International Journal of Gastroenterology Sciences
Online ISSN: 2664-9217, Print ISSN: 2664-9209
Received: 01-11-2018; Accepted: 02-12-2018
www.gastroenterologyjournals.com
Volume 1; Issue 1; January 2019; Page No. 01-07

Diseases of the alimentary tract: A basic review

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Abstract
Alimentary tract diseases (basic review) are to understand very easily for graduate, post graduate and post-doctoral ayush, dental, medical etc., students. I am explaining main and important diseases in alimentary tract in day to day practical life for medical students and professionals. Diseases are gastro oesophageal reflux disease, gastritis, peptic ulcer disease, inflammatory bowel disease.

Keywords: alimentary tract diseases, causes, clinical features, investigation.

Introduction
We have lot of diseases to explain in alimentary tract system. But only main/few diseases are reviewing for under graduate, post graduate and post-doctoral AYUSH, dental, medical, nursing etc., for entrance and main examination purpose.

Gastroesophageal reflux disease
Gastroesophageal reflux disease (GERD) is a very common digestive disorder worldwide with an estimated prevalence of 18.1–27.8% in North America. Approximately half of all adults will report reflux symptoms at some time. It's defined as "Gastroesophageal reflux disease is a condition of troublesome symptoms and complications that result from the reflux of stomach contents into the esophagus". Diagnosis of Gastroesophageal reflux disease is typically based on classic symptoms and response to acid suppression after an empiric trial. Gastroesophageal reflux disease is an important health concern as it is associated with decreased quality of life and significant morbidity. Successful treatment of Gastroesophageal reflux disease symptoms has been associated with significant improvement in quality of life, including decreased physical pain, increased vitality, physical and social function, and emotional well-being. While Gastroesophageal reflux disease medications are not particularly expensive, the cost of treating Gastroesophageal reflux disease patients has been deemed 2-fold more costly than comparable individuals without Gastroesophageal reflux disease [1, 2, 3].

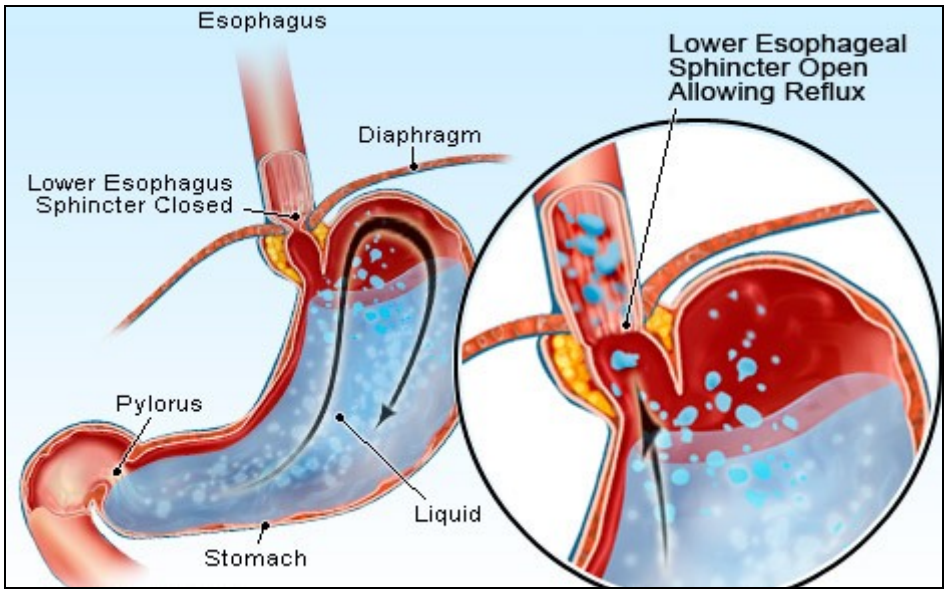


Fig 1: Gastroesophageal reflux disease

Etiology and pathophysiology
Risk factors for Gastroesophageal reflux disease include older age, excessive body mass index (BMI), smoking, anxiety/depression, and less physical activity at work.6–8 Eating habits may also contribute to Gastroesophageal reflux disease, including the acidity of food, as well as size and timing of meals, particularly with respect to sleep. Recreational physical activity appears to be protective, except when performed post prandially. Gastroesophageal reflux is primarily a disorder of the lower esophageal

