FOUR LEG NEWS

MARCH – APRIL 2015 VOLUME 4, ISSUE 2

HI EVERYONE!

Fair warning the information in this newsletter is a bit 'brain taxing'… a bit more of a wrap your head around the articles than my normal selection…

However, the content is good. I chose 3 'outside of the box' topics and two articles on each to share with you:

1) Arteriothrombosis / Thromboembolism

2) Duchenne Muscular Dystrophy

3) Synovial Hemangioma

These are all conditions that I have witnessed in one form or another and I thought that it would be mind-expanding to share with the group. BUT… be awake when you read them or they just might put you to sleep. (Not a good sales pitch, I know… but the information is very important!)

Enjoy the learning!

Cheers,
Laurie

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ARTERIAL THROMBOSIS AFTER VEHICULAR TRAUMA AND HUMERAL FRACTURE IN A DOG.

Objective

This case study describes the treatment of a 3-year old, 19kg mixed breed dog who developed an arterial thrombosis following surgery to repair a midshaft humeral fracture sustained after being hit by a car.

Injuries sustained included pneumothorax, hemothorax, pulmonary contusions, a full-thickness axillary skin wound, and a transverse fracture of the midshaft of the right humerus. The dog was stabilized and supportive care provided in the form of IV fluids, antibiotics, and analgesics. A thoracocentesis was performed and a thoracotomy tube placed. The axillary wound was cleaned and bandaged, with fracture repair (open reduction and internal fixation) and closure of the axillary wound being performed 48 hours after admission.

The dog was discharged 4 days after surgery. Physical therapy instructions included passive range of motion of the elbow and shoulder joints for 10 to 15 minutes 3 times daily with strict exercise restriction. Antibiotics and pain medication were continued at home. On discharge the dog had a weight bearing lameness.

Four days after discharge the dog represented for severe pain and non–weight-bearing lameness. Physical examination found knuckling of the surgical limb. The limb was also cool to the touch compared to the contralateral limb with absent digital pulses. The surgical sites were healing well and radiographs revealed appropriate fracture alignment and apposition. A thromboelastogram revealed mild hypercoagulability, and an arterial thrombus affecting the surgical limb was suspected. Treatment of IV unfractionated heparin was initiated, ultrasonography of the limb and axillary region the next day confirming a thrombus in the right brachial artery with minimal flow to the affected limb. The heparin infusion was continued, and clopidogrel was added. Within 48 hours, pain had subsided and the dog was using the limb with a weight-bearing lameness.

The dog was discharged on oral anticoagulants, antibiotics and pain medications. Five weeks later, ultrasonography showed complete resolution of the thrombus and radiographs showed early bone healing bridging the fracture. Anticoagulant administration was discontinued at this time. Seven months after cessation of anticoagulant administration, the dog had full mobility of the limb and was clinically normal.

Discussion

Hemostatic complications related to trauma are common in humans, thought to be caused by dysfunction of components of Virchow's triad (hypercoagulability, blood stasis, and endothelial injury). Venous thrombi occur in up to 50% of human trauma patients. Arterial thrombosis after trauma is less common but is noted to occur after humeral trauma and fracture repair, especially when fractures are displaced. Potential mechanisms of arterial thromboembolism include direct injury to the artery, the development of an arteriovenous fistula, or endothelial disruption secondary to arterial stretching or spasm.

You are never too old to set another goal or to dream a new dream.
C.S Lewis

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… Arterial Thrombosis contd

Arterial thrombosis has been reported in dogs with a range of diseases. This case, there were no clinical signs that could be attributed to other disease either before the traumatic event or at the 7-month follow-up. Underlying disease contributing to the arterial thrombus formation was considered unlikely. The dog described in this case had a displaced humeral fracture; however, the fracture location made it unlikely that the thrombus was secondary to direct arterial injury from the fracture itself. The thromboelastogram results made it unlikely that a hypercoagulable state was a factor in the development of the arterial thrombus. The degree of blunt trauma and the humeral fracture may have both contributed to shear injury of the axillary vessels, it being possible that changes in blood flow and endothelial injury played a more important role in thrombus formation than did a hypercoagulable state.

