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Use of ethylene-vinyl alcohol copolymer as a liquid embolic agent to treat a peripheral arteriovenous malformation in a dog

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Abstract

Case Description—An 11-year-old castrated male Tibetan Mastiff was evaluated because of a visibly enlarged blood vessel and progressively worsening swelling of the right hind limb.

Clinical Findings—On physical examination, the right hind limb was markedly larger than the left hind limb and the dog was minimally weight bearing on the affected limb. A bruit was auscultated over the affected region. Ultrasonography of the tarsal region of the right hind limb revealed an artery with turbulent flow that communicated with venous drainage. A CT scan confirmed the presence of an arteriovenous malformation (AVM).

Treatment and Outcome—Embolization of the AVM with a liquid embolic agent (ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide) was elected. An arteriogram was performed prior to treatment and delineated the vessels that were targeted for embolization. The embolic agent was infused into the AVM, and a postinjection arteriogram confirmed complete occlusion of the AVM nidus and normal arterial flow to the paw with subsequent normal venous drainage. The circumference of the abnormal paw was 51 cm before the procedure and 22.9 cm at 4 weeks after the procedure. Additionally, the gait of the dog dramatically improved. No complications associated with the procedure developed.

Clinical Relevance—Peripheral AVMs in dogs are uncommon, and described treatment options are limited and generally associated with serious morbidity. A liquid embolic agent, ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide, was successfully administered in this case, and no morbidity was observed secondary to the procedure. Clinical success was characterized by substantial improvement in limb swelling and marked improvement in the gait of the dog.

An 11-year-old castrated male Tibetan Mastiff (weight, 48.9 kg [108 lb]) was evaluated because of diffuse swelling of the right hind limb and paw. The dog's owners noticed a visibly enlarged blood vessel in the right hind limb that had been increasing in size for 7 months prior to evaluation. There was no known history of trauma or neoplasia of the right

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hind limb. No changes in appetite were apparent, and there was no history of lethargy, anorexia, vomiting, diarrhea, or polyuria and polydipsia.

On physical examination, the dog was lame in the right hind limb and the limb was diffusely swollen from the level of the mid tibia to the paw. Additionally, a 10-cm-long, easily compressible mass-like lesion was evident in the region of the right lateral saphenous vein. The right popliteal lymph node was also enlarged. Minor signs of pain were elicited on palpation of the lumbosacral region.

No abnormalities were detected on CBC and serum biochemical analysis. Cytologic evaluation of fine-needle aspirates of the right popliteal lymph node revealed reactive lymphoid and histiocytic hyperplasia with chronic and ongoing hemorrhage.

Three-view (right lateral, left lateral, and dorsoventral) thoracic radiography revealed no abnormalities. A 4-view (lateromedial, dorsoplantar, dorsomedial-plantarolateral oblique, and dorsolateral-plantaromedial oblique) radiographic assessment of the right hind limb was performed and revealed moderate, diffuse soft tissue swelling of the distal aspect of the crus and pes and multiple regions of periosteal proliferation on the metatarsal bones and phalanges.

During ultrasonographic evaluation of the affected limb, the proximal region of the femoral artery appeared normal to the level of the caudal femoral artery. Approximately 3 cm proximal to the tarsus, turbulent arterial flow was encountered and continuity with the venous drainage was seen as a plexus of abnormal arterial branches that communicated directly with associated veins. Doppler assessment of the vascular plexus indicated multidirectional high-velocity blood flow with a mixture of color signals (Figure 1) consistent with a mosaic pattern. Spectral Doppler assessment of the vascular plexus was not recorded. Veins in the tarsal region contained turbulent blood flow with an arterial pulsatile flow pattern.

