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CASE REPORT
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Esophageal Diverticulum and Megaesophagus in a Dog and a Cat

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ABSTRACT

***Background*:** Esophageal diverticula are pouch-like dilatations in the esophageal wall that rarely affect dogs and cats, and may have the megaesophagus as a base cause. The definitive method of diagnosis is the contrasting chest X-ray that will visualize the sacculation.

***Cases*:** *Case 1.* A feline male, mixed breed, with 40 days of life was seen with complaint of postprandial regurgitation that was repeated at each meal. On physical examination, the animal was below ideal weight, apathetic, with pale mucosa and bristly. It was suspected of megaesophagus, which was confirmed by contrasting thoracic lateral-lateral (LL) radiography, and the patient also had anterior partial esophageal dilation to the topographic image of the heart. The image was suggestive of persistence of the right aortic arch, and corrective surgery was indicated but it was not authorized by the tutors. Therefore, conservative dietary treatment was instituted. The animal remained stable for a period of one and a half years. After this period the patient returned with an aggravated condition of vomiting, anorexia and apathy. The same clinical condition was maintained in the chest X-ray. An esophageal flushing was indicated, which was not authorized by the tutors, being prescribed mucosal protector and antibiotic. The medication was maintained by the tutor and after 1 month of the initial care, the clinical condition worsened and the animal died. *Case 2.* A 6-month-old male Pinscher dog weighing 1 kg was seen with postprandial regurgitation complaint since the beginning of the weaning transition period. In the esophagography performed a compatible radiographic image was seen with a diverticulum of the cranial thoracic esophagus and congenital total megaesophagus. The treatment adopted was conservative. Twelve months after the initial care, the animal presented radiographically resolution of the diverticulum and megaesophagus and was in good health.

***Discussion*:** These cases report two rare conditions of esophageal diverticulum associated with megaesophagus in a feline and canine. In the case of the feline, it was possible to conclude that the formation of the diverticulum was secondary to not surgical resolution of the persistence of the right aortic arch, with consequent maintenance of the megaesophagus, since the diverticulum was not present in the first radiographic examination. In the second case, when the radiograph was taken, the animal already presented the cranial diverticulum to the total megaesophagus, which, together with the history of postprandial vomiting since the 45 days of life, suggested a delay in the maturation of the esophageal nervous system that caused the megaesophagus and impairment of normal peristalsis of the organ culminating in the formation of the diverticulum. Due to the rare condition in both species, there are no reports of predilection for race, age or sex of the animals affected by the diverticulum, whereas for the megaesophagus, when in the congenital form, it preferentially occurs in young dogs and cats [less than 2 years of age], at the time of transition from the liquid to the solid diet. Both reports were of young animals that had recently undergone a food transition, which would justify the appearance of the megaesophagus as the main cause of the esophageal diverticulum. The clinical signs presented by the animals were compatible with those reported in the literature. It is concluded that although the esophageal diverticulum occurred in both cases as a consequence of megaesophagus, the resolution of the underlying cause is decisive for the resolution of the clinical condition determining the patient's prognosis.

Keywords: regurgitation, food impacts, esophageal dysfunction.

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INTRODUCTION

Esophageal diverticula are pouch-like dilatations in the esophageal wall that rarely affect dogs and cats [8]. Megaesophagus, esophageal stricture, esophagitis, foreign body, hiatal hernia and abnormalities in the vascular ring are possible causes of this change when acquired. In these cases, the diverticula can be differentiated into: by traction or by pulsion [2,4].

Traction occurs when there is a chronic inflammatory process adjacent to the esophagus, which is cured by fibrosis, generating adhesions that pull the esophagus leading to a distortion [12]. Pulsion diverticula develop due to disturbances in motility and altered esophageal peristalsis as well as stenotic lesions that generate an increase in intraluminal esophageal pressure as abnormalities of the vascular ring or foreign bodies [6,7].

One of the causes of pulsion-acquired diverticulum is the megaesophagus, because it is an esophageal alteration characterized by total or partial dilatation of the organ and consequent reduction of its peristalsis, or even loss of tone [6,7,12]. Congenital megaesophagus is usually diagnosed in animals when still puppies and the acquired form are more common in adult animals [15]. The congenital form may mainly be due to vascular ring abnormalities and esophageal dysmotility [10].

