Ftplectures Gastrointestinal system Lecture Notes

GASTROLOGY

Medicine made

Ftplectures Clinical Medicine
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Adeleke Adesina, DO
Clinical Medicine

1133 Broadway Suite 706,
New York, NY, 10010

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Achalasia

Objectives for learning

Definition, pathophysiology, signs, symptoms, diagnosis, treatment and management.

Definition

Achalasia is the failure of lower esophageal sphincter to relax with swallowing.

Pathophysiology

It is due to loss of inhibitory ganglion cells of the myenteric plexus of the lower esophageal sphincter. There is impaired relaxation of lower esophageal sphincter which causes obstruction.

Signs and symptoms

Chest pain.
Progressive dysphagia i.e. from solids to liquids.
Nocturnal cough because undigested food products in the esophagus goes into trachea.
Weight loss

Diagnosis

Barium swallow reveal bird's beak sign.
Manometery of lower esophageal sphincter shows elevated pressure.
Endoscopy of lower esophagus shows constriction.

Treatment and management

Nitrates/Calcium channel blockers.
Endoscopic injection of botulinum toxin relaxes the sphincter.
Pneumatic balloon dilatation.
Heller's myotomy- surgical incison of lower esophageal sphincter.

Acute Appendicitis

Objectives for learning: Definition, Causes/ Risk factors, Pathophysiology, Clinical symptoms and signs, Physical exam, Diagnosis, Treatment and Complication.

Definition:

An inflammation of the appendix is known as appendicitis.

Causes/ Risk factors

- Lymphoid hyperplasia
- Fecalith
- Tumor/carcinoid tumor
- Foreign bodies

Pathophysiology

The afferent fibers supplying the appendix pick up the nerve sensations, cross and cause inflammation of the sympathetic truck at the T10 sympathetic level, causing periumbilical pain. As the appendix starts to get inflamed and gets biggers, it starts to rub against the peritoneal cavity and eventually the skeletal muscles get inflamed. Now as the inflammation has been spread, for that reason the periumbilical pain transmits to the lower abdominal quadrant.

Clinical Symptoms And Signs

- Nausea and Vomiting (Hamburger signs)
- Right lower quadrant pain
- Anorexia
- Low grade fever

Physical exam

Rovsing's sign

A patient is laid down and deep palpation is done from the left iliac fossa upwards. It will cause a pain in the right iliac fossa.

Obturator sign

A leg of the patient is flexed and internally rotated while he is lying. This will cause pain and is due to the spasm of obturator internus muscles.

Psoas sign

Psoas sign is a right lower-quadrant pain produced when the right hip of a patient is extended. It can also be elicited by flexing the right hip of a patient while it is in supine position. The pain is due to inflammation of the psoas muscles and inflammation of the peritoneum overlying the iliopsoas muscles.

Diagnosis

Appendicitis is a clinical diagnosis however following tests are recommended.

- KUB
- Ultrasound
- CT scan which may show peri-appendicular
- Increase WBC (1100 to 1500)

Treatment

- Bowel rest—Keep NPO.
- Antibiotics are given (usually cefotetan).
- IV fluids are given.
- Laparoscopy appendectomy is performed.

Complication

- ✓ Abscess can develop after surgery which requires drainage.

Acute pancreatitis

Objectives for learning

Anatomy, physiology, acute pancreatitis, etiology, symptoms, signs, diagnosis, treatment, management and complications.

Anatomy and physiology of pancreas

Pancreas is both endocrine and exocrine organ. It consists of body, head and tail. Duct of santorini runs from tail through the body into the head where it may or may not join with the bile duct to open into the duodenum.

Exocrine function of the pancreas is associated with digestion. Pancreas produce various enzymes:

- Bicarbonate neutralize the acidic chime from stomach.
- Chymotrypsin
- Lipase

Type of cells	**Secretion**
Alpha cells	Glucagon
Beta cells	Insulin
Delta cells	Somatostatin

Definition

Acute pancreatitis is the inflammation of the pancreas.

Etiology

Gallstone
Ethanol/alcohol
Trauma
Steroids
Mumps
Autoimmune
Scorpion bite
Hypertriglyceridemia
Drugs i.e. furosemide, thiazide, valproic acid, HIV drugs, metronidazole etc.

Symptoms

Severe epigastric pain going to the back
Nausea
Vomiting
Weakness

Signs

Grey turner sign- ecchymotic patch at the lower back

Cullen's sign- priumbilical ecchymotic patch

Diagnosis

Elevated amylase/lipase (more specific)
Decreased calcium level
Abdominal X-ray may reveal sentinel loop or colon-cutoff sign.
CT scan may show abscess, blood, pseudocyst etc.
Ultrasound may show clogging of stone at the ampulla of Vater.

Treatment and management

Stop oral intake.
IV fluid- normal saline, potassium ampoules, dextrose.
Analgesic.
Nasogastric tube suction of the undigested food.
Bowel rest.
Antibiotics and metronidazole.

