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Use of percutaneous transvenous coil embolization in the treatment of intrahepatic portosystemic shunts in four cats

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CASE DESCRIPTION

4 cats (6 to 9 months old) were evaluated because of clinical signs consistent with a portosystemic shunt (PSS).

CLINICAL FINDINGS

Among the 4 cats, 3 had neurologic abnormalities including ataxia, head pressing, disorientation, and obtundation. One cat was evaluated because of urethral obstruction; a retrieved urethral stone was determined to have urate composition. Clinicopathologic findings (hypoproteinemia, low BUN concentration, and high serum bile acids concentration) were consistent with a PSS in all cats. A diagnosis of intrahepatic PSS (IHPSS) was made for all cats on the basis of ultrasonographic and CT findings.

TREATMENT AND OUTCOME

All cats underwent percutaneous transvenous coil embolization (PTCE). No major intraprocedural complications were encountered, and all cats were discharged from the hospital. For the 3 cats that were presented with neurologic signs, an evaluation performed at 12, 14, or 48 months after the procedure revealed resolution of the neurologic signs, and owners reported that the behavior of each cat appeared normal. One cat that initially had neurologic and gastrointestinal signs had lower urinary tract signs after PTCE and developed an acquired extrahepatic PSS.

CLINICAL RELEVANCE

Although IHPSSs in cats are uncommon, the outcomes of PTCE for the 4 cats of the present report suggested that this treatment may benefit cats with an IHPSS. No short-term complications were encountered, and all cats had improvement in clinical signs following PTCE, although an acquired extrahepatic PSS was later identified in 1 cat. Further investigation of the use of endovascular techniques for the treatment of IHPSSs in cats and other species is warranted. (*J Am Vet Med Assoc* 2020;257:70–79)

A 7-month-old spayed female domestic shorthair cat (cat 1) was evaluated at the UCD-VMTH for treatment of an IHPSS. Neurologic signs of ataxia, head pressing, and disorientation were first noted by the owner when the cat was approximately 2 months old (1 month after its adoption). Initial laboratory diagnostic testing (a CBC, serum biochemical panel, and assessment of plasma ammonia concentration) performed by the primary veterinarian at that time revealed hyperglycemia (168 mg/dL; reference interval, 79 to 126 mg/dL), low-normal BUN concentration (11 mg/dL; reference interval, 10 to 30 mg/dL), low creatinine concentration (0.4 mg/dL; reference interval, 0.6 to 1.4 mg/dL), high alkaline phosphatase activity (145 U/L; reference interval, 10 to 90 U/L), and high plasma ammonia concentration (499 µmol/L; reference interval, 0 to 95 µmol/L). Abdominal ultrasonography had been performed and revealed an IHPSS, renomegaly, cystoliths, and ureteroliths. Shortly prior to the cat's referral evaluation, a CBC revealed thrombocytopenia (119,000 platelets/µL; reference interval, 200,000 to 500,000 platelets/µL), and a serum biochemical panel revealed high alanine transaminase activity (404 U/L; reference interval, 20 to 100 U/L), low-normal BUN concentration (10 mg/dL), and mild hyponatremia (134 mmol/L; reference interval, 142 to 164 mmol/L). The cat was started on a protein-restricted renal diet[a] in addition to a protein-restricted hepatic diet.[b] Medical management was initiated with lactulose (1.7 mg/kg [0.8 mg/lb], PO, q 8 h), amoxicillin–clavulanic acid (11.6 mg/kg [5.3 mg/lb], PO, q 12 h), levetiracetam (13.0 mg/kg [5.9 mg/lb], PO, q 6 to 8 h), and metronidazole (9.3 mg/kg [4.2 mb/lb], PO, q 12 h).

At the referral evaluation, the owner reported that the cat had possible mild polydipsia (without poly-

ABBREVIATIONS

CTA	CT angiography
EHPSS	Extrahepatic portosystemic shunt
IHPSS	Intrahepatic portosystemic shunt
LRS	Lactated Ringer solution
PSS	Portosystemic shunt
PTCE	Percutaneous transvenous coil embolization
PTFE	Polytetrafluoroethylene
UCD-VMTH	University of California-Davis Veterinary Medical Teaching Hospital

uria), liquid stools (with lactulose administration), and occasional coughing. No hematuria, pollakiuria, stranguria, or dysuria was reported. On physical examination, the cat was quiet but alert and responsive. It had a rectal temperature of 38.3°C (100.9°F), pulse rate of 200 beats/min, and respiratory rate of 44 breaths/min. The cat weighed 2.7 kg (5.9 lb) and had a body condition score of 5/9. The remainder of the physical examination findings were unremarkable.

The cat underwent general anesthesia for abdominal dual-phase CTA. The cat was premedicated with butorphanol tartrate (0.3 mg/kg [0.1 mg/lb], SC) and atropine sulfate (0.02 mg/kg [0.009 mg/lb], SC), and induction of anesthesia was achieved with IV administration of propofol (3 mg/kg [1.4 mg/lb]) following preoxygenation. Levetiracetam (20 mg/kg [9.1 mg/lb]) was administered IV at the time of induction of anesthesia. Anesthesia was maintained via inhalation of isoflurane with oxygen, and reported anesthetic complications included hypothermia (36.7°C [98.1°F]) and hypotension (systolic blood pressure, 85 mm Hg). Dopamine hydrochloride (5 µg/kg/min [2.3 µg/lb/min], IV) was administered to correct hypotension. Helical CT[c] images were obtained in the arterial, portal, and venous phases of abdominal CTA after the cat was administered 2.38 mL of iopamidol[d]/kg (1.08 mL/lb) IV at an injection rate of 5 mL/s; the settings used were 120 kVp, 200 mA, and 0.625-mm collimation. Computed tomographic angiography revealed a large, anomalous blood vessel that arose from the portal vein and coursed ventrally and to the left within the liver and subsequently dorsally and to the right, where it joined the left hepatic vein 2 cm distal to its confluence with the caudal vena cava. These features were consistent with a left divisional IHPSS. The cat also had an anomalous tortuous vessel that arose from the splenic vein, coursed cranially and dorsally along the lesser curvature of the stomach through the left gastric vein, and joined the phrenic vein at its insertion on the caudal vena cava near the diaphragm. This vessel was consistent with a congenital EHPSS with phrenic vein termination. Branches from the portal vein were visualized extending into the right side of the liver. The liver was diffusely hypoattenuating with the exception of a portion of the left lateral lobe that was soft tissue attenuating; this finding was consistent with hepatic lipidosis. The cat had multiple wedge-shaped contrast-enhancing regions in the renal cortices bilaterally, consistent with renal infarcts. Multiple mesenteric lymph nodes were mildly enlarged (maximum dimension, approx 5 mm), consistent with reactive lymph nodes or possibly associated with the juvenile status of the cat. No uroliths were detected, and the remainder of the CTA findings were unremarkable.

