ULCERATIVE COLITIS/CHRONIC COLITIS

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GROUP 11

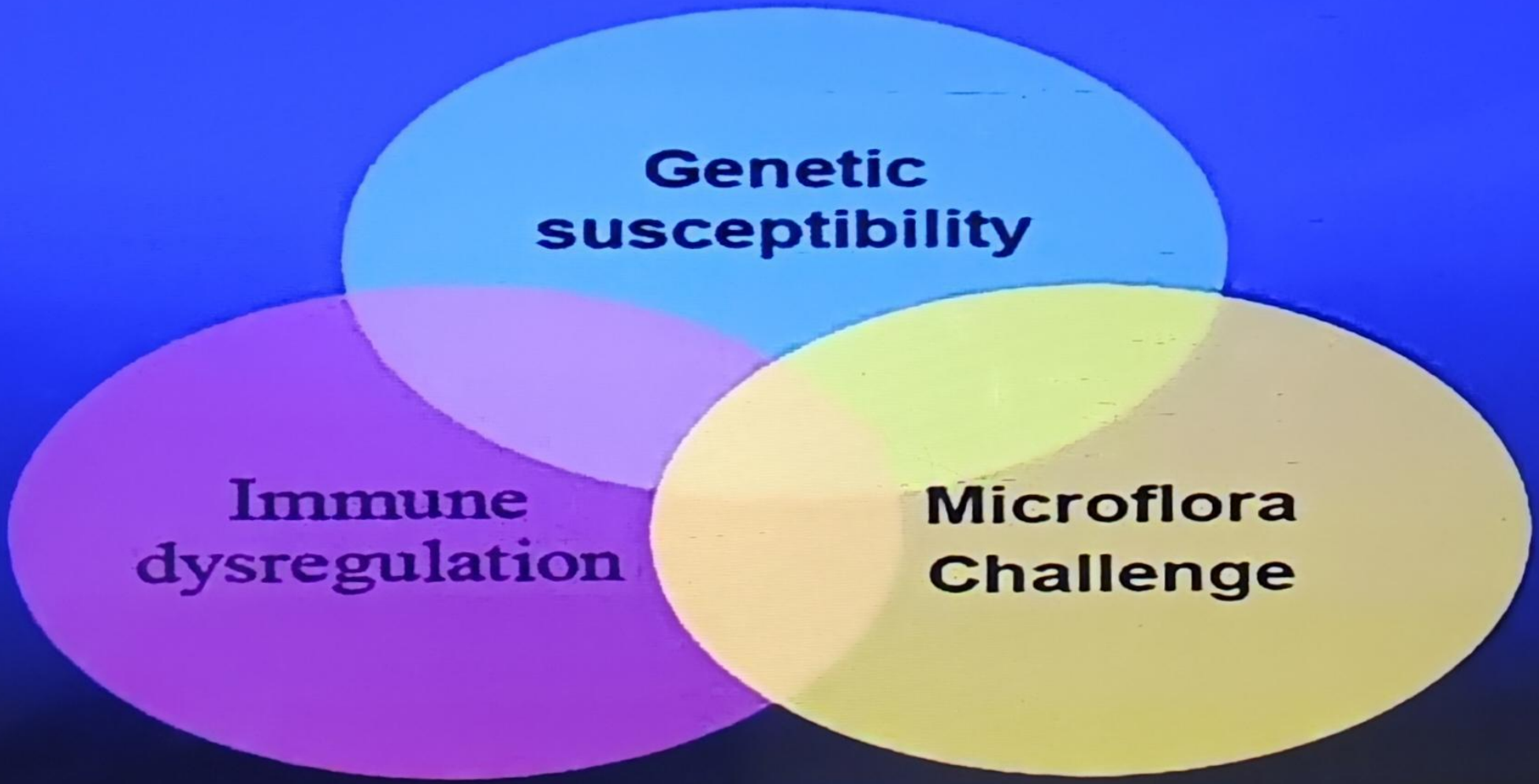
INFLAMMATORY BOWEL DISEASE

- Refers to two chronic diseases of unknown etiology that cause inflammation of the intestine with extra intestinal manifestations
- Ulcerative colitis and Crohn's disease
- Although the diseases have some features in common, there are some important differences.

