of the four routes connected to the pharynx, three are shut off i.e., nasal, oral and laryngeal. When larynx is fully elevated (1-3cm) the Upper Oesophageal Sphincter relaxes and pharyngeal peristalsis propels food into the oesophagus. Upper Oesophageal Sphincter subsequents contracts and descends to its original position. This process and sequence of events is under control of swallowing centre in the brain stem. Afferent impulses from the oral cavity and pharynx and size of bolus etc. influence the centre.

Dysphagia: It is a swallowing disorders in geriatric age group. A number of changes occur which worsen swallowing with age. Some of these changes are a direct effect of aging process, but many others are either from the disease process that occur more often in elderly or due to medication used to treat various medical conditions.

In geriatric age changes occur in all three phases of swallowing.

Oral phase: In elderly person number of changes occur like increase in connective tissue in tongue, loss of dentition, reduced masticatory strength, which interfere with oral preparatory phase of swallowing leading to poorly formed food bolus.

Pharyngeal phase: There is delay in the initiation and decrease in duration of pharyngeal swallow along with decreased opening duration of cricopharyngeous. Thus the coupling between oral and pharyngeal phase of swallowing is disturbed leading to dysphagia and aspiration.

Oeseophageal phase: With age there is decrease in number of cells in Auerbach's plexus and a decrease in peristaltic amplitude. Primary peristalsis remains unchanged but secondary peristalsis is less frequent or absent.

1.3 DYSPHAGIA

The prevalence of dysphagia increases with age. Most studies abroad have shown 50-60% prevalence in nursing homes and 30% in general medical wards.Indian literature on this subject is meagre.

Dysphagia could be oropharyngeal or oesophageal. A good clinical history will help to diagnose the type of dysphagia. This will help you in selecting further diagnostic aids.

Videofluroscopy can reveal some change in upto 84% of asymptomatic elderly—out of these about 1/3rd are due to Upper Oesophageal Sphincter dysfunction. It is advisable to exclude treatable pathology and not consider the problem as normal aging.

1.3.1 Oropharyngeal Dysphagia

Oropharyngeal dysphagia is characterised by difficulty in transferring food bolus from the oral cavity past the pharynx to the upper oesophagus.

The patient usually presents with the following symptoms:

- Difficulty in gathering food bolus in the back of tongue.
- Difficulty in transferring food from oral cavity to back of throat and food pipe.
- Inability to propel food into food pipe.
- Food sticks in throat.
- Nasal regurgitation.
- Coughing in voice after meal.
- Repeated pneumonia.

Oropharyngeal dysphagia can be caused by a variety of neruological, muscular and structural disorders as shown in Table 1.1.

Table 1.1: Causes of Oropharyngeal Dysphagia

1) **Medical and surgical lesions of peripheral and central nervous system**
   - Cerebrovascular accidents
   - Multiple sclerosis
   - Parkinson's disease
   - Central Nervous System tumour
   - Amyotrophic lateral sclerosis
   - Bulbar poliomyelitis
   - Peripheral neuropathies
   - Alzheimer's disease
   - Head injury.

2) **Muscular or Neuromuscular causes**
   - Myasthemia gravis
   - Muscular dystrophies
   - Endocrine myopathy
   - Inflammatory muscle disease
   - Alcoholic myopathy.

3) **Structural Lesions**
   - Carcinoma
   - Postcricoid welis
   - Anterior cervical osteophytes
   - Scarring after operation or radiation or burn
   - Inflammatory conditions
   - Abnormal relaxation of U.E.S.
   - Abnormal opening of U.E.S.
   - Zenkers diverticuum.

4) **Pharmacological therapy**
   - Anticholinergic
   - Antihistamines
   - Phenothiazines.

1.3.2 Oesophageal Dysphagia

There are two oesophageal sphincters — Upper oesophageal sphincter and lower oesophageal sphincter.

The function of the upper oesophageal sphincter (U.O.S.) is to prevent air swallowing in between meals. It is made up of striated muscle while the lower sphincter (LOS) is made up of smooth muscle and its function is to limit reflux of acid gastric contents to the oesophagus. It offers some resistance to the food bolus which is overcome by effective peristalsis.

Common causes of Oesophageal Dysphagia are shown in Table 1.2.

Table 1.2: Common Causes of Oesophageal Dysphagia in Elderly Patients

Disorders of Motility	1) Achalasia	**Mechanism** Absence of peristalsis Failure of LOS to relax Obstruction and dilatation. Result:Dysfunctional oesophagus
	2) Scleroderma	Ineffective oesophageal motility Weak peristaltic waves and food stops in oesophagus. Result is ineffective motility of oesophagus.
	3) Diffuse Oesophageal Spasm	Increased number of simultaneous Contractions which are weak in amplitude.
	4) Hypertensive LOS	Incomplete relaxation of sphincters.
Mechanical lesions	1) Carcinoma	Cancer of oesophagus Cardia, lymphoma, paraneoplastic tumour. infiltrate the sphincter.
	2) Stricture	Medications Potassium supplement quinidine Lesions after therapy-tetracycline iron supplement
	3) Lower oesophageal webs	
	4) Oesophagitis	
	5) Diverticula	
	6) Compression from outside	Abnormal right subclavian artery or annular aorta.
Neurological Cause	Parkinson's disease	
	Multiple Sclerois	
	Myasthenia Granis	Weakness of muscles
	Amyotrphic Lateral Sclerosis	

1.3.3 Achalasia Cardia

Achalasia is motor-disturbance of the oesophagus characterised by a poorly relaxing lower oesophageal sphincter. Failure of the sphincter to relax produces a functional obstruction of the esophagus.

