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REVIEW

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Non-neoplastic disorders in an aging gut: concise review

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Abstract

The spectrum of gastrointestinal (GI) issues in the older population varies from common physiological age-related changes to devastating, less common sinister pathological illness. GI system has direct exposure to external environment. Thus, it is modeled to embrace the pathophysiological changes that occur due to interaction with external factors. Gastrointestinal tract (GIT) per se is more resilient to aging as compared to other organ systems. On the other hand, elderly may present with a large plethora of GI symptoms. This presents a challenge to all echelons of medical consultation for accurate attribution for the aging process or pathophysiological causation of GI symptoms. This dichotomy leads to hindrance in adequate and appropriate treatment of GI ailments. In GI system, non-neoplastic disorders are far more common than neoplastic disorders. Hence, it becomes imperative to understand the aging evolution of the GI system and management of GI disorders in the older population.

Keywords Geriatrics, Gastrointestinal tract, Non-neoplastic disorders

Background

The geriatric boom of the human population has presented new challenges. GI and hepatobiliary disorders are one of the most common presenting complaints in elderly. The physiology of aging affects GI motility, digestion, absorption, and hormone secretion, giving rise to various pathophysiological manifestations. We review the predominant non neoplastic disorders affecting the elderly with emphasis on the underlying pathophysiology, clinical presentation, diagnosis, and available treatment options.

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Pathophysiological changes in aging GUT

Anatomical changes in an aging GIT lead to various physiological alterations. Clinical presentations of common GI disorders can be explained by these physiological changes. Tables 1 and 2 summarize the various anatomical and physiological changes affecting the GIT in elderly.

Oral cavity

Xerostomia

Sensation of the dry mouth or hyposalivation is a common and often neglected problem in geriatric population. The reported prevalence in elderly is around 27 % [1]. Salivary secretion which occurs as a reflex response to chewing or speaking is reduced in a multitude of conditions. These can be drug side effects (antihypertensives, antipsychotics, anticholinergics), chronic diseases like diabetes mellitus (DM), autoimmune disorders like Sjogren's syndrome, nutritional deficiencies like vitamin (Vit) B deficiency states, alcohol abuse, or exposure to radiation therapy [2]. The reduced function of salivary glands coupled with physiochemical changes in the salivary secretions (increased viscosity and decreased ptyalin

Table 1 Age related anatomical & physiological changes and their clinical manifestations

Age-related anatomical changes	Physiological effects	Clinical manifestation
Oropharynx & foregut		
↓ Nerve fibers in the olfactory bulb & olfactory receptors	Reduced perception of taste & smell	Dysgeusia (distorted ability to smell & taste)
Diminution of maxillary & mandibular bones with gradual erosion of tooth sockets	Gum recession & teeth loss	↓Chewing capacity leading to Inadequate food consumption
↓ Salivary secretion	Dry mouth	Xerostomia (dry mouth). Severity increases with reduced fluid consumption & increased drug usage
↓Esophageal peristalsis	↑ Transit time of food	Increase chances of GERD Increased likelihood of aspiration
↓ Tensile strength of UES & LES	Esophageal conduit dysfunction	
↓ Elasticity of stomach wall	↓ Storage capacity of stomach	Decreased oral intake, ↑ risk of under nutrition

Table 2 Age related anatomical & physiological changes and their clinical manifestations

