
UNIT 1 DISORDERS OF GASTROINTESTINAL TRACT

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1.0 OBJECTIVES

After reading this unit, you should be able to:

- describe the common gastrointestinal disorders in the elderly;
- distinguish clinically between these common gastrointestinal disorders;
- evaluate dysphagia;
- evaluate dyspepsia;
- recognize presentations of GERD including atypical ones;
- evaluate constipation;
- assess for treatable causes of faecal incontinence;
- treat the clinical problems; and
- Institute preventive measures wherever possible.

1.1 INTRODUCTION

In this unit, you will learn about common 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. The role of physiological changes in normal aging which may act as predisposing factor in some of the diseases conditions to a variable degree has been highlighted in the beginning of the unit. Geriatric patients have greater likelihood for functional and cognitive disorders and additional co-morbidity. Hence it is crucial to anticipate difficulties in preparing these patients for interventions like endoscopic procedures. The risk-benefit ratio should be kept in mind during diagnostic testing or management. A proper understanding of risks involved, adherence to the patient's wishes and keeping quality of life foremost in mind are essential while providing healthcare for elderly.

1.2 NORMAL AGE RELATED CHANGES IN GIT

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.

- (a) 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. Dry mouth or xerostomia is fairly common in elderly and may be due to medications or diseases affecting salivary glands.

1.3 DYSPHAGIA

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

- (a) Oral phase: In elderly person number of changes occur like increase in connective

Did You Know?

The 3 phases of swallowing are

- (a) oral phase
- (b) pharyngeal phase
- (c) oesophageal phase.

tissue in tongue, loss of dentition, reduced masticatory strength, which interfere with oral preparatory phase of swallowing leading to poorly formed food bolus. Dry mouth or xerostomia is fairly common in elderly and may be due to medications or diseases affecting salivary glands.

- (b) Pharyngeal phase: Reduction in URS pressure occurs with aging. There is delay in the initiation and decrease in duration of pharyngeal swallow along with decreased opening duration of cricopharyngeous. Delayed elevation of larynx and a reduced ability to clear the pharynx are associated with aging. Thus the coupling between oral and pharyngeal phase of swallowing is disturbed leading to dysphagia and aspiration.
- (c) Oesophageal 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.

The prevalence of dysphagia increases with age. Most studies abroad have shown 50-60% prevalence in nursing homes and 30% in general medical wards. Prevalence of dysphagia in elderly in India is likely to be similar but is not well studied. Some patients have limited awareness of their dysphagia, so lack of the symptom does not exclude an underlying disease. When dysphagia goes undiagnosed or untreated, patients are at a high risk of pulmonary aspiration and subsequent aspiration pneumonia secondary to food or liquids going the wrong way into the lungs. Some people present with "silent aspiration" and do not cough or show outward signs of aspiration. Undiagnosed dysphagia can also result in dehydration, malnutrition, and renal failure. Dysphagia may sometimes be the first symptom of a systemic disorder.

Dysphagia could be **oropharyngeal** or **oesophageal**. Oropharyngeal (or transfer) dysphagia and esophageal dysphagia can result from inflammatory, mechanical, or functional causes. A good clinical history will help to diagnose the type of dysphagia. This will help you in selecting further diagnostic aids. Concurrent neurologic symptoms, nasopharyngeal regurgitation, dysphonia, drooling, aspiration, and coughing after eating suggest oropharyngeal dysphagia. Dysphagia localized below the sternum, chronic GERD, caustic ingestion, or immunocompromised states suggests esophageal dysphagia.

Videofluoroscopy 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 neurological, muscular and structural disorders as shown in **Table 1.1**.

Table 1.1: Causes of Oropharyngeal Dysphagia

<p>1) Medical and surgical lesions of peripheral and central nervous system</p> <p>Cerebrovascular accidents</p> <p>Multiple sclerosis</p> <p>Parkinson's disease</p> <p>Central Nervous System tumour</p> <p>Amyotrophic lateral sclerosis</p> <p>Bulbar poliomyelitis</p> <p>Peripheral neuropathies</p> <p>Alzheimer's disease</p> <p>IHead injury.</p>	<p>2) Muscular or Neuromuscular causes</p> <p>Myasthenia gravis</p> <p>Muscular dystrophies</p> <p>Endocrine myopathy</p> <p>Inflammatory muscle disease</p> <p>Alcoholic myopathy.</p>
<p>3) Structural Lesions</p> <p>Carcinoma</p> <p>Postericoid webs</p> <p>Anterior cervical osteophytes</p> <p>Scarring after operation or radiation or burn</p> <p>Inflammatory conditions</p> <p>Abnormal relaxation of U.E.S.</p> <p>Abnormal opening of U.E.S.</p> <p>Zenkersdiverticulum.</p>	<p>4) Pharmacological therapy</p> <p>IAnticholinergic</p> <p>Antihistamines</p> <p>IPhenothiazines.</p>

Investigations

The tests used to evaluate include barium radiography, a videofluoroscopic swallowing study (VFSS; modified barium swallow), fiberoptic endoscopic evaluation of swallowing (FEES).

Management of neurogenic dysphagia

Most elderly patients with neurogenic dysphagia will require swallowing therapy. During swallow training, two parallel courses are often tried, which are having the patient (1) eat some foods orally while preventing aspiration via compensatory postural techniques, sensory stimuli, voluntary swallow maneuvers, and dietary changes and (2) exercise to build up strength and coordination to regain full swallowing function without compensation.

Five postural techniques (chin-down, chin-up, head turned, head tilted, and lying down) and several postural combinations are currently used for swallow compensation. The chin-down posture is well suited in patients with a tongue base disorder, and a reclining

posture is useful in patients with bilateral pharyngeal damage or reduced laryngeal elevation. Chemical, thermal, and tactile stimulation through changing the taste, volume, temperature, and carbonation of the food (bolus) and even additional pressure on the tongue with a spoon as food is presented have been effectively used to modulate human swallowing behavior. Some swallow maneuvers such as the supraglottic, super-supraglottic, and effortful swallow and the Mendelssohn maneuver have been used by normal subjects and dysphagic patients to compensate for pharyngeal swallow and are employed in swallow rehabilitation. Dietary changes, particularly thickened liquid diets, are commonly used to prevent liquid aspiration in patients with oropharyngeal dysphagia.

Several range-of-motion exercises are used to boost pharyngeal swallow and could take up to 6 weeks to be effective. T-strengthening exercises are aimed at improving oral and pharyngeal transit times and have been reported to be beneficial in dysphagic patients who have had strokes and in patients who have been treated for head and neck cancer. Shaker exercise (a series of sustained and repetitive head lifting exercises to enhance the strength of infrahyoid and suprahyoid muscular activity) improves hypopharyngeal movement and upper esophageal sphincter opening.

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.

Oesophageal dysmotility as well as mechanical causes are common in elderly. Esophageal cancer is common malignant cause for dysphagia. Eosinophilic esophagitis can sometimes present very late. Dysphagia may be the first symptom of a systemic disorder or a rare paraneoplastic symptom.

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
2) Scleroderma
3) Diffuse Oesophageal Spasm
4) Hypertonic (jackhammer) oesophagus
5) Ineffective esophageal motility
6) Fragmented peristalsis
Mechanical lesions
1) Carcinoma oesophagus.
2) Medications (Pill esophagitis)
3) Lower oesophageal webs

- 4) Oesophagitis
- 5) Diverticula
- 6) Extrinsic compression: Abnormal right subclavian artery or annular aorta, cervical osteophytes, enlarged left atrium

Neurological Causes

Parkinson's disease

Multiple Sclerosis

Myasthenia Gravis

Amyotrophic Lateral Sclerosis

Investigations

The tests used to evaluate include barium radiography, endoscopy, and esophageal manometry.