Aetiology

The cause of achalasia remains unknown. A viral cause has been postulated but epidemiologic data does not support it.Genetic factors are also not involved as is suggested by the occurrence of achalasia in only one of a pair of monozygotic twins.

Pathology

The disease involves abnormalities in both muscle and nerve components. A decrease in the number of ganglion cells in the intramural oesophageal nerve plexus has been observed.

Lewy bodies, characteristics of Parkinson's disease have also been noticed in some patients. Degenerative changes in the oesophageal branches of the vagus and in the dorsal motor nucleus of the vagus in the brainstem have also been noted. The circular muscle of the lower oesophagus is thickened, but muscular changes are thought to be secondary to the underlying neuronal changes.

Clinical Presentation

The duration of symptoms at presentation averages 2 years. Dysphagia is the predominant symptom. Chest pain occurs in 1/3 to 1/2 of patients and improves with time, is retrosternal and is after precipitated by eating. Regurgitation, weight loss and heart burn are other symptoms. Pulmonary symptoms may occur due to aspiration of oesophageal contents.

Diagnosis and Investigation

A detailed history usually gives sufficient clues towards the presence of the disease. The investigations of value are given below:

1) ***Radiographic Studies*** are the primary screening tests. A Barium swallow with fluoroscopy is the appropriate radiographic investigation and confirmatory of the disease. The detailed oesophagus ends in a pointed 'beak' that represents the non- relaxing lower oesophageal sphincter. Fluoroscopy during the swallow reveal no meaningful peristalsis in the oesophageal body.

2) ***Endoscopy:*** Typical endoscopic findings include dilatation and atony of the oesophageal body and a punctured, closed lower sphincter that does not open during the procedure.

3) ***Manometry:*** It is practically important when radiographs are normal or inconclusive. It confirms or establishes the diagnosis of achalasia. Three differences in the LES characteristic are often observed in achalasia: resting pressure is elevated, the length of the high pressure zone is greater and intraoesophageal pressure exceeds intra- gastric pressure in patients with achalasia.

Management

Treatment is directed at palliation of symptoms and prevention of complications

Three pallitative procedures are available to reduce lower sphincter pressure and improve emptying by gravity. These palliative procedures must be adjusted only when proper facilities are available generally in hospitals.

Pharmacotherapy: Oesophageal sphincter relaxation can be achieved by some drugs. These include nitrates (amyl nitrate) and calcium channel blockers (nifedipine).

Dilation: Bougienage with large caliber dilators produces transient improvement. Forceful dilation to a diameter of approximately 3 cm is necessary to tear the circular muscle and effect lasting reduction in lower sphincter pressure. Good response occurs in at least 60% of patients. The response rate varies with patient age (older patients respond better) and duration of symptoms (a longer history is associated with a better response).

Eosophagomyotomy: A modification in the original heller procedure is employed most commonly in the surgical management of achalasia. An anterior myotomy is performed by dividing the circular muscle fibres down to a level of the mucosa. Good results from myotomy ocuur in 80-90 % or more of the patients. The most significant complication is gastroesophageal reflux.

Check Your Progress 1

1) How does achalasia produce oesophageal obstruction?

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2) The main symptom of achalasia is:

   a) Heart burn

   b) Chest pain

   c) Dysphagia

   d) Weight loss

3) What is the confirmatory test for diagnosing achalasia?

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4) Describe the treatment options available for achalasia cardia?

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1.3.4 Diagnosis of Dysphagia

1) **History :** History is very important as to whether the dysphagia is gradual or sudden in onset, it is for solid or liquid, associated with coughing (aspiration), speech defects or

