Case Report


Esophageal Varices due to a Probable Arteriovenous Communication in a Dog

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A 6-year-old 40-kg castrated male Samoyed dog was presented for evaluation of chronic remittent lameness of the right forelimb. The dog had a history of polyuria-polydipsia (PU/PD) and lethargy over the previous year. Physical examination was unremarkable except for a grade II systolic murmur at the apex of the heart on the left side. A 6-lead ECG was within normal limits. Arterial blood gas analysis indicated mild respiratory alkalosis (pH, 7.457, reference range, 7.370–7.450; pCO₂, 30.6–39.1 mm Hg; pO₂, 90 mm Hg; HCO₃, 20.9 mmol/L; reference range, 19.1–25 mmol/L). No clinically relevant abnormalities were found on CBC, serum biochemistry, serum protein electrophoresis, or hemostasis profile. Urinalysis revealed markedly reduced urine osmolality (142 mOsm/kg; reference range, 600–2400 mOsm/kg), consistent with the PU/PD, reported by the owner. Both the urinary cortisol/creatinine ratio and urinary bile acid concentration were within normal limits, excluding hyperadrenocorticism and hepatic failure as underlying causes of the PU/PD.

Thoracic radiographs disclosed a generalized increase in the size of the cardiac silhouette and pulmonary vascular enlargement, suggesting pulmonary overcirculation. An ELISA for Dirofilaria immitis antigen was negative. Two-dimensional, M-mode echocardiography (transducer frequency, 2.0–3.0 MHz) revealed left atrial enlargement, left ventricular eccentric hypertrophy, and impaired systolic function (end-diastolic dimension, 65.7 mm; end-systolic dimension, 44.2 mm; shortening fraction, 32.7%) with normal valves. Spectral and color-flow Doppler examination disclosed mild mitral, aortic, and pulmonic valve insufficiency. Both the tricuspid and the telediastolic pulmonic valvular peak regurgitant jet velocities were increased as follows: 3.26 m/s (normal, ≤2.5 m/s) and 2.44 m/s (normal, ≤2.0 m/s), respectively. According to the Bernoulli’s equation modification, the systolic pulmonary artery pressure was estimated to be 42.5 mm Hg and the diastolic pulmonary artery pressure was estimated to be 23.9 mm Hg, values consistent with mild pulmonary hypertension.

The dog was anesthetized and subjected to radiography of the right forelimb and total body multidetector computed tomography (MDCT). The radiographs were negative for abnormalities, and synovial fluid examination of the shoulder and stifle joints did not indicate evidence of any inflammatory pathology. MDCT scans of the brain, thorax, and abdomen were obtained. For the thoracic and abdominal scans, the dog was positioned in dorsal recumbency, and we employed the following parameters: helical modality, 120 kV, 200 mA, 0.7-second rotation tube, 0.526 pitch, and 1.2-mm slice thickness. For the brain scan, the dog was positioned in sternal recumbency and the scanner parameters were as follows: axial modality, 120 kV, 310 mA, 2-second rotation tube, 0.625 slice thickness, and 10-mm intervals. For an enhanced series, 2-mL/kg ioxidixanod 320 mg I/mL was injected via a 22-gauge catheter into the right cephalic vein at a 3 mL/second infusion rate, through a computed tomography injector system.

The brain and abdominal MDCT scans were normal. However, MDCT of the neck and chest revealed 12 pairs of ribs, an enlarged heart, and enlarged pulmonary vessels. Both of the bronchoesophageal arteries were enlarged and connected with an enormous network of homogeneously enhancing serpentine structures involving the thoracic esophagus (esophageal and paraesophageal varices). The bronchoesophageal vein was extremely dilated (Figs 1, 2). The right aygous and hemizygous veins were normal. The cranial vena cava was dilated as was the cervical vertebral venous system, which protruded into the vertebral canal. These findings were consistent with an arteriovenous communication (single or multiple fistulas, possibly between the thoracic aorta and the aygous system) with resultant venous distension and esophageal varicosity formation.

To further examine for suspected esophageal varices, we subjected the dog to an immediate videoendoscopic examination while still anesthetized. The proximal esophagus was of normal diameter and had normal mucosal features. However, numerous tortuous submucosal structures protruding into the esophageal lumen consistent with esophageal varices were encountered in the distal 3rd of the esophagus. Varix diameter ranged from 1 to 4 mm, and varices are submucosal esophageal vessels. Endoscopic criteria predictive factor for variceal bleeding include the size of esophageal varices as well as the “red signs” on the mucosa overlying esophageal varices. In our case, the characteristics of the mucosa overlying the varices were normal,
and no red signs were observed in the mucosal aspect on varices (Fig 3A,B). Endoscopic examination of the stomach and duodenum revealed no abnormalities.

On the basis of clinical findings and imaging and endoscopic examination, the diagnosis of “downhill” esophageal varices due to a probable anomalous arteriovenous communication was made. The owner elected medical management and did not consent to selective angiography and possible surgical intervention. Two months after diagnosis, the owner reported that the dog was in good condition, with no apparent pain in the forelimb and decreased PU/PD. The dog is being treated for the varices with an angiotensin converting enzyme (ACE)-inhibitor (enalapril, 10 mg PO q12h).