Complications

Pancreatic pseudocyst-a cyst that has no true epithelial lining and is formed by the inflammatory process leading to scarring and entrapment of inflammatory fluid and pancreatic enzymes in the cyst. Elevated amylase level after resolution of acute inflammation is suggestive of pseudocyst. If the cyst is more than 5cm or persists for more than 6 weeks then it must be aspirated or surgically excised repectively.
Fistula formation.
Hypocalcemia.
Peritonitis.
Sepsis

Carcinoid syndrome

Objectives of learning

Definition, location, pathophysiology, signs, symptoms, diagnosis, treatment, precautions and complications.

Definition

Carcinoid syndrome is the tumor of endocrine system. It is also called neuroendocrine syndrome. The tumor usually produce chemical substances or hormones that are responsible for the symptoms.

Location

These rare tumors, if present, are commonly found inside the appendix.
2nd most common site is at the terminal ileum.
3rd site is inside lungs where it is called bronchial carcinoid.

Pathophysiology

Enterochromaffin cells of the gut produce normal amounts of serotonin in gastrointestinal system. Serotonin is helpful in gut motility. Carcinoid produces lot of serotonin which is made from tryptophan (an amino acid) by tryptophan hydroxylase. The excessive amount of serotonin, secreted in blood, goes to the liver via portal circulation. The liver tends to detoxify all the serotonin. Liver converts serotonin into 5'-hydroxyindoleacetic acid (5'HIAA), through monoamine oxidase (MAO), which is secreted out of the body through urinary system. But liver cannot degrade excessive amount of serotonin. So the remaining serotonin is sent back to the circulation which explains the signs and symptoms.

Signs and symptoms

Flushing of skin.
Diarrhea as a result of positive motility effect of serotonin.
Broncho constriction which causes wheeze.
Tricuspid/pulmonary valve insufficiency which is due to hypertrophy cardiac myocytes.
Pellagra (Vitamin-B3 i.e. Niacin deficiency) because tryptophan, which is also a precursor of niacin, is forced to produce great amounts of serotonin.

Diagnosis

5'-HIAA (5'-hydroxyindoleacetic acid) levels are very high in the urine.

Treatment

Octreotide is actually a somatostatin analog.
Surgical resection is used if tumor is localized.
Niacin is given.

Precautions

Diet low in tryptophan is helpful

Complication

Metastasis of tumor from appendix to liver and lungs.

Cholecystitis

Objectives for learning

Definition, pathophysiology, symptoms, signs, diagnosis, treatment, management and complications.

Definition

Inflammation of gallbladder is called cholecystitis.

Etiology

Gallstones

Pathophysiology

Gallstones when stuck produce biliary colic and when stone fall away, the pain is relieved. When gallstone permanently stuck in the cystic duct it results in continuous pain. The gallbladder is inflamed.

Symptoms

Continuous pain in the right upper quadrant.
Nausea.
Vomiting.
Low grade fever.

Signs

Boa's sign- pain radiating to back at the tip of shoulder girdle.
Murphy's sign- pressing the right upper quadrant with fingers along with deep breath results in sudden arrest of breathing as soon as the inflamed gallbladder is touched.

Diagnosis

Ultrasound may reveal lot of fluid around gallbladder (pericystic fluid), distended gallbladder and gallstone.
Abdominal X-ray may reveal calcified stone or calcified wall of gallbladder (porcelain gallbladder).
HIDA scan (Hepatoimidoacetic acid) is said to be positive when gallbladder is not visible upon injecting a dye because cystitis blocks the passage of dye into the gallbladder.

Treatment and management

NPO
IV fluids
Analgesics
Antibiotics
Cholecystectomy is the ultimate treatment.

Complications

- Rupture of gallbladder leading to biliary peritonitis and sepsis.
- Gallstone ileus- huge and inflamed gallbladder in conjunction with duodenum results in fistula formation and all the gallstones are shunted into the intestines. These stones block the GI tract at the ileocecal junction resulting in signs and symptoms of intestinal obstruction.
- Emphysematous cholecystitis.
- Gangrene of the gallbladder wall.

Choledocholithiasis:

Objectives for learning

Definition, symptoms, signs, clinical examination, lab investigations, diagnosis, treatment and management.

Ascending cholangitis, symptoms, signs, lab investigations, treatment and management.

Definition:

Choledocholithiasis means that stone has obstructed the common bile duct and, thus, the bile flow.

Signs and symptoms

- Right upper quadrant pain.
- Pain in the epigastrium.
- Yellowing of the eyes.
- Dark colored urine.
- Clay colored stool.

Clinical examination

On physical examination patient might have positive Murphy's sign (when you press the abdomen in the right hypochondrium then patient with hold his breath when inflamed gall bladder touches the parietal peritoneum).

Laboratory investigations:

Liver function tests (LFTs) ALT/AST

ALP (alkaline phosphatase)/GGT (gamma glutamyl transferase). Among all the liver function tests GGT is more specific for gall bladder and biliary tree.

Complete blood count (CBC).

Diagnosis and treatment:

Diagnosis and treatment overlaps each other and we make diagnosis via:

- Endoscopic retrograde cholangio pancreatography (ERCP). ERCP is done along with sphincterectomy.
- Laproscopic choledocho-lithotomy is also used.

ERCP is the gold standard approach for both diagnosis and treatment.

