



## CASE REPORT

### A Blind-End Atresia of the Small Intestine in a Puppy. A Case Report

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#### ARTICLE HISTORY (18-095)

Received: March 18, 2018  
Revised: September 04, 2018  
Accepted: November 17, 2018  
Published online: January 24, 2019

#### Key words:

Dog  
Histopathology  
Small intestine atresia

#### ABSTRACT

Intestinal atresia and stenosis is one-third of intestinal obstruction cases in animals and is a common surgical problem in neonates. In human the most common causes of intestinal obstruction in newborns are intussusception and intestinal atresia. The pathogenesis of intestinal atresia in both human and animals is not clear. The present study describes the clinical and histomorphological features of a case of congenital intestinal atresia in a dog. The animal presented with single ileal blind-end atresia combined with the extension of proximal segments of intestines. The distal part of intestines including anus were patent. Histologically intestinal wall congestion and flattening of intestinal villi were noted.

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**To Cite This Article:** Janus I and Dzimira S, 2019. A blind-end atresia of the small intestine in a puppy. A case report. Pak Vet J, 39(1): 142-144. <http://dx.doi.org/10.29261/pakvetj/2019.004>

#### INTRODUCTION

The intestine congenital segmental anomalies may vary in the degree from stenosis with incomplete occlusion of the lumen to atresia with its complete occlusion that can be further subdivided into three types: membrane atresia, cord atresia, and blind-end atresia. The latter is described as an absence of a segment of the intestine, with disconnected blind ends and a gap in the mesentery. (Brown *et al.*, 2007; Nichol *et al.*, 2011; Lombardero and Yllera, 2014). It can occur in different parts of the intestines, although in dogs it has been found only in the small intestine and anus (Gough, 1999; Vianna and Tobias, 2005). The defect prevents the normal movement of gut content and meconium in the fetus and neonate, leading to dilation of the proximal segment, with progressive abdominal distension. The bowel beyond the discontinuity is small in diameter, and devoid of content other than mucus and exfoliated cells. (Brown *et al.*, 2007; Lombardero and Yllera, 2014) In human intestinal atresia occurs in approximately one-third of all cases of neonatal intestinal obstruction. (Ali *et al.*, 2011). Although the defect was reported in various animals (Gough, 1999; Brown *et al.*, 2007, Lejeune *et al.*, 2011; Lombardero and Yllera, 2014), it occurs mostly in cattle, horses, and pigs with dogs being affected less frequently.

**Clinical report:** A 48-hours-old female Chihuahua puppy was admitted for necropsy. According to owners' report, the dog presented with a normal appetite, abdominal

enlargement, and difficulties with defecating. The post-mortem examination showed a distension of the abdominal cavity. Stomach and small intestines (duodenum and ileum) were visibly filled; the intestines were 1 cm in diameter; the intestinal mucous membrane was dark pink (Fig. 1). In approximately half of length, the ileum was blind-ended with the posterior part also blind-ended and smaller in diameter (3 mm). The posterior part of ileum normally passed in a large intestine and ended with a patent anus (Fig. 2). The liver was dark red, congested and fragile. Other organs were normal. Histological sections of the proximal part of intestines, routinely stained with hematoxylin and eosin, showed a very thin strongly congested intestinal wall with flattened and rarely arranged intestinal villi and blood stasis in the widened vessels of the intestinal wall (Fig. 3). The distal segment of intestines showed villi arranged densely; the wall was strongly congested, and thicker, with less stretched bowel musculature. The duodenum specimens showed a moderate degree of villi congestion and presence of cellular detritus in the lumen.

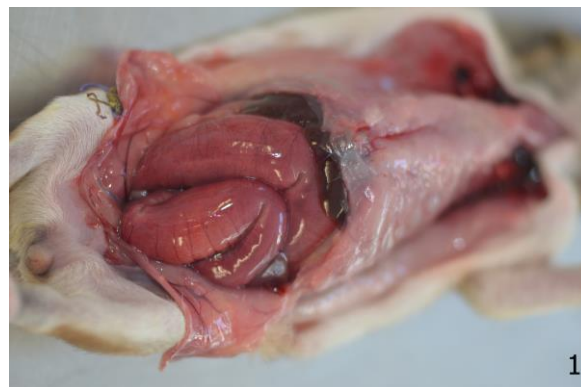
#### DISCUSSION

Intestinal obstruction can be congenital or acquired. The latter in dogs is usually caused by foreign bodies, abscesses, intestinal or mesenteric volvulus (Jones *et al.*, 2017; Plavec *et al.*, 2017; Smeak *et al.*, 2018). In those cases the treatment method of choice is laparotomy with enterotomy or enterectomy (Jones *et al.*, 2017; Plavec *et*

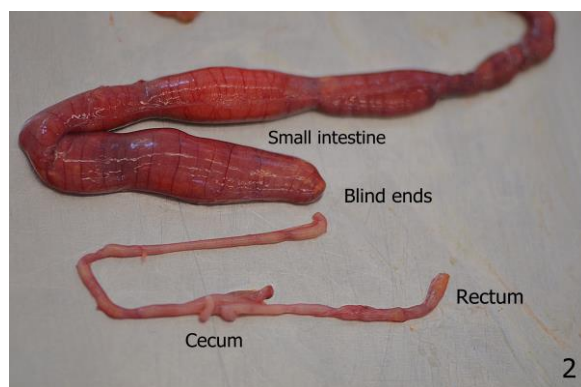
al., 2017). However, these causes are less likely in a 48-hour-old puppy pointing to a congenital disease.

