

Case Report

Congenital intestinal polyp associated with intussusception in a 3-day-old foal

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Introduction

Hyperplastic or adenomatous polyps have been previously described in both human medicine and domestic species (Crawford 1999). This condition has been most frequently reported in cats (Orr *et al.* 1980) and also in dogs and sheep (Barker *et al.* 1993). To date, only one report of an adenomatous polyp in the equine species exists (Patterson-Kane *et al.* 2000). To the authors' knowledge, an intussusception secondary to a hyperplastic polyp has not been reported previously in an equine neonate. **The case reported here describes the finding of a congenital polyp *post mortem* in a neonatal foal and the history leading to euthanasia.**

Case details

History

A 9-hour-old Trakehner-cross filly (40 kg) was admitted to the Mid-Atlantic Equine Medical Center with the primary complaint of mild colic, poor suck reflex and generalised weakness. Gestational length had been normal and delivery uneventful. Examination of the placenta revealed no abnormalities. An unknown quantity of colostrum was administered via nasogastric intubation. Passage of meconium was observed. The referring veterinarian was called to re-evaluate the foal 3–4 h later because of abdominal discomfort, and administered dipyrone (50% solution 10–20 mg/kg bwt i.m.) prior to shipping to the referral centre.

Physical examination

The foal was lethargic, with a slightly obtunded mentation and a weak suck reflex. Mucous membranes were pink with mild injection and capillary refill time was delayed at 2–3 secs.

Rectal temperature (37.2°C; reference range [rr] 37.2–38.8°C), heart rate (84 beats/min; rr 80–120 beats/min) and respiratory rate (24 breaths/min; rr 20–40 breaths/min) were within normal reference values. Auscultation of the heart and lungs revealed no abnormalities. Peripheral pulses were weak and extremities were cool to touch. Gastrointestinal borborygmi were present in all quadrants and no abdominal distention was noted. The umbilicus and all joints palpated normally. The foal exhibited bilateral flexor tendon contracture in the forelimbs. She was able to stand without support, but required assistance to lie down.

Clinical pathology

Results of haematological and serum biochemical analyses revealed elevated packed cell volume (47%; rr 32–42%) and creatinine (43 mg/l; rr 6–22 mg/l), compatible with hypovolaemia and/or placental compromise. Arterial blood pressure and arterial blood gas values were within normal reference values.

Ultrasonography

Abdominal ultrasonography revealed a distended, fluid-filled stomach. Moderately distended fluid-filled immotile loops of small intestine were imaged in the caudal abdomen. An indwelling nasogastric tube was placed and approximately 600 ml gastric reflux removed.

Treatment

Initial treatment consisted of potassium penicillin (22,000 u/kg bwt i.v. q. 6 h) and amikacin (25 mg/kg bwt i.v. q. 24 h). A total of 2.5 l isotonic fluid with 5% dextrose (100 ml/l) were administered at interval dosing over an 8 h period. This was followed by continuous rate infusion of an isotonic polyionic solution at a rate of 150 ml/kg bwt/day (1.5 x maintenance rate). Assessment of physical examination parameters and reflux of gastric contents were performed every 2 h. Over the ensuing 10 h period, the foal refluxed a total of 3 l. With the

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exception of a mild intermittent bruxism, she exhibited no signs of colic and continued to pass meconium.

Differential diagnosis

A presumptive clinical diagnosis of perinatal asphyxia syndrome (PAS) with secondary functional obstruction of the stomach and small intestinal tract was made. Although parturition and the placenta itself were reported to be normal, premature placental separation, placental insufficiency, placental dysfunction or reduced umbilical blood flow may lead to perinatal asphyxia that ultimately impacts upon function of the gastrointestinal tract and urinary system (Slovis 2003; Wilkins 2004). The gastrointestinal effects of PAS may be manifested as ileus, recurrent excessive reflux and gas distention (Slovis 2003; Wilkins 2004). Other differential diagnoses included congenital malformation of the gastrointestinal tract, partial blockage of the gastrointestinal tract due to an intraluminal or nonstrangulating extraluminal mass, and intermittent intussusception.

