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Use of adjunctive prednisolone in the management of a cat with bilateral quadriceps contracture following trauma

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Abstract

Case summary A 6-month-old cat was successfully treated for bilateral quadriceps contracture. Conventional treatments including surgery, dynamic flexion apparatus and physical therapy along with analgesics and non-steroidal anti-inflammatory drugs resulted in temporary clinical improvement that was relapsing. The initiation of supplementary corticosteroid treatment with prednisolone coincided with an immediate and sustained clinical improvement and long-term resolution.

Relevance and novel information Successful treatment of bilateral quadriceps contracture has not previously been reported in a cat. Quadriceps contracture remains a challenging condition to treat with some cases unresponsive to therapy. Systemic prednisolone treatment appeared to be of benefit in the management of this case and may have a role in some cats where muscle contracture appears relapsing in nature. Further prospective investigations in cats with muscle contracture, including muscle biopsies of affected cats, are warranted.

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Case description

A 6-month-old prepubertal entire domestic shorthair cat presented with bilateral closed femoral fractures due to unobserved trauma that had occurred up to 4 days prior. The cat had indoor/outdoor access and road trauma or a fall was considered likely. Radiographs revealed Salter Harris type-1 physeal fracture in the right distal femur, left distal femoral diaphyseal fracture and fracture of the left femoral neck. Surgical stabilisation with internal fixation was performed. The right limb was treated by cross pins in the distal femur. The left femoral fracture was repaired with a laterally applied 1.5/2.0 veterinary cuttable bone plate and screws, and left femoral head and neck osteotomy was performed. The cat was discharged on meloxicam (0.05 mg/kg orally q24h) and with instructions for 6 weeks of strict cage-style rest. The owner was encouraged to perform passive range-of-motion exercises of the stifles and left hip and allow supervised walking exercise in the house. Effective physical therapy was not performed owing to the cat's demeanour.

Two weeks postoperatively the cat showed good clinical recovery and was walking well with mild bilateral lameness. Periarticular thickening of the right stifle joint and mildly reduced joint flexion were noted, but no comment regarding left stifle range of motion was recorded. A course of pentosan polysulfate injections (3 mg/kg SC, weekly for 4 weeks) was initiated. At 6 weeks postoperatively, the owner reported the cat had developed worsening left hindlimb lameness and difficulty using the litter tray over several weeks. On examination, there was weightbearing left pelvic limb lameness with stiff

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hindlimb gait. The left stifle joint was fixed in mild extension, with minimal flexion possible even under anaesthesia. There was a large firm fracture callous palpable and the distal left quadriceps muscle was firm, non-painful and fixed distally to the femur. Proximally, the quadriceps muscle was soft with normal consistency. Radiographs showed stable implants and advanced fracture healing with marked periosteal callous formation. A diagnosis of left quadriceps contracture was made. The right stifle and both hips demonstrated an acceptable range of motion and comfort on palpation at that time.

Surgery was performed, including removal of the bone plate and screws, mobilisation of adhesions between the quadriceps and femur with placement of a free fat graft between the bone and muscle and attachment of a dynamic flexion apparatus.[1] The proximal apparatus was created by inserting three threaded mini-external fixation pins in the caudal aspect of the left ischium that were incorporated into a blob of hardware acrylic cement (Knead-it; Selleys) in which a metal hook was also embedded. The distal apparatus was a single circular external fixator ring block secured in the distal tibia with two crossed wires. The proximal and distal elements were connected via elastic bands to maintain stifle flexion. Normal range of stifle flexion was possible immediately after surgery. The cat was discharged to the owner with ongoing oral meloxicam and oral buprenorphine (0.01 mg/kg orally q12h). The stifle was maintained in a flexed position when the cat was confined, but the apparatus was unhooked when it was possible to have supervised house exercise, to encourage walking.

In a follow-up examination 2 weeks later (8 weeks post-fracture repair), the left stifle demonstrated near normal range of motion in flexion. Mild wire tract discharge was present distally and there was loosening of the ischial attachment so the flexion apparatus was removed. Decreased flexion (90 degrees) was noted in the right stifle at that time consistent with developing quadriceps contracture on the contralateral side. Physical therapy was initiated in hospital in an attempt to prevent progression of contracture. This comprised passive range of motion exercises of both stifles and hips and muscle stretching performed in 10–15 min sessions twice daily under anaesthesia, owing to patient demeanor.[2] After 1 week the right hindlimb showed no persistent improvement in stifle range of motion. When physical therapy was not performed for over 12 h there was notable decrease in stifle range of motion.

