

EXPERIENTIAL HIGHER INSTITUTE OF SCIENCE AND TECHNOLOGY (EXHIST)
COURSE TITLE: NURSING MANAGEMENT OF PATIENTS WITH DISORDERS OF
DIGESTIVE SYSTEM

LEVEL: 300

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Course Outline

Module 1: Review of Anatomy and Physiology of the GI System

- Overview of the digestive system
- Structure and function of accessory and primary organs of digestion
- Physiological processes of digestion and absorption

Module 2: Nursing Assessment – History and Physical Assessment

- Physical assessment techniques specific to GI disorders
- Diagnostic tests for GI assessment

Module 3: Common GI Disorders

- Oral cavity disorders: gums, and teeth
- GI bleeding, infections, inflammation, tumors
- Obstruction, perforation, and peritonitis

Peptic and duodenal ulcers

- Malabsorption disorders
- Appendicitis and hernias
- Hemorrhoids, fissures, and fistulas

Module 4: Pancreatic Disorders

- Pancreatitis: inflammation, cysts, and tumors
- Assessment and nursing management

Module 5: Liver Disorders

- Liver inflammation and abscess
- Cirrhosis and portal hypertension
- Hepatic failure

Module 6: Gallbladder Disorders

- Cholecystitis: inflammation of the gallbladder
- Cholelithiasis: gallstones

Module 7: Gastric Decompression and Lavage

- Indications for gastric decompression
- Enteral feeding techniques
- Stoma care for patients with feeding tubes

Module 8: Alternative Therapies and Complementary Approaches

Module 2. Nursing Assessment

1.1. History of present illness and review of systems

Bowel habits

- Frequency, quantity, colour and consistency of stool – Presence of blood, mucus
- Pain before, during or after defecation

Abdominal pain

- Location – Frequency – Duration
- Character (for example, crampy or constant, sharp or stabbing
- Radiation – Onset (sudden or gradual
- Progression from onset – Aggravating and relieving factors – Associated symptoms

Vomiting

- Frequency – Volume – Colour – Hematemesis – Relationship to food intake

Other characteristics and symptoms

- Fever – Growth history – Dysphagia – Appetit
- Food and fluid intake since onset of illness – Usual nutrition and food habits: type of foods eaten, variety of foods in diet, quantity of food eaten, dietary balance, fibre content of diet – Weight loss or weight gain – Colour (for example, presence of jaundice) – Skin (for example, pruritus, rash) – Activity level – History of previous GI diseases or abdominal surgery – Medications (for example, iron)
- Allergies, especially known allergies to food (for example, lactose intolerance)

1.2. Physical examination

General appearance

- Apparent state of health – Appearance of comfort or distress
- Position of child and presence of guarding (child's behavior can also give very good clues as to the severity of any abdominal pain) – Colour (for example, flushed, pale, jaundiced) – Nutritional status (obese or emaciated) – State of hydration (skin turgor, mucous membrane moisture)

Vital signs

- Temperature may be elevated in infection – Blood pressure usually normal
- Tachycardia or bradycardia may be present – Respiratory rate
- Obtain weight, height and head circumference when possible and/or applicable

Inspection

– Size, shape and contour; note any distension or asymmetry (in infancy, abdomen is typically protuberant; in early childhood the abdomen is still protuberant, but flattens when the child is lying down).

Auscultation

Auscultation, to listen for bowel sounds, should be done before palpation. Listen for quality and quantity of bowel sounds.

An increase in bowel sounds alone is not significant, because this can occur with anxiety or mild gastroenteritis. However, it may also be a sign of obstruction.

Percussion

- General percussion in all four quadrants for normal tympany
- Delineate span of liver; upper border is in the mid-clavicular line, between the fourth and sixth intercostal spaces
- Determine spleen size
- Shifting dullness to assess for ascites in the abdomen. There will be dullness to percussion on the dependent side when the patient is in a side-lying position; the border of the percussion note will change to a new more lateral position several moments after the patient assumes a supine recumbent position

Palpation

Ideally, palpation is performed with the patient lying supine, with hands by the sides and relaxed. Be sure your hands are warm. Examine all four quadrants in succession. If there is pain, start with the painless areas, and palpate the painful area last. Palpation should be light at first, with progression to deep palpation by the end of the examination.

Light Palpation

- Assess for tenderness, guarding, superficial masses

Deep Palpation

- Feel for organs (liver, spleen, bladder and kidneys) and masses
- Assess for rebound tenderness (pain that occurs upon suddenly releasing the hand after deep palpation)
- Assess for referred tenderness (pain that is felt in an area distant to the area being palpated), which can be a clue to the location of the underlying disease

Rectal examination

- Anal patency (check this feature only in newborns)

- Skin tags
- Sphincter tone
- Fissures
- Tenderness
- Masses
- Observe for frank blood or melena

1.3. Diagnostic Tests

1. Endoscopy: This procedure involves inserting a flexible tube with a camera through the mouth to visualize the esophagus, stomach, and the upper part of the small intestine (duodenum). It is used to diagnose conditions like gastroesophageal reflux disease (GERD), ulcers, and Barrett's esophagus.

Colonoscopy: A colonoscope is used to examine the rectum and the entire colon. This test is valuable for detecting colorectal cancer, polyps, and IBD.

Sigmoidoscopy: Similar to colonoscopy but focuses on the rectum and the lower part of the colon.

2. Capsule Endoscopy:

A small, pill-sized camera (capsule) is swallowed, allowing for the visualization of the small intestine. It is helpful in diagnosing conditions such as Crohn's disease, celiac disease, and small bowel tumors.

3. Upper GI Series (Barium Swallow) and Lower GI Series: These radiographic studies involve the use of barium contrast to visualize the upper (esophagus and stomach) and lower (colon) GI tract. They are used to diagnose structural abnormalities, such as strictures, ulcers, and obstructions.

4. Abdominal Ultrasound: Ultrasound imaging is used to assess the liver, gallbladder, pancreas, and other abdominal organs for abnormalities, including gallstones, liver tumors, and pancreatic cysts.

5. CT Scan (Computed Tomography): A CT scan provides detailed cross-sectional images of the abdomen, allowing for the evaluation of various GI conditions, including tumors, abscesses, and inflammatory bowel diseases.

6. MRI (Magnetic Resonance Imaging): MRI can be used to assess the abdominal and pelvic organs, providing high-resolution images that are useful for diagnosing conditions like liver and pancreatic tumors.

7. X-rays: X-rays may be used to detect foreign objects, assess abdominal gas patterns, and evaluate the presence of obstructions or perforations.

8. Stool Tests: Fecal occult blood test (FOBT) checks for the presence of blood in the stool, which may indicate GI bleeding. Stool culture tests can identify bacterial, parasitic, or viral infections in the GI tract.

9. Blood Tests: Blood tests, such as liver function tests, complete blood count (CBC), and serologic tests, can provide valuable information about GI conditions, including liver disease, anemia, and celiac disease.

10. Breath Tests: Hydrogen breath tests are used to diagnose conditions like lactose intolerance and bacterial overgrowth in the small intestine.

11. H. pylori testing: Tests, such as a breath test or stool antigen test, can identify the presence of *Helicobacter pylori*, a bacterium associated with peptic ulcers and gastritis.

13. Biopsy: Tissue samples may be obtained through endoscopy or surgery and examined under a microscope to diagnose conditions like cancer or inflammatory disorders.

Module 3. COMMON GI DISORDERS

3.1. ORAL CAVITY-LIPS, GUMS AND TEETH

The oral cavity includes the gingival or gums, the buccal mucosa (the lining of the inside of the mouth), the area under the tongue, the hard palate (roof of the mouth), the area behind the wisdom teeth, and the front two-thirds of the tongue.

There are a number of infections that affect the oral cavity. Their impact can range from annoying symptoms that are fairly easily resolved to major problems that can have long-ranging effects.

3.1.1. STOMATITIS

Stomatitis is defined as an inflammation of the oral mucosa that may spread to the lips, palate and buccal mucosa. It is a common infection and generally classified as **acute herpetic stomatitis** or **aphthous stomatitis**.

Acute herpetic stomatitis is caused by infection of the **herpes simplex virus** and is common in children aged 1 to 3 years. It is usually self-limiting but can be severe, and even fatal, in neonates.

Symptoms

Symptoms have an abrupt onset and include malaise, anorexia, irritability, mouth pain and fever, which may last for one to two weeks. The patient's gums are swollen, bleed easily, and the mucous membrane is painful.

Ulcers develop in the mouth and throat that eventually acquire the appearance of "punched-out" lesions with red areolae. Pain usually ceases about two to four days

before the ulcers are completely healed.