The dog was placed under general anesthesia for CT-angiographic evaluation of the right hind limb arteriovenous communication. The dog was premedicated with hydromorphone (0.5 mg/kg [0.23 mg/lb], SC), acepromazine (0.01 mg/kg [0.0045 mg/lb], SC), and atropine (0.02 mg/kg [0.009 mg/lb], SC). After premedication, an IV catheter was placed in the cephalic vein. Induction of anesthesia was performed with propofol (2.5 mg/kg [1.1 mg/lb], IV) and midazolam (0.2 mg/kg [0.09 mg/lb], IV). Anesthesia was maintained with isoflurane in oxygen. The dog was positioned within the CT gantry in dorsal recumbency with the hind limbs extended. By use of a standard 16-slice helical CT unit, contiguous, 0.6-mm collimated images of both hind limbs were obtained from immediately proximal to the hip joints distally to include the digits. A dual-phase angiogram was then obtained from the right stifle joint to the digit. A low dose of nonionic, iodinated contrast medium[a] (200 mg/kg [90.9 mg/lb], IV) was injected at 2 mL/s into the cephalic catheter, and a dynamic CT acquisition was initiated. Images were acquired every 2 seconds at a selected location where the right femoral artery and vein could be easily seen. The time to maximal arterial and

[a]Isovue 370, Bracco Diagnostics Inc, Princeton, NJ.

venous enhancement was calculated. The dual-phase angiogram was then obtained by administering a full dose of contrast medium (800 mg/kg [363.6 mg/lb], IV) followed by 2 additional CT sequences, one beginning at the calculated time of maximal arterial enhancement and the second at the time of maximal venous enhancement.

Computed tomography revealed a large plexus of abnormal blood vessels in the caudolateral aspect of the right hind limb surrounding the caudal branch of the lateral saphenous vein and extending along the distal half of the tibia (Figure 2). The main arterial supply to the AVM was a branch of the popliteal artery (ie, the cranial tibial artery). Proximal to the vascular anomaly, the saphenous vein was markedly distended. Distal to the vascular plexus, the limb was markedly, diffusely swollen with prominently distended veins. There was smooth periosteal proliferation of some of the metatarsal bones. The right popliteal lymph node was also markedly enlarged. The CT interpretation was a large AVM adjacent to the distal aspect of the right tibia with secondary venous congestion, edema in the distal portion of the limb, and metatarsal periosteal productive reaction.

The owners were offered several treatment options and elected to pursue treatment with liquid embolization approximately 6 months later. On physical examination at that time, the right hind limb swelling had progressed considerably and the dog was only minimally weight bearing on that limb. A bruit had developed and was auscultated over the affected region of the right hind limb. Fluid (presumed to be lymph) was observed as coming from the skin of the paw (Figure 3). The circumference of the abnormal right hind paw (from most lateral aspect of the fifth digit to most medial aspect of the second digit) was 51 cm, and the circumference at the right metatarsophalangeal joint was 35.6 cm. The circumference of the unaffected left hind paw was 18.4 cm, and the circumference at the left metatarsophalangeal joint was 15.2 cm. The weight of the patient at this visit was 50 kg (110 lb); the rest of the physical examination findings were unchanged.

The dog was premedicated with morphine (0.5 mg/kg, IM) and glycopyrrolate (0.01 mg/kg, IM), and a cephalic IV catheter was placed. Anesthesia was induced with propofol (2.6 mg/kg [1.2 mg/lb], IV) and diazepam (0.3 mg/kg [0.14 mg/lb], IV) and maintained with isoflurane in oxygen. The patient was transferred to the catheterization laboratory and positioned in dorsal recumbency. Hair over the right femoral region was clipped; the area was prepared with sterile technique and draped.

The right femoral artery was digitally palpated through the skin, and a 1-cm incision was made over the femoral artery. The fascia surrounding the femoral artery was dissected away to expose the femoral artery. Two 3-0 sutures of polydioxanone[b] were placed around the femoral artery. The proximal suture was ligated around the femoral artery. An 18-gauge over-the-needle catheter[c] was introduced into the femoral artery (oriented distally), and the needle was removed. An 0.035-inch hydrophilic guide wire[d] was introduced into the over-the-needle catheter and subsequently into the femoral artery. The over-the-needle catheter was removed over the guide wire. A 5F vascular access sheath and dilator[e] were introduced

[b]PDS, Ethicon Inc, Somerville, NJ.
[c]Angiocath, Becton, Dickinson and Co, Franklin Lakes, NJ.
[d]Weasel wire, Infiniti Medical, Menlo Park, Calif.

into the femoral artery over the guide wire, and the proximal polydioxanone suture was ligated around the femoral artery and vascular access sheath combination. The dilator was then removed over the guide wire. The fluoroscopy unit was positioned over the proximal right hind limb of the patient.