sphincter (LES) but there are several factors that may contribute to its development. The factors influencing Gastroesophageal reflux disease are both physiologic and pathologic. The most common cause is transient lower esophageal sphincter relaxations (TLESRs). TLESRs are brief moments of lower esophageal sphincter tone inhibition that are independent of a swallow. While these are physiologic in nature, there is an increase in frequency in the postprandial phase and they contribute greatly to acid reflux in patients with Gastroesophageal reflux disease. Other factors include reduced lower esophageal sphincter (LES) pressure, hiatal hernias, impaired esophageal clearance, and delayed gastric emptying [4].

Clinical features

Gastroesophageal reflux disease (GERD) is defined as symptoms or mucosal damage produced by the abnormal reflux of gastric contents into the esophagus or beyond, into the oral cavity (including larynx) or lung. Gastroesophageal reflux disease can be classified as non-erosive reflux disease (NERD) or erosive reflux disease (ERD) based on the presence or absence of esophageal mucosal damage seen on endoscopy. The following document will provide a brief overview of the epidemiology, clinical symptoms and complications of Gastroesophageal reflux disease as well as a more comprehensive review of the current approach to diagnosis and management. Typical symptoms are Acid regurgitation, heartburn, Epigastric fullness, epigastria pressure, epigastric pain, dyspepsia, nausea, bloating, belching (atypical symptoms). Extra oesophagal symptoms are chronic cough, bronchospasm, wheezing, hoarseness, sore throat, asthma, laryngitis, dental erosions [5].

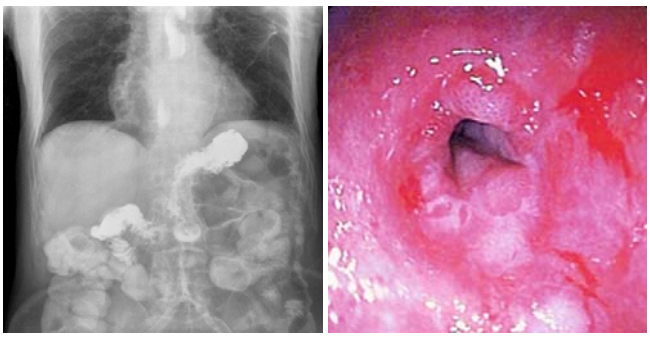


Fig 2: Barium esophagram and Upper endoscopy of Gastroesophageal reflux disease

Complications

- Oesophagitis
- Barrett's oesophagus
- Anaemia
- Benign oesophageal stricture

Diagnosis

The diagnosis of Gastroesophageal reflux disease is typically made by a combination of clinical symptoms, response to acid suppression, as well as objective testing with upper endoscopy and esophageal pH monitoring. For example, the combination of moderate to severe typical symptoms and endoscopic changes (erosive esophagitis or Barrett's esophagus) are highly specific (97%) for Gastroesophageal reflux disease (confirmed with pH testing). However, a well ken history alone can prove very valuable in the diagnosis, especially in the setting of heartburn and acid regurgitation which have a very high specificity (89% and 95%, respectively), albeit low sensitivity (38% and 6%) for Gastroesophageal reflux disease. This can allow one to make a presumptive diagnosis and begin empiric therapy, thereby avoiding a comprehensive and costly evaluation in every patient presenting with uncomplicated symptoms [6, 7].

Ambulatory PH monitoring

Ambulatory reflux monitoring is the only modality allowing direct measurement of esophageal acid exposure, reflux episode frequency and association between symptoms and reflux episodes. It is typically used to evaluate patients with persistent symptoms despite medical therapy, particularly those without endoscopic evidence of Gastroesophageal reflux disease, in order to confirm the diagnosis. It can also be employed to monitor the control of reflux in those on therapy with persistent symptoms

Upper endoscopy

Upper endoscopy is the primary modality used in the evaluation of the esophageal mucosa in patients with Gastroesophageal reflux disease and also allows for biopsies of concerning lesions (*e.g.*, Barrett's metaplasia, strictures or masses). It is important though to understand that there are limitations with the use of upper endoscopy in the diagnosis of Gastroesophageal reflux disease. For instance, while an endoscopy showing esophagitis or Barrett's esophagus essentially confirms the diagnosis of Gastroesophageal reflux disease (high specificity), a normal endoscopy does not refute the diagnosis. In fact, most patients with typical symptoms of Gastroesophageal reflux disease will have no endoscopic evidence of Gastroesophageal reflux disease on esophagogastroduodenoscopy [8. 9].

