**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.