Dyspepsia 2

DR. ADORATA COMAN
Irritable bowel syndrome

The irritable bowel syndrome (IBS) is a physiologic disorder presumably of multifactorial etiology.

**Histology:**
- the most frequent syndrome in gastro-enterology
- associated with neurovegetative syndrome
- it is defined like an "vagal autonomic neuropathy" consisting of: central nervous system, enteral nervous system, bowel wall with its contents.

From *anamnesis*:
- abdominal pain
- diarrhea, constipation (or emptying disability)
- flatulence ("wind") onset on stress.

**Treatment** with:
- fibres in constipation
- imodium (loperamide) on diarrhea
- added: – Anticholinergics
  – Ca antagonists
  – increases digestion with supplements
  – antibacterial agents/eubiotics, proбиotics
  – sedatives, anxiolytics, antidepressants.

**Physical measures**
The use of baths, hot water bottles, exercise, relaxation techniques, and periods of examination can be benefit when individualized to the patient's needs.
Cancer of the colon

61% localized – recto – sigmoid

**Symptoms**
1). Functional disturbance of bowel transit – constipation, diarrhoea +/- mucous/ both
2). Low digestive bleeding – occults, rectoragy
3). Anaemia – secondary of bleeding
4). Occlusion (König syndrome – pain, increase peristalsis, garguiments)
5). Pain – late in evolution
6). Fever
7). Weight lost
8). General signs of cancer

**Exploration:**
- Rectoscopy → Colonoscopy

±Byopsy
- X-Ray – barium enema

**Signs:** – palpation/rectal palpation – tumour
**Laboratory:** – haemoglobin, iron levels, electrolytes.
Rectal cancer

**Specific symptoms**
1). Rectorhagy
2). Tenesmus + Pencil shaped stools with mucous
3). Constipation
4). Pain – increased by defecation

**Signs** – rectal palpation
- palpation of the sigmoid zone
- indirect sign: – cecal distention
## Ulcerative Colitis

### A recto–sigmoidal syndrome
- stools with diarrhoea, mixed with
- mucus
- pus
- abdominal pain
- tenesmus

### Inflammation syndrome
- fever
- tachycardia
- arthritis
- skin nodules
- uveitis

### A general syndrome
- anorexia, nausea, vomiting
- weight lost
- oedema
- dehydratation signs
- tympanism (acute toxic dilatation)
Ulcerative colitis

**Signs** – abdominal palpation – “sigmoidal tube”
– rectal palpation – spastic sphincterus

**Endoscopy + Biopsy** – ulcerations, bleeding
- mucus, pus
- pseudopolips

**X-ray:** - narrowing
- “spiculi”
- “map”- like

**Laboratory:** - inflamation
- ↓ proteins
- ↓ electrolytes
- for acutisation: α2 globulin
- oesinophiles ↑
Causes of hepatomegaly

1. **Inflammatory diseases**: a) acute + chronic hepatitis  
   b) cirrhosis

2. **Tumours**: a) benign tumours (haemangioma, lymphangiom)  
   b) malignant tumours (carcinoma – primary, metastasis)  
   c) parasites (hydatic cysts)

3. **Infiltrative diseases**: a) with cells (leukaemia, lymphomas)  
   b) with abnormal substances (glicogenosis, lipoidosis, amiloidosis, haemochomathosis)

4. **Infectious diseases** (abces) – Parasites (Ecchinococcus canis)

5. **Vascular congestions** – stasis liver, Budd – Chiarri syndrome

6. **Biliary stasis** (Obstructive jaundice)
Palpation of the liver on inspir:
- inferior edge
- consistency
- sensibility
- surface
- dimension (dullness on mid – clavicle line)

Echography

- **diffuse** → cirrhosis
- **localized** → solid – cancer, benign tumor
- **liquid** → cyst, haemangioma
Abnormal liver function

