**INTESTINAL OBSTRUCTION**

The causes of intestinal obstruction can be classified under the following 3 broad groups:

**1. Mechanical obstruction:**

1. *internal obstruction (intramural and intraluminal):*
2. Inflammatory strictures (e.g. Crohn’s disease)
3. Congenital stenosis, atresia, imperforate anus
4. Tumours
5. Roundworms
6. Gallstones, faecoliths, foreign bodies
7. *External compression*:
8. Peritoneal adhesions and bands
9. Strangulated hernias
10. Intussusception
11. Volvulus
12. Intra-abdominal tumour.

**2. Neurogenic obstruction:** It occurs due to paralytic ileus i.e. paralysis of muscularis of the intestine after abdominal operation or by acute peritonitis.

**3. Vascular obstruction:**

1. Thrombosis
2. Embolism
3. Accidental ligation.

Out of the various causes listed above, conditions producing external compression on the bowel wall are the most common causes of intestinal obstruction (80%).

* Patient present with abdominal pain and vomotting. Patient not pass stool or gases
* On examination there will be absence of bowel sound
* Major complications are 1) necrosis of the affected segment 2) loss of fluid and electrolytes.

**INTUSSUSCEPTION**

Intussusception is the telescoping of a segment of intestine into the segment below due to peristalsis. The telescoped segment is called the *intussusceptum* and lower receiving segment is called the *intussuscipiens.* The condition occurs more commonly in infants and young children. The main *complications* of intussusception are intestinal obstruction,infarction, gangrene, perforation and peritonitis.

**VOLVULUS**

Volvulus is the twisting of loop of intestine upon itself through 180° or more. This leads to obstruction of the intestine as well as cutting off of the blood supply to the affected loop. The usual causes are **bands and adhesions** (congenital or acquired).

**INFLAMMATORY BOWEL DISEASE**

**(CROHN’S DISEASE AND ULCERATIVE COLITIS)**

**DEFINITION.** The term ‘inflammatory bowel disease (IBD)’ is commonly used to include 2 idiopathic bowel diseases having many similarities but the conditions usually have distinctive morphological appearance. Both diseases produce inflammation of the bowel, both lack confirming evidence of a proven causative agent, both have a pattern of familial occurrence, and both can be accompanied by systemic manifestations.

**Pathogenesis:**

**1. Genetic factors.** Genetic factors are implicated in the etiopathogenesis of IBD. HLA studies show that ulcerative colitis is more common in HLA-DRB1- alleles while Crohn’s disease is more common in HLA-DR7 and DQ4 alleles.

**2. Immunologic factors.**

In both types of IBD, activated CD4+ T helper (TH) cells are present in the lamina propria and in the peripheral blood. There are two main types of CD4+ T cells in IBD:

1. *TH1 cells* secrete proinflammatory cytokines IFN-γ and TNF which induce transmural granulomatous inflammation seen in Crohn’s disease. IL- 12 initiates TH1 cytokine pathway (cell mediated immune pathway).
2. *TH2 cells* secrete IL-4, IL-5 and IL-13 which induce superficial mucosal inflammation characteristically seen in ulcerative colitis (humeral immune response pathway).

**3. Exogenous factors.**

i) Microbial infection

ii) Psychosocial factors

iii) Smoking and

iv) Oral contraceptives.

Both these disorders primarily affect the bowel but may have systemic involvement in the form of polyarthritis, uveitis, ankylosing spondylitis, skin lesions and hepatic involvement. Both diseases can occur at any age but are more frequent in 2nd and 3rd decades of life. Females are affected slightly more often.

**1. Crohn’s disease or Regional enteritis** is an idiopathic chronic ulcerative IBD, characterised by transmural, non-caseating granulomatous inflammation, affecting most commonly the segment of terminal ileum and/ or colon, though any part of the gastrointestinal tract may be involved.

***G/A*** Characteristic feature is the multiple, sharply demarcated, granulomatous lesions that are surrounded by normal-appearing mucosal tissue called ***skip lesions***: segmental bowel involvement with intervening uninvolved ‘skip areas’.

***M/E:*** The features are as under:

1. *Transmural inflammatory cell infiltrate* consisting of chronic inflammatory cells.

2. *Non-caseating granulomas* are present in all the layers of the affected bowel wall in 60% of cases.

