ACUTE DYSPNEA IN A DOG

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History

A 13-year-old intact mixed breed dog weighing 20 kg., was presented to the emergency service at the Koret School of Veterinary Medicine, University Teaching Hospital for acute dyspnea, salivation and non-productive vomiting.

Medical history included a dry cough of 6 months duration that partially responded to theophylline. The dog was current on vaccination and deworming, and lived in-and-outdoors with another healthy dog.

Physical examination

On presentation the dog was cyanotic, orthopneic and had significant inspiratory dyspnea with a respiratory rate of 24 breaths per minute. Heart sounds were muffled and abdominal distention was noted. Auscultation of the lungs was difficult due to referred upper-respiratory noise.

The dog was adequately hydrated, with a rectal temperature of 38.5 C°, pulse rate of 100 beats per minute and a body condition score of 5/5.

Initial stabilization included flow-by oxygen and a single dose of furosemide (2 mg/kg IV). An intravenous catheter was placed and thoracic and cervical radiographs were obtained (Figures No. 1, 2).

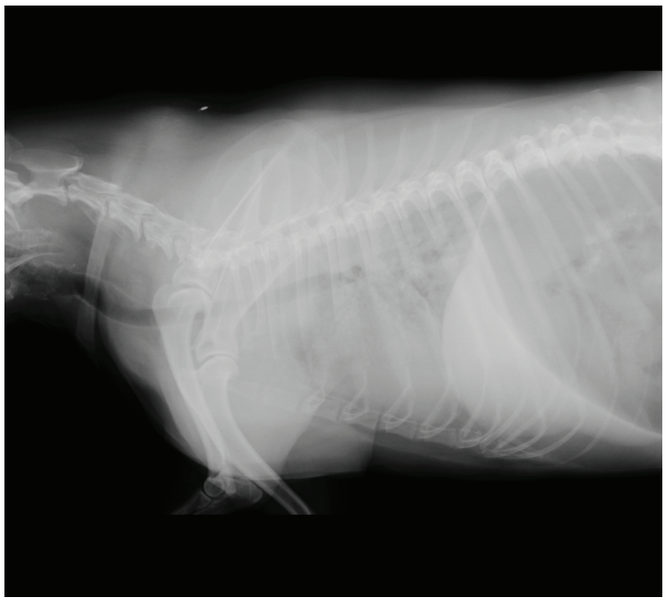

Figure 1.

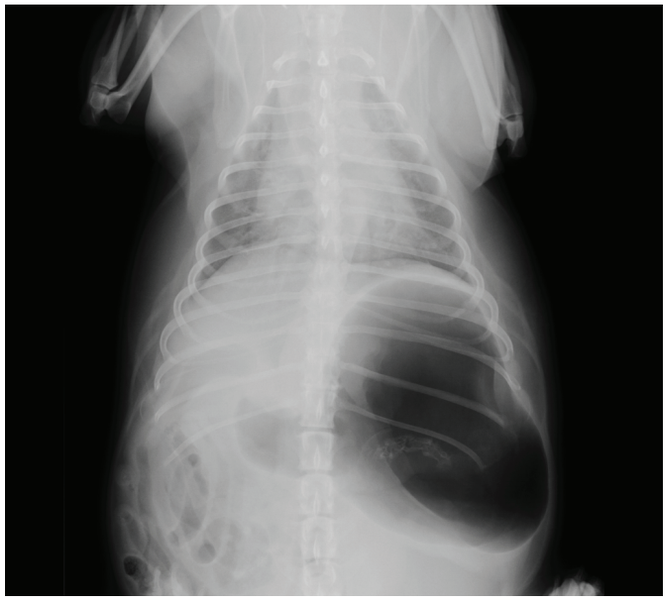

Figure 2.

Describe the radiographic findings.

