

INSIGHTS IN IMAGING

Radiographic Features of Pulmonary Hypertension in Dogs and Cats

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Pulmonary hypertension is defined as increased pulmonary arterial pressure, commonly secondary to cardiovascular, pulmonary, or systemic disease.^{1,2} Right-sided cardiomegaly can ensue, possibly followed by cardiac decompensation into right-sided heart failure with increased severity. More commonly, dogs and cats with pulmonary hypertension exhibit signs of dyspnea, syncope, exercise intolerance, lethargy, or coughing.¹ A diagnosis is most practically achieved with Doppler echocardiography due to its accessibility and noninvasiveness. However, a definitive diagnosis is best obtained by using more invasive catheterization techniques under fluoroscopic guidance to directly measure pulmonary arterial pressure,^{1,2} although the risks, higher cost, and training needed to perform such a procedure make it a less favorable choice for routine

clinical use.¹ Thoracic radiography may precede and is more accessible than echocardiography and may show abnormalities supportive of pulmonary hypertension and changes indicative of the underlying cause. Although thoracic radiography is reported to be insensitive for patients with mild pulmonary hypertension, abnormalities may be more reliably observed in dogs and cats with at least moderate pulmonary hypertension.² Management of pulmonary arterial hypertension depends on the underlying cause. The American College of Veterinary Internal Medicine recently published a consensus statement with guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs.¹ This article focuses on the use of thoracic radiographs to support a diagnosis of pulmonary hypertension.

Abstract

Pulmonary hypertension commonly results from cardiovascular, pulmonary, or systemic disease. Clinical signs include dyspnea, syncope, exercise intolerance, lethargy, and/or coughing. Diagnostics can begin with thoracic radiography; more advanced techniques include Doppler echocardiography and pressure measurement by catheterization under fluoroscopic guidance. Although certain abnormalities may indicate pulmonary hypertension on radiographs, a radiographic assessment of the intrathoracic structures as a whole can help screen for comorbidities, which may either be a cause or sequela of pulmonary hypertension and may require further diagnostic work-up. Early diagnosis and treatment can help prevent progression to right-sided heart failure. Management depends on the underlying cause.

Take-Home Points

- Causes of pulmonary hypertension are categorized as primary arterial hypertension (including congenital cardiac shunts), left-sided cardiac disease (left ventricular dysfunction or valvular disease), respiratory disease and/or hypoxia, pulmonary thromboembolization, parasitic disease, and multifactorial or idiopathic.
- Thoracic radiography is a practical, accessible, and noninvasive screening tool.
- Recognition of the radiographic features of pulmonary hypertension can help guide further diagnostics.
- Radiographs are useful for identifying comorbidities and an underlying cause.
- Early diagnosis and treatment of pulmonary hypertension help prevent progression to right-sided heart failure.
- Management of pulmonary hypertension depends on the underlying cause.

RADIOGRAPHIC FEATURES

Dogs

Radiographic features of pulmonary hypertension include right-sided cardiomegaly and pulmonary arterial distension. Right-sided cardiomegaly is characterized by rounding of the cranial and right lateral margins of the cardiac silhouette with associated caudal and left displacement of the cardiac apex, resulting in a “reverse D” appearance of the cardiac silhouette on a ventrodorsal/dorsoventral projection (**FIGURE 1**).^{2,3} In the absence of concurrent left-sided cardiomegaly, the cardiac silhouette may become wider in relation to its height.³ An objective means of measuring this change, the “3/5 to 2/5 cardiac ratio” method involves drawing a line from the carina, extending ventrally parallel to the caudal border of the heart. Approximately 3/5 of the cardiac silhouette surface area (right heart) should be cranial to this line, and 2/5 of the cardiac silhouette surface area (left heart) should lay caudal to this line (**FIGURE 2**).⁴ If the cardiac silhouette cranial to this line has a fractional surface area greater than 3/5, right-sided cardiomegaly should be considered.⁴

Using the clockface analogy (**FIGURE 3**), an opaque soft tissue bulge confluent with the cardiac silhouette at the 1 to 2 o’clock position on the dorsoventral or ventrodorsal projections is suggestive of main pulmonary artery enlargement (**FIGURE 4**). This finding may be associated with lobar pulmonary artery enlargement. Although asymmetry of the lobar pulmonary arteries and veins (characterized by enlargement of the lobar pulmonary arteries compared with the corresponding lobar pulmonary veins) is supportive of lobar pulmonary artery enlargement, this

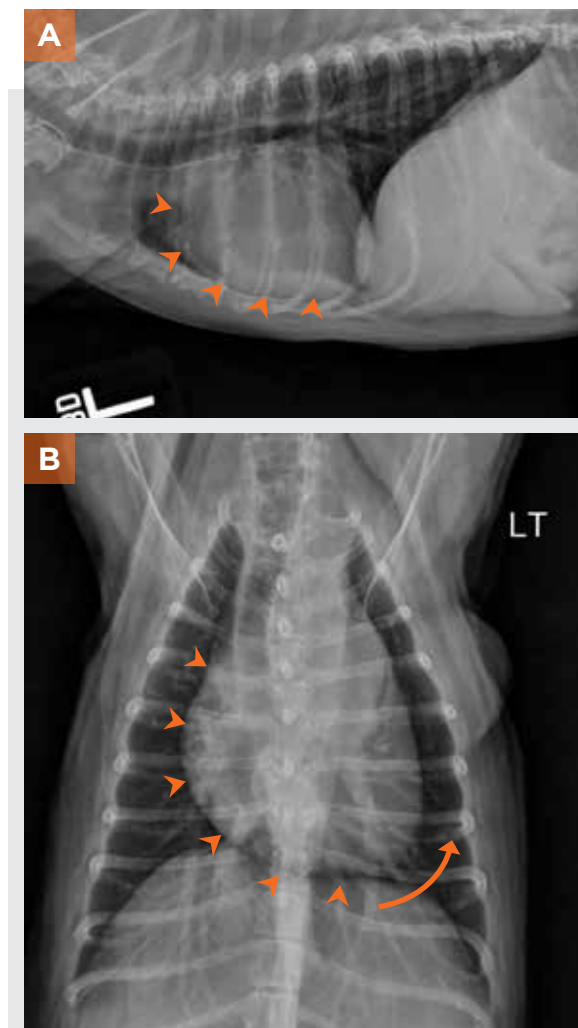


FIGURE 1. (A) Lateral and (B) dorsoventral projections of a dog with severe right-sided cardiomegaly. The cranial and right lateral margins of the cardiac silhouette are severely rounded (**arrowheads**), and the apex of the cardiac silhouette is displaced to the left (**arrow**). The cardiac silhouette has a “reverse D” appearance.

