# Traumatic Reticuloperitonitis in Cattle

Traumatic reticuloperitonitis of cattle occurs when animals ingest nails, pieces of wire, and other nonmetallic materials that injure the reticular wall. Sharp objects can puncture the reticulum, allowing ingesta and bacteria to leak into the peritoneal cavity, resulting in peritonitis and often leading to adhesions in the abdomen. Clinical signs include rumenoreticular atony, decreased milk production, and signs of abdominal pain. Treatment can be medical (rumen magnet and antimicrobial therapy) or surgical (foreign body removal via rumenotomy).

Traumatic reticuloperitonitis of cattle is localized inflammation in the wall of the reticulum, usually due to perforation by a sharp object (eg, nail or wire) ingested by the animal. The most common clinical findings include decreased feed intake, decreased rumen motility, mild fever, poorly digested feces, and signs of pain. Foreign body tests (eg, back grip, percussion of the reticulum with a mallet, or pole test) to elicit a grunt are an important step in clinical examination. Ultrasonographic evaluation is essential to identify inflammatory lesions of the reticulum, and radiography enables visualization of foreign bodies. Traumatic reticuloperitonitis is treated with a rumen magnet and antimicrobial therapy, and laparorumenotomy is an option when there is no response to medical treatment. Disease is much less common in other ruminants such as goats and sheep, which tend to be discriminating eaters.

## Etiology of Traumatic Reticuloperitonitis in Ruminants

Ingested wires can come from cut tires used to weigh down tarps covering silage, and other metallic fragments may come from components of mixer wagons. Production systems often use wire fencing. Aluminum and other nonmagnetic foreign bodies are uncommon.

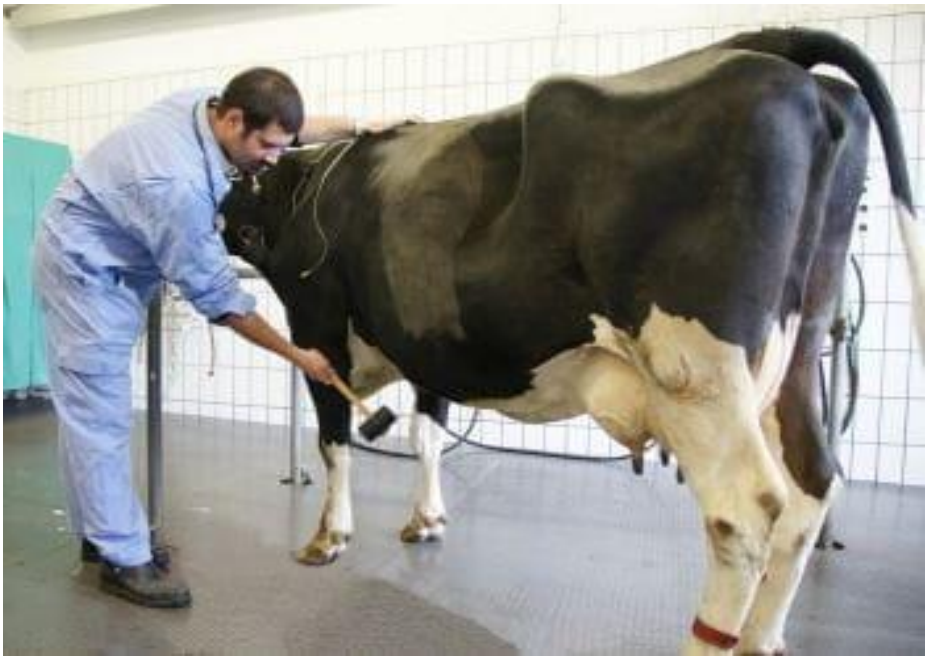
Swallowed metallic objects, such as nails or pieces of wire, fall directly into the reticulum and can penetrate or perforate its wall and cause traumatic reticuloperitonitis. Contractions of the reticulum promote penetration of the wall by the foreign object.

Localized peritonitis in traumatic reticuloperitonitis, cow

Perforation of the wall of the reticulum allows leakage of ingesta and bacteria, which contaminates the peritoneal cavity. The resulting peritonitis is generally localized and frequently results in adhesions. Less commonly, more severe diffuse peritonitis, pleuritis, pericarditis, hepatic, or splenic abscess develops.