3. There is *patchy ulceration* of the mucosa which may take the form of deep fissures.

4. There is *widening of the submucosa* due to oedema and foci of lymphoid aggregates.

5. In more *chronic cases,* fibrosis becomes increasingly prominent in all the layers disrupting muscular layer.

**2. Ulcerative colitis** is an ulcerative colitis affecting chiefly the mucosa and submucosa of the rectum and descending colon, though sometimes it may involve the entire length of the large bowel.

***G/A*** The characteristic feature is the continuous involvement of the rectum and colon without any uninvolved skip areas when compared to Crohn’s disease. The intervening intact mucosa may form inflammatory ‘pseudopolyps.’

***M/E*** Ulcerative colitis because of remission and exacerbations, is characterised by alternating ‘active disease process’ and ‘resolving colitis.’The changes in the ‘active disease process’ are :

1. *Crypt distortion, cryptitis* and focal accumulations of neutrophils forming *crypt abscesses.*

2. *Marked congestion, dilatation and haemorrhages*.

3. *Superficial mucosal ulcerations.*

4. *Goblet cells* are markedly *diminished* in cases of active disease.

5. Areas of *mucosal regeneration and mucodepletion* of lining cells.

6. In long-standing cases, epithelial *cytologic atypia* , dysplasia which may progress to carcinoma *in situ* and adenocarcinoma.

Comparison between Ulcerative colitis and Crohn's disease:

|  |  |  |
| --- | --- | --- |
|  | **U.C** | **Crohn's disease** |
| **Incidence** | **More common** | **Less common** |
| **Site** | **Rectum always involved and extend proximally,it may involve whole colon** | **Any part of GIT from**  **mouth to anus** |
| **Pattern of involvement** | **Continuous** | **Skip lesion** |
| **Gross** | **Diffuse ulceration with pseudo-polyp** | **Cobblestone with fissure** |
| **Fistula** | **Less common** | **Common** |
| **Serosa** | **Normal (disease of mucosa and**  **submucosa )** | **Inflamed (transmural )** |
| **Fibrosis** | **Mild** | **Marked** |
| **Granuloma** | **No** | **60%** |
| **Risk of malignant transformation** | **5%** | **Very rare** |
| **Stricture and intestinal obstruction** | **Late or rarely** | **Early** |
| **Wall appearance** | **Thin** | **Thickened** |
| **Dilatation** | **Yes** | **No** |

**COMPLICATIONS.** These are:

**Crohn’s disease:**

1) Malabsorption, 2) Fistula formation, 3) Stricture formation and 4) Development

of malignancy.

**Ulcerative colitis:**

1) Toxic megacolon (Fulminant colitis), 2) Perianal fistula formation,

3) Carcinoma and 4) Stricture formation.

**INTESTINAL TUBERCULOSIS:**

**1. PRIMARY INTESTINAL TUBERCULOSIS:** an uncommon disease in the developed countries of the world, primary tuberculosis of the ileocecal region is quite common in developing countries. Occur by ingestion of unpasteurized cow’s milk infected with *Mycobacterium bovis.* But now-a-days due to control of tuberculosis in cattle and pasteurisation of milk, virtually all cases of intestinal tuberculosis are caused by *M. tuberculosis.*

***G/A*** The affected lymph nodes are enlarged, matted and there is healing by fibrosis and calcification

***M/E*** In the initial stage, there is primary complex or Ghon’s focus in the intestinal mucosa as occurs elsewhere in primary tuberculous infection. Subsequently, the mesenteric lymph nodes are affected which show typical tuberculous granulomatous inflammatory reaction with caseation necrosis.

**2. SECONDARY INTESTINAL TUBERCULOSIS.** Self-swallowing of sputum in patients with active pulmonary tuberculosis may cause secondary intestinal tuberculosis, most commonly in the terminal ileum and rarely in the colon.

***G/A*** The intestinal lesions are prominent than the lesions in regional lymph nodes as in secondary pulmonary tuberculosis. The lesions begin in the Peyer’s patches or the lymphoid follicles with formation of small ulcers that spread through the lymphatics to form large ulcers which are *transverse to the long axis of the bowel*. In advanced cases, transverse fibrous strictures and intestinal obstruction are seen.

***M/E*** The granulomatous tuberculous lesions in the intestine are similar to those observed elsewhere. Mucosa and submucosa show ulceration and the muscularis may be replaced by variable degree of fibrosis.