Further treatment

During the first 24 h of hospitalisation, approximately 300–500 ml gastric reflux were obtained every 2–4 h. The foal's overall mentation also improved during this period. Gastroscopic examination was performed and mucosal erosions in the distal oesophagus and adjacent to the oesophageal-gastric sphincture were noted. Following removal of approximately 2 l gastric fluid, a hyperaemic and diffusely ulcerated squamous nonglandular mucosa was visualised. Ranitidine (6.6 mg/kg bwt i.v. q. 4 h) was subsequently added to the treatment protocol. Supportive care continued and additional medical therapy included administration of 1 litre i.v. plasma to correct the hypogammaglobulinaemia (4 g/l; rr >8 g/l). Total parenteral nutrition and intranasal oxygen supplementation at 5 l/min were also commenced.

At 48 h post presentation the foal began to salivate excessively. The bruxism and abdominal discomfort also worsened. Surgical exploration was discussed at this point, but declined by the owner. Lidocaine (2% solution, 1.3 mg/kg bwt i.v. loading dose, 0.05 mg/kg bwt/min constant rate infusion) was administered in an attempt to promote intestinal motility and provide analgesia. On the third morning of hospitalisation, the foal appeared dull and lethargic. She was also consistently painful despite the addition of butorphanol (0.01–0.04 mg/kg bwt i.v. as necessary q. 1–4 h) to the treatment protocol.

Repeated ultrasonography

Repeat abdominal ultrasonographic examination revealed more diffusely distended, fluid- and gas-filled, immotile loops of small intestine and a large, distended, fluid-filled stomach. A target-like lesion, sometimes seen via ultrasound in cases of intestinal intussusception, was not observed (Bernard *et al.* 1989; Fontaine-Rodgers and Rodgers 2001). Given the lack of response to medical therapy, surgical intervention was again discussed with the owners who instead elected to subject the foal to euthanasia.

Necropsy

Post mortem examination revealed a 6 cm intussusception of the proximal jejunum, approximately 60 cm from the pylorus. The wall of the jejunum appeared moderately oedematous. Upon examining the mucosal surface of the jejunum, a dark red, pedunculated filamentous mass (5 x 15 x 5 mm) was identified on the mucosa at the leading edge of the intussusceptum (**Fig 1**). The polyp occupied approximately 60% of the lumen of the jejunum. A small amount of grossly normal peritoneal fluid was present in the abdominal cavity. Erosions of the distal oesophagus, as well as the gastric squamous mucosa near the oesophogastric sphincter, were noted. No other gross abnormalities were observed.

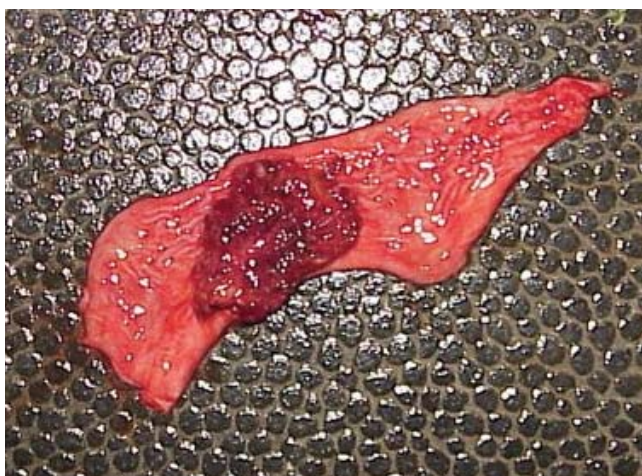


Fig 1: Photograph of the hyperplastic polyp found in a 3-day-old Trakehner-cross foal; a dark red, pedunculated filamentous mass (5 x 15 x 5 mm) was identified on the mucosa at the leading edge of the intussusceptum.

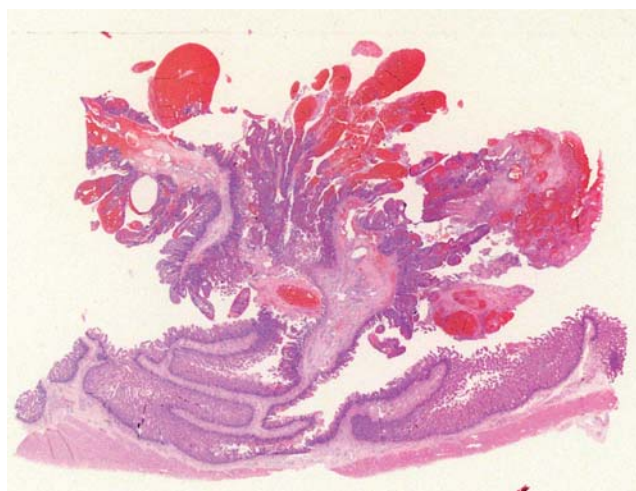


Fig 2: Histological slide of the hyperplastic polyp obtained from the jejunum at the leading edge of the intussusceptum in a 3-day-old Trakehner-cross foal.