Note that if a child with stomatitis sucks his thumb, the lesions might spread to the hand.

Treatment

Treatment is focused on symptom relief. Salt-water mouth rinses, topical antihistamines, antacids or corticosteroids may be used to reduce discomfort. Bland or liquid diets may be recommended to reduce the discomfort of eating. In extreme cases, when the patient cannot ingest adequate amounts of food and/or liquids, intravenous therapy may be indicated.

3.1.2. Candidiasis (thrush)

This is a fungal infection that causes cream or bluish-white patches of exudates to appear on the tongue, mouth, and/or pharynx. Persons at high risk include premature neonates, older adults, those with suppressed immune systems, persons taking antibiotics, or persons taking steroids for a long period of time.

For infants, the oral mucosa is swabbed with nystatin after feedings because feedings wash away the medication. The mother should also be treated to avoid passing the infection back and forth.

Older children and adults swish nystatin solutions around the mouth for a few minutes before swallowing it. Eating yogurt with active cultures may be helpful in treating the infection.

3.1.3. Gingivitis

It is an inflammation of the gums. This condition can be an early warning sign of diabetes, blood dyscrasias and lack of vitamins. Gingivitis is occasionally due to the use of hormonal contraceptives. More often, it is due to poor oral hygiene, ill fitting dentures or other irritants.

Signs include redness, painless swelling, bleeding and evidence of gum detachment from teeth.

Treatment includes elimination of triggering factors (e.g improving the fit of dentures), regular dental check-ups, and good oral hygiene. Sometimes oral or topical corticosteroids are recommended.

3.1.4. Glossitis

Inflammation of the tongue. It can be due to bacterial infection, irritation or injury, or a lack of vitamin B. Spicy foods, smoking and alcohol intake may also promote its

development.

The tongue becomes red, ulcerated or swollen to the point that the airway is obstructed. Swallowing is difficult and painful, as is chewing. Speech is impaired. Treatment focuses on eliminating the underlying cause. Regular dental checkups are important, and good oral hygiene should be encouraged.

3.1.5. Periodontitis

This is an inflammation of the gums accompanied by recession and loosening of the teeth. Often caused by poor dental hygiene, it is sometimes related to the use of hormonal contraceptives and may also be an early sign of diabetes, blood dyscrasias or vitamin insufficiency. Onset is abrupt.

Signs include bright red swollen gums and loosening of the teeth. There may be evidence of systemic infection, such as the presence of fever and chills.

Treatment includes initiating and maintaining good oral hygiene, participating in regular dental check-ups, and, if necessary, surgery to remove infected areas and prevent recurrence.

3.2. GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux disease (GERD), often referred to as “**heartburn**,” is the backflow of gastric and/or duodenal contents into the esophagus past the lower esophageal sphincter (LES). This backflow is not accompanied by belching or vomiting. Many people, even health care professionals, sometimes dismiss “heartburn” as a minor concern. However, ongoing reflux may cause inflammation of the esophageal mucosa and complications such as esophageal ulcers, strictures, or Barrett’s esophagus

Pathophysiology/etiology

Under normal conditions, the LES sustains enough pressure around the lower end of the esophagus to close it, thus preventing reflux of gastric and/or duodenal contents. The sphincter relaxes after every swallow to permit food to pass into the stomach. In GERD, the sphincter does not remain closed. This is generally due to inadequate LES pressure or if pressure in the stomach propels gastric contents into the esophagus.

Stomach contents are very acidic, thus causing pain and irritation when they move into the esophagus. The esophageal mucosa becomes inflamed, which can decrease

LES pressure more and more until there is a recurrent cycle of reflux and heartburn.

Risk factors

- Pyloric surgery.
- Nasogastric (NG) tube intubation for more than four days
- Hiatal hernia.
- Conditions that increase abdominal pressure, such as pregnancy, obesity, persistent vomiting or coughing.
- Medications such as calcium channel blockers, morphine, diazepam, anticholinergics and meperidine.
- Alcohol.
- Cigarettes.
- Orange juice.
- Tomatoes.
- Whole milk.
- Peppermint.
- Chocolate.

Signs and symptoms

- The most common presenting symptom of GERD is burning pain in the epigastric area, commonly referred to as “heartburn.” The pain may radiate to the arms and chest and even to the neck and jaw.
- Some patients may think that they are having a heart attack.
- The pain often occurs after a meal or when lying down.
- Patients may complain of feelings of fluid accumulation in the throat.
- A chronic cough may result from the reflux of gastric contents into the throat.
- There may also be hoarseness upon awakening in the morning.

Diagnosis

Diagnostic tests focus on identifying the underlying cause of GERD.

In addition to a careful history and physical, a number of specific tests are conducted.

- **Esophageal acidity test:** checks the competence of the LES and measures reflux.
- **Acid perfusion test:** Determines whether reflux is the cause of the problem and distinguishes it from cardiac problems.
- **Esophagoscopy**
- **Barium swallow:** Used to identify hiatal hernia as a causative factor or esophageal stricture as a complication of GERD.

Treatment

The goals of treatment are: • To control symptoms. • To promote healing of the esophagus and • To prevent or manage complications.

Medical therapy

- **Antacids** to neutralize acidic gastric contents.
- **Foaming agents** (such as Gaviscon) that prevent reflux.
- **H2 blockers** that reduce the amount of gastric acid production.
- **Protein pump inhibitors (PPIs)** limit gastric acid secretion and facilitate rapid resolution of symptoms and esophageal healing in 80-90 percent of patients. Examples of PPIs include Prilosec, Nexium and Kapidex.

- **Bard EndoCinch system:** An endoscopic device that puts stitches in the LES to make it stronger. This device is inserted for chronic GERD.

Symptom control focuses primarily on lifestyle modifications.

- **Dietary habits:** Avoid foods that trigger GERD symptoms, such as caffeine products, beans, chocolate, spicy food, carbonated beverages, orange juice, alcohol, onions, fatty foods, tomato juice and tomato sauce.

Avoid eating large meals, which put pressure on the LES

- **Positioning:** Patients should sit upright and not lie down for up to 2 hours after eating. Sleep with the head of the bed raised 6 to 8 inches.

- **Weight control - Avoid Smoking - Limit/avoid alcohol consumption**

- **Stress:** Stress itself does not cause GERD. However, it can cause individuals to drink, smoke or overeat, which can trigger GERD symptoms.

- **Medication:** Patients need to be taught about the medications they take, the importance of taking them, and any medication side effects that may occur.

Lifestyle modifications can promote healing of the esophagus and control symptoms.

Complications

Barrett's esophagus.

Barrett's esophagus is a disorder characterized by the replacement of "normal" tissue that lines the esophagus with tissue similar to the lining of the intestine. This is referred to as **intestinal metaplasia**. It does not produce signs or symptoms. Its cause is unknown, but it is often found in persons with GERD.

Nursing Care

3.3. GASTRITIS

Gastritis is a common condition characterized by inflammation of the lining of the stomach. It may be acute or chronic. In most people, gastritis is not severe. However, some people may develop gastric ulcers and an increased the risk for stomach

cancer. **Acute gastritis** has an abrupt onset and causes reddening of the gastric mucosa, edema, hemorrhage and erosion.

Chronic gastritis is generally common in the elderly and in people who have pernicious anemia, although either acute or chronic gastritis can occur at any age. In chronic gastritis, all stomach mucosal layers to become inflamed.

Pathophysiology and etiology

When the stomach's protective layer is weakened or injured, gastric digestive juices penetrate the stomach and damage the gastric lining, leading to inflammation. The factors that predispose to this include;

- Bacterial infection with *Helicobacter pylori* (*H. pylori*)
- Drug use: Regular use of drugs such as aspirin and NSAIDs can cause gastritis.
- Alcohol ● Smoking ● Old age ● Bile reflux disease: ● Autoimmune gastritis

Signs and symptoms

Signs and symptoms of acute gastritis are abrupt in onset and include: indigestion, abdominal cramps, nausea, vomiting and hematemesis. These signs and symptoms can last from a few hours to several days.

Chronic gastritis may produce similar signs and symptoms or may cause only mild discomfort such as slight pain or an inability to tolerate fatty or spicy foods.

Diagnosis

- **Upper gastrointestinal endoscopy:** This allows visualization of the stomach lining and removal of tissue samples for biopsy.
- **Tests for occult blood:** Stools and vomitus can be tested for the presence of blood, indicating gastric bleeding.
- **Blood tests:** Hemoglobin and hematocrit levels are assessed to determine whether the patient has developed anemia from gastric bleeding.
- Test for the *H-pylori*

Treatment

Treatment focuses on eliminating the cause.

