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Intrapulmonary arteriovenous anastomoses in dogs with severe *Angiostrongylus vasorum* infection: clinical, radiographic, and echocardiographic evaluation☆

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Abstract *Background:* In both humans and dogs the pulmonary vasculature is able to recruit large-diameter anatomical intrapulmonary arteriovenous anastomoses (IPAVAs). In healthy people the opening of these anastomoses affects the degree of exercise-induced increase in pulmonary arterial pressure. The presence of these IPAVAs can be demonstrated using saline contrast echocardiography.
Objectives: The aims of the present study were to characterize severely affected, naturally infected dogs with *Angiostrongylus vasorum*, to evaluate if these dogs can open IPAVAs, and to assess if the recruitment of such anastomoses affects the severity of pulmonary hypertension (PH).

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Animals: Eight client-owned dogs with severe *A. vasorum* infection were recruited.
Methods: Dogs with *A. vasorum* infection that presented with severe dyspnea and/or syncope were prospectively screened by echocardiography for the presence of PH and IPAVAs. Only severely affected dogs, based on a combination of clinical, radiographic and echocardiographic abnormalities, were enrolled.
Results: Opening of IPAVAs could be demonstrated in three dogs with no to moderate PH, and could not be demonstrated in five dogs with severe PH. In two dogs thoracic radiographs showed only mild interstitial changes, while computer tomography and postmortem examination revealed severe pulmonary interstitial and vascular disease.
Conclusions: These results suggest that dogs may open IPAVAs and that opening of such anastomoses may play a regulatory role in the development of PH. There may be a marked discrepancy between radiographic changes and disease severity in *A. vasorum*.

Abbreviation Table

AT	acceleration time
CT	computed tomography
ET	ejection time
IPAVAs	intrapulmonary arteriovenous anastomoses
LV	left ventricle
PAP	pulmonary arterial pressure
PG	pressure gradient
PH	pulmonary hypertension
PR	pulmonic regurgitation
PVR	pulmonary vascular resistance
RV	right ventricular
SCE	saline contrast echocardiography
sPAP	systolic pulmonary arterial pressure
TAPSE	tricuspid annular plane systolic excursion
TR	tricuspid regurgitation

Introduction

The pulmonary vasculature is able to recruit large-diameter intrapulmonary arteriovenous anastomoses (IPAVAs) in healthy humans and dogs [1,2]. These are dynamic preformed silent vascular conduits that, when opened, create a bypass through the pulmonary microcirculation as these anastomoses allow arterial blood to bypass the capillary beds and join up with postcapillary venous blood [3]. The presence of such anastomoses can be demonstrated by contrast echocardiography [4]. There is anatomical [2,5,6] and biophysical [2,7,8] evidence of the presence of IPAVAs in humans and dogs. The ability to recruit IPAVAs varies between individuals, and not all humans or dogs, healthy or diseased, are able to open IPAVAs [1,2,7,9]. These physiological anastomoses are not to be confused with macroscopic arteriovenous-fistula malformations, even though the consequences may be similar [10,11].

Intrapulmonary arteriovenous anastomoses may be involved in both physiological and pathological conditions, although their precise role remains controversial [12,13]. One potential important role may be the regulation (decrease) of pulmonary arterial pressure (PAP) in health, e.g. during exercise, and disease. Specifically, IPAVA may work as "pop-off valves" in the face of increased flow and pressure thus minimizing damaging effects on the pulmonary microcirculation and reducing right ventricular (RV) afterload [14].

Pulmonary hypertension (PH) is a hemodynamic abnormality of considerable and growing importance in dogs, *Angiostrongylus vasorum* being an increasingly recognized cause [12—17]. This parasite causes severe pulmonary vascular and parenchymal lesions initiated by an intense immune response against eggs and larval antigens in (peripheral) pulmonary arteries. In some dogs, widespread pulmonary thrombosis can be found predominantly in more peripheral, but sometimes even in the main pulmonary arteries. In other dogs, widespread essentially complete pulmonary parenchymal destruction with severe hemorrhage and loss of recognizable architecture is found. Finally, there is some pulmonary artery remodeling