Case Report

Atresia coli in a foal: Diagnosis made with colonoscopy aided by N-butylscopolammonium bromide

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Summary

Atresia coli, a rare congenital defect, was diagnosed in a foal via colonoscopy after N-butylscopolammonium bromide was used to aid visualisation of the intestinal defect. Colonoscopy is a cost effective tool for diagnosing atresia of the terminal colon. N-butylscopolammonium bromide aided colonoscopy has not been previously reported in horses.

Introduction

Intestinal atresia has been reported in numerous species including man, cattle, sheep, pigs, dogs, cats and foals (van der Gaag and Tibboel 1980; Johnson 1986a). In foals, the defect is considered rare and is most commonly reported in the large and small colon. Intestinal atresia can be categorised as type 1, 2 or 3 and there is some argument over the pathogenesis of the problem. Invariably intestinal atresia carries a poor prognosis (Young et al. 1992). Attempts at surgical correction often fail. In an industry where one foal may have significant financial value, accurate diagnosis of this rare defect ante mortem can be very important and assessing the feasibility of surgical correction prior to incurring the cost of surgery is sound economic practice.

Case details

History

A 30 h old Thoroughbred colt was referred to Mid-Atlantic Equine Medical Center for evaluation and treatment of colic. The foal had appeared normal and had nursed shortly after birth. The barn manager reported seeing passage of a small amount of meconium. Six hours after birth, the foal began to show signs of mild abdominal discomfort and was nursing less vigorously. In the 24 h following the initial colic signs, the foal passed no manure. He stopped nursing. Colic signs progressed and his abdomen became distended. Two soapy water enemas were nonproductive. Treatment on the farm with oral laxatives, analgesics, i.v. fluids and plasma failed to alleviate the colic.

Clinical findings and initial treatment

On presentation, the foal had a normal temperature of 37.8°C. An elevated pulse rate was present (150 beats/min) and its oral mucous membranes were hyperaemic. The respiratory rate was normal (36 breaths/min), but inhalation was laboured due to the presence of significant bilateral abdominal distension. Gastrointestinal sounds were present. Digital rectal examination revealed a complete absence of faeces and the perineum was clean. Treatment, consisting of i.v. polyionic fluids with 5% dextrose (Normosol-M) and a warm water enema, was begun with a presumptive diagnosis of a meconium impaction. The enema was completely unproductive and the enema tube seemed to meet a blockage several cm oral to the anus. Treatment was continued with the assumption of a high meconium impaction, but differential diagnoses such as strangulation of the small colon, or atresia coli began to be considered. Further diagnostics were pursued to assess the patency of the gastrointestinal tract. In addition to the colic related abnormalities, the colt also had significant contracture of the coffin (distal interphalangeal) and pastern (proximal interphalangeal) joints of both hindlimbs.

Further diagnostics and treatment

Abdominal ultrasound revealed normal umbilical structures and a gas distended large colon and caecum. A 9.8 mm diameter GIF 130-3M videoscope was inserted into the foal’s rectum. On initial colonoscopy, the mucosal

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wall of the small colon appeared healthy and pink, but a severe narrowing of the lumen 20 cm oral to the anus could be seen. Despite copious air insufflation into the rectum, normal peristalsis obscured visualisation enough that it could not be determined if the bowel was completely obstructed (Fig 1). Abdominal radiographs were taken following a barium enema. Severe stenosis of the small colon was apparent on the contrast study (Fig 2), however, complete obstruction of the colon lumen could not be verified. The owner wished to pursue further diagnostics and treatment despite the abnormal findings on radiographs and colonoscopy. With a history of meconium passage having been seen on the farm, the owner wished to continue treating the colt until a definitive diagnosis could be reached.

A blood sample was taken for serum biochemistry analysis and a complete blood count (CBC). The CBC was normal while the serum biochemistry showed a mild azotaemia with blood urea nitrogen at 330 mg/l (reference range [rr] = 100–250 mg/l) and creatinine at 28 mg/l (rr = 10–22 mg/l), and an elevated creatine kinase at 962 u/l (rr = 120–470 u/l). These elevations were considered within acceptable limits for a newborn foal. Blood gas analysis of an arterial blood sample showed normal values for blood pH, CO2 and O2 partial pressures, and electrolytes.

A nasogastric tube was passed and yellow curdled reflux was obtained. A 16 gauge, 20 cm, over the wire catheter was placed into the foal’s left jugular vein. A muzzle was placed to prevent nursing and the foal was monitored throughout the night. One litre of polyionic fluid with 5% dextrose (Normosol-M)1 was given i.v. every 6 h to maintain hydration. When abdominal distension reached a critical stage, the gas distended caecum was trocharised with a 14 gauge, 5 cm catheter and the hind gut was decompressed. Once deflated, the colt slept in comfort for about 3 h. By the following morning, abdominal distension had recurred and respiration was laboured.