method may be unreliable in the presence of concurrent lobar pulmonary vein enlargement. For objective assessment of the lobar pulmonary vasculature, the width of the right cranial lobar pulmonary artery can be compared with the width of the right fourth rib just ventral to the vertebral column on a left lateral projection (FIGURE 5). A lobar pulmonary artery width to fourth rib width ratio greater than 1.2 is indicative of lobar pulmonary artery enlargement.⁵

A similar approach involves measuring the ratio of the caudal lobar pulmonary artery width to the ninth rib width, where the lobar pulmonary artery and rib intersect on a ventrodorsal/dorsoventral projection (FIGURE 6). A recent study found a greater correlation between lobar pulmonary artery size and pulmonary hypertension by comparing the width of the caudal

lobar pulmonary arteries to the width of the ipsilateral third rib (FIGURE 7).² Using the ninth and third ribs as reference, a ratio greater than 1:1 was suggestive of lobar pulmonary artery enlargement.² In chronic and severe cases of pulmonary hypertension, findings may include tortuosity and blunting of the pulmonary vasculature.¹⁻³

Cats

Evaluation of cardiac chamber enlargement in cats is limited due to the relative positioning of the cardiac

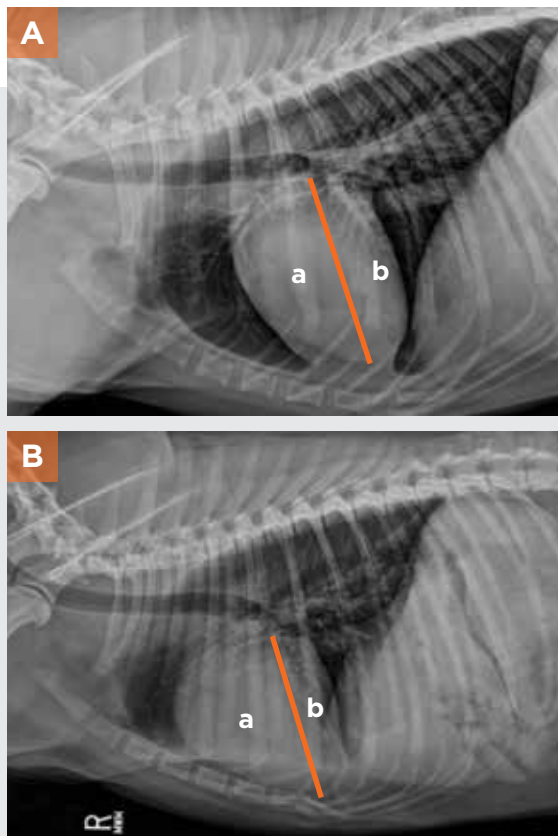


FIGURE 2. Right lateral thoracic radiographs of a dog (A) without and (B) with right-sided cardiomegaly. In the absence of cardiomegaly (A), a line drawn from the carina and extending ventrally parallel to the caudal border of the heart divides the cardiac silhouette into a normal “3/5 to 2/5 cardiac ratio,” as correlated by comparing the surface areas of **a:b**. In patients with right-sided cardiomegaly (B), more than 3/5 of the cardiac silhouette surface area is identified cranial to this line (i.e., **a:b** is greater than 3/5:2/5).

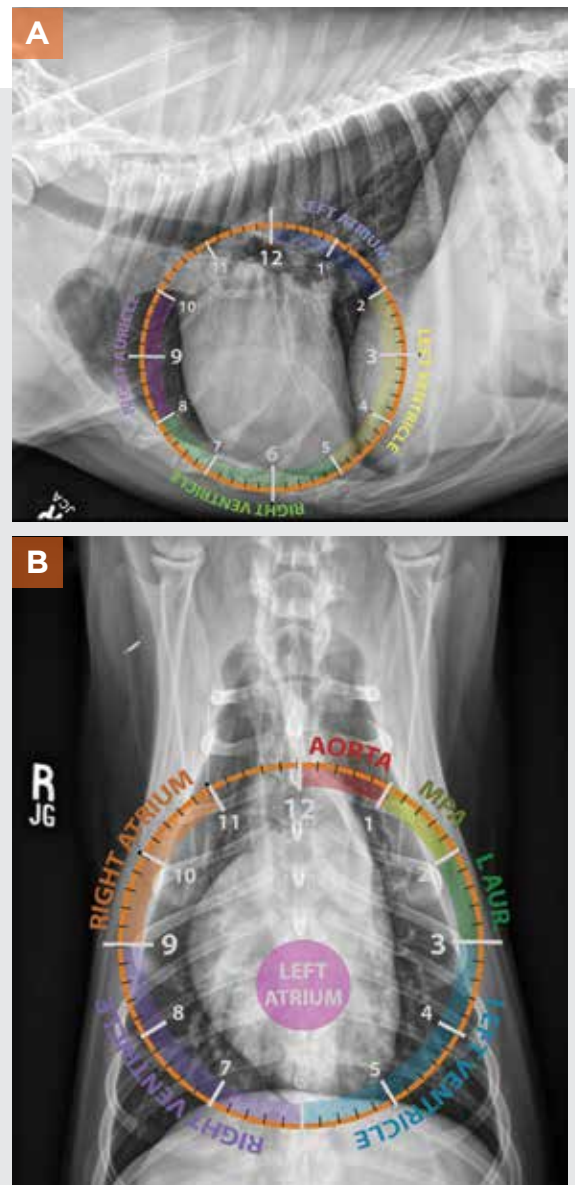


FIGURE 3. Clockface representation of cardiac chambers location on (A) right lateral and (B) ventrodorsal radiographs. L. aur, left auricle; MPA, main pulmonary artery.

chambers and variable orientation of the longitudinal axis of the heart. Generalized cardiomegaly may be observed in cats with right-sided cardiomegaly but is not specific and may also be seen exclusively or concurrently with left-sided cardiomegaly. Similar to dogs, an opaque soft tissue bulge at the 1 to 2 o'clock position along the cardiac silhouette in cats is supportive of main pulmonary artery enlargement (FIGURE 8). However, the main pulmonary artery tends to be more medially positioned in cats and may not always be apparent.³ Although there are no published objective measurements for lobar pulmonary vessel enlargement in cats, subjective enlargement of

the lobar pulmonary arteries may support a diagnosis of pulmonary hypertension, especially in the presence of pulmonary vessel tortuosity (FIGURE 9).

CAUSES OF PULMONARY HYPERTENSION

For dogs and cats, the patient's signalment, clinical signs, and physical examination findings, along with baseline diagnostics, including thoracic radiography and echocardiography, are useful for diagnosing and investigating possible causes of pulmonary hypertension. Etiologies can be broken down into the following disease categories¹:

- **Group 1:** Primary arterial hypertension (including congenital cardiac shunts)
- **Group 2:** Left-sided cardiac disease (left ventricular dysfunction or valvular disease)
- **Group 3:** Respiratory disease and/or hypoxia
- **Group 4:** Pulmonary thromboembolization
- **Group 5:** Parasitic disease
- **Group 6:** Multifactorial or idiopathic

Some radiographic abnormalities may aid in the recognition of such diseases. The remainder of this article describes the radiographic features of specific



FIGURE 4. Dorsoventral projection of a dog with pulmonary hypertension. A large, opaque soft tissue bulge at the 1 to 2 o'clock position along the cardiac silhouette (**arrowheads**) in the region of the main pulmonary artery is indicative of main pulmonary artery enlargement.

