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Differential Diagnosis of Persistent Dysphagia and Regurgitation in the Young

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Introduction:
Veterinarians in practice are constantly encountered with young dogs presented with a history of persistent dysphagia and/or regurgitation since weaning. Persistent dysphagia and regurgitation implies the continual inability to swallow solid foods and pass the formed bolus normally through the esophagus to the stomach; where it is digested enzymatically and physically to a liquified state, allowing the passage through the pyloric sphincter into the duodenum. Although there is a multitude of possible etiologies, those conditions which are detected symptomatically at or shortly after weaning are considered; the clinical signs being related to the introduction of solid food to the diet. Therefore, the differential would include those conditions that are primarily congenital in origin. Historically, megaesophagus and vascular ring anomalies are the congenital conditions most frequently reported.

Acquired conditions unrelated to age must also be included, i.e., foreign body obstruction and esophageal stricture; but conditions manifested as transient vomiting, i.e., infectious diseases or their sequelae and intestinal obstruction are excluded.

Diagnosis is based on history, age of the patient, physical examination, but most importantly radiology. Examination of the thoracic esophagus should include lateral and DV projections. Frequently, the esophagus can be evaluated more completely by placing the dog in an oblique position so that the right dorsal thoracic wall is closest to the collimator. On plain films the normal esophagus cannot be identified due to its collapsed state (except in deglutition) which contains neither food or air, and the similarity in density to the muscles of the neck and mediastinum.

The objective of the plain film examination is recognition of disorders, which requires: 1) decreased density (gas) within the esophagus, 2) increased density within, or 3) decreased density outside the esophagus. Therefore, radiographic interpretation is directed at classifying the disorder as intraluminal, intramural, or periesophageal. A combination of these lesions may also be seen.

A radiographic study, using positive contrast, permits evaluation of function and morphology of the esophagus as well as the location and size of the disorder. The only preparation necessary is withholding food from the patient four to six hours prior to the study. Barium sulfate is the agent usually employed, but iodine-containing solutions are recommended in cases where plain film studies are suggestive of esophageal perforations.

Fluoroscopic studies allow the dynamic process of swallowing to be assessed but are usually limited to the university hospitals.

Esophagoscopy can be performed in private practices and can be of special benefit in certain cases, but anesthesia is always required.

The principal differential features of the possible etiologies are now discussed in detail.

Megaesophagus:
Of all the conditions discussed, this is the most frequent. Initially the dog has intermittent difficulty in swallowing—possibly with some discomfort and choking. As the condition progresses, regurgitation begins,
with the interval between ingestion of the meal and regurgitation becoming shorter. With time, the dog may begin regurgitating before the meal is completely consumed, extending the neck and holding the mouth low. This regurgitated food is frequently "reconsumed" and may be retained in the thoracic esophagus for up to an hour before further regurgitation. Fluids may be completely retained but are usually partially regurgitated. Many animals vomit copious quantities of frothy saliva.

In long-standing cases, regurgitation may be delayed for an hour or more after feeding. The symptoms may then simulate pyloric stenosis and pylorospasm but because in megaesophagus the regurgitated material has not been in the stomach, it contains no acid. This feature distinguishes the two conditions.

After the ingestion of fluid, palpating the lower cervical esophagus often reveals a pocket of fluid at this point. Ballooning of the esophagus at the thoracic inlet may be detected by compressing the chest while holding the nose and mouth closed. Affected animals are usually bright and active, except after feeding, if unaffected by secondary pneumonic problems caused by the aspiration of regurgitated fluids.

The condition is diagnosed by radiography. Lateral views show massive dilation of the entire thoracic esophagus. The trachea and heart may be depressed ventrally, due to the dorsally enlarged fluid-filled esophagus. On contrast studies, the esophagus may be only partially outlined because of the presence of air, fluid, and debris preventing the even dispersal of the barium. There is no evidence of intramural or periesophageal constrictions, nor is peristalsis evident. Vento-dorsal views show the characteristic and diagnostic coning appearance of the cardia.

**Vascular Ring Anomalies**

Ectopic vascular structures due to developmental anomalies of the aorta can compress the esophagus over the base of the heart, restricting the passage of food. In the majority of cases the "vascular ring" is a result of a persistent right aortic arch with the ligamentum arteriosum (the post-natal remnant of the ductus arteriosus connecting the aorta and the pulmonary artery) passing dorsally over the esophagus.

Less frequently, the compression is caused by a left subclavian artery or carotid artery and double aortic arch.

Except in rare cases, clinical signs appear soon after weaning, due to the introduction of solid food to the diet and the inability of solid food to pass through the compromised esophageal lumen.

Affected animals regurgitate, often with some distress, within minutes of feeding. Depending on the degree of compression, fluids may pass through the area without retention. Occasionally, however, the compression may be less severe and frequent, and small meals may then pass unobstructed to the stomach.

