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Esophageal varices in dogs: A retrospective case series

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Abstract

Background: Esophageal varices (EV) are abnormally dilated veins in the esophagus caused by alterations of blood flow or pressure. Esophageal variceal hemorrhage is a major complication of hepatic disease in humans, but a lack of information exists regarding associated adverse events in dogs.

Objective: To describe the clinical manifestations and associated etiologies and outcomes of dogs with EV.

Animals: Twenty-five client-owned dogs with EV diagnosed via computed tomography (CT), endoscopy, or fluoroscopy.

Methods: Retrospective case series. Cases were identified by review of the hospital imaging records database between 2010 and 2020. Signalment, clinical signs, and outcomes were documented. When present, additional collateral vasculature was also recorded. Cases were subcategorized into suspected etiology based upon the anatomic location or absence of an attributable underlying disease process, as well as the direction of blood flow.

Results: Twenty-four of 25 cases were identified via CT, with a prevalence of 0.012% (24/1950 total studies). Presenting clinical signs were nonspecific, and more likely because of the underlying cause as opposed to complications secondary to EV themselves. Etiologic anatomic locations were similar in occurrence between the abdomen (N = 14) and thorax (N = 11). All cases with an abdominal etiologic location had presumed or confirmed portal hypertension and 9/11 cases with a thoracic etiologic location had pulmonary, caval, or systemic hypertension. No cases died or were euthanized as a direct result of EV or associated hemorrhage.

Conclusions and Clinical Importance: Esophageal varices are rarely reported in dogs and commonly identified concurrently with portal, pulmonary, and caval hypertension. Hemorrhage is not a common clinical manifestation of EV.

KEYWORDS
collateral circulation, varix, vascular abnormality

Abbreviations: CT, computed tomography; EV, esophageal varices; GI, gastrointestinal.

1 | INTRODUCTION

Esophageal varices (EV) are a leading cause of death in people with cirrhotic hepatic disease and occur in dogs secondary to a number of disease processes resulting in obstruction or abnormal portal or caval blood flow.[1-4] Esophageal varices are defined as normal veins of the esophagus that are abnormally dilated because of increased vascular resistance in the portal venous system or superior/cranial vena cava (SVC), and further characterized as "uphill" or "downhill" depending on the direction of venous flow.[1-3,5,6] Downhill varices are rare, extend downwards, or caudally, and usually develop secondary to superior vena cava obstruction. Uphill varices extend upwards, or cranially, are commonly associated with portal hypertension from cirrhotic hepatic disease, and are more often responsible for hemorrhage and associated death in humans. While this variation accounts for nearly half of EV reported in dogs, only 1 report of variceal hemorrhage currently exists.[7]

Previous studies have characterized both advanced imaging features and proposed pathophysiology of EV in a small number of dogs.[1,2] Reported etiologies have included venous thrombi, pulmonary thrombosis, portal hypertension, space occupying masses, and bronchoesophageal arterial hypertrophy. In 2 case reports of dogs with EV, both had final diagnoses of arteriovenous fistulae formation.[3,7] However, clinical signs and outcomes of dogs in reported cases are either unreported or largely unrelated to their vascular anomaly.

With the increased availability of computed tomography (CT) and endoscopy, the diagnosis of EV in dogs is likely to become more common. As such, clinicians will need to be able to assess their clinical importance. Accordingly, our primary aim for this study was to evaluate the clinical manifestations and associated etiologies of EV in dogs, with a secondary aim to determine if the presence of EV was associated with death or euthanasia. We hypothesized that dogs with EV do not commonly present with clinical signs reported for humans with EV, and that dogs are unlikely to die because of complications directly related to the presence of EV.

2 | MATERIALS AND METHODS

The low prevalence of EV in small animals has resulted in various classifications of associated acquired collaterals. As such, the term EVs will be utilized as an umbrella term inclusive of esophageal, paraesophageal, bronchoesophageal, and gastroesophageal varices henceforth.

