Compartment syndrome associated with expansile antebrachial tumors in two dogs

Lynn C. Maki, DVM; Stanley E. Kim, BVSc, MS; Matthew D. Winter, DVM; Kelvin Y. Kow, DVM, MS; Julia A. Conway, DVM; Daniel D. Lewis, DVM

Case Description—A 10-year-old spayed female Jack Russell Terrier and a 7-year-old neutered male mixed-breed dog were evaluated because of acute, progressive, unilateral forelimb lameness associated with signs of pain and turgid antebrachial swelling.

Clinical Findings—For each dog, there were no palpatory abnormalities. A diagnosis of compartment syndrome was confirmed on the basis of high caudal antebrachial compartmental pressure in the affected forelimb.

Treatment and Outcome—Both dogs underwent surgical exploration of the affected forelimb. In each case, an intramuscular tumor (mast cell tumor in the Jack Russell Terrier and suspected sarcoma in the mixed-breed dog) was detected and presumed to be the cause of the high compartmental pressure. At 6 months following tumor excision, the dog with the mast cell tumor did not have any clinical signs of disease. The dog with a suspected sarcoma underwent tumor excision and forelimb amputation at the proximal portion of the humerus followed by chemotherapy; the dog was euthanized approximately 1 year following treatment because of pulmonary metastasis.

Clinical Relevance—Compartment syndrome is a serious but rarely reported condition in dogs and is typically ascribed to intracompartmental hemorrhage. These 2 cases illustrate the potential for expansile intramuscular antebrachial tumors to cause compartment syndrome in dogs. (J Am Vet Med Assoc 2014;244:346-351)

A 10-year-old spayed female Jack Russell Terrier weighing 5.6 kg (12.3 lb) was referred to the University of Florida Small Animal Hospital because of intermittent signs of pain and swelling of the right antebrachium. The signs of pain and swelling recurred every 3 to 5 months over the preceding 2 years. On each occasion, the dog developed spontaneous swelling and pitting edema centered subjacent to the right elbow region. The episodes were self-limiting and usually resolved without treatment within 24 hours after onset. No traumatic or other potential inciting events were ever observed.

On initial evaluation, the dog would not bear weight on the right forelimb. Firm, diffuse, turgid swelling of the right antebrachium with minimal swelling of the paw and without pitting edema was evident (Figure 1). Signs of severe pain were elicited on gentle palpation of the antebrachium. No obvious puncture wounds or other skin lesions were found. Thoracic auscultation revealed apparently normal lung sounds with no obvious pleural friction rub. The remainder of the physical examination revealed no abnormalities.

Radiographic views of the right antebrachium obtained by the referring veterinarian provided evidence of only generalized soft tissue swelling. A CBC revealed a stress leukogram and mild anemia (Hct, 38.6%; reference range, 40% to 56%). The only abnormalities detected by serum biochemical analyses were mildly high alanine aminotransferase activity (103 U/L; reference range, 22 to 68 U/L) and aspartate aminotransferase activity (304 U/L; reference range, 15 to 32 U/L) and hyperfibrinogenemia (700 mg/dL; reference range, 100 to 400 mg/dL). Echocardiography revealed tricuspid stenosis and mitral valve regurgitation with no evidence of vegetative endocarditis.

Ultrasonographic examination of the affected limb revealed multiple anechoic regions among the muscle fibers of the proximal portion of the antebrachium, generalized decreased echogenicity of the muscles in the caudal aspect of the antebrachium, and a large volume of echogenic fluid in the elbow joint. Doppler ultrasonographic examination of the affected areas revealed minimal, inconsistent blood flow. Cytologic evaluation of an initial fine-needle aspirate sample from the affected forelimb area revealed proteinaceous fluid, RBCs, nondegenerate neutrophils, and bacteria. A sample of synovial fluid from the right elbow joint, along with another fine-needle aspirate from a muscle belly in the caudal aspect of the right antebrachium, was obtained and submitted for cytologic evaluation and bacterial culture. Cytologic evaluation of the synovial fluid sample revealed red opaque fluid with normal viscosity, a high total protein concentration (5.0 g/dL; reference range, 2 to 2.5 g/dL), and a high cell count (98,980 WBCs/µL and 234,000 RBCs/µL) with mostly mature neutrophils (77%; reference range, < 5%) and some evidence of degraded intracellular diplococci; however, microbial culture of the synovial fluid sample did not yield bacterial growth. Cytologic evaluation of...
the aspirate sample from the caudal antebrachial muscle yielded few cells but also a predominance of mature neutrophils with evidence of digested intracellular bacteria as well as low numbers of extracellular bacilli.