**Cytolysis**
- ↑ ASAT
- ↑ ALAT
  - Viral hepatitis: Ag Hbs, Ac anti HVC
  - Toxic hepatitis: γGT

**Liver failure**
- ↓ albumine
- ↓ cholesterol
- ↓ prothrombine complex
  - Drugs hepatitis: therapy stop

**Cholestasis**
- ↑ bilirubin
- ↑ alkaline phosphatase
- ↑ Cholesterol
  - stasis
    - intra
    - extra
    - echo

- Nondilated biliary tract
- Dilated biliary tract
Jaundice

Corpuscular jaundice:
- Infarction - bleeding - cancer

Extracorpuscular jaundice:

- Prehepatic
  - Haemolysis → Massive destruction of blood
  - Intrinisic obstacle: inflammation

- Posthepatic
  - Intrinsic obstacle: stones, parasites, tumors, fibrosis

- Intrahepatic
  - Fail of enter
  - Fail of excretion
  - Fail of conjugating

- Gylbert sdr., Crigler-Najar
- Congenital, Rotor, Dubin-Jones
- Structural abnormalities of liver cells
  - Carential
  - Cirrhosis
  - Cholestasis
  - Toxicity: alcohol, antimytotic drugs
I. Splenomegaly with named cause (secondary)
   1. Acute infectious diseases: – bacterian
      – viral
   2. Chronic inflammatory diseases: – Tuberculosis
      – Lues
   3. Parasites: – Malaria, Bylchariosis, Echinococcus
   4. Colagenosis

II. Primary splenomegaly (pure spleen syndrome)
   1. Mobil spleen syndrome
   2. Splenic artery aneurism
   3. Tumoral spleen: benign (haematoma, cysts, parasites)
      - malignant, primitive, metastatic.
   4. By overcharged
      - congenitally – Gaucher
      - Nieman pick
      Secondary – amiloidosis
      - haemochromathosis

III. Blood diseases
   – with hyperplasic syndrome
   (lymphoid/miloid)
   – with citopenia – haemolytic anaemia
   – neutropenia/pancitopenia

IV. On portal hypertension
Clinical criteria

depending on the latency, cholestatic and colangitică
a. bilioduodenale dyspepsia
(b). hepatalgii and postprandial afort
(c). fatigue, fatigabilitate, decreased intellectual performance
(d). sleepy postprandială
+ anemia, weight loss, subfebrilitate
Subjective:- absent
Objective:
- jaundice
- Vascular stars
- hemorrhagic syndrome
- edema gambiere oliguria
- endocrine syndromes
- flatulence
- hepatomegalie (smooth, increased consistency in self-defense), splenomegaly
Functional and biochemical criteria

It outlines 5 syndromes:
Hepatocitoliză
- GOT, GPT, GOT/GPT (Rittis) = 1.3-1.6
- sideremia, B12 rose
Excretory irinel
- increasing the total Bi (I + D), FAS, total cholesterol/lipid modified by esterification
- GTP, 5NT, leucinaminotransferaza
- hipoprotrombinemie according to the vit.K
Hepatopriv
- decrease total protein, cholesterol, esterified applied;
- decrease in coagulation factors (prothrombin complex II, V, VII, IX and vit.K)
Inflammatory-immunological
- VSH, -Ig, globuline, Cs low
- Ac HCV, HBe
- AgHBis present, AutoAc, FR
- RBW, CIC, transformation, blast
Hepatic encephalopathy

Definition- neuropsychic syndrome secondary to acute hepatitis (viral, toxic) or accompanying hepatopatiale cornice, with or without shunt porto-systemic, spontaneous or surgically

Precipitanti factors
- gastro-intestinal bleeding;
- renal azotemia-amoniogeneza; intestinal; IRA
- paracenteze unexpected evacuatorii low blood pressure;
- Excess dietary protein
- constipation
- infections
- sedative drugs, morphine, codeine,
- diuretics decrease in blood K+ ; + ammonium chloride
- alcohol
- surgery
- acute liver failure:
- viral hepatitis fulminating
- toxic hepatitis-halotan, others
- acute hepatic steatosis-sdr. Raye;
- hepatotoxine direct (mushroom poisoning).
Hepatic encephalopathy

