Original Investigation

CT morphology of anomalous systemic arterial supply to normal lung in dogs

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Abbreviations: ASANL, anomalous systemic arterial supply to the normal lung

ABSTRACT

Anomalous systemic arterial supply to the normal lung (ASANL) is a rare congenital anomaly in humans, in which the systemic arteries supply the basal segments of the lower lobe. It has a normal bronchial connection, but lacks a normal pulmonary artery. This anomaly has never been reported in the veterinary literature. The objectives of this retrospective descriptive study were to characterize the CT findings and clinical features of ASANL, and to determine the breed predisposition in a population of referral canine cases. Thoracic CT images, in which the caudal lung lobes were fully inflated and the pulmonary artery could be traced to the periphery, were reviewed. A total of 1,950 dogs were enrolled, and the aberrant vasculature equivalent to ASANL in humans was detected in 48 dogs. Shetland Sheepdogs (7/48, OR=8.0, $P<0.00001$), Miniature Dachshunds (19/48, OR=3.9, $P<0.00001$), and Labrador Retrievers (6/48, OR=4.5, $P=0.0009$) were over-represented. The affected lung lobes were the right caudal lobe (24/48, 50%), the left caudal lobe (21/48, 43.8%), and bilateral caudal lobes (3/48, 6.3%). The aberrant vessels originated from the left gastric artery (14/48), descending thoracic aorta (8/48), celiac artery (6/48), and splenic artery (1/48). In the remaining 19 cases, the origin of the aberrant vessels could not be determined. Although the clinical significance of ASANL in dogs remains unclear, surgeons should be aware of this finding prior to lobectomy of the caudal lung lobes to avoid intraoperative systemic arterial bleeding.

INTRODUCTION

The normal lung is supplied by the pulmonary and bronchial arteries. Pulmonary arteries supply the distal portion of the respiratory bronchiole, alveolar duct, alveoli, and the thin pleura, and the bronchial arteries supply the tracheobronchial lymph nodes, peribronchial connective tissue, and the bronchial mucous membrane.[1] The bronchial arteries mostly arise from the bronchoesophageal artery, which arises from the right fifth intercostal artery close to its origin from the aorta, crosses the left face of the esophagus, and branches at the tracheal bifurcation of the left and right bronchial arteries. The bronchial arteries communicate with the pulmonary arteries through the capillary bed at the level of the respiratory bronchioles.[1,2] Systemic arterial supply to the lungs refers to all abnormal vessels supplying the lung, arising directly or indirectly from the aorta in humans.[3] Among several types of systemic arterial supply to lungs, anomalous systemic arterial supply to normal lung (ASANL), also known as anomalous systemic arterial supply to the basal segments of the lung, is the rarest form; in this type, a portion of the lung lacking a normal pulmonary artery receives systemic arterial supply and drains through the normal pulmonary veins.[4-6] Based on our review of the literature, ASANL has never been reported in the veterinary literature.

ASANL is often asymptomatic and may be diagnosed following detailed examination of an incidental cardiac murmur, for example.[7] When symptomatic, clinical signs include hemoptysis, heart murmur, exertional dyspnea, tachypnea, and cough.[8] Although most adult patients are asymptomatic, patients with this disease are recommended to undergo surgery or endovascular embolization, because of the potential risk of hemoptysis due to pulmonary hypertension and heart failure due to left-to-left shunt.[7] It is very important to differentiate ASANL from other pulmonary vascular anomalies, while selecting the appropriate surgical procedure. Therefore, the computed tomographic morphology of ASANL has been investigated. Reports suggest that Asians are predisposed to have ASANL.[9] If a pulmonary vascular anomaly equivalent to ASANL exists in dogs, it is important to investigate its clinical findings.

The aim of the present retrospective descriptive study was to investigate whether ASANL exists in dogs, and if it does, to evaluate its CT morphology, clinical significance, and breed predisposition in dogs by reviewing CT images in a population of canine referral cases.

METHODS

Selection and Description of Subjects

This retrospective descriptive study was performed at the Hokkaido University Veterinary Teaching Hospital. Approval by the Animal Care and Use Committee of our institution was not required due to the retrospective nature of the study. Consent was obtained from the owners of all dogs recruited for this study. Inclusion criteria were applied to the client-owned dogs that underwent thoracic CT examination for diagnostic and monitoring purposes at a single referral hospital (Hokkaido University Veterinary Teaching Hospital) between April

2015 and June 2020. The CT images where the pulmonary artery of the caudal lung lobes could not be traced to the periphery because of inadequate inflation, motion artifacts, or any type of intra-thoracic airway abnormality (e.g., bronchiectasis or bronchial plugging) or parenchymal abnormality (e.g., ground-glass opacity or consolidation) were excluded. The following data were collected from the medical records: signalment, clinical signs, abnormalities detected at the physical examination, purpose of CT studies, and when available, the results of echocardiography. All decisions for exclusion and data collection was made by a veterinary radiology specialist with 10 years of experience in veterinary radiology (G.S.).