After positioning the guide wire in the femoral artery, a 4F Berenstein catheter[f] was passed through the sheath over the guide wire into the femoral artery. An arteriogram was obtained at this level by the injection of a 50:50 combination of saline (0.9% NaCl) solution and nonionic, iodinated contrast medium.[a] An enlarged feeding artery (confirmed as the cranial tibial artery) was observed at the middle aspect of the tibia, which was rapidly shunting into dilated draining veins through several communicating vessels (AVM; Figure 4).

The guide wire was then passed from the femoral artery into the cranial tibial artery. The feeding artery of the nidus was observed at the level of the mid tibia, and the guide wire was passed as far into the feeding vessel of the nidus as possible. The Berenstein catheter was passed over the guide wire to the level of the nidus, and then the guide wire was removed. A dimethyl sulfoxide–compatible microcatheter[g] and microwire[h] combination were coaxially introduced into the Berenstein catheter and passed into the AVM nidus (Figure 4). Once the microcatheter was in position in the nidus, the microwire was removed and a contrast angiogram (100% contrast) was performed to confirm appropriate location within the target vessel. The contrast medium was then flushed from the microcatheter with 10 mL of saline solution.

Liquid embolization was performed with a commercially available nonadhesive, radiopaque liquid embolic agent: ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide and suspended micronized tantalum powder.[i] Preparation of the liquid embolic agent was performed according to manufacturer recommendations after the microcatheter was appropriately placed in the nidus of the AVM. In short, the ethylene-vinyl alcohol copolymer was mixed for at least 20 minutes prior to injection, and dimethyl sulfoxide was injected into the microcatheter to fill the catheter dead space. The catheter hub was maintained in a vertical position, and the Luer hub was overfilled with the dimethyl sulfoxide. A 1-mL syringe containing ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide was immediately connected to the hub, ensuring that no air was introduced. The syringe was held vertically, and the liquid embolic agent was injected at a slow, steady rate controlled by thumb pressure under fluoroscopic observation until the nidus was completely filled (Figure 4). After completion of the injection, the syringe was slightly aspirated and the microcatheter was gently pulled to separate it from the embolic cast; the microcatheter was then removed.

An angiogram was obtained from the Berenstein catheter after the embolic had solidified and revealed no flow through the AVM nidus, improved arterial blood flow in the distal

[e]Vascular introducer sheath with dilator, Infiniti Medical, Menlo Park, Calif.
[f]Berenstein catheter, Infiniti Medical, Menlo Park, Calif.
[g]Marathon, ev3, Plymouth, Minn.
[h]Microwire, Infiniti Medical, Menlo Park, Calif.
[i]Onyx, ev3, Plymouth, Minn.

aspect of the limb, and appropriate venous drainage (Figure 4). The Berenstein catheter and introducer sheath were removed, and the femoral artery was ligated. The subcutaneous tissue and skin were closed routinely.

The dog recovered from anesthesia without incident and was discharged the next morning. At 1 month after treatment, the dog was ambulating well on the right hind limb. The circumference of the abnormal paw was 22.86 cm, and the circumference at the meta-tarsophalangeal joint was 19.05 cm (Figure 5). The circumference of the normal left hind paw and meta-tarsophalangeal joint was unchanged. At 13 months after treatment, the dog developed weakness in the hind limbs and signs consistent with a T3-L3 myelopathy; advanced diagnostic testing was not performed. Physical therapy improved the signs secondary to the myelopathy for many months, but the dog was euthanized nearly 18 months after embolization for worsening of myelopathy. No complications developed secondary to the embolization in the posttreatment period, and there was no recurrence of signs associated with the AVM. A necropsy was not performed.

