DIAGNOSIS AND TREATMENT OF A PERITONEOPERICARDIAL HERNIA IN AN ADULT DOG: A CASE REPORT

SUMMARY

Peritoneopericardial diaphragmatic hernia is a common congenital pericardial anomaly in dogs and cats, characterized by a communication between the abdomen and the pericardial sac. Animals may be asymptomatic or show nonspecific clinical signs related to the gastrointestinal and cardiorespiratory systems. In this report, we present a case of a 3-year-old female Schnauzer, weighing 7.7 kg, with a history of easy fatigue and cyanosis triggered by stress. The diagnosis of peritoneopericardial diaphragmatic hernia was confirmed through echocardiography and simple and contrast radiography findings that showed the presence of hepatic lobes in the pericardial sac. The animal underwent supra-umbilical celiotomy to correct the hernia and subsequently presented immediate improvement of clinical signs.


RESUMO

A hérnia diafragmática peritoniopericárdica é uma anomalia congênita pericárdica comum em cães e gatos, sendo caracterizada pela comunicação entre abdômen e saco pericárdico. Os animais podem ser assintomáticos ou apresentar sinais clínicos inespecíficos relacionados aos sistemas gastrointestinal e cardiorrespiratório. Expõe-se um caso de um cão Schnauzer, fêmea, 3 anos de idade, pesando 7,7 kg, com histórico de cansaço fácil e cianose em momentos de estresse. O diagnóstico de hérnia peritoniopericárdica foi confirmado por meio de ecocardiografia e exame radiográfico simples e contrastado que evidenciaram a presença de lobos hepáticos no saco pericárdico. O animal foi submetido a celiotomia pré-umbilical para correção do defeito, apresentando melhora imediata dos sinais clínicos após a correção cirúrgica.

INTRODUCTION

Diaphragmatic hernias may be congenital or acquired, the latter being rare in dogs and cats. Peritoneopericardial diaphragmatic hernia (PPDH) is the most common cause of congenital pericardial anomaly in dogs and cats (PEREIRA and LARSSON, 2015). It originates from defects in diaphragmatic embryogenesis that results in incomplete development of the pleuroperitoneal folds or transverse septum (PARK, 2002).

PPDH is characterized by a communication between the abdomen and pericardial sac (FOSSUM, 2014), allowing migration of some abdominal organs into the pericardium. The most common migratory organs are hepatic lobes, intestinal loops, the spleen, and the stomach. Although it is not certain, its etiology may be genetic and associated with the occurrence of congenital heart diseases (PEREIRA and LARSSON, 2015). Some breeds have higher predisposition for occurrence of the disease, like Weimaraner and Cocker Spaniel dogs, and Himalayan and long-haired cats (REIMER et al., 2004; FOSSUM, 2008).

Animals with PPDH are generally asymptomatic, and clinical manifestations are directly related to severity of the diaphragm defect and the specific organs involved. When clinical signs such as vomiting, diarrhea, weight loss, tachypnea, dyspnea, exercise intolerance, syncope, and cough are present, they are nonspecific and may be related to the gastrointestinal and cardiorespiratory systems. Cardiorespiratory signs may appear under conditions of stress and excitement (FOSSUM, 2014; PEREIRA and LARSSON, 2015).

Diagnosis of PPDH should be based on anamnesis, physical examination, electrocardiography, and complementary imaging tests such as simple contrast radiography, ultrasonography, and echocardiography. Radiographic signs that may be indicative of PPDH include increased cardiac silhouette, dorsal elevation of the trachea, overlapping of the heart with the diaphragmatic borders, and structures filled with gas in the pericardial sac (FOSSUM, 2014). Treatment consists of surgical correction; however, corrective surgery is not recommended for asymptomatic elderly animals or for patients who do not present organs herniated into the pericardium that may be strangulated, such as the omentum (PEREIRA and LARSSON, 2015).

