

# Surgical treatment of noncommunicating duplication of the colon in a dog

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- ▶ Colonic duplication is a rarely reported congenital anomaly in humans, dogs, and horses.
- ▶ Diagnosis of this condition requires specialized imaging techniques and surgical exploration of the abdomen with histologic evaluation of excised tissue.
- ▶ Excision of a conjoined noncommunicating duplicate colon, with use of porcine small intestinal submucosa to reinforce repair of serosal defects in the true colon, may provide a means for successful treatment of this or similar conditions.

A 4-month-old 2.3-kg (5-lb) sexually intact male Jack Russell Terrier was evaluated at the University of Missouri-Columbia Veterinary Medical Teaching Hospital (UMC-VMTH) because of tenesmus and stranguria that had developed rapidly during the preceding 48 hours. Twenty-four hours prior to admission, the dog had been examined by the referring veterinarian. At that time, the dog was pollakiuric and able to defecate only small amounts of mucoid feces with great difficulty. Urinalysis revealed hematuria and bacteriuria. A fecal flotation was performed but revealed no evidence of intestinal parasitism. Cystographic evaluation revealed a soft-tissue opacity between the colon and the bladder. The dog was treated with penicillin and dexamethasone (dosages and administration routes unknown) and hospitalized overnight for observation. The following day, the tenesmus and stranguria persisted, and the dog was referred to UMC-VMTH for additional diagnostic tests. Prior to transportation of the dog to UMC-VMTH, its urinary bladder was catheterized and emptied.

On physical examination, the dog appeared to be in good health; its size and weight were normal for a Jack Russell Terrier of that age. The dog had signs of depression, assumed a hunched position when standing, and frequently strained while posturing to defecate. Rectal temperature was 38.2°C (100.8°F), heart rate was 152 beats/min, and respiratory rate was 20 breaths/min. Abdominal palpation did not elicit signs of pain and revealed a large (2 × 7 cm), firm, tubular mass in the caudal dorsal abdomen. Via digital rectal examination, a firm, spherical mass was palpable along

the ventral wall of the rectum; the mass was located approximately 3 cm into the rectum and occluded the full luminal diameter. Blood was obtained via jugular venipuncture for CBC and serum biochemical analyses; results of these tests were within reference ranges.

Examination of radiographs provided by the referring veterinarian revealed that the urinary bladder was radiopaque because of administration of contrast medium. Serosal detail was poor. A large, homogeneous, ill-defined soft-tissue opacity was present between the colon and the bladder (Fig 1). This resulted in a mass effect in which the trigone of the bladder was displaced ventrally, and the distal portion of the descending colon was displaced dorsally. On the ventrodorsal radiographic view, a tubular soft-tissue opacity was seen laterally and to the left of the descending colon. A large amount of fecal material of granular appearance was evident throughout the colon cranial to the mass; however, at the level of the mass, the descending colon and rectum were void of feces. To obtain a retrograde urethrogram, the lumen of an 8-F Foley catheter was filled with 3 mL of 2% lidocaine solution; the catheter was inserted approximately 1 cm into the urethra and the bulb expanded with 3 mL of physiologic saline (0.9% NaCl) solution. Iohexol<sup>®</sup> (240 mg of iodine/mL) contrast medium was diluted to a 1:4 ratio with sterile saline solution, and approximately 10 mL of the resultant solution was injected into the urethra. Lateral and ventrodorsal oblique radiographic views of the caudal portion of the abdomen were obtained during administration of the solution and revealed that the prostatic urethra was narrowed and displaced ventrally. On ultrasonographic examination of the caudal portion of the abdomen, a large, round, fluid-filled structure was detected dorsal to the bladder. Multiple intensely hyperechoic linear foci were dispersed throughout the mass (Fig 2), and its fluid content resulted in distal acoustic enhancement in the ultrasonographic image. This structure was tubular in shape in a sagittal plane, and it compressed the trigone of the bladder. Differential diagnoses included paraprostatic cyst, pericolonic abscess or cyst, or duplicate colon.