Potential cases with EV were identified by search of the Radiology Information System (RIS) at the North Carolina State University Veterinary Hospital for the years of 2010-2020. This database includes endoscopic studies. Search terms used were "esophagus" or "esophageal" and "varices," "varix," or "collateral circulation." Radiology reports from these dogs were reviewed and cases were included in this study if EV were confirmed using CT, ultrasonography, fluoroscopy, or endoscopy. Medical records were reviewed for signalment, presenting clinical signs, and outcome. Clinical signs were identified through review of medical history from case summaries. Clinical signs/complications that were considered to be complications of EV were specifically noted and defined as hemoptysis, hematemesis, melena, or syncope and weakness related to anemia/blood loss. The presence of hypertension (caval, portal, pulmonary, systemic) was documented if explicitly stated in the medical record or imaging report(s). Caval hypertension was considered presumed in the presence of an obstructive caval lesion with concurrent caval distention, right ventricular stiffness and without overt pulmonary hypertension. Portal hypertension was considered confirmed if documented on ultrasound as reduced portal velocity (<10 cm/s) via pulsed-wave Doppler or hepatofugal flow with color Doppler interrogation, and considered presumed in dogs in which acquired portosystemic shunts were identified without confirmation of portal blood flow alterations on ultrasound, or in dogs with EV with the presence of an obstructive lesion in the portal venous system on imaging. Pulmonary hypertension was considered presumed using estimated systolic pulmonary arterial pressures (PAPs) measured via echocardiography. Systemic hypertension was considered confirmed using indirect methods of Doppler and/or oscillometric blood pressure measurements. Outcomes were assessed through review of documented communications between the owner or primary care veterinarian. If outcome was not available after discharge, primary veterinarians were contacted to obtain the most recent medical information and health status of the individual patient. Outcome was defined as alive or dead/euthanized related or unrelated to EV associated complications.

Computed tomography images were reviewed by 2 board-certified radiologists (Christine L. Gremillion, Eli B. Cohen). Varices were classified as uphill or downhill based upon the direction of blood flow, location/category of collateral vasculature (esophageal, paraesophageal, gastroesophageal, bronchoesophageal), and arterial or venous. Arterial varices were further subclassified based on connections to the aorta, pulmonary artery, or both. Varices were considered esophageal if vessels were located within the esophageal wall. For paraesophageal and gastroesophageal varices, the esophageal hiatus was used as a break point, with extramural varices located at the level of or caudal to the esophageal hiatus categorized as gastroesophageal, and extramural varices cranial to the esophageal hiatus categorized as paraesophageal. Varices were classified as bronchoesophageal if they communicated with the bronchoesophageal artery, and were further subclassified based on location, with bronchoesophageal varices considered "proximal" when located at the level of the esophagus and "distal" when located at the level of the lung extending along bronchi. When additional collateral vasculature or varices were identified in included portions of the imaged anatomy (eg, neck, abdomen), these were also included and described based on prior literature into the following categories: thyroidal, left gastric, left gastrophrenic, pancreaticoduodenal, omental/mesenteric, colic, cholecystic, choledochal, phrenic or subcutaneous/body wall varices.[1] Other abnormally large tortuous vessels were also included in this description (eg, splenogonadal acquired shunts). Dogs were then subcategorized into suspected etiology based upon anatomic location (thoracic,

abdominal) and presence or lack thereof (idiopathic) of an attributable underlying disease process.

3 | RESULTS

Twenty-five dogs were identified as having EV and included in the study. Twenty-five had outcome data available. EV were diagnosed via CT (n = 22), fluoroscopy (n = 1), endoscopy (Figure 1) and CT (n = 1), and ultrasound and CT (n = 1). The single dog for which fluoroscopy was utilized underwent said imaging to aid in the placement of an Amplatz Occluder for a suspected Patent Ductus Arteriosus; the EV was an unexpected finding thought to be responsible for the clinical and echocardiographic findings previously attributed to a PDA. Prevalence of EV diagnosed on CT throughout the study period was 0.012% (24 out of 1950 total studies).

The study population consisted of 9 spayed females, 10 castrated males, and 6 intact males. The median and mean age was 5 years (range, 0.16-11 years). Breeds represented included the Golden Retriever (n = 3), Labrador Retriever (n = 3), Old English Sheepdog (n = 2), Bassett Hound (n = 1), Cavalier King Charles Spaniel (n = 1), Coonhound (n = 1), English Bulldog (n = 1), French Bulldog (n = 1), German Shepherd Dog (n = 1), Greyhound (n = 1), Jack Russell Terrier (n = 1), Mixed Breed Dog (n = 1), Pekingese (n = 1), Pembroke Welsh Corgi (n = 1), Rottweiler (n = 1), Staffordshire Terrier (n = 1), Standard Poodle (n = 1), Standard Schnauzer (n = 1), and Yorkshire Terrier (n = 1).

