Intussusception in Horses

Bradley B. Nelson, DVM
Sabrina H. Brounts, DVM, MS, DACVS, DECVS
University of Wisconsin–Madison

Abstract: Intussusception is thought to be associated with abnormal motility in the intestinal tract. The clinical signs vary depending on the associated intestinal damage. Therefore, intussusception should be included in the differential diagnosis for any horse with acute or chronic colic. A diagnosis may be made by ultrasonography or rectal palpation, but exploratory laparotomy is the predominant method. Treatment varies according to the location of the intussusception, but all treatments involve surgical intervention. The prognosis for intussusception can be good if the condition is diagnosed and treated promptly.

Although intussusception is a relatively uncommon cause of colic in adult horses, it must be considered in the differential diagnosis of horses with acute or chronic abdominal pain. Intussusception can cause serious pathology and must be diagnosed and treated promptly with surgery before irreversible damage occurs. During intussusception, a portion of bowel telescopes into a more distal section. The proximal end is the intussusceptum, and the distal, receiving portion is the intussuscipiens.1 As the intussusceptum invaginates into the distal bowel, the mesenteric blood flow becomes occluded. If the intussusceptum is short, mural flow is preserved and the intussusception is a simple obstruction. With a longer intussusceptum, mural flow is occluded, leading to edema of the intussusceptum. The swelling can retain this segment within the intussuscipiens, leading to further vascular obstruction, adhesion formation, and bowel necrosis. Typically, a significant portion of involved bowel produces a large obstruction, leading to acute colic and a lack of response to analgesics.

Intussusception can occur in multiple locations throughout the alimentary tract. Small intestinal intussusceptions can affect the jejunojejenum, jejunoleum, ileoleum, or ileocecum; large intestinal intussusceptions can affect the cecocecum, cecocolon, colon, or colorectum. A rare case report described a pyloric-duodenal intussusception.2 This article focuses on the more common locations.

Small Intestinal Intussusception

Etiology and Clinical Signs

Small intestinal intussusception is thought to develop because of abnormal intestinal peristalsis.1 Horses may be predisposed to intussusception because of enteritis, rapid dietary changes, tape-worm or ascarid parasitism (e.g., *Parascaris equorum* infection), mesenteric arteritis, previous abdominal surgery, intraluminal foreign bodies, and intramural masses.1,3,4 There is no obvious breed or sex predisposition, but intussusception is more common in young horses (6 months to 3 years of age).5,6 Ileoceleal intussusception is the most prevalent type, with an incidence of 74% in one report.1

The various clinical signs of small intestinal intussusception can range from mild, intermittent abdominal pain with reduced appetite and fecal output to severe colic with severe dehydration due to complete obstruction of the small intestine. The clinical signs depend on the extent of bowel involved, the location, and whether the lumen is obstructed.7 A horse with chronic intussusception may present with a reduced appetite, reduced fecal output, and pyrexia, making diagnosis difficult.1,5 Ileoleal intussusception has a short length, and affected horses usually present with chronic colic;
jejunojejunal intussusception is longer and causes more intense colic.1

Diagnosis
The patient history, clinical signs, and physical examination may confirm abdominal pain that is not specific for intussusception. Rectal palpation can reveal dilated loops of small intestine, but the diagnostic sensitivity of rectal palpation alone is low. On occasion, a clinician may be fortunate enough to palpate a firm tubular structure.1,4 For diagnosis of ileocecal intussusception, reports have suggested that a painful, tight, tubular mass in the right dorsal quadrant near the base of the cecum can be palpated.4,5

Transabdominal ultrasonography is potentially useful for diagnosing intussusception. The sensitivity of this diagnostic test is low, but if a “target” or “bull’s eye” lesion is observed, the diagnosis is definitive. The diagnostic unreliability of ultrasonography is predominantly due to the likelihood that the lesion is too close to the body wall to be detected. On an ultrasonogram, two concentric rings appear around a central hypoechoic structure (the lumen of the intussusceptum; FIGURE 1). Between the two bowel segments, there may be another hypoechoic line indicating intestinal fluid with or without fibrin. Both segments may be thickened due to mural edema.8,9