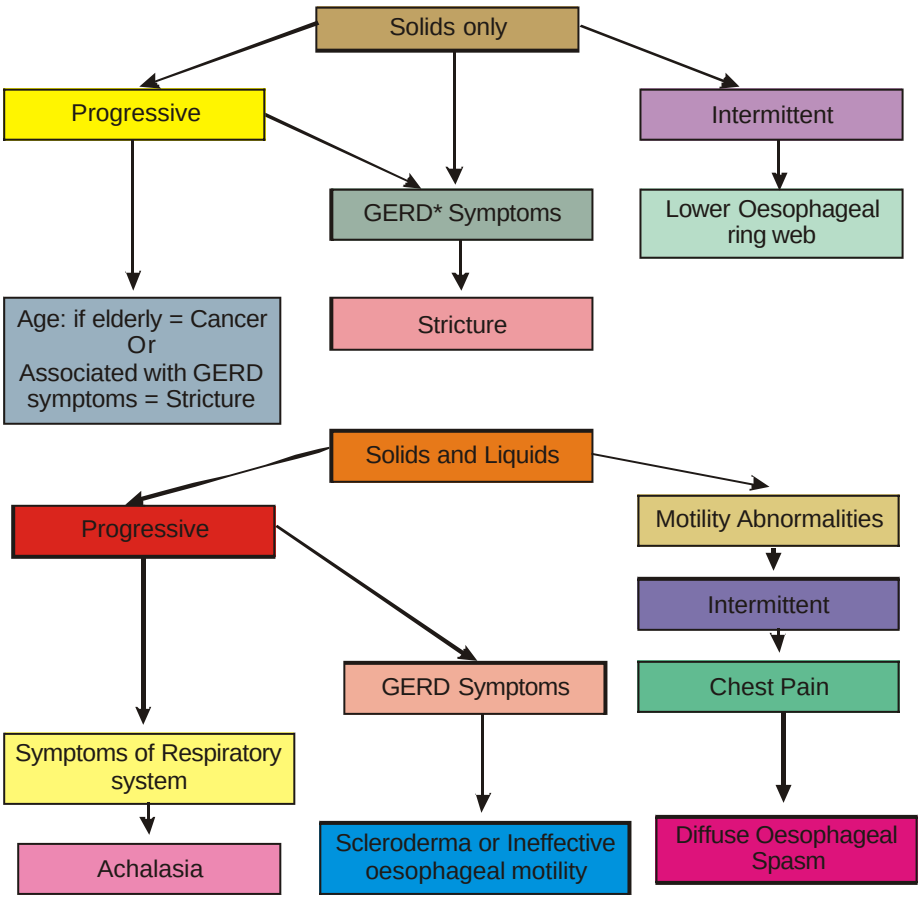

Fig. 1.1: Diagnosis of dysphagia

* GERD - Gastroesophageal reflux disease

other neurological defects. In obstructive lesion to begin with dysphagia is for solids and gradually progresses to liquid also, contrary to neurological dysphagia which is for liquids primarily and is associated with aspiration and nasal regurgitation while solids can be swallowed comparatively easily.

2) **Radiological Examination :** X-ray Soft tissue neck and barium swallow.

3) **Endoscopic :** Examination of oesophagus should be done.

Useful investigations are:

- Video radiographic study
- Larynxgoscopic examination
- Barium swallow
- Upper GIT endoscopy

In some cases, manometric study of pharynx and oesophagus may have to be undertaken. If needed after neurological examination, a computerised tomography or magnetic resonance imaging scans can be useful.Thyroid evaluation cannot be overlooked in the elderly.

1.3.5 Management

In elderly patient, with short history, it is important to rule out malignant tumours so that early management can give better results.

When certain diseases are causing dysphagia, specific therapy is indicated for example, in case of Parkinson's disease with dysphagia. In hypothyroidism again substitution therapy is indicated.

In sphincter abnormalities, to improve dysphagia, dilations can be carried out in specialised centers to improve dysphagia. The same is true for Myotomy of U.O.S. Patients learn adjustments in diet and eating habits with regard to quantity, texture and quality of food.

Oesophageal dysphagia is treated effectively with antisecretory and prokinetic medications.

Primary treatment for achalasia is dilations under guidance of specialists.

Diffuse oesophageal spasm is treated by drugs like calcium channel antagonists. Botulinum toxin injection has been used for hypertensive LES. Finally, help of speech therapist may be useful in teaching patients.Sample measures to improve swallowing and reduce aspiration. These are usuals posture modification during meals and changes in consistency and quality of food.

1.4 GASTROESOPHAGEAL REFLUX DISEASE IN ELDERLY (GERD)

Entry of gastric contents into oesophagus is called Gastroesophageal Reflux. When it produces clinical symptoms or histological changes, it is called gastroesophageal reflux disease and it is a very common and challenging disease of upper GIT in the elderly. In the elderly, it is more common to find evidence of endoscopic and pathological findings of advanced disease. GERD is more common in elderly because the intra abdominal segment of lower oesophageal sphincter is shorter. Secondary peristalsis is reduced and the number of tertiary contractions increase. There is also relaxation of the tone of oesophageal sphincter. In these patients, the co-existing disease and multiple medicine therapy. Many atypical symptoms (chest pain, cough, laryngitis) present challenges in diagnosis and treatment.

True prevalance is difficult to estimate in elderly because of atypical presentation in some subjects. Most studies indicate about 14-15% of patients over 65 years of age have weekly heartburn.

1.4.1 Pathophysiology

Pathophysiology revolves round factors protecting oesophageal mucosa and those causing damage. For example, prolonged contact with higher acid contents of stomach. Factors contributing to reflux disease are shown diagrammatically in Fig 1.2.