The cat was hospitalized following CTA and administered lactulose (0.5 mg/kg [0.23 mg/lb], PO, q 8 h), amoxicillin (18.5 mg/kg [8.4 mg/lb], PO, q 12 h), and levetiracetam (20 mg/kg, IV, q 8 h). The following day, the cat was anesthetized for PTCE of the IHPSS. The cat was premedicated with methadone (0.3 mg/kg, SC) and atropine (0.02 mg/kg, SC), and induction of anesthesia was achieved with IV administration of propofol (9.3 mg/kg) following preoxygenation. Levetiracetam (20 mg/kg) was administered IV at the time of induction of anesthesia, and cefazolin (22 mg/kg [10 mg/lb], IV) was administered prior to the procedure. Anesthesia was maintained via inhalation of isoflurane with oxygen, and hypothermia (33.8°C [92.8°F]) was a reported complication that occurred during the procedure. Mean arterial blood pressure was noted to decrease to 60 mm Hg, and dopamine (5 µg/kg/min, IV) was administered to correct hypotension. Dextrose (added to IV fluids in a quantity sufficient to create a 2.5% dextrose solution) was administered to correct hypoglycemia.

For the PTCE procedure, the cat was placed in dorsal recumbency **(Figure 1)**, and hair on the ven-

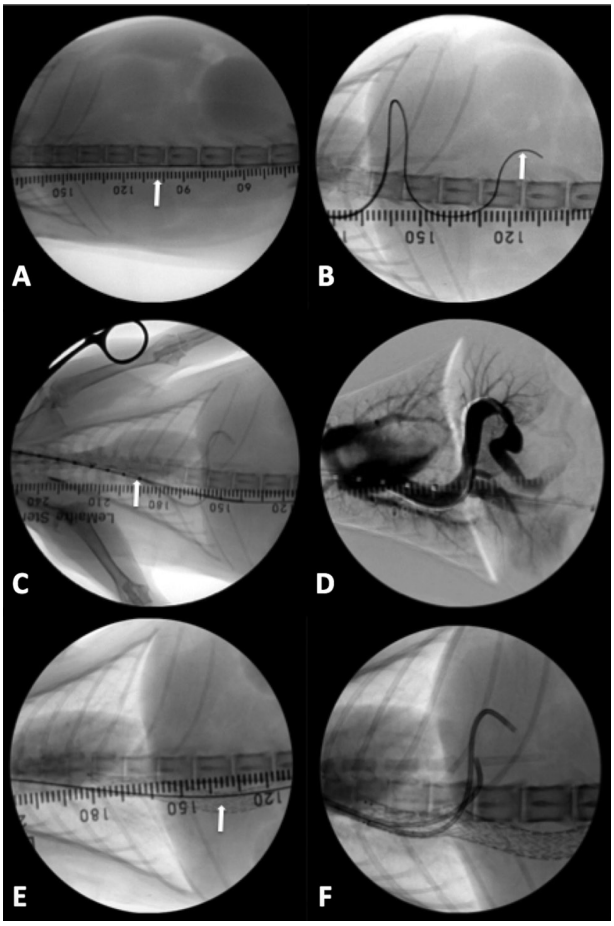

Figure 1—Fluoroscopic images of a 7-month-old spayed female domestic shorthair cat (cat 1) undergoing PTCE for treatment of a left divisional IHPSS. A—The cat is positioned in dorsal recumbency, and a stent guide (arrow) on the dorsum of the cat can be seen. B—A hydrophilic guidewire (arrow) has been advanced from the vena cava into the IHPSS and further into the portal vein. C—A marker catheter (arrow) is placed in the vena cava (next to the catheter that is present in the IHPSS) to allow for simultaneous cavography and portography. D—A simultaneous cavogram and portogram are obtained to identify the vena cava, portal vein, and IHPSS. E—The caval stent (arrow) has been deployed across the ostium of the IHPSS. F—Two catheters can be seen in the IHPSS, one for coil delivery and the other for portal blood pressure monitoring.

tral cervical region was clipped. The surgical site was prepared with sterile technique and draped. A stab incision was made into the skin overlying the right jugular vein. A 22-gauge over-the-needle catheter[e] was introduced into the jugular vein, and the needle was removed. A 0.018-inch hydrophilic guidewire[f] was introduced into the jugular vein, and a transition dilator[g] was placed to allow for introduction of a 0.035-inch hydrophilic guidewire after removal of the 0.018-inch guidewire. A 9F vascular access sheath with a dilator[h] was introduced into the jugular vein over the guidewire. The dilator was removed, and the sheath was sutured to the skin with 3-0 nylon suture material.[i] The guidewire was manipulated into the shunt and guided further into the portal vein. An angled catheter[j] was placed over the guidewire and into the shunt. The guidewire was removed, and a portal pressure of 5 mm Hg was obtained. A 5F marker catheter[k] was then introduced into the caudal vena cava over a 0.035-inch guidewire. A mixture of equal volumes of contrast agent and saline (0.9% NaCl) solution was injected into both the angled catheter and marker catheter simultaneously to generate a cavogram and a portogram. An image of the combined cavogram and portogram was saved, and shunt length and location were determined. A stent (10 X 60-mm stent[l]) was chosen on the basis of data obtained from the previously performed CTA scan. The angled catheter was removed, and a 0.035-inch PTFE wire[m] was placed into the caudal vena cava through the marker catheter. The marker catheter was removed. The stent was introduced into the caudal vena cava over the PTFE wire, and the stent was deployed. The stent delivery system was removed over the PTFE wire, and the angled catheter was reintroduced over the PTFE wire. The PTFE wire was removed, and the 0.035-inch hydrophilic guidewire was then reintroduced into the angled catheter. The guidewire was passed through the interstices of the stent into the shunt and further into the portal vein. A second catheter[j] was introduced over the guidewire, and portal pressure was assessed after the guidewire was removed. A PTFE wire was used to push a thrombogenic coil[n] (5 mm in diameter) through the second angled catheter; after delivery of the coil, the portal and caval pressures were measured. A guidewire was then introduced into the angled catheters to remove the catheters. The guidewire was left in the vena cava, and the vascular access sheath was removed over the guidewire. A 7F, 20-cm-long triple-lumen catheter[o] was introduced into the cranial vena cava over the guidewire, and the guidewire was removed. The triple-lumen catheter was sutured to the skin. Initial caval and portal pressures were 4 and 5 mm Hg, respectively; after coil placement, caval and portal pressures were 5 and 13 mm Hg, respectively. The cat recovered from anesthesia and the PTCE procedure without complication. Total procedure time (the interval from stab incision to placement of the triple-lumen catheter) was 120 minutes, and total anesthesia time (the interval from induction of anesthesia to extubation) was 170 minutes.

