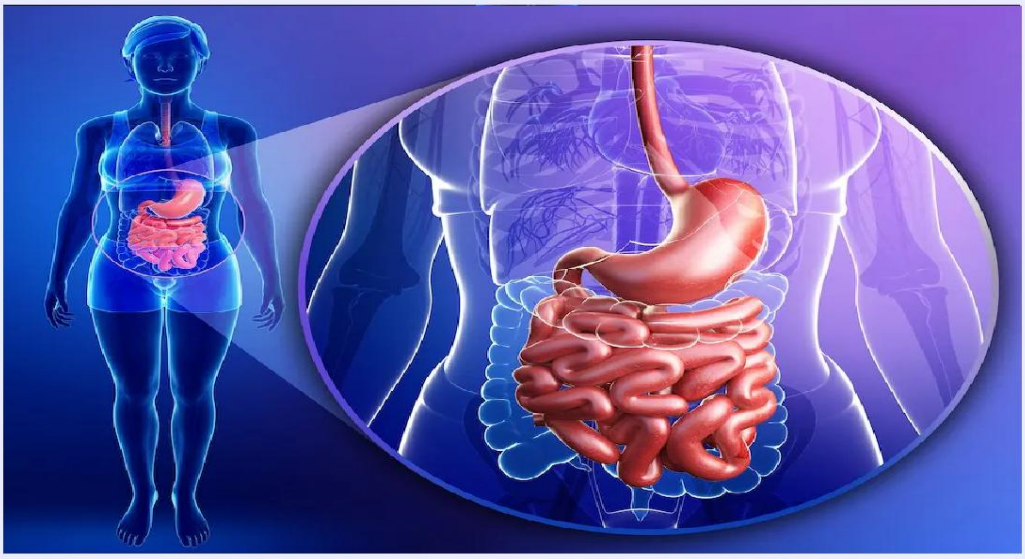
May -2024

S.TENTISHEV ASIAN MEDICAL INSTITUTE

Kant-Kyrgyzstan

Inter-Professional Discipline Department
"Gastrointestinal Tract"



T.S.A.M

"THE STUDENT'S ABSTRACT OF MEDICINE"

FOR

Interpreofessional Communication And Partnership In Health Care & Medical Education

5th

Dr.AFTAB SHEIKH
Senior Lecturer
Orthopedic Surgeon & Traumatologist

AsMI
Pharmacological
Society

S. Tenishev Asian Medical Institute

ABSTRACT

Department of Interprofessional Disciplines

A collection of works by AzMI students under the guidance of

Senior lecturer Dr.Aftab Sheikh in the specialty

General Medicine & Dentistry

1-Collection of works by AzMI students under the guidance of Senior lecturer ***Dr.Aftab Sheikh*** was approved and recommended in meeting of
The Department of Interprofessional Disciplines ,Protocol No.01 ,dated 5th of September 2023 academic year.
2-***Approved by Head of the Department Ryspekova Altynay Erkinbekovna on specialities GENERAL MEDICINE 560001 and Stamotology 540004***
3-The collection consists of 40-45 pages.
4-The collection will be published 7 times in an academic year
5-Release date from the 20th to 30th of the month.
6-Work of students of 3-4-5 years of 5 and 6 year programs as included and recommended
7-Relevance of the collection of information about the importance of thetopic of disease of organ systems and other diseases like infections etc.
8-Rules and requirement for students research paper students submit their topic by the 10th of the month included REFERENCES

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Gastritis

OVERVIEW

Gastritis is a general term for a group of conditions with one thing in common: Inflammation of the lining of the stomach. The inflammation of gastritis is most often the result of infection with the same bacterium that causes most stomach ulcers or the regular use of certain pain relievers. Drinking too much alcohol also can contribute to gastritis.

Gastritis may occur suddenly (acute gastritis) or appear slowly over time (chronic gastritis). In some cases, gastritis can lead to ulcers and an increased risk of stomach cancer. For most people, however, gastritis isn't serious and improves quickly with treatment.

EPIDEMIOLOGY

At the global level, there were 30.9 million (95% UI: 25.4 to 36.7) incident cases of GD in 2019 , with an ASIR of 379.9/100,000 persons (95% UI:312.42 to 448.12) . Over the 30-year period, only the low SDI region experienced an increasing ASIR for GD, whereas the ASIR in the other four SDI regions decreased. Among the 21 GBD regions, the Central Sub-Saharan Africa region showed the most significant increase in ASIR, while the Tropical Latin America region had the most significant decrease in ASIR. Examining the countries-level data, the three countries with the highest ASIR are Angola, Congo and Central African Republic, while the three countries with the lowest ASIR are Brunei Darussalam, Japan and Singapore

SYMPTOMS

Gastritis doesn't always cause symptoms. When it does, the symptoms of gastritis may include:

- ✓ Gnawing or burning ache or pain, called indigestion, in your upper belly. This feeling may become either worse or better after eating.
- ✓ Nausea.
- ✓ Vomiting.
- ✓ A feeling of fullness in your upper abdomen after eating.

CAUSES

Gastritis is an inflammation of the stomach lining. The stomach lining is a mucus-lined barrier that protects the stomach wall. Weaknesses or injury to the barrier allows digestive juices to damage and inflame the stomach lining. Several diseases and conditions can increase the risk of gastritis. These include inflammatory conditions, such as Crohn's disease.

RISK FACTORS

Factors that increase your risk of gastritis include:

- ✓ **Bacterial infection.** A bacterial infection called Helicobacter pylori, also known as H. pylori, is one of the most common worldwide human infections. However, only some people with the infection develop gastritis or other upper gastrointestinal disorders. Healthcare professionals believe sensitivity to the germs could be inherited. Sensitivity also may be caused by lifestyle choices, such as smoking and diet.
- ✓ **Regular use of pain relievers.** Pain relievers known as nonsteroidal anti-inflammatory drugs, also called NSAIDs, can cause both acute gastritis and chronic

gastritis. NSAIDs include ibuprofen (Advil, Motrin IB, others) and naproxen sodium (Aleve, Anaprox DS). Using these pain relievers regularly or taking too much of these medicines may damage the stomach lining.

- ✓ **Older age.** Older adults have an increased risk of gastritis because the stomach lining tends to thin with age. Older adults also have an increased risk because they are more likely to have H. pylori infection or autoimmune disorders than younger people are.
- ✓ **Excessive alcohol use.** Alcohol can irritate and break down your stomach lining. This makes your stomach more vulnerable to digestive juices. Excessive alcohol use is more likely to cause acute gastritis.
- ✓ **Stress.** Severe stress due to major surgery, injury, burns or severe infections can cause acute gastritis.
- ✓ **Cancer treatment.** Chemotherapy medicines or radiation treatment can increase your risk of gastritis.
- ✓ **Your own body attacking cells in your stomach.** Called autoimmune gastritis, this type of gastritis occurs when your body attacks the cells that make up your stomach lining. This reaction can wear away at your stomach's protective barrier.

    - Autoimmune gastritis is more common in people with other autoimmune disorders. These include Hashimoto's disease and type 1 diabetes. Autoimmune gastritis also can be associated with vitamin B-12 deficiency.

- ✓ **Other diseases and conditions.** Gastritis may be associated with other medical conditions. These may include HIV/AIDS, Crohn's disease, celiac disease, sarcoidosis and parasitic infections.

PATHOGENESIS

- ✓ The continuous mucosal injury due to long-standing H. pylori infection, leads to atrophy of stomach.
- ✓ This continuous pathological process results in erosion or ulceration of the mucosa leading to the destruction of the glandular layer and followed by fibrous replacement.
- ✓ The destruction of the glandular basement membrane and the sheath of supporting cells prevents orderly regeneration. This uneven regeneration follows a divergent differentiation pathway producing metaplastic glands (pseudo-pyloric appearance) which are composed of cells of the 'ulcer-associated cell lineage' (UACL).

PATHOLOGY

- Reduced number of oxyntic cells. No intestinal metaplasia
- The mucosa is infiltrated with neutrophils
- H. pylori is not seen on H&E stain.
- Immunohistochemical stain of H. pylori detects the organisms.

DIAGNOSIS OF GASTRITIS & GASTROPATHY

How do doctors diagnose gastritis and gastropathy?

Your doctor will ask about your medical history, symptoms, and any medicines you take. Your doctor will also perform a physical exam and may order an upper gastrointestinal (GI) endoscopy with biopsies or other tests.

UPPER GI ENDOSCOPY

Upper GI endoscopy is a procedure in which a doctor uses an endoscope—a flexible tube with a camera—to see the lining of your upper GI tract, including your esophagus, stomach, and duodenum. During upper GI endoscopy, a doctor obtains biopsies by passing an instrument through the endoscope to take small pieces of tissue from your stomach lining. A pathologist will examine the tissue with a microscope. Doctors may use upper GI endoscopy to diagnose gastritis or gastropathy, determine the cause, and manage complications.

BLOOD TEST

Doctors may use blood tests to check for other causes of gastritis or signs of complications. For a blood test, a health care professional will take a blood sample from you and send the sample to a lab.

STOOL TEST

Doctors may use stool tests to check for *H. pylori* infection and for blood in your stool, a sign of bleeding in your stomach.

UREA BREATH TEST

Doctors may use a urea breath test to check for *H. pylori* infection. For the test, you will swallow a capsule, liquid, or pudding that contains urea that is "labeled" with a special carbon atom. If *H. pylori* is present, the bacteria will convert the urea into carbon dioxide. After a few minutes, you will breathe into a container, exhaling carbon dioxide. A health care professional will test your exhaled breath. If the test detects the labeled carbon atoms, the health care professional will confirm an *H. pylori* infection in your digestive tract.

UPPER GI SERIES

Doctors may use an upper GI series to check for signs of gastritis or gastropathy. An upper GI series is a procedure in which a doctor uses x-rays and a chalky liquid called barium to view your upper GI tract.

TREATMENT

Treatment of gastritis depends on the specific cause. Acute gastritis caused by NSAIDs or alcohol may be relieved by stopping use of those substances.

Medicines used to treat gastritis include:

- **Antibiotics to kill H. pylori.** For H. pylori in your digestive tract, your healthcare professional may recommend a combination of antibiotics to kill the germs. Be sure to take the full antibiotic prescription, usually for 7 to 14 days. You also may take a medicine to block acid production. Once treated, your healthcare professional will retest you for H. pylori to be sure it has been destroyed.
- **Medicines that block acid production and promote healing.** Medicines called proton pump inhibitors help reduce acid. They do this by blocking the action of the parts of cells that produce acid. You may get a prescription for proton pump inhibitors, or you can buy them without a prescription.

Long-term use of proton pump inhibitors, particularly at high doses, may increase your risk of hip, wrist and spine fractures. Ask your healthcare professional whether a calcium supplement may reduce this risk.

- **Medicines to reduce acid production.** Acid blockers, also called histamine blockers, reduce the amount of acid released into your digestive tract. Reducing acid relieves gastritis pain and encourages healing. You may get a prescription for an acid blocker, or you can buy one without a prescription.
- **Medicines that neutralize stomach acid.** Your healthcare professional may include an antacid in your treatment. Antacids neutralize existing stomach acid and can provide rapid pain relief. These help with immediate symptom relief but are generally not used as a primary treatment. Side effects of antacids can include constipation or diarrhea, depending on the main ingredients. Proton pump inhibitors and acid blockers are more effective and have fewer side effects.

COMPLICATIONS

Left untreated, gastritis may lead to stomach ulcers and stomach bleeding. Rarely, some forms of chronic gastritis may increase your risk of stomach cancer. This risk is increased if you have extensive thinning of the stomach lining and changes in the lining's cells.
Tell your healthcare professional if your symptoms aren't improving despite treatment for gastritis.

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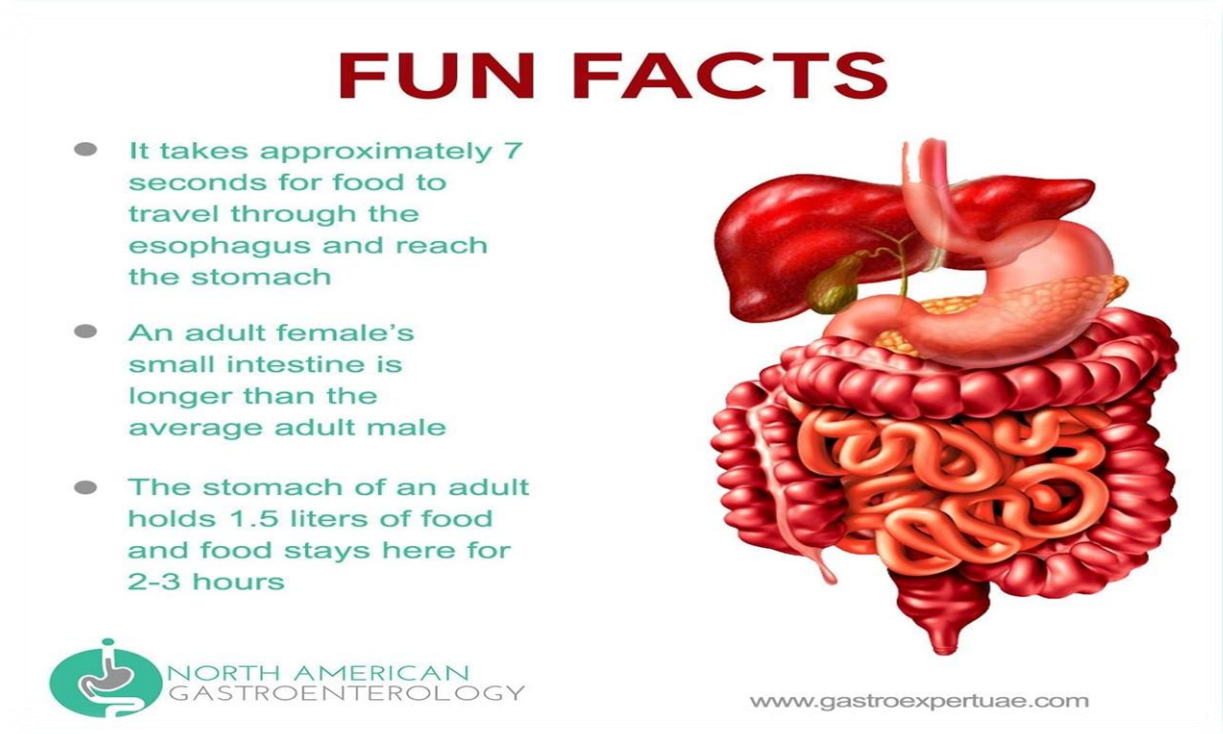

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2020-2025

Pancreatic Pseudo cyst

OVERVIEW:

A pancreatic pseudocyst is an encapsulated collection of homogenous fluid with little or no necrotic tissue located near the pancreas. It often presents with nonspecific symptoms in patients with a history of chronic pancreatitis and less commonly acute pancreatitis. It is crucial to identify any complications that may arise to minimize the associated morbidity and mortality. This activity reviews the etiology and evaluation of pancreatic pseudocysts and highlights the role of the interprofessional team in the various treatment modalities available for patients with this condition.

INTRODUCTION TO PANCREATIC PSEUDOCYST:

A true cyst is a localized fluid collection that is contained within an epithelial-lined capsule. In contrast, a pseudocyst is a fluid collection that is surrounded by a *non-epithelialized* wall made up of fibrous and granulation tissue, hence the name "pseudo" cyst. A pancreatic pseudocyst is an encapsulated collection of homogenous fluid with little or no necrotic tissue within it. It is usually well-circumscribed and located outside of the pancreas, often in the lesser sac. Pancreatic pseudocysts are often seen as a complication of chronic pancreatitis and less commonly from acute pancreatitis. They occur when the damage of the pancreatic ducts, frequently from biliary stones or alcohol, causes extravasation and collection of the pancreatic fluid. Regardless of the cause, the overall incidence of pseudocysts is low; 0.5 to 1 per 100,000 adults per year.

ETIOLOGY:

- ✓ Pseudocysts are formed when disruption of the main pancreatic duct or its branches, either from inflammation or direct injury, causes extravasation of pancreatic enzymes into the parenchyma and eventually forms a distinct collection.
- ✓ It appears that alcohol-related pancreatitis is the major cause of pancreatic pseudocyst formation in countries where alcohol consumption is higher as it contributes to over 70% of the cases.