Abdominocentesis can be used as a general diagnostic aid to detect intestinal injury or peritonitis, but it should not be used as the primary tool for definitively diagnosing intussusception. The intussuscipiens partially seals the intussusceptum or at least delays leakage of cellular evidence of bowel injury; therefore, results of abdominocentesis may not accurately reflect the condition of the bowel. Although many diagnostic modalities are available, definitive diagnosis is most often attained by exploratory ventral midline celiotomy or by necropsy if the horse does not survive (FIGURE 2). Although this method is the most reliable, the other procedures discussed above should be used to aid diagnosis. On abdominal exploration during celiotomy, the surgeon finds the proximal portion of the intestine and its subsequent mesentery invaginated within the intussuscipiens. Proximal dilation of the intestine with intramural thickening (edema), fibrinous adhesions, and ecchymoses on the antimesenteric surface can be documented.1 Depending on the level of vascular compromise, the proximal and distal segments may be hemorrhagic, necrotic, or friable; however, the intussuscipiens may appear normal.10

Treatment
Specific treatment of small intestinal intussusception depends on several factors, but surgical intervention is always required. Once the lesion is located, reduction may be attempted by gentle traction on the proximal portion and massage of the intussuscipiens.1,4,5,11 For cases involving extensive necrosis, severe bowel ischemia, or adhesion formation, reduction is contraindicated1,4,11–13 because of increased risk of intestinal rupture, abdominal contamination, and peritonitis. If the intussusception is not reducible, incomplete bypass or resection of the entire affected segment can be performed.4,5,12

Incomplete bypass is mainly an option for ileocecal intussusceptions. This quick technique is an option if bowel necrosis is not present. However, for other sites of small intestinal intussusception, complete resection and anastomosis of the small intestinal segment is more often indicated because of the presence of devitalized bowel. The literature describes multiple resection techniques, including the hand-sewn end-to-side, hand-sewn end-to-end, stapled functional end-to-end, and stapled side-to-side techniques.1,4,11–13 In a study involving the latter three techniques for jejunojejunal intussusception, no technique was associated with a better long-term survival rate. However, the stapled side-to-side technique was associated with reduced duration of postoperative ileus.13

Complications associated with small intestinal intussusception repair depend on the location and extent of viable bowel, the ability for reduction, and the surgeon’s experience. No treatment is universal; therefore, treatment must be tailored to the individual patient. Potential postoperative complications include colic, ileus, diarrhea, adhesions, fever, tachycardia, incisional infection, peritonitis, a decrease in stoma size, ileal hypertrophy, and recurrence of intussusception.1,13,17 In chronic cases involving incomplete bypass via side-to-side ileocecostomy, ileal hypertrophy and subsequent rupture have been noted.17 Ileal hypertrophy may not always progress to rupture, but it may lead to luminal obstruction. After jejunocecostomy in one case, the cecocolic orifice was obstructed by a large, hypertrophied ileal stump, resulting in ileoceccocolic intussusception.18

Patient care following surgical treatment of intussusception should be determined by each patient’s needs. Hypovolemia should be corrected with intravenous balanced polyionic crystalloid solutions. Intraabdominal lavage may be indicated if peritonitis is expected to affect patient recovery. Additional therapy, such as administration of analgesics, antimicrobials, and/or promotility agents, should be used as needed.
**Prognosis**

The prognosis for small intestinal intussusception depends on many factors, including the length of bowel affected, duration of the lesion, viability of the bowel, whether resection and anastomosis are needed, and the health status of the horse at the time of surgery. The longer the intussusception is present and the intestine compromised, the worse the prognosis. In generalized studies of small intestinal intussusceptions, >50% of patients survived long term and returned to normal function, with the ileoileal group having the best outcome. While the other factors affecting prognosis depend on individual case reports, survival rates at hospital discharge after jejunal resection and anastomosis were 81% to 91%, with rates of 48% to 68% 1 year later. If resection was not required, the rate of survival to discharge was 92% and the 1-year survival rate was 77%. The use of side-to-side anastomosis instead of end-to-end anastomosis for jejunojejunostomy resulted in no significant difference in long-term survival. Decreased anesthetic time with the use of an automatic stapling device has been shown to improve the prognosis.