Ascending cholangitis:

Ascending cholangitis means ascend of infection, by over growth of the bacteria, through the common bile duct into the biliary system.

Clinical signs and symptoms:

Clinically this condition is diagnosed by the classical triad of charcot:

- Fever.
- Right upper quadrant pain.
- Jaundice.

Laboratory investigations:

- Liver function test (LFTs).
- ALP/GGT.
- Conjugated bilirubin.
- White blood cells increase (left shift).
- Blood culture.

Treatment:

ERCP is done in cases of any obstruction in the bile duct. If stones are the cause then stones can be retrieved through the sphincter of oddi and stone retrieval associated with the sphincterotomy.

Cholelithiasis:

Objectives of learning:

- Definition.
- Risk factors for cholesterol stones.
- Risk factors for pigment stones.
- Presentation.
- Diagnosis.
- Treatment.

Definition:

Cholelithiasis is the stone formation in the gall bladder.

Classification:

Gall bladder stones are divided into following types:

- Cholesterol stones (yellow-green stones).
- Pigment stones (black stones).

Risk factors for cholesterol stones:

- Obesity.
- Diabetes mellitus (type 2).
- Hyper-cholesterolemia.
- Oral contraceptive pills (OCP).
- Pregnancy.
- Cystic fibrosis.

Risk factors for pigment stones:

- Sickle cell anemia.
- Hereditary spherocytosis.
- Thalassemia.
- Prosthetic heart valves.

Presentation:

Patient having gall stones presents to physician with following problems:

- Biliary colicky pain.

- Boas sign (tingling sensations on the back of the shoulder).

Diagnosis:

Diagnosis is made through the following investigations:

- Ultrasonography.
- Computed-tomography.
- Magnetic resonance imaging (MRI).

Treatment:

Treatment for cholelithiasis is symptomatic, when they cause problems they are treated with surgical removal of the gall bladder (**cholecystectomy**).

Chronic pancreatitis:

Objectives for learning

Definition, etiology, symptoms, signs, lab investigations, treatment and complications.

Definition:

Chronic pancreatitis is the scarring of the pancreatic tissue after recurrent attacks of acute pancreatitis.

Etiology:

Alcoholics (90%).

Signs and symptoms:

- Recurrent episodes of epigastric pain radiating towards back
- Steatorrhea(fatty stool).
- Weight loss.
- Diabetes (type 1)
- Anorexia.

Laboratory investigations:

- Elevated amylase or lipase but most of the times these are in normal range in case of chronic pancreatitis.
- Specific laboratory investigation for chronic pancreatitis is stool elastase.

Treatment:

- Pain relief by various methods (mepridine/fentanyl, celiac ganglion block)
- Exogenous enzymes (trypsin/lipase).
- Diet containing medium chain fatty acids.
- Ask the patient to give-up drinking.

Complications:

Continuous alcohol intake will lead to **pancreatic cancer**.

Diffuse esophageal spasm:

Objectives of learning:

- Definition.
- Cause.
- Signs and symptoms.
- Diagnosis.
- Treatment.
- Differential diagnosis.

Definition:

Diffuse esophageal spasm is the high amplitude non-peristaltic contractions.

Cause:

Cause is unknown (idiopathic).

Signs and symptoms:

- Chest pain.
- Dysphagia.
- Odynophagia.

Diagnosis:

Barium swallow (cock screw appearance of the esophagus).

Treatment:

Treatment is symptomatic:

- Nitroglycerine.
- Calcium channel blockers.

Differential diagnosis:

- Angina pectoris.
- Myocardial infarction.

Diverticular disease

Objectives of learning

Definition, pathophysiology, risk factors, symptoms, signs, diagnosis, treatment and management.

Definition

Diverticulosis is the outpouchings of the colonic mucosa and submucosa due to weakening of the wall of colon.

Pathophysiology

Raised intra luminal pressure gradually weakens the wall of colon. Soon the weak part of colon expands and form a pocket called diverticula.

Risk factors

Very low fiber diet
Fatty food intake
More than 40 years old

Symptoms and signs

Fatigue
Painless bloody stools
Shortness of breath with exertion
Light headedness

Diagnosis

CBC may reveal anemia i.e. Hb less than 10, leukocytosis
Abdominal X ray may rule out perforation of gut
CAT scan is most important test

Treatment and management

IV fluids
Blood transfusion in case of severe anemia

Diverticulitis

It is the inflammation of diverticula. Unlikely to diverticulosis, there is no bleeding.

Etiology

It is usually caused by hard fecal matter called fecalith.

Symptoms

Left lower quadrant abdominal pain
Fever
Nausea
Vomiting
Constipation

Signs

Left lower quadrant tenderness

Diagnosis

CBC may show leukocytosis
CAT scan confirms the diagnosis

Treatment and management

Bowel rest i.e. NPO
Antibiotics usually metronidazole
Nasogastric tube
In case of perforated diverticulitis: Hartmann's procedure is done. In this procedure, the inflamed area is cut off and stump is formed. After 6 weeks, the two fresh parts of colon are rejoined.