Twelve hours after admission the colt was sedated with xylazine (TranquiVed)3 i.v. at a dose of 1.1 mg/kg bwt and laid in lateral recumbency. N-butylscopolammonium bromide (Buscopan)4 was administered at a dosage of 0.3 mg/kg bwt, and colonoscopy repeated. With peristalsis ablated by the N-butylscopolammonium bromide, the colonoscope clearly visualised an area 20 cm oral to the anus where the small colon lumen narrowed to approximately 2 mm (Fig 3a). A stylet was passed through the 2.8 mm channel in the videoscope2 into the stenosis and met a dead end within 5 mm (Fig 3b). The mucosal wall of the small colon still appeared pink and healthy 12 h after initial colonoscopy. This, in combination with the presence of normal peristalsis on initial colonoscopy, suggested that a strangulating lesion of the small colon was unlikely. A diagnosis of atresia coli was made. Due to the poor prognosis associated with surgical correction, the owner opted to have the colt humanely subjected to euthanasia.

**Gross pathology**

On necropsy, a normally developed, gas distended caecum and large colon were found with the pelvic flexure retroflexed ventrally. The transverse colon was grossly normal, but the descending colon ended in a distended blind ending sac that was connected to an
abnormally narrow terminal colon by a 2–3 mm cord of fibrous tissue (Figs 4a,b). The wall of the distended descending colon was thin and lacking teniae. No lumen was apparent within the fibrous tissue cord. All other aspects of the gastrointestinal tract were normally formed and the mesentery was normally attached to all intestines, including the atretic segment.

Discussion

Intestinal atresia is an unusual congenital defect in any species, but particularly rare in foals. In this case, the history of meconium having been seen shortly after birth was misleading. Although intestinal stenosis was obvious on all diagnostic imaging, the owner was unwilling to cease treatment if any possibility of a patent lumen existed. Thus, a completely accurate diagnosis of intestinal atresia prior to euthanasia was considered to be essential.

Intestinal atresiae can be categorised into one of 3 types (van der Gaag and Tibboel 1980). In type 1, a normal intestinal wall is present, but the intestinal lumen is completely occluded by a membranous septum. Two pieces of intestine end in blind ending sacs in type 2, but these segments are connected by a fibrous band that lacks a lumen. The mesentery may or may not be attached to the defective bowel. In type 3, oral and aboral segments of intestine end in blind ending sacs that are completely unconnected. The majority of reported cases of atresia coli in foals have been type 3 (Rakestraw and Hardy 2006); however, the connecting fibrous band in the present case would classify it as a type 2 atresia.

Fig 3: a) Colonoscopy immediately following N-butylscopolammonium bromide administration. b) Introduction of a stylet into the stenosis to feel the blind end of the atresia.

Fig 4: a) Post mortem visualisation of descending colon atresia (arrow). b) Atresia coli. Note the severely dilated segment of small colon proximal to the atresia.
Surgical repair of atresia coli has been attempted with limited success (Rakestraw and Hardy 2006). Approaching through a ventral midline celiotomy, large colon and transverse colon defects can be exteriorised and resected, along with the dilated area of the oral segment. An end-to-end or end-to-oblique anastomosis is preferred rather than a side-to-side anastomosis in an effort to avoid the creation of a blind-ending sac. The high rate of failure associated with surgical correction is due primarily to the disparity in diameter of the anastomosed ends. Surgical correction of the terminal small colon is further complicated by the fact that the bowel cannot be exteriorised to perform the necessary resection and anastomosis (Fischer 2006). Correction could be attempted using an end-to-end anastomosis gun within the abdomen but, in this case, the dilation of the oral segment would have made this technique challenging (Fig 4b).

The definitive pathogenesis for atresia coli has yet to be determined. Hereditary factors have been implicated. Occurrence of atresia ilei in Swedish Highland cattle appears consistent with autosomal recessive inheritance, as does atresia jejuna in Jersey cattle (Schneider et al. 1981). Children with the genetic disorder Down’s syndrome are reported to have an increased incidence of duodenal atresia (Johnson 1986b). Infectious and environmental causes for intestinal atresiae have little support. In one report, a foal with atresia coli was found to be concurrently infected with equine herpesvirus-1, but no connection could be found between the 2 problems (Anderson et al. 1987). In another report, 2 foals with atresia coli were born on neighbouring farms within 4 days of each other (Cho and Taylor 1986) but the timing and geographic proximity were considered to be coincidental.