The most indicated method of diagnosis of the diverticulum is the association of the complete history with the contrasting thoracic radiograph, in which the esophageal dilation will be seen [7].

The objective of this study is to report two cases of esophageal diverticula resulting from partial megaesophagus in one cat and one in a dog.

CASES

Case 1. A male feline, mixed breed, with approximately 40 days of life was seen at a veterinary clinic with postprandial regurgitation that was repeated at each meal. The guardian administered vermifuge on his own, without improvement of the clinical condition.

The animal was below ideal weight, apathetic, with pale mucosa and bristly in the clinical examination. Food was offered in the office, the cat ingested, but regurgitated the contents soon after.

After anamnesis and clinical examination, the suspicion was of megaesophagus, which was confirmed by contrast-enhanced lateral-lateral (LL) chest X-ray, in which partial esophageal dilatation was visualized anterior to the base of the heart (Figure 1A). From this image it was suspected of megaesophagus due to persistence of the right aortic arch, and corrective surgery was indicated. However, there was no consent from the tutor for such intervention.

Upon the refusal of the tutor for the surgical procedure, conservative dietary treatment was recommended initially with hypercaloric paste feed (Hiperkal Cat®)[1], and later, paste feed pates for cats. The animal remained stable for a period of an year and a half with the proposed treatment.

After 1 and a half years, the patient returned to the clinic presenting with vomiting, progressing to anorexia and apathy. Another contrast-enhanced chest X-ray was performed in the LL position, where the megaesophagus and the presence of esophageal diverticulum were observed near the base of the heart (Figure 1B). Esophageal flushing was indicated in an attempt to reduce the volume of food retained in the diverticulum, which was not authorized by the tutor. Thus, the use of a mucosal protectant based on ranitidine (Antak®)[2] at the dose of 2 mg/kg BID and antibiotic based on Amoxicillin (Amoxil®)[2] at a dose of 20 mg/kg BID was prescribed.

Hematology, urea, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and serology for feline immunodeficiency virus (FIV) and feline leukemia virus (FELV) were performed as complementary tests. The hemogram showed leukopenia (4,100 mm/3) with lymphopenia (656 mm/3) [Table 1]. The biochemical tests were within normal range and the serology for FIV/FELV was negative. Maintaining the refusal of the surgical procedure by the tutors, was added the previous medication Thymomodulin syrup (Leucogen®)[3] 150 mg / kg, BID, for 30 days.

After 4 days of the first examinations, a new hemogram and repetition of the biochemical tests were performed, where the leukopenia was on the way to leukocytosis with increased band neutrophils and lymphopenia (Table 1), the biochemistry remained within normal range.

The prescription was maintained by the tutor and 1 month after the initial care, the clinical condition was aggravated by recurrent episodes of vomiting and the animal died. The guardian did not allow necropsy, but it is suggested that septicemia occurred due to rupture of the diverticulum because of the non-

