Abdominal disease in calves: A diagnostic challenge

Erin Fierheller, DVM

The diagnosis of colic in calves presents a challenge for veterinarians. The main goal in examining calves with suspected abdominal disease is to determine whether a surgical or medical condition is present. Once the nature of the condition has been ascertained, decisions regarding treatment and prognosis can be made. Since every case of abdominal disease presents differently, often the diagnosis cannot be made on the physical examination alone; therefore, some general principles will assist in forming rational diagnostic and therapeutic protocols. This issue of Large Animal Veterinary Rounds provides a summary of specific painful gastrointestinal disorders in calves and discusses their diagnosis, treatment, and prognosis. Two case discussions of acute abdominal problems in calves are included at the end of the article.

Abdominal disorders are commonly encountered in young calves < 2 months of age. Diagnoses may range from simple gas colic to strangulating obstructions that require immediate surgical intervention. The most difficult decision for veterinarians is determining whether medical or surgical therapy is needed. In horses with colic, the prognosis can be predicted from the physical examination and laboratory indices of shock. In calves, however, similar processes have different manifestations, and signs must be interpreted carefully. Therefore, the history and physical examination findings become very important in evaluating colicky calves.

History

The history should include the number of affected animals in the herd and the presence of concurrent disease. Some calves with enteritis may be colicky in the peracute stages of enteritis or may develop an intussusception. Most calves presenting with obstructive gastrointestinal disease exhibit abdominal distension, acute signs of abdominal pain, or inappetence (Table 1). Abdominal distention is the most common presenting complaint and may be present in 75% of cases. Signs of abdominal pain include kicking at the belly, paddling of the feet, getting up and down, rolling, bruxism, and pain on abdominal palpation. Of these, pain on abdominal palpation is the most common sign of colic and is seen in about 75% of cases.

The age of the affected calf is an important consideration. Calves < 1 week old are much more likely to have a lesion requiring surgical therapy (73% have surgical lesions), with intestinal aplasia being the most commonly diagnosed condition. These calves have a significantly poorer prognosis than older calves that are more likely to have medical lesions (only 26% have surgical lesions). In a retrospective study by Naylor and Bailey that excluded cases of enteritis, 69% of the calves presenting with abdominal disorders in their first week of life, died.

The progression of clinical signs over time is also of diagnostic value. Rapidly progressive disorders where the calf becomes depressed, dehydrated, and recumbent over a period of hours suggest...
a more severe lesion.² Calves with a slower progression of deterioration are more likely affected by either medical disorders or non-strangulating obstructions.²³ In many cases, the decision for surgical exploration is based on the failure to respond to medical therapy and the deterioration of the calf’s physical condition.

The physical examination

A thorough physical examination is essential in arriving at a diagnosis and in assessing prognosis. Concurrent diseases will influence treatment and prognosis. A list of differential diagnoses for abdominal distention and colic in calves is given in Table 2. Temperature, heart rate, and respiratory rate should be recorded and the cardiovascular status and degree of dehydration should be assessed. In calves with abdominal disease (excluding enteritis), the heart rate and the degree of abdominal pain are not useful prognostic indicators. However, prognosis worsens as the severity of dehydration, hypothermia, and recumbency increase. In a retrospective study, 75% of calves that were recumbent on presentation, died from their condition.¹ The umbilicus should be auscultated for evidence of pneumonia. The umbilicus should be examined for evidence of herniation, urachal patency, and/or omphalophlebitis. In bull calves, the scrotum and inguinal regions should be palpated for hernias.

Although clinical examination of the digestive tract and abdomen of the calf is more difficult than in adult cattle, it is of critical importance for an accurate diagnosis. Examination of the abdomen includes:

- Deep palpation of abdominal organs
- Percussion and auscultation of the presence of fluid-splashing sounds indicative of a fluid-filled viscus,
- Abdominal distention
- Abdominal pain
- Depression
- Inappetence
- Dehydration
- Melena
- Decreased fecal production

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<thead>
<tr>
<th>Table 1: Clinical signs of abdominal disorders in calves with lesions that may require surgical correction</th>
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<tr>
<td>• Abdominal distention</td>
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<td>• Abdominal pain</td>
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<td>• Depression</td>
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Diagnoses for abdominal distention and pain in calves