- **Antibiotics** to treat bacterial gastritis (*H pylori*).
- **Histamine-2 receptor** antagonists to block gastric secretions.
- **Antacids** to buffer the effects of the acidity of gastric secretions.
- **Acid blockers** such as cimetidine, ranitidine (Zantac) or famotidine (Pepcid) may be prescribed if antacids do not work.

- In case of severe blood loss, blood replacement may be necessary.
- Lifestyle changes may also be helpful in reducing the signs and symptoms.
 - **Avoid** foods that trigger or exacerbate signs and symptoms.
 - **Maintain** a healthy weight. Digestive problems are common in people who are overweight.
 - Exercise to help all body systems to function at their highest potential

Nursing care

3.4. GASTROENTERITIS

Gastroenteritis is generally a self-limiting illness characterized by irritation and inflammation of the stomach and intestines, which produces nausea, vomiting, diarrhea and abdominal cramping. Children and the elderly are especially susceptible to complications of this disease as are people who are in a debilitated state. The very young and the elderly patients can quickly become severely dehydrated and also experience electrolyte imbalances as a result of vomiting and diarrhea.

Gastroenteritis affects all age groups and is commonly referred to as intestinal flu, traveler's diarrhea or viral enteritis. It is the fifth leading cause of death in young children.

Causes

- **Bacteria** ● **Amebae**: ● **Viruses** ● **Food allergies**
- **Medication reactions**: Adverse reactions to drugs such as antibiotics may trigger gastroenteritis.
- **Ingestion of poisons or heavy metals**: Poisonous substances and water contaminated with heavy metals such as lead and mercury can trigger gastroenteritis.

Symptoms

Signs and symptoms vary depending on the cause, age and general health of the person affected. Typical symptoms include:

- Low grade fever
- Nausea
- Vomiting.
- Diarrhea.
- Malaise.

NB: The nurse should remain alert to the signs and symptoms of dehydration and electrolyte imbalance, especially in children, the elderly and debilitated patients.

Signs and symptoms of dehydration include:

- Thirst.
- Poor skin turgor.
- Dry skin.
- Fever.
- Dizziness.
- Weakness.
- Elevated heart rate.
- Decreased blood pressure.
- Changes in mental status.
- Seizures.
- Coma.

Common electrolyte imbalances that can occur with severe vomiting and diarrhea are **hypernatremia** and **hypokalemia**. Electrolyte imbalances are more common in children and the elderly.

Symptoms of hypernatremia are:

- Low-grade fever
- Flushed skin.
- Weakness
- Lethargy
- Tremors
- Confusion
- Seizures
- Coma

Symptoms of hypokalemia;

- Weakness, especially in the legs
- Leg cramps
- Arrhythmias
- Rapid heart rate

Medical and Nursing management

The disease is usually self-limiting with complete recovery. However, in severe cases, it may be necessary to seek emergency medical attention.

- IV fluids and antibiotics may be necessary.
- Patients need rest and nutritional support. - Lost fluid and electrolytes can be replaced with broth, ginger ale, ice chips and lemonade as tolerated.
- Milk and milk products should be avoided because they may exacerbate vomiting and diarrhea.
- Teach Patients about preventive measures to reduce the risk of gastroenteritis.

3.5. INTESTINAL OBSTRUCTION

Intestinal obstruction is a complication of various GI disorders and. It can be partial or complete blockage of the lumen in either the small or large intestine. The site in 90% of patients is the small bowel, which is usually more serious than large bowel obstruction. Untreated complete obstruction, regardless of location, can be fatal within hours from shock and vascular collapse.

Intestinal obstruction can be classified in three categories:

- 1. Simple:** Intestinal contents cannot move through the bowel, but there are no complications or blood flow alterations.
- 2. Strangulated:** Blood supply is cut off to the affected section of bowel. This may be partial or complete.
- 3. Close-looped:** both ends of a portion of the bowel are blocked, thus isolating it from the rest of the intestine.

Obstruction of the intestine causes GI secretions, gas and swallowed air to accumulate near the location of the obstruction. Peristalsis increases above and below the obstruction as the bowel tries to force its contents through the blockage. This damages the intestinal mucosa and leads to distention at and above the area of blockage. Distention interferes with venous blood flow and blocks normal absorption. The inability to absorb causes the bowel to secrete water, sodium and potassium into fluids collecting in the lumen.

Small bowel obstruction leads to metabolic alkalosis due to dehydration and loss of acidic gastric contents (gastric hydrochloric acid). Obstruction in the lower bowel causes a loss in alkaline fluids leading to metabolic acidosis.

Causes: The leading cause of small bowel obstruction is adhesions followed by malignancy, Crohn's disease and hernias.

- Large bowel obstruction is generally due to malignancy.
- Mechanical obstruction is due to blockage from impacted stool, foreign objects, such as gallstones or fruit pits; bowel wall compression due to intussusception; or tumors.
- Non-mechanical obstruction is due to physiologic alterations such as electrolyte imbalances, the effects of medications that slow peristalsis (e.g. opioids), thrombosis or paralytic ileus.

Signs and symptoms

Signs and symptoms can begin with pain, nausea and vomiting and, if untreated, progress to shock, sepsis and death. They can vary depending on the location of the obstruction and whether it is partial or complete.

- **Small intestine obstruction:** Nausea, vomiting, constipation, abdominal distention and colicky pain.

- Extreme thirst, malaise, and dry oral mucous membranes and tongue may develop.
- Bowel sounds heard upon auscultation may be quite loud, and may even be heard without the use of a stethoscope.
- Pain upon abdominal palpation and rebound tenderness if bowel strangulation.
- Untreated obstruction may cause hypovolemic shock.
- If the small bowel is completely obstructed, extreme peristalsis may develop and bowel contents may be moved toward the mouth, and vomitus contains gastric juice, then bile, and eventually, the contents of the ileum.

● **Large intestine obstruction:** Symptoms progress more slowly because the large bowel can absorb fluid and distend well beyond its typical size.

- Constipation may be the only sign at the onset. - Then colicky abdominal pain may develop quite swiftly, producing frequent spasms. - There is significant abdominal distention. - Eventually, vomiting occurs (vomitus may contain fecal matter)
- Continuous abdominal pain develops, and peritonitis may occur.

If the obstruction is partial, these symptoms can occur in a less severe form

Diagnosis

- History and physical examinations
- Lab results may show decreased sodium, chloride and potassium levels due to vomiting and an elevated white blood cell count due to infection (peritonitis).
- Diagnostic tests such as CT scans, barium enema, upper GI and small-bowel series, and abdominal films are conducted.
- Small bowel obstruction findings include alternating levels of fluid and gas, often referred to as a “**stepladder**” pattern.
- Large bowel obstruction findings include a distended, air-filled colon.

Treatment

- Immediate treatment includes fluid resuscitation, correction of electrolyte imbalances and administration of analgesics and anti-emetics as indicated.
- Bowel decompression is accomplished by the insertion of a nasogastric tube to suction GI contents and avoid aspiration.
- Antibiotics that are effective against gram-negative and anaerobic organisms.
- Strangulated obstruction requires blood transfusions. Patients must be monitored closely for evidence of shock indicated by pallor, tachycardia and hypotension.
- Surgical interventions may include removal of tumors and other masses, hernia repair or bowel resection.

- For large bowel obstruction, surgical resection with anastomosis, colostomy or ileostomy may be necessary.

Nursing Care

- **Monitor closely for signs and symptoms of metabolic alkalosis:** Changes in levels of consciousness, tetany, twitching, shallow respirations, cardiac arrhythmias and confusion.
- **Monitor for signs and symptoms of metabolic acidosis:** Headache, lethargy, deep, rapid respirations (Kussmaul's respirations), hypotension, anorexia, stupor.
- **Monitor input and output closely:** Record amount and color of NG tube drainage.
- **Monitor for signs of dehydration:** Thick, swollen tongue, dry oral mucous membranes and poor skin turgor.

3.6. GI Inflammation

Ulcerative Colitis and Crohn's disease are both types of inflammatory bowel disease. However, they affect different portions of the GI tract. Ulcerative colitis is confined to the colon, while Crohn's disease can affect the entire GI tract.

Symptoms may include abdominal pain, diarrhea, rectal bleeding, and weight loss.

Diagnosis involves endoscopy, colonoscopy, imaging, and laboratory tests.

Treatment aims to reduce inflammation and may include medications (anti-inflammatory drugs, immunosuppressants), lifestyle modifications, and, in some cases, surgery.

In order to properly diagnose and treat inflammatory bowel disease, it is important to determine the exact nature of the disease affecting the bowel.