Management of esophageal dysphagia

Esophageal cancer is managed with surgery, radiation and chemotherapy depending on stage of disease. Benign structures, webs or rings can be treated with endoscopic dilatation using bougie or ballon dilators.

Motility disorders are often treated with prokinetics like domperidone, itopride for decreased motility; and with nitrates or calcium channel blockers (nifedipine or diltiazem) for spastic conditions. Acid suppression with proton pump inhibitors are needed for acid reflux related esophageal spasm. Management of achalasia cardia is detailed in a later section.

1.3.3 Pill esophagitis

Medication-induced esophageal injury usually caused by local irritation of the esophageal mucosa and referred to as pill may lead to swallowing difficulties. It is mostly caused by doxycycline, potassium chloride formulations, nonsteroidal anti-inflammatory drugs, alendronate, and quinidine.

The most common site for pill-associated esophagitis is near the level of the aortic arch, an area characterized by compression from the arch, skeletal to smooth muscle transition, and physiologic reduction in the amplitude of the esophageal peristaltic wave. The risk of the development of pill esophagitis is enhanced if medications are taken in the supine position and prior to sleeping as the frequency of swallowing and the ability of the saliva to dilute medications in the esophagus are diminished during sleep. Other factors include drug intake without sufficient water/fluid, polypharmacy, the size and shape of a pill, esophageal motility disorders or compression, or entrapment by fixed mediastinal structures and adhesions following prior chest surgery.

Elderly patients are particularly at risk for development of drug-related dysphagia and pill esophagitis because they consume more medications and are more likely to have anatomic and motility abnormalities, cardiac enlargement with concomitant compression of the mid-esophagus, and decreased saliva production. Thus, a detailed review of medications and when they are taken is an important component of dysphagia evaluation.

Think and Reflect

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Management of esophageal dysphagia

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1.3.4 Achalasia Cardia

Achalasia is motor-disturbance of the oesophagus characterised by a poorly relaxing lower oesophageal sphincter (LES). Failure of the sphincter to relax produces a functional obstruction of the esophagus. Patients with long standing achalasia are at an increased risk for developing esophageal cancer although absolute risk is low.

Aetiology

Idiopathic achalasia is an inflammatory disease of unknown etiology characterized by esophageal aperistalsis and failure of LES relaxation due to loss of inhibitory nitroergic neurons in the esophageal myenteric plexus. **Secondary achalasia** occurs in diseases such as Chagas disease or as a consequence of tumour infiltration into the myenteric plexus. Malignancy induced secondary achalasia is rare, accounting for 2-4% of patients presenting with achalasia. Secondary achalasia can also be seen as part of a paraneoplastic syndrome as in small cell carcinoma. Rare association with sarcoidosis has been described with inflammatory cells associated with the myenteric plexus. It can also be due to amyloidosis, Nissen's fundoplication. It is important to differentiate primary achalasia from secondary achalasia as treatment for the latter will depend on the cause.

Pathology

There is inflammatory degeneration of the nitric oxide-producing, inhibitory neurons that affect the relaxation of esophageal smooth muscle; the cholinergic neurons that contribute to lower esophageal sphincter (LES) tone by causing smooth muscle contraction may be relatively spared. In some patients, associated degenerative changes are also found in the ganglion cells of the dorsal motor nucleus of the vagus in the brainstem, and Wallerian degeneration has been observed in the vagal fibers that supply the esophagus. However, the disordered motility of achalasia is primarily due to the loss of inhibitory neurons in the esophageal wall resulting in aperistalsis, associated with loss of inhibitory innervation of the LES rendering the sphincter muscles incapable of normal relaxation and consequent high basal sphincter pressure.

Clinical Presentation

Achalasia should be suspected in the following patients:

- Dysphagia to solids and liquids
- Heartburn unresponsive to a trial of proton pump inhibitor therapy
- Retained food in the esophagus on upper endoscopy
- Unusually increased resistance to passage of an endoscope through the esophagogastric junction

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.

In the early days, achalasia was diagnosed radiographically with the demonstration of esophageal dilation along with retention of swallowed food and contrast material. Subsequently, esophageal manometry became the method of choice, with the defining characteristics of incomplete LES relaxation and absent peristalsis. More recently, high-resolution manometry (HRM) has further objectified the diagnosis, using carefully validated metrics to quantify LES relaxation and peristaltic function.

The commonly used investigations are detailed below:

- 1) **Barium esophagram** — Findings on barium esophagram that are suggestive of achalasia include:
 - Dilation of the esophagus. In patients with late- or end-stage achalasia, the esophagus may appear significantly dilated (megaesophagus), angulated, and tortuous, giving it a sigmoid shape.
 - Narrow esophagogastric junction with “bird-beak” appearance caused by the persistently contracted LES
 - Aperistalsis.
 - Delayed emptying of barium
- 2) **Endoscopy:** Typical endoscopic findings include a dilated esophagus that contains residual material, sometimes in large quantities. In patients with achalasia, the LES usually does not open spontaneously to allow effortless passage of the endoscope into the stomach but, unlike obstruction caused by neoplasms or fibrotic strictures, the contracted LES can usually be traversed easily with gentle pressure on the endoscope. Nonspecific changes in the esophageal mucosa include erythema and ulceration due to retained food .
- 3) **Esophageal manometry:** Diagnostic manometric findings of achalasia are
 - *incomplete relaxation of the lower esophageal sphincter (LES) which is manifested as integrated relaxation pressure [IRP] above the upper limit of normal*
 - *aperistalsis in the distal two-thirds of the esophagus.*
 - *Basal LES pressure may be elevated in some patients*

High resolution esophageal manometry (HRM) can help subtype achalasia cardia (**types I, II, III**) which helps in directing management.

Management

Treatment is directed at palliation of symptoms and prevention of complications

Treatment options include mechanical disruption of the muscle fibers of the LES (eg, pneumatic dilation, surgical myotomy, or peroral endoscopic myotomy [POEM]) or biochemical reduction in LES pressure (eg, injection of botulinum toxin, use of oral nitrates)

Pneumatic balloon dilation and surgical myotomy have comparable high success rates. However, the efficacy of both treatments decreases over time. The response rate to esophageal dilatation varies with patient age (older patients respond better) and duration of symptoms (a longer history is associated with a better response). Good results from myotomy occur in 80-90 % or more of the patients. The most significant complication is gastroesophageal reflux.

POEM is an effective submucosal endoscopic technique for performing myotomy of the LES and more proximal esophageal muscle. In addition, good results for POEM have been reported in patients with achalasia conditions that often do not respond well to conventional therapies such as type III (spastic) achalasia and “end stage” achalasia (markedly dilated, sigmoid esophagus), and in patients who have failed prior endoscopic dilatation and surgical achalasia treatments.

In elder patients with co-morbidities who carry high risk for undergoing esophageal dilatation or motomy, the alternatives include injection of botlinuminto the LES or pharmacotherapy with nitrates.

Check Your Progress 1

1) How does achalasia produce oesophageal obstruction?

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2) The main symptom of achalasia is:

Respiratory Disorders

- 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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1.4 GASTROESOPHAGEAL REFLUX DISEASE (GERD) IN ELDERLY

Gastroesophageal reflux disease (GERD) is one of the most common gastrointestinal disorders in the elderly. 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 increased. There is also relaxation of the tone of oesophageal sphincter. Age-related physiologic changes seen in the aged, along with comorbidity and polypharmacy, promote LES incompetence and/or injure the esophageal mucosa are factors in development of GERD. Elderly are more likely to develop severe disease, esophagitis, and complications. It is more common to find evidence of endoscopic and pathological findings of advanced disease. The anti reflux mechanism is depicted in Fig.1.1.