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<th>Table 2: Differential diagnoses for abdominal distention and pain in calves</th>
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<tr>
<td>Medical</td>
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<td>• Acute enteritis</td>
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<td>• Omphalophlebitis</td>
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<td>• Pneumonia</td>
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<tr>
<td>• Diffuse peritonitis</td>
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<tr>
<td>• Gastrointestinal tympany</td>
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<td>• Non perforating abomasal ulceration</td>
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<tr>
<td>Surgical</td>
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<tr>
<td>Peracute to acute, severe onset</td>
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<tr>
<td>• Abomasal torsion</td>
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<td>• Mesenteric torsion</td>
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<tr>
<td>• Perforating abomasal ulcerization</td>
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<tr>
<td>Acute to subacute onset</td>
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<tr>
<td>• Abomasal dilation (LDA, RDA)</td>
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<td>• Intussusception</td>
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<td>• Intestinal atresia</td>
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Diagnostic tests

Abdominocentesis may be attempted with the calf in left lateral recumbency. To avoid puncture of the abomasum, the site should be dorsal and caudal to the umbilicus, or in the center of the inguinal region.⁵ Although peritoneal fluid cytology is a highly sensitive indicator of peritoneal disease, it does not often indicate the definitive diagnosis. Failure to obtain peritoneal fluid does not preclude the possibility of inflammatory disease.⁵ Hemograms may be useful for diagnosis because substantial inflammatory changes indicate the presence of severe vascular compromise to the gut or diffuse peritonitis.¹ However, they may not be practical without hospital laboratory facilities. Severe hypoproteinemia usually indicates diffuse peritonitis; severely dehydrated calves are more likely to have a surgical lesion (except in calves with enteritis), and severe leukopenia and a left shift are suggestive of a poor prognosis.¹

Abdominal radiography can be used to identify gaseous distention, but often it will not provide a definitive diagnosis, and therefore, it may not be economically feasible. In cases where atresia coli is suspected, contrast radiography with a barium enema may support the diagnosis.

Based on physical examination findings and history, a list of likely differential diagnoses can be formulated and a decision made about whether the calf will be managed medically, surgically, or not at all. In many cases, a definitive diagnosis cannot be made without exploratory laparotomy. Table 3 lists the differential diagnoses for colicky calves. The diseases most commonly diagnosed include: atresia coli, diseases of the abomasum, diffuse peritonitis, and enteritis.

Atresia coli

Intestinal atresia is the most common abdominal disease in calves < 8 days old.¹ Affected calves are usually normal at birth, but develop clinical signs 24–48 hours after birth. Characteristic clinical findings include inappetence, progressive bilateral abdominal distention, and the absence of feces. Clear or blood-tinged mucus can be found in the rectum and intermittent paroxysms of straining may occur.⁵ Abdominal radiography reveals distended intestinal loops.
and the absence of feces in the rectum. A barium enema may aid in identifying the site of the lesion. However, atresia coli is most often definitively diagnosed by performing an exploratory laparotomy.

Passing a catheter up the rectum is not a useful diagnostic tool since the majority of atresia coli cases in calves involve the spiral loop of the ascending colon. In addition, this procedure carries the risk of colonic perforation.

Treatment of atresia coli involves surgical resection and anastomosis. Resection of the proximal dilated blind end of the spiral colon and end-to-side or end-to-end anastomosis with the descending colon is the recommended surgical technique. An enterotomy to evacuate retained meconium and fluid is performed at the apex of the cecum. Short-term survival rates after surgery range from 43% to 71%, with 85% of surviving calves having postoperative complications. Long-term survival rates are in the range of 35%–40%, but many of these calves have chronic loose feces and poor growth rates. Therefore, surgical treatment is usually not recommended or economical.

The cause of atresia coli is unknown. There have been reports suggesting that ileal and jejunal atresia have a hereditary basis. Rectal and anal atresia are known to be an inherited defect. However, evidence suggests that atresia coli is not an inherited defect.

### Diseases of the abomasum

#### Abomasal volvulus

Abomasal volvulus occurs in calves >8 days old. Calves usually present with an acute or peracute onset of abdominal pain, dehydration, and shock. These clinical signs often progress rapidly. The abdomen is usually distended (often more prominent on the right), and a viscus may be palpated or visualized caudal to the ribs on the right side. A large ping and fluid-splashing sounds may be found on auscultation over the right side of the abdomen. Treatment involves emergency right flank laparotomy, abomasal decompression, and correction of the torsion. Most abomasal torsions occur in a counter-clockwise direction when viewed from the rear of the calf. Prognosis for survival is around 50% if the calf is diagnosed and treated early. If the calf is recumbent upon presentation, the prognosis for survival is poor (25% survival).