Age-related anatomical changes	Physiological effects	Clinical manifestation
Midgut & hindgut		
Intestinal villi shrink & broaden with age	↓ Absorptive surface area	Malabsorption
Small intestinal bacterial overgrowth (SIBO)	Normal gut flora equilibrium is compromised	Bloating, pain ↓ Absorption of minerals like Ca, Fe, Folic acid
Reduced peristalsis of large intestine	Increased large bowel transit time	Chronic constipation, diverticulosis
Internal anal sphincter thickens & muscle strength of large intestine reduces	Increased rectal pressure ↓ Contraction in colon	Fecal impaction
Hepatobiliary system		
Total liver mass decreases with reduction in hepatic blood flow	↓ Albumin production	Undernutrition, impaired drug metabolism

content), causes dry mouth, cracking of the lips, and fissuring of the tongue. Patients have trouble in swallowing and chewing, often requiring modification of diet. Treatment options include topical application of artificial saliva, sialagogues like pilocarpine, and treatment of underlying disease (e.g., Sjogren's syndrome). Nonpharmacological treatment includes cessation of alcohol or tobacco, moistening the oral mucosa with frequent sips of water. Certain investigational therapeutic options that have been tried are use of oral iron supplements, Omega 3 and Vit E supplements, intraoral electrostimulation, and mouthwashes containing propylene glycol, citric acid, and aloe vera in various combinations. However, none of these has shown any proven benefit [2].

Oropharyngeal dysphagia

It is often underreported, misdiagnosed, and under treated condition in elderly. A prevalence has been estimated to be around 10 % in individuals beyond fifth decade of life to around 30 % in hospitalized patients [3]. Muscle mass and elasticity of connective tissue decreases with age. This coupled with neurodegenerative disorders (dementia, Parkinson's disease), neurological disorders (stroke, head injury, poliomyelitis), and cancers of the head and neck, drugs or irradiation may lead to progressive deterioration of swallowing. The patient often presents with history of difficulty in initiating a swallow, inability to chew food, drooling of the saliva, or spillage of food. The patient may also complain of coughing or nasal regurgitation of food intake. This severely impairs the nutritional status besides increasing the risk of pneumonia, more so in hospitalized patients [4]. Treatment involves modification of diet, e.g., national dysphagia diet, postural adjustments in hospitalized patients, swallow therapy, and neuromuscular praxis.

Esophagus

Esophageal dysphagia on the other hand may occur secondary to structural or motility disorders of the esophagus. Structural causes may be intrinsic, e.g., esophageal web, stricture or carcinoma, or etiologies which cause extrinsic compression, e.g., lymphoma, lymph node masses, aberrant right subclavian artery (*dysphagia lusoria*), or right-sided aorta (*dysphagia aortica*). Achalasia cardia is the classical presentation of primary motility disorder of esophagus. Connective tissue diseases, scleroderma, or paraneoplastic syndromes may secondarily affect esophageal motility [5]. Esophageal dysphagia

classically presents a few seconds after swallowing and is often accompanied by chest pain, heart burn, or regurgitation of undigested food. Opioid-induced esophageal dysfunction (OIED) is recently described condition which may mimic achalasia [6]. Evaluation should include diligent history and clinical examination, neurological examination followed by targeted investigation: video fluoroscopic swallowing study (VFSS), fiberoptic endoscopic evaluation, and esophageal manometry if indicated [7]. Elderly patients with esophageal dysphagia, odynophagia, and those with alarm symptoms like anemia, GI bleed, or weight loss should undergo esophagogastroduodenoscopy (EGD).

