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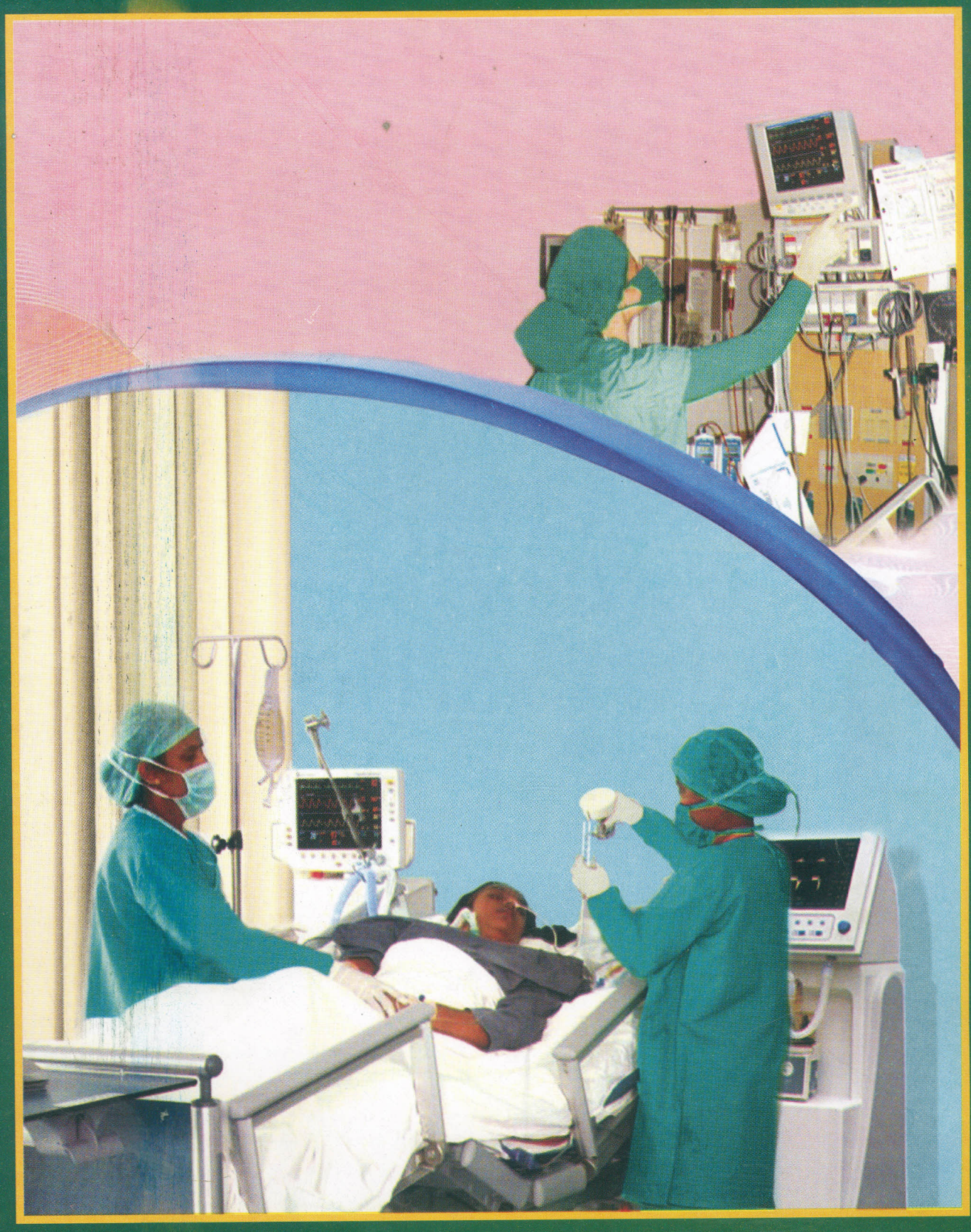
Indira Gandhi National Open University
School of Health Sciences

BNS-032
Nursing Management in Critical Care Conditions



CARE OF CRITICALLY ILL PATIENTS WITH PROBLEMS OF GASTROINTESTINAL SYSTEM 1

"शिक्षा मानव को बन्धनों से मुक्त करती है और आज के युग में तो यह लोकतंत्र की भावना का आधार भी है। जन्म तथा अन्य कारणों से उत्पन्न जाति एवं वर्गगत विषमताओं को दूर करते हुए मनुष्य को इन सबसे ऊपर उठाती है।"

— *इन्दिरा गांधी*

"Education is a liberating force, and in our age it is also a democratising force, cutting across the barriers of caste and class, smoothing out inequalities imposed by birth and other circumstances."

– Indira Gandhi

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Block

1

CARE OF CRITICALLY ILL PATIENTS WITH PROBLEMS OF GASTROINTESTINAL SYSTEM

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COURSE INTRODUCTION

In theory course-1, you have learnt about the general concepts and principles of critical care nursing. In course-2, you will learn in depth about various critical conditions of all the body systems. The content emphasizes on using the nursing process approach in caring critically ill patients. This will help you to focus on specific and advanced assessment of each critical condition, the possible nursing diagnosis and the collaborative management including medical, surgical and nursing intervention.

This course has been organised in six blocks for your convenience. Each block has 4-6 units which will help you to develop advanced knowledge and skills regarding care of critically ill patients with problems of various body systems. The details of the Blocks are outlined below:

Block 1 focuses on Care of critically ill patients with problems of gastrointinal systems.

Block 2 deals with Care of critically ill patients with problems of Respiratory system.

Block 3 deals with Care of critically ill patients with cardiothoracic problems.

Block 4 describes Care of critically ill patients with problems of nervous system.

Block 5 describes Care of critically ill patients with problems of renal system and endocrinal system.

Block 6 deals with care of critically ill patients with various emergency conditions.

BLOCK INTRODUCTION

This block deals with various gastrointestinal disorders which require critical and prompt assessment and subsequent intervention. This block will help you to apply nursing process in care of patients with gastrointestinal disorders commonly seen in the critical care environment; and will guide you for prompt assessment and intervention by applying your critical thinking.

This block comprises four units:

Unit 1 Deals will Acute Gastrointestinal bleeding.

Unit 2 Focuses on Acute intestinal obstruction, intra abdominal compartment syndrome, Perforative Peritonitis.

Unit 3 Describes Hepatic disorders – Fulminent hepatic failure and hepatic encephalopathy.

Unit 4 Relates to Acute Pancreatitis.

UNIT 1 ACUTE GASTROINTESTINAL BLEEDING

Structure

1.0 OBJECTIVES

After completing this unit, you will be able to:

- Enumerate the various causes of acute Gastrointestinal (GI) bleeding;
- Identify signs and symptoms in a patient with acute GI bleeding;
- State your role in different diagnostic tests to identify the source of bleeding;
- Manage a patient with GI bleeding; and
- Advice a patient to prevent the recurrent attacks of GI bleeding.

1.1 INTRODUCTION

Acute gastrointestinal (GI) bleeding is a potentially life-threatening abdominal emergency that remains a common cause of hospitalization. The source can be

upper or lower, overt or occult. The patient can be either hemodynamically stable or unstable on presentation. Bleeding from the upper GI tract is approximately four times as common as bleeding from the lower GI tract and is a major cause of morbidity and mortality. Recognizing the signs and symptoms of upper versus lower gastrointestinal bleeding is crucial for prompt and appropriate treatment as it may point to many significant diseases and conditions. In this unit you will learn about concepts, definitions, causes, pathophysiological basis of diseases, signs and symptoms, assessment and management focused on acute Gastrointestinal (GI) bleeding.

1.2 DEFINITION AND CLASSIFICATION

In this section we shall focus on definition and classification of Acute gastrointestinal (GI) bleeding.

1.2.1 Definition

Gastrointestinal bleeding refers to any bleeding that starts in the gastrointestinal tract, which extends from the mouth to the large bowel. The degree of bleeding can range from nearly undetectable to acute, massive, and life-threatening.

1.2.2 Classification

Gastrointestinal bleeding can be roughly classified into two clinical syndromes: Upper gastrointestinal bleeding and lower gastrointestinal bleeding.

Upper gastrointestinal bleeding

Upper gastrointestinal bleeding is defined as the bleeding proximal to the ligament of **Trietz (Fig. 1.1).** This type of bleeding may be the result of a variety of pathological processes and characterized by hematemesis (vomiting up blood) and melena (tarry stool containing altered blood).

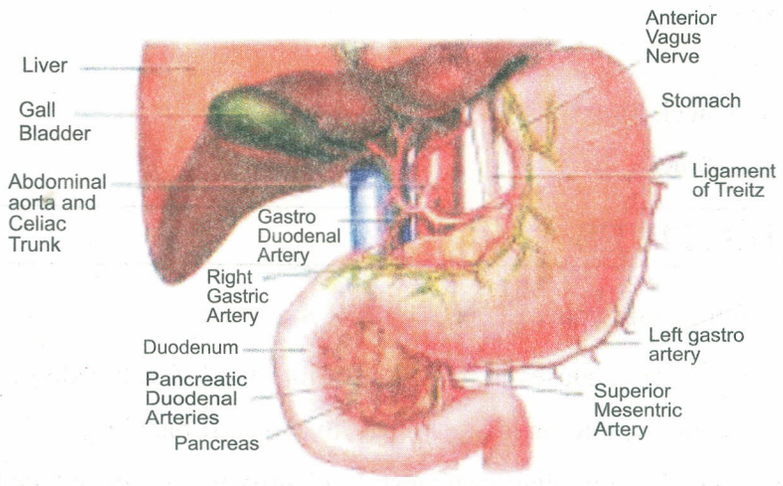

Fig. 1.1: Gastro Intestinal Bleeding (G.I. Tract above and below ligament of trietz)

Lower gastrointestinal bleeding

Usually the bleeding per rectum especially in the absence of hematemesis is the indication of lower gastrointestinal bleeding. Tarry stool containing altered blood may be due to the bleeding originating from anywhere between the stomach and the proximal colon.

1.3 ETIOLOGICAL FACTORS

Gastrointestinal bleeding occurs due to number of etiological factors as given below:

1.3.1 Causes for Upper GI Bleeding (UGIB)

The causes of upper GI bleeding are as follows:

a) Peptic ulcer disease
b) Mucosal erosive diseases
c) Esophageal varices
d) Mallory-Weiss tear
e) Stress-related erosive syndrome (stress ulceration)
f) Vascular anomalies.

Let us explain each one of them.

a) Peptic Ulcer disease

You may recall that Peptic ulcers are localized erosions of the wall of the digestive tract. Ulcers usually occur in the stomach or duodenum. Breakdown of the walls results in damage to blood vessels, causing bleeding. When the mucous membranes break down, they are unable to counteract the harsh effects of stomach acid. Non-steroidal anti-inflammatory drugs (NSAIDs), aspirin, alcohol, and cigarette smoking promote gastric ulcer formation. Helicobacter pylori are a type of bacteria that also promote formation of ulcers. About 50% of UGI bleeds are due to peptic ulcers.

b) Mucosal erosive diseases

Esophagitis is reported to be a cause of acute UGI bleeding. Inflammation of the stomach wall can result in bleeding in response to an inability of the gastric lining to protect itself from the acid it produces. Erosive gastritis is the second leading cause of UGI hemorrhage. NSAIDs, steroids, alcohol, burn, and trauma infrequently cause acute, massive UGI bleeding. Hematemesis is the most common presentation in acute UGI bleeding.

c) Esophageal varices

This is the third leading cause of UGIB and has the highest mortality rate, between 40% and 70%. Swellings in veins of esophagus or stomach usually result in patients with portal hypertension and from liver diseases. Varices most commonly result from alcoholic liver cirrhosis. When varices bleed, the bleeding can be massive and catastrophic and occur without warning signals. Almost 90% of variceal bleeding presents with hematemesis, with or without melena.

d) Mallory-Weiss tear

Mallory-Weiss tears occur within 2 cm of the gastro esophageal junction and usually associated with a hiatus hernia. It is believed to be caused by increase in pressure across the wall of the esophageal or stomach usually at the esophago-gastric junction, often as a result of vomiting or retching and can also occur after seizures, forceful coughing or laughing, lifting, straining, or childbirth. Physicians often find tears in people who have recently binged on alcohol.

e) Stress-related erosive syndrome (stress ulceration)

Stress-related erosive syndrome (SRES) is usually found among critically ill patients. The mucosal ischemia and gastric acidity play important role in SRES resulting in bleeding. There used to be superficial mucosal lesions mostly in the acid producing portion of the stomach.

f) Vascular anomalies

Abnormal vascular structures are also the cause of bleeding among patients with chronic renal failure. Arteriovenous malformations most commonly present with massive acute hemorrhage.

1.3.2 Causes of Lower Gastro Intestinal Bleeding (LGIB)

The causes of lower GI bleeding are as follows:

a) Diverticulosis

b) Angiodysplasia

c) Ischemic Colitis

d) Polyps

e) Hemorrhoids and fissures.

a) Diverticulosis: Colonic diverticula are one of the most common causes of LGI bleeding. Small out-pockets, or diverticula, form on part of the wall of colon (large intestine), usually in a weakened area of the bowel wall. A person may develop several pockets more common in those who have constipation and strain at stool. These are most common in the sigmoid colon and to a lesser extent in the proximal colon. The bleeding is usually painless and not accompanied by diverticulitis. Diverticulitis of large bowel results from protrusions of mucosa through muscular layers, generally at the site of nutrient vessel.

b) Angiodysplasia: Angiodysplasia is one of the vascular anomalies, a malformation in the blood vessels in the wall of the GI tract. About 25% of LGI bleeding occurs in patients over 50 years of age with vascular anomalies. The lesions are usually multiple and most common in the large intestine and often bleed. The elderly and people with chronic kidney failure develop the disease most often. Bleeding usually occurs as bright red blood per rectum or as maroon-colored stool.

c) Ischemic Colitis: Mostly patients develop LGI bleeding while in Intensive care unit. Ischemia of colon usually affects older patients. Ischemic colitis involves the left colon, the splenic flexure, and sigmoid colon; the areas between superior and inferior mesenteric arteries. The patient complains of sudden onset of abdominal pain and bleeding.

d) Polyps: Intestinal polyps are noncancerous tumors of the GI tract, occurring mostly in people older than 40 years. A small proportion of these polyps may transform into cancer. Colonic polyps may bleed rapidly, or they may bleed slowly and go undetected.

e) Hemorrhoids and fissures: Hemorrhoids are swellings of veins in and around rectum. Straining on passing of stool can cause them to bleed. Bleeding from hemorrhoids is usually mild, intermittent, and bright red. Massive bleeding is rare. Anal fissures, or tears in the anal wall, also may

trigger small amounts of bright red bleeding from the anus. Forceful straining during passage of hard stool usually causes such tears, which can be very painful.

1.4 PATHOPHYSIOLOGY

Any etiological factors when leads to bleeding, the blood in GI tract irritate to the stomach causing nausea and vomiting that is in the form of hematemesis. But when remains in the stomach for a long period, give coffee ground appearance as partially digested. The accumulation of blood in the tract stimulates peristalsis, leads to increase in bowel sounds and diarrhea. Stool may be black and tarry-melena or frankly bloody.

GI bleeding with great loss of blood can rapidly decrease the blood volume, brings the changes in the hemodynamic status: tachycardia, hypotension, pallor, and decrease urine output. Unless the blood volume is restored, patient can go into hypovolemic shock that can lead to acidosis, renal failure, bowel infarction, acute coronary syndrome, coma, and death (as shown in flow chart (Fig.1.2)).

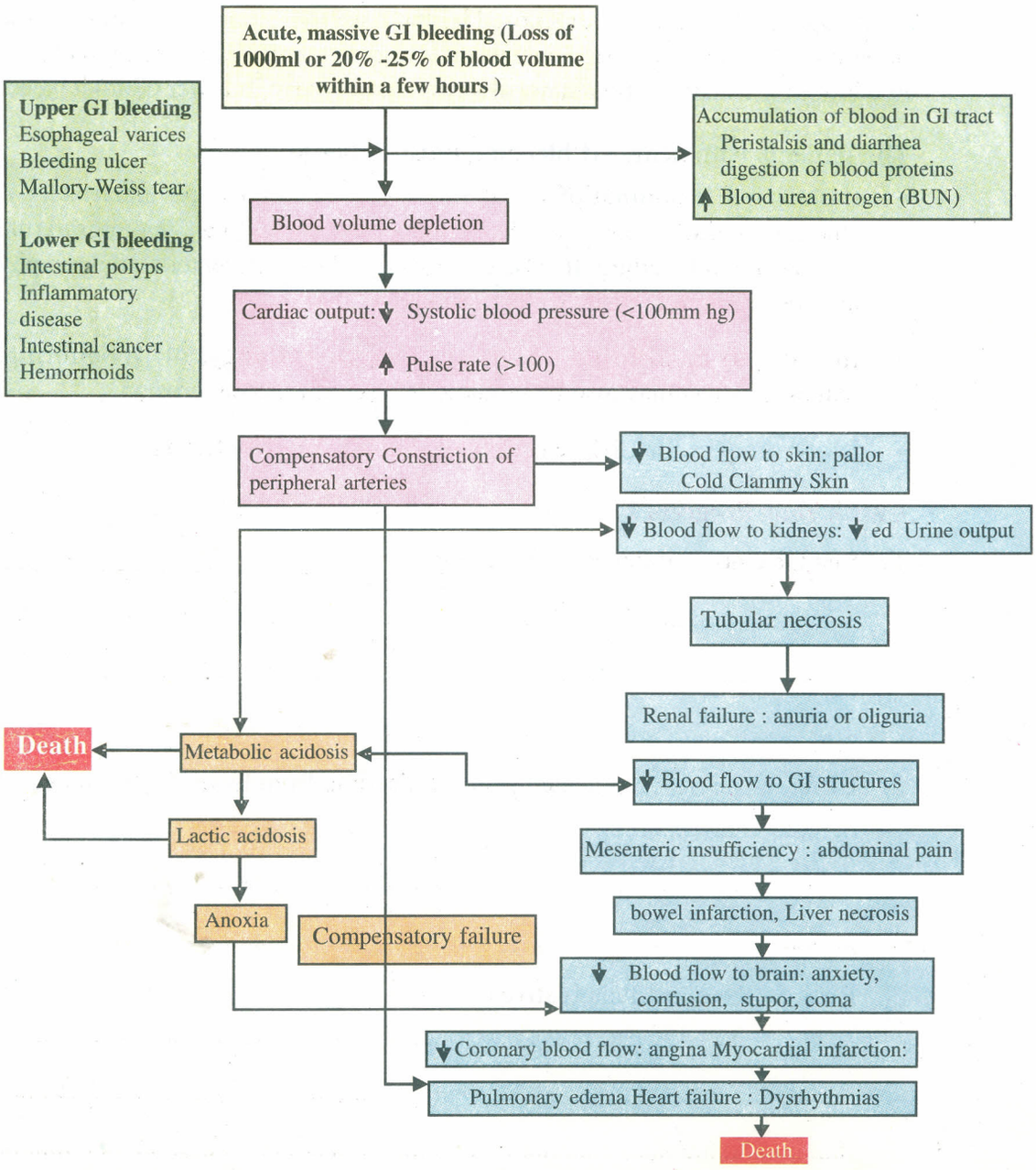

Fig. 1.2: Flow chart of Pathophysiology of acute gastrointestinal hemorrhage

1.5 CLINICAL MANIFESTATIONS

Patients with GI hemorrhage present with:

- Hematemesis, coffee ground vomiting, melena or hematochezia. The presentation of bleeding depends on the amount and location of hemorrhage.
- Complications of anemia, including chest pain, syncope, fatigue and shortness of breath.
- Hemodynamic instability i.e., Low Blood pressure, increased pulse rate, altered level of consciousness, decreased urine output, signs of aspiration.
- Decreased peripheral circulation- Pallor, Cold Clamy Skin
- Anxiety.