1. WHAT ARE THE RADIOGRAPHIC DIAGNOSES?

2. PROVIDE A LIST OF DIFFERENTIAL DIAGNOSES TO THE ABNORMAL RADIOGRAPHIC FINDINGS.

3. WHAT WILL BE YOUR NEXT DIAGNOSTIC AND THERAPEUTIC PROCEDURES?

See the following page for the diagnosis and explanation

RADIOGRAPHIC FINDINGS

Cervical (close-up)

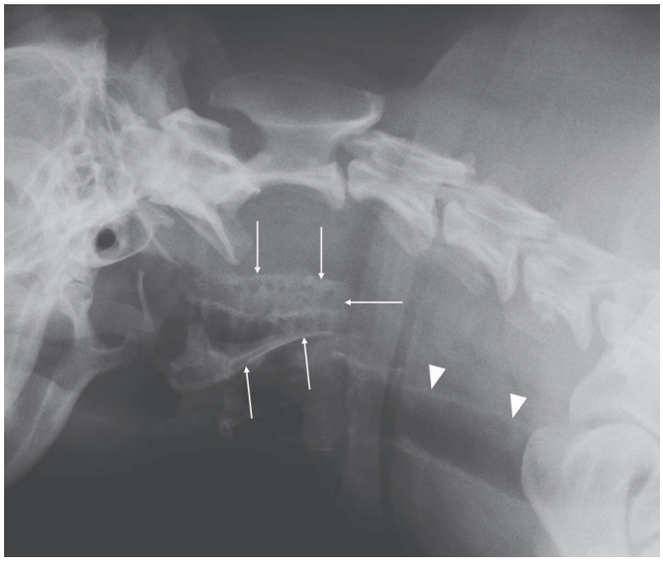

Figure 3.

A demarcated mass of bony opacity is seen within the pharynx (figure 3–arrows). The cervical esophagus is mildly distended with gas causing distinction of the tracheal wall- "tracheoesophageal strip sign" (figure 3-arrow heads).

Thoracic

Bilateral diffuse increased opacity within all lung fields, with reduced visibility of the pulmonary vasculature. There is border effacement of the cardiac silhouette ("cardiac silhouette sign") on the lateral view. In addition, widening of the cranial mediastinum is visible on the dorsoventral view.

Abdomen

Within the viewable abdomen, the stomach is severely distended with gas and a bony foreign material is visible within it.

Soft tissues & skeleton

Increased subcutaneous fatty tissue accumulation is seen mainly at the extra- thoracic region, and multiple thoracic and lumbar spondylosis deformans.

RADIOGRAPHIC DIAGNOSIS

1. Suspected foreign body (bone) within the pharynx.
2. Diffuse interstitial to alveolar pulmonary pattern *.
3. Cranial mediastinum widening.
4. Gastric dilation.
5. Spondylosis deformans.

*The dominant lung pattern is interstitial; however some focal alveolar infiltrations are visualized. The prominent increase in opacity seen on the lateral view is also the result of subcutaneous fatty tissue accumulation in the thoracic region

DIFFERENT DIAGNOSIS

Pulmonary interstitial pattern (1)

The list of differential diagnosis for pulmonary interstitial pattern is extensive. Prioritization of the various differential diagnoses is important, such that the most likely differentials are placed at the top of the list.

1. Edema, either cardiogenic or non-cardiogenic (e.g. as a result of upper obstructive airways or acute respiratory distress syndrome-"shock lung").
2. Hemorrhage (e.g. trauma, coagulopathy)
3. Toxins (e.g. Paraquat poisoning)
4. Pneumonia (due to aspiration, bacterial, viral, fungal, parasitic, etc.)
5. Neoplasia (e.g. lymphosarcoma)
6. Allergic (e.g. Pulmonary Infiltrate with eosinophils, usually will have also a bronchial component)
7. Pulmonary thromboembolism
8. Artificial increased lung opacity (e.g. poorly inflated lungs, obesity, underexposure etc.)

Other differential diagnoses which are less likely or irrelevant to our case include pulmonary fibrosis, pancreatitis, uremia, hyperadrenocorticism, age related, long-term corticosteroid administration and radiation (localized to the irradiated area of the lung).