Computed tomography, performed with the dog under anesthesia, revealed diffuse, heterogeneous soft tissue thickening extending from the distal aspect of the brachium to the distal metacarpal region; the soft tissue thickening was most severe at the caudolateral aspect of the antebrachium, centered on the flexor muscles. Following administration of contrast medium, minimal, heterogeneous contrast enhancement was seen at the periphery of the most severely swollen tissues, but no contrast enhancement was evident centrally. With the dog still anesthetized and in left lateral recumbency, the caudal antebrachial compartmental pressures were measured (Figure 2). After sterile preparation, a standard 20-gauge hypodermic needle connected via a noncompressible, fluid-filled line to an invasive arterial pressure transducer was inserted percutaneously into the caudal compartment approximately 5 mm deep to ensure that the end of the needle was beneath the fascia. Pressure in the left forelimb was 0 mm Hg, whereas pressure in the right forelimb was 19 mm Hg (25.8 cm H₂O).

Immediately following pressure measurement, surgical exploration of the caudomedial aspect of the right antebrachium was performed via an incision extending distal from the right elbow region to just proximal to the central carpal pad. The caudal antebrachial fascia was distended. The antebrachial fascia was longitudinally incised to expose the flexor musculature. The deep digital flexor muscle bellies were diffusely and severely swollen with a soft, fluctuant consistency and dark gray discoloration. The swollen muscle bellies immediately protruded from the fascial compartment after the fasciotomy was made. A small portion of the medial capsule of the elbow joint was excised en bloc with the entire deep digital flexor muscle because these tissues were intimately adhered. The joint capsule was not closed because of the limited amount of remaining capsule. The excision site and elbow joint were copiously lavaged with sterile saline (0.9% NaCl) solution. The adjacent flexor muscles appeared grossly normal. A Jackson-Pratt drain was placed in the caudal antebrachial defect, and the antebrachial fascia was closed with 2-0 polydioxanone suture in a simple continuous pattern. The subcutaneous and skin layers were closed routinely. Excised tissue was submitted for histologic evaluation as well as aerobic and anaerobic bacterial culture.

The dog recovered well from anesthesia but became anxious overnight. Pain and anxiety were controlled with methadone hydrochloride (0.2 mg/kg [0.09 mg/lb], IV, q 4 to 6 h) and acepromazine maleate (0.02 mg/kg [0.009 mg/lb], IV, as needed). The dog was also administered IV fluid therapy at a maintenance rate (60 mL of isotonic fluids/kg/d [27.3 mL/lb/d]), ampicillin-sulbactam (30 mg/kg [13.6 mg/lb], IV, q 8 h), and metronidazole (18 mg/kg [8.2 mg/lb], IV, q 12 h). The morning following surgery, there was mild to moderate edema of the right antebrachium; however, the closure of the incision was intact. By the third day following surgery, the drain was removed; 60 mL of serosanguineous fluid had been collected. The dog was discharged from hospital, and the owner was instructed to administer tramadol (2 mg/kg [0.9 mg/lb], PO, q 6 h), amoxicillin-clavulanic acid (14 mg/kg [6.4 mg/lb], PO, q 12 h), and metronidazole (11 mg/kg [5 mg/lb], PO, q 12 h) pending histologic evaluation and bacterial culture results.

Histologic evaluation of sections from the center and a section from each longitudinal margin of the excised tissue was performed. An intramuscular mast cell tumor of the right deep digital flexor muscle with secondary necrotizing and eosinophilic myositis was iden-
The dog was anesthetized and positioned in left lateral recumbency, and the caudal aspect of the antebrachium was prepared in accordance with sterile technique. The compartmental pressure was then measured with an 18-gauge hypodermic needle attached to an invasive arterial transducer via sterile noncompressible, fluid-filled tubing. The pressure in the compartment was high (19 mm Hg).

A 12-cm incision was made along the caudal antebrachial compartment. A 1 cm incision was recommended, close such as chemotheraphy with vinblastine and lomustine or use of toceranib phosphate, a tyrosine kinase inhibitor. The owner declined further treatment. The dog was anesthetized, and the pressure in the caudal antebrachial compartment was measured in each forelimb with the same technique as described for the Jack Russell Terrier. The pressure in the left forelimb was 20 mm Hg (27 cm H₂O), and pressure in the contralateral limb was 3 mm Hg (4 cm H₂O).

