Asian Clin. Vet. Med., 2018, 1(1): 1-5
ISSN : 2433-2313 (Online)

Case Report

Submit : 17/1/2018 Accept : 12/4/2018 Published : 31/5/2018

A juvenile cat with megaesophagus and a hiatal hernia after diaphragmatic hernia repair

Akiko Uemura, DVM., Pinkarn Chantawong, DVM., Pitipat Kitpipatkun, DVM., Seijirow Goya, DVM., Kazumi Shimada, DVM., Ryou Tanaka*, DVM, Ph.D
*Department of Veterinary Surgery, Faculty of Veterinary Medicine, Tokyo University of Agriculture and Technology

Abstract

A case of a juvenile cat with complications after diaphragmatic hernia repair is reported. In the present case, a diaphragmatic hernia was surgically repaired in a six-month-old intact, male, crossbreed cat. The cat showed vomiting as a clinical symptom from the day after the surgical repair, and megaesophagus and hiatal hernia were found as post-repair complications. The aim was to control the cat's condition with surgical esophago-gastropexy, provision of nutrients through an artificial opening to the stomach, protection of the esophageal mucous membrane, and the application of pro-kinetic drugs. Similar to the few previous reports of megaesophagus and hiatal hernia after surgical repair of diaphragmatic hernia, the clinical symptoms disappeared over a number of weeks following surgery. However, the megaesophagus and hiatal hernia were seen simultaneously in the present case, and the findings are reported based on the progress and transition in clinical symptoms in this animal. Such complications following diaphragmatic hernia repair present difficulties from the viewpoint of nutritional management in young and weak animals, but they may be transient.

Keywords: cat, megaesophagus, hiatal hernia, diaphragmatic hernia, complication

Introduction

Feline diaphragmatic hernia (1) is a relatively frequently observed condition that is classified into acute (2) and chronic (3) forms. Surgical repair is a recommended treatment for both forms, but there is some differences in the recommend treatment theory; the repair should be performed quickly in acute but stable cases (2), but highly urgent surgical repair is regarded as less of a necessity in chronic cases (3). Widely known complications following diaphragmatic hernia repair in cats and dogs include pneumothorax, pulmonary edema, pleural fluid, arrhythmia, and incarceration in the digestive tract (4).

On the other hand, megaesophagus has been reported as the rarer post-repair complications in few reports (5). Megaesophagus is a disorder of the esophagus observed in both dogs and cats and is classified into hereditary and acquired forms. The acquired form is known to occur as a result of various disorders, including esophagitis (6), myasthenia gravis and polymyositis (7). However, these primary diseases is not popular in cats compared with dogs (8). According to the report of Joseph *et al*., a cat showed megaesophagus following surgical diaphragmatic hernia repair even though there was no sign of megaesophagus before the correction of diaphragmatic hernia. In that case, a cat suffered respiratory deterioration and cyanosis during the anesthesia induction. The case showed clinical symptoms such as vomiting from directly after the diaphragmatic hernia repair. Megaesophagus was identified and treated with gastrostomy tube, H_2 blockers and metoclopramide. These symptoms disappeared over a number of subsequent weeks.

Another rare complication, hiatal hernia, has been reported in

*Corresponding Author: Ryou Tanaka Department of Veterinary Surgery, Faculty of Veterinary Medicine, Tokyo University of Agriculture and Technology 3-5-8 Saiwaicho, Fuchu, Tokyo 183-8509, Japan Email: ryo@vet.ne.jp

a number of dogs following surgical repair for chronic diaphragmatic hernia (9). Hiatal hernia has both hereditary and acquired forms, and it often results from congenital hiatal abnormalities; from congenital hiatal abnormalities; however, acquired form of hiatal hernia may develop with phrenicoesophageal ligament damage or flaccidity (10). However, we could not find any reports of post-repair hiatal hernia in cats, and its progression, treatment, and prognosis remain unclear.

In the present case, a diaphragmatic hernia was surgically repaired in a six-month-old juvenile cat. The cat showed vomiting as a clinical symptom from the day after the surgical repair, and megaesophagus and hiatal hernia were found as post-repair complications. Gastrostomy and esophago-gastropexy were performed at the same time, and the aim was to achieve medical control with H_2 blockers and pro-kinetic drugs. We have provide commentaries on the rare complication an compared to that in the previously reported cases.