The mediastinal widening is most likely due to fat accumulation (obese dog), and without clinical significance in this case. The gastric dilatation is assessed to be secondary to the severe dyspnea and thus, due to aerophagia. The spondylosis deformans are age-related, incidental, and clinically insignificant in this case.

ASSESSMENT

A bony foreign body is located in the pharynx, blocking the upper airways, causing severe inspiratory dyspnea and increased upper-respiratory sounds. In addition, post-obstructive pulmonary edema, a form of non-cardiogenic pulmonary edema resulting in poorly inflated lungs, is suspected. The abdominal distention is most likely due to gastric dilation secondary to aerophagia.

THE NEXT DIAGNOSTIC STEP

The dog was anesthetized with intravenous propofol and diazepam to effect for oral and laryngeal examination. Orotracheal intubation was not attempted to avoid advancing the bony foreign body into the trachea.

A few bony fragments were seen and easily removed from the entrance to the pharynx and the larynx, the largest being 10x5 cm (figure 4). Following this, endotracheal intubation was performed and the dog received 100% oxygen for several hours. Recovery was uneventful.

A repeat thoracic radiograph was performed the following day (figure 5). Mild diffuse pulmonary interstitial pattern was noted, with marked improvement compared to the previous radiographs. The caudal thoracic was moderately distended with gas. The heart size and shape and pulmonary vasculature was within normal limits.

Figure 4

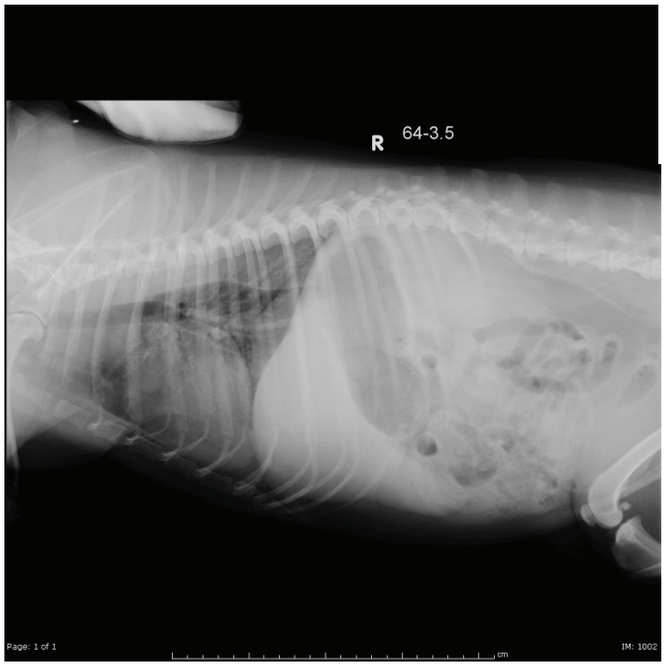

Figure 5

DISCUSSION

Pulmonary interestitial pattern

The interstitium is the supporting structure of the lungs and includes the alveoli walls and ducts, the interlobular septa, the capillaries, and the supporting tissue of the lymphatics, bronchioles, and pulmonary vasculature (2).

An interstitial pattern is categorized as either structured (nodular) or unstructured. An unstructured interstitial pattern, as seen in this case, is caused by a collection of fluid, cells, or fibrin within the connective tissue framework of the lung, between the alveoli, and around vessels and airways. This will result in a generalized increase in lung opacity and loss in vessel definition (3).

Since many lung pathologies are dynamic and "on-going processes", interstitial disease may have progressed to the stage of alveolar involvement before it becomes detectable on radiographs (2).

Many abnormal lung patterns consist of a combination of two or three constituent patterns. The alveolar and interstitial patterns may be difficult to distinguish, and often co-exist. Usually, one pattern is dominant and will help to elucidate the etiology (1).