Esophageal cancer:

Objectives for learning

Classification, risk factors of squamous/adenocarcinoma, symptoms, signs, diagnosis, treatment and prognosis.

Classification:

- Squamous cell carcinoma.
- Adenocarcinoma.

Risk factors for squamous cell carcinoma:

- Smoking.
- Alcohol.

Risk factors for adenocarcinoma:

- Gastro-esophageal reflux disease (acid reflux).
- Barrett's esophagus (columnar metaplasia).

Signs and symptoms:

- Progressive dysphagia.
- GERD (gastro esophageal reflux disease).
- Weight loss.
- Odynophagia.
- Nausea and vomiting.

Diagnosis:

- Barium swallow helps us to check the level of obstruction.
- Upper GI endoscopy with biopsy.

Treatment:

- Chemo-radiation to shrink the tumor.
- Surgical resection if it possible.
- Stent placement if the surgical resection is not possible.

Prognosis:

Prognosis for squamous cell carcinoma is poor.

Esophageal diseases:

Dysphagia:

Definition:
Dysphagia is difficulty in swallowing.

Causes:
- Strictures.
- Schatzki rings.
- Webs (plummer vinson syndrome).
- Esophageal cancer.

Diagnosis:
- Barium swallow.
- Endoscopy.
- pH monitoring.
- Esophageal manometery.

Odynophagia:
Odynophagia is painful swallowing.

Causes:
- Infections.
- Drug induced.

Diagnosis:
Endoscopy is used for diagnosis of the condition.

Esophageal diverticula:

Objectives of learning:

- Definition.
- Presentation.
- Diagnosis.
- Treatment.

Definition:

Esophageal diverticular is the cervical out pouching through the cricopharyngeal muscle also called as zenker's diverticulum.

Presentation:

- Bad breath (halitosis).
- Chest pain.
- Coughing up undigested food products.
- Dysphagia.

Diagnosis:

- Barium swallow.

Treatment:

- Surgical removal of the diverticula
- Myotomy.

Esophagitis:

Objectives for learning:

Definition, etiology, symptoms, signs and treatment.

Definition:

Esophagitis is the inflammation of the esophagus.

Etiology:

Infections:

- Pharyngitis.
- Candidiasis (oral thrush).
- Herpes simplex infection.
- Cytomegalo-virus (CMV) esophagitis.

Drug induced esophagitis:

- Potassium chloride (KCL).
- Aspirin.
- NSAIDs.
- Bisphosphonates.

Signs/symptoms:

- Painful swallowing.

Treatment:

Treatment of the esophagitis depends upon the cause which is causing this condition:

- Oral fluconazole is used to treat the candida associated esophagitis.
- Acyclovir I/V is used to treat the herpes simplex virus associated esophagitis.
- Gancyclovir is used to treat the cytomegalovirus associated esophagitis.

Gastroesophageal reflux disease (GERD)

Objectives for learning

Definition, risk factors, diagnosis, complications treatment and replication.

Definition

Reflux of acidic contents from stomach into the esophagus is gastroesophageal reflux disease.
Symptoms and signs
Sour taste
Unexplained cough
Hoarseness

Risk factors

Spicy foods
Alcohol
Caffeine
Garlic onions
Mints
Nicotine

Treatment and management

Lifestyle modification
Antacids i.e. calcium carbonate
H_2 blockers i.e. nizatidine, cimetidine
Proton pump inhibitors (best drug) i.e. omeprazaole, esomeprazole
Nissen fundoplication is invasive procedure for long term treatment

Complications

Esophagitis
Esophageal strictures
Upper GI bleeding
Aspiration of GI contents
Barrett's esophagus

Gastritis

Objectives for learning

Definition, acute gastritis, chronic gastritis, etiology, pathogenesis, signs, symptoms, diagnosis, treatment and complications.

Definition

'Gastric' means 'stomach' and 'itis' means 'inflammation.

Acute gastritis

Acute gastritis is the severe inflammation of stomach lining. It is also called 'erosive gastritis' because the mucosal lining is eroded.

Etiology and pathogenesis of acute gastritis

1. NSAIDS (non-steroidal anti-inflammatory drugs) are the most common cause of acute gastritis e.g. aspirin, ibuprofen, ketorolac etc. They interfere with arachidonic acid pathway and reduce the amount of prostaglandins that are necessary for mucosal wall protection of the stomach.
2. Alcohol intake, especially binge drinking, is responsible for removing the mucosal layer of stomach lining.
3. Stress
4. Burns result in thin skin and loss of water and, thus, hypovolemia, which results in decreased perfusion of visceral organs including stomach. The partial ischemia is responsible for ulcer that is also known as Cushing's ulcer.

Signs and symptoms

Epigastric pain
Hematemesis (vomiting blood)
Melena (black stools due to blood)

Diagnosis

Endoscopy reveal the gastric lining
Urease breath test, if positive, indicate H. Pylori infection.
Biopsy is required for confirmation.
IgG and IgM reveal chronic and acute infections respectively.

Treatment and management of acute gastritis

Antacids
H_2 blockers i.e. Ranitidine
PPI (proton pump inhibitor) reduce HCl production.
Sucralfate projects the lining of the stomach.
Stop alcohol intake

Chronic Gastritis

It is also called non-erosive gastritis.