- ✓ Having gallstones and drinking a lot of alcohol are the 2 most common causes of pancreatitis.
- ✓ Some other causes of it include:
- ✓ Pancreas injury
- ✓ Pancreas infection
- ✓ Pancreatic tumor
- ✓ High calcium levels in your blood
- ✓ Very high levels of blood fats (cholesterol)
- ✓ Pancreatic damage from medicines
- ✓ Autoimmune diseases
- ✓ Conditions that run in your family that harm the pancreas, such as cystic fibrosis

EPIDEMIOLOGY:

Pseudocysts can occur after pancreatitis in any age group. The incidence of pseudocysts is higher in males as it follows the incidence of pancreatitis, which is slightly male predominant. In acute pancreatitis, the incidence of pseudocysts ranges from 5% to 16%. Pseudocysts tend to be more common in the setting of chronic pancreatitis, with incidence rates between 20% to 40%. This can be explained by its long course posing an increased risk of damaging the pancreatic ducts with fibrosis, calculi, or protein plug formation. Data collected from over 357 admissions at Wayne University Hospital in Detroit for pancreatic pseudocyst recognized that the majority of the cases were alcohol-induced (70%) followed by biliary tract disease (8%), trauma (6%), and the remaining attributed to the idiopathic cause.

PATHOPHYSIOLOGY:

Injury to the pancreatic ducts, either from pancreatitis or direct trauma, can lead to extravasation of the pancreatic fluid. In some instances, this fluid organizes into a discrete collection, confined by walls of adjacent organs such as the stomach, pancreas, omentum, and colon, forming a pseudocyst. It can take anywhere between 4 to 6 weeks for the pseudocyst wall to mature. Up to two-thirds of pseudocysts have demonstrable connections to the pancreatic duct. In the remaining one-third of cases, an inflammatory reaction can sometimes seal off the connection making it difficult to identify. The significance of this connection will be discussed in detail under management.

CLASSIFICATION SYSTEM:

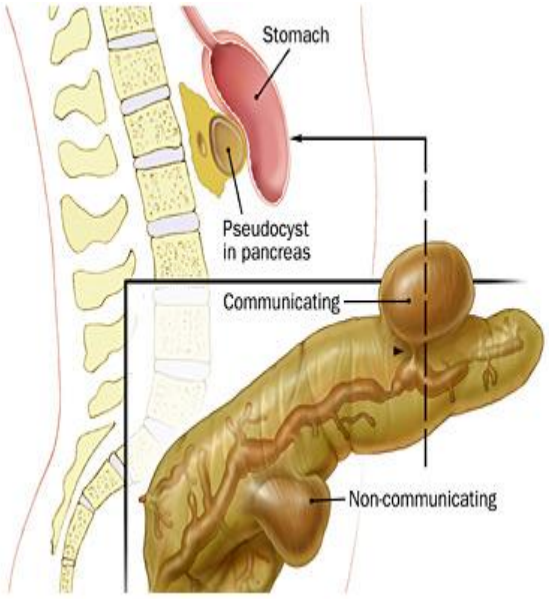
Pancreatic Pseudocysts are classified based on:

- ✓ Pancreatic Duct association with pancreatic pseudocyst

Although PPC as a term is well-established, there is no classification system widely accepted. The First classification system widely proposed by D'Edigio and Schein is based on the underlying etiology of pancreatitis (Acute or Chronic), the pancreas ductal anatomy, and the presence of communication between the cyst and pancreatic duct. Using this classification system, the cyst may be divided into three distinct types:

- ✓ **Type I, or Acute "Post-necrotic" pseudocysts**, occur after an episode of acute pancreatitis and are associated with normal duct anatomy and rarely communicate with the pancreatic duct.
- ✓ **Type II, also post-necrotic pseudocysts,** occur after an episode of acute-on-chronic pancreatitis (the pancreatic duct is diseased, but not structured, and there is often a duct-pseudocyst communication.
- ✓ **Type III, defined as "retention" pseudocysts,** occur in chronic pancreatitis and are uniformly associated with duct stricture and pseudocyst-duct communication.

Type	Description of Pancreatic Pseudocyst
I	<5 cm and without complications, symptom, and neoplasia
II	Suspected cystic neoplasia
III	The lLocation of pancreatic pseudocyst is uncinate
IIIa	Pseudocyst communication with the pancreatic duct
IIIb	Without communication between pseudocyst and pancreatic duct
IV	Location of pancreatic pseudocyst is head, neck, and body
IVa	Exist communication between pseudocyst and pancreatic duct (1)
IVb	Distance from the cyst to the gastrointestinal wall is <1 cm (2)
IVc	Neither 1 nor 2
V	Location of pancreatic pseudocyst is tail
Va	Splenic vein involvement or upper gastrointestinal bleeding
Vb	Distance from the cyst to the gastrointestinal wall is <1 cm, without splenic vein involvement or upper gastrointestinal bleeding



✓

SIGNS & SYMPTOMS:

✓ Patients with acute pancreatitis who are not treated within seven days or those whose symptoms reappear after a transient improvement period should be suspected of pancreatic pseudocyst.

Some of the signs and symptoms that are suggestive of pseudocyst are:

- Persistent abdominal pain
- Anorexia
- New abdominal mass after an episode of pancreatitis
- Jaundice or shock (less commonly)

Findings that are of limited sensitivity:

- Abdominal tenderness
- Palpable abdominal mass
- Signs of peritonitis, including guarding and rigidity (in case of a ruptured cyst)
- Fever
- Scleral icterus
- Pleural effusion

RISK FACTORS:

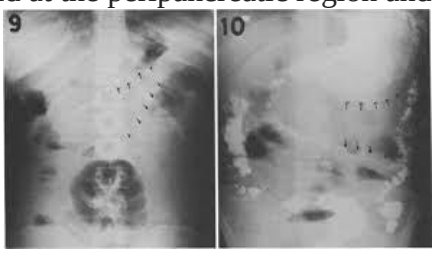
✓ You may have a higher risk for pseudocysts if you have a health issue that can cause pancreatitis:
✓ These include gallstones.
✓ You may be able to decrease your risk by treating your health condition.
✓ Drinking less alcohol may also lower your risk.

DIAGNOSIS:

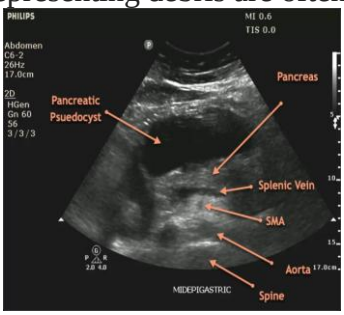
Diagnosis of Pancreatic Pseudocyst based on the following:

✓ Laboratory Methods:
✓ CBC
✓ Serum Electrolytes
✓ Liver Function Tests (LFTs)
✓ Serum Amylase/Lipase showing persistently elevated serum amylase
✓ Radiographic Features:

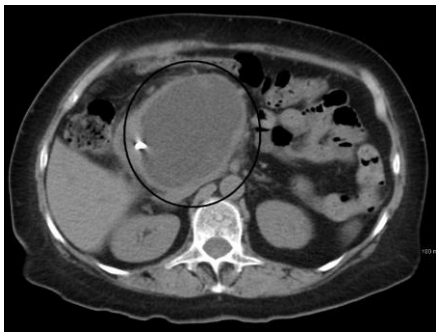
Plain Radiographs (X-rays) of the abdomen are often not sensitive in the workup for pancreatic pseudocysts. However, if the cyst is large, it may demonstrate a gastrocolic separation sign which suggests fluid at the peripancreatic region and into the lesser sac.



Ultrasonography of the abdomen showing hypoechoic or anechoic collections, with dependent low-level echoes representing debris are often seen.



Computed Tomography (CT-Scan) is the most accurate method (Sensitivity 90-100%) showing Pseudocysts appear as well-circumscribed, usually round or oval peripancreatic fluid collections of homogeneously low attenuation, that are usually surrounded by a well-defined enhancing wall. Calcification of the wall of a pseudocyst is rare and an alternative diagnosis should always be considered.



TREATMENT:

Initial treatment for pancreatic pseudocysts is:

- ✓ Patient is made NPO (Nill-Patient Oral) to rest the pancreas
- ✓ Intravenous narcotic analgesics to control abdominal pain
- ✓ Supplemental Oxygen (2L) via nasal cannula
- ✓ Intravenous fluids of lactated Ringer's or normal saline are initially bolused at 15-20 mL/kg (1050-1400mL), followed by 2-3mL/kg per hour (200-250mL/h) to maintain the urine output >0.5mL/kg per hour.
- ✓ Octreotide can be useful as an adjunct to catheter drainage.

Indications for the drainage of pancreatic pseudocysts are:

- ✓ Pseudocysts that are large and thick-walled (> 6cm)
- ✓ Pseudocysts that have lasted from > 6 weeks.

The following are the methods for draining pancreatic pseudocysts:

- ✓ Percutaneous drainage (CT-guided or Ultrasound-guided)
- ✓ Endoscopic Drainage
- ✓ Cystogastrostomy
- ✓ ERCP guided Trans-papillary drainage
- ✓ EUS (Endoscopic Ultrasound) guided Cystogastrostomy
- ✓ Surgical drainage

COMPLICATIONS:

The goal is to avoid complications which include:

- ✓ Infection
- ✓ Rupture into the gastrointestinal tract causing signs and symptoms of bleeding
- ✓ Free rupture into the peritoneal cavity causing abdominal pain, peritonitis, or even death
- ✓ There have been case reports of large pseudocysts causing increased intra-abdominal pressure, which can present with orthopnea, dyspnea, abdominal pain, distension, and new organ failure
- ✓ Pancreatic Pseudo-aneurysm
- ✓ Compression of the surrounding structures causing biliary complications or portal hypertension

PROGNOSIS:

Spontaneous resolution of pseudocyst is common, especially for those that occur after an episode of acute pancreatitis. The maturation of pancreatic pseudocysts takes approximately 2 to 6 weeks, and during this time, 33% of cysts resolve spontaneously.

PREVENTION:

The best way to avoid pseudocysts is to avoid pancreatitis, which is usually caused by gallstones or heavy alcohol use. If gallstones are triggering pancreatitis, you may need to have your gallbladder removed. If your pancreatitis is due to alcohol use, not drinking can reduce your risk.

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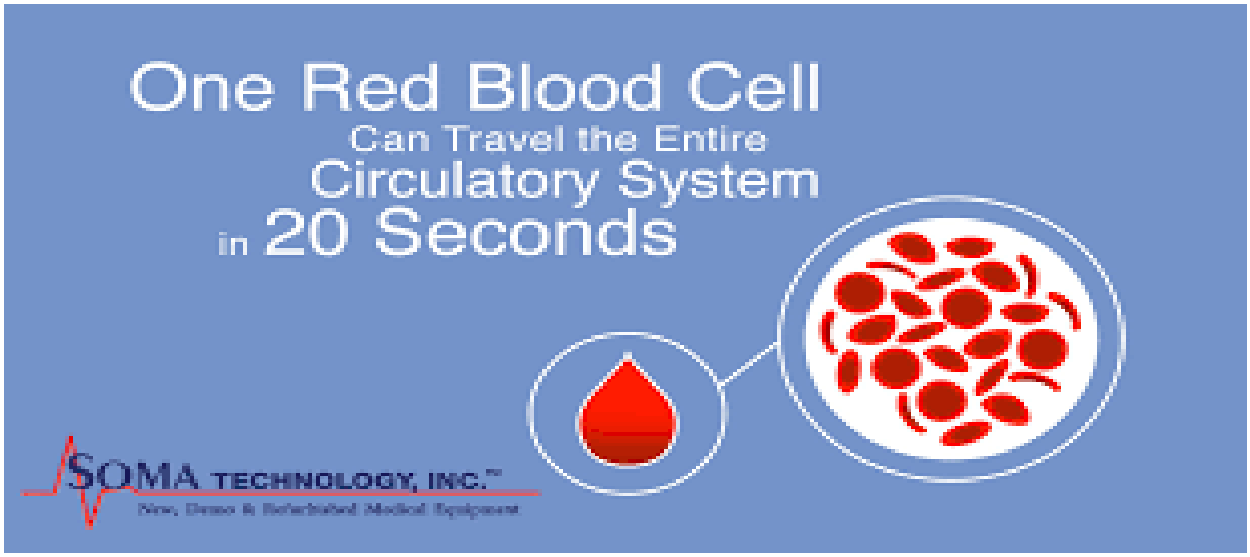

SHARUHITHA DONEPALLI
5th Semester 3rd Year- MBBS

Irritable Bowel Syndrome

DEFINATION

Irritable bowel syndrome (IBS) is a group of symptoms that occur together, including repeated pain in your abdomen and changes in your bowel movements, which may be diarrhoea, constipation, or both. With IBS, you have these symptoms without any visible signs of damage or disease in your digestive tract.

EPIDEMOLOGY

The number of newly diagnosed cases of irritable bowel syndrome (IBS) is increasing worldwide. International IBS prevalence is estimated at 11.2% with variations in geographic regions ranging from 7% to 21% [1,2]. IBS is more prevalent among women with the incidence being 1.5 to 3 fold higher than in men. Mortality During follow-up, there were 3,290 deaths in individuals with IBS (9.4/1,000 person-years) compared with 13,255 deaths in reference individuals (7.9/1,000 person-years), resulting in an HR of 1.10 (95% confidence interval [CI] = 1.05

ETIOLOGY

The Etiology of IBS is broad and not clearly understood. However motility, visceral sensation, brain-gut interaction, and psychosocial distress can all play a role in the development of IBS. It can occur after a bacterial infection or a parasitic infection (giardiasis) of the intestines.

RISK FACTOR

1. Are young. IBS occurs more frequently in people under age 50.
2. Are female, IBS is more common among women. Estrogen therapy before or after menopause also is a risk factor for IBS.
3. Have a family history of IBS. Genes may play a role, as may shared factors.
4. Have anxiety, depression or other mental health issues.
5. smoking and alcohol consumption.

PATHOGENESIS

The pathogenesis of irritable bowel syndrome (IBS) is quite complex, IBS is thought to be caused by a combination of factors, including abnormalities in the gut-brain axis, changes in gut motility, and alterations in the gut microbiome. These factors can lead to increased sensitivity in the intestines. Underlying mechanisms that could lead to irritable bowel syndrome include genetic factors (most notably an identified mutation of SCN5A); post-infectious changes, chronic infections and disturbances in the intestinal microbiota; low-grade mucosal inflammation, immune activation, and altered intestinal permeability.

DIAGNOSIS

Blood test : blood tests to check for conditions other than IBS, including anemia, infection, and digestive diseases.

Stool test : stool tests to check for blood in your stool or other signs of infections or diseases. stool study is also can check to see intenstine has trouble taking in nutrients(malabsorption)

Hydrogen breath test : To check for small intestinal bacterial overgrowth or problems digesting certain carbohydrates, such as lactose intolerance. if lactose intolerance tests are positive they may have problems similar to those caused by IBS.

Upper GI endoscopy : A biopsy to check for celiac diseases.

CT Scan : produces images of abdomen and pelvis that might rule out other causes of symptoms, especially during belly pain.

Colonoscopy : Can help your provider determine if you have certain bowel disorders that may be causing your symptoms, including polyps, IBD and cancerous growths. For this procedure, a provider inserts a scope that allows them to view your entire colon

TREATMENT

Counselling/stress Management/Diet
Physical activity
Laxatives (IBS-C)

- ✓ Osmotic laxatives (PEG)
- ✓ CI-channel activator (lubiprostone)
- ✓ Guanylate cyclase agonist (linaclotide)

Antidiarrheals (IBS-C)

- ✓ Loperamide
- ✓ Bile acid sequestrants (eg.cholestyramine)
- ✓ 5-HT antagonists (alosetron)

Antibiotics (IBS-D)

- ✓ Rifaximin

Abdominal Pain

- ✓ Antispasmodics
- ✓ Tricyclic antidepressants (low dose)
- ✓ SSRIs
- ✓

COMPLICATIONS

The complications of IBS can affect not only physically but also mentally and emotionally.

Chronic constipation or diarrhea can cause hemorrhoids.

Mood disorder Anxiety, depression.

Vitamin and minerals deficiencies due to intolerance to certain foods.

Poor quality of life people with moderate to severe IBS miss there time as many days from work as do those without bowel symptoms.

