PATHOLOGY OF STOMACH AND INTESTINE.
DISEASES OF LIVER, GALL BLADDER AND PANCREAS.

(gastric ulcer, chronic gastritis,
ulcerous colitis, hepatitis, liver cirhosis, chronic cholecystitis,
acute hemoragic necrosis of pancreas)

Dentistry

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Peptic ulcer (64)

- *ulcer* = defect extending deeper than *lamina muscularis mucosae*
- *erosion* = more shallow defect
- 70% prox. duodenum (young, ♂), dist. stomach (A)
- disbalance between **protective and aggressive factors**

### Protective factors
- Mucus
- $\text{HCO}_3^-$
- epithelium regeneration
- neutralisation with saliva, bile, pancreatic fluid
- PGE
- good blood supply

### Aggressive factors
- acid
- Pepsin
- **H. pylori**
- Nicotin
- Alcohol
- Drugs
- Stress

- **G**: decreased mucosal protection
- **D**: increased gastric acid and pepsin secretion
Peptic ulcer (64)

1. **ACUTE**

   - severe stress – shock, trauma, sepsis, extensive burns, intracranial lesions, NSAIDs…
   
   - multiple, more common in the stomach, smaller, more shallow

2. **CHRONIC**

   - **H. pylori gastritis**, NSAIDs, local irritants, psychological stress, genetic factors, hormones (gastrin – gastrinoma – Zollinger-Ellison sy)
   
   - solitary, flat or slightly elevated firm margins, mucosal folds converge towards the ulcer, superficial / deep
Peptic ulcer (64)

**Clinics:** epigastric pain (G – after meal, bad appetite - weight loss, D – on empty stomach, good appetite - weight gain),

**DG:** GFS, H.pylori

**TH:** H.pylori, decreasing of acidity (inhibitor PP, H2, antacids), surgery

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**CHRONIC G. ULCER**

1. necrotic zone
2. spfc exsudative z.
3. granulation tissue
4. fibrous conn.tissue
Chronic gastritis (216)

- chronic inflammation of gastric mucosa

- **E:** infection with *H. pylori*, reflux of duodenal contents, associated disease of stomach, AutoAb, chemical and physical factors

- --- cytotoxic effect on mucosa – inflammatory response

- **type A** – rare, corpus and fundus, AutoAb – gastric atrophy, hypo/achlorhydria

- **type B** – more frequent, antrum, *H. pylori*, associated ulcer ... Ca!!!

- **type AB** (mixed gastritis) – both areas, many factors

- superficial, atrophic, hypertrophic, eosinophilic, hemorrhagic, granulomatous
**Inflammatory bowel disease – unknown etiology**

- idiopathic inflammatory disease, 2 forms:

<table>
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<tr>
<th>Morbus Crohn (Crohn disease)</th>
<th>Ulcerative colitis (250)</th>
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<tr>
<td>• ileitis terminalis (whole GIT, extraGIT)</td>
<td>• rectum and colon (extraGIT)</td>
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<tr>
<td>• segmental transmural inflammation</td>
<td>• continuous superficial inflammation</td>
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Inflammatory bowel disease – unknown etiology

Clinics:
- change exacerbation and remission
- abdominal pain
- diarrhea
- extraintestinal manifestation

COMPLICATIONS:
- mimics appendicitis
- rarely bloody
- rare
- above symphysis, tenesmus
- blood and mucus
- often
- perianal fistulas, abscesses
- stenosis, ileus,
- malabsorption,
- perforation
- Ca

- toxic megacolon
- massive bleeding
- Ca!!!
Inflammatory bowel disease – unknown ethiology

- cobblestone mucosa
- solitary ulcerations, fistulas, stenosis
- noncaseating granulomas

DG:
- X-ray
- colonoscopy
- histology

- teeth-like defects
- bleeding, confluent ulcers, pseudopolyps, loss of haustration
- crypt abscesses
Ulcerative colitis

- continuous involvement by spfc ulcers
- inflammatory pseudopolyps
- mucosa is reddish and friable

- ulcers – not penetrating into the muscle layer
- nonspecific inf.inf. in lamina propria
- goblet cells diminished
- congestion of mucosal capillaries - hemorrhages

- crypts – deformed
- with accumulated Neu – crypt abscesses
Adenocarcinoma of the colon (47)

- **E:** genetic factors (FAP, HNPCC), environmental (alcohol, smoking, diet low in fiber and rich in fat), adenomatous polyp, ulcerous colitis