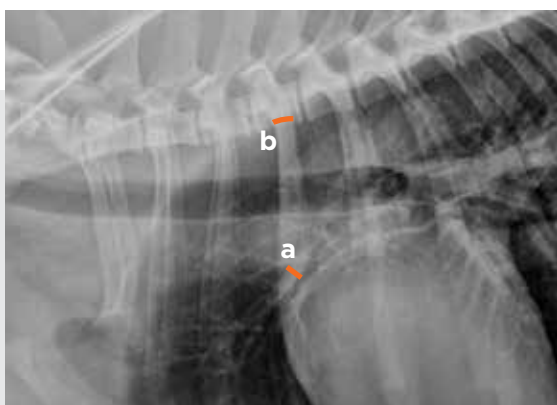


FIGURE 5. Left lateral projection of a dog without cardiovascular disease and (**a**) a normal right cranial lobar pulmonary artery width compared to (**b**) fourth rib width.

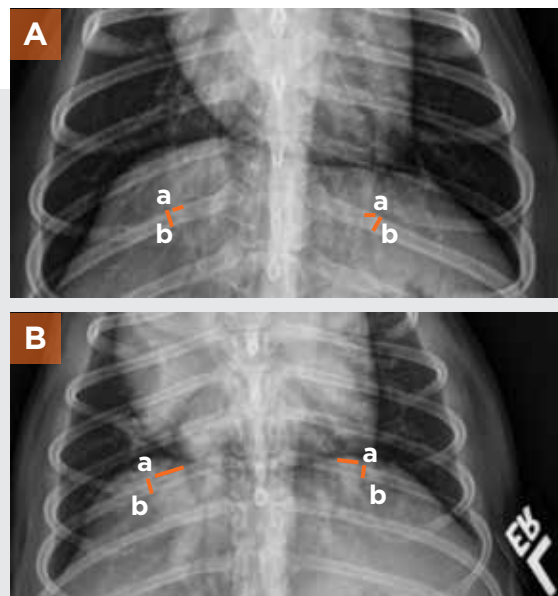


FIGURE 6. (A) Dorsoventral projection of a dog without cardiovascular disease and (**a**) normal right and left caudal lobar pulmonary artery width to (**b**) ninth rib width ratios. **(B)** Dorsoventral projection of a dog with pulmonary hypertension and (**a**) an increased caudal lobar pulmonary artery width to (**b**) ninth rib ratios ($a > b$), indicative of pulmonary artery enlargement.

diseases that may cause pulmonary hypertension. A directly related higher degree of pulmonary hypertension is expected with increased disease severity.

Group 1: Primary Arterial Hypertension

In the absence of radiographic abnormalities, a diagnosis of pulmonary hypertension may be presumed to be secondary to a primary or secondary arteriopathy, sometimes with an unknown underlying etiology (i.e., idiopathic).^{1,6} Radiographic evidence of pulmonary

hypertension may be apparent, and in some patients, cor pulmonale and right-sided heart failure may develop.⁶ Many patients may be falsely placed in this category antemortem if radiographic evidence of pulmonary disease is not apparent, as the absence of disease cannot be ruled out without histopathologic confirmation.¹ Rarely recognized in veterinary patients,

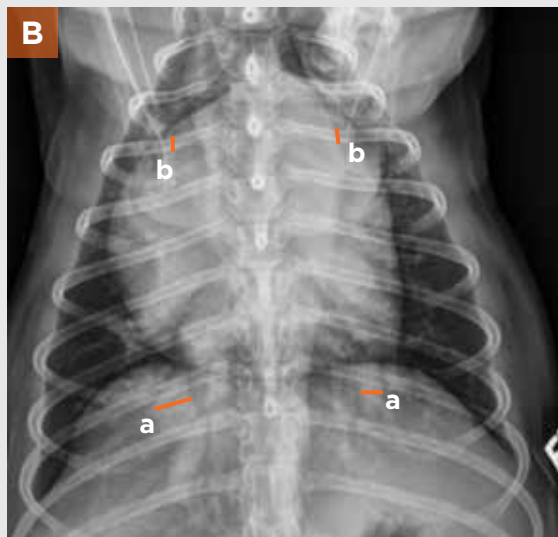
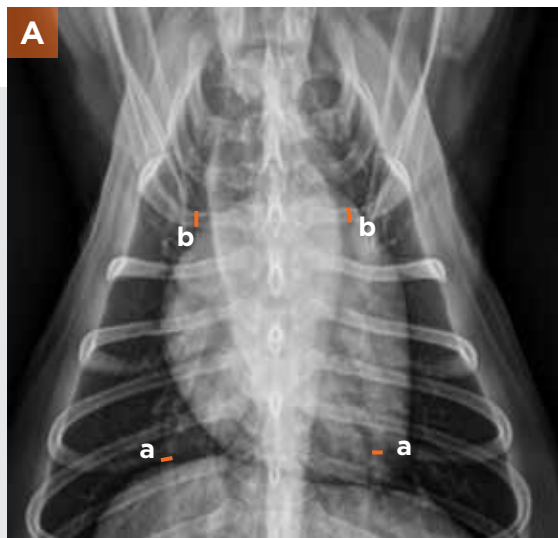


FIGURE 7. (A) Dorsoventral projection of a dog without cardiovascular disease and **(a)** normal right and left caudal lobar pulmonary artery width to **(b)** third rib width ratios. **(B)** Dorsoventral projection of a dog with pulmonary hypertension and **(a)** an increased caudal lobar pulmonary artery width to **(b)** third rib ratios ($a > b$) indicative of pulmonary artery enlargement.

