Combined hiatal and pleuroperitoneal hernia in a shar-pei

Jerome M. Auger, Steve M. Riley

Abstract — This article presents an unusual combination of a type IV hiatal hernia and a pleuroperitoneal hernia in a young shar-pei. Pathogenesis, diagnosis, and treatment of both conditions are discussed. At surgery, close examination and palpation of the whole diaphragm are recommended to allow perioperative diagnosis of unexpected defects.

Résumé — Hernie hiatale et pleuropéritonéale combinée chez un Shar-peï. L'article traite de la combinaison inhabituelle d'une hernie hiatale de type IV et d'une hernie pleuropéritonéale chez un jeune Shar-peï. Il est question de la pathogenèse, du diagnostic et du traitement des deux conditions. Lors d'une chirurgie, il est recommandé de procéder à un examen attentif et à la palpation du diaphragme pour permettre le diagnostic de défauts imprévus.

(Traduit par Hélène Petitclerc)

Can Vet J 1997; 38: 640–642

A 13.5 kg, 9-month-old, castrated male shar-pei was presented to the Ontario Veterinary College, Veterinary Teaching Hospital (OVC, VTH) because of chronic vomiting and regurgitation, hypersalivation, and poor body condition. Signs had started approximately 6 mo prior to presentation, shortly after weaning. The vomit consisted of food or, more often, white or, occasionally, yellow-tinged frothy material. The dog had been tried on various diets and it appeared that canned food elicited less postprandial vomiting than did dry food. An endoscopy, performed 6 mo prior to referral by the referring veterinarian, revealed hyperemic esophageal and gastric mucosae. Treatment for gastritis and reflux esophagitis with cimetidine, sucralfate, and metoclopramide had been given, at recommended doses, for several months prior to referral, without significant improvement.

On examination, the dog appeared abnormally thin and was drooling profusely. The dorsal aspect of the front paws was brown-stained, attributable to persistent soiling by saliva. Auscultation of the thorax discerned shallow breathing and decreased lung sounds over the left caudo dorsal thorax. Results from a complete blood cell count, a biochemistry profile, and a urinalysis were within normal limits. Chest radiographs showed a megaesophagus and revealed an oval, thick-walled structure, compatible with the stomach, cranial to the diaphragm in the area of the esophageal hiatus (Figure 1). On the basis of the breed, clinical signs, and radiographic findings, we made a tentative diagnosis of hiatal hernia.

A barium esophagram confirmed the megaesophagus and herniation of a large part of the stomach into the thorax. Fluoroscopy of the barium swallow revealed good esophageal motility and gastroesophageal reflux. The gastroesophageal junction was located several centimeters cranial to the diaphragm, and the gastric fundus protruded into the left thorax to the level of the 7th rib. These findings were characteristic of a combined axial and paraesophageal hiatal hernia (1,2). Medical and surgical options were discussed with the owners, who opted for surgical correction, since an appropriate medical treatment had previously failed to bring about significant improvement.

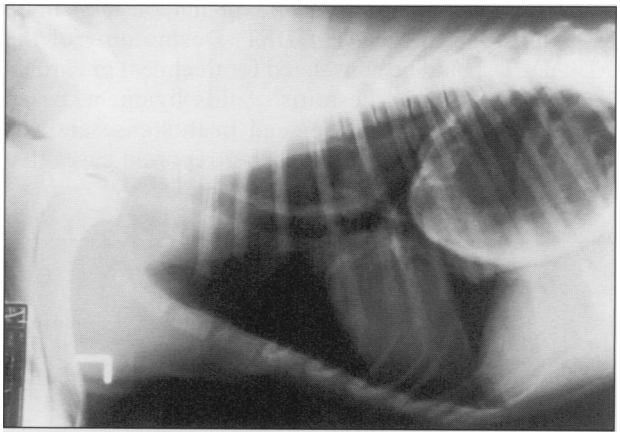

Figure 1. Left lateral thoracic radiograph. Megaesophagus and an air-filled oval structure protruding into the thorax are clearly visible.