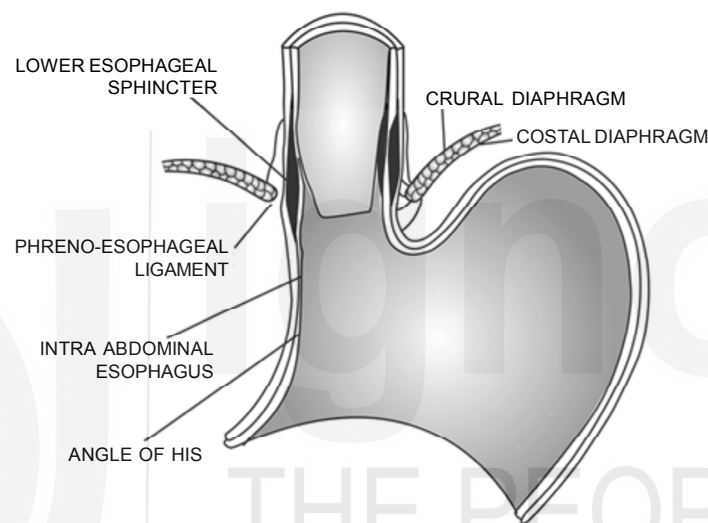


Fig.1.1 Anti Reflux Mechanism

Did You Know?

Common extra-esophageal symptoms of GERD are chronic cough, hoarseness, and wheezing.

Atypical manifestations such as dysphagia, vomiting, and extraesophageal symptoms (cough, laryngeal or bronchospasm) are more apparent. A more severe complication of GERD is Barrett's metaplasia, which increases the risk of esophageal adenocarcinoma. True prevalence is difficult to estimate in elderly because of atypical presentations in some patients. . Most studies indicate about 14-15% of patients over 65 years of age have weekly heartburn.

1.4.1 Pathophysiology

Gastroesophageal reflux disease (GERD) develops due to an imbalance between injurious/symptom-eliciting factors (reflux events, acidity of refluxate, oesophageal hypersensitivity) and defensive factors (oesophageal acid clearance, oesophageal mucosal integrity). The extent of symptoms and mucosal injury is proportional to the frequency of reflux events, the duration of mucosal acidification, and the caustic potency of refluxed fluid.

Other aggravating factors are obesity and presence of a hiatus hernia. Specific foods (fat, chocolate, peppermint), caffeine, alcohol, smoking, and several drugs (e.g. anticholinergics, nitrates, calcium channel blockers, tricyclic antidepressants, opioids, theophylline, diazepam, barbiturates) can cause reflux by lowering LES pressures. The various causative factors for development of GERD have been illustrated in Fig.1.2

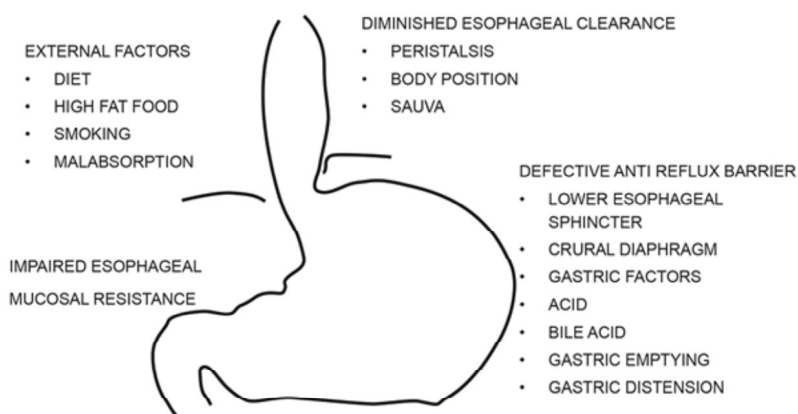


Fig. 1.2 Pathophysiology of GERD

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. Classic symptoms of gastroesophageal reflux disease (GERD) are heartburn (pyrosis) and/or regurgitation. Other symptoms include dysphagia, chest pain, water brash, globus sensation, odynophagia, extraesophageal symptoms (eg, chronic cough, hoarseness, wheezing), and infrequently, nausea.

Table 1.3: Symptoms of GERD

<ul style="list-style-type: none"> • 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

Points to Ponder

The common complications of GERD are oesophagitis, peptic stricture, and Barrett's oesophagus which may lead to oesophageal cancer.

Complications of GERD

Complications are more common in elderly and are:

- Oesophagitis with erosions and ulcers in the oesophagus
- Peptic stricture resulting from oedema & scar tissue in distal oesophagus, usually are associated with hiatus hernia.

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

1.4.3 Diagnosis

The diagnosis of GERD can often be based on clinical symptoms alone in patients with classic symptoms such as heartburn and/or regurgitation. Usual tests include endoscopy, barium studies and oesophageal pH testing.

Early endoscopic evaluation is important in the elderly who have more severe disease even with mild or atypical or alarming symptoms. It can detect esophageal manifestations of GERD (eg, Barrett's metaplasia, erosive esophagitis) and can rule out an upper gastrointestinal tract malignancy. It can also rule out other etiologies in patients with GERD symptoms that are refractory to a trial of proton pump inhibitor therapy. Upper endoscopy may be normal in patients with GERD, or there may be evidence of esophagitis of varying degrees. Among untreated GERD patients, approximately 30 percent will have endoscopic esophagitis.

In patients with suspected GERD with chest pain and/or dysphagia and a normal upper endoscopy, an esophageal manometry should be performed to exclude an esophageal motility disorder. Oesophageal pH monitoring be used to confirm the diagnosis of GERD in those with persistent symptoms (whether typical or atypical, particularly if a trial of twice-daily PPI has failed) or to monitor the adequacy of treatment in those with continued symptoms and prior to undertaking anti-reflux surgery.

1.4.4 Management

Non Pharmacological Treatment

- Life style modifications like raising the head end of bed by six inches using blocks under the legs of the head end of the bed (not by pillows which raise the head only). Some benefit is there by sleeping on left side.
- Patients should be advised to avoid smoking, alcohol and heavy meals at least 3 hours before sleep.
- They should be advised selective elimination of dietary triggers (caffeine, chocolate, spicy foods, food with high fat content, carbonated beverages) if there is symptom correlation with ingestion and an improvement in symptoms with elimination.
- Avoidance of tight-fitting garments to prevent increasing intragastric pressure and the gastroesophageal pressure gradient
- Promotion of salivation through oral lozenges/chewing gum to neutralize refluxed acid and increase the rate of esophageal acid clearance.
- Avoidance of tobacco and alcohol, as both reduce lower esophageal sphincter pressure and smoking also diminishes salivation.
- Abdominal breathing exercises to strengthen the antireflux barrier of the lower esophageal sphincter.
- Yoga

Pharmacological Therapy

Antacids are useful in only symptom relief and are to be used on demand basis only. They are ineffective in providing longer term relief, preventing GERD or in achieving significant rates of healing. Sucralfate (aluminum sucrose sulfate), a surface agent, adheres to the mucosal surface, promotes healing, and protects from peptic injury is also useful for symptom relief.

H₂ blockers or H₂ receptor antagonists (H₂RA) are useful in GERD and symptoms are relieved in 6-12 weeks. They decrease the secretion of acid by inhibiting the histamine 2 receptor on the gastric parietal cell. Usual doses are

Famotidine 20 mg BID

Ranitidine 150 mg BID

Proton pump inhibitors (PPIs) are the most potent inhibitors of gastric acid secretion by irreversibly binding to and inhibiting the hydrogen-potassium (H-K) ATPase pump. PPIs are most effective when taken 30 minutes before the first meal of the day because the amount of H-K-ATPase present in the parietal cell is greatest after a prolonged fast. PPIs should be administered daily rather than on-demand because continuous therapy provided better symptom control, quality of life, and higher endoscopic remission rates. Usual dose used is once daily.

