CASE REPORT

Recurrent rectal prolapse caused by colonic duplication in a dog

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A 9-month-old female Shar Pei cross-bred dog was presented with a history of recurrent rectal prolapse over 7 months. Repeated reduction and anal purse string sutures and subsequent incisional colopexy failed to prevent recurrent rectal prolapse. Digital rectal examination following reduction of the prolapse identified a faeces-filled sac within the ventral wall of the rectum and an orifice in the ventral colonic wall, cranial to the pubic brim. A ventral, communicating tubular colonic duplication was diagnosed by means of a barium enema. Surgical excision of the duplicated colonic tube was performed via a caudal ventral midline laparotomy. At 20 weeks post-operation, there has been no recurrence of rectal prolapse.

Key words: rectal prolapse, colonic duplication, dog

Colonic duplication is a rare congenital defect that has been reported in dogs, humans, horses and one cat. Within the veterinary literature, there have been only five previously reported cases of canine colonic duplication. In these five dogs, clinical signs included tenesmus, dyschezia and frequent defaecation. Rectal prolapse was documented in one previously reported case and that dog was euthanased intraoperatively.

The classification of colonic duplications has previously been defined. These classifications include type I: the duplication is limited to colon and rectum; and type II: the duplication is associated with other abnormalities such as urogenital duplications and vertebral column abnormalities. These types can then be further subclassified as spherical (non-communicating), tubular non-communicating and tubular communicating. In reported human cases of tubular colonic duplications, no two exactly identical cases have ever been reported. Furthermore, human cases often involve the duplicated tube terminating as a rectovaginal fistula or rectourinary fistula.

The aetiology of colonic duplication in humans and animals is unknown. It is postulated the condition is due to faulty division of the hind gut during fetal development. One possible theory is an early aberration in the development of the primitive hind gut which results in a division of the intestines resulting in duplication of the terminal gut. Incomplete recanalisation of portions of the gastrointestinal tracts that go through a solid stage has also been proposed.

This case report is the first to describe recurrent rectal prolapse associated with colonic duplication and the successful surgical excision of a ventrally located colonic duplication in a dog.

Case report
A 9-month-old entire female Shar Pei cross-bred was presented with a 7 month history of recurrent rectal prolapse. The prolapse would recur periodically at weekly to monthly intervals and was managed by the referring veterinarian with reduction of the prolapse and anal purse string sutures on four occasions. Initial surgical management had involved ventral midline laparotomy and an incisional colopexy from the left lateral aspect of the descending colon to the left internal abdominal wall. The dog also underwent routine ovariohysterectomy at this time. Colonoscopy was performed at the time of the colopexy and no gross abnormalities were observed in the descending colon.

Two weeks following colopexy and ovariohysterectomy, the dog was re-presented for a recurrent rectal prolapse. Due to the extremely aggressive nature of this dog, physical examination of the conscious animal was not possible. The dog was sedated with a combination of 100 mg tiletamine hydrochloride/100 mg zolazepam (Zoletil, Virbac Pty Ltd, NSW) intramuscularly and the perineum was examined. Examination of the external genitalia revealed no anatomical abnormalities. Digital vaginal examination was normal. A 15 cm diameter fleshy mass was protruding through the anal orifice. There was resistance to passage of a lubricated thermometer ventrally and laterally into the anus at the periphery of the extruded mass. This appeared to be consistent with partial rectal prolapse. The rectal mucosa appeared viable. Palpation of the extruded mass found it to contain faecal material. The prolapsed tissue was reduced. On digital rectal examination a 1 cm diameter ostium was found in the ventral colonic wall 2.5 cm cranial to the pubic brim. A warm water enema was performed. A 14 French foley catheter was placed just
inside the anal opening and the balloon inflated with 5 mL of water. Liquid barium (200 mL) was injected into the rectum and colon. The barium enema revealed a septal division coursing caudally along the ventral descending colon and rectum (Figure 1). A warm water enema was repeated and the dog recovered uneventfully. Surgery was planned for 48 h later. The dog was fasted for 24 h prior to surgery.

Anaesthesia was induced with 140 mg zolazepam/tiletamine intramuscularly and maintained with isoflurane / oxygen mixture. Intravenous compound sodium lactate was administered at 10 mL/kg/h. Intravenous amoxycillin 10 mg/kg and enrofloxacin 5 mg/kg were administered immediately following anaesthetic induction.