The cat was hospitalized for 3 days after the PTCE procedure for continued monitoring and supportive care. The lactulose, amoxicillin, and levetiracetam treatments were continued, and the cat also received LRS containing 20 mEq of KCl/L (delivered at a rate of 2.6 mL/kg/h [1.2 mL/lb/h], IV), famotidine (1.0 mg/kg [0.45 mg/lb], IV, q 12 h), and buprenorphine (0.01 mg/kg [0.005 mg/lb], IV, q 12 h). The cat developed no signs of portal hypertension and had no seizures or other neurologic signs. Two days after the PTCE procedure, the cat was quiet but alert, and it became mildly hyperthermic (39.4°C [103°F]). Serosanguineous discharge was noted from the region of the jugular catheter; the catheter was subsequently removed. On recheck evaluation approximately 6 hours later, the cat's rectal temperature was considered normal (38.6°F [101.4°F]). Focused abdominal ultrasonography revealed no peritoneal effusion. Throughout the postprocedural hospitalization period, the cat maintained a good appetite and urinated and defecated (soft to liquid feces) regularly. The cat was discharged from the hospital 3 days after the PTCE procedure, and the owners were instructed to monitor its condition and restrict its activity. Treatments with amoxicillin (18.5 mg/kg, PO, q 12 h for 4 weeks), lactulose (0.4 mg/kg [0.18 mg/lb], PO, q 8 h for 8 weeks), levetiracetam (13.0 mg/kg, PO, q 8 h for 2 days), and famotidine (0.9 mg/kg [0.4 mg/lb], PO, q 12 h for life) were to be administered to the cat. The owners were also instructed to provide the cat with the protein-restricted diets for 8 weeks with subsequent transition to a maintenance feline diet. At 48 months after the PTCE procedure, the cat was clinically normal.

A 2-month-old castrated male Siamese cross (cat 2) was evaluated at the UCD-VMTH because of a 2-week history of hyporexia, ptyalism, increased respiratory effort, ataxia, weakness, and inappropriate mentation. The cat was initially evaluated by the primary veterinarian and on physical examination was noted to be both intermittently obtunded and hypersensitive to stimuli, 5% dehydrated, and tachypneic (respiratory rate, 120 breaths/min) with harsh lung sounds bilaterally, and it had a distended abdomen. At that time, the cat was hospitalized for 1 day and administered IV fluids (isotonic crystalloids) with dextrose supplementation, ampicillin IV, lactulose PO, doxycycline IV, and a warm water and lubricant enema (all at unknown dosages). The cat's mentation improved and it was discharged from the hospital. Recommendations made to the owner were to administer lactulose (0.3 mg/kg, PO, q 8 h) and metronidazole (10.0 mg/kg [4.5 mg/lb], PO, q 12 h) to the cat and feed it a protein-restricted renal diet.[b]

When the cat was presented to the UCD-VMTH, it was bright, alert, and responsive. Clinicopathologic findings were consistent with a PSS, and abdominal ultrasonography revealed an IHPSS that was suspected to be central divisional in nature. The portal

vein was observed entering the liver at the hilus and then extending cranially into an enlarged shunt vessel that traversed dorsally and became more dilated. The portal blood flow was tortuous in the region of the ampulla. Portal blood flow was mildly pulsatile and had a velocity of ≤ 16.2 cm/s. The shunting vessel entered into the caudal vena cava just caudal to the diaphragm. Mild microhepatica was observed, and the kidneys were fused medially. The aorta coursed dorsally relative to the kidneys, and the caudal vena cava was displaced ventrally relative to the kidneys. Echogenic debris (suspected urate crystalluria) was evident in the urinary bladder. Continued medical management was recommended prior to possible PTCE to allow for growth of the cat. The cat was discharged from the hospital. The owner was to continue administration of lactulose (0.3 mg/kg, PO, q 8 h) to the cat, discontinue the metronidazole treatment, and start administration of amoxicillin (22.5 mg/kg [10.2 mg/lb], PO, q 12 h). The cat was to be provided with a protein-restricted hepatic diet formulated for cats during the growth period instead of the protein-restricted renal diet.

The cat was reevaluated at the UCD-VMTH at 9 months of age for PTCE of the IHPSS. No additional neurologic signs, polyuria, polydipsia, or periuria had been noted by the owner. Helical CT images were obtained as described for cat 1. Computed tomographic angiography revealed a large right divisional IHPSS. The anomalous vessel arose from the portal vein as it entered the right caudal aspect of the liver. The shunt vessel coursed laterally and cranially, where it reached a large, focal dilation in the caudate or right lateral lobe of the liver prior to draining into the right lateral hepatic vein and intrahepatic caudal vena cava. The connection between the dilated anomalous vessel and caudal vena cava measured approximately 1.7 cm in length, and the vena cava cranial and caudal to this region measured approximately 7 mm in diameter. The liver appeared small. The left and right kidneys were fused at their caudal poles, and the caudal vena cava was observed to bifurcate at the level of the renal veins with duplication of the caudal vena cava caudally **(Figure 2)**. Mineral debris without overt uroliths was present in the urinary bladder lumen. The remainder of the CTA findings were unremarkable.

