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# Inguinal hernia causes mortality in an adult American black bear

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***Abstract:*** Herniation of viscera induced by medical intervention has been described in carnivores, yet occurrence of hernias causing wild carnivore mortality, including in bears, remains unknown. We describe an inguinal hernia, intestinal entrapment and rupture, and peritonitis causing mortality in a male American black bear (*Ursus americanus*). In the autumn of 2014, a free-ranging, adult bear was housed at Virginia Tech's Black Bear Research Center. After 13 days in captivity, the bear showed signs of lethargy and intermittent inappetence consistent with the onset of hibernating behaviors. However, the bear suddenly displayed rapid deterioration and died before medical assistance could be provided. During necropsy examination, a devitalized portion of small intestine was found entrapped in the left inguinal ring. An intestinal perforation was evident near the entrapment area, which caused a subsequent peritonitis. To our knowledge this is the first report of bear mortality due to inguinal herniation with subsequent perforation and peritonitis.

***Key words:*** black bear, entrapment, inguinal hernia, mortality, peritonitis, *Ursus americanus*

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Inguinal herniation is the protrusion of an organ or tissue through the inguinal canal (Waters et al. 1993). Inguinal hernias can be classified as congenital or acquired due to weakness or damage of muscle and/or ligaments in the inguinal area (Brown et al. 2007, Matthews and Neumayer 2008). Determining the causality of inguinal hernias in wildlife is challenging, though size of inguinal rings may play an important role in this pathology (Brown et al. 2007).

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Naturally occurring gastrointestinal accidents in ursids, including intestinal entrapment due to herniation, are rarely recognized; and hence, there is limited information regarding these conditions. In contrast, abdominal herniation caused by human interventions is fairly well-known in wild carnivores, including bears. Most intestinal accidents in wild carnivores have been associated with placement of radiotransmitter implants in the abdominal cavity. A polar bear (*Ursus maritimus*) developed an abdominal hernia due to dehiscence of the ventral muscle layers, resulting from a suspected infection of the surgical site or an immune reaction to the transmitter (Philo et al. 1979). A free-ranging American badger (*Taxidea taxus*) died due to an omental torsion associated with an intra-abdominal radiotransmitter (Quinn et al. 2010). Ursids subject to bile farming (Asiatic black bear [*U. thibetanus*], sun bear [*Helarctos malayanus*], and brown bear [*U. arctos*]) have been reported to have a high incidence (approx. 30%) of abdominal hernias (Bourne et al. 2010).

In late autumn of 2014, the Virginia Department of Game and Inland Fisheries in the state of Virginia, USA, used a culvert trap to capture a 2-year-old male American black bear (*U. americanus;* hereafter, black bear) from the wild. The bear was immediately transported and housed in captivity at the Black Bear Research Center (BBRC). At admission to the BBRC, we chemically immobilized the black bear with a mixture of ketamine hydrochloride (7.4 mg/kg; ZooPharm Inc., Windsor, Colorado, USA) and xylazine (1.6 mg/kg; Rompun, Bayer Health Care LLC., Leverkusen, Germany) delivered intramuscularly with a Dan-Inject $CO_2$ pistol-dart system (DanWild LLC., Austin, Texas, USA). The bear weighed 94.4 kg and had no apparent physical abnormalities during examination. We reversed anesthesia with an intramuscular injection of yohimbine (0.3 mg/kg; ZooPharm Inc.). We provided the bear with water ad libitum and introduced dry food (80 kcal/kg/day; Southern States® Complete Active Dog Food, metabolizable energy = 3,526 kcal/kg; Pro Pet LLC, Saint Marys, Ohio, USA) over a 6-day period. All procedures were previously approved by the Institutional Animal Care and Use Committee at Virginia Tech under protocol #12-112. The bear was monitored at least twice a day and exhibited normal behavior and normal water and food consumption, defecation, and urination in the first 13 days of captivity. During the subsequent 4 days, the bear began spending most of the time in the denning area, showed signs of lethargy,