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SWETHA DURAIRAJ,
5th SEM,3rd YEAR

DUODENAL ULCER

DEFINITION:A duodenal ulcer is a sore that develops on the lining of the duodenum,which is the first part of the small intestine. It is typically caused by stomach acid damaging the lining of the duodenum. Symptoms can include abdominal pain, bloating, and nausea.

EPIDEMIOLOGY: Duodenal ulcers are relatively common worldwide. In some regions, such as parts of Asia and Africa,the prevalence may be higher compared to Western countries,particularly in developing countries, may have higher rates of duodenal ulcers due to factors such as dietary habits, prevalence of Helicobacter pylori infection, and access to healthcare. Duodenal ulcers are more common in men than in women, and they often occur between the ages of 30 and 60. However, the exact age and gender distribution can vary between populations. The prevalence of duodenal ulcers has been decreasing in many parts of the world,possibly due to improved hygiene, decreased prevalence of Helicobacter pylori infection qand better management of risk factors**.**

ETIOLOGY: The two primary causes for duodenal ulcers are a history of recurrent or heavy NSAID use and a diagnosis of H. pylori.The majority of patients carry a secondary diagnosis of H. pylori; however, as infection rates have declined, other previously uncommon etiologies are becoming more prevalent. Other causes of duodenal ulcers include etiologies that, in similar ways to NSAIDs and H. pylori, disrupt the lining of the duodenum. Some of these include Zollinger-Ellison syndrome, malignancy, vascular insufficiency, and history of chemotherapy.

RISK FACTOR:

- ✓ Helicobacter pylori infection
- ✓ Chronic obstructive pulmonary disease
- ✓ llicit drugs, e.g., cocaine, that reduce mucosal blood flow
- ✓ NSAIDs (potentiated by corticosteroids)
- ✓ Alcoholic cirrhosis ,smoking
- ✓ Psychological stress (can increase gastric acid secretion)
- ✓ Zollinger-Ellison syndrome
- ✓ Viral infection (CMV, herpes simplex virus)

PATHOGENESIS: Due to etiological factors, erosion occurs which is caused by the increased concentration or activity of acid pepsin or by decreased resistance of mucosa. A damage in mucosa cannot secretes enough mucus to act as a barrier against HCl.
Patient with duodenal ulcer disease secretes more acid then normal it leads to damage of gastroduodenal mucosa allows for decreased resistance to bacteria.Thus,infection occurs from h.pylori bacteria.

DIAGNOSIS: Diagnosis of H. pylori based on a history of presenting illness and physical exam findings is a possibility, studies are necessary to establish a definite diagnosis and underlying etiology further.Computed tomography performed for the evaluation of abdominal pain can identify non-perforated ulcers.

The majority of patients will need a referral for esophagogastroduodenoscopy (EGD) for further evaluation. Duodenal ulcers occur most frequently in the first portion of the duodenum (over 95%), with approximately 90% located within 3 cm of the pylorus and are usually less than or equal to 1 cm in diameter. Barium endoscopy is an option for patients with contraindications to EGD.

Lab test(For H.pylori infection):Serological test,Stool antigen test,Urea breath test.

TREATMENT:

- ✓ Antibiotics
- ✓ H2- blockers(Histamine Receptor blockers)
- ✓ Proton pump inhibitors or PPIs
- ✓ Mucosal protective agents
- ✓ Antacids
- ✓ Lifestyle changes :Quit smoking,Limited alcohol and caffeine.

COMPLICATIONS: The three main complications associated with duodenal ulcers are bleeding, perforation, and obstruction.

For patients who present with bleeding, the majority are manageable via endoscopic intervention. However, a minority of patients will require surgical intervention.

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Robbins & Cotran pathology textbook (10th edition)
https://www.healthdirect.gov.au

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5th SEMESTER, 3rd YEAR.

INFLAMMATORY BOWEL DISEASE

DEFINITION: Inflammatory Bowel Disease (IBD) is characterized by repetitive episodes of inflammation of gastrointestinal tract caused by an abnormal immune response to gut microflora which encompasses Crohn's disease and ulcerative colitis.

EPIDEMIOLOGY: The North American incidence of IBD ranges from 2.2 to 19.2 cases per 1,00,000 person-years for ulcerative colitis and 3.1 to 20.2 cases per 2,00,000 person-years for Crohn's disease.

IBD is much more prevalent in North America and Europe than in Asia or Africa. Although most IBD occurs in people aged 15-30 years, up to 25% of patients will develop IBD by adolescence. CD is slightly more common in females than males, UC appears to be equally present in both genders. The pooled standardized mortality was nearly 1.39.

ETIOLOGY: The etiology of IBD is idiopathic; but is likely to be multifactorial.

It can also be due to: Genetic predisposition, Environmental triggers like bacterial/viral infections, NSAIDS, Smoking, Mucosal immune system damage or Immunoregulatory defect, Antibiotic exposure, dietary factors, stress.

RISK FACTORS:

1. Age – Most people develop IBD before they are 30 years old.
2. Race or Ethnicity
3. Family history
4. Smoking, alcohol consumption, Excessive intake of sugars
5. Nonsteroidal Anti-Inflammatory Drugs
6. Oral Contraceptive Pills
7. Parenteral contraception and Appendectomy.

PATHOGENESIS: ~~The intestinal immune system is the key to the pathogenesis of IBD.~~

In IBD, intercellular junctions are defective due to primary barrier function failure, so that dietary and bacterial antigens penetrate into the intestinal wall and activates the immune system. This causes increased production of pro-inflammatory mediators which will lead to excessive inflammation of the mucosal layer.Excessive inflammatory reactions lead to continued deterioration of the epithelium and further exposure to intestinal microbes, thereby further worsening the inflammation

In Ulcerative colitis, there is always mucosal inflammation, whereas in Crohn's disease there are skip lesions which can induce strictures, or development of fistulas.

CLINICAL MANIFESTATIONS: Diarrhea, Abdominal pain, Bloating due to bowel obstruction, Hematochezia, Rectal bleeding, Weight loss, Fatigue, Arthritis.

DIAGNOSIS:

1.CBC- will identify anemia, leukocytosis, thrombocytosis and albumin levels.

2.Fecal calprotectin levels- can be used as marker for intestinal inflammation.

3. Stool studies- done to rule out ova and parasitic organisms.

4. Abdominal X-ray – can assess for presence of free air, bowel obstruction, toxic megacolon.

5.Barium studies- are done to characterize bowel disease; a lead pipe appearance can be seen which indicates ulcerative colitis.

6. US, CT, MRI can be used in diagnosis of IBD to assess for complications.

7.Colonoscopy- to determine the pattern of colonic and terminal ileum inflammation.

TREATMENT: Treatment is divided based on the severity of the disease: Mild, Moderate, Severe.

MILD: Antibiotics like Metronidazole, and drugs like Sulfasalazine and Mesalamine can be given which has anti-inflammatory effects.

MODERATE: Corticosteroids like Prednisolone are effective in decreasing disease activity and inducing remission in most patients. Immunomodulators such as Methotrexate and TNF inhibitors like Infliximab are preferred.

SEVERE: Surgeries can be done like: Proctocolectomy, Resection, Colectomy, etc.,

COMPLICATIONS: The complications of IBD are divided into two categories, Intestinal and Extraintestinal.

INTESTINAL: Hemorrhage, Strictures, Anal fistulas, Toxic megacolon, Colon cancer, Perirectal abscesses.

EXTRAINTESTINAL: Osteoporosis, Deep Vein Thrombosis, Anemia, Primary sclerosing arthritis, Cholangitis, Gall stones,

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BARRETT'S ESOPHAGUS

INTRODUCTION :-

Barrett's oesophagus is a condition in which the normal stratified squamous epithelium (pink lining or mucosal lining) of the oesophagus (food/swallowing pipe that connects mouth and stomach) is damaged by acid reflux and thickens and reddens, as detected on endoscopic examination and pathologically confirmed by the presence of intestinal metaplasia columnar epithelium (the presence of thick cells similar to those present in intestine).

EPIDEMIOLOGY :-

Barrett's esophagus is usually discovered during endoscopic examinations of middle-aged and older adults; the large majority of cases go unrecognized .

In the United States it is estimated that as many as 5.6 percent of adults have Barrett's esophagus. However, estimates of the prevalence of Barrett's esophagus in the general population have varied widely ranging from 0.4 to more than 20 percent depending, in part, upon the population studied and the criteria used to establish the diagnosis .

In studies performed in the United States, Barrett's esophagus appears to have a higher prevalence in White individuals as compared with individuals who identify as being of Hispanic descent or Asian descent, and prevalence appears to be lowest in Black individuals. The prevalence of short-segment Barrett's esophagus is substantially higher than long-segment Barrett's esophagus .

ETIOLOGY:-

The exact cause of Barrett's esophagus isn't known. While many people with Barrett's esophagus have long-standing GERD, many have no reflux symptoms, a condition often called "silent reflux."

Whether this acid reflux is accompanied by GERD symptoms or not, stomach acid and chemicals wash back into the esophagus, damaging esophagus tissue and triggering changes to the lining of the swallowing tube, causing Barrett's esophagus.

RISK FACTOR:-

Common risk factors in the development of Barrett's esophagus include:

- ✓ Family history. Your odds of having Barrett's esophagus increase if you have a family history of Barrett's esophagus or esophageal cancer.
- ✓ Being male. Men are far more likely to develop Barrett's esophagus.
- ✓ Being white. White people have a greater risk of the disease than do people of other races.

- ✓ Age. Barrett's esophagus can occur at any age but is more common in adults over 50.
- ✓ Chronic heartburn and acid reflux. Having GERD that doesn't get better when taking medications known as proton pump inhibitors or having GERD that requires regular medication can increase the risk of Barrett's esophagus.

SIGNS AND SYMPTOMS:-

Barrett's oesophagus is asymptomatic, but it can manifest symptoms of long-term GERD, such as heartburn and acid regurgitation. Along with this, the symptoms might also include:

- ✓ Difficulty in swallowing food
- ✓ Difficult to sleep due to heartburn.
- ✓ Chest pain
- ✓ Constant sore throat
- ✓ Unintentional weight loss
- ✓ Blood in stool
- ✓ Vomiting

COMPLICATIONS :-

- ✓ Barrett's oesophagus complication includes oesophageal cancer (rare). However, the probability of having oesophageal cancer is statistically minimal (one in every 860 Barrett's esophagus patients). In some patients, Barrett's oesophagus might be associated with complications such as stricture, ulcer, and dysplasia.

PATHOGENESIS OF BARRETT'S ESOPHAGUS:-

- ✓ GERD is a condition, where the acid and bile from the stomach come back up through the lower oesophageal sphincter (LES), a critically important valve, present between the oesophagus and the stomach that prevents the reflux of gastric contents back into the oesophagus. The lining of the stomach is made of mucinous columnar epithelium, which can handle the acidic environment needed for digestion, whereas the lining of the oesophagus, on the other hand, is made of squamous epithelium.
- ✓ The LES may eventually fail, allowing stomach acids to reach the lower part of the oesophagus. The acidic irritant causes the squamous epithelium (oesophageal lining) to become red and swollen. When oesophageal lining exposed to gastric acids for a long time, there is persistent inflammation (due to release of inflammatory cytokines such as interleukin-8, interleukin-6) and a columnar metaplasia reaction occurs, commonly known as Barrett's oesophageal metaplasia. Eventually, goblet cells develop, which is a sign of an intestinal-type phenotype.

DIAGNOSIS:-

- ✓ Barrett's oesophagus diagnosis includes a biopsy and upper gastrointestinal (UGI) endoscopy. While investigating the root of a patient's GERD symptoms, gastroenterologist may make the diagnosis of Barrett's oesophagus

TREATMENT:-

Depending on the patient's health status and severity of dysplasia, the gastroenterologist will discuss the best course of treatment, which includes:

- ● Endoscopic ablative therapies
- ● Photodynamic therapy
- ● Radiofrequency ablation therapy
- ● Cryotherapy
- ● Endoscopic mucosal resection
- ● Surgery
- ● Esophagectomy
- ● Fundoplication (prophylactic)
- ● GERD medications

CONCLUSION:-

People with Barrett's esophagus have an increased risk of esophageal cancer. The risk is small, even in people who have precancerous changes in their esophagus cells. Fortunately, most people with Barrett's esophagus will never develop esophageal cancer.

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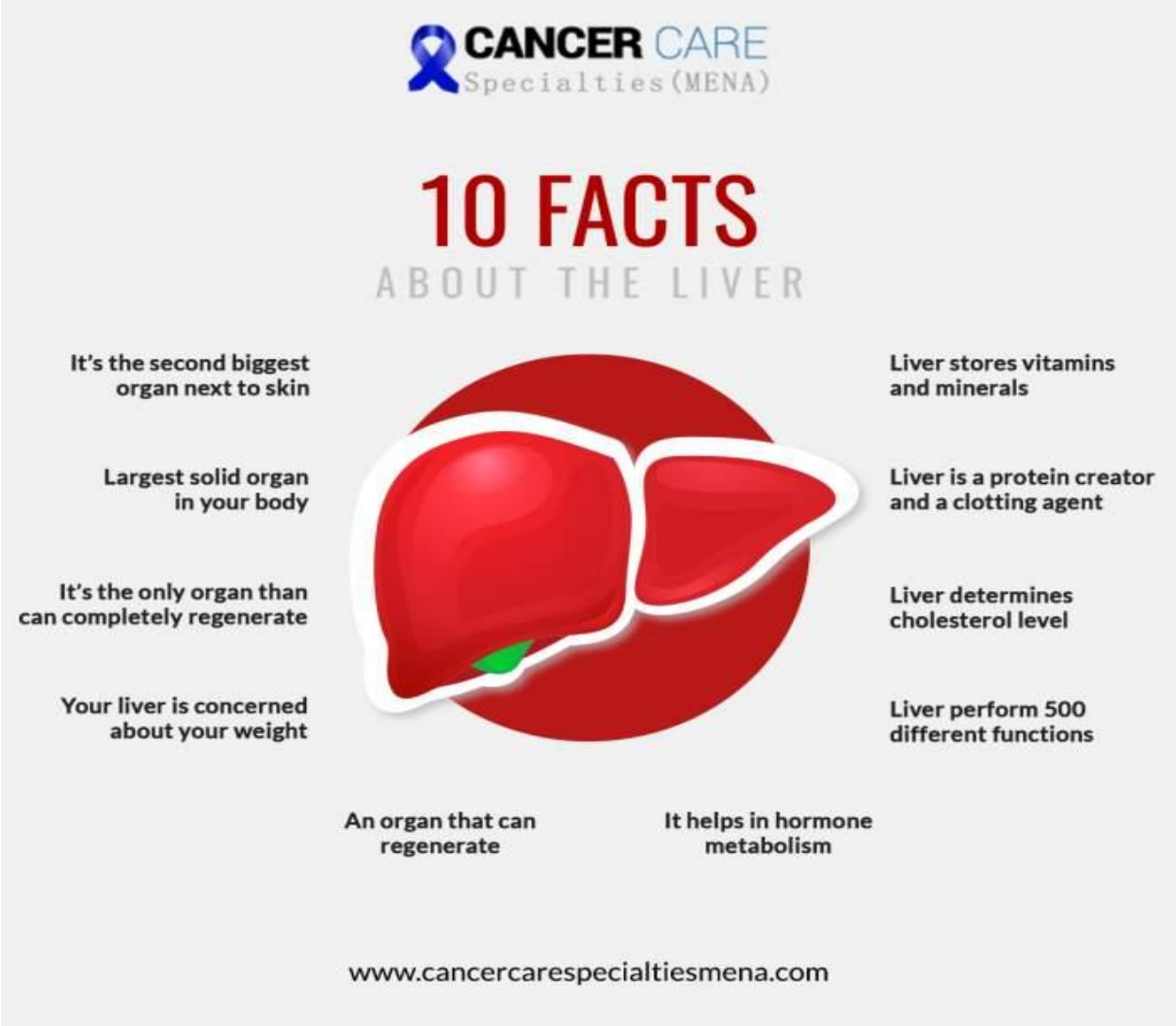

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UPPER GASTROINTESTINAL BLEEDING

INTRODUCTION :-

Upper gastrointestinal (GI) bleeding refers to bleeding that occurs anywhere in the esophagus, the stomach, or the upper part of the small intestine. It is a symptom of an underlying disorder, and it can be serious. The manifestations depend on the location and rate of bleeding.Hematemesis is vomiting of red blood and indicates upper GI bleeding, usually from a peptic ulcer, vascular lesions or varix. Coffee-ground emesis is vomiting of dark brown, granular material that resembles coffee grounds. It results from upper GI bleeding that has slowed or stopped, with conversion of red hemoglobin to brown hematin by gastric acid. Melena is black, tarry stool and typically indicates upper GI bleeding, but bleeding from a source in the small bowel or right colon may also be the cause. About 100 to 200 mL of blood in the upper GI tract is required to cause melena, which may persist for several days after bleeding has ceased.

