



DOI: 10.33188/vetheder.661451

Olgu Sunumu / Case Report

Megaesophagus development after surgical treatment of diaphragmatic hernia in three cats

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MAKALE BİLGİSİ /

ARTICLE
INFORMATION:

Geliş / Received:

24 Aralık 19

24 December 19

Kabul / Accepted:

21 Nisan 20

21 April 20

ABSTRACT:

A one year old and two three year old domestic short hair cats presented with trauma induced diaphragmatic hernia at Ankara University Animal Hospital. After clinical and radiographic examination all three cats diagnosed with diaphragmatic hernia. All cats gone under surgical repair for hernias. Several days after surgery all cats brought back to the clinic with vomiting, regurgitation and anorexia. Direct radiographs show esophageal dilatation and right shift in esophagus. Megaesophagus persisted for several weeks and was an unexpected complication as no association between diaphragmatic hernia (and diaphragmatic rupture) and megaesophagus has never been described in the veterinary literature. With medical treatment using prokinetic and antacid and high ground feeding, all cats recovered from megaesophagus without need for surgical intervention. There is no report of gastroesophageal reflux or megaesophagus associated with DH (or DY) in veterinary literature although there were three cases in very little amount of time in our clinic thus megaesophagus due to DH can be more common in cats than believed to be.

Anahtar Sözcükler:

Kedi
Megaözefagus
Diyafram fitiği
Cerrahi

Keywords:

Cat
Megaesophagus
Diaphragmatic hernia
Surgery

Üç kedide diyafram fitiğinin cerrahi sağaltımı sonrası megaözefagus gelişmesi

ÖZET :

Ankara Üniversitesi Veteriner Fakültesi Hayvan Hastanesine travma şikayeti ile başvuran bir adet bir yaşında ve iki adet üç yaşında kediyeye çekilen radyografiler ve klinik muayene sonucu diyafram fitiği tanısı konuldu. Diyafram fitiği tedavisi cerrahi olarak yapıldı. Tüm kediler birkaç gün sonra kliniğe kusma, regürjitasyon ve iştahsızlık şikayetleri ile geri getirildi. Direkt radyografilerde özefajial genişleme ve özefagusun sağa doğru yer değiştirdiği görüldü. Veteriner literatüründe megaözefagus ve diyafram fitiği arasında bağlantı çok az sayıda olguda tanımlandığı için megaözefagus, diyafram fitiğinin beklenmeyen bir komplikasyonudur. Tüm kediler prokinetik ve antiasid ilaç sağaltımı ve yüksek zeminden besleme ile cerrahi müdahaleye ihtiyaç duymadan iyileşti. Kedilerde diyafram fitiği sonucu gelişen megaözefagus da gastroözefajial reflüye dair çok az sayıda olgu tanımlanmasına rağmen kliniğimizde kısa sürede üç hastada bu duruma rastlanması, bu durumunun düşünüldüğünden daha yaygın bir komplikasyon olduğunu göstermektedir.

How to cite this article: Şen Y, Başer Mİ, Ergin İ, Sönmez G, Bumin A: Megaesophagus development after surgical treatment of diaphragmatic hernia in three cats. *Veteriner Hekimler Derneği Dergisi*, 91(2): 169-175, 2020, DOI: 10.33188/vetheder.661451

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1. Introduction

Megaesophagus is generalize dilatation of esophagus following neuromuscular disorders. It is classified as congenital and acquired depends on its reason or primer (idiopathic) and seconder depends on its etiology (3, 21). Seconder megaesophagus results from different conditions including neuromuscular disorders, endocrine or inflammatory diseases, toxic and infective agents and esophageal obstructions (11, 12). All forms of megaesophagus is rarely seen in cats (5). Idiopathic congenital megaesophagus has been identified before (14). Congenital megaesophagus is rear in cats but Siamese cats may be predisposed for this condition. Pathogenesis of congenital megaesophagus has not been understood completely but it is considered that an esophageal disorder can be developed by vagal nerve afferent stimulation (5).

Pleuroperitoneal hernias are rarely reported in cats (16, 29) and approximately %85 of diaphragmatic hernias (DH) and diaphragmatic ruptures (DR) is traumatic in cats and dogs (1, 32, 33) Congenital DH is associated with many anomalies in humans and transient megaesophagus is reported in newborns with congenital DH (19). Other most common complication of congenital DH in humans is gastroesphagial reflux (GOR) (17, 23). There is no report of gastroesophageal reflux or megaesophagus associated with DH (or DY) in veterinary literature although vagal and recurrent laryngeal nerve anomalies has seen in mice with experimentally induced congenital DH (20).