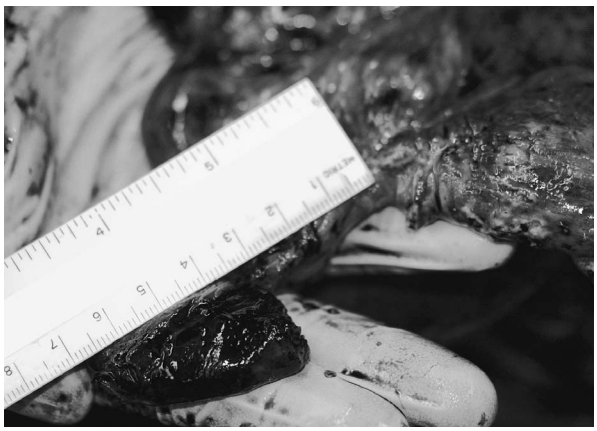

**Fig. 1. Small intestine (jejunum) entrapped in the left inguinal canal of an adult male American black bear (*Ursus americanus*).**

and intermittent inappetence, which were associated with the onset of normal hibernation behavior as 100 individual bears have previously shown at our facility. Finally, after 18 days in captivity, the bear showed erratic walking and difficulty breathing. The bear suddenly collapsed and died before any medical assistance could be provided. At this stage, the bear presented gray coloration in oral mucous membranes. We immediately submitted the body for necropsy examination at the Virginia Tech Animal Laboratory Services, Virginia-Maryland College of Veterinary Medicine.

During external inspection, we observed a sexually intact black bear with appropriate coat condition and weight for the time of the year. The bear weighed 96 kg. The abdominal cavity contained approximately 1 L of tan, cloudy fluid with occasional fibrin clots. Upon microscopic analysis, the fluid contained reactive neutrophils and a smaller proportion of macrophages and lymphocytes, as well as numerous bacteria of varying morphology. The small intestines were purple and uniformly dilated. A 7-cm portion of dark purple, friable jejunum was entrapped in the left inguinal canal (Fig. 1). At the level of the inguinal ring, the jejunum was white-gray, thickened, and markedly constricted with multiple fibrinous adhesions to the inguinal ring. A 1-cm jejunal perforation was immediately proximal to the entrapped intestine at the entry into the left inguinal ring (Fig. 2). The left testis and spermatic cord were dark purple and grossly enlarged (Fig. 3), suggestive of strangulation of vascular supply to the testis by the entrapped intestine. Incidentally, we found a 0.22-caliber, lead bullet in a fibrous

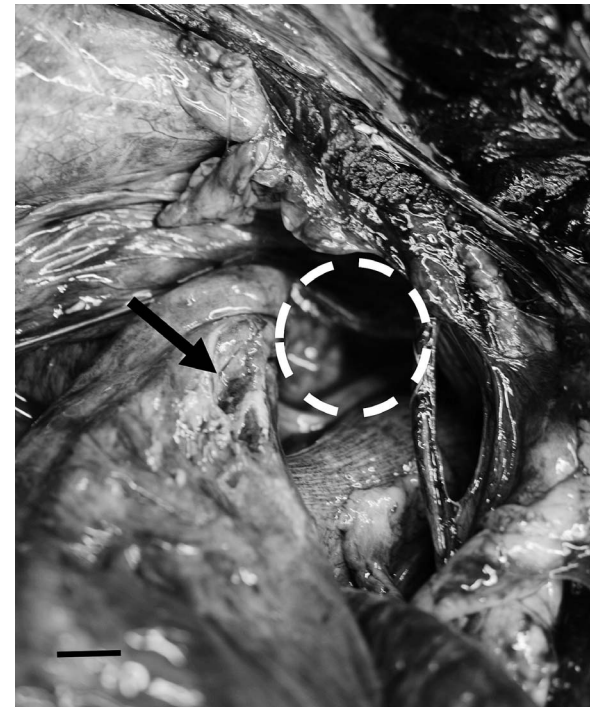

**Fig. 2. Jejunal perforation (arrow) proximal to a left inguinal canal (circle) entrapment in an adult male American black bear (*Ursus americanus*). Scale bar = 1 cm.**