Twenty-four of 25 (96%) dogs did not experience complications secondary to EV. One (4%) dog had melena, though this animal also exhibited vomiting without blood while hospitalized and is thought unlikely to have had variceal hemorrhage. The most common presenting clinical signs included vomiting (n = 8 [location of EV etiology: 2 = thoracic, 6 = abdominal), lethargy (n = 6 [location of EV etiology: 1 = thorax, 5 = abdominal]), abdominal distention (n = 5 [location of EV etiology: 5 = abdominal]), and cough (n = 3 [location: 3 = thoracic]). Additional clinical signs included altered mentation (n = 1), ataxia (n = 1), exercise intolerance (n = 1), head pressing (n = 1), and syncope (n = 2).

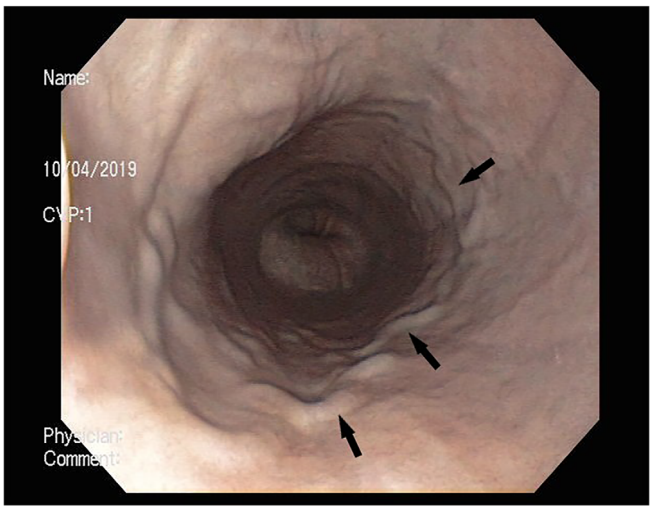

FIGURE 1 Video endoscopy of esophageal lumen, thoracic section in a dog with esophageal varices, suspected to be secondary to an anomalous arterial branch arising for the left pulmonary artery

Eleven dogs had EV with an attributable cause located in the thorax. Eight of these dogs had cardiovascular abnormalities, while the remaining 3 had neoplastic disease. Cardiovascular abnormalities included arteriopulmonary shunts (n = 2), cardiac AV fistula (n = 1), congenital anomalous pulmonary arterial branch (n = 1), major aortopulmonary collateral arteries (n = 1), pulmonary artery stenosis (n = 1), pulmonary arterial thrombus (n = 1), and pulmonary thromboemboli (n = 1). Neoplastic diseases included heart base masses (n = 2) and a mediastinal mass (n = 1). Pulmonary, caval, or systemic hypertension was present in all but 2 dogs within this category. Pulmonary hypertension was diagnosed using PAP estimation via echocardiography in all dogs (n = 6). Caval hypertension was presumed in all dogs (n = 3) with obstructive mass lesions without overt pulmonary hypertension. Systemic hypertension was diagnosed using Doppler in 1 dog with concurrent pulmonary hypertension. All dogs (11/11) had downhill EV (Figures 2 and 3). Two dogs had a single category of collaterals while the remainder had multiple (range, 2-4). Categories of varices associated with the esophagus included paraesophageal (n = 9), esophageal (n = 4), and bronchoesophageal (n = 6), with 3 subclassified as proximal bronchoesophageal and 3 subclassified as proximal and distal bronchoesophageal. Varices associated with the esophagus were classified as arterial with aortic connection (n = 4), arterial with aortic and pulmonic arterial connections (n = 3), and venous (n = 3). Additional varices or collateral circulation visible in the provided imaging included thyroidal (n = 1) and subcutaneous (n = 1) varices. One of these dogs only had imaging performed via fluoroscopy so the presence of multiple varices could not be ruled out.