1.6 DIAGNOSTIC ASSESSMENT

Acute upper GI bleeding is diagnosed by the presence of melena, hematemesis, or blood in a patient's gastric contents on lavage in comparison, bright red blood in the stool is usually—though not always—indicative of a lower GI tract bleed.

The patients with acute GI bleeding must undergo

- Endoscopic examinations that include, esophagoscopy, gastroscopy, colonoscopy, sigmoidoscopy depending on the presenting symptoms to rule out the site of bleeding. In case of Angiodysplasia, mesenteric angiography may be required to visualize the lesions.
- In order to confirm the diagnosis, biopsy, cytology, pH monitoring, manometry test may also be required in case of mucosal erosion.
- Blood samples for CBC, electrolytes, BT/CT, cross matching.

Check Your Progress 1

1) List the causes of upper GI Bleeding.

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2) Differentiate between acute upper GI bleeding from lower GI bleeding.

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3) Encircle the correct alternatives.

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i) Which of the following signs and symptoms is the most common presentation of upper GI bleeding?

   a) Bright red blood from the rectum with feces.

   b) Bright red blood from the rectum without feces.

   c) Tarry stools associated with vomiting of blood.

   d) Tarry stools without vomiting of blood.

ii) All the following factors indicate a poor prognosis in patients with upper GI bleeding, except:

   a) Bloody nasogastric aspirate.

   b) Ulcer size less than 2 cm.

   c) Variceal bleeding source.

   d) Age older than 60 years.

1.7 THERAPEUTIC MANAGEMENT

Caring for patients with acute GI bleeding can be challenging. But your nursing interventions on both the physical and emotional levels can make a difference.

1.7.1 Goals of Management

The aims of therapeutic management must focus on following

- Stemming the bleeding.
- Restoring hemodynamic status.
- Prevent further Gastrointestinal bleeding
- Identify and treat cause of the bleeding.
- Experience minimal or no symptoms of pain or anxiety.

1.7.2 Management

- Stemming the bleeding,
- Administration of IV fluids such as normal saline or a balanced electrolyte solution,
- Administration of whole blood to restore the blood volume and components,
- Administration of packed red cells if required to restore oxygen carrying capacity of blood,
- Upper endoscopy whenever possible to seal the bleeding vessel,
- Gastric lavage.

1.7.3 Endoscopy

For most types of GI bleeding, endoscopic therapy is the treatment of choice, and is often successful (Fig. 1.3.). The most common form of endoscopic treatment is given below:

1) **Sclerotherapy:** Sclerotherapy involves injecting the bleeding vessel with a necrotizing agent; this traumatizes the endothelial layer of the GI mucosa, causing necrosis and eventual sclerosis of the bleeding vessel.

2) **Heater probe, laser therapy, electro coagulation:** In this a bleeding site and surrounding tissue is cauterized or heat treated with a heater probe or electro coagulation

3) **Rubber band ligation and hemoclips:** During band ligation bands are placed around the varices to create an obstruction to stop the bleeding.

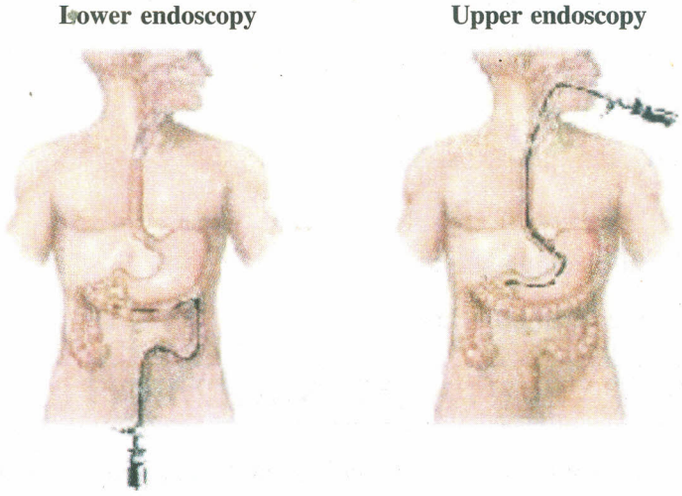

Fig. 1.3 : Endoscopy

If the above therapies fail then following procedure may be followed:

Tamponade therapy: Treatment of variceal upper GI bleeding may also include tamponade therapy, typically with the placement of **a Sengstaken-Blakemore tube**

Porto systemic shunt/Transjugular intrahepatic shunt (TIPS): A channel between the systemic and portal venous systems is crealed to redirect portal blood thereby reducing portal hypertension and decompressing the varices to control bleeding.

Baloon tamponade: Baloon tamponade tubes stop hemorrhage by applying direct pressure against bleeding vessel while decompressing the stomach.

1.7.4 Drug Therapy

Acid suppression: In an upper bleeding, proton pump inhibitors reduce gastric acid production and enhance healing of bleeding lesions.

Correction of coagulopathy: If coagulation parameters (e.g., prothrombin time) are deranged, vitamin K or fresh frozen plasma may need to be administered.

Reduction of portal pressure: If the bleeding is thought to be due to esophageal varices (a complication of cirrhosis of the liver), vasopressin analogues and rarely octreotide may be administered. Rarely, a **Sengstaken-Blakemore tube** may be inserted to mechanically compress varices.

1.7.5 Surgical Intervention

In severe bleeding in some cases **Laparotomy** may be required to identify the bleeding source.

- Prepare patient for the surgery (physical, psychological and legal) and give postoperative nursing care.
- When bleeding stops and the patient is haemo-dynamically stable, start **nutritional therapy,** the patient is observed for symptoms of nausea and vomiting and a recurrence of bleeding.

  1) Feedings initially consist of clear fluids or milk and are given hourly until tolerance is determined.

  2) Gradual introduction of bland foods follows if the patient exhibits no signs of discomfort.

1.8 NURSING MANAGEMENT

Nursing management of gastro-intestinal bleeding is discussed in following sub-sections:

1.8.1 Assessment

i) **Rapid assessment:** You need to do Rapid assessment of bleeding which is very crucial

Irrespective of cause of acute GI bleeding, a rapid clinical assessment is essential and you should focus on the immediate crises. Patients with massive Gastrointestinal bleeding remains a common reason for intensive care unit admission or in emergency department with hypovolemic shock. Along with shock, you will find hematemesis (bright red/brown/coffee ground emesis, hematochezia (bright red stools), melena (black tarry stools).Patients with low-volume bleeding have no change in vital signs when they stand. They may appear anxious but have no other signs or symptoms. So, in a patient with GI bleeding you should **immediately assess for:**

- Airway, breathing, and circulation
- Blood pressure
- Rate and character of pulse
- Peripheral perfusion with capillary refill
- Observation for the presence or absence of neck vein distention
- Mental status (level of consciousness)
- Skin color and temperature
- The presence or absence of bowel sounds should be assessed
- Urine output and specific gravity
- Increased thirst
- Complete history of events leading to bleeding episode.

- Place the acutely ill patient on cardiac monitor, obtain oxygen saturation level. An indwelling catheter may be inserted to evaluate urine output.

You may refer the box 1.1 below for a quick clinical classification of Hemorrhage.

Class	Blood Loss (%)	Clinical Signs/Symptoms
1.	≤ 15	Pulse rate normal or <100 beats/min (Supine)
		Capillary refill: <3 sec
		Urine output: adequate (30-35ml/hr)
		Orthostatic hypotension Apprehensive
2.	15-30	Pulse rate: increased (>100 beats/min)
		Capillary refill: sluggish
		Pulse pressure: normal (Supine)
		Tachypnea
		Urine output: low (25-30 ml/hr)
3.	30-40	Pulse rate: 120+ beats/min (Supine)
		Hypotension
		Skin: Cool, Pale
		Confused
		Hyperventilating
		Urine output: low (5-15 ml/hr)
4.	≥ 40	Profoundly hypotensive
		Pulse rate: 140+ beats/min
		Confused, lethargic
		Urine output minimal

Fig. 1.3: Clinical classification of hemorrhage

ii) Help the patient for undergoing various laboratory studies to assess the level of various parameters

Blood for:

- CBC
- blood urea nitrogen (BUN)
- serum electrolytes
- blood glucose
- prothrombin time
- liver enzymes
- arterial blood gases (ABGs)
- type and cross-match for possible blood transfusions.

Other laboratory studies:

- Vomitus and stools should be tested for the presence of gross and occult blood;

- Urinalysis provides information on presence of blood in the urine, and the specific gravity gives an immediate indication of patient's hydration status.

iii) Assist for any endoscopic procedure/ tamponade therapy if necessary.

1.8.2 Nursing Diagnoses

1) Fluid volume deficit related to acute loss of blood
2) Ineffective peripheral tissue perfusion related to loss of circulatory volume
3) Risk for aspiration related to active bleeding and altered level of consciousness
4) Altered renal and cerebral tissue perfusion related to decreased blood volume
5) Anxiety related to GI Bleeding/Uncertain outcome
6) Ineffective coping related to situational crisis
7) Potential complication : hypovolemic shock related to loss of blood.

1.8.3 Implementation

Your goal as a critical care nurse is to restore and maintain an effective cardiac output and tissue perfusion and controlling the hemorrhage and further bleeding. To achieve these aims you may follow the following protocol:

Follow resuscitation protocol for Upper Gastro Intestinal (UGI) Bleeding:

- In severe bleeding; obtain adequate intravenous access; give rapid fluid resuscitation with lactate Ringer's solution. Blood (Whole blood, packed RBCs, and fresh frozen plasma) may be administered to replace loss blood due to hemorrhage.
- Protect the airway by intubating if required to avoid respiratory compromise from aspiration of blood, especially in patients with altered mental status.
- Monitor vital signs every 15 to 30 minutes.
- Give gastric lavage in case of upper GI bleed.
- Check signs and symptoms of shock and accordingly treatment should be started as soon as possible.
- Keep the head of the bed elevated.
- Oxygen Inhalation may help to increase blood oxygen saturation (it not intubated).
- Insert indwelling urinary catheter.
- Urine output should be measured hourly (at least 0.5 ml/kg per hour indicates adequate renal perfusion).
- Urine specific gravity should be measured.
- Intake and output record should indicate an equal or positive balance.

- Assist in obtaining central venous access when necessary to monitor central venous pressure/ cardiac output.
- Once the patient has been stabilized, early endoscopy will likely be performed. Although the history and physical examination are used to differentiate between upper and lower GI bleeding, endoscopic examination is required to determine the exact site of the bleeding.
- Vasopressive and stomatostanin may be used to arrest bleeding.

Nursing alert

Whatever type of endoscopic procedure your patient undergoes, watch for signs of treatment complications, including fever; mediastinitis; pain caused by esophageal spasm; motility disturbances of the esophageal sphincter, resulting in esophageal reflux and heartburn; and perforation, as indicated by abdominal distention, a board-like abdomen, and decreased bowel sounds.

Be alert for systemic complications as well. Cardiovascular complications include heart failure, heart block, and pericarditis. Respiratory complications include atelectasis, aspiration pneumonia, pneumothorax, pulmonary embolism, and acute respiratory distress syndrome.

1.8.4 Follow up Advice

Patient who has had one major bleeding episode is likely to have another. Following an acute GI hemorrhage, continuous care focusing on the underlying cause of disease if possible and preventing the future episode of bleeding the patient must be instructed to:

- Avoid irritating foods
- Prevent or decrease stress-inducing situations.
- Take only prescribed medications. Patient who requires regular administration of ulcerogenic drugs — such as aspirin, corticosteroids, or NSAIDs — should receive instructions regarding the potential adverse effects these agents may have on the GI mucosa Taking the medications with meals or snacks lessens the potential irritating effects.
- Smoking and alcohol should be stopped because they are sources of irritation.
- Need for long-term follow-up care may be necessary.

Check Your Progress 2

1) List the main goals of management of acute GI bleeding?

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ii) Encircle the correct answers

i) All the following include recommended therapy for upper GI hemorrhage, except:

a) FFP in a patient who requires a blood transfusion

b) Oral omprazole in a patient with peptic ulcer disease

c) Octreotide in a patient with nonvariceal bleeding when endoscopy is delayed

d) Antibiotic therapy in a patient with variceal bleeding.

ii) Which of the following is the diagnostic study of choice in patients with lower GI bleeding?

a) Endoscopy

b) Radionuclide imaging

c) Angiography

d) Colonoscopy

iii) Which of the following is the most common causes of upper and lower GI bleeding, respectively?

a) Ulcers and angiodysplasia

b) Varices and diverticulosis

c) Esophagitis and colitis

d) Gastric erosions and polyps.

1.9 LET US SUM UP

Gastrointestinal bleeding is a relatively frequent problem in the critically ill patients. Common causes of UGI bleeding include acute stress ulceration (ASU), peptic ulceration and esophageal varices. Non-variceal upper gastrointestinal bleeding requires resuscitation and correction of coagulation disturbances before endoscopy is performed. If a bleeding ulcer is detected it is often managed by an adrenaline injection or electrocautery into the base of the lesion and a proton pump inhibitor. Surgery is considered for all patients in whom bleeding persists despite endoscopic or medical therapy.

For lower gastrointestinal bleeding or if there is no obvious upper gastrointestinal lesion during endoscopy, then selective mesenteric angiography with embolisation of the bleeding point or colonoscopy with electrocautery or adrenaline injection (for diverticula hemorrhage) may be considered as an alternative to surgery.

1.10 GLOSSARY

Hematemesis : Hematemesis is vomiting of red blood and indicates upper GI bleeding, usually from an arterial source or varices. Coffee-ground emesis is vomiting of dark brown, granular material results from upper GI bleeding that has slowed or stopped, with conversion of red Hb to brown hematin by gastric acid.

Hematochezia : Hematochezia is the passage of gross blood from the rectum and usually indicates lower GI bleeding but may result from vigorous upper GI bleeding with rapid transit of blood through the intestines.

Melena : 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. However, black stool that does not contain occult blood may result from ingestion of iron, bismuth, or various foods and should not be mistaken for melena.

1.11 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

i) The causes of upper GI bleeding in the following conditions are as follow:

- Peptic ulcer disease
- Mucosal erosive diseases
- Esophageal varices
- Mallory-Weiss tear
- Stress-related erosive syndrome (stress ulceration)
- Vascular anomalies

ii) Acute upper GI bleeding is diagnosed by the presence of melena, hematemesis, or blood in a patient's gastric contents on lavage in comparison, bright red blood in the stool is usually—though not always—indicative of a lower GI tract bleed.

iii) i) c ii) b

Check Your Progress 2

1) The aims of therapeutic management must focus on

- Stemming the bleeding
- Resorting hemodynamic status
- Have no further GI bleeding
- Have the cause of the bleeding identified and treated
- Experience minimal or no symptoms of pain or anxiety

2) i) c ii) d iii) a

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UNIT 2 ACUTE INTESTINAL OBSTRUCTION, PERFORATIVE PERITONITIS, INTRA ABDOMINAL COMPARTMENT SYNDROME

Structure

2.0 OBJECTIVES

After completing this unit, you will be able to:

- Enumerate the various etiological factors in acute Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome;
- Explain the pathophysiological changes in Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome;

- Identify the various signs and symptoms in a patient with acute Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome;
- Appreciate your role in different diagnostic tests to identify the cause of Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome;
- Identify and describe your role as a nurse in the prevention, care and management of Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome; and
- Assist the patient in preventing the potential complications of acute Intestinal obstruction, perforative peritonitis and intra abdominal compartment syndrome.

2.1 INTRODUCTION

Acute Intestinal Obstruction, perforative peritonitis and intra abdominal compartment syndrome are some of the most common emergencies in critical care units. As a critical care nurse working in critical care units, you should be able to identify the impending signs and symptoms of these emergencies and prevent the life threatening complications in the patients by your accurate observations and timely interventions. In this unit we shall discuss about acute Intestinal Obstruction, perforative peritonitis and intra abdominal compartment syndrome.

2.2 ACUTE INTESTINAL OBSTRUCTION

We shall discuss about definition, etiology diagnostic assessment, pathophysiology, clinical manifestation, therapeutic and nursing management of acute intestinal obstruction in the following subsections:

2.2.1 Definition

Partial or complete impairment of the forward flow of intestinal contents is known as intestinal obstruction. It is the failure of intestinal contents to move through the bowel lumen. Obstruction of large bowel occurs much less frequently than small bowel obstruction.

2.2.2 Etiological Factors

The main etiological factors are:

i) **Mechanical Obstruction:** A mechanical bowel obstruction is something that decreases the diameter of the bowel's opening from either the inside or outside. It physically blocks the passage and thereby movement of intestinal contents through the intestines resulting into distension and accumulation of fluids and gas. Possible mechanical obstructions could be due to problems outside the intestine, within intestine and in the intestinal lumen as given below (Fig 2.1).

- Strangulation by bands or adhesions or through apertures.
- Volvulus (twisting of bowel on its mesentery).

- Impaction of foreign bodies including gall stones.
- Acute intussusceptions (telescoping of one segment of bowel into another).
- Neoplasms.
- Hernias.
- Strictures.
- Fecal impaction.