Surgical intervention on right hindlimb was performed as described for the left side, although implants were embedded in bone and were not recovered. The cat remained hospitalised or was treated as an outpatient daily over this period with ongoing meloxicam and buprenorphine, and once- or twice-daily physical therapy on both hindlimbs. The flexion apparatus was connected with the right stifle joint in full flexion while the cat rested, but was released at other times to promote walking and exercise. The fixator on the right side loosened at the site of the ischial pins and was removed after 2 weeks. After removal of the dynamic fixation device daily physical therapy was continued. Despite physical therapy, the range of movement in the left hindlimb became progressively restricted to approximately 50 degrees of motion, and in the right limb contracture had worsened to 60–70 degrees of movement. Surgery was then repeated on the left side to free the reformed adhesions, and reattach a dynamic flexion apparatus (12 weeks after fracture repair). The apparatus was also maintained for 1 week until it loosened. Daily physical therapy was continued after fixator removal; however, this was not adequate to maintain range of motion, even while hospitalised. Meloxicam was withdrawn and following a washout period of 48 h prednisolone treatment was initiated empirically in an attempt to limit reoccurrence of fibrosis and formation of adhesions. Prednisolone therapy commenced approximately 13 weeks post-fracture repair, initially at 1.85 mg/kg orally q12h for 3 days then tapered over time. By day 10 the cat was on 0.93 mg/kg prednisolone q48h this was continued for 8 weeks in total. No further physical therapy was performed.

Within 3 days of initiating prednisolone treatment, the cat showed both a lack of worsening and significant improvement in range of motion in both hindlimbs, and after 4 weeks of treatment, the left stifle had a normal range of movement, and the right was only slightly decreased. The cat was continued on 0.93 mg/kg q48h for 4 more weeks. Two weeks after prednisolone treatment ended, and approximately 23 weeks post-fracture repair, a follow-up evaluation was performed. The cat had maintained good range of motion in both hindlimbs, and contracture had not reoccurred. Fifteen months later the cat was represented for examination and radiographs. The owner reported that the cat had continued to improve over time, becoming more adventurous and mobile particularly during jumping. Occasional self-limiting right hindlimb lameness was noted. On palpation both quadriceps muscles felt soft, compliant and non-painful, and near-normal stifle joint range of motion had been maintained. Mild right stifle crepitus was noted and radiographs confirmed mild stifle osteoarthritis; however, the abnormal periosteal callus associated with the femurs had remodelled.

Discussion

Muscle contracture is abnormal shortening of skeletal muscle and loss of ability to stretch due to pathological changes in muscle fibres or support tissues.[3] As in this case, contractures are usually secondary to bone fractures and soft tissue trauma.[4,5] They may also be associated with other non-traumatic injury triggers, including

immune-mediated myositis, neuromuscular disease,[6] protozoal infection (neospora or toxoplasma), neoplasia and ischaemia.[5,7] In cats, acquired muscle contracture is uncommon but is sporadically reported in a variety of muscles, including the semitendinosus,[5] brachialis muscle,[5] digital flexors of the forelimbs,[8–10] and, most commonly, the quadriceps muscles.[1,4,5,11–13] Quadriceps contracture most frequently occurs in young animals subsequent to femoral diaphyseal fractures with fixation, or following immobilisation of the limb in extension.[4] Risk factors for quadriceps contracture in cats include young age, major trauma with severe or multiple injuries, exuberant callous formation, and inadequate or delayed surgical repair or prolonged postoperative disuse.[4,5] The prognosis for restoring normal limb function with chronic quadriceps contracture is considered guarded or poor,[5] but in the earlier stages treatment to restore stifle range of motion may be successful.[1,12,13] Prevention is optimal with active physical therapy in at-risk patients advised.[5] The presence of bilateral muscle contracture may not affect the prognosis if appropriate treatment is given,[14,15] however, no previous accounts of successful treatment of bilateral quadriceps contracture in cats were identified.

Initial treatment in this cat was based upon published recommendations and included physical therapy, surgical mobilisation, use of a free fat graft to try and limit adhesion formation and placement of a dynamic flexion apparatus across the stifle.[1,5,12] This treatment did not prevent recurring contracture in this patient and several factors may have contributed to this outcome. The flexion apparatus was maintained for limited periods (2 weeks at a time) before it loosened as a result of limited bone purchase in the ischium; this was a slightly shorter time frame than in similar cases reported in dogs (4 weeks)[1] and cats (3 weeks)[12] that were successfully treated, and may have affected treatment outcome. Ischial pin loosening led to removal of the apparatus in each case and that may have been minimised with a different surgical construct in the pelvis. Physical therapy in cats needs to be tailored to the behavioural peculiarities of the species,[16] and was poorly tolerated in this patient, often requiring anaesthesia. This delayed initiation of physical therapy after surgery reduced the frequency of treatment and may have reduced treatment efficacy.[2] More proactive early physical therapy or involvement of a qualified therapist, if available, may have prevented development or progression of contracture. Non-steroidal anti-inflammatory drugs and analgesia with buprenorphine was administered to try and facilitate limb use and mobilisation, as is widely recommended during management and rehabilitation of muscle contractures in dogs and cats.[12,17] In this case, conventional treatments resulted in initial improvement, followed by recurrence of contracture. Initiating treatment with the corticosteroid prednisolone coincided with an almost immediate improvement in clinical state and stopped the apparent rapid tendency for contracture to recur. Continued treatment with prednisolone was associated with a full recovery with no need for additional surgical treatments or physical therapy. Fifteen months on from the end of treatment, contracture has not recurred, and the cat is considered normal with occasional lameness only.