#### Gastrointestinal tympany

Gaseous distention of the intestinal tract is the most common cause of abdominal pain in calves >1-week old. It is frequently seen in calves on milk replacer or ad libitum diets. The pathogenesis is thought to involve the consumption of large amounts of milk resulting in bacterial proliferation in the gastrointestinal tract. These bacteria increase gas production in the abomasum and cecum. Clinical signs include an acute onset of colic and abdominal distention. A large ping may be auscultated over the right flank. Fluid-splashing sounds may be elicited over the right, left, or both sides of the abdomen. These clinical signs are similar to those seen with abomasal torsions and displacements. Therefore, a definitive diagnosis is often made at surgery. If calves with tympany are managed medically, they usually remain standing and do not become severely dehydrated or hypothermic, unlike calves with volvulus. The prognosis for survival in calves with gastrointestinal tympany is good.

#### Abomasal dilatation/displacement

The prevalence and etiology of displaced abomasum in calves is poorly understood, although displacements are typically seen in calves >1-week old. The abomasum in calves can displace to the left or the right, however, left displacements are rare. Clinical signs include depression, anorexia, and decreased fecal production. In chronic cases, poor weight gain and recurrent tympany may be seen. With simple dilatations, abdominal enlargement is evident predominantly on the side of the displacement. A large, gas-distended viscus can be percussed and auscultated, and there may be associated fluid-splashing sounds on abdominal succussion.

Differentiation between abomasal volvulus, abomasal displacement, and gastrointestinal tympany can be very difficult. These disorders may in fact be varying severities of the same disease process. There is speculation that abomasal displacement may result from gas or volatile fatty acid accumulation. The introduction of grain or pelleted feeds to the diet is considered one possible cause of this increased gas and volatile fatty acid production. Other primary diseases that can cause abomasal atony (eg, pneumonia or peritonitis), should also be considered.

#### Abomasal ulceration

There is a perception among veterinarians and cow-calf producers that the incidence of fatal abomasal ulcers in unweaned beef calves is on the increase. Although the cause is unknown, there are 4 general theories:

- trace mineral deficiencies
- bacterial or fungal agents,
- abrasive agents
- stress.

In a field investigation by Jelinski et al, the incidence of fatal abomasal ulcers per farm was 1.9%–2.4%. This study found no correlations between cases of perforating ulcers
and mineral supplementation, *Clostridium perfringens* type A, abrasive agents, or stress. However, only cases of fatal perforating abomasal ulcerations were included and cases of subclinical nonperforated ulcers were not examined. Other studies have linked abomasal trichobezoars to ulcer formation. Although many calves with abomasal ulcers also have hairballs, there is no evidence to suggest that trichobezoars are a precipitating factor. In addition, many calves without ulcers have hairballs.

Clinical signs can vary and include depression, abdominal pain, abdominal distention, and inappetence. Abdominal pain may be expressed as bruxism. In most cases, calves are found dead without any premonitory signs. Affected calves are usually average or above average in growth performance.

One theory for ulcer formation involves the age when it occurs most commonly. The majority of calves with fatal abomasal ulcers are <2 months old. Forestomach development generally occurs between 3 to 8 weeks after birth. It is speculated that there may be some form of “undifferentiated abomasal dysfunction,” associated with forestomach development, predisposing calves to developing abomasal disorders such as ulcers, tympany, and dilatations.

Ulcers are also seen in hand-fed calves when started on roughage diets, and suckling veal calves between 3 to 5 months of age. The prevalence in healthy veal calves is 57%-75%. Clinical signs similar to those seen in beef calves may occur and in some cases, melena is evident. These calves more commonly have subclinical ulcers that cause no apparent illness. Recent studies on the treatment of abomasal ulcers describe the effectiveness of type 2 antihistamine blockers and antacids. Administered orally, these drugs decrease gastric acid pH. Recommended dosages include cimetidine (50-100 mg/kg PO, q 8 hr), or ranitidine (10-50 mg/kg PO, q 8 hr). The use of aluminum hydroxide and magnesium hydroxide compounds (50 ml containing 5.0 g of Al[OH]₃ and 4.5 g of Mg[OH]₂ PO, q 8 hr) also increase gastric pH. However, higher doses may have adverse effects such as metabolic alkalosis, diarrhea, and hypermagnesemia. Feeding milk or milk replacers alone will cause transient increases in gastric pH. These studies were done on healthy veal calves and their effect on intestinal ulcers is unknown. In addition, the cost of therapy may preclude treatment as a practical option.