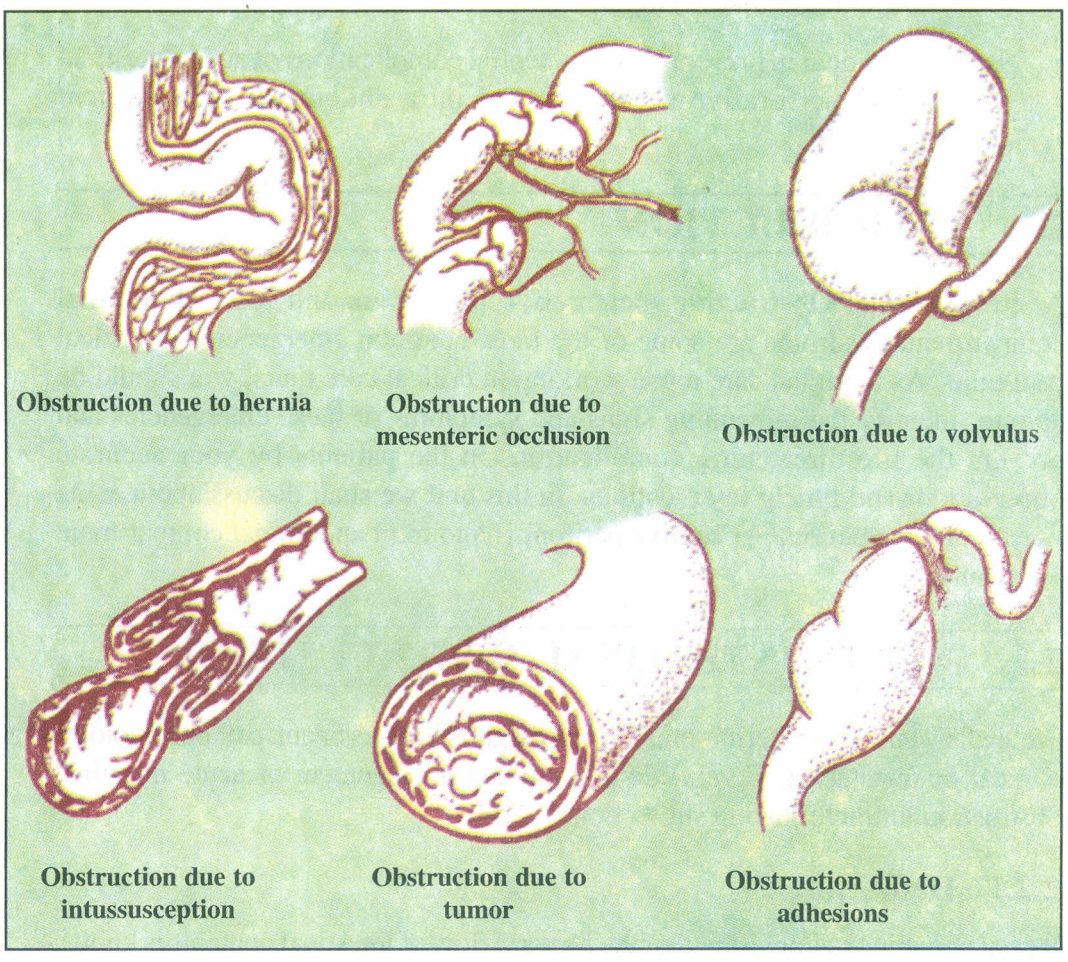

Fig 2.1: Intestinal Obstruction due to Mechanical Obstruction

ii) **Neurogenic or adynamic Obstruction:** (Paralytic ileus i.e., decreased impulses to bowel for propulsive movements): Interference with the nerve supply to the intestine resulting in the decrease or absence of peristalsis. The causes can be as follows:

- Inflammatory reactions (acute pancreatitis, appendicitis),
- Electrolyte abnormalities (especially hypokalemia),
- Lumbar spinal fractures or spinal cord injury,
- Abdominal surgery,
- Shock.

iii) **Vascular Obstruction:** Interference or the impaired blood supply to a portion of the intestines resulting in ischemia and gangrene of the bowel. It is due to following:

- Clot, thrombosis,

- Compression of the vessel,
- mesenteric artery occlusion,
- Mesenteric vein thrombosis,
- Arthrosclerosis.

2.2.3 Pathophysiological Changes

When the bowel is obstructed, ingested fluids and food, digestive secretions, and gas accumulate above the obstruction. The proximal bowel distends, and the distal segment collapses. The normal secretary and absorptive functions of the mucosa are depressed, and the bowel wall becomes edematous and congested.

Normally 7-8 liters of electrolyte rich fluid is secreted by the bowel and most of it is reabsorbed. Increased pressure within the bowel causes a decrease in the absorption ability of the bowel which increases the fluid retention still further.

Soon the increased intra luminal pressure causes a reduction in the venous return which increases the venous pressure. This in turn increases the capillary permeability and allows plasma to extravagate into the bowel lumen and into the peritoneal cavity.

The bowel wall also becomes permeable to bacteria and bowel organisms enter the peritoneal cavity. The retention of fluids in the bowel and peritoneal cavity can lead to a severe reduction in the circulating blood volume and can result in hypotension and hypovolemic shock.

Strangulating obstruction is obstruction with compromised blood flow; it occurs in nearly 25% of patients with small-bowel obstruction. It is usually associated with hernia, volvulus, and intussusceptions. Strangulating obstruction can progress to infarction and gangrene in as little as six hours. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcts, leading to gangrene and perforation. In large-bowel obstruction, strangulation is rare (except with volvulus).

2.2.4 Clinical Manifestations

We shall discuss about clinical manifestation under two categories as given below:

i) Related to Small bowel Obstruction

Symptoms occur soon after the onset —

- **Epigastric or umbilical abdominal cramping:** The patient with a small-bowel obstruction presents with a pain that is colicky and intermittent. The pain is episodic and generally occurs in the mid-to-upper abdomen. If the obstruction is partial, the pain worsens right after the patient eats and improves with digestion. Distention and generalized discomfort without colicky pain may indicate a lack of movement in the intestines caused by paralysis of the bowel (paralytic ileus). Sometimes the patient gets pain relief after changing position or vomiting.
- **Vomiting:** Nausea and vomiting occur as a result of increased peristaltic activity, but the intestinal contents reverse the direction instead of moving

forward. The vomiting is often **projectile and nonfecal**, especially if there is obstruction high in the small bowel. Another sign of obstruction high in the small intestine is vomit that is odorless or looks or smells like bile (a greenish yellow fluid that has a bitter, offensive odor).

- **Constipation** is a common sign of small-bowel obstruction. However, in a partial obstruction, the patient may have diarrhea and pass some gas. In a complete obstruction, the patient may have a bowel movement if the obstruction is above the stool that's already in the bowel.
- **Obstipation** (complete obstruction).
- **Hyperactive high pitched bowel sounds**. The bowel sounds range from hyperactive bowel sounds (increased loudness, tone, and regularity) to totally absent bowel sounds, typical of a paralytic ileus.
- **Palpable loops of bowel.**
- **Shock, oliguria, and constant abdominal pain** seen with late partial obstruction, or strangulated bowel, and or perforation.

ii) Related to Large Bowel Obstruction

Milder symptoms with more gradual onset —

- **Lower abdominal cramping**,
- **Occasional fecal-type vomitus,**
- **Increasing constipation** leads to obstipation with abdominal distention,
- **Nausea and vomiting** may be absent at first. As the large-bowel obstruction worsens, the patient's vomit may smell like feces,
- **Bloating** is more visible in patients with a large-bowel obstruction,
- **Palpable mass,**
- **Tympany** with percussion.
- Systemic symptoms are relatively mild and fluid and electrolyte deficits are uncommon.

2.2.5 Diagnostic Assessment

- When a patient has abdominal pain and complains of nausea and vomiting, it's critical that you begin your assessment by taking a complete and detailed history. Ask the patient about his bowel habits, and find out about any surprising changes. Ask when he had his last bowel movement, any bloating of abdomen or constipation? Were there prior surgeries? Abdominal trauma? Hernias? Peptic ulcer disease? Does the patient experience constipation or indigestion? Has he had gallstones? Tumors? Radiation therapy to the abdomen or the peritoneal area? Has he ever had an eating disorder? Find out about current and past medications.
- Ask and determine the location, duration, intensity, frequency and the type of abdominal pain. Ask what, if anything, relieves the pain.
- Find out if he has nausea or vomiting, and, if so, with what frequency, consistency, color, amount and odor and record the onset, frequency, amount color and odor.

- Record bowel function; Any passage of flatus.
- Once you obtain a thorough history, it's time to assess the patient.
- Inspect the abdomen for any scar, abdominal mass and distention.
- Measure the abdominal girth.
- Monitor vital signs. If temperature is elevated, it could be a sign of infection or possible perforation, tachycardia can be related to possible hypovolemic shock or septicemia and when you measure blood pressure, keep in mind that hypotension is secondary to low circulating fluid volume).
- Monitor hydration status (capillary refill time, BP, Pulse Skin turgor, urine output).
- Assess bowel sounds including character and location: Auscultate to all four quadrants of the abdomen.

You should be able to hear some bowel sounds at least once every 5 to 15 seconds. They might last one to a few seconds each. In a normal bowel, the sounds may be high-pitched gurgling sounds.

- If you don't detect bowel sounds, there may be a problem, such as paralytic ileus or a bowel obstruction. Observe for muscle guarding and tenderness.
- High-pitched or tinkling sounds may correspond to a hyperactive bowel with increased peristalsis. They are associated with diarrhea and typically occur anterior to an obstruction.

After initial assessment, a number of diagnostic tests are done to determine the location, extent, and severity of the obstruction.

Help the patient physically and psychologically to undergo these diagnostic tests. These tests include following:

- A complete blood cell (CBC) count to look for signs of infection and dehydration. An elevated white blood cell count (15,000 to 20,000/mm^3) is a sign of infection and may indicate bowel strangulation or perforation.
- An increased hematocrit level may mean dehydration.
- Serum electrolytes i.e., Sodium, potassium, chlorides decreased, BUN increased.
- Serum amylase levels may be elevated, particularly when strangulation is present.
- Serum Osmolality may increase.
- ABGs may reveal metabolic alkalosis (pH>7.45, bicarbonates >26 mEq/L, PCo_2>45 mmHg) with small bowel obstruction due to loss of HCl from the stomach.
- Urinalysis — urine specific gravity increases.
- Type and crossmatch (if there's a chance the patient needs surgical intervention).

- Abdominal X-rays, flat and upright views to determine the location, pattern, and types (mechanical or non-mechanical, partial or complete) of the obstruction. It shows distended loops of intestine with fluid and gas in a small bowel obstruction. Free air under diaphragm indicates a perforation.
- Barium enema may be used to confirm the diagnosis of large bowel obstruction.
- Computed tomography can also determine the location and degree of the obstruction; it's about 90% sensitive and specific in diagnosing small-bowel obstruction and is the preferred diagnostic imaging test.
- Barium enema to determine the exact location and confirm the presence of an obstruction (barium is used with great caution, and not at all if a perforation is suspected).
- Colonoscopy to help in the assessment and diagnosis of a large-bowel obstruction.
- Gastroscopy tests, which can indicate an upper gastrointestinal mass.

2.2.6 Therapeutic Management

Treatment of acute intestinal obstruction must proceed simultaneously with diagnosis:

i) Conservative medical management

- Nasogastric tube or intestinal tubes (Cantor or Miller-Abbott tubes) may be used to decompress the bowel so as to relieve distention and nausea. Note the color and amount of aspirate.
- IV fluids that contain normal saline and potassium to restore, balance, and/or replace lost fluid. The types and amounts of fluids ordered depend on the results of lab tests and the overall condition of the patient.TPN may be necessary in some cases to correct the nutritional status.
- IV antibiotics if bowel ischemia is suspected and to minimize the risk of infection that may result from the contents of the intestines spilling into the peritoneal and abdominal cavities.
- Pain management.

ii) Surgical Treatment

The choice of surgical procedure depends on the type and location of the bowel obstruction.

Surgery in the small bowel: It can be a resection with end-to-end anastomosis. In this procedure, the surgeon removes the diseased tissue and reattaches either end of the healthy intestinal tissue to the other.

When surgery is chosen for a large-bowel obstruction, the predominant cause of the obstruction is often a malignant tumor. If there's perforation or diverticula, the surgery may be a resection with anastomosis. If there's a tumor in the colon, a hemicolectomy (removal of the diseased part of the colon) may be appropriate. You should prepare the patient physically and mentally for the type of surgery and the rehabilitation required in the postoperative period. If colostomy /ileostomy

is to be performed, psychological preparation of the patient and of significant others is required. You should actively participate in the rehabilitation of the patient and family members.

2.2.7 Nursing Management

i) Nursing Assessment

It should start with detailed health history and physical examination as discussed below:

Health history and Physical examination

- Complains of abdominal pain, bloating, constipation.
- Determine the location, duration, intensity and frequency of the abdominal pain.
- Record the onset, frequency, colour, odor and amount of vomitus.
- Bowel function e.g., passage of flatus should be recorded.
- Auscultate abdomen for bowel sounds; character and location.
- Inspect the abdomen for any scar, abdominal mass, distension.
- Observe for muscle guarding and tenderness.
- Previous history of intestinal obstruction or risk factors such as hernia, diverticulosis, previous abdominal surgery.
- Vital signs, temperature BP, skin colour, texture and turgor.
- Colour and moisture of mucus membrane.

ii) Nursing Diagnoses

- Acute pain related to abdominal distension and increased peristalsis
- Reabsorbtion and loss of fluids secondary to vomitting.
- Deficient fluid volume related to decrease in intestinal fluid.
- Ineffective tissue perfusion related to severe reduction in the circulating blood volume.
- Ineffective breathing pattern related to abdominal distension.
- Imbalanced nutrition related to intestinal obstruction and vomiting.

iii) Planning

Your main goals are:

To decrease distension or remove gas and fluid

To relieve or remove obstruction (decompression)

To maintain fluid and electrolyte balance

To improve nutritional status.

iv) Nursing Interventions

- Monitor Vital Signs (Blood pressure, Pulse, Respiration and Temperature).
- Oxygen inhalation to supply adequate oxygen to tissue organs.
- Intake and out put record (urine out put and nasogastric aspirate) Note the colour and amount of aspirate.

- Adequate Hydration is very important to maintain renal function and tissue perfusion, to prevent shock, and to maintain adequate blood pressure. Taking care of I/V lines and recording the amount and type of fluid administered.
- Pain medications are useful to control pain and the patient's anxiety.
- If patient has abdominal distention, measure his abdominal girth every shift. Each time, make sure the patient is in the supine position if s/he's comfortable and it is not contraindicated;
- Use the same measuring tape, measure at the same time, and mark the site on his abdomen to ensure accuracy.
- Determine if his bowel function has improved by noting the absence of nausea and vomiting. Listen for bowel sounds and note any expulsion of flatus and stools.
- Examine the incision to check if there is drainage from the surgical wound.
- Check If patient has an ostomy, a surgically created opening in the abdomen for the discharge of intestinal waste.

A colostomy is created for problems associated with the blockage of the large intestine. An ileosotomy is an opening created for problems in the small intestine. This is when your support, understanding, and ability to educate are an essential part of your patient care.

Assess the stoma and ensure that the pouch protects the skin and contains drainage.

- Observer the parastomal skin and prevent excoriation due to drainage coming in contact with the skin. The stoma may be closed afterwards.
- Comfort and reassure the patient. Teach him what to expect during his recovery period. Ensure to include the patient's family and caregivers in the plan of care when appropriate.
- Explain the patient the purpose of any tubes and clarify the sequence of procedures to alleviate his anxiety.
- While patient can't take nutrition by mouth, provide good mouth care. Use a water-soluble lubricant for lip care and care of the nasal mucosa. If s/he has an NG tube in place, provide the appropriate care for the tube as well as for the patient. When your patient is ready to eat, usually within 24 to 48 hours after surgery or at the first sounds of peristalsis, a progressive diet will be ordered as tolerated.
- Provide comfort measures for relief of the patient as and when possible. Simply raising the head of the bed to 45 degrees helps the patient breathe better and can help create a more restful environment.
- Prevent Infection by maintaining aseptic technique and taking care of the patient's hygiene and of physical surroundings.

Follow up advice

Teach the patient to recognize signs and symptoms of recurrent problems, such as infection, recurrence of obstruction, so that s/he knows when to seek help from his health care provider.

Check Your Progress 1

i) Intestinal obstruction can occur due to —

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ii) List the Clinical manifestations of following

a) Small bowel obstruction.

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b) Large bowel obstruction

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iii) Outline the Immediate nursing management of a patient with intestinal obstruction.

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2.3 PERFORATIVE PERITONITIS

Perforative peritonitis is the most common surgical emergency in India. Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex. Any part of the GI tract may become perforated from a variety of causes, releasing gastric or intestinal contents into the peritoneal space. Symptoms develop suddenly, with severe pain followed shortly by signs of shock. Peritonitis is life threatening without prompt treatment. While working in the critical care unit you should identify the patients with the signs and symptoms of perforative peritonitis to decrease morbidity and mortality in these patients. **We shall discuss about Definition Etiological factors, Pathophysiology, Clinical manifestations, diagnosis, therapeutic management and nursing management in following sub sections.**

2.3.1 Definition

Peritonitis is an inflammation (irritation) of the peritoneum, the tissue that lines the wall of the abdomen and covers the abdominal organs.

2.3.2 Etiological Factors

Sites and etiological factors of bowel perforations leading to peritonitis are:

- **Duodenum:** The most common site of perforation in the bowel is duodenum. Duodenal perforation is a complication of untreated chronic duodenal ulcer and administration of NSAIDS. The duodenum gets perforated commonly on the anterior surface.
- **Stomach:** Either a peptic ulcer or carcinoma of stomach may get perforated. Administration of NSAIDS may contribute to perforations of stomach.
- **Small intestines:** Perforation of small intestines is due to typhoid fever and regional enteritis or as a complication of unrelieved intestinal obstructions.
- **Large intestines:** The large bowel perforations are mainly due to malignancy, diverticulosis, and ulcerative colitis and during medical procedures like colonoscopy, sigmoidoscopy and biopsies.
- **Other appendages:** Intestinal appendages like vermiform appendix, Meckels diverticulum or colonic diverticula may get inflamed and get perforated.
- **Other causes:**
  - o Intestinal obstructions, strangulations may lead to perforations.
  - o External penetrating injuries and injuries to intestines during laparoscopic and surgical procedures may cause perforations of intestines.
  - o Ingestion of caustic substances: Accidental or intentional ingestion of caustic substances may result in acute intestinal perforation and peritonitis. Delayed perforation may occur up to 4 days after acid exposure.
  - o Foreign bodies: These may cause perforation of the esophagus, stomach, or small intestine, with intra-abdominal infection, peritonitis, and sepsis.

2.3.3 Pathophysiology

Normally, the stomach is relatively free of bacteria and other microorganisms because of its high intraluminal acidity. Most persons who experience abdominal trauma have normal gastric functions and are not at risk of bacterial contamination following gastric perforation. However, those who have a preexisting gastric problem are at risk of peritoneal contamination with gastric perforation. Leakage of acidic gastric juice into the peritoneal cavity often results in profound chemical peritonitis. If the leakage is not closed and food particles reach the peritoneal cavity, chemical peritonitis is succeeded by gradual development of bacterial peritonitis. Patients may be free of symptoms for several hours between the initial chemical peritonitis and the later occurrence of bacterial peritonitis.