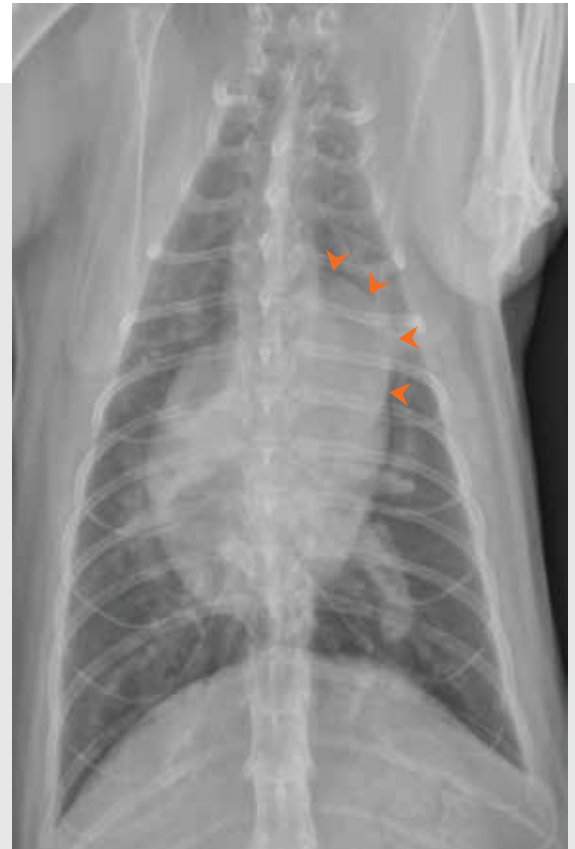


FIGURE 8. Ventrodorsal thoracic radiograph of a cat with a large, opaque soft tissue bulge at the 1 to 2 o'clock position along the cardiac silhouette (**arrowheads**), indicative of main pulmonary artery enlargement.

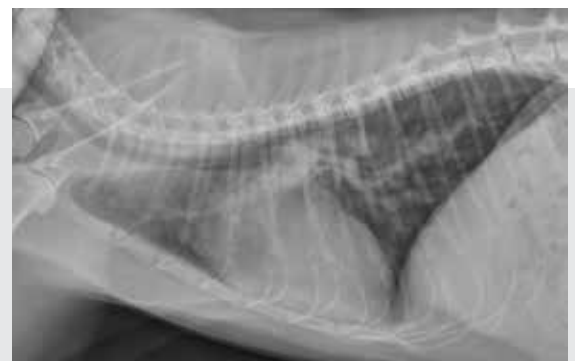


FIGURE 9. Right lateral thoracic radiograph of a cat with severe pulmonary hypertension and tortuosity of the lobar pulmonary arterial vasculature.

veno-occlusive disease resulting from alveolar remodeling associated with capillary hemangiomatosis can result in severe hypertension.⁷ Although best identified on computed tomography, nodular soft tissue opacities within the lungs may be radiographically apparent and seen in combination with a multifocal, unstructured, interstitial pulmonary pattern (most severe caudodorsally) and correlated to primarily represent a combination of vascular thickening, secondary edema, and/or hemorrhage (FIGURE 10).⁷

Increased pulmonary arterial pressure may result from increased circulating volume caused by shunting of blood from the left cardiac chambers into the right cardiac chambers secondary to a congenital heart defect.³ The most common shunting congenital cardiovascular anomalies include ventricular and atrial septal defects and patent ductus arteriosus (PDA). In cats, the most common congenital heart disease is

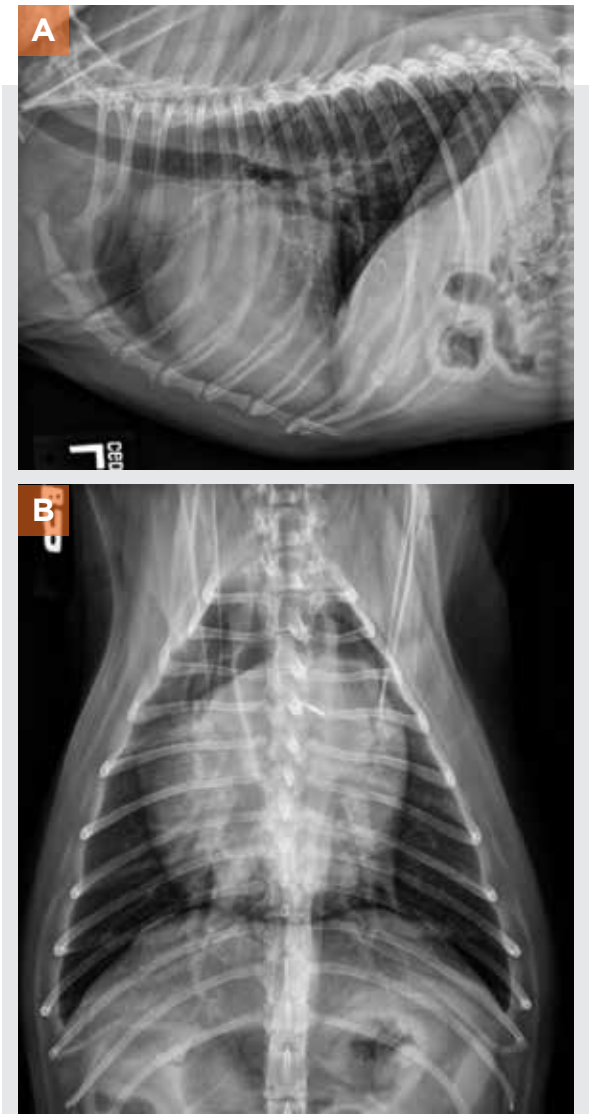


FIGURE 10. (A) Left lateral and (B) dorsoventral projections of a dog with moderate right-sided cardiomegaly and lobar pulmonary artery enlargement; scant pleural effusion; and mild, diffuse, unstructured, interstitial pulmonary pattern. In the absence of identifiable primary pulmonary disease, these findings were most consistent with pulmonary veno-occlusive disease and secondary cor pulmonale from underlying pulmonary hypertension.

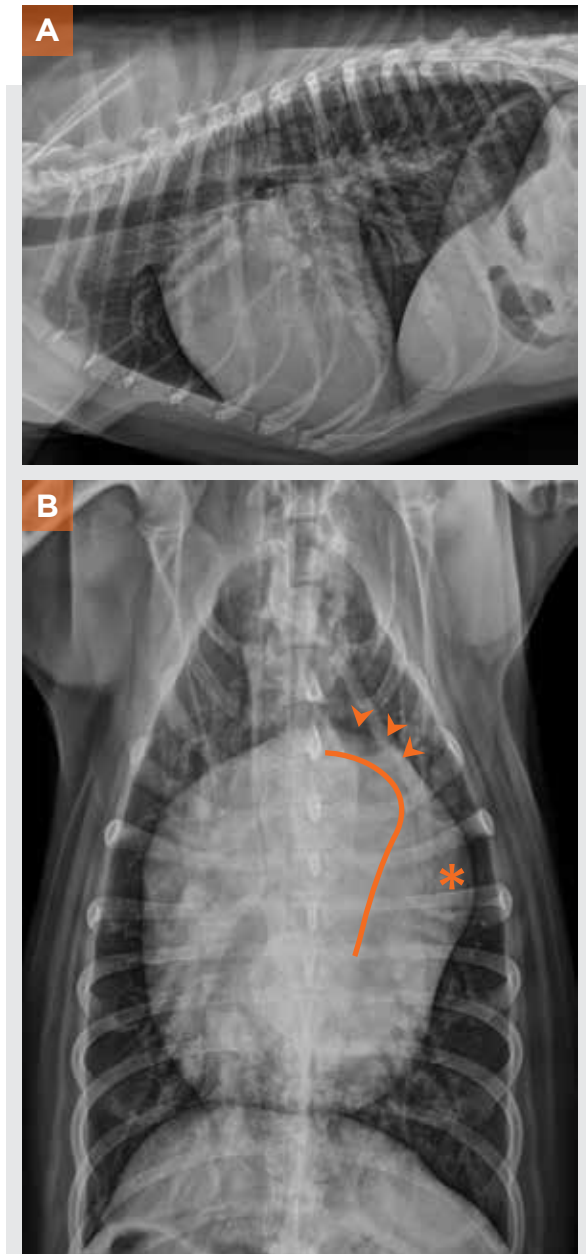


FIGURE 11. (A) Right lateral and (B) dorsoventral projections of a dog with patent ductus arteriosus, supported by aortic arch (outlined), main pulmonary artery (arrowheads), and left auricular (asterisk) enlargement on the ventrodorsal projection (“triple knuckles”).