**Other diseases that may require surgical correction**

Other surgical intestinal disorders include intussusceptions, small intestinal volvulus, cecal torsion, and intestinal enteroliths or other foreign bodies. Often diagnosis of these conditions is made on exploratory laparotomy since clinical signs are variable. In calves with intussusception, there may be evidence of melena. Affected calves are depressed, weak, inappetent, show abdominal discomfort, and pass scant feces. There may be a history of diarrhea and the abdominal contour may appear normal or slightly distended. Often, a definitive diagnosis is difficult to make without an exploratory laparotomy. Prognosis after surgical resection and anastomosis is good, provided the segment of small intestine affected is capable of being exteriorized through the surgical incision.

**Diffuse peritonitis**

Acute diffuse peritonitis is usually seen in calves <3 weeks old, often within the first week of life, and is associated with septicemia or another concurrent infection (enteritis, omphalophlebitis, polyarthritis). Definitive diagnosis can be made with cytology of peritoneal fluid or with an exploratory laparotomy. The prognosis for survival is poor, although antibiotic therapy and abdominal lavage have been attempted.

**Enteritis**

Acute enteritis is the most common neonatal disease affecting young calves <2 months of age. Diarrhea, dehydration, and depression are common clinical findings. However, some cases may present peracutely with abdominal enlargement and pain, without evidence of diarrhea. In these early stages, dehydration may not be evident so that these calves closely resemble calves with other intestinal diseases. Fluid splashing is often evident on abdominal succussion. Loud, intestinal, peristaltic sounds may be heard over the abdomen and a digital rectal examination may stimulate defection of diarrheic feces.

The differentiation between acute enteritis and abdominal obstruction can be very difficult. In cases where the need for surgery is questionable, supportive care and monitoring for progression of clinical signs is warranted. Exploratory laparotomy in calves with enteritis does not affect the prognosis.

**Case discussions**

**Case 1**

A 4-day-old Simmental bull calf is presented with a 1-day history of progressive abdominal distension and inappetence. The calf was born without assistance, but has not sucked well since birth. Colostrum, then milk, were tube-fed twice daily. There is no history of calf diarrhea in the herd.

On presentation, the calf is standing, depressed, and intermittently kicking at its belly. Temperature, heart
rate, and respiratory rate are 39.4°C (38.5-39.5°C), 180 (100-120 beats/minute), and 40, (20-50 breaths/minute), respectively (normal values). The calf is not clinically dehydrated and the mucous membranes and capillary refill time are normal. On palpation, the external umbilicus contains a fluid-filled sac; proximally, the umbilicus is slightly thickened, but not painful. There is no umbilical hernia. A sample of red-tinged fluid is obtained by fine needle aspiration of the distal umbilicus. The fluid contains high urea, as determined by a blood urea test strip. The abdomen is bilaterally distended, but not painful on palpation. On percussion, there is a large ping over the right flank. Fluid-splashing sounds are heard over the left abdomen on ballottement. Examination of the perineum reveals no evidence of fecal staining and on digital rectal palpation, red-tinged mucus is present in the rectum, but no feces are found. On percussion and auscultation with the calf in dorsal recumbency, the ping encompasses the right central abdomen, but does not cross the ventral midline. A stomach tube is passed and small amounts of milk clot, but no gas, are siphoned from the rumen.

**What are the differential diagnoses, therapeutic options, and prognosis for survival in this calf, with and without surgical intervention?**

The differential diagnoses in this calf should include atresia coli (no evidence of feces), abomasal dilatation (left-sided ping), and a urachal defect (urine-filled umbilical pocket). Peracute enteritis can also be considered; however, the calf is relatively alert. To differentiate intestinal obstructions from peracute enteritis, the calf should be monitored for the progression of clinical signs and continued absence of feces. A strangulating obstruction is not suspected since the calf has remained relatively alert over the previous day and there is no evidence of cardiovascular compromise other than an elevated heart rate, possibly due to pain. Atresia coli is the major differential diagnosis due to the lack of feces, slow clinical progression of the disease, and the age of the calf. The abomasal dilatation may be a separate problem, but the cause and significance of this finding is unknown.

Surgical treatment may be offered, however, this option is often not economically feasible due to the poor prognosis for survival and the potential for concurrent diseases (urachal defect, abomasal dilatation). Surgery would, however, provide a definitive diagnosis. In this case, the calf was taken home and died 5 days later. Segmental aplasia of the ascending colon was found at necropsy.