## Diagnosis of Traumatic Reticuloperitonitis in Ruminants

- Clinical examination
- Testing for presence of a reticular foreign body (eg, back grip, pain percussion, or pole test)
- Laboratory testing (eg, serum-to-peritoneal fluid glucose concentration difference or peritoneal fluid D-dimer concentration)
- Ultrasonographic evaluation of the ventral abdomen to identify localized peritonitis adjacent to the reticulum
- Radiography to visualize metallic foreign bodies in the reticulum



Eliciting pain response in traumatic reticuloperitonitis, cow.

Cattle suspected of traumatic reticuloperitonitis should be tested for signs of pain, including grunting and bruxism. Foreign body tests to elicit a grunt are an important part of clinical examination; however, the same response may be elicited in other painful abdominal and thoracic conditions as well. Cattle with acute traumatic reticuloperitonitis generally grunt in pain more readily than do those with chronic traumatic reticuloperitonitis. The back grip, percussive pain, and pole tests are the most important tests in diagnosing traumatic reticuloperitonitis. Based on a retrospective study of 508 cattle, foreign body tests (pole test, back grip, percussion) have only fair diagnostic reliability and may result in false negatives.

Although not always necessary, laboratory tests may be helpful in diagnosing traumatic reticuloperitonitis. A differential WBC count more accurately indicates traumatic reticuloperitonitis than does a total WBC count. During the first 3 days of acute localized peritonitis, a characteristic clinicopathologic finding is an increased neutrophil count. After the third day, the findings are normalized in uncomplicated cases. Hyperfibrinogenemia and hyperproteinemia are more reliable than WBC counts at indicating traumatic reticuloperitonitis. Increased fibrinogen concentration may be evident as early as 2–3 days after traumatic reticuloperitonitis onset. In chronic cases, an increase in gamma-globulin concentration accounts for the increase in total protein concentration.

The gamma-globulin and fibrinogen concentrations can be determined simply and quickly by the glutaraldehyde coagulation test, in which they are positively correlated with the time to gel formation. The diagnostic sensitivity of the test is 97.8% for a coagulation time of 3 minutes, and 87.9% for 6 minutes.

Results of rumen fluid analysis are not diagnostic for traumatic reticuloperitonitis. Peritoneal fluid analysis can help determine whether peritonitis is present. Ideally,

ultrasonography-guided abdominocentesis using a spinal needle with stylet yields samples that can be assessed for amount, color, transparency, odor, and consistency as well as for the presence of other material. The specific gravity and total solids concentration in a sample can be evaluated by refractometry. Exudates that indicate traumatic reticuloperitonitis are generally cloudy and watery to viscous fluids that may have a foul odor because of bacteria and may clot quickly after being collected. Flecks of fibrin are often present, the specific gravity is >1.015, and the total solids concentration is >30 g/L. When assessing aspirated peritoneal fluid in cases of traumatic reticuloperitonitis, standard definitions differentiating abdominal transudates from abdominal exudates do not always apply because healthy and sick animals may show similar concentrations of protein and fibrinogen in the peritoneal fluid.

The concentrations of other substances (eg, glucose and D-dimer) can be measured in the peritoneal fluid and serum, and their values from the two sources compared to clarify the diagnosis. In healthy cattle, concentrations of glucose in blood and peritoneal fluid are usually similar. A comparatively lower concentration of glucose in the peritoneal fluid indicates the presence of bacteria, which metabolize glucose. Serum-to-peritoneal fluid glucose concentration difference is therefore considered a very specific criterion for the diagnosis of septic peritonitis. Peritoneal fluid D-dimer concentration is considered the best criterion for the diagnosis of peritonitis because both the sensitivity and the specificity are high.

Ultrasonographic examination of the ventral abdomen using a 3.5- to 5.0-MHz linear or convex transducer is the most accurate way to diagnose localized peritonitis near the reticulum and characterize reticular contraction frequency. The transducer is applied to the ventral aspect of the thorax on both sides of the sternum and to the lateral aspects of the thorax up to the level of the elbow. If abnormalities are detected,

the area being examined is expanded to determine the extent of the lesions. Inflammatory changes appear as echogenic deposits, with or without hypoechoic or anechoic fluid pockets, and structures of various shapes and echogenicities with central echogenic fluid collections. Inflammatory lesions may involve the adjacent organs (typically spleen, liver, and rumen).