ventricular septal defect; in dogs, PDAs are most prevalent.⁶ Thoracic radiographs of dogs and cats with a small shunting defect can be normal. Left-sided cardiomegaly may be observed in the presence of larger defects with left-to-right shunting, and the increased volume of blood into the right heart will precipitate main pulmonary artery enlargement. With increased severity and chronicity, shunt reversal (flow of blood from right to left cardiac chambers) can ensue from the increased resistance in the right ventricular outflow tract (e.g., Eisenmenger syndrome).⁶ A similar process may be observed in dogs with PDAs. Although not identified consistently in patients with PDAs, the combination of aortic arch, main pulmonary artery, and left auricular enlargement on a ventrodorsal projection (“triple knuckles”) may be pathognomonic for PDA (FIGURE 11).^{3,8}

Group 2: Left-Sided Cardiac Disease (Congenital or Acquired)

Pulmonary hypertension can develop as a sequela of heart failure from acquired left-sided heart disease, such as degenerative mitral valve disease (FIGURE 12), dilated cardiomyopathy, mitral valve dysplasia, or mitral or aortic valve stenosis.⁸ Borgarelli et al. identified that prognosis was poorer for dogs with degenerative mitral valve disease and pulmonary hypertension than those without pulmonary hypertension. Early phases of pulmonary hypertension may be reversible; irreversible remodeling of the pulmonary vasculature typically occurs with chronicity as increased vascular resistance causes a permanent elevation in pulmonary pressures.^{8,9}

Characteristic radiographic features of left-sided cardiomegaly include left atrial and ventricular enlargement. Left atrial enlargement is recognized by an opaque soft tissue bulge along the caudodorsal aspect of the cardiac silhouette in the region of the left atrium on lateral projections, which corresponds to a focal region of increased soft tissue opacity caudal to the tracheal bifurcation, resulting in splaying of the principal bronchi on the ventrodorsal/dorsoventral projections.³ Left-sided cardiomegaly is further supported by increased apical-to-basilar length of the cardiac silhouette and flattening of the caudal margin on lateral projections. On ventrodorsal/dorsoventral projections, a soft tissue bulge may be observed at the 3 o’clock position along the cardiac silhouette in the region of the left auricular appendage, indicative of concurrent left auricular enlargement.⁶ Although

rounding of the cranial and right lateral margins of the cardiac silhouette may be observed in dogs with severe left-sided cardiomegaly, caused by morphologic distortion of the heart from severe cardiac chamber enlargement, concurrent left- and right-sided cardiomegaly should also be considered, especially if main pulmonary artery enlargement, with or without lobar pulmonary artery enlargement, is also present.¹⁰ Cardiogenic pulmonary edema may not be observed at the time of pulmonary hypertension diagnosis; however, prior left-sided cardiac decompensation in those patients should be considered.⁸

Group 3: Respiratory Disease and/or Hypoxia

Pulmonary hypertension can be a sequela to a wide

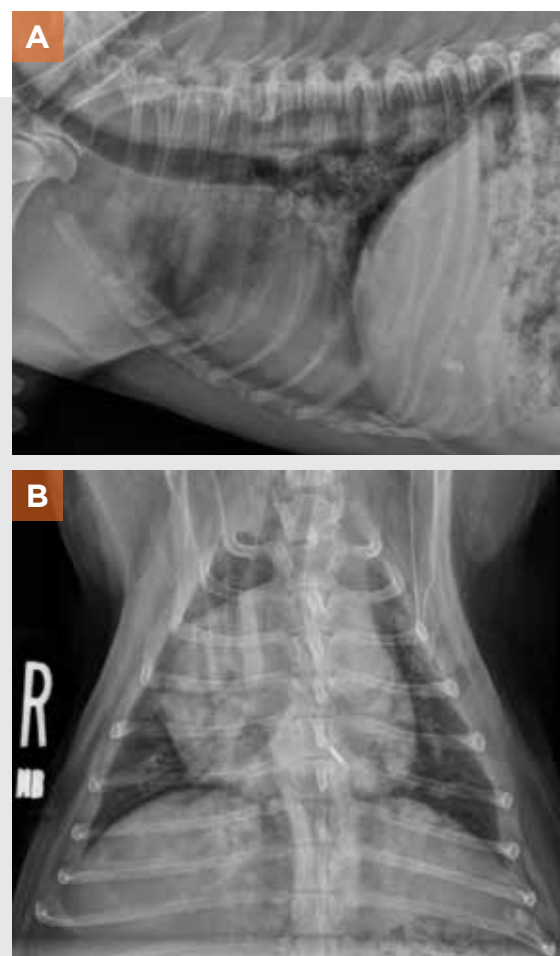


FIGURE 12. (A) Left lateral and (B) dorsoventral projections of a dog with acquired left-sided cardiomegaly secondary to degenerative mitral valve disease with pulmonary hypertension and concurrent right-sided cardiomegaly.

array of airway diseases in dogs and cats. Diseases of the lower airways primarily affect the bronchi (e.g., bronchitis, asthma) or interstitium (e.g., pneumonia) and etiology can be inflammatory, neoplastic, or infectious.¹ If the pulmonary hypertension is secondary to lower airway disease, radiographic evidence of bronchial or pulmonary parenchymal disease may be seen, although advanced imaging such as computed tomography or bronchoscopy may be necessary for a definitive diagnosis.⁶ Bronchial disease is recognized by thickened bronchi forming prominent rings and peripheral parallel “tram” lines.³ Although commonly seen in older patients, bronchial mineralization may be suggestive of chronic bronchitis. Asthma is most common in cats and is a cause of chronic pulmonary

obstruction. Although no studies support a link between the 2 disease processes, pulmonary hypertension and feline asthma may be diagnosed concurrently (**FIGURE 13**).^{6,11} In contrast, an unstructured/structured interstitial pulmonary pattern and/or alveolar pattern is indicative of primary interstitial disease.⁶ With increased duration, some interstitial diseases (e.g., neoplasia, bronchopneumonia) can result in pulmonary hypertension.¹ Some dog breeds (e.g., West Highland white terriers, Pekingese) are predisposed to interstitial pulmonary diseases and are at higher risk for the complication of pulmonary