CASE REPORT

A three-year-old female Schnauzer weighing 7.7 kg was brought to the Governador Laudo Natel Veterinary Hospital, FCAV/UNESP, Jaboticabal Campus, for treatment. The owner reported hematuria, easy fatigue, and mild cyanosis under exciting situation, such as arrival of visitors or before walks. There was no history of cough, syncope, and convulsions. Hematuria and cardiorespiratory signs were reported to have started 10 days and 18 months before examination, respectively, with slow progressive increase in frequency. At physical examination, physiological parameters were normal. Auscultation revealed muffled cardiac sounds in the left hemithorax. A midline defect in the abdominal musculature was palpated just distal to the xiphoid cartilage.

Complete blood count and serum biochemistry test results showed normal for the species. Urinalysis through cystocentesis showed bacteriuria and the presence of leukocytes, suggestive of bacterial cystitis, which was confirmed through isolation of coagulase-positive and cephalosporin-sensitive Staphylococcus sp. in urine culture.

The patient was referred for chest radiography, which indicated increased cardiac silhouette, overlapping of the heart with the diaphragmatic borders, and discontinuity of the diaphragm. The chest X-ray image findings suggested PPDH (Figure 1A and 1B). Abdominal radiography showed the presence of bladder stones.

Electrocardiography revealed sinus arrhythmia with episodic migratory pacemaker and heart rate varying between 94 and 125 bpm with prolonged QRS complex duration (63 ms), indicative of left ventricular overload, and T wave larger than 25% of R wave, suggestive of hypoxia and/or electrolyte imbalance. In addition, echocardiography of the right parasternal window showed the presence of parenchymal organ similar to the hepatic lobe surrounding the right ventricle and part of the left ventricle (Figure 2A and 2B). Nevertheless, the heart showed no remodeling and no hemodynamic repercussion during examination. Contrast radiography of the gastrointestinal tract using 100% barium sulfate (11 mL/kg, Bariogel®, Cristália Produtos Químicos Farmacêuticos, Itapira, Brazil) through nasogastric tube was used to confirm non-herniation of the stomach or intestinal loops, followed by surgery to remove uroliths and correct the PPDH.

The anesthetic protocol comprised tramadol hydrochloride (Tramal®, Pfizer, New York, USA), 4 mg/kg, IM, as a preanesthetic sedative; propofol (Propovan®, Cristália Produtos Químicos Farmacêuticos, Itapira, Brazil), 5 mg/kg, IV, for induction of anesthesia; isoflurane 3% (Forane®, Abbott, São Paulo, Brazil) for maintenance of anesthesia. Under dorsal decubitus positioning, the abdomen and caudal two-thirds of the thoracic cavity were prepared for aseptic surgery. Ventral midline abdominal skin incision was made in the retroumbilical region to access the urinary bladder. The uroliths were removed and subsequently sent for laboratory analysis. The incision was sutured. In the second part of the surgery, an abdominal midline incision was made in the pre-umbilical region and section of the abdominal muscles to access the diaphragmatic dome. The diaphragm defect (Figure 3A) showed herniation of the quadrates and left medial hepatic lobes, as well as partial herniation of the gallbladder (Figure 3B). The organs without adhesions were carefully reduced into the abdominal cavity (Figure 3C). The edges of the diaphragmatic defect were debrided with a scalpel blade and approximated with a simple continuous suture using synthetic contrast radiography, ultrasonography, and echocardiography. Radiographic signs that may be indicative of PPDH include increased cardiac silhouette, dorsal elevation of the trachea, overlapping of the heart with the diaphragmatic borders, and structures filled with gas in the pericardial sac (FOSSUM, 2014). Treatment consists of surgical correction; however, corrective surgery is not recommended for asymptomatic elderly animals or for patients who do not present organs herniated into the pericardium that may be strangulated, such as the omentum (PEREIRA and LARSSON, 2015).
nonabsorbable suture material (Figure 3D). The excess of pericardial sac was removed, and a small remaining defect was corrected using omentalization. The remaining air was evacuated from the pericardium via pericardiocentesis through the diaphragm, using a closed system with a three-way valve coupled to a syringe and a scalp vein set. Finally, the musculature was closed with Sultan suture using synthetic absorbable suture material (Caprol® 2-0, Ethicon, New Jersey, EUA). Subcutaneous tissues were closed with continuous zig-zag suture using synthetic absorbable suture material (Caprol® 3-0, Ethicon, New Jersey, EUA). The skin was closed with simple interrupted suture using synthetic nonabsorbable suture material (Mononylon® 3-0, Ethicon, New Jersey, EUA).

