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Medical Diagnosis/Disease: Crohn's Disease

NCLEX IV *(8)*: Physiological Integrity/Physiological Adaptation

NCLEX IV *(7)*: Reduction of Risk

Anatomy and Physiology

Normal Structures

GI tract extends around 30ft from mouth to anus. Large tube composed of 4 layers, mucosa lining, submucosa connective tissue, muscle, and serosa. 3 smooth muscle layers, oblique (inner), circular (middle), and longitudinal (outer). PNS is mainly excitatory and SNS is inhibitory. Both afferent fibers relay sensory information. GI tract has its own nervous system called ENS (enteric) which regulates motility and secretions along the entire GI tract. Its composed of 2 networks, Meissner plexus in the submucosa and Auerbach plexus between muscle layers. Submucosal plexus controls secretions and is involved in many sensory functions. Myenteric plexus is the major nerve supply to the GI tract and controls GI movements. Venous blood draining the GI tract organs empties into portal vein which perfuses the liver which allows it to clean blood of bacteria and toxins. Celiac artery, superior mesenteric artery, and inferior mesenteric artery supply arterial blood to GI tract. Stomach and duodenum receive their blood from celiac axis, distal small intestine to mid large intestines receives its blood from branches of hepatic and SMA. Distal large intestine through the anus receives blood from the IMA. GI and accessory organs receive 25-30% of CO at rest and 35 or more after eating. The peritoneum almost completely covers the abdominal organs. 2 layers of peritoneum are the parietal layer (lines abdominal cavity wall) and visceral layer (covers abdominal organs. 2 folds are mesentery (attaches the small intestine and part of the large intestine to the posterior abdominal wall; contains blood and lymph vessels) and omentum (hangs like an apron from stomach to intestines; contains fat and lymph nodes). Main function of the GI system is to supply nutrients to body cells which is accomplished through process of ingestion, digestion, absorption, and elimination.

Ingestion: intake of food, appetite is controlled in the hypothalamus, which is affected by factors as hypoglycemia, empty stomach, and decrease in body temperature. Hormone ghrelin released from stomach mucosa stimulates appetite and leptin suppressed appetite. Deglutition or swallowing is the mechanical portion of ingestions. Three organs involved mouth, pharynx, and esophagus. Mouth: lips and oral (buccal) cavity. Lips functions in speech. Hard and soft palates from roof of oral cavity. OC contains teeth use for mastication(chewing) and tongue which is a solid muscle mass that aids in chewing and moving food to back of throat for swallowing; it is covered in taste buds and is also important for speech. Salivary glands (parotid, submaxillary, and sublingual) which produce saliva (consists of water, protein, mucin, inorganic salts and salivary amylase). Pharynx: muscular tube lined with mucous membrane. 3 divisions nasopharynx, oropharynx and laryngeal pharynx. Mucous membrane is continuous with nasal cavity, mouth, auditory tubes, and larynx. Oropharynx is route for food from mouth to esophagus by receptors that are stimulates by food or liquid to stimulate swallowing reflux. Epiglottis closes over opening to larynx to prevent aspiration. Tonsils and adenoids composed of lymphoid tissue help body prevent infection. Esophagus: hollow, muscular tube that receives food from pharynx and moves it into stomach. 7-10in long and 0.8in in diameter. Structurally composed of 4 layers inner mucosa, submucosa, muscularis propria, and outermost adventitia. Upper third of esophagus is composed of striated skeletal muscle while distal 2/3 are smooth muscle. Between swallows the UED closes and the esophagus collapses. With swallowing the UES relaxes, and a peristaltic wave moves the bolus into the esophagus. Muscular layers contract (peristalsis) and propel the food to the stomach. The LES at the distal end of the esophagus controls opening of esophagus into the stomach. Stays contracted except during swallowing, belching or vomiting. LES prevents reflux of acidic gastric contents into esophagus.