The diagnosis is based on contrast radiography in two planes. The lateral view shows a conical narrowing of a small segment of the proximal thoracic esophagus at the base of the heart, causing secondary dilatation and sacculcation of the thoracic esophagus cranial to the stricture. The distal half of the thoracic esophagus is usually of normal size and has good peristaltic activity. The ventro-dorsal view shows the characteristic narrowing, often with kinking of the esophagus at the point of compression. The descending aorta may also be seen on the right side.

**Congenital Hypertrophic Pyloric Stenosis and Pylorospasm**

These conditions are characterized by persistent regurgitation and postprandial projectile vomiting in young dogs due to impaired movement of ingesta through the pyloric canal and/or pyloric sphincter. When diagnosing this condition, it is important to note that the vomitus is always food from the last meal and may be virtually unchanged, rather than frothy material or gastric secretions characteristic for other conditions.

Pyloric stenosis is related to a developmental structural alteration of the musculature resulting in a persistent problem, while pylorospasm is associated with an intermittent problem due to functional impairment. The symptoms usually develop soon after weaning and affected animals are usually presented with a history of marked distension of the anterior abdomen after feeding, often with obvious discomfort or pain caused by vigorous gastric contractions.
against a closed pylorus. They are usually grossly undersized and in poor condition, with a pot-bellied appearance. Exaggerated borborygmi are frequently reported.

Diagnosis is confirmed by determining a delayed gastric emptying time by feeding barium mixed with solid food and taking radiographs at thirty minute intervals. The normal stomach begins to empty within thirty minutes. If the gastric emptying time is delayed for an hour or more, there is probably some degree of pyloric stenosis or pylorospasm (gastric emptying time implies the time taken for the stomach to begin to empty—not to empty completely.) On ventrodorsal radiographs, the entire stomach is found to be enlarged, particularly the diameter of the pyloric canal and antrum, with an hour glass contraction close to the pylorus.

**Developmental Herniation of the Stomach Through the Esophageal Hiatus**

Congenital gastroesophageal invagination is quite rare in the young dog. The puppy is usually presented with signs of cachexia and severe persistent regurgitation, starting directly after weaning. If much of the stomach is herniated, there may also be dyspnea and cyanosis.

On endoscopy, the characteristic appearance of the severely congested gastric mucous membrane is easily distinguished from the paler esophageal mucosa.

The important radiographic findings include the presence of gas trapped in the dilated thoracic esophagus which is well circumscribed, increased density in the terminal thoracic esophagus, and restricted passage of contrast medium into the abdomen. The rugal pattern of the stomach may be identified in the obstructing density at the terminal thoracic esophagus, particularly if positive contrast is administered.

It is important to emphasize that any obstruction, functional or mechanical, of the terminal esophagus or cardia of the stomach can produce a generalized esophageal dilatation, and therefore contrast studies should always be made with this finding in scout films.

**Esophageal Diverticula**

Circumscribed outpouching of the esophageal wall is an uncommon finding in the young dog. It usually results from defective development of the muscular layer of the esophagus and protrusion of the mucosal lining at the weakened region, resulting in a diverticulum. A diverticulum usually does not produce clinical signs unless the stomata of the outpouching becomes large and filled with ingesta causing retching and regurgitation. Therefore the symptoms may or may not be directly related to weaning.

Also, in contrast to megaesophagus, the retching in these cases is usually persistent and painful and may not be related to feeding, because the contents of the diverticula cannot be expressed by regurgitation.

The condition can be diagnosed by viewing the stomata via endoscopy or by radiographs taken in two planes. Careful examination of survey radiographs may show a pocket of gas, sometimes with an accumulation of radio-opaque debris. Contrast esophageal studies are usually needed to reach a diagnosis. Although the diverticula may not fill completely with contrast, they are usually clearly separate from the main esophageal lumen.

**Esophageal Stenosis**

Congenital intramural strictures of the esophagus must be differentiated from periesophageal causes such as vascular anomalies. The location of the stenotic area can usually be of benefit. The symptoms are dysphagia and immediate regurgitation of food and sometimes fluids related to weaning.

In contrast to this, the onset of clinical signs in acquired stenosis is gradual. Initially, semi-solids will pass through the stenotic area but solid foods will be regurgitated. With time, all ingested material will be unable to pass. The thoracic inlet is a common location for acquired esophageal stenosis, with possible causes being foreign bodies, caustic agents, etc. Another common site is above the base of the heart. Trauma and/or regurgitated gastric juices may damage the esophageal mucous membranes and muscular layers, eventually resulting in a stricture. In both acquired and congenital stenosis, contrast radiology is necessary for diagnosis. The use of an esophagscope may especially be of value in this condition, enabling the visualization of scarred mucosa.
Cricopharyngeal Achalasia

The exact etiology of this condition is unknown. But abnormal innervation of the cricopharyngeal muscle is suspected, resulting in failure of the cricopharyngeal sphincter to relax, thereby preventing the passage of food from the pharynx into the esophagus.