3.7. Peptic & Duodenal ulcers

Peptic ulcer disease (PUD) is defined as an erosion of the GI mucosa.

Peptic ulcers are circumscribed lesions that can develop in the lower esophagus, stomach, pylorus, the duodenum or the jejunum. Approximately 80 percent of all peptic ulcers are located in the duodenum.

Gastric ulcers develop most often in middle-aged and elderly men, particularly those who are chronic users of nonsteroidal antiinflammatory drugs (NSAIDs), alcohol or tobacco.

Pathophysiology

Under normal circumstances, a thick layer of mucus protects the stomach from chemical trauma, mechanical trauma and auto digestion. This mucus acts as a barrier that prevents harmful effects of gastric acid and digestive enzymes.

Prostaglandins also provide a measure of gastric protection. When the gastric mucosal barrier is damaged or destroyed, gastric ulcers can develop.

The duodenum is protected by a mucoid alkaline secretion of the Brunner's glands.

This secretion neutralizes the acid chyme. When there is an excessive production of acid that cannot be neutralized, duodenal ulcers can develop.

Causes

There are three main causes of PUD.

1. Infection with *Helicobacter pylori* (*H. pylori*)
2. The use of NSAIDs
3. Effects of illnesses like pancreatitis, hepatic disease, Crohn's disease, pre-existing gastritis and Zollinger Ellison syndrome.

Predisposing factors for PUD.

- Physical trauma, emotional stress and aging.
- Irritants such as alcohol, coffee and tobacco.

Signs and symptoms

- Heartburn and indigestion. - Eating may both cause and relieve pain.
- If eating causes pain, there may be weight loss.
- In severe cases, there may be significant GI bleeding.
- Duodenal ulcers also cause heartburn and localized mid-epigastric pain. Eating relieves this pain. Weight gain is likely because patients eat to diminish the pain.
- An unusual sensation of hot water bubbling in the back of the throat is characteristic of duodenal ulcers.
- Duodenal ulcer attacks usually take place about two hours after meals or whenever the stomach is empty.
- Attacks may also occur after ingesting orange juice, aspirin or alcohol.

Complications

N.B: Ulcers can penetrate the pancreas and cause serious back pain. Perforation of the ulcer may occur, causing abrupt, severe pain, a rigid abdomen, grunting respirations and diminished bowel sounds.

Bleeding that can lead to hypovolemic shock. Bloody vomitus or stools, low

hemoglobin and hematocrit levels, tachycardia and hypotension may signal this serious complication.

Diagnosis.

- Esophagogastroduodenoscopy
- Barium swallow, upper GI series, and small bowel series
- Hemocult test
- White blood cell count
- Hematocrit and hemoglobin
- H. pylori antibody assay

Management

Treatment focuses on destruction of H. pylori and protecting the lining of the stomach. Experts in the treatment of PUD recommend treatment with two antibiotics (e.g., tetracycline, bismuth subsalicylate or metronidazole) to eliminate H. pylori.

Additional treatment measures to heal and protect the lining of the stomach include:

- Administration of proton gastric acid pump inhibitors to decrease secretion of gastric acid.
- Administration of a prostaglandin analog such as misoprostol.
- Administration of histamine-2 blockers to reduce gastric acid secretion and anticholinergics to inhibit the action of acid-secreting cells.
- Patient education.

N.B: In case of severe GI bleeding, emergency measures must be initiated.

Nursing care

3.8. CELIAC DISEASE

Celiac disease (also referred to as idiopathic steatorrhea, nontropical sprue, gluten enteropathy and celiac sprue) is a disease that damages the small intestine. It is characterized by an inability to properly absorb food and an intolerance of gluten, a protein found in wheat, wheat products, rye and barley. Gluten is found in a wide variety of foods but may also be found in everyday items such as vitamins, lip balms and some medicines.

Celiac is a disease of malabsorption and an immune reaction to gluten. When gluten is ingested, there is injury to the villi in the upper portion of the small intestine. This leads to a reduction in surface area and malabsorption of most nutrients.

Under normal conditions, villi allow absorption of nutrients from food into the bloodstream. If villi are not healthy, nutrients cannot be absorbed. This causes the patient to become malnourished, no matter how much food is eaten.

Cause

It is caused by an intramucosal enzyme defect that prevents the body from digesting gluten. Tissue toxicity occurs, resulting in swift turnover of cells, increased number of epithelial lymphocytes and damage to the surface epithelium of the small bowel. The disease affects women and girls twice as often as men and boys. There is a strong genetic association among persons who have the disease.

Complications

- Anemia secondary to malabsorption
- Syncope, angina and heart failure due to anemia
- Bleeding disorders due to vitamin K deficiency
- Intestinal lymphoma

Signs and symptoms

The disease affects people in different ways relative to the extent of damage to the small intestine, a person's age and the length of time the patient has had symptoms without being diagnosed and treated.

- Damage to the small intestine may cause cramping, recurrent diarrhea, abdominal distention, weakness and an increase in appetite without weight gain.
- Anemia due to malabsorption of folate, iron, and vitamin B12.
- Loss of calories, fat-soluble vitamins(A,D,K), calcium, minerals, electrolytes and malabsorption of fat, carbohydrates and protein.
- Osteomalacia, osteoporosis, tetany and bone pain due to calcium loss and vitamin D deficiency.
- Paresthesia, seizures, peripheral neuropathy.
- Dry skin, eczema, psoriasis, dermatitis, brittle nails.
- Amenorrhea, hypometabolism and adrenocortical insufficiency.
- Irritability, lethargy, and mood changes.

Diagnosis

- **Esophagogastroduodenoscopy:** Histologic changes seen include a mosaic like pattern of alternating flat and bumpy areas, a nearly total absence of villi, and an irregular, blunt, unorganized network of blood vessels confirm diagnosis.
- **Blood studies:** Elevated alkaline phosphatase levels, low cholesterol and albumin levels, slightly elevated liver enzymes, abnormal blood clotting and anemia.
- **Antibody blood tests:** High levels of anti-tissue transglutaminase (+TGA) antibody or anti-endomysium antibodies (EMA) are indicative of the disease.

Treatment

The only treatment for celiac disease is implementation of a gluten free diet for the remainder of the patient's life. Patients usually notice improvement within days of eliminating gluten from their diets. The small intestine heals within three to six months in children but may take several years in adults.

Here are some tips for patients as they adapt to their new way of eating, which must continue for the rest of their lives!

- Read all food labels carefully! Many foods contain gluten in varying amounts.
- Ask pharmacists whether prescribed medications contain wheat or other gluten products.
- Some non-food products, such as lipstick, may contain gluten as an additive. Find out about the contents of any product that can be ingested through the GI tract! If ingredients are not provided on the label or package insert, contact the manufacturer for a product list.
- When eating out, ask the waiter if a gluten-free menu is available.

Nursing Care and nursing diagnosis

3.9. HERNIAS

A hernia occurs when an organ or tissue pushes through a weak spot or opening in the muscles or connective tissue that surrounds it. Hernias can develop in various parts of the body and can include:

- **Inguinal Hernias:** An inguinal hernia occurs when intra-abdominal fat or a portion of the small intestine protrudes through a weakened area in the lower abdominal wall. This type of hernia is located in the groin because the fat or the intestine moves through a weak area at the inguinal ring, which is the opening to the inguinal canal. An inguinal hernia appears as a bulge on one or both sides of the groin.
- **Hiatal hernia**, also referred to as **hiatus hernia**, is a deficiency or defect in the diaphragm that allows part of the stomach to pass through the opening in the diaphragm into the chest.
- **Femoral Hernia:** Develops when a fatty deposit within the femoral canal grows and creates an opening large enough for part of the peritoneum and bladder to move through.

- **Incisional Hernia:** Occurs at the site of a previous surgery due to a weakness in the abdominal wall, inadequate wound healing or an infection.

- **Umbilical Hernia:** Common in neonates, but also occurs in obese women, or in women who have had a number of pregnancies. It is due to abnormal muscular structure around the umbilical area.

Signs and symptoms of Hernias

Hernias typically present as a visible bulge or swelling at the site of the hernia. They may be painful or cause discomfort, especially when lifting, coughing, or straining. They are usually reduced (manipulated back into place) without much difficulty. However, the hernia may become incarcerated due to adhesions that interfere with intestinal flow.