The occurrence, treatment possibilities and various hypotheses on the cause of intestinal atresia have been studied since XVII century (Ali *et al.*, 2011; Nichol, 2011; Lombardero and Yllera, 2014). At first, atresia was speculated to result from the obliterative embryonic events such as the atrophy of the vitelline duct (Ali *et al.*, 2011; Lombardero and Yllera, 2014). In the end of XIX century also the failure of normal recanalization of the solid cord stage of the fetal bowel was suspected to lead to the atresia (Ali *et al.*, 2011; Nichol, 2011; Lombardero and Yllera, 2014). In 1912 Spriggs proposed the theory of fetal vascular accident as a possible cause of the defect. The theory was further developed and proven in the second half of XX century on various animal models (Ali *et al.*, 2011; Nichol, 2011). Since the 1950s' also familial origin of the disease was reported (Ali *et al.*, 2011; Lombardero and Yllera, 2014). Nowadays, the lack of recanalization of the solid cord stage of the intestine during early fetal development and the late mesenteric vascular accident seem to be the two most reliable theories. (Ali *et al.*, 2011; Lejeune *et al.*, 2011; Nichol, 2011). However, also other mechanisms e.g. improper molecular signaling related to gut specification early in development might be related to the development of the defect (Sadler and Rasmussen, 2010; Nichol, 2011). Genetic defects of different proteins (including membrane-bound tyrosine kinase receptor, Hedgehog signaling proteins and Cdx-2 protein) result in a disruption in endoderm development and in atresia formation (Nichol, 2011). This challenges the vascular disorders hypothesis and points to a disruption in endoderm development or endoderm to mesoderm signaling as the leading events of atresia formation (Nichol, 2011). What is interesting, intestinal obstruction seems not only to be caused by proteins disorders, but also contribute to further neuroendocrinal signaling disturbances (Ballouhey *et al.*, 2017). Gough (1999) in a case similar to ours in a Kerry blue terrier discusses different possible causes of intestinal atresia, including familial inheritance. In human, intestinal atresia accounts for approximately one-third of all cases of neonatal intestinal obstruction (Ali *et al.*, 2011; Nichol, 2011). The defect can be sporadic or hereditary (Ali *et al.*, 2011; Nichol, 2011). It can be also a component of genetic syndromes such as chromosome 15q24 microdeletion syndrome, where intestinal atresia is one of many defects present in the affected patient (Magoulas and El-Hattab, 2012). There is relatively little evidence to show that the defect is inherited in any animal species, although atresia is reported to be an inherited lethal defect in calves and foals (Lombardero and Yllera, 2014). According to data obtained from the history, we do not suspect a familial origin playing a role in the described case, although no other mechanisms can also be confirmed due to lack of evidence. Subburayan *et al.* (2015) analyze 147 cases of intestinal obstruction in the newborn in a period of 5 years, including 39 cases of intestinal atresia with various histomorphological changes. The most common type of atresia was type II combined with other anomalies: gastroschisis, volvulus, anal stenosis, microcolon, annular pancreas, meconium cyst and duplication cyst. Histologically they observed ulceration, flattening and abnormal villous

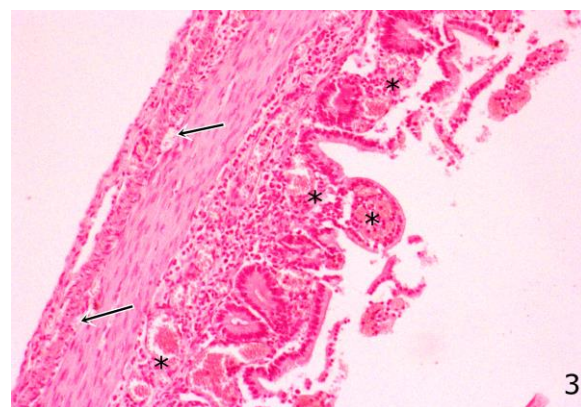
configuration in the intestinal wall, luminal obliteration and haemangiomas proliferation of blood vessels. These authors observed also hemorrhage, calcification and mesenchymal condensation around blood vessels. In our case, we observed only a thinning of intestinal wall, flattening of villi, and a strong blood stasis and small extravasation of blood. We did not find any changes such as extensive hemorrhages or dystrophic tissue calcification. The number of Auerbach's myenteric plexuses in the proximal section of the intestine was smaller than in the normal sections of the gastrointestinal tract. An intrauterine intestinal ischemia due to vascular pathology followed by a resorption of the bowel is the possible explanation for the development of intestinal atresia.



**Fig. 1:** Macroscopic view of abdominal organs. Extremely filled small intestine.



**Fig. 2:** A view of blind end of thick, small intestine and a thin part of distally part of digestive tract.



**Fig. 3:** Histopathological picture of blind, thick segment of the small intestine. Visible congested mucosa, strongly filled blood vessels (asterisks) and single Auerbach's plexus (arrow) HE stain, 100x.

**Conclusions:** Although intestinal atresia is a rare defect reported in dogs, it should be considered as a possible cause of abdominal extension and problems with defecation in newborn animals. The possible causes including familial origin should be a subject of further studies.

**Acknowledgments:** Publication supported by Wrocław Centre of Biotechnology, programme the Leading Notional Research Centre (KNOW) for years 2014-2018.

**Authors contribution:** SD and IJ performed the necropsy; SD performed the histopathological examination; all authors critically revised the manuscript and approved the final version.

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