Fourteen dogs with EV had an attributable cause located in the abdomen. Hepatic disease was the most common and accounted for 9 of these cases. Of the dogs with hepatic disease, 5 were diagnosed with congenital arterioportal malformations, 2 with portal vein hypoplasia (PVH) and 2 with hepatitis (lymphoplasmacytic). Shunting was diagnosed via imaging (ultrasound and CT for all). Hepatitis and hypoplasia were diagnosed via liver biopsy; minimal to moderate fibrosis was identified on histopathologic analysis of both cases with hepatitis. Three dogs had a neoplastic cause (mesenteric mass, intraluminal portal vein mass, and splenic mass) while the remaining 2 had vascular abnormalities (portal thrombi). All (14/14) of the dogs with attributable disease located in the abdomen had either confirmed or presumed portal hypertension, with 5 cases confirmed with ultrasound with and 9 cases presumed. All (14/14) dogs had uphill EV (Figure 4). All patients had at least 2 categories of collateral circulation. Categories of varices associated with the esophagus included gastroesophageal (n = 14), paraesophageal (n = 11), and esophageal (n = 6). All varices were classified as venous (n = 14). Additional varices or collateral circulation visible in the provided imaging included splenogonadal (n = 7), colic (n = 6), left gastric (n = 5), omental/mesenteric (n = 3), left gastrophrenic (n = 2), pancreaticoduodenal (n = 1), cholecystic

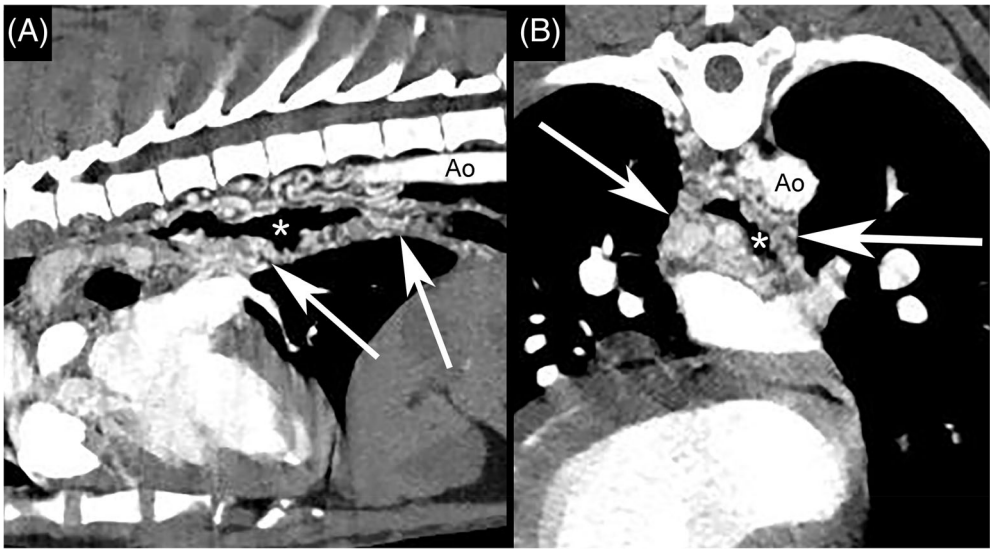

FIGURE 2 Sagittal (A) and transverse (B) computed tomographic images of a patient with downhill arterial esophageal varices (white arrows) secondary to bronchoesophageal artery hypertrophy with an anomalous connection to the left main pulmonary artery. The esophageal lumen is denoted by white asterisks (*). Ao denotes the descending aorta