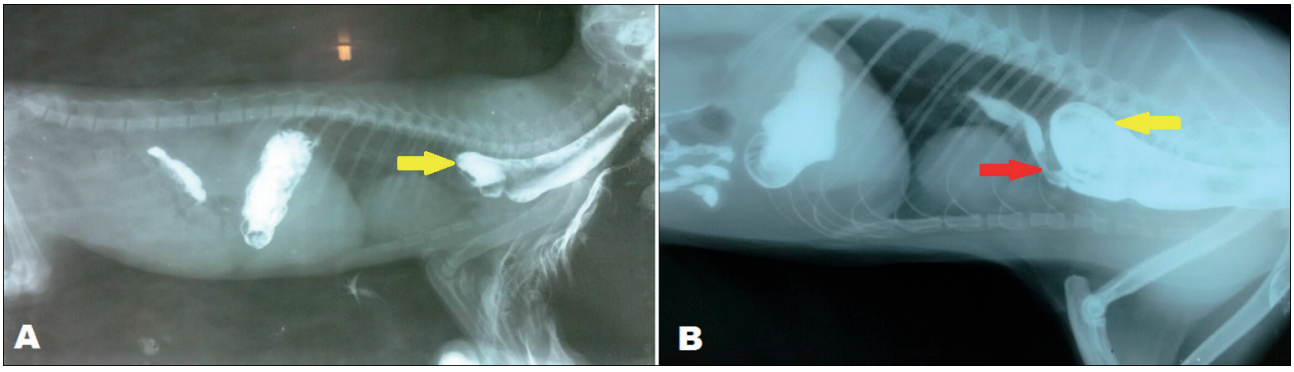

Figure 1. Macrograph of the radiographic image presented by the feline of case 1, presenting: A- the first thoracic X-ray of the animal showing partial esophageal dilatation (yellow arrow), with possible proximal constriction at the base of the heart; B- radiography performed 1 and a half years after diagnosis of megaesophagus, presenting esophageal diverticulum in the caudal portion of the megaesophagus (yellow arrow), esophageal constriction (red arrow) proximal to the base of the heart.

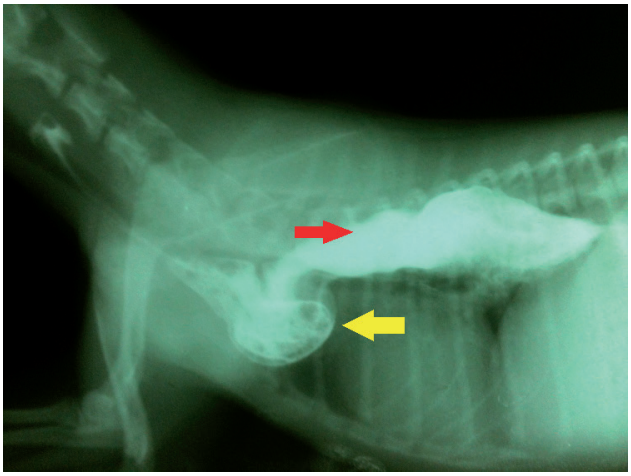

Figure 2. Macrophotography of the radiographic image presented by the canine of case 2, presenting: diverticulum in the thoracic esophagus (yellow arrow) and enlargement of the remainder of the esophagus to the hiatal region, image compatible with that of megaesophagus (red arrow).

-achievement of the surgical procedure and progressive compression by the aortic arch in the esophageal, further reducing the passage of food and increasing the size of the diverticulum.

Case 2. A 6-month old male Pinscher dog, weighing 1 kg, was seen at the Veterinary Hospital of the State University of Santa Cruz with complaints of dermatological alterations. During the anamnesis the tutor reported that the animal eventually had episodes of postprandial regurgitation since 45 days of age. At physical examination there was no change noteworthy, with all vital parameters within normalcy.

The animal was then referred to the radiology department for a lateral-lateral (LL) chest X-ray followed by contrast to show the cervical and thoracic esophagus. On the contrast-enhanced examination, a exuberant ventral sac of the thoracic esophagus was visualized in cranial mediastinum topography, followed by enlargement of its entire extension to the hiatal region (Figure 2). The radiographic images were compatible with diagnosis of cranial thoracic esophageal diverticulum and congenital total megaesophagus.

The treatment adopted was conservative with orientations of alimentary management with ingestion of pasty diet with the animal in upright position to avoid accumulation of food. The tutor was also informed that there might be a need for a surgical procedure to resolve the diverticulum.

The clinical status of the animal was monitored by telephone contact, where information was always obtained that the patient was stable and feeding normally.

Twelve months after the initial consultation, the guardian returned with the patient to the institution mentioning that the episodes of regurgitation had ceased and that the dog was feeding on dry feed and at four months were no longer adopting the erect position after feeding. The animal was referred to the radiology department for repeated radiography. In this new examination, there was total contrast passage through the esophagus with resolution of the diverticulum and megaesophagus.

DISCUSSION

These cases report two rare conditions of esophageal diverticulum associated with megaesophagus in a feline and canine. As it is a rare condition in both species [8], there are no reports of predilection for breed, age or sex of the animals affected by the diverticulum, whereas for the megaesophagus, when in the congenital form, it occurs preferentially in young dogs and cats (below 2 years of age) at the time of transition from the liquid to solid diet [9], with a higher prevalence described in Siamese cats [17]. Both reports were of young

