Small Intestinal Strangulating Lipoma

Brandon D. Pate

Clinicopathologic Conference

Mississippi State University

Class of 2019

CPC Advisor: Cathleen Mochal-King, DVM, MS, DACVS-LA

Introduction:

Colic is an important cause of morbidity and mortality in the equine population. It is a common emergency in equine practice with approximately 4 out of every 100 horses having an episode of colic every year (2). Of the horses that are evaluated by a veterinarian in private practice, approximately 7% to 10% have a lesion that requires it to be surgical corrected (2). The fatality rate was 11%, and 1.4% of colic events resulted in surgery (2). Statistics such as these clearly illustrate the importance of veterinarians being knowledgeable regarding the causes, workup and treatment of colics, and perhaps most importantly, when to refer these colics.

Colic as a term used to refer to clinical signs of abdominal pain. This descriptor is non-specific, and these signs of pain can be caused by a number of different things, both within the gastrointestinal tract, as well as elsewhere such as the reproductive tract or urinary tract. The most common, however, is primary gastrointestinal disease, which will thus be the focus of this report.

Equine practitioners often classify gastrointestinal causes of colic into two pathophysiologic categories- a true obstruction or a functional obstruction. With a true obstruction there is something physically preventing a normal flow of ingesta along the tract. The could be an obstruction within the lumen (intraluminal) within the wall of the intestinal tract (intramural), or external to the wall of the intestinal tract (extraluminal). With a functional obstruction, in comparison, there is no physical obstruction but rather a motility disorder, impeding the flow of ingesta. This disordered motility is referred to as ileus. Ileus will be discussed at length below due to its relevance in this case.

The classification of a true obstruction can be further broken down into strangulating and non-strangulating lesions. With the former, something has disrupted the vascular integrity of that

segment of the gastrointestinal tract. Regardless of the etiology, strangulating obstructions will eventually lead to the loss of viability in the effected section of bowel. With a non-strangulating lesion, the obstruction does not interfere with the vascular supply or return and thus are less likely to effect bowel viability. Determining whether a horse is suffering from a strangulating versus non-strangulating lesion is of critical importance. These cases need to be identified earlier rather than later because these are emergency surgical cases that will not resolve with medical therapy alone. As approximately 58-85% of small intestinal colics are caused by a strangulating lesion, including them on the differential diagnosis list is critical (2).

History:

Hartz is a 17-year-old Clydesdale Gelding who presented to the Mississippi State University, College of Veterinary Medicine (MSU-CVM)'s Equine Surgery service for colic on the night of December 10, 2017. The owners reported finding him down in the pasture on that morning. Hartz was then trailered to the referring veterinarian where an IV catheter was placed, and fluids were given along with pain medication. Hartz continued to worsen despite therapy and was then referred to the MSU-CVM Equine service for further evaluation. He was used as a Fox Hunting horse and spent most of his time out on pasture. There were no recent changes in feed or exercise. His typical diet consisted of a half cup of Equine Senior and a half cup of Equine Strategy pelleted feed and bermuda grass hay. He was last dewormed a few months ago, prior to arrival, and he was up to date on vaccinations and equine infectious anemia testing.

Presentation and Initial Examination:

On presentation, Hartz weighed 1,420 pounds with a body condition score of 5/9 (ideal). He appeared quiet, depressed, and dull. His initial exam revealed a normal rectal temperature of 98.3 degrees Fahrenheit, mild tachycardia, with a pulse of 64 beats per minute with no murmurs or

arrhythmias and mild tachypnea with a respiratory rate of 32 breaths per minute with no abnormal lung sounds heard. His mucus membranes were tacky to the touch with a normal capillary refill time of 2 seconds. Hartz was estimated to be 5% dehydrated. A gastrointestinal auscultation revealed reduced GI sounds in all four quadrants. Nasogastric intubation was performed and no reflux was obtained. Digital pulses palpated normally in all four limbs. Prior to further workup, 200 mg of xylazine and 140 mg of buscopan (N-butylscopolammonium bromide) were administered intravenously. Subsequent rectal palpation revealed multiple loops of dilated small intestine. An abdominal ultrasound examination was performed and confirmed amotility in the small intestine. Ventrally, a moderate amount of free abdominal fluid was noted and there were several distended loops of small intestine with an abnormal wall thickness measuring up to as much as 14.5mm (normal <3mm) were seen. The right side imaged normally. A stall-side lactate meter was used to quickly assess systemic tissue perfusion. Hartz systemic lactate was 3.4mmol/L (normal being <2.0), this is consistent with hypovolemia or secondary to intestinal ischemia.