At the initial evaluation at UMC-VMTH, it was reported that the puppy had been unable to urinate that day; therefore, placement of an indwelling urinary catheter connected to a closed urine collection system was undertaken, and the 5-F red rubber catheter passed easily through the urethra. Exploratory surgery was scheduled for the following day.

A 24-gauge catheter was placed in a cephalic vein, through which lactated Ringer's solution was administered during surgery (5 mL/kg/h [2.3 mL/lb/h], IV). The dog was premedicated with glycopyrrolate (0.01

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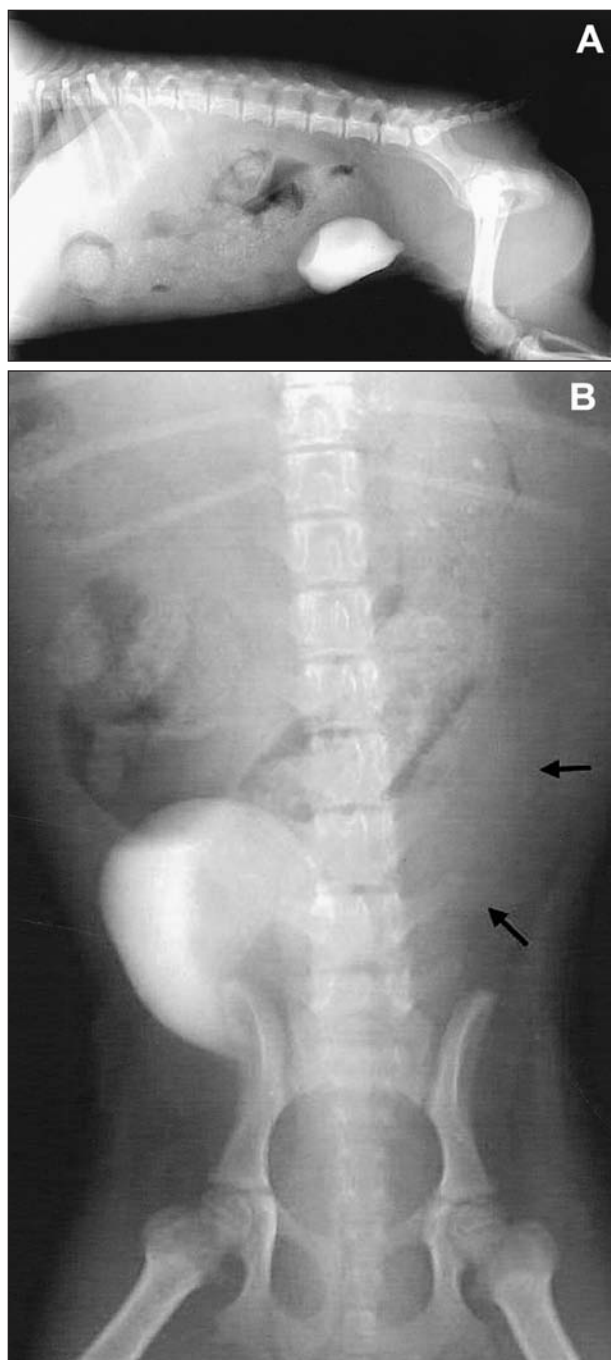


Figure 1—Lateral (A) and ventrodorsal (B) radiographic views of the caudal portion of the abdomen of a dog with colonic duplication. Notice the residual contrast medium in the urinary bladder from a cystographic evaluation performed by the referring veterinarian. A large homogeneous soft tissue opacity is evident between the bladder and the colon. The trigone of the bladder is compressed ventrally. In the ventrodorsal view, a tubular-shaped soft tissue opacity is seen adjacent to the lateral aspect of the descending colon (black arrows).

mg/kg [0.005 mg/lb], IM), xylazine (0.65 mg/kg [0.30 mg/lb], IM), and morphine (0.65 mg/kg, IM). Anesthesia was induced with sodium pentothal (11 mg/kg [5 mg/lb], IV) and maintained with isoflurane. Cefoxitin<sup>b</sup> (22 mg/kg [10 mg/lb], IV) was administered slowly at induction and every 90 minutes during