EPIDEMIOLOGY :-

Upper Gastrointestinal Bleeding is described as blood loss from a gastrointestinal source above the ligament of Treitz. UGIB accounts for 75% of all acute gastrointestinal (GI) bleeding cases. Its annual incidence is approximately 80 to 150 per 100,000 population. Patients on long-term, low-dose aspirin have a higher risk of overt UGIB compared to placebo. When aspirin is combined with P2Y12 inhibitors such as clopidogrel, there is a two-fold to three-fold increase in the number of UGIB cases. When a patient requires triple therapy (i.e., aspirin, P2Y12 inhibitor and vitamin K antagonist), the risk of UGIB is even higher. Upper gastrointestinal bleeding (UGIB) is a common problem that is estimated to occur in 80 to 150 out of 100,000 people each year. Estimated mortality rates are between 2 and 15 percent.

ETIOLOGY:-

Upper GI Bleeding

- Peptic ulcer disease (can be secondary to excess gastric acid, H. pylori infection, NSAID overuse, or physiologic stress)
- Esophagitis
- Gastritis and Duodenitis
- Varices
- Portal hypertensive gastropathy (PHG)
- Angiodysplasia
- Dieulafoy lesion (bleeding dilated vessel that erodes through the gastrointestinal epithelium but has no primary ulceration; can be at any location along the GI tract.
- Gastric antral valvular ectasia (GAVE; also known as watermelon stomach)
- Mallory-Weiss tears
- Cameron lesions (bleeding ulcers occurring at the site of a hiatal hernia.

- Aortoenteric fistulas
- Foreign body ingestion
- Post-surgical bleeds (post-anastomotic bleeding, post-polypectomy bleeding, post-sphincterotomy bleeding)
- Upper GI tumors
- Hemobilia (bleeding from the biliary tract)
- Hemosuccus pancreaticus (bleeding from the pancreatic duct)

RISK FACTOR:-

Common risk factors in the development of upper gastrointestinal bleeding include:

- Advancing age (Age 60 years or older)
- Previous history of gastrointestinal bleed
- Chronic kidney disease
- Underlying cardiovascular disease
- Cirrhosis and portal hypertension
- Presence of H.pylori infection
- NSAID's or aspirin use in patients with a history of ulcer disease
- Patients on dual antiplatelet therapy
- Patients taking glucocorticoids
- Dyspepsia
- Gastroesophageal reflux disease.

SIGNS AND SYMPTOMS:-

Symptoms of GI bleeding can be easy to see, called overt, or not so obvious, known as occult.

- Haematemesis : vomiting of bloodwhether fresh and red or digested and black.
- Melana: passage of loose, black tarry stools with a characteristic foul smell.
- Coffee ground vomiting : blood clot in the vomitus.
- Hematochezia: passage of bright red blood per rectum (if the haemorrhage is severe).

Overt bleeding might show up as:

- Vomiting blood, which might be red or might be dark brown and look like coffee grounds.
- Black, tarry stool.
- Rectal bleeding, usually in or with stool.

With occult bleeding, you might have:

- Lightheadedness.
- Difficulty breathing.
- Fainting.
- Chest pain.
- Abdominal pain.

COMPLICATIONS :-

Upper Gastrointestinal bleeding if not managed timely or properly can lead to serious consequences. Following complications can occur in a patient with upper gastrointestinal bleeding:

- Respiratory Distress
- Myocardial Infarction
- Infection
- Shock
- Death

PATHOGENESIS OF UPPER GI BLEEDING:-

•The pathophysiology of gastrointestinal (GI) bleeding involves disrupting the blood arteries that supply the GI tract, resulting in bleeding.
Conditions that are associated with the pathophysiology of GI bleeds are discussed below:

- **Peptic Ulcer**

The majority of GI bleeds are caused by stomach and duodenal ulcers. Persons with peptic ulcers exhibit bleeding in the upper gastrointestinal tract as their primary symptom. Duodenal ulcers are four times as likely than stomach ulcers to cause bleeding. The proximity of posterior duodenal ulcers to GDA branches makes them more likely to hemorrhage than other duodenal ulcers.

- **Stress Ulcers**

Stress ulcers can result in multisystem trauma, hypotension, respiratory failure, sepsis, and jaundice. It may be caused by bile reflux, which damages the stomach's protective barrier, or by splanchnic vasoconstriction, which restricts blood supply to the liver. Acute gastroduodenal lesions may result from a shock, an infection, surgery, trauma, burns, or a brain condition that leads to GI bleeding.

- **Erosive Gastritis**

One-third of all upper GI bleeding is caused by diffuse gastritis. The condition is characterized by several erosions, with the majority occurring in the fundus and body of the stomach. NSAIDs, alcohol, and steroids increase the likelihood of bruising since they are detrimental to the stomach lining. H. pylori is also associated with slow, protracted bleeding.

- **Esophageal and Gastric Varices**

Varices are enlarged veins in the submucosa caused by increased pressure in the portal vein. Varix ulceration, which can be brought on by reflux esophagitis or increased pressure within varix, is the initial stage in the path to variceal bleeding.

- **Dieulafoy's Vascular Malformations**

Dieulafoy's lesions are large, intertwining blood arterioles in the submucosa of the stomach. Most lesions occur in the fundus and body of the stomach, along the stomach's slight curve. Since there is a hole in the gastric mucosa, Dieulafoy's lesions induce bleeding. This hole results from pressure exerted by the bulging and pulsing arteriole.

- **Gastric Neoplasms**

Both malignant and benign cancers of the upper gastrointestinal tract can produce bleeding. Neoplasms are known to induce light and consistent bleeding, and patients frequently exhibit symptoms of anemia.

- **Aortoenteric Fistulas**

Aortoenteric fistulas occur when a prosthetic graft in a patient who has had aortic repair degrades into the intestine due to an infection surrounding the graft.

DIAGNOSIS:-

- **Blood tests.** You may need a complete blood count, a test to see how fast your blood clots, a platelet count and liver function tests.
- **Stool tests.** Analyzing your stool can help determine the cause of occult bleeding.
- **Nasogastric lavage.** A tube is passed through your nose into the stomach to remove stomach contents. This might help find the source of the bleeding.

- **Upper endoscopy.** An upper endoscopy is a procedure that uses a camera to view the upper digestive system. The camera is attached to a long, thin tube, called an endoscope, and passed down the throat to examine the upper gastrointestinal tract.
- **Capsule endoscopy.** In this procedure, you swallow a vitamin-size capsule with a tiny camera inside. The capsule travels through your digestive tract taking thousands of pictures that are sent to a recorder you wear on a belt around your waist.
- **Angiography.** A contrast dye is injected into an artery, and a series of X-rays are taken to look for and treat bleeding vessels or other issues.
- **Imaging tests.** A variety of other imaging tests, such as a CTscan of the belly, might be used to find the source of the bleed.

TREATMENT:-

GI bleeding often stops on its own. If it doesn't, treatment depends on where the bleed is from. In many cases, bleeding can be treated with medicine or a procedure during a test. For example, it's sometimes possible to treat a bleeding peptic ulcer during an upper endoscopy or to remove polyps during a colonoscopy. If you have an upper GI bleed, you will be given an IV drug known as a proton pump inhibitor (PPI) to suppress stomach acid production. Once the source of the bleeding is identified, your doctor will determine whether you need to continue taking a PPI.Depending on the amount of blood loss and whether you continue to bleed, you might need fluids through a needle (IV) and, possibly, blood transfusions. If you take blood-thinning medicines, including aspirin or nonsteroidal anti-inflammatory medications, you might need to stop.

CONCLUSION:-

Bleeding in the upper gastrointestinal tract is a medical emergency that, if not treated quickly, might be fatal. The blood arteries supplying the esophagus, stomach, and duodenum are damaged in the pathophysiology of upper GI bleeding, which results in hemorrhage. A multidisciplinary strategy is used to treat upper GI bleeding, including resuscitation, locating the cause, and administering the proper medications. For identifying and treating upper GI bleeding, endoscopy is frequently the first line of treatment; however, in more serious situations, angiography, embolization, or surgery may be necessary.

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DYSPEPSIA

INTRODUCTION:-

Dyspepsia is a medical term for indigestion or discomfort in the upper abdomen, often characterized by symptoms like bloating, nausea, burping, and heartburn. It's a common condition and can be caused by various factors such as overeating, spicy or fatty foods, stress, or underlying digestive disorders.

EPIDEMIOLOGY:-

The epidemiology of dyspepsia indicates that it's a prevalent condition worldwide, affecting people of all ages. Its exact prevalence varies depending on factors like geographic location, age group, and diagnostic criteria. Studies suggest that anywhere from 20% to 40% of the general population may experience dyspepsia at some point in their lives. It's more common in adults and tends to be slightly more prevalent in women than in men. Additionally, the prevalence of dyspepsia tends to increase with age.

CLASSIFICATION OF DYSPEPSIA:-

Dyspepsia can be categorized into two main types:

1. Functional Dyspepsia: This type of dyspepsia occurs when there's no identifiable cause or underlying structural abnormality that can explain the symptoms. It's also known as non-ulcer dyspepsia and is characterized by recurrent or persistent symptoms of indigestion.

2. Organic Dyspepsia: Organic dyspepsia is associated with identifiable causes or underlying conditions such as gastroesophageal reflux disease (GERD), peptic ulcers, gastritis, gallbladder disease, or certain medications. Identifying and treating the underlying cause is crucial in managing this type of dyspepsia.

PATHOGENESIS:-

The pathogenesis of dyspepsia is multifactorial and not fully understood. However, several mechanisms are believed to contribute to the development of symptoms:

1. Delayed Gastric Emptying: Dysfunction in the emptying of the stomach contents into the small intestine can lead to feelings of fullness and bloating, contributing to dyspeptic symptoms.

2. Gastric Acid and Mucosal Integrity: Excessive production of gastric acid or disruption of the protective mucosal barrier lining the stomach can result in inflammation and irritation, leading to symptoms such as heartburn and upper abdominal discomfort.

3. Helicobacter pylori Infection: This bacterium can infect the stomach lining, leading to inflammation (gastritis) and possibly contributing to dyspeptic symptoms, especially in individuals with peptic ulcers.

4. Visceral Hypersensitivity: Some individuals with dyspepsia may have heightened sensitivity to normal gastric stimuli, leading to exaggerated symptoms even with minor changes in gastric function.
5. Psychological Factors: Stress, anxiety, and depression can exacerbate dyspeptic symptoms through the brain-gut axis, influencing gastric motility and perception of discomfort.
6. Dietary Factors: Consumption of certain foods, such as spicy, fatty, or acidic foods, can exacerbate symptoms by irritating the stomach lining or increasing gastric acid production.
7. Medications: Certain medications, particularly NSAIDs, aspirin, and some antibiotics, can irritate the stomach lining and contribute to dyspeptic symptoms.
8. Gastroesophageal Reflux: Acid reflux from the stomach into the esophagus (GERD) can manifest as dyspeptic symptoms such as heartburn and regurgitation.

RISK FACTOR:-

Several factors can increase the risk of developing dyspepsia:

1. Dietary Factors
2. Lifestyle Factors
3. Medications
4. Gastrointestinal Disorders
5. Psychological Factors
6. Age and Gender
7. Obesity
8. Helicobacter pylori Infection

SIGNS AND SYMPTOMS:-

Signs and symptoms of dyspepsia can vary from person to person, but commonly include:
1. Upper Abdominal Discomfort: This is the hallmark symptom, often described as a burning, gnawing, or dull ache in the upper abdomen.
2. Bloating: Feeling of fullness or abdominal bloating, especially after eating.
3. Nausea: Feeling queasy or having an urge to vomit.
4. Belching: Excessive burping or belching, often with a sour or acidic taste.
5. Heartburn: Burning sensation or discomfort in the chest, especially after eating or lying down.
6. Regurgitation: Backflow of stomach contents into the throat or mouth, often accompanied by a sour taste.
7. Early Satiety: Feeling full soon after starting a meal, even if only a small amount of food is consumed.
8. Loss of Appetite: Decreased desire to eat, often due to discomfort or feeling full.
9. Fatigue: Feeling tired or lacking energy, which may be related to disrupted sleep patterns or chronic discomfort.
10. Unintentional Weight Loss: Some individuals with severe or chronic dyspepsia may experience weight loss over time due to decreased appetite or difficulty eating.

COMPLICATIONS:-

While dyspepsia itself is not typically associated with serious complications, it can lead to various issues if left untreated or if underlying conditions are not addressed. Some potential complications include:

1. Impaired Quality of Life
2. Malnutrition
3. Esophageal Complications
4. Peptic Ulcers
5. Psychological Effects
6. Medication Side Effects
7. Reduced Work Productivity

DIAGNOSIS:-

The diagnosis of dyspepsia typically involves a combination of clinical evaluation, medical history, physical examination, and sometimes additional tests. Here's an overview of the diagnostic process:

1. Medical History

2. Physical Examination

3. Diagnostic Tests:

- Laboratory Tests: Blood tests may be ordered to check for signs of infection (such as Helicobacter pylori), inflammation, or other abnormalities.

- Endoscopy: In some cases, an upper gastrointestinal endoscopy may be recommended to visually inspect the esophagus, stomach, and duodenum for signs of inflammation, ulcers, or other structural abnormalities.

- Imaging Studies: Imaging tests such as abdominal ultrasound or CT scan may be performed to evaluate the gallbladder, pancreas, or other organs if indicated by the clinical presentation.

- Functional Tests: Tests to assess gastric emptying, such as gastric emptying scintigraphy or breath tests, may be ordered in cases where delayed gastric emptying is suspected.

4. Trial of Therapy

TREATMENTS:-

The treatment of dyspepsia depends on the underlying cause and specific symptoms experienced by the individual. Here are some general approaches to managing dyspepsia.

1. Lifestyle Modifications:

- Avoiding trigger foods
- Eating smaller, more frequent meals
- Avoiding lying down or going to bed immediately after eating
- Managing stress

2. Dietary Changes:

- Increasing fiber intake
- Avoiding excessive caffeine and alcohol

3. Medications:

- Antacids
- Acid-suppressing medications: Proton pump inhibitors (PPIs) or H2-receptor antagonists (H2 blockers)
- Prokinetic agents
- Antibiotics

4. Psychological Interventions

CONCLUSIONS:-

In conclusion, dyspepsia is a common gastrointestinal condition characterized by symptoms such as upper abdominal discomfort, bloating, nausea, and heartburn. It can be classified into functional dyspepsia, where no identifiable cause is found, and organic dyspepsia, which is associated with underlying conditions such as GERD, peptic ulcers, or gastritis. While dyspepsia itself does not typically lead to serious complications, it can significantly impact quality of life if left untreated. The diagnosis of dyspepsia involves a thorough clinical evaluation, medical history, physical examination, and sometimes additional tests such as endoscopy or imaging studies.

Reference:-

By Robin's pathology
By Clinical Pharmacology by Dr. Aftab Sheikh
By K. D. Tripathi

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GASTROESOPHAGEAL REFLUX DISEASE

INTRODUCTION:-

Gastroesophageal reflux disease (GERD) occurs when stomach acid repeatedly flows back into the tube connecting your mouth and stomach (esophagus). This backwash (acid reflux) can irritate the lining of your esophagus.

PATHOPHYSIOLOGY:-

The pathophysiology of GERD is multifactorial and is best explained by various mechanisms involved, including the influence of the tone of the lower esophageal sphincter, the presence of a hiatal hernia, esophageal mucosal defense against the refluxate and esophageal motility.