The following day, the cat was anesthetized for PTCE of the IHPSS. Reported anesthetic complications included hypotension (mean arterial blood pressure, 55 mm Hg), oscillating hyperglycemia and hypoglycemia, anemia, and arrhythmias. Dextrose (added to IV fluids in a quantity sufficient to create a 2.5% dextrose solution) was administered to correct hypoglycemia. The PTCE procedure was performed as described for cat 1; a 10-mm-wide, 60-mm-long stent was used, and 9 coils (5 mm in diameter) were placed. Initial caval and portal pressures were 6 and 7 mm Hg, respectively; after coil placement, caval and portal pressures were 6 and 13 mm Hg, respectively. The cat recovered from anesthesia and the PTCE procedure without complication. Total procedure time was 165 minutes, and total anesthesia time was 255 minutes.

The cat was hospitalized for 3 days after the PTCE procedure for continued monitoring and supportive

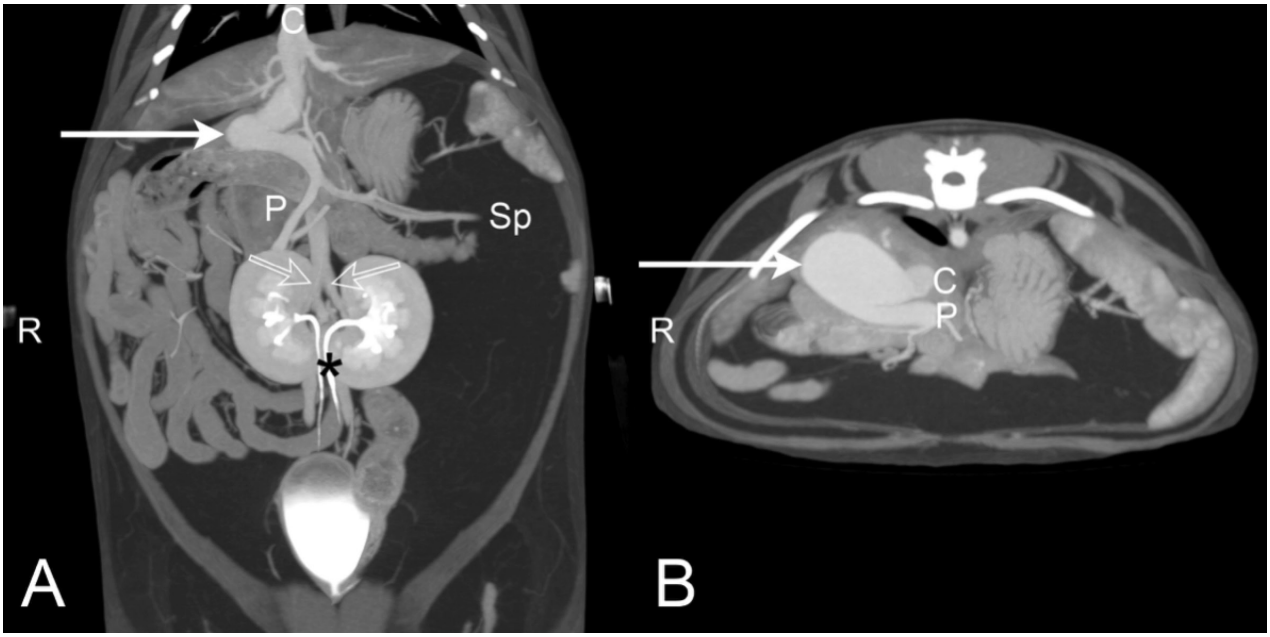

Figure 2—Dorsal plane-multiplanar reformatted (A) and transverse plane maximum intensity projection (B) CT images of a 9-month-old Siamese cross cat (cat 2) with a right divisional IHPSS. The shunt arises from the portal vein (P) at the porta hepatis cranial to the splenic vein (Sp) and courses through the right division as a large dilation (solid arrow) before joining the caudal vena cava (C). The caudal poles of the kidneys are fused medially (asterisk), and the caudal vena cava divides proximal to the renal pelves (open arrows).

care. The lactulose and amoxicillin treatments were continued, and the cat also received LRS (1.4 mL/kg/h [0.6 mg/lb/h], IV), famotidine (0.5 mg/kg, IV, q 12 h), phenobarbital (2.5 mg/kg [1.1 mg/lb], IV, q 12 h), and buprenorphine (0.01 mg/kg, IV, q 12 h as needed for pain). The cat developed no signs of portal hypertension and had no seizures or other neurologic signs. Throughout the postprocedural hospitalization period, the cat maintained a good appetite and urinated and defecated normally. The cat was discharged from the hospital 3 days after the PTCE procedure, and the owners were instructed to monitor its condition and restrict its activity. Ten days after the PTCE procedure, the cat was evaluated by the primary veterinarian following 1 episode of stranguria and hematuria. Treatment with prazosin (0.1 mg/kg [0.045 mg/lb], PO, q 12 h) and buprenorphine (0.01 mg/kg, PO, q 8 to 12 h) was prescribed. Two weeks later, the cat was presented to the primary veterinarian because of persistent stranguria and hematuria. Abdominal radiography revealed 2 calculi in the distal portion of the urethra. Voiding urethrohydropulsion was performed. However, the cat's clinical signs recurred 2 days later, and a cystotomy was performed. Analysis of the uroliths revealed a composition of 100% urate.

Approximately 14 months after the PTCE procedure, when the cat was approximately 2 years old, it was evaluated by the primary veterinarian because of hyporexia and ptyalism. Radiography revealed no evidence of upper airway obstruction or material lodged in the pharynx or upper portion of the gastrointestinal tract; however, cystoliths were detected. After the cat was sedated, an oral examination revealed an apparently inflamed soft palate, everted left tonsil, glossal lesions, and no laryngeal abnormalities. The only pertinent serum biochemical finding was high alanine aminotransferase activity (153 U/L; reference interval, 12 to 130 U/L). The cat was started on buprenorphine (0.02 mg/kg, PO, q 8 to 12 h), and cystotomy was recommended. Two weeks later, the cat was reevaluated by the primary veterinarian because of lesions associated with the tongue and pharynx. Preprandial (17.4 µmol/L; reference interval, < 13 µmol/L) and postprandial (82.1 µmol/L; reference interval, < 30 µmol/L) serum bile acids concentrations were high. Further assessment at the UCD-VMTH was recommended, which occurred approximately 2 weeks later. The owners reported signs of apparent abdominal pain and hematuria, pollakiuria, periuria, and stranguria of a few weeks' duration. The cat was not receiving any medications and was no longer being fed a protein-restricted diet. On physical examination, the cat was bright, alert, and responsive. It had a rectal temperature of 38.7°C [101.6°F], heart rate of 160 beats/min, and respiratory rate of 30 breaths/min. The cat weighed 4 kg (8.8 lb) and had a body condition score of 6/9. It had mild periodontal disease, but no oral ulcerations were detected. The cat had nonpositional, rotational, dysconjugate nystagmus and a grade 2/6 left parasternal systolic heart murmur. Abdominal palpation of the cat did not elicit signs of pain, and the remainder of the physical examination findings were unremarkable.