The microbiology of the small bowel changes from its proximal to its distal part. Few bacteria populate the proximal part of the small bowel, whereas the distal part of the small bowel (the jejunum and ileum) contains aerobic organisms

(e.g., *Escherichia coli*) and a higher percentage of anaerobic organisms (e.g., *Bacteroides fragilis*). Thus, the likelihood of intra-abdominal or wound infection is increased with perforation of the distal bowel.

The presence of bacteria in the peritoneal cavity stimulates an influx of acute inflammatory cells. The omentum and viscera tend to localize the site of inflammation, producing a phlegmon. (This usually occurs in perforation of the large bowel). The resulting hypoxia in the area facilitates growth of anaerobes and produces impairment of bactericidal activity of granulocytes, which leads to increased phagocytic activity of granulocytes, degradation of cells, hypertonicity of fluid forming the abscess, osmotic effects, shift of more fluids into the abscess area, and enlargement of the abdominal abscess. If untreated, bacteremia, generalized sepsis, multi organ failure, and shock may occur.

2.3.4 Clinical Manifestations

The main manifestations of peritonitis are acute **abdominal pain**, **abdominal tenderness**, and **abdominal guarding**, which are exacerbated by moving the peritoneum, e.g., coughing, flexing the hips, or eliciting the Blumberg sign (i.e., rebound tenderness, meaning that pressing a hand on the abdomen elicits less pain than releasing the hand abruptly, which will aggravate the pain, as the peritoneum snaps back into place). The presence of these signs in a patient is sometimes referred to as **peritonism**. Along with these patient will present with following signs and symptoms:

- Anorexia, nausea, vomiting,
- Abdominal distention,
- Weakness, pallor, excessive sweating, and cold skin because of excessive loss of fluid, electrolytes, and protein into the abdominal cavity.
- Hypotension,
- Tachycardia
- Signs of dehydration
- Temperature of 103° F (39.4° C) or higher
- Abdominal distention and resulting upward displacement of the diaphragm may decrease respiratory capacity. Typically, the patient with peritonitis tends to breathe shallowly.

2.3.5 Diagnostic Assessment

Patient in severe pain will be very anxious. You should help the patient physically and psychologically to undergo various diagnostic evaluations.

Rectal and bimanual vaginal and pelvic examination: These examinations may help in assessing conditions such as acute appendicitis, ruptured tubo-ovarian abscess, and perforated acute diverticulitis.

Laboratory Investigations

- CBC is done to determine leukocytosis (greater than 20,000/µl).

- Paracentesis is done and aspirate is sent for blood, bile, pus, bacteria, fungus and amylase.
- Serum electrocytes are also measured.

Other tests

- Abdominal computed tomography scan or *X*-rays showing edematous and gaseous distention of the small and large bowel support the diagnosis. In the case of perforation of a visceral organ, the X-ray shows air in the abdominal cavity.
- Chest X-ray may show elevation of the diaphragm.

Peritoneoscopy

This may be necessary to identify the underlying cause. It may be helpful in patients without ascites.

2.3.6 Therapeutic Management

After peritonitis develops, emergency treatment must combat infection, restore intestinal motility, and replace fluids and electrolytes.

When peritonitis results from perforation, **surgery** is necessary. The aim of surgery is to eliminate the source of infection by evacuating the spilled contents and repairing any organ perforation.

Prevention of complications

To prevent perforative peritonitis, steps should be taken for:

- Early treatment of GI inflammatory conditions.
- Avoiding over the counter medications.
- Preoperative and postoperative antibiotic therapy after GI surgery prevents perforation and subsequent peritonitis.

2.3.7 Nursing Management

i) Nursing Assessment

History taking and physical examination

Ask patients about the time of onset of pain, the duration and location of pain, the characteristics of pain, relieving and aggravating factors, and other symptoms associated with abdominal pain.

- Sharp, severe, sudden-onset epigastric pain that awakens the patient from sleep often suggests perforated peptic ulcer. Differentiate this from conditions such as cholecystitis and pancreatitis.
- Painless perforation of a peptic ulcer can occur with steroid use.
- The presence of shoulder pain suggests involvement of the parietal peritoneum of the diaphragm.
- In elderly patients, consider the possibility of perforated diverticulitis or ruptured acute appendicitis if the pain is located in the lower abdomen.

- In young adults with pain in the lower abdominal quadrant, consider perforated appendicitis as a possible diagnosis. Acute appendicitis with sudden perforation is usually associated with illness of several hours. The pain is typically localized in the right lower quadrant of the abdomen, unless the disease process has progressed to generalized peritonitis.
- In young women, also consider ruptured ovarian cyst and ruptured tubo-ovarian abscess in the differential diagnosis.
- Assess for Signs of Impending shock i.e., low BP, Tachycardia, and pale skin.
- Take vital signs and assess for any hemodynamic changes.
- Take pulse and blood pressure measurements with the patient lying in bed and sitting, and note any postural changes.

Abdominal Examination

- Examine the abdomen for any external signs of injury, abrasion, and/or ecchymosis. Observe patients' breathing patterns and abdominal movements with breathing, and note any abdominal distension or discoloration. (In perforated peptic ulcer disease, patients lie immobile, occasionally with knees flexed, and the abdomen is described as boardlike.)
- Bowel sounds are usually absent in generalized peritonitis.
- Carefully palpate the entire abdomen, noting any masses or tenderness. Tachycardia, fever, and generalized abdominal tenderness may suggest peritonitis. Abdominal fullness and doughy consistency may indicate intra-abdominal hemorrhage.
- Tenderness on percussion may suggest peritoneal inflammation.

ii) Nursing Diagnoses

- Abdominal pain related to perforation and inflammation of the peritoneum.
- Risk for fluid volume deficit related to collection of fluid in the peritoneum, nausea and vomiting.
- Altered nutrition related to perforation of GI tract, nausea and vomiting.
- Anxiety related to pain, uncertainty of cause and outcome.
- Potential complications: hypovolemic, and septic shock.

iii) Planning

- Resolution of inflammation
- Relief of abdominal pain
- Prevention of complications
- Improvement in nutritional status.

iv) Implementation

The patient with peritonitis is acutely ill, needs intensive nursing care:

- Assess pain.
- Analgesic as prescribed for pain management
- Position the patient in a comfortable position
- Maintain airway, breathing and respiration
- Oxygen inhalation for adequate tissue perfusion
- Monitor vital signs i.e., Blood pressure, Pulse, Respiration and Temperature
- NPO and nasogastric (NG) aspiration and recording the amount and color of aspirate
- Accurate monitoring of fluid intake and output
- Total parenteral nutrition to meet the nutritional requirements
- Urinary catheterization is used to assess urinary flow and fluid replacement
- Measuring abdominal Girth and recording
- Administer systemic antibiotics as prescribed.

Preoperative nursing management

- Pain management
- To decrease peristalsis the patient should receive nothing by mouth
- I.V. fluids are administered.
- Nasogastric (NG) suction should be done
- Antibiotic therapy should be given as advised usually.It includes the administration of appropriate antibiotics, depending on the infecting organisms
- Preparing patient for the surgery.

Post operative nursing management

- Patient is managed for airway, breathing and respiration
- Administering parenteral fluids and electrolytes as ordered and accurately recording fluid intake and urine output, including drainage from the NG tube and the incision.
- Placing the patient in semi-Fowler's position to promote drainage (through drainage tube) by gravity.
- Encouraging the patient to deep-breathe, cough effectively, and use an incentive Spiro meter.
- Teaching the patient how to splint the incision. Observe and record character of drainage from postoperative wound drains if inserted; take care to avoid dislodging drains.

- Regular cleaning and lubrication of oral cavity to counteract dryness due to NG intubation.
- Encouraging and assisting in ambulation as ordered, usually on the first postoperative day.
- Observing for signs of dehiscence (may complain that, "something gave way") and abscess formation (persistent abdominal tenderness and fever).
- Frequently assessing for peristaltic activity by listening for bowel sounds and evaluating for passage of flatus, bowel movements, and soft abdomen.
- When peristalsis returns and temperature and pulse rate are normal or when NG output diminishes (less than 200 ml/24 hr), the NG tube is removed.
- Gradually decreasing parenteral fluids and increasing oral intake.
- Postoperatively, preparing patient and family for discharge; teaching them how to care of incision and drains if still in place at discharge.

Check Your Progress 2

i) List the main clinical manifestations in a patient of peritonitis.

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ii) How can you prevent the chances of peritonitis in a patient after surgery?

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2.4 INTRRA ABDOMINAL COMPARTMENT SYNDROME

Abdominal Compartment Syndrome (ACS) is a manifestation of severe intra-abdominal hypertension and is characterized by an acute rise in intraabdominal pressure that results in impaired intraabdominal organ perfusion. The patient presents massive abdominal distension with attendant cardiovascular, respiratory, and renal insufficiency. It occurs when a fixed compartment, defined by myofascial elements or bone, becomes subject to increased pressure, leading to ischemia and organ dysfunction. Well recognized to occur in the extremities, it also occurs in the abdomen, and some believe, in the intracranial cavity.

Abdominal compartment syndrome is probably under recognized because it primarily affects patients who are already quite ill and whose organ dysfunction may be incorrectly ascribed to progression of the primary illness. Untreated, ACS has a high mortality rate. Since treatment can improve organ dysfunction, it is important that the diagnosis be considered in the appropriate clinical situation. You being working in the critical care units can identify the impending signs and symptoms of abdominal compartment syndrome and can prevent the life threatening complications in the patients.

2.4.1 Definition

Organ dysfunction caused by intra-abdominal hypertension (IAH) is considered to be abdominal compartment syndrome. The dysfunction may be respiratory insufficiency secondary to compromised tidal volumes, decreased urine output caused by falling renal perfusion, or any organ dysfunction caused by increased abdominal compartment pressure.

Intra-abdominal pressure is the steady state pressure concealed within the abdominal cavity. For most critically ill patients, an intra-abdominal pressure of 5 to 7 mmHg is considered normal and is directly related to body mass index. Patients with increased abdominal girth that developed slowly may have higher baseline intra-abdominal pressures. As an example, morbidly obese and pregnant individuals can have chronically intra-abdominal pressure as high as 10 to 15 mmHg without adverse sequelae.

The morbidity of abdominal compartment syndrome is attributed to its effects on multiple organ systems. Because of this, abdominal compartment syndrome has a high mortality rate even with treatment. Furthermore, abdominal compartment syndrome is often a sequelae to severe injuries that independently carry high morbidity and mortality rates.

2.4.2 Classification of Abdominal Compartment Syndrome (ACS)

Intra-abdominal hypertension (IAH) is a sustained intra-abdominal pressure =12 mmHg which can be further classified as:

- **Hyper acute IAH** i.e., elevation of the intra-abdominal pressure lasting only seconds. It is due to laughing, coughing, straining, sneezing, defecation, or physical activity.
- **Acute IACH** i.e., elevation of the intra-abdominal pressure that develops over hours. It is usually the result of trauma or intra-abdominal hemorrhage and can lead to the rapid development of ACS.
- **Sub acute IAH** i.e., elevation of the intra-abdominal pressure that develops over days. It is most common in medical patients and can also lead to ACS.
- **Chronic IAH** i.e., Elevation of intra-abdominal pressure that develops over months (pregnancy) or years (morbid obesity). It does not cause ACS, but does place the individual at higher risk for ACS if they develop superimposed acute or sub acute IAH.

Abdominal compartment syndrome is organ dysfunction caused by intra-abdominal hypertension (IAH) and can be divided into the following 3 categories:

- **Primary or acute abdominal compartment syndrome:** This occurs when intra-abdominal pathology is directly and proximally responsible for the compartment syndrome.
- **Secondary abdominal compartment syndrome:** This occurs when no visible intra-abdominal injury is present but injuries outside the abdomen cause fluid accumulation.
- **Chronic abdominal compartment syndrome:** This occurs in the presence of cirrhosis and ascites, often in the later stages of the disease.

Conditions like severe adynamic ileus, bowel obstruction, retropeitoneum hematoma, necrotizing pancreatitis, hemoperitoneum, hepatic ascites etcmay cause to intraabdominal hypertension, leading to ACS.

Now you know that Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are distinct clinical entities and should not be used interchangeably. Elevated pressure in the abdomen is referred to as intra-abdominal hypertension (IAH) while the end stage organ failure that occurs due to the patho-physiologic derangements that resulted by increased intra-abdominal pressure, referred as the abdominal compartment syndrome (ACS).

2.4.3 Etiological Factors

The 3 types of abdominal compartment syndrome have different and sometimes overlapping causes.

i) Primary or acute ACS

- Penetrating trauma
- Intraperitoneal hemorrhage
- Pancreatitis
- External compressing forces, such as debris from a motor vehicle collision or after a large structure explosion
- Pelvic fracture
- Rupture of abdominal aortic aneurysm
- Perforated peptic ulcer.

ii) Secondary

Secondary ACS may occur in patients without an intra-abdominal injury, when fluid accumulates in volumes sufficient to cause IAH.

- Large-volume resuscitation.
- Large areas of full-thickness burns may lead to ACS within 24 hours if they resave average of 237 ml/kg over a 12-hour period.
- Penetrating or blunt trauma without identifiable injury.
- Postoperative.
- Packing and primary fascial closure, which increases incidence.
- Sepsis.

ii) Chronic

- Peritoneal dialysis
- Morbid obesity
- Cirrhosis.

2.4.4 Pathophysiology

Normal intraabdominal pressure is less than 10 mm Hg. The abdominal cavity that includes peritoneal and, to a lesser extent, retroperitoneal cavities, are much more distensible than an extremity, they reach an endpoint at which the pressure rises dramatically due to intraabdominal hemorrhage, surgical packing, or third space fluids may cause a rise in the intraabdominal pressure.

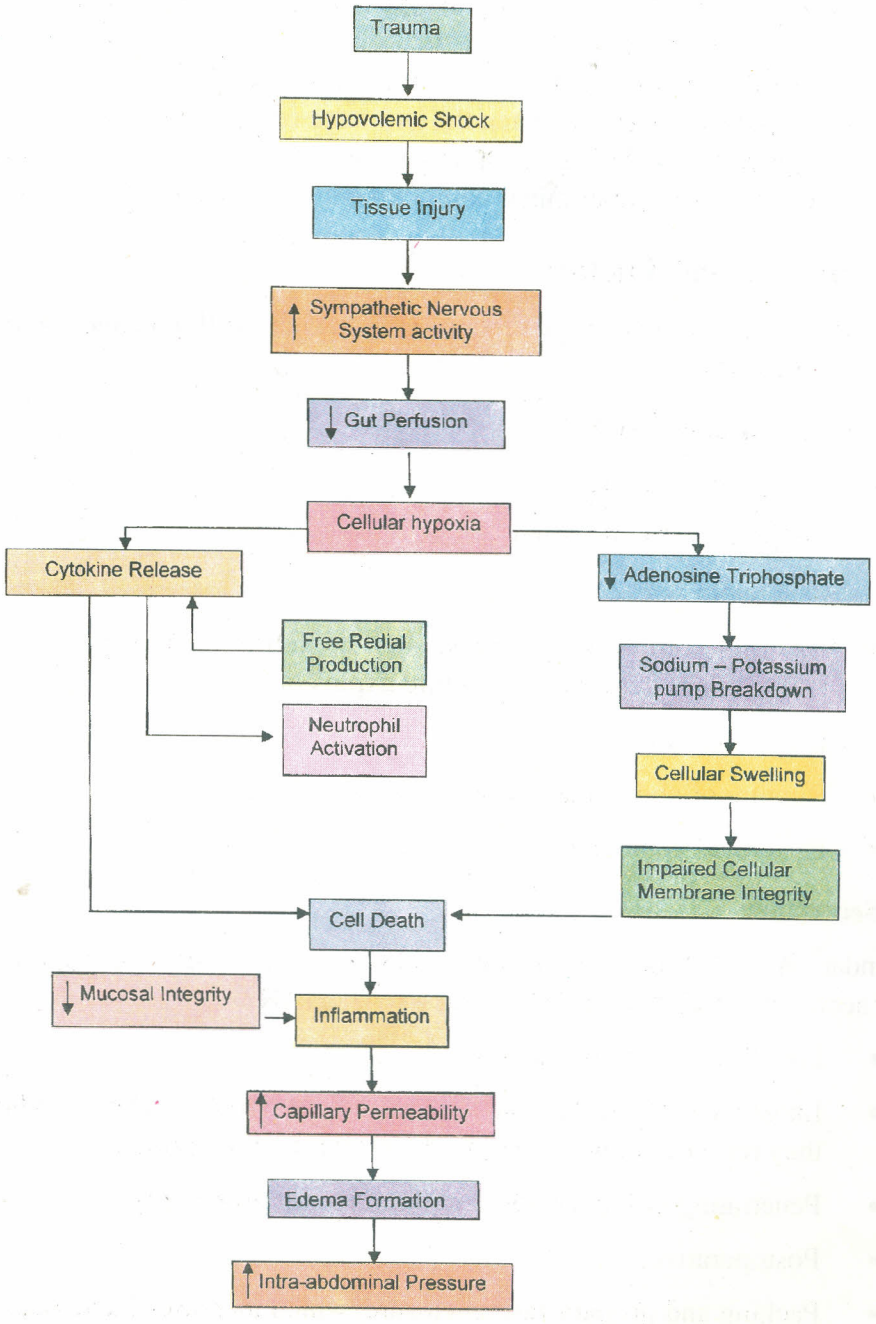

Fig.2.2: Pathophysiology changes in intra abdominal compartment syndrome

The increase in pressure within the abdomen has both direct and indirect on organ perfusion. It causes progressive hypo perfusion and ischemia of the intestines and other peritoneal and retroperitoneal structures. In the kidney, there

may be direct compression of the renal artery or vein resulting in Patho physiological effects including release of cytokines, formation of oxygen free radicals, and decreased cellular production of adenosine triphosphate. These processes may lead to translocation of bacteria from the gut and intestinal edema, predisposing patients to multi organ dysfunction syndrome.

As pressure rises, abdominal compartment syndrome impairs not only visceral organs but also the cardiovascular and the pulmonary systems; it may also cause a decrease in cerebral perfusion pressure.

Abdominal compartment syndrome follows a destructive pathway similar to compartment syndrome of the extremity. Problems begin at the organ level with direct compression; hollow systems such as the intestinal tract and portal-caval system collapse under high pressure. Immediate effects such as thrombosis or bowel wall edema are followed by translocation of bacterial products leading to additional fluid accumulation, further increasing intra-abdominal pressure. At the cellular level, oxygen delivery is impaired leading to ischemia and anaerobic metabolism. Vasoactive substances such as histamine and serotonin increase endothelial permeability, further capillary leakage impairs red cell transport, and ischemia (See Fig.2.2).

