Small Colon Strangulating Lipoma in the Mare

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Introduction:

The diagnosis and treatment of colic is one of the most common challenges presented to equine practitioners today. In a survey of 1427 horses on 31 different farms, an incidence rate of 10.6 colic cases/100 horse years was reported, illustrating how common colic is as a presenting complaint to equine practitioners.

The term colic is very non-specific, and only describes an acute state of abdominal pain or discomfort. The term itself does not provide any information regarding the cause of the condition. There are a variety of conditions that can cause colic, as well as numerous risk factors that predispose horses to these conditions. Specific risk factors known to be associated with colic include previous history of colic, changes in concentrate feeding, changes in hay feeding, feeding high levels of concentrate, and even vaccination with a monocytic ehrlichiosis vaccine.[3] Specifically, changes in type of hay fed has been shown to increase the risk of colic in equine patients.[16] The sources of abdominal pain described as colic may range from relatively minor gastrointestinal conditions including spasmodic or gas colic, to more severe conditions such as large colon impaction or intestinal strangulation. Additionally, there are several non-gastrointestinal causes exist, such as disorders involving the urogenital and reproductive tracts, further confusing the picture.

The pathological causes of colic can collectively be organized into the following categories: obstruction, strangulation, nonstrangulating infarctions, enteritis, peritonitis, ulceration, or ileus.[2] Two studies have documented that 75 – 80% of colic cases are caused by gas colic or a colic of unknown cause, with patients recovering following little to no treatment at all.[1,2] Because colic can be quite painful and thus pose a safety risk to both the horse and the people involved, a swift yet judicious diagnostic work-up should be performed by the practitioner in order to facilitate the

most appropriate next step in treatment. Although a high percentage of colic episodes do not require emergency medical attention and have the potential to resolve spontaneously, each case should be approached with the goal of determining the cause of the colic. In many cases, the exact cause of the colic symptoms will not be determined, therefore the practitioner should take into consideration all information available when determining which cases can be managed medically, and those that should be taken to surgery. For example, causes of colic such as bacterial colitis are typically managed medically, whereas colonic displacements would warrant surgery. Strangulating lesions cannot be managed medically, and require surgery for correction. Strangulating lesions typically involve the small intestine, and the most common small intestinal strangulating lesions are pedunculated lipomas and epiploic foramen entrapments.[5] The small colon is more rarely involved in strangulating lesions in comparison to the small intestine, however strangulating small colon lesions may involve the inguinal ring, flank musculature defects, tears in omentum, uterine broad ligament tears, mesenteric tears, or uterine or vaginal tears.[11] Pedunculated lipomas may also cause strangulations of the small colon, but are much more rare than those found involving the small intestine. It has been reported that in cases of strangulating lipomas, 93% involved the small intestine, while only 7% involved the small colon.[17] This disparity is further illustrated by one review which found that of 75 pedunculated lipoma cases examined, 70 involved the small intestine, while only 5 involved the small colon.[4] Strangulating lesions of the small colon caused by pedunculated lipomas must often be managed differently than small intestinal strangulating lesions due to the inherent differences in blood supply and bacterial load between the small intestine and small colon. This report will describe the diagnosis and treatment of a small colon strangulating lesion caused by a pedunculated lipoma.

History:

March 18, 2017 a 21 year old Paint horse mare presented to the Mississippi State University College of Veterinary Medicine (MSU CVM) Equine Department on referral for colic. The mare had been observed to be rolling in her turnout the day before, with a decreased appetite. The mare had been administered 480 mg of flunixin meglumine by her owners upon noticing her behavior and her referring veterinarian was contacted. Upon arrival to the boarding facility, the referring veterinarian administered 3 mg of detomidine intravenously for analgesia and sedation, and passed a nasogastric tube. No net reflux was obtained, and 8 L of electrolyte water was administered through the nasogastric tube. Rectal palpation at this time revealed what the referring veterinarian suspected to be a large pelvic flexure impaction. Approximately 30 minutes following intravenous administration of the detomidine, the mare was obviously painful once again, and the dose was repeated along with a 5 L bolus of LRS intravenously via a jugular catheter. Sand and other debris was observed in and around the mare's eyes presumably from rolling. This debris was removed by thorough rinsing, and a fluorescein stain was used to check for any corneal defects. No corneal ulcers were observed. Due to her uncontrollable pain, the mare was referred to the MSU CVM Equine Department for further evaluation and treatment.