Abdominal ultrasonography was performed and revealed that the stent was appropriately positioned in the caudal vena cava. There was a heterogeneous soft tissue opacity in the right side of the liver in the region of the IHPSS (findings of Doppler ultrasonography in this region were inconclusive), normal portal venous flow (20 cm/s), 3 cystic calculi, and an EHPSS vessel arising from the terminal portion of the splenic vein and coursing caudally. The soft tissue opacity in the region of the IHPSS was consistent with thrombosis of the IHPSS vessel. The EHPSS was suspected to be associated with acquired shunting. A urine sample was collected via cystocentesis and submitted for aerobic microbial culture. The culture yielded 2 colony types of coagulase-negative *Staphylococcus* spp. Medical management for the EHPSS was recommended as well as cystotomy, liver biopsy, and dual-phase CTA to further assess the extent of portosystemic shunting. The owners were instructed to administer the cat lactulose (0.3 mg/kg, PO, q 8 h, titrated to effect) and amoxicillin, transition to feeding the cat a protein-restricted hepatic diet,[b] and attempt to increase the cat's water intake. In addition, the owners were instructed to monitor the cat's urination and neurologic signs. Antimicrobial treatment (unknown drug) for the cat's urinary tract infection was initiated by the primary veterinarian. A follow-up conversation with the referring veterinary office, approximately 29 months after the PTCE procedure, revealed that the cat had no abnormal clinical signs.

A 6-month-old castrated male Himalayan (cat 3) was evaluated at the UCD-VMTH for treatment of a PSS. Two months earlier, the cat had developed a urethral obstruction. Diagnostic testing performed by the cat's primary veterinarian revealed high serum liver enzyme activities and bile acids concentration. Cystotomy was performed for urolith removal. Shortly thereafter, the cat developed a recurrent urethral obstruction, and cystotomy and urethrostomy were performed. The cat was started on lactulose (dosage ranged from 0.1 to 0.2 mg/kg [0.045 to 0.09 mg/lb], PO, q 8 h), amoxicillin (unknown dosage), and a protein-restricted hepatic diet.[b] No neurologic signs were noted. At the time of the referral evaluation, the owner reported that the cat was urinating normally.

Clinicopathologic findings were consistent with a PSS. Abdominal ultrasonography revealed an IHPSS that coursed dorsally toward the caudal vena cava at the level of the diaphragm; however, the insertion of the shunt into the systemic circulation was not visualized. Ultrasonography also revealed mild biliary sludge, hyperechoic foci along the distal and ventral periphery of a liver lobe (suspected to represent a prior biopsy site), prominent bilateral renal medullary rim signs with renal mineralization, thickening of the urinary bladder wall with hyperechoic far-shadowing material along the ventral and cranial aspects of the bladder wall (suspected to represent suture material or cystotomy sites), and thickened small intestinal

submucosa (suspected to represent inflammatory or infiltrative gastrointestinal tract disease).

Helical CTA images were obtained as previously described for cats 1 and 2. A large, tortuous, anomalous vessel arose from the portal vein just cranial to the right portal vein branch and coursed through the left lobe of the liver and then dorsally and toward the left side, where it subsequently converged with the phrenic vein at the level of the diaphragm. This shunt was assessed as a left divisional IHPSS. The liver was of normal size and attenuation, and the kidneys and ureters appeared normal. The urinary bladder was subjectively thickened with regions of hyperattenuating suture material, but no cystoliths were identified. The remainder of the CTA findings were unremarkable.

A PTCE procedure was performed as described for cats 1 and 2. A 10-mm-wide, 60-mm-long stent was used, and 1 coil (4 mm in diameter) was placed. Initial caval and portal pressures were 0 and 4 mm Hg, respectively; after coil placement, caval and portal pressures were 1 and 8 mm Hg, respectively. There were no anesthetic complications; the cat recovered uneventfully from anesthesia and the PTCE procedure. Total procedure time was 95 minutes, and total anesthesia time was 220 minutes.

The cat was hospitalized for 3 days after the PTCE procedure for continued monitoring and supportive care. The cat developed no signs of portal hypertension and had no seizures or other neurologic signs. Throughout the postprocedural hospitalization period, the cat maintained a good appetite and urinated and defecated normally. The cat was discharged from the hospital 3 days after the PTCE procedure, and the owners were instructed to monitor its condition and restrict its activity. The owners were instructed to administer amoxicillin (16 mg/kg [7.3 mg/lb], PO, q 12 h for 8 weeks), lactulose (0.1 mg/kg, PO, q 12 h for 10 weeks), and famotidine (1.0 mg/kg, PO, q 12 h for life). They were also instructed to continue to feed the protein-restricted hepatic diet[b] for 12 weeks. A follow-up conversation with the owners at approximately 30 months after the PTCE procedure revealed that the cat had no abnormal clinical signs and was urinating normally.

A 9-month-old spayed female domestic shorthair cat (cat 4) was presented to the Veterinary Medical Center at the University of Florida for evaluation of an IHPSS. The cat was initially adopted from a rescue shelter at 8 weeks of age and was reported to have intermittent episodes of trembling, hypersalivation, lethargy, and so-called stargazing since that time. Abnormal clinicopathologic findings noted at that time included high circulating concentrations of preprandial bile acids (97.9 µmol/L; reference interval, 0 to 5 µmol/L) and ammonia (718 µg/dL; no reference interval available). The cat underwent dual-phase CTA[p] performed with settings of 120 kVp, 400 mA, and 2-mm slice collimation. A short anomalous vessel with terminal mild tortuosity connecting the caudal vena cava to the central hepatic vein, consistent with a central divisional IHPSS, was identified. Bilaterally, the kidneys were mildly enlarged, and the urinary bladder contained small mineral-attenuating calculi. The cat was started on a commercial renal support diet,[q] lactulose (1 mL, PO, q 8 h), metronidazole (10 mg/kg, PO, q 12 h), and levetiracetam (20 mg/kg, PO, q 8 h) at the time of diagnosis. Clinical signs resolved with medical management, and the cat was clinically normal at the time of presentation at the veterinary medical center.