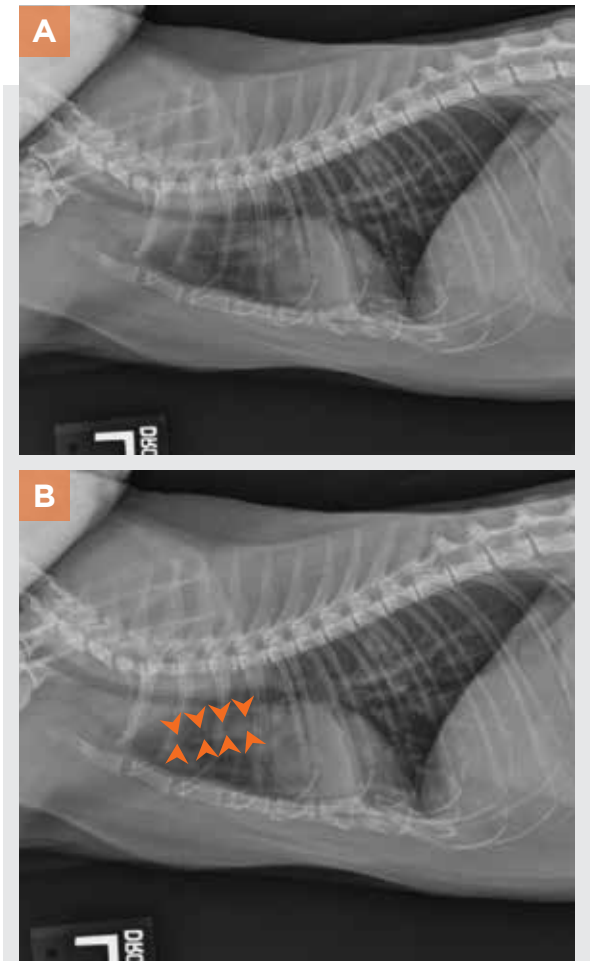


FIGURE 13. (A AND B) Left lateral projections of a cat with feline lower airway disease, characterized by the presence of a severe bronchial pattern. **(B)** The mild cardiomegaly and lobar pulmonary artery enlargement (**arrowheads**) are subsequent to pulmonary hypertension (diagnosed with echocardiography) and likely secondary to the chronic lower airway disease.

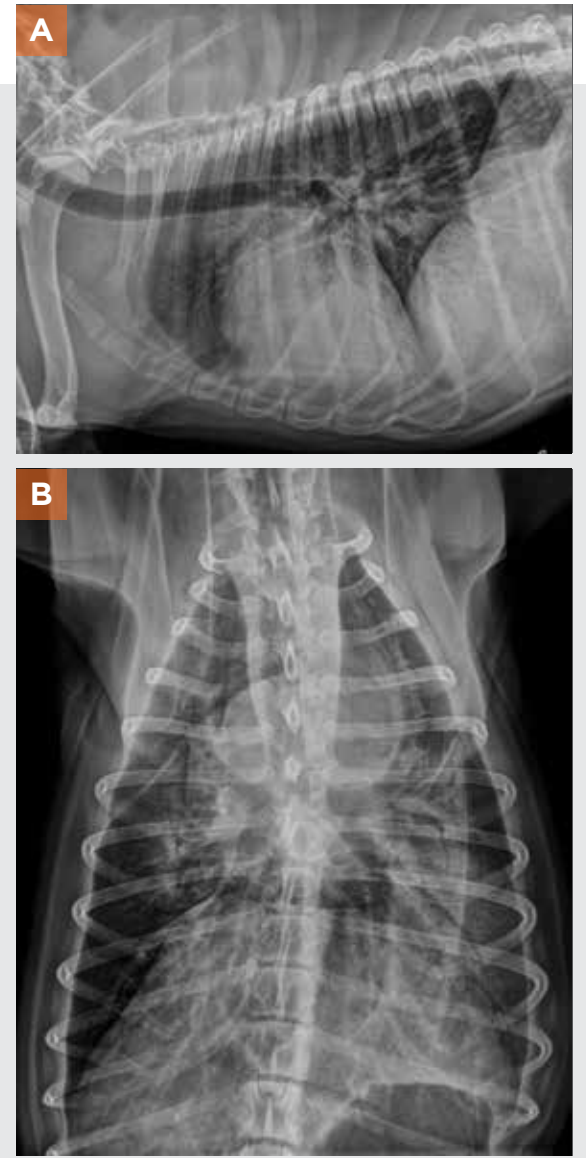


FIGURE 14. (A) Left lateral and **(B)** dorsoventral projections of a West Highland white terrier with idiopathic pulmonary fibrosis and cor pulmonale (main and lobar pulmonary enlargement and right-sided cardiomegaly).

hypertension (**FIGURE 14**).^{12,13} In other instances, onset of pulmonary hypertension may be acute.¹

The absence of radiographic abnormalities does not exclude the possibility of disease, and radiographic abnormalities may lag behind clinical signs. Other causes of obstructive airway disease may precipitate pulmonary hypertension.^{1,14} An example is bronchomalacia with or without tracheal and/or principal bronchial collapse, especially in dogs. Radiographically, undulation of the tracheal margins with variations in the diameter of the trachea or principal bronchi may raise concerns for bronchomalacia; however, radiography is not diagnostic and computed tomography and/or bronchoscopy are favored for a diagnosis.¹⁴ If dynamic tracheal and/or bronchial collapse is suspected, the diagnosis may require serial inspiratory and expiratory lateral radiography or a respiratory videofluoroscopic study.^{3,6}

Group 4: Pulmonary Thromboembolization

A pulmonary thromboembolism (PTE) represents material such as coagulated blood, neoplasia, or bacterial conglomerates lodged within the pulmonary arterial vasculature, occluding blood flow to the lungs.⁶ This process may be a sequela to altered blood flow, vascular injury, or a hypercoagulable state secondary to protein-losing diseases, hyperadrenocorticism, or disseminated intravascular coagulation (DIC).⁶ This obstruction may be partial or complete, resulting in varied disease severity and complications. Depending on the magnitude of the obstruction, pulmonary hypertension may result and can lead to right-sided heart failure in more severe cases.^{1,3,6,15}

For some patients with PTEs, radiographic abnormalities may not be seen. Although a nonspecific finding, the sole radiographic abnormality may be evidence of pulmonary hypertension (main and lobar pulmonary artery enlargement with or without right-sided cardiomegaly). The lobar pulmonary arteries may be peripherally blunted and tortuous.^{3,6} In the periphery of affected lung lobes, regions of pulmonary hypoperfusion (oligemia) may be observed. Other affected regions of the lung may have an unstructured interstitial to alveolar pulmonary pattern with a wedge shape and similar peripheral distribution (**FIGURE 15**).⁶

