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
- HCO3 –
- 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 antibiotic treatment, decreasing acidity (inhibitor PP, H2, antacids), surgery

**Complications:** bleeding, stricture, perforation, penetration, recurrence

**Micro appearance:** superficial necrotic layer, exudative zone with necrosis, granulation tissue, fibrous tissue
Multiple shallow defects

Multiple acute stomach ulcerations
Chronic stomach ulcer

Deep tissue defect with elevated margins

Mucosal folds converging towards the ulcer
Duodenal ulcer with perforation
Acute diffuse peritonitis as a result of a perforated ulcer
Chronic stomach ulcer, HE

Unaffected gastric mucosa

Deep ulceration reaching beneath the muscle layer
Chronic duodenal ulcer, PTAH

- Deep ulceration penetrating into underlying pancreatic tissue
- Duodenal mucosa
- Duodenal muscularis externa
- Erosion of a blood vessel with thrombus

Chronic duodenal ulcer, PTAH
Chronic stomach ulcer, HE

- Necrotic layer
- Exudation zone
- Granulation tissue
Chronic stomach ulcer, HE

Unaffected mucosa

Superficial necrotic layer

Exudation with hemorrhage

Granulation 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, chronic medication

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

• superficial, atrophic, hypertrophic, eosinophilic, hemorrhagic, granulomatous
Chronic gastritis, HE

Flattened gastric villi

Chronic inflammatory infiltrate in submucosal layer
Intestinal metaplasia of gastric mucosa

Chronic gastritis, HE
Intestinal metaplasia of gastric mucosa

Chronic gastritis, Alcian blue
Helicobacter pylori
Inflammatory bowel disease – unknown ethiology

- idiopathic inflammatory disease, 2 forms:
  - Morbus Crohn (Crohn disease)
    - ileitis terminalis (whole GIT, extraGIT)
    - segmental transmural inflammation
  - Ulcerative colitis (250)
    - rectum and colon (extraGIT)
    - continuous superficial inflammation

Clinics:
- Switching between exacerbation and remission
  - abdominal pain
  - diarrhea
  - extraintestinal manifestation
- 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
Ulcerative colitis, HE

- Superficial ulcer without deeper involvement
- Inflammatory pseudopolyp
- Vascular congestion
Ulcerative colitis, HE

Crypt abscess with neutrophils

Nonspecific chronic inflammatory infiltrate

Ulcerative colitis, HE
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:** asymptomatic → 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)
Colon adenocarcinoma, HE

Adenomatous tubular structures with atypical cells

Mucus production
Colon adenocarcinoma, HE

- Carcinoma
- Transition zone from normal epithelium into dysplastic epithelium
- Normal epithelium
Infiltration of muscularis layer

Colon adenocarcinoma, HE
Atypical cells with apoptotic bodies

Inflammatory response

Colon adenocarcinoma, HE
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
Coagulative hepatocyte necrosis

Lymphoplasmocytic infiltration

Chronic hepatitis, HE
Chronic hepatitis, HE

Piecemeal hepatocyte necrosis

Lymphoplasmocytic infiltration
Liver cirrhosis (62, 63)

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

clinics:
- skin changes (icterus, spider naevi,...)
- endocrine (gynecomastia, menstruation disorders)
- hypocoagulative condition, generalized oedema, ascites

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

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

- multiple nodules: **macro (>3mm) / micronodular (<3mm)**
- **Macro:** liver more firm, larger / normal / shrunk, colour variable
- **Micro:** normal lobular structure not present – replaced by nodules (disorganised proliferation of HC, no CV), nodules divided by fibrous septa with chronic inflammatory infiltrate, steatosis, necrosis (in active form), pseudotubules in portal regions
Liver cirrhosis

Prominent nodular architecture of liver with variable size of nodules
Liver cirrhosis, HE

Nodular architecture separated by fibrous septa

Thick fibrous septa with chronic inflammatory infiltrate

Steatosis

Bile ducts
Nodule of hepatocytes surrounded by fibrous tissue with chronic inflammatory infiltrate

Bile pool

Liver cirrhosis, HE
Liver cirrhosis, PTAH

- Pseudolobule
- Bridging fibrous septa
- Pseudolobule
Liver cirrhosis, van Gieson

- Pseudolobule
- Bridging fibrous septa
- Steatosis
Chronic cholecystitis (99)

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

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

- **Micro**: thickening of the muscle layer
  - fibrosis of the wall
  - chronic inflammatory infiltrate (mucosa, subserosal layer)
  - invaginations of epithelial cells into musculature (*Rokitansky-Aschoff sinuses*)
Chronic cholecystitis, HE

- Thickened muscle layer
- Rokitansky-Aschoff sinus
- Chronic inflammatory infiltrate
Chronic cholecystitis, HE

Rokitansky-Aschoff sinus

Chronic inflammatory infiltrate
Acute hemorrhagic necrosis of pancreas (130)

- **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 calcium → saponification (**Balser necrosis**)
• **manifestation:**
  - 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 inferior

• mortality is high!!! 20-30%
Acute pancreatitis

Edematous pancreas with reddened adjacent fat tissue

Balser necrosis of fat tissue
Acute pancreatitis

Balser necrosis of fat tissue
Acute pancreatitis, HE

Unaffected pancreatic tissue

Acute inflammatory infiltrate (neutrophils)

Fat necrosis (shadows of fat cells with blurred margins and content)

Hemorrhage
Acute inflammatory infiltrate (neutrophils)

Hemorrhage

Unaffected pancreatic tissue (with autolysis – no inflammation)

Acute pancreatitis, HE
Acute pancreatitis, HE

Fat necrosis (shadows of fat cells with blurred margins and content)

Acute inflammatory infiltrate (neutrophils)
Questions

1. Is there a relationship between peptic ulcers and gastric carcinoma? Can ulceration be observed in a carcinoma?
2. Can colon carcinoma be caused by IBD? If so, which IBD has higher probability of developing a carcinoma?
3. Name typical clinical signs of hepatitis. Can they mimic a different disease affecting liver? How would you distinguish between said diseases?
4. What is the mechanism behind fat necrosis in acute pancreatitis?