Types

Type A gastritis is autoimmune and incidence is 10% of all types of chronic gastritis.
Type B gastritis is caused by *H. Pylori* infection and incidence is 90% of all types of chronic gastritis.

Complications

Vitamin B12 deficiency and intrinsic factor deficiency causes pernicious anemia.
MALT (lymphoma) is complication of chronic *H. Pylori* infection.

Treatment and management

Triple management of amoxicillin, clarithromycin and omeprazole not only relieve the patient but also reduce the incidence of this disease.

Guillain Barre Syndrome

Objectives for learning

Definition, etiology, signs and symptoms, diagnosis, differential diagnosis, treatment and management, and prognosis.

Definition

Guillain Barre Syndrome is demyelinating disease of the motor neurons. It is characterized by the inflammation and polyneuropathy.

Etiology

The GB syndrome is preceded by the infections e.g. upper respiratory diseases, gastrointestinal diseases
GI infections include *campylobacter jejuni*
Viral infections include CMV, Hepatitis, and HIV

Signs and symptoms

It has an abrupt onset which is severe and manifests itself in the form of ascending paralysis i.e. starts from the extremities and goes towards the center of the body. The person is unable to move the extremities but the sensory nerves are intact. Once the disease reaches the center of the body and involve diaphragm, the consequences are disastrous because of respiratory failure.
This disease may also include the autonomic features such as arrhythmia, tachycardia, and postural hypotension.

Diagnosis

CSF analysis by lumbar puncture reveal elevated protein but normal cell count.
Nerve conduction studies reveal decrease motor conduction velocity because of demyelination of the nerves.

Differential diagnosis

Multiple sclerosis. The sphincter and bladder control are intact in GB syndrome as compared to multiple sclerosis.

Treatment and Management

Pulmonary function monitoring is compulsory.
IVIG for severe weakness are useful.
Plasmapheresis is used to filter the antibodies.
NO steroids because it will worsen the disease.

Prognosis

Most of the patient recover in 1-3 weeks.
After 6 weeks, the prognosis is bad.

Hemochromatosis:

Objectives of learning:

- Definition.
- Causes.
- Presentation.
- Laboratory investigations.
- Treatment.
- Complications

Definition:

Hemochromatosis is the excess deposition of iron in the body.

Causes:

It is genetically predisposed condition and transmitted as autosomal recessive trait.

Signs and symptoms:

- Tiredness.
- Joint aches.
- Cirrhosis of liver.
- Congestive heart failure (CHF).
- Diabetes mellitus.
- Abdominal pain.
- Polyphagia, Polydypsia, Polyuria.
- Testicle atrophy.
- Erectile dysfunction.
- Restrictive cardio-myopathy.
- Skin is bronze looking.

Diagnosis:

For diagnosis of the hemochromatosis we shall order following laboratory investigations:

- Increased ferritin levels.
- Increased serum iron.
- Increased serum transferrin.
- Increased blood glucose.
- Disturbances of ALT/AST.

Liver biopsy gives the definitive diagnosis for the condition.

Treatment:

- Phlebotomy.
- Defroxamine (iron chelating agent).

Complications:

People suffering from this condition are more predisposed to infections caused by following organisms:

- Listeria monocytogenes.
- Vibrio vulnificus.
- Yersinia enterocolitis.

Hepatitis B:

Objectives of learning:

- Structure.
- Transmission.
- Pathogenesis.
- Serologic course.
- Diagnosis.
- Treatment.

Structure:

Hepatitis B virus belongs to a family known as hepadna virus. According to the structure of the hepatitis B virus there are 3 antigens:

- Surface antigen (HBsAg).
- Early antigen (HBeAg).
- Core antigen (HBcAg).

DNA of the hepatitis B virus is enclosed within the icosahedral nucleo-capsid. DNA polymerase is an enzyme attached with the DNA and is essential for its replication.

Transmission:

Transmission occurs via:

- Blood transfusion.
- Needle-stick injury.
- Sexual activity.

Pathogenesis:

Hepatitis B virus attacks the hepatocytes by attaching to the receptors on the hepatocytes and after attachment virus moves inside the cell by endocytosis. Body produces response against the virus by releasing the virus specific cytokines. These cytokines activates the T-lymphocytes. T-lymphocytes are cytotoxic and kill the virus and during this killing process hepatocytes are destroyed in this way hepatocellular damage occur. Enzymes are released when hepatocytes are destroyed these are ALT (alanine amino-transferase) and AST (aspartate amino-transferase) being ALT is more specific for liver damage.

Serological course:

70% of the people infected with hepatitis B virus are asymptomatic that's why we call it a "silent disease".

20% of the people infected with hepatitis B virus develop severe symptoms.

5% of the people infected with hepatitis B become a chronic carrier.

During acute attack of the disease HBsAg elevates in the blood. Body produces antibodies (IgM) against the HBcAg. HBeAg is also detected in the blood during acute phase that's why it is called as early antigen. HBeAg shows acute phase reaction and high infectivity. After acute phase reaction antibodies are produce in the body:

- Anti HBc antibodies (IgG): Detects in the body during window period
- Anti HBe antibodies (IgG).
- Anti HBs antibodies (IgG): Depicts that's acute infection of the hepatitis B with complete recovery.