INTRODUCTION

- Ulcerative colitis is an idiopathic form of acute and chronic ulcero-inflammatory 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.
- these disorders primarily affect the bowel but may have systemic involvement in the form of polyarthritis, uveitis, ankylosing spondylitis, skin lesions and hepatic involvement.

Etiologic Factors in IBD



ETIOLOGY

- 1 Genetic factors. Genetic factors are implicated in the etiopathogenesis of IBD is supported by the following evidences:
- i) There is about 3 to 20 times higher incidence of occurrence of IBD in first degree relatives.
- Ii) Overall, there is approximately 50% chance of development of IBD in

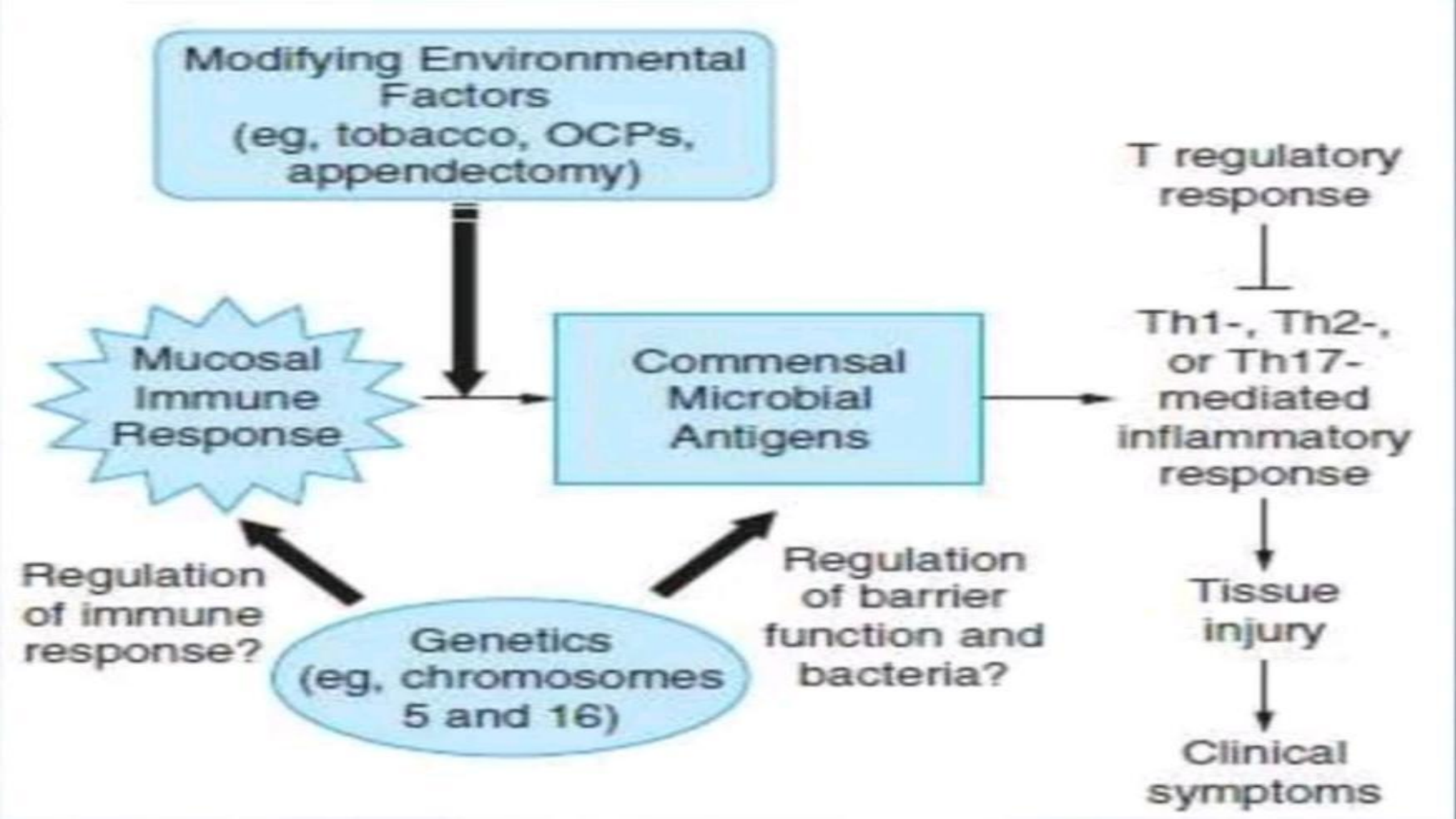
  iii) Genome wide search has revealed that disease-predisposing loci are present in chromosomes 16q, 12p, 6p, 14q and 5q. Iv) HLA studies show that ulcerative colitis is more common in HLA-DRB1 alleles while Crohn's disease is more common in HLA-DR7 and DQ4 alleles.