In humans anticoagulants are recommended after major trauma or long bone fracture with systemic thrombolytics sometimes administered for rapid reduction of thrombus size and restoration of perfusion. The use of thrombolytics for acute thrombosis is however controversial, with randomized trials documenting medication associated risks of bleeding without sufficient evidence of a clear survival benefit.

In the dog described here, unfractionated heparin administration was initiated to achieve rapid therapeutic anticoagulation and prevent additional thrombus formation. Fibrinolytics were not administered because of the risk of bleeding and that the presence of a thrombus could not be confirmed.

The dog improved rapidly so thrombolytics were not used. Clopidogrel was added to broaden the anticoagulant effect and Dalteparin was substituted for unfractionated heparin allowed for outpatient treatment.

There is no consensus in veterinary medicine regarding which anticoagulants to use to after thrombosis has been confirmed. The multiple anticoagulants in this case were based on clinician preference, however, single-agent therapy might have provided similar results. There is also no consensus on the duration of time required for an artery to heal and to restore endothelial integrity. In this case, given the clinical improvement and resolution of the thrombus at 5 weeks, a decision was made to discontinue anticoagulants.

Arterial thrombosis should be considered a potential complication after severe trauma or humeral fracture and should be suspected in any dog with acute pain after fracture repair.

My thoughts on clinical relevance:
You just have to know that this can happen! If a post-fracture dog comes for rehab but shows clinical signs as described above, ensure that the dog received proper veterinary care as soon as possible.

Source
DePaula KM, deLaforcade AM, King RG, Hughs H, Boudrieau RJ. Arterial Thrombosis after Vehicular Trauma and Humeral Fracture in a Dog. JAVMA, Vol 243, No. 3, August 1, 2013

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CLINICAL AND NEUROLOGICAL CHARACTERISTICS OF AORTIC THROMBOEMBOLISM IN DOGS.

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Dogs feel very strongly that they should always go with you in the car, in case the need should arise for them to bark violently at nothing right in your ear.
Dave Barry

Four days after discharge the dog represented for severe pain and non–weight-bearing lameness. Physical examination found knuckling of the surgical limb. The limb was also cool to the touch compared to the contralateral limb with absent digital pulses. The surgical sites were healing well and radiographs revealed appropriate fracture alignment and apposition. A thromboelastogram revealed mild hypercoagulability, and an arterial thrombus affecting the surgical limb was suspected.

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… Clinical and Neurological Characteristics Contd.

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CANINE MODELS OF DUCHENNE MUSCULAR DYSTROPHY AND THEIR USE IN THERAPEUTIC STRATEGIES.

Duchenne muscular dystrophy (DMD) is an X-linked recessive disorder in which the loss of dystrophin causes progressive degeneration of skeletal and cardiac muscle. Potential therapies such as gene and cell-based approaches, are not without risk and are first tested in animals. Dogs affected by muscular dystrophy are used to study the disease as clinical features may be predictive of disease pathogenesis and treatment outcome in humans.

DMD gene mutations are found in several breeds including Rottweilers, German shorthaired Pointers, Cavalier King Charles Spaniels, and Golden Retrievers. Studies typically involve the form of muscular dystrophy originally characterized in golden retrievers (GRMD).

GRMD studies have included measurements of muscle strength, joint contractures, and timed function tests. Results are used to track disease progression and clinical milestones, such as the loss of ability to walk.

In dogs, signs are observed soon after birth, affected puppies having difficulty suckling and stunted growth. They go on to develop stilted gait, muscle atrophy, excessive drooling, a plantigrade stance, and lumbar kyphosis progressing to lordosis. As with DMD, some muscles hypertrophy. Pharyngeal or esophageal muscle involvement can result in aspiration pneumonia and cardiomyopathy leads to cardiac failure.