Hemogram			
	Result 01/15	Result 01/19	Reference*
Red blood cell (mm³)	7.33	8.49	5.0 - 10.0
Hemoglobin (g/dl)	13.4	12.4	8.0 - 15.0
Hematocrit (%)	38.1	40.1	24 - 45
VCM (fL)	52	47	39 - 55
CHCM (%)	35	31	31 -35
Platelets (µL)	610,000	471,000	300,000 - 800,000
Leukogram			
Total Leukocytes (mm³)	4,100	19,100	5,500 - 19,500
Segmented (mm³)	2,460	7,373	2,500 - 12,500
Band neutrophils (mm³)	738	1,515	0 - 300
Lymphocytes (mm³)	656	909	1,500 - 7,000
Monocytes (mm³)	82	202	0 - 850
Eosinophils (mm³)	0	101	0 - 1,500
Basophils (mm³)	0	0	rare

* Weiss D.J. & Wardrop K.J. 2010. *Schalm's Veterinary Hematology*. 6th edn. Willey-Blackwell, 1232p.

animals that had recently undergone a food transition, which would justify the appearance of the megaesophagus as the main cause of the esophageal diverticulum. However, the onset of megaesophagus was diagnosed for different causes in the feline and in the dog.

The clinical signs presented by the animals were postprandial regurgitation, anorexia and weight loss, and are compatible with those reported in the literature [2,6,7].

In the case of the feline, it was possible to conclude that the formation of the diverticulum was secondary to the megaesophagus since it was not present in the first radiographic examination. Due to non-surgical resolution of aortic arch persistence and maintenance of esophageal hypomotility, esophageal lumen obstruction progressed as a consequence of the growth of the animal, with impaction of food in the esophagus and formation of the diverticulum. However, the cause of the megaesophagus was possibly related to the persistence of the right aortic arch due to its location with esophageal compression at the level of the cardiac base as described [13], as well as being the most common vascular ring anomaly in dogs and cats according to reports of literature [3].

In the case of the dog, the animal already presented the cranial diverticulum to the total megaesophagus at the moment of the radiographic diagnosis, which together with the history of postprandial vomiting since the 45 days of life, was determinant for the suspicion of an esophageal nervous maturation delay which caused the megaesophagus and impairment of normal peristalsis of the organ, culminating in the formation of the diverticulum. Both cases corroborate with the literature mentioning megaesophagus, stenosis, esophagitis and foreign bodies as the main causes of diverticula formation [5,12,14].

Both diverticula were classified according to their etiology as pulsion diverticula [6,7], precisely because they are consequences of the megaesophagus that favors the reduction of peristalsis and even loss of tonus in the organ in question [7,12]. Basically, it develops because of the herniation of the mucosa at a certain fragile point, developed as a result of an increase in intraluminal pressure. Most, if not all, epifrenic diverticula developed by functional or mechanical esophageal obstruction, which promotes a specific characteristic of the diverticula sac: to be more pendular, with walls largely composed of normal mucosa, with only an occasional portion of attenuated and loose connective tissue [12].

For the feline case, the treatment consists in identifying the type of vascular ring anomaly for surgical referral with posterior section and esophageal release [5]. The choice of tutors in not performing the surgical intervention to correct the persistence of the right aortic arch worsened the reduction of the esophageal lumen leading to a greater accumulation of content and impaction with formation of the diver-

ticulum. Authors [16] claim that surgical intervention should be performed as soon as possible avoiding further damage to the esophagus, reducing the chances of irreversible megaesophagus and loss of its motility. Evaluating the hematological alterations and the clinical condition of the animal, it is proposed that rupture of the diverticulum occurred, culminating with a generalized infection, leading to death, as described in the literature that the diverticula can result in esophageal impaction, chronic esophagitis and rupture of the wall of the diverticulum [5].

The second case was a total megaesophagus, therefore, hypomotility of the organ caused by delayed maturation of esophageal function was suspected [1]. According to the literature, the prognosis for this case is reserved, because it is not certain whether maturation will occur or not [1]. In the case reported, the tutor followed the instructions of conservative treatment when using high-level liquid feeding as described [11], reducing the chances of content impaction, sepsis and/or disruption of the diverticulum. One year after the diagnosis the animal had no clinical signs, as described in the literature [18], which cites that in some animals the dilated esophagus, may return partially to its normal size and function.

Both cases reported were esophageal diverticula secondary to megaesophagus confirmed by thoracic radiography. In the case of the feline, the refusal of the tutors in the surgical authorization to resolve the persistence of the right aortic arch, aggravated the case, leading the patient to death due to rupture of the diverticulum and sepsis. In the case of the dog, the nutritional management was enough to resolve the clinical condition, because according to expectation, there was nerve maturation of the esophageal function.

