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Case Report

ATRESIA COLI IN AN ARABIAN FOAL

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ABSTRACT

Atresia coli is a congenital defect that result in an inability to pass feces since birth. In the present study, type IIIa atresia coli (blind ends) was identified in a male Arabian foal with clinical signs of abdominal distension, colic, failure in defecation and depression. At necropsy, segmental agenesis affected left ventral and right dorsal colon. The dorsal right colons, transverse, small colon and rectum were atrophic with narrow lumen. The prognosis was poor in this case. Atresia coli was reported in the various breed of foals but it seems this is the first report in an Arabian breed foal.

Key words: Atresia coli, foal, colon

INTRODUCTION

Congenital anomalies of the digestive tracts are uncommon in the domestic animals, but atresia of anus and rectum is more common than other anomalies in calves and piglets (1). Intestinal atresia is characterized by complete obstruction of the lumen in human beings (2) and animal species (1). Any intestinal segment from the duodenum to the anus may be involved (3, 4). In animals, small intestine atresia is less common than the large intestine (1, 5). The most frequent intestinal atresia is observed in colon particularly the spiral colon of calves (6, 7), and large and small colon of foals (6). Intestinal atresia is classified into 4 categories. Type I or membrane atresia that the intestinal lumen is closed with a membranous diaphragm. Type II: cord atresia. Two blind ends of intestines are connected together by a fibrous or muscular cord-like strip. Type IIIa is a blind-end atresia that a segment of intestine is absent. Type IIIb is similar to type IIIa, but the distal segment of affected part is coiled. Type IV, identified by multiple sites of atresia (8, 9). The present study describes the occurrence of intestinal atresia in an Arabian foal.

CASE REPORT

A four day-old, male Arabian foal was presented to the Veterinary Medical Teaching Hospital,

Shahid Bahonar University of Kerman, Iran for treatment of colic. Defecation was not observed from the birth time by owner. Foals had a normal anal orifice with an empty rectum. Clinical examination revealed hypotermie, tachycardia and elevated respiratory rate. The mucous membranes were dark which shows a possible toxemia. Complete absence of faeces was determined by digital rectal examination and response to warm water enemas was negative. Foal was unable to stand. There was no good result of recovery and the case referred to postmortem examination. At necropsy, small intestine was normal. Cecum developed normally and right ventral colon was appeared as a blind-end sac that impact by feces. Left ventral colon and left dorsal colon were not formed. The dorsal right colons, transverse, small colon and rectum were atrophic with narrow lumen. No other abnormality was observed in other organs. Atresia coli was diagnosed in this case. (Figure 1)

DISCUSSION

Congenital anomalies at birth originate from faults during embryogenesis. Genetic factors or geographic influence, or both may be responsible. Developmental defects vary as lethal, semi-lethal, or compatible with life. (10, 11). One of these anomalies in digestive system is atresia. Intestinal atresia is an unusual congenital defect that occurs at various locations of intestinal tract. In human, the incidence rate is 1 in 5,000 birth in the neonate (12). The affected

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neonates may die due to intoxication within a few days after birth (5). In the most domestic species, intestinal atresia occurs sporadically with more frequency in calf and rare in the foal. Clinical signs are recognized 24-48 hours after birth. Foals affected with atresia coli show abdominal distension, colic, failure in defecation, depression and malaise (5, 13).

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Figure 1. Atresia coli in foal. Agenesis of the left ventral colon and left dorsal colon. RVC: Right ventral colon; RDC: Right dorsal colon; TC: transverse colon.

In the present study, type IIIa atresia coli (blind ends) was identified in an Arabian breed foal. Clinical signs were similar to the previous reports of intestinal atresia in the domestic animals. Segmental agenesis had occurred in a wide part of colon. The prognosis was poor in this case.

Atresia coli was reported in the various breed of foals but it seems our case is the first report in an Arabian breed foal. Nappert et al. (1992) described an occurrence 1.3% incidence of atresia coli in foals with history of progressive abdominal distension, colic and lack of faecal production at the Large Animal Hospital, University of Montreal. The breed distribution of cases was included Appaloosa (N = 2), Morgan (N = 1), Standardbred (N = 1), Throughbred (N = 1), Paint Horse (N = 1) and Quarter Horse (N = 1). Five foals had type III (blind-end) and 2 foals type II (cord atresia). Surgical treatment was done in 3 foals but was unsuccessful. They stated a grave prognosis should be considered when this condition is diagnosed in foals (14).

Young et al. (1992) confirmed atresia coli in five foals (4 females and 2 males) with signs of acute colic and absence of meconium during surgery. Severity of atresia was variable from lack of the large, transverse and/or small colon in all foals and also in an eight month aborted fetus. Other concurrent congenital defects were found including ventricular septal defect, common truncus arteriosus, right ventricular hypertrophy, hydrocephalus, cerebellar dysplasia, cerebral atrophy dermal and hemangioma on the dorsal thorax (15).

Hunter and Belgrave (2010) found type II atresia coli in a Thoroughbred foal. On necropsy, descending colon ended in a distended blind-end sac that was connected to a narrow terminal colon by a 2–3 mm fibrous cord. Distended descending colon had thin wall without teniae. No lumen was apparent within the fibrous tissue cord (16).

The pathogenesis of intestinal atresia is poorly understood but this condition could be resulted from a simple recessive gene, developmental arrest, or vascular insufficiency that resulting in ischemic necrosis of the affected intestinal portion. One of the main theories regarding intestinal atresia is interruption of the blood supply to a segment of intestine and resulting in segmental atrophy in the embryonic course (12). Vascular insufficiency may be due to intussusception, volvulus, herniation or strangulation of the intestines during pregnancy period (17, 18). This etiology is supported in experimental studies (19). Tibboel et al. (1980) demonstrated that occlusion of a mesenteric vessel for more than 20 min resulted in atresia in >50% of experimental subjects in chicken embryos (20). Other opinion suggests that atresia results from a failure of revacuolization or recanalization of the intestinal development (21). Infectious and environmental risk factors for intestinal atresia have little support. In one report, a foal with atresia coli was found to be infected with equine herpesvirus-1, but no

relationship was found between two problems (22).

In contrary to the literature, colonic atresia suggests a non-heritable trait (8), atresia ani is believed to be hereditary in cattle and pigs. Autosomal recessive gene is responsible for atresia jejuni in Jersey breeds and atresia ilei in Swedish Highland (4).

Diagnosis of intestinal atresia requires taking history, clinical signs, physical examination, rectal palpation, ultrasonography, endoscopic examination and radiography using barium enemas (3, 12). Surgical repair is recommended (23). Treatment is based on intestinal anastomosis of the atretic segment (9, 24). In the most animals that intestinal atresia affected especially the proximal sites, surgical treatment is not recommended due to limited accessibility, low survival rate after operation and economic considerations.

CONCLUSION

If cause of atresia has genetic base and/or the affected animal is intended for breeding, surgery is not a useful way due to probably propagation of genetic defects (13). For preventing program, the, parents of the affected foal should be avoided for breeding purpose (12). It seems the present study is the first report of atresia coli in Arabian breed of foal.

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