A ventral midline laparotomy was performed from the level of the umbilicus to the pubis. Balfour retractors were placed to maintain exposure of the abdominal cavity. The urinary bladder was retracted caudally with two stay-sutures of 3-0 polydioxanone. Sterile laparotomy sponges were packed around the descending colon. Faeces were milked cranially along the descending colon. A curved Doyen forcep was applied to the descending colon 20 cm cranial to the pubic brim. A transverse ventral colotomy was performed approximately 5 cm cranial to the pubic brim. A longitudinal partial thickness colotomy through the seromuscular layer of the descending colon was made approximately 1 cm caudal to the colotomy and extended to the level of the pubic brim. The ostium of the duplicated colonic segment was identified by passing a finger through the transverse colostomy into the duplicated tube (Figure 2). The seromuscular layer of the colon was undermined at the longitudinal colotomy to expose the mucosa of the duplicated colonic segment. The mucosa of the duplicated segment was incised at the level of the ostium. A stay suture was placed across the ostium to provide traction to the tubular duplication during its excision. The septum, or ventral aspect of the colonic mucosa, was excised using Metzenbaum scissors. The excision was continued caudally, past the level of the pubic brim, allowing excision of the duplicated segment to the level of the perineum. Excision of the duplicated colonic tube is summarised diagrammatically in Figure 3.

The longitudinal and transverse colotomies were closed with full thickness, simple interrupted 2-0 polydioxanone sutures. A serosal patch comprising jejunum was applied to colonic wounds with 3-0 polydioxanone interrupted sutures. The Doyen forceps were removed from the descending colon and laparotomy sponges were withdrawn from the abdominal cavity. The abdomen was lavaged with warmed, sterile isotonic saline. Gloves, drapes and surgical instruments were changed and the abdominal incision was closed routinely in three layers.

The dog made an uneventful recovery. Postoperatively, morphine 0.3 mg/kg was administered subcutaneously every 4 h for 12 h. Oral amoxycillin/clavulanate 10 mg/kg twice daily and enrofloxacin 5 mg/kg once daily were administered for 7 days postoperatively. Docusate sodium (Coloxyl, Fawns & McAllan Pty Ltd) 120 mg was administered orally with food once daily for 10 days. The dog was noted to have haematochezia for 3 days postoperatively. Defaecation has been reported as normal by the owner at 20 weeks post-operation and there has been no recurrence of rectal prolapse.

The excised duplicated colonic segment was submitted for histopathological evaluation. The septum included intact mucosa and muscularis mucosa separated by connective tissue (Figure 4). The mucosa was of normal colonic type. The histopathological diagnosis was incomplete tubular colonic duplication.
Discussion

The duplication in this case represented a type I tubular communicating colonic duplication and the opening of the duplicated segment was located ventrally in the colonic mucosa. The duplicated segment ended as a blind tube at the level of the perineum. There was no external opening to the duplicated segment. The case does vary from the colonic tubular duplication reported by Shinokazi et al in that the duplication in that case terminated 1.5 cm cranial to the anus.

The shared septum can range from two mucosal layers with intervening connective tissue to two attached, colonic walls, with the outer wall having all the tissue layers of a normal colon. It has been reported that the mucosa of the duplicated segment may resemble mucosa of a distant gastrointestinal viscus such as stomach or small intestine. Histopathological evaluation of the resected duplicate tube in this case identified normal colonic mucosa. The septum comprised connective tissue between a mucosae and muscularis mucosae and the histopathological classification in this case was incomplete colonic duplication (Figure 4). Differential diagnosis for this case was colonic diverticulum. A serosal cavity between the colon and duplicate tube was not demonstrated histologically, as would be expected with a colonic diverticulum.

To the authors’ knowledge this is the first report of the excision of a communicating tubular duplicated colon in the dog. A submuscularis resection of the duplicated segment was performed, leaving its common ventral colonic muscular wall intact. A pubic sympysiotomy was not required to improve access, as traction with stay sutures provided the necessary exposure to reach the termination of the duplicated segment. The seromuscular layer was elevated from the underlying mucosa of the duplicated segment easily. The termination of the duplicated tube was dissected free from its attachment at the perineum. The subseromuscular layer at this point was not able to be sutured to the ventral rectal wall. This has not resulted in faecal retention or difficult defaecation. In humans, various operative techniques have been utilised to remove the duplicated segment. These include dividing the common septum in order to create a common colonic channel, side-to-side anastomosis of adjacent normal colon and duplicated bowel, as well as mucosal stripping. Closure of the ventral rectal wall in our case resulted in a reduction of lumen diameter, but this has had no long term adverse clinical effects. Mild complications of tenesmus and haematochezia were noted for the first 5 d postoperatively but then resolved. Tenesmus during these first 5 d was controlled with the administration of a faecal softener. No further rectal prolapse was noted in the immediate or long term postoperative period.