Digestion and absorption: stomach: store and mix food with gastric secretions and empty contents into small boluses into small intestine. Only absorbs small amounts of water, alcohol, electrolytes, and certain drugs. J shaped and lies obliquely in epigastric, umbilical and left hypochondriac regions of abdomen. Always contains gastric fluid and mucus. 3 main parts: fundus, body, antrum. Pylorus us a small portion of antrum proximal to pyloric sphincter. LES and pyloric sphincter guard the entrance to and exit from the stomach. Stomach has 4 layers: serous(outer; continuous with peritoneum) muscular layer consists of longitudinal layer, circular layer, and oblique layer. Mucosal layer forms fold called rugae that have many small glands. Un the fundus, the glands contain chief cells(secrete pepsinogen) and parietal cells (secrete HCI acid, water, and intrinsic factor.) HCI acid makes gastric juice acidic. Acidic pH helps protected against ingested organisms. Intrinsic factor promotes cobalamin (B12) absorption in small intestines. Small intestines: digestion and absorption, uptake of nutrients from gut lumen to bloodstream, Coiled tube ab 23 ft in lengths and 1-1.1in in diameter. Extends from pylorus to ileocecal valve. Composed of duodenum, jejunum, and ileum. Ileocecal valve prevents reflux of large intestine contents into small intestine. Mucosa of SI is thick, vascular, and glandular. Functional unties are villi (minute, fingerlike projections in mucous membrane) they contained epithelial cells that produce the intestinal digestive enzymes and also produce microvilli. Circular folds in the mucous and submucous

Pathophysiology of Disease

Exact cause is unknow, but it is an autoimmune disease involving an immune reaction to a persons own intestinal tract. Results from an overactive, inappropriate or sustained immune response to environmental and bacterial triggers in a genetically susceptible person. The inflammation causes widespread tissue destruction. Incidences and frequency of any inflammatory bowel disease (IBD) varies depending on geographic location and racial or ethnic background. Higher rates are in northern hemisphere and industrialized nations, risk being greater urban compared with rural areas. White and Ashkenazic Jewish origin than in other racial and ethnic groups. Diet, smoking, and stress increase susceptibility by changing the environment of the GI microbial flora, especially diets in industrialized countries are thought to contribute to the development of IBD. High intake of refined sugar, total fats, polyunsaturated fatty acid and omega-6 fatty acids seem to increase the risk. Use of NSAIDs, antibiotics, and oral contraceptives also increase risk

Anticipated

Labs

CBC (iron deficiency and blood loss),
WBC (toxic megacolon or perforation),
CMP(fluid and electrolyte losses),
CRP/sed rate (inflammation)
stool cx (blood, pus , and mucus)
liver functions (AST, ALT, bilirubin)

Additional Diagnostic

Double contrast barium enema, small bowel series, transabdominal US, CT, MRI, colonoscopy, capsule endoscopy

layers along with the villi and microvilli increase surface area for digestion and absorption. The digestive enzymes on the brush border of the microvilli chemically break down nutrients for absorption. Btw bases of villi lie crypts of lieberkuhn which contain multipotent stem cells. These are precursors for other epithelial cell types. Brunners glands in submucosa of the duodenum secrete an alkaline fluid containing bicarbonate that neutralizes acidic fluids and protects mucosa. Intestinal goblet cells secrete mucus that protects the mucosa.
Physo of digestion: the physical and chemical breakdown of food into absorbable substances. The timely movement of food through GI tract and secretion of specific enzymes promote digestion. Enzymes break down food to particles of appropriate size for absorption. It begins in mouth where food I chewed, mechanically broken down and mixed with saliva which helps by lubricating food. Saliva contains amylase which breaks down starches to maltose. Chewing and the sight, smell, through and taste of food stimulate the release of saliva. Hormones are released into the bloodstream which control HCI acid secretion, production and release of digestive enzymes and motility. In stomach, muscle action mixes the food with gastric secretions to form chyme which is now ready for absorption. Protein digestion begins with release of pepsinogen from chief cells. Stomachs acidic environment results in conversion of pepsinogen to active from pepsin, which begins the breakdown of proteins. Length of time food stays in the stomach depends on the composition of the food. Average meal stays in stomach for 3-4hr. in small intestine, physical presence and chemical nature of chyme stimulates motility and secretion. Secretions from the lover and enzymes from the small intestine. Carbohydrates are broken down to monosaccharides, fats to glycerol and fatty acids and proteins to amin acids. Enzymes of the brush border of microvilli complete the digestion process. These enzymes break down disaccharides to monosaccharides and peptides to amino acids from absorption. The absorption of most of the end products of digestion occurs in the small intestine. Movement of the villi enables these ends products to come in contact with the absorbing membrane.
Elimination: large intestine: hallow muscular tube around 5-6ftx2in. most important functions are water and electrolyte absorption. Also a reservoir for fecal mass until defecation occurs. Feces are composed of water, bacteria, unabsorbed minerals, undigested foodstuffs, bile pigments, and desquamated epithelial cells, large intestines secrete mucus which acts as lubricant and protects mucosa. Microorganisms in colon contribute to digestion by producing vit k and b and breaking down proteins that are not digested or absorbed in the small intestine into amino acids. Bacteria deaminate the amino acids, resulting in ammonia. ammonia is carried to liver where is converted to urea. Movements of large intestine are usually slow, however propulsive peristalsis does occur. Food entering the stomach and duodenum triggers gastrocolic and duodenocolic refluxes resulting in peristalsis in the colon. These refluxes are more active after first daily meal and often result in bowel evacuation. Defecation is a reflex action involving voluntary and involuntary control. Feces in the rectum stimulate sensory nerve endings that produce the desire to defecate. The reflex center or defecation is in the parasympathetic nerve fibers in the sacral part of spinal cord. These fibers produce contraction of rectum and relaxation of internal anal sphincter. Valslva maneuver or bearing down can promote defecation. Close airway and contract abdominal muscles and bearing down. Increases intraabdominal and intrathoracic pressures and reduces venous return to heart. Creates drop in BP and then when relaxing again there is a sudden flow of blood into heart.