The severity of the disease in GRMD affected dogs varies widely, studies looking at clinical signs suggest that homozygous females and smaller dogs have milder signs, supporting a potential influence of either gender or body weight. In DMD treatment trials, reducing growth hormone did not support a relationship with disease severity. Additionally one GRMD study found that lower growth hormone levels tended to correlate with a more severe phenotype, another finding that larger GRMD affected dogs had milder signs at 6 months of age. Measures of weight and joint angles in affected dogs indicate that larger dogs have less severe postural instability and potentially a less severe phenotype.

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… Canine Models of Duchenne Muscular Dystrophy Contd.

Researchers have developed phenotypic tests to characterize disease progression. Comparing serial measurements from treated and untreated groups, improvement or delayed progression of disease can be documented. Functional outcome values have been found to vary considerably, even among dogs from the same litter, suggesting that modifier genes significantly influence phenotype. The effects of phenotypic variation on statistical analysis is offset by obtaining baseline values prior to treatment, each dog thus being its own control.

Composite scores have been used in some preclinical GRMD trials. Postural and gait changes, function (breathing, drooling) and posture have all been assessed and assigned a score based on severity; the scores being added to indicate the degree of dysfunction.

GRMD dogs have a slow, short-stepped, and swaying gait. Accelerometry has been used to characterize this gait, results indicating that total power, stride length and speed, and stride frequency, were significantly decreased, in GRMD versus normal dogs. Subsequent studies showed that gait changes could be identified as early as 2 months of age, dogs walking significantly more slowly and having a more extended stifle and less flexed tibiotarsal joint than normal dogs. A 6-minute-walk test is a standard outcome parameter in DMD patients and has also been used to assess dogs with heart disease. GRMD affected dogs had difficulty completing this test.

At 6 months of age GRMD dogs have abnormally contracted tibiotarsal joint angles, more restricted flexion of the hip joint, increased maximal stifle extension, and more acute tibiotarsal flexion. A cranioventral shift of the pelvis is also seen. It is thought that these joint and postural changes may contribute to the plantigrade stance typically seen in GRMD affected dogs.

Dystrophin serves to buttresses the muscle cell membrane. Without dystrophin, the membrane is prone to tearing with minimal exercise. Dystrophic muscles are particularly susceptible to injury subsequent to eccentric contractions.

Cardiac syndromes are also seen in DMD, the severity varying from subclinical to fatal congestive heart failure. GRMD dogs have ventricular arrhythmias and increased Q/R ratios on ECG evaluation, changes seen at 6 months and progressing with age. There is evidence of ventricular dilation and decreased fractional shortening, and myocardial fibrosis and mineralization is evident as early as 6.5 months of age.

MRI and EMG have been used provide data on the natural history and response to therapy of DMD. MRI changes in DMD include an accumulation of fat in affected muscles and an associated increase in whole body fat and decrease in muscle mass. Electromyography (EMG) in DMD affected individuals is characterized by small, polyphasic, short-duration motor unit potentials (MUPs), with associated spontaneous activity. EMG in GRMD dogs showed spontaneous activity in all dogs, particularly after 10 weeks. Abnormalities were rare at 6 weeks but easily detected at 8 to 10 weeks.

Histopathologic lesions vary among dystrophin-deficient muscles. Fiber size variation occurs due to myofiber hypertrophy and an increase in small regenerating fibers. Necrotic myofibers, in various states of degeneration are also seen.

Not all muscles are affected equally. Like DMD affected individuals, extraoccular muscles are largely spared in GRMD dogs. In contrast, muscles that are used heavily early in life show acute necrosis, others being spared early on but developing lesions later.

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… Canine Models of Duchenne Muscular Dystrophy Contd

Muscles that undergo early necrosis may regenerate and even hypertrophy.

Treatments for DMD are categorized as molecular, cellular, or pharmacologic (Chakkalakal et al 2005; Goyenvalle et al 2011). The most common molecular approaches are *gene therapy* and *gene correction*. Cell-based therapies take normal myoblasts or stem cells and transplant them into diseased muscle. Pharmacologic approaches target specific pathogenetic mechanisms that contribute to the dystrophic phenotype.