***Clostridium Difficile* Colitis**

*C. difficile*, which is part of normal flora in 2% to 10% of humans, is a gram-positive spore-forming bacillus that has been implicated as the offending organism in colitis associated with antibiotic therapy. The spores are resistant to the acid environment of the stomach and convert to vegetative forms in the colon. Treatment with broad-spectrum antibiotics predisposes to disruption of the normal bacterial flora of the colon, leading to colonization by *C. difficile*. Almost any antibiotic may cause

*C. difficile* colitis, but broad-spectrum antibiotics with activity against the gram-negative bacteria of the normal intestinal flora are the most common agents.

**DYSENTERIES**

The term ‘dysentery’ is used to mean bloody diarrhea with abdominal cramps, tenesmus and passage of mucus in the stools, from any cause. There are 2 main forms of dysenteries.

**1. BACILLARY DYSENTERY.** Bacillary dysentery is the term used for infection by *shigella* species: *S. dysenteriae, S. flexneri, S. boydii and S.sonnei.*

route of infection:Infection occurs by foeco-oral route

predisposing factors: 1) poor personal hygiene, 2) in densely populated areas, 3) ingestion of contaminated food and water.

The common housefly plays a role in spread of infection.

***G/A*** The lesions are mainly found in the colon and occasionally in the ileum. Superficial transverse ulcerations of mucosa of the bowel wall occur in the region of lymphoid follicles but perforation is seldom seen.

***M/E*** The mucosa overlying the lymphoid follicles is necrosed. The surrounding mucosa shows congestion, oedema and infiltration by neutrophils and lymphocytes. The mucosa may be covered by greyish-yellow ‘*pseudomembrane’* composed of fibrinosuppurative exudate.

**2. AMOEBIC DYSENTERY.** This is due to infection by *Entamoeba histolytica.* It is more prevalent in the tropical countries and primarily affectsthe large intestine. Infection occurs from ingestion of cyst form of theparasite.

***G/A*** flask-shaped ulcers having narrow neck and broad base are seen. They are more conspicuous in the caecum, rectum and in the flexures.

***M/E*** The ulcerated area shows chronic inflammatory reaction consisting of lymphocytes, plasma cells, macrophages and eosinophils. The trophozoites of *Entamoeba* are seen in the inflammatory exudate and are concentrated at the advancing margin of the lesion. Intestinal amoebae characteristically have ingested red cells in their cytoplasm.

**Irritable Bowel Syndrome IBS**

The term *irritable bowel syndrome* is used to describe a functional gastrointestinal disorder characterized by a variable combination of chronic and recurrent intestinal symptoms not explained by structural or biochemical abnormalities. There is evidence to suggest that 10% to 20% of people in Western countries have the disorder. The condition is characterized by persistent or recurrent symptoms of **abdominal pain, altered bowel function, and varying complaints of flatulence, nausea and anorexia, and anxiety or depression.** Irritable bowel syndrome is believed to result from:

1. Dysregulation of intestinal motor and sensory functions modulated by the CNS. Persons with irritable bowel syndrome tend to experience increased motility and incomplete digested food enters the colon.
2. Abnormal intestinal contractions in response to psychological and physiologic stress.
3. Change in intestinal flora

**Diverticular Disease**

***Diverticulosis*** is a condition in which the mucosal layer of the colon herniates through the muscularis layer. Often, there are multiple diverticula, and most occur in the sigmoid colon. Diverticular disease is common in Western society, affecting approximately 5% to 10% of the population older than 45 years and almost 80% of those older than 85 years. This suggests that dietary factor (*e.g.*, lack of fiber content), a decrease in physical activity, and poor bowel habits (*e.g.*, neglecting the urge to defecate), along with the effects of aging, contribute to the development of the disease.

***Diverticulitis***is a complication of diverticulosis in which there is inflammation and gross or microscopic perforation of the diverticulum. One of the most common complaints of diverticulitis is **pain in the lower left quadran**t, may accompanied by nausea and vomiting, tenderness in the lower left quadrant, a slight fever, and an elevated white blood cell count. These symptoms usually last for several days, unless complications occur. Complications include abscess, perforation with peritonitis, hemorrhage, Fistulas formation especially vesicosigmoidal (ie bladder) fistula .

**MALABSORPTION SYNDROME**

DEFINITION AND CLASSIFICATION

The malabsorption syndrome (MAS) is characterized by impaired intestinal absorption of nutrients especially of fat; some other substances are proteins, carbohydrates, vitamins and minerals. MAS is subdivided into 2 broad groups:

**Primary MAS,** which is due to primary deficiency of the absorptive mucosal surface and of the associated enzymes.

**Secondary MAS:** in which mucosal changes result secondary to other factors such as diseases, surgery, trauma and drugs.

