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Occurrence of jejunojejunal and ileocecal intussusceptions in a thoroughbred stallion

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PEER REVIEW

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Comments

This is a good study in which the authors evaluated JII in a German thoroughbred gelding horse and provided treatment and prevention for future studies. Clinicopathologic findings of this report in Iran was for the first time in this breed horse. And the results are very interesting. Details on Page 494

ABSTRACT

A 4-year-old, 600.4 kg stallion horse of German thoroughbred breed was presented at the Tehran University Veterinary Medicine Teaching Hospital, Iran, with a history of weakness of 2–3 days duration. The stallion died of unknown cause on the previous day. Abnormal findings during physical examination included an estimated dehydration of 3% to 5%, dark red oral mucous membranes, elevated heart and respiratory rates, stiffened feces, and absence of intestinal motility on auscultation. Postmortem examination indicated the presence of a jejunojejunal and ileocecal intussusceptions, 46 cm in length; a torsion of jejunum proximal to the intussusceptions was also noted. An ulcerative lesion, 6 cm in diameter, was found on the mucosa of duodenum. Large amounts of sand, coat hair, and hay mantling in bloody discharge were observed in a colonic impaction. It was an acute case that was evident by the involvement of intussusception and ulceration, indicating desquamation of the necrotic epithelial cells with edema and distinct infiltration of neutrophils. Histopathological examination confirmed that the ileum was the intussiscipiens to the jejunum that subsequently passed through into the caecum, and the mesentery probably tore as a result of the intussusception.

KEYWORDS Intussusceptions, Histopathological, Jejunojejunal, Ileocecal, Horse

1. Introduction

Intussusceptions are believed to result from abnormal motility patterns of various causes, such as sudden dietary changes, intestinal neoplasia, obstruction secondary to foreign bodies, previous jejunal resection and anastomosis, parasitism, maldigestion secondary to gastroduodenal ulcer disease, and enteritis^[1]. The cause of an intussusception is often unknown, but any condition that alters intestinal motility has been implicated^[2,3]. It is suggested that intussusception can represent a diagnostic challenge in horse medicine. History, clinical and clinicopathologic examinations are important in the diagnosis of intussusception^[3,4]. Most intussusceptions that develop in horses are jejuno–jejunal, ileal–ileal, or ileocecal. A few cases of jejunojejunal and ileocecal intussusception (JII) have been reported in horses^[5,6]. However, this case report described a special case of jejunojejunal and ileocecal intussusceptions with cause idiopathic in an thoroughbred horse.

2. Case report

2.1. Case history

In February 2013, a four-year-old German thoroughbred

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gelding, with a history of anorexia, ecchymotic hemorrhage of mucous membranes, fever, lethargy, reluctance to move, colic and a lack of defaecation was submitted to the clinic of the Faculty of Veterinary Medicine, University of Tehran. After two days without treatment, values for rectal temperature, pulse and respiratory rate were 40.8 °C, 88 beats/min and 30 breaths/min, respectively. The horse showed episodes of mild to severe abdominal pain. Auscultation with percussion over the right flank was negative and abdominal distention was not evident. Rectal examination revealed the presence of a firm palpable abdominal mass on the left of the median line and cranial to the pelvis. There was no fecal material in rectum. Distended loops of small intestine were palpable on rectal examination.

2.2. Clinical pathology

The horse was slightly dehydrated. Hematocrit value, total serum protein and fibrinogen concentrations were 36% (reference range 24%–46%), 7 g/dL (reference range 6.7–7.5 g/dL), and 600 mg/dL (reference range 100–600 mg/dL), respectively. Clinical pathology revealed that there was a mild hyperglycemia [5.1645 mmol/L (reference range 2.4975–4.1625 mmol/L)], hypocalcaemia [1.675 mmol/L (reference range 2.425–3.1 mmol/L)] and a slight hypochloraemia [93 mmol/L (reference range 97–111 mmol/L)]. Serum urea, creatinine, potassium and sodium concentrations were all within the normal range

After an initial positive response to therapy with benzylpenicillin, flunixin and dexamethasone, the horse condition deteriorated suddenly on day 3 of hospitalization, with signs of severe endotoxaemia and small-intestine obstruction. The horse was euthanized because of its poor prognosis and because of economic considerations.

2.3. Macroscopic pathology

After the opening of the abdominal cavity, dilated jejunal, ileal and cecal loops and hemorrhagic spots on them were apparent (Figure 1). In small intestines, severe hyperemia of mesentery veins with ecchymosis and petechiae, intussusception of small intestine in two points (jejunojejunal and ileocecal intussusceptions), partial fibrin organization at intussusception sites and thickening of intussusception site may induce severe colic which seemed to produce at least one week. Serosanguinous fluid in intestines, pulmonary hyperemia and edema were indicative for shock which may resulted from neurogenic shock, and concurrently septic shock may have occurred following intestinal lesions and slightly cardiac hypertrophy. Petechiae and ecchymosis hemorrhages were observed on mesentery and gastric serosa accompanying gastric distention. In addition, pericardial cavity included hydropericardium about 70 mL, and severe emphysema even in bullae shape were also detected in cranioventral regions of lung. The content in veins and cardiac chambers was more as chicken fat clot which some parts of it was adhered to valves and chordae tendineae in thrombus form (Figure 1).