**Large Intestinal Intussusception**

**Etiology and Clinical Signs**

Cecal intussusception results from invagination of the cecal apex into the base (ceccocum) or through the cecocolic orifice into the right dorsal colon (ceccocolon; **Figure 3**). These are the most common large intestinal intussusceptions. Intussusception of the large colon has also been observed. The left ventral colon can telescope on itself proximal to the pelvic flexure, and the left dorsal colon can intussuscept on itself.20–22 The small colon can also become intussuscepted, but this is extremely rare, with only a few case reports in the literature.20–24

Large intestinal intussusception is also thought to be due to altered intestinal motility resulting from tapeworm (*Anoplocephala perfoliata*) infestation, cecal wall abscessation, deworming with organophosphates, vascular compromise by infection with *Strongyloides vulgaris* or *Eimeria leuckarti*, or administration of parasympathomimetic drugs. Some authors dispute the clinical significance of tapeworm infestation as a cause of intussusception, but many report at least a correlation between the two, even if it is not clinically significant. There is no age or breed predilection for these types of intussusception, but these causes are most common in horses aged 2 to 3 years.

Clinically, three syndromes are associated with large intestinal intussusception:

- Acute syndrome
- Subacute syndrome
- Chronic wasting syndrome

These syndromes are related to the severity of colic. Depending on the clinical syndrome, an affected horse displays various levels of abdominal pain and clinicopathologic abnormalities. In general, the severity of clinical signs decreases with chronicity. The clinical signs range from severe abdominal pain in the acute syndrome to weight loss, fever, and mild intermittent colic in the chronic wasting syndrome. All the syndromes can progress to a devitalizing lesion and more severe colic. In one report, 55% of horses presented with acute syndrome, 30% presented with subacute syndrome, and 13% presented with chronic wasting syndrome.

**Diagnosis**

Diagnosis of large intestinal intussusception requires the same diagnostic aids used in small intestinal intussusception. The patient history, clinical signs, and physical examination are related to the form of disease and usually reveal nonspecific colic. Retraction of the cecum may be painful on rectal palpation in the right dorsal quadrant. In some cases of cecocolic intussusception, the base of the cecum cannot be palpated because of its movement into the cranial abdomen.

Ultrasonography can be used to diagnose large intestinal intussusception. The cecum is normally close to the body wall; therefore, ultrasonography may be more useful for diagnosing cecocolic intussusceptions than for small intestinal lesions. Ultrasonography shows the outer intussusciens as sacculated and separated from the thick-walled, congested, edematous inner intussuscipiens. Cecocolical intussusceptions are found in the right ventral abdomen because the apex is invaginated and displaced from its normal location. Cecocolical intussusceptions are located in the upper right abdominal quadrant because the cecum invaginates into the right ventral colon.

Diagnosis is predominantly by exploratory ventral midline celiotomy or necropsy. On abdominal exploration, the cecal apex is absent and a firm mass is palpated in the base of the cecum or the right ventral colon. Pathologic findings include adhesions, bowel ischemia and necrosis, and a friable, hemorrhagic, edematous cecum.

**Treatment**

Treatment of intussusception always includes surgical correction. Cecocolical intussusception can often be reduced manually and
may require resection only if the apex is devitalized. As described for small intestinal intussusceptions, simple reduction may predispose patients to recurrence of intussusception. However, because most ceca are devitalized at the time of surgery, partial typhlectomy is commonly performed.