2.4.5 Clinical Manifestations

Clinical manifestations of ACS are not only seen in the abdominal cavity, but are evident in all organ systems of the body. The pulmonary, cardiovascular, renal, gastrointestinal, neurological, and immune systems can all exhibit signs of dysfunction when IAH/ACS develops.

i) Pulmonary Manifestations

Increased IAP creates a restrictive effect on the lungs. The restrictive effect on the lung leads to reduced ventilation, decreased lung compliance, increase in airway pressures, and reduction in tidal volumes; the end result being respiratory acidosis.

ii) Cardiovascular manifestations

Increased IAP reduces cardiac output, increases central venous pressure, increases systemic vascular resistance, increases pulmonary artery pressure, and pulmonary artery wedge pressure. The rises in pressures can be misleading as the rises in pressures are not necessarily due to increased volume.

iii) Renal manifestations

Increased IAP can cause reduction in renal plasma flow and glomerular filtration rate. Most likely this is due to increase in renal vascular resistance and decreases in cardiac output, leading to oliguria.

iv) Gastrointestinal manifestations

IAP decreases abdominal perfusion pressure, decreases celiac blood flow, decreases superior mesenteric artery blood flow, decreases the blood flow to all intra-abdominal organs, and decreases mucosal blood flow. Enteral feeding becomes difficult, intestinal permeability increases and bacterial translocation can occur. There is an increased risk for gastrointestinal bleeding.

v) Neurological Manifestations

Increased IAP can cause obstruction of cerebral venous outflow, leading to vascular congestion and increased intracranial pressure. Decreased cardiac output, in combination with increased intracranial pressure lead to decreased cerebral perfusion pressure.

2.4.6 Diagnostic Assessments

Patients with following history and clinical parameters should be confirmed for the diagnosis of IAH/ACS by IAP.

History

- Increase in abdominal girth
- Difficulty in breathing
- Decreased urine output
- Syncope
- Malena
- Non-steroidal anti-inflammatory drug (NSAID) use
- Alcohol abuse
- Nausea and vomiting
- History of pancreatitis

Clinical Parameters

- Distended, tense abdomen
- IAP > 20 mm of Hg
- Elevated peak airway pressure
- Massive I.V. fluids requirements
- Renal insufficiency: Oliguria to anuria, not responding to volume repletion
- Decreased or diminished cardiac output with high filling pressure
- Hypoxemia refractory to increase F i O_2 and PEEP
- Hypercarbia
- Hypercapnia
- Wide pulse pressure
- Acidosis

Intra Abdominal Pressure (IAP)

Intra Abdominal Pressure is helpful to decide the severity of the condition and the need for decompression. Measurement is mostly accomplished through trans-vesical pressure measurements. Other methods are rectal route measurement, direct intra peritoneal measurement, and Intra gastric method (via a naso gastric tube).

a) **Direct intra peritoneal measurement** (using a peritoneal dialysis catheter)

Direct measurement via a peritoneal dialysis catheter is the most accurate measurement, but is not a realistic method for the majority of ICU patients since it is invasive and involves the insertion of an intra peritoneal catheter into the abdomen.

b) **Measurement via urinary catheter in the bladder**

IAP measurement is mostly accomplished through this method. The "gold standard" measurement in the ICU has been the urinary catheter connected to a pressure transducer monitoring set in order to get a bladder pressure measurement. Bladder pressure measurement is reflective of IAP and is measured in millimeters of mercury (mmHg).

Indications for IAP monitoring

- Post-operative abdominal surgery patients
- Patients with open or blunt abdominal trauma
- Mechanically ventilated patients with other organ dysfunction
- Patients with symptoms and signs consistent with ACS including oliguria, increased ventilatory requirements and unexplained acidosis.
- Patients with burns and massive fluid resuscitation.

Method

100 ml of sterile saline is instilled into the bladder via the aspiration port of the Foleys catheter with the drainage tube clamped. An 18-gauge needle attached to a pressure transducer is then inserted in the aspiration port, and the pressure is measured. The transducer should be zeroed at the level of the symphysis pubic. (Fig 2.3)

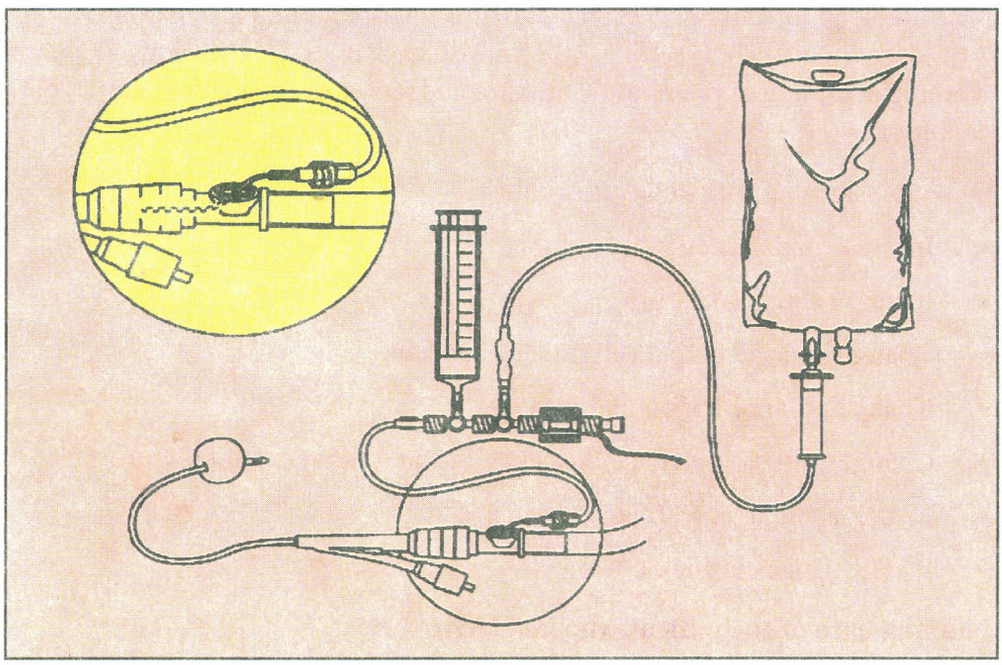

Fig 2.3: Intra abdominal pressure monitoring device

2.4.7 Therapeutic Management

The best way to treat ACS is to prevent it. Recognizing early signs and symptoms is the best form of treatment for ACS.

i) **Nonsurgical options** for management and treatment of IAH and ACS focus on reducing IAP:

   a) Improve abdominal wall compliance by using sedation and analgesia to reduce muscle tone, reducing abdominal muscle tone by initiating neuromuscular blockade, and keeping the head of bed in the lowest position possible.

   b) Evacuating intra-luminal contents by performing nasogastric decompression, rectal decompression or enemas, or administration of prokinetic motility agents.

   c) Evacuating abdominal fluid collections via per cutaneous decompression.

   d) Correcting positive fluid balance with fluid restriction, diuretics and colloids to mobilize the third-space edema, or intermittent or continuous hemodialysis/ultra filtration to remove fluids.

ii) **Surgical decompression** is a life-saving intervention when IAH is refractory to all other treatments and organ dysfunction is present. When surgical decompression is done the abdomen is left open and must be closed with a dressing. Temporary abdominal closure techniques are used to cover the abdomen until the condition subsides and definitive closure of the patient's abdomen is completed with either a skin graft or flap. Negative pressure wound therapy or iodine-impregnated plastic adhesive drape are two types of temporary abdominal closures that may be used.

2.4.8 Nursing Management

Nursing care involves vigilant monitoring for early detection, including serial measurements of intra-abdominal pressure. Intra-abdominal hypertension and ACS occur in many ICU settings (PICU, MICU, and SICU. Therefore, as a nurse working in these areas you should monitor and report any changes in the patient's condition.

- Vital signs i.e. BP, Pulse, Respiration
- Increase in tenseness of abdominal wall
- Increase abdominal girth
- Change in urine output (oliguria or anuria)
- Change in intra- abdominal pressure
- Changes in level of consciousness (Elevated intracranial pressure)
- Hypoxia and hypercarbia
- F i O_2Changes in Vision

Nursing care of the patient who has IAH/ACS

- Maintaining a patent airway
- Frequent abdominal assessments i.e., Abdominal Girth, Intra abdominal pressure

- Blood pressure monitoring
- Heart rate monitoring
- Pulse oximetry monitoring
- Monitoring of respiratory rate
- Hemodynamic monitoring: i.e., central venous pressure, pulmonary artery pressures, and cardiac output
- Fluid intake and urine output
- Electrolyte balance
- Maximal positioning for lower IAP, usually head of bed in the lowest position.

Check Your Progress 3

i) Intra abdominal pressure is

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ii) Intra abdominal compartment syndrome is defined as

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iii) Nursing assessment for anticipating the Intra abdominal compartment syndrome

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2.5 LET US SUM UP

In this unit we discussed about acute intestinal obstruction, perforative peritonitis, and abdominal compartment syndrome conditions.

Intestinal obstruction exists when blockage prevents the normal flow of intestinal contents through the intestinal tract. The obstruction can be partial or complete.

Its severity depends on the region of bowel affected, the degree to which the lumen is occluded and especially the degree to which the vascular supply to the bowel is compromised.

In perforative peritonitis, any part of the GI tract may become perforated from a variety of causes, releasing gastric or intestinal contents into the peritoneal space. Symptoms develop suddenly, with severe pain followed shortly by signs of shock. Diagnosis is usually made by the presence of free air in the abdomen on imaging studies. Treatment is with fluid resuscitation, antibiotics, and surgery. Mortality is high, varying with the underlying disorder and the patient's general health.

Abdominal compartment syndrome is a potentially lethal condition caused by any event that produces intra-abdominal hypertension Normal intra-abdominal pressure is 0-5 mm Hg. Physiologic compromise begins when the pressure rises above 8-10 mm Hg. Once the pressures increase beyond about 20 mm Hg irreversible tissue injury occurs, ultimately resulting in ACS and multiple organ failure. Early recognition of rising abdominal pressure is critically important because it allows prompt intervention which will prevent ACS from developing, thereby leading to a much better prognosis for the patient.

2.6 GLOSSARY

Colostomy : Surgical opening into the colon by means of a stoma to allow drainage of bowel contents

Ileostomy : Surgical opening into the ileum by means of a stoma to allow drainage of bowel contents

Obstipation : Pain with constipation

Perforation : Tear or hole

Paracentesis : Is the removal of fluid (ascites) from the peritoneal cavity through a small surgical incision made through the abdominal wall under sterile conditions

Intra-abdominal pressure (IAP) : The steady-state pressure concealed within the abdominal cavity expressed in millimeters of mercury (mmHg)

Abdominal perfusion pressure (APP) : Abdominal perfusion pressure (APP) = mean arterial pressure (MAP) – intra-abdominal pressure (IAP)

Intra-abdominal hypertension (IAH) : A sustained or repeated pathological elevation in IAP $\geq$ 12 mmHg. Grades of IAH are:

- Grade I: IAP 12-15mmHg
- Grade II: IAP 16-20mmHg
- Grade III: IAP 21-25mmHg
- Grade IV: IAP > 25mmHg
- Normal IAP = 5 to 7 mmHg
- meigs syndrome

2.7 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

i) a) adhesions and paralytic ileus.

b) intussception and dehydration

c) volvulus and varices

d) atresias and telangiectasia

ii) a) Clinical manifestations in small bowel obstruction are epigastric or umbilical abdominal cramping, Vomiting (sooner with proximal obstruction, Bilious if obstruction distal to pylorus), Obstipation, Diarrhea and flatus (partial obstructions) Hyperactive high pitched bowel sounds, Palpable loops of bowel .

b) Clinical manifestations in large bowel obstruction are milder with more gradual onset, Lower abdominal cramping, Increasing constipation with abdominal distention, Vomiting uncommon, if it is then feaculent, Palpable mass,Tympany with percussion.

iii) Immediate nursing management of a patient with intestinal obstruction is Assessment of patients pain and pain management, intestinal decompression, Fluid resuscitation, monitoring vital signs, abdominal girth, and urine output .preparing and facilitating for the diagnostic evaluation.

Check Your Progress 2

i) Main clinical manifestations in a patient of peritonitis are abdominal pain, tenderness and guarding nausea and vomiting, high fever, abdominal distension.

ii) Peritonitis can be prevented by early treatment of GI inflammatory conditions, avoiding over the counter medications, preoperative and postoperative antibiotic therapy after GI surgery prevents perforation and subsequent peritonitis.

Check Your Progress 3

i) IAP is the pressure within the abdomen. This pressure is normally very low, but can be increased during illness and injury due to swelling or internal bleeding. Normal IAP is <10 mm of Hg.

ii) Abdominal compartment syndrome is Organ dysfunction caused by intra-abdominal hypertension (IAH).

iii) Nursing assessment for anticipating the Intra abdominal compartment syndrome is measuring abdominal girth, urine output, vital signs, level of consciousness, central venous pressure, intra abdominal pressure.

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UNIT 3 HEPATIC DISORDERS – FULMINENT HEPATIC FAILURE AND HEPATIC ENCEPHALOPATHY

Structure

3.0 OBJECTIVES

After completing this unit, you will be able to:

- Enumerate the various causes of fulminant hepatic failure and hepatic encephalopathy;
- Identify the various signs and symptoms in a patient going into fulminant hepatic failure and hepatic encephalopathy;
- Discuss the pathophysiological changes leading to various signs and symptoms in a patient with fulminant hepatic failure and hepatic encephalopathy;
- Describe role of critical care nurse in the conservative management of a patient with fulminant hepatic failure and hepatic encephalopathy;

- Explain the role of nurse in the various diagnostic tests for a patient planned for liver transplantation;
- Describe the care of patient with liver transplantation; and
- Advice a patient to prevent the potential complications after orthotropic Liver transplantation.

3.1 INTRODUCTION

Fulminant hepatic failure (FHF) and hepatic encephalopathy are one of the most catastrophic and challenging gastrointestinal emergencies encountered in clinical practice and encompasses a pattern of clinical symptoms and pathophysiological responses associated with the rapid arrest of normal hepatic function. FHF carried a very high mortality rate, widely reported to be in excess of 80%. However, with an improved understanding and recognition of the syndrome, more aggressive medical therapy, intensive care and the advent of orthotropic liver transplantation (OLT) as a treatment option, survival rates have improved considerably. In this unit we shall discuss about definition, etiologic factors, pathophysiology clinical manifestations, diagnostic assessment therapeutic and nursing management of fulminant hepatic failure and hepatic encephalopathy.

3.2 FULMINANT HEPATIC FAILURE

We shall discuss various aspects of fulminant hepatic failure in following sub sections:

3.2.1 Definition

Fulminant hepatic failure refers to the rapid development of severe acute liver injury with impaired function of synthesis and encephalopathy often in association with coagulopathy, jaundice and multisystem organ failure in a person who previously had a normal liver or had well-compensated liver disease.

Several definitions of the time course for which liver failure should be considered Fulminant have been proposed:

- The development of encephalopathy within eight weeks of the onset of symptoms in a patient with a previously healthy liver.
- The appearance of encephalopathy within two weeks of developing jaundice, even in a patient with previous underlying liver dysfunction.

Fulminant or acute hepatic failure is a sudden and severe impairment of liver or hepatic functions due to massive necrosis of liver cells. It is characterized by severe liver dysfunction, hepatic encephalopathy, metabolic derangements, neurological complications and, ultimately, multi organ failure in a patient without preexisting liver disease.

Patients who have rapid deterioration of liver function with the development of encephalopathy within six months but fall outside the boundaries of the above time intervals are considered to have "subfulminant" hepatic failure.

Fulminant and subfulminant hepatic failure differs in their clinical features and prognosis. As an example, cerebral edema is common in fulminant disease and rare in subfulminant disease. In contrast, renal failure and portal hypertension are more frequently observed in patients with subfulminant hepatic failure.

3.2.2 Etiological Factors

The Etiological factors of fulminant hepatic failure may include following:

- **Viral hepatitis** (Hepatitis A and B) is the commonest cause accounting for 60-75% of all cases. All viral infections causing acute hepatits may be complicated by FHF. Other non-hepatotropic viruses, such as Epstein-barr virus, cytomegalovirus, adenovirus and herpes simplex virus can also cause FHF, but very rarely.
- **Vascular:** Right heart failure, Budd-Chiari syndrome, veno-occlusive disease, shock liver (ischemic hepatitis), heat stroke.
- **Metabolic:** Acute fatty liver in pregnancy, Wilson's disease, Reye's syndrome, galactosemia, hereditary fructose intolerance, tyrosinemia,
- **Miscellaneous:** Malignant infiltration (liver metastases, lymphoma), autoimmune hepatitis, sepsis.
- **Toxins:** Carbon tetrachloride, Phosphorus, Industrial cleaning solvents.
- **Hepatotoxic drug reactions** e.g., anaesthetic agents, nonsteroidal anti-inflammatory drugs, antidepressants, Acetaminophen in Tx doses with alcohol, Idiosyncratic reaction — halothane, sulfonamides, phenytoin, and others.
- Acute alcoholic hepatitis.
- Mushroom poisoning.
- Pregnancy complicated with eclampsia.

3.2.3 Pathophysiology

The Pathogenesis of fulminant hepatic failure is uncertain. But it is thought that **direct cytotoxic effects** and/or hyper immune response to antigen, co-infection with two or more viruses may be the initial events that, for still unknown reason, trigger rapid progress to liver failure instead of the usual recovery. Pathophysiologic alterations seen in FHF include. Cerebral oedema, due to alteration in permeability of blood brain barrier and passage of ammonia into the astrocytes (Brain cells). Damaged liver lack in gluconeogenesis, which leads to elevated level of insulin followed by hypoglycemia.

Liver damage alter the functions of synthesis and metabolism which gives rise to symptoms of toxemia ceagulopahty, portal hypertension, ascites, altered bowel and bladder function.