In humans, esophageal varices are described as normal veins (submucosal and subepithelial) of the esophagus that are abnormally dilated because of increased blood flow resistance in the portal venous system or superior vena cava (SVC). Esophageal varices, which project directly into the esophageal lumen, are of clinical importance in humans because they are prone to life-threatening bleeding. Varices may be called “uphill” or “downhill” depending on the direction of venous flow to the central circulation. In human patients, uphill varices are more common and usually result from portal hypertension. Downhill esophageal varices (DEVs) serve as collateral branches directing blood flow “downward,” either to bypass SVC obstruction via the azygous vein or to drain the superior systemic system to the portal vein when both the SVC and azygous vein are obstructed. In the human medical literature, DEVs have been often described in cases of SVC obstruction or compression due to lung tumors or mediastinal masses. Alternatively, this type of esophageal varices may develop as a result of excessive blood drainage into the esophageal veins caused due to systemic venulitis, thyroid disease or intervention, Bechet’s disease, Castleman’s disease, or pulmonary hypertension.

Predominant factors influencing downward extension of esophageal varices are obstruction level and duration. The more distal the increase in blood-flow resistance, the greater the possibility of DEVs developing. When the obstruction or flow resistance is proximal to the azygous veins, blood flows to the heart through mediastinal collaterals, and DEVs are confined to the upper esophagus. When the obstruction is below or involving the azygous vein, blood returns via the hemiazygous vein, and esophageal and portal veins and may be associated with varices extending throughout the entire esophagus.

DEVs have not been reported previously in the veterinary literature. We recently documented, by MDCT angiography, the gastroesophageal and mesenteric varices in a small case series of dogs affected by portal hypertension due to a variety of causes (eg, portal thromboembolism, primary portal vein hypoplasia, intrahepatic portal-venous fistula, and hepatic cirrhosis). Neither clinical nor imaging features of portal hypertension were detected in the present dog. Increased blood flow in the azygous system due to an arteriovenous communication appeared to underlie the clinical presentation.
Arteriovenous communications generally have been reported as congenital lesions that arise as a result of disordered mesodermal differentiation during gestation, with an associated lack of development of the local capillary bed. Congenital arteriovenous fistula (AVF) results from persistence of the embryonic communicating branches between arteries and veins. Acquired AVFs have been reported both in humans and dogs after trauma (eg, penetrating wound or surgery). Congenital AVFs have been described previously in the liver of dogs, but rarely in other sites. An AVF can cause an imbalance between vasoconstrictor and vasodilatory systems, which leads in decompensated patients to a reduction in sodium and water excretion. Accordingly, ACE inhibitors have been used in the medical management of AVF in human patients, and the effects of ACE inhibitors on fluid balance may have been responsible for the good response to therapy in the dog of this report.

The clinical and imaging findings in the dog of the present report were consistent with single or multiple arteriovenous communications, but the AVF route could not be precisely determined. The lack of 1 pair of ribs in this dog is suggestive of a more complex congenital malformation. The dilated cervical vein system described may be the result of retrograde filling of the paravertebral and epidural veins via an arteriovenous communication and could be responsible for the chronic lameness and pain, due to spinal cord compression. As previously reported in a cat with a aortocaval communication, AVF-provoked increases in blood volume, resistance, and systemic blood pressure may have caused blood flow to be diverted to the azygous system, basivertebral veins, and internal venous plexus. Dilatation of the latter in the epidural space can result in compression of the spinal cord and clinical signs of myelopathy. Moreover, a pathway between the vertebral and esophageal veins has been reported to play an important role in the genesis of DEVs in humans, making the varices plausible consequences of excessive blood flow. In humans, selective angiography remains the gold standard for analysis of the anatomic and morphologic features of anomalous vascular connections. Conventional radiology, ecodoppler ultrasound examination, MDCT nonselective angiography, and flexible videendoscopy were used in the dog described here. MDCT angiography played a primary diagnostic role, providing information about the arteriovenous connection, demonstrating dilatation of the vertebral venous system with spinal cord compression, and identifying the esophageal varices. In human medicine, videendoscopy is considered to be the reference standard in the detection and bleeding risk evaluation of esophageal and gastric varices. The high mortality rate associated with esophageal variceal bleeding in humans has made early identification of potentially dangerous varices a clinical priority. The main endoscopic parameters by which dangerous varices are identified are variceal diameter and appearance. Large, tortuous varices measuring >5 mm in diameter have a greater predisposition to spontaneous rupture than smaller varices, and the presence of longitudinal red streaks and red, flat, or raised spots on varices (so-called “red signs”) are considered a sign of high bleeding risk. To the best of the authors’ knowledge, the endoscopic features of esophageal varices have not been described in dogs previously, and the predictive value of variceal features is unknown. Nevertheless, on the basis of the parameters described above, the esophageal varices in the dog of this report are considered to be of low bleeding risk.

Footnotes

a Logiq 5 Expert, GE Healthcare, Milwaukee, WI
b LightSpeed 16, GE Healthcare, Milwaukee, WI
Visipaque 320, Amersham Health, Princeton, NJ
Envision CT Injector System, Medrad, Indianola, PA
Small Animal Videendoscope 60814 PKS, KARL STORZ, Goleta, CA
Bertolini G, Zotti A, Caldin M. Multidetector CT angiography in acquired anomalies of the canine portal venous system due to portal hypertension: three-cases. 2006 Joint International Scientific Conference of the International Veterinary Radiology Association (IVRA)-American College of Veterinary Radiology (ACVR)-European College of Veterinary Diagnostic Imaging (ECVDI) and European Association of Veterinary Diagnostic Imaging (EAVDI), Vancouver, British Columbia, Canada, August 7–11 2006 (abstract)

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References