Group 5: Parasitic Diseases

Dirofilaria immitis and *Angiostrongylus vasorum* parasite infections are separated from other types of infectious etiologies because they cause pulmonary hypertension through a combination of pathophysiologic factors.¹

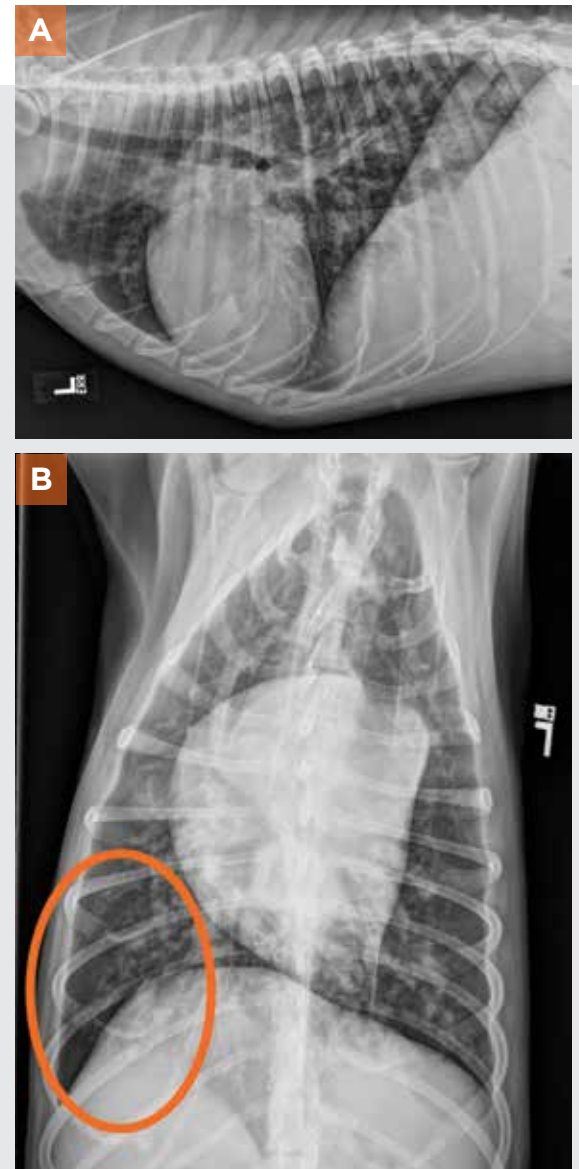


FIGURE 15. (A) Right lateral and (B) dorsoventral projections of a dog with mild right-sided cardiomegaly, mildly enlarged pulmonary arteries, mild pleural effusion, and multifocal left-sided unstructured interstitial coalescing to alveolar pulmonary pattern and regions of oligemia (circled). These findings are consistent with pulmonary hypertension and consequent right-sided heart failure (cor pulmonale), attributed to pulmonary thromboembolism.

***Dirofilaria immitis* Infection**

Of the 2 parasitic diseases, the most common in dogs and cats is dirofilariasis, especially in tropical and subtropical regions. Given cats' inherent resistance to infection, dirofilariasis is diagnosed at much lower frequency for cats than dogs. Although the parasite's life cycle differs for dogs and cats, transmission depends on a mosquito vector for both species.¹⁶

In animals with a higher worm burden, *D immitis* worms reside in the pulmonary arteries or the right heart.^{3,6} Cats have a propensity to exhibit a more severe clinical response to a lower worm burden than dogs, and pulmonary disease may develop in response to the presence of immature worms.¹⁷ Dogs may not show clinical signs of heartworm disease and the severity of clinical signs depends on the worm load, duration of infection, and degree of cardiovascular damage.⁶ Increased pulmonary arterial pressure can result from embolization of the pulmonary arteries by the worms, arteritis, and eosinophilic pneumonitis. Right-sided cardiomegaly and right-sided heart failure may ensue.^{3,6}

Radiographic features of *D immitis* worm infection include enlargement and tortuosity of the lobar pulmonary arteries, with or without right-sided cardiomegaly, as well as evidence of pleural effusion, caudal vena cava congestion, hepatomegaly, and peritoneal effusion (FIGURE 16).^{1,3,6} In patients with chronic infection, mineralization of the pulmonary vasculature may be apparent and represent an irreversible chronic change.^{3,6} Localization of cardiac chamber enlargement in cats is limited and most often characterized by generalized nonspecific cardiomegaly.⁶ A diffuse unstructured interstitial pulmonary pattern may be observed in all lung lobes in the presence of eosinophilic pneumonitis. Small, ill- or well-defined, opaque soft tissue and/or mineral nodules are most indicative of granulomatosis and increased chronicity.^{3,6} The lobar pulmonary arteries can also be truncated by pulmonary thromboemboli and/or physical obstruction by the worms themselves.^{3,6} Occasionally, in patients with pulmonary thromboembolization, regions of increased soft tissue opacity and/or hypovascularized lucent regions, mostly with a peripheral distribution, are identified.⁶ A superior method for assessing pulmonary perfusion to support a diagnosis of arterial occlusion is computed tomography.³

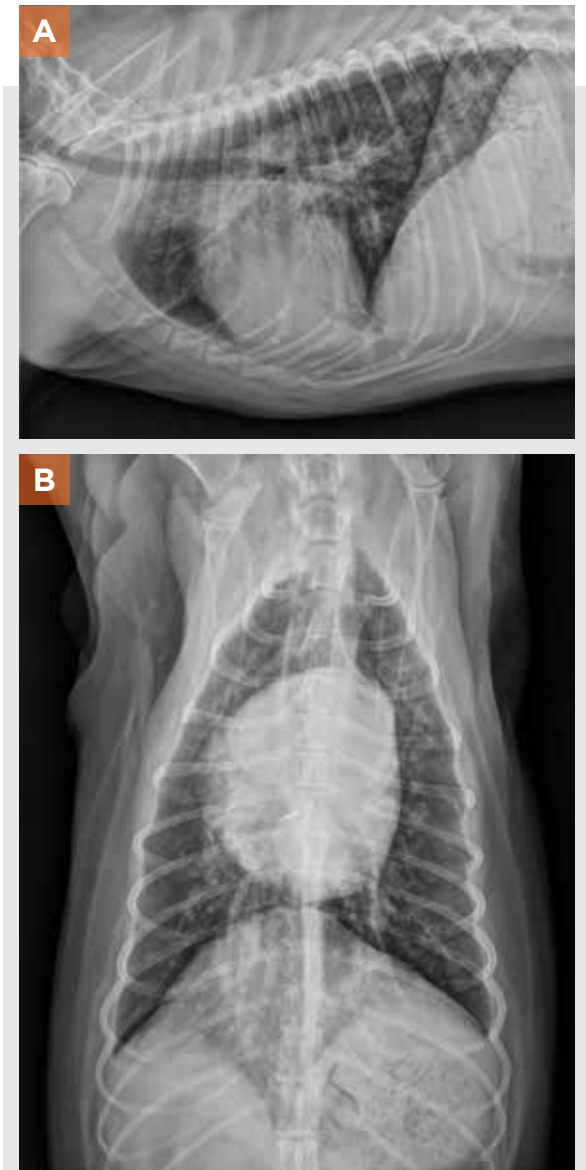


FIGURE 16. (A) Left lateral and (B) dorsoventral projections of a dog with moderate right-sided cardiomegaly and lobar pulmonary artery enlargement secondary to dirofilariasis with cor pulmonale. The mixed moderate bronchial and unstructured interstitial pulmonary patterns and nodules are consistent with eosinophilic bronchitis and pneumonitis with granulomatosis.