0. neuropsychic normal
I. PRODROMAL
- fatigue + apathy/euphoria, impaired sleep/wakefulness, dysarthria, hiperROT
- megalomanice, paranoid tendencies
II. DELIRIUM
- personality changes + excitation/apathy
Neurological changes: hyperetonic-extrapiramidal
- cerebelare disorders, hyper REV clonus, dysarthria, nystagmus, flapping, asteryxis
- EEG changes
III. Advanced CONFUSION
- pyramidal signs, foetor hepaticus
IV. STUPOR
- hypotonia, hipoROT, (asteryxis), responding to strong stimuli
V. DEEP COMA (1) + (2) + (3) + (4)
- hypothermia, seizures, the absence of response to the stimulation of the pain, deep breathing
**Hepato-renal syndrome (HRS)**

**Definition** - a particular form of kidney failure functional, potentially reversible, produced by various causes that affect both the liver and kidney, characterized by oliguria, azotemia, hyponatremia of blocking.

- SHR. "true" – in which the liver is initially affected/- "pseudo" SHR – in both organs at the

**Predisposing factors:** - paracenteze in large quantities
- excess diretice
- digestive bleeding
- infection of ascites
- hepatic encephalopathy

**Clinical picture**
- ascites + jaundice
- oligo/anuria ± signs of dehydration

**Humoral-biochemical picture**
- blood: increases azotemia, urea/creatinine ± 20/1, hiponatriemie dilution
- urine: urinary osmolaritate ≥ 450 mOsm/kg (≥ osm. 1.5), decreased Na+ urinary ≥ 10 mEq/l, U.u. ≥ 14 g%, decrease diuresis, proteinuria is insignificant; ± hematuria, cilindrurie
Alcohol consumption
Mechanism of metabolism

Alcohol dehydrogenase (mitochondria) – converts alcohol into acetaldehyde which enters the Krebs cycle. Excess lead to dependency and injuries: tissues and organs.
A unit of alcohol is 8 g
MAXIMUM DAILY
- 6 U in men
- 4 U in women
For live:
- 160 g ethanol/daily □ increased risk
- 80 g ethanol/daily □ environmental risk
- 40 g ethanol/daily □ low risk
## Effects of excessive alcohol consumption (chronic)

<table>
<thead>
<tr>
<th>SNC</th>
<th>Gastro-intestinal</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Epilepsy</td>
<td>- acute gastritis</td>
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<tr>
<td>- sdr. Wernicke-Korsakoff</td>
<td>- cancer of the esophagus and rectum</td>
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<tr>
<td>- polineuropatia</td>
<td>- liver and pancreatic diseases</td>
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<tr>
<td>Muscle</td>
<td>Hematopoiesis</td>
</tr>
<tr>
<td>- acute or chronic idiopathic</td>
<td>- macrocitoză through injury or toxic effect of folați</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>- thrombocytopenia</td>
</tr>
<tr>
<td>- cardiomyopathy, arrhythmias</td>
<td>- leukopenia</td>
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<tr>
<td>Metabolism</td>
<td>Bones</td>
</tr>
<tr>
<td>- hiperuricemi, Hyperlipidemia</td>
<td>- Osteoporosis</td>
</tr>
<tr>
<td>- Hypoglycemia</td>
<td>- osteomalacia</td>
</tr>
<tr>
<td>Endocrine-sdr. pseudo Cushing</td>
<td>Respiratory infections</td>
</tr>
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Liver impairment in chronic alcoholism

Clinic
- asymptomatic (liver increased in sizes varying degrees)
- symptomatic
- moderate pain with all the symptoms of chronic hepatitis
- severe, associate
- jaundice
- ascites
- hepatomegalie
- the classic signs and symptoms of cirrhosis

Men
- GTP injury of liver function
- Leukocytosis + increased bilirubin hipoalbuminemie sdr. Zieve
- increased hemolysis, hypertriglyceridemia, increased total cholesterol
- GTP, GTO, elevated
Acute alcoholic hepatitis