Samples for bacteriological examination were collected proximal and distal to the lesion and in the lumen of the intussusceptum. The samples were processed to isolate Enterobacteriaceae; the cultures revealed the presence of *Escherichia coli*.

2.4. Microscopic pathology

Histological examination of the invaginated jejunum, ileum and cecum revealed signs of transmural necrosis and large hemorrhagic areas. Intense hyperemia, acute catarrhal enteritis and severe necrosis affected the portion of small intestine preceding the intussusception and diffuse necrotic fibrino hemorrhagic and purulent enteritis (Figure 1).

In the intestinal myenteric plexi, there were swollen neurons with a central clear cytoplasmic halo, peripheral nuclear displacement, and nuclear degeneration (central chromatolysis), surrounded by glial satellite cells with hyperchromatic chromatin and small amounts of mildly basophilic cytoplasm (satellitosis/activated satellite cells). Shrunken neuronal cells with hypereosinophilic cytoplasm and non-distinguishable nuclear borders (neuronal necrosis) were also visible, and were surrounded by glial cells (neuronophagia). Moderate numbers of lymphocytes and

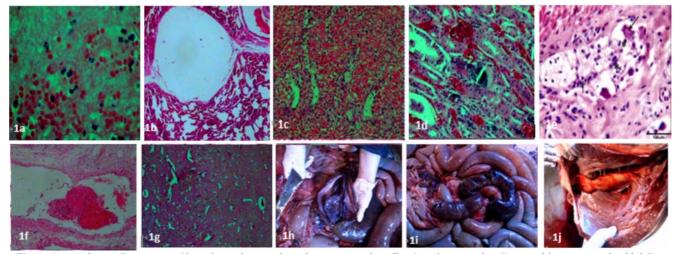


Figure 1. 1a and 1c: Diffuse necrotic fibrino hemorrhagic and purulent enteritis, 1b: Bullae & emphysema, 1d: Diffuse renal hyperemia and mild diffuse calcification, 1e: Intestinal Auerback plexus involved in degenerative and inflammatory changes of central neuronal chromatolysis with peripheral displacement of nuclei (arrow), satellitosis by glial cells (*) and a moderate number of infiltrating lymphocytes (†). Hematoxylin and eosin, bar=100 µm, 1f: cerebellar meningeal hyperemia and edema, 1g: Cerebral perivascular edema, 1h: partial fibrin organization at intussusception sites and thickening of intussusception site, 1i: intussusception of small intestine, 1j: The content in cardiac chambers was more as chicken fat clot which some parts of it was adhered to valves.

plasma cells were also detected. In addition, in the central nervous system, cerebellar meningial hyperemia and edema, microhemorrhage in cerebellum, cerebral diffuse hyperemia and cerebral perivascular edema were observed.

The liver contour was enlarged with rounded lobe edges; parenchymatous degeneration of hepatocytes was noted in the liver with focal mononuclear cell infiltration.

The kidney histology lesions such as diffuse renal hyperemia, mild acute tubular necrosis, mild diffuse calcification and focal lymphocytic interstitial nephritis were observed.

The lung sub lobar, interlobar edema and bullous emphysema were noted. The mesenteric lymph nodes was perceived subcortical and paracortical hemorrhage associated with lymphadenitis.

3. Discussion

Different types of intussusceptions are recognized in horse such as enteric, ileoileal, ileocecocolic, cecocolic and colocolic. Usually, the ileum is involved at or close to the ileocaecal junction^[7,8]. Jejuno–jejunal intussusception is less frequently encountered, hence its incidence is very low (2.26%) in horses^[7]. Clinical signs are extremely variable depending on the degree of compromise or obstruction of the affected portion of the gut. Pain may range from intermittent mild to severe. Pain may be so uncontrollable that immediate surgical intervention is required^[7,9–11].

The horse showed slight signs of colic, anorexia, lack of defaecation and lethargy but no abdominal distention. In the diagnosis of small intestinal obstruction, rectal palpation is considered valuable. Although intussusception is palpable in only a minority of affected adult horses (23%), distended loops of small intestine are palpable per rectum in 50% of cases with intussusception^[5]. In this case, while the intussusception was diagnosed on the rectal examination, neither rectal palpation examination of the right flank revealed distended loops of small intestine.

In present case, the horse had mild to severe respiratory compromise, tachycardia, mild cyanosis likely an increased intra–abdominal pressure, and a mild hyperglycemia, hypocalcaemia and a slight hypochloraemia. Hematological and blood biochemical data may be normal, as was shown by Vieitez *et al.* in horse with jejuno–jejunal intussusceptions^[12]. On the other hand, clear hematological and biochemical abnormalities are commonly seen. The findings of the current case are similar to those described by Albanese *et al.*^[13] and Lin *et al.*^[14].