A 7-year-old neutered male mixed-breed dog weighing 30 kg (66 lb) was referred to the University of Florida Small Animal Hospital because of left forelimb lameness that was unassociated with any known trauma. Four days prior, the owners had noticed a subtle left forelimb lameness that progressed to a non-weight-bearing lameness over a period of 3 days. A referring veterinarian initially examined the dog. The left antebrachium was swollen, and signs of pain were elicited on palpation. Radiography of the limb revealed only diffuse soft tissue swelling of the affected region. Results of a urinalysis, CBC, and serum biochemical analysis were all considered normal. The dog was administered a constant rate infusion of fentanyl citrate (4 µg/kg/h [1.8 µg/lb/h], IV) for analgesia and was referred the following day.

On initial evaluation, the dog was non-weight bearing on the left forelimb. There was turgid, diffuse swelling of the caudal portion of the left antebrachium without pitting edema; signs of pain were evident during palpation. Ultrasonographic examination of this region revealed circumscribed, severe, heterogeneous thickening within the fascial planes of the left flexor carpi radialis muscle with central, irregular, ill-defined hypoechogenic regions within the swelling. Doppler ultrasonographic examination of the affected region was not performed. Prothrombin time was 6.6 seconds (reference range, 6.8 to 10.2 seconds), and partial prothrombin time was 10.2 seconds (reference range, 10.7 to 16.4 seconds). Cytologic examination of fine-needle aspirate samples of the extensor muscles revealed few cells, mainly occasional unremarkable mesenchymal cells of possible fibroblastic or myocytic origin. Vacuolated mononuclear phagocytes were also observed; however, no erythropagia, infectious agents, or neoplastic cells were found. Thoracic radiography and abdominal ultrasonography were performed, and no abnormalities were detected. The dog was anesthetized, and the pressure in the caudal antebrachial compartment was measured in each forelimb with the same technique as described for the Jack Russell Terrier. The pressure in the left forelimb was 20 mm Hg (27 cm H₂O), and pressure in the contralateral limb was 3 mm Hg (4 cm H₂O).

The mixed-breed dog underwent surgery to explore and decompress the left caudal antebrachial compartment. A 12-cm incision was made along the caudal aspect of the antebrachium, initiated 1 cm distal to the elbow joint. The distended caudal antebrachial compartment was turgid, and when the fascia was incised, a diffuse, infiltrative, multilobulated, cystic mass protruded through the fasciotomy. The mass, which arborized between the muscular fascial planes, was dissected from the surrounding musculature while preserving nerves and was incompletely excised. Routine closure was performed after copious lavage with warm saline solution. Samples of the excised tissue were submitted for cytologic evaluation of an impression smear, bacterial culture, and histologic evaluation. After surgery, pressure in the left caudal antebrachial compartment was 7.2 mm Hg (10 cm H₂O).

The dog appeared comfortable after recovering from anesthesia and required only 1 dose of morphine sulfate (0.5 mg/kg [0.23 mg/lb], IV) overnight. A dose of cefazolin sodium (30 mg/kg, IV, q 6 h) was admin-