RISK FACTORS:-

Conditions that can increase your risk of GERD include:

*Obesity

*Bulging of the top of the stomach up above the diaphragm (hiatal hernia)

*Pregnancy

*Connective tissue disorders, such as scleroderma

*Delayed stomach emptying

*Factors that can aggravate acid reflux include:

*Smoking

*Eating large meals or eating late at night

*Eating% certain foods (triggers) such as fatty or fried foods

*Drinking certain beverages, such as alcohol or coffee

*Taking certain medications, such as aspirin

CAUSES:-

GERD is caused by frequent acid reflux or reflux of non-acidic content from the stomach.When you swallow, a circular band of muscle around the bottom of your esophagus (lower esophageal sphincter) relaxes to allow food and liquid to flow into your stomach. Then the sphincter closes again.If the sphincter does not relax as it should or it weakens, stomach acid can flow back into your esophagus. This constant backwash of acid irritates the lining of your esophagus, often causing it to become inflamed.

SIGNS AND SYMPTOMS :-

*A burning sensation in your chest (heartburn), usually after eating, which might be worse at night or while lying down.

*Backwash (regurgitation) of food or sour liquid.

*Upper abdominal or chest pain.

*Trouble swallowing (dysphagia)

*Sensation of a lump in your throat.

DIAGNOSIS:-

To confirm a diagnosis of GERD, or to check for complications

(1) Upper endoscopy
(2) Ambulatory acid (pH) probe test
(3) X-ray of the upper digestive system.
(4) Esophageal manometry
(5) Transnasal esophagoscop

TREATMENT:-

Nonprescription medications:

Antacids that neutralize stomach acid. Antacids containing calcium carbonate, such as Mylanta, Rolaids and Tums, may provide quick relief. But antacids alone won't heal an inflamed esophagus damaged by stomach acid. Overuse of some antacids can cause side effects, such as diarrhea or sometimes kidney problems.

Medications to reduce acid production. These medications — known as histamine (H-2) blockers — include cimetidine (Tagamet HB), famotidine (Pepcid AC) and nizatidine (Axid AR). H-2 blockers don't act as quickly as antacids, but they provide longer relief and may decrease acid production from the stomach for up to 12 hours. Stronger versions are available by prescription.

Medications that block acid production and heal the esophagus. These medications — known as proton pump inhibitors — are stronger acid blockers than H-2 blockers and allow time for damaged esophageal tissue to heal. Nonprescription proton pump inhibitors include lansoprazole (Prevacid 24 HR), omeprazole (Prilosec OTC) and esomeprazole (Nexium 24 HR).

Prescription medications:

Prescription-strength treatments for GERD include:

Prescription-strength proton pump inhibitors. These include esomeprazole (Nexium), lansoprazole (Prevacid), omeprazole (Prilosec), pantoprazole (Protonix), rabeprazole (Aciphex) and dexlansoprazole (Dexilant).Although generally well tolerated, these medications might cause diarrhea, headaches, nausea, or in rare instances, low vitamin B-12 or magnesium levels.Prescription-strength H-2 blockers. These include prescription-strength famotidine and nizatidine. Side effects from these medications are generally mild and well tolerated.

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HEPATITIS- C

OVERVIEW :-

Hepatitis C is a viral infection that causes liver swelling, called inflammation. Hepatitis C can lead to serious liver damage. The hepatitis C virus (HCV) spreads through contact with blood that has the virus in it.

EPIDIMOLOGY:-

Globally, an estimated 50 million people have chronic hepatitis C virus infection, with about 1.0 million new infections occurring per year. WHO estimated that in 2022, approximately 242 000 people died from hepatitis C, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer).

MANAGEMENT :-

Antiviral medications, including sofosbuvir and daclatasvir, are used to treat hepatitis C. Some people's immune system can fight the infection on their own and new infections do not always need treatment. Treatment is always needed for chronic hepatitis C.

National Guidelines for the Management of Viral Hepatitis

Viral hepatitis is defined as inflammation of the liver cells due to viral infection. The burden of liver disease in South Africa is mostly underestimated as viral hepatitis, in particular chronic infection, is a silent and neglected cause of morbidity and mortality. However, the burden of disease is likely substantial given the prevalence of chronic viral hepatitis. This burden is further compounded by the lack of screening and access to care and treatment as well as inadequate disease surveillance, human and financial resources. The National Guidelines for the Management of Viral Hepatitis were developed, with the purpose to: inform healthcare workers in the public and private sectors about the disease, its epidemiology in South Africa and current methods of diagnosis and therapy strengthen the healthcare response to viral hepatitis empower communicable diseases workers and stakeholders to make informed decisions regarding appropriate and cost effective interventions

PATHOGENESIS:-

HCV is a non-cytopathic virus[35] that enters the liver cell and undergoes replication simultaneously causing cell necrosis by several mechanisms including immune-mediated cytolysis in addition to various other phenomena such as hepatic steatosis, oxidative stress and insulin resistance.

DIAGNOSIS:-

HCV-people who infection is diagnosed in 2 steps: Testing for anti-HCV antibodies with a serological test identifies have been infected with the virus. If the test is positive for anti-HCV

antibodies, a nucleic acid test for HCV ribonucleic acid (RNA) is needed to confirm chronic infection and the need for treatment.

TREATMENT:-

Hepatitis C is treated using direct-acting antiviral (DAA) tablets.DAA tablets are the safest and most effective medicines for treating hepatitis C.They're highly effective at clearing the infection in more than 90% of people.The tablets are taken for 8 to 12 weeks. The length of treatment will depend on which type of hepatitis C you have.Some types of hepatitis C can be treated using more than 1 type of DAA. NHS- approved hepatitis C medicines include:

sofosbuvir

a combination of ledipasvir and sofosbuvir

a combination of ombitasvir, paritaprevir and ritonavir, taken with or without dasabuvir

a combination of elbasvir and grazoprevir

a combination of sofosbuvir and velpatasvir

a combination of sofosbuvir, velpatasvir and voxilaprevir

a combination of glecaprevir and pibrentasvir

Ribavi

COMPLICATION:-

Chronic hepatitis C can be a serious disease resulting in long-term health problems, including liver damage, liver failure, cirrhosis, liver cancer, and even death. It is the most common reason for liver transplantation in the United States.

REFERENCE:-

By Robins pathology

By Clinical Pharmacology by Dr Aftab sheikh

By K D Tripathi

SAKSHI YADAV
5th SEM 3rd YEAR (2021-2026/MBBS)

TRAVELLER'S DIARRHEA

INTRODUCTION-

Traveller's diarrhea is common condition that affect individual travelling to foreign countries , particularly in region with poor sanitation or unsafe drinking water . it is characterized by sudden onset of loose water , often accompanied by abdominal cramps , nausea by consuming food or water contaminated with baacteria, viruses, or parasites.

EPIDEMIOLOGY-

Traveller's diarrhea is prevalent among travelers to developing countries affecting up to 50% of individual . I it's primarily caused by bacterial pathogens like ESCHERICHI COLI , with viral and parasitic causes also possible. Prevention includes practising good hygiene and avoiding risky and drinks.

RISK FACTORS-

- ✓ consumptoin of risky foods
- ✓ poor sanitation
- ✓ inadequate and hygiene
- ✓ weakened immune system
- ✓ previous history of the condition

SIGN AND SYMPTOMS-

- ✓ Loose watery stools
- ✓ abdominal cramps
- ✓ nausea, vomiting
- ✓ fever , malaise

COMPLICATIONS-

Dehydrations:- excessive fluid loose from diarrhea and vomiting can lead to dehydration, particularly in young children, older adults, and individuals with weakened immune system . Electrolyte imbalance :- Dehydration can disrupt the balance of electrolyte in body including sodium, potassium, and chloride leading to electrolyte imbalance . persistent diarrhea

PATHOGENESIS-

The pathogenesis of travelers diarrhea involes ingestion of contaminated food or water containing bacteria, viruses, or parasites. this pathogens inter the digestive tract and colonize the intestine , leadind to inflammation and increased fluid secreation . this results in the characteristic symptoms of diahhea , including loose stools , abdominal cramps, and sometimes vomiting . most cases are self limiting and resolve within a few days, but in severe cases, dehydration and complication may occur.

DIAGNOSIS-

Dianosing travelers diarhhea typically involves assessing clinical symptoms , recent traveler history, and potential exposure to contaminted food or water. in most cases stool sample analysis is not required anless symptoms are severe or complication are suspected.

TREATMENT-

Treatment of travelers diarrhea typically involves hydration, rest and symptomatic relief with medication like loperamide(Imodium) or bismuth subsalicylate(Pepto-Bismol). In severe cases or if symptoms persist, antiboitics may be prescribed . Its essential to stay hydrated and avoid high risk foods and drinks to prevent futher iliness.

CONCLUSION-

Traveler's diarrhea is common ailment among travelers to foreign countries characterized by sudden onset of loose stools and abdominal discomport. treatment involves hydration , rest, and symptomatic relief, with antibodies reserved for severe cases . prevention through good hygiene and food safety practices is essentials to minimize risk.

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2.https://www.mayoclinic.org/diseases-conditions/travelers-diarrhea/symptoms-causes/syc-20352182

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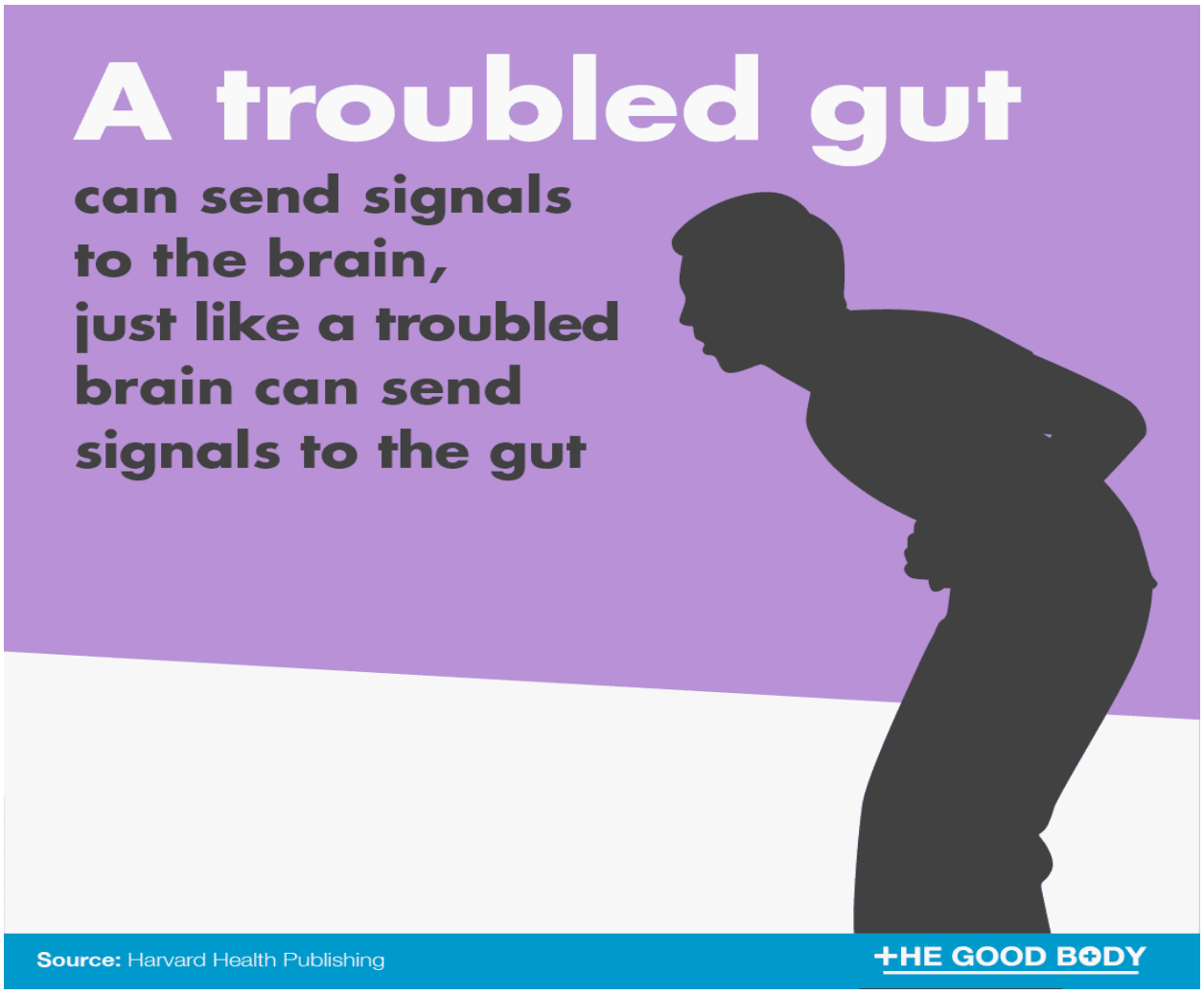

ANJALI
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HEMORRHOIDS

INTRODUCTION

It is also known as piles, are swollen veins located around rectum or in the anus. They can cause discomfort, itching,pain and sometimes bleeding, especially during bowel movement .

EPIDEMIOLOGY-

Hemorrhoids are common, affecting about 44% of general population. They occur more frequently in adults agd 45 to 65 and are slightly more common in men. Hemorrhoids are quite common, affecting about 4% of the adult population globally. Factors like age, genetics, pregnancy, and lifestyle habits such as prolonged sitting, low-fiber diet, and obesity can contribute to their development. While they can occur at any age, they're more common in adults aged 45-65. Though not typically serious, they can cause discomfort and sometimes bleeding. Treatment often involves lifestyle changes, topical creams, or in severe cases, procedure like rubber band ligation or surgery.

RISK FACTOR-

- Constipation
- Chronic diarrhea
- Pregnancy
- Obesity
- Sedentary lifestyle

SIGN AND SYMPTOMS-

- Rectal bleeding- First sign of hemorrhoids
- Itching and irritation
- Pain or Discomfort
- Protrusion
- Mucus discharge

COMPLICATION-

Thrombosis -External hemorrhoids can develop blood clot leads to thrombosis .
Strangulation-Prolonged internal hemorrhoids become trapped outside the anus.
Anemia
Anal fistula and fissure
1.Thrombosis: When a blood clot forms within a hemorrhoids, causing severe pain and swelling.
2.Bleeding: Hemorrhoids can bleed, sometimes leading to anemia if the bleeding is persistent.
3.Strangulation: This occurs when the blood supply to an internal hemorrhoid is cut off, leading to tissue death.
4.Chronic blood loss: prolonger Or recurrent bleeding can lead to iron deficiency anemia.
5.Prolapse: Internal hemorrhoids may protrude outside the anus, especially during bowel movements, and can sometimes become trapped outside the anus.
6.Infection: In rare cases, if hemorrhoids are not properly cleaned if hygiene is poor, they can

become infected.

PATHOGENESIS-

Hemorrhoids develop due to increased pressure on the vein in rectal and anal area, weakning their walls and causing swelling.

DIAGNOSIS-

Diagnosing hemorrhoids typically involves a physical examination of anal by healthcare provider. They vidsually inspect for selling blood vessels may perform digital rectal exam to check for internal hemorrhoids. In some cases additional test like ANOSCOPY or SIGMOIDOSCOPY or COLONOSCOPY may be used for clear examination.

TREATMENT-

Typiaclly involves lifestyle modification such as increasing fibre intake and staying hydrated along with over the counter cream for symptoms relief.In more severe cases ,medical procedure like rubber band ligation or suegery may be necesary.

CONCLUSION-

Hemorrhoids are common conditon characterised by swolen veins in rectal and anal area while they can cause discomfort, itching, and bleeding, they are usually manageable with lifestyle modification, over-the-counter treatment and medical procedure if necessary. Seeking prompt medical attention can help alleviate symptoms and prevent complications. With proper care and management, most people can find relief from hemorrhoids symptoms and improve their quality of life.

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ADITYA JAISWAL
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MEGACOLON DISEASE

INTRODUCTION

Megacolon,as well as megarectum,is a descriptiveterm.It denotes dilatation of thebcolon that is not caused by a mechanical obstruction. Although the definition of megacolon has varied in theliterature, most researchers use the measurement of greater than 12 cm for the cecum as thestandard.

Mega colon can be divided into the following three categories:

Acute megacolon (pseudo-obstruction)

Chronic megacolon , which includes congenital,acquired, and idiopathic causes

Toxicmegacolon

EPIDEMIOLOGY

United State Data

No large-scale studies have been conducted to determine prevalence/incidence of acquired megacolon.