Presentation and Emergency Stabilization:

Upon arrival to the MSU CVM Equine Department, the mare's mental status was dull. She weighed 436 kg (960 lbs.) and was a body condition score of 5/9. She was tachycardic 52 beats per minute (rr= 24-36 bpm) with a second degree atrioventricular block, normal respiration 12 breaths per minute (rr = 8-12 bpm), and normothermic of 99.4 Fahrenheit (rr = 99.5-101.5 F). Her mucous membranes were observed to be pink and mildly tacky, and a capillary refill time was measured to be less than 2 seconds. She was estimated to be 5% dehydrated. Abdominal

auscultation revealed significantly decreased gastrointestinal motility in all four quadrants. Digital pulses palpated normally in all four limbs, and a moderate amount of mucopurulent ocular discharge was observed bilaterally. Prior to rectal palpation, xylazine and buscopan (N-butylscopolammonium bromide) were administered intravenously. Rectal palpation revealed a large impaction of the pelvic flexure occupying the right ventral abdomen. On abdominal ultrasound, several loops of small intestine including the duodenum were visualized and determined to be normal. Cecum and colon distention was observed on the right side of the abdomen with the colon wall measuring 6.5 mm (normal = less than 5 mm)[18]. Colonic teanial bands were observed horizontally on ultrasound, raising suspicion of colonic displacement. A complete blood count and blood chemistry were performed upon arrival. The following abnormalities were observed on the complete blood count: mildly decreased RBC count, mildly decreased PCV, a mild mature neutrophilia, and a moderate lymphocytopenia. Abnormalities observed on the blood chemistry include the following: mild hypochloremia, mildly elevated CO2, mild hyperglycemia, mild hyperglobulinemia, mild hypocalcemia, mild hypophosphatemia, moderately elevated creatinine kinase, and moderate hypomagnesemia.

Emergency stabilization was implemented including a 20 L intravenous bolus of Lactated Ringer's solution, followed by Plasmalyte at a rate of 1L/hour. A nasogastric tube was placed and secured in order to facilitate continual oral fluid therapy as well as monitoring for reflux. During the initial 12 hours of her hospital stay, the nasogastric tube was checked every 4 hours for reflux, and 4 L of water was administered if the net reflux was less than 2 L. Approximately 1 gallon of mineral oil was administered via the nasograstric tube over the first 12 hours. Once initial stabilization and diagnostic procedures were concluded, the mare was moved to a stall for supervision throughout the night. Three hours following her arrival, the mare was observed to be

painful and was attempting to roll in her stall. At this time, 480 mg of banamine were administered intravenously, however no improvement was seen. Thirty minutes later, 150 mg of xylazine and 5 mg of butorphanol were administered intravenously for analgesia. Following this administration, the mare appeared to be more comfortable for the rest of the night. The following day the mare was stable but uncomfortable. She did not pass any feces, and no net reflux was obtained from the nasogastric tube throughout the day. Rectal palpation was repeated 36 hours after arrival revealing the suspected pelvic flexure impaction to be slightly reduced in size, but displaced and occupying a large portion of the right ventral abdomen. Based on her clinical condition and lack of improvement, the mare's owners elected for an exploratory celiotomy. Upon exploration, a strangulating lipoma of the small colon was diagnosed as the source of the colic symptoms.

Pathophysiology:

Abdominal pedunculated lipomas in horses are benign hyperplastic adipocyte deposits that typically originate in mesenteric tissue.[7] These abdominal lipomas may be pedunculated or broad-based, solitary or multiple in nature, and most commonly originate from the mesentery of the small intestine, however they have been reported to originate from the mesentery of the descending colon, rectum, and cecum as well.[7] Specifically, the mesocolon typically contains a large amount of fat deposits, making the development of lipomas more probable, however the thickness of the wall of the descending colon along with the presence of fecal balls usually acts as a deterrent for the formation of a strangulating lesion.[17] In comparison to other diseases of the small colon, strangulating lipomas make up a relatively small percentage at approximately 11%.[17] Ponies, Arabians, and Quarter Horses are reported to be more commonly affected by pedunculated lipomas, and castrated males are reported to be more commonly affected than

females or sexually intact males.[4,7,17] One study indicated that Saddlebred and Arabian horses greater than 14 years of age were among the highest category at risk for pedunculated lipoma formation.[7] Geriatric horses are predisposed to the development of strangulating lipomas, with the mean age of horses with small intestinal strangulating lipomas being 19.2 years.[10] In a demographic study of equids 20 years of age and older, it was reported that strangulating lipomas were the second most common specific disease process in geriatric horses[12], further illustrating the effect of age on the prevalence of strangulating lesions caused by lipomas within the equine population.