**Figure 2**—Photograph of the caudal aspect of the right antebrachium of the same dog as in Figure 1. The dog was anesthetized and positioned in left lateral recumbency, and the caudal aspect of the antebrachium was prepared in accordance with sterile technique. The compartmental pressure was then measured with an 18-gauge hypodermic needle attached to an invasive arterial transducer via sterile noncompressible, fluid-filled tubing. The pressure in the compartment was high (19 mm Hg).
Compartment syndrome develops when interstitial pressure within an osteofascial compartment becomes sufficiently increased to impede tissue perfusion. Compartment syndrome is caused by an increase in interstitial pressure that may be the result of swelling, hemorrhage, or fluid accumulation. The symptoms of compartment syndrome include pain, paresthesia, paresis, and, in severe cases, necrosis. The diagnosis of compartment syndrome is clinical and is based on the history, physical examination, and laboratory findings. In this case, the dog was referred to the hospital for evaluation of a pruritic, limb swelling. The history, physical examination, and laboratory findings were consistent with compartment syndrome. The limb was amputated, and the dog was discharged from the hospital. The chemotherapeutic protocol consisted of metoclopramide hydrochloride (0.3 mg/kg [1.4 mg/lb], SC) and carprofen (2.5 mg/kg [1.1 mg/lb], PO, q 8 h). The preliminary diagnosis was synovial cell sarcoma. The histologic examination of the excised tissue revealed a highly cellular tissue with atypical mesenchymal cells identified individually as well as in large, poorly cohesive sheets. The cells had mild to moderate anisocytosis and anisokaryosis with a variable but generally high nuclear-to-cytoplasmic ratio. Histologic examination of tissue sections revealed a poorly differentiated, anaplastic sarcoma. There were 3 to 6 mitotic figures/400X field with frequent bizarre mitotic figures. Extensive hemorrhage, multifocal fibrin deposition, and scattered necrotic debris were also visible. Neoplastic cells extended to all borders of the submitted excised tissue. The preliminary diagnosis was synovial cell sarcoma. Aerobic and anaerobic bacterial cultures of the excised tissues yielded no growth. Given the diagnosis of synovial cell sarcoma, limb amputation was recommended. The dog was returned 9 days after the initial surgery for amputation of the left forelimb at the proximal region of the humerus. Surgery was uneventful, and the dog was ambulatory and appeared comfortable the following morning. Histologic evaluation of a representative sample of the amputated limb revealed muscle necrosis, with focally extensive, subacute, marked vasculitis. Fibrinoid necrosis and thrombosis of the vessels, with multifocal fibroplasia of the soft tissue, surrounded the surgical site. There was no evidence of neoplasia within the examined sections of the amputated limb. The day following surgery, the dog received its initial cycle of chemotherapy (carboplatin [300 mg/m², IV] and metoclopramide hydrochloride [0.3 mg/kg [1.4 mg/lb], SC]) and was later discharged from the hospital. The chemotherapeutic protocol consisted of alternating administration of carboplatin (300 mg/m², IV) and doxorubicin hydrochloride (30 mg/m², IV) every 3 weeks for 6 treatments. At the last treatment, the dog was doing well; however, the dog was euthanized 10 months later as a result of pulmonary metastasis. Discussion Compartment syndrome develops when interstitial pressure within an osteofascial compartment becomes sufficiently increased to impede tissue perfusion. Prolonged periods of compromised tissue perfusion can lead to neuromuscular ischemia and irreversible tissue damage. A diagnosis of compartment syndrome is determined by recognition of characteristic clinical abnormalities and substantiated by detection of high compartment pressure. Treatment is aimed at rapid surgical decompression to restore effective tissue perfusion. Compartment syndrome was first described in humans. The account was published in 1881 by Dr. Richard von Volkmann, who recognized that placing a bandage too tightly on a forearm could cause blood stasis and ischemia, resulting in muscular damage and subsequent fibrosis. Subsequently, it has been established that any anomaly causing external compression or internal expansion of an osteofascial compartment can increase internal pressure and potentially cause compartment syndrome. Trauma, snake bites, neoplasia, muscular excision, surgery, infiltrative infusions, nephritic syndrome, vascular injury, thermal injuries, and hemophilia have been cited as possible causes of compartment syndrome in people. The mean incidence rate for compartment syndrome in people is reported to be 7.3/100,000 for men and 0.7/100,000 for women, with fractures being the most common cause. Compartment syndrome in dogs has only been reported sporadically, with hemorrhage being the only suspected ascribed cause. Four susceptible osteofascial compartments have been identified in dogs: the femoral compartment, the cranialateral compartment of the crus, the caudal compartment of the crus, and the caudal antebrachial compartment. Despite numerous reported causes of compartment syndrome in people, high intracompartmental pressure secondary to hemorrhage is the only documented cause of compartment syndrome in dogs. The 2 dogs described in the present report are unique in that compartment syndrome developed secondary to an expansile intracompartmental soft tissue tumor. One prior case report describes femoral compartment syndrome secondary to an intramuscular hemangiosarcoma in a dog; however, it is unclear from that report whether the increased compartmental pressure was due to the physical presence of the neoplastic mass or hemorrhage secondary to the neoplasia. Although uncommon, these cases highlight the need for clinicians to be cognizant that compartment syndrome can develop secondary to an underlying malignancy. Both dogs described in this report had forelimb swelling, signs of pain, and lameness, with no history of trauma. The cyclic history of recurring self-limiting signs for the Jack Russell Terrier was most likely attributable to periodic spontaneous degranulation of a mast cell tumor. Degranulation of mast cells leads to the release of inflammatory cytokines, which can cause intermittent swelling and pain. The signs of severe pain and turgid swelling raised concern for possible compartment syndrome in this dog. To our knowledge, there has been only 1 previous report describing an intramuscular mast cell tumor in a dog. Clinicopathologic or imaging findings obtained prior to anesthesia were not helpful in diagnosing compartment syndrome or neoplasia in either dog described in the present report. The Jack Russell Terrier had WBC count and serum enzyme activity abnormalities consistent with inflammation and muscle damage, but the radiographic and ultrasonographic findings were only consistent with generalized soft tissue swelling and did not indicate that the soft tissue swelling was compartmentalized. Contrast-enhanced ultrasonography is somewhat useful in identifying vessel compression, but assessment of such vessels in small dogs, like the 2 described in this report, would likely have been difficult.
Although cytologic evaluation of fine-needle aspirate samples obtained from the affected forelimb regions did not yield evidence of underlying neoplasia in either dog, the findings did establish the presence of sepsis in the affected muscle and adjacent elbow area in the Jack Russell Terrier. It was suspected that the infection was likely the result of hematogenous contamination secondary to the necrotic myositis.

