Strangulating Intestinal Obstructions in Four Captive Elephants (*Elephas maximus* and *Loxodonta africana*)

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STRANGULATING INTESTINAL OBSTRUCTIONS IN FOUR CAPTIVE ELEPHANTS (*ELEPHAS MAXIMUS* AND *LOXODONTA AFRICANA*)

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Abstract: Three captive-born (5-day-old, 8-day-old, and 4-yr-old) Asian elephants (*Elephas maximus*) and one captive-born 22-yr-old African elephant (*Loxodonta africana*) from three private elephant facilities and one zoo in the United States presented with depression, anorexia, and tachycardia as well as gastrointestinal signs of disease including abdominal distention, decreased borborygmi, tenesmus, hematochezia, or diarrhea. All elephants showed some evidence of discomfort including agitation, vocalization, or postural changes. One animal had abnormal rectal findings. Nonmotile bowel loops were seen on transabdominal ultrasound in another case. Duration of signs ranged from 6 to 36 hr. All elephants received analgesics and were given oral or rectal fluids. Other treatments included warm-water enemas or walking. One elephant underwent exploratory celiotomy. Three animals died, and the elephant taken to surgery was euthanized prior to anesthetic recovery. At necropsy, all animals had severe, strangulating intestinal lesions.

Key words: Elephant, *Elephas maximus*, *Loxodonta africana*, colic, intestinal strangulation, surgery.

INTRODUCTION

Although colic has been reported with some frequency in elephants, the majority of cases have been associated with infectious organisms including *Salmonella* spp.,[12,30,37] *Clostridium* spp.,[5,10,15] and elephant endotheliotropic herpesvirus (EEHV).[21] In range countries, nematodes and tapeworms are causes of impaction colics.[29,39] Reported noninfectious causes of colic in elephants include cholelithiasis,[2] plant toxicities,[36] malabsorption,[25] sand and foreign body ingestion,[24] and feed impactions.[40]

Strangulating intestinal lesions constrict both vasculature and intestinal lumen diameter. As this occurs, the gut becomes distended, motility alters drastically, and ischemia results leading to metabolic derangements, dehydration, and circulatory shock.[31] The degree of pain and the presenting signs can be highly variable in domestic species[9,22,28,32] and the etiology is often unknown. If damage is not extensive, surgical intervention can be curative. In this report, four different strangulating lesions in three juvenile Asian elephants (*Elephas maximus*) and one young adult African elephant (*Loxodonta africana*) are documented. To the authors' knowledge, this is the first report of strangulating intestinal lesions in elephants.

CASE REPORTS

Case 1 was a 5-day-old, 143-kg male Asian elephant (*E. maximus*) born at a zoo in New York who was evaluated for hematochezia and respiratory abnormalities following an episode of near-drowning. The calf was normal at birth and was

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nursing well but developed a patent urachus on the third day of life which was successfully treated with application of silver nitrate sticks (Grafco silver nitrate applicators, GF Health Products, Inc., Atlanta, Georgia 30360, USA). For the next 36 hr, the calf acted normally, nursed well, and passed normal urine and feces. On day 5, the calf fell into a pool in the enclosure while playing. Although rescued by keepers in less than 2 min, the total amount of time the calf was submerged is unclear. Following rescue, the calf was pale, agitated, dyspneic, and tachycardic with a heart rate of 80 beats/min (reference heart rate, 39 beats/min)[27] and was unable to stand. Treatment included nasal oxygen (10 L/min), furosemide (Furoject, Boehringer Ingelheim Vetmedica, Inc., St Joseph, Missouri 64506, USA; 0.83 mg/kg i.m.), dexamethasone (Dexaject, Phoenix Scientific, Inc., St. Joseph, Missouri 64503, USA; 0.05 mg/kg i.v.), and ceftiofur (Naxcel, Pharmacia & Upjohn, Kalamazoo, Michigan 49001, USA; 1.2 mg/kg i.m.). The dyspnea improved although the calf remained tachypneic with a respiratory rate of 44 breaths/min (reference respiratory rate 10–12 breaths/min).[8] Over the next 3 hr, the calf became brighter and stood to nurse, although it began to defecate small amounts of frank blood and mucus. Six hours after the near-drowning, tachycardia worsened to 140 beats/min; the calf also became febrile with a temperature of 38.7°C (reference temperature 35.9°C)[7] and started to vocalize. Eight hours after falling into the water, the calf collapsed and arrested. Resuscitation efforts were unsuccessful. At necropsy, a 360° clockwise torsion around the root of the mesentery was found incorporating 80% of the small intestine, the cecum, and the entire large colon. Affected intestines were dark red to black and gas-distended with a necrotic mucosal surface. The mesenteric vessels were markedly engorged with multiple blood clots at the root of the mesentery. The abdominal cavity contained approximately 3 L of serosanguinous fluid. The urachus was closed. No evidence of aspiration or other lung changes was noted. Histopathology showed multifocal large areas of hemorrhage in the small intestine characterized by marked diffuse congestion and moderate edema. The mucosal epithelium had necrosed and sloughed leaving only lamina propria remaining. Polymerase chain reaction (PCR) for EEHV in whole blood was negative.