Data Recording and Analysis

Data on computed tomographic examinations of patients were retrieved and assessed using a PACS workstation DICOM viewer (OsiriX, Pixmeo, Geneva, Switzerland). All thoracic CT images had been reconstructed using 0.5 to 2.0 mm reconstruction slice thicknesses, and the same interslice gap as the slice thickness, as recommended by the attending veterinary radiology specialist; however, the raw data were not available because of the retrospective nature. All CT images were reviewed by the aforementioned veterinary radiology specialist (G.S.). The diagnostic criteria for ASANL were as follows: 1) portions of the caudal lobe(s) had no normal pulmonary arterial supply, 2) an anomalous systemic artery fed that region, 3) bronchial connection of the region was normal, and 4) the venous return to the left atrium occurred via the pulmonary vein.[10,11] A bronchial connection was considered normal when it connected the normal pulmonary parenchyma that it ventilated to the tracheobronchial tree. The origin of the aberrant vessels were determined where possible.

Statistics

All statistical analyses were selected and performed by a veterinarian with a PhD degree in veterinary science (G.S.), using commercial software (JMP Pro version 14, SAS Institute, Inc., Cary, NC, USA). To evaluate the predisposition of ASANL in each breed, the odds ratios and 95% confidence intervals (CIs) were calculated if the number of the control population was at least 20 in each breed category. The control population for breed-specific odds ratio calculations consisted of all dogs included in this study for each breed, with the number of dogs from that breed with a diagnosis of ASANL subtracted from the total. The formula used for the calculation of odds ratios was as follows: odds ratio = [(number of cases for a breed)/(Number of control population for the breed)]/[(number of cases for all other breeds)/(Number of control population for all other breeds)]. Logistic regression analysis was used to determine the 95 % confidence interval, and to calculate P-values for assessing the statistical significance of the predisposition of each breed. A significance level of $P< 0.05$ was modified using a Bonferroni correction, such that $P<0.0038$ was considered significant. The differences in morbidity among males and females were also examined using the odds ratio.

RESULTS

Data on a total of 2,287 dogs who had undergone thoracic scans were found in the hospital database during the study period; 337 dogs were excluded owing to the following imaging findings in the caudal lobes: evidence of atelectasis (i.e., ground glass opacity [GGO] or consolidation associated with a decrease in lung volume often found, but not limited, to the dependent portions of the lungs) (170), motion artifacts (84), and lung pathology (83). Thus, a total of 1,950 dogs (1,075 males and 875 females) were included in this study. The purpose of the thoracic CT studies was to perform staging in neoplastic conditions (1636), to evaluate thoracic diseases (60), and to screen for lung diseases in cases of abdominal diseases (158), head and neck diseases (64), musculoskeletal diseases (19), and intervertebral disc disease (12).

All CT examinations were performed using an 80-row multidetector CT scanner (Aquilion Prime, Canon Medical Systems, Tochigi, Japan). Patients were examined in ventral recumbency (1,926) or dorsal recumbency (24) with the dogs anesthetized (1,907), sedated (4), or conscious (39). In cases where CT studies was performed under general anesthesia using a ventilator, apnea was induced during the acquisition by stopping the ventilator (acquisition at the end of expiration). The scan settings included a pitch of 0.813, tube rotation time of 0.5 s, scan slice thickness of 0.5 mm, tube potential of 120 kV, and tube current of 60–500 mA; these were automatically calculated by a commercial software package (Sure Exposure 3D; Canon

Medical Systems, Tochigi, Japan). The performance of contrast studies was not standardized. For contrast studies, iohexol (Omnipaque 300, GE Healthcare, Oslo, Norway) was used as a contrast medium; it was administered at a dose of 600 mgI/kg via the cephalic vein, through a power injector (Dual Shot GX7, Nemoto Kyorindo Co., Ltd., Tokyo, Japan). For small and medium sized dogs (approximate weight <30 kg), the injection duration was set to 20 s; it was set to 30 s for larger dogs. Contrast studies consisted of three phases; in cases where the duration of injection was 20 s, the arterial, portal, and delayed phases were acquired at 26, 50, and 180 s after the injection of the contrast medium, respectively. In cases where the duration of injection was 30 s, the arterial, portal, and delayed phases were acquired 36, 60, and 180 s after the injection of the contrast medium, respectively. Total body scans were performed in all patients. When a detailed examination of the lung was included for the purpose of the CT examination, additional thoracic scans were acquired with an airway pressure of 10 cmH_2O, which has been reported to improve lung inflation.[12]