Strangulation differs from simple internal obstruction in that a strangulating lesion simultaneously occludes the intestinal lumen as well as its blood flow.[8] It is often difficult to differentiate an internal obstruction such as an impaction from a strangulating lesion clinically, as they can present similarly. However, strangulating lesions often lead to necrosis of the intestinal mucosa and subsequent endotoxemia and sepsis sooner than internal obstructions, due to a more rapid loss of blood supply.[8] Strangulating lesions can be divided into two general categories: hemorrhagic strangulations and ischemic strangulations.[8] Hemorrhagic lesions occur when the venous blood supply is occluded, but the arterial supply remains viable. This leads to an increase in size of the affected section due to continual inflow of blood from the arterial supply. The affected section of bowel becomes engorged with blood and takes on a characteristic purple to black appearance.[8] Ischemic strangulation occurs during instances of intestinal volvulus or other tight strangulations where both the venous and arterial supply is occluded by the strangulation. Bowel affected by ischemic strangulation will often appear pale in comparison to neighboring bowel, and can be smaller in size due to a lack of blood flow.[8]

When small intestinal strangulating lesions are present, fluid from ingesta within the lumen of the intestine is unable to reach the large bowel where it would normally be absorbed. This leads to sequestration of the fluid orad to the strangulation and can cause hypovolemia, decreased cardiac output, and acid-base disturbances if severe enough.[9] Reperfusion injury is a concern when a strangulating lesion is corrected surgically, as inflammatory products and oxygen-derived free radicals may be released from the previously strangulated section of bowel, leading to ileus or post-operative infarction.[9] The mucosa of the small intestine is very vascular, accounting for about 80% of the blood supply to the small intestine, and as a result is very sensitive to hypoxia from lack of blood supply or blood stasis.[9] Hypoxic damage to the mucosal layer allows gram-negative bacteria and endotoxins to penetrate the laminal propria and submucosa, entering circulation through the vasculature of the tissue adjacent to the strangulated lesion.[9] Bacteria and endotoxins eventually end up in the peritoneal cavity once the muscularis layer begins to degrade, allowing them to leak through the serosa.[9] Upon gaining entry into circulation, endotoxins begin the cascade of white blood cell activation and endothelial cell damage known as endotoxic shock. Endotoxic shock results in increased vascular permeability and decreased cardiovascular function.[9]

In general, the clinical course of disease of strangulating lesions of the small colon is slower than that of the more proximal segments of bowel.[11] Some indications of a strangulating lesion involving the small colon are an increased concentration of nucleated cells and total protein within abdominal fluid, as well as distention of the large colon and absence of feces detected during rectal palpation.[11] In comparison to the small intestine, the bacterial load of the small colon is exponentially greater, and the blood supply much lower, making it imperative to relieve a strangulating lesion as soon as possible. Following surgery of the small colon, fever, diarrhea

and laminitis are all common complications, and are thought to be due to increased bacterial toxin absorption across the compromised intestinal tissue.[11,15]

Diagnostic Approach:

Diagnosing a strangulating lesion caused by a pedunculated lipoma can be very difficult to achieve without exploratory surgery. It is therefore imperative that a standard diagnostic approach to equine colic cases be used so that the practitioner does not neglect clinical signs or symptoms that may suggest a serious and life-threatening strangulating lesion. Information derived from the physical exam and diagnostic tests may be grouped into 5 basic categories that can be used to help differentiate between the need for medical or surgical treatment of a colic case. These categories are as follows: pain, cardiovascular status, palpation per rectum, degree of nasogastric reflux, and the results of abdominal paracentesis.[13]

Pain associated with a strangulating lesion is often severe and unresponsive to analgesics. The degree of pain is related to the amount of bowel involved in the strangulation, as well as the length of time the strangulation has been present. It is thought that pain results directly from ischemia to the affected bowel, and that over time, the horse may become more depressed, but slightly less painful as the section of bowel becomes necrotic.[9] Intestinal distention and mesenteric tension may also be significant sources of pain as well.[13] In specific cases of small colon obstruction, clinical signs may take a slower course of development than those involving the small intestine. As is often the case with strangulating pedunculated lipomas, complete obstruction of the small colon can often lead to severe abdominal distention, decreased to absent fecal production, depression and pain.[11]The degree of pain and its responsiveness to a relatively low dose of alpha-2 agonist analgesics or NSAIDs can indicate to the practitioner the likelihood that the lesion may be treated with medical therapy alone.[13] If the relief of pain provided by

medication is short-lived or absent, a more severe lesion such as a strangulation should be suspected.[13] Pain should be interpreted within the overall context of the patient however, as decreasing amounts of pain in the face of increasing mental depression or worsening cardiovascular parameters may signal the presence of necrotic tissue.[13]