- 2. Immunologic factors. Defective immunologic regulation in IBD has been shown to play significant role in the pathogenesis of IBD:
- i) Defective regulation of immune suppression. The mechanism responsible for this is by activation of CD4+ T cells secreting cytokines inhibitory to inflammation (IL-10, TGF-B) which suppress inflammation in the gut wall.

  Ii) Transgenic mouse experimental model studies. Gene 'knock out' studies on colitis in mice have revealed that multiple immune abnormalities may be responsible for IBD as under:

- 3. Exogenous factors. In addition to role of genetic factors and deranged T-cell mediated immunity, a role for several exogenous and environmental factors has been assigned:
- i) Microbial factors, ii) Psychosocial factors, iii) Smoking and iv) Oral contraceptives.
  i) Microbial factors: At different times, role of a variety of microbes in initiation of inflammatory response by the body has been suspected. Accordingly, several microorganism species (bacteria, viruses, protozoa and fungi) have been suspect but without definite evidence: Mycobacterium paratuberculosis, Salmonella, Shigella, Helicobacter, Clostridia, bacteroides, Escherichia, Measles virus etc.

- Ii) Psychosocial factors: It has been observed that individuals who are unduly sensitive, dependent on others and unable to express themselves, or some major life events such as illness or death in the family, divorce, interpersonal conflicts etc, suffer from irritable colon or have exacerbation of symptoms.
- iii) Smoking: Role of smoking in causation of Crohn's diseasehas been reported.
- iv) Oral contraceptives: An increased risk to develop Crohn's disease with long-term use of oral contraceptives has been found in some studies but there is no such increased risk for ulcerative colitis.


Modifying Environmental Factors (eg, tobacco, OCPs, appendectomy)
Mucosal Immune Response
Commensal Microbial Antigens
T regulatory response
Th1-, Th2-, or Th17-mediated inflammatory response
Tissue injury
Clinical symptoms
Regulation of immune response?
Genetics (eg, chromosomes 5 and 16)
Regulation of barrier function and bacteria?

RISE OF INCIDENCE IN IBD IN INDIA

- Familial aggregation
- Nicotine Consumption
- Oral Contraceptives
- • Dietary Habits-Refined sugars, Fast food, cereals, bakers yeast etc
- Physical inactivity Early weaningHygiene
- • Infectious diseases- TB, MeaslesSave ▭ O

MORPHOLOGY

- Mucosa shows linear and superficial ulcers, usually not penetrating the muscular layer. The intervening intact mucosa may form inflammatory 'pseudopolyps. The muscle layer is thickened due to contraction, producing shortening and narrowing of the affected colon with loss of normal haustral folds giving 'garden-hose appearance'