NCLEX II *(3)*: **Health Promotion and Maintenance**

NCLEX IV *(7)*: **Reduction of Risk**

Contributing Risk Factors	Signs and Symptoms	Possible Therapeutic Procedures	Prevention of Complications
Family hx, smoking, stress, diet with high saturated fat, refined carbs, and processed meats, use of antibiotics, NSAIDs, air pollution	Bloating, blood in stool, fever, mouth sores, reduced appetite, urgent need to move bowels, skin changes, malnutrition	Non-surgical Pain control, electrolyte balance, nutritional support, IV fluids, Surgical: Resecting diseased segments with anastomosis, strictureplasty, fistulas, drain abdominal abscesses	(What are some potential complications associated with this disease process) Hemorrhage, strictures, perforation, abscesses, fistulas, CDI, toxic megacolon, small intestine cancer, multiple sclerosis, ankylosing spondylitis, malabsorption, liver disease, osteoporosis

NCLEX IV *(6)*: **Pharmacological and Psychosocial/Holistic**

Parenteral Therapies

NCLEX IV *(5)*: **Basic Care and Comfort**

NCLEX III *(4)*:

Care Needs

Anticipated Medication Management	Non-Pharmacologic Care Measures	What stressors might a patient with this diagnosis be experiencing?
Aminosalicylates, antimicrobials, corticosteroids, immunomodulators, biologic therapies	High calorie, high vitamin, high protein diet, physical and emotional rest, referral for counseling or support group	Fear, pain, medical expenses, no cure, sudden bathroom needs, psychosocial life, disease progression, body image, dietary restrictions

Client/Family Education **NCLEX I** *(1)*: **Safe and Effective Care Environment**

List 3 potential teaching topics/areas	Multidisciplinary Team Involvement (Which other disciplines do you expect to share in the care of this patient)
• no cure, but can be managed • understanding medications and side effects • avoid diet in high sat fats, refined carbs, and processed meats.	Gastroenterologist, surgeons, pharmacists, IBD nurse specialists, psychologist, dietitian, dermatologist, nutritionist, radiologist, oncology

Potential Patient Problems (Nursing Diagnoses)

To Be Completed Before the Simulation

Anticipated Patient Problem: malabsorption

Clinical Reasoning: reduced oral intake, weight loss, medication side effects, inflammation due to active disease

Goal 1: pt will maintain adequate weight during my time of care

Relevant Assessments (Prework) What assessments pertain to your patient's problem? Include timeframes.	**Multidisciplinary Team Intervention** (Prework) What will you do if your assessment is abnormal?
Assess pt weight q shift	Encourage nutritional supplements like ensure drink during meal times
Assess electrolytes as posted by lab	Notify provider if any major changes
Assess fluid intake/output q1hr	Provide bladder scan to determine retention
Assess meal trays after meal times	Educate on importance of eating proteins first and discuss what kinds of food they would prefer to eat
Assess food on meal trays as provided	Discuss with dietitian if needed to provide ensure drinks or more high protein/ high calorie meals
Assess muscle movements during assessment	Notify provider if any serious changes to muscle mass (atrophy)

Goal 2: pt electrolyte status will be maintained within normal limits during my time of care

To Be Completed Before the Simulation

Anticipated Patient Problem: acute pain

Clinical Reasoning: cramping from Crohn's, inflammation of digestive tract,

Goal 1: pt will report pain of 2/10 or less by the end of my care

Relevant Assessments (Prework) What assessments pertain to your patient's problem? Include timeframes.	**Multidisciplinary Team Intervention** (Prework) What will you do if your assessment is abnormal?
Assess pt pain q1-2hr, PRN	Provide medications prescribes by provider for pain
Identify inflammatory foods with pt when eating/ discussing pain habits	Educate pt on foods that could possibly making inflammation worse
Assess vitals q4hr/PRN	Notice vital spikes when in pain and provide medication
Pain medication not working, breakthrough pain	Provide distraction techniques
Assess knowledge of systemic symptoms	Educate on what kind of symptoms could be related that are no in GI system
Assess	