Barium esophagram

Barium esophagram was once recommended as a screening test for Gastroesophageal reflux disease, but is no longer part of the diagnostic evaluation. A 1996 study of 125 patients compared barium esophagram to esophageal pH monitoring to assess the accuracy of barium screening as a predictor of abnormal esophageal acid exposure. A significantly greater degree of abnormal esophageal acid exposure occurred in patients who had a hiatal hernia or spontaneous reflux on barium radiography. However, the sensitivity and specificity of barium radiography for abnormal degrees of acid reflux were insufficient and therefore this test is no longer recommended in the diagnosis of Gastroesophageal reflux disease [10, 11, 12].

Esophageal manometry

Esophageal manometry is most useful for the evaluation of dysmotility and has only limited utility in the evaluation of Gastroesophageal reflux disease [13]. Gastroesophageal reflux disease is a chronic disease that typically requires long term management in the form of lifestyle modification, medical therapy and, for a subset of patients, surgical therapy and constitutional homoeopathic treatment.

Gastritis

Gastritis is a histological diagnosis, although it can sometimes be recognized at endoscopy.

Acute gastritis

It is often erosive and haemorrhagic. Neutrophils are the predominant inflammatory cell in the superficial epithelium. Many cases result from aspirin or NSAID ingestion. Acute gastritis often produces no symptoms but may cause dyspepsia, anorexia, nausea or vomiting, haematemesis or melaena. Treatment should be directed to the underlying cause. Short term symptomatic therapy with antacids, acid suppression or antimetics, homoeopathic medicines may be necessary [14].

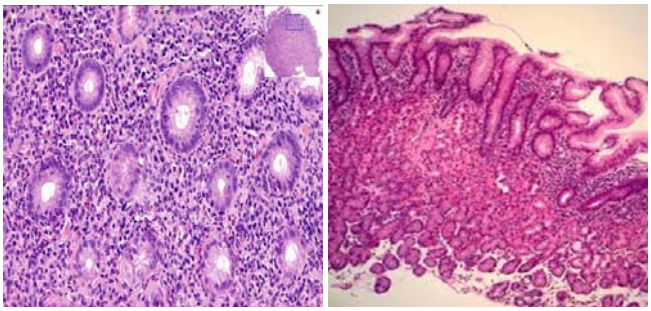


Fig 3: Histopathology of Chronic gastritis

Chronic gastritis

The most common cause of chronic gastritis is H. Pylori. The predominant inflammatory cells are lymphocytes and plasma cells. Correlation between symptoms and endoscopic or pathological findings I spoor. Most patients are asymptomatic and do ot require any treatment. Socioeconomics and environmental hygiene are inevitably the most important background factors in transmission of *H. pylori* infection worldwide, these socioeconomic factors being, thereby, the background factors also in epidemiology of chronic gastritis and its sequelae. The infection rate in childhood and the age-specific prevalence of *H. pylori* gastritis are high in the "old" birth cohorts born decades earlier than the prevalence in the "young" birth cohorts born more recently and in whom the infection rate of *H. pylori* at childhood is low. Thus, the mean prevalence of gastritis at the population level reflects the average of the prevalences of chronic gastritis in different birth cohorts, and the mean rate of *H. pylori* infection at pediatric age [15, 16].

Peptic ulcer disease

The term peptic ulcer refers to an ulcer in the lower oesophagus, stomach or duodenum in the jejunum after surgical anastomosis to the stomach, or, rarely in the ileum adjacent to a meckel's diverticulum. Ulcer in the stomach or duodenum may be acute or chronic: both penetrate the muscularis mucosae but the acute ulcer shows no evidence of fibrosis.

