CROHN'S DISEASE

- Chronic multifocal relapsing & remitting, progressive Inflammatory Bowel disease.

GROSS

Sites: any area of GIT from mouth to anus

m/c – Terminal ileum
I-C valve
Caecum.

- multiple lesions.
"Skip lesions."

Mucosal lesions:

- aphthous ulcer → earliest lesion.
- multiple lesions coalesce longitudinally
→ serpentine ulcers.
→ linear clefts or fissures:
  - fistula tracts.
  - perforation.

→ Islands of normal mucosa between ulcers shows edema and produces Cobble stone appearance.

Intestinal wall

- Fibrotic – thickened – rubbery.
lumen is narrowed, strictures are seen.
→ hosepipe strictures.
→ radiological 'string sign'

Serosal/External surface.

- Red, opaque, hyperemic & covered with serosal exudate.
- Creeping fat – along anti-mesenteric border.

MICROSCOPY

- Transmural inflammation.
- crypt abscesses
- mucosal aphthous ulcers & inflammation
- distortion of mucosal crypt architecture - bizzare branching
- non caseating granuloma – Hallmark * m/c → submucosa
- Epithelial metaplasia
→ pseudopyloric metaplasia
→ paneth cell metaplasia

CLINICAL FEATURES

Extraintestinal.

- uveitis
- migratory polyarthritis.
- ankylosing spondylitis.
- erythema nodosum.

Intestinal.

- Intermittent mild diarrhoea, fever, abdominal pain
- waxing & waning course.
- disease reactivation by external triggers

Lab

- ESR, CRP ↑

Complications

- Fe def anemia.
- malabsorption.
- strictures.
- perforation.

ULCERATIVE COLITIS

It is a chronic crypt destructive, ulcerating IBD limited to colon & rectum.

- Involves only mucosa & submucosa.

Clinical features

- young adults - m/c.
- bloody diarrhea.
- lower abd pain & cramps

Morphology

GROSS

Site : - diffuse - No skip lesions
- Colon & rectum

Early active colitis

- mucosa - red & fine granular (sandpaper)
- frequently covered with yellowish exudate and bleeds easily.
- Small superficial erosions or broad based ulcers.

Chronic colitis

- pseudopolyps.
- mucosa - granular or smooth
- diffuse, no skip lesions.
- limited to mucosa & submucosa
- Bowel wall is not thickened.

MICROSCOPY

EAC

- chronic inflammatory infiltrates.
- Neutrophils - hallmark of acute ds.
- crypt injury & architect distortion
- crypt abscess

Chronic ulcerative colitis

- crypts distortion — bizzare branching crypt atrophy.
- ↑ in chronic inflammatory cells
- loss of goblet cells & mucin depletion.
- dysplastic changes

Complications

- Toxic megacolon.
- Colorectal ca.
- Hemorrhage.

IBD

Introduction

It is a chronic inflammatory condition.
Characterized by:

- altered interaction between Intestinal microbiota & Host Immune system.
- genetically predisposed
- inappropriate mucosal Immune activation

- IBD (KU)
- M/c feature to distinguish UC & Cr
- Morphology of crohns.
- Etiopatho of U.C.
- Morpho - UC.
- Crohns.
- dif btwn Cr & UC

IBD

Crohn's disease.
- any part of GI

Etiology - diagram

Ulcerative colitis.
- colon & rectum.
- start at rectum.

① Genetic factors.

1. familial occurence and concordance amongst twins:
   CD > UC.
2. Susceptibility genes:
   - NOD 2 / CARD 15 - recognition & response against microbes.
   - ATG 16L1
   - IRGM.

② Environmental factors

Gut environment - major factor.

- Intestinal microbiota can be modified by diet & exercise
- Smoking - CD in smokers
  UC in non/ex smokers

③ Host factors

- epithelial defect →
  - impaired mucosal barrier function.
  - abnormal intestinal defensins.
- Immune dysregulation
  - ↑ of CD4+ Tcells.

Pathogenesis - hypothesis.

① Trans epithelial influx of luminal bacteria

↓

APC presents to CD4+ T cell

↓

activation of CD4 T cell

differentiate to

- Th1 by IL-12 → TNF ↑ permeability → ↑ macrophage; IFN-γ
- Th2 by IL-4 → IL-13 → mucosal inflammation
- TH17 by IL-23 → release → IL-17 → recruits neutrophils