GERD

GERD is the most common GI disorder in elderly [8]. The worldwide incidence has been increasing along with increase in associated complications like Barret's esophagus and adenocarcinoma [9]. Defective anti-reflux barrier: transient lower esophageal sphincter relaxation (tLESR), increased frequency of hiatus hernia in elderly, coupled with age-related changes in esophageal motility and saliva production, disturbance in esophageal mucosal resistance, and delayed gastric emptying time, have all been postulated to be responsible for GERD. The use of drugs like nitrates, anticholinergics, and benzodiazepines have been shown to reduce resting LES pressures. The presence of obesity and sedentary lifestyle are other risk factors. Heart burn, acid regurgitation, water brash, and nausea are the most common symptoms while dysphagia, odynophagia, weight loss, and GI bleed herald an ominous diagnosis [10]. The elderly may have reduced symptom severity but increased the risk of mucosal injury. Patients can also present with atypical symptoms like chest pain, globus sensation, ear, nose throat (ENT) symptoms, chronic cough, sleep apnea, and aspiration pneumonia [11]. Older patients are more likely to present with complications. These include erosive esophagitis, esophageal strictures, Barret's esophagus, adenocarcinoma esophagus, dental erosions, laryngitis, sinusitis, chronic cough, aspiration pneumonia, and sleep apnea. Diagnostic armamentarium includes upper GI barium series, EGD to rule out mucosal lesions/esophageal carcinomas. The video capsule endoscopy (VCE) has been postulated to have a diagnostic role. Ambulatory pH monitoring and esophageal manometry may be used in patients with atypical symptoms or those with poor response to therapy [8]. The goal of treatment is to alleviate symptoms, promote mucosal healing, prevent complications, and prevent recurrence of symptoms. Lifestyle management with emphasis on weight loss, elevation of the head end of the bed, promoting time interval between bedtime and the last meal, and review of polypharmacy play an important role. Proton pump inhibitors (PPIs) are the cornerstone of pharmacological therapy. Motility agents like metoclopramide and GABA agonist like baclofen may be used in a select subset of patients. Some of the common drug-related effects of these medications are increased risk of osteoporosis and pneumonia with long-term PPI use; risk of cardiac arrhythmia, muscle tremors, and tardive dyskinesia with metoclopramide; confusion, dizziness, and light headedness with GABA agonists [12].

Esophagitis

GERD is by far the most common cause of esophagitis. However, certain other etiologies that deserve special mention are pill esophagitis, defined as injury to esophageal mucosa related to the direct effects of medication. Although it is a self-limiting condition, persistent symptoms require evaluation for ulceration, stricture, or perforation. The most commonly implicated drugs are antibiotics: tetracycline or doxycycline, non-steroidal anti-inflammatory drugs (NSAIDs), and bisphosphonates: Alendronate or ibandronate, potassium chloride, and warfarin. EGD is the investigation of choice. It should be done if alarm symptoms (odynophagia, hematemesis, malena, or pain abdomen) are present or persistence of symptoms after discontinuation of suspected drug [13].

Eosinophilic esophagitis (EE) is uncommonly reported in the geriatric age group with reported prevalence of 0.05%. Elderly with EE are most likely to present with dysphagia, are more likely to use PPIs, undergo dilatation for their symptoms, and less likely to receive topical steroids. The elderly is also less likely to present with associated disorders like asthma or food allergies [14].

Barret's esophagus

Barret's esophagus is a preneoplastic lesion characterized by the presence of columnar metaplasia in place of normal squamous epithelium at the lower end of esophagus. Prevalence increases with age, white race, long standing GERD, tobacco use, and obesity. The lesions follow the metaplasia—dysplasia—carcinoma sequence with the estimated risk of developing adenocarcinoma being 0.5 % per year. The American Society of Gastrointestinal Endoscopy (ASGE) recommends screening in individuals with multiple risk factors. The use of chromoendoscopy and tissue sampling using "Seattle protocol" has been recommended [15]. Individuals with low-grade dysplasia (LGD) should undergo repeat EGD after 6 months. Individuals in whom LGD has been reconfirmed and in individuals with high-grade dysplasia (HGD) should be referred to multidisciplinary team. Treatment options include endoscopic and surgical therapy [16].

Stomach

Mucosal changes occur in the gastric mucosa with advancing age. Popularly termed as *aging gastropathy*, these changes occur because of atrophy of gastric glands, reduced prostaglandin, and bicarbonate secretion which coupled with reduced sensory innervation brings about impaired mucosal defense against a variety of agents especially drugs like NSAIDs and aspirin. Besides, there is reduced responsiveness to ulcer healing drugs as well [17]. Research indicates that gastric acid secretion does not decrease with age although a small fraction of individuals may have reduced secretion because of chronic atrophic gastritis [18].