- **CP:** asympt. ... alteration of diarrhea and obstipation, blood in stool, abdominal pain, meteorism, weight loss, palpable Tu, ileus

- **DG:**
  - colonoscopy, imaging techniques (X-ray, CT), staging

- **prevention:** per rectum + OB (yearly after 45 years of age)
Adenocarcinoma of the colon (47)

• variously differentiated tubular structures with atypia and mitosis
• infiltration of muscle and fat tissue
• grading
• mucus production (ec / ic)
Chronic hepatitis (136, 218)

- chronic / relapsing hepatic disease lasting over 6 months with evidence of inflammation and necrosis

- **E**: viruses (HCV, HBV, HDV), drugs, AOI

- **CP**: fatigue, malaise, loss of appetite – hepatomegaly and splenomegaly, hepatic tenderness

DG (in HBV hepatitis):

- elevated serum levels of HBsAg and HBcAg
- immunohistochemical evidence of HBV infection
- + elevated liver enzymes, bilirubin, decreased coagulation factors and proteins
Chronic hepatitis

- necrosis of hepatocytes
- piecemeal necrosis
- apoptotic bodies
- lymphoplasmocytic infiltration
- steatosis
- fibrosis
- Mallory’s hyalin
- regressive changes of bile ducts,
- activation of Kupffer cells,

- necrotic lobule with collapsed reticulin framework
Liver cirrhosis (62, 63)

- progressive irreversible destruction of hepatic architecture by fibrous septa that encompass regenerative nodules of hepatocytes
- alcohol, HBV, HCV, AOI diseases, metabolic, biliary occlusion, cardiac failure, toxic agents

- clinics:
  - skin changes (icterus, spider nevi...)
  - endocr. (gynecomastia, menstruation disorders)
  - hypocoagulative condition, oedema, ascites

- COMPLICATIONS:
  - portal hypertension (esophageal varices, ascites, splenomegaly)
  - hepatic failure, encephalopathy, hepatorenal syndrome, Ca

- DG:
  - lab. tests
  - USG, liver biopsy
Liver cirrhosis (62, 63)

- multiple nodules: **macro (>3mm) / micronodular (<3mm)**
- liver more firm, larger / normal / shrunk, colour variable

- normal lobular structure not present – replaced by nodules (disorganised proliferation of HC, no CV)
- nodules divided by fibrous septa with chronic inf.inf.
- steatosis
- necrosis (in active form)
- pseudotubules in portal regions
Chronic cholecystitis (99)

- E: gallstones, bacterial infection (streptococcus, Escherichia coli), repeated attacks of acute CHC

- fibrous thickening of the wall of the gallbladder
- shrinking of the gallbladder
- calcification of the wall (porcelain gallbladder)

- thickening of the muscle layer
- fibrosis of the wall
- chronic inf.inf. of the mucosa and subserosal layer
- invagination of epithelial cells into musculature (Rokitansky-Aschoff sinuses)
Acute hemorrhagic necrosis of pancreas (130)

• severe acute inflammation of pancreatic tissue

• **autodigestion by pancreatic enzymes** (acinic cell damage / duct obstruction / increased exocrine production of enzymes)

• **E**: idiopathic, **gallstone**, **alcohol**, fatty foods, trauma, steroids, mumps, autoimmune disease, hyperlipidemia, hypercalcemia, drugs

• **P**:
  
  • enzymes digest the pancreas and surrounding tissues
  
  • **proteases** – proteolysis
  
  • **elastases** – destroy the elastic fibres of the vessel walls
  
  • **lipases** and **phospolipases** – lipolysis – fat necrosis – combine with Ca – saponification (**Balser necrosis**)
**Clinics:**
- severe epigastric pain with belt-like irradiation below the ribs
- fever, nausea, vomiting
- ascites, peritoneal irritation (defens, meteorism, subileus)

**Complications:**
- peritonitis (chemical and bacterial) – shock – acute renal failure
- DIC
- pancreatic abscess / pseudocyst (formed after clearing of necrotic debris)
- obstruction of duodenum
- thrombosis v.portae, v.cava

- mortality is high!!! 20-30%
- pancreas larger, swollen, edematos
- necrosis
- hemorrhage
- Balser necrosis
- blood stained ascitic fluid

- hyperemia
- oedema
- necrosis of pancreatic tissue
- necrosis of vessel walls - hemorrhage
- fat necrosis
- acute inf.inf.