Associated with steatosis, necrosis and inflammatory infiltrates + with polymorphonuclear and broad spectrum of clinical manifestations

**Laboratory test**
- OGT>PGT, gammaGTP increased,
- bilirubin (direct) increased with haemolitic components,
- increases g-globulin levels (IgA),
- macrocitosis (reduction of B12, folic acid hepatic pool)
- Hypertriglyceridemia.
Diagnosis

- medical history
- clinical trial (jaundice, fever)
- laboratory

**Differential diagnosis**
- other acute hepatitis, obstructive jaundice

**Evolution**
Acute form 25% severe
- high fever, increased bilirubin, increasing leukocytes, high amonemia, urea rises
- + absence of infection lipid (serum lactescent)
- death occurs by hepatic-renal failure

Subacute form
- Subacute yellow atrophy of the liver
- cirrhosis (60%)

**Treatment**
- Suppression of alcohol, bed rest, diet, vitaminoterapie, Corticoids, sedatives.
Pancreatic cancer

– usually – adenocarcinoma, head of pancreas

**Symptoms and signs**

1). **Loss of weight** – with/ no anorexia, lack of appetite
2). **Abdominal pain**
   – localisation – epigastrium + right upper quadrant
   – irradiation on left upper quadrant
   – irradiation on lumbar column
   – characteristics – steady
   – profound (deep)
   – persistent
3). **Obstructive jaundice** – +”Courvoisier sign” (large and tender gallbladder)
   – progressive

**Other symptoms:** – nausea, vomiting
   – fever
   – depression
Pancreatic cancer

Other signs: – hepatomegaly (stasis/metastasis)
– epigastric tumour
– epigastric murmur (spleenic artery stenosis)
– thromboflebitis migrans (“Trousseau sign”)

Other possible diagnosis – chronic pancreatitis (steatorhea)
– cysts of pancreas (7 general signs)

Laboratory tests – non specific
- anaemia syndrome, cholestatic syndrome
- cytology/ faeces exam (for chronic pancreatitis)

Imagistic tests
  o X Ray: – selective arteriography
    – spleno – portography
  o Endoscopy: – endoscopical retrograde colangio – pancreatograpgy (ERCP)
  o CT scan, scinti – scan
  o Echography
  o Byopsy – percutaneus
  o Laparotomy
Acute pancreatitis

**Causes**
1. Gall bladder stones/ obstruction of the pancreas ducts
2. Alcohol
3. Viral infections (mumps)
4. Hyperparathyroidism, hyperlipidemias
5. Allergy
6. Ischemia
7. Corticoids
8. Traumatism, surgery, endoscopy

**Semiology**
- acute on set
- hard pain
- resistant to antalgics
- possible abdominal contraction or meteorism

**Symptoms**
- Dyspepsy
- Diarrhoea
- weight lost
- Pain
- Colaps
Acute pancreatitis

Other signs: – jaundice
- diabetes mellitus signs
- upper GIT bleeding
- echimosis on abdominal wall (Cullen sign)

of complications: – ascitis, peritonitis
– ileus
– spasmofilia
– diffuse intravascular coagulation
– portal trombosis
– acute renal failure
– pleurisy, acute respiratory failure
– pericarditis, miocarditis
– dermal necrosis – overinfection
Acute pancreatitis

**Laboratory tests**
- blood/urine amylase, clearance amylase
- lipase concentration
- ↓ calcemia
- ↑ urea in blood
- leucocitosis
- ↓ haematocrit
- ↑ glycemia
- ↑ LDH, γGPT

**Explorations**
- X Ray – simple
- Echography
- Tomography
Acute peritonitis

**Germs** E. coli, Pneumococcus, Streptococcus

**Causes** – secondary peritonitis – diseases of intraabdominal organs

1). Necrosis of the bowel due to – obstruction
   – infarction
   – neoplasm

2). Inflammatory disease: – appendicitis
   – ulcerative colitis (fulminant)
   – diverticulitis
   – perforation of a peptic ulcer

3). Over infection of ascites – cirrhosis, nephritic syndrome
   – primary peritonitis – without a local condition