Omeprazole 20 mg

Esomeprazole 40mg

Lanoprazole 30 mg

Rabeprazole 20 mg

Pantoprazole 40 mg

Dexlansoprazole 60mg

In current practice, standard-dose PPI once daily for eight weeks in addition to lifestyle and dietary modifications is preferred option. In cases with nocturnal acid breakthrough may benefit from addition of a nighttime dose of H₂RA to the morning once daily PPI regimen. Use of prokinetic agents like domperidone or itopride can be considered as adjunctive in case of poor response to PPIs but should be used preferably after assessment for other conditions especially esophageal dysmotility by doing esophageal manometry studies. There is no role for empiric eradication of H. pylori. Early referral to a specialist is warranted for patients who fail to respond to once daily PPI therapy (refractory GERD), and patients who cannot tolerate long-term PPIs or want to discontinue therapy.

Anti-reflux surgery

Antireflux surgery is reserved for patients who require high doses of PPIs to control symptoms, for persistent proven GERD symptoms or esophageal mucosal damage despite maximal medical therapy, and when there is significant structural disruption at the esophagogastric junction (eg, hiatus hernia). Surgery is not recommended in patients who demonstrate a complete lack of response to PPI therapy

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 PEPTIC ULCER DISEASE (PUD)

Peptic ulcer are defects in gastrointestinal mucosa (stomach and duodenum) extending through the muscularis mucosae that persists as a function of the acid peptic activity in the gastric juice. Most ulcers occur when the normal secretory, defense, or repair mechanisms of the stomach are disrupted by superimposed processes such as *H. pylori* infection and the ingestion of NSAIDs. Ulcer incidence increases with age for both duodenal ulcers (DUs) and gastric ulcers (GUs), but the incidence of uncomplicated PUD reached a plateau with age, whereas for complicated PUD, the incidence increases with age.

1.5.1 Etiology and Pathophysiology

Peptic ulcer disease (PUD) is associated with two major factors: *Helicobacter pylori* (*H. pylori*) infection and the consumption of nonsteroidal anti-inflammatory drugs (NSAIDs). NSAID use and *H. pylori* infection represent independent and also synergistic risk factors for uncomplicated and bleeding peptic ulcer disease.

H. pylori — *H. pylori* affects a number of aspects of gastrointestinal physiology, including gastric acid secretion, and mucosal defense mechanisms leading to peptic ulcer disease.

NSAIDs, including aspirin — NSAID use is associated with a fourfold increase in the risk of peptic ulcer disease. In addition, NSAIDs are associated with an increase in risk of complications from peptic ulcer disease which include gastrointestinal bleeding, perforation, and pyloric obstruction. NSAID-induced ulcers may also be more refractory to conventional therapy.

Other factors: Smoking is a risk factor for PUD. Alcohol in high concentrations damages the gastric mucosal barrier. Dietary factors may have a role in that certain foods may aggravate while other may have protective effects.

Table 1.5 : Etiology and associations of peptic ulcers

<p><i>Infections</i></p> <p>Helicobacter pylori</p> <p>HSV</p> <p>CMV</p> <p><i>Drug exposure</i></p> <p>NSAIDs and aspirin including low dose aspirin</p> <p>Bisphosphonates (probably when combined with NSAIDs)</p> <p>Clopidogrel (when combined with NSAIDs or in high risk subjects)</p> <p>Corticosteroids (when combined with NSAIDs)</p> <p>Sirolimus</p> <p>Spirolactone (probable, no data with NSAID cotherapy)</p> <p>Mycophenolate mofetil</p> <p>Potassium chloride</p> <p>Chemotherapy (eg, hepatic infusion with 5-fluorouracil)</p> <p><i>Hormonal or mediator-induced, including acid hypersecretory states</i></p> <p>Gastrinoma (Zollinger-Ellison syndrome)</p> <p>Systemic mastocytosis</p> <p><i>Post surgical</i></p> <p>Antral exclusion</p> <p>Post-gastric bypass</p> <p><i>Vascular insufficiency</i></p> <p><i>Radiation therapy</i></p> <p><i>Infiltrating disease</i></p> <p>Sarcoidosis</p> <p>Crohn disease</p> <p><i>Idiopathic peptic ulcer</i></p> <p><i>Non-Helicobacter pylori, non-NSAID peptic ulcer</i></p> <p><i>Comorbid ulcers associated with decompensated chronic disease or acute multisystem failure</i></p> <p>Stress intensive care unit ulcers</p> <p>Cirrhosis</p> <p>Organ transplantation</p> <p>Renal failure</p> <p>Chronic obstructive pulmonary disease (secondary to smoking)</p>
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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. In the elderly the manifestation of peptic ulcers is more likely to be silent. Elderly also tolerate the complications poorly as well as their ulcers take longer time to heal as compared to younger patients.

Physical examination may reveal mild epigastric tenderness or evidence of associated diseases.

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, melaena or dizziness. Upper gastrointestinal bleeding can be life-threatening in the elderly.

Did You Know?

The common complications are perforation, bleeding and gastric outlet obstruction.

1.5.3 Diagnosis

- A) Diagnostic tests for *H. pylori*:
- i) Urease test: Breath, Biopsy
 - ii) Histology
 - iii) Culture
 - iv) Serology
- B) Upper GI Barium 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.
- C) Endoscopy:

Endoscopy is now the gold standard for diagnosis and allows direct inspection and biopsy. In case of gastric ulcer, malignancy is to be ruled out and biopsy taken. Duodenal ulcers are usually benign and not biopsied routinely.

1.5.4 Management

The main aims of management are:

- 1) Relieve symptoms
- 2) Healing of ulcer
- 3) Prevent recurrence.

Points to Ponder

In case of gastric ulcer, malignancy is to be ruled out and biopsy is taken at endoscopy. Duodenal ulcers are usually benign and not biopsied routinely.

All patients with peptic ulcers should receive antisecretory therapy with a proton pump inhibitor (PPI) (eg, omeprazole 20 to 40 mg daily or equivalent) to facilitate ulcer healing. PPI use results in faster control of peptic ulcer disease symptoms and higher ulcer healing rates as compared with H₂RA as a consequence of stronger acid suppression. PPIs also heal NSAID-related ulcers more effectively as compared with H₂RAs

Duration — The duration of initial antisecretory therapy varies based on the ulcer characteristics, the underlying etiology (*H. pylori*, NSAID use) and the presence of ulcer complications (eg, bleeding, perforation, penetration, or gastric outlet obstruction)

H. pylori-positive ulcer

- **Uncomplicated ulcer** – In patients with uncomplicated ulcers, PPI (eg, omeprazole 20 mg BD) given for 14 days, along with the antibiotic regimen to treat *H. pylori*, is usually adequate to induce healing. Eradication of infection should be confirmed four or more weeks after the completion of therapy.
- **Complicated ulcer** – All patients with complicated peptic ulcers (ulcers with bleeding, perforation, penetration, or gastric outlet obstruction) should initially receive acid suppressive therapy with an intravenous PPI. Once patients are tolerating oral medications, they should be switched to an oral PPI at high-dose twice daily to enhance healing (eg, omeprazole 40 mg BD). Dosing should generally be reduced to once daily after four weeks and is treatment duration is 6-8 fweeks in DU and 8 to 12 weeks in GU.

NSAID-induced ulcer — Patients with NSAID-associated ulcers should be treated with a PPI (eg, omeprazole 20 to 40 mg daily) for four to eight to weeks based on the size of the ulcer. In patients with peptic ulcers who need to remain on NSAIDs or aspirin, maintenance antisecretory therapy with a PPI (eg, omeprazole 20 mg daily) can reduce the risk of ulcer complications or recurrence

Non-H. pylori, non-NSAID ulcer — In patients with *H. pylori*-negative ulcers that are not associated with NSAID use, we suggest initial PPI therapy for four weeks for uncomplicated duodenal ulcers, and eight weeks for a gastric ulcer or any complicated ulcer before repeat endoscopic evaluation to assess for ulcer healing.