Case 2 was an 8-day-old, 162-kg female Asian elephant, born at a private facility in Florida, who was evaluated for decreased appetite and possible discomfort. The calf appeared normal at birth and was nursing well and gaining 1 kg daily. On day 8 of life, the calf's nursing activity decreased and the calf appeared restless. She repeatedly ran to her mother, and placed her mouth on the mammary gland without nursing. Amount and frequency of defecation were normal. She was tachycardic with a heart rate of 85 beats/min. Borborygmi were normal. Fecal examination was negative for protozoans and nematodes.

Flunixin meglumine (Banamine Injectible Solution, Schering-Plough, Pointe Claire, Quebec, Canada H9R 1B4; 0.5 mg/kg i.m. b.i.d.) was administered, which increased the calf's comfort level. Although the calf resumed nursing, its heart rate remained elevated at 60 beats/min. Twelve hours later, the calf stopped nursing again and was observed straining to defecate, occasionally passing small amounts of clear mucus with frank blood. Tachycardia worsened to 95 beats/min. Borborygmi were absent. Transabdominal ultrasound, using a 5-2 MHz curved transducer (Sonosite Titan, Bothell, Washington 98021, USA), identified a few mildly dilated loops of small intestine with decreased motility. The leukogram showed a left shift (3% band cells; reference range 0%), with a leukocytosis ($24 \times 10^3/\mu l$; reference range $10–18 \times 10^3/\mu l$). Hematocrit, plasma biochemistry, and urinalysis were normal. A warm-water enema was administered without effect. The calf was also given 4 L of plain water per rectum for hydration. Ceftiofur (2.2 mg/kg i.m. b.i.d.) was initiated because of the left shift. In the afternoon, the calf showed additional signs of discomfort including lying down and rising repeatedly. A single dose of butorphanol (Torbugesic Injectable, Fort Dodge Animal Health, Fort Dodge, Iowa 50501, USA; 0.1 mg/kg i.m.) failed to improve her comfort level. A second ultrasound, performed 4 hr after the first, showed numerous loops of distended, nonmotile bowel.

Because of the elephant's continued deterioration and abnormal ultrasound findings, an exploratory ventral celiotomy was performed. Following sedation with a combination of xylazine (Xylazine HCl Injection, IVX Animal Health, Inc., St. Joseph, Missouri 64503, USA; 0.06 mg/kg i.m.) and butorphanol (0.01 mg/kg i.m.), anesthesia was induced with a mixture of ketamine (Ketaset, Fort Dodge Animal Health; 0.5 mg/kg i.m.) and xylazine (0.06 mg/kg i.m.). After intubation, anesthesia was maintained with isoflurane (Aerrane, Baxter Healthcare Corporation, Deerfield, Illinois 60015, USA). At surgery, a 17.6-cm long,