Diagnostic Workup:

The key to performing a good diagnostic work up is being meticulous with each step of the exam. The first step of working up any colic is getting a good history and performing a physical exam. The history is often variable for colic cases and consists of having an acute onset of abdominal pain that is persistent. Additional things that need to be evaluated for every colic are are feed or feed management changes, exercise changes or routine changes, deworming protocol, and finally, any previous episodes of colic (7). These are major risk factors to cause colic in reported horses. The physical exam findings can be great help with respect to distinguish strangulating from non-strangulating lesions. For strangulating lesions, the patients usually have

moderate to severe abdominal pain. Pain is a good indicator to clinicians whether to refer this case for surgery. If the horse is not responding to analgesic medication and medical therapy, then this is an important factor pointing to a strangulating lesion. Initially, they will be responsive to analgesics, but the response diminishes as the disease progresses. They will be tachycardic, with a heart rate usually greater than 60 beats/min. Their temperature is within normal limits acutely, but can change depending on the timing of the lesion. Cardiovascular status diminishes with progression of diseases, and signs of dehydration can occur rapidly in patients. As the disease progresses, horses may become depressed and show progressive signs of endotoxemia including tachycardia, tachypnea, congested mucous membranes, and a prolonged capillary refill time (11). After a thorough physical exam is completed, an initial database should be run on the patient. This includes rectal examination, nasogastric reflux, abdominocentesis, ultrasonography, and a lactate should be run to examine perfusion parameters (7). The rectal examination of a small intestinal strangulating lesions may reveal multiple loops of tight dilated small intestinal loops throughout the abdominal cavity (7). As the disease progresses, the size and the loops will slowly increase. The cause of the strangulating lesion is rarely palpated on rectal. Late in the disease, it may be possible to palpate thickened turgid small intestine consistent with congestion and devitalization.

Nasogastric reflux should be performed apart of the initial database as well. This is a valuable tool in order to determine whether the lesion is strangulating vs. non-strangulating. It will also help to tell you the location of the colic or lesion. The amount of reflux increases as the time progresses and may be absent early in the disease. Larger amounts of reflux generally point to the small intestine, whereas variable amounts tell you it is more likely the large intestines. The pH of the fluid is also important. The more alkaline the pH, the more likely it is in the small

intestine. The smell of the fluid is valuable as well. The strangulating lesions typically have a malodorous smell to them.

An abdominocentesis should be performed on these horses as well. This is the most helpful diagnostic tool when differentiating strangulating vs. non-strangulating. Strangulating lesions have an increase in total protein, white blood cells, and red blood cells to the same degree (7). In other words, the total nucleated cell counts will be higher with strangulating lesions. In reality, there is a bit of time difference in the way these three parameters change. These changes are due to a compromise in the gastrointestinal tract wall and circulation. This is the major factor responsible for the difference between a strangulating lesion and a non-strangulating lesion.

Abdominal ultrasounds should also be included in a thorough diagnostic work up of a colic. It is an excellent tool for evaluating the gastrointestinal system in real time. In strangulating lesions of the small intestines, you will see multiple loops of distended intestinal loops that are not motile. You will also be able to identify free fluid within the abdominal cavity.

Pathophysiology:

A strangulating lipoma is a smooth-walled, benign fat tumor of variable size. It is suspended by mesenteric fibrovascular pedicle of variable length within the abdomen (5). Lipomas cause colic in horses when the stalk of the lipoma wraps around the small intestine and mesentery as it gets longer (5). As the loop tightens it prevents ingesta and blood flow from passing into the remaining intestine causing a strangulating of the entrapped intestine (5). This is primarily a disease of older horses, with the mean presenting age of 14 to 19.2 years (6). Geldings are at higher risk than mares and stallions (5). Ponies, Arabians, Saddlebreds, and Quarter Horses are reported to be at higher risk than Thoroughbreds (5). Ponies alone have increased odds of strangulation of the small intestine by lipomas above other breeds (3). The disease was once

thought to be associated with obesity, but it is now thought that there is an increased incidence seen in aged geldings because of an alteration in fat deposition caused by endocrine abnormalities such as insulin resistance, Cushing's disease, and metabolic syndrome (5).