- A lump or bulge over the herniated area when the patient is in a standing position or strains. The lump or bulge disappears when the patient is lying down.
- Sharp, steady pain that could be exacerbated with lifting, straining or exercising, and is relieved with rest or being in the supine position.
- Feelings of weakness or pressure in the groin for inguinal hernias

Strangulation of the hernia can lead to partial or complete bowel obstruction. It produces the following signs and symptoms:

- | | |
|--------------------------------------|--------------------------------------|
| • Severe pain. | • Vomiting. |
| • Redness in the area of the hernia. | • Infection. |
| • Fever. | • Absent or diminished bowel sounds. |
| • Tachycardia. | • Bloody stools. |
| • Anorexia. | • Shock. |

N.B: An **incarcerated** hernia is one that becomes trapped and is unable to be **reduced** (massaged back into the normal anatomical position). An incarcerated hernia can become **strangulated**, meaning there is serious interference with normal blood flow to the incarcerated area. This can lead to obstruction and necrosis. A strangulated hernia is a surgical emergency

Diagnosis

- Physical examination and Imaging studies

Treatment

- The treatment of choice is **herniorrhaphy**, which is a surgical procedure during which the contents of the hernia sac are replaced into the abdominal cavity. The

procedure is often performed under local anesthesia.

- Another surgical option is **hernioplasty**, during which the weakened area is reinforced or supported with steel mesh, wire or fascia.
- Patients who delay surgery should be cautioned about the potential complications of the disorder and taught to recognize the signs and symptoms of strangulation and bowel obstruction.
- After surgery, patients should be taught how to recognize signs and symptoms of infection and to avoid lifting heavy objects or straining during bowel movements.

Nursing Care and Nursing Diagnosis

3.10. APPENDICITIS

Appendicitis is inflammation of the appendix and is the most common disease that requires emergency surgery. It can occur at any age but has a peak incidence in late teens and early 20s.

Causes

Appendicitis is due to obstruction of the appendiceal lumen (inside of the appendix) that could be caused by viral infection, stricture, fecal mass or ingestion of barium.

Signs and symptoms

- Abdominal pain that can be localized to the right lower quadrant (at the McBurney's point) or generalized over the abdomen.
- Rebound tenderness is common.
- Anorexia
- Nausea or vomiting
- Low-grade fever
- Elevated WBC count with an increased number of immature cells.

Diagnosis

Patient history and physical examination

CT scan or abdominal echography may be used to visualize the appendix.

N.B: Abrupt termination of abdominal pain suggests perforation of the appendix.

Treatment

The only treatment for appendicitis is removal of the appendix or appendectomy. Laparoscopic appendectomies reduce recovery time and enhance the speed of recovery.

Caution! An ice bag may be used for pain relief prior to surgery. NEVER apply heat

to the right lower abdominal quadrant because heat may rupture the appendix. Never give enemas or laxatives because these may also rupture the appendix.

Nursing Care and Nursing diagnosis

3.11. PERITONITIS

Peritonitis is an inflammation of the peritoneum (the membrane that lines the abdominal cavity and covers the internal abdominal organs. Peritonitis can be acute or chronic.

Causes

Under normal conditions, the peritoneum is sterile. Peritonitis occurs when bacteria enter the peritoneum as a result of appendicitis, diverticulitis, peptic ulcer, ulcerative colitis, abdominal cancers, strangulated obstruction or stab wounds.

Chemical inflammation may also cause peritonitis. Chemical inflammation occurs with events such as perforation of a gastric ulcer or rupture of a fallopian tube. At times, it may be associated with peritoneal dialysis.

Signs and symptoms

- Abrupt, severe and widespread abdominal pain. This pain generally intensifies and localizes in the area of the case (e.g., right lower quadrant with appendicitis) with accompanying rebound tenderness.
- Excessive diaphoresis • Weakness. • Pallor • Cold skin • Hypotension
- Tachycardia • hyperthermia • Distended abdomen. • Decreased intestinal motility
- With accompanying intestinal obstruction, there will be nausea, vomiting and rigidity of the abdomen.

Diagnosis

In addition to patient history, the following are used to identify peritonitis:

- **Abdominal x-rays:** Reveal distension of the small and large intestine due to gas and edema; if internal organs are perforated, air may be noted underneath the diaphragm.
- **Chest x-ray:** May reveal elevated diaphragm.
- **Blood studies:** Reveal leukocytosis.
- **Paracentesis:** Shows the presence of bacteria, blood, pus or urine.

- **Laparotomy:** to determine the underlying cause of the peritonitis.

Treatment

Antibiotic therapy, dependent upon the organisms causing the infection, is initiated.

Patients are not to receive food or drink by mouth.

Fluids and electrolytes are administered intravenously.

Supportive measures include administration of analgesics, insertion of a nasogastric (NG) tube for bowel decompression, and if needed, a rectal tube to promote the passage of flatus.

If peritonitis is due to a perforation (e.g., a perforated fallopian tube, or a ruptured appendix) surgery must be performed as soon as possible.

The goals of surgery are to remove the contents spilled into the peritoneum from the perforation and to insert drains to facilitate this removal.

Nursing Care and nursing diagnosis

HEMORRHOIDS, FISSURES AND FISTULAS

3.12. Hemorrhoids

Hemorrhoids are varicosities or enlarged veins in the lower part of the rectum and the anus. **Internal hemorrhoids** cannot be felt and are found in the inside lining of the rectum. **External hemorrhoids** are found underneath the skin that surrounds the anal area. These types of hemorrhoids are felt when they swell, and they may cause itching or pain as well as bleeding. A thrombosed external hemorrhoid is the result of blood clotting within veins and can cause the patient significant pain.

Internal hemorrhoids are covered by mucosa and actually protrude into the rectal lumen. They may prolapse when the patient has bowel movements.

External hemorrhoids are covered by skin and protrude from the rectum. These hemorrhoids are more likely to thrombose than internal hemorrhoids.

Hemorrhoids can be classified according to their severity.

- **First-degree hemorrhoids:** These are confined to the anal canal.
- **Second-degree hemorrhoids:** These prolapse when the patient strains (e.g., during defecation) but reduce spontaneously.
- **Third-degree hemorrhoids:** These are prolapsed hemorrhoids but must be manually reduced after each bowel movement.
- **Fourth-degree hemorrhoids:** They are severe and cannot be reduced.

Causes and incidence

Hemorrhoids are most likely due to increased venous pressure. Factors that predispose the development of hemorrhoids include:

- Prolonged periods of sitting or standing.
- Straining during defecation (often associated with constipation).
- Straining during coughing, sneezing or vomiting
- Heart failure.
- Liver disease.
- Alcoholism.
- Anal/rectal infections.
- Rectal surgery.
- Pregnancy.
- Anal intercourse
- Loss of muscle tone (often due to old age).

Signs and symptoms

Hemorrhoids may not cause any symptoms at all. But symptoms generally increase with the severity of the problem.

First-degree hemorrhoids, although they may be painless, characteristically cause painless, intermittent bleeding with defecation. The patient may report bright red blood on the toilet paper or on stool. Bleeding occurs as the result of injury to the mucosa that covers the hemorrhoids. These hemorrhoids may cause itching if anal hygiene is poor.

Second-degree hemorrhoids result in more severe characteristic symptoms with the addition of hemorrhoid prolapse, which spontaneously resolves itself after the patient has a bowel movement. They are usually painless.

Third-degree hemorrhoids cause ongoing discomfort and prolapsed whenever there is an increase in intra-abdominal pressure. They must be manually reduced. Thrombosis of external hemorrhoids causes abrupt rectal pain and a large, firm lump that can be felt by the patient. Such hemorrhoids may cause bleeding that is severe enough to cause secondary anemia. In these cases, the patient may complain of fatigue and weakness, exhibit significant pallor.

Diagnosis

Physical examination confirms the diagnosis of external hemorrhoids.

A proctoscopy can be performed to diagnose internal hemorrhoids and to rule out polyps or other conditions.

Treatment

Treatment depends on the type and severity of the hemorrhoids. For hemorrhoids that are not severe, lifestyle modifications are often the foundation of treatment as well as measures initiated for symptoms relief. The goals of treatment are to reduce or eliminate pain, inflammation and congestion, and to facilitate defecation without

straining.

- Eliminate constipation and teach patients measures to prevent constipation.
- Patient education on hydration and diet
- Stool softeners may be recommended in cases of severe constipation.
- Local anesthetic agents in the form of lotions, creams or suppositories may be used to decrease local swelling and pain.
- Hydrocortisone cream and suppositories may be used to reduce edema, itching and prolapsed hemorrhoids.
- Warm sitz baths may also provide pain relief.
- Hemorrhoidectomy by cauterization or excision is the most effective treatment and is required for patients experiencing severe pain or bleeding, significant prolapse and itching.
- However, surgical intervention is contraindicated in persons with diseases of the blood such as leukemia, aplastic anemia or hemophilia, GI cancer, or during the first trimester of pregnancy.