Discussion

Vascular malformations are uncommon in all species and are generally considered a subcategory of vascular anomalies, with neoplastic or neoplastic-like lesions being the other major subcategory.[1,2] Historically, debate has existed about whether vascular malformations are a form of neoplasia (often called hemangiomas) or represent a separate entity.[3] Unlike vascular malformations, hemangiomas are not present at birth, grow rapidly during a proliferative phase, go through a period of stabilization, and then involute (often completely).[2–6] Conversely, vascular malformations are always present at birth, grow with the affected individual, and never regress.[3–7]

More recently, vascular malformations have been better characterized on the basis of the presence of histologic and immunohistochemical properties, allowing for a more accurate assessment. Vascular malformations are composed of mature endothelium that does not undergo tumor-like endothelial proliferation[1,5]; interestingly, use of α-smooth muscle antibody stain reveals an absence of smooth muscle in the vascular wall, which is generally considered the reason for ectasia and the increase in size of the malformation that occurs during the growth of the affected individual.[5]

Several classification systems for humans have been developed to categorize the myriad of vascular malformations that have been documented. One of the earliest systems divided the malformations into categories according to the endothelial characteristics; in that classification scheme, the malformations were identified as fistulae or as either capillary, venous, arterial, or lymphatic.[3] Jackson et al[6] developed a separate classification system complementary to the endothelial classification scheme based on the identification of the blood flow rate. In that scheme, vascular malformations were classified into low-flow (venous) and high-flow (arteriovenous) malformations.[6]

The modified Hamburg classification system is likely the most accepted means of classifying vascular malformations in humans and takes into account a primary classification

centered on whether the malformation is arterial, venous, arteriovenous, capillary, lymphatic, or combined vascular.[1,5] After a primary classification has been assigned, subclassification is established on the basis of embryological characteristics, with major categories being extratruncular and truncular.[1,5] Extratruncular and truncular forms are distinguished by the time at which the defect in embryogenesis occurred[5,8]; generally, extratruncular forms are derived from the remnants of the extratruncular primitive capillary network and truncular forms are dysembryoplasias of a differentiated vascular truncus.[8] Arteriovenous malformations that are truncular often result in aplasia or obstruction, whereas extratruncular forms are infiltrating.[5,8]

Although a classification system for vascular malformations in dogs has not been developed, some of the established principles in humans[8] can be applied to the dog of this report. The vascular anomaly in the dog of this report could be classified as an AVM with high-flow rate and extratruncular characteristics. The importance of classification lies in the ability to impact clinical decision making (ie, choice of an appropriate embolic agent).

It was interesting that this malformation was observed later in life, given that a developmental abnormality might be anticipated to manifest earlier; however, the medical history of the dog of this report suggested a developmental etiology. Connections between the arterial and venous system can occur secondary to trauma or neoplasia; however, the dog of this report had a history of neither. The owners had owned the dog since birth and confirmed that a tumor had never been removed from that region and no trauma had occurred. Additionally, imaging results supported a lack of trauma and neoplasia in the region of the vascular malformation. A periosteal reaction can be encountered secondary to trauma, but the reaction in this case was presumed to be secondary to the altered blood flow in this region.

The clinical signs in the dog of this report were similar to what is often found in humans with an AVM in the limbs.[5,9,10] Patients with AVMs often develop edema, cellulitis, and signs of pain.[5,9,10] Additionally, ischemia to the skin or tissue distal to the AVM can occur.[5,9,10] As the blood volume increases over time, the involved veins subsequently become dilated and tortuous, as occurred in the dog of this report.

Several physiologic processes occur with AVMs that result in increased systemic blood volume, which in turn results in greater venous return to the heart, pulmonary arterial hypertension, and potentially high-output cardiac failure.[5,11] High-output cardiac failure has been described for a cat with an aortocaval fistula, and clinical signs resolved after surgical treatment.[12]

Although adverse effects such as cardiac dysfunction had not occurred at the time of treatment in the dog of this report, the potential for eventual cardiovascular effects was a factor in treatment selection. The presence of lameness and limb swelling all necessitated the pursuit of treatment in this case. Amputation of the hind limb was considered, but certain factors including signs of pain on palpation of the lumbosacral region made this option less attractive.