This condition causes a persistent dysphagia starting at weaning, and can result in nasopharyngeal regurgitation or aspiration of the oral contents. Bilateral suppurative rhinitis and/or aspiration pneumonia rapidly ensues and causes a secondary dyspnea. Affected animals, although almost cachectic, may survive several weeks or even months.

The diagnosis is confirmed by radiography. The upper cervical esophagus is excessively dilated with air and during a barium swallow, most of the contrast remains in the pharynx until it is regurgitated.

Further description is best illustrated by a typical case study.

Case Report

A nine week old female terrier-type dog was admitted to the Iowa State University Veterinary Hospital on the 22nd of September, 1979, with a history of persistent dysphagia since weaning. Repeated attempts to swallow solid food resulted in gagging and expulsion of the food from the mouth by forward movements of the tongue. With persistent masticatory and deglutitory efforts, the dog was able to pass small amounts of the semi-liquid, "re-ingested" food to the stomach without further difficulty. Liquids, if then taken in moderate amounts, would pass with minimal effort to the stomach.

The dog was presented with "open-mouth" breathing and would periodically have spells of a moist hacking cough. Moist rales were auscultated in the lungs and the external nares were crusted over with a mucopurulent exudate. Examination of the pharynx did not reveal any inflammatory or obstructive lesions.

Lateral and ventro-dorsal radiographs were taken of the thorax. Extensive bronchial and interstitial densities were noted throughout most of the lung field. The interstitial densities coalesced in the ventral portions of the lung field with patchy areas of alveolar density. These changes suggested an increased inflammatory response within the lungs, i.e., severe chronic bronchopneumonia, probably due to aspiration, considering the patient's previous history. A large amount of air was also seen within the stomach and intestinal tract due to aerophagia.

A tentative diagnosis of cricopharyngeal achalasia was confirmed by fluoroscopic studies of a barium swallow. A bolus of barium sulfate was seen to pass into the posterior portion of the pharynx (in the area of the esophagus) by normal movements of the tongue and contracture of the pharyngeal musculature. Despite a force sufficient to cause marked distension of the posterior pharyngeal wall, inadequate relaxation of the cricopharyngeal sphincter prevented normal movement of the bolus into the esophagus, and subsequently the bolus fell back into the pharynx. The thin stream of barium that passed through the sphincter was moved by normal peristaltic waves to the stomach. As the epiglottis opened for inspiration, the residual barium refluxed into the laryngeal pharynx, aspirated into the trachea, and was discharged by coughing.

Cricopharyngeal myotomy was performed three days later, allowing the pneumonia to begin to be resolved with the aid of antibiotics. The dog was placed on solid food the day following surgery, and experienced no difficulty in swallowing. The secondary aspiration bronchopneumonia and nasopharyngitis continued to be treated medically with systemic antibiotics.

Recovery progressed smoothly. Post-myotomy fluoroscopic studies were made utilizing barium sulfate mixed with dog food. Good pharyngeal motility was seen with opening of the cricopharyngeal area and passage of the bolus into the proximal esophagus. Normal primary and secondary esophageal peristaltic contractions were seen as the bolus proceeded through the remainder of the esophagus.

Successful correction of the cricopharyngeal achalasia was concluded.

Summary:

On initial examination of the patient with a history of persistent dysphagia and regurgitation since weaning, it is important to observe the manner of food intake and the character of the regurgitation.

For example, there is no difficulty in swallowing with congenital or acquired disorders involving the esophagus (strictures,
Clinical Pathology Review

E. D. Lassen, D.V.M., Ph.D.*

Analysis of the blood from a 5 year old, female Holstein cow revealed the following leukogram:

| WBC (4,000–12,000) | 6,000 |
| Band neutrophils (0–120) | 1,080 |
| Segmented neutrophils (600–4,000) | 1,320 |
| Lymphocytes (2,500–7,500) | 2,880 |
| Monocytes (25–850) | 720 |
| Eosinophils (0–2,400) | 0 |

The cow had freshened 3 days before the blood sample was taken. One quarter of the mammary gland was swollen and hard. A vaginal discharge was evident. What is your interpretation of the leukogram?

Interpretation:

This is an inflammatory leukogram (i.e., there is an increased tissue demand for neutrophils). The major feature of an inflammatory leukogram is a significant left shift (i.e., increased numbers of immature neutrophils). In the bovine, WBC counts are typically in the normal range or slightly above normal during inflammatory episodes. In peracute inflammations and/or overwhelming bacterial infections, WBC counts may be below normal and immature WBC’s may outnumber mature WBC’s. Possible causes of the tissue demand for neutrophils are mastitis and/or metritis.

References


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