The other pathophysiology that I will focus on is post-operative ileus. Approximately 20% of post-operatively surgical cases develop ileus (9). This is associated with 40% of post op deaths in horses treated for colic (7). Ileus is impairment of normal propulsive bowel motility (9). This is important to talk about when discussing surgical procedures in the equine patient. Ileus is a cause and effect type of situation, meaning the ileus can be the underlying cause of the colic. But in this case, it is the result of the colic. So, when you have a horse that starts colicing, they are generally going to have a lot of underlying issues that worsens the colic. This is the reason you need to look at ileus as a cause and effect type of event. Ileus also needs to be discussed in degrees as well. This is not an all or none thing because ileus occurs in grades such as mild, moderate, and severe. This is why we characterize it as an impairment of propulsive motility.

The relevance of ileus in this case is that ileus tends to develop from many different causes and is very common post-operatively. These include but are not limited to pain, distention of gastrointestinal system, decreased GI perfusion, electrolyte abnormalities, inflammation of the peritoneum, endotoxemia, drugs, and post-operatively (1). Pain is usually the initiator of the ileus, but with a lipoma it is secondary. It decreases peristalsis because it is a sympathetic and parasympathetic tone issue. When horses are painful, they increase their sympathetic nervous system, which results in a decrease in motility. The other cause of ileus that I will focus on is post-operative ileus. There are two current theories on why this occurs post-operatively. First, it is caused by simple manipulation of the GI tract during surgery. This is thought to cause activation of mechanoreceptors and nociceptors in the GI tract (4). Activation of these receptors

in the GI tract are thought to stimulate adrenergic pathways which ultimately inhibits smooth muscle activity (12). This occurs with just anesthetizing the animal, but it should be short lived and is an uncomplicated post-operative ileus.

The other type of ileus is what we refer to as late stage post-operative ileus. This is the ileus that we usually are referring to when we talk about post-operative ileus in the horse. It is adynamic ileus due to inflammatory changes in circular and longitudinal muscle layers in the gut (1). These inflammatory changes lead to activation of local macrophages that result in nitric oxide and prostaglandin synthesis (12). These local cells lead to a leukocyte infiltration within the muscle layers and impairs the motility and initiates inhibitory motor reflexes in the GI system (12). Inflammatory mechanisms are likely the major underlying cause of ileus regardless of insult.

Treatment and Management:

As mentioned in the introduction, these strangulating lesions will not resolve with medical therapy alone. Therefore, surgical correction is the mainstay treatment for this disease. This consists of performing an exploratory celiotomy. Next, you have to identify the inciting problem and correct it. In cases of strangulating lipomas, you have to locate the lipoma and transect it. Following this, you will have to determine the vascular integrity of the small intestines, and whether the tissue is devitalized. The devitalized tissue will need to be transected, and a resection and anastomosis will need to be performed.

The surgery is just the beginning of the battle. Complications after jejunal resection include leakage at the anastomotic site with subsequent local or diffuse peritonitis, adhesions, stricture at the anatomic site, and post-operative ileus (6). Adhesions are the most common cause of death in these after small intestinal resection (6). The most difficult part of these cases is reestablishing gastrointestinal motility. It has been reported that 72% of horses with pedunculated lipoma will

experience post-operative complications (8). When the 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 (5).

The main goal of treating the ileus is to address the primary problem. In this case, the strangulating lipoma and dead gut was the problem. This is also accomplished by relieving the pain, treating the shock and dehydration and correcting the metabolic alterations. Motility enhancers are also very useful in the treatment of ileus. This is where medications come in to help with these cases. In the proximal gastrointestinal tract, lidocaine and metoclopramide are the two mainstays of motility enhancers (10). The lidocaine is important in these cases because it not only increases the motility, but it has anti-inflammatory effects as well. It also helps with systemic analgesia in these patients. The prognosis for long-term survival for horses after resection and anastomosis is guarded (8). But, if these horses are caught early, small intestinal strangulating lesions can have 80% prognosis for survival (8).

Case Management:

Over the course of the night, his heart rate increased, and he began pawing and turning back towards his flank, which were signs consistent with severe colic. An abdominocentesis was performed and a serosanguinous fluid was produced. Based on the clinical picture upon presentation, most notably the evidence of significant small intestinal distension in conjunction with an abnormal abdominocentesis, the recommendation was then made to pursue an exploratory celiotomy.