Non-steroidal anti-inflammatory drug (NSAID)-induced gastropathy

NSAIDs are known to cause gastric mucosal erosions, ulceration, and GI bleed. The term NSAID-induced gastropathy was coined in 1986 to differentiate it from classic peptic ulcer disease (PUD) which is predominantly seen in young men and is identified by occurrence of gastric or duodenal ulcers. Apart from advancing age, concurrent *H. pylori* infection, history of previous GI tract disease, and the use of drugs like anticoagulants are the other risk factors. Postulated pathophysiology has highlighted the role of inhibition of proinflammatory prostaglandin (PG) synthesis by inhibiting the enzyme cyclooxygenase, direct injury to gastric mucosa by production of pro inflammatory mediators like TNF α, leukotrienes, and disruption of epithelial barrier by altering the membrane permeabilization. Symptoms can vary from dyspepsia and pain abdomen to potentially serious complication like GI bleed, strictures, and gastric outlet obstruction (GOO). Treatment involves discontinuation of culprit drug, treatment of concurrent *H. pylori* infection, and the use of PPIs. The use of PG analog Misoprostol, although reduces the incidence of ulcers, has no proven benefit in dyspepsia and other side effects [19, 20]. The use of nitric oxide releasing NSAIDs (NO—NSAID) and hydrogen sulfide releasing NSAIDs (H2S—NSAID) are being evaluated for providing safer efficacy. However, none of the trials has evaluated the use of these newer agents in geriatric age group.

Peptic ulcer disease

Elderly patients are at high risk of developing PUD and its complications. Advancing age per se is a risk factor for severe disease [21]. The incidence of PUD has been steadily decreasing while the incidence of complications remains high [22]. *H. pylori* infection is the commonest cause of PUD (both duodenal and gastric ulcer) followed by medication like aspirin and NSAIDs. Other etiological factors are gastric bypass surgery, Zollinger—Ellison syndrome, critical illness, and infections like cytomegalovirus (particularly in immunocompromised patients). Individuals may often present with nonspecific symptoms like dyspepsia, pain abdomen, nausea, and early satiety. There is often poor co-relation of the symptoms with endoscopic findings more so in the elderly whose initial presentation may be in the form of complications like perforation or GI bleed. Hence, it is recommended that elderly individuals with dyspepsia be evaluated with an EGD more so if they have alarm features like anemia, weight loss, history of GI bleed, and any h/o long-term NSAID or aspirin use [23]. *H. pylori* eradication should be done in all patients with PUD. The patient with endoscopically confirmed gastric ulcer should be treated with PPI till the ulcer has healed. Healing should be documented by a repeat EGD. Biopsy should be taken in gastric ulcers to rule out malignancy. For duodenal ulcers, *H. pylori* eradication for 7–14 days is usually sufficient. Long-term PPI, usually for 4–8 weeks may be given after which a thorough reassessment should be done [24]. Complications may require urgent endoscopic, interventional radiology, or surgical intervention.

Small intestine

The small intestine with its absorptive, immunological, and endocrine function assumes great importance in the pathophysiology of energy homeostasis in elderly. There are wide gaps in our knowledge in understanding the changes occurring in the GI tract and small intestine in particular [25]. Interestingly, on contrary to animal studies, the human studies have shown that morphological changes (e.g., height of villi and crypt depth) and intestinal permeability remain largely unaffected while there is variation in the post-prandial release of intestinal hormones like cholecystokinin (CCK) and ghrelin with advancing age [25].

Small bowel bleeding

Small bowel is the most common cause of obscure GI bleed. This can be a perplexing problem in elderly who are more likely to have vascular anomalies, ulceration, NSAID-induced enteropathy, small intestinal tumors, and nonspecific enteritis [26]. Usually, small bowel ulcerations are commonly associated with chronic NSAID use (60–70 % of patients with chronic NSAID use). Antithrombotic agents like aspirin are other common culprit agent [27]. Though majority of lesions are asymptomatic, abdominal pain, diarrhea, anemia, or overt GI bleed can be the clinical presentation. NSAIDs can also lead to stricture formation. Evaluation would include small bowel enteroscopy, colonoscopy, and VCE. Computed tomography (CT), enterography, and small intestinal ultrasound can also be useful.