On physical examination, the cat was bright, alert, and adequately hydrated; it had a body weight of 3.6 kg (7.9 lb) with a body condition score of 5/9. The cat's irises were copper colored, but the remainder of the physical and neurologic examination findings were unremarkable. Clinicopathologic findings were consistent with the presence of a PSS.

The following day, the cat was anesthetized for PTCE of the IHPSS. The cat was placed in dorsal recumbency, and hair on the ventral cervical region was clipped. The surgical site was prepared with sterile technique and draped. A stab incision was made into the skin overlying the right jugular vein. A 22-gauge over-the-needle catheter was introduced into the jugular vein, and the needle was removed. A 0.018-inch guidewire was introduced into the jugular vein, and a transition dilator was placed to allow for introduction of a 0.035-inch hydrophilic guidewire after removal of the 0.018-inch guidewire. A 9F vascular access sheath with dilator was introduced into the jugular vein over the guidewire. The dilator was removed, and the sheath was sutured to the skin with 3-0 nylon suture material. A 0.035-inch J-tip guidewire[r] and 4F angled catheter were used to select the shunt ostium and ultimately the portal vein. The guidewire was removed, and a portal pressure of 4 mm Hg was measured. A 5F marker catheter was then introduced into the caudal vena cava over a 0.035-inch guidewire. The caval pressure was 3 mm Hg. A mixture of equal volumes of contrast agent and saline solution was injected into both the angled catheter and marker catheter to generate a cavogram and a portogram. Caval diameter and shunt morphology and location were documented. A 10-mm-wide, 40-mm-long open-cell vascular stent[s] was placed in the caudal vena cava across the shunt ostium. The guidewire and angled catheter were introduced into the shunt ostium and portal vein via the stent interstices. An angled 0.027-inch microcatheter[t] over a 0.021-inch microwire[u] was guided through the angled support catheter into the portal vein. An angiogram was obtained and a portal pressure measurement was performed. A 0.021-inch guidewire was used to push a 4-mm-diameter tornado-configured microcoil[v] through the second angled catheter; after delivery of the coil, the portal pressure was 6 mm Hg. A second microcoil was deployed, and portal pressure was 8 mm Hg following deployment. After the delivery of the second and final coil, all catheters and wires were removed. Total procedure time was 141 minutes, and total anesthesia time was 220 minutes. The cat recovered from anesthesia and the PTCE procedure without complica-

tions apart from a few intermittent premature ventricular contractions.

The cat was hospitalized for 2 days after the PTCE procedure for continued monitoring and supportive care. At 2 days after the PTCE procedure, the cat was discharged from the hospital; initial follow-up evaluations at 1 week, 4 weeks, and 3 months after the procedure were recommended to the owners. The owners were instructed to continue feeding the cat the commercial renal support diet and administering lactulose (1 mL, PO, q 8 h), metronidazole (10 mg/kg, PO, q 12 h), and levetiracetam (20 mg/kg, PO, q 8 h) until the 3-month follow-up evaluation. The cat was to receive lifelong treatment with famotidine (0.75 mg/kg [0.34 mg/lb], PO, q 12 h).

At the first follow-up evaluation 7 days after the PTCE procedure, the cat was clinically normal with no complications reported by the owners. At 3 months after the PTCE procedure, the cat had reportedly developed sudden-onset hyporexia and had a high rectal temperature (40.0°C [104.1°F]). A serum biochemical panel and CBC were performed, and the results were unremarkable apart from mild hyperglycemia (216 mg/dL; reference interval, 74 to 159 mg/dL) and lymphocytosis (6,080 cells/µL; reference interval, 850 to 5,850 cells/µL). On cytologic examination of a blood smear, epierythrocytic organisms consistent with *Mycoplasma hemofelis* were detected. Results of urinalysis were unremarkable. The cat was treated with 100 mL of LRS (SC, once), amoxicillin–clavulanic acid (16 mg/kg, PO, q 12 h), enrofloxacin (6.2 mg/kg [2.8 mg/lb], PO, q 24 h), prednisone (1.5 mg/kg [0.68 mg/lb], PO, q 24 h), maropitant (1.0 mg/kg, SC, q 24 h), and vitamin B_{12} (500 µg, SC, once).

The cat was reexamined 4 weeks later (4 months after the PTCE procedure) and was doing well with no clinical signs of infection or complications associated with PTCE. Circulating protein C concentration was normal at 68% (reference interval, 65% to 120%). Repeated cytologic examination of a blood smear revealed a reduced number of organisms consistent with *M hemofelis*. The cat had completed the prednisone and enrofloxacin treatments 1 week prior to this reevaluation and was no longer receiving metronidazole, amoxicillin–clavulanic acid, or maropitant. Current medications included lactulose (1 mL, PO, q 8 h) and famotidine (0.75 mg/kg, PO, q 12 h). The cat had also been weaned onto a commercial maintenance feline diet. Owing to the continued presence of *M hemofelis*, treatment with enrofloxacin (6.2 mg/kg, PO, q 24 h) was reinstituted, and administration of prednisone (1.5 mg/kg, PO, q 48 h) was commenced.

At the last follow-up evaluation (5 months after the PTCE procedure), the cat was clinically normal. It had gained 0.45 kg (1 lb; body condition score, 6/9) since the PTCE procedure; the irises remained copper colored. The owners were directed to discontinue the enrofloxacin and prednisone treatments over the following week. Circulating protein C concentration was normal at 92% and increased from the value 1 month after the PTCE procedure. At 12 months following the procedure, the cat was no longer receiving any medications apart from famotidine and was being fed a commercial maintenance feline diet.

Discussion

Portosystemic shunts are congenital vascular anomalies that result in aberrant communication between the systemic and portal venous systems. Because this abnormal communication causes blood to be diverted away from the liver, hepatic growth is altered, and substances that are normally metabolized by the liver accumulate intravascularly. Although there are some publications[1–20] describing the diagnosis and treatment of PSSs in cats, the overall prevalence of PSSs appears to be lower in cats relative to their prevalence in dogs.