The next day, the dog was anesthetized and the skin of the ventral abdominal wall was prepared for a sterile procedure. A cranial midline celiotomy was performed. The cranial part of the abdominal cavity and esophageal hiatus were examined. Reduction of the hernia by gentle traction on the stomach revealed that the left lateral lobe of the liver and the spleen were displaced into the thoracic cavity along with the cardiac and fundic portions of the stomach. These organs were easily pulled from the thorax and repositioned in the abdominal cavity. A membranous hernial sac was present and there was no communication between the abdominal and thoracic

Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1.

Present address for Dr. J.M. Auger: Department of Clinical Sciences, Faculty of Veterinary Medicine, University of Montréal, C.P. 5000, Saint-Hyacinthe, Quebec J2S 7C6.

cavities. The loose hiatus was reduced to an opening 2 cm in diameter, using simple interrupted sutures of 2/0 polypropylene to imbricate the crura of the diaphragm ventral to the esophageal hiatus (2,3). The index finger was used for palpation of the esophagocardiac junction to evaluate the degree of reduction in diameter thus achieved. A fundic belt-loop gastropexy was performed to anchor the stomach to the left abdominal wall and avoid reherniation. Thoracocentesis was performed transdiaphragmatically to restore intrathoracic negative pressure. Closure of the celiotomy was routine.

The immediate postoperative period was uneventful; however, 24 h after surgery, the dog was still salivating abundantly and vomiting more frequently than expected. Thoracic and abdominal radiographs were taken and appeared surprisingly similar to those taken preoperatively; it was debated whether the surgical repair had ruptured and herniation had reoccurred. After further discussion with the owners, a 2nd surgery was scheduled. The previous incision was opened and the belt-loop gastropexy, which was found intact, was released to allow manipulation of the stomach and deeper exploration of the abdominal cavity. The latter revealed that the pyloric portion of the stomach and the pylorus had herniated into the thoracic cavity, not through the esophageal hiatus but through the dorsal or lumbar part of the diaphragm (5), which consisted of a flaccid membrane instead of the muscular layer. During the 1st surgery, palpation of this area had not detected obvious abnormalities, and visual examination was not performed. The location of this diaphragmatic defect corresponded to that of the pleuroperitoneal folds during embryogenesis, and this defines the resulting pleuroperitoneal hernia (6). A stomach tube was inserted orally to keep the cardia open for the next surgical step. The flaccid portion of the diaphragm was plicated using 2/0 polypropylene mattress sutures, dorsal to the esophageal hiatus. Thoracocentesis was performed transdiaphragmatically to restore intrathoracic negative pressure. A 12 cm longitudinal incision was made in the seromuscular layer of the ventral gastric wall, midway between the attachments of the lesser and greater omenta and ending over the pyloric canal. It was then included in the closure of the linea alba using 0 polydioxanone and 2/0 polypropylene simple interrupted sutures to create a permanent midline gastropexy. The rest of the closure was routine.

The dog recovered well from the 2nd surgery and immediate abatement of the clinical signs was observed. Prior to discharge, instructions were given to the owners to feed their animal from an upright position and to watch for signs of aspiration pneumonia. Sucralfate was prescribed at 0.5 g, PO, q8h for 3 wk for possible esophagitis. At recheck 2 wk postoperatively, hypersalivation was no longer present and only occasional vomiting was reported by the owners. Chest films did not reveal any abnormalities. Six months after the procedure, the owners were contacted and they reported that the dog was asymptomatic.

Esophageal hiatal hernia has been reported previously in the dog (2,4,7–12). It is usually a congenital condition; however, acquired hiatal hernia has been described (4,7,12). A predilection to this disorder is suspected in the Chinese shar-pei, since this breed is over-represented in the literature reports. An investigation of the mode of inheritance has not been attempted at this time, probably because most cases are isolated. Four main types of hiatal hernia are described in the human medical literature (1) and have been transposed to the veterinary field. The type I hiatal hernia is a sliding or axial hernia and seems to be, by far, the most common type diagnosed in the dog (3,4,7–9,12). It corresponds to a cranial displacement of the gastroesophageal junction into the thoracic cavity. The type II, rolling or paraesophageal, hiatal hernia corresponds to a herniation of the stomach through an enlarged hiatus while the gastroesophageal junction remains in a nearly normal position. Although they are frequently referred to synonymously, the true paraesophageal hiatal hernia differs from the type II hernia in that it occurs through a separate diaphragmatic defect adjacent to the esophageal hiatus. The type III hiatal hernia is a combination of types I and II. The type IV hernia is a type III hernia complicated by herniation of abdominal organs, in addition to the stomach, into the thoracic cavity. It, therefore, applies to the case described here.