Because cecolic intussusceptions are associated with more edema and adhesion formation, they typically are not manually reducible. Colotomy may be indicated to reduce a cecum that is wedged within the right ventral colon because of adhesion and edema. Performing a right ventral colotomy increases the risk of abdominal contamination and subsequent peritonitis. Before colotomy, pelvic flexure enterotomy should be performed to evacuate edema. Reduction from within the right ventral colon can then be attempted; success depends on the condition of the bowel.

If reduction is not achievable from within the right ventral colon, partial typhlectomy through the ventral colotomy incision should be performed to attempt reduction with less edematous cecal tissue. If the cecum cannot be reduced or the remaining cecal stumps are necrotic and friable, complete cecal bypass is indicated. Complete cecal bypass has been documented for ileocolostomy or jejunocecostomy. Postoperatively, patients are treated on an individual basis as described for small intestinal intussusceptions.

**Prognosis**

The prognosis depends on the amount of cecum intussuscepted, the extent of necrotic bowel, the amount of contamination at surgery, and the experience of the surgeon. Generally, the survival rate of horses that undergo surgical correction of large intestinal intussusception is good (83%). Surgical reduction and partial typhlectomy was successful in four of five horses that underwent the procedure. If right ventral colotomy is performed in irreducible cases, the prognosis is also good. Previously, ileocolostomy was documented as having a high failure rate and was recommended only as a last resort. However, many successful outcomes have been published. Reports have suggested that correction of cecocolic intussusceptions is associated with a better prognosis than correction of cecocolic intussusceptions, but patients with either condition have a good prognosis for recovery if surgery is performed.

**Conclusion**

Clinical signs of intussusception vary greatly, making diagnosis challenging. Intussusception should be included in the differential diagnosis for any horse with acute or chronic colic. Surgical intervention is the treatment of choice. The prognosis for intussusception can be good if the condition is diagnosed and treated promptly.

**References**

1. Which of the following parasites is not a suggested cause of small or large intestinal intussusception?
   a. Parascaris equorum
   b. Oxyuris equi
   c. Strongylus vulgaris
   d. Anoplocephala perfoliata

2. Which of the following is typically required to definitively diagnose intussusception?
   a. abdominocentesis
   b. ultrasonography
   c. rectal palpation
   d. exploratory celiotomy

3. In intussusception, the ________ (the proximal segment of the intussusception) telescopes into the ________ (the distal segment).
   a. intussusception; intussusceptum
   b. intussuscipiens; intussusception
   c. intussusceptum; intussuscipiens
   d. intussuscipiens; intussusceptum

4. Which age group of horses most commonly develops small intestinal intussusceptions?
   a. younger than 6 months
   b. 6 months to 3 years of age
   c. 5 to 10 years of age
   d. older than 10 years

5. Which of the following is not a common location for small intestinal intussusceptions?
   a. the pylorus-duodenum
   b. the jejunoojejunum
   c. the jejunileum
   d. the ileocecum

6. Which of the following is not a clinical syndrome associated with cecal intussusceptions?
   a. acute syndrome
   b. subacute syndrome
   c. chronic wasting syndrome
   d. chronic active syndrome

7. What is the best treatment option for ileocecal intussusception with no devitalized bowel?
   a. manual reduction by gentle traction and abdominal closure
   b. manual reduction by gentle traction followed by incomplete bypass (ileocecostomy) without resection
   c. complete resection and anastomosis (jejunocecostomy)
   d. no intraoperative reduction, followed by medical treatment

8. The prognosis for uncomplicated large intestinal intussusceptions is
   a. excellent
   b. good
   c. poor
   d. grave

9. Which of the following is/are a prognostic factor(s) for small intestinal intussusception?
   a. the length of bowel affected
   b. the extent of bowel necrosis
   c. the preoperative health status of the horse
   d. all of the above

10. Which of the following is not a common preoperative or postoperative sequela of intussusceptions?
    a. adhesion formation
    b. peritonitis
    c. ileus
    d. pollakiuria