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Lecture title: **ABOMASAL ULCERS**

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Summary:

Abomasal ulceration occurs in mature cattle and calves and may cause acute abomasal hemorrhage with indigestion, melena, and sometimes perforation, resulting in a painful acute local peritonitis or acute diffuse peritonitis and rapid death, or a chronic indigestion with only minimal abomasal hemorrhage. Ultrasonography and laboratory tests may aid making a diagnosis. Treatment is based on decreasing acid secretion in the abomasum.

ETIOLOGY

A. Primary ulceration

1. Abomasal hyperacidity in adult cattle, although there is no direct evidence to support the hypothesis except in animals subject to fasting.
2. Mechanical abrasion of the pyloric antrum caused by the ingestion of coarse roughage, such as straw.
3. Bacterial infections such as *C. perfringens* type A or fungi such as *Aspergillus fumigatus* and *Mucor* spp.
4. Concurrent stress as in cattle with severe inflammatory processes or in severe pain that result in hypercortisolemia.

B. Secondary ulceration

1. Reflux of bile into the abomasal lumen, particularly in ruminants with LDA or RDA, is strongly associated with abomasal ulceration. Bile acids are a well-documented cause of gastric injury.
2. Lymphoma of the abomasum and erosions of the abomasal mucosa in viral diseases such as bovine virus diarrhea (BVD) and bovine malignant catarrh.

PATHOGENESIS

Type 1: Nonperforating ulcer

- ✓ There is incomplete penetration of the abomasal wall resulting in a minimal degree of intraluminal hemorrhage, focal abomasal thickening, or local serositis.
- ✓ Nonbleeding chronic ulcers commonly cause a chronic gastritis.
- ✓ Erosions are discrete mucosal defects that do not penetrate the muscularis mucosa of the abomasum.

Type 2: Ulcer causing severe blood loss

- ✓ There is penetration of the wall of a major abomasal vessel, usually in the submucosa, resulting in severe intraluminal hemorrhage and anemia.
- ✓ In acute ulceration with erosion of a blood vessel there is acute gastric hemorrhage with reflex spasm of the pylorus and accumulation of fluid in the abomasum, resulting in distension, metabolic alkalosis, hypochloremia, hypokalemia, and hemorrhagic anemia.
- ✓ Usually within 24 hours there is release of some of the abomasal contents into the intestine, resulting in melena.

Type 3: Perforating ulcer with acute, local peritonitis

There is penetration of the full thickness of the abomasal wall, resulting in leakage of abomasal contents. Resulting peritonitis is localized to the region of the perforation by adhesion of the involved portion of abomasum to adjacent viscera, omentum, peritoneal surface, or diaphragm.

Type 4: Perforating ulcer with diffuse peritonitis

There is penetration of the full thickness of the abomasal wall, resulting in leakage of abomasal contents. Resulting peritonitis is not localized to the region of the perforation; thus digesta is spread diffusely throughout the peritoneal cavity.

CLINICAL FINDINGS

1. The clinical syndrome varies depending on whether ulceration is complicated by bleeding or perforation.
2. In the common clinical form of bleeding abomasal ulcers there is a **sudden onset of anorexia**, **mild abdominal pain**, **tachycardia (90–100 beats/min)**, **severely depressed milk production,** and **melena**.
3. Acute hemorrhage may be severe enough to cause death in less than 24 hours.
4. More commonly there is subacute blood loss over a period of a few days with the development of hemorrhagic anemia.
5. The feces are usually scant, black, and tarry. There are occasional bouts of diarrhea.
6. Melena may be present for 4 to 6 days, after which time the cow usually begins to recover into a stage of chronic ulceration without evidence of hemorrhage.
7. Abomasal ulceration secondary to lymphoma of the abomasum is characterized by chronic diarrhea and melena. The ulcer does not heal.
8. Moderate dehydration is common, and affected cows commonly sip water continuously and grind their teeth frequently.
9. The prognosis in chronic ulceration is poor because of the presence of several ulcers and the development of chronic abomasal atony.
10. Some cows improve temporarily but relapse several days later and fail to recover permanently.
11. The case–fatality rates for cattle with types 1, 2, 3, or 4 are 25, 100, 50, and 100%, respectively.

CLINICAL PATHOLOGY

1. Melena, the dark brown to black color of the feces is usually sufficient indication of gastric hemorrhage but tests for blood may be necessary. When perforation has occurred with acute local peritonitis, there is neutrophilia with a regenerative left shift for a few days, after which time the total leukocyte and differential count may be normal.
2. Hemogram, in acute gastric hemorrhage, there is acute hemorrhagic anemia.

TREATMENT

1. Blood transfusions
   - Blood transfusions and fluid therapy may be necessary for acute hemorrhagic ulceration.
   - In the case of severe blood loss, a dose of 20 mL/kg BW may be necessary.
2. Antacids
   - The goal of antacid treatment is to create an environment that is favorable to ulcer healing. This can be done by decreasing acid secretion (oral or parenteral administration of histamine type-2 receptor antagonists [H_2 antagonists] and proton pump inhibitors). Oral and parenteral administration of cimetidine and ranitidine increases abomasal pH in sheep and cattle. High doses of cimetidine (20 mg/kg BW intravenously, or 50–100 mg/kg orally) increased abomasal pH in weaned lambs for more than 2 hours. Daily oral administration of cimetidine (10 mg/kg BW for 30 days) to calves may facilitate healing of abomasal ulcers.
   - Neutralizing secreted acid (oral administration of magnesium hydroxide and aluminum hydroxide). Aluminum hydroxide directly absorbs pepsin, decreasing the proteolytic activity of pepsin in the stomach. Both compounds bind bile acids, protecting against ulceration induced by bile reflux.
3. Kaolin and pectin

   Large doses of liquid mixtures of kaolin and pectin (2–3 L twice daily for a mature cow) to coat the ulcer and minimize further ulcerogenesis have been suggested and used with limited success.
4. Surgical excision

   Surgical excision of abomasal ulcers has been attempted, with some limited success. The presence of multiple ulcers may require the radical excision of a large portion of the abomasal mucosa and hemorrhage is usually considerable. A laparotomy and exploratory abomasotomy are required to determine the presence and location of the ulcer.