University of Mosul
Lecture No.: 6
College of Veterinary Medicine
Date:22/10/2024
Unit of Scientific Affairs
Website:

Lecture title: **VAGUS INDIGESTION (Hoflund syndrome)**

Lecturer Affiliation: **Department of Internal and Preventive Medicine**
College of Veterinary Medicine, University of Mosul, Mosul, Iraq

Summary:

Vagus indigestion in cattle was first described in 1940 by Hoflund. It is characterized by a dysfunction in the tenth pair of cranial nerves that leads to changes in forestomach motility. There is accumulation of ingesta in the forestomach, reduced appetite or anorexia, scant faeces, decreased milk production, acid base abnormalities, dehydration, weight loss, and eventually weakness and recumbancy. Prognosis is usually unpredictable and often considered poor.

ETIOLOGY

1. The etiology of vagus indigestion has been divided into two major complications of traumatic reticuloperitonitis: **vagal nerve injury** and **reticular adhesions**, with the latter being the most common cause.

A. Vagal Nerve Injury and Dysfunction:

- ✓ It was hypothesized that the inflammatory and scar tissue lesions (traumatic reticuloperitonitis) affected vagal nerve fibers supplying the forestomach and abomasum.
- ✓ In dorsal vagal nerve injury resulted in affected on the reticuloomasal orifice (anterior stenosis) inhibit the passage of ingesta from the reticulorumen into the omasum and abomasum, resulting in an enlarged rumen with abnormal rumen contents.
- ✓ Injury of the pyloric branch of the ventral vagus nerve resulted in affected on the **pylorus** (posterior stenosis) and inhibited the flow of ingesta from the abomasum, resulting in abomasal impaction.
- ✓ Both abnormalities resulted in scant feces containing undigested feed particles.

B. Reticular Adhesions

Mechanical impairment of reticular motility as a result of reticular adhesions is probably the most important cause of vagal indigestion syndrome.

2. In addition, there are some other causes:

- ✓ Actinobacillosis of the rumen and reticulum is a less common cause.
- ✓ Perireticular abscesses near the reticuloomasal orifice of cattle can cause the disease.
- ✓ Following surgery for abomasal volvulus.

EPIDEMIOLOGY

1. Vagal indigestion is most common in dairy cows that have a history of traumatic reticuloperitonitis.
2. The disease also occurs in beef cattle and in mature bulls.

PATHOPHYSIOLOGY

The current classification method divides the types of vagal indigestion into anterior functional stenosis and posterior functional stenosis.

A. Anterior Functional Stenosis

- ✓ This is characterized by accumulation of ingesta in the reticulorumen, known also as failure of omasal transport.
- ✓ If the ruminal wall is atonic, the ingesta accumulates; if it has normal motility, the ruminal wall responds to the distension by increased motility and the production of frothy bloat.
- ✓ Ruminal motility will be almost continuous (3–6 contractions per minute) but the contractions are ineffective in propelling the ingesta into the omasum.
- ✓ As a result, the rumen enlarges to fill the majority of the abdomen, which accounts for the gross distension of the abdomen.
- ✓ \The dorsal sac of the rumen enlarges to the right of the midline, and the ventral sac enlarges to fill most or all of the right lower quadrant of the abdomen; this results in the "L-shaped" rumen as viewed from the rear of the animal.
- ✓ The continuous rumen contractions also result in frothy rumen contents, which can be fatal if progressive and not relieved.

B. Posterior Functional Stenosis

- ✓ This is characterized by failure of transpyloric outflow resulting in abomasal impaction with large particles.
- ✓ Abomasal fluid containing hydrochloric acid may reflux into the rumen if the fluid does not move from the abomasum into the small intestines. This is known as the abomasal reflux syndrome.
- ✓ The chloride concentrations in the rumen fluid increase and there is a hypochloremia and hypokalemia.