Among the 1,950 dogs included in this study, 48 (2.4%) dogs (33 males and 15 females) met the diagnostic criteria of ASANL. Among the cases with ASANL, the number of males was twice that of females, but no statistically significant difference (OR=1.8, P=0.058) was found. The median age at the time of the CT study was 11 years (range: 1-16 years). The affected breeds and odds ratios are summarized in Table 1. Shetland Sheepdogs (7 cases, OR=8.0, P<0.0001), Miniature Dachshunds (19 cases, OR=3.9, P<0.0001), and Labrador

Retrievers (6 cases, OR=4.5, P=0.0009) were over-represented relative to the control population.

The purposes of the CT studies included assessment of the staging in patients with neoplastic disease (n = 35), evaluation of non-neoplastic abdominal disease (10), whole body screening in extremely debilitated patients (2), and evaluation of intervertebral disc disease (1). Clinical signs related to cardiovascular or respiratory diseases were detected in 5 patients; 3 had epistaxis with nasal tumors, 1 had nasal discharge with rhinitis, and 1 had upper respiratory obstruction with dynamic nasopharyngeal collapse. On physical examination, 3 cases had a mild heart murmur (murmur intensity of 2/6). Echocardiography was performed in 13 cases; 2 had stage B1 myxomatous mitral valve disease (MMVD), 1 had stage B2 MMVD, and the others had no abnormalities. Therefore, only 1 dog (an 11-year-old chihuahua) was found to have left atrial enlargement.

In all ASANL cases, the connection of the bronchial tree was normal; however, the pulmonary arteries to the caudomedial portions of the right and/or left lung lobes were lacking. The aberrant vessels were running from within the mediastinum toward the bronchi in the region lacking pulmonary arteries; from there, they ran caudally along the bronchi similar to pulmonary arteries (Figures 1-4). The median diameter of the aberrant vessels at the insertion of the mediastinum was 1.0 mm (range, 0.7-2.6 mm). Aberrant vessels were found in the right caudal lobe in 24 cases, in the left caudal lobe in 21 cases, and in bilateral caudal lobes in 3 cases. The origin of the aberrant vessels was determined in 29/48 cases (14 cases, left gastric artery; 8 cases, descending thoracic aorta; 6 cases, celiac artery; and 1 case, splenic artery) (Figures 2, 4). In the remaining 19 cases, the origin of the aberrant vessels could not be determined due to the lack of contrast studies or arterial phase images, the lack of images reconstructed using soft-tissue reconstruction kernels, or the extremely narrow diameter of the aberrant artery. There were 4 cases with 2 aberrant vessels. One case has 2 aberrant vessels originating from thoracic aorta at the level of 11th and 12th thoracic vertebra, both of which supplied the right caudal lobe. In another case, one aberrant vessel originating from the thoracic aorta supplied the right caudal lobe; the other originating from the left gastric artery supplied left caudal lobe. In the other 2 cases, the origin of the aberrant vessels could not be determined. The results of the CT findings are summarized in Table 2.

DISCUSSION

This study demonstrated that pulmonary vascular malformations equivalent to ASANL in humans also exist in dogs. Shetland Sheepdogs, Miniature Dachshunds, and Labrador Retrievers were significantly overrepresented; however, it is necessary to note that these results are based on patients that presented to a single referral veterinary institution. At the time of the current study, we did not find any published reports describing the incidence of ASANL in humans; however, Asian populations appear to have a higher incidence of the disease (> 90%), suggesting a probable genetic etiology.[9,10]

In cases where systemic arterial supply to the lung is identified in humans, the differential diagnoses include hypertrophied systemic arteries associated with chronic pulmonary parenchymal inflammation, the hypogenetic lung syndrome, and intra- or extralobar pulmonary sequestration.[7,11] Diagnoses are obtained based on the description of the venous return and pulmonary arterial supply of the involved lung, and the absence of a normal bronchial tree. CT angiography is the best suited modality, because it allows the evaluation of all components.[11] In our cases, there was no evidence of chronic pulmonary parenchymal inflammation, abnormal pulmonary venous return, or abnormal bronchial connections. Furthermore, the aberrant systemic artery solely supplied the normal caudomedial portion of the right or left lobes without normal pulmonary arterial supply to the involved area; these findings are consistent with the diagnosis of ASANL.[8,13]