The role that prednisolone played in the successful treatment of this cat remains speculative, but the use of steroids coincided with a marked change in the clinical course of the patient that continued over time. Various potential mechanisms of action for corticosteroids exist to modify the outcome of muscle contracture and more than one mechanism may be involved. Prednisolone is an anti-inflammatory drug, which, like all corticosteroids, achieves its biological effects by binding to and inhibiting proinflammatory factors, while also up-regulating anti-inflammatory factors.[18] Specifically, it binds to and activates glucocorticoid receptors, which cause increased transcription and translation of anti-inflammatory proteins, such as interleukin-10, lipocortin-1, interleukin 1 receptor antagonist and neutral endopeptidase. Furthermore, activated glucocorticoid receptors have a direct inhibitory effect on activated transcription factors, including nuclear factor kappa B and activator protein-1, regulating inflammatory protein expression.[18] Steroids inhibit all wound-healing processes and are broad-spectrum antifibrotics, downregulating cytokines that lead to fibrosis, including transforming growth factor-beta1 and fibroblast growth factor,[18] and decreasing collagen synthesis by inhibiting prolyl hydroxylase and amplifying collagenolysis.[19]

Successful treatment with corticosteroids administered both systemically and/or intralesionally is reported in other fibrosing conditions,[20,21] including oesophageal, urethral and colonorectal strictures, and for hepatic cirrhosis in humans.[22] Steroids are often used in treatment for similar conditions in dogs and cats, although limited published evidence of efficacy is available.[23,24] The potential utility of corticosteroids specifically on muscular contractures are not well documented in the literature in any species. Steroids could also work by other mechanisms. They have a demonstrated beneficial effect on the clinical course of muscle dysfunction due to Duchenne muscular dystrophy in boys, including delaying onset of scoliosis and contractures, although the mechanism of this action is uncertain.[25,26] In this disease, corticosteroids may potentially enhance myoblast proliferation and promote muscle regeneration and may inhibit muscle deterioration by a membrane stabilising effect or inhibiting lysosomal-bound proteases.[26] Improved muscle function is also apparent in dogs, with an analogous disease known as Golden Retriever muscular dystrophy that was treated

with chronic oral prednisone.[27] Flexion contractures in humans may rarely be the sentinel sign for hypoadrenocorticism, resulting in a condition known as 'stiff man' syndrome, which rapidly responds to glucocorticoid treatment.[28] The pathophysiology of this condition is unknown,[28] and while there is no suggestion the cat described herein was hypoadrenocorticoid there may be another specific effect of cortisol on muscle that inhibits contracture.

Histopathology of muscle is rarely performed in cats or dogs affected with quadriceps contracture, presumably because there is a clear traumatic cause evident in most cases. For this reason the pathological features of quadriceps muscle contracture specific to cats are not known. Histological changes may also vary over the time course of muscle injury from the acute to the recovery phase. Histological findings are reported in other forms of muscle contracture in dogs, and fibrosis with muscle fibre atrophy and fibre size variation were the predominant findings with minimal inflammatory changes.[29,30] There may, however, be species differences in these findings. Histological findings in a single cat with idiopathic digital flexor tendon contracture identified fibrosing myofascitis; however, the diagnosis was made post mortem and no specific anti-inflammatory treatment was provided.[10] In tissue from another cat with brachialis muscle contracture, mild inflammatory infiltrates, oedema and fibrosis were reported in the muscle and tendon with no infectious agents seen. When contracture reoccurred after 8 months, further tissue biopsy showed bundles of fibroblasts and collagen with a similar mild-to-moderate mixed inflammatory infiltrate.[5] This cat was given a single intramuscular injection of triamcinolone, at a dose of 2.2 mg/kg, purportedly to help prevent further adhesions, and at 5 month follow-up good recovery with no contracture was reported.[5]

Steroid treatment may have some