Etiology

Peptic ulcer disease includes both gastric and duodenal ulcers which posed a major threat to the world's population over the past two centuries with a high morbidity and mortality. The evolution of knowledge regarding etiopathogenesis of peptic acid disease from acid-driven disease to an infectious disease has opened up this topic for various studies to find the best possible options for management of this disease. The discovery of *Helicobacter pylori* has evinced great interest in the role played by this microbe. The eradication of this organism has been found to be of paramount importance to minimize the complications of peptic ulcers. The management of peptic ulcer disease and its complications remain a challenge. In addition, non-steroidal anti-inflammatory drugs (NSAIDs), low-dose aspirin, smoking, excessive alcohol use, emotional stress and psychosocial factors are increasingly important causes of ulcers and their complications even in *H. pylori* negative patients. Other rare causes of peptic ulcer disease in the absence of *H. pylori*, NSAIDs, and aspirin also exist. Epidemiological studies reveal a very strong association between *H. pylori* infection and peptic ulcer disease. More than half the world's population has a chronic *H. pylori* infection of the gastroduodenal mucosa, yet only 5-10% develops ulcers. Factors that determine whether the infection will produce the disease depends on the pattern of histological changes, gastritis induced changes in homeostasis of gastric hormones and acid secretion, gastric metaplasia in the duodenum, interaction of *H. pylori* with the mucosal barrier, immunopathogenesis, ulcer genic strains, and genetic factors. Management of peptic acid disease varies from using H_2 receptor antagonist, proton pump inhibitors (PPI) to triple chemotherapy and sequential regimen for *H. pylori*. Similarly treating perforation varies from a conservative non operative approach to a surgical approach [17, 18].

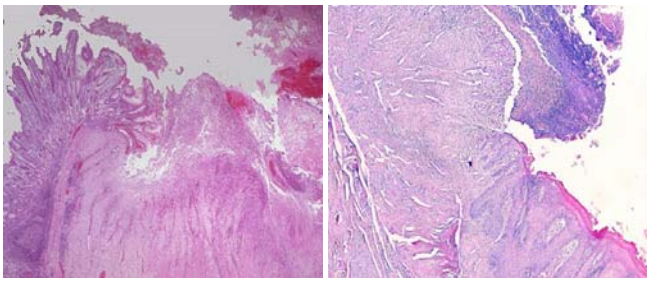
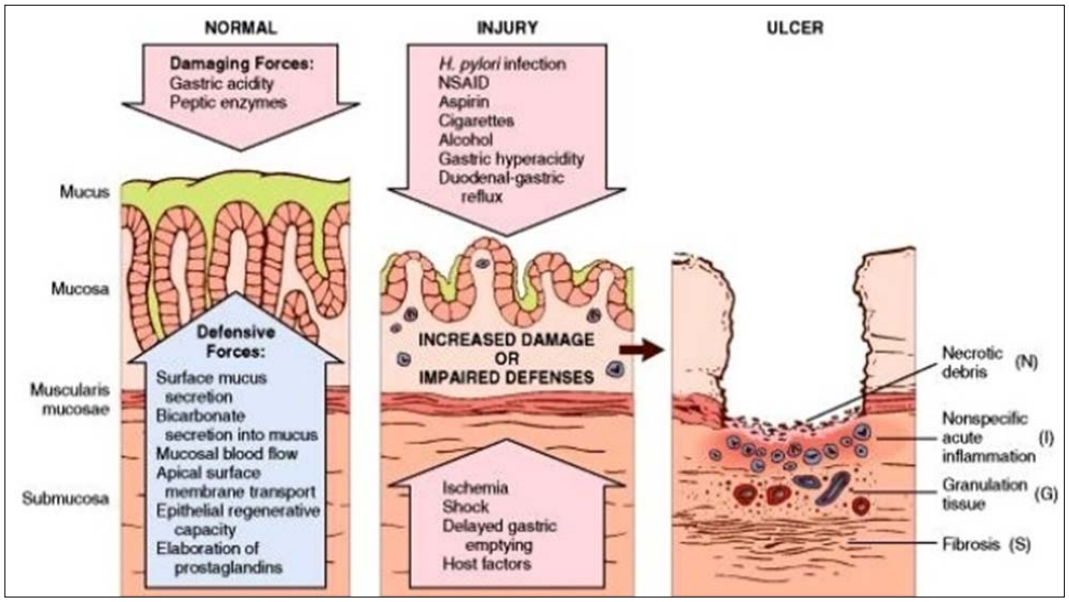


Fig 4: Histopathology of peptic ulcer

Pathology

Chronic gastric ulcer is usually single, 90% are situated on the lesser curve within the antrum or at the junction between body and antral mucosa. Chronic duodenal ulcer usually occurs in the first part of the duodenum just distal to the junction of pyloric and duodenal mucosa, 50% are on the anterior wall. Gastric and duodenal ulcers coexist in 10% of patients and more than one peptic ulcer is found in 10 -15% of patients [19, 20].



