Abdominal ventral hernia in a pigeon (*Columba livia*): a case report

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**ABSTRACT:** Reports of abdominal hernias in birds are often not clearly described. Abdominal hernias have been characterized as a separation in the aponeurosis of the abdominal musculature on the ventral midline. In this article one case of abdominal hernia is described. A painless, reducible swelling was located in the ventral abdominal region close to the cloaca. Surgical repair was performed because the bird was stable clinically and an hernioraphy was indicated. A ventral midline celiotomy was performed. For repair of the abdominal hernia the abdominal muscles and skin were sutured in a standard two-layer closure using a simple continuous suture pattern. This procedure is safe and uncomplicated and this approach can be recommended as a curative method for abdominal hernia repair.

**Keywords:** pigeon; hernia; repair; adhesion

Abdominal hernias in birds can be congenital or acquired (Bennet, 1994). The etiology of abdominal hernias in birds is unknown. Abdominal hernias occur fairly frequently in female psitacine species, particularly in the budgerigar. Hernias are frequently associated with weakening of the abdominal musculature caused by egg-laying, egg-binding, or hyperestrogenism. Rarer causative factors of abdominal hernias applicable to either sex include trauma, straining, or abdominal masses. Abdominal hernias in birds are not true hernias because there is no opening in the aponeurosis of abdominal muscles and typically there is no hernial ring. Therefore, entrapment and strangulation of abdominal viscera do not occur.

Any avian surgeon must first become a competent small animal surgeon. In view of the small body size and increased metabolic rate, avian surgery requires exactness because any errors are magnified. Surgery on birds of less than 2 kg requires microsurgical techniques and equipment, as well as a significant degree of manual dexterity. The patient must be assessed with respect to energy and nutritional status and any circulatory fluid or blood deficits. Intraoperative and postoperative hypothermia, analgesia, sepsis and shock must be controlled. Any longer period of starvation will be detrimental in view of risk of a negative energy balance. All birds over 100 g are intubated to protect the airway from gastric reflux. Smaller birds are generally not intubated because of the increased risk of blockage of small-diameter tubes by respiratory secretions (Forbes, 2002).

**Case presentation**

A two-year old pigeon was referred with a 15-day history of abdominal swelling. On admission, the pigeon had a rectal temperature of 39.7°C, its pulse rate was 195 beats per minute and its respiratory rate was 28 breaths per minute. Its diet consisted of a commercially available food. No indication of diarrhea was observed in the cloacal region.

A painless, reducible swelling was located in the ventral abdomen region close to the cloaca. The swelling appeared as a spherical body, about 5 cm in diameter. The presented data enabled us to make a diagnosis of abdominal hernia (Figure 1).

For surgery, the pigeon was placed under general anaesthesia using ketamine hydrochloride (Narketan® 10; Vetoquinol) 30 mg/kg and xylazine...
(Xylapan®, Vetoquinol) 1 mg/kg administered intramuscularly into the pectoral muscle. The pigeon was positioned in dorsal recumbency. After preparation of the operation field, a ventral midline celiotomy were performed. This approach gives only poor visibility of most of the abdominal cavity. It does facilitate surgery of the small intestine, pancreatic biopsy, liver biopsy, or cloacopexy and is used in diffuse abdominal disease such as peritonitis, egg binding, and cloacal prolapses. The skin of the abdominal wall is tented, and an initial incision is made with scissors from the cranial border of the cloaca to 2 cm caudal of sternum (Figure 2).

The incision is then extended with fine scissors. Corrective surgery involved a procedure in which an elliptical transabdominal incision through the skin and abdominal muscles was performed to reduce the size of the distended hernial sac by removing part of the abdominal wall. This approach can be extended along the costal border cranially and to the pubis caudally to create a flap unilateral or bilateral of the midline to increase access.
The hernia consisted of enlarged liver and small intestine loops glued with adhesions in the form of a fibrin mesh which formed one ball (Figure 3).

