



## A Case of Duodenal and Pancreatic Agenesis in A Calf

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### ABSTRACT

A three-day-old Montafon calf was brought to the clinic and died in spite of intervention attempts. In the necropsy of this calf, abdominal cavity was filled with caseous material and intestines were distended due to gas accumulation, had an anemic texture. The liver tissue was pale gray-white in color. The duodenum was not present where it should normally be between the jejunum and the pyloric region of the abomasum. Instead, there was a structure presenting hyperemic and adipose tissue about 25-30 cm, along with lymph nodules. At the end of the Pylorus and at the beginning of the jejunum, there were luminal structures near the thick, crumpled crater-like circle. The pancreas was not present where it anatomically should be found. In the chest cavity, the lungs were congestion and not fully collapsed. The heart muscle was pale. In the lights of necropsy and histopathological findings, case was diagnosed as congenital duodenal agenesis, according to the authors knowledge this is the first report in a calf in Turkey.

**Keywords:** *Duodenal Agenesis, Calf, Congenital, Pancreas, Intestine*

### ÖZ

### Bir Buzağıda Duodenum ve Pankreatik Agenesis Olgusu

Agoni halinde kliniğe getirilen 3 günlük Montofon buzağının yapılan müdahaleye rağmen öldü. Bu buzağıya yapılan nekropside barsakların anemik ve gazla dolu olduğu, karın boşluğunda kazeöz yapılar görüldü. Karaciğer dokusu soluk gri-beyaz renkte idi. Duodenum normalde jejunum ve abomasumun pilorik bölgesi arasında olması gereken yerde mevcut değildi. Bunun yerine, lenf nodülleri ile birlikte yaklaşık 25-30 cm'lik hiperemik ve yağ dokusu sunan bir yapı vardı. Pylorus'un sonunda ve jejunumun başında, kalın, buruşuk krater benzeri dairenin yakınında lüminal yapılar vardı. Pankreas anatomik olarak bulunması gereken yerde yoktu. Göğüs boşluğunda akciğerler konjensyonlu ve kollabe olmadığı gözlemlendi. Kalp kası soluk renkte idi. Buzağılarda ender görülen, nekropsis bulguları ve histopatolojik incelemeler sonucunda konjenital duodenal agenezis olarak tanımlanan bu olgu Türkiye'den bildirilen ilk vakadır.

**Anahtar Kelimeler:** *Bağırsak, Buzağı, Duodenum Agenesisi, Konjenital, Pankreas*

### INTRODUCTION

Numerous cases of congenital malformations involving various parts of the intestines have been reported in domestic animals (Priour and Dargatz 1984). The cause of defects in the intestines of animals is ischemia, which usually occurs in a segment of the intestine in the early stages of fetal life (Azizi et al. 2010; Jubb et al. 1993). Intestinal atresia is the most common in domestic animals and the most common intestinal anomalies in ruminants are atresia coli, atresia ani, atresia recti (Demiraslan et al. 2014; Göksel and Saritaş 2015; Jubb et al. 1993).

It has been reported that atresia ani is inherited in cattle, atresia jejuni is an autosomal disease in Highland cattle and Jersey breeds, but atresia coli is not genetically understood (Priour and Dargatz 1984) and segmental intestinal agenesis is rarely reported (Priour and Dargatz 1984). The submitted duodenal agenesis case is thought

have a worth of publication since the number of literatures on it is very limited both in world and our country.

### CASE HISTORY

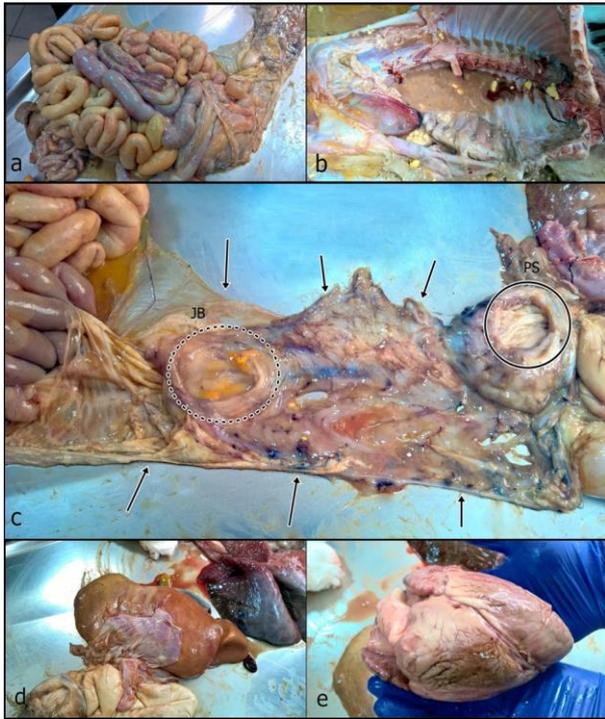
The 3-day-old male Montafon calf was brought to the clinic in poor general condition and in agony. Clinical history, taken from the owner, anorexia, weakness and inability to defecate. Clinical examination revealed that the in the lymph nodes growth, dehydrated, difficulty breathing and tympanic abdominal was presented. The hemoglobin values were low and leukocyte, lymphocyte and monocyte cells were high, suggesting a septic infection, but the died without detailed intervention.