Other general measures — Underlying risk factors for peptic ulcer disease should be addressed and treated. Smoking cessation should be advised.

Management of complications:

- a) Haemorrhage: Endoscopy is performed to classify the ulcers and asses risk risk for rebleeding. If required endotherapy for bleeding ulcer is performed. Common endoscopic interventions included use of (i) adrenaline injections (ii) thermal method like gold probe (iii) argon plasma coagulation (iv) hemostatic powder application (v) application of mechanical devices like clips at the bleeding ulcer.
- b) Perforation: Surgical management
- c) Gastric outlet obstruction: endoscopic dilatation or surgical bypass procedure (gastrojejunostomy).

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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Gastroparesis

You have already read about the delayed emptying time with age in unit 1 of block 2 of MME 104. Symptoms of gastroparesis or delayed gastric emptying include nausea, vomiting and abdominal distension. Etiological factors for delayed gastric emptying include effects of diseases and drugs; common causes include diabetes mellitus, Parkinson’s disease, hypothyroidism, chronic renal failure; but it is often idiopathic. Clinical presentation is nonspecific, with nausea, vomiting, and bloating. Diagnosis involves history, examination, endoscopy, and scintigraphy. Management includes lifestyle modification, pharmacotherapy, and gastric electrical stimulation.

Gastrointestinal bleeding

Old age is associated with increasing incidence, morbidity, and mortality from gastrointestinal bleeding. Upper gastrointestinal bleeding presents with hematemesis, melena, or blood inn nasogastric tube lavage and results mainly from peptic ulcers, gastric or esophageal varices, and esophagitis. Diverticulosis, hemorrhoids, colon cancer, polyps, and angioectasias are the main causes of lower gastrointestinal bleeding, which generally manifests as hematochezia. Initial evaluation of the patient includes history taking, physical examination with attention to signs of hypovolemia, performance of nasogastric tube lavage, and rectal examination. The diagnosis can is mostly confirmed and treated by endoscopy. On presentation, blood should be sent for a complete blood count, typing and crossmatching, liver function tests, and coagulation studies. Resuscitative measures ought to be immediately undertaken during the patient’s assessment in the emergency department.

1.6 CONSTIPATION

Constipation is characterized by infrequent and difficult passage of hard stools. The patient passes hard stools at too long intervals.

According to the Rome IV criteria, functional constipation is defined as any two of the following features: straining, lumpy hard stools, sensation of incomplete evacuation, use of digital maneuvers, sensation of anorectal obstruction or blockage with 25 percent of bowel movements, and decrease in stool frequency (less than three bowel movements per week) for the last three months with symptom onset six months prior.

Studies have reported that the prevalence of constipation in the older adult ranges from 24 to 50%. In addition to age, risk factors for chronic constipation include female gender, physical inactivity, concurrent medication use, and depression. The pictorial depiction of the external and internal sphincter in resting phase is shown in **Fig. 1.3** and normal defecation is shown in **Fig. 1.4**.

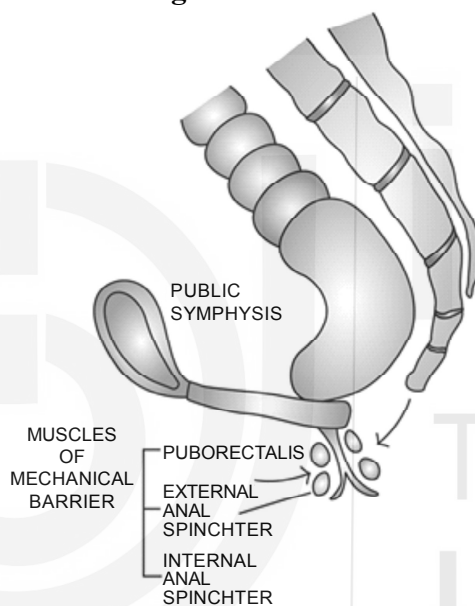


Fig. 1.3 Resting phase

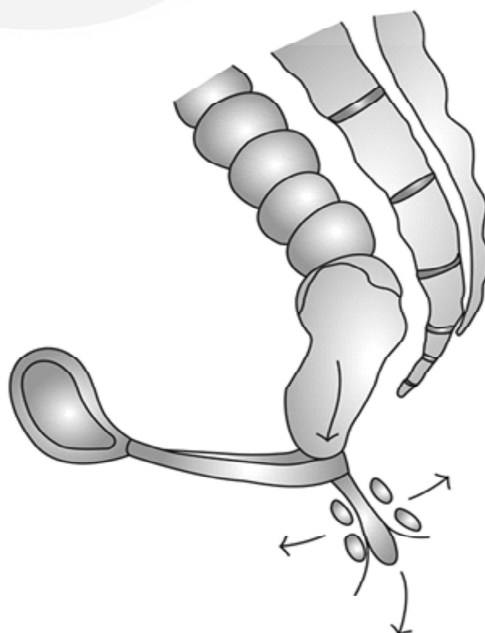


Fig. 1.4 Normal Defecation

1.6.1 Aetiology

Constipation in the older adult may be due to primary colorectal dysfunction or secondary to several etiologic factors.

Primary causes are as follows:

Slow transit constipation — Slow transit constipation is characterized by prolonged delay in stool transit throughout the colon. This could be due to a primary dysfunction of colonic smooth muscle (myopathy) or neuronal innervation (neuropathy) or secondary to dyssynergic defecation.

Dyssynergic defecation — Dyssynergic defecation is caused by difficulty with bowel movement or inability to expel stool from the anorectum. (Fig.1.5).

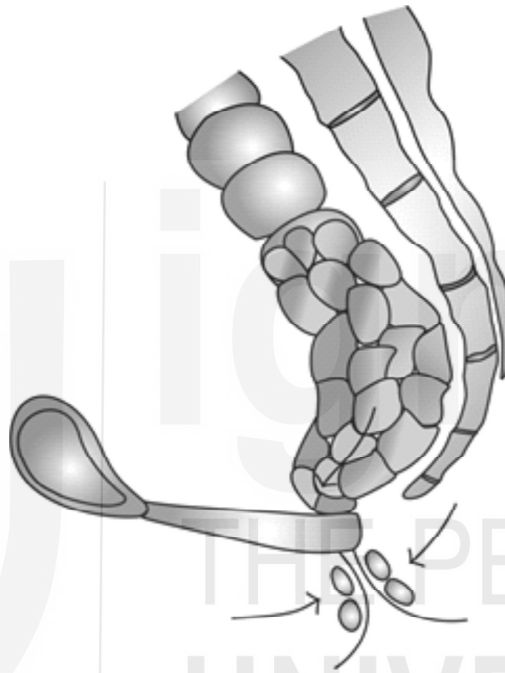


Fig. 1.5 Dyssynergic Defecation

Irritable bowel syndrome — Irritable bowel syndrome with predominant constipation (IBS-C) is characterized by abdominal pain with altered bowel habits. Many of these patients have visceral hypersensitivity

Following table shows the secondary causes for constipation:

Table 1.6 : Causes of secondary constipation

Organic	:	Colorectal cancer, extra intestinal mass, post inflammatory, ischemic, or surgical stenosis
Endocrine or metabolic	:	Diabetes mellitus, hypothyroidism, hypercalcemia, porphyria, chronic renal insufficiency, panhypopituitarism, pregnancy
Neurological	:	Spinal cord injury, Parkinson's disease, paraplegia, multiple sclerosis, autonomic neuropathy, Hirschsprung disease, chronic intestinal pseudo-obstruction
Myogenic	:	Myotonic dystrophy, dermatomyositis, scleroderma, amyloidosis, chronic intestinal pseudo-obstruction

Did You Know?

Constipation in elderly may be **primary** (slow transit/dyssynergia/IBS-C) or **secondary** constipation due to various causes.