1.5-cm wide focal adhesion was identified in the distal small intestine constricting the large colon near the cecocolic junction. Several smaller adhesions scattered throughout the jejunum caused additional bowel strangulation. All adhesions originated from the antimesenteric large colon just distal to the cecum. The strangulated colon was friable with areas of acute hemorrhage. No evidence of a perforating foreign body could be found. The intestinal lesions were deemed nonresectable due to their size, location, and severity and the animal was euthanized with pentobarbital-sodium (Beuthanasia-D, Schering-Plough Animal Health Corporation; 40 ml i.v.) prior to recovery from anesthesia. PCR for EEHV in whole blood was negative. Fecal culture for *Salmonella* spp. was also negative. Histologic examination of the intestinal mucosa suggested that the adhesions, which were composed of fibrous connective tissue, had been present for several weeks, including antepartum, and represented a congenital abnormality of unknown cause. In addition, the intestinal submucosa was distorted by edema and disorganized fibrous tissue. Deep to the submucosa, marked irregularity and disorganization of the external muscular layers were seen, with large accumulations of extravasated erythrocytes, fibrin, disorganized fibrous tissue, and occasional lymphoid follicles. Discontinuity of smooth muscle layers was associated with severe hemorrhage. The adjacent mesentery contained numerous immature lymphoid follicles, extravasated erythrocytes, fibrin, and disorganized fibrous connective tissue.

Case 3 was an 865 kg, 4-yr-old male Asian elephant from a private facility in California who was examined for anorexia and diarrhea of a few hours duration. This animal had been surgically castrated without complications 6 mo earlier and had no history of medical problems. His normal diet of oat hay with some grass hay was unchanged. Treatment consisted of oral fluids and mineral oil (amounts not recorded) and flunixin meglumine (1.75 mg/kg i.m. b.i.d.). Over the course of the day, this male showed mild signs of abdominal discomfort, including stretching and attempting to lie down, as well as tenesmus. Although he refused feed, he continued to drink well. The next day, he became progressively tachycardic (heart rate, 92 beats/min), and abdominal distention was noted. A large gas pocket located distally on the right side of the abdomen could be palpated per rectum. Thirty-six hours after initial presentation, the elephant collapsed and died. At necropsy, a mesenteric rent incarcerating 1.5 m of distal small intestine was found along with 25 L of serosanguinous fluid in the abdomen. The wall of the incarcerated intestine was dark red, thick, and friable and had ruptured in several places, causing content leakage and a severe septic peritonitis. Fibrin was adhered to most of the abdominal serosa, and all abdominal organs had petechial and echymotic hemorrhages. Histopathology showed severe transmural congestion, hemorrhage, and edema in the incarcerated loop of intestine. The mucosa was characterized by diffuse, severe necrosis and invasion of all layers by mixed bacterial flora. There was diffuse neutrophilic infiltration of the lamina propria and submucosa, and the blood vessels of these two layers were congested, engorged with marginated neutrophils, and occluded by fibrin thrombi. These changes were segmental, and there was a well-delineated line of demarcation between the affected intestine and the more-normal adjacent areas. Fecal cultures were negative for *Salmonella* spp., *Shigella* spp., and *Campylobacter* spp. Fecal examination was negative for nematodes and protozoans.

Case 4 was a 22-yr-old, 2,950-kg female African elephant (*L. africana*) owned by a private facility in California who was examined because of anorexia and mucoid diarrhea starting shortly after she had grazed on a small amount of lush forage in an area adjoining her usual pasture. This female's regular diet consisted of grass hay and elephant pellets and she had no prior medical problems. Although she refused food, she drank normal amounts of water. Bismuth salicylate (Bismusol, First Priority, Inc., Elgin, Illinois 60123, USA; amount not recorded) was administered orally before its keepers commenced walking her around the compound. Over the next few hours, the female developed abdominal distention and would repeatedly lie down and stretch. Borborygmi and heart rate were difficult to assess because of the animal's size. The elephant was given a single dose of flunixin meglumine (1.0 mg/kg i.m.) but refused further oral medication. Within 6 hr of initial presentation, the animal collapsed and died. At necropsy, a 360° torsion around the root of the mesentery, involving the entire large colon, was identified. The bowel was severely congested and hemorrhagic throughout its length. Areas of the intestinal wall were thickened up to 2.5 cm. Histopathology showed submucosal edema and multifocal hemorrhages throughout the colon wall, and the colonic vasculature was engorged

with blood. Small venules in the colon were characterized by fibrin and neutrophils adherent to the endothelium. Cultures for *Salmonella* spp. were negative.