The Following flowchart will help to understand pathophysiological basic of FHF. Fig.3.1

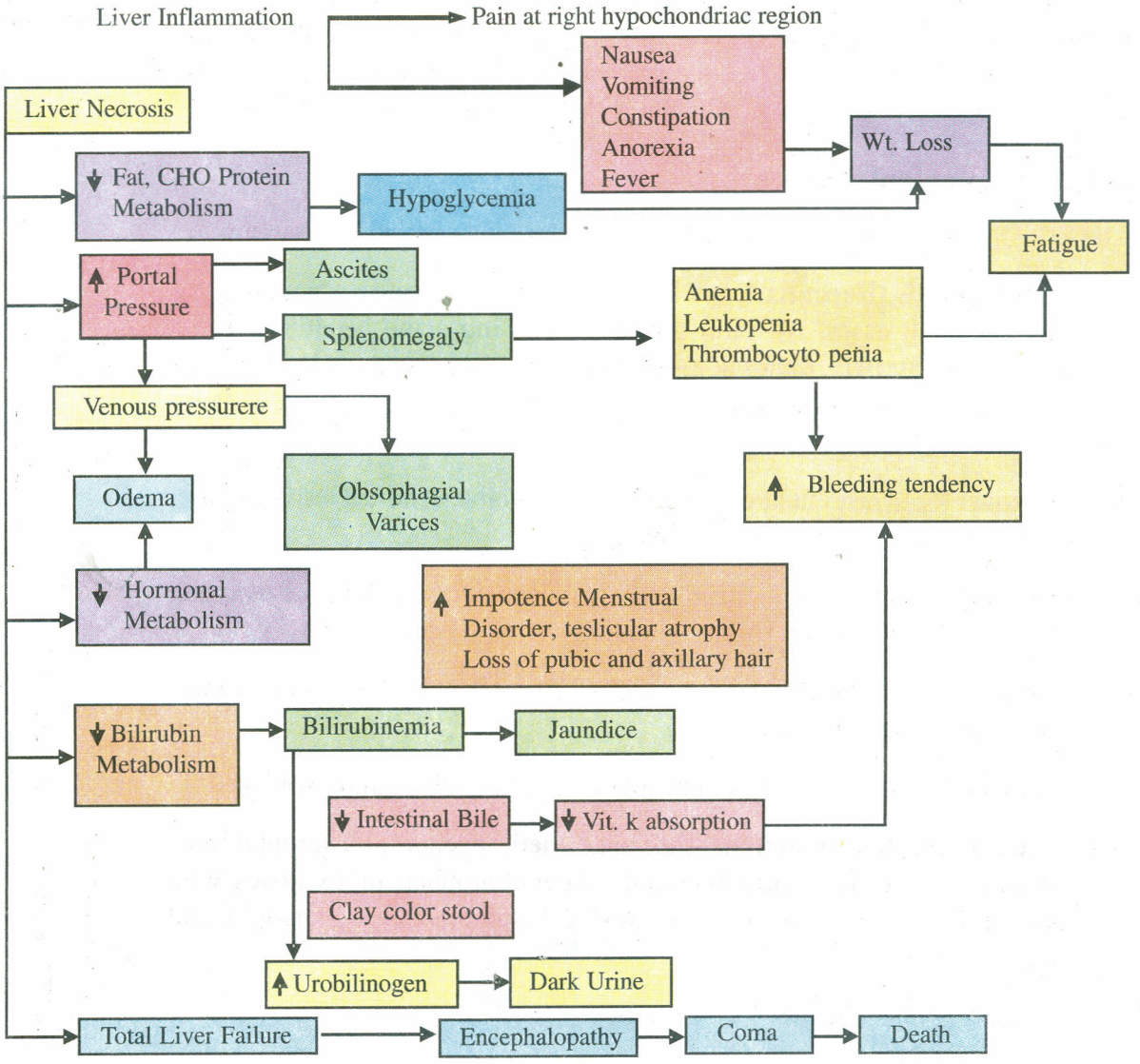

Fig. 3.1: Flowchart showing pathophysiology of hepatic failure

3.2.4 Clinical Manifestations

The clinical manifestations are given in following table

Subjective Findings	Objective Findings
Headache Dizziness Night mares	i) Altered mental status with agitation, uncooperative or violent behaviour, delirium, mania, somnolence and coma.
Jaundice Vomiting	a) Asterixis b) fetor hepaticus c) Small liver d) Decerebrate rigidity and posturing in late stages e) Abnormal pupil reflexes (usually dilated in late stages). f) Hypotension, tachycardia, arrhythmias g) Tachypnoea, respiratory arrest h) Elevated temperature i) Oliguria and uremia j) Abnormal bleeding k) Ascitis

3.2.5 Diagnostic Assessment

The diagnostic evaluation includes following:

i) Laboratory investigation of Blood for —

- Bilirubin **> 20 mg%**
- Transaminases usually high (>1000)
- INR prolongation, decrease in coagulation factors 5,7
- Ammonia — usually elevated
- Coagulopathy with prolonged PT, PTT
- Arterial blood Glas (ABG) analysis may reveal – alkalosis or acidosis
- BUN, Creatinine increased

ii) CT scan of liver may present decreased size with areas of necrosis.

iii) Percutaneous needle biopsy of the liver may reveal degree of hepatocellular necrosis

iv) Hepatic ultrasonography may demonstrate extra hepatic biliary obstruction and abnormal size of liver.

Other assessment include ECG, EEG, Chest X ray

3.2.6 Therapeutic Management

The essentials of management aims at:

- Diagnosis of cause of liver injury and encephalopathy.
- **Skilled intensive care** to minimize aggravating factors and complications until liver function recovers or transplantation can be performed.

Conservative Treatment

- Parenteral hydration with attention to maintain normal electrolytes balance,
- Prevention or correction of hypoglycemia.
- Nutritional support with protein restriction aiming at the reduction of urea synthesis.
- Neomycin and lactulose aimed to reduce the colonic production and absorption of ammonia.
- Prostacyclin (PGI2), a well-known vasodilator, may improve tissue hypoxia.
- Steroids.
- Coagulation abnormalities may be corrected with parenteral Vitamin K. In cases of no response to vitamin K, or suspicion of disseminated intravascular coagulopathy, fresh frozen plasma is indicated with or without platelet concentrate infusion. Plasma exchange has been suggested as another means that may correct homeostasis.
- The reduction of gastric acidity by early use of H2-antagonists is important to prevent stress-induced gastro-duodenal disease and subsequent hemorrhage.

- Cerebral edema is a major complication of increased ICP and a common cause of death. Early detection is the key for effective treatment. Patients at risk of increased ICP should be placed in a head-up position with elevation of the trunk about 20° above the horizontal. However, higher elevation (≥ 40°) possibly produces paradoxical elevation of the ICP. Monitoring of the ICP by extradural transducers allows not only early detection of increased ICP but also guidance of therapy to prevent brainstem herniation. This has considerable implications for transplantation. Mannitol bolus reduces ICP and more importantly increases the overall survival rate.
- Antibiotics are frequently administered to combat infection.
- **Artificial liver support devices** are designed not only to remove circulating toxins but also to perform complex metabolic functions. In this Extracorporeal Liver Assist Devices (ELAD) the blood of the patient is perfused through fiber cartridge containing living hepatocytes.
- Hepatic regeneration by growth factors stimulating hepatocyte proliferation, especially insulin and glucagon are reported to be successful.
- **Mediator therapy:** Immunotherapies, selective bowel decontamination and liver dialysis system are under trial.

Liver transplantation: Despite all these medical measures, the mortality rate remains very high varying from 50-85%. This has led to the introduction of **Orthotropic Liver transplantation (OLT)** which reversed the prognosis with survival rates from 60-80%.

Guidelines to Select Patients for Orthotropic Liver transplantation (OLT) :

These criteria include:

a) **Prothrombin time > 100 seconds or any three of the following:**
   - Age <10 or > 40 years.
   - Non-A, Non-B hepatitis, halothane or other drug related etiology.
   - Duration of jaundice before onset of encephalopathy > 2 days .
   - Prothrombin time > 50 seconds. 5) Serum bilirubin > 20 mg/dl.

b) **In Paracetamol induced FHF:**
   - pH> 7.3 or
   - Prothrombin time > 100 seconds and
   - Creatinine > 2 mg/dl. 4) Grade III or IV encephalopathy.

c) **Other Criteria included are as follows:**

Factor V < 20% or < 30% in patients below 30 years of age with confusion or coma.

These criteria although still widely used were found to be poor when tested in other centers. Accordingly, it has been suggested that every patient with FHF should be evaluated for OLT. If the need for transplantation becomes clear or persists, the operation should proceed.

Contraindications: Contraindications to liver transplantation can be divided into those that are absolute and those that are relative, i.e., those which are expected to complicate and increase the risk of transplantation. Absolute contraindications to liver transplantation include following:

- AIDS or HIV positivity
- Irreversible brain damage
- Multi-system failure that is not correctable by liver transplantation
- Malignancy outside the liver (not skin cancer)
- Infection outside the hepatobiliary system
- Active alcohol or substance abuse
- Advanced cardiopulmonary or other systemic disease.

Factors that increase the risk of liver transplantation:

These include —

- Advanced age
- Advanced chronic renal failure
- Cholangiocarcinoma
- Chronic hepatitis B virus infection
- Hepatocellular carcinoma
- Hypoxemia from intrapulmonary shunts.

3.2.7 Nursing Management

i) Assessment

You have to do thorough assessment of all the body systems with special emphasis on metabolic dysfunction involving liver, pancrease and biliary system by keeping in mind that you have gathered objective and subjective data (refer 3.2.3) for prompt and accurate diagnosis and interventions.

ii) Nursing Diagnoses

Fulminant Hepatic failure

- Ineffective breathing pattern related to decreased lung expansion
- Impaired gas exchange related to ventilation / perfusion mismatching or intrapulmonary shunting
- Decreased cardiac output related to alterations in preload
- Decreased intracranial adaptive capacity related to failure of normal compensatory mechanisms
- Ineffective renal tissue perfusion related to decreased renal blood flow
- Disturbed body image related to actual change in body structure, function or appearance
- Deficient knowledge about discharge regimen related to lack of previous exposure to information
- Potential for bleeding related to impaired synthesis of clotting factors.

iii) Intervention

While you are taking care of patient with FHF, you have to keep in mind that your nursing priorities are directed towards the following points :

i) Protecting the patient form injury

ii) Providing comfort and emotional support

iii) Maintaining surveillance for complications and

iv) Educating the patients and family for effective home care.

In order to fulfill the above mentioned priorities you have to carry out following nursing interventions for providing care to the patients:

- Assess LOC
- Assess respiratory status and initiate measures to prevent complications
- Elevate head of the bed to at least 30 degrees, change position every 2 hours
- Assess signs of increased intracranial pressure and elevate the head by 30 degree to normalize ICP and administer medications as advised like mannitol, steroids etc.
- If there are signs of coagulopathy you should prevent injury during nursing procedures
- Checking of vital signs (BP, Pulse, Respiration and Temperature)
- Blood sugar level need to be checked every 3-6 hours
- Maintain normal BP, and CVP
- Administer Fluid and electrolytes as advised
- Monitor urine output hourly
- Lactulose-to produce 3-4 loose stools per day
- Administer medications as advised like Cimetidine, Vitamin K, Preventive antibiotics.
- Give Lactulose enemas to reduce ammonia level as advised
- Measure abdominal girth to assess the degree and abdominal distention / ascites.
- Use of benzodiazepines and other sedatives to be discouraged, because of the "masking" of pertinent neurological changes.

Check Your Progress 1

i) Define Fulminant hepatic failure.

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ii) List the absolute Contraindications for the orthotropic liver transplantation

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3.3 HEPATIC ENCEPHALOPATHY

Hepatic encephalopathy is considered to be a reversible metabolic encephalopathy, which occurs as a complication of hepatocellular failure.

Hepatic encephalopathy refers to the changes in the brain that occur in patients with advanced acute or chronic liver disease. If liver cells are damaged, certain substances that are normally cleansed from the blood by the healthy liver are not removed (ammonia mainly, and other toxins). Nurses working in ICU settings should be aware of the different precipitant to hepatic encephalopathy so that specific measures can be initiated to prevent the patient from going into encephalopathy and hence aiding in the early recovery of the patient.

3.3.1 Definition

Hepatic encephalopathy is a syndrome observed in patients with cirrhosis of the liver. It is a central nervous system dysfunction resulting from liver disease; frequently associated with elevated ammonia levels producing changes in mental status, altered level of consciousness and coma.

Hepatic encephalopathy may develop in patients without cirrhosis who have undergone portocaval shunt surgery.

Classification /Categorization of Hepatic encephalopathy: Hepatic encephalopathy can be divided into three Categories:

i) Encephalopathy associated with acute Liver Failure.

ii) Encephalopathy associated with porto-systemic bypass and no intrinsic hepatocellular disease.

iii) Encephalopathy associated with cirrhosis and portal hypertension.

3.3.2 Etiological Factors

Two Proposed theories of hepatic encephalopathy are as follows:

a) Ammonia hypothesis

Ammonia is produced in the gastrointestinal tract by bacterial degradation of amines, amino acids, purines, and urea. Normally, ammonia is detoxified in the liver by conversion to urea and glutamine by the Krebs-Henseleit cycle. In liver disease or in the presence of Porto systemic shunting, the portal blood ammonia is not efficiently converted to urea. Increased levels of ammonia may enter the systemic circulation because of Porto systemic shunting.

b) (Glutamine amino butyric acid) GABA hypothesis

GABA is a neuro inhibitory substance produced in the gastrointestinal tract. Of all brain nerve endings, 24-45% may be GABAergic. Increased GABAergic tone is observed in patients with cirrhosis, perhaps because of decreased hepatic metabolism of GABA.

c) Other Precipitating etiological factors are as follows:

Metabolic disturbances may precipitate hepatic encephalopathy. Common among them are hyponatremia (often arising as a result of diuretic treatment or simply as a complication of the edema typically found in advanced cirrhosis), hypokalemia (again, often as a result of diuretic use), alkalosis, dehydration, hypoglycemia (a condition to which people with cirrhosis are susceptible).

- **Renal failure:** Renal failure leads to decreased clearance of urea, ammonia, and other nitrogenous compounds.
- **Infection:** Infection may predispose to impaired renal function and to increased tissue catabolism, both of which increase blood ammonia levels.
- **Constipation:** Constipation increases intestinal production and absorption of ammonia.
- **Blood transfusions** may result in mild hemolysis, with resulting elevated blood ammonia levels.
- **Diuretic therapy:** Decreased serum potassium levels and alkalosis may facilitate the conversion of NH_4^+ to NH_3.
- There are several **medications** the use of which may lead to hepatic encephalopathy. These include benzodiazepine (e.g., diazepam, lorazepam), narcotics, and diuretics. Alcohol ingestion, (whether or not it was the cause of the patient's liver disease), may also precipitate hepatic encephalopathy.
- In patient of **non-compliance with dietary protein restriction.**
- **G I Bleeding** may also lead to hepatic encephalopathy. Blood contains large quantities of protein in the form of plasma proteins and hemoglobin. Hence, the presence of blood in the stomach or small intestine represents a protein load which, as a result of bacterial metabolism in the lumen of the gut, is converted to potentially toxic products such as ammonia.
- **Certain surgical procedures:** For example, operations to relieve pressure in the portal vein by connecting it to the splenic vein or other systemic venous vessels have the effect of diverting incoming intestinal venous blood away from the liver. This means that such ammonia-carrying blood will not be able to be "purified" by the liver. Encephalopathy can result. In a similar manner, the more-recently-developed "TIPS (transjugular intrahepatic portosystemic shunt" procedure (as you have learned in previous unit) often precipitates hepatic encephalopathy.

3.3.3 Pathophysiology

Due to the presence of scarring within the liver, cirrhosis leads to obstruction of the passage of blood through the liver, causing portal hypertension (Fig.3.2). This means it is difficult for blood from the intestines to go through the liver to get back to the heart. Portal-systemic anastamoses ("shunts") develop, and portal

blood (from the intestinal veins) will bypass the liver and return to the heart via another route without undergoing first-pass detoxification by the liver.

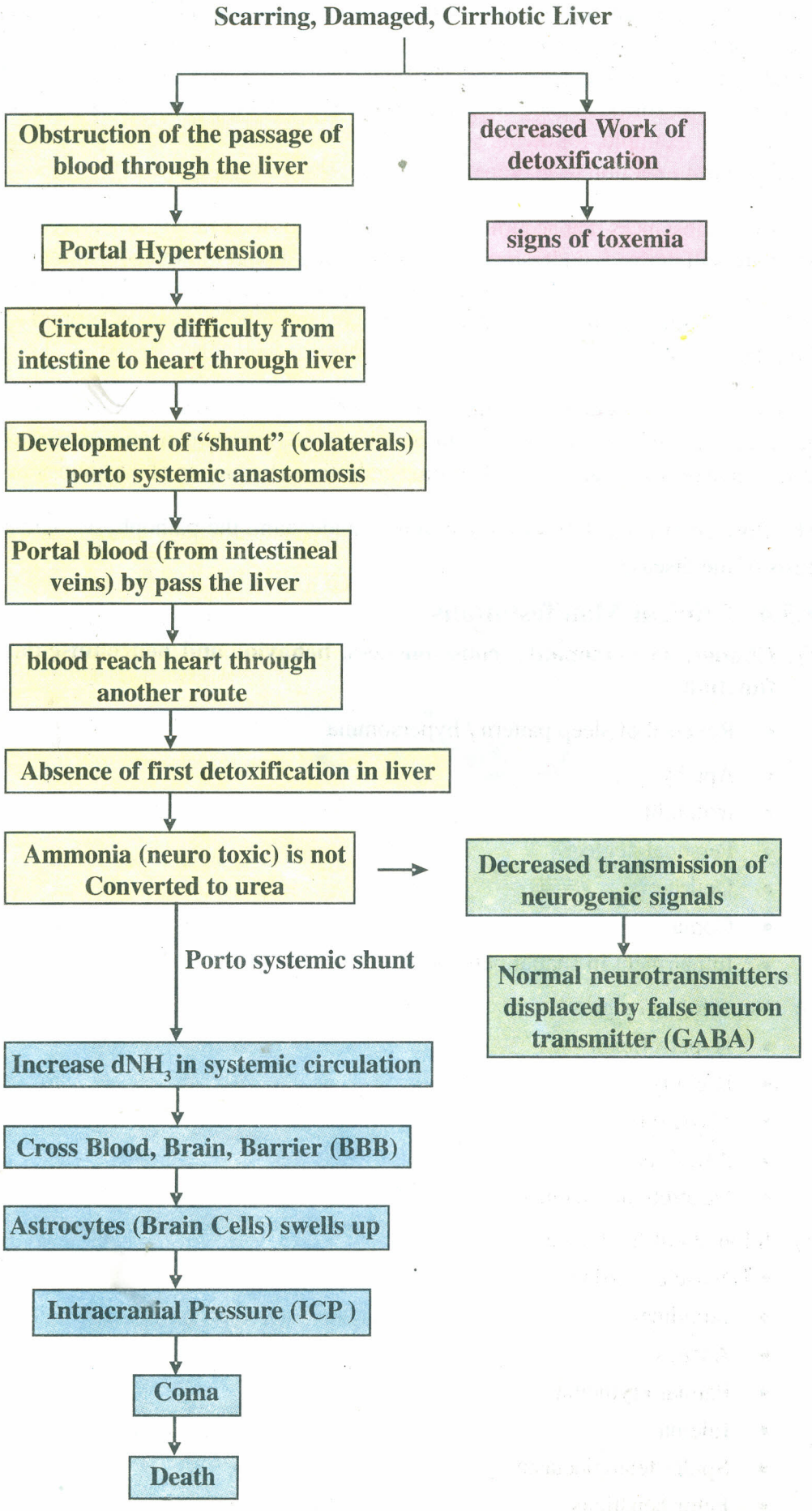

Fig. 3.2: Flow chart of Pathophysiology hepatic encephalopathy

Furthermore, in cirrhosis and other forms of liver disease, the damaged liver will not be functioning as well as it should be, so even blood that does travel through the liver may not be as detoxified as it otherwise would be. In fact, if the degree of liver damage and malfunction is severe, then, even in the absence of portal hypertension and the consequent bypassing of the liver by blood coming in from the intestines, hepatic encephalopathy will still occur. Such may well be the case, for example, following severe injury due to acetaminophen poisoning or acute viral infection (e.g., hepatitis A).