Case

The cat was a six-month-old, intact, male crossbreed. The cat had shown labored breathing since the day the cat was sheltered three weeks ago, and diagnosed with diaphragmatic hernia by the local veterinarian. The cat was accordingly admitted to our hospital for the desired treatment (Day 1). The cat showed fatigability and labored abdominal breathing, but no loss of appetite, vomiting, or diarrhea, and visible mucus membranes were pink. He weighed 1.5 kg, with a body condition score of 2/5. Radiography showed disappearance of the heart shadow, and the presence of a gaseous shadow of the alimentary canal in the thoracic cavity (Figure 1). A complete blood count (CBC) on hematology showed a white blood cell count (WBC) of 7800/μL, a red blood cell count (RBC) of $1001*10^4$ /μL, and a hemoglobin (HGB) concentration of 12.6 g/dL. Blood biochemistry showed glucose (GLU) of 106 mg/dL, blood urea nitrogen (BUN) of 24.2 mg/dL, creatinine (CRE) of 0.9 mg/dL, alanine aminotransferase (ALT) of 60 U/L, alkaline phosphatase (ALP) of 256 U/L, total protein (TP) of 7.3 g/dL, and albumin (ALB) of 3.4 g/dL.

On Day 2, the cat underwent surgical diaphragmatic hernia repair. Anesthesia was induced with atropine (0.05 mg/kg, s.c.), butorphanol (0.2 mg/kg, i.v.), and propofol (5 mg/kg, i.v.), and maintained with isoflurane. Despite the plentiful oxygen supply, oxygen saturation (SpO_2) decreased to 80% under 100% oxygen inhalation immediately after anesthetization and intubation, and then to 55% when the cat was placed in the dorsal position for disinfection of the surgical field. Accordingly, the cat was placed in a recumbent position on its lower left side rather than a dorsal position, and an incision was made in a caudal direction toward the navel along the abdominal and an incision was made in a caudal direction toward the navel along the abdominal viscera were observed in the peritoneal cavity, and the liver, spleen, stomach, and the majority of the intestinal tract were found in the thoracic cavity. The liver showed morphological distortion in all lobes, dark red coloration, and pronounced enlargement, and was observed in the thoracic cavity. No peristaltic alimentary canal. The diaphragm was substantially ruptured in three directions on its movement was observed in the abdominal side, and hyperplastic connective tissue was observed at the stump; however, that was no major absence of the diaphragm. The lungs showed hepatization across almost all lobes in the thoracic cavity. A simple interrupted suture with 3-0 nylon sutures was performed on the diaphragm. A silicon tube was introduced to drain the thoracic cavity through the most ventral side of the diaphragm. The surgical incision could be closed without excessive tension at the time of suturing the abdominal wall. The duration of the surgical procedure from opening to closing of the incision was 45 minutes.

On Day 3, the cat's breathing was stable, but it showed vomiting from the morning. Multiple nematodes were present in the vomitus and were suspected to be *Toxocara cati*. Abdominal ultrasonography showed almost no peristalsis in the alimentary canal, and treatment with famotidine (1.0 mg/kg, i.v., BID) and metoclopramide (1.0 mg/kg/day, CRI) was therefore started. Air and pale, bloody pleural fluid were removed from the thoracic drain at 5.2 mL/head/day and 5.0 mL/head/day, respectively.

On Day 4, vomiting (which was occurring several times daily) persisted, and the cat underwent radiography of the alimentary canal using a contrast agent (1.5 mL/kg, p.o.). There were no constricted areas in the esophagus; however, almost the entire amount of the contrast agent was retained in the esophagus 70 minutes after it had been administered orally. A total of 4.0 mL/head of pleural fluid were removed from the thoracic drain on that day, but the color of the fluid had become pale pink, and the thoracic drain was removed on the evening of the same day.

By Day 5, megaesophagus had developed (Figure 2), and feeding had become problematic due to vomiting. Accordingly, it was judged that compulsory feeding bypassing the esophagus was necessary, and a surgical gastrostomy was performed. The cat underwent midline laparotomy under anesthesia in the same manner as on Day 2. There was a hiatal hernia in the abdominal esophagus. The abdominal esophagus was drawn into the abdominal cavity, and an esophagopexy was performed by fixing the esophagus to the diaphragm with simple interrupted suturing using 3.0-nylon sutures. A button-type gastrostomy tube was introduced, and a simultaneous gastropexy was performed. The incision was then closed in the prescribed manner. The duration of the surgical procedure from the opening to the closing of the incision was 35 minutes.