Congenital pleuroperitoneal hernia is rare in small animals (3). One case has recently been reported in a cat (11). Embryological formation and malformation of the diaphragm have been well described by Noden and de Lahunta (6). Pleuroperitoneal hernia results from incomplete closure of the peritoneal canals (dorsal part of the diaphragm) or from failure of the pleuroperitoneal folds to incorporate muscular components of the body wall. In the former situation, an opening persists between the thoracic and abdominal cavities, which leads to rapid death by respiratory insufficiency (3). In the latter, the lumbar part of the diaphragm remains membranous instead of becoming a strong muscular layer. Thus, it acts like a hernial sac, as in the case we are reporting. This was not recognized by palpation at the 1st surgery. It is possible that the corresponding weakened area became significant only after reduction of the abdominal organs and correction of the hiatal hernia.

The history and clinical signs of this dog were highly suggestive of an esophageal hiatal hernia. Clinical signs commonly appear shortly after weaning, at the time of transition from liquid to solid food. Hypersalivation and vomiting or regurgitation, especially in the immediate postprandial period, are reported by most authors (3,4,7–9). Often, poor body condition results from this altered digestive function. Dyspnea and exercise intolerance have also been observed and seem secondary to either aspiration pneumonia or lung compression by the herniated abdominal organs. The risk of fatal complications, such as, gastric volvulus, torsion, obstruction, strangulation, and intrathoracic dilatation, is dreaded in humans with a type II hiatal hernia and prompts surgical correction, even in asymptomatic cases (1). Callan *et al* (8) reported 2 fatal cases with "acute large gastric herniation" in the shar-pei. In our patient, both the esophageal hiatal and pleuroperitoneal hernias probably contributed to the clinical signs; however, it is difficult to determine their respective importance.

An isolated pleuroperitoneal hernia can be diagnosed by paracostal ultrasonography and, possibly,

by positive-contrast peritoneography to outline the diaphragmatic defect (11). Diagnosis of esophageal hiatal hernia is based on history, clinical signs, and survey radiographs of the thorax, which may reveal the presence of a gas-filled, soft tissue structure cranial to the diaphragm, megaesophagus, and masses with a soft tissue density when the spleen or liver have herniated (2,3). Contrast studies with oral administration of barium are necessary to outline the hernia precisely and to diagnose more subtle sliding or type I hernias. Fluoroscopic examination following barium administration should be performed to assess esophageal motility and the degree of gastroesophageal reflux. It is particularly important when dealing with the shar-pei, in which various degrees of esophageal dysfunction have been demonstrated (13). In this breed, an apparently isolated megaesophagus should raise suspicion of an associated esophageal hiatal hernia. Ultrasonography would probably enable identification of the herniated organs. This was not done in our case.