Histopathology

Histologically, the mass consisted of narrow-based polyp with papillary projections of highly vascularised loose connective tissue and a thin layer of smooth muscle continuous with the submucosa and muscularis mucosae of the adjacent jejunum (Fig 2). The papillary projections were lined by hyperplastic mucosa consisting of hypercellular *lamina propria* and highly irregular villi of variable length and width. The villi were lined by a single layer of epithelial cells that varied from cuboidal to columnar. Intestinal crypts were also variable in size and irregularly spaced within the hypercellular *propria* mucosae. There were numerous erosions of the mucosa of the polyp with large fibrinocellular clots adhered to the surface. Both the mucosa and submucosa were moderately oedematous and haemorrhagic. The final histopathological diagnosis was hyperplastic polyp of the jejunal mucosa.

Discussion

In man, polyps have been classified as either hyperplastic or adenomatous. The former are composed of well-formed glands and crypts lined by non-neoplastic epithelial cells (Crawford 1999). Hyperplastic polyps are thought to develop as a result of abnormalities in the mucosal development and structure, or as a result of inflammation (Crawford 1999). Hyperplastic polyps may become malignant, but most have little potential to do so (Crawford 1999) and are usually considered benign. Alternatively, adenomatous polyps (polypoid adenomas) arise from proliferation and dysplasia of epithelial cells and may transform into carcinomas or adenocarcinomas (Crawford 1999). Differentiating between the 2 types of polyp can be difficult, with the single distinguishing criterion being the presence or absence of epithelial dysplasia (Crawford 1999). In man, adenomatous polyps most frequently occur in the colon (Barker *et al.* 1993; Jones 1997; Crawford 1999). In the small intestine, the duodenum is most commonly and the ileum least commonly affected (Barker *et al.* 1993; Jones 1997; Crawford 1999). Adenomatous polyps may occur spontaneously or as a component of an inherited syndrome (Barker *et al.* 1993; Jones 1997; Crawford 1999).

In domestic animals a clear distinction between hyperplastic and adenomatous polyps has not been well defined (Barker *et al.* 1993; Jones 1997; Crawford 1999; Patterson-Kane *et al.* 2000). This may be because of a lack of data examining the biological behaviour of polyps in domestic species. Adenomatous polyps are most commonly reported in cats (MacDonald *et al.* 1993), with Asian feline breeds exhibiting a genetic syndrome similar to that described in man (Orr *et al.* 1980; MacDonald *et al.* 1993). Adenomatous polyps have also been reported in dogs, cattle and sheep (Barker *et al.* 1993). There has been one report of adenomatous polyposis in a mature horse with a history of weight loss (Patterson-Kane *et al.* 2000).

Polyps have also been classified based upon the age of the individual affected in man. For example, congenital polyps

may be recognised in neonates as early as 2 days of age (Lee *et al.* 1970). These tumours tend to be benign, inflammatory or hyperplastic polyps. Juvenile polyps possessing normal epithelium and hypertrophic *lamina propria* (Jacoby *et al.* 1997) occur in children aged 2–10 years (Ko *et al.* 1995). Other types of polyp reported include adenomatous, infantile and inflammatory polyps in children aged 6 months to 19 years (Ko *et al.* 1995; Pallai and Tolia 1998). Congenital and infantile polyps are seen at early ages. The common presenting complaint with these polyps is rectal bleeding (Lee *et al.* 1970; Ein 1976; Ong and Beasley 1990; Chen *et al.* 1998), although intestinal blockage may also occur. Several reports in human neonates attribute intussusception to congenital polyps (Lee *et al.* 1970; Ein 1976; Ong and Beasley 1990; Chen *et al.* 1998). The polyps are thought to alter intestinal motility and lead to intussusception (Lee *et al.* 1970; Ein 1976; Ong and Beasley 1990; Chen *et al.* 1998).

Intussusceptions have also been reported to occur frequently in foals and young horses and occur when a segment of intestine invaginates (intussusceptum) into the adjacent segment of intestine (intussusciens; usually a distal portion) (Barker *et al.* 1993; Jones 1997; Chen *et al.* 1998; Crawford 1999). The aetiology is uncertain, but has been attributed to changes in peristalsis (hypo- or hyper-) and/or to the existence of a small mass, foreign body or parasite at the leading edge of the intussusception (Barker *et al.* 1993; Jones 1997; Crawford 1999; Blikslager and Jones 2004). This is the first time an intussusception secondary to a hyperplastic polyp has been described in an neonatal foal.