A chronic ulcer extends to below the muscularis mucosa and the histology shows four layers: surface debris, an infiltrate of neutrophils, granulation tissue and collagen.

Fig 5: Pathophysiology of Peptic ulcer

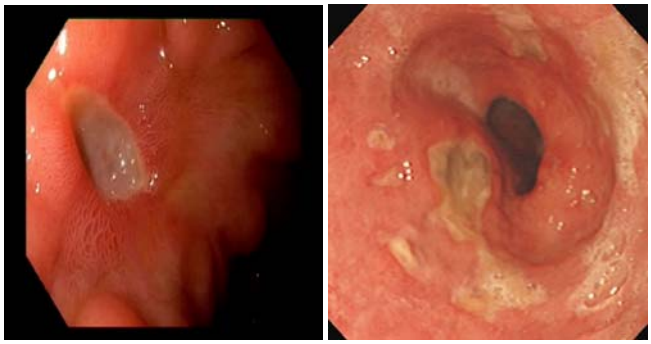


Fig 6: Endoscopy of peptic ulcer

Clinical features

Peptic ulcer disease is a chronic condition with a natural history of spontaneous relapse and remission lasting for decades. Pain is referred to the epigastrium and is often so sharply localized that the patient can indicate its site with two or three fingers(the pointing sign). Hunger pain occurs intermittently during the day, often when the stomach is empty. Pain wakes the patients from sleep and may be relieved by food, a drink of milk or antacids; this symptoms is very characteristic of duodenal ulcer. Pain is ameliorated by food, milk and by belching and vomiting. Relief by vomiting is more typical of gastric ulcer than of duodenal ulcer. Periodicity pain present and last for several weeks at a time. Between episodes the patient feels perfectly well. Other symptoms that occur, especially during episodes of pain, include water brush, heartburn, loss of appetite and vomiting. Persistent vomiting occurring daily suggests gastric outlet obstruction [21, 22].

Investigation

The diagnosis can be made by double contrast barium meal examination or by endoscopy. Endoscopy is the preferred investigation because it is more accurate and has the enormous advantage that suspicious lesions and HP status can be evaluated by biopsy.

Complication

- Perforation.
- Gastric outlet obstruction.
- Bleeding

Management

Cigarette smoking, aspirin and NSAIDs should be avoided. Alcohol in moderation is not harmful and no special dietary advice is required, but to avoid very spicy food. All patients with proven acute or chronic duodenal ulcer disease and those with gastric ulcer who are helicobater pylori positive should be offered eradication therapy as primary therapy. Treatment is based upon a proton pump inhibitor taken simultaneously with 2 antibiotics for one week and along with homoeopathic treatment.

Inflammatory bowel disease

Ulcerative colitis and crohn's disease are chronic inflammatory bowel disease which pursues a protracted relapsing and remitting course, usually extending over years. The incidence of inflammatory bowel disease (IBD) varies widely between populations: Crohns disease appears to be very rare in the underdeveloped world yet ulcerative colitis, although still unusual, is becoming more common.

Ulcerative colitis

Ulcerative colitis (UC) is a chronic disease with recurrent uncontrolled inflammation of the colon. The rectum is always affected with inflammation spreading from the distal to the proximal colonic segments. The terminal ileum is typically not involved but some patients with extensive disease may show endoscopic signs of "backwash ileitis". As the course of disease and extent vary considerably among patients, an individualized diagnostic and therapeutic approach is necessary.