Nursing Care and Nursing diagnosis

3.13. Anal Fissures

Anal fissures are small tears or cracks in the lining of the anus, often caused by trauma during a hard or large bowel movement. They can be acute (short-term) or chronic (long-lasting).

Signs and symptoms

- Pain during and after bowel movements, often described as a sharp, burning, or tearing sensation.
- Bright red blood on toilet paper or in the stool.
- Muscle spasms in the anal sphincter.

Management

- Dietary changes to soften stools (e.g., increased fiber intake).
- Topical treatments (ointments or creams) to relax the anal sphincter and promote healing.
- Warm sitz baths to relieve discomfort and promote healing.
- Surgical procedures, such as sphincterotomy, for chronic or severe fissures.

3.14. Anal Fistulas

Anal fistulas are abnormal tunnels or tracts that connect the rectum or anus to the skin around the anal area. They often result from an infection that forms an abscess near the anus.

Signs and Symptoms

- Pus or discharge from an opening near the anus.
- Pain, swelling, and tenderness around the anal area.
- Recurrent abscess formation.

Treatment

Surgical intervention is usually required to treat anal fistulas. The procedure involves draining the abscess, removing the affected tissue, and creating a drainage channel to allow for proper healing. Surgery aims to prevent recurrence and complications.

Nursing care and nursing diagnosis for Anal fissures and Fistulas

MODULE 4. DISORDERS OF THE PANCREAS

4.1. Pancreatitis

Pancreatitis is an inflammation of the pancreas. It can be classified as **acute or chronic**.

Acute pancreatitis can be a medical emergency associated with a high risk for life-threatening complications and mortality, whereas chronic pancreatitis often goes undetected until 80% to 90% of the exocrine and endocrine tissue is destroyed.

Acute pancreatitis does not usually lead to chronic pancreatitis unless complications develop. However, chronic pancreatitis can be characterized by acute episodes.

Chronic pancreatitis is an inflammatory disorder characterized by progressive anatomic and functional destruction of the pancreas. As cells are replaced by fibrous tissue with repeated attacks of pancreatitis, pressure within the pancreas increases. The end result is mechanical obstruction of the pancreatic and common bile ducts and the duodenum.

4.1.1. ACUTE PANCREATITIS

Acute pancreatitis ranges from a mild, self-limiting disorder to a severe, rapidly fatal disease that does not respond to any treatment. Mild acute pancreatitis is

characterized by edema and inflammation confined to the pancreas. Minimal organ dysfunction is present, and return to normal usually occurs within 6 months.

Pathophysiology

Self-digestion of the pancreas by its own proteolytic enzymes, principally trypsin, causes acute pancreatitis. Gallstones enter the common bile duct and lodge at the ampulla of Vater, obstructing the flow of pancreatic juice or causing a reflux of bile from the common bile duct into the pancreatic duct, thus activating the powerful enzymes within the pancreas. Normally, these remain in an inactive form until the pancreatic secretions reach the lumen of the duodenum. Activation of the enzymes can lead to vasodilation, increased vascular permeability, necrosis, erosion, and hemorrhage.

Causes and predisposing factors

- Long-term use of alcohol.
- Bacterial or viral infection, with pancreatitis a complication of mumps virus.
- Blunt abdominal trauma, peptic ulcer disease, ischemic vascular disease.
- The use of corticosteroids, thiazide diuretics, and oral contraceptives.

Clinical Manifestations

- Severe abdominal pain, typically located in the mid epigastrium.
- Acute pain, occurring 24 to 48 hours after a very heavy meal or alcohol ingestion, and it may be diffuse and difficult to localize. The pain is not relieved by antacids.
- Vomiting or nausea.
- A rigid or board-like abdomen may develop and is generally an ominous sign; the abdomen may remain soft in the absence of peritonitis.
- Ecchymosis in the flank or around the umbilicus may indicate severe pancreatitis.
- Fever, jaundice, mental confusion, and agitation also may occur.
- Hypotension is typical and reflects hypovolemia and shock caused by the loss of large amounts of protein-rich fluid into the tissues and peritoneal cavity. The patient may develop tachycardia, cyanosis, and cold, clammy skin in addition to hypotension.

Diagnosis

- History and physical examination
- Serum amylase and lipase levels rise in excess within 24 hours.
- Elevated urinary amylase levels – Elevated WBC count
- Hypocalcemia - X-ray

- Peritoneal fluid, obtained through paracentesis or peritoneal lavage, may contain increased levels of pancreatic enzymes

Management

Management of the patient with acute pancreatitis is directed toward relieving symptoms and preventing or treating complications.

- All oral intake is withheld to inhibit pancreatic stimulation and secretion of pancreatic enzymes. The patient is fed parenterally.
- Nasogastric suction may be used to relieve nausea and vomiting, to decrease painful abdominal distention and paralytic ileus, and to remove hydrochloric acid so that it does not enter the duodenum and stimulate the pancreas. Histamine-2 (H2) antagonists (eg, cimetidine) may be prescribed to decrease pancreatic activity by inhibiting HCl secretion.

Pain management: Demerol is prescribed because it is less likely to cause spasm of the sphincter of Oddi contrary to morphine. Antiemetics may be prescribed to prevent vomiting.

Respiratory care: Aggressive respiratory care is indicated because of the high risk for elevation of the diaphragm, pulmonary infiltrates and effusion, and atelectasis. Hypoxemia occurs in a significant number of patients with acute pancreatitis even with normal x-ray findings. Respiratory care may range from close monitoring of arterial blood gases to use of humidified oxygen to intubation and mechanical ventilation.

Biliary drainage: Placement of biliary drains (for external drainage) and stents (indwelling tubes) in the pancreatic duct through endoscopy is performed to reestablish drainage of the pancreas. In most cases, this has resulted in decreased pain and increased weight gain.

Surgical intervention: surgery may be performed to assist in the diagnosis of pancreatitis (diagnostic laparotomy), to establish pancreatic drainage, or to resect or débride a necrotic pancreas.

Nursing Care and nursing diagnosis

4.1.2. CHRONIC PANCREATITIS

Assignment

4.2. Pancreatic Cysts

As a result of the local necrosis that occurs at the time of acute pancreatitis, collections of fluid may form in the vicinity of the pancreas. These become walled off by fibrous tissue and are called **pancreatic pseudocysts**. They are the most common type of pancreatic cysts. Less common cysts occur as a result of congenital anomalies or are secondary to chronic pancreatitis or trauma to the pancreas.

Diagnosis

Diagnosis of pancreatic cysts and pseudocysts is made by ultrasound, computed tomography, and ERCP. ERCP may be used to define the anatomy of the pancreas and evaluate the patency of pancreatic drainage.

Pancreatic pseudocysts may be of considerable size, so because of their location behind the posterior peritoneum, when they enlarge they impinge on and displace the stomach or the colon, which are adjacent. Eventually, through pressure or secondary infection, they produce symptoms and require drainage.

Drainage into the gastrointestinal tract or through the skin and abdominal wall may be established. In the latter instance, the drainage is likely to be profuse and destructive to tissue because of the enzyme contents. Hence, steps must be taken to protect the skin near the drainage site from excoriation.

Ointments protect the skin if they are applied before excoriation takes place.

Another method involves the constant aspiration of digestive secretions from the drainage tract by means of a suction apparatus, so that skin contact with the digestive enzymes is avoided.

4.3. Pancreatic Tumors

Pancreatic tumors can be benign (non-cancerous) or malignant (cancerous).

Malignant tumors include pancreatic adenocarcinoma, which is a highly aggressive cancer.

The symptoms of pancreatic tumors may include abdominal pain, jaundice (yellowing of the skin and eyes), weight loss, and changes in bowel habits.

Diagnosis involves imaging studies (e.g., CT scans, MRIs), biopsies, and blood tests.

The treatment of pancreatic tumors depends on their type and stage. Options may include surgery, chemotherapy, radiation therapy, targeted therapy, and

immunotherapy. Pancreatic cancer is often diagnosed at an advanced stage, which can make it challenging to treat.

MODULE 5. HEPATIC DISORDERS

5.1. Hepatitis (Liver Inflammation)

Hepatitis, is the inflammation of the liver tissue. It can result from various causes, including viral infections, alcohol abuse, autoimmune diseases, and medications. There are several types of hepatitis, including hepatitis A, B, C, D, and E, each with different modes of transmission and potential complications.