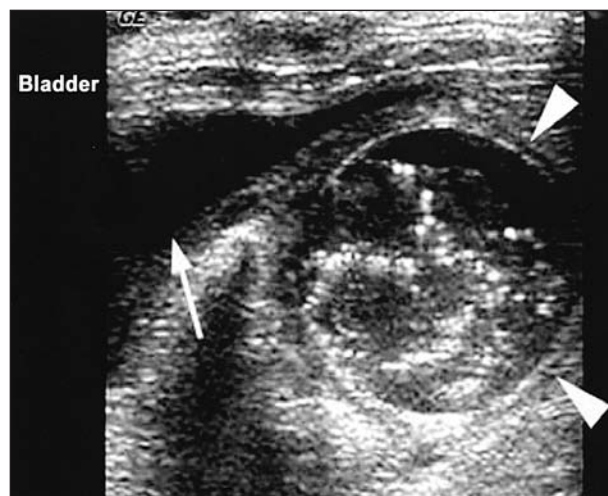


Figure 2—Ultrasonographic image of the caudal portion of the abdomen of a dog with colonic duplication. Notice the large, round, fluid-filled structure with multiple intensely hyperechoic foci (white arrowheads) located dorsal to the trigone of the bladder (white arrow). The prostate could not be seen.

surgery. The dog was clipped and prepared for ventral midline laparotomy.

On examination of the abdominal cavity, a large, tubular cystic mass (10 × 1.5 cm) that was adherent to the antimesenteric border of the descending colon was identified (Fig 3). The mass extended along the descending colon from the region approximately 2 cm aboral to the left colic flexure to the level of the prostate. In the aboral direction, the extent of the mass could not be defined, because it was adhered between the colon, prostate, and dorsal aspect of the bladder. The tubular mass was greatly distended with a clear, viscous fluid that was mixed with a yellow mucoid substance. The fluid was aspirated from the mass with a 22-gauge needle and syringe for cytologic evaluation (performed concurrently with the operative procedure) and aerobic and anaerobic bacteriologic culture. The colon was compressed by the large mass and was filled with feces oral to the conjoined tubular structure. The urinary bladder was retracted caudally and secured with a stay suture. An apparent depression in the serosal surface of the colon and conjoined mass was explored as a dissection plane; the shared region of serosa was incised over this depression, and the underlying tunica muscularis seemed to be associated with both structures. Via blunt dissection, the muscularis layer between the colon and mass was split to enable removal of the tubular mass. The mass was removed from the antimesenteric colon as far caudally as possible without disruption of the trigonal and prostatic adhesions. Excision of the mass left a partial thickness seromuscular defect in the wall of the descending colon. However, there was no communication between the lumina of the cystic structure and colon along the length of the adhered mass. A small portion (1 × 1 cm) of the seromuscular wall and underlying mucosa of the cystic mass that was adhered to the bladder trigone and dorsal prostatic region could not be excised. Both kidneys and ureters were examined and appeared to be normal (ie, no evidence of obstruction). All excised tis-

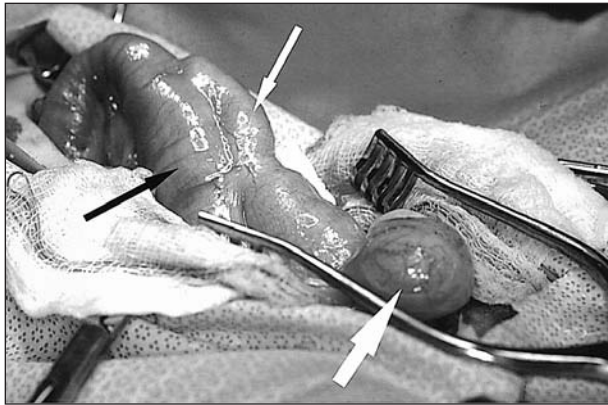


Figure 3—Photograph obtained during surgery of the abdomen of a dog with colonic duplication. Notice the duplicate colon (narrow white arrow) adhered to the antimesenteric border of the descending colon (black arrow). The duplicate colon shared the seromuscular layer with the colon aborally to the level of the bladder (large white arrow).

sues were placed in neutral-buffered 10% formalin solution for subsequent histologic examination.