Anorectal	: Anal fissure, anal strictures, inflammatory bowel disease, proctitis
Drugs	: Opiates, antihypertensive agents, tricyclic antidepressants, iron preparations, antiepileptic drugs, anti-Parkinsonian agents (anticholinergic or dopaminergic), barium
Diet or lifestyle	: Low fiber diet, dehydration, inactive lifestyle

1.6.2 Clinical presentation and complications

Constipation is characterized either by unsatisfactory defecation, infrequent stools, or difficulty with stool passage. In older adults, constipation may be associated with fecal impaction and overflow fecal incontinence. Fecal impaction can cause stercoral ulceration, bleeding, and anemia.

Alarm symptoms include hematochezia (blood in stools), positive fecal occult blood test, obstructive symptoms, acute onset of constipation, severe persistent constipation that is unresponsive to treatment, weight loss, a change in stool caliber, family history of colon cancer or inflammatory bowel disease; and these indicate the need for more extensive evaluation.

1.6.3 Diagnostic evaluation

It is important to elicit a thorough history noting the onset and duration of constipation.

Co-morbid issues such as immobility, chronic medical problems, and a medication list should be reviewed. Concurrent psychosocial problems such as social isolation, decreased mobility, poor nutrition, and lack of independence should also be addressed as they can contribute to constipation.

Physical examination — A comprehensive physical examination should be performed that includes a rectal exam to palpate for hard stool, assess for masses, anal fissures, hemorrhoids, sphincter tone, push effort during attempted defecation, prostatic hypertrophy in males, and posterior vaginal masses in females.

Laboratory tests include complete blood count, FBS, PPBS, HbA1c, serum calcium, and thyroid function tests can identify metabolic conditions that may be causative as well as secondary anemia that will indicate further evaluation.

Flexible sigmoidoscopy or a full length **colonoscopy** allows for direct visualization of the colon to exclude mucosal lesions (eg, solitary rectal ulcer syndrome, inflammation, malignancy) and should be performed in patients if they have alarm symptoms and also as indicated for colorectal cancer screening.

Physiological testing includes (a) **colonic transit studies** where abdominal X-rays are taken at 36 hours and 60 hours after ingestion of radio opaque pellets; and (b) **anorectal manometry**.

1.6.4 Management

The first step in the treatment of chronic functional constipation is with lifestyle and dietary modification. A daily fiber intake of 20 to 25 g/day is generally recommended. Patients should be advised to consume foods like unpolished rice, wheat, ragi, maize, legumes, dates, and green leafy vegetables.

Laxative usage in the older adults should be individualized based on the patient's history,

Points to Ponder

Alarm symptoms in patients with constipation include hematochezia (blood in stools), positive fecal occult blood test, obstructive symptoms, acute onset of constipation, severe persistent constipation that is unresponsive to treatment, weight loss, a change in stool caliber; or a family history of colon cancer /inflammatory bowel disease.

co morbidities, drug interactions, and side effects. **Bulk laxatives** are the first line of therapy in older patients with chronic constipation who do not respond to dietary and lifestyle modification. **Osmotic laxatives** can be used in patients not responding satisfactorily to bulking agents. Low-dose **polyethylene glycol (PEG)** is efficacious and well tolerated in older adults. **Lactulose** is less effective than low-dose PEG and also had a higher incidence of flatus.

Stool softeners and **suppositories** have limited clinical efficacy. **Enemas** should be used only as needed after several days of constipation in order to prevent fecal impaction. In patients over the age of 70 years being treated with enemas for constipation, warm water enemas are safer than sodium phosphate enemas. Newer drugs include **lubiprostone** (type 2 chloride channels activator) and **linaclotide** (guanylate cyclase agonist) significantly enhance bowel movement frequency and relieve other constipation-related symptoms.

Biofeedback therapy is a painless, noninvasive means of cognitively retraining the pelvic floor and the abdominal wall musculature to facilitate evacuation and is available in referral centers performing anorectal manometry.

Check Your Progress 4

1) What is constipation?

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2) What are the complications encountered with faecal impaction.

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1.7 DIARRHEA IN ELDERLY

Introduction

Chronic diarrhoea may place an elderly patient at risk of dehydration and malnutrition as well as impacting significantly on their quality of life and functional status. Diarrhea in elderly is associated with multiple and diverse causes. The many predisposing and risk factors related to diarrhoea reflect aspects of the aging process directly and indirectly, as with altered defence mechanisms and physiological processes, iatrogenic causes and the effects of institutionalization.

Additional factors to consider in the elderly population are the increased likelihood of colorectal cancer, the second peak of inflammatory bowel disease (IBD) incidence, the potential for polypharmacy contributing to diarrhoea and awareness that faecal incontinence may be more likely in older persons, with or without, associated diarrhoea.

1.7.1 Common diarrheal diseases in elderly

Infective diarrhea

The risk factors for infective diarrhea in elderly include malnutrition, severe co-existing illnesses, cognitive impairment and physical infirmities that compromise hygiene. Institutionalization significantly increases the risk of infection from common source outbreaks such as food-borne epidemics and by person to person spread. Shared toilet facilities with patients with infective diarrhea (especially those with fecal incontinence) increases the risk of infection.

Small bowel bacterial overgrowth (SIBO)

This is an important cause for diarrhea and consequent malabsorption. Age related achlorhydria and use of PPIs are predisposing factors for SIBO. Hydrogen breath testing can be used to make a diagnosis. Breath testing is not a very sensitive; and empirical trial of antibiotics may be needed in cases of high clinical suspicion. SIBO is often under-diagnosed as a cause of chronic diarrhea in the elderly population. Blind loop syndrome or Stagnant loop syndrome can be often seen in elderly.

Clostridium difficile infection

Recent use of antibiotics e.g. clindamycin is usual clinical setting. Hospitalization, proton pump inhibitor use are some predisposing factors. Positive stool samples for *C. difficile* and characteristic appearance of pseudo membrane on colonoscopy are helpful in diagnosis. Appropriate antibiotic therapy with metronidazole or vancomycin results in rapid resolution.

Radiation enteropathy

Diarrhea is a common side-effect of radiation therapy for malignancies in the elderly such as carcinoma of the cervix in females, and rectum and prostate in males. Bloody diarrhea with abdominal pain, urgency, tenesmus can occur. Symptoms can occur weeks to years after abdominal/pelvic irradiation.

Surgery

The most common cause of diarrhea due to surgery is from bowel resection or *diversion colitis* which occurs in a surgically-diverted bowel loop.

Fecal incontinence

Fecal incontinence (FI) is common in the elderly. **Spurious diarrhea** or **overflow diarrhea** is common in elderly in with constipation. Other causes of fecal incontinence associated with aging are impaired rectal sensation and reservoir capacity, impaired puborectalis function and cognitive impairment. FI might be categorized as **urge** (limited time from sense of need to defecate to defecation), **passive** (no awareness of need to defecate; involuntary loss of stool), or **seepage** (involuntary leakage after normal defecation). First-line treatments are nonpharmacologic strategies such as reducing functional barriers, dietary changes, and education about positioning and environmental factors. Medical therapies include psyllium for mobile patients with loose stools. Antimotility agents might help chronic diarrhea and those with past anorectal surgery and passive FI. Digital evacuation of stools is needed if there is fecal impaction (spurious diarrhea). Refractory cases with sphincter dysfunction or injury might benefit from interventions such as sacral neuromodulation, sphincter repair, or sphincteroplasty.