Small bowel vascular lesions

Vascular lesions are an important cause of obscure GI bleed. Angio ectasia (AE), Dieulofoy's lesion (DL) and Arteriovenous malformation (AVM) contribute to around 5% of all causes of GI bleed [26]. Increasing age, apart from chronic kidney disease (CKD), aortic stenosis (Heyde's syndrome), venous thromboembolism (VTE), and the use of warfarin are associated with increasing frequency of angiodysplasia [28]. Clinical manifestations vary with the type of lesion. AE being abnormally dilated and tortuous vessels in the mucosa or submucosa, present with chronic intermittent venous bleeding. DL are large but histologically normal arteries that protrude though a mucosal defect. They may present with acute onset bleed causing hemodynamic compromise once overlying epithelium gets denuded. AVM are histologically abnormal aberrant vessels (thickened hypertrophic vessel walls). Ongoing GI bleed with vascular compromise would warrant CT angiography as the initial investigation of choice. A stable patient may be considered for VCE [29]. VCE can visualize the full small intestine in almost 90 % patients with a diagnostic accuracy of 50 % to pick up AD [30]. This is especially true with ongoing SI bleed. Therapeutic intervention however cannot be done. Therapeutic procedures can be performed using a deep enteroscopy (DE) in suspected small bowel lesions. Single-balloon enteroscopy (SBE), double balloon enteroscopy (DBE), and spiral enteroscopy (SE) are available. DBE has maximum insertion depth [31]. Endoscopic therapeutic modalities are argon plasm coagulation (APC), endoclip application for DL and AVM, injection sclerotherapy, and combination of APC along with injection sclerotherapy. Radiological embolization can be considered in patients with active bleeding and failed or contraindicated endotherapy. Surgical management may be considered only when other therapeutic options have failed. The use of thalidomide and long-acting somatostatin analogs is an area of active research [29].

Mesenteric ischemia

Acute mesenteric ischemia (AMI) is an important differential diagnosis in elderly presenting with acute abdomen. Classically divided into non-occlusive (NOMI) and occlusive disease. Mesenteric arterial embolism is seen in 50 % of all individuals diagnosed with AMI followed by mesenteric arterial thrombosis (15–25%) and mesenteric venous thrombosis (5–15%) [32]. Atherosclerotic involvement of mesenteric vessels is the most common cause of AMI. Acute obstruction over underlying chronic atherosclerotic disease of mesenteric vessels can present with atypical features like poorly localized pain abdomen, diarrhea, and vomiting [33]. High index of suspicion is required as patients may have history of hospitalization in past with similar complaints in which an exact diagnosis could not be established [34]. The diagnostic accuracy of a CECT (Abd) varies from 67% when performed for routine evaluation to 89–100 % when a clinical diagnosis of AMI was established prior to the investigation [35]. Treatment options include endovascular therapy (mechanical aspiration, stenting) and open revascularization (embolectomy, retrograde recanalization, and stenting of SMA, surgical SMA bypass, damage control surgery ± embolectomy/SMA stenting). The reported mortality rate is lower for endovascular therapy than open revascularization [36].