Portosystemic shunts are often characterized by the location and course of the abnormal vessel.[3] In both dogs and cats, EHPSSs are identified much more commonly than are IHPSSs.[5,7,21,22] Intrahepatic PSSs are often subcategorized into 3 forms, depending on the affected liver lobe, namely right divisional, central divisional, and left divisional. In the 4 cats of the present report, PTCE was successfully performed in patients with all IHPSS forms (2 central divisional IHPSSs and 1 each of left and right divisional IHPSSs). In dogs, it is common to alter either patient or fluoroscopic position for treatment of central divisional IHPSSs because these shunts are often very short and can be difficult to delineate from the portal vein and vena cava in the ventrodorsal plane. However, in the 2 cats with central divisional shunts in the present report, a typical ventrodorsal orientation was successfully used throughout the PTCE procedure.

In dogs and cats, surgical attenuation of an IHPSS is generally considered to be more challenging owing to shunt location relative to the hepatic parenchyma and is often associated with greater frequencies of intra- and postoperative treatment-related complications, compared with surgical attenuation of an EHPSS.[1,16] As a result, evaluation of alternative (nonceliotomy-based) treatment options has been performed in dogs with the goals of decreasing the occurrence rate of complications and improving outcomes associated with treatment.[23–25] Transvenous coil embolization was first described for small numbers of dogs[26–30]; more recently, 3 reports[23–25] of retrospective and prospective studies have been published that detail outcomes for larger cohorts of dogs. In 1 study,[24] 95 dogs with IHPSSs were treated with transvenous embolization, of which 79 (83%) had 1 treatment and 16 (17%) had multiple treatments. Overall, in 70 of 86 (81%) treated dogs, outcome was considered excellent or fair in 57 (66%) and 13 (15%) dogs, respectively, and median survival time after treatment was 2,204 days.[24] Several factors, including improvements in various clinicopathologic variables, were shown to be significantly associated with an excellent outcome.[24] In another study,[23] 25 dogs undergoing PTCE were prospectively assessed to evaluate

changes in clinical signs, clinicopathologic variables, and diagnostic imaging findings as a result of treatment. Significant improvements in clinical signs, liver volume, and hepatic arterial fraction were identified after PTCE.[23] In a third study,[25] cellophane banding was compared with PTCE as treatments for dogs with IHPSSs. The procedural duration between open cellophane banding management in 31 dogs and PTCE in 27 dogs for treatment of IPHSSs did not differ significantly.[25] Five years after treatment, the percentage of dogs surviving was 75% among those treated with cellophane banding and 80% among those treated with PTCE.[25]

Two reports[15,18] describe the use of interventional radiology methods in the treatment of IHPSSs in 3 cats. In 1 case report,[18] coil embolization was performed in 1 cat without stent support. No complications were encountered during the procedure in that cat, and the cat was reportedly doing well with clinical improvement at 10 weeks after treatment. More recently, a hybrid approach with coil embolization in 2 cats was reported.[15] For those cats, celiotomy was performed and either the portal vein or a mesenteric vein was directly catheterized to allow access to the shunt for coil placement. Coils were placed directly into the shunt, and the cats were considered to have good outcomes at 10 and 22 months, respectively, after treatment.[15]

To the authors' knowledge, this is the first report of a stent-supported PTCE procedure used to treat IHPSSs in cats. Patient size is an important factor when considering different treatment options. For interventional radiology procedures, the vascular access point has to be sufficiently large to allow placement of the largest device necessary during that particular procedure. Currently, the caval stents used in canine PTCE procedures have a relatively wide delivery system (10F) and are available in diameter sizes of ≥ 16 mm; thus, the placement of a 10F (or greater) sheath in the jugular vein is necessary in dogs. In the cats of the present report, smaller stents (10 mm in diameter) with a smaller delivery system were used. However, if the clinician performing the procedure is planning dual angiographic evaluation (ie, simultaneous cavography and portography), as described for the cats of the present report, then an 8F introducer or larger (2 catheters of at least 4F in diameter each) should be inserted in the jugular vein. The authors prefer to have simultaneous catheter placement within the shunt to allow portal pressures to be continuously recorded while coils are being delivered. However, as noted in 1 cat (cat 4) in the present study, the portal pressure can be measured in between coil placements. Given the small size of the portocaval shunt in that cat, only 2 coils were placed, and navigation in and out of the abnormal vessel with the microcatheter was considered reasonable. Coil size should also be considered during PTCE of cats. In most dogs, 8-mm-diameter coils are well tolerated,[23,24] but the authors selected smaller coils for the cats of the present report because of the comparatively narrower lumen of the anomalous portocaval vessels in this species. Further, the shape of the coils is also a consideration because some shunts have very short lengths at the caval ostium. Tornado-configured microcoils were used in 1 cat (cat 4) of the present report. These coils maintain a lower profile yet provide equivalent surface area contact at the level of the stent, compared with coils not of tornado configuration. Regardless, the 4- to 5-mm-diameter coils appeared to be well tolerated by the cats in the present report.

The clinical signs of cats with PSSs can be variable, but neurologic abnormalities are common.[6,7] Three of the 4 cats of the present report initially had neurologic abnormalities such as ataxia, head pressing, disorientation, and obtundation. At the long-term follow-up evaluations, these clinical signs appeared to have resolved, and owners reported normal behavior of each cat. Similar to findings in dogs, the gastrointestinal and urinary systems are commonly affected in cats with PSSs. One of the 4 cats of the present report was evaluated because of urinary tract signs (urethral obstruction secondary to urate urolithiasis); other clinical signs of an IHPSS were not evident in this cat prior to treatment. Copper-colored irises are a relatively common finding in cats with PSSs,[6,7] and this characteristic was detected in 1 of the 4 cats of the present report.