Prior to initiating treatment, several imaging modalities were used to diagnose and characterize the AVM in the dog of this report. Ultrasonography (B-mode and Doppler) was useful for confirming an abnormal communication between the arterial and venous systems, which allowed for a more focused consideration of available treatment options. Computed tomography–angiography provided a pretreatment vascular map of the region and helped to rule out the presence of an underlying disease process such as previous trauma or neoplasia. In this case, as in humans, the intraprocedural fluoroscopic-angiography was the most useful imaging modality for defining the specific flow pattern and abnormal arteriovenous communication in the complex vascular plexus. During fluoroscopic angiography, the AVM could be clearly identified and the appropriate feeding vessels could be targeted. Further, the abnormal venous drainage pattern was recognized. Angiograms obtained at the conclusion of embolization provided evidence of successful treatment in that arterial flow to the distal aspect of the limb improved, venous drainage was corrected, and flow through the AVM was no longer seen.

Because of the high-velocity blood flow of the AVM in the dog of this report, arterial embolization with an intravascularly delivered embolic agent was considered the treatment of choice. A coaxial catheter system allowed for placement of the embolic agent directly in the nidus, which maximized dose administration and decreased the chance of nontarget embolization. Percutaneous treatment with a sclerosing agent such as ethanol is a commonly chosen option for low-flow vascular malformations; however, there was concern about use of this treatment because of the high-flow rate in this lesion.[7] Infusing ethanol into a high-flow lesion can result in diffuse spread of the agent and resultant non-target embolization; other complications may include cardiovascular collapse and seizures.

Transcatheter embolization has emerged as the treatment of choice for AVMs in humans.[5] The goal of treatment of AVMs is to embolize the feeding arteries as close to the lesion as possible while completely eliminating the nidus.[7,10,13] It is desirable to leave the main access vessels patent to allow for future treatments. Additionally, embolization of vessels delivering blood to normal structures adjacent to the lesion should be avoided.[13]

Several embolic agents have been investigated in the treatment of AVMs. Embolic agents that have been used include detachable balloons, coils, particulate agents (eg, gelfoam, polyvinyl alcohol particles, and spherical embolic agents), and liquid or sclerosing agents.[1,5,14] Balloons are often not currently used as permanent occlusion agents as a result of the development of more effective options. The use of a coil was successful in treating a congenital arteriovenous fistula of the saphenous artery in a dog previously,[15] and coils are often used in the treatment of portosystemic shunts (mostly intrahepatic) in dogs.[16] However, the use of coils is controversial in human medicine because the nidus is often not destroyed, resulting in recruitment of more vessels; additionally, future interventions are not generally possible after coil embolization. The delivery of particulate agents needs to be considered thoroughly because recanalization can occur; if blood flow is reestablished, further treatment would be indicated.[17] Liquid embolic agents commonly used in human patients include glue and ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide. Cyanoacrylate is the glue option that has been most evaluated and has been shown to be an effective option in the treatment of AVMs. Ethylene-vinyl alcohol copolymer as a liquid

embolic agent was selected for treatment of the dog in this report because of success with its use in several human studies[18–20] as well as several advantages, including a nonadhesive nature, slower polymerization, good visibility, and excellent delivery control.

The ethylene-vinyl alcohol copolymer is a biocompatible liquid that can form an embolus when contact is made with blood.[10,18–21] It is embedded with micronized tantalum powder to provide contrast for visualization with fluoroscopy. The liquid embolic agent used in the present study (ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide) has been investigated for cerebral use in many studies,[10,19,22,23] but use for peripheral indications is increasing as well.[20,24–28] Peripheral uses result in clinical success in many human cases with a low complication rate.[20,25,27,28]

Several cases of arteriovenous fistulae and AVMs have been described in the veterinary literature.[12,15,29–34] Hepatic AVMs, which are congenital, predominate, but other causes such as trauma (surgical and other forms) and neoplasia have also been reported.[12,15,29–34] Liquid embolic agents have been used with success in a few patients, but experience with this technique in the treatment of peripheral AVMs is limited.[32]

The use of a liquid embolic agent, such as ethylene-vinyl alcohol copolymer, and its performance for peripheral AVM embolization with the techniques described here has not been previously reported. Additionally, the dramatic response with only a single treatment while maintaining sufficient blood flow to the limb was highly promising. Peripheral AVMs in dogs are uncommon, and available treatment options are limited and are associated with severe morbidity. No morbidity was associated with this procedure, and the successful use of this embolic agent was characterized by substantial improvement in swelling of the distal aspect of the limb and marked improvement in the gait of the patient. The long-term outcome in this case was also encouraging; however, it will be important to continue to monitor the success of this treatment option in future cases.