Conclusion

GRMD affected dogs develop progressive, fatal disease similar to DMD in humans. To better utilize the GRMD model in therapeutic trials, phenotypic tests have been developed to objectively characterize disease progression. This has set the stage for use of GRMD dogs in preclinical trials and a range of genetic, cellular, and pharmacologic studies, setting the stage for clinical trials in DMD.

My thoughts on clinical relevance:

Muscular Dystrophy has always been a confusing disease for me to 'wrap my head around'. I recall one adolescent (human) patient that came to the clinic (back when I treated humans). He was having soft tissue injury after soft tissue injury… and it didn't make sense. He was later diagnosed with Duchenne Muscular Dystrophy, and I don't know what became of him. In my canine clinic, we've only had one dog with a presumed diagnosis of GRMD, and this article is just my selfish desire to learn more but then to share with you!

Source:

Kornegay JN, Bogan JR, Bogan DJ, Childers, MK, Li J, Nghiem P, Detwiler DA, Larsen CA, Grange RW, Bhavaraju-Sanka RK, Tou S, Keene BP, Howard Jr. JF, Wang J, Fan Z, Schatzberg SJ, Styner MA, Flanigan KM, Xiao X, Hoffman EP. Canine Models of Duchenne Muscular Dystrophy and Their Use in Therapeutic Strategies. Mamm Genome. 2012 February ; 23(0): 85–108.

I ask people why they have deer heads on their walls. They always say because it's such a beautiful animal. There you go. I think my mother is attractive, but I have photographs of her.

Ellen DeGeneres

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MOTOR PHYSICAL THERAPY AFFECTS MUSCLE COLLAGEN TYPE I AND DECREASES GAIT SPEED IN DYSTROPHIN-DEFICIENT DOGS.

Introduction:
GRMD (Golden Retriever Muscular Dystrophy) has muscle abnormalities close to the ones seen in humans (increased creatine kinase activity, muscle hypotrophy, contractures, degeneration, endomysial and perimysial fibrosis). Also seen are cycles of muscular necrosis and regeneration, muscle wasting, postural abnormalities and respiratory or heart failure, as seen in DMD patients.

Physical therapy (PT) is one of the supportive therapies available for DMD, however there is no consensus regarding the type and intensity of physical therapy. This study looked at the effect of physical therapy on gait biomechanics and muscular collagen deposition in dystrophin-deficient dogs.

Method
Four dystrophic dogs were selected from a GRMD kennel in Brazil. Two of the dystrophic dogs underwent a PT protocol of active walking exercise while the other two control dogs maintained their routine of activities of daily living. Pre and post physical therapy, collagen type I and III were analyzed using immunohistochemistry and gait biomechanics.

Physical therapy sessions were 3 times a week, with a rest day between sessions. Sessions ran for 40 minutes over a 12 week period. PT was conducted within a 36 x 5 m arena and consisted of walking 8 times around the arena at a speed at least 10% more than their walking speed. Velocity was recorded using a chronometer and dogs were allowed to rest between each circuit.

Pre and post PT muscle fragments were collected from biceps femoralis of each dog for morphological analysis. Video records of the gait and Ground Reaction Forces (GRF) were maintained.

Discussion
Even with intervals of rest to avoid fatigue between days and during PT, decreased gait velocity and an increase in the amount of type I collagen was found. There was no influence on angular displacement or GRF. In contrast, control dogs who maintained their usual daily activities did not show any muscular damage.

Fibrosis in dystrophic muscle is poorly understood, but appears to be the consequence of inflammatory infiltrate from muscular damage. This study showed that skeletal muscle of GRMD animals had increased muscular fibrosis deposition prior to PT when compared to skeletal muscle of healthy dogs. The PT protocol used in this study influenced type I collagen, treated dogs having increased muscle damage, suggesting a functional loss of these muscles.

Kinematics and gait dynamics in both study and control dogs showed a gait velocity lower than healthy dogs, the treatment group being slower than the control group, possibly a result of fatigue. PT was found to play an important role decreasing walking speed and in functional muscle deterioration.