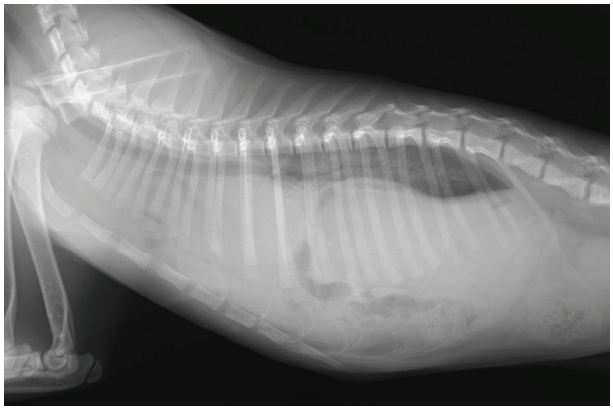

Figure 1: Right lateral chest radiograph on Day 1
Radiography showed disappearance of the heart shadow, and the presence of a gaseous shadow of the alimentary canal in the thoracic cavity.

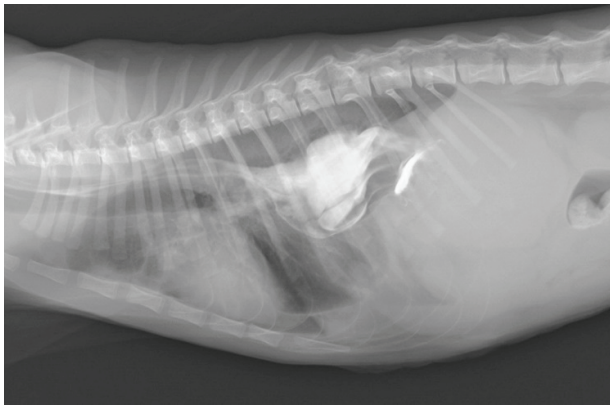

Figure 2: Right lateral chest radiograph on Day 5
Gastrointestinal contrast examination showed megaesophagus and a contrast medium had stayed in the esophagus.

On Day 6, fluid infusion of peripheral venous nutrition was initiated, along with provision of a comprehensive liquid diet through the artificial opening to the stomach. Almost no peristaltic movement in the alimentary canal was seen at that time, and, accordingly, the cholinesterase inhibitor edrophonium (0.1 mg/kg, i.m.) was administered under abdominal ultrasonographic guidance in addition to metoclopramide, and the liquid diet and pyridostigmine (0.5 mg/kg) were administered through the artificial opening to the stomach with gastrointestinal peristalsis activated. The pyridostigmine dose was modulated (0.5 to 2.0 mg/kg, BID) depending on the movement in the alimentary canal and the internal volume as observed on abdominal ultrasonography each time the diet was provided.

Medical management was continued in this manner, and on Day 10, the cat was observed to defecate for the first time since admission to hospital (on Day 1). Abdominal ultrasonography showed improved peristalsis in the alimentary canal, and the cat was now being supplied with around 70% to 80% of its daily energy requirement (DER) through the artificial opening to the stomach.

On Day 11, in line with the strong wishes of the owner, the cat was provisionally discharged from hospital on a continuing regimen of metoclopramide and famotidine administration through the artificial opening to the stomach. The cat was also analyzed for anti-acetylcholine receptor antibody on the same day, and the results were within the normal range (0.08 nmol/L, 0 to 0.3 nmol/L).

On Day 14, the cat was re-admitted to hospital because the owner reported difficulties providing nutrition through the artificial opening to the stomach at home. The cat's body weight had decreased to 1.2 kg, and radiography and abdominal ultrasonography showed a large volume of retained gas in the stomach and markedly decreased peristalsis in the alimentary canal. The medical treatment prescribed at discharge was re-initiated.

The cat's body weight recovered to 1.4 kg by Day 24. Vomiting persisted; however, the cat was discharged after the owner received further training on managing the artificial opening to the stomach. Metoclopramide and famotidine continued to be administered through the artificial opening to the stomach.

By Day 28, the cat was largely receiving food orally at home, and its body weight had increased to 1.6 kg. Vomiting was

not seen from Day 25 onward, and the artificial opening to the stomach was closed in line with the owner's strong wish. Vomiting and fatigability were not observed after the second discharge from hospital. Although this cat was smaller than its littermates, its body weight passed 2.0 kg on Day 50, and at the time of this report (Day 98), its condition continues to improve gradually.