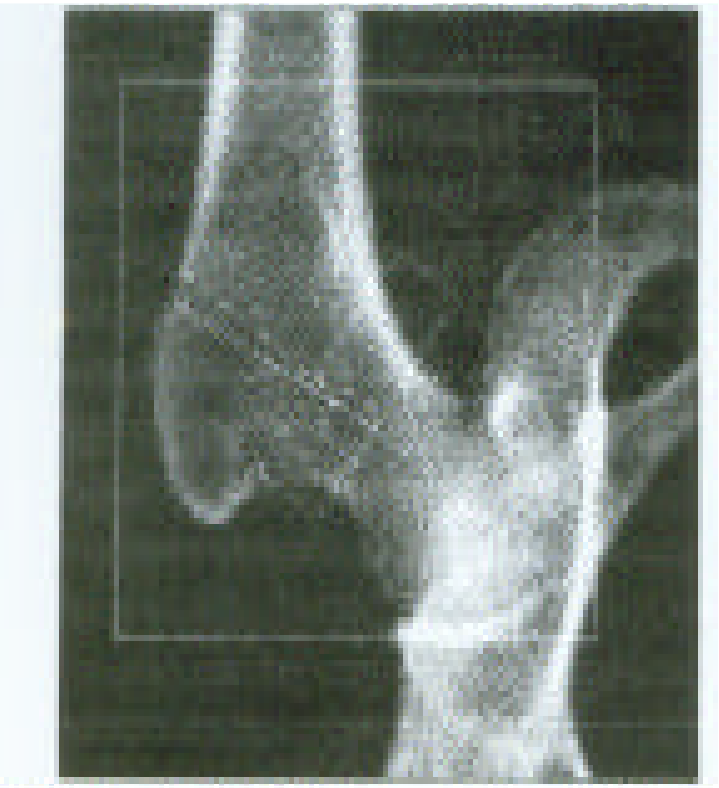

Fig. 1.2: Pathophysiology of GERD syndrome

1.4.2 Clinical Presentation

Elderly patients with GERD present with a variety of symptoms. These are shown in Table 1.3. The classic GERD symptom is heartburn after meal or exercise in reclining or bending position. With aging and decline in oesophageal sensitivity, the symptoms are less severe in elderly. Other symptoms are acid regurgitation, water brash, belching and nausea.

Table 1.3: Symptoms of GERD

Typical	Atypical	Alarm Symptoms
Heart burn	Unexplained chest pain	Dysphagia
Regurgitation	Asthma	Weight loss
	Cough	Gastrointestinal bleeding
	Wheezing	Anaemia
	Hoarseness	
	Laryngitis	
	Hiccup	
	Nausea	
	Globus sensation	
	Aspiration Pneumonia	

Diagnosis

The classic history of heart burn and acid regurgitation is suggestive of diagnosis. Other tests are oesophageal pH testing endoscopy, radiologic investigations, barium studies and therapeutic tests. All these need appropriate settings and available in special centres. Early endoscopic evaluation is important in the elderly who have more severe disease even with mild or atypical or alarming symptoms.

1.4.3 Complications of GERD

Complications are more common in elderly and are:

a) Oesophagitis with erosions and ulcers in the oesophagus

b) Peptic stricture resulting from oedema & scar tissue in distal oesophagus, usually are associated with hiatus hernia.

c) Barretts Oesophagus when normal squamous cell epithelium is replaced by intestinal metaplasia. These changes are precursors of adenocarcinoma and endoscopic surveillance is important.

1.4.4 Management

GERD is chronic and recurrent in majority. There are three broad steps in the treatment.

Non Pharmacological Treatment

Life style modifications like raising the head end of bed by six inches (not by pillows which raise the head only). Some benefit by sleeping on left side. Patients should be advised to avoid smoking, alcohol and heavy meals at least 3 hours before sleep. S/he should be advised to avoid citrus juices, coffee, chocolate, tomato products, fatty meals.

Pharmacological Therapy

Available H_2 blockers are used and symptoms are relieved in 6-12 weeks.

Cimetidine	400 mg BID	All to be taken before breakfast and before dinner
Tamotidine	20 mg BID	
Ranitidine	150 mg BID	
Hizatidine	150 mg BID	

Eighty per cent relapse with six months of ceasation of treatment and maintenance therapy is needed. More effective treatment of GERD, today is with proton pump inhibitors acting on enzyme needed in final step of parietal cell secretion of acid. These are used in once daily dose as

Omeprazole	20 mg
Lanoprazole	30 mg
Rabeprazole	20 mg
Pantoprazole	40 mg

Use of prokinetic agents like cisapride is not to be addressed without gastroenterologists supervision.

Surgery

Now Anti reflux surgery can be done by laparoscopy but careful selection of patients is needed and consultation with gastroenterologist is very essential.

1.5 PEPTIC ULCER DISEASE

Peptic ulcer are defects in gastrointestinal mucosa extending through the muscularis mucosae that persists as a function of the acid peptic activity in the gastric juice.

1.5.1 Etiology and Pathophysiology

The regulation of acid pepsin secretion reflects an intricate balance of input from neural, endocrine, paracrine and autocrine pathways and involves both stimulatory and inhibitory mechanisms. Likely, several mechanisms underline the ability of normal gastroduodenal mucosa to defend itself against injury from the acid peptic activity in gastric juice and to repair injury rapidly when it does occur. Ulcers are formed only when these repair and healing mechanism fail .