Table 1.7.1: Causes of fecal incontinence

<p>Anus</p> <ul style="list-style-type: none">• Traumatic: surgical or obstetric injury• Nontraumatic: radiation, fibrosis, neuropathy (eg, diabetes) <p>Pelvic floor</p> <ul style="list-style-type: none">• Traumatic: surgical or obstetric injury, chronic straining• Nontraumatic: obesity, sarcopenia, poor muscle coordination <p>Rectum</p> <ul style="list-style-type: none">• Traumatic: surgical injury• Inflammation: inflammatory bowel disease, radiation, infection• Reduced sensation: neuropathy, constipation <p>Bowel</p> <ul style="list-style-type: none">• Diarrhea: infection, inflammation, medications (magnesium, antibiotics, metformin, proton pump inhibitors, cholinesterase inhibitors, antifungals, calcium channel blockers)• Constipation with overflow diarrhea <p>Central nervous system</p> <ul style="list-style-type: none">• Brain: neurodegenerative disorders, stroke, brain tumour, multiple sclerosis• Spinal cord: injury, spinal stenosis, myelopathy
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Diverticular disease

The risk of developing diverticula of the colon increases with age and diarrhoea is a consequence of diverticulitis when acute or chronic inflammation due to a mechanical obstruction within the diverticula occurs. The clinical picture of diverticulitis is a febrile illness with bloody diarrhoea, lower abdominal pain, tenderness, and a possible mass due to an abscess.

Carcinoma colon

Diarrhea or diarrhea alternating with constipation, associated with rectal blood loss, are prominent symptoms of carcinoma of the colon, which is the most common malignancy in old age after carcinoma of the prostate. The rectosigmoid is the site of tumour in two thirds of cases. A malignancy should be urgently excluded when iron deficiency anemia without an obvious cause, changes in bowel habit or caliber of stool, the passage of frank blood, or occult bleeding, or weight loss.

Ischemic colitis

Ischemic colitis occurs more frequently in elderly patients and presents with diarrhea, cramping lower (usually left-sided) abdominal pain and the passage of frank blood or clots.

1.7.2 Inflammatory bowel disease (IBD)

IBD is relatively common in elderly subjects, with up to 35% of patients with IBD aged ≥ 60 years. There are 2 distinct groups of elderly patients with IBD: those who have had IBD for several decades and those who have received a diagnosis later on in life (elderly-onset IBD).

Elderly patients with CD have more colonic involvement with lower frequency of strictures and fistulas as compared to younger individuals, while elderly UC patients experience more commonly left-sided or extensive colitis rather than proctitis. Furthermore, in UC patients, the disease location tends to remain stable, with only 16% of individuals showing proximal disease extension at follow-up visit. Age-related differences in clinical presentation are more evident in CD patients than in UC patients. CD diagnosed in advanced age presents more often with rectal bleeding and less often with diarrhea, abdominal pain and weight loss. Isolated colonic inflammation with less frequent penetrating or perianal disease is a more common finding in elderly-onset CD and small bowel and upper gastrointestinal disease is less common. The severity of symptoms of UC (especially rectal bleeding and diarrhoea) is usually milder in elderly than that in younger individuals.

Although the available drugs for the treatment of IBD in the elderly are the same as those used in younger patients, the response may be slower in advanced age. Treatment of IBD in elderly is complicated by the presence of co-morbidities and by the risk of drug interactions, which is exacerbated by polypharmacy. Potential drug-drug interactions for 5-ASA derivatives, corticosteroids and immunomodulators, are factors needing consideration in deciding the choice of therapy in elderly IBD patients, as they can affect the efficacy and safety of treatments for both IBD and co-morbidities. Cognitive decline and psychiatric diseases, which are relatively common in elderly, further contribute to decreased adherence to therapy.

It is important to differentiate Crohn's disease from TB as misdiagnosing Crohn's disease instead of TB, can have deleterious effects for the patient due to the immunosuppressive medications given for Crohn's disease.

1.7.3 Gastrointestinal Tuberculosis

The most common site of gastrointestinal involvement is the ileocecal region which is involved in 64% of cases of gastrointestinal TB and is thought to be due to the abundant lymphoid tissue and the stasis of the stools around that segment. The rest of the colon is affected with decreasing frequency further away from ileo-caecal area with rectosigmoid being less frequent.

In TB, cell-mediated immunity plays a key role in controlling infection. As age advances the related decline in immunity increases the chance of reactivation of latent disease. Co-morbidities that are more common in the elderly, such as diabetes mellitus, chronic kidney disease, malnutrition, underlying malignancy, alcohol abuse and the use of immunosuppressant drugs (can impair the cell-mediated immune system, increasing the risk of both reactivation and new infection progressing to disease).

The symptoms are not specific and can mimic diseases like Crohn's disease and colon cancer. Commonest complaints are abdominal pain 80.6%, weight loss 74.63%, loss of appetite 62.69%, fever 40.3%, loose stools 16.42% and alternate constipation and diarrhea 25.37%.

Successful treatment of colonic TB can be achieved with conservative management with oral anti-TB medications unless a surgical emergency like perforation or obstruction occurs.

Adverse drug events occur more commonly with increasing age, due to age-related physiological changes and the coexistence of other diseases leading to polypharmacy. Presence of renal or liver dysfunction warrants rigorous monitoring. Increasing age is a predictor of hepatotoxicity due to both isoniazid and rifampicin. Non-pharmacological factors may also increase the risk of hepatotoxicity, such as liver involvement by extensive mycobacterial disease, underlying chronic liver disease and poor nutritional status. Ethambutol can potentially cause reduction in visual acuity, central scotomas and colour vision defects due to optic neuritis. Streptomycin may cause oto- and nephrotoxicity more frequently in those with renal dysfunction, and these adverse events are generally irreversible. Drug interaction is more frequently a problem because elderly patients often receive polypharmacy. A variety of drugs may interact, notably, anticonvulsants like phenytoin; digoxin and steroids. The incidence of drug resistance is also higher in elderly.

1.7.4 Irritable bowel syndrome

While functional bowel disorders clearly exist in the elderly population, new onset lower gastrointestinal (GI) symptoms should always alert the clinician to the likelihood of an organic pathology and the priority in this age group is to exclude colonic neoplasia.

Due to its strong association with psychosomatic factors the irritable bowel syndrome (IBS) is particularly relevant to the elderly. Significant life-events occur in old age such as bereavement of a spouse, ill-health, physical and mental incapacity, relocation from the family home or dependence on a care-giver. There can be varying but characteristic combinations of abdominal pain, bloating (distension) and symptoms of disordered defecation, especially urgency, straining, feeling of incomplete evacuation and altered stool form and frequency". The types of IBS are diarrhea predominant (IBS-D), constipation predominant (IBS-C), missed-type (IBS-M) unclassified type (IBS-U). The presence of pain differentiates IBS-D from functional diarrhea.

Therapy is directed towards pain relief (antispasmodic drugs) diarrhea (loperamide, diphenoxylate) and constipation.

Microscopic colitis

Microscopic colitis, encompassing collagenous colitis and lymphocytic colitis, is more common in elderly females. It typically presents with chronic, non-bloody, secretory diarrhea, frequently with nocturnal symptoms and episodes of faecal incontinence. Macroscopically, the colonic mucosa appears normal at colonoscopy and the diagnosis is made at histological assessment of colonic mucosal biopsies.²⁸ An increase in intraepithelial and lamina propria lymphocytes (>20/100 cells) on colonic biopsy indicates lymphocytic colitis and the presence of a thickened subepithelial collagen band (>10 µm) indicates collagenous colitis.

Pancreatic exocrine insufficiency

Pancreatic exocrine insufficiency, resulting from impairment of pancreatic enzyme and bicarbonate secretion, results in maldigestion and consequently, diarrhoea. Steatorrhea, secondary to maldigestion of fat, may not be clinically apparent: the appearance of stools is not a reliable marker of its presence; stool fat estimation can be performed.

Did You Know?

Faecal incontinence might be **urge** (limited time from sense of need to defecate to defecation), **passive** (no awareness of need to defecate; involuntary loss of stool), or **seepage** (involuntary leakage after normal defecation).

Think and Reflect

The most common site of GI tuberculosis is the ileocecal region due to the abundant lymphoid tissue and the stasis of the stools around that segment.