Goal 2: pt will understand what foods to avoid to avoid pain by the end of my care

To Be Completed During the Simulation:

Actual Patient Problem: decreased blood volume

Clinical Reasoning: light headed, blood in ostomy, active bleed, transfusion ordered

Goal: pt will have restored circulation by the end of my care Met: yes ☐ Unmet:☐

Goal: pt will understand how to prevent bleeds in the future by the end of my care Met: yes ☐ Unmet:☐

Actual Patient Problem: malnutrition

Clinical Reasoning: light headed, weight of 109, improper diet

Goal: pt will verbalize understanding of proper diet by the end of my care Met: yes ☐ Unmet:☐

Goal: pt will maintain optimal body weight by the end of my care Met: yes ☐ Unmet:☐

Additional Patient Problems:

Below will be your notes, add more lines as needed. **Relevant Assessments:** Indicate pertinent assessment findings. **Multidisciplinary Team Intervention:** What interventions were done in response to your abnormal assessments? **Reassessment/Evaluation:** What was your patient's response to the intervention?

Patient Problem	Time	Relevant Assessments	Time	Multidisciplinary Team Intervention	Time	Reassessment/ Evaluation
Decreased blood volume		Reports light headedness and dizziness, "I feel like im going to faint and throw up"		Provide cold cloth		"that would be great"
Decreased blood volume		VS 94/56, HR 110, RR 26, SAO2 94%		O2 applied at 2L NC, lowered HOB		Improved circulation to prevent hypovolemic shock
Decreased blood volume		Blood ready		Discuss if pt experienced any reaction with previous blood transfusion		Prevent reaction
Decreased blood volume		Restless, flushed face, headache, temp 101.8, RR22		Stop blood transfusion immediately		Prevented further exposure to sensitizing component
Decreased blood volume		Scheduled for endoscopy, pt education is needed		Educated that medication to reduce oral secretions may be administered		Pt verbalizes understanding

Malnutrition		States feeling stressed before episode occurred that brought her to hospital		Educated ways to manage stress not involving alcohol		Pt states understanding
Malnutrition		Requests ibuprofen, states is taken a lot during week		Educated that ibuprofen is not appropriate due to Crohn's		Pt states she will not be taking it anymore
Malnutrition		Improper diet discussed, inadequate nutrition		Provide educational material providing correct diet		Pt verbalizes understanding of eating high protein and carb diet

ATI Virtual Clinical Questions and Reflection:

1) Identify two members of the healthcare team collaborating in the care of this patient:
    a. Esther
    b. Bonnie
2) What were three steps the nursing team demonstrated that promoted patient safety?
    a. **Hand hygiene each time pt room was entered**
    b. **Confirmed all pt information was correct before administering blood**
    c. **Stayed with pt when she was in distress**
3) Do you feel the nurse and medical team utilized therapeutic communication techniques when interacting with individuals, families, and health team members of all cultural backgrounds?
    a. If **yes**, describe**: yes, the team used therapeutic techniques when working together to give report and provide SBAR to each other. They also asked the pt questions that made her feel safe in order for the patient to be able to express her concerns**
    b. If **no**, describe: _____
    _____

Reflection

1) Go back to your Preconference Template:
    a. Indicate (circle, star, highlight, etc.) the components of your preconference template that you saw applied to the care of this patient.
2) What was the priority nursing problem? Provide rationale. **decreased blood volume due to the active bleed. Pt was showing multiple signs of blood loss including lightheadedness, dizziness, and n/v**
3) Review your Patient Problem Form: Did you see many of your anticipated nursing assessments and interventions used?
    a. Were there interventions you included that *were not* used in the scenario that could help this patient?
        i. If **yes**, describe: **I believe that monitoring pt weight would be an intervention that would be beneficial to the pt due to malabsorption**
        ii. If **no**, describe:
        _____
        _____
4) After completing the scenario, what is your patient at risk for developing?
    a. Hypovolemic shock
    b. Why? **Severe blood loss**
5) What was your biggest "take-away" from participating in the care of this patient? How did this impact your nursing practice?

a. **My biggest takeaway is to assess signs early of what could be happening to your patient. Catching the transfusion reaction saved the pt from further complications, which showed me how important it is to check on your patient when administering anything to a patient. I also learned about timely interventions through this. This will impact my nursing practice by diligently watching a pt after administration of anything**