CLINICAL FINDINGS

A. Ruminal distension with hypermotility

1. The occurrence of ruminal distension with hypermotility is not particularly related to pregnancy or parturition.
2. Moderate to severe bloat is common.
3. There is evidence of loss of body weight.
4. The animal has usually been anorexic intermittently for the past few weeks.
5. The abdomen is prominently distended and the rumen movements represented by the abdominal ripples are often unusually prominent and may occur at the rate of 4 to 6 per minute**.**
6. The sounds of the rumen contractions are often reduced or almost absent in spite of hyperactivity because the rumen contents are pasty and frothy.
7. The feces are scant and pasty and contain undigested particles.
8. The temperature is usually normal and bradycardia (44–60 beats/min) may be present because of decreased feed intake.
9. Viewed from the rear the enlarged rumen is **L-shaped**, the left flank distended from top to bottom and the right flank distended only in the lower half-the papple-shaped abdomen.
10. An important aspect of the clinical history of vagal indigestion cases is that standard treatments for ruminal tympany and impaction usually have no effect on the course of the disease.

B. Ruminal distension with atony

1. Ruminal distension with atony is most common in late pregnancy and may persist after calving.
2. The cow is clinically normal in all respects except that is anorexic; passes only scant amounts of soft pasty feces; and has a distended abdomen and will not respond to treatment with purgatives, lubricants, or parasympathetic stimulants.
3. Ruminal movements are seriously reduced or absent and there may be persistent mild bloat.
4. Fluid-splashing sounds may also be audible on ballottement of the left and right flanks if the rumen is distended with excessive quantities of fluid.
5. There is no pain on deep palpation of the ventral abdomen.
6. The animal loses weight rapidly, becoming weak and recumbent. At this stage the heart rate increases markedly. The animal dies slowly of inanition.

DIAGNOSIS

1. Obtaining an accurate history is very importance. Most cases of vagal indigestion have been affected for at least several days or a few weeks.
2. The clinical examination should focus on the state of the rumen and the abomasum.
3. A left-side exploratory laparotomy and rumenotomy will often be necessary to make a diagnosis. This will allow the determination of the presence of reticular adhesions, obstructive of the reticuloomasal orifice and the state of the abomasum.
4. Peritoneal fluid is indicative of a chronic reticuloperitonitis.
5. Serum biochemistry, in abomasal impaction there is metabolic hypochloremic and hypokalemic.

NECROPSY FINDINGS

1. The rumen is grossly enlarged and the contents are pasty and may be frothy and may have undergone some putrefaction.
2. The reticulum and omasum are usually grossly enlarged.
3. The omasum may be almost twice its normal size.
4. The abomasum may be up to twice its normal size and firm on palpation.
5. The intestines may be relatively empty and the feces in the large intestine are pasty, containing an increased amount of undigested particles.
6. Lesions between the reticulum and ventral abdominal floor and the diaphragm vary considerably from thick fibrinous suppurative adhesions to multiple abscesses.

DIFFERENTIAL DIAGNOSIS

The functional disorders of ruminant stomach have to be differentiated from conditions such as ruminal tympany, displaced abomasum, diaphragmatic hernia, intestinal obstruction, paralytic ileus, ascites and ruptured bladder.

TREATMENT AND PREVENTION

1. The prognosis in most cases in unfavorable but also unpredictable. The problem is to determine the location and extent of the lesion.
2. Earlier, the treatment adopted in cases of functional disorders of the stomach included evacuation of rumen contents through a stomach tube or rumenotomy. This treatment used to be ineffective and very few cases used to recover.
3. The left paralumbar exploratory laprorumenotomy was also used as a treatment method to palpate the reticular adhesions or abscess. Following this, any abscess that may have formed was drained, and fluids, calcium, broad spectrum antibiotics, analgesics, and ruminal transfaunation were administered.
4. Prevention is dependent on preventing traumatic reticuloperitonitis through management of the environment and the administration of reticular magnets.