The cardiovascular status of the patient is often a good indicator of the severity of the lesion, and can be assessed by recording heart rate and rhythm, pulse rate and quality, mucous membrane color and capillary refill time.[13] Endotoxic shock is a common cause of cardiovascular collapse, and occurs frequently with severe strangulating lesions involving pedunculated lipomas. In cases of small colon strangulation, fluid may accumulate orad to the strangulation within the lumen of gastrointestinal tract, leading to a decrease in circulating fluid volume and hypovolemic shock.[13] Elevations in heart rate must be interpreted carefully, as these may signal that the patient is in hypovolemic shock and attempting to maintain tissue perfusion, or it may simply be a response to pain.[13] Other factors such as pale mucous membranes or increased capillary refill time present concurrently with elevations in heart rate may signal a more compromised cardiovascular state, rather than a simple response to pain. Vasodilation caused by endotoxic shock as a result of necrosis of a section of bowel can lead to very dark and congested mucous membranes with a rapid capillary refill time.[13] Therefore, it is essential to monitor cardiovascular parameters during a standard colic workup, as a patient that is decompensating from a cardiovascular standpoint may be in need of surgical intervention, but is not a good surgical candidate.

Rectal palpation alone has the potential to provide a near definitive diagnosis of a small colon strangulating lesion caused by a pedunculated lipoma if the practitioner is able to detect a tight stricture within the rectum. However, if the strangulating lesion is causing distention of the large

intestine as well, or if it is simply beyond the reach of the practitioner, it may be difficult to confirm the presence of the strangulating lesion. The primary goal of rectal palpation is to determine if a surgical lesion is present by detection of displaced abdominal viscera, abnormal orientation of a tenial band, or edema or distention of viscera can often indicate a surgical lesion.[13]

Nasogastric intubation is a very useful tool in determining the status of the stomach and small intestine.[13] Strangulating lesions of the distal gastrointestinal tract often do not produce the amount of reflux that more proximal lesions do, at least in the acute phase of the disease, with only 30% of horses with strangulating lesions of the small colon displaying gastric reflux.[17] If the strangulation is prolonged however, distention of the ascending colon may occlude the flow of ingesta through the proximal small intestine, producing more gastric reflux.[17] Because rupture of the stomach is a risk for every colic patient where reflux is produced, a nasogastric tube should be placed in every patient that is being treated for colic to ensure that any distention of the stomach is relieved.[13]

Abdominal paracentesis is another tool that can be used to differentiate between medical or surgical management of colic. Abdominal paracentesis is not warranted in every case of colic, and is best suited for cases where there is mild pain, normal cardiovascular status, no net reflux from nasogastic intubation, and a normal rectal exam.[13] The inherent risk of enterocentesis when performing abdominal paracentesis prohibits the use of this diagnostic technique when the practitioner suspects compromised bowel may be present, as the bowel wall may not seal by the normal mechanisms if inadvertently punctured during the procedure.[13] Characteristics of the fluid obtained by abdominal paracentesis such as color, turbidity, the presence of red blood cells,

neutrophil count, protein level, or the presence of bacteria can all provide information regarding the health of the gastrointestinal tract.[13]

In summary, there is no definitive method of diagnosis for a strangulating pedunculated lipoma affecting the small colon or any part of the gastrointestinal tract. The colic workup must therefore be approached systematically in order to gain as much information as possible prior to making decisions on treatment. In the case of the mare described in this case report, the presence of unrelenting pain and discomfort, coupled with no resolution of the suspected pelvic flexure impaction warranted the decision to proceed with surgery.

Treatment and Management:

Surgical correction is the only viable treatment option for strangulating lesions involving any section of bowel. Surgical correction of strangulating lesions caused by pedunculated lipomas is not without risk however, as it has been reported that as many as 72% of horses with pedunculated lipomas will experience post-operative complications.[7] The same study identified negative prognostic indicators associated with post-operative survival time including heart rate greater than 80 beats per minute, abnormal color of abdominal fluid, pale mucous membranes, surgery in which intestinal resection is required, and inability to attain a mean arterial pressure of greater than or equal to 100 mmHg during surgery.[7] The author of this report questions whether these negative prognostic indicators are specific to lesions involving pedunculated lipomas requiring surgery, or if any cause of colic that must be corrected surgically and displays the before-mentioned negative prognostic indicators would be associated with a lower survival rate.