Significant changes were found in elbow, hip and stifle joints, however PT was not found to have been a factor, the changes instead reflecting the natural progression of the disease. Shoulder, carpal and tarsal joints also changed with changes being individual to each dog and not related to PT.

Propulsion was almost absent in the study animals, suggesting a loss of limb strength in these limbs. GRMD is seen to be almost absent by 5 months of age, a finding supported by this study and again not related to PT.

Contd overleaf …

… Motor Physical Therapy Contd

Conclusion:
DMD is a progressive disease presenting early in life. Physical therapy can increase quality of life, helping maintain muscle flexibility and maintaining activities of daily life. Using PT to improve motor function however requires further investigation, given this study found a negative effect on gait speed and muscular of collagen in GRMD dogs. The authors recommend DMD patients be encouraged to perform independent daily activities supported by environmental adaptations, but that individual limitations should be respected.

Physiotherapists also must know how to find a balance between the preservation of motor function of patients while minimizing muscle damage.

My thoughts on clinical relevance:
This study is rather disconcerting. However, it also confirms what I witnessed – nothing seemed to help the young man with muscular dystrophy from back when I was a new grad PT… but then again, he wasn't diagnosed when we were treating him. The conclusion is most important in this study, working on activities of daily living is likely the most important part for disease management, and pushing for 'progress' will likely be deleterious in these cases.

Source:
Gaiad TP, Araujo KPC, Serrao JC, Miglino MA, Ambrosio CE. Motor Physical Therapy Affects Muscle Collagen Type I and Decreases Gait Speed in Dystrophin-Deficient Dogs. PLoS ONE 9(4): e93500.

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SYNOVIAL HEMANGIOMA IN A DOG

Introduction
In humans, knee arthroscopy allows minimally invasive evaluation and treatment of synovial hemangioma. This report describes the use of diagnostic arthroscopy and arthrotomy for surgical resection of a synovial hemangioma in the stifle joint of a dog

The dog, an 8-year-old male neutered Standard Poodle presented for evaluation of chronic, recurrent left pelvic limb lameness unresponsive to NSAIDS. There was no history of trauma or previous disease. On examination marked quadriceps and biceps femoris muscle atrophy and weight-bearing lameness of the left pelvic limb were apparent. Left stifle joint manipulation and palpation was painful and there was a slight cranial drawer sign.

Synovial fluid aspiration confirmed hemarthrosis of the stifle. Radiographs of the left stifle showed a well-defined, circular, soft tissue mass on the proximocaudal aspects of the infrapatellar fat pad

Arthroscopy was performed, finding a well-defined, pedunculated, cherry red, villonodular synovial mass in the intercondylar notch, extending behind the intermeniscal ligament. Pressure from the mass had stretched the soft tissues including the CCL. The mass was removed via an arthrotomy lateral to the patellar ligament. The mass was determined to be a benign cavernous synovial hemangioma

The dog was discharged home with instructions for physical therapy and rehabilitation without use of ultrasound, thermotherapy, or any physical agents that could cause hyperemia.

Rechecks were at 10 day intervals for 1 month and then monthly. Recovery was rapid and at the initial 10-day recheck the dog had a near normal gait with no signs of pain. Increased limb use reversed muscle atrophy, and at 20 days, cranial drawer was almost absent. By 40 weeks gait was normal, there was no joint pain, and no hemarthrosis. Radiographs of the left stifle showed no degenerative changes or evidence of recurrence.

Discussion
Arthroscopic and histologic features of this synovial hemangioma are similar to those reported in human synovial hemangiomas. Complete resolution is usually achieved with complete resection.

Likely causes of chronic recurrent lameness unresponsive to anti-inflammatories and associated with hemarthrosis without associated trauma origin, and having a well-defined soft tissue lesion evident on radiographs include fibrosarcoma, rhabdomyosarcoma, osteosarcoma, malignant fibrous histiocytoma , liposarcoma, hemangiosarcoma, and undifferentiated sarcoma - the more common canine articular tumors.