Intussusceptions are believed to arise either as a result of segmental motility disorder or local changes in the intestinal wall^[15]. Forthermore, in a special case, jejuno–jejunal intussusception was reported as a complication of jejuno–jejunal anastomosis by researchers^[16,17].

In the reported case, a jejunoileo–caecal intussusception was released by midline necropsy. Intussusception is an invagination of one segment of intestine and its mesentery into the lumen of an adjacent segment^[18]. The invaginated segment is the intussusceptum while the enveloping segment is the intussuscipiens^[19]. Predisposing factors that may lead to segmental motility differences are enteritis, mesenteric arteritis, severe ascarid impaction, tapeworm infection (*Anoplocephala perfoliata*) and sudden dietary changes^[20].

In animals and in human beings[3,21,22], intestinal

intussusception may be related to severe mesenteric lymph node enlargement caused by mechanical stress^[22]. In the present case, lesions consistent with lymphadenomegaly or mesenteric masses were not identified and this excluded any correlation with both intussusceptions.

Moreover, bacteriology identified non adhesive *Escherchia coli*, were not conclusive in demonstrating the role of a single bacterium in the pathogenesis of both intussusceptions.

In the current case, microscopic features of the lesion from jejunoileo–caecal ulceration (the presence of necrotic epithelial cells with distinct infiltration of neutrophils) suggest that the lesion could have been a secondary cause. The cause of the ulcerative lesion was most likely the presumed stress from poor nursing and maybe associated with administration of flunixin meglumine. A study was conducted to determine the negative effect of a high dose of flunixin meglumine (6.6 mg/kg·d⁻¹) on the gastrointestinal tract of horses[²²].

Based on history, clinical examination, necropsy and pathological findings, the cause of intussusception couldn't be determined in this case. Results suggested that the weakness in this thoroughbred horse could have originated from inadequate farm management. The progression of this case was rapid, and the horse died because of its poor general condition associated with colic syndrome. This case highlights the importance of astute clinical diagnosis for potential risks in thoroughbred horse and the need for prompt surgical intervention.

In conclusion, the ileum was the intussiscipiens to the jejunum that subsequently passed through into the caecum, and the mesentery probably tore was as a result of the intussusception.

Conflict of interest statement

We declare that we have no conflict of interest.

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Comments

Background

Although intussusception is a relatively uncommon cause of colic in adult horses, it must be considered in the differential diagnosis of horses with acute of 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 intussusception, and the distal, receiving portion is the intussusception, As the intussusceptum invaginates into the distal bowel, the mesenteric blood flow becomes occluded. Intussusception can occur in multiple locations throughout the alimentary tract. Small intestinal intussusception (SII) can affect the jejunojejunum, jejunoileum, ileoileum, or ileocecum. Large intestinal intussusception can affect the cecocecum, cecocolon, colon, or colorectum. SII is an uncommon but well documented cause of colic and abdominal pain in horses. Diagnosis of SII is complicated by the variability in its clinical presentation; affected horses may be presented for treatment of acute, severe colic or recurrent signs of mild to moderate abdominal pain which persist in spite of conservative treatment for several days. Etiological factors which lead to the motility perturbations responsible for SII have not been defined for horses.

Research frontiers

Intussusceptions are believed to result from abnormal motility patterns of various causes, such as sudden dietary changes, intestinal neoplasia, *etc.* The cause of an intussusception is often unknown, but any condition that alters intestinal motility has been implicated.

Related reports

The study nearly agreed with Vieitez *et al.*, who reported the jejunojejunal intussusception following umbilical herniorrhaphy in a foal, and Lin *et al.*, who reported the jejunojejunal intussusception and colonic impaction in a 12–day–old orphan foal. Forthermore, Gaughan and Hackett reported chronic ileocecal intussusception in horses. But this research focuses more on JII that is rare in horses in these areas than that of in other intussusceptions.

Innovations & breakthroughs

The study emphasized on intussusception incidence, diagnosis, and considering thoroughbred horse breed. Due to JII rarity and severe spread to all parts of the gastrointestinal tract, it's study is the most important in horse breed because reports of JII are limited in the veterinary literature. There is conflicting information regarding gender predisposition. In this report, horse with JII was male. Based on reporting of Iran, we can get an effective step for treatment and prevention for future studies to provide.

Applications

It is significant to know the distribution of JII in horses. The results of the present study suggest that the bowel telescopes were diagnosed as intussusceptions in the light of clinical and pathomorphological findings. It was considered as substantial by means of being the first case reported in a thoroughbred horse.

Peer review

This is a good study in which the authors evaluated JII in a German thoroughbred gelding horse and provided treatment and prevention for future studies. Clinicopathologic findings of this report in Iran was for the first time in this breed horse. And the results are very interesting. This paper was recommended because it was considered as substantial by means of being the first case reported in a thoroughbred stallion.

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