After removal of the conjoined tubular mass, a 1-cm<sup>2</sup> diverticulum in the colonic mucosa and submucosa of the descending colon (at the most caudal limit of the excision site) was noted. This diverticulum was resected and closed with a simple continuous suture pattern with 4-0 polydioxanone suture.<sup>c</sup> The cut edges of the colonic seromuscular layer were apposed along the length of the defect with the same type of suture in a simple continuous suture pattern, thereby inverting the exposed subserosal colonic layers. Because the impacted colon was dilated, removal of the mass and subsequent closure of the defect resulted in minimal narrowing of the colonic lumen. A single layer of 4-ply porcine small intestinal submucosa<sup>d</sup> was placed over the sutured serosa and secured with simple interrupted sutures of 4-0 poliglecaprone 25.<sup>e</sup> An omental pedicle was created and advanced to the caudal portion of the abdomen. The most caudal aspect of this omentum was attached with simple interrupted sutures of 4-0 poliglecaprone 25 to the remnant of nonresectable mucosa that was adherent to the dorsal aspect of the bladder and prostate.

The abdominal exploration was completed without any other important findings. The abdomen was lavaged with warm saline solution prior to routine closure. The dog recovered from anesthesia without complication in the UMC-VMTH intensive care unit. Evaluation of the distal colon via digital rectal examination suggested resolution of the obstructing mass. For 24 hours after surgery, lactated Ringer's solution was administered at a maintenance rate (0.5 mL/kg/h [0.23 mL/lb/h]), and buprenorphine was provided for analgesia (0.01 mg/kg, q 6 h, IV). Cefoxitin was continued (22 mg/kg, q 6 h, IV). Within hours of completion of the surgery, the dog began to urinate without evidence of stranguria, although diarrhea and dyschezia persisted. Urination continued to be normal following surgery. The diarrhea gradually resolved over the ensuing 5 days, and the tenesmus subsided a day later at which time normal defecation was first

observed. Whereas the puppy was mildly anorectic the day of referral to the UMC-VMTH, its appetite was excellent immediately after recovery from anesthesia and remained so throughout hospitalization. At discharge (1 week after admission), the owners were instructed to keep the dog at rest for 4 weeks and observe closely its urination and defecation habits.

Results of the cytologic examination of the fluid contents of the cystic mass were inconclusive. The sample contained few intact nucleated cells and low numbers of RBCs; neither microorganisms nor neoplastic cells were found. The fluid component of the sample contained eosinophilic material, which suggested a largely proteinaceous composition. Bacteriologic culture of the fluid, with or without enrichment broth, did not produce aerobic or anaerobic growth. Histologically, the excised tissue was lined by well-developed normal colonic epithelium that was supported by submucosa and muscular tunics; this finding was consistent with colonic duplication. There were multifocal areas of hemorrhage in the mucosa and submucosa and focal areas of fibrosis and perivascular accumulations of plasma cells and lymphocytes within the submucosa and muscular tunics.

Four weeks after surgery, the puppy was reexamined at UMC-VMTH. At that time, the owners reported that the dog's condition was excellent and that all episodes of stranguria and tenesmus had subsided. Via rectal examination, there was no evidence of mass recurrence or stricture formation. Abdominal radiography and ultrasonography revealed a thin soft-tissue opacity between the colon and bladder, which the authors believe to be the omental flap. During the subsequent 4 months, the dog continued to develop normally without recurrence of clinical signs.

Colonic duplication is a rare congenital anomaly that has been reported in humans,<sup>1</sup> dogs,<sup>2</sup> and 1 horse.<sup>3</sup> It is estimated that 1 in 4,000 children is born with alimentary tract duplication, most commonly involving the jejunum or ileum.<sup>1,4</sup> Duplication of the colon has been reported in fewer than 75 humans<sup>4,5</sup>; the veterinary medical literature includes reports of only 5 cases (involving 4<sup>2,6-8</sup> dogs and 1 horse<sup>3</sup>). Three of the dogs with duplication of the colon were euthanized after the identification of other profound congenital malformations, including genitourinary anomalies, vertebral duplication, and cecal malformation.<sup>2,3,6</sup> Surgical correction of a duplicate colon has been reported<sup>7</sup> in 1 dog in which there was communication between the colons; the communicating ostium was extended by incising the remaining septum to form a common lumen between the true and duplicate colon. That surgical procedure was successful, and the dog was free of clinical signs associated with the duplication at an examination performed 6 months after surgery. To the authors' knowledge, the surgical correction of a noncommunicating duplicate colon in a dog has not been reported.

