

Systemic pathology
Large intestine (Hemorrhoids, Malabsorption Syndrome)

Lecture (10)

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Hemorrhoids

Hemorrhoids are dilated anal and perianal collateral vessels that connect the portal and caval venous systems to relieve elevated venous pressure within the hemorrhoid plexus. Thus, although hemorrhoids are less serious than esophageal varices, the pathogenesis of these lesions is similar. They are common, affecting about 5% of the general population.

Common predisposing factors include constipation and associated straining, which increase intraabdominal and venous pressures, venous stasis of pregnancy, and portal hypertension.

Collateral vessels within the inferior hemorrhoidal plexus are located below the anorectal line and are termed **external hemorrhoids**, while those that result from dilation of the superior hemorrhoidal plexus within the distal rectum are referred to as **internal hemorrhoids**.

On histologic examination, hemorrhoids consist of thin-walled, dilated, submucosal vessels beneath anal or rectal mucosa. These vessels are subject to trauma, which leads to rectal bleeding. In addition, they can become thrombosed and inflamed.

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Hemorrhoids often manifest with pain and rectal bleeding, particularly bright red blood seen on toilet tissue.

Hemorrhoids also may develop as a result of portal hypertension, where the implications are more ominous. Hemorrhoidal bleeding generally is not a medical emergency; treatment options include sclerotherapy, rubber band ligation, and infrared coagulation. In severe cases, hemorrhoids may be removed surgically by *hemorrhoidectomy*.

Malabsorptive Diarrhea

Malabsorption manifests most commonly as *chronic diarrhea* and is characterized by defective absorption of fats, fat- and water-soluble vitamins, proteins, carbohydrates, electrolytes, minerals, and water. Chronic malabsorption causes weight loss, anorexia, abdominal distention, borborygmi, and muscle wasting. A hallmark of malabsorption is *steatorrhea*, characterized by excessive fecal fat and bulky, frothy, greasy, yellow, or clay-colored stools.

Malabsorption results from disturbance in at least one of the four phases of nutrient absorption:

- *Intraluminal digestion*, in which proteins, carbohydrates, and fats are broken down into absorbable forms

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- *Terminal digestion*, which involves the hydrolysis of carbohydrates and peptides by disaccharidases and peptidases, respectively, in the brush border of the small-intestinal mucosa
- *Transepithelial transport*, in which nutrients, fluid, and electrolytes are transported across and processed within the small-intestinal epithelium
- *Lymphatic transport* of absorbed lipids

In many malabsorptive disorders, a defect in one of these processes predominates, but more than one usually contributes. As a result, malabsorption syndromes resemble each other more than they differ.

Signs and symptoms include diarrhea (from nutrient malabsorption and excessive intestinal secretion), flatus, abdominal pain, and weight loss. Inadequate absorption of vitamins and minerals can result in anemia and mucositis due to pyridoxine, folate, or vitamin B₁₂ deficiency; bleeding due to vitamin K deficiency; osteopenia and tetany due to calcium, magnesium, or vitamin D deficiency; or neuropathy due to vitamin A or B₁₂ deficiency. A variety of endocrine and skin disturbances may also occur. Mycobacterial infection, which can be lead to lymphatic transport defects.