Peritoneopericardial diaphragmatic hernia in the dog and cat

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Peritoneopericardial diaphragmatic hernia in the dog and cat

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ABSTRACT. Peritoneopericardial diaphragmatic hernia (PPDH) is a congenital communication between the pericardial sac and the abdominal cavity allowing displacement of abdominal organs to the pericardial sac. Peritoneopericardial diaphragmatic hernia is thought to occur due to a failure of the development of septum transversum. Vomiting, exercise intolerance and respiratory distress are the most common clinical signs. Diagnosis of PPDH is based on plain radiography. Physical examination and diagnostic imaging may detect the presence of other congenital anomalies. Surgical repair of the PPDH is the treatment of choice for animals with clinical signs. Prognosis following surgical repair of PPDH is favorable. Animals with no clinical signs related to PPDH may not undergo surgical repair.

Keywords: Cat, dog, peritoneopericardial diaphragmatic hernia

Περιτονεοπερικαρδιοδιαφραγματοκήλη στο σκύλο και τη γάτα

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ΠΕΡΙΛΗΨΗ. Η περιτονεοπερικαρδιοδιαφραγματοκήλη (ΠΠΔΚ) είναι μια συγγενής διαμαρτία που φέρει σε επικοινωνία τον περικαρδικό σάκο με την κοιλιακή κοιλότητα και μεταφέρει οργάνους από την κοιλιακή κοιλότητα στον περικαρδικό σάκο. Η ΠΠΔΚ οφείλεται σε αποτυχία ανάπτυξης του διαφράγματος. Η συμπτωματολογία της περιλαμβάνει συχνά έμετα, εύκολη κόπωση και αναπνευστική δυσχέρεια και η διάγνωση γίνεται με απλή ακτινογραφία. Η κλινική εξέταση και ο απεικονιστικός έλεγχος μπορεί να καταδείξουν τη συνύπαρξη άλλων συγγενών διαμαρτιών. Σε ζώα με συμπτώματα η χειρουργική θεραπεία είναι η θεραπεία εκλογής για την ΠΠΔΚ. Η πρόγνωση μετά την επέμβαση είναι καλή, εκτός αν αναπτύχθηκαν συμπτώματα άλλων συγγενών διαμαρτιών. Τα ασυμπτωματικά ζώα μπορεί να επιβιώσουν χωρίς να υποστούν χειρουργική αποκατάσταση της κήλης.

Keywords: Γάτα, σκύλος, περιτονεοπερικαρδιοδιαφραγματοκήλη
INTRODUCTION

Peritoneopericardial diaphragmatic hernia (PPDH) is a congenital communication between the pericardial sac and the abdominal cavity allowing displacement of abdominal organs to the pericardial sac (McClaran, 2013). Peritoneopericardial diaphragmatic hernia is the most common congenital anomaly affecting the pericardium and the diaphragm in small animals (Dettweiler et al., 1960, Clinton, 1967, Evans and Biery, 1980, Wallace et al., 1992, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). Organs more commonly found within the pericardial sac include the liver, small intestine, gallbladder, omentum, spleen, colon, pancreas and falciform ligament (Evans and Biery, 1980, Wallace et al., 1992, Neiger, 1996, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013).

EMBRYOLOGICAL DEVELOPMENT

Peritoneopericardial diaphragmatic hernia is thought to occur due to failure of the development of septum transversum, the structure that constructs the ventral diaphragm (Fig. 1). Failure of closure of the septum transversum or failure of fusion of the septum transversum and pleuroperitoneal folds are the proposed mechanisms for PPDH development. Possible causes of failure include genetic defects, prenatal injury and teratogenic agents (Clinton, 1967, Evans and Biery, 1980, Reimer at al., 2004).

DEMOGRAPHIC DATA

Peritoneopericardial diaphragmatic hernia is an uncommon anomaly in dogs and cats accounting for 0.025% of the small animal population in two veterinary teaching hospitals (Burns et al., 2013), that occurs more commonly in cats than in dogs (Reed, 1951, Evans and Biery, 1980, Willard and Aronson, 1981, Wallace et al., 1992, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). Breeds that are overrepresented include Weimeraners, domestic longhair and Persian cats (Evans and Biery, 1980, Cowan and Feeney 1989, Lunney, 1992, Reimer at al., 2004, Rexing and Coolman, 2004, Chalkey et al., 2006, Banz and Gottfried, 2010, Burns et al., 2013). Median age at diagnosis is 14.4 months for dogs and 30 months for cats (Wallace et al., 1992, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). No sex predilection is reported.