The toxic substances that accumulate in the setting of liver failure and affect the brain are still not well understood. They have been thought to include ammonia (NH_3) and **mercaptans**. Ammonia is normally converted to urea by the liver and, as with **mercaptans**, is produced by the bacterial breakdown of protein in the intestines.

Ammonia can cross the blood-brain barrier, where it causes the support cells of the brain (astrocytes) to swell. The swelling of the brain tissue increases intracranial pressure, and can lead to coma or death via herniation of the brainstem.

The flow chart (Fig. 3.2) will guide you to understand the pathophysiological basis of the disease.

3.3.4 Clinical Manifestations

a) **Changes of personality, consciousness, behavior and neuromuscular function**:

- Reversal of sleep pattern / hypersomnia
- Apathy
- Irritability
- Personal neglect
- Delirium
- Coma
- Impairment in spatial perception:

b) Neurological signs

- Hyperreflexia
- Rigidity
- Myoclonus
- Asterixis
- Decerebrate posturing

c) Stigmata of liver failure

- Muscle wasting
- Jaundice
- Ascites
- Palmar erythema
- Edema
- Spider telangiectasia
- Fetor hepaticus

Grading of Symptoms

The symptoms can be divided in to following four stages Fig 3.3

Stage 1 • Euphoria or depresión, mild confusion, slurred speech, irritability, decreased attention, disordered sleep rhythem, slight asterixis and normal EEG.

Stage 2 • Lethargy, moderate confusion, marked asterixis and abnormal EEG, personality changes, intermittent disorientation, suppressed level of conciousness but responsive to painful stimuli.

Stage 3 • Marked confusion incoherent speech, (sleeping but arousable) gross disorientation, asterixis present and abnormal EEG.

Stage 4 • Coma, initially responsive to noxious stimuli later unresponsive asterixis absent and abnormal EEG, decerebrate posturing.

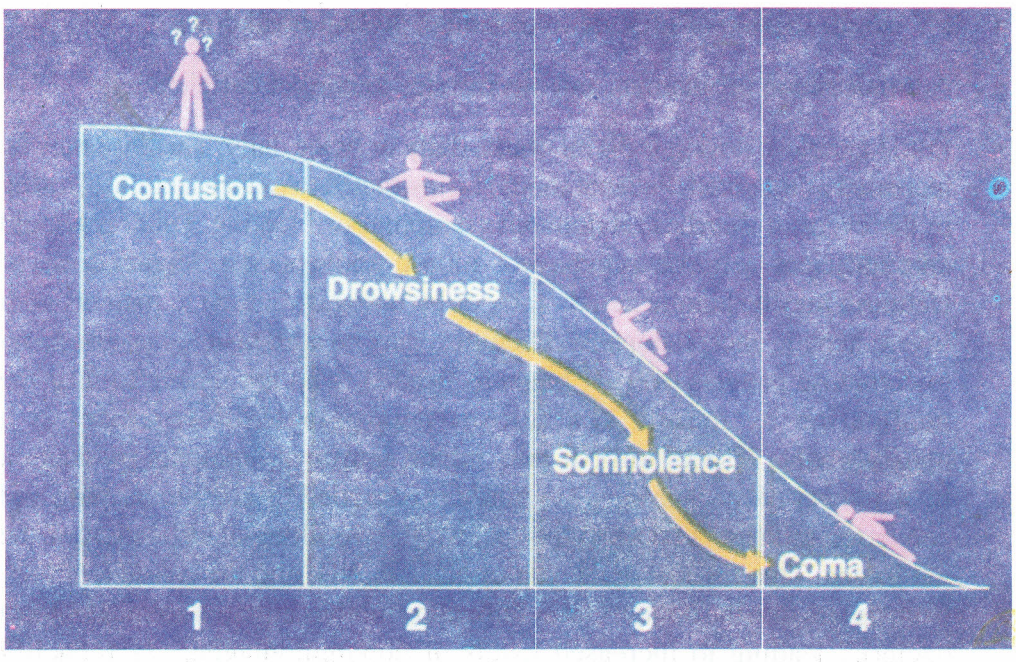

Fig 3.3: Stages of Hepatic encephalopathy

3.3.5 Diagnostic Assessment

- History and physical examination
- Blood examination for the following:
  - o Ammonia – Increase serum level
  - o Liver Enzymes – Increase
  - o ABG analysis – metabolic acidosis
  - o Serum albumin – decrease with progressive liver disease
- EEG Shows increasing Amplitude with decreasing frequency and the development of triphasic waves in late stage of coma
- MRI of brain to exclude intracranial lesion
- CT scan of liver as well as brain
- Needle biopsy of liver – to demonstrate level of necrosis
- Hepatic USG – will rule out extrehepatic obstruction if any.

3.3.6 Therapeutic Management

The goal of management would be focused on mainly to:

- Reduce the protein break down
- Reduce the level of serum ammonia as rapid as possible
- Restore the normal functioning of other systems
- Minimize the complications.

Conservative Medical Treatment

To achieve the above mentioned goals, following interventions need to be carried out:

- I/V administration of glucose to minimize protein breakdown.
- Administration of vitamins and electrolytes to correct deficiencies.
- Initial lactulose dosing is 30 ml orally, daily or twice daily. The dose may be increased as tolerated. Patients should be instructed to reduce lactulose dosing in the event of diarrhea, abdominal cramping, or bloating. Patients should take sufficient lactulose as to have 2-4 loose stools per day. Higher doses of lactulose may be administered orally or by nasogastric tube to patients with severe hepatic encephalopathy. Lactulose may be administered as an enema to comatose patients every 4 hours as needed. who are unable to take the medication by mouth. (Lactulose (beta-galactosidofructose) is a nonabsorbable disaccharide. It may inhibit intestinal ammonia production by a number of mechanisms. Lactulose is degraded by colonic bacteria and converted to lactic acid and other acids, with resulting acidification of the gut lumen. This favors conversion of NH_4^+ to NH_3 and the passage of NH_3 from tissues into the lumen. Gut acidification inhibits ammoniagenic coliform bacteria, leading to increased levels of nonammoniagenic lactobacilli. Lactulose works as a cathartic, reducing colonic bacterial load).
- **Antibiotics,** such as metronidazole, oral vancomycin, paromomycin, and oral quinolones, are administered in a effort to decrease the colonic concentration of ammoniagenic bacteria.
- **Ornithine-aspartate** lowers serum ammonia levels through stimulation of the urea cycle and urea formation.
- **Sodium benzoate** reacts with glycine to form hippurate, increases ammonia excretion in urine.
- Osmotic diuretics to reduce cerebral oedema
- **Serum ammonia levels** are measured daily.

3.3.7 Nursing Management

i) Assessment

As you know, a prompt and accurate nursing assesment is the pillar of effective management. So the following box will help you identify the severity of disease by analyzing the key clinical feautres of HE.

Box 3.2: Staging of Hepatic Encephalopathy alongwith signs and sympotms to be assessed

Stage 1	• Euphoria or depresión, mild confusion, slurred speech, irritability, decreased attention, disordered sleep rhythm, slight asterixis and normal EEG.
Stage 2	• Lethargy, moderate confusion, marked asterixis and abnormal EEG, personality changes, intermittent disorientation, suppressed level of conciousness but responsive to painful stimuli.
Stage 3	• Marked confusion incoherent speech, (sleeping but arousable) gross disorientation, asterixis present and abnormal EEG.
Stage 4	• Coma, initially responsive to noxious stimuli later unresponsive asterixis absent and abnormal EEG, decerebrate posturing.

Along with this you need to assess following parameters:

A daily record of handwriting and performance in arithmetic is kept to monitor mental status.

Checking of Vital signs i.e., blood pressure, pulse, respiration and temperature.

ii) Nursing diagnosis

- Altered level of sensorium related to cerebral oedema secondary to NH_3 accumulation and disease process.
- Activity intolerance related to disease process.
- Deficient fluid volume related to bleeding, decreased intake.
- Altered nutrition less than body requirement related to disease process.
- Ineffective breathing pattern related to ascites.
- Potential for altered tissue perfusion related to disease process.
- Potential for infection.

iii) Intervention

You need to carry out following activities to provide nursing care to critically ill patients with hepatoencephaopathy:

- Monitor levels of consciousness, mood and cognitive functioning and orientation.
- Maintain patent airway to prevent hypoxia
- Maintain fluid and electrolyte balance
- Meet the nutritional requirement (less protein and high carbohydrate diet is given)
- Maintain fluid intake and output record. Body weight is recorded daily.
- Note the frequency and quality of stools
- Watch the signs of internal bleeding
- Encourage bowel cleansing

- Protect the patient from self injury
- Watch the signs of infection maintain aseptic technique while attending the patient; care of urinary catheter, personal hygiene, and care of tubing etc.
- Protect renal functions by – restricting Sodium intake, monitoring and maintaing fluid intake.
- Follow-up advice
- Give advise related to the following aspects of care:
  - o Medications as advised
  - o Diet as advised. Use of vegetable rather than animal proteins
  - o Preventing Infection
  - o Avoiding over the counter medications
  - o Frequent liver enzyme studies.

3.4 COMPLICATIONS

You may find the following complications when your patient is suffering from HE

- Ascitis,
- Jaundice,
- Hepato-renal syndrome.

Check Your Progress 2

i) Define hepatic encephalopathy.

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ii) List the different precipitants for hepatic Encephalopathy.

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3.5 LET US SUM UP

Fulminant hepatic failure is a complex clinical syndrome that has a diverse etiology, an unpredictable course, and a high mortality rate. The acutely ill patients admitted to the ICU with this condition often presents a challenge to the intensive care staff because of the life-threatening, multi-system complications.

Hepatic encephalopathy is a common complication of liver cirrhosis occurring in up to 80% of patients and is characterized by neuropsychiatric complications ranging from slight altered mental status to coma. While the underlying mechanisms remain poorly understood with several proposed hypothesized theories, the main stay of treatments is aimed at identifying and treating the precipitating causes or reducing intestinal ammonia production. In this unit we have focused on definition,etiology pathophysiology,clinical manifestations, diagnostic assessment, therapeutic management and nursing management.The various flow charts have been presented through out the unit to clarify the content.

3.6 GLOSSARY

Orthotropic liver transplantation : Grafting of a donor liver into the normal anatomic location (with removal of diseased native liver)

ICP : Intracranial pressure

Cirrhosis : A chronic liver disease characterized by fibrotic changes and the formation of dense connective tissue with in the liver, subsequent degenerative changes and loss of functioning cells.

Portal Hypertension : Elevated pressures in the portal circulation resulting from obstruction of venous flow into and through the liver

Asterixis : Flapping tremors of the hands

Fetor hepaticus : Characteristic breath odor like acetone or freshly mowed grass

Myoclonus : Twitching or cloning spasm of a muscles

Spatial perception defect : This can be made apparent by noting the patient's poor ability to copy or draw various simple images, e.g., cube, star, clock. This deficit can also be demonstrated by administering a test that has the patient connect a number of randomly-placed dots on a sheet of paper (the "trail test" or "numbers connecting test".

3.7 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Fulminant hepatic failure is a clinical syndrome developing as a result of massive necrosis of liver cells, characterized by severe metabolic derangements, neurological complications and, ultimately, multi organ failure.

2) Absolute Contraindications for the orthotropic liver transplantation are AIDS or HIV positivity, irreversible brain damage, multi-system failure that is not correctable by liver transplantation, malignancy outside the liver, infection outside the hepato-biliary system, active alcohol or substance abuse, advanced cardiopulmonary or other systemic disease.

Check Your Progress 2

1) Hepatic encephalopathy is impaired brain function due to advanced liver damage, occurring when the liver can no longer effectively filter toxins from the bloodstream.

2) Precipitants for hepatic encephalopathy can be any metabolic disturbance, several medications, infections, bleeding into the stomach or small intestine, portal hypertension etc.

3.8 FURTHER REFERENCES

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UNIT 4 ACUTE PANCREATITIS

Structure

4.0 OBJECTIVES

After completing this unit, you will be able to:

- Enumerate the various causes of acute Pancreatitis;
- Explain the pathophysiological changes leading to various signs and symptoms in Pancreatitis;
- Discuss the role of nurse in various diagnostic tests;
- Describe the role of nurse in collaborative management of a patient with Pancreatitis using nursing process; and
- Advise patients to prevent the potential complications after pancreatitis.

4.1 INTRODUCTION

In units 1, 2 and 3 you have learnt about acute gastric, intestinal and hepatic conditions. In this unit you will learn about acute pancreatitis The pancreas is a

gland which produces two main types of substances; digestive juices and digestive hormones. Digestive juices include enzymes and bicarbonate. They travel through a small tube called the pancreatic duct to the small intestine (duodenum). There, the enzymes break down the proteins and fats in the foods that we eat to permit the nutrients to be absorbed. The bicarbonate neutralizes stomach acid. Digestive hormones, mainly insulin and glucagon, are released into the bloodstream. They control the body's blood sugar, a major source of energy.The pancreas may get inflamed due to various reasons. Once the gland becomes inflamed, it may lead to swelling of the gland and surrounding blood vessels, bleeding, infection, and can thus damage the gland. The digestive juices become trapped and start "digesting" the pancreas (autodigestion of the gland) itself. If this damage persists, the gland may not be able to carry out normal functions. Pancreatitis may be acute (short-term) or chronic (long-term). Acute pancreatitis can be a life-threatening illness with severe complications. Most people with acute pancreatitis recover completely from their illness. The pancreas returns to normal with no long-term effects. Pancreatitis may however reoccur, if the underlying cause is not eliminated.

Patients with acute severe pancreatitis may be admitted in ICU directly .The patients may develop pancreatitis as a complication of another disease, surgical procedure, or trauma. It accounts for 10% of all patients with episodes of pancreatitis. The patients with severe pancreatitis usually die as a result of infection, pulmonary complications or hemodynamic collapse. As a critical care nurse you have to be prepared to provide quality care to such patients.This unit will focus on definition, etiology, pathophysiological changes, clinical manifestations, diagnostic assessment, therapeutic and nursing management. The complications of pancreatitis have also been discussed.

4.2 DEFINITION

Acute pancreatitis is an inflammation of the pancreas that occurs suddenly and usually resolves in a few days with treatment. Acute pancreatitis can be a life-threatening illness with severe complications and varies from mild edema to severe hemorrhagic necrosis. The severity of the disease varies according to the extent of pancreatic destruction.

4.3 ETIOLOGICAL FACTORS

- Alcohol abuse and gallstones are the two main causes of pancreatitis, accounting for 80%-90% of all cases. The remaining 10%-20% of cases of pancreatitis have various causes, including the following:
- Certain medications like corticosteroids, thiazide diuretics, oral contraceptives, NSAIDS etc. are associated with increased incidence of pancreatitis.
- Exposure to certain chemicals
- Certain metabolic disorders such as hyperparathyroidism, hyperlipidemia,
- After surgical procedures on pancreas, stomach, duodenum or biliary tract,and after ERCP:

- Viral infections like post-mumps complication, coxsackie B virus
- Calculus, hepato-biliary disease
- Ischemia of the pancreas as seen after cardiopulmonary bypass
- In some cases the cause can be idiopathic

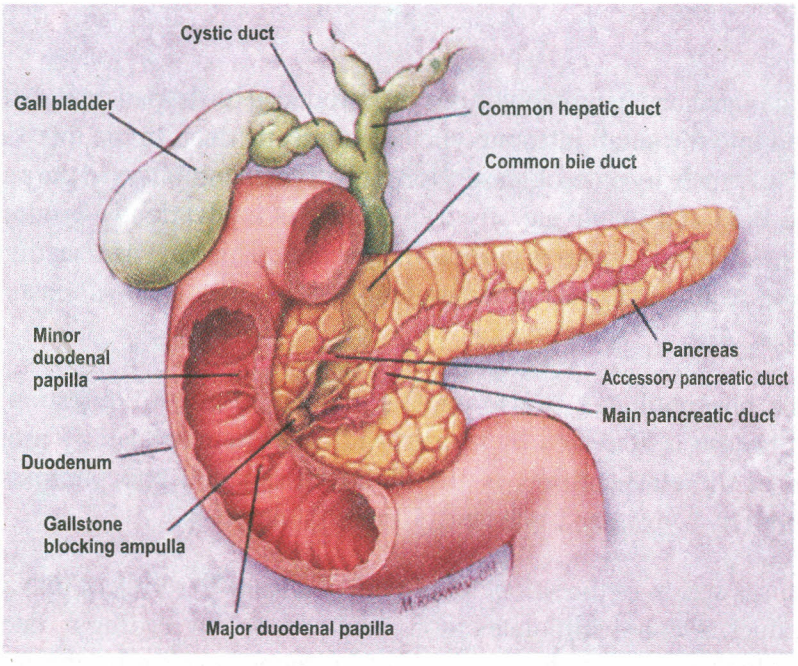

Fig 4.1: Inflammed pancreas due to Gall stone Blocking ampulla

You can use the Ranson's criteria for a quick assessment of the severity of pancreatitis.

Ranson's Criteria

On Admission:

- Age > 55 yrs.
- WBC > 16,000/mm
- Glucose > 200 mg/
- AST > 250 u/ml
- LDH > 350 u/l

Within 48 hrs of admission:

- Decreased HCT<10%
- Increased BUN > 5 mg/dl
- Decreased Serum Ca < 8 mg / dl
- Base Deficit > 4mEq / l
- Fluid sequestration > 6 l
- PO_2 < 60 mm hg.

Severity Assessment

- 2 or less signs = 1% mortality
- 3 to 4 signs = 15% mortality
- 4 to 6 signs = 40% mortality
- > 6 signs = 100% mortality

4.4 PATHOPHYSIOLOGICAL CHANGES

Pancreatitis is caused by the autodigestion of the pancreas by its own enzymes. One possible theory believed for Pancreatitis is the reflux of bile acids into the pancreatic ducts through an open or distended sphincter of oddi. This reflux may occur because of gall stones impacted at ampula of vater, atony or edema of sphincter.