Many causes should be considered when an unstructured interstitial pattern is observed (see the differential diagnostic list above). Thus, it is recommended to rule in/rule out a potential disease by integrating the signalment, relevant history, clinical signs as well as radiographs and other diagnostic procedures. It should be mentioned that obtaining the definitive diagnosis for an interstitial pattern may require direct cytologic sampling as fine needle aspiration or biopsy. That is because many diseases causing an interstitial pattern do not involve the air way, and sampling by transtracheal aspiration or bronchoalveolar lavage may not be helpful (4).

Post-obstructive pulmonary edema

Post-obstructive pulmonary edema is a poorly-defined cause of non-cardiogenic pulmonary edema in human medicine and, to our knowledge, has not been reported in companion animals. A single case report of a horse developing prominent pulmonary edema following upper airway obstruction during recovery from surgery for left cricoarytenoideus dorsalis muscle reinnervation and ventriculocordectomy has been made (5). Gross, histological, and electron microscopic postmortem examination of this horse showed severe hemorrhagic pulmonary edema. The following discussion is based on a review by McConkey *et al.* (6).

In 1960 Swann reported pulmonary edema at autopsy in human victims from hanging. It was not clear whether this was a pre- or post-mortal change. Since than, over 100 human cases of post-obstructive pulmonary edema have been reported, with the underlying causes being strangulation, neoplasia, laryngospasm, hanging, epiglositis, bilateral vocal cord palsy, acromegaly, goitre and obstruction of the endotracheal tube.

The common pattern in these cases is the occurrence of an episode of airway obstruction followed by the rapid onset of respiratory distress, hemoptysis and bilateral radiological changes consistent with pulmonary edema. After the elimination of the etiological cause there is then rapid and complete resolution of both clinical and radiological features within 24 hours. In 1993 Cascade reviewed the radiological features, including alveolar edema and predominating interstitial edema and commented both on the typical rapid resolution and the previous lack of recognition in the radiological literature (7).

The terms "post-obstructive pulmonary edema", "negative pressure pulmonary edema" and "laryngospasm induced pulmonary edema" have been used. Various mechanisms have been proposed, although the precise pathophysiology is uncertain (8-10). Negative intra-alveolar pressure will directly

alter Starling forces across the pulmonary capillary by lowering the pulmonary interstitial hydrostatic pressure. This negative pressure also disturbs cardiovascular physiology. Increased right heart filling, decreased left heart filling, increased left ventricular (LV) afterload (transmural pressure) and decreased LV ejection lead to increased pulmonary capillary hydrostatic pressure. Thus the pulmonary capillary transmural pressure is increased by two mechanisms, favoring transudation of fluid into the pulmonary interstitium. The resultant mechanical stress may disrupt the integrity of pulmonary or bronchial capillaries.

Edema fluid analysis has consistently shown a high protein content. This suggests that the fluid is an exudate and is evidence for disruption of capillary integrity rather than simple rearrangement of Starling forces. When measured, cardiac filling pressures have been low or normal, as may be expected with a non-cardiogenic pulmonary edema. Bronchoscopy, performed on a few human patients, showed haemorrhagic lesions lining the mucosa of the trachea and large bronchi (11, 12). This led to the interesting speculation that airway bleeding rather than pulmonary edema was the dominant event and that this in turn was due to disruption of the high-pressure bronchial rather than the low-pressure pulmonary capillaries.

Differential diagnoses include aspiration pneumonitis, occult cardiac disease, fluid overload and anaphylaxis. Aspiration can produce a clinical picture similar to that seen here. Treatment modalities range from nasal oxygen to intubation and positive pressure ventilation.

We suspect that post-obstructive pulmonary edema exists and is under-recognized in veterinary medicine as well. In our hospital, several dogs presented with severe pulmonary edema following an episode of upper airway obstruction such as accidental strangulation with a neck lead or laryngeal edema, especially in brachiocephalic breeds. While we cannot definitively conclude that this dog had post-obstructive pulmonary edema, the purpose of this brief communication was to increase the awareness to this potential sequel of upper airway obstruction in the veterinary community.

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