Discussion

The etiology of the development of postoperative megaesophagus in the cat is unknown. Joseph *et al*. proposed that a post-repair increase in abdominal pressure was one factor in the development of megaesophagus following diaphragmatic hernia repair. Specifically, diaphragmatic repair leads to negative pressure in the thoracic cavity and positive pressure in the abdominal cavity, and this phenomenon is one cause of megaesophagus. Hiatal flaccidity and increased abdominal pressure play an important role in the formation of hiatal hernias, as reported by Pratschke *et al* (9).

We proposed two factor to develop postoperative megaesophagus in the cat from the clinical findings in the present case. First, esophagitis was produced as a reaction to the invasive anesthetic, and the esophagitis induced megaesophagus. The megaesophagus may also have been aggravated by the increased abdominal pressure. We consider that the hiatal hernia may have been induced by the increase in abdominal pressure. Second, we suspect that physiological ileus occurred postoperatively in this case. Recovery of peristaltic movement in the alimentary canal following surgery requires 24 hours for the small intestine, between 24 and 48 hours for the stomach, and between 48 and 72 hours for the large intestine (11). Postoperative ileus (POI) may be diagnosed when these recovery times are exceeded and peristalsis has not resumed in the alimentary canal, but in the present case, almost no peristalsis was observed at 72 hours postoperatively. Functional ileus has been proposed as a severe complication produced after diaphragmatic hernia repair (4), and POI is produced as its precursor. This case was not characterized by complete disappearance of peristalsis; however, it is possible the physiological ileus was not alleviated by postoperative management, and that the condition progressed beyond POI to functional ileus. Risk factors for POI in human medicine include prolonged duration of surgery, respiratory tract conditions, and opioid use (12). In the present case, the duration of surgery was not prolonged, at 45 minutes; however, all lung lobes showed hepatization. In particular, we consider that chronic insults to the bronchi, bronchioles, and alveoli occurred with the decline of SpO_2 to 55% even though oxygen inhalation was set at 100% when the cat was in the dorsal position during surgical repair. The absence of observed intestinal peristalsis during the surgical repair of the diaphragmatic hernia suggests the possibility of unsatisfactory peristalsis in the alimentary canal predating surgery. Handling of the intestinal canal is also another important mechanism of POI; accordingly, we speculate that physiological ileus arose due to multiple causes. We also consider that the physiological ileus may have aggravated the hiatal flaccidity and megaesophagus.

The cat in the present case did not show high anti-acetylcholine receptor antibody levels or other symptoms of muscle weakness. Accordingly, we surmise that this cat had megaesophagus induced as explained above with an additionally complicating hiatal hernia, and not megaesophagus related to myasthenia gravis.

Minihan *et al*. classified diaphragmatic hernias with a clinical duration of two weeks or more as chronic (3). We consider that the cat in the present case had a chronic diaphragmatic hernia based on its history of labored breathing and fatigability over the three-week period since it had been in protective care. A case of feline megaesophagus following diaphragmatic hernia repair has been reported by Joseph *et al*., but it cannot be ascertained whether that case was chronic or acute, since the clinical course remained unclear (5). However, we suspect that case may have been chronic, because the animal manifested previously unseen symptoms when exposed to the invasiveness of general anesthesia. In veterinary medicine, hiatal hernia has been reported following diaphragmatic repair only in dogs, but the diaphragmatic hernia in each of those cases was chronic (9). Surgical repair is not highly urgent in cases of chronic diaphragmatic hernia (3). However, based on consideration of those two reports (5) (9), as well as this case study, it may be that post-repair complications of the sort seen in this case develop more readily when the diaphragmatic hernia is chronic.

In a previously reported case of feline megaesophagus following surgical diaphragmatic hernia repair (5), clinical symptoms disappeared over a number of weeks postoperatively. Similarly in the present case, clinical symptoms disappeared by Day 25. Accordingly, it may be considered that a prognosis following the manifestation of these complications is not poor.