Medical treatment can be attempted in animals with a small sliding hernia. It consists of feeding modifications (upright feeding and change in the consistency of the diet) and the use of antacids, like H_2 agonists (cimetidine, ranitidine) and prokinetic agents (metoclopramide). Bright *et al* (7) recommended that medical treatment be administered for 1 mo before contemplating surgical intervention. However, most authors have reported poor results with medical treatment (4,8,9) and, although it should be attempted, owners should be informed of its low success rate. Another reason for not delaying surgical treatment is the possibility of serious complications, such as aspiration pneumonia or acute worsening of the hernia. Early methods of surgical treatment for canine esophageal hiatal hernia were modelled after their human counterparts and aimed at restoring a competent lower esophageal sphincter (Nissen fundoplication). They have been associated with many complications in the dog and are not recommended (3,4,12). Currently, the advocated surgical treatment consists of hiatal plication (closure of esophageal hiatus to reduce its size to an opening 1 to 2 cm in diameter), best achieved after orally inserting a gastric tube of the appropriate size or using a finger to assess the proper diameter of the hiatus, circumferential esophagopexy, and left flank gastropexy. An esophagopexy was not performed on our patient, which, apparently, did not influence the final outcome. Any gastropexy technique can be used; however, a tube gastropexy also allows enteral feeding and subsequent resting of the esophagus. The midline gastropexy performed at the 2nd surgery was done to provide stabilization of a larger area of the stomach than would have been possible with a belt-loop gastropexy. Very good results have been obtained with the above techniques (3,4,8,12). Moreover, the associated megaesophagus and decreased esophageal motility seem to resolve postoperatively, in most cases. Likewise, the megaesophagus was no longer present at the 2-week re-evaluation of our patient. The megaesophagus observed probably resulted from cranial displacement of the cardia, and it could be more appropriate to refer to it as esophageal dilatation. The medical treatment described above may be given for 1 to 3 wk after surgery.

Congenital pleuroperitoneal hernia is a rare occurrence in the dog. To our knowledge, the combination of this hernia and a type IV hiatal hernia has not been described in the veterinary literature. Multiple degrees of congenital diaphragmatic hernia may be found in the dog; however, congenital absence of portions of the lumbar part of the diaphragm can rapidly be fatal after birth. The defect seen in this dog was probably as severe as it can be in a near-adult animal.

During surgical treatment of a hiatal hernia, the entire diaphragmatic surface should be closely inspected after the herniorraphy has been completed, to allow identification and subsequent closure of any concurrent defect. Finally, the increasing likelihood of a breed predisposition for hiatal hernia in the shar-pei should motivate future genealogical and genetical studies of affected animals. CVJ

References

1. Skinner DB, Belsey RHR. Management of Esophageal Disease. Philadelphia: WB Saunders, 1988: 487–501.
2. Williams JM. Hiatal hernia in a Shar-pei. J Small Anim Pract 1990; 31: 251–254.
3. Johnson KA. Diaphragmatic, pericardial, and hiatal hernia. In: Slatter DH, ed. Textbook of Small Animal Surgery. 2nd ed. vol 1. Philadelphia: WB Saunders, 1993: 455–470.
4. Prymak C, Saunders HM, Washabau RJ. Hiatal hernia repair by restoration and stabilization of normal anatomy: An evaluation in four dogs and one cat. Vet Surg 1989; 18: 386–391.
5. Hermanson JW, Evans HE. The muscular system. In: Evans HE, ed. Miller's Anatomy of the dog, 3rd ed. Philadelphia: WB Saunders, 1993: 258–384.
6. Noden DM, de Lahunta A. The Embryology of Domestic Animals. Baltimore: Williams & Wilkins, 1985: 287–291.
7. Bright RM, Sackman JE, DeNovo C, Toal C. Hiatal hernia in the dog and cat: A retrospective study of 16 cases. J Small Anim Pract 1990; 31: 244–250.
8. Callan MB, Washabau RJ, Saunders HM, Kerr L, Prymak C, Holt D. Congenital esophageal hiatal hernia in the Chinese Shar-pei dog. J Vet Intern Med 1993; 7: 210–215.
9. Ellison GW, Lewis DD, Phillips L, Tarvin GB. Esophageal hiatal hernia in small animals: Literature review and a modified surgical technique. J Am Anim Hosp Assoc 1987; 23: 391–399.
10. Miles KG, Pope ER, Jergens AE. Paraesophageal hiatal hernia and pyloric obstruction in a dog. J Am Vet Med Assoc 1988; 193: 1437–1439.
11. Mann FA, Aronson E, Keller G. Surgical correction of a true congenital pleuroperitoneal diaphragmatic hernia in a cat. J Am Anim Hosp Assoc 1991; 27: 501–507.
12. White RN. A modified technique for surgical repair of oesophageal hiatal herniation in the dog. J Small Anim Pract 1993; 34: 599–603.
13. Stickle R, Sparschu G, Love N, Walshaw R. Radiographic evaluation of esophageal function in Chinese Shar-pei pups. J Am Vet Med Assoc 1992; 201: 81–84.