Diarrhea in elderly

Diarrhea is a common problem in elderly and one which adversely affects quality of life. Infectious diarrhea remains predominant in the developing countries. Drug-induced diarrhea, malabsorption (bacterial overgrowth), diabetes-associated diarrhea, besides uncommon causes like celiac disease, inflammatory bowel disease (IBD), pancreatic insufficiency, small bowel tumors, and amyloidosis may all contribute to impaired quality of life and functional disability [37]. Acute diarrhea warrants maintenance of intravascular volume and empirical antibiotics (if infectious etiology is suspected). For hospitalized patients and those on antibiotics, chemotherapeutic drugs, clostridium difficile infection should be excluded. Immunocompromised individuals require evaluation for atypical organisms like Cryptosporidium, Isospora, Microsporidia, or Microbium avium complex. Chronic diarrhea should be evaluated by thorough history and clinical examination, meticulous drug history, evaluation for malabsorption syndromes, and small bowel series and EGD with duodenal biopsy and/or ileocoloscopy [38].

Small bowel bacterial overgrowth

The overgrowth of bacteria in the small intestine which normally harbors 10 [5, 6] organisms/mL is termed as SIBO [39]. Decreased gastric acid secretion, altered gut motility, breakdown of normal mucosal immune barrier, and anatomical abnormalities predispose to SIBO. Thus, elderly who have co-morbidities like DM which alters the gut motility, hospitalized patients with multiple antibiotics, and multiple drugs including PPI, with structural abnormalities like small intestinal diverticula, are at a greater risk. The disease can have variable presentation: chronic diarrhea, bloating, abdominal distention, and fatigue, leading to complications like nutritional deficiencies, metabolic bone disorders, and malabsorption. Diagnosis at times can be difficult. The direct methods of measuring the bacterial colony counts are particularly cumbersome and have multiple limitations. Breath test indirectly measures bacterial overgrowth based on

bacterial modification of a substrate like glucose, xylose, or lactulose [40]. Modification of substrate by addition of radiolabeled C [13] or C [14] to xylose or glycocholic acid followed by mass spectroscopic measurement of breath samples can also be done. The recent use of antibiotics and PPI interferes in the interpretation. Thus, they need to be discontinued at least 2 weeks prior to testing. During the initial 90 min, the increase from baseline fasting hydrogen concentration by 10–12 ppm after 50-g glucose load or 20 ppm after lactulose consumption is suggestive of SIBO with sensitivity of 60–70 % and specificity of 40–80 % [41]. Treatment involves complimentary role of dietary, pharmacological, and if necessary surgical therapy. Prokinetics may be used in gastroparesis or small bowel dysmotility. Antibiotics reduce overgrowth which in turn reduces mucosal inflammation and thus further reducing chances of overgrowth and malabsorption. The choice and duration of antibiotics are based on weak evidence. Ciprofloxacin, doxycycline, amoxycillin/clavulanic acid, and rifaximin have been tried with duration varying between 7 and 10 days in different trials. The use of probiotic therapy in SIBO is debatable [42].

Large intestine

Diverticular disease of colon

Diverticulosis is the commonest anatomical alteration in the colon. Diverticula are formed when the colonic mucosa and submucosa herniates through the muscular layer of the colonic wall. The site of herniation is usually the part with least resistance, "the site of insertion of vasa recta" [43]. Diverticulosis is expected to be present in almost 50 % of individuals by sixth decade of life. Ten to 25% of these end up with complications like diverticulitis [44]. This disease, which was thought to be rare previously, is now being reported with increasing frequencies in Asian countries as well [45]. The left colon especially the sigmoid colon is the most common site in the Western world. For unknown reasons, the right side is affected commonly in Asian countries. Structural variations in the colonic wall, altered gut motility, and reduced fiber intake have all been postulated to be responsible for the development of diverticula. Another widely accepted but debatable theory is that reduced dietary fiber intake predisposes to diverticular disease. The presence of subclinical inflammation, role of NSAIDs, sedentary lifestyle, and the causative effect of colonic microbiota are also being investigated [45]. Only around 10–25 % individuals with diverticular disease develop symptoms. Diverticulitis or inflammation involving one or the adjacent diverticula may present as acute or subacute onset of lower or left lower quadrant abdominal pain. Fever, vomiting, and recent onset change of bowel habits with elevated white blood cell count/inflammatory markers are usually seen. This inflammatory process may be complicated by development of abscess, fistula, obstruction, hematochezia, or perforation. Other possible differential diagnosis like ischemic colitis, infectious colitis, inflammatory bowel disease, acute appendicitis, and colorectal carcinoma require exclusion. Emergency management requires maintenance of hemodynamic stability and broad-spectrum antibiotic support. CECT(Abd) with intravenous and luminal contrast detects diverticulitis with a sensitivity and specificity of 95 % and 96 %, respectively [46]. Modified Hinchey's classification which is a CECT(Abd)-based classification system is an accepted criterion to manage acute diverticulitis [47]. Surgical management is warranted in a complicated disease setting with development of abscess, stricture, fistula, or perforation. Abscess with diameter < 4 cm may be managed medically while those > 4 cm require either percutaneous or surgical drainage [48]. Laparoscopic-guided aspiration of pus, abdominal lavage with placement of abdominal drains prior to elective surgery has been tried. Resection of colon with Hartman's pouch is the surgical procedure in those presenting with peritonitis with multiple co morbidities. For recurrent uncomplicated diverticulitis, the decision to perform an elective surgery is guided by clinical conditions like frequency of attacks, need for immunosuppression, post-op morbidity, and patients' preferences' [49].