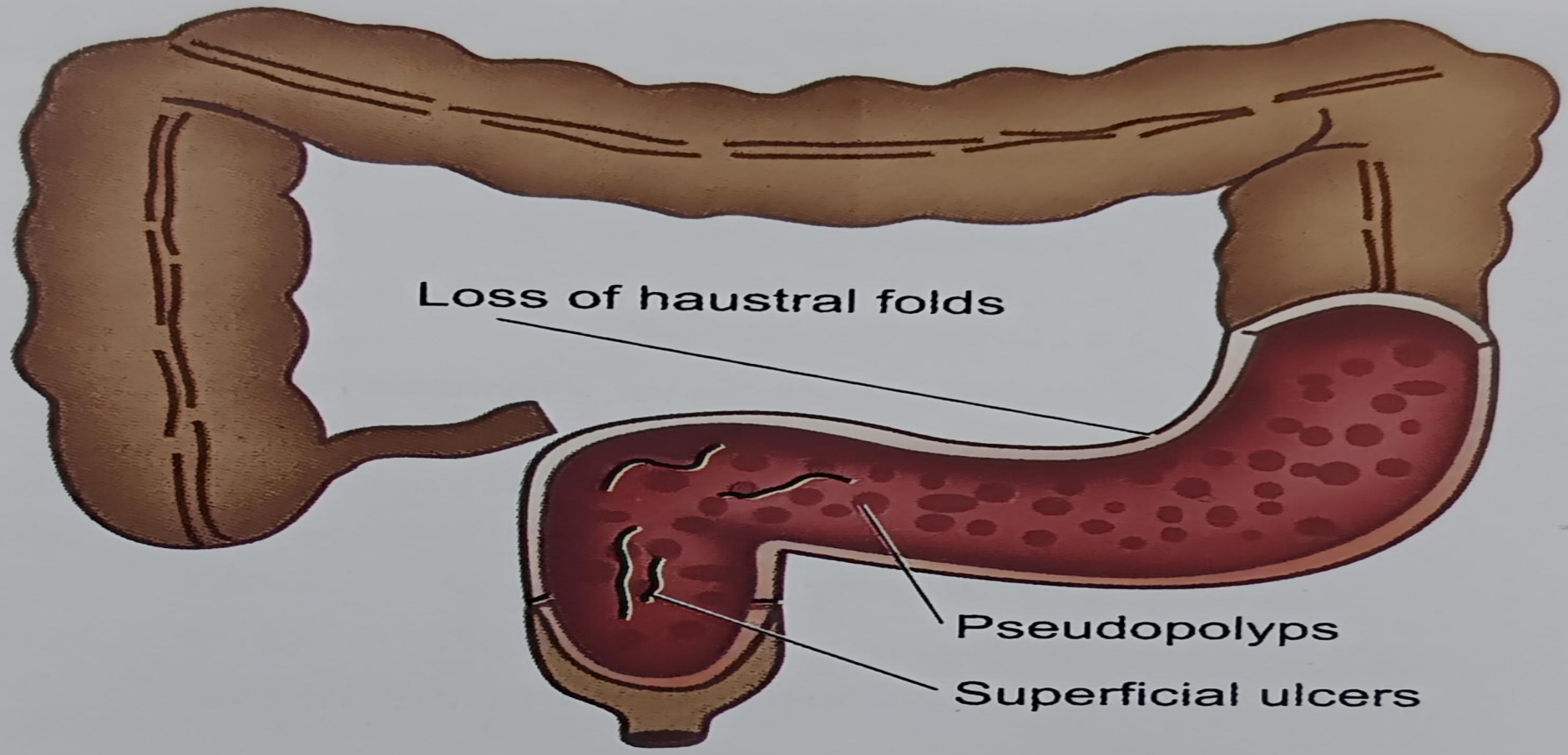
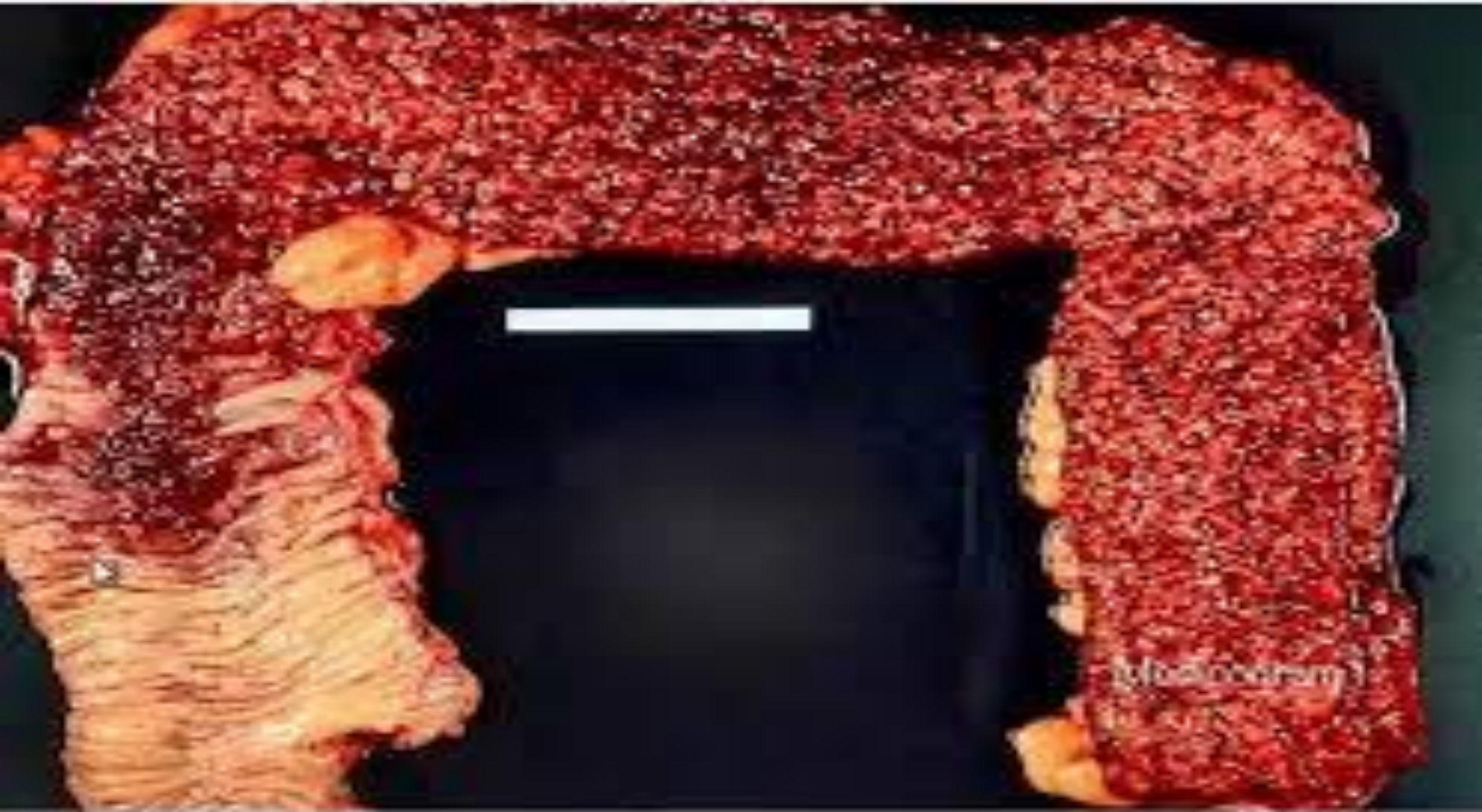