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Abbreviation

AVM Arteriovenous malformation

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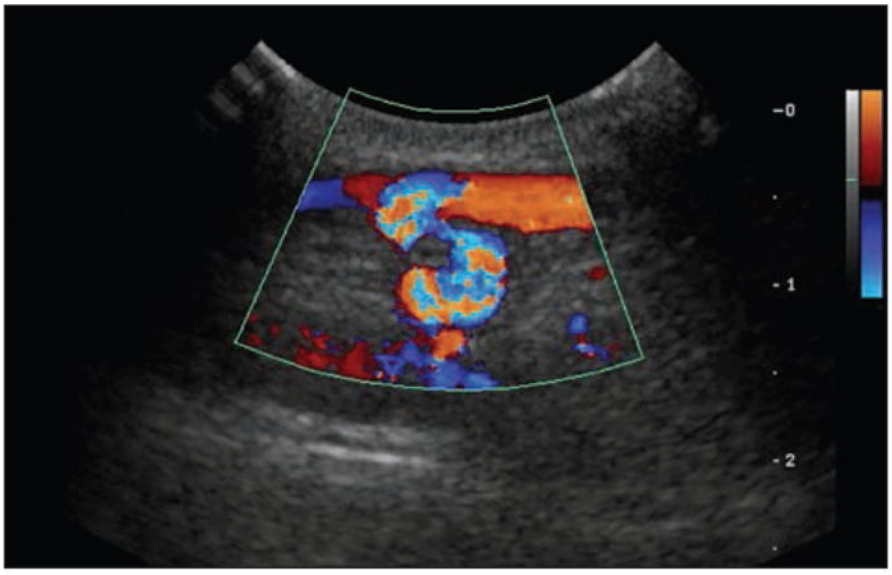

Figure 1.

Ultrasonographic image of an AVM of the right hind limb in an 11-year-old castrated male Tibetan Mastiff. Notice the typical mosaic pattern of Doppler signals. The 2-color scale indicates direction (red = toward the transducer; blue = away from the transducer) and relative velocity of flow (lighter shade corresponds to greater velocity). The mosaic pattern is indicative of turbulent flow within the AVM.

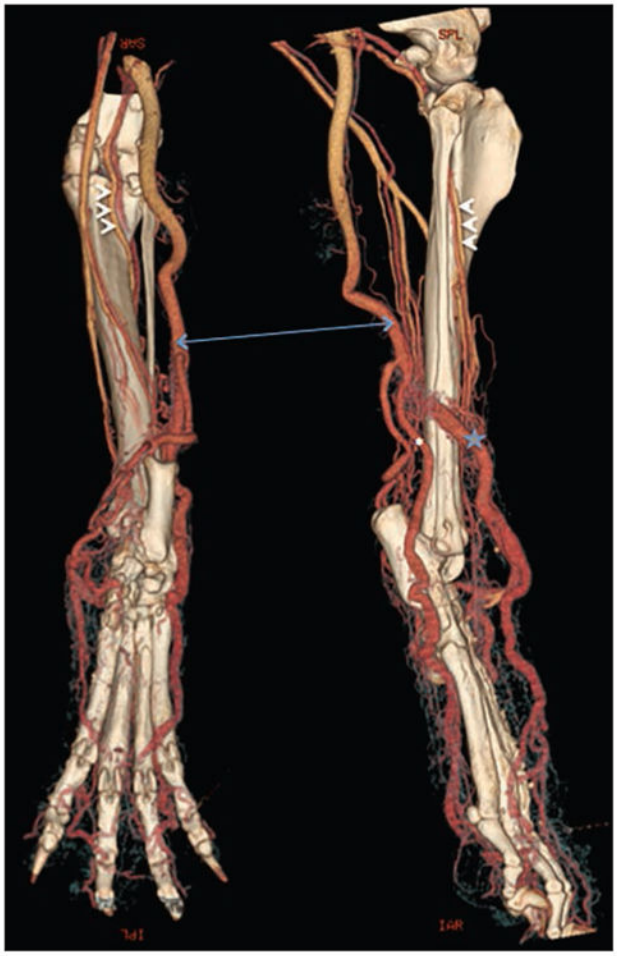

Figure 2.