In humans, a tentative diagnosis of compartment syndrome can be made on the basis of clinical abnormalities, including pain on manipulation of involved limb segments, muscle belly tension, and muscle paresis or paralysis.\textsuperscript{2,6,8} In humans and dogs, pulse rate, pulse quality, and body temperatures are typically within reference limits.\textsuperscript{1,2,6,14} Definitive diagnosis can be established by measuring intracompartamental pressure.

In dogs, normal compartmental pressures range from 2 to 8 mm Hg.\textsuperscript{2,6} There are several described invasive methods for obtaining compartmental pressures, such as use of a mercury manometer, an arterial line, a wick catheter, a slit catheter, and a handheld device.\textsuperscript{1,6,8,20} Unfortunately, no veterinary studies have analyzed a specific technique for obtaining intracompartamental pressures, to our knowledge. For both dogs described in the present report, a standard 18- to 20-gauge hypodermic needle was attached to a calibrated invasive pressure transducer via a noncompressible, fluid-filled line. The measurement was then obtained percutaneously after a sterile preparation with the dog anesthetized and in lateral recumbency. Although not done in the cases described in this report, other veterinary reports\textsuperscript{6,16} describe obtaining a mean compartmental pressure from 3 measurements obtained at either the same site\textsuperscript{6} or at 3 locations within the suspect compartment.\textsuperscript{6}

Treatment for compartment syndrome is aimed at rapid decompression of the involved compartment to allow restoration of adequate tissue perfusion. A fasciectomy is typically performed to relieve both circumferential tension and intracompartamental pressure.\textsuperscript{1,6,7,11} Although reapposition of the incised fascia is often not feasible with compartment syndrome, the 2 dogs of the present report had space-occupying lesions that were excised, enabling closure of the fascial layer. In human medicine, there is extensive debate as to when treatment for compartment syndrome is indicated. Some sources\textsuperscript{2,10} support a fasciectomy when the compartmental pressure reaches 30 mm Hg. Others\textsuperscript{12,21} advocate for intervention when the difference between the diastolic arterial blood pressure and the compartmental pressure is $\leq 30$ mm Hg, regardless of whether the compartmental pressure is $> 30$ mm Hg.

Compartmental pressures previously reported for dogs with compartment syndrome range between 25 and 41 mm Hg.\textsuperscript{2,6,13,16} In the 2 dogs described in the present report, we suspect that there was relief of pressure following a fasciectomy, considering that, in both dogs, the intramuscular mass immediately protruded through the fascial incision. Both dogs also markedly improved, with substantially less severe signs of pain, and began to bear weight on the affected limb within 4 days after surgical decompression and removal of affected tissue. Our observations following decompression in the 2 dogs with compartment syndrome are similar to those reported for other dogs\textsuperscript{2,8,13–16} and suggest that surgical intervention in affected dogs should be contemplated when compartmental pressure is lower than the pressure threshold recommended in human medicine.

Ideal treatment for a mast cell tumor involves complete excision of the tumor with a 2-cm margin of normal tissue and 1 adjacent fascia plane.\textsuperscript{17} This was not performed in the Jack Russell Terrier of the present report because there was no evidence of neoplasia prior to the exploratory surgery and compartmental decompression. Moreover, resection with wide margins would have required a forequarter amputation (ie, removal of forelimb and scapula). After a diagnosis of mast cell tumor was made, the option for an amputation of the affected limb was offered but was declined by the owner. During the recheck evaluation 14 days after tumor excision from the right forelimb, physical examination revealed a mildly enlarged right precapsular lymph node. Cytologic evaluation of a fine-needle aspirate from this lymph node revealed low numbers of well-granulated mast cells. Regional lymph node excision was offered for more accurate staging but was also declined. Although marginal excision of the mast cell was not ideal, there was no evidence of local recurrence at the dog’s 6-month recheck evaluation.

Compartment syndrome in dogs has principally been attributed to intracompartamental hemorrhage, although reports are sparse.\textsuperscript{2,8,13–16} The 2 cases described in the present report have established that compartment syndrome can develop secondary to expansile intramuscular tumors in dogs. Although mass excision effectively alleviated the acute clinical abnormalities of compartment syndrome in both affected dogs, clinicians should consider that the long-term prognosis for similar patients ultimately depends on the inciting cause.

References