Clinical Manifestations

Acute Hepatitis

- | | | |
|--------------------------------------|----------------|-------------------|
| • Anorexia | • Malaise | • Pruritus |
| • Nausea, vomiting | • Headache | • Dark urine |
| • Right upper quadrant discomfort | • Fever | • Bilirubinuria |
| • Constipation or diarrhea | • Arthralgias | • Light stools |
| • Decreased sense of taste and smell | • Urticaria | • Fatigue |
| | • Hepatomegaly | • Continued |
| | • Splenomegaly | hepatomegaly with |
| | • Weight loss | tenderness |
| | • Jaundice | • Weight loss |

Pathophysiology

During an acute viral hepatitis infection, liver damage is mediated by cytotoxic cytokines and natural killer cells that cause lysis of infected hepatocytes.

Inflammation can interrupt bile flow (cholestasis). After resolution of an acute infection, liver cells can regenerate and, if no complications occur, resume their normal appearance and function.

A chronic viral hepatitis infection causes chronic inflammation and can cause fibrosis that (over decades) can progress to cirrhosis.

Complications

Acute liver failure, chronic hepatitis, cirrhosis of the liver, and hepatocellular carcinoma.

Diagnostic Studies

The only definitive way to distinguish among the various forms of viral hepatitis is by testing the patient's blood for the specific antigen or antibody

- History and physical examination
- Liver function tests
- Alanine aminotransferase
- Aspartate aminotransferase (AST)
- Serum bilirubin

Management

The treatment of hepatitis depends on its cause and type. Some forms of viral hepatitis (e.g., hepatitis A and E) are often self-limiting and resolve without specific treatment.

For chronic hepatitis B and C, antiviral medications may be prescribed.

Management may also include supportive care to relieve symptoms, rest, and maintaining proper hydration and nutrition.

In cases of drug-induced or autoimmune hepatitis, identifying and discontinuing the causative agents or using immunosuppressive medications may be necessary.

Preventative measures, such as vaccination for hepatitis A and B and practicing safe sex and safe injection practices, can help reduce the risk of viral hepatitis.

Nutritional therapy: Emphasis is placed on a well-balanced diet that the patient can tolerate. During acute viral hepatitis, adequate calories are important because the patient usually loses weight.

If fat content is poorly tolerated because of decreased bile production, it should be reduced. Vitamin supplements, particularly B-complex vitamins and vitamin K, are frequently used. If anorexia, nausea, and vomiting are severe, IV solutions of glucose or supplemental enteral nutrition therapy may be used. Fluid and electrolyte balance must be maintained.

Nursing Care and Nursing Diagnosis

5.2. Drug and chemical-induced liver diseases (Assignment)

5.3. Liver Cirrhosis

Cirrhosis is a chronic progressive disease of the liver characterized by extensive degeneration and destruction of the liver cells. The development of cirrhosis is an insidious, prolonged course, usually after decades of chronic liver disease.

Etiology

Any chronic liver disease, including disease from excessive alcohol intake can cause cirrhosis. The specific cause of cirrhosis may not be determined in all patients. The most common causes of cirrhosis are chronic hepatitis C infection and alcohol-induced liver disease.

Environmental factors and genetic predisposition may also lead to the development of cirrhosis, regardless of dietary or alcohol intake.

Approximately 20% of patients with chronic hepatitis C and 10% to 20% of those with chronic hepatitis B develop cirrhosis. Chronic inflammation and cell necrosis result in fibrosis and, ultimately, cirrhosis.

Chronic hepatitis combined with alcohol ingestion is synergistic in accelerating liver damage.

Pathophysiology

In cirrhosis the liver cells attempt to regenerate, but the regenerative process is disorganized, resulting in abnormal blood vessel and bile duct architecture. The overgrowth of new and fibrous connective tissue distorts the liver's normal lobular structure, resulting in lobules of irregular size and shape with impeded blood flow. Eventually, irregular and disorganized liver regeneration, poor cellular nutrition, and hypoxia (from inadequate blood flow and scar tissue) result in decreased functioning of the liver.

Clinical manifestations

The onset is usually insidious. Early symptoms include fatigue. Many patients with compensated cirrhosis may not be aware of their liver condition. The diagnosis may not be discovered until when they manifest advanced symptoms.

Later symptoms may be severe and result from liver failure and portal hypertension

Gastrointestinal

- | | | |
|--------------------|--------------------------|----------------------------------|
| • Anorexia | • Change in bowel habits | • Esophageal and gastric varices |
| • Dyspepsia | • Dull abdominal pain | • Gastritis |
| • Nausea, vomiting | • Fetor hepaticus | |

- Hematemesis
- Hemorrhoidal varices

Integumentary

- Jaundice
- Spider angioma
- Palmar erythema
- Purpura
- Petechiae
- Caput medusa

Hematologic

- Anemia
- Thrombocytopenia

Complications

- Portal hypertension - Esophageal and gastric varices - Peripheral edema and ascites - Hepatic encephalopathy (mental status changes, including coma).

Patients without complications of cirrhosis have **compensated cirrhosis**. Those who have one or more complications of their liver disease have decompensated cirrhosis.

- Leukopenia
- Coagulation disorders
- Splenomegaly

Metabolic

- Hypokalemia
- Hyponatremia
- Hypoalbuminemia

Cardiovascular

- Fluid retention
- Peripheral edema
- Ascites

Reproductive

- Amenorrhea
- Testicular atrophy
- Gynecomastia (male)

- Impotence

Neurologic

- Hepatic encephalopathy
- Peripheral neuropathy
- Asterixis

a) Portal Hypertension and Esophageal and Gastric Varices

Structural changes in the liver result in compression and destruction of the portal and hepatic veins and sinusoids. These changes cause obstruction to the normal flow of blood through the portal system, resulting in portal hypertension.

Portal hypertension is characterized by increased venous pressure in the portal circulation, splenomegaly, large collateral veins, ascites, and gastric and esophageal varices. Collateral circulation develops in an attempt to reduce high portal pressure and the increased plasma volume and lymphatic flow. The collateral channels commonly form in the lower esophagus (the anastomosis of the left gastric vein and the azygos veins), anterior abdominal wall, parietal peritoneum, and rectum.

Varicosities may develop in areas where the collateral and systemic circulations communicate, resulting in esophageal and gastric varices, *caput medusae* (ring of varices around the umbilicus), and hemorrhoids.

b) Ascites

Ascites is the accumulation of serous fluid in the peritoneal or abdominal cavity. It is a common manifestation of cirrhosis. With portal hypertension, proteins shift from the

blood vessels via the larger pores of the capillaries into the lymph space. When the lymphatic system is unable to carry off the excess proteins and water, they leak through the liver capsule into the peritoneal cavity. The osmotic pressure of the proteins pulls additional fluid into the peritoneal cavity

A second mechanism of ascites formation is hypoalbuminemia resulting from the liver's inability to synthesize albumin.

A third mechanism is hyperaldosteronism, which occurs when aldosterone is not metabolized by damaged hepatocytes. The increased level of aldosterone causes increased sodium reabsorption by the renal tubules. This retention of sodium, combined with an increase in antidiuretic hormone, causes additional water retention.

- Ascites **is manifested by** abdominal distention with weight gain. If the ascites is severe, the umbilicus may be everted.
- Abdominal striae with distended abdominal wall veins may be present.
- The patient has signs of dehydration (e.g., dry tongue and skin, sunken eyeballs, muscle weakness) and a decrease in urine output.
- Hypokalemia is common and is due to an excessive loss of potassium caused by hyperaldosteronism.
- Low potassium levels can also result from diuretic therapy used to treat the ascites.

Diagnostic Studies

- History and physical examination • Liver function studies
- Esophagogastroduodenoscopy • CT scan • Liver ultrasound • Serum electrolytes

Management

The goal of treatment is to slow the progress of cirrhosis and prevent and treat any complications.

- Rest • Administration of B-complex vitamins • Avoid alcohol • Minimize/ avoid aspirin, acetaminophen, and NSAIDs

Encourage a Low sodium diet • serve diuretics • carry out a paracentesis if indicated.

- Serve antibiotics and other medications as prescribed

Nursing care and nursing diagnosis

5.4. Acute Liver Failure

Acute liver failure, or fulminant hepatic failure, is a clinical syndrome characterized by severe impairment of liver function associated with hepatic encephalopathy. The most common cause is drugs, usually acetaminophen in combination with alcohol. People who abuse alcohol are particularly susceptible to detrimental effects of acetaminophen on the liver.

Other drugs that can cause acute liver failure include isoniazid, halothane, sulfa-containing drugs, and NSAIDs. Drugs can cause liver cell failure by disrupting essential intracellular processes or causing an accumulation of toxic metabolic products. Viral hepatitis, in particular HBV, is the second most common cause. Acute liver failure is characterized by the rapid onset of severe liver dysfunction in someone with no prior history of liver disease. Although the disease can run its course over 8 weeks, it can last as long as 26 weeks.