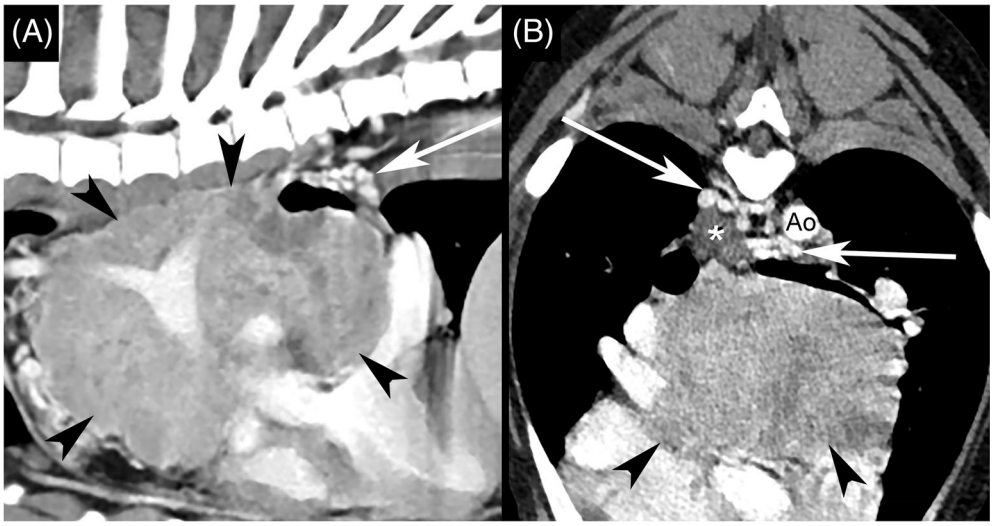

FIGURE 3 Sagittal (A) and transverse (B) computed tomographic images of a patient with downhill venous esophageal varices (white arrows) secondary to a heart-base tumor (black arrowheads) resulting in impaired venous return. The esophageal lumen is denoted by a white asterisk (*). Ao denotes the descending aorta

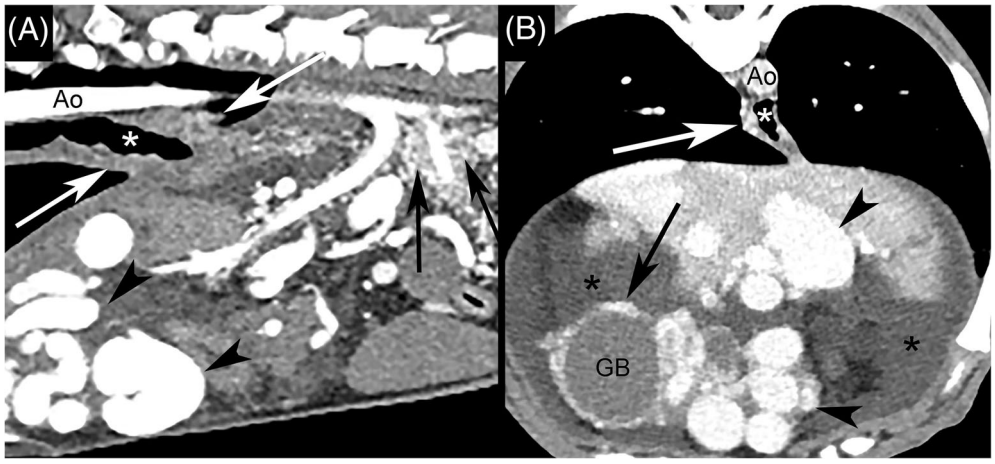

FIGURE 4 Sagittal (A) and transverse (B) computed tomographic images of a patient with uphill venous esophageal varices (white arrows) secondary to a congenital hepatic arterioportal malformation (black arrowheads). Note the presence of numerous concurrent intra-abdominal varices in this patient (black arrows) and moderate peritoneal fluid (black asterisks). The esophageal lumen is denoted by white asterisks (*). Ao denotes the descending aorta. GB denotes the gallbladder

(n = 1), choledochal (n = 1), phrenic (n = 1), and subcutaneous/body wall varices (n = 1).

Follow-up varied from 1 day to 9 years (median, 6.1 years) and was obtained by medical records review or through discussion with the owner or primary veterinarian. No dogs were documented to have been euthanized or died directly because of complications associated with their EV, and 13 out of 20 dogs with nonneoplastic disease were alive at the time of submission of this manuscript (range, 96-3089 days; median, 866 days). All dogs (n = 6) with neoplastic disease were euthanized in-hospital or shortly following diagnosis (range, 1-17 days; median, 7 days) because of financial constraints or poor prognosis associated with treatment/lack thereof for the associated cancer. Three of 7 dogs with cardiovascular disease were euthanized (range, 4-257 days; median, 90 days); 1 was euthanized following an episode of respiratory distress, suspected to be secondary to a pulmonary thromboembolism, while the remaining 2 were euthanized following diagnosis because of financial constraints and/or quality of life concerns. Similarly, 2 dogs with hepatic disease (arterioportal malformations) were euthanized because of financial constraints (1 and 39 days). One owner experienced financial constraints before performing surgical attenuation of the arterioportal malformation. The other patient underwent surgery for attenuation but was euthanized because of financial constraints associated with complications of postoperative sepsis.

4 | DISCUSSION

In the present study, we describe the clinical manifestations of EV in dogs. To our knowledge, there are no other reports in which the clinical implications of EV in a relatively large number of dogs, as outlined by presenting clinical signs, attributable disease processes, and outcomes, are described. Dogs in the present study did not exhibit complications secondary to the presence of EV despite approximately half having a suspected etiology of hepatic disease and portal hypertension; this is in contrast to humans in which variceal hemorrhage is a common (~50%) and often life-threatening sequelae of EV secondary to cirrhotic hepatic disease.[8] Similarly, dogs with EV did not appear to have a poor long-term prognosis, as 13 of the 20 dogs with nonneoplastic disease were alive at the time of submission of this manuscript. Those that were euthanized were done so because of reasons unrelated to complications secondary to EV.