The most common clinical symptom of ASANL in humans is hemoptysis; however, the majority of patients are asymptomatic, and ASANL is often discovered incidentally.[10,14] Computed tomography may reveal ground glass opacity (GGO) in the affected area, which indicates an area of relative hypervascularity and/or intra-alveolar hemorrhage.[7,15] Other manifestations include exertional dyspnea, heart murmur, and congestive heart failure secondary to the cardiac overload produced by left-to-left shunts.[8,16,17] In this study, none of the cases had hemoptysis or GGO. A heart murmur was present in 3 of the 11 cases in which the results of auscultation were derived from the medical records. However, it was assumed that the cases without any records of auscultation did not have a heart murmur. In addition, among the 13 cases with echocardiography, only an 11-year-old Chihuahua was found to have left atrial enlargement. Since this is a breed with a high incidence of MMVD, it is unlikely that ASANL solely caused the volume overload to the left atrium in this case. Since the CT studies were not performed for cardiovascular or respiratory symptoms, ASANL was presumed to be an incidental finding in all cases; however, further prospective studies are needed to clarify the clinical significance of ASANL in dogs.

A possible explanation for these differences in the clinical and imaging findings between humans and dogs is the differences in the diameter of the aberrant arteries. The clinical symptoms and GGO in humans are caused by the volume overload to the heart due to aberrant systemic arteries, and the higher pressure of the systemic circulation compared to the pulmonary circulation.[15,17] The diameter of the aberrant systemic arteries in humans has been reported to be 6-30 mm.[8,10,15] Conversely, the median diameter of the aberrant arteries in this study was only 1.0 mm. One hypothesis is that the volume of aberrant arterial flow was considerably low for affecting the cardiovascular system in dogs.

In the present study, ASANL had equal incidence in the right and left caudal lobes, and the most common origin of the aberrant arteries was the left gastric artery, followed by the descending thoracic aorta. These findings differ from those reported in humans, where the left lower lobe is most commonly affected, and the right lower and upper and middle lobes are rarely involved.[7] The aberrant arteries most frequently originate from the descending thoracic aorta, followed by the celiac axis.[14,17] Although the cause of ASANL is unknown, it is believed that it may result from the abnormal persistence of embryonic aortic postbranchial arches, that supply the lung buds before development of the main pulmonary artery.[8,18] Differences in the distribution of aberrant arteries between dogs and humans may be attributed to anatomical differences in the lung lobes.

One potential clinical significance of ASANL in dogs would be the risk of bleeding during lung lobectomy of the caudal lobes. If the surgeon attempts to resect the caudal lobe without being aware of the presence of ASANL, unexpected systemic arterial bleeding may occur. Therefore, it is recommended to conduct CT in patients scheduled for caudal lung lobectomy to check for the presence of the ASANL. In this instance, it is crucial to perform the CT study while the patient is in a ventral recumbent position to prevent gravity-dependent atelectasis of the caudodorsal area of the caudal lobes, and to obtain arterial phase images, which cover as far as the level of the celiac artery. If the imaging is performed in dorsal recumbency, atelectasis often occurs mostly in part of the periphery of the caudal lobes, even if the airway pressure is positive.[12] Since the aberrant systemic artery is considerably small and supplies the caudal peripheral area of the caudal lobes, even small volumes of atelectasis would mask the aberrant artery, making detection difficult.

This study has several limitations. It included a population that presented to a single referral institution; therefore, the results on breed predisposition may reflect the geographical breed distribution or genetic bias. In addition, the CT and echocardiographic procedures were not standardized due to the retrospective nature of the study. The origin of the aberrant systemic arteries could also not be determined in 19 of 48 cases; this was partly due to the absence of images of the arterial phase, or the availability of arterial phase images of only 2 mm thickness. Additionally, echocardiography was not performed in all CT cases, and only 13 of 48 ASANL cases underwent echocardiography. In addition, functional abnormalities could not be completely excluded in dogs with ASANL in this study, even if no parenchymal abnormalities were found on CT images. Therefore, the true incidence and clinical significance of ASANL in dogs could not be determined. Furthermore, since no cases of ASANL underwent lobectomy of the caudal lung lobes in this study, it is not clear whether ASANL actually affects surgical procedures.

