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Various degrees of ectopic viscera in new born puppies of three years old Pomeranian bitch

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Abstract

This report describes the diagnostic evaluation of a day-old Pomeranian puppies with the anamnesis of ectopic viscera along with eventration, a rare congenital syndrome in puppies. It is characterized by a ventral midline closure defect. The reports proposed a familial inheritance in animals. Ectopic viscera commonly harbors a poor prognosis, posing an ethical and surgical dilemma whereas eventration has a better prognosis when corrected surgically. In this study, a total of three puppies of the same litter were studied. One had thoracic Ectopia cordis due to partial sternal agenesis, liver was located extra-abdominally (*Ectopia hepatica*) and peritoneal layer was intact but the intestines protruded out (eventration), due to agenesis of abdominal muscles. It also had fully developed Diaphragm and no abnormalities were noticed in the thoracic and abdominal organs and other two puppies were reported with mild degree of eventration. All the puppies were surgically treated and the prognosis was recorded.

Keywords: Canine, ectopic viscera and eventration

Introduction

Multiple congenital abnormalities are less common in companion animals. Etiology of congenital anomalies is considered to be caused by genetic factors, mutations, chromosomal abnormalities, infectious and environmental factors (Maas *et al.*, 2009) [2]. Ectopia is a congenital anomaly defined as an organ located anatomically in another part of the body or in another organ or tissue (Ozdemir-salci ES *et al.*, 2024) [3]. It results from abnormal development of the mesoderm during early embryogenesis which is characterized by failure of proper development of the transverse septum, sternum, diaphragm, pericardium, and abdominal wall during embryogenesis (Floek *et al.*, 2008) [1]. The condition results from a failure of mesodermal differentiation and migration between 14-18 days of embryonic development (Parvari *et al.*, 1996) [4] as it affects the developmental field of the ventral midline, leading to the incomplete formation of transverse septum (diaphragm formation), somatic mesoderm (body wall closure) and splanchnic mesoderm (cardiac development) (Snowball *et al.*, 2015) [7].

Ectopia cordis is the presence of the heart partially or totally outside the thoracic cavity (Floek *et al.*, 2008) [1]. Similarly, *Ectopia hepatica* is an anomaly characterized by different location of the hepatic parenchyma (Ozdemir-salci ES *et al.*, 2024) [3]. According to the position of the heart, ectopia cordis is classified as Cervical, cervico-thoracic, thoracic, thoraco-abdominal and abdominal (Floek *et al.*, 2008) [1]. In cases of *Ectopia hepatica*, a small liver tissue may be attached to the gallbladder, intraabdominal organs and ligaments separately, or a small accessory liver lobe may be seen in different localization (Ozdemir-salci ES *et al.*, 2024) [3]. The reported sternum anomalies include branched xiphoid process, V-shaped bifurcation, *Sternum bifidum*, *Synchondrosis sternum*, wedge-Shaped or asymmetric sternum, *Sternum gallinaceum* and sternal Foramen (Snowball *et al.*, 2015) [7]. *Fissura abdominalis* is a defect caused by the absence of complete fusion of the abdominal wall in the median line (Floek *et al.*, 2008) [1].

These defects include a spectrum of malformations from incomplete xiphoid to thoraco-abdominoschisis with the latter commonly observed with exposed internal viscera and is thus lethal to the fetus's survival and resulting in pregnancy loss (So *et al.*, 2023) [8].

Three puppies in a litter of six were born with variably severe midline defects (thoraco-abdominoschisis), including *Ectopia cordis*, *Ectopia hepatica*, partial agenesis of sternum and agenesis of abdominal muscles leading to extrusion of abdominal viscera in one of them. The three other puppies in the litter were born alive without any gross congenital defects, according to the owner's report.

According to the owner's report, the dam was approximately three years old at the birth of this litter. The dam had its first litter of 4 puppies which included one of the puppy was born with these features of mid-ventral defects and the puppy had survived for a day and then it succumbed. The consecutive pregnancy of the dam was reported with the incidence of inbreeding. The incidence of congenital mid-ventral defects has been increased in these littermates.

Materials and Methods

Under local analgesia and physical restraint, the puppy with ectopia cordis, *Ectopia hepatica*, and agenesis of sternum and abdominal muscles was positioned in dorsal recumbency. The viscera (liver, small intestine, large intestine, stomach) were lavaged with normal saline and the heart was repositioned back to thorax and the diaphragm was sutured with thoracic muscles. Abdominal organs were separated from the lesions. Hepatic lobectomy was performed by ligating the supplying vessels. Stomach was deflated using sterile 24G needle by aspiration of stomach contents. Leakage was checked and lavaged. Systematic repositioning of organs (liver, stomach, small intestine, and large intestine) was done. The abdominal wall was opposed by interlocking suture pattern with 3-0 PGA suture material.