Inflammatory bowel disease (IBD) in elderly

The prevalence of IBD in elderly age group varies from 3 to 21 % for Crohn's disease (CD) to 7–29 % for UC. Elderly have declining immunity, altered gut motility, and alteration in gut microbiota which bring about altered drug metabolism and pharmokinetics [50]. The differential diagnosis includes ischemic colitis, vascular anomalies, malignancy, and drug-induced colitis. For IUC, the disease behavior and presentation more or less resemble that of the younger age group although the first presentation may be severer and elderly individuals are less likely to present with fever and pain abdomen. The left-sided colitis is the most common site (45%) followed, in decreasing order of involvement: pancolitis (31%) and proctitis (22%). For CD, colonic (44%), ileal (32%), and ileocolonic (26%) are involved with the upper GI tract involvement seen in around 7% of cases [51]. Stricturing and penetrating disease is seen in 24% and 8% cases, respectively, while perianal involvement has been seen in around 12 % cases [52]. 5-ASA remains the mainstay of treatment both for IUC and CD. There is general trend towards the restricted use of immunomodulators and biologicals in the elderly [50]. Elderly with IUC is more at risk of undergoing surgery. Total colectomy with permanent ileostomy is the most performed procedure.

Post-operative complications mainly pulmonary and cardiovascular complications are higher in the elderly. The all-cause mortality rates are also higher in the elderly [53].