Three-dimensional CT-angiographic rendering of the arterial feeding vessels of the AVM and draining veins of the dog of this report. The cranial tibial artery (branching from the popliteal artery) is the primary feeding artery of the AVM (white arrowheads). The lateral saphenous vein serves as the primary venous return of the AVM (blue arrows) with the cranial (blue star) and caudal (white asterisk) branches of the lateral saphenous vein present immediately distal to the AVM.

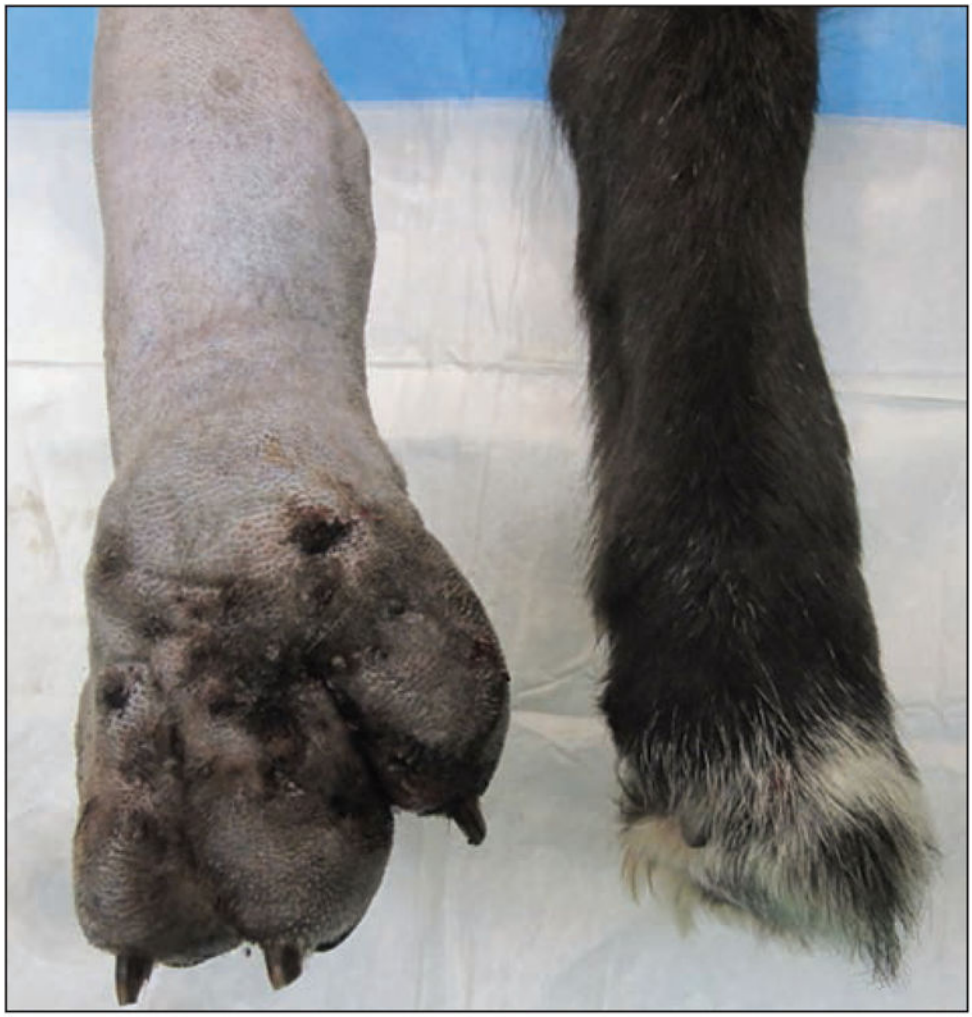

Figure 3.
Photograph of the hind paws of the dog of this report. Notice the diffuse swelling of the right hind paw, compared with the contralateral paw.