1). Infection with rare germs (Cryptococcus) 2). General infection (septicemia).
Acute peritonitis

– acutization of chronic peritonitis
1). Tuberculosis peritonitis
2). Fungal and parasitic peritonitis
3). Overinfection of pseudomyxoma peritonei (Meigs tumours)
4). Starch – granulomatous peritonitis (talc powder hypersensitivity)
5). Familial paroxysmal peritonitis (familial Mediterranean fever)
Acute peritonitis

**Clinics**
- abdominal pain
- guarding with rebound tenderness
- absent bowel sounds
- vomiting
- in evolution: – tachycardia
  - paralytic ileus
  - hypertension
  - shock
  - pyrexia
  - oliguria
  - acute tubular necrosis

**Paraclinics**
- plain x – Ray of the abdomen (for air)
- hydro – aerial levels (bowel obstruction)
- inflammation tests
Classification-DUKES

Stage I
a. caught epithelium – 80% surgical healing.
(b)1. muscularis mucosal,
(b)2. muscle structure.
Stage II
(c)1. mesenteric nodes – up 30-40% survival,
(c)2. passed over the muscular layer.
Location- 61% of the rectum and sigmoid.
## Symptoms

<table>
<thead>
<tr>
<th><strong>Functional disorders</strong></th>
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<tbody>
<tr>
<td>- constipation/diarrhea/mucoreea</td>
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<td>+ various changes in transit; discomfort</td>
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<th><strong>Bleeding</strong></th>
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<td>- systematic research on the occult (diet 2-3 days without iron)</td>
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<td>- rectorhagies</td>
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<th><strong>Anemia</strong></th>
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<td>- any unknown cause anemia needs colon investigations</td>
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<tr>
<td>- anemia – 90% cases hemorrhage</td>
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<th><strong>Others</strong></th>
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<tr>
<td>Suboclusion-sdr. König/ Occlusion (78% – cause cancer)</td>
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<tr>
<td>Pain</td>
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<tr>
<td>Fever-caused by tumor-necrosis sign tardy</td>
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<tr>
<td>Causes of fever: liver cancer, kidney cancer, lung cancer</td>
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<tr>
<td>Altered general state</td>
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Diagnosis

Physical examination
- palpation of the tumor, abdominal masses,
- Konig syndrome, pseudo-oclussion,
- rectorrhagies.

Explorations:
- Rectal touche, cancer of the rectum (-10%), rectorrhagies – especially the anal ring checks 10%,
- Rectoscopy – 30 cm in depth exploration – you can see the tumor, rectorrhagies – associated – 60%,
- Colonoscopy (fibrosigmoidoscopul-70 cm),
- Rx exam, Barium enema,
- Complete blood count, syderemia, feritine, total, protein, electrolyte balance (in case of diarrhea), renal function (acute enterocolitis, which can cause kidney failure).
Differential diagnosis

- familial polyposis-total colectomy with preservation of the rectum; removal of the polyp of rectal level+ biopsy; It is 6 in endoscopic 6 months.
- haemorrhagic rectocolitis (RCUH)
- invagination
- TB
- foreign body
- Crohn's disease
- parasitosis (amoebioza), colic diverticulosis
Complications

- Occlusion,
- Uterine fistulas,
- Hemorrhages - HDI (HDS can manifest through HDI when is massive),
- Enterocolitis with fever,
- Perforation with/without peritonitis – rare (4%).

Therapy:
- surgery – hemycolectomy + rectal amputation,
- chemo-radiation therapy, according to the guidelines,
- symptomatic-palliative.
Overposed on a chronic conditions:
- with usual constipation,
- dolico/megacolon,
- functional colopathy, IBS,
- needs investigations, repeated controls.

An acute episode which can be:
- acute enteropathy, infection, dysbiosis,
- hemorrhoids, anal fissures,
- perirectale fistulae or abscesses.

A condition which evokes neoplasie:
- hypochromic anemia,
- thrombophlebitis,
- weight loss,
- prolonged fever.