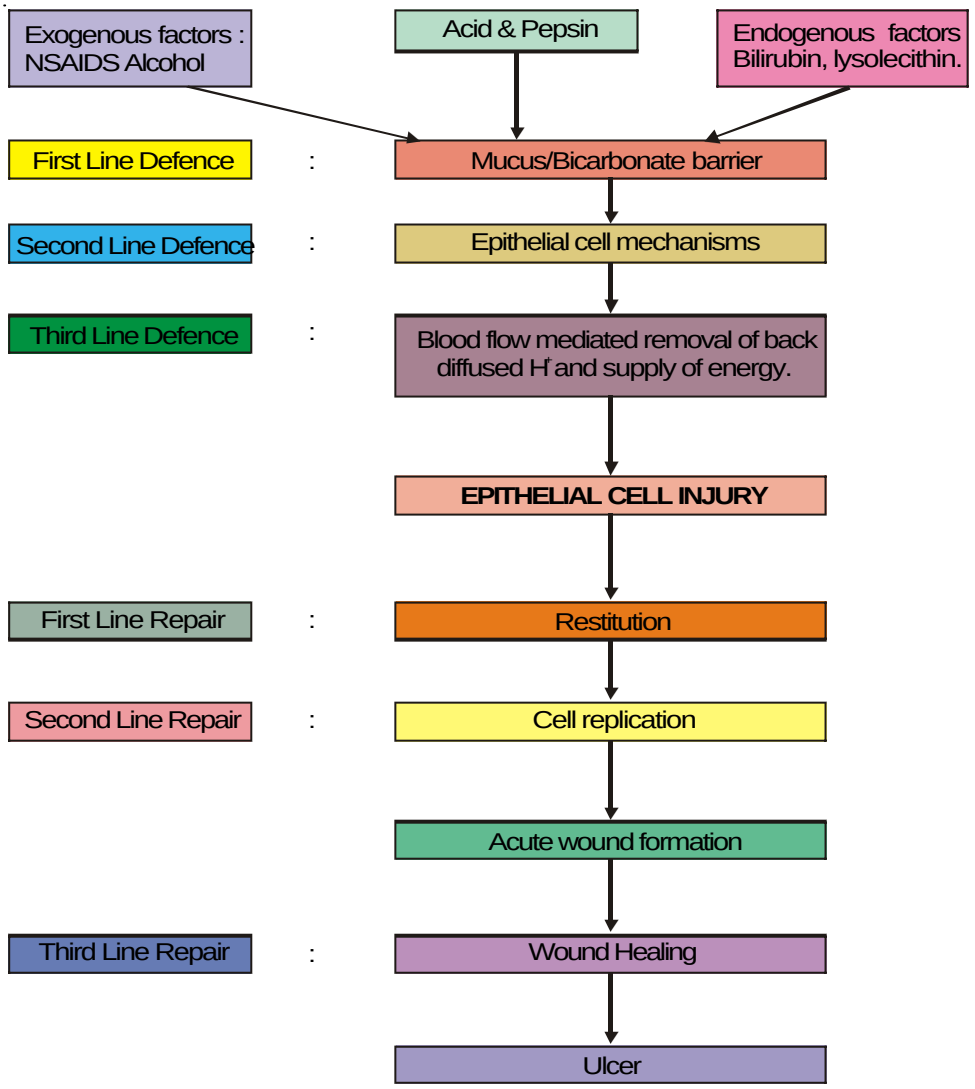

Fig. 1.3: Pathophysiology of ulcer formation

Thus ulcer occurs when three sets of lines of defense and three sets of repair mechanisms fail.

Causes of Peptic Ulcer

Common causes:

H.pylori associated

NSAID induced

Stress Ulcers

Pathology

Erosions are superficial mucosal defects whereas ulcers extent through muscularis mucosae into sub-mucosa or muscularis propria . Based upon the location, ulcers are divided into the following four subtypes. (Kindly refer to Fig 1.4 for diagnostic representation)

(a) Duodenal Ulcers

(b) Distal gastric ulcers, comprising distal antral and prepyloric ulcers.

(c) Proximal gastric ulcers

(d) Ulcers in gastric cardia or hiatal hernia pouch. These are special subclass as these more inclined to complicate.

Peptic Ulcer Disease
Fuctas helping repair
Mucus Bicarlorly barrier
Cpithetial cel mechanisms
Blood supplies energy and removes H^2
Helicobactors
pylon
(90%) mechanism of Ulcer formation and repair
Factors Precipetating
helicopacter Pysoions (75%)
$AlcoHo_2$
Cigarette
Acid pepsu
endogenus factor