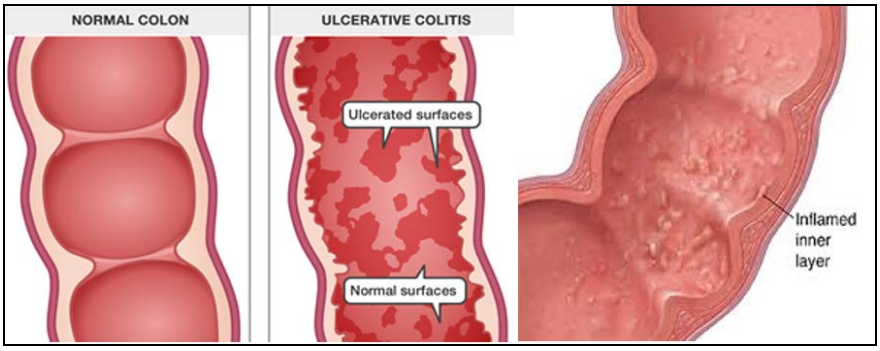


Fig 7: ulcerative colitis

Only a minority of patients have chronic, unremitting symptoms. Emotional stress, intercurrent infection, gastroenteritis, antibiotics or NSAID therapy may provoke a relapse. Rectal bleeding and mucus discharge. Some-time accompanied by tenesmus.

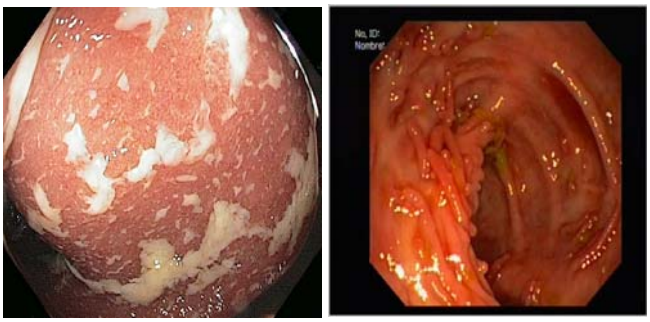


Fig 8: Endoscopy of ulcerative colitis

The disease remains confined to the rectum in approximately 25% of cases, and in the remainder of cases, ulcerative colitis spreads proximally and contiguously. Pancolitis occurs in 10% of patients. The distal terminal ileum may become inflamed in a superficial manner, referred to as backwash ileitis. Even with less than total colonic involvement, the disease is strikingly and uniformly continuous. As ulcerative colitis becomes chronic, the colon becomes a rigid foreshortened tube that lacks its usual haustral markings, leading to the lead pipe appearance observed on barium enema.

Clinical features

Some patients pass frequent, small volume fluid stools, while other are constipated and pass pellet stools. Weight loss, malaise, anorexia and abdominal pain occur and the patient is toxic with fever.

Complications

- Severe, life threatening inflammatory of the colon.
- Perforation of the small intestine or colon.
- Life threatening acute haemorrhage.
- Fistula and perianal disease.
- Cancer.
- Seronegative arthritis.
- Erythema nodosum, pyoderma ganrenosum, oral aphthous ulcers, conjunctivitis, iritis, primary sclerosing cholangitis, gall stones, fatty liver, portal pyaemia, liver abscess, amyloidosis, oxalate calculi, deep vein thrombosis, portal or mesenteric vein thrombosis.

Investigations

Blood tests are like complete blood test, vit B12, ESR, serum albumin etc., Bacteriology like stool cultures are performed to exclude superimposed enteric infection in patients who present with exacerbations of inflammatory bowel disease. Sigmoidoscopy, barium studies, plain radiographs and radionuclide scans.

Management

Drug treatment, nutritional therapy, surgical treatment and homoeopathic medicines.

Crohns disease

The sites most commonly involved in order of frequency are terminal ileum and right side of colon, colon alone, terminal ileum alone, ileum and jejunum. Characterized, the entire wall of the bowel is oedematous and thickened. There are deep ulcers which often appear as linear fissures, thus the mucosa between them is described as cobblestone. Deep ulcer may penetrate through the bowel wall to imitate abscesses or fistulae. Its prevalence has continually increased over the past 50 years with the highest incidence being reported in northern Europe, the United Kingdom and North America [23, 24].