Diagnosis of duplicate colon requires histologic evaluation to differentiate aberrant tissue from a diverticulum (the latter is a defect in the tunica muscularis that allows the mucosa to bulge outward). The tissue of an intestinal duplication must be attached to the gastrointestinal tract and must include both alimenta-

ry epithelium and a central layer of smooth muscle.<sup>9</sup> After histologic confirmation of an intestinal duplication, its description is based on gross appearance, anatomic association, and communication with the functional tract.

The classification system for colonic duplication that is used in human medicine can also be applied to colonic duplication in dogs. Type I duplications are limited to discrete segments of the colon or rectum, whereas type II duplications are complete and often associated with genitourinary anomalies. Type I duplications may be further described as spherical noncommunicating (Ia), tubular noncommunicating (Ib), tubular and communicating (Ic), loop form with separate blood supply (Id), or multiple combined duplications (Ie).<sup>7</sup> In the dog of this report, the colonic duplication was type Ib, because the redundant segment was tubular in shape and did not communicate with the true colon.

Duplication of additional body systems, such as the genital or urinary tracts, has been reported to occur in as many as 80% of humans with colonic duplication.<sup>10</sup> This relationship, however, may vary among the different types of colonic duplication. It has been suggested that type II colonic duplication is more often found in conjunction with duplications of other body systems and may be related to faults at various stages in development.<sup>2</sup> The primitive digestive tract develops from the folded yolk sac endoderm. The midgut region becomes the small intestine, cecum, and proximal colon, whereas the distal colon, rectum, and urogenital tract are products of the hindgut.<sup>11</sup> Concurrent colonic and urogenital duplication may result from partial formation of a twin at the level of the hindgut.<sup>2</sup> Duplications that do not involve other organ systems may originate later in development, after the distal hindgut has folded back on itself and established the future development of the rectum and urogenital sinus.<sup>2</sup> The hindgut changes from a solid to a tubular structure when epithelial cells at its center become rearranged, and their vacuoles become joined. If these vacuoles coalesce at several positions, multiple lumina may potentially develop. Ischemic intestinal insult has also been suggested as a cause of duplication, wherein a small portion of devitalized intestine persists to become an extraneous intestinal tract segment.<sup>2,4,7</sup> Because the embryogenesis of intestinal duplications is still poorly understood, it is difficult to speculate about the origin of the congenital defect in the dog of this report.

The clinical signs of alimentary tract duplications in humans depend on the anatomic location, communications, and space occupancy of the duplication.<sup>1,5,10</sup> Specifically, colonic duplications have been reported to cause vomiting, abdominal pain and distension, and constipation, although these duplications have also been described as incidental findings in asymptomatic adults.<sup>1,5,10</sup> Because of the nonspecific nature of these signs, diagnosis requires sophisticated imaging techniques, often with surgical exploration of the abdomen.<sup>1,7</sup> If untreated, complications may include intestinal obstruction or perforation, hemorrhage, and pain.<sup>12</sup> In dogs with colonic duplication, clinical signs

include increased frequency of defecation, tenesmus, constipation, urinary incontinence, fecal retention, abdominal distension, and asymptomatic presentation.<sup>2,6,7</sup> In the dog of this report, the most severe signs were tenesmus and stranguria. Stranguria has not been reported as a clinical sign associated with colonic duplication. Because of the noncommunicating nature of this dog's duplicate colon, the secretions produced into its lumen had no route of evacuation and thus exacerbated its distension. Within the caudal abdomen and pelvic canal entrance, it seemed likely that the increasing distension of the duplicate colon was causing compression of the descending colon and urethra and resulted in the observed clinical signs.