Figure 18.27 Ulcerative colitis. Continuous involvement of the rectum and colon without any uninvolved skip areas. The ulcers are superficial with intervening inflammatory pseudopolyps. The lumen is narrow and the haustral folds are lost giving 'garden-hose appearance'.



CHARACTERISTICS FORM OF CHRONIC COLITIS ACCORDING TO BIOPSY

- 1Crypt distortion, cryptitis and focal accumulations of neutrophils forming crypt abscesses.
- 2 Marked congestion, dilatation and haemorrhages frommucosal capillaries.
- 3 Superficial mucosal ulcerations, usually not penetrating Into the muscle coat, except in severe cases, and is accompanied by nonspecific inflammatory cell infiltrate of lymphocytes, plasma cells, neutrophils, some eosinophils and mast cells in the lamina propria.

- 4 Goblet cells are markedly diminished in cases of activedisease.
- 5. Areas of mucosal regeneration and mucodepletion of lining cells.6.
- In long-standing cases, epithelial cytologic atypia ranging from mild to marked dysplasia and sometimes developing into carcinoma in situ and frank adeno carcinoma.

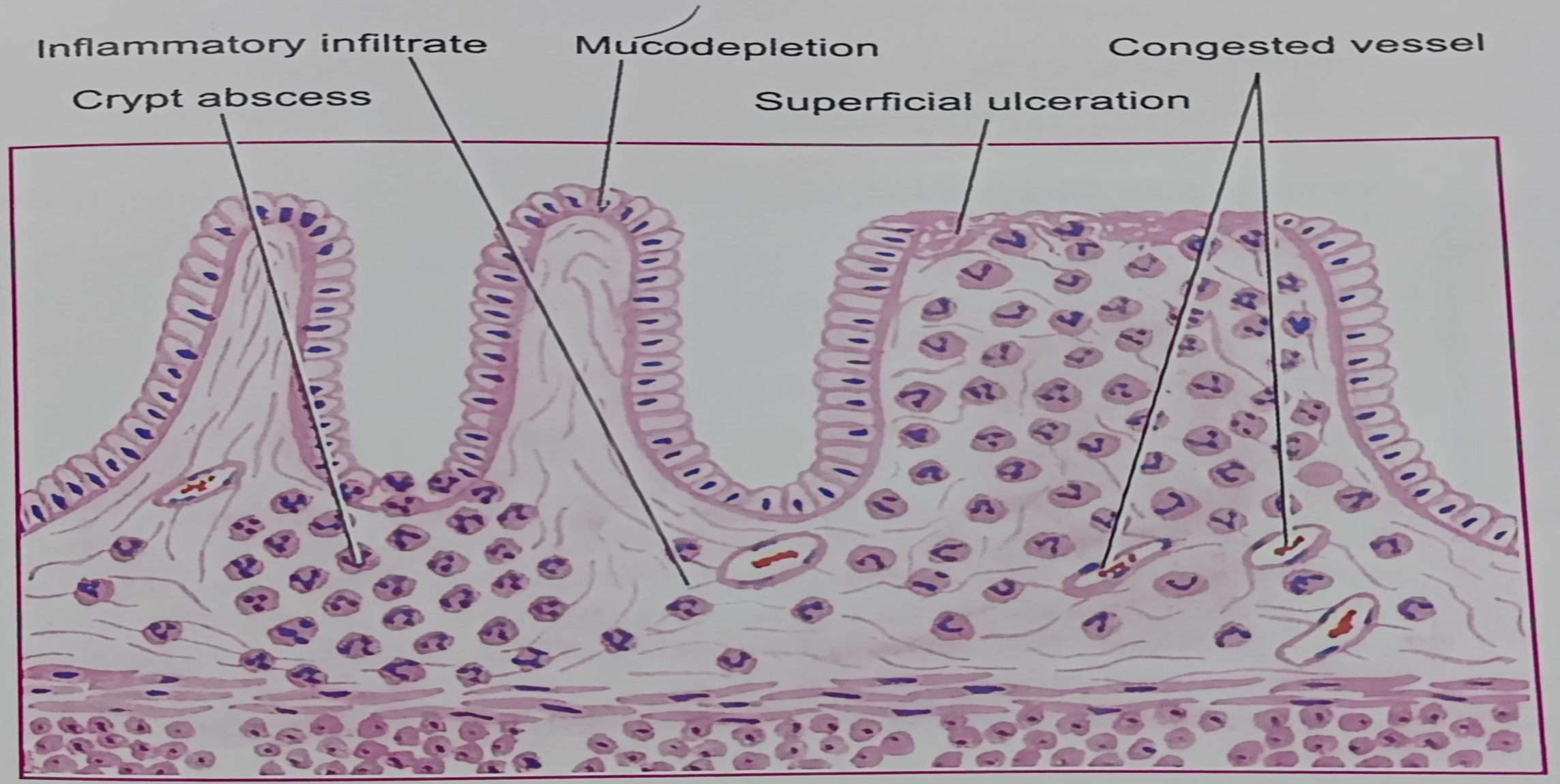
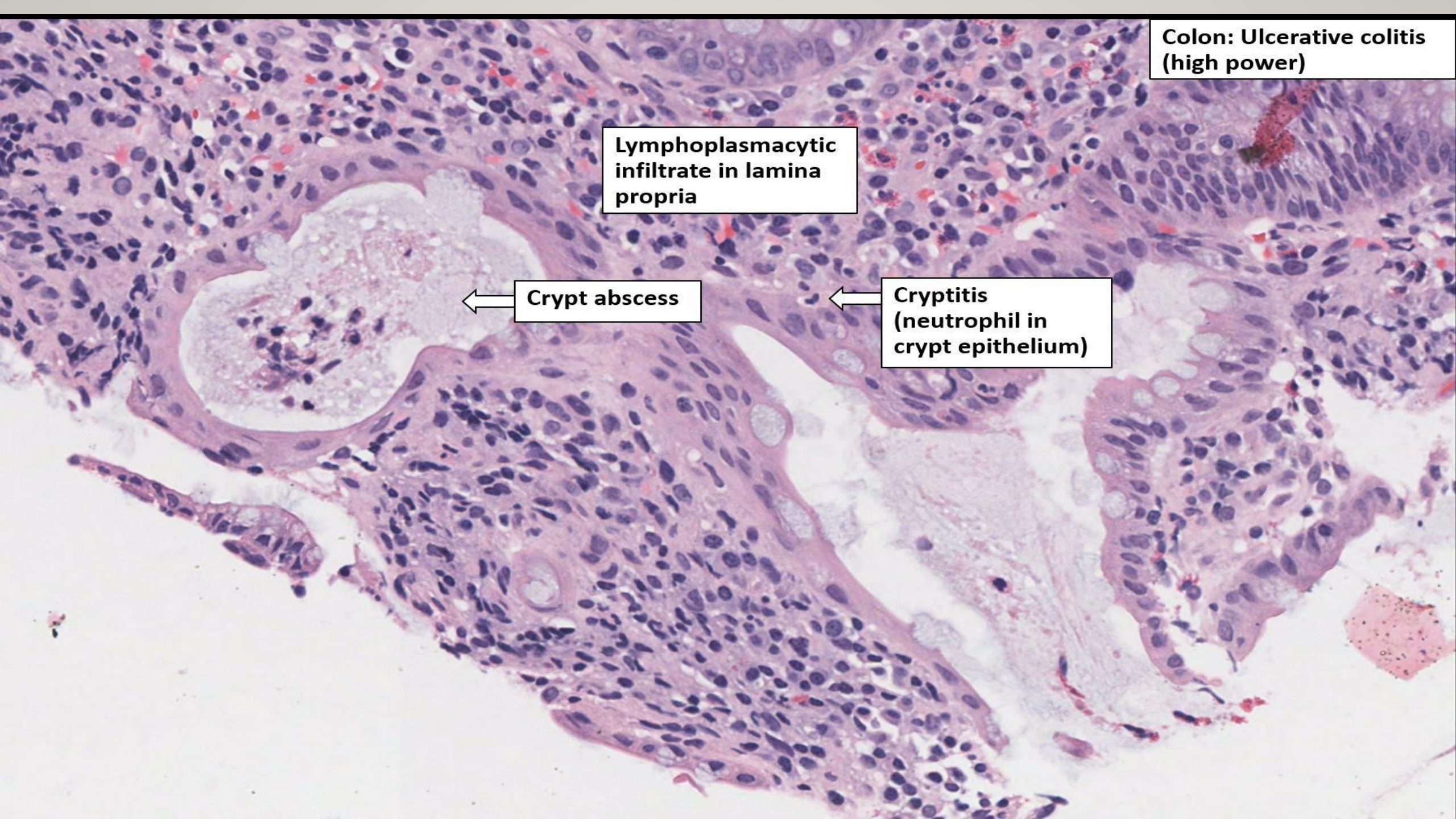