When the abdominal cavity was opened, the intestines was gas filled (Figure 1a). It was observed that the contents of the stomach containing the cheesy milk clots were in the abdominal cavity (Figure 1b). At the end of the abomasal



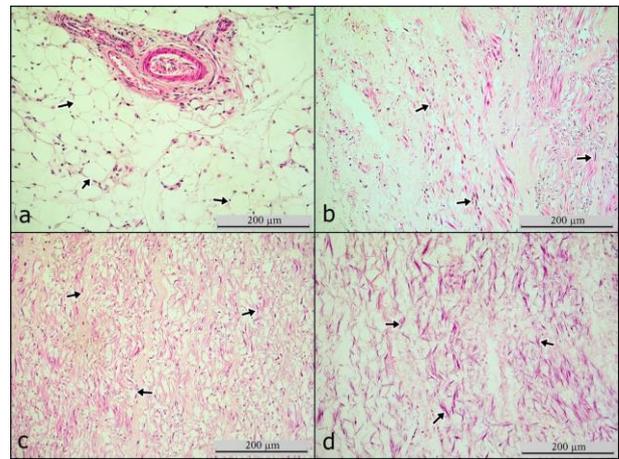
pylorus region (PS), there was no duodenum, which should be between the jejunum (JB), and a 25-30 cm membrane structure consisting of hyperemic and adipose tissue and lymphoid nodules was observed (Figure 1c). It was seen that pylorus reached abomasum from this structure while it was connected to small intestines by luminal opening by jejunum (Figure 1c).

In addition, the pancreas was not present where it anatomically should be found. The liver was pale gray-white color (Figure 1d). Pelvic of the urinary bladder is grown inside was full of urine. When the chest cavity was opened, it was observed that the lungs were reddish and not well collapsed. The heart was anemic and the muscles were pale (Figure 1e).

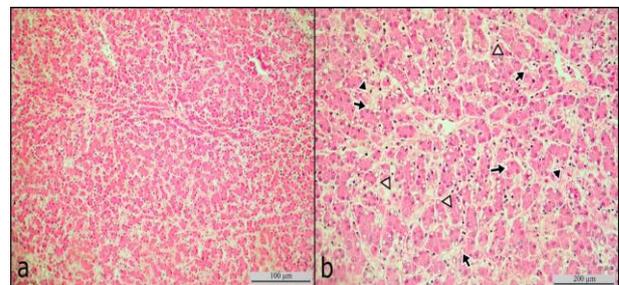


**Figure 1.** a. Gas-filled intestine appearance. b. Appearance of stomach contents, including cheeses, in the abdomen-chest cavity. c. The appearance of the duodenum and pancreas instead of the membranous at the end of the abomasal pylorus (PS) region and between the jejunum (JB). d. Appearance of liver in gray-yellowish color. e. Appearance of heart muscle was pale.

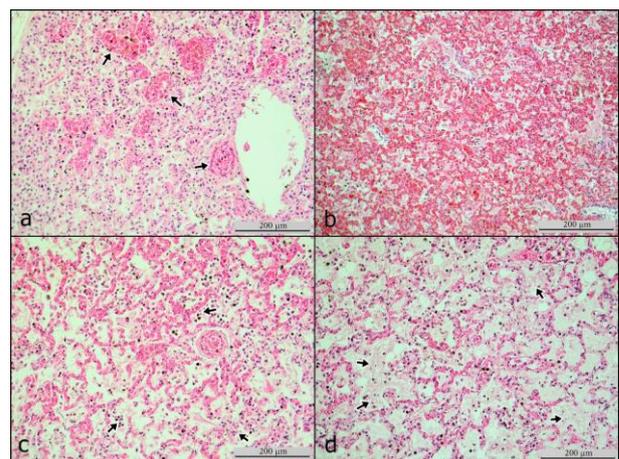
In the sections prepared with membranous tissue, hyperemic blood vessels, fatty tissues composed of fat cells in large areas, collagen and fibrous tissue cells (fibrocytes-fibroblasts) and structures composed of myocytes were observed (Figure 2 a,b,c,d). Hepatocyte cytoplasm was found to have sharp-edged rounded vacuole formation in the liver (Figure 3a). While lymphocytic cells were seen in the sinusoids, an increase in the number of Kupffer cells was detected (Figure 3b). Hyperemic blood vessels (Figure 4a) and severe congestion (Figure 4b) were noted in the lungs. Additionally, in some areas, the alveolar epithelium detached into the exudative mass (Figure 4c) admixed with fibrin (Figure 4d).



**Figure 2.** a. Fatty appearance of fat cells forming large areas composed of fat cells, Membranous Mass (arrows), HxE, x20. b-c. The appearance of collagen and fibrous tissue cells (fibrositis-fibroblasts), Membranous Mass (arrows) HxE x20. d. Appearance of Myocytes Membranous Mass (arrows) HxE, x20.