For repair of the abdominal hernia the abdominal muscle and skin were sutured in a standard two-layer closure using a simple continuous suture pattern with Maxon 3/0 (Figure 4 and Figure 5).

DISCUSSION

Reports of abdominal hernias in birds are somewhat ambiguous. Some studies characterize them as a separation in the aponeurosis of the abdominal musculature on the ventral midline. Abdominal hernias have also been rejected as true hernias because of the absence of an opening in the aponeurosis of the abdominal muscles. Affected birds are most commonly middle-aged to older hens with variable degrees of abdominal swelling. Skin ulceration and hemorrhage can also occur. (MacWhirter, 1994; Altman, 1997).

The present study on this abdominal protrusion in *Columba livia* returned a diagnosis of abdominal hernia. Previously, abdominal or umbilical hernias as a flock problem in turkey poults (Carlson, 1962; Ranck, 1973). Ranck (1974) suggested that umbilical hernias may have at least two different causes. One cause may be genetic factors, as in the case of swine or other animals, in which hernias become a flock problem and the hernial rings are apparently not inflamed. Another cause may be an unhealed navel, in which some omphalitis may occur, causing the hernial rings to be inflamed or future healing resulting in a fibrous covering of the umbilical area.

Furthermore, there was no sign or indication of thinning or weakness of the abdominal muscles to contribute to the hernial protrusion. The present hernia seems to have been caused by acquired factors like inflammation or a developmental disorder, although the definitive etiology is unknown.

Early surgical repair of abdominal hernias should be determined individually. Prompt surgical repair of the hernia is important if the bird traumatizes its abdomen by rubbing on surfaces, experiences respiratory distress, has difficulty passing urates and feces from its cloaca, or has the entire abdominal viscera within the hernial sac (MacWhirter, 1994). Surgical repair was performed because the bird was stable clinically and an hernioraphy was indicated.

Adhesions are pathological bonds between surfaces within body cavities. These bonds can be thin connective tissues, thick fibrous bridges containing blood vessels and nerve tissue, or a direct contact between two organ surfaces (Diamond and Freeman, 2001). Adhesion can be found in abdominal, pericardial, pleural, uterine and joint cavities, and in the chamber of the eyes. Peritoneal adhesions may be classified, according to the etiology, as congenital or acquired, which in turn can be classified as post-inflammatory or postoperative. Depending on their location and structure, adhe-
sions may remain silent or cause clinically important complications such as intestinal obstruction, chronic pelvic pain, female infertility and difficulties at the time of re-operation (Ellis, 1997).

Intestinal obstruction is the most serious complication of peritoneal adhesions as it can be life threatening due to strangulation. Adhesions increase the technical difficulty for surgeries, impede access to the abdomen and the operation site, enhance complication rates, and also complications relating to anaesthesia, operating recovery time and use of surgical materials (Ray et al., 1998).

Peritoneal injury, infection or irritation, initiates an inflammatory reaction that increases all components of the peritoneal fluid, i.e., proteins and cells, generating fibrous exudates and the formation of fibrin. Fibrin formation is the result of the activation of the coagulation cascade, which includes two pathways, i.e., the contact factor or intrinsic pathway and the tissue factor or extrinsic factor. These fibrinous exudates and fibrin deposition are an essential part of normal tissue repair. The degradation of fibrin is regulated by the plasminogen system. In this system, the inactive proenzyme plasminogen is converted into active plasminogen activators (Pas), (Holmdahl, 1997).

The balance between fibrin deposition and degradation is critical in determining normal peritoneal healing or adhesion formation. If fibrin is completely degraded, normal peritoneal healing will occur. In contrast, if fibrin not completely degraded, it will serve as a scaffold for fibroblasts and capillary ingrowth. Indeed, fibroblasts will invade the fibrin matrix and extracellular matrix (ECM). The ECM can be completely degraded by MMPs, leading to normal healing. However, if the process is inhibited by tissue inhibitors of MMPs peritoneal adhesions will be formed (Murphy et al., 1992; Holmdahl et al., 1997).

REFERENCES


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