When small intestine is involved in a strangulating lesion by a pedunculated lipoma, it has been reported that these horses are at a greater risk for developing post-operative ileus in comparison

to other surgical diseases of the small intestine.[14] When a pedunculated lipoma creates a strangulating lesion of the small colon, resection and anastomosis is often needed if non-viable colon is present. The recommended technique for anastomosing small colon at present time is a two-layer technique, involving a full-thickness appositional pattern oversewn with a partial-thickness continuous inverting pattern.[17] This pattern has been reported to pose less risk for dehiscence, peritonitis, and adhesion formation, while also decreasing the risk of impaction at the anastomosis site.[17]

Characteristics unique to the small colon that may adversely affect the outcome of the surgery include high concentrations of collagenase in the small colon, large numbers of bacteria, particularly anaerobic bacteria, poor vascular supply compared to other sections of the gastrointestinal tract, and mechanical stress on the surgical site produced by formed fecal balls.[17] Aggressive use of broad spectrum antibiotics including potassium penicillin, gentamicin sulfate, and metronidazole are recommended when resection and anastomosis of the small colon is performed.[17] If the surgical procedure was without complication, healthy margins of small colon were obtained, and no abdominal contamination was incurred during the procedure, then 24 hours of antibiotic therapy may be adequate.[17] However, if compromised bowel is present or if there are any signs of peritonitis or contamination during the surgery, extended use of antibiotic therapy is imperative.[17] Consideration should be given to the use of antiendotoxin therapy such as polymyxin B or hyperimmune serum if the patient displayed any signs of endotoxemia prior to surgery.[17] In order to mitigate any mechanical damage to the resection and anastomosis site, evacuation of the large colon and post-operative feeding management should be performed to prevent fecal balls from causing dehiscence.[17] The overall prognosis of horses undergoing resection and anastomosis of the small colon is guardedfor discharge from the hospital and poor

for survival beyond 6 months .[15] Ultimately, the veterinary surgeon should take into consideration the details of each case individually to determine a prognosis prior to and during surgery for lesions involving strangulation of the small colon.

Case Management:

In the case of the mare described in this report, surgery was deemed the best option to create a favorable prognosis. In preparation for surgery, the mare was sedated with xylazine and butorphanol, followed by anesthetic induction with ketamine and diazepam, and general anesthesia maintained by isoflurane inhalant. The mare was placed in dorsal recumbency, her ventral abdomen was clipped and sterilely prepped, and sterile drapes were applied. An approximately 30 cm incision was made along the linea alba to facilitate visualization of the abdominal contents. Upon entry of the abdomen, the cecum was encountered immediately and was largely distended with gas. The gas was removed from the cecum, and the pelvic flexure impaction was easily identified and noted to be softening. An enterotomy was performed at the pelvic flexure and copious amounts of sterile saline was used to remove the impacted feed material. Upon complete removal of the impaction, the enterotomy site was lavaged with sterile and gentamicin saline, and closed in a one layer full-thickness appositional and oversewn with a partial thickness inverting pattern. The surface of the colon was thoroughly lavaged with sterile and gentamicin saline. Simultaneous to the pelvic flexure enterotomy, the small intestine was identified and explored from orad to aborad, beginning at the duodenum. Upon initial entry into the abdomen, a small 2cm x 2cm x 1cm free floating saponified lipoma was encountered and removed. Although no abnormalities were detected involving the small intestine, upon evaluation of the small colon, a pedunculated lipoma measuring 4cm x 5cm x 5cm was encountered strangulating a section of small colon measuring approximately 12 cm in length and