Figure 3. (a) Partial thickness incision in the seromuscular layer of the colon. (---) represents incision into the page. (b) The seromuscular layer of the duplicate colonic tube is undermined and reflected. (c) The rectal floor is excised ensuring complete removal of the duplicate colonic tube. (---) represents excision into the page. (d) Arrows indicating direction of closure of colonic / rectal mucosa and seromuscular layer.
There have been two surgical reports for colonic duplication in dogs: one was for a tubular communicating colonic duplication and the other for a non-communicating spherical colonic duplication. In the case of the tubular communicating duplication, the septum between the colon proper and duplicated segment was incised with Metzenbaum scissors in a longitudinal direction, thus dividing the septum. This technique created a single, larger lumen, although no tissue was obtained for histopathological evaluation. In our case the duplicated tube was excised. This novel approach was chosen because it was speculated that incision of the septum longitudinally may have allowed healing along points of the incised septum, resulting in multiple outpouchings and resulting problems with faecal passage.

The second previous case report describes the excision of a non-communicating colon and this represented a spherical duplication that was cystic in nature, in contrast to the type of colonic duplication in our case. Furthermore, the authors in this second case reported reinforcement of the colonic wall closure with porcine small intestinal submucosa. Due to the mild reduction of the colonic/rectal lumen diameter in our dog, we reinforced the closure with serosal patching acquired from an adjacent loop of jejunum. No clinical evidence of colonic wall dehiscence was noted. Utilising autogenous tissue prevented any problems associated with tissue incompatibility.

There have been a number of reports in the human literature discussing the potential for malignant tumours to develop in association with the duplicated part of the intestine. The age at time of diagnosis in these patients ranged from 12 years in one case, to 57 years in another report. No such incidents of associated neoplasia have yet been reported in the veterinary literature and this may be a result of few reported cases of intestinal duplication or death of affected animals before neoplasia development. Nevertheless, the potential, association with malignancy that has been described with intestinal duplications in the human literature influenced our decision to excise the duplicate colonic tube.

A postoperative radiographic contrast barium enema was offered but declined by the owners. As the duplicated tube has been excised, we would anticipate a normal radiographic image. Twenty weeks postoperatively, the dog is defaecating normally and there have been no further episodes of rectal prolapse.

Dogs afflicted with colonic duplication can have signs ranging from asymptomatic to tenesmus, increased frequency of defaecation, faecal retention and abdominal distension. To the authors' knowledge there is only one other case of rectal prolapse secondary to duplicating colon in the veterinary literature. In that case, rectal prolapse occurred once and the puppy was euthanased intraoperatively. The prolapse reported in the present case should be considered recurrent and partial as the ventral and lateral rectal wall was prolapsed with the terminal end of the duplicated tube. This case serves to highlight that an underlying cause in cases of rectal prolapse and colonic and/or rectal duplication should be investigated in the young dog. In this case it is presumed that the condition was congenital because the dog was displaying signs of tenesmus and partial prolapse at 6 weeks of age. We were unable to contact the breeders to establish further historical information.

In the previously reported case of communicating tubular colonic duplication, the opening to the duplicated segment was identified with colonoscopy. In the present case, colonoscopy failed to identify the opening to the ostium, possibly because of inadequate air distension of the descending colon, with resulting mucosal folds obscuring an opening. Alternatively, the ventrally located and, possibly, caudally directed ostium, or faecal material, may have obscured the opening. Because colonoscopy had failed to reveal the opening to the duplicated segment, an incisional colopexy was performed initially, and the rectal prolapse recurred.

This case highlights the importance of a diagnostic barium enema. At initial presentation a barium enema was offered but declined. At a later stage the owners consented to a barium enema following recurrence of the prolapse. A barium enema has been diagnostic in previous cases of colonic duplication in the dog. The barium enema was useful in our case, but would not be useful in cases of non-communicating intestinal duplications. Sonography may be a useful tool for investigation of non-communicating intestinal duplications.

This report describes the first successful excision of an incomplete tubular, communicating colonic/rectal duplication in a dog with a novel surgical procedure and no recurrence of partial rectal prolapse postoperatively. Colonic or rectal duplication should be considered as a differential diagnosis for rectal prolapse in the juvenile dog and a barium enema is recommended in cases of recurrent rectal prolapse.

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References
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