Internationaldata

The most common cause of megacolon worldwide is infection with trypanosoma Ruiz (Chagasdisease).

Race-,sex-, and age-old demographics

Race has not been documented to play a role I megacolon.

The frequency of acquired megacolon is equally distributed between the sexes.the congenital megacolon, Hirschsprungdisease, predominantly occurs in males.

Although clinically chronic megacolon can occurinany age group, inherited types usually presentin young patients,and acquired types usually presenting older patients.

ETIOLOGY

Causes of acquired megacolon Neurologic diseases include the following:

- ✓ Chagasdisease
- ✓ Parkinson disease
- ✓ Myotonicdystrophy
- ✓ Diabeticneuropathy
- ✓ Spinalcordinjury
- ✓ Paraneoplasticneuropathy
- ✓ Amyloidosis
- ✓ Systemic diseases include the following:
- ✓ Scleroderma
- ✓ Dermatomyositis/polymyositis

- ✓ Systemiclupuserythematosus

Mixed connective tissue disease Metabolicdiseasesincludethefollowing:

- ✓ Hypothyroidism
- ✓ Hypokalemia
- ✓ Porphyria
- ✓ Pheochromocytoma

Medication-induced conditions can cause acquired megacolon.Idiopathic causes in clude the following:

- ✓ Nonfamilial visceral neuropathy (sporadic hollow visceral neuropathy or chronic idiopathic intestinal pseudo-obstruction)
- ✓ Results from damage to the myenteric plexus from drugs or viral infections
- ✓ The most common non mechanical cause of acquired megacolonis infection with rusi (Chagasdisease).
- ✓ This infection results in the destruction of the enteric nervous system.
- ✓ Although this disease was originally confined to south America,recentestimatesindicatethat350,000 people in the United States are seropositive, one third of whom are thought to havechronic Chagasdisease.

Causes of congenital megacolon

- ✓ Entericneuropathies include the following:
- ✓ Hirschsprungdisease(congenitalaganglionosis)
- ✓ It is caused by a single gene mutation of the RET proto-oncogene on band 10q11.2. The defect occurs in1in 5000livebirths.somecasesarefamilial,with an overall incidence of 3.6% among siblings of index cases.
- ✓ Waardenburg-Shahsyndrome(piebaldism,neuraldeafness,megacolon)
- ✓ idiopathic
- ✓ In the newborn period,an unrecognized imperforate anus maybe the cause of megacolon.As inflammation progresses into the smooth-muscle layers of the colon, NO and local inflammatory modulator sapper to be involved intoxicated colon PATHOGENESIS.

PATHOGENESIS

The exact mechanism by which megacolon develops is not known.however, the end result is the same: severely decreased intestinal motility causes a buildup of feces, air, and intestinal secretions in the colon, which presents as dilation of the colon.

There are several suggested mechanisms by which intestinal motility might be decreased:

In acute, non-toxicmegacolon, there is damage to the autonomi nervous system

I chronic megacolon ,there is inherent neurological and/ or muscular dysfunction in the bowels

In toxic megacolon, there is reduced smooth muscle activity,likely as the result of inflammation. This may be related to increase nitric oxide synthesis.

DIAGNOSES

Mega colon can be diagnosed by observing the size of the colon an abdominal x-rays can.Most physician sagree that a colon diameter greater than 12 centimeters at the cecum should be classified as megacolon.

A contrast enhanced CT scan isused to confirm these findings, additionally showing the colonist ree of mechanical obstruction.If a CT scan is not possible, colonoscopy can be performed to verify the colon is free of mechanical obstruction. However, in toxic megacolon, colonoscopy should not be performed due to high risk of perforating the colon.

TREATMENT

- ✓ Treatment for megacolon starts by addressing the underlying cause (such as the offending medication or disease),if known.In acute megacolon, all food and drink should be withheld ANDA nasogastric tube placed. If non-toxic, neostigmine should be administered, and if necessary,the colon itself should be decompressed by means of a colonoscopy. If toxic, steroids and broad-spectrum antibiotics should've given.
- ✓ In chronic megacolon, both dietary and pharmacological methodsshould beused to increase intestinal motility. Laxatives and enemas may also be used to prevent fecal impaction.
- ✓ If the patient does note spond to the streat ments with in one to three days,it may be necessary to use surgery to remove all or part of the colon.Following colectomy, options include ileocecal anastomosis and ileostomy.

Is megacolon reversible?

In most cases,megacolon reversible

SUMMARY

Megacolon is an abnormal dilation of the colon that can be categorized as acute, toxic, or chronic. Acute megacolon, also known as Ogilvie syndrome, is associated with damage to the autonomic nervous system and often occurs in ill or postoperative patients with no clear cause.Toxic megacolon is characterized by concurrent systemic toxicity, usually as the result of colon inflammation following an infection. Chronic megacolon is caused by bowel dysfunction as a result of neurological or muscular disorders.

In all three types, symptoms include constipation, bloating, and abdominal pain. Diagnosis of megacolon relies on the use of x-ray and tuscans, and a variety of treatment options can be employed depending on the cause and nature. In rare cases, surgery may be needed to treat megacolon.

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Appendicitis

WHAT IS APPENDICITIS

The appendix is a thin tube that is joined to the large intestine. It sits in the lower right part of your belly (abdomen). When you are a young child, your appendix is a working part of your immune system, which helps your body to fight disease. When you are older, your appendix stops doing this and other parts of your body keep helping to fight infection.

The appendix can get infected. If not treated it can burst (rupture). This can happen as soon as 48 to 72 hours after you have symptoms. Because of this, appendicitis is a medical emergency. If you have symptoms, see a doctor right away to avoid more infection, which can be life-threatning.

CAUSES

Appendicitis happens when the inside of your appendix is blocked. Appendicitis may be caused by various infections such as virus, bacteria, or parasites, in your digestive tract. Or it may happen when the tube that joins your large intestine and appendix is blocked or trapped by stool. Sometimes tumors can cause appendicitis.

The appendix then becomes sore and swollen. The blood supply to the appendix stops as the swelling and soreness get worse. Without enough blood flow, the appendix starts to die.

SYMPTOMS

- ✓ Symptoms of appendicitis include:
- ✓ dull pain centred around the navel, which progresses to a sharp pain in the lower right side of the abdomen.
- ✓ pain in the lower back, hamstring or rectum (less commonly)
- ✓ fever.
- ✓ vomiting.
- ✓ diarrhoea or constipation.
- ✓ loss of appetite.

SIGNS

- ✓ Dull pain near the naval (easily appendicitis)
- ✓ Pain in the persistence and increase in intensity
- ✓ Nausea and vomiting
- ✓ Abdominal swelling
- ✓ Pain intensifies and move to lower right abdomen
- ✓ Loss of appetite
- ✓ Fever inability to pass gas

DIAGNOSIS

To help diagnose appendicitis, your health care team will likely take a history of your symptoms and examine your abdomen.

Tests used to diagnose appendicitis include:

Physical exam:- A member of your health care team may apply gentle pressure on the painful area. When the pressure is suddenly released, appendicitis pain will often feel worse. This is because of inflammation of the lining of the abdominal cavity, called the peritoneum.

Your care team also may look for abdominal stiffness and a tendency for you to flex your abdominal muscles in response to pressure over the inflamed appendix. This is called guarding.

A member of your health care team also may use a lubricated, gloved finger to examine your lower rectum. This is called a digital rectal exam. People of childbearing age may be given a pelvic exam to check for other problems that could be causing the pain.

Blood test:- This test checks for a high white blood cell count. A high white blood cell count may indicate an infection.

Urine test:- You may have to take a urine test, also called a urinalysis. A urinalysis makes sure that a urinary tract infection or a kidney stone isn't causing your pain.

Imagine test:- You also may have imaging tests to help confirm appendicitis or find other causes for your pain These tests may include an abdominal X-ray, an abdominal ultrasound, a CT scan or an MRI

TREATMENT

Appendicitis treatment usually involves surgery to remove the appendix. Before surgery, you may be given antibiotics to treat infection.

Surgery to remove the appendix

Appendectomy is a surgery to remove the appendix. Appendectomy can be performed as open surgery using one abdominal cut about 2 to 4 inches long. This is called laparotomy. The surgery also can be done through a few small abdominal cuts. This is called laparoscopic surgery. During a laparoscopic appendectomy, the surgeon places special tools and a video camera into your abdomen to remove your appendix.

In general, laparoscopic surgery allows you to recover faster and heal with less pain and scarring. It may be better for older adults and people with obesity.

But laparoscopic surgery isn't right for everyone. You may need an open appendectomy if your appendix has ruptured and infection has spread beyond the appendix, or you have an abscess. An open appendectomy allows your surgeon to clean the abdominal cavity.

Expect to spend 1 to 2 days in the hospital after your appendectomy.

Draining an abscess before appendix surgery

If your appendix has burst and an abscess has formed around it, the abscess may be drained. To drain it, a tube is placed through your skin into the abscess. Appendectomy can be performed several weeks later, after the infection is under control.

If your appendicitis isn't serious and doesn't require surgery, antibiotics may be used alone. However, if the appendix isn't removed, there is a higher chance of appendicitis coming back.

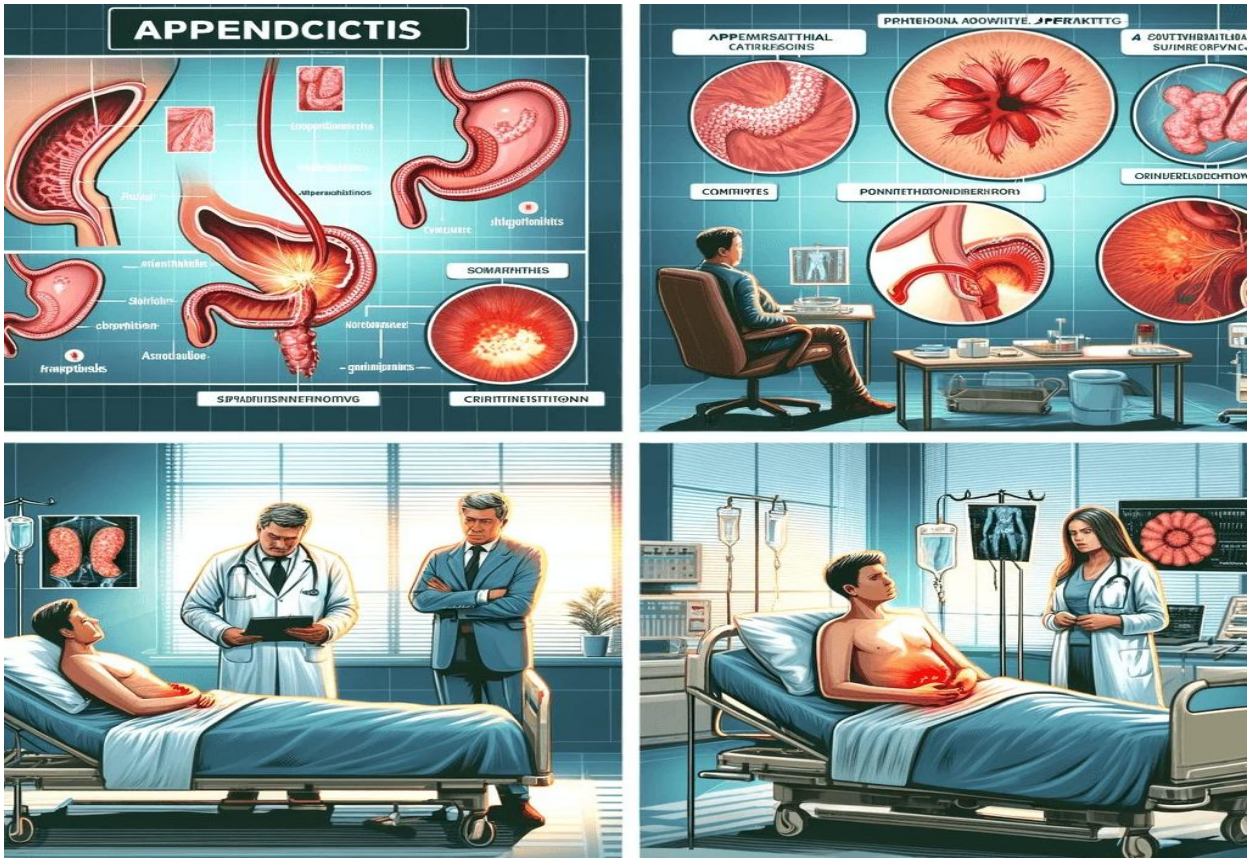
COMPLICATIONS Complications of appendicitis and appendectomy include surgical site infections, intra-abdominal abscess formation (3% to 4% in open appendectomy and 9 to 24% in Laparoscopic appendectomy),prolonged ileus, enterocutaneous fistula, and small bowel obstruction.

SUMMARY

anatomy
appendix
epidemiology
typically children and young adults
peak incidence 2nd to 3rd decade of life
presentation
initial periumbilical pain with fever, nausea and vomiting
progresses to localized right iliac
fossa pain
pathophysiology
obstruction of the appendiceal lumen
fluid accumulation, infection, venous congestion, ischemia/necrosis
causes
lymphoid hyperplasia (60%)
appendicolith (33%)
rare: foreign body, Crohn's disease, tumor
investigation
US is often all that is required (quick, dynamic and no radiation)
cross-sectional imaging is more sensitive (CT and MRI)
•treatment
appendectomy (laparoscopic or open)

REFRENCE

By clinical pharmacology Dr.aftab Sheikh, By K.D tripathi pharmacology



MUHAMMAD YASIR MAJEED
8th Semester 4th Year Session: 2020-2025

MALABSORPTION SYNDROME

DEFINATION:

Malabsorption refers to alterations of the gastrointestinal tract (GIT) affecting the digestion, absorption and transport of nutrients across the bowel wall. Malabsorption is defined as intestinal absorption capacity falling short of 85%. It is regarded as an important clinical indicator of intestinal failure.1 The latter refers to the inability of the GIT to digest and absorb sufficient nutrients to maintain the GIT mucosa integrity, fluid balance, nutritional status and overall health.

CAUSES:

The causes, diagnostic tests and treatment of malabsorption can be described in terms of premucosal, mucosal and postmucosal aberrations.

Causes of premucosal malabsorption include diseases and conditions that result in impaired digestion . Such clinical settings include chronic pancreatitis, cystic fibrosis and pancreatic cancer, all of which are associated with inadequate pancreatic enzyme secretion as well as cholestatic liver disease and bacterial overgrowth that could lead to lack of solubilising bile salts.

Causes of mucosal malabsorption include conditions that affect the gut mucosa itself, and result in a reduced absorptive area. Examples include coeliac disease, inflammatory bowel diseases and Whipple's disease.

Causes of postmucosal malabsorption comprise conditions that result in altered nutrient transport, i.e. vascular or lymphatic obstruction.

Table I: Pathophysiology of malabsorption [3,4]

Premucosal
Impaired digestion
Bile acid/enzyme deficiencies
Mucosal
Reduced absorption
Bowel resection
Diseases affecting absorption
Postmucosal
Altered nutrient transport
Vascular or lymphatic abnormalities

CONSEQUENCES

The consequences of malabsorption are directly linked to the extent and duration of the underlying causes. This may include

- ✓ Abdominal pain and bloating (due to bacterial gas production and bacterial overgrowth), diarrhoea and steatorrhoea, fluid and electrolyte losses, anaemia (iron, folate and vitamin B12), growth retardation and osteopenia (malabsorption of calcium, vitamin D, phosphate and magnesium results in secondary hyperparathyroidism).3–5
- ✓ The overarching consequence of malabsorption is malnutrition. Because malnutrition can be regarded as an independent risk factor for morbidity and mortality, malabsorption is a condition that needs to be identified early and treated timeously.
- ✓ According to a recent article by Richard et al describing the consequences of malnutrition, hospitalised malnourished patients had a significantly greater risk of developing infectious complications, respiratory failure, cardiac arrest, cardiac failure, arrhythmias and wound dehiscence.
- ✓ The malnourished patients also had a significantly longer duration of hospitalisation, regardless of the underlying disease and its course of treatment. Furthermore, the risk for hospitalisation of longer than 12 days' duration is four to five times higher in malnourished patients.

DIAGNOSIS

Diagnostic tests to identify malabsorption also include those tests that can identify impaired digestion (premucosal), reduced absorption (mucosal) and altered nutrient transport (postmucosa).