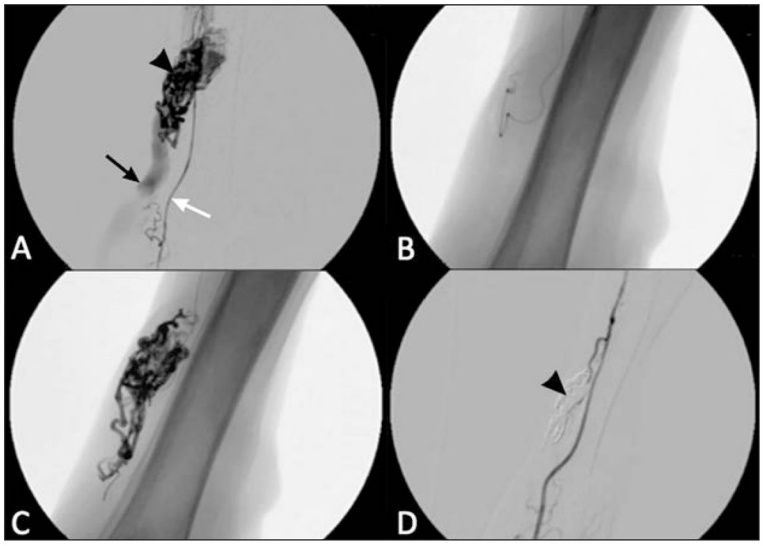

Figure 4.

Intraprocedural fluoroscopic images of the AVM of the dog of this report. A—Digital subtraction image in which an injection from the femoral artery has been performed. The AVM nidus (arrowhead), draining vein (black arrow), and artery providing blood to the distal aspect of the limb (white arrow) can be seen simultaneously. B—A microcatheter has been placed into the AVM nidus, and injection of ethylene-vinyl alcohol copolymer has been initiated. C—The nidus has been filled with ethylene-vinyl alcohol copolymer. D—Digital subtraction image obtained during an injection. Notice the lack of contrast enhancement of the AVM nidus (black arrowhead).

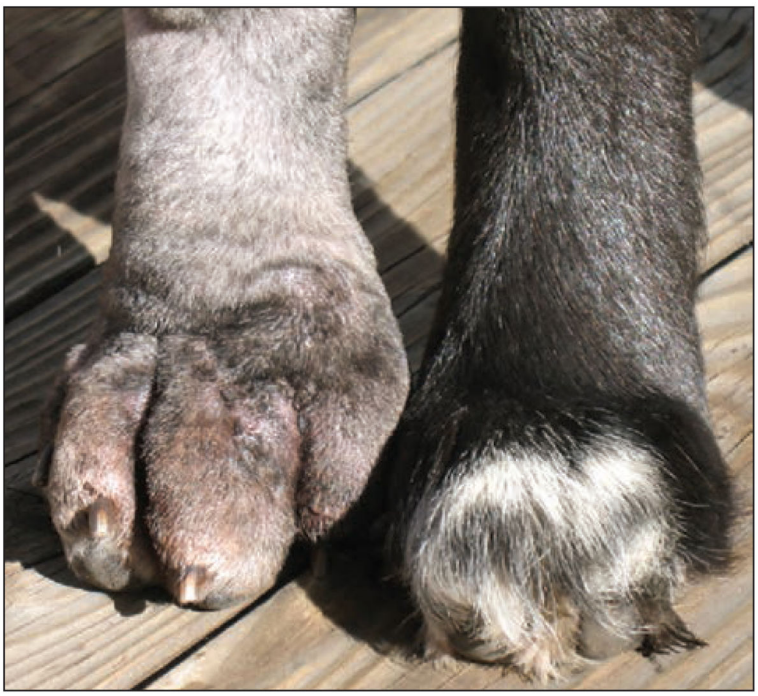

Figure 5.

Photograph of the distal aspect of the hind limbs of the dog of this report at 4 weeks after treatment. Notice the decrease in size of the distal aspect of the right hind limb, compared with Figure 3.