In summary, congenital anomalies such as intraluminal intestinal polyps should be considered as a contributing cause of colic in equine neonates exhibiting progressive signs of colic, when other more common causes have been excluded (Slovic 2003; Wilkins 2004).

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References

- Barker, I.K., Vandreamel, A.A. and Palmer, N. (1993) The alimentary system. In: *Pathology of Domestic Animals*, Vol. 2, 4th edn., Eds: K.V.F. Jubb, P.C. Kennedy and N. Palmer, Academic Press, San Diego. pp 130-140.
- Bernard, W.V., Reef, V.B., Reimer, J.M., Humber, K.A. and Orsini, J.A. (1989) Ultrasonographic diagnosis of small-intestinal intussusception in three foals. *J. Am. vet. med. Ass.* **194**, 395-397.
- Blikslager, A.T. and Jones, S.L. (2004) Ischemic disorders of the intestinal tract. In: *Equine Internal Medicine*, 2nd edn., Eds: S.M. Reed, W.M. Bayly and D.C. Sellon, W.B. Saunders Co., Philadelphia. pp 913-921.
- Chen, J.Y., Lin, R.I., Chao, H.C. and Kong, M.S. (1998) Neonatal ileoileocolic intussusception associated with ileal polyp report of one case. *Zhonghua Minguo xiao er ke yi xue hui za zhi* **39**, 203-205.
- Crawford, J.M. (1999) The gastrointestinal tract. In: *Pathologic Basis of Disease*, 6th edn., Eds: R.S. Conran, V. Kumar and T. Collins, W.B. Saunders Co., Philadelphia. pp 826-843.

- Ein, S.H. (1976) Leading points of childhood intussusception. *J. Pediatr. Surg.* **11**, 209-211.
- Fontaine-Rodgers, G. and Rodgers, D.H. (2001) Diagnosis of small intestinal intussusception by transabdominal ultrasonography in 2 adult horses. *Can. vet. J.* **42**, 378-380.
- Jacoby, R.F., Schlack, S., Gurbax, S. and Laxova, R. (1997) Del(10)(q22.3q24.1) associated with juvenile polyposis. *Am. J. med. Genet.* **70**, 361-364.
- Jones, T.C. (1997) The digestive system. In: *Veterinary Pathology*, 6th edn., Eds: T.C. Jones, R.D. Hunt and N.W. King, William and Wilkins, Baltimore. pp 106-1080.
- Ko, F.Y., Wu, T.C. and Hwang, B. (1995) Intestinal polyps in children and adolescents - a review of 103 cases. *Zhonghua Minguo xiao er ke yi xue hui za zhi* **36**, 197-202.
- Lee, H.S., Kwon, H.C., Pyung, K.K. and Yun, D.J. (1970) A neonatal intussusception induced by a congenital ileal polyp in a two day old male new born. *Yonsei med. J.* **11**, 54-59.
- MacDonald, J.M., Mullen, H.S. and Moroff, S.D. (1993) Adenomatous polyps of the duodenum in cats: 18 cases (1985-1990). *J. Am. vet. med.* **202**, 647-651.
- Ong, N.T. and Beasley, S.W. (1990) The lead point in intussusception. *J. Pediatr. Surg.* **25**, 640-643.
- Orr, C.M., Gruffydd-Jones, T.J. and Kelly, D.F. (1980) Ileal polyps in Siamese cats. *J. small anim. Pract.* **21**, 669-674.
- Pallai, R.B. and Tolia, V. (1998) Colonic polyps in children: frequently multiple and recurrent. *Clin. Pedi.* **37**, 253-257.
- Patterson-Kane, J.C., Sanchez, L.C. and MacKay, R.J. (2000) Small intestinal adenomatous polyposis resulting in protein losing enteropathy in a horse. *Vet. Pathol.* **37**, 82-85.
- Slovis, N.M. (2003) Gastrointestinal failure. *Clin. Tech. equine Pract.* **2**, 79-86.
- Wilkins, P. (2004) Disorders of foals. In: *Equine Internal Medicine*, 2nd edn., Eds: S.M. Reed, W.M. Bayly and D.C. Sellon, W.B. Saunders Co., Philadelphia. pp 1390-1397.