However, we also consider that nutritional management is important during periods of severe vomiting, particularly for young and weak cats. The cat in the present case vomited on multiple occasions daily, and it produced a large volume of vomitus, giving rise to concern over whether it was achieving the necessary caloric intake. Control of vomiting is difficult to achieve with famotidine and metoclopramide; accordingly, we also used cholinesterase inhibitors as pro-kinetic drugs in addition to infusion of peripheral venous nutrition of nutrients and compulsory diet provision through the artificial opening to the stomach. Veterinarians have few opportunities to use drugs of this sort, except in cases of myasthenia gravis. However, pro-kinetic drugs have great utility in human medicine, especially for postoperative paralytic ileus (13). We consider that gastro-esophageal reflux was prevented in the present case with rapid transportation of diet from the stomach to the intestines after its introduction through the artificial opening to the stomach, resulting in an achievement of a consistently low vomitus volume. However, we cannot rule out the possibility that vomiting was induced by an accumulation of saliva in the esophagus resulting from an increase in salivary volume with the promotion of peristalsis in the alimentary canal by the cholinesterase inhibitor. Accordingly, abdominal ultrasonography was used to monitor movements in the esophagus, stomach, and intestines, and internal volumes for each of the many daily dietary provisions through the artificial opening to the stomach. During that monitoring, the therapeutic dosage was modulated based on thorough consideration to the applied pharmaceutical volume.

Conclusion

A case of a juvenile cat with complications after diaphragmatic hernia repair was reported. The complications were extremely limited megaesophagus and hiatal hernia, which were expressed at the same time. The aim was to control the cat's condition with surgical esophago-gastropexy, provision of nutrients through an artificial opening to the stomach, protection of the esophageal mucous membrane, and the application of pro-kinetic drugs. Similar to the few previous reports, clinical symptoms disappeared over a number of weeks following surgery. Such post-diaphragmatic hernia repair complications present difficulties from the viewpoint of nutritional management in young and weak animals, but they may be transient.

References

1. Stokhof AA. Diagnosis and treatment of acquired diaphragmatic hernia by thoracotomy in 49 dogs and 72 cats. Vet Q. 1986;8(3):177-183.
2. Gibson TW, Brisson BA, Sears W. Perioperative survival rates after surgery for diaphragmatic hernia in dogs and cats: 92 cases (1990-2002). J Am Vet Med Assoc. 2005;227(1):105-109.
3. Minihan AC, Berg J, Evans KL. Chronic diaphragmatic hernia in 34 dogs and 16 cats. J Am Anim Hosp Assoc. 2004;40(1):51-63.
4. Garson HL, Dodman NH, Baker GJ. Diaphragmatic hernia. Analysis of fifty-six cases in dogs and cats. J Small Anim Pract. 1980;21(9):469-481.
5. Joseph R, Kuzi S, Lavy E, Aroch I. Transient megaoesophagus and oesophagitis following diaphragmatic rupture repair in a cat. J Feline Med Surg. 2008;10(3):284-290.
6. Mace S, Shelton GD, Eddlestone S. [Megaesophagus in the dog and cat]. Tierarztl Prax Ausg K Kleintiere Heimtiere. 2013;41(2):123-131; quiz 32.
7. Dewey CW, Bailey CS, Shelton GD, Kass PH, Cardinet GH, 3rd. Clinical forms of acquired myasthenia gravis in dogs: 25 cases (1988-1995). J Vet Intern Med. 1997;11(2):50-57.
8. Hague DW, Humphries HD, Mitchell MA, Shelton GD. Risk Factors and Outcomes 2015;29(5):1307-1312.
9. Pratschke KM, Hughes JM, Skelly C, Bellenger CR. Hiatal herniation as a complication of chronic diaphragmatic herniation. J Small Anim Pract. 1998;39(1):33-38.
10. Lorinson D, Bright RM. Long-term outcome of medical and surgical treatment of hiatal hernias in dogs and cats: 27 cases (1978-1996). J Am Vet Med Assoc. 1998;213(3):381-384.
11. Vather R, O'Grady G, Bissett IP, Dinning PG. Postoperative ileus: mechanisms and future directions for research. Clin Exp Pharmacol Physiol. 2014;41(5):358-370.
12. Bragg D, El-Sharkawy AM, Psaltis E, Maxwell-Armstrong CA, Lobo DN. Postoperative ileus: Recent developments in pathophysiology and management. Clin Nutr. 2015;34(3):367-376.
13. Orlando E, Finelli F, Colla M, Giotto E, Terragni P, Olivero G. [A double-blind study of neostigmine versus placebo in paralytic ileus as a result of surgical interventions]. Minerva Chir. 1994;49(5):451-455.