Figure 18.28 Ulcerative colitis in active phase. The microscopic features seen are cells and a 'crypt abscess'.


Colon: Ulcerative colitis (high power)
Lymphoplasmacytic infiltrate in lamina propria
Crypt abscess
Cryptitis (neutrophil in crypt epithelium)

SYMPTOMS

- Rectal bleeding and tenesmus are universallypresent.
- Diarrhea and abdominal pain are more frequent with proximal colon involvement.
- Nausea and weight loss in severe cases.
- Severe abdominal pain or fever suggestsfulminant colitis or toxic megacolon.

COMPLICATIONS

- 1. Toxic megacolon (Fulminant colitis) is the acute fulminating colitis in which the affected colon is thin-walled and dilated and is prone to perforation and faecal peritonitis. There is deep penetration of the inflammatory cell infiltrate into muscle layer which is disrupted. The summariGISInflabowulceCrohIBD,granthe.
- 2. Perianal fistula formation may occur rarely.
- 3. Carcinoma may develop in long-standing cases of ulcerativecolitis of more than 10 years duration.
- 4. Stricture formation almost never occurs in ulcerative colitis.

	Ulcerative colitis	Crohn's disease
Localization GI tract	Especially colon and rectum	Whole GI tract
Ileum	Not except backwash-ileitis	Often involved
Colon	Left> right	Right>left
Rectum	Commonly involved	Typically spared
Distribution GI tract	Diffuse (continuous)	Segmental (discontinuous)
Ulcers	Superficial ulcers	Aphtoid ulcers, confluent deep linear ulcers
Pseudopolyps	Common	Uncommon
Skip-lesions	Absent	Present
Cobblestone-pattern	Absent	Present
Deep fissures	Absent except in fulminant colitis	Present
Fistulae	Absent except in fulminant colitis	Present
Mucosal atrophy	Marked	Minimal
Thickness of the wall	Normal	Increased
Fat wrapping	Absent	Present

Crohn's Disease