Vomiting, lethargy, abdominal distention, and coughing were the most common clinical signs within the study dogs and could be explained by the underlying disease processes as opposed to direct sequelae of EV. Indeed, none of the dogs that were presented for vomiting exhibited hematemesis. The lack of overt hemorrhage could reflect the lack of intraluminal varices identified within the study population, though this characteristic is not reported as a positive prognostic indicator for bleeding in humans. The high incidence of vomiting could be the result of the small numbers of EV reported and the large number of disease processes for which this clinical sign might be associated. However, increasing intra-abdominal pressure has deleterious effects on variceal hemodynamics in people.[9] Therefore, vomiting might play a role in the development or exacerbation of EV in dogs. Further studies are indicated to determine if the associations between vomiting and EV are repeatable on prospective analysis.

The development of EV in humans with cirrhotic hepatic disease is attributed to increased sinusoidal resistance and subsequent collateral overcirculation from the portal to the esophageal venous systems.[7,10] This type of esophageal varix is classified as uphill, in which the direction of venous blood flow is toward the heart and central circulation; this classification is more common in humans.[10] In the present group of dogs, uphill varices were also more common and similarly associated with disease affecting portal circulation. Half of human patients with portal hypertension caused by hepatic cirrhosis are expected to develop uphill EV with subsequent variceal hemorrhage being a leading cause of death within this population.[11] It is unclear why a similar relationship between the presence of EV and likelihood of bleeding is not readily appreciable in dogs, though the underlying etiology of hepatic disease is potentially to blame. Alcoholic cirrhosis is the most common form of cirrhosis in humans and therefore minimally translatable to small animal medicine.[12] Meanwhile, chronic hepatitis (CH) has been implicated as a common cause of hepatic cirrhosis in dogs.[13,14] In the present study, only 2 dogs had primary inflammatory hepatic disease, while the remainder of dogs with hepatic disease had suspected hepatic arterioportal malformation or PVH. There is important overlap in the pathophysiology of these vascular abnormalities, hepatic cirrhosis, and the development of portal hypertension as a result of overlapping effects on presinusoidal, sinusoidal, and postsinusoidal pressures in each category. However, subcategories of noncirrhotic portal hypertension exist within these canine hepatic vascular classifications, as do cases of idiopathic portal hypertension.[15,16] As such, the similarities between this group of hepatic vascular abnormalities in dogs, alcohol-induced cirrhosis in humans, and the pathophysiology behind EV formation and associated complications remain uncertain.

In humans, downhill EV are less common and account for approximately 0.1% of all cases of variceal hemorrhage.[6] Over half of the reported causes of downhill EV are malignant mediastinal lesions; however, benign causes are more likely to result in bleeding.[6,17] In contrast, only 27% of the cases of downhill EV in the present study were because of neoplasia. The relatively high percentage of dogs with benign causes of downhill EV should be considered during the diagnostic evaluation of dogs with clinical signs such as hematemesis and melena. The most common etiology of bleeding downhill varices in humans is complication related to central venous catheterization.[6] This has yet to be reported in dogs, though prospective analysis is required to determine if the presence of downhill EV are an underreported sequela of dogs with thrombosis secondary to central venous catheters.

This case series included a wide variety of varices of differing etiologies, including both venous and arterial varices. Arterial varices might result from aberrant arteries or hypertrophied arteries that anastomose with pulmonary arteries, such as bronchoesophageal

artery hypertrophy which was observed in a subset of our patients. Of the arterial varices in this case series, some communicated with the pulmonary arterial vasculature while others communicated with the systemic arterial vasculature (aorta), and some communicated with both. The varied pattern of communication with the pulmonary and systemic arterial systems indicates that these varying patterns of varices are likely being subjected to different pressures, which likely also has an impact on the potential for development of variceal hemorrhage. Venous varices occur as a result of increased blood flow resistance in the portal or systemic venous systems, or increased blood drainage from pulmonary hypertension, with each of these varying etiologies likely having differing risks of hemorrhage.