Cattle with traumatic reticuloperitonitis frequently have peritoneal effusion in which the accumulation of fluid lacks an echogenic border and is limited to the reticular region. The fluid appears anechoic to hypoechoic depending on the amount of fibrin and cellular material it contains. Fluid surrounding the lesions may contain strands of fibrin that stand out on an ultrasonogram. Reticular abscesses that have an echogenic capsule and a cavity with content that ranges from hypoechoic to moderately echogenic and that is either homogeneous or heterogeneous are present in about 20% of cattle with traumatic reticuloperitonitis. Rarely, ultrasonography may show magnets in the reticulum or foreign bodies either inside or outside of the reticulum.

Most cattle with traumatic reticuloperitonitis have a change in the frequency of reticular contractions.

Radiography is the usual initial modality of choice to visualize metallic foreign bodies in the reticulum. To be considered clinically relevant, linear foreign bodies must be ≥1 cm long. Even more important than the presence of a foreign body is its position. Foreign bodies most likely to have penetrated the reticulum are those that do not contact the reticulum's ventral aspect or those that are situated at an angle >30° to the reticulum's ventral aspect. Any foreign body that is outside of the reticulum's outer contour to some extent has punctured the reticular wall. Foreign bodies that lie flat on the reticulum's ventral aspect or whose positions vary on serial

radiographs are considered nonpenetrating, but they still pose a risk for traumatic reticuloperitonitis. Foreign bodies on the ventral aspect of the reticulum but not attached to a magnet in the reticulum may be nonmagnetic.

## Treatment of Traumatic Reticuloperitonitis in Ruminants

- Oral administration of a rumen magnet and antimicrobial therapy over several days (medical treatment)
- Rumenotomy if medical (conservative) treatment fails

Initial treatment of cattle suspected of having traumatic reticuloperitonitis consists of oral administration of a rumen magnet (if the animal does not already have one; give only one magnet per animal) and antimicrobial therapy (most commonly penicillin or broad-spectrum antimicrobials such as ampicillin, ceftiofur, and tetracyclines). Anti-inflammatory drugs, including flunixin meglumine, ketoprofen, and meloxicam, are administered to manage pain. By capturing ferromagnetic foreign bodies in the reticulum and rumen, magnets prevent trauma. How well the rumen magnet works depends mainly on where the foreign body is situated in the reticulum; a magnet can pick up foreign bodies that on the ventral aspect of the reticulum or that are upright more easily than those that either are not in contact with the ventral aspect of the reticulum or are penetrating the reticulum.

Treatment is considered successful when the rectal temperature normalizes and eating and rumination improve within 3–5 days. Radiographic evidence that a foreign body is completely attached to a magnet also confirms the success of treatment. If conservative treatment fails, the animal may need to be treated surgically or euthanized. The decision of which course to follow should be made

only after radiographic evaluation confirms that the foreign body is not attached to the magnet.

Two main surgical techniques are used for rumenotomy. In laparorumenotomy, the rumen is permanently sutured to the peritoneum and transverse fascia so that the entire procedure—including access to the rumen and healing of the incision—can take place outside of the peritoneum. In temporary extra-abdominal fixation, the surgeon repositions the sutured rumen to its normal position in the abdomen. Laparotomy is carried out in the left flank, the rumen is partially emptied so that the reticular lumen can be palpated, and foreign bodies are removed.

Abscesses that are firmly and broadly attached to the reticulum can be incised and the content drained into the reticulum. For abscesses attached to the thoracic or abdominal wall, transcutaneous drainage guided by ultrasonography is the treatment of choice.

## Prevention of Traumatic Reticuloperitonitis in Ruminants

Measures to prevent traumatic reticuloperitonitis include avoiding the use of baling wire, passing feed over magnets to remove metallic objects, keeping cattle away from sites of new construction, and completely removing old buildings and fences. In addition, magnets may be administered by mouth. There is good evidence that giving magnets to all cattle at ~1 year of age minimizes the incidence of traumatic reticuloperitonitis.

# Traumatic pericarditis

Perforation of the pericardial sac by a sharp foreign body originating in the reticulum causes pericarditis with the development of toxemia and congestive heart failure. Tachycardia, fever, engorgement of the jugular veins, anasarca, hydrothorax and ascites, and abnormalities of the heart sounds are the diagnostic features of the disease.

## Etiology

Traumatic pericarditis is caused by penetration of the pericardial sac by a migrating metal foreign body from the reticulum.

The incidence is greater during the last 3 months of pregnancy and at parturition than at other times. Approximately 8% of all cases of traumatic reticuloperitonitis will develop pericarditis. Most affected animals die or suffer from chronic pericarditis and do not return to completely normal health.