CLINICAL SIGNS AND PHYSICAL EXAMINATION FINDINGS

The presence of clinical signs may depend on the degree of herniation (Burns et al., 2013). In large diaphragmatic defects abdominal organ displacement into the pericardial sac may result in adhesion formation of the organs to the pericardium, organ entrapment or strangulation leading to gastrointestinal, respiratory or cardiac disorders (Burns et al., 2013, McClaran 2013). Small defects which do not allow organ protrusion may be asymptomatic (Burns et al., 2013). The most common clinical signs are anorexia, vomiting, exercise intolerance, dyspnoea or tachypnoea (Burns et al., 2013); other signs include lethargy, weight loss, diarrhoea, coughing, or collapse (Evans and Biery, 1980, Wallace et al., 1992, Neiger, 1996, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). On physical examination the most common findings are muffled heart sounds, heart murmurs or decreased lung sounds (Burns et al., 2013); other findings include thoracic borborygmi or an empty abdomen on palpation (Evans and Biery, 1980, Wallace et al., 1992, Neiger, 1996, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). However, many animals are asymptomatic (Evans and Biery, 1980, Wallace et al., 1992, Neiger, 1996, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). Concurrent conditions seen in cats include cardiomyopathy, renal failure, respiratory tract infection, pericardial cyst and effusion, polycystic kidney disease, hyperthyroidism, urinary tract obstruction, chylothorax, seizures, diabetes mellitus, linear foreign bodies, intussusception, inflammatory bowel disease, gastroenteritis (Banz and Gottfried, 2010, Reimer at al., 2004). Pregnancy and cholelithiasis were reported in two dogs with PPDH (Rosenstein et al., 2001, Statz et al., 2007). The most common haematological finding detected in dogs and cats was elevated serum alanine aminotransferase activity and the most common finding seen in cats was increased serum calcium concentration (Banz and Gottfried, 2010, Burns et al., 2013). Other nonspecific laboratory abnormalities observed in dogs were elevated serum alkaline phosphatase activity, neutrophilia, elevated total bilirubin, increased packed cell volume and decreased platelet count (Banz and Gottfried, 2010). Nonspecific laboratory abnormalities detected in cats include anaemia, mild hyperglycaemia, mildly increased creatinine kinase, mildly increased alkaline phosphatase and aspartate aminotranferase,
elevated creatinine and bilirubin, eosinophilia, neutrophilia, hyperkalaemia and hypocholesterolaemia (Banz and Gottfried, 2010). The diagnosis of PPDH was an incidental finding in 50% of the cats and 46% of the dogs of a recent study during diagnostic imaging performed for unrelated reasons (Burns et al., 2013).

DIAGNOSTIC IMAGING

Diagnosis of PPDH is usually based on plain radiography. Thoracic radiography may detect an enlarged cardiac silhouette or cardiomegaly, presence of abdominal organs within the pericardial sac or loss of distinction between the heart and the diaphragm (Evans and Biery, 1980, Wallace et al., 1992, Neiger, 1996, Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013) [Figs. 2, 3, 4]. Barium studies to aid in identification of intestinal loops within the pericardial sac may also be performed. Ultrasonographic examination, echocardiography and computed tomography may be performed to aid in diagnosis and to rule out any other congenital anomalies (Lamb et al., 1989, Hay et al., 1989, Wallace et al., 1992, Neiger, 1996, Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013).

OTHER CONGENITAL ANOMALIES

Physical examination and diagnostic imaging may detect the presence of other congenital anomalies affecting mostly the abdominal wall, skeleton, heart and vessels (Oyster et al., 1977, Evans and Biery, 1980, Bellah et al., 1989, Wallace et al., 1992, Lunney, 1992, Neiger, 1996, Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). A list of other congenital anomalies identified in dogs and cats with PPDH are presented in Table 1.
SURGICAL TREATMENT

Surgical repair of the PPDH is the treatment of choice for animals with clinical signs. Surgery is performed under general gas anaesthesia and intermittent positive pressure ventilation. Premedication and anaesthetic induction can be performed using routine protocols. The hernia is approached through a ventral midline celiotomy (Figs. 5, 6). In most of the cases entrance to the pleural cavity is not required. Debridement of the hernia margins will lead to entrance to the pleural cavity. The defect can be closed by apposition of the diaphragmatic margins in one or two layer closure using a simple interrupted or continuous pattern with polydioxanone, polypropylene or nylon suture material, from a dorsal to ventral direction (Bellah et al., 1989, Wallace et al., 1992, Banz and Gottfried, 2010, Burns et al., 2013) [Fig. 7]. A caudal median sternotomy is required to improve surgical access and when adhesions are present between the liver or intestines and the pericardium (Burns et al., 2013). Adhesions of the herniated organs to the pericardium may necessitate partial pericardiectomy (Banz and Gottfried, 2010). Reconstruction of the diaphragm with pericardial flaps or grafts can also be done in cases of large defects or in agenesis of the diaphragm (Burns et al., 2013, McClaran 2013). A thoracostomy tube is placed if the pleural cavity is entered; thoracostomy tube is removed shortly after recovery from anaesthesia or within 12 hours after surgery (Banz and Gottfried, 2010). Median time for tube removal is 22.5 hours (Burns et al., 2013). Management of pneumopericardium is usually not required unless cardiopulmonary function is affected associated with large volumes of air accumulation within the pericardial sac (Papazoglou et al., 2015).