***Angiostrongylus vasorum* Infection**

A vasorum lungworms are widely distributed in regions of Africa, North and South America, and Europe and are transmitted by ingestion of gastropods.^{6,18,19} In dogs and cats, larvae migrate to the right ventricle and pulmonary arteries, where they reach maturity approximately 1 month after infection.¹⁸ Eggs are produced and released into the pulmonary capillaries where they hatch, and larvae then migrate into the alveoli and pulmonary interstitium.⁶ For a high percentage of dogs with *A vasorum* infection, thoracic

radiographs are reported to have a mixed bronchial, alveolar, and unstructured interstitial pulmonary pattern. When an alveolar pattern is present, a multifocal or peripheral distribution is most common (FIGURE 17).^{6,18} A similar pulmonary pattern is reported for dogs in which coagulopathy did or did not develop, which is a poorly understood complication of *A vasorum* infection and may be a manifestation of DIC.^{6,20} For a smaller percentage of cases, a small amount of pleural effusion has been reported.¹⁸ In addition, pulmonary hypertension may develop in some dogs, with radiographic evidence of right-sided cardiomegaly.²¹ Less commonly reported are main and lobar pulmonary artery enlargement, and radiographic abnormalities in the lungs may persist beyond therapeutic resolution of the infection.^{6,18}

Group 6: Multifactorial or Idiopathic

A diagnosis of pulmonary arterial hypertension may result from a combination of the aforementioned diseases.^{1,6} It may also be associated with other diseases, such as a mass compressing the pulmonary lobar arteries, for which radiography may be a useful diagnostic tool.¹ In other instances, the underlying etiology may be unclear.¹

SUMMARY

In combination or alone, main and lobar pulmonary arterial enlargement and right-sided cardiomegaly can be indicative of pulmonary hypertension. When in combination, pulmonary hypertension should be more strongly suspected; however, echocardiography remains the modality of choice for establishing a diagnosis. Radiography is noninvasive and widely accessible and therefore is one of the most practical imaging modalities for screening for an underlying cause of pulmonary hypertension and associated complications. However, for patients with mild cases of pulmonary hypertension, radiographic abnormalities may not be apparent. Radiographic abnormalities may help identify or suggest a primary cause; however, in some cases, advanced imaging such as computed tomography or additional diagnostic testing such as histopathology is necessary to confirm a diagnosis. Early diagnosis and treatment of pulmonary hypertension itself as well as the underlying cause may substantially improve or alleviate clinical signs associated with pulmonary hypertension. Without a prompt diagnosis, patients may be treated inappropriately and the pulmonary hypertension may result in right-sided cardiac

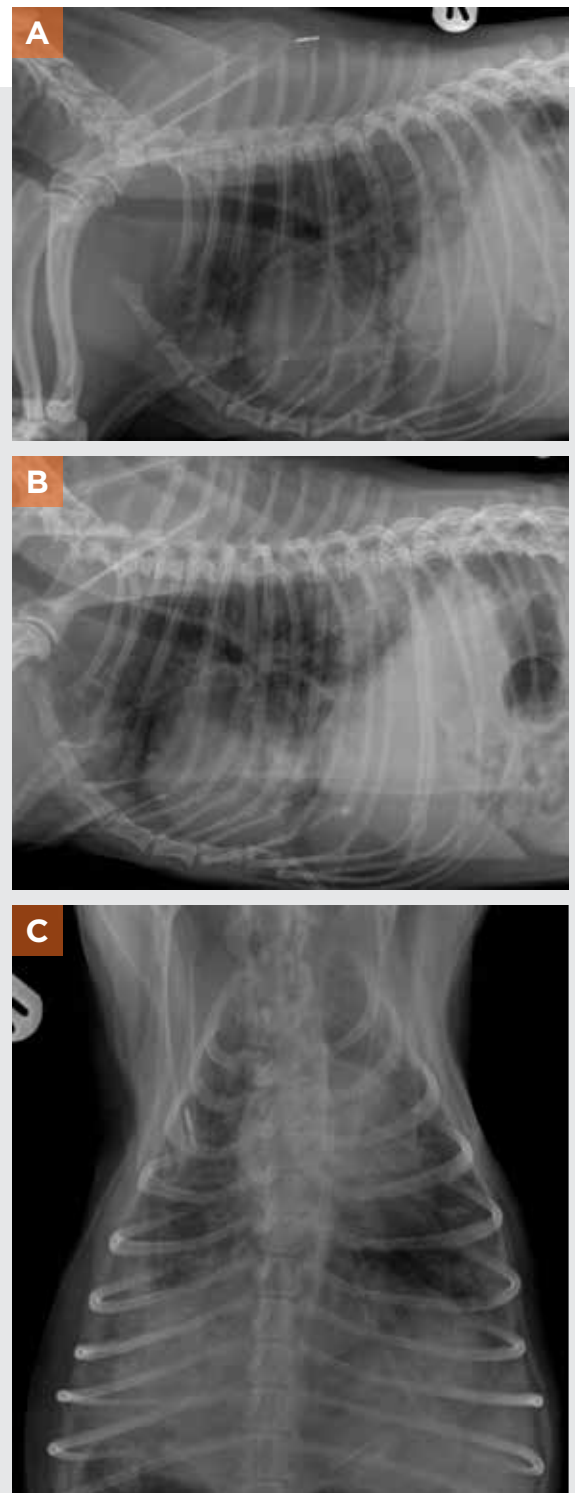


FIGURE 17. (A) Right lateral, (B) left lateral, and (C) ventrodorsal projections of a dog with a confirmed diagnosis of *Angiostrongylus* infection. A multifocal marked peripheral alveolar pattern can be identified in all lung lobes and is a common radiographic feature of angiostrongylosis. The rounding of the right cranial and lateral margins of the cardiac silhouette is attributed to subsequent pulmonary hypertension (confirmed with electrocardiography).



decompensation, carrying a poorer prognosis with increased severity and chronicity.⁸ **TVP**

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