**Figure 3.** a. The appearance of rounded fat vacuoles of different sharp edges with multiple sizes in the cytoplasm of hepatocytes scattering diffusely in the parenchyma Liver, HxE, x10. b. Numerous increase in number of lymphocytic serial cells (white arrows), fat vacuoles (arrows) and Kupffer cells (arrow heads) in the sinusoids, Liver, HxE, x20.



**Figure 4.** a. Hyperemic blood vessels (arrows), Lung HxE, x20. b. Hyperemia and severe congestion in interalveolar vessels in the form of large areas, Lung HxE, x20. c. Appearance of spilled alveolar epithelium in the exudative mass of the alveolar lumen (arrows), Lung HxE, x20. d. Appearance of fibrin in the alveolar lumens (arrows), Lung HxE, x20.

## DISCUSSION

Congenital malformations involving parts of the intestines in domestic animals have been reported alone or in combination with other system malformations (Prieur and Dargatz 1984). The cause of malformation and defective occurrences in intestines is ischemia, which usually occurs in a segment of the intestine in the early stages of fetal life (Azizi et al. 2010; Jubb et al. 1993). The most known and most studied are atresia which appears as partial or complete intestine obstruction. Most of them are known as the determined by inherited autosomal recessive genes (Prieur and Dargatz 1984). It is known that the etiologies of segmental intestinal agenesis cases are similar (Prieur and Dargatz 1984), while others are originated from viral infection causing multiple malformations of the nervous-skeletal-muscular system (Elbers et al. 2012; Jubb et al. 1993; Smolec et al. 2010).

Prieur and Dargatz (1984) reported segmental intestinal agenesis in a 2-day calf and reported that there was no ileocecal region and colon, but fat and mesenteric lymph nodules were present in the rectum and anus (Prieur and Dargatz 1984). However, intestinal agenesis is similar to our case but the anatomical region and multiple anomalies are different.

Pancreatic congenital agenesis in human medicine is rarely reported as the absence of the dorsal part of the pancreas (Gold 1993; Schnedl et al. 2009). Among the anomalies seen is the division of the pancreas followed by the ectopic pancreas (Schnedl et al. 2009). In the agenesis of the dorsal pancreas, the ventral part is accompanied by hypertrophy (Gold 1993). No reported cases of congenital agenesis of the pancreas were encountered in veterinary medicine.

Gastro-intestinal perforations in humans and animals are usually caused by gastric dilatation and volvulus, obstruction, blunt abdominal and piercing abdominal trauma, and intestinal changes (Boysen et al. 2003; Hinton et al. 2002). Acute obstruction in the intestines is mostly found in duodenum and jejunum, while chronic obstruction is found in the ileum in the large intestine (Jubb et al. 1993). Intestinal perforation can be caused by an extrinsic obstruction such as external ileus (Blikslager et al., 1992), abscess, peritonitis, as well as occlusion of intestinal lumen mechanically by foreign body (Hayes 2009), parasite (Beyazit and Selver 2011), enterolith, phytobezoar and trichobezoar (Barrs et al. 1999). There are cases of gastro-intestinal perforation reported in zebra (Beyazit and Selver 2011), horse (Hawkins et al. 1993), cats and dogs (Boysen et al. 2003; Hinton et al. 2002). Gastro-intestinal perforation is a common cause of septic peritonitis and is a condition that should be urgently intervened (Boysen et al. 2003; Hayes 2009; Hinton et al. 2002). In our case; Similar to gastro-intestinal perforations observed in humans and animals, it is septic peritonitis that can only occur when the stomach intestinal contents are poured into the abdominal cavity and the animal can live for several days. The separated points were not a perforation of the disease but duodenal agenesis.

While gastro-intestinal perforation may differ clinically from perforation, it often appears as fatigue, loss of appetite, vomiting, and food intake. The signs and symptoms of mucous membranes are jaundice, difficulty in breathing, abdominal pain, decrease in albumin level, white cell number and variability in liver enzymes and

thrombocytopenia (Boysen et al. 2003; Braun 2016; Van Metre et al. 2005). Hypochloremia, hyponatremia, hypokalemia and hyperglycemia are frequently seen in serum biochemistry in cases of gastro-intestinal perforation (Van Metre et al. 2005). In the blood examination, low hemoglobin values and high leukocyte, lymphocyte and monocyte cells initially suggested that it was a septic infection. However, necropsy has also been shown to have duodenal agenesis and not associated with infection.

As a result; If there are complaints, anorexia, weakness, vomiting and defecation in the calves for a few days, it is necessary to consider intestinal atresia, intestinal perforations as well as partial agenesis in the intestines, which are more important than these. In addition, abdominal radiography should be used, and if the animal is dead, it is concluded that determining the cause of clinical findings by performing necropsy will be enlightening for later cases

## CONFLICT of INTEREST

The authors declare that they have no conflict of interest.

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