- ✓ Serum levels of electrolytes, minerals and vitamins may serve as a good proxy marker of nutritional status and hence low values can serve as an indicator of impaired digestion and premucosal malabsorption. .
- ✓ A faecal fat excretion test is a simple and quick, albeit cumbersome, test to measure fat malabsorption, where a fat content of less than 7 g/day following a 100 g fat intake for 72 hours is regarded as normal. The only disadvantage is that the test cannot differentiate between enteric and pancreatobiliary causes of malabsorption.1,3,4
- ✓ The hydrogen breath test can be used to identify carbohydrate malabsorption. This test is based on the assumption that undigested carbohydrates will be fermented by colonic bacteria, resulting in the accumulation of hydrogen.3,4 The gas is absorbed by the intestinal mucosa and excreted through the lungs. However, it is important to remember that about 18% of individuals are hydrogen nonexcretors,4 and in such individuals the test results are unreliable.
- ✓ Various carbohydrates may be measured, but glucose and lactulose are probably most commonly used for the identification of bacterial overgrowth and hence carbohydrate maldigestion.7,8 Irrespective of the type of carbohydrate used, exhaled hydrogen is measured in parts per million (ppm), and an increase of more than 20 ppm above baseline values is considered a positive result. The test results may be influenced by various factors, for example the use of antibiotics or laxatives, periods of fasting, diet over the previous 24 hours, and therefore adequate patient preparation before the test is important.

Table II: Diagnostic tests for malabsorption[1,3–5,7,8]

Premucosal
Impaired digestion
Serum electrolyte, mineral and vitamin values
Faecal fat excretion, hydrogen breath test
Ultrasound for obstructions/calcifications
Mucosal
Reduced absorption
Bowel resection
Endoscopy and histology
Xylose test, Schilling test
Postmucosal
Altered nutrient transport
Ultrasound/contrast for fistulae

TREATMENT

The treatment plan for malabsorption must be, by necessity, cause specific,3 with appropriate adaptation to a diet that would best support a given setting. **For instance**, pancreatic enzymes may be added to food to aid its absorption. Normally between 25 000 to 40 000 IU of lipase is required per meal. However, this dose should be titrated against the clinical response. **For best results** it is recommended that meals should be divided into five or six smaller meals throughout the day. A lipase dosage in excess of 75 000 IU per meal is not recommended.9 **In the case of altered nutrient transport caused by obstruction, surgery is the best option.**

Table III: Treatment of malabsorption

Premucosal
Impaired digestion
Partially digested food
Pancreatic enzyme supplementation
Surgery for obstruction
Mucosal
Reduced absorption
Partially digested food
Disease-specific treatment
Postmucosal
Altered nutrient transport
Surgery for obstruction

Table V: Malabsorption index: implementation[10]

Total points	Degree of malabsorption	Recommended nutritional therapy
0	Low	Utilise whole protein diet
2–6	Moderate	[a]MCT-containing whole protein-based diet, advanced to peptide-based diets if not tolerated
7–14	High	Use peptide-based MCT-containing diets; if <60% goal achieved, advance to [b]TPN
15+	Very high	TPN with/without elemental diet

a = medium-chain triglycerides, b = total parenteral nutrition

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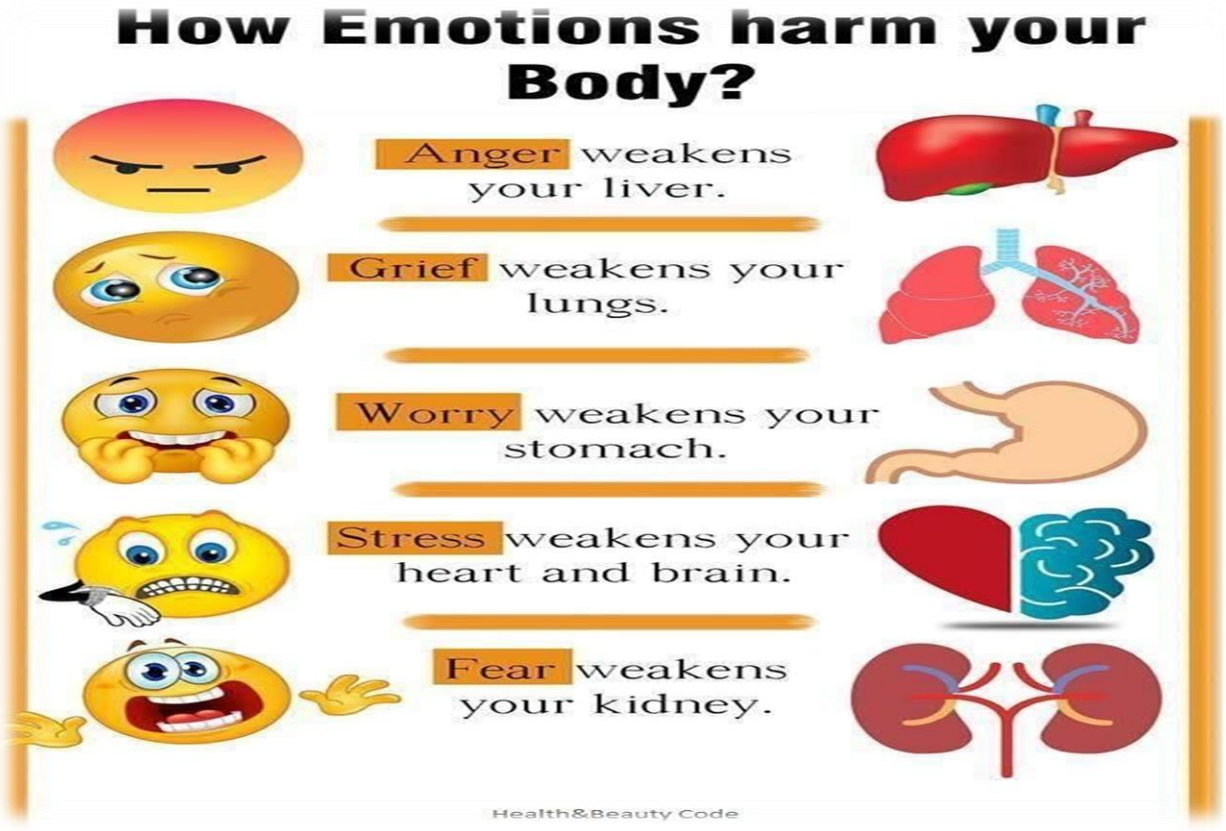

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GALLSTONE (Cholelithiasis)

INTRODUCTION

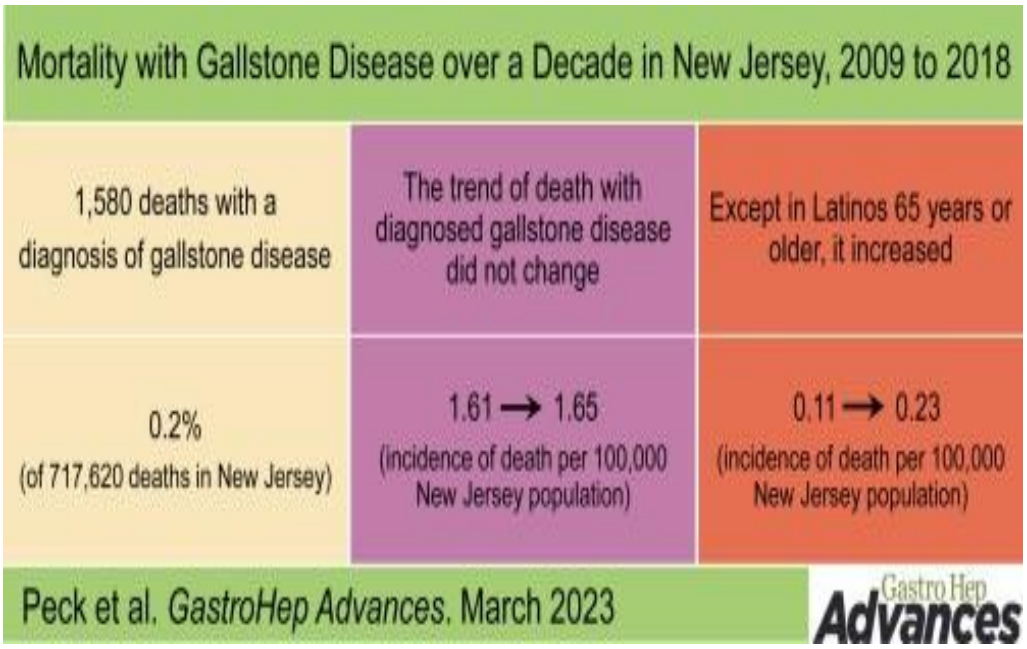
Calculous disease of the biliary tract is the general term applied to diseases of the gallbladder and biliary tree that are a direct result of gallstones. Gallstone disease is the most common disorder affecting the biliary system.

DEFINATION

Cholelithiasis is the formation of gallstones, which are composed of cholesterol, calcium salts, and bile pigments.

INCIDENCE & EPIDEMOLOGY

In the United States, about 20 million people (10-20% of adults) have gallstones.Every year 1-3% of people develop gallstones and about 1-3% of people become symptomatic.



Prevalence of cholesterol cholelithiasis in other Western cultures is similar to that of the United States, but it appears to be somewhat lower in Asia and Africa.

Types of Gallstones

Classified according to the cholesterol content of the stones:

Cholesterol stones: 80% or more cholesterol. They appears yellow in colour and are oval in shape with a dark spot in the centre.

Pigment stones: less than 20% cholesterol. They are either black or brown and form when bile has high bilirubin concentration.

Mixed stones: Between 20 and 80% cholesterol along with other constituents such as bile salts, calcium, etc.

Stages and classification

1. Initial stage or "prestone"Viscous, nonhomogeneous bile

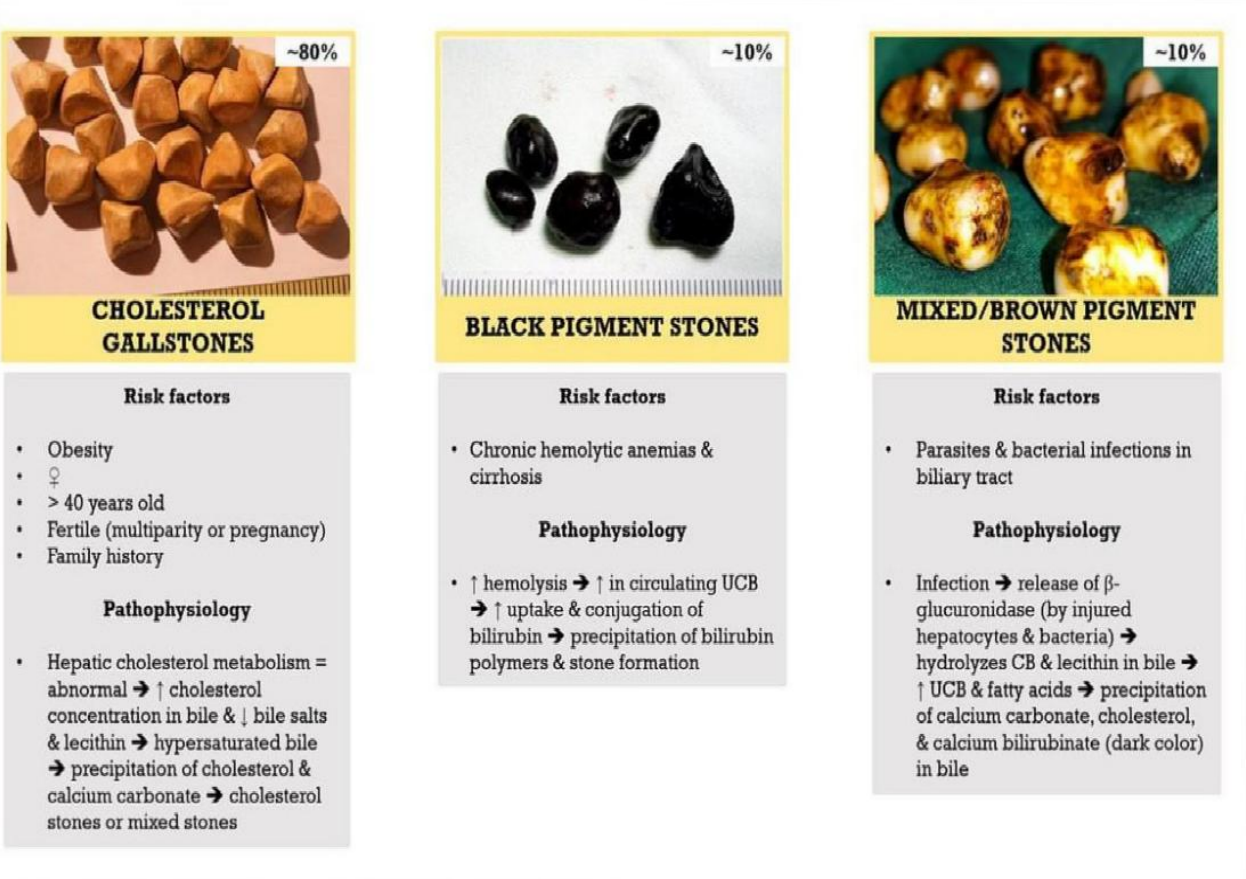
II. Formation of stones

- Localization:

in gallbladder,
in common bile duct,
in hepatic ducts
Composition:cholesterol,pigment, mixed
Clinical forms: asymptomatic (latent) and
manifesting Pain form with typical bile colics

III. Stage of chronic, recurrent cholecystitis with concremental

IV. Stage of complications



Predisposing Factors For Gallstone Formation

Cholestrol and mixed stones

Demographic and genetic factors - familial disposition; hereditary aspects; greater prevalence in Northern Europe and North America, lower - in Asia

Obesity - increased biliary secretion of cholesterol

Weight loss - mobilization of tissue cholesterol leads to increased biliary cholesterol secretion while enterohepatic circulation of bile acids is decreased

Female sex hormones - Estrogens stimulate hepatic lipoproteins receptors, increase uptake of dietary cholesterol, and increase biliary cholesterol secretion

-Natural and synthetic estrogens lead to decrease bile salt secretion and decreased conversion of cholesterol to cholesterol esters

signs and symptoms

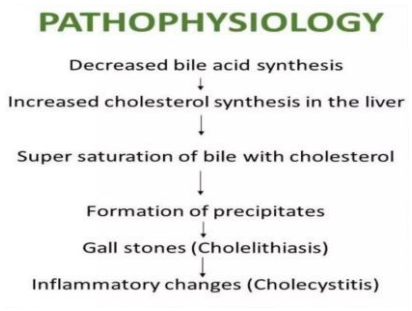
There are three stages of gallstones: asymptomatic, symptomatic, and with complications.