INTRAOPERATIVE COMPLICATIONS

Intraoperative complications are observed mostly in cats including haemorrhage associated with adhesions of the liver to the pericardium or myocardium, hypotension, respiratory acidosis, hypventilation, hypoxia, loss of palpable pulses and multifocal ventricular premature contractions (Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). Blood transfusion is performed for the...
management of haemorrhage. Hypotension is treated using intravenous fluids and inotropic drugs. Oxygen is delivered for the management of respiratory complications.

**POSTOPERATIVE COMPLICATIONS**

Postoperative care is usually performed in the critical care unit; animals are under continuous cardiorespiratory monitoring and receive opioids for pain management and oxygen via a face mask or intranasal catheter in case of poor oxygenation. Postoperative complications are detected in both cats and dogs. Major complications in cats include hyperthermia, tachypnoea, dyspnoea, hypoventilation, persistent acidaemia, hypoxia, refractory pneumothorax, pleural effusion, pulmonary oedema, respiratory arrest, chylothorax, incisional dehiscence, hernia recurrence and partial blindness (Wallace et al., 1992, Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013). Dogs suffer fewer postop-

| Dogs                             | Cats                             |
|----------------------------------|----------------------------------|
| **Abdominal wall**               | **Abdominal wall**               |
| Ventral abdominal hernia         | Ventral abdominal hernia         |
| Umbilical hernia                 | Umbilical hernia                 |
| **Sternum**                      | **Sternum**                      |
| Pectus excavatum                 | Pectus excavatum                 |
|Incomplete xyphoid               | Sternaeae fusion                 |
| Sternal defects                  | Sternal defects                  |
| **Ribs**                         | **Ribs**                         |
| Costochondral deformities        |                                 |
| **Genital system**               | **Genital system**               |
| Cryptorchidism                   | Cryptorchidism                   |
| **Head and Neck**                | **Head and Neck**                |
| Cleft palate                     | Prognathism                      |
| Stenotic nares                   |                                 |
| **Eyes**                         | **Eyes**                         |
| Persistent pupillary membranes   | Eyelid atresia and microphthalmia|
| **Cardiovascular**               | **Cardiovascular**               |
| Pulmonic stenosis                | Pulmonic stenosis                |
| Subaortic stenosis               | Mitral stenosis                  |
| Ventricular septal defects       | Atrial septal defect             |
| Persistent left cranial vena cava| Double chambered right ventricle with pulmonic stenosis|
| Portoazygous shunt               | Pseudotruncus arteriosus         |

Table 1. Congenital anomalies that are identified in dogs and cats with PPDH (Eyster et al., 1977, Evans and Biery, 1980, Bellah et al., 1989, Wallace et al., 1992, Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013).
operative complications including incisional dehiscence, regurgitation, pericardial steatitis or pneumothorax (Wallace et al., 1992, Banz and Gottfried, 2010, Burns et al., 2013). Hyperthermia usually resolves within 12 hours of detection (Banz and Gottfried, 2010). Oxygen delivery is often required to combat respiratory complications. Short-term postoperative mortality ranged from 3-14% for cats (Reimer et al., 2004, Banz and Gottfried, 2010, Burns et al., 2013) and from 5-12.5% for dogs (Banz and Gottfried, 2010, Burns et al., 2013).

PROGNOSIS

Prognosis following surgical repair of PPDH is usually good with 81-87.5% of the dogs and 86-97% of the cats survive (Evans and Biery, 1980, Reimer at al., 2004, Banz and Gottfried, 2010, Burns et al., 2013).

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MANAGEMENT OF ASYMPTOMATIC HERNIAS

Animals with no clinical signs or with fewer clinical signs related to PPDH may not undergo surgical repair (Reimer at al., 2004, Burns et al., 2013). Monitoring by the owners for respiratory or gastrointestinal signs has been reported in these cases. The median age of these animals was significantly greater than the median age of animals that had surgical reconstruction of PPDH (Burns et al., 2013). Interestingly, long-term survival was not different between animals undergoing surgical repair and those without (Burns et al., 2013).