Clinical Manifestations and Diagnostic Studies

- Manifestations include jaundice, coagulation abnormalities, and encephalopathy.
- Changes in mentation are the first clinical sign.
- Patients are susceptible to a wide variety of complications, including cerebral edema, renal failure, hypoglycemia, sepsis, and multiorgan failure.
- Serum bilirubin is elevated, and the prothrombin time is prolonged.
- Liver enzyme levels (AST, ALT) are often markedly elevated.
- CT or magnetic resonance imaging (MRI) is helpful in providing information about the liver size and contour, presence of ascites, tumors, and patency of the blood vessels.

Nursing and collaborative management

- Since acute liver failure may progress rapidly, with hour-by hour changes in consciousness, early transfer to the ICU is preferred once the diagnosis is made.
- Renal failure is a frequent complication in patients with liver failure. Protect renal function by maintaining adequate fluid balance, avoiding nephrotoxic agents (e.g., aminoglycosides, NSAIDs), and promptly identifying and treating infection.
- Liver transplantation is the treatment of choice and increases survival rates in patients with acute liver failure.
- Monitoring and management of hemodynamic and renal function, as well as glucose, electrolytes, and acid-base status, are critical.

- Conduct frequent neurologic evaluations for signs of elevated intracranial pressure. Position the patient with the head elevated at 30 degrees. Avoid patient stimulation. Maneuvers that cause straining or Valsalvalike movements may increase intracranial pressure (ICP).
- Avoid the use of any sedatives because of their effects on mental status.
- Additional measures include padding bedrails to avoid injury from possible seizures, closely observing the patient to prevent injuries, monitoring intake and output for renal function, and providing good skin and oral care to avoid breakdown and infection.

5.5. Liver Abscesses

Two categories of liver abscess have been identified: **amebic and pyogenic**. Amebic liver abscesses are most commonly caused by *Entamoeba histolytica*.

Pyogenic liver abscesses are less common and could be caused by cholangitis and abdominal trauma.

Pathophysiology

Whenever an infection develops anywhere along the biliary or GI tract, infecting organisms may reach the liver through the biliary system, portal venous system, or hepatic arterial or lymphatic system. Most bacteria are destroyed promptly, but occasionally some gain a foothold. The bacterial toxins destroy the neighboring liver cells, and the resulting necrotic tissue serves as a protective wall for the organisms. Meanwhile, leukocytes migrate into the infected area. The result is an abscess cavity full of a liquid containing living and dead leukocytes, liquefied liver cells, and bacteria.

Clinical manifestations

- Fever with chills and diaphoresis, malaise, anorexia, nausea, vomiting, and weight loss may occur.
- Patient may complain of dull abdominal pain and tenderness in the right upper quadrant of the abdomen.
- Hepatomegaly, jaundice, anemia, and pleural effusion may develop. Sepsis and shock may be severe and life-threatening.

Diagnostic Findings

Blood cultures are obtained but may not identify the organism.

Aspiration of the liver abscess, guided by ultrasound, CT, or MRI, may be performed

to assist in diagnosis and to obtain cultures of the organism. Percutaneous drainage of pyogenic abscesses is carried out to evacuate the abscess material and promote healing

Medical Management

Treatment includes IV antibiotic therapy; the specific antibiotic used in treatment depends on the organism identified. Continuous supportive care is indicated. Open surgical drainage may be required if antibiotic therapy and percutaneous drainage are ineffective.

Nursing Care and nursing diagnosis

MODULE 6. DISORDERS OF THE GALL BLADDER

6.1 Cholecystitis

Acute inflammation (cholecystitis) of the gallbladder causes pain, tenderness, and rigidity of the upper right abdomen that may radiate to the mid-sternal area or right shoulder and is associated with nausea, vomiting, and the usual signs of an acute inflammation. An empyema of the gallbladder develops if the gallbladder becomes filled with purulent fluid.

Calculous cholecystitis is the cause of more than 90% of cases of acute cholecystitis. In calculous cholecystitis, a gallbladder stone obstructs bile outflow. Bile remaining in the gallbladder initiates a chemical reaction; autolysis and edema occur; and the blood vessels in the gallbladder are compressed, compromising its vascular supply.

Acalculous cholecystitis describes acute gallbladder inflammation in the absence of obstruction by gallstones. It occurs after major surgical procedures, severe trauma, or burns. Other factors associated with this type of cholecystitis include torsion, cystic duct obstruction, primary bacterial infections of the gallbladder, and multiple blood transfusions. It is speculated that acalculous cholecystitis results from alterations in fluids and electrolytes and in regional blood flow in the visceral circulation. Bile stasis (lack of gallbladder contraction) and increased viscosity of the bile are also thought to play a role.

Nursing care and nursing diagnosis

6.2. CHOLELITHIASIS

Calculi, or gallstones, usually form in the gallbladder from the solid constituents of bile; they vary greatly in size, shape, and composition.

Pathophysiology

There are two major types of gallstones: those **composed predominantly of pigment** and those **composed primarily of cholesterol**. Pigment stones probably form when unconjugated pigments in the bile precipitate to form stones.

The risk of developing such stones is increased in patients with cirrhosis, hemolysis, and infections of the biliary tract. Pigment stones cannot be dissolved and must be removed surgically.

Cholesterol stones account for other cases. Cholesterol, a constituent of bile, is insoluble in water. Its solubility depends on bile acids and phospholipids in bile. In gallstone-prone patients, there is decreased bile acid synthesis and increased cholesterol synthesis in the liver, resulting in bile supersaturated with cholesterol, which precipitates out of the bile to form stones. The cholesterol-saturated bile predisposes to the formation of gallstones and acts as an irritant, producing inflammatory changes in the gallbladder.

Clinical Manifestations

- Gallstones may be silent, producing no pain and only mild gastrointestinal symptoms. The patient with gallbladder disease from gallstones may develop two types of symptoms: those due to disease of the gallbladder itself and those due to obstruction of the bile passages by a gallstone. The symptoms may be acute or chronic. Epigastric distress, such as fullness, abdominal distention, and vague pain in the right upper quadrant of the abdomen, may occur. This distress may follow a meal rich in fried or fatty foods.
- Pain and biliary colic - Jaundice - Changes in urine and stool color
- Vitamin deficiency

Diagnostic Findings

- History and physical examination • Ultrasound • Liver function studies
- White blood cell count • Serum bilirubin
- Endoscopic retrograde cholangiopancreatography (ERCP)
- Percutaneous transhepatic cholangiography

Management

The major objectives of medical therapy are to reduce the incidence of acute episodes of gallbladder pain and cholecystitis by supportive and dietary management and, if possible, to remove the cause of cholecystitis by pharmacologic therapy, endoscopic procedures, or surgical intervention.

Although nonsurgical approaches have the advantage of eliminating risks associated with surgery, they are associated with persistent symptoms or recurrent stone formation. Most of the nonsurgical approaches, including lithotripsy and dissolution of

gallstones, provide only temporary solutions to the problems associated with gallstones. other treatment approaches include;

The use of **laparoscopic cholecystectomy** (removal of the gallbladder through a small incision through the umbilicus).

Nutritional and supportive therapy

Approximately 80% of the patients with acute gallbladder inflammation achieve remission with rest, intravenous fluids, nasogastric suction, analgesia, and antibiotic agents. Unless the patient's condition deteriorates, surgical intervention is delayed.

The diet immediately after an episode is usually limited to low-fat liquids.

The patient should avoid eggs, cream, pork, fried foods, cheese and rich dressings, gas-forming vegetables, and alcohol. It is important to remind the patient that fatty foods may bring on an episode. Dietary management may be the major mode of therapy in patients who have had only dietary intolerance to fatty foods and vague gastrointestinal symptoms.

Drug therapy

Ursodeoxycholic acid (UDCA) and chenodeoxycholic acid (chenodiol or CDCA) have been used to dissolve small, radiolucent gallstones composed primarily of cholesterol.

Nonsurgical removal of gallstones

Dissolving Gallstones: Various methods have been used to dissolve gallstones by infusion of a solvent into the gallbladder.

Stone Removal by Instrumentation; Several nonsurgical methods are used to remove stones that were not removed at the time of cholecystectomy or have become lodged in the common bile duct.

Nursing care and nursing diagnosis

Assignment: (Group work), Read, make short notes and do a presentation on;

- ❖ **Gastric decompression, lavage and stoma care, different feeding techniques**
- ❖ **Alternative therapies used in treatment of disorders of digestive system**