Constipation in elderly

Constipation is estimated to affect 26 % of all females and 16 % of all males by 65 years of age. In hospitalized patients, this could be as high as 80 % [54, 55]. It has adverse impact on the individuals' quality of life. Classically, constipation can be either primary or secondary. The primary process can have normal transit time through the colon, slow transit, or anorectal dysfunction. Individuals with slow onset constipation have altered colonic motility, characterized by reduced high amplitude propulsive contractions (HAPC), i.e., < 5/day [56]. This can occur in several endocrine and metabolic disorders like hypothyroidism, DM, and hypercalcemia. Delay in colon transit time has a minor role in elderly when compared to other factors like polypharmacy and immobility. Anorectal constipation may occur because of anorectal anomalies, uro–gynecological diseases, or incoordination between pelvic floor muscle relaxation or abdominal muscle contraction [57]. Secondary causes of constipation include systemic diseases, drugs like anticholinergics, NSAIDs, or calcium channel blocker. The present Rome IV criteria describe symptom-based criteria to classify constipation under the following subheadings: functional constipation, anorectal disorders, and opioid-induced constipation. Elderly often relates to straining while passing stools when they describe symptom suggestive of constipation. Thus, a good history training is imperative while analyzing the pathophysiological process leading to constipation. Bristol stool scale is a useful tool to describe the stool consistency in clinical practice [58]. A digital rectal examination (DRE) is useful to detect anorectal tone and pelvic floor pathologies like rectal prolapse/growth/mass or fecal impaction. In the absence of alarm features like GI bleed, fever, weight loss, etc., trial of high fiber diet and laxative can be offered. Secondary causes, if identified, need to be treated. In those who do not respond anorectal manometry should be done to rule out possible outlet obstruction [59]. Balloon expulsion test as a part of anorectal manometry or independently can be done to detect pelvic dyssynergia [60]. High fiber intake, regular physical exercise, and toilet time training needs to be emphasized at every visit. Bulk laxatives (psyllium, methylcellulose) and polyethylene glycol3350 (PEG) are better tolerated. Stool softeners like docusate can be useful in acute setting like post-operative period. Stimulant laxatives like senna should be avoided in chronic setting. Chloride channel activator, Lubiprostone, decreases stool transit time and is a useful adjunct in chronic idiopathic constipation [61]. Guanyl cyclase C receptor-Linaclotide has shown neuromodularity effects in functional constipation [62]. Biofeedback has shown benefit in dyssynergia in combination with standard treatment [63, 64]. Colchicine and Misoprostol are being evaluated for use in refractory chronic constipation in clinical studies. Opioid-induced constipation should be managed with a combination of non-pharmacological methods like bulk laxatives and peripherally acting opioid receptor inhibitors like methylnaltrexone, alvimopam, and naloxegol, especially in refractory cases.

Conclusion

Elderly may present with a large plethora of GI symptoms, non-neoplastic disorders being more common than neoplastic disorders. With this, it is imperative for the treating physician to understand the aging evolution of the GI system and management of GI disorders in the geriatric population.

Abbreviations

GIT	Gastrointestinal tract
DM	Diabetes mellitus
Vit	Vitamin
EGD	Esophagogastroduodenoscopy
OIED	Opioid-induced esophageal dysfunction
GERD	Gastroesophageal reflux disease
tLESR	Transient lower esophageal sphincter relaxation
ENT	Ear, nose, throat
VCE	Video capsule endoscopy
PPIs	Proton pump inhibitors
NSAIDs	Non-steroidal anti-inflammatory drugs
EE	Eosinophilic esophagitis
ASGE	American Society of Gastrointestinal Endoscopy
LGD	Low-grade dysplasia
HGD	High-grade dysplasia
CCK	Cholecystokinin
CT	Computed tomography
AE	Angio ectasia
DL	Dieulofoy's lesion
AVM	Arteriovenous malformation
CKD	Chronic kidney disease
VTE	Venous thromboembolism
SBE	Single-balloon enteroscopy
DBE	Double balloon enteroscopy
SE	Spiral enteroscopy
AMI	Acute mesenteric ischemia
NOMI	Non-occlusive
IBD	Inflammatory bowel disease
SIBO	Small bowel bacterial overgrowth
CD	Crohn's disease
HAPC	High amplitude propulsive contractions

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Authors' contributions

CRediT (Contributor Role Taxonomy) author role in the manuscript (for more information read about the various roles here Dr. Saurabh Dawra: Conceptualization, data curation, formal analysis, investigation/methodology, project administration, resources, software, supervision, validation, visualization, writing—original draft, writing—review & editing, Dr. Pradeep Behl: Conceptualization, data curation, formal analysis, Dr. Sharad Shrivastava:

Conceptualization, writing—review & editing, Dr. Manish Manrai: Conceptualization, formal analysis, supervision/validation, writing—review & editing, Dr. Alok Chandra: Conceptualization, investigation, Dr. Anupam Kumar: Conceptualization, methodology, resources, software, Dr. Ankit Kumar: Conceptualization, methodology, resources, software, Dr. Manveer Singh Tevatia: Conceptualization, investigation. The authors read and approved the final manuscript.

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