The owners consented to surgery and were apprised of the risks of anesthesia, colic surgery and recovery as well as the complications commonly seen in association with strangulating lesions

(i.e. need for resection, post-operative ileus, endotoxemia, colitis, laminitis, etc.). Hartz received a 1 L bolus of hypertonic saline followed by 5L of isotonic fluids to replenish his circulating volume. He also received pre-operative broad-spectrum antimicrobial therapy (Enrofloxacin and Potassium Penicillin) in order to reduce the incidence of infection. He was also given flunixin meglumine pre-operatively for its analgesic and anti-inflammatory properties. In preparation for surgery, General anesthesia was then maintained using isoflurane inhalant. The patient was then sterilely draped, and an approximately 30 cm incision was made along the linea alba entering the abdominal cavity. Immediately upon entering the peritoneal cavity, several loops of dilated small intestine were encountered, and the cecum was not readily identifiable. The small intestine was diffusely dilated with gas and contained fluid ingesta. Upon further exploration, an approximately 12-foot-long, firm, congested and edematous portion of jejunum was identified. Further investigation identified the bowel was strangulated secondary to incarceration by a pedunculated lipoma. Upon relieving the strangulation, the bowel retained its necrotic color and no mesenteric pulse was identified. Based on these signs, the section of jejunum was deemed devitalized. A jejunojejunostomy was then pursued.

Once the jejunojejunostomy was completed, the site was lavaged and meticulously checked for contamination. Following completion of the resection and anastomosis, the entire length of the intra-abdominal gastrointestinal tract was exteriorized and explored. No additional lipomas were found and the resection site was assessed for any sign of leakage. Prior to closure, the abdomen was lavaged with several liters of saline followed by an additional liter of penicillin and gentamicin saline. A large visceral retainer was used to facilitate closure of the linea. The abdomen was then closed in a routine three-layer fashion. A bandage was stapled to the abdomen to cover the incision site during recovery. Recovery from anesthesia was uneventful.

Case Outcome:

Following recovering from anesthesia, Hartz remained hospitalized for 8 days. Hartz's condition failed to improve following the small intestinal resection and anastomosis. Hartz was maintained initially on intravenous potassium penicillin (22,000 IU/kg q6h), enrofloxacin (5.5 mg/kg q24h), polymyxin B (5,000 U/kg in 1 L of saline q8h), flunixin meglumine (1.1 mg/kg q12h), and DMSO (1 pint in 5 L of LRS q24h) following surgery. Hartz remained on intravenous LRS fluids (supplemented with calcium gluconate), intravenous 2% lidocaine constant rate infusion, intravenous metoclopramide constant rate infusion, oral ranitidine (7mg/kg q8h), sucralfate (25mg/kg q8h), and aspirin (25mg/kg q24h). Heparin (40 U/kg q8h) was administered subcutaneously. Hartz was also started on cryotherapy for laminar support in order to reduce the risk of post-operative laminitis. Over the course of 48 hours, he had several episodes of increased discomfort and agitation indicated by an elevated heart rate, pawing, flank watching, and attempting to roll. An abdominal ultrasound was performed, and small intestinal ileus was evident by dilated small intestines. His heart rate maintained between 60-64 beats/min consistently. Refeeding was attempted on the third day of his hospital stay, however, the following day, he was refluxing larger volumes, indicating that ingesta was not passing through the small intestine.

On day 5 post-operatively, Hartz seemed as if he was improving. He appeared brighter and was offered feed and water. However, he did continue to spontaneously reflux gastrointestinal fluid in excess of normal amounts. His lidocaine constant rate infusion was discontinued, but he was still receiving the metoclopramide constant rate infusion for motility. His pain was being maintained with flunixin meglumine. Hartz was started on additional medical therapy in order to induce small intestinal motility (acepromazine 0.01 mg/kg q4h) as well as receiving hypertonic

saline and hetastarch to reduce edema in the small intestine. On day 6 post-operatively, Hartz still seemed as if he was slowly improving in attitude, but his spontaneous reflux was not. He was then restarted on a lidocaine constant rate infusion, DMSO, and given another bolus of hetastarch and hypertonic saline.

On day 7 post-operatively, Hartz started to take a turn for the worst. He appeared dull and depressed. He was laying down for prolonged periods of time. He was refluxing large volumes of gastrointestinal contents and his heart rate and temperature increased. There was a mucoid discharge coming from both nostrils again, and he was noted to have increased digital pulses in all four limbs indicating a possible laminitic event. He was then started on chloramphenicol (50 mg/kg q6h) given the prolonged increased in his temperature to combat bacterial translocation. A single dose of neostigmine (0.01 mg/kg) was also added to stimulate gastrointestinal motility and magnesium sulfate was added to his fluids.