**Case 2**

A 1-month-old Hereford heifer calf has a 1-day history of inappetence and depression. She received colostrum at birth and has had no previous illness. The calf was seen lying down and kicking at her belly. Her condition has rapidly deteriorated over the past few hours and she is now in lateral recumbency and unwilling to stand. The temperature, heart rate, and respiratory rate are 38.5°C (38.5-39.5°C), 140 (100-120 beats/minute), and 72 (20-50 breaths/minute, respectively. The mucous membranes are pale and the capillary refill time is >5 seconds. Dehydration is between 5%-10% based on the evaluation of a skin tent and the sunken eyes. The abdomen is bilaterally distended and a large ping and fluid-splashing sounds are elicited on auscultation, percussion, and succussion over the right abdomen. The lung sounds and the navel are normal. An abdominocentesis is not performed. The area of tympany does not resolve after a stomach tube is passed.

**What are the differential diagnoses, therapeutic recommendations, and prognosis for this calf?**

The physical examination findings and history for this calf suggest an acute gastrointestinal disorder requiring surgical therapy. The evidence of shock (dehydration and recumbency) suggest a possible strangulating obstruction. Differential diagnoses for this calf include abomasal torsion, intussusception, perforated abomasal ulcer, and mesenteric torsion. Although recumbency indicates a poor prognosis for survival, surgical exploration is warranted early in the course of the disease for more valuable calves.

The calf is surgically prepared for a right flank laparotomy and started on intravenous Lactated Ringers fluids at 4 L/hr. Analgesia is obtained with a lidocaine inverted-L line block. An 180°, counter-clockwise, abomasal torsion is found at surgery. After decompression and correction of the torsion, the abomasal serosa is found to be slightly reddened and edematous, but is considered viable. There are no other lesions found on abdominal exploration. The abdomen is closed and the heifer recovers uneventfully from surgery. She is discharged from the hospital 3 days later.

**Conclusion**

Clinical signs of colic, abdominal distention, and decreased feces in calves with abdominal disease are non-specific and many cases are a diagnostic challenge. Whether an individual case requires surgical or medical
therapy is frequently difficult to determine and often, an exploratory laparotomy is necessary to obtain a diagnosis. Although unnecessary surgeries are not economical for the producer, surgery does not affect the prognosis in calves with medical lesions. In cases where the calf is in stable physical condition and the diagnosis is unclear, medical therapy is feasible, as well as monitoring the calf for progression of clinical signs.

Calves presenting with abdominal disorders (other than enteritis), <8-days old, are more likely to have surgical lesions and a poorer prognosis compared to older calves. Approximately three-quarters of calves <8 days of age who present with colic and abdominal distention have surgical lesions. Seventy-five per cent of animals that are recumbent on presentation die, despite aggressive therapy.

Abstract of Interest

Effect of orally administered cimetidine and ranitidine on abomasal luminal pH in clinically normal milk-fed calves

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Objective: To characterize the change of pH in the abomasal lumen throughout a 24-hour period, to determine whether pH of the abomasal body differs from pH of the pyloric antrum, and to determine whether oral administration of cimetidine and ranitidine alters pH of the abomasal lumen in milk-fed calves.

Animals: 5 male dairy calves (4 Holsteins-Friesian, 1 Ayshire), 5 to 15 days old.

Procedure: Cannulas were surgically positioned in the abomasal body and pyloric antrum of each calf. Calves received the following treatments in a randomized crossover design: milk replacer (60 m/kg of body weight, q 12 h [untreated control calves]), milk replacer and cimetidine (50 or 100 mg/kg, q 8 h), or milk replacer and ranitidine (10 or 50 mg/kg, q 8 h). The pH of the abomasal body and pyloric antrum was measured for 24 hours, using miniature glass pH electrodes.

Results: Suckling of milk replacer immediately increased abomasal luminal pH from 1.4 to 6.0, followed by a gradual decrease to preprandial values by 6 hours. Preprandial and postprandial pH values were not significantly different between the abomasal body and pyloric antrum, indicating lack of pH compartmentalization in the abomasum of milk-fed calves. Administration of cimetidine and ranitidine caused a significant dose-dependent increase in mean 24-hour abomasal luminal pH.

Conclusions and Clinical Relevance: Abomasal acid secretion in milk-fed calves is mediated in part by histamine type-2 receptors. Cimetidine and ranitidine may be efficacious in the treatment of abomasal ulcers in milk-fed calves.


Dr. Erin Fierheller received the WCVM gold medal award on graduation in 2000. After graduation, she entered into mixed practice for one year at the Leduc Veterinary Hospital Ltd., in Alberta. She is currently completing her first year of a mixed practice for one year at the Leduc Veterinary Hospital on graduation in 2000. After graduation, she entered into

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