The aim of this paper was to evaluate clinical and radiographic findings and treatment results of esophageal inflammation and megaesophagus in three cats, which were formed in different degrees after diaphragmatic hernia repair operations.

2. Case Story

Three domestic short hair, male, one one year old and two three years old cats were brought to animal hospital of Ankara University Veterinary Faculty Animal Hospital with a history of difficulty in breathing and cyanotic membranes. The history was, all cat was perfectly healthy before and symptoms started after they went outside. Clinical examination revealed, tachypnea, tachycardia, decreased respiratory and heart sounds, as well as the presence of stomach sounds in the right thoracic area in auscultation. In direct radiography abdominals organs (liver lobes and gas filled intestines) were seen in thoracic cavity and peritoneopleural communication suspicions was confirmed (Fig 1).



Figure 1: L/L and V/D pre-op radiographies of case number two. Daggers are showing the soft tissue opacity in the right thoracic region of the patient.

Şekil 1: 2 numaralı olgunun operasyon öncesi L/L ve V/D pozisyonlarda çekilmiş göğüs radyografileri. Artı işareti toraksın sağ yarımında görülen yumuşak doku opasitesinesi göstermekte

Surgical intervention was chosen for all three patients' diaphragmatic hernia repair. CBC and biochemistry values were in normal ranges in all three patients in pre-op checks. Before the operation, 100% oxygen was given to the cats by mask for 30 minutes and infusion pump was set to infuse lactated ringer solution at a rate of 10 ml/kg/hr.

Prednisolon was injected 1 mg/kg IV intraoperatively. Operating table was positioned in 45 degree reverse Trendelenburg from the beginning of premedication, till the end of operation. All animals received preemptive analgesia with morphine HCl, general anaesthesia with propofol (4 mg/kg, IV) and isoflurane (by using cuffed endotracheal tubes), setting the ventilator to “pressure controlled ventilation” mode with an inspiratory pressure value of 10 cm H₂O, with respiratory rates of 16 breaths/minute. PEEP (positive end-expiratory pressure) mode was activated and was set to 2 cm H₂O for recruitment maneuver and to prevent atelectasis. After anesthesia, median incision laparotomy was performed. In case number one and two, ventrolateral radial rupture was seen in right side of the diaphragm and case number three had a ventral radial rupture in right side of the diaphragm (Fig2). In all cases, diaphragmatic rupture edges were thick and had fibrosis, in according to this findings it was decided all hernias was acquired. In case number one, three of liver lobes and most of small intestine segments was in thoracic cavity, in cases two and three, two of liver lobes and most of small intestine segments was in thoracic cavity. Pylorus and esophagus were shifted cranially to diaphragm rupture. All the herniated organs replaced in abdominal cavity. Diaphragmatic rupture repaired with 3/0 poliprolen in simple continues sutures. After diaphragm repair, 22G needle inserted through muscle part of diaphragm and free air drained from thoracic cavity using three-way cock and syringe to obtain negative pressure. Operation ended after abdominal muscles, subcutaneous tissue and cutaneous tissue closed with appropriate suture technique. All cats recovered from anesthesia in the intensive care unit, received fluids, analgesics, oxygen and were monitored for hypothermia, pain and dyspnea. Analgesics were administered 6 and 12 hours after surgery in all cats.



Figure 2: Intraoperative photograph of the case number two. On the right side of the diaphragm, a ventrolateral radial tear (dagger) can be seen.

Şekil 2: 2 numaralı olgunun operasyon sırasında çekilmiş görüntüsü. Diyaframın sağ tarafında ventrolateral radial yırtık (artı) görülmekte.