**Fig. 3. Testicles and spermatic cords of an adult male American black bear (*Ursus americanus*) with a left inguinal hernia. Left testicle and spermatic cord (left) presented darker coloration and larger size than those on the right side (right). Scale bar = 1 cm.**

capsule in the left lung lobe without any associated gross pathology.

During histopathological examination, the section of entrapped jejunum revealed hemorrhage and large numbers of neutrophils that extended from the serosal to the luminal surfaces in the area of perforation. The serosa was covered by a thick layer of fibrin with admixed neutrophils, basophilic cellular debris, and mixed bacteria. Blood vessels throughout the sections were severely dilated. Moderate necrosis of the tunica muscularis and mucosa were evident in the most severely inflamed portion of the intestine. In less affected areas, the inflammation extended into adipose tissue and the serosal layer was hypertrophied. The left testis revealed generalized congestion and areas of hemorrhage. Based on these findings the diagnosis was an inguinal hernia of unknown duration, with entrapment and perforation, and subacute peritonitis.

To our knowledge this is the first report of bear mortality due to an inguinal hernia with entrapment, subsequent perforation, and peritonitis. We were not able to determine whether the inguinal hernia was of congenital or acquired origin. Nevertheless, inguinal hernias are likely to be caused by multifactorial aspects of individuals. Some risk factors in other species experiencing inguinal hernias have been identified, including abdominal muscle weakness and/or muscle and fascial weakness in the growing area coupled with increased intra-abdominal pressure (Conner and Peacock 1973, Matthews and Neumayer 2008). Cryptorchidism in humans and mice (*Mus musculus*) can either be concomitant to inguinal hernias or pose an increased risk factor for herniation (Clarnette and Hutson 1996, Thonneau et al. 2003). However, it remains unclear whether specific body postures, age, or seasonality could also represent risk factors in ursids. We suggest the 2 most possible scenarios that may explain the occurrence of inguinal hernia in our case. First, the black bear arrived at our facility with the preexisting condition due to genetic and/or muscle-strength issues, and the entrapped intestine experienced rupture while at our facility. Second, the bear was predisposed to inguinal herniation and a change in intra-abdominal pressure during capture or transport created the "ideal conditions" to facilitate herniation, entrapment, and subsequent intestinal rupture at our facility. Even though the latter scenario is possible, we believe it is the less likely of the 2 scenarios given that we have conducted multiple, closely monitored, capture procedures in 130 adult American black bears with only 1 individual (current report) presenting herniation and entrapment.

Interestingly, clinical signs associated with severe pathological findings in ursid intestinal accidents can differ widely. A captive polar bear showed lethargy, depression, and intermittent vomiting due to an omental torsion (Mendez-Angulo et al. 2014). In this case, an exploratory laparotomy revealed a severe inflammatory response producing a large amount of malodorous, serosanguineous fluid in the abdominal cavity, and necrotic areas surrounding the torsion (Mendez-Angulo et al. 2014). Similarly, another captive polar bear with a recurrent umbilical hernia with omentum entrapment showed acute signs of extreme abdominal discomfort, which included restlessness, rolling, and pawing at the ground (Velguth et al. 2009). It is possible that ursids are stoic (e.g., highly tolerant to pain) and exhibit clinical signs late in the course of illness, thereby increasing the difficulty of diagnosis and treatment in this species.

We suggest that intestinal accidents should be considered as a differential diagnosis for the clinical signs shown by the black bear in our case. Herniation and subsequent intestinal accidents should be investigated as a possible cause of mortality in ursids when no apparent cause of death is identified during external inspection.

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