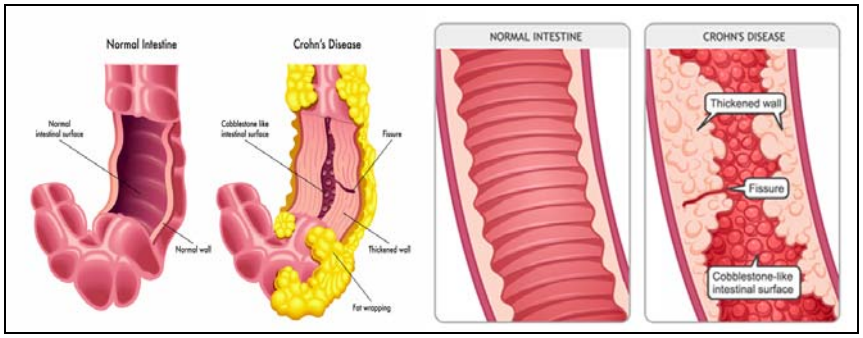


Fig 9: Crohns disease

Fistula may develop between adjacent loops of bowel or between affected segmens of bowel and the bladder, uterus or vagina and may appear in the perineum. Characteristically, the changes are patchy. Even when a relatively short segment of bowel is affected, the inflammatory process is interrupted by islands of normal mucosa and the change from the affected part is abrupt. A small lesion separated in this way from a major area of involvement is referred to as a "skip" lesion. The mesenteric lymph nodes are enlarged and the mesentery thickened. Histologically, chronic inflammation is seen through all the layer of the bowel wall, which is thickened as a result. There are focal aggregates of epithelioid histiocytes, which may be surrounded by lymphocytes and contain giant cells. Lymphoid aggregates or microgranulomas are also seen, and when these are near to the surface of the mucosa they often ulcerate to form tiny aphthous like ulcers [25].

Clinical features

Chronic diarrhoea, defined as a decrease in faecal consistency for more than 4 weeks, is the most common presenting symptom. Abdominal pain (70%), weight loss (60%) and blood, mucus or both in stools (40–50%) are also common findings in Crohns disease. Extraintestinal manifestations affect approximately a third of patients with inflammatory bowel disease. The most commonly observed extraintestinal manifestation is primary peripheral arthritis (33%); aphthous stomatitis, uveitis, erythema nodosum and ankylosing spondylitis can be seen whilst pyoderma gangrenosum, psoriasis and primary sclerosing cholangitis are relatively uncommon. Fistulae, a complication of Crohns disease, occurs in up to 35% of patients with Crohns disease, with perianal fistula occurring in 20%.

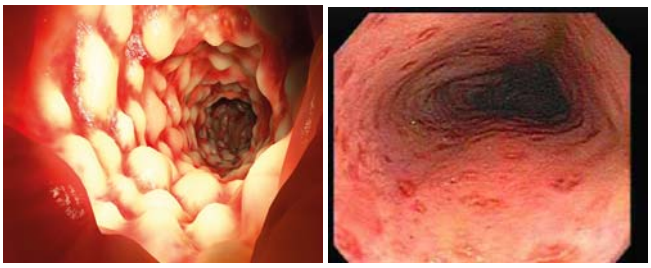


Fig 10: Endoscopy of Crohns disease

Risk factors

Crohns disease has a peak age prevalence of 30–39 years old and gender influence differs in various demographics. In a Canadian and New Zealand population, females are 10–30% more likely to acquire the disease than males. Other inflammatory diseases have been implicated with Crohns disease including asthma, psoriasis, pericarditis, ankylosing spondylitis, atopic dermatitis and primary sclerosing cholangitis. Their impact tends to be most influential during childhood.

Different diagnosis:

- Other cases of right iliac fossa mass: caecal carcinoma, appendix abscess, infection (tuberculosis, yersinia, actinomycosis), mesenteric adenitis, pelvic inflammatory disease, lymphoma.

Management

Intravenous fluids, antibiotics for proven infection, nutritional support, avoidance of opiates, antidiarrhoeal agents and homooepathic management.