A previous study detailing portal collateral circulation in dogs and cats proposed a classification system for small and large collaterals as seen on CT angiography based upon the site of increased resistance or occlusion.[1] These sites, as pertaining to causation of esophageal collaterals, include the portal vein, cranial vena cava, azygous vein, and pulmonary veins.[1] Twenty-four of 25 dogs in the present study had identifiable disease affecting these sites. Unsurprisingly, all dogs with disease affecting the portal circulation (ie, arterioportal malformation, primary liver disease, abdominal neoplasia, abdominal vessel thrombus formation) had confirmed or presumed portal hypertension. Moreover, all but 1 dog with intrathoracic disease had disease affecting the pulmonary veins or cranial vena cava. Interestingly, all but 2 of these dogs also had pulmonary or suspected caval hypertension. While this finding aligns with the suspected pathophysiology for variceal development and has been described in human medicine, pulmonary hypertension has only been reported in association with the development of EV in 3 dogs.[1,3,6] It is unknown if the lack of previously reported cases of EV with pulmonary hypertension is because of a perceived lack of clinical importance and therefore not presented in text or if it was truly not detected. Prospective analysis is required to determine a true association between pulmonary hypertension and the presence of EV. However, given the prevalence of pulmonary hypertension in the present study, we recommend prioritizing echocardiography for estimation of PAP for the diagnostic workup of EV of unknown cause.

The mortality rate associated with esophageal variceal bleeding in humans is the primary driving force for early identification. Risk factors for spontaneous rupture in humans are identified via video endoscopy and include a larger size (>0.5 mm in diameter) and longitudinal red streaks or red spots overlaying the protruding vessels.[18] Only 1 dog in the present study had video endoscopy performed and neither of these risk factors were seen (Figure 1). Similarly, in 2 previous studies in which endoscopic EV images were provided, these risk factors were not observed. However, in the single reported case of EV-induced hemorrhage in the veterinary literature, red spots were identified; this dog lived 4 more years and ultimately succumbed to disease unrelated to its EV. Therefore, while identification of endoscopic risk factors is similarly as uncommon as variceal hemorrhage itself, the translatability of endoscopically identified risk factors is questionable in the dog.

Based on our findings, we believe that early identification of EV in dogs is not necessary to mitigate the risk of spontaneous variceal rupture and death. However, recognition of EV is important given the commonality of interventional procedures in veterinary medicine such as endoscopic biopsy acquisition and the placement of feeding tubes. Therefore, the presence of pulmonary or portal hypertension or known disease that might affect caval or portal blood flow should prompt further evaluation for EV before performing placement of feeding tubes or biopsy of the esophagus.

Limitations of this study include its retrospective nature and relatively small sample size. While the study population represents the largest group of dogs with EV to date, it was conducted at a single center. Inclusion of cases using records from multiple institutions and thus allowing inclusion of a larger number of cases is needed to verify the aforementioned findings. Diagnostic imaging findings were reviewed for standardization; however, the remainder of the diagnostic workup was not. Because of this, there was no standardized approach to EV diagnosis or medical record keeping. Additionally, the described clinical features (eg, hypertension) were documented from coded case summaries that contained variable explanation of the techniques utilized to suspect or confirm the diagnosis.

Diagnosis of EV requires CT in a majority of cases. Given the relatively low prevalence of EV in dogs, failure to utilize techniques that may aid in the diagnosis of EV, such as contrast or inclusion of additional body cavities in the scan, might have resulted in some dogs going undiagnosed with EV. Moreover, given the known association between portal hypertension and gastrointestinal (GI) ulceration in dogs suffering from severe hepatic disease, evidence of GI hemorrhage could be wrongly and presumptively diagnosed as ulcerative blood loss, rather than variceal hemorrhage.[19] These animals are also frequently deemed poor anesthetic candidates for imaging (CT) or endoscopy. Thus, it remains possible that EV and variceal hemorrhage is misdiagnosed as GI hemorrhage in a subset of severely affected dogs with chronic hepatitis or cirrhosis, and therefore underrepresented in the present study.

In conclusion, EV are uncommon in dogs and likely of minimal clinical importance when compared to humans. However, many parallels exist between humans and dogs in regard to underlying etiology and the presence of portal hypertension with uphill varices. These parallels should prompt evaluation for EV when there is known disease affecting portal or caval blood flow. Alternatively, incidental identification of an EV should prompt evaluation for portal or caval hypertension.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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