Although surgical correction of a duplicate colon was performed in a dog in which there was communication between the colons, resection in that dog was not elected because of the shared blood supply with that of the true colon.<sup>7</sup> Surgical resection of the duplicate colon was elected as treatment for the dog of this report because of its antimesenteric attachment, absence of a well-developed blood supply, and lack of communication with the true colon. The pelvic diameter of the puppy was assessed to be too small to accommodate the entrance of a terminal colon twice its normal diameter, because the duplicate colon was compressing the urethra sufficiently to compromise urine flow. For these reasons, removal of the duplicate colon was recommended; interestingly, at the time of surgery, the duplicate colon was extremely thin walled, which suggested potential compromise of peristalsis had incorporation with the true colon been attempted.

After resection of the duplicate colon and primary repair of the remaining seromuscular defect, the colonic repair site was reinforced and enhanced with a graft of porcine small intestinal submucosa and overlying omental flap. Porcine small intestinal submucosa is an acellular, naturally occurring extracellular matrix comprised predominantly of collagen type I with glycosaminoglycans and growth factors, including basic fibroblast growth factor, transforming growth factor- $\beta$ , and vascular endothelial growth factor.<sup>13</sup> Its use as a grafting substrate for reconstruction or reinforcement of abdominal wall, urinary bladder, tendons, blood vessels, dura mater, and small intestine has been reported.<sup>14</sup> Although not fully understood, small intestinal submucosa induces site-specific tissue regeneration in a variety of applications.<sup>15</sup> It is believed to provide a scaffold for migration of normal cells to repair tissue defects and to promote appropriate tissue regeneration, rather than replacement with scar tissue.<sup>15</sup> The milieu of growth factors, collagen types I and IV, and fibronectin are thought to increase the ability of local cells to attach and migrate into areas of injury.<sup>13,16</sup> Similar to all xenografts, small intestinal submucosa is immunogenic but appears unique in its preferential activation of a T helper cell 2 response following acute inflammation. This T helper cell 2 response leads to graft remodeling with tissue acceptance and regeneration, rather than a T helper cell-mediated graft rejection.<sup>17</sup> Small intestinal submucosa may be particularly useful in gastrointestinal tract surgery, because its ability to facilitate

healing of full-thickness small intestinal defects has been demonstrated.<sup>14</sup> Another method of colonic surgical reinforcement involves serosal patching. However, because of the limited space of the narrow caudal abdomen in the puppy of this report, the application of a thin layer of small intestinal submucosa was more appropriate.

Advancement of a pedicle of omentum over the ventral surface of the colon was performed primarily to establish omentalization of an unresectable portion of the duplicate colon. The omental pedicle was placed to provide drainage for potential continued secretions from the duplicate colonic mucosal remnants and prevent postoperative cyst formation. Omentum can facilitate abdominal and thoracic drainage, because it contains the major system of lymphatic drainage from the peritoneal cavity, a rich network of small vessels and capillaries, and the ability to induce and promote angiogenesis.<sup>18</sup> For these reasons, it may have also been secondarily beneficial in the healing of the colonic surgical wound.

Despite the rarity of colonic duplication, its occurrence should be considered as a differential diagnosis in the evaluation of young dogs with acute onset of signs of abdominal disease. Furthermore, from the assessment of the dog of this report, stranguria should also be considered as a potential clinical sign of colonic duplication in young dogs. Because of its tissue-engineering capabilities in the small intestine, small intestinal submucosa was used for reinforcement of colonic serosa in the dog of this report; the success of this procedure expands the surgical applications in which small intestinal submucosa may be beneficial.

<sup>a</sup>Omnipaque, Nycomed Inc, Princeton, NJ.

<sup>b</sup>Mefoxin, Merck & Co Inc, West Point, Pa.

<sup>c</sup>PDS suture, Ethicon Inc, Somerville, NJ.

<sup>d</sup>Vet BioSISt, Cook Biotech Inc, West Lafayette, Ind.

<sup>e</sup>Monocryl suture, Ethicon Inc, Somerville, NJ.

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