![FIGURE 1 - Pre- and postoperative simple and contrast radiography images in latero-lateral (A), ventral-dorsal (B), latero-lateral (C), and ventral-dorsal (D) positions. (A) Contrast radiography image after oral administration of barium showing loss of definition between the diaphragmatic dome and the heart (red arrow), and absence of stomach and intestines in the pericardial sac. (B) Contrast radiography image showing increased cardiac silhouette and the presence of parenchymal organ in the pericardial sac displacing the heart (yellow arrows). (C) Simple postoperative radiography of peritoneopericardial herniorrhaphy with definite limitation between the diaphragmatic dome and cardiac silhouette (black arrow). (D) Postoperative radiography image of peritoneopericardial herniorrhaphy with restoration of the cardiac silhouette (green arrow) and well-defined limitation between the thoracic and abdominal cavities (black arrow).](image)

Postanesthetic medication included dipyrone 25 mg/kg, SC (D-500®, Zoetis, Campinas, São Paulo, Brazil), tramadol hydrochloride 4 mg/kg, SC (Medley, Campinas, São Paulo, Brazil), and meloxicam 0.1 mg/kg, SC (Eurofarma, Itapevi, São Paulo, Brazil). The patient underwent postoperative chest radiography, which showed complete correction of the defect (Figure 1C and 1D). Postoperative medication included cephalexin 30 mg/kg, VO, q.12 h, for 21 days (EMS, Hortolândia, São Paulo, Brazil); tramadol hydrochloride 1 drop/kg, q.12 h for 7 days (Neo Química, Rio de Janeiro, Brazil); dipyrone 1 drop/kg, q.12 h, for 7 days (Medley, Campinas, São Paulo, Brazil); meloxicam 0.1 mg/kg, VO, q.24 h, for 3 days (Ourofino Saúde Animal, Cravinhos, São Paulo, Brazil); and ranitidine hydrochloride 2.2 mg/kg, VO, q.12 h, for 21 days (Label®, Aché, Guarulhos, São Paulo, Brazil).

At 7-day post-surgery, the patient showed excellent general condition. Sutures were removed 15 days after surgery; at the time, the patient showed no fatigue under stressful situation. Twenty-five days after surgery, urine culture was negative which indicated that antimicrobial therapy was no longer necessary, and the patient was discharged.
FIGURE 2 - Echocardiography images of the right parasternal window of the canine patient showing the presence of parenchymal structure compatible with that of the liver inside the pericardium. (A) Modified longitudinal image of the four heart chambers and parenchymal structure (asterisk) identified near the right ventricle and cardiac apex. (B) Modified transverse image showing parenchymal structure (asterisk) inside the pericardium (arrow). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

FIGURE 3 - Intraoperative photographs of the canine patient in dorsal decubitus showing pre-umbilical celiotomy to correct peritoneopericardial diaphragmatic hernia. The white star shows the cranial side, and the yellow star shows the right side. (A) Image of diaphragmatic defect after skin incision and subcutaneous divulsion (black arrow). (B) Left lateral hepatic lobe herniation (blue arrow) is identified through the defect (black arrow) with visible diaphragmatic borders (white arrow) after completion of celiotomy. (C) Image of the heart covered by visceral pericardium (yellow arrow) and excess parietal pericardium (green arrow) after reduction of the hepatic lobes. (D) Herniorrhaphy after debridement of diaphragmatic borders to close the defect (black arrow) with subsequent removal of excess parietal pericardium (green arrow).
RESULTS AND DISCUSSION

The defect was properly created in all animals, but one that an injury in the distal sis-cortex of the ulna was observed. Moreover, on the 7th postoperative day, this animal presented complete transverse ulna fracture and it was removed from the study. Except for Our patient had no history of trauma, and intraoperatively, adhesions and signs of inflammation and hemorrhagic areas were absent, which suggested the occurrence of congenital PPDH (CHISCO et al., 2016). True diaphragmatic hernias are defined as subtotal diaphragmatic defects in which the serosa on the thoracic surface of the diaphragm remains intact, preventing direct communication between the pleural and peritoneal cavities. It is a congenital defect in which the internal growth of collagen or muscle tissue between the pleura and the peritoneum ceases prematurely (CARIOU et al., 2009).