**Clinical features**: persons with intestinal malabsorption usually have symptoms directly referable to the gastrointestinal tract that include diarrhea, steatorrhea (fatty stools. If person consuming a diet containing 80 to 100 g of fat each day, excretion of 7 to 9 g of fat indicates steatorrhea ), flatulence, bloating, abdominal pain, and cramps. Weakness, muscle wasting, weight loss despite normal or excessive caloric intake, and abdominal distention often are present. Failure to absorb the fat-soluble vitamins can lead to easy bruising and bleeding (*i.e.*, vitamin K deficiency), bone pain, a predisposition to the development of fractures and tetany (*i.e.*, vitamin D and calcium deficiency), macrocytic anemia, and glossitis (*i.e.*, folic acid deficiency). Neuropathy, atrophy of the skin, and peripheral edema may be present. MAS involve the following diseases:

1. Celiac disease
2. Pancreatic insufficiency (exocrine glands)
3. Crohns disease

**Celiac Sprue (Non-tropical Sprue, Gluten-Sensitive Enteropathy,**

**Idiopathic Steatorrhoea)**

This is the most important cause of primary malabsorption. The condition is characterised by significant loss of villi in the small intestine and hence diminished absorptive surface area due to an immunologic response to the gliadin fraction of gluten.

**Pathogenesis:**

1. There is an association with certain type II human leukocyte antigens (HLA). HLA-DQ2 is found in up to 95% of patients with celiac disease. The remaining patients mainly have HLA-DQ8.
2. Celiac disease results from activation of both a cell-mediated (TH1-cell) and humoral (TH2 B-cell) immune response. This activation results from exposure to the glutens.
3. Environmental factor eg microbial infection(eg rota virus)

Gliadin peptides pass through the epithelial barrier and activate T-lymphocytes located in the lamina properia. Activation of CD4+ T-lymphocytes occur when macrophages and other APC present gliadin with either HLA-DQ2 or HLA-DQ8 to CD4+ T-lymphocytes. Activated CD4+ T-lymphocytes produce high levels of pro-inflammatory cytokines, inducing both T-helper 1 immune response pattern dominated by IFN-γ, and a T-helper 2 pattern, which causes a clonal expansion of B-lymphocytes that subsequently differentiate in plasma-cells secreting anti-gliadin and anti-tissue-transglutaminase antibodies.

The symptoms are usually relieved on elimination of gluten from the diet.

Tissue samples taken from subjects affected by celiac disease mainly show:

(1) Decreased enterocyte height;

(2) Crypt hyperplasia;

(3) Villous atrophy;

(4) Increased intraepithelial T lymphocytes.

**Benign lesions of the intestine:**

By far the most common types of benign growth of the intestine are polyps. A gastrointestinal polyp can be described as a mass that protrudes into the lumen of gut.

**Classification:**

I//Polyps can be subdivided according to their attachment to the bowel wall

1. sessile [raised mucosal nodules]
2. pedunculated [attached by a stalk])

II//their histopathologic appearance

1. hyperplastic
2. adenomatous

III// their neoplastic potential

1. benign
2. malignant

**Types:**

**I//non neoplastic Polyps:** non neoplastic benign lesion of the colon and rectum include the followings:

hyperplastic polyp, inflammatory polyp, hamartomatous polyp and lymphoid polyp.

**II//Adenomatous polyp: intraepithelial** neoplasm that range from small, often pedunculated lesions to large neoplasm that is usually sessile. Its prevalence increases progressively with age. Males and females affected equally. It composed the followings:

1. Tubular adenomas: composed of tubular glands
2. Villous adenomas: composed of villous projections
3. Tubulovillous adenomas: mixed

Adenomatous polyp usually associated with dysplasia which range from low to high grade dysplasia. Adenomatous polyp is a precursor for invasive colorectal adenocarcinoma (risk of malignancy 40%).

III//**Familial polyposis syndrome: familial adenomatous polyposis (FAP) is the commonest type.**

It is AD genetic disorder due to mutation in adenomatous polyposis coli gene (APC) on chromosome number 5. The presence of 100 polyps is necessary for diagnosis. Life time risk of cancer development is 100%. Some patients already have cancer of colon or rectum at time of diagnosis. Cancer prevention measures include early detection of the condition and prophylactic colectomy.