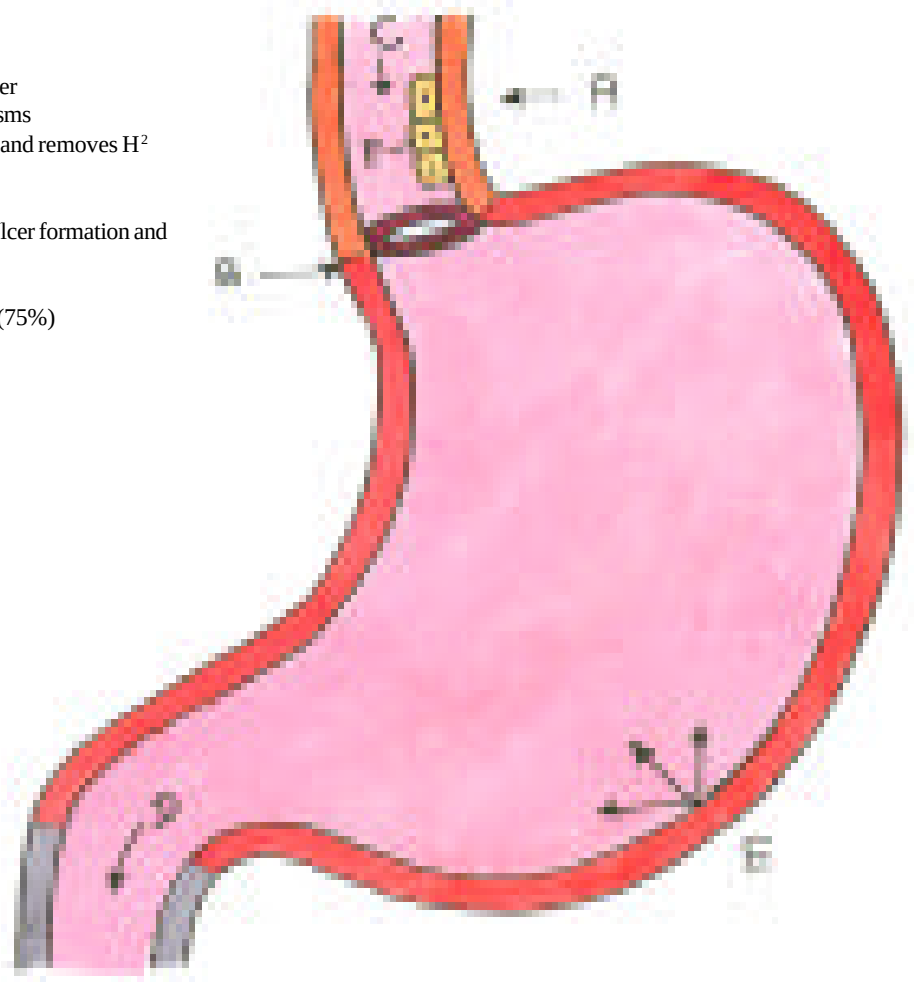

Fig. 1.4: Mechanisms of ulcer formation and repair

In the elderly the manifestation of peptic ulcers is more likely to be silent. The proportion of gastric ulcer catches up with the duodenal ulcers in the elderly. Elderly also tolerate the complications poorly as well as their ulcers take longer time to heal as compared to younger counterparts.

Potential risk factors of ulcers are:

a) NSAIDS

b) Cigarette smoking

c) Alcohol

Duodenal ulcers emerges two decades earlier than gastric ulcers particularly in males. Estimation of annual incidence of peptic ulcer drawn from pre-H.pylori range from 0.1% - 0.3%. Several studies indicate an ulcer incidence of about 1% per year in H. pylori infected individuals, a rate that is 6-10 fold higher than for non-infected individuals.

Check Your Progress 2

1) Why GERD is more common in elderly?

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2) What are the diagnostic modalities available for diagnosing GERD?

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1.5.2 Clinical Presentations

Peptic ulcer classically manifests as symptoms of acid dyspepsia which appear to reflect gastroduodenal sensitization to hydrogen ions. Importantly, some patients are free of heralding symptoms until they manifest with ulcer haemorrhage or perforation. Peptic ulcer is overdiagnossed in clinical practice because similar symptoms are found in other conditions also.

Other clinical presentations may be:

a) Symptomatic gastroesophageal reflux.

b) Feature suggestive of irritable bowel.

Physical examination may reveal mild epigastric tenderness or evidence of disease associated or complicating the manifestation and management of peptic ulcer such as cirrhosis.

Presentation of Ulcer Complications

The majority of complications are associated with chronic peptic ulcers. Penetrating ulcers manifest with a shift from typical vague visceral discomfort to a more localised and intense pain that radiates to back and has lost relief with antacids. A sudden development of severe diffuse abdominal pain may indicate perforation. Vomiting is the feature of gastric outlet obstruction. Haemorrhage may be heralded by nausea, haematemesis, malena or dizziness.

1.5.3 Diagnosis

A) Diagnostic tests for H. pylori:

   i) Urease test : Breath, Biopsy

   ii) Histology

   iii) Culture

   iv) Serology

B) Upper GI Radiography: Three aspects of peptic ulcer is to be looked into:

   i) Identification of crater

   ii) Secondary changes in duodenum and stomach

   iii) Differentiation of benign ulcers from malignant ones.

Endoscopy is now the gold standard for diagnosis. Fibreoptic endoscopy provides a sensitive, specific and safe method for diagnosing peptic ulcers, allowing direct inspection and biopsy. In case of gastric ulcer, malignancy is to be ruled out and biopsy taken. Duodenal ulcers are to be ruled out and biopsy taken. Duodenal ulcers are usually benign.

1.5.4 Management

The main aims of management are:

1) Relieve symptoms

2) Healing of ulcer

3) Prevent recurrence.

In the past the first two were easily achieved. Now even the third aim is fulfilled with eradication of H pylori.

Balanced diet, rest and sedation are no longer advised in management of ulcer disease.

1) **Diet :** Substances like tea, coffee stimulate acid secretion and are therefore to be avoided. Same applies to anytime which causes injury to gastric mucosa, for example alcohol. Adequate fibre in diet helps in healing.

2) Injury producing substances: Smoking, NSAIDS.

3) Drugs used for ulcer healing

   a) Antacids which give symptomatic relief also.

   b) H_2 receptors antagonists block the receptors on parietal cell and effectively control acid secretion. Drugs are:

Cimetidine	40 mg twice daily	Affects enzyme P450 in liver Alters levels 01 drugs like theophyllin, adrenergic block in aged.
Ranitidine	150 mg	To be avoided in case of alcohol
Tamotidine	20 mg	Avoid in patients with heart Disease.
Nizatidine	150 mg	Avoid in patients with heart Disease.
Roxatidine	75 mg	

Double dose can be used as a single dose at night. Maintenance dose is used at night once daily instead of twice daily.