A developmental arrest theory has been suggested as a possible aetiology. This theory suggests that the fetal intestine is patent on initial development and then plugs with mucus but recanalises by the end of the 8th week of development (Schneider et al. 1981; Johnson 1986b). Failure to recanalise the intestinal lumen was thought to result in intestinal atresia. This theory was disproved when evidence of bile, squamous epithelial cells and gross meconium was found distal to atretic sites (Schneider et al. 1981). As bile does not begin to flow from the bile duct until the 11th week of development, it would be impossible for bile to occur distal to an intestinal atresia if the intestinal lumen never recanalised by Week 8. The history of witnessed meconium passage in this case further supports these earlier reported findings. Intestinal ischaemia, either temporary or permanent, has also been suggested as a cause of intestinal atresia and this aetiology has significant experimental support (Schneider et al. 1981; Johnson 1986b). A study in chicken embryos by Tibboel et al. (1980) demonstrated that occlusion of a mesenteric vessel for >20 min resulted in atresia of the temporarily ischaemic intestine in >50% of experimental subjects.

In the past, atresia coli in foals has been most commonly diagnosed post mortem, intraoperatively or on contrast radiographs taken after a barium enema or nasogastric administration of barium. Colonoscopy provided a definitive diagnosis in a case reported by Young et al. (1992), in which the terminal colon was found to end in a blind ending sac 8 cm oral to the anus. However, in a report by Overbaugh (1983), colonoscopy was not useful in diagnosing a small colon atresia >30 cm oral to the anus. In this case, a significant stenosis could be appreciated 20 cm oral to the anus on colonoscopy, but visualisation was impaired by normal peristaltic movement and the atresia could not be definitively diagnosed. Surgical correction of the atresia carried a poor prognosis and exploratory laparotomy simply to diagnose the suspected atresia seemed unjustified economically. The foal was refluxing at the time of presentation, therefore oral administration of barium was not indicated and contrast radiographs from a barium enema were not completely conclusive. Ablation of peristalsis with N-butylscopolammonium bromide improved visualisation on colonoscopy enough to make a definitive diagnosis.

N-butylscopolammonium bromide is most commonly used in horses to facilitate rectal examination and treat spasmodic colic. The drug exerts an anticholinergic effect by competitively inhibiting muscarinic receptors (Renner et al. 2005). In addition to its gastrointestinal effects, N-butylscopolammonium bromide also causes tachycardia, pupil dilation and mucous membrane dryness similar to other parasymphatheticolytic drugs such as atropine. In a study by Luo et al. (2005), the smooth muscle relaxation in the gastrointestinal tract of horses was clearly demonstrated when a 68% decrease in rectal pressure was measured after i.v. administration of N-butylscopolammonium bromide at a dose of 0.3 mg/kg bwt.

Indications for colonoscopy in this species are limited and, to the authors’ knowledge, i.v. administration of N-butylscopolammonium bromide to facilitate colonoscopy has not been reported in this species. The drug has been used for this purpose in man, but increased pain post colonoscopy and risks associated with the tachycardia occurring after i.v. administration have limited its use (Mui et al. 2004). In a clinical study by Bertone (2002), the adverse haemodynamic effects of this drug after i.v. administration in horses were not considered significant enough to decrease its safety when used at the recommended dose of 0.3 mg/kg bwt. In this particular case, the N-butylscopolammonium bromide could also have been used to achieve better contrast radiographs had the repeat colonoscopy not been definitive.

Although the usefulness of colonoscopy as a diagnostic tool in horses is limited, this report demonstrates that, in foals, it can be a very effective method of diagnosing descending colon atresia. It is more cost effective and less time consuming than other routine methods of ante mortem diagnosis such as contrast radiographs, exploratory laparotomy or laparoscopy. It can be done in the field and requires minimal assistance. One person to
restrain the mildly sedated foal is sufficient. Colonoscopy can be performed with the foal standing or recumbent and the superior visualisation achieved with N-butylscopolammonium bromide can aid an immediate and accurate diagnosis. As surgical repair of atresia coli defects within the pelvic canal carry a poor prognosis, accurate diagnosis of these lesions prior to surgical exploration can save clients significant cost.

Manufacturers’ addresses
1Hospira Inc., Lake Forest, Illinois, USA.
2Olympus, Center Valley, Pennsylvania, USA.
3Vedco Inc, St Joseph, Missouri, USA.
4Boehringer Ingelheim Vetmedica Inc., St Joseph, Missouri, USA.

References