In conclusion, the incidence of ASANL in dogs is 2.4%, with an equal incidence in the right and left caudal lobes. In this population from a single referral institution, analysis demonstrated significantly higher incidence among Shetland Sheepdogs, Miniature Dachshunds, and Labrador Retrievers. Although the clinical significance of ASANL is not clear, surgeons should be aware of this finding prior to lobectomy of the caudal lung lobes, as it may affect surgical planning. Therefore, when performing CT studies in patients scheduled for caudal lung lobectomy, it is strongly recommended that the arterial phase is obtained in ventral recumbency, including sections from the thorax to the celiac artery.

List of Author Contributions

Category 1

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Category 2

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Tables

Table 1. Odds ratios of dog breeds diagnosed with anomalous systemic arterial supply to normal lung

	Dogs with ASANL (n=48)	Dogs without ASANL (n=924)	Odds Ratio	Lower 95% CI[a]	Upperr 95% CI	P value
Shetland Sheepdog*	7	39	8.0	3.4	19	<.0001
Miniature Dachshund*	19	270	3.9	2.2	7.1	<.0001
Labrador Retriever*	6	57	4.5	1.9	11	.0009
American Cocker Spaniel	3	43	2.9	0.86	9.6	.0860
Chihuahua	3	168	0.66	0.20	2.2	.493
Border Collie	1	25	1.6	0.21	12	.649
Boston Terrier	1	27	1.5	0.20	12	.677
Toy Poodle	2	126	0.6	0.14	2.5	.479

Golden Retriever	2	54	1.5	0.35	6.3	.589
Welsh corgi	1	100	0.36	0.05	2.7	.320
West Highland White Terrier	1	10	ND[b]	ND	ND	ND
Scottish Terrier	1	5	ND	ND	ND	ND
Bolognese	1	0	ND	ND	ND	ND

a: confidence interval
b: not determined
P<.0038 is considered significant
*Breeds significantly predisposed to ASANL

Table 2. Summary of the distribution, origin, and number of aberrant arteries.

		Number of cases
Affected lung lobe	Right caudal lobe	24
	Left caudal lobe	21
	Bilateral caudal lobes	3
Origin of aberrant artery	Left gastric artery	14
	Descending thoracic aorta	8
	Celiac artery	6
	Splenic artery	1
	Uncertain	19

Number of aberrant arteries	1	44
	2	4

Figure legends

Figure 1

The lung window (WL = -150 HU, WW = 1800 HU) (A-E) and the corresponding soft tissue window (WL = 40 HU, WW = 350 HU) (F-J) for post contrast transverse images of the left caudal lung lobe of left-sided ASANL in a 12-year-old spayed female Border Collie (soft-tissue reconstruction algorithm, 0.5 mm slice thickness, ventral recumbency).

The bronchus in the caudomedial portion of the left caudal lobe lacks the pulmonary artery (B, C, D: arrowheads). From that level, an aberrant vessel (C, D, E: arrows) arising from the mediastinum supplies that region, as if it were a pulmonary artery.

Figure 2

Maximum intensity projections of 3D multiplanar reconstruction images (A – D: transverse, E: oblique sagittal, F: oblique dorsal) of the same case as shown in Figure 1 (WL = -150 HU, WW = 1800 HU, soft tissue reconstruction algorithm, 0.5 mm slice thickness). The aberrant vessel (arrowheads) originates from the left gastric artery (arrow) and supplies the caudal periphery of the left caudal lung lobe. The aorta (open arrow) and the celiac artery (open arrowhead) are indicated.

Figure 3

The lung window (WL = -150 HU, WW = 1800 HU) (A-E) and the corresponding soft tissue window (WL = 40 HU, WW = 350 HU) (F-J) for post contrast transverse images of the right caudal lung lobe of right sided ASANL in a 7-year-old male Golden Retriever (soft-tissue reconstruction algorithm, 0.5 mm slice thickness, ventral recumbency). As in the case of figures 1 and 2, the bronchus in the caudomedial portion of the right caudal lobe lacks the pulmonary artery (arrowhead), and an aberrant vessel (arrows) supplies the normal lung instead of the normal pulmonary artery.

Figure 4

Maximum intensity projection images of the caudal thorax of right sided ASANL in a ten-year-old neutered male Shetland Sheepdog (WL = -150 HU, WW = 1800 HU, soft tissue reconstruction algorithm, 0.5 mm slice thickness, ventral recumbency). The aberrant vessel (arrow) originates from the descending thoracic aorta and supplies the caudal periphery of the right caudal lung lobe (arrowheads).