The measurement of human faecal elastase-1 is generally the preferred test of pancreatic function being a sensitive biomarker for moderate to severe but not mild pancreatic insufficiency. A faecal elastase value of <200 ug/g should prompt pancreatic enzyme replacement and subsequent investigation for causes of pancreatic exocrine insufficiency. In the elderly, an initial CT scan, if not already performed, will help exclude pancreatic malignancy and other advanced pancreatic disease or age-related atrophy as a cause for exocrine insufficiency.

1.7.5 Drugs causing diarrhea in elderly

Various drugs may be implicated in diarrhea in elderly. Alterations in body composition, as well as hepatic and renal dysfunction are more common in the elderly and may impact on drug pharmacokinetics with a consequent influence on drug-related chronic diarrhea and subsequent management.

- (a) **Laxative abuse:** Laxatives used excessively and for prolonged periods of time are a cause of diarrhea and surreptitious laxative use occurs in the elderly. Cognitive impairment also may lead to inadvertent laxative abuse, to treat constipation which increases with age and is a problem in about one-third of elderly persons. **Factitious diarrhea** is due to laxative abuse.
- (b) **Other drugs:** see table.

Table 1.7.2 : Common causes of drug-induced diarrhea in the elderly.

PPIs	: omeprazole,
H2RAs	: ranitidine
NSAIDs	: ibuprofen, naproxen, diclofenac
Antibiotics	
Biguanides	: metformin
Alpha glucosidase inhibitors	: acarbose
Motility agents	: macrolides, metoclopramide
Cardiac glycosides	: digoxin
Chemotherapy agents	: epirubicin, 5-fluorouracil, methotrexate, cisplatin
Magnesium supplements	
ACE inhibitors	: ramipril, lisinopril
Colchicine	
Thyroid hormones	: L-thyroxine
SSRIs	: sertraline, citalopram

1.7.6 Investigations for diarrhea

Abdominal examination and pelvic examination including digital rectal examination for masses, sphincter tone is needed.

Routine and microscopic examination of stool is useful in acute infective episodes.

Fecal occult blood testing may be used in patients with symptoms suggestive of colorectal cancer without rectal bleeding in primary care settings. However, the exclusion or diagnosis of colorectal cancer relies on colonoscopy. In the elderly population, there

are a number of factors that may determine suitability for colonoscopy including co-morbidities, frailty, and tolerability of bowel preparation. In unfit patients, the alternatives are flexible sigmoidoscopy, CT virtual colonoscopy and capsule endoscopy.

If a colonoscopy is possible, then an ileoscopy should be performed, as also, right-sided and left-sided colonic biopsies and rectal biopsies with a view to assessing for IBD and microscopic colitis. In those unable to undergo full colonoscopy, a flexible sigmoidoscopy may still allow for assessment and biopsy of the left sided colonic mucosa and thus the potential to diagnose these conditions. In suspected TB, the biopsy specimens should be send for AFB smear and culture; and often ancillary tests like Gene X pert or TB-PCR are useful.

Marked elevations in erythrocyte sedimentation rate (ESR) are seen in active TB. For IBD, the more sensitive marker is C- reactive protein (CRP).

CT abdomen with contrast is usual radiological evaluation for cancer staging; and is also useful in IBD, TB and ischemic colitis.

Fecal calprotectin can be used in primary care to aid differentiation between IBS and potential IBD A raised calprotectin is non-specific and can occur in IBD, microscopic colitis and colorectal cancer, as well as other GI conditions. Fecal calprotectin can also be used as a non-invasive method of monitoring disease activity in IBD.

Check Your Progress 5

1) What are the two most common forms of IBD?

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2) What are the three most common symptoms of GI tuberculosis?

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1.8 LET US SUM UP

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 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 dyspepsia 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 *H pylori* have revolutionised the treatment of peptic ulcer disease.

The frequency of bowel movements varies from three movements a day to three a week. As a rule, constipation should be suspected if more than 3 days pass between bowel movements or if there is difficulty or pain when passing a hardened stool.

Diarrhea in elderly is associated with multiple and diverse causes. Increased likelihood of colorectal cancer, the second peak of inflammatory bowel disease (IBD) incidence, the potential for polypharmacy contributing to diarrhea; and awareness about faecal incontinence should be kept in mind.

1.9 GLOSSARY

DYSPHAGIA is a sensation of food or liquid being stuck in the esophagus or chest. If this sensation is associated with pain, it is labeled **ODYNOPHAGIA** and if it is associated with persistent obstruction and bolus retention, it is categorized as a **FOOD IMPACTION**.

GASTROESOPHAGEAL REFLUX DISEASE (GERD) is a condition of troublesome symptoms and complications that result from the reflux of stomach contents into the esophagus.

PEPTIC ULCER is a defect in the gastric or duodenal wall that extends through the muscularis mucosa (the lowermost limit of the mucosa) into the deeper layers of the wall (submucosa or the muscularis propria).

HELICOBACTER PYLORI is a spiral, microaerophilic, gram-negative bacterium with flagella that have urease, catalase, and oxidase activity.

GASTROPARESIS is a disorder characterized by delayed gastric emptying of solid food in the absence of a mechanical obstruction of the stomach, resulting in the cardinal symptoms of early satiety, postprandial fullness, nausea, vomiting, belching and bloating.

DIARRHEA may be defined in terms of stool frequency, consistency, volume or weight: more than three bowel movements daily, more than 200 grams of the stool daily, and its water content exceeding 75-85%. (The patient's concept of diarrhea often focuses around stool consistency.)

CHRONIC DIARRHEA is the persistent alteration from the norm with stool consistency between types 5 and 7 on the Bristol stool chart and increased frequency greater than 4 weeks' duration.

CONSTIPATION is defined by bowel disturbances (ie, reduced frequency of bowel habits, hard stools, excessive straining to defecate, a sense of anorectal blockage, anal digitation, and a sense of incomplete evacuation after defecation).

INFLAMMATORY BOWEL DISEASE (IBD) is a heterogeneous group of chronic inflammations in the gastrointestinal tract, which traditionally consists of two types: Crohn's disease (CD) and ulcerative colitis (UC).

ULCERATIVE COLITIS is a chronic disease characterized by diffuse mucosal inflammation of the colon. Ulcerative colitis always involves the rectum (i.e., proctitis), and it may extend proximally in a contiguous pattern to involve the sigmoid colon (i.e., proctosigmoiditis), the descending colon (i.e., left-sided colitis), or the entire colon (i.e., pancolitis).

CROHN'S DISEASE is a chronic idiopathic inflammatory bowel disease condition characterized by skip lesions and transmural inflammation that can affect the entire gastrointestinal tract from the mouth to the anus.

FECAL INCONTINENCE is “the involuntary loss of liquid or solid stool that is a social or hygienic problem.”

1.10 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

- 1) Achalasia produces 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. Manometry 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 botulinum toxin can bring about transient improvement.

Check Your Progress 2

- 1) GERD is more common in 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 tolerate 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) Constipation is characterised by infrequent and difficult passage of hard stools. Persons
- 2) passes hard stools at too long intervals.
- 3) The complications include faecal incontinence, false diarrhoea and mechanical ileus.

Check Your Progress 5

- 1) The two most common forms of IBD are ulcerative colitis and Crohn's disease.
- 2) The three most common symptoms of abdominal TB are abdominal pain, weight loss, and loss of appetite.

1.11 REFERENCES AND FURTHER READINGS

- 1) Paul GS. Gastrointestinal problems in the elderly. *In*: Balakrishnan V, Rajesh G (eds). Practical Gastroenterology 2nd edition. 2016, Jaypee Brothers, New Delhi
- 2) Pitchumoni CS, Dharmarajan TS (eds) Geriatric Gastroenterology. 2012; Springer, New York