## Pathogenesis

1-The penetration of the pericardial sac may occur with the initial perforation of the reticular wall. However, the animal may have had a history of traumatic reticuloperitonitis sometime previously, followed by pericarditis,

2- usually during late pregnancy or at parturition. In this case it is probable that the foreign body remains in a sinus in the reticular wall after the initial perforation and penetrates the pericardia! Sac at a later date.

3-Physical penetration of the sac is not essential to the development of pericarditis, infection sometimes penetrating through the pericardium from a traumatic

mediastinitis.

4-Introduction of a mixed bacterial infection from the reticulum causes a severe local inflamation , and persistence of the foreign body in the tissues is not essential for the further progress of the disease.

5-The first effect of the inflammation is hyperemia of the pericardial surfaces and the production of friction sounds synchronous with the heart beats.

6-Two mechanisms then operate to produce signs: the toxemia due to the infection and the pressure on the heart from the fluid which accumulates in the sac and produces congestive heart failure.

6- an affected animal may be severely ill for several weeks with edema developing only gradually, or extreme edema may develop within 2-3 days. The rapid development of edema usually indicates early death.

7-If chronic pericarditis persists there is restriction of the heart action due to adhesion of the pericardium to the heart.

8-Congestive heart failure results in most cases but some animals may recover.

9- An uncommon sequel after perforation of the pericardial sac by a foreign body is rupture of a coronary artery or the ventricular wall. Death usually occurs suddenly due to acute, congestive heart failure.

**Clinical findings**

1-Depression, anorexia, habitual recumbency and rapid weight loss are common.

2-Diarrhea or scant feces may be present and grinding of the teeth, salivation and nasal discharge are occasionally observed.

3-The animal stands with the back arched and the elbows abducted.

4- Respiratory movements are more obvious, being mainly abdominal, shallow, increased in rate to 4O- 50/min and often accompanied by grunting.

5-Engorgement of the jugular veins, and edema of the brisket and ventral abdominal wall are common

5-A prominent jugular venous pulse is usually visible and extends proximally up the neck.

6-Pyrexia (40-41oC,) is common in the early stages and an increase in the heart rate to 100/min and a diminution in the pulse amplitude are constant.

7-Rumen movements are usually present but depressed. Pinching of the withers to depress the back or deep palpation of the ventral abdominal wall behind the xiphoid sternum commonly elicits a marked painful grunt.

8-. Auscultation of the thorax reveals the diagnostic findings. In the early stages before effusion commences, the heart sounds are normal but are accompanied by a pericardial friction rub, which may wax and wane with respiratory movements. Care must be taken to differentiate this from a pleural friction rub due to inflammation of the mediastinum. In this case the rub is much louder and the heart rate will not be so high.

9-Several days later when there is marked effusion, the heart sounds are muffled and there may be gurgling, splashing or tinkling sounds.

10-Most affected animals die within a period of 1-2 weeks, although a small proportion persist with chronic pericarditis.

11-The obvious clinical findings in the terminal stages are gross edema, dyspnea, severe watery diarrhea, depression, recumbency and complete anorexia.

12-Enlargement of the liver may be detectable by palpation behind the upper part of the right costal arch in the cranial part of the right paralumbar fossa.

13-Death is usually due to asphyxia and toxemia. Animals which have recovered from an initial pericarditis are usually affected by the chronic form of the disease.

14-Body condition is poor, the appetite is variable, there is no systemic reaction and the demeanor is bright.

## Clinical pathology

1-Hemogram A pronounced leukocytosis with a total count of 16 000-30 OOO/pL accompanied by a neutrophilia and eosinopenia is usual.

## Necropsy findings

1-In acute cases there is gross distension of the pericardial sac with foul-smelling, grayish fluid containing flakes of fibrin, and the serous surface of the sac is covered by heavy deposits of newly formed fibrin.

2-A cord-like, fibrous sinus tract usually connects the reticulum with the pericardium.

3-Additional lesions of pleurisy and pneumonia are commonly present.

4-In chronic cases the pericardial sac is grossly thickened and fused to the pericardium by strong fibrous adhesions surrounding loculi of varying size which contain pus or thin straw-colored fluid.

## Treatment

The results of treatment are usually unsatisfactory but salvage of up to 50% of cases can be achieved by long-term treatment with antimicrobials. Selected cases of traumatic pericarditis have been treated satisfactorily by pericardiotomy.