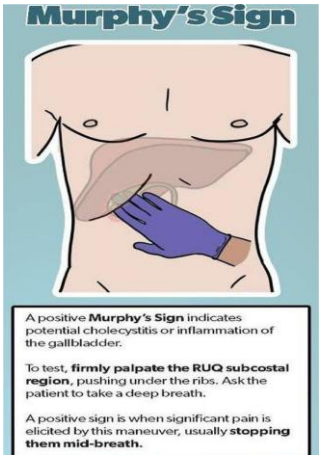
following symptoms:

- ✓ a feeling of abdominal bloating and excessive gas
- ✓ nausea and sometimes vomiting
- ✓ pain that is usually in the upper right or middle part of the abdomen
- ✓ radiation of the pain through to the back or into the shoulder
- ✓ worsening of the pain after a heavy or fatty meal

If complications occur, the individual develop further symptoms:

- ✓ abnormally light-colored stools
- ✓ blockage of the bowels
- ✓ dark-colored urine
- ✓ fever
- ✓ itching
- ✓ jaundice, or yellowing of the eyes and skin
- ✓ severe, constant abdominal pain





SYNDROMES

Pain syndrome Dyspeptic syndrome:

. Gastric Intestinal

Inflammatory syndrome (during exacerbation) **Cholestatic syndrome** (in obstruction of common bile duct)

Dyslipidemia Complications: Cholangitis

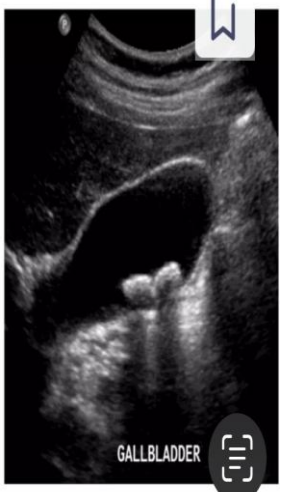
Mechanical obstruction of bile ducts (choledocholithiasis) Galbladder perforation and bile peritonitis

Empyema of gallbladder Gallbladder hydrops Pericholecystitis

Management

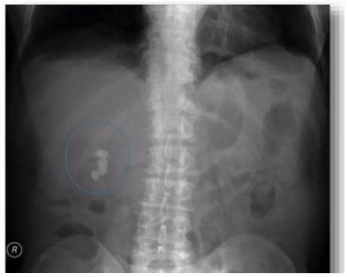
Investigations

- USG abdomen – posterior acoustic shadowing
- Plain X RAY abdomen
- LFT- Increased conjugated bilirubin
  Increased Alkaline Phosphate, GGT, 5'-Nucleotidase
- TLC



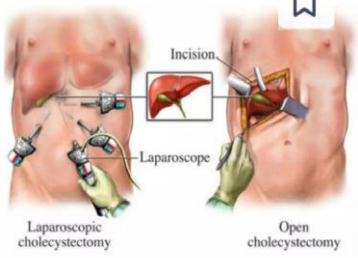
D/D of radio-opaque shadow on x-ray

- Renal stone
- Calcified 12th rib tip
- Phlebolith
- Faecolith
- Calcified lymph node
- Renal cell Ca - calcification
- Calcified Adrenal tumor



Surgical therapy

- Laparoscopic cholecystectomy is ideal.
- Open cholecystectomy is done if patient unfit for laparoscopy through Right Sub-costal(KOCHERS's) incision



Complications of Gall stones

In Gall Bladder-

- Acute cholecystitis
- Chronic cholecystitis
- Empyema of gall bladder
- Mucocele gall bladder
- Perforation – leading to biliary peritonitis
- Gangrene of gall bladder
- Carcinoma

In Bile duct-

- Obstructive jaundice
- Cholangitis
- Acute pancreatitis

In Intestine-

- Acute intestinal obstruction

Treatment

Medical therapy-

GALL STONE DISSOLUTION

Ursodeoxycholic acid (UDCA) - with a functioning Gall bladder with stone less than 10 mm,10-15 mg/kg/day

Pigment stones are non responsive to medical therapy

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Intussusception

OVERVIEW

Intussusception is a serious condition in which part of the intestine slides into an adjacent part of the intestine. This telescoping action often blocks food or fluid from passing through. Intussusception also cuts off the blood supply to the part of the intestine that's affected. This can lead to infection, death of bowel tissue or a tear in the bowel, called perforation

SYMPTOMS

Children

The first sign of intussusception in an otherwise healthy infant may be sudden, loud crying caused by belly pain. Infants who have belly pain may pull their knees to their chests when they cry.
The pain of intussusception comes and goes, usually every 15 to 20 minutes at first. These painful episodes last longer and happen more often as time passes.
Other symptoms of intussusception include:

- ✓ Stool mixed with blood and mucus — sometimes referred to as currant jelly stool because of its appearance.
- ✓ Vomiting.
- ✓ A lump in the belly.
- ✓ Weakness or lack of energy
- ✓ Diarrhea..

Adults

Intussusception is rare in adults. Also, because symptoms of the disorder often overlap with the symptoms of other disorders, it's more challenging to identify. The most common symptom is belly pain that comes and goes. Nausea and vomiting also may occur. People sometimes have symptoms for weeks before seeking medical attention

CAUSES

- ✓ Your intestine is shaped like a long tube. In intussusception, one part of your intestine — usually the small intestine — slides inside an adjacent part. This is sometimes called telescoping because it's similar to the way a collapsible telescope slides together.
- ✓ In some cases in adults, the telescoping is caused by a growth in the intestine, such as a polyp or a tumor, called a lead point. The typical wavelike contractions of the intestine grab this lead point and pull it and the lining of the intestine into the bowel ahead of it. In most cases, however, no cause can be found for intussusception.

Children

In most cases of intussusception in children, the cause is unknown. Intussusception seems to occur more often in the fall and winter. And because many children with the problem also have flu-like symptoms, some suspect a virus may play a role in the condition. Sometimes, a lead

point can be identified as the cause of the condition — most frequently the lead point is a pouch in the lining of the small intestine (Meckel's diverticulum).

Adults

- ✓ In adults, intussusception is usually the result of a medical condition or procedure, including:
- ✓ A polyp or tumor.
- ✓ Scar-like tissue in the intestine, known as adhesions.
- ✓ Weight-loss surgery such as gastric bypass or other surgery on the intestinal tract.
- ✓ Swelling in the intestines due to diseases such as Crohn's disease

RISK FACTORS

Risk factors for intussusception include:

- ✓ **Age.** Children — especially young children — are much more likely to develop intussusception than adults are. It's the most common cause of bowel obstruction in children between the ages of 6 months and 3 years.
- ✓ **Sex.** Intussusception more often affects boys.
- ✓ **Irregular intestinal formation at birth.** Intestinal malrotation is a condition in which the intestine doesn't develop or rotate correctly. This increases the risk of intussusception.
- ✓ **Certain conditions.** Some disorders can increase the risk of intussusception, including:

- Cystic fibrosis.
- Henoch-Schonlein purpura, also known as IgA vasculitis.
- Crohn's disease.
- Celiac disease.

COMPLICATIONS

Intussusception can cut off the blood supply to the affected portion of the intestine. If left untreated, lack of blood causes tissue of the intestinal wall to die. Tissue death can lead to a tear in the intestinal wall, called a perforation. This can cause an infection of the lining of the abdominal cavity, known as peritonitis.

Peritonitis is a life-threatening condition that requires immediate medical attention. Symptoms of peritonitis include:

- ✓ Belly pain.
- ✓ Swelling in the belly area.
- ✓ Fever.
- ✓ Vomiting.

Peritonitis may cause your child to go into shock. Symptoms of shock include:

- ✓ Cool, clammy skin that may be pale or gray.
- ✓ A weak and rapid pulse.
- ✓ Breathing that may be either slow and shallow or very rapid.
- ✓ Anxiety or agitation.
- ✓ Extreme listlessness

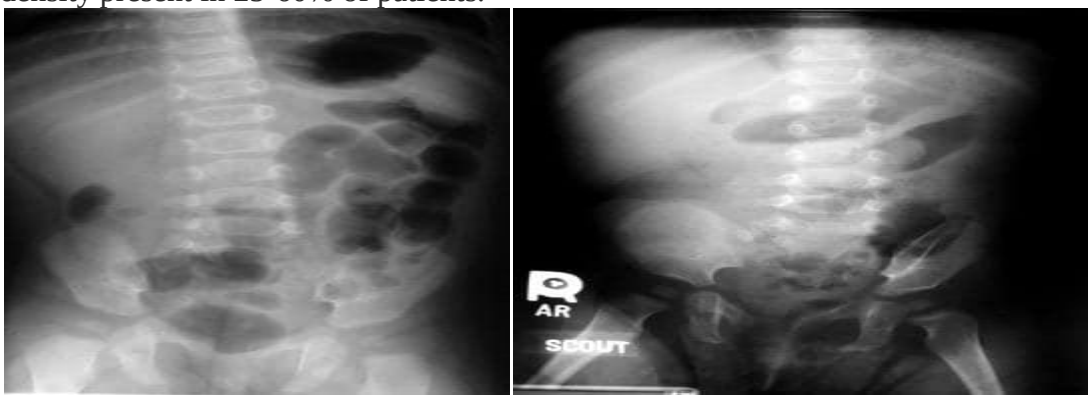
DIAGNOSIS

Your or your child's health care provider will start by getting a history of the symptoms of the problem. The provider may be able to feel a sausage-shaped lump in the belly. To confirm the diagnosis, your provider may order:

Radiographs

After obtaining a thorough history and performing a careful physical examination, obtain plain radiographs of the abdomen with the patient in the supine and upright positions.

Plain abdominal radiography reveals signs that suggest intussusception in only 60% of cases. (See the images below.) Plain radiograph findings may be normal early in the course of intussusception. As the disease progresses, the earliest radiographic evidence includes an absence of air in the right lower and upper quadrants and a right upper quadrant soft tissue density present in 25-60% of patients.



Abdominal radiograph shows small bowel dilatation and paucity of gas in the right lower and upper quadrants and in 2nd image Note intussusception in the left upper quadrant on this plain film of an infant with pain vomiting.These findings are followed by an obvious pattern of small bowel obstruction, with dilatation and air-fluid levels in the small bowel only. If the distention is generalized and the air-fluid levels are also present in the colon, the findings more likely represent acute gastroenteritis than intussusception.

A left lateral decubitus view is also helpful. If the view exhibits air in the cecum, the presence of ileocecal intussusception is highly unlikely.

Ultrasonography and CT Scanning

Hallmarks of ultrasonography include the target and pseudokidney signs. (See the image below.)

Abdominal ultrasonography reveals the classic target sign of an intussusceptum inside an intussuscipiens

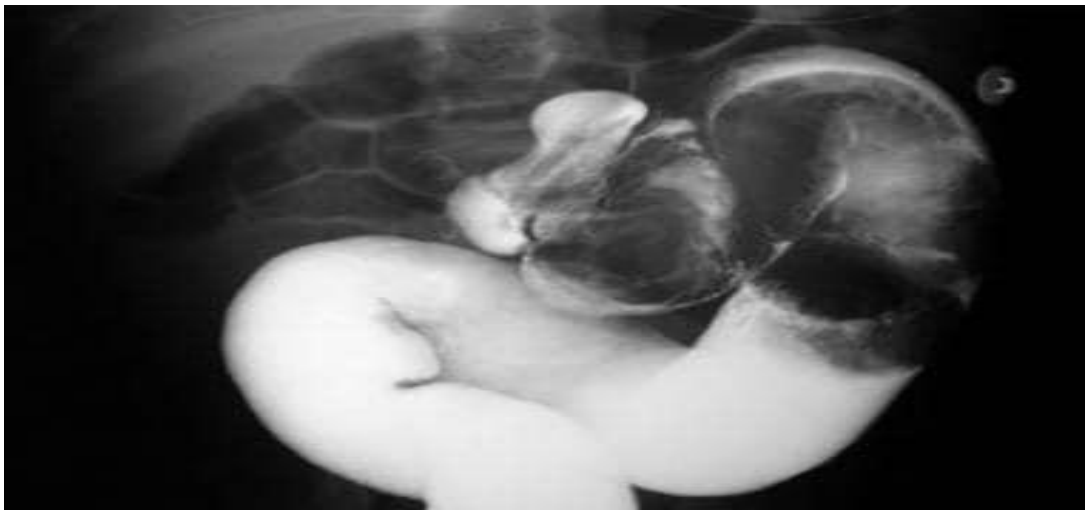
Ultrasonography eliminates the risk of exposure to ionizing radiation and can help to depict lead points and residual intussusceptions. It also helps to rule out other possible causes of abdominal pain. Even so, ultrasonography is highly operator dependent; therefore, interpret results with caution.

The presence of ascites and long segments of intussusception can be used as sonographic predictors of failure for nonoperative management. Sonographic detection of ascites, air, and absence of blood flow in the intestinal wall strongly suggest bowel gangrene.

Computed tomography (CT) scanning has also been proposed as a useful tool to diagnose intussusception (see the image below); however, CT scan findings are unreliable, and CT scanning carries risks associated with intravenous contrast administration, radiation exposure, and sedation

Contrast Enema

The traditional and most reliable way to make the diagnosis of intussusception in children is to obtain a contrast enema (either barium or air). Contrast enema is quick and reliable and has the potential to be therapeutic. (See the images below.)



Barium enema shows intussusception in the descending colon.

Intussusception evident during air contrast enema prior to reduction

Exercise caution when performing contrast enema in children older than 3 years, because most of these patients have a surgical lead point, usually in the small bowel. The diagnostic and therapeutic yield of the enema is lower in these patients. Enema is contraindicated in patients in whom bowel gangrene or perforation is suspected.

TREATMENT

Nonoperative Reduction

Tailor treatment of the child with intussusception to the stage at presentation. For all children, start intravenous fluid resuscitation and nasogastric decompression as soon as possible.
The presence of peritonitis and any evidence of perforation revealed on plain radiographs are the only 2 absolute contraindications to an attempt at nonoperative reduction with a therapeutic enema. Therapeutic enemas can be hydrostatic, with either barium or water-soluble contrast, or pneumatic, with air insufflation. Therapeutic enemas can be performed under fluoroscopic or ultrasonographic guidance. The technique chosen is not important as long as the radiologist performing the enema is comfortable with the method. Preferably, the pediatric surgeon involved is present at the reduction. Enema reduction is more likely to be successful if initiated early (eg, within 4 hours of hospitalization).
A study by Flaum et al presented their experience in intussusception reductions using saline enema under ultrasound control and concluded that it is an efficient and safe procedure
Surgical Reduction

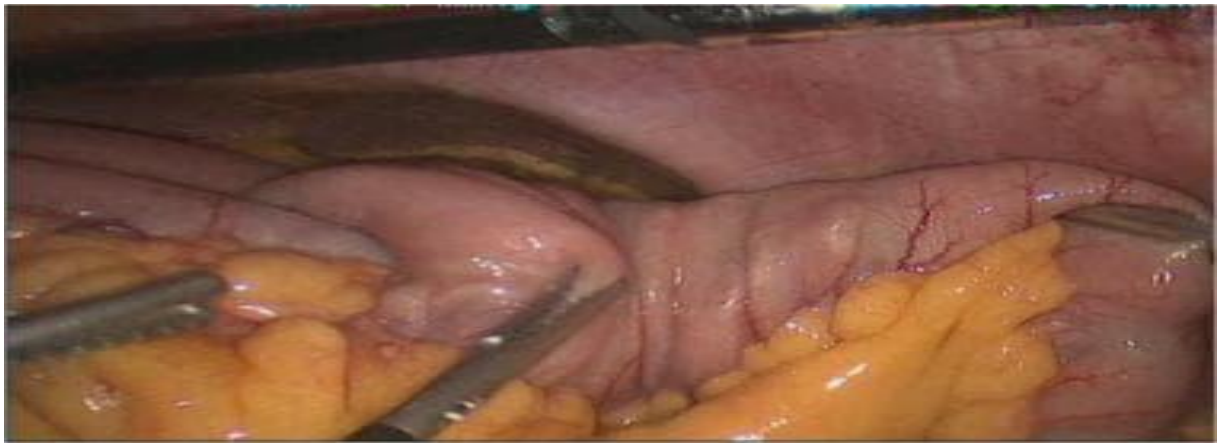
If nonoperative reduction is unsuccessful or if obvious perforation is present, promptly refer the infant for surgical care.
Traditional entry into the abdomen is through a right paraumbilical incision. Deliver the intussusception into the wound and attempt nonoperative reduction. Milking the intussusceptum out of the intussuscipiens is important. Sustain gentle manual pressure rather than pulling out the intussusceptum to avoid risk of iatrogenic perforation. If operative reduction is successful, appendectomy is often performed if the blood supply of the appendix is compromised. A

cecopexy is not necessary. Risk of recurrence of the intussusception after operative reduction is less than 5%.

If manual reduction is not possible or perforation is present, perform a segmental resection with an end-to-end anastomosis. A diligent search for any lead points is warranted, especially if the patient is older than 2-3 years.

Laparoscopy has been added to the surgical armamentarium in the treatment of intussusception. Laparoscopy can be performed in all cases of intussusception. Reduction of the intussusception, confirmation of radiologic reduction, and detection of lead points have all been reported.

Laparoscopy is associated with faster recovery times, decreased length of stay, decreased time to full feeds, and lower requirements of pain medication. (See the image below.)



Laparoscopic view of a jejuno-jejunal intussusception

MEDICATION SUMMARY

Drug therapy is not currently a component of the standard of care for intussusception. Medications are limited to those used for pain control after surgery. In the immediate postoperative period, weight-adjusted intravenous morphine is usually administered. As the oral diet is resumed, acetaminophen with codeine or ibuprofen is given orally.

Patients with HSP or hemophilia and intussusception require standard therapy for the individual disease. Some investigators have advocated the use of steroids in intussusception secondary to HSP and lymphoid hyperplasia, with varied results

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