Diagnosis and Treatment of Hardware Disease

Matt D. Miesner, DVM, MS*, Emily J. Reppert, DVM, MS

KEYWORDS

• Hardware • Rumen • Peritonitis • Pericarditis • Ruminant • Rumenotomy

KEY POINTS

- Cattle frequently ingest irregular objects with potential risk of rumeno-reticular damage and perforation, followed by peritonitis, pleuritis, and/or pericarditis, in addition to sepsis, restrictive adhesions, and/or abscess formation.
- Patients with hardware disease often present with client complaints of rumeno-reticular dysmotility (bloat), abdominal discomfort (colic), anorexia, lethargy, and weight loss (falling behind).
- During complete physical examination, heed evidence of pain localized to the cranioventral abdomen, abnormal auscultation findings consistent with rumen dysmotility, and pleural/peritoneal/pericardial effusion and inflammation.
- Ancillary diagnostics including an inflammatory leukogram, radiographs, and ultrasound are advantageous for diagnosis before exploratory laparotomy or rumenotomy.
- Treatment of hardware disease is directed at controlling infection and removing foreign bodies when possible: however, prevention should be the primary emphasis.

Video content accompanies this article at http://www.vetfood.theclinics.com.

INTRODUCTION: NATURE OF THE PROBLEM

Traumatic reticuloperitonitis (TRP), resulting from penetration of the reticulum by a foreign body, is a common gastrointestinal (GI) disorder affecting adult dairy cattle and, less commonly, beef cattle. TRP results from the indiscriminate grazing habits and accidental ingestion of foreign bodies. Ingestion of a foreign body has 4 potential outcomes[1,2]:

1. Attachment to a previously administered magnet with no development of clinical disease
2. Penetration of the reticular wall without entering the peritoneal cavity, causing focal reticulitis and mild clinical disease

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Veterinary Clinical Sciences, Kansas State University, 1800 Denison Avenue, Manhattan, KS 66506, USA
* Corresponding author. 103J Mosier Hall, Manhattan, KS 66506.
E-mail address: mmiesner@vet.k-state.edu

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3. Perforation of the reticular wall and entrance to the peritoneal cavity causing acute localized TRP
4. Perforation of the reticular wall and entrance to the peritoneal or thoracic cavity resulting in pericarditis, myocarditis, abscessation, vagal indigestion, or other secondary disease

TRP remains a primary cause of vagal indigestion in cattle. Type II vagal indigestion (failure of omasal transport) is the most common type of vagal indigestion associated with TRP.

PATIENT HISTORY

In acute cases of TRP, animals typically develop clinical signs within 24 hours of the foreign body penetrating the reticular wall. Animals become anorexic, agalactic, reluctant to move, anxious, and in some cases may have an arched back.[2] Uncomplicated cases may resolve within 3 to 5 days after the initial acute episode. Resolution of disease is marked by increased appetite and return to normal milk production. In more complicated cases, development of clinical disease is protracted and clinical signs may remain static for days to weeks. Progression of disease may be the result of failure to localize the peritonitis or involvement of other organs resulting in ongoing disease. Cattle with chronic TRP may have decreased feed intake, milk production, and fecal output for prolonged periods of time.

PHYSICAL EXAMINATION

Early signs of TRP develop shortly after the foreign body perforates the reticular wall resulting in localized peritonitis. Acute disease caused by TRP is characterized by fever, anorexia, decreased to absent rumen contractions, and cranial abdominal pain. Evidence of cranial abdominal pain includes any of the following:

1. Absent ventroflexion with pressure applied to the withers
2. Grunt with pressure applied to the withers
3. Grunt with dorsal pressure applied to the xyphoid
4. Reluctance to move
5. Arched back when standing
6. Forelimbs held in an abducted position

TRP remains the most common cause of cranial abdominal pain in adult cattle. Differentials for cranial abdominal pain in cattle include[3]:

1. Localized peritonitis secondary to TRP, perforating abomasal ulcer, superficial hepatic abscess
2. Pleuritis
3. Pericarditis
4. Endocarditis

Other nonspecific clinical findings of TRP include abduction of the elbows, tachycardia, and tachypnea. Persistence or progression of clinical signs may indicate failure to contain the peritonitis or involvement of other structures. Sequela associated with TRP depends on the size, shape, and location of penetration. Penetration of the reticular wall and diaphragm can lead to bacterial seeding of the peritoneal, thoracic, and pericardial spaces resulting in peritonitis, pleuritis, and pericarditis. Inflammation, abscessation, and adhesions of the reticulum can lead to abnormal reticuloruminal motility and the development of vagal indigestion. Papple (L-shaped) abdominal