The authors of the present report routinely perform CTA in all cats prior to treatment with PTCE. This practice allows for detailed assessment of the vasculature as well as the development of a vascular map that can be used during the fluoroscopic procedure. Additionally, the stent size used in cats undergoing PTCE is determined prior to the procedure from images obtained via CTA. Although none of the 4 cats of the present report underwent post-PTCE CTA, this follow-up assessment would be an option to consider to enhance assessment of response to treatment and evaluate the patient for development of additional shunting blood vessels. Recently, the use of CTA to compare hepatic arterial blood flow and hepatic volume before and after PTCE was shown to be useful in dogs.[23]

Percutaneous transvenous coil embolization was performed without major intraprocedural complications in the 4 cats of the present report. It is routine practice for the authors to discuss with owners the potential complications that may occur in the perioperative period, including hemorrhage, portal hypertension, posttreatment seizures, blindness, and implant- and anesthesia-related complications. Such complications did not occur in the 4 cats, but clinicians performing PTCE procedures should be cognizant of their potential development, especially as these types of complications have been reported after PTCE in dogs.[24] In dogs, major reported intraprocedural complications have included substantial portal hypertension and severe acute gastrointestinal hemorrhage.[24] In that study[24] of 95 dogs with IHPSSs, life-threatening postprocedural complications included seizures or hepatic encephalopathy (7/111 [6%] procedures), cardiac arrest (2/111 [2%] proce-

dures), and hemorrhage from the jugular site requiring transfusion (2/111 [2%] procedures); pneumonia, suspected portal hypertension, and sudden death as a result of an unknown cause were each associated with 1 procedure.

At 14 months after the PTCE procedure, 1 cat (cat 2) that was originally presented for neurologic and gastrointestinal signs developed lower urinary tract signs after treatment. It was determined that this cat had developed an acquired EHPSS. No findings during the procedure were noted that would suggest why this occurred in this particular cat; portal vein hypoplasia was not suspected on the basis of CTA or intraprocedural findings. Acquired PSSs most commonly develop secondary to underlying hepatic disease that is associated with portal hypertension, but the development of acquired PSSs in dogs with congenital PSSs has also been reported.[31] In dogs with IHPSSs, the presence of multiple IHPSSs both before and after PTCE treatment has also been described.[23] Further evaluation of the use of PTCE and the potential for development of acquired PSSs after treatment in cats is needed.

Although the diagnosis of IHPSSs in cats is uncommon and the number of treated cats described in the present report was limited to 4, the use of PTCE as a treatment for feline patients with IHPSS appears to hold promise. No short-term complications were encountered, and all cats had posttreatment improvement in clinical signs, although an acquired EHPSS developed in 1 cat. However, one should consider that although clinical follow-up information was obtained, posttreatment imaging assessments of these cats were not performed. Additionally, the retrospective nature of data collection was associated with inherent challenges and possible error. Further investigation of the use of endovascular techniques for the treatment of IHPSSs in cats and other species is warranted.

Footnotes

a. Purina Pro Plan Veterinary Diet NF Kidney Function Feline Formula, St Louis, Mo.
b. Prescription Diet l/d Feline, Hill's Pet Nutrition Inc, Topeka, Kan.
c. GE Lightspeed 16, GE Medical Systems, Milwaukee, Wis.
d. Isovue (370 mg/mL), Bracco Diagnostics Inc, Princeton, NJ.
e. Becton, Dickinson and Co, Franklin Lakes, NJ.
f. Weasel Wire, Infiniti Medical, Redwood City, Calif.
g. Merit MAK mini access kit, Merit Medical, Jordan, Utah.
h. Introducer sheath and dilator, Infiniti Medical, Redwood City, Calif.
i. Ethilon nylon suture, Ethicon US LLC, Bridgewater, NJ.
j. Berenstein catheter, Infiniti Medical, Redwood City, Calif.
k. Marker catheter, Infiniti Medical, Redwood City, Calif.
l. Urethral stent, Infiniti Medical, Redwood City, Calif.
m. Bassett wire, Infiniti Medical, Redwood City, Calif.
n. Cook Medical Inc, Bloomington, Ind.
o. Arrow, Teleflex, Morrisville, NC.
p. Toshiba Prime 160 multidetector row computed tomography, Toshiba America Medical Systems, Tustin, Calif.
q. Prescription Diet k/d Feline, Hill's Pet Nutrition Inc, Topeka, Kan.
r. J-tip guidewire, Terumo Medical Corp, Somerset, NJ.
s. Zilver Vascular Self-Expanding Stent, Cook Medical, Bloomington, Ind.
t. Renegade STC-18 microcatheter, Boston Scientific, Marlborough, Mass.
u. Microwire, Terumo Medical Corp, Somerset, NJ.
v. VortX Tornado Coil, Boston Scientific, Marlborough, Mass.

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From this month's *AJVR*

Activities of matrix metalloproteinase-2, matrix metalloproteinase-9, and serine proteases in samples of the colorectal mucosa of Miniature Dachshunds with inflammatory colorectal polyps

Noriyuki Nagata et al

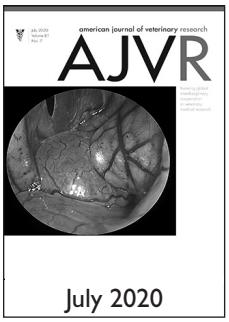
OBJECTIVE
To investigate the activities of gelatinases (matrix metalloproteinase [MMP]-2 and MMP-9) and serine proteases in the colorectal mucosa of Miniature Dachshunds (MDs) with inflammatory colorectal polyps (ICRPs).

ANIMALS
15 MDs with ICRPs and 5 dogs with non–ICRP-related large bowel diarrhea (controls).

PROCEDURES
Zymographic methods were used to evaluate the activities of MMP-2, MMP-9, latent forms of MMP-2 and MMP-9 (pro–MMP-2 and pro–MMP-9), and serine proteases in inflamed and noninflamed tissue samples from MDs with ICRPs and in noninflamed tissue samples from control dogs. The associations of serine protease activities with MMP-2 or MMP-9 activity were also analyzed.

RESULTS
Activities of pro–MMP-2 and pro–MMP-9 were detected in most tissue samples from MDs, regardless of the tissue type, whereas activities of MMP-2 and MMP-9 were not detected in control tissue samples. In the inflamed tissue samples from MDs with ICRPs, the activities of MMP-2, pro–MMP-9, and MMP-9 were significantly higher than those in the noninflamed tissue samples from those dogs. Serine protease activities were significantly higher in the inflamed and noninflamed tissue samples from MDs with ICRP, compared with findings for control tissue samples. A weak correlation was detected between serine protease activities and MMP-9 activity.

CONCLUSIONS AND CLINICAL RELEVANCE
Study results suggested that gelatinase and serine protease activities are upregulated in the colorectal mucosa of MDs with ICRPs, possibly contributing to the pathogenesis of this disease through the functions of these enzymes in degradation of extracellular matrix and promotion of inflammatory cell migration and inflammatory responses. (*Am J Vet Res* 2020;81:572–580)



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