Hensel (2014) reported that in 46.4% of dogs and 50% of cats, PPDH is an incidental finding; in contrast, in the present case, diagnosis of PPDH was based on clinical signs and results from prospective diagnostic testing. In addition, the author reported that 57.1% of the dogs had congenital anomalies associated with PPDH, such as umbilical hernia, hernia of the umbilical wall cranial to the navel, and sternal anomalies, which corroborates the finding on palpation in our case of midline defect in the abdominal musculature distal to the xiphoid cartilage. Umbilical hernia is present in most cases as well as in the dog reported here (KHEIRANDISH et al., 2014; CÂMARA et al., 2020).

In a retrospective study, Burns et al. (2013) verified prevalence of PPDH in 28 dogs and 30 cats. The mean age at diagnosis for dogs was 1.2 years, ranging from 12 months to 12.3 years, and the most common clinical signs were exercise intolerance, tachypnea, dyspnea, cough, vomiting, and anorexia, which is in agreement with the findings in the present case, despite the difference in the age of our patient compared with the age-range reported by the author. According to Kheirandish et al. (2014) and Smolec et al. (2018), only 6% of animals are diagnosed after eight years of age, while 48% are diagnosed in the first years of life.

A previous study indicated presence of increased alanine amino transferase level (BURNS et al., 2013); however, Fossum (2014) reported that this change is uncommon, and hence, normal laboratory test results in our case were expected. Muffled cardiac sounds at auscultation are common in patients with PPDH, as are ascites, murmurs caused by heart displacement due to the presence of visceral organs, and heart defects. Thoracic radiography findings of increased cardiac silhouette and the heart overlapping with the diaphragmatic borders are useful to identify the disease (VOGES et al., 1997; FOSSUM, 2014). Serial contrast radiography findings contributed in ruling out the presence of intestines or stomach in the pericardial sac (NELIDA and FEIJOÓ, 2012), since the occurrence of stomach herniation, although uncommon, would significantly aggravate the patient’s condition.

Although electrocardiography was not decisive in the diagnosis of PPDH, finding of increased T wave corroborated the patient’s clinical condition of easy fatigue and discreet cyanosis. Echocardiography is a fast, easy, safe, and noninvasive method to diagnose PPDH, through which it was possible to verify the presence of parenchymal tissue near the pericardial sac, differentiate the disease from pericardial effusion or cardiomegaly, and evaluate the patient’s heart condition (DEBIJAK et al., 2009; FOSSUM, 2014). Pereira and Larsson (2015) reported that of the abdominal organs, the liver is at highest risk for herniation; in agreement, our patient showed migration of the hepatic lobes into the pericardial cavity and the presence of pericardial effusion.

Hypotension is expected under diaphragmatic hernia repair, mainly when the viscera are reduced into the abdominal cavity (CLARKE et al., 2014); however, during the anesthetic and surgical protocol, we observed that our patient remained stable throughout the procedure, including during the reduction of herniated organs. Additionally, Clarke et al. (2014) reported intraoperative cardiac arrhythmias in patients with chronic hernias with adhesions. The present case showed no adhesions between the organs or presence of arrhythmias, possibly due to extreme care in organ manipulation during reduction with minimal cardiac manipulation, resulting in a better prognosis. Reimer et al. (2004) reported that hyperthermia is the most common postoperative complication, which was not observed throughout post-operative follow-up in the present case.

Burns et al. reported that surgery is effective to treat clinical signs related to PPDH with small or absent self-limiting postoperative complications (REIMER et al., 2004). Cariou et al. (2009) recommended surgical manipulation to confirm the origin of the diaphragmatic defect and to avoid potential risk of ventilatory compromise and late strangulation and/or necrosis of the herniated abdominal content.

CONCLUSIONS

Correlation of the history, clinical signs, and muffled heart sounds on auscultation with simple and contrast chest radiography and echocardiography findings was effective for confirming the diagnosis of PPDH. Peritoneopericardial herniorrhaphy was useful to correct the defect and treat the patient’s clinical signs without complications.

REFERENCES