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REFERENCES

1 **Bexfield N.H., Watson P.J. & Herrtage M.E. 2006.** Esophageal dysmotility in young dogs. *Journal of Veterinary Internal Medicine*. 20(6): 1314-1318.

2 **Borku M.K., Ural K., Kibar M., Ozkanlar Y., Guzel M. & Cingi C. 2009.** Esophageal diverticulum in a cat. *Turkish Journal of Veterinary and Animal.* 33(2): 171-174.

3 **Buchanan J.W. 1999.** Prevalence of cardiovascular disorders. In: Fox P., Sisson D. & Moise N.S. (Eds). *Canine and Feline Cardiology*. 2nd edn. Philadelphia: WB Saunders, pp.457-470.

4 **Durocher L., Johnson S.E. & Green E. 2009.** Esophageal diverticulum associated with a trichobezoar in a cat. *Journal of the American Animal Hospital Association*. 45(1): 142-146.

5 **Fossum T.W. 2012.** Surgery of the esophagus. In: Fossum T.W. (Ed). *Small Animal Surgery*. 4th edn. Missouri: Elsevier Mosby, pp.372-394.

6 **Gualtieri M. 2001.** Esophagoscopy. *Veterinary Clinics of North America: Small Animal Practice*. 31(1): 605-630.

7 **Guilford W.G. & Strombeck DR. 1996.** Diseases of Swallowing. In: Strombeck D.R. (Ed). *Strombeck´s Small Animal Gastroenterology*. 3rd edn. Philadelphia: W.B. Saunders, pp.211-238.

8 **Hill F.W., Christie B.A., Reynolds W.T. & Lavelle R.B. 1979.** An oesophageal diverticulum in a dog. *Australian Veterinary Journal*. 55(1): 184-187.

9 **Lipsitz D., Berry J.L. & Shelton G.D. 1999.** Inherited predisposition to myasthenia gravis in New-foundlands. *Journal of the American Veterinary Medical Association*, 215(7): 946-958.

10 **Mace S., Shelton G.D. & Eddlestone S. 2012.** Megaesophagus. *Compendium: Continuing Education for Veterinarians*. 34(2): 1-7.

11 **Oliveira E.C., Gaiga L.H., Colomé L.M., Stedile R., Mello F.P.S., Martins J.M. & Freire C.D. 2004.** Persistência do arco aórtico direito em um cão - Relato de caso. *Revista da Faculdade de Zootecnia, Veterinária e Agronomia*. 11(1): 174-180.

12 **Pearson H., Gibbs C. & Kelly D.F. 1978.** Oesophageal diverticulum formation in the dog. *Journal of Small Animal Practice*. 19(1): 341-355.

13 Ricardo C., Augusto A. & Canavese S. 2001. Double aortic arch in a dog (*Canis familiaris*): a case report. *Anatomia, Histologia, Embryologia*. 30(6): 379-381.

14 Strombeck D.R. & Guilford W.G. 1990. Esophageal Diverticula: Diseases of Swallowing. In: Strombeck D.R. & Guilford W.G. (Eds). *Small Animal Gastroenterology*. 2nd edn. Santa Ana: Stonegate Publishing, 157p.

15 Tanaka M.N., Hoogevonink N., Tucholski A.P., Trapp S.M. & Frehse M.S. 2010. Megaesôfago em cães. *Revista Acadêmica, Ciência Agrária Ambiental*. 8(3): 271-279.

16 Twedt D.C. 1997. Afecções do esôfago. In: Ettinger S.J. & Feldman E.C. (Eds). *Tratado de Medicina Interna Veterinária - Moléstias do cão e do gato.* 4.ed. São Paulo: Manole, 3020p.

17 Washabau R.J. 2003. Gastrointestinal motility disorders and gastrointestinal prokinetic therapy. *Veterinary Clinics: Small Animal Practice*. 33(5): 1007-1028.

18 Willard M.D. 2006. Distúrbios da cavidade oral, faringe e esôfago. In: Nelson R.W. & Couto C.G. (Eds). *Medicina interna de pequenos animais* 3.ed. Rio de Janeiro: Elsevier, pp.398-399.