Given Hartz's continued small intestinal ileus and non-response to prokinetics, the options of continued medical management or repeat laparotomy were discussed with the owners prior to the first weekend post-op. Given all he had been through, the owners elected to continue with medical management over the weekend to see if he would improve. During this time, he was started on parenteral nutrition to meet a portion of his caloric needs. However, on his 8th day post-op, Hartz was noted to be very dull and depressed and refluxing the largest amounts seen during his hospitalization (87.5L).

On Monday, December 18, 2017, due to his failure to improve over the weekend, the owners elected for humane euthanasia. Hartz was sedated and subsequently euthanized with an overdose of pentobarbital administered intravenously. He was subsequently submitted for necropsy in an attempt to identify the cause of his continued ileus.

Necropsy exam revealed that the jejunojejunostomy site was intact and did not appear to leak following surgery. The primary findings were of some jejunal discoloration on either side of the resection site, some evidence of hemorrhage and necrosis in the associated mesentery and fibrinous adhesions throughout the small intestine. This was likely due to the initial colic insult, surgical trauma and prolonged ileus.

Conclusion:

In conclusion, strangulating lesions in the equine, and specifically strangulating lipomas, are common in geriatric horses. These lesions are life threatening and will require surgery in order to alleviate the colic. It is better to recognize these types of colic earlier in the course of the disease and deal with them accordingly in order to prevent post-operative complications.

References

1. Blikslager AT. Ischemic Disorders of the Intestinal Tract. In: Reed SM, Bayly WM, Sellon DC, eds. Equine Internal Medicine. 3rd ed. St. Louis: Saunders Elsivier, 2010; 876-882
2. Cook VL, Hassel DM. Evaluation of the Colic in Horses: Decision for Referral. Veterinary Clinics of North America: Equine Practice. 2014; Vol 30, Issue 2, 383-398
3. Dunkel B, Buonpane A, Chang YM. Differences in gastrointestinal lesions in different horse types. Vet Rec. 2017 Sep 16; 181 (11): 291. Doi: 10.1136/vr.104098. Epub 2017 Aug 5.
4. Edwards GB, Proudman CJ. An analysis of 75 cases of intestinal obstruction caused by peduculated lipomas. Equine Vet. Journal 1994; 26:18-21.
5. Eggleston R. Small Intestine: Strangulating Lipoma. Clinical Veterinary Advisor. 2012; 554.
6. Fessler FF. End to End Anastomosis: jejunal resection and anastomosis. In Adams SB, Fessler FF. eds. Atlas of Equine Surgery. St Louis: Saunders Elsivier, 2000; 97-102.
7. Freeman DE, Schaeffer DJ. Age Distributions of horses with strangulation of the small intestine by a lipoma or in an epiploic foramen: 46 cases (1994-2000). In J AM Vet Med Assoc. 2001; Jul 1;219 (1):87-9.
8. French NP, Smith J, Edwards GB, Proudman CJ. Equine surgical colic: risk factors for postoperative complications. Equine Vet. Journal. 2002; 34:444-449.
9. Gonda KC. Byars TD. Equine History, Physical Examination, and Records. In: Smith BP, eds. Large Animal Internal Medicine. 4th ed. Dt. Louis: Saunders Elsevier, 2014.
10. Rakestraw PC, Hardy J. Small Intestine. In: JA, Stick JA, eds. Equine Surgery. 4th ed. St. Louis: Saunders Elsevier, 2012
11. Rose JR; Hodgeson DR. Alimentary System. In: Ethell MT, Dart AJ, Hodgson DR, Rose RJ, eds. Manual of Equine Practice. 2nd ed. St. Louis: Saundaers Elsevier, 273-341.
12. Sanchez CL. Examination for disorders of the gastrointestinal tract. In: Reed SM, Bayly WM, Sellon DC, eds. Equine Internal Medicine 3rd ed. St. Louis: Saunders Elsevier, 2010; 777-784
13. Southwood LL. Acute Abdomen. Clinical techniques in Equine Practice. 2006; 5:112-1266
14. Zachary JF. Alimentary System and the Peritoneum, Omentum, Mesentery, and Peritoneal Cavity. In Zachary JF. Pathologic Basis of veterinary Disease. 6th ed. St. Louis: Sauders Elsevier