References

1. El-Serag HB, Sweet S, Winchester CC, Dent J. Update on the epidemiology of gastro-oesophageal reflux disease: a systematic review. Gut. 2014; 63:871-880.
2. Locke GR, 3rd, Talley NJ, Fett SL, Zinsmeister AR, Melton LJ. 3rd Prevalence and clinical spectrum of gastroesophageal reflux: a population-based study in Olmsted County, Minnesota. Gastroenterology. 1997; 112:1448-1456.
3. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R. Global Consensus Group. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol. 2006; 101:1900-1920.
4. Bloom BS, Jayadevappa R, Wahl P, Cacciamanni J. Time trends in cost of caring for people with gastroesophageal reflux disease. Am J Gastroenterol. 2001; 96:S64-69.
5. Zheng Z, Nordenstedt H, Pedersen NL, Lagergren J, Ye W. Lifestyle factors and risk for symptomatic gastroesophageal reflux in monozygotic twins. Gastroenterology. 2007; 132:87-95.
6. Tefera L, Fein M, Ritter MP, Bremner CG, Crookes PF, Peters JH, Hagen JA, DeMeester TR. Can the combination of symptoms and endoscopy confirm the presence of gastroesophageal reflux disease? Am Surg. 1997; 63:933-936.
7. Giannini EG, Zentilin P, Dulbecco P, Vigneri S, Scarlata P, Savarino V. Management strategy for patients with gastroesophageal reflux disease: a comparison between empirical treatment with esomeprazole and endoscopy-oriented treatment. Am J Gastroenterol. 2008; 103:267-275.
8. Katzka DA, Paoletti V, Leite L, Castell DO. Prolonged ambulatory pH monitoring in patients with persistent gastroesophageal reflux disease symptoms: testing while on therapy identifies the need for more aggressive anti-reflux therapy. Am J Gastroenterol. 1996; 91:2110-2113.
9. Kwiatek MA, Pandolfino JE. The Bravo pH capsule system. Dig Liver Dis. 2008; 40:156-160.
10. Aksglaede K, Funch-Jensen P, Thommesen P. Intra-esophageal pH probe movement during eating and talking. A videoradiographic study. Acta Radiol. 2003; 44:131-135.
11. Ravi K, Francis DL. New technologies to evaluate esophageal function. Expert Rev Med Devices. 2007; 4:829-837.
12. Weusten BL, Roelofs JM, Akkermans LM, Van Berge-Henegouwen GP, Smout AJ. The symptom-association probability: an improved method for symptom analysis of 24-hour esophageal pH data. Gastroenterology. 1994; 107:1741-1745.
13. DeVault K, McMahon BP, Celebi A, Costamagna G, Marchese M, Clarke JO *et al*. Defining esophageal

Landmarks, gastroesophageal reflux disease, and Barrett's esophagus. Ann NY Acad Sci. 2013; 1300:278-295.
14. Schindler R. Chronic gastritis. Klin Wochenschr. 1966; 44:601-12.
15. Siurala M. The story of gastritis. Scand J Gastroenterol Suppl. 1991; 186:1-3.
16. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol. 1996; 20:1161-81.
17. Lau WY, Leow CK. History of perforated duodenal and gastric ulcers. World J Surg. 1997; 21:890-6.
18. Bertleff MJ, Lange JF. Perforated peptic ulcer disease: A review of history and treatment. Dig Surg. 2010; 27:161-9.
19. Lagoo S, McMahon RL, Kakihara M, Pappas TN, Eubanks S. The sixth decision regarding perforated duodenal ulcer. JSLS. 2002; 6:359-68.
20. Van Doorn LJ. Detection of *Helicobacter pylori* virulence-associated genes. Expert Rev Mol Diagn. 2001; 1:290-8.
21. Van Doorn LJ, Figueiredo C, Sanna R, Plaisier A, Schneeberger P, de Boer W *et al*. Clinical relevance of the cagA, vacA, and iceA status of *Helicobacter pylori*. Gastroenterology. 1998; 115:58-66.
22. Ballinger A, Smith G. COX-2 inhibitors vs. NSAIDs in gastrointestinal damage and prevention. Expert Opin Pharmacother. 2001; 2:31-40.
23. Beaugerie L, Seksik P, Nion-Larmurier I, Gendre J, Cosnes J. Predictors of Crohn's disease. Gastroenterology. 2006; 130:650-656.
24. Bernstein C, Blanchard J, Rawsthorne P, Yu N. The prevalence of extraintestinal diseases in inflammatory bowel disease: a population-based study. Am J Gastroenterol. 2001; 96:1116-1122.
25. Buisson A, Chevaux J, Allen P, Bommelaer G, Peyrin-Biroulet L. Review article: the natural history of postoperative Crohn's disease recurrence. Aliment Pharmacol Ther. 2012; 35:625-633.