Over the next three days, all cats were in good health conditions but case number one and three vomited two times and case number two vomited several times. Vomiting kept under control with metoclopramide HCl (0,2mg/kg iv 12q h) usage. In post-operative day 6 and 7, cats brought to the hospital with history of anorexia, vomiting or regurgitation and mild dehydration. On direct radiography, severe gastric dilatation, megaesophagus and esophageal deviation to the right side of the chest cavity was observed (Fig 3). In all cases megaesophagus was perfectly clear on direct radiographs, indirect radiography which is relatively invasive and require sedation, was decided not necessary. Diaphragm was intact in all radiographs. All cats treated with intravenous lactated Ringer's solution (20mg/kg/h), ranitidine (2 mg/kg IV q12 h) and metoclopramide (0,2 mg/kg iv q12 h) for three days. Also oral surfactant (2mg/kg

oral q24) was administrated. Patients' owners told to give cats' food and water from someplace in higher ground.

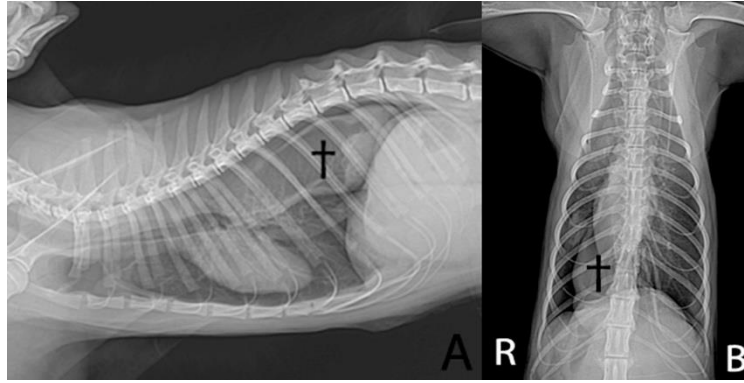


Figure 3: L/L and V/D direct radiograph taken on the postoperative third day of case number three. The esophageal dilatation (dagger) and the right deviation of esophagus (figure B dagger) can be seen.

Şekil 3: 3 numaralı olgunun operasyon sonrası üçüncü günde çekilen L/L ve V/D direkt radyografileri. Özefagusteki genişleme (Şekil A artı) ve özefagusun sağa deviyasyonu (şekil B artı) izlenebilmekte.

On day ten after medical treatment start, the signs of ileus, regurgitation and hypersalivation decreased. After three weeks, cats was eating more and getting some weight. After six mounts owners reported, cats were eating perfectly and back to normal live standards (Fig 4).

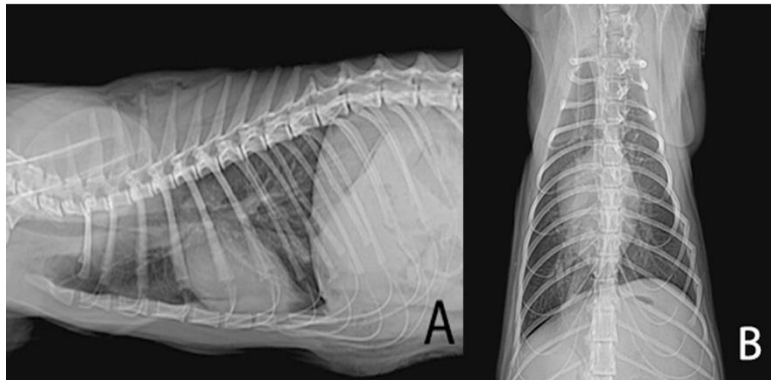


Figure 4: L/L and V/D direct radiograph of case number two; taken at six mounts after operation.

Şekil 4: 2 numaralı olgunun operasyondan altı ay sonra çekilen L/L ve V/D radyografileri

3. Discussion and Conclusion

Congenital diaphragm defects in cats and dogs are usually formed in the dorsolateral region when the intermedial regions of the left lumbar muscles or the crura and tendons absence, and such patients generally cannot survive the neonatal period. Because of this reason it is unlikely that these cats had congenital hernias (4, 6, 13).

Traumatic diaphragmatic hernias in cats reported to be circular, linear and combination of circular and linear, respectively %59, %18 and %23 (9). In congenital diaphragm ruptures, hernia usually occur in dorsolateral diaphragm with or without including center tendon (4, 29). The idea of acquired hernia in this cats is strengthens by no history of respiratory sign before going out, fibrosis tissues in diaphragm edges, hernia doesn't including center tendon and rupture being in right lateral area (15).