Window period is the period when serological test shows negative HBsAg, but positive HBcAb it means body is clearing the virus from the body.

If the person remians positive for HBsAg even after 6 months then it means that he is **chronic carrier** for hepatitis B.

Signs and symptoms:

Flu like symptoms:

- Fever.
- Chills.
- Myalgia.
- Joint aches.
- Nausea.
- Headache.
- Loss of appetite.

Icteric symptoms:

- Right upper quadrant pain.
- Jaundice.
- Fever.

Laboratory investigations:

- HBsAg+, HBeAg+, and HBsAb- = acute infection of hepatitis B.
- HBsAg-, HBsAb+ = vaccinated.
- HBcAb+, HBsAg-, HBsAb+ = past infection and completely recovered.
- HBcAb+, HBsAg-, HBsAb- =window period.

Treatment:

- Alpha interferon.
- Adefovir.
- Lamuvidine.
- Entacavir.
- Tenofovir/telbuvidine.

Complications:

- Cirrhosis of the liver.
- Portal hypertension.
- Hepatocellular carcinoma (HCC).

Hepatocellular tumors:

Classification:

Liver tumors are divided into two types:

- Adenoma.
- Hepatocellular carcinoma.

Adenoma:

Incidence:

Common in middle aged women.

Etiology:

- Oral contraceptive pills (increase in estrogen).

Diagnosis:

Ultrasonography shows mass in the liver with well-defined borders.

Laboratory investigations:

- Increase in AST/ALT.
- Disturbances in the gamma glutamyl-transferase (GGT).

Treatment:

Treatment is symptomatic.

Hepatocellular carcinoma:

Risk factors:

- Most common cause of the liver cancer is metastasis from different organs in the body.
- Liver cirrhosis.
- Chronic alcohol intake.
- Hepatitis C (80% develops HCC)
- Hepatitis B (20% develops HCC).
- Aflatoxins (substances from mushrooms).

Clinical symptoms:

- Right upper quadrant pain.
- Abdominal tenderness.
- Jaundice.
- Disturbances in the coagulation cascade.

Investigations:

- Abdominal ultrasonography shows ill-defined border mass.
- CT-scan shows metastasis and mass in the liver.
- Alpha-fetoproteins (AFP) are elevated.

Definitive diagnosis can only be made only on the basis histo-pathological examination

Treatment:

- Surgical resection if the tumor is surgically resectable.
- Chemotherapy/radiation therapy.
- Liver transplant is the last resort.

Complications:

- Gastro-intestinal bleeding.
- Liver failure.

Jaundice

Objectives for learning

Definition, pathology, causes of conjugated/unconjugated jaundice, symptoms, physical examination, diagnosis, treatment and management.

Definition

Yellow discoloration of the skin, sclera and mucous membranes due to over production or under clearance of bilirubin.

Pathologic jaundice

Levels of bilirubin greater than 2mg/dl. Also known as hyperbilirubinemia
There are two types of jaundice:

1. Unconjugated
2. Conjugated

Causes of unconjugated hyperbiliruinemia

The large amount of bilirubin reaches the liver and liver cannot conjugate the excessive amounts of bilirubin. So unconjugated bilirubin levels are high.

1. Increased hemolysis i.e. microangiopathic hemolytic anemia, aortic stenosis, valvular stenosis, autoimmune hemolysis, hereditary spherocytosis
2. Gilbert's syndrome appear when body is under stress and liver cannot conjugate the bilirubin.
3. Crigler-Nijjar syndrome is genetic defect in which the conjugating enzymer 'uridine glucuronosyltransferase' is absent.
4. Physiologic jaundice in newborn
5. Cirrhosis of liver because the liver parenchyma is dead and the remaining healthy liver has lost its ability to conjugate
6. Drugs such as sulfonamides, penicillin, rifampin etc

Causes of conjugated hyperbilirubinemia

1. Extra-hepatic obstruction of biliary tract i.e.
    - Gallstones,
    - primary sclerosing cholangitis,
    - primary biliary cirrhosis,
    - cholangiocarcinoma,
    - Klatskin's tumour,
    - bilary atresia and
    - pancreatic cancer.
2. Hereditary inability to excrete the conjugated bilirubin out of the liver i.e.

- Dubin-Johnson syndrome (liver is black on biopsy)
- Rotor syndrome

Symptoms

Yellow eyes

Physical examination

Yellow sclera.
Yellow mucous membrane.
Urine color may be dark in conjugated hyperbilirubinemia.

Diagnosis

Liver function tests may be high in case of hepatitis, cirrhosis.
Alkaline phosphatas/GGT levels show the status of bilary tree.
Blood levels of conjugated and unconjugated bilirubin.
Serum albumin
PT/APTT

Treatment and management

Diagnose and treat the original cause of jaundice i.e. hepatitis, primary biliary cirrhosis, gallstone etc.