Arthroscopic exploration in this dog clarified diagnostic findings and directed arthrotomy over amputation for the removal of this well-defined mass.

Contd overleaf …

… Synovial Hemangioma Contd.

Synovial hemangioma is rare in dogs, however based on experience in this case and in another reported in humans, they can likely be resolved by complete surgical excision

My thoughts on clinical relevance:
This is an important report for two reasons: 1) to be aware of this pathology (and others) and be aware that severe atrophy is not normal with cruciate strains… the rehab practitioner may need to advocate for advanced diagnostics in cases of suspected partial CCL tears where atrophy is beyond expectation; 2) to make note of the post-operative recommendations in this case.

Source: Arias JI, Torres C, Saez D. Synovial Hemangioma in a Dog. Veterinary Surgery 38:463–466, 2009

SYNOVIAL HEMANGIOMA IN THE STIFLE JOINT OF A DOG

This study takes the form of a case study involving synovial hemangioma in the stifle joint of a dog. Vascular tumors are common in the skin and subcutis of dogs but not in the canine synovium. In humans, synovial hemangioma is defined as a benign vascular proliferation or hamartoma arising in articular or bursal synovial membrane.

The dog, a 28kg, 8-year-old, male neutered Belgian Sheepdog presented for left hind limb lameness of 3 months' duration. Soft-tissue swelling was noted around the left stifle, aspirated synovial fluid being hemorrhagic. There was no cranial drawer movement of the stifle. Radiographs showed a soft tissue swelling in the cranial and medial aspect of the stifle, displacing the infrapatellar fat pad. There were no bony changes in the stifles or coxofemoral joints and thoracic radiographs were within normal limits.

Surgical exploration of the left stifle was performed, findings including hemarthrosis and several reddish-purple nodular nodular masses attached by stalks to the synovial membrane. Nodules that were accessible were removed and sent for histological analysis.

A diagnosis of hemangiosarcoma, based upon the presence of focal papillary endothelial hyperplasia and nuclear atypia was considered, although no tumors were seen in the heart or abdomen on ultrasound. The affected limb was amputated 1 week after the initial biopsy and dissected to determine the extent of the tumor.

Endothelial atypia without invasiveness has been seen in human synovial hemangiomas and does not warrant a diagnosis of angiosarcoma. The tumor in this dog was well differentiated without atypia or multi layering of endothelial cells. The tumor was also within the synovial membrane, with only limited invasion of the joint capsule. Findings were thus more consistent with hemangioma or vascular hamartoma than with hemangiosarcoma.

The dog recovered without complications and physical examination, abdominal ultrasound, and 3-view thoracic radiographs 6 months later, revealed no evidence of recurrence or spread of the tumor.

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… Synovial Joint Hemangioma Contd

Synovial hemangioma is a benign proliferation of blood vessels within the synovium of a joint or bursa. It is classified as a deep hemangioma, usually involving skeletal muscle, less commonly, synovium, bone, nerve, or lymph node. Since the first description in 1856, fewer than 200 cases of human synovial hemangioma have been reported

One retrospective study involving 20 cases found, the knee joint involved in 12 cases. Thirteen patients were male with age at presentation ranging from 9 to 49 years. Symptoms were usually pain and swelling and onset usually during adolescence, only occasionally reports in older adults.

Synovial hemangioma or vascular hamartoma is seen in carpal and digital tendon sheaths of young horses, usually presenting by 2 or 3 years of age. It is a benign proliferation that may recur after incomplete excision but does not have histologic atypia and does not metastasize. Hemangioma of tendon sheaths or articular synovium is not recognized in dogs who are not prone to developing juvenile hemangiomas or hamartomas

A vascular tumor should be a differential diagnosis for any dog with non-traumatic hemarthrosis and red synovial nodules.

My thoughts on clinical relevance:
Again, it's just important to know of this condition!

Source:
Miller MA, Pool RR, Coolman BR. Synovial Hemangioma in the Stifle Joint of a Dog. Vet Pathol 44:240–243 (2007)