The two other puppies were surgically operated under local analgesia and physical restraint. The puppies were positioned in dorsal recumbency. The abdominal viscera (intestines) were lavaged with normal saline. The abdominal wall was sutured after reducing the intestinal loops.

The diagnosed Pentalogy of Cantrell and a novel maternally inherited copy number variant were detected by Chromosome Microarray analysis. Among the genes within the duplicated region is *ALDH1A2* which has previously been demonstrated in animal models to have a strong association with congenital heart disease and diaphragmatic hernia, the two elements comprising the Pentalogy of Cantrell (Steiner *et al.*, 2013) [5]. It is possible that perturbation of retinoic acid levels during development, secondary to this microduplication could underlie the pathology.

ALDH1A2 encodes the enzyme Retinaldehyde dehydrogenase type 2 (Steiner *et al.*, 2013) [5]. Retinaldehyde dehydrogenase type 2 is critical for the conversion of Vitamin A into all trans-retinoic acid, which are morphogen that is known in vertebrate models to be integral for the normal cardiac development and diaphragm formation in the early stages of gestation (Maas *et al.*, 2009) [2]. Retinoic acid is essential for axial patterning and later development in organogenesis.

Retinoic acid cannot be synthesized *de novo* by animals (Maas *et al.*, 2009) [2] and must be obtained from preformed dietary Vitamin A, which is then bound to retinol binding protein, which is then transported into the cells by STRA-6, a membrane receptor (Steiner *et al.* 2013) [5].

Within the cell, retinol is first oxidized to retinaldehyde, which is then further oxidized to retinoic acid. The oxidation to retinoic acid is catalyzed by the retinaldehyde dehydrogenase family of three enzymes. Of these three,

retinaldehyde dehydrogenase type 2 is the major form involved in early embryonic and cardiac development.



Fig 1: Picture showing puppies with various degrees of ectopic viscera



Fig 2: Picture showing a puppy with severe degree of ectopic viscera along with eventration



Fig 3: Picture showing a puppy with mild degree of ectopic viscera



Fig 4: Picture showing hepatic lobectomy



Fig 5: Picture showing lobectomized liver lobes



Fig 6: Picture showing aspiration of stomach content using sterile 24G needle (2 ml syringe)



Fig 7: Picture showing apposition of abdominal wall by interlocking suture pattern with 3-0 PGA suture material



Fig 8: Picture showing the puppies in incubator post-operatively

Results and Discussion

The 14-18 days of embryological period is associated with failed progression of the lateral mesoderm into the transverse septum of the diaphragm, which has been implicated to underlie the Pentalogy of Cantrell (Parvari *et al.*, 1996) [4]. Studies reveal that retinoids play a key role in the formation of pleuroperitoneal fold, the embryonic structure that gives rise to diaphragm, in which deficiency of retinoic acid leads to diaphragmatic hernia (Steiner *et al.*, 2013) [5]. Cardiac malposition has also been suggested to have caused by a mesodermal defect resulting in partial or complete failure of septum-transversum development (Floek *et al.*, 2008) [1]. Septum transversum arises from the mesodermal tissues that lies in the region of the thoracic cavity and the yolk sac on the ventral embryonic body wall.

Gene mutations including those affecting the Wnt/ β -catenin pathway that is critical for midline development such as mutations in the Porcupine O-acyltransferase (PORCN) gene have been linked to this condition in humans and puppies suggesting a link between them (Snowball *et al.*, 2015) [7], (Maas *et al.*, 2009) [2]. It is currently re-mapped to Xq25-q26.1 and to Goltz-Gorlin Syndrome (Xp11.23 loci) that have rarely occurred concurrently in some cases of POC. Goltz-Gorlin Syndrome is an X-linked human autosomal dominant disorder, caused by a mutation in the PORCN gene (Smigielski *et al.*, 2011) [6]. This gene belongs to the Wnt signaling pathway, which interestingly involves the development of the sternum and the thoracic body wall.

The vertebrate heart is particularly sensitive to variations in retinoic acid signaling (Maas *et al.*, 2009) [2]. Avian embryos subjected to Vitamin A deficiency have been used to model Vitamin A effects on very early heart development (Maas *et al.*, 2009) [2].

The congenital developmental diaphragmatic defects in these puppies were unusual when compared with the normal communication of peritoneal cavity with pericardial cavity. The puppies with smaller defects without severe gross eventration have good prognosis post-operatively (So *et al.*, 2023) [8]. The one puppy that had severe mid-ventral defects which had been surgically intervened, survived six hours from birth. The two other puppies survived 72 hours from birth. The detailed examination of the internal heart was unfortunately not possible in either of the affected cases, due to very small size.

Conflict of Interest

Not available

Financial Support

Not available

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