Liver function tests:

Objectives of learning:

- Anatomy of the liver.
- Hepatocytes.
- Hepatocyte injury.

Anatomy of the liver:

Inferior mesenteric vein +superior mesenteric vein+ splenic vein combines to form the portal vein that enters the liver.

Intra-hepatic biliary ducts combined to form the right and left hepatic ducts which combines to form the common hepatic duct and it combines with the cystic duct to form the common bile duct and that drains into the duodenum.

Hepatocytes:

Hepatocytes are the liver cells and contains following enzymes:

- Aspartate transaminase (AST).
- Alanine transaminase (ALT).

Alanine transaminase (ALT) is more specific to the liver than aspartate transaminase (AST).

Hepatocyte injury:

Alcohol is converted to aldehyde and aldehyde activate stellate cells which normally stores vitamin A and lay down collagen and in this way fibrosis occurs.

Mesenteric ischemia

Objectives for learning

Definition, pathophysiology, signs, symptoms, diagnosis, treatment & management, complications.

Definition

Mesenteric ischemia is the ischemia of gut resulting from blockage of blood supply in the mesenteric arteries.

Pathophysiology

Atherosclerosis is the most common cause. The atheroma formation gradually blocks the lumen of the artery. Hyperlipidemia i.e. low HDL and high LDL enhances the atheroma formation.
Embolism is the clot that breaks off from some other part of the body (i.e. mural thrombus in atrial fibrillation or heart failure) travels down to the mesenteric artery.
Venous thrombosis as a result of hypercoaguble states is responsible for blood compromise e.g. FactorV deficiency, antithrombin III deficiency, protein C deficiency, homocystinemia

Symptoms

Severe abdominal pain immediately after eating.
Nausea
Vomiting
Diarrhea with blood because ischemia results in necrosis and infarcted bowel wall bleeds.

Signs

Tender abdomen. Pressing the abdomen produces severe pain.

Diagnosis

CBC may reveal leukocytosis
Lactate levels may be raised because ischemia results in anaerobic metabolism which results in lactic acid production and greater amount of lactic acid is responsible for metabolic acidosis.
Amylase/ LDH levels may be high.
Abdominal X-ray/CT scan reveal the air-fluid levels i.e. pneumatosis intestinalis also called thumb-printing sign.
Mesenteric angiography in the gold standard for diagnosis os acute mesenteric ischemia.

Treatment and management

IV fluids for compensating the water loss.
Antibiotics and Metronidazole.
Anticoagulants i.e. Heparin or warfarin.
Angioplasty.

Complications

Sepsis may lead to shock and death.
Complete necrosis of part of gut.

Peptic ulcer disease

Objectives of learning

Definition
Etiology
Pathophysiology
Symptoms and signs
Diagnosis
Treatment and management
Complications

Definition

Peptic ulcer is the ulcer residing in the stomach or in the duodenum.

Etiology

Helicobacter pylori is most common cause
NSAIDs are the second most common cause
Zollinger-Ellison syndrome
Alcohol, smoking, caffeine intake

Pathophysiology

The bacteria has urease enzyme that increases the pH of the stomach and creates suitable environment for the bacteria.
NSAIDs reduce the prostaglandins and ultimately affects the protective lining of the stomach.
In Zollinger-Ellison syndrome, there is increase secretion of gastrin which stimulates the cells to produce greater amount of HCl.

Symptoms and signs

Epigastric pain
Nocturnal pain (duodenal ulcer)
Relationship with food: Pain increases (stomach ulcer) and pain decreases (duodenal ulcer)

Diagnosis

Endoscopy and biopsy is done (gold standard)
Barium swallow
Urease breath test
Elevated gastrin levels in Zollinger-Ellison syndrome

Treatment and management

Stop NSAIDs, alcohol, smoking, caffeine intake
H_2 blockers i.e. cimetidine, ranitidine etc.
Proton pump inhibitors (PPI) i.e. omeprazole, esomeprazole
For treating bacteria: clarithromycin, amoxicillin, bismuth and PPI (triple therapy) is given
Sucralfate- forms a protective covering over the ulcer
Misoprostol- a prostaglandin analog

Complications

Ulcer can perforate leading to spillage of contents into the peritoneum resulting in the peritonitis leading to shock culminating to death.
Hemorrhage

Portal hypertension

Objectives of learning

Definition
Physiology of portal system
Portal hypertension

Definition

Portal hypertension is the raised venous pressure in the portal veins due to some underlying cause.

Physiology of portal system

The hepatic portal vein is primarily formed by the union of two veins i.e.

I. Superior mesenteric vein
II. Inferior mesenteric vein

Superior mesenteric vein
It drains blood from the duodenum, jejunum, ileum, appendix, cecum, ascending colon and half of the transverse colon. It also drains blood from the pancreas and gallbladder.

Inferior mesenteric vein
It drains blood from lower esophagus, stomach, distal half of large colon, descending colon, sigmoid colon and rectum.

Portal hypertension

Esophageal veins are affected by the portal hypertension and result in esophageal varices.
Middle and inferior rectal veins are dilated and may form hemorrhoids in case of portal hypertension.
Paraumbilical veins when dilated appear in the form of veins radiating from the umbilicus. This is termed as caput medusa.