In human medicine, asymptomatic esophageal dilatation in neonatal area was reported but no data is available in veterinary literature (7). No megaesophagus signs in pre-op radiographs, no vomiting or regurgitation before

operation and symptoms' beginning in the post-op day two suggest that megaesophagus is a post-operative complication. Hypersalivation could be result of, abdominal pressure rises after operation, pancreas irritation during operation, post-anesthesia esophagitis syndrome or drugs that used. Esophagitis in cats because of some drugs and combinations of this drugs has been reported (2, 10, 30). Although all drugs that used in this study is given cats intravenously except amoxicillin which is given orally after operation. Amoxicillin is not reported to cause esophagitis, to the best of our knowledge. Also the patients has taken amoxicillin orally for minimum times so it is unlikely to cause esophagitis.

Pathogenesis of megaesophagus in these cats is not clear. Although, esophagus' longterm dysplasia before surgery, stomach shift to cranially, edema and dysfunction of lower esophageal sphincter could be cause of this condition. However, even if gastroesophageal reflux was present before surgery, it was subclinical and likelihood of causing megaesophagus development was minimum. The moderate pressure increase caused by the abdominal pressure results in increased pressure in the lower esophageal sphincter, which prevents the formation of gastroesophageal reflux because the pressure is above the gastric pressure. However, a further increase in the gastric pressure causes lower esophageal sphincter loosening and gastroesophageal reflux (12, 28).

Abdominal organs repositioning and suturing of diaphragm is likely to rapidly increase abdominal pressure for a short time in these cats. At the same time, correction of displacement of esophagus to the left side and stretching of the esophagus may cause reducing in the resistance of the gastroesophageal junction thus resulting gastroesophageal reflux, esophageal erosion and inflammation (15).

In a study conducted in brachiocephalic dogs shown that, respiratory depression leads to esophageal reflux in %80 and correction of upper airways by surgery reduces reflux. It is reported that the increase in abdominal pressure due to recurrent vomiting combination with increase in negative thoracic pressure due to difficulty in breathing results gastroesophageal reflux (26).

Bilateral vagal nerve lesions because of surgery, inflammation or trauma can effect esophageal mobility and cause megaesophagus(5). These lesions can be result of trauma that cause the diaphragmatic hernia, congenital or surgical mistakes. In cats that has been motioned in this paper; it is unlikely to have congenital megaesophagus because they responded to medical treatment. In addition, only a secondary correlation between diaphragmatic hernia and megaesophagus due to vagal or laryngeal recurrence nerve damage observed only in rats and the cause was more traumatic than congenital lesions (20). In the cases that has been described in this paper, it is unlikely that there was any vagal nerve damage that cause the megaesophagus because the surgery sides or diaphragmatic rupture was not near the vagal nerve.

Some correlation between megaesophagus and late gastric emptying was reported in cats but pathogenesis is unclear (25). It was reported that experimentally induced gastric dilatation decreases esophageal mobility in cats (22, 27) and repeated gastric dilatation and volvulus causes intermittent megaesophagus in dogs (24).

In this report, it is seen in post-operative radiographs and clinical history that diaphragmatic hernia repair caused gastroesophageal and lower esophageal sphincter disorders in these cats. This leads to gastric dilatation, gastroesophageal reflux, esophagitis and megaesophagus. Although the pathogenesis of gastroesophageal reflux is not clear, the authors think that gastroesophageal reflux is caused esophagitis and esophagitis caused megaesophagus.

The esophagitis associated with gastroesophageal reflux in the cat has been reported in very few cases and there is little information on the clinical appearance (13, 15, 18, 31).

Acquired idiopathic megaesophagus has been described in cats but the pathogenesis has not yet been clarified (8). Secondary megaesophagus can be formed after many conditions such as central nervous system neoplasia or Myasthenia gravis, which disrupts the swallowing reflex and esophageal muscle functions (11, 20). Development of secondary megaesophagus in cats is less likely than in dogs, it is believed the reason for this is, cats have less smooth muscles in esophagus and innervation of esophagus is via autonomic nerves than somatic nerves (5). It has been shown that esophageal obstructions reduces esophageal contractions, movement and time in cats and causes serious dysfunctions in esophageal function (22). An experimental study has shown that abdominal pressure increase causes slowing in esophagus peristaltic motion and prolongs the duration of lower esophageal sphincter to open (27).

It is believed that combination of long-term use of metoclopramide and sulphate with high ground feeding has made it possible to cure megaesophagus without operative intervention in the cats that has been described in this report.

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