DISCUSSION

The elephant is a monogastric hindgut fermenting species. The gastrointestinal tract consists of a simple stomach, small intestine, cecum, large colon, and rectum. The small intestine is short compared to other herbivores, particularly in African elephants. Reported lengths of the small intestine are 9.2 m in *L. africana* and 20.2 m in *E. maximus*. The large colon has been measured at 9.4 m in *L. africana* and 11.8 m in *E. maximus*.[13] The large intestine maintains a single diameter throughout its length and is without sacculations.

In the wild, elephants are both grazers and browsers, consuming leaves, grass, fruit, bark, and entire small trees and shrubs. Proportions of these items can vary tremendously.[6,38] In North America, hay is the primary dietary component for elephants along with varying amounts of browse, commercially made elephant pellets, fresh fruits and vegetables, and bread. It is unclear how or if diet contributes to the likelihood of colic in captive elephants.

Inciting or underlying causes of the strangulating lesions can only be speculative in these 4 elephants. Aerophagia may have been an inciting cause in the near-drowning case (case 1). Aerophagia is a possible, but unproven, cause of colic in horses, ostensibly due to creation of air pockets within the intestines.[4]

Anomalous congenital bands (case 2) are rare occurrences in humans and animals that are thought to develop in utero from mesenteric abnormalities and almost always result in intestinal incarceration or strangulation.[3,35,42] At birth, this elephant's gastrointestinal tract may have been too small to be constricted by the bands, so that the abnormality only became clinical as the elephant (and its digestive tract) grew.

Case 3 is more difficult to explain, as no obvious abnormalities or underlying issues were reported, and the surgical castration had occurred a half-year earlier without complications. In case 4, the dietary change involving lush pasture may have been an inciting cause, as sudden changes in diet have been correlated with increased incidence of colic in horses.[14]

All elephants in this report showed some evidence of gastrointestinal disease including abdominal distention and abnormal fecal output, and signs of discomfort including tachycardia, unusual posturing, and vocalization. However, signs of pain seemed minimal compared to the severity of the lesions. More conclusive diagnostic techniques used in colicky horses and cattle, such as rectal palpation, nasogastric intubation, and abdominocentesis, are sometimes possible in small elephants, although safety-related issues for handlers can preclude further examination.[17,18] Although both rectal and transabdominal ultrasound are possible in elephants,[26,41] the maximum image depth of 25 cm of most ultrasound probes prevents adequate organ visualization in animals larger than 2,270 kg.[23] Currently, normal ultrasound anatomy and measurements of the elephant gastrointestinal tract remain undescribed. Nevertheless, the use of ultrasound in case 2 illustrates its utility in smaller elephants suspected of having abdominal disease.

A conundrum of diagnosis is that, at the present time, successful abdominal surgery in elephants has been limited to castrations using a flank approach,[11] laparascopic vasectomies,[43] (elephants have intra-abdominal testicles), and abdominal hernia repairs in young animals.[1] Unfortunately, even these surgeries can be associated with high morbidity and mortality.[18,33] Failure to obtain adequate access to the surgical site was a problem in case 2 and is a common complication of open abdominal surgery in elephants. Other complications include inability to close the incision, incisional dehiscence, and surgical infection.[19] General anesthesia on elephants is similarly difficult, requiring experienced personnel, specially constructed mattresses or waterbeds to prevent muscle damage, and multiple large-animal anesthesia machines connected together to achieve adequate tidal volume as well as sufficient anesthetic gas and oxygen flows.[16,20] Specialized laparoscopic tools and techniques in development for use in elephants may provide additional surgical options in the future.[34,43] If surgery is feasible, however, then prompt diagnosis and treatment are critical.

In conclusion, this report demonstrates that surgical causes of colic are important differentials in evaluating sick elephants. Elephants with severe disease can present with only mild signs of discomfort. Additionally, signs of colic can be nonspecific and difficult to distinguish from other disease processes that affect elephants, including EEHV. Techniques such as ultrasound and rectal palpation can be useful for diagnosing strangulating lesions in smaller elephants.

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