Trypsinogen is an inactive proteolytic enzyme produced by the pancreas. Normally it is released into the small intestine via the pancreatic duct. In the intestine it is inactivated to trypsin by enterokinase. Normally trypsin inhibitors in the pancreas and plasma bind and inactivate any trypsin that is inadvertently produced. In pancreatitis, activated trypsin is present in pancreas. This enzyme can digest the pancreas and it also activates other proteolytic enzymes such as elastase and phospholipase A.

Elastase and phospholipase A also plays a major role in auto digestion of the pancreas. Elastase is activated by trypsin and cause hemorrhage by producing dissolution of the elastic fibres of the blood vessels and phospholipase A is activated both by trypsin and bile acids causes fat necrosis.

Alcohol causes spasm of the sphincter of oddi and leads to reflux of bile acids in pancreatic duct. Alcohol stimulates more acid production leading to decreased gastric ph which in turn leads to release of hormone secretin by the intestine, ultimately stimulating pancreatic secretions.

Thus pathological changes are because of premature activation of proteolytic and lipolytic pancreatic enzymes which are normally activated in the duodenum which leads to auto digestion of pancreas.

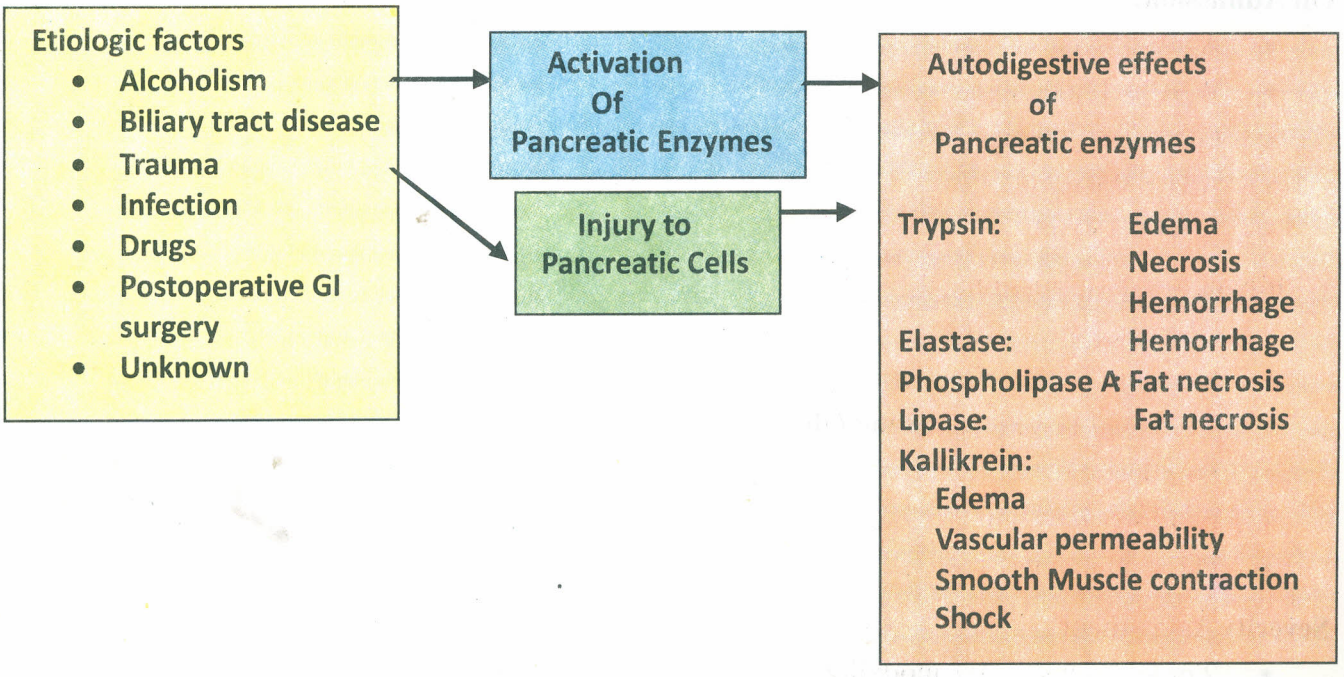

Fig 4.2: Flow chart of Pathological changes in pancreatitis

4.5 CLINICAL MANIFESTATIONS

- Patient will complain of abdominal pain in mid epigastric region radiating to back, because of retroperitoneal location of the pancreas. Pain has sudden onset and is severe, deep, piercing and continuous. It is aggravated by eating and is not relieved by vomiting.
- Nausea (Some people do vomit, but vomiting does not relieve the symptoms.)
- Low grade fever
- Decreased bowel sounds
- Hypotension
- Rapid heartbeat (A rapid heartbeat may be due to the pain and fever, or it may be due to compensatory mechanism in a situations; if a person/patient is bleeding internally).
- Feeling of lightheadedness or fainting
- Intravascular damage because of circulating trypsin may cause areas of cyanosis and blue discoloration on the abdominal wall.
- Increased formation of kenin peptides like kallikerin, bradykenin causes increased capillary permeability and altered vasomotor tone.
- Hypovolemia can occur as a result of exudation of blood and plasma proteins into the retroperitoneal space.
- Shock may develop because of hemorrhage into the pancreas or toxemia from the activated pancreatic enzymes. in this case turners sign (bluish discoloration in flank) and cullar's sign (bluish discoloration in periumbilical region) are manifested.

4.6 DIAGNOSTIC ASSESSMENT

i) **Laboratory investigation of blood reveals increased level of following:**

- Serum amylase may elevate to levels greater than 200 U/L within 24 hours upto 7 to 14 days
- Increased Serum Lipase
- Urinary amylase may be increased to more than 3600 U/day
- Hyperglycemia
- Hyperlipidemia
- Hypocalcemia
- Increase LFT
- Increase WBC > 1000 /ml

ii) **Diagnostic imaging tests**

- Abdomen X ray
- CT Scan
- Ultrasound: if biliary stone is suspected
- Magnetic resonance cholangiopancreatography

4.7 COMPLICATIONS OF PANCREATITIS

The acute and late complications of pancreatitis are given below:

4.7.1 Acute Complications

- Shock,
- Hypocalcemia (low blood calcium),
- High blood glucose,
- Dehydration, and kidney failure (resulting from inadequate blood volume which, in turn, may result from a combination of fluid loss from vomiting, internal bleeding, or oozing of fluid from the circulation into the abdominal cavity in response to the pancreatic inflammation, a phenomenon known as Third Spacing).
- Respiratory complications are frequent and are major contributors to the mortality from pancreatitis. Some degree of pleural effusion is almost ubiquitous in pancreatitis. Some or all of the lungs may collapse (atelectasis as a result of the shallow breathing which occurs because of the abdominal pain). Pneumonitis may occur as a result of pancreatic enzymes directly damaging the lung, or simply as a final common pathway response to any major insult to the body (i.e., ARDS or Acute Respiratory Distress Syndrome).

4.7.2 Late Complications

Late complications include recurrent pancreatitis and the development of pancreatic pseudocysts. A pancreatic pseudocyst is essentially a collection of pancreatic secretions which has been walled off by scar and inflammatory tissue. Pseudocysts may cause pain, become infected, rupture and/or cause hemorrhage, and may press and block the structures such as the bile duct, thereby leading to jaundice. It may even migrate around the abdomen.

4.8 THERAPEUTIC MANAGEMENT

The objectives and details of therapeutic management is given in following subsections:

Objectives

Treatment is usually focused on relieving symptoms and preventing further aggravation of symptoms. So objectives for the treatment are:

- Relieve pain
- Prevention or alleviation of shock
- Reduction of pancreatic secretions
- Control of fluid and electrolyte imbalance
- Prevention or treatment of infection
- Removal of precipitating cause
- Preventing complications.

4.8.1 Conservative Treatment

- Medication for pain management using NSAIDS, anticholinergics, and histamine receptors and antagonist to reduce pancreatic secretion.
- Nil orally. No food or liquid should be given to patient by mouth for a few days. This is called bowel rest. By refraining from food or liquid intake, the intestinal tract and pancreas are given a chance to start healing.
- Nasogastric aspiration to reduce vomiting and preventing gastric secretions from entering duodenum which may stimulate pancreatic secretions.
- Fluid resuscitation through replacement of I/V fluids to maintain fluid and electrolyte balance, or fluid sequestration within bowel lumen. Continued fluid administration is necessary to prevent secondary organ dysfunction.
- Foleys catheterization if necessary
- CVP line care
- May require early intubation and respiratory support
- Require meticulous critical care
- Prophylactic use of antibiotics.

4.8.2 Endoscopic Cholangio Pancreatography(ERCP) and Surgical Procedures

The following surgical procedures are performed by the physician. You need to understand this to be able to provide appropriate nursing care —

After lightly sedating the patient and giving medication to numb the throat, an endoscope is inserted through the mouth, throat, and stomach into the small intestine. The physician guides the endoscope and injects a special dye into the pancreatic or bile ducts that help the pancreas, gallbladder, and bile ducts appear on the screen while X-rays are taken.

The following procedures can be performed using ERCP:

Sphincterotomy: Using a small wire on the endoscope, the muscle that surrounds the pancreatic duct or bile ducts is accessed and a tiny cut is made to enlarge the duct opening. When a pseudocyst is present, the duct is drained.

Gallstone removal: The endoscope is used to remove pancreatic or bile duct stones with a tiny basket. Gallstone removal is sometimes performed along with a sphincterotomy.

Stent placement: Using the endoscope, a tiny piece of plastic or metal that looks like a straw is placed in a narrowed pancreatic or bile duct to keep it open.

Balloon dilatation: Some endoscopes have a small balloon that is used to dilate, or stretch, a narrowed pancreatic or bile duct. A temporary stent may be placed for a few months to keep the duct open.

4.8.3 Surgical Debridement

Infected pancreatic tissue, abscess, or large areas of necrosis should be considered for early debridement. Percutaneous drainage of the pseudocyst is performed, and drainage tube is left in place. Necrosectomy is done in case patient is not improving with the conservative treatment.

4.8.4 Nutritional Support

Initially the patient is on NPO status to reduce the pancreatic secretion. Decision about. Total parental nutrition (TPN) verses enteral feedings must be made. Patient with paralytic ileus secondary to the pancreatitis or abdominal complications, require TPN. Enteral feeding distal to the Ampulla of Vater is well tolerated. When patient's condition improves, fluids are allowed, small frequent meals which are high in carbohydrates and low in fat, are given to patient.

4.9 NURSING MANAGEMENT

4.9.1 Assessment

Subjective and objective data is obtained regarding past health history, medication or surgery or other treatments. General and system wise objective data is taken. General data includes following:

- ABC
- Pain severity, relieving and aggravating factors
- Nausea and vomiting, signs of dehydration and electrolyte imbalance
- Impending signs of shock i.e. low blood pressure, tachycardia
- Signs and symptoms of infection
- Abdominal distension and tenderness
- Bowel sounds

4.9.2 Nursing Diagnosis

- Acute pain related to inflammation of pancreas and surrounding tissues
- Risk for imbalanced fluid volume related to vomiting, nasogastric suctioning, NPO status, fever, shifting of body fluids.
- Imbalanced nutrition related to inability to ingest or digest food or absorb nutrients, vomiting, nasogastric suctioning, NPO status.
- Ineffective breathing pattern related to abdominal distension, ascites, pain or respiratory complication.
- Risk for infection.
- Ineffective therapeutic management related to lack of knowledge of preventive measures, diet restrictions, and restriction of alcohol intake and follow up care.

4.9.3 Planning

- Reduce the degree of pain or relieve the pain
- Maintenance of fluid and electrolytes
- Maintenance of balanced nutrition
- Maintain effective breathing pattern
- Maintain effective therapeutic regimen.

4.9.4 Implementation

The nursing intervention related to various problems is given below:

Related to pain

- Assess degree and nature of pain.
- Administer pain medications as instructed for relieving pain
- Promote pancreatic rest by NPO and NG suctioning
- Semi fowler's position to decrease pressure on the diaphragm by distended abdomen
- Bed Rest to decrease the metabolic demands.

Related to fluid and electrolytes

- Check the color and amount of gastric aspirate
- Monitor Vital signs i.e., BP, Pulse, Respiration and Temperature
- ECG
- Monitor urine output
- Maintain intake and output record
- Observe for the manifestation of electrolyte imbalance
- Check blood glucose levels
- Check for the signs of hypocalcaemia; symptoms of tetany such as jerking, irritability, and muscular twitching.
- Assess for a positive Chvostek's or Trousseaus's sign.

Related to nutritional support

- Monitor weight and laboratory values
- Observe stool for steatorrhea
- Administer nasogastric feeds.
- Nutritional support is paramount and the decision about Total Parenteral Nutrition (TPN) verses enteral feedings is made. Thus accordingly care is required.
- Frequent oral and nasal care to relieve the dryness of the mouth and nose will add to the comfort of the patient.

Related to breathing pattern

- Assess respiratory functions
- Provide comfortable position
- If acute respiratory distress syndrome develops, the patient may require intubation and mechanical ventilation so you need to assist in the procedure and provide care to the patient with ventillator.

Related to infection

- Observe for fever
- Chest physiotherapy and turning, coughing, deep breathing exercises and assuming semi-fowler's position to prevent respiratory infection.

Related to ERCP and other surgical procedures

- Prepare the patient for the procedure and assist in ERCP i.e., Taking Consent for the procedure, Care of dentures, administering premedication if ordered, monitoring vital signs during and after the procedure and observing the patient for potential complications like perforation, bleeding, and infection.
- Povide Pre and post operative care in case of surgical procedure as learnt in your basic training.
- Provide Special wound care and Pouch care.
- Assess and measure fluid and electrolyte loss.

Related to therapeutic regimen

To prevent further attacks, you should advice the patient that s/he should —

- Stop alcohol consumption and smoking.
- Eat a diet high in carbohydrates and low in fats.
- Should report immediately of any untoward symptoms e.g., constant severe abdominal pain, jaundice, high blood glucose level, steatorrhea.

Check Your Progress 1

Encircle the correct answer

i) The most common pathogenic mechanism in acute pancreatitis is —
   a) Cellular disorganization
   b) Lack of secretion of enzymes
   c) Auto digestion of the pancreas
   d) Over production of enzymes

ii) In a patient with acute pancreatitis nursing management should include which of the following?
   a) Monitoring for infection particularly respiratory infection
   b) Observing stools for signs for steatorrhea
   c) Checking for the signs of hypercalcemia
   d) Providing a diet low in carbohydrates with moderate fat.

iii) Which of the following diagnostic tests is most appropriate to determine if gallstones are the cause of the patient's pancreatitis?

a) Endoscopic retrograde cholangiopancreatography

b) CT scan of the abdomen

c) Ultrasonography of the right upper quadrant

d) Plain radiographs of the abdomen

iv) Write the follow up advice you will give to a patient to prevent the recurrent attacks of pancreatitis?

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4.10 LET US SUM UP

Acute pancreatitis is an inflammatory condition of the pancreas characterized clinically by abdominal pain and elevated levels of pancreatic enzymes in the blood. Abnormal exocrine and endocrine function can occur during an acute attack. In patients with interstitial or mild acute pancreatitis the gland returns to histological and functional normalcy after recovery. Endocrine function returns to normal soon after the acute phase, while exocrine function may take up to one year for full recovery. Depending on its severity, it can have severe complications and high mortality despite treatment. Therefore, nurses should not only manage these patients but also should advise them to prevent the recurrent attacks and complications. In this unit we have discussed the definition, etiological factors, pathophysiology, clinical manifestations, diagnostic assessment, complications of pancreatitis therapeutic and nursing management.

4.11 GLOSSARY

ERCP : (Endoscopic Retrograde Cholangio Pancreatography): An endoscopic procedure utilizing fiberoptic technology to visualize the biliary system

Steatorrhea : Frothy foul-smelling stools with a high fat content

4.12 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) c

2) a

3) a

4) Follow up advice to a patient should include:

- Stop alcohol consumption and smoking

- Eat a diet high in carbohydrates and low in fats
- Should report immediately of any untoward symptoms e.g., constant severe abdominal pain, jaundice, high blood glucose level, steatorrhea

4.13 FURTHER REFERENCES

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BNS-032 Nursing Management in Critical Care Conditions

Block 1 Care of Critically ill Patients with Problems of Gastrointestinal System

Unit 1 Acute Gastrointestinal bleeding
Unit 2 Acute intestinal obstruction, Intra abdominal Compartment Syndrome, Perforative Peritonitis.
Unit 3 Hepatic disorders – Fulminent hepatic failure and hepatic encephalopathy
Unit 4 Acute Pancreatitis

Block 2 Care of Critically Ill Patients with Problems of Respiratory System
Unit 1 Acute Respiratory Distress Syndrome
Unit 2 Chest trauma (Hemothorax, Pneumothorax Chylothorax, Pyothorax, Hydrothorax)
Unit 3 Pulmonary Edema, Pulmonary Embolism, Atelectasis
Unit 4 Acute exacerbation of Chronic Obstructive Pulmonary Diseases And Status Asthmatics
Unit 5 Interstitial Lung Disease, Pneumonia, Pleural Effusion

Block 3 Care of Critically Ill Patients with Problems of Cardiothoracic System
Unit 1 Acute Coronary Syndrome
Unit 2 Hypertensive Crisis
Unit 3 Cardiac dysrrhythemia and cardiac arrest
Unit 4 Conductive Disturbances and Heart Block (Pacemaker)
Unit 5 Heart Failure, Aneurysm (Aortic LV)
Unit 6 Open heart surgery, cardiac Tampanade and Heart Transplantation

Block 4 Care of Critically Ill Patients with Problems Of Nervous System
Unit 1 Altered Sensorium And Increased Intracranial Pressure (↑ ICP)
Unit 2 Cerebro-vascular Accident (Stroke)
Unit 3 Traumatic Brain injury, Spinal Cord Injury and Statin Epileptics[
Unit 4 Myasthenic Crisis Multiple Scterosis And Guillain Barre Syndrome

Block 5 Care of critically ill patients with problem of renal and endocrine system
Unit 1 Care of Patients With Renal Transplantation
Unit 2 Acute Tubular Necrosis And Bladder Trauma
Unit 3 Diabetic Ketoacidosis
Unit 4 Thyroid crisis, Myxedema coma, Adrenal crisis and Syndrome of inappropriate hypersecretion of anti diuretic hormone (SIADH))

Block 6 Care Of Critically Ill Patients With Various Emergency Conditions
Unit 1 Burn
Unit 2 Poly Trauma (Multiple Organ Failure /organ transplantation/ triage/coding)
Unit3 Shock, Septicemia, Multiple Organ Dysfunction Syndrome, Disseminated Intravascular Coagulation, Status Epilepticus
Unit 4 Drug Over Dose And Poisoning, Anaphylaxis
Unit 5 Drowning

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