located approximately 36 cm proximal to the rectum. Using a hemostat and sharp transection of the stalk, the lipoma was removed and the previously strangulated section was inspected to determine viability. A noticeable color change began immediately after removing the strangulation from the section of colon. The strangulated section that had originally been dark purple to black improved in coloration upon removal of the strangulation, although it remained light purple in color with numerous areas of petechiae and ecchymosis visible on the serosal surface. Doppler ultrasound was used to evaluate blood flow to the previously strangulated section, and a satisfactory level of blood flow was determined to be present. Due to observation of a significant color change upon removal of the strangulation as well as detecting blood flow using the Doppler ultrasound, in conjunction with the aforementioned concerns regarding resection and anastomosis of the small colon, the decision was made not to perform a resection and anastomosis of the previously strangulated section. While exploring the remainder of the small colon, a third lipoma was found associated with the mesocolon measuring approximately 3cm x 3cm x 2cm. The stalk of this lipoma was ligated and it was removed. No other abnormalities were detected upon further abdominal exploration. The abdominal contents were verified to be in normal anatomical positions and the abdomen was thoroughly lavaged with sterile saline followed by application of gentamicin saline and carboxymethlycellulose to the abdomen prior to closure. A visceral retainer was used to protect the abdominal viscera during abdominal closure, and a three-layer closure of the linea alba, subcutaneous tissues, and skin was performed. Recovery from anesthesia was prolonged but smooth and uneventful.

Case Outcome:

Following recovery from anesthesia, the mare remained hospitalized for 7 days. During the next week, her appetite and mentation improved. The mare was maintained on intravenous potassium

penicillin (22,000 IU/kg q6h), gentamicin sulfate (6.6 mg/kg q24h), polymyxin B (5,000 U/kg in 1 L of saline q8h), DMSO (1 pint in 5 L of LRS q24h), and flunixin meglumine (1.1 mg/kg q12h) for the first 48 hours following surgery. During the post-operative period, the mare remained on intravenous plasmalyte fluids, intravenous 2% lidocaine constant rate infusion, and oral ranitidine (7 mg/kg q8h) and sucralfate (25 mg/kg q8h). Injectable antibiotics were discontinued after 48 hours and oral chloramphenicol (50mg/kg q8h) was initiated. DMSO was discontinued 72 hours following surgery, and at 96 hours, all injectable medications including intravenous fluids had been discontinued, and flunixin meglumine was administered orally. Throughout the week, the mare's appetite improved and at approximately 48 hours following surgery, the mare produced feces. Her appetite progressively increased, and she began passing feces regularly. On March 26, 2017, seven days following surgery, the mare's owners were visiting when it was noted that she appeared uncomfortable, was looking at her sides, and attempting to lie down. She was disinterested in grazing when offered the opportunity outside, and her heart rate was noted to be elevated in her stall. Upon passage of a nasogastric tube, a large amount of feed material was evacuated from her stomach. Rectal palpation at this time revealed no significant abnormalities. The nasogastric tube was left in place and the mare was started on intravenous fluids and lidocaine once again. Physical exam revealed a toxic line along her gums, and cool extremities. Multiple abdominal ultrasounds were performed, and a large amount of abdominal fluid was discovered. When the fluid was sampled via sterile preparation and abdominal paracentesis, a large amount of serosanguinous fluid was retrieved. Analysis of the fluid revealed a total nucleated cell count of 89,000/µL and a protein content of 6.4 g/dl (normal nucleated cell count = less than 5,000/µL; normal protein content is less than 2.5 g/dl)[13]. Lactate was noted to be markedly elevated at 16.3 mmol/L. Cytological analysis of the fluid

revealed numerous degenerate neutrophils with phagocytosed rods. Over the course of the evening, the mare became increasingly more uncomfortable and it became difficult to control her pain through medication. Due to a poor prognosis, her owners elected to euthanize.

The mare was presented for necropsy to the diagnostic laboratory service at MSU CVM following euthanasia. Gross necropsy revealed peritonitis characterized by a large amount of exudative, yellow fluid within the abdomen. Multiple areas of hemorrhage within the serosa of the gastrointestinal tract were observed. An approximately 3 feet long section of ileum was dark purple in color and located between the ventral colon and cecum with sharp margins between healthy and congested tissue readily apparent. A focal small colon torsion was present and tightly adhered to the enterotomy site at the pelvic flexure by fibrinous attachments. The diagnosis as provided by Dr. Ann McBride of the MSU CVM Diagnostic Laboratory Service was a multifocal small colon and ileal venous infarction with congestion and extensive fibrinous adhesions, along with peritonitis and a small colon torsion that was adhered to the pelvic flexure enterotomy site.

In conclusion, it is likely that the strangulated section of small colon was unable to fully recover from its vascular insult despite removal of the pedunculated lipoma, although it appeared viable at the time of surgery. The congestion that resulted from the strangulation, coupled with the decompensated bowel likely caused the thromboembolic event evident on necropsy. The source of the peritonitis was most likely the section of strangulated small colon that became necrotic following surgery, since the enterotomy site at the pelvic flexure was observed to be intact on necropsy.

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