Primary biliary cirrhosis

Objectives of learning

Definition
Pathophysiology
Symptoms
Signs
Diagnosis
Treatment and
Management

Definition

It is the autoimmune destruction of the intra-hepatic bile duct.

Pathophysiology

The autoimmune destruction of the bile ducts within the liver resulting in obstruction of the bile channels leading to fibrosis. The scar tissue is formed and it obstructs the flow of the bile. The extra bile is secreted into the blood and produce symptoms of jaundice.

Symptoms

Right upper quadrant pain
Yellowish eyes
Itching
Yellowish eyeballs

Signs

Yellow conjunctiva
Yellow mucous membranes

Diagnosis

Cholestatic LFT i.e. elevated ALT and Alkaline phosphatase
Elevated total bilirubin (usually conjugated)
Positive anti-mitochondrial antibody (AMA-IgM immunoglobulin)
Biopsy is the gold standard for diagnosis

Treatment and management

Cholestyramine-bile acids binder
Ursodeoxycholic acid-hydrophillic bile acid
Liver transplant is the ultimate treatment

Primary sclerosing cholangitis

Objectives for learning

Definition
Etiology
Symptoms
Signs
Diagnosis
Association with other disease
Treatment and
Management

Definition

It is chronic condition of intra-hepatic or extra-hepatic bile walls destruction leading to stricture formation and blockage of the bile.

Etiology

It is idiopathic condition

Symptoms

Right upper quadrant pain
Yellowish eyes
Itching
Yellowish eyeballs

Signs

Yellow conjunctiva
Yellow mucous membranes

Association with other disease

Ulcerative colitis (70%)

Diagnosis

ERCP (endoscopic retrograde cholangiopancreatography)
PTC (percutaneous trans-hepatic cholangiogram)

Elevated ALT and Alkaline phosphatase

Treatment and management

Cholestyramine
ERCP
Liver transplant is the ultimate treatment

Small bowel obstruction

Objectives for learning

Definition
Pathophysiology
Etiology
Symptoms
Signs
Diagnosis
Treatment and
Surgical management

Definition

Obstruction of small bowel i.e. duodenum, jejunum and ileum can be of two types

I. Partial obstruction
II. Complete obstruction

On the basis of loop, there are two types of obstruction

I. Open loop obstruction
II. Closed loop obstruction

Pathophysiology

Dehydration is the main etiological factor of obstruction. GI tract needs lot of fluids to keep the fecal matter semi-solid and maintain the motility of thee feces. The solid fecal matter gradually accumulates in the intestines leading to increase in diameter of the bowel and ultimately obstruction. There is metabolic alkalosis and hypokaelemia. The low potassium levels cause tachycardia and altered mental status.

Etiology

Adhesions after surgery are most common cause
Incarcerated hernia is the 2nd most common cause
Intussusception
Cancer

Symptoms

Abdominal pain: diffuse and continuous
Distension
Nausea
Vomiting

Signs

Distended abdomen
Non-specific tenderness all over the abdomen
Hernia may be seen clearly, if present

Diagnosis

Abdominal plain film may show dilated loop of bowels with air-fluid level
Barium enema
Upper GI series

Treatment

IV fluids
NG tube
Potassium chloride

Surgical management

Complete obstruction
Strangulated hernia
Adhesions exploratory laparotomy

Ulcerative colitis:

Objectives of learning:

- Definition.
- Incidence.
- Gross appearance.
- Presentation.
- Extra-intestinal presentation.
- Complications.
- Treatment.

Definition:

Ulcerative colitis is the chronic inflammatory disease of the colon or the rectum.

Incidence:

It occurs in young adults/adolescents.

It can involve rectum and colon:

- Only rectum-10%.
- Rectum and left colon-40%.
- Rectum, left and right colon-30%.
- Backwash ileitis-10%.

Gross appearance:

- Uninterrupted involvement of the rectal mucosa.
- Only restricted to the mucosa lining and submucosa.
- Poly morphonuclear neutrophils.

Presentation:

- Hematochezia (bloody diarrhea).
- Pain abdomen.
- Frequent bowel movement
- Fever.
- Anorexia.
- Weight loss.
- Tenesmus (incomplete evacuation of the rectum).

Extra-intestinal presentations:

- Skin lesions-pyoderma gangreosum.
- Eye-anterior uveitis.
- Joints-arthritis.

Diagnosis:

- Stool culture-check for clostridium difficile, ova, parasites
- Fecal leukocytes.
- Colonoscopy-we see the extent of the disease.

Complications:

Patient can have following complication:

- Iron deficiency anemia.
- Bleeding.
- Dehydration.
- Increased risk of developing the colo-rectal cancer.
- Primary sclerosing cholangitis (PSC).
- Cholangiocarcinoma.
- Toxic mega-colon.
- Depression.
- Narcotic abuse just to get relief from pain.

Treatment:

Following treatment options are available for ulcerative colitis:

- Systemic corticosteroids.
- Sulphasalazine-5 amino-salicyclic acid (5-ASA).
- Azathioprine/6-merceptopurine.
- Total colectomy.