Disease tends to recur when treatment is stopped. It is expensive and is suitable only for the following selected categories:

a) Frequent relapses

b) Complications like Gastro-Intestinal bleeding

c) In elderly subjects with multiple diseases

d) Cases when NSAID therapy has to be continued.

In older patients, treatment is carried out with PPI (Proton Pump Inhibitors). The ulcer healing results are better and achieved earlier. These are omeprazole, pantoprazole etc. The following are suitable for treatment.

a) Ulcer not healing with 3-4 month treatment with H2 receptor antagonists.

b) Complicated ulcers.

c) Zollinger Eilison syndrome.

d) Associated reflux oesophagitis.

A) **Duodenal Ulcers:** With recognition of fact that H. pylori plays a central role in pathogenesis of duodenal ulcer, the main objective is to eradicate H. pylori. Numerous drugs have been evaluated against H. pylori but no single agent is optimally effective. Currently, the most successful therapy is triple therapy which consists of bismuth compound, metronidazole and either amoxycllin or tetracycline, tripotassium dicitrate bismultiate 120 mg QID f— mouth.Tetracycline 500 mg TDS 2 weeks, Metronidazole 400 mg TDS 2 weeks. In the past the drugs that reduce acid secretion or neutralised acid were mainstay for treatment of duodenal ulcer but now these drugs are used as a adjunctive or for symptomatic relief of duodenal ulcers. These include antacids, hydrogen receptor antagonists, anticholinergics, coating agents, prostaglandins and proton pump inhibitors.

B) **Gastric Ulcer:** Gastric ulcers associated with H. pylori infection should be treated with tripple therapy combined with acid suppressive therapy. These ulcers take longer to heal. If gastric ulcer does not heal at satisfactory rate, malignancy should be ruled out.

Complications and Management

a) ***Haemorrhage:*** In most patients it is self-limited. In some cases endoscopic procedures are to be used.

b) ***Perforation:*** Surgical management

c) ***Gastric outlet obstruction:*** Drainage procedure

Check Your Progress 3

1) What are the causes of peptic ulcer disease?

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2) How clinical profile of peptic ulcer in elderly is different from adults?

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3) What is the treatment modality available for treating peptic ulcers?

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4) Which is the diagnostic procedure most useful in diagnosing peptic ulcer disease?

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1.6 TUBERCULOSIS OF INTESTINE

Any region of the gastrointestinal tract can be involved with tuberculosis. This disease constitutes an important public health problem in developing countries like India.

Etiology and Pathogenesis

The pathogen responsible for most of the cases is acid fast bacilli mycobacterium tuberculosis, although in some parts of the world where dairy products are extensively used, mycobacterium bovis is an important cause.

The routes of infection are :

a) swallowed organism

b) haematogeneous dissemination from active pulmonary focus. The frequency of intestinal involvement is directly related to the severity of pulmonary involvement.

Classification

The most frequent site of intestinal involvement is the distal ileum and caecum with 85-90% of patients having infection at this site. Multiple areas of bowel can be involved but the most frequent site is ileocaecal region. The other locations, in order of incidence are ascending colon, jejunum, appendix, duodenum, stomach, esophagus, sigmoid colon and rectum.

Pathology

The gross appearance of intestinal tuberculosis has been divided into three categories :

1) Ulcerative, seen in 60% of patients. There are multiple superficial lesions confined largely to the epithelial surface. It is considered to be highly virulent process.

2) Hypertrophic, occurring in 10% of patients. This conditions consists of scarring, fibrosis and heaped up mass lesions that mimic carcinoma.

3) Ulcerohypertrophic, seen in 30% of patients. This type has mucosal ulcerations combined with the healing and scar formation.

Histologically, the distinguishing lesion is a granuloma. Caesation is not always seen, especially in the mucosa although caesating granulomas are found with regularity in regional lymph nodes.

Clinical Features

Only a minority of patients with intestinal tuberculosis will have specific symptoms. The most common complaint is chronic abdominal pain, which is non-specific and is reported in 80-90% of patients. Weight loss, fever, diarrohea or constipation and blood in stool may be present. An abdominal mass, usually in the right iliac fossa, deep and rather posterior, can be appreciated in about two third of patients.

Laboratory Findings

There is usually mild anaemia with normal total white blood cell count. The tubercle b acillus can be isolated from the stool in about one third of patients. But this finding is not useful in patients with coexisting pulmonary tuberculosis, because it may represent only swallowed organisms.

Complication

The complications include haemorrhage, perforation, obstruction, fistula formation and malabsorption syndromes.

Diagnosis

The definitive diagnosis of intestinal tuberculosis is made by identification of organism in tissues, either by direct identification with acid fast stain, by culture of the excised tissue or

by PCR assay. A presumptive diagnosis of intestinal tuberculosis can be established in patients with active pulmonary tuberculosis who have radiographic and clinical findings suggestive of intestinal involvement.Radiographic examination of the bowel reveals a thickened mucosa with distortion of mucosal folds, Ulceration, various degrees of thickening and stenosis of bowel and psudopolyp fromation. CT can be helpful in showing preferential thickening of ileococcal valve and medial valve of caecum, extension to terminal ileum and massive lymphadenopathy with central caesation necrosis. The caecum is contracted with disease on both sides of valve and the valve itself is often distorted and incompetent. Tuberculosis has tendency to involve small segments of intestine with stenosis and fistula formation.

Treatment

Standard antituberculosis treatment gives a higher cure rate for intestinal tuberculosis. There are no controlled trials to determine the optimal type of therapy or duration. In case of extrapolation from other forms of pulmonary TB, a period of 12 months would be adequate treatment.

Role of Surgery

Surgical interventions is required especially in case of obstructions and fistula formation. Mass lesions associated with hyertrophic form may still need surgical intervention if they cause obstruction.

Surgery may also be required in patients of perforation, confined perforation with abscess formation or if massive haemorrhage occur.

Check Your Progress 4

1) What are the routes of infection for intestinal tuberculosis?

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2) Which is the most frequent site of involvement by intestinal Koch?

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3) What is the role of surgery in intestinal Koch?

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1.7 LET US SUM UP

Upper gastrointestinal disorders are common medical problems in the elderly.

Achalasia cardia is a disease involving the muscle and nerve components of the oesophagus and is characterised by a poorly relaxing lower oesophageal sphincter. The cause remains unknown. The disease usually presents dysphagia. It can be easily diagnosed by a detailed

history and appropriate investigations including a barium swallow, endoscopy and manometry. Treatment by dilation and oesophagomyotomy is directed at palliation of symptoms.

GERD is very common in the elderly and can cause diagnostic confusions. It can be managed efficiently by drugs but some refractory cases require surgery. Peptic ulcer disease causes wide range from symptoms ranging from dysplesia and epigastric discomfort to serious complications like perforation or haemorrhage The definition of role of H pylori in the peptic ulcer disease and effective drugs for eradicating Hpylori have revolutionised the treatment of peptic ulcer disease.

Because of being widely prevalent in India, tuberculosis of GI tract has also considerable clinical importance. This disease causes a difficult diagnostic problem as well is very difficult to treat.

1.8 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Achalasia producers functional obstruction of the oesophagus because of an inability of the lower oesophageal sphincter to relax. A decrease in the number of ganglion cells coupled with the degenerative changes in the oesophageal branches of the vagus have been noted. Thickening of the circular muscle of the lower oesophagus is thought to be secondary to the neuronal changes.

2) c)

3) A barium swallow with fluoroscopy can be used for screening of patients suspected to have achalasia cardia. Dilation of the lower oesophagus terminating in appointed “beak” is characteristic.

   Manonetary clinches the diagnosis of achalasia. It is characterised by an increase in resting pressure. The intra-oesophageal pressure exceeds intra-gastric pressure.

4) Dilation of the oesophagus produces a significant response in 60% of patients . Older patients with a longer history respond better.

   Oesophagomyotomy shows good results in 80-90% of patients. However, it is commonly associated with gastroesophageal reflux.

   Drugs like nitrates, calcium channel blockers and botulinium toxin can bring about transient improvement.

Check Your Progress 2

1) GERD is more common is elderly because:

   a) The intra abdominal segment of lower oesophageal sphincter is shorter.

   b) The secondary peristalsis is reduced.

   c) The number of tertiary contractions increase.

   d) Relaxation of the tone of lower oesophageal sphincter.

2) The diagnostic modalities available are:

   a) Upper Gastrointestinal Endoscopy.

   b) Barium swallow.

   c) Radiological investigations

   d) Therapeutic studies.

Check Your Progress 3

1) Peptic ulcer disease occurs when the intricate balance of repair and healing mechanisms of gastric mucosa fail. These mechanisms can be disturbed by a wide variety of factors which include:

   - H. pylori
   - NSAIDs
   - Stress

2) Peptic ulcer is more likely to be silent in elderly with proportion of gastric ulcers being more. There is high likelihood of complications and these subgroups of patients colerate complications poorly. The ulcers in these patients can take a long time to heal.

3) The discovery of H. pylori has radically changed the treatment for peptic ulcer disease. At present, the goal is to eradicate the H. pylori which will lead to cure of peptic ulcer disease. For this, multiple drug therapy along with proton pump inhibitors is commonly used with considerable success.

   Peptic ulcer disease if untreated can lead to wide variety of complications which may require surgical intervention.

4) Upper GI endoscopy is the gold standard for diagnosing peptic ulcer disease.

Check Your Progress 4

1) The routes of infection are:

   a) Swallowed organism in case of pulmonary Koch's (most commonly).

   b) Haematogeneous dissemination.

2) The most frequent site of involvement of gut by Koch's is distal ileum and caecum. The other sides in order of incidence are ascending colon, jejunum, duodenum, stomach, oesophagus, sigmoed colon and rectum.

3) Surgical intervention is required if patient with intestinal Koch's develops obstruction or perforation or abscess formation.

1.9 FURTHER READINGS

Sainani, G.S. (ed.), *AP1 Textbook of Medicine*, 6th edn., Association of Physician of India, 1999.

Fauci, Braunwald, Isselbacher, et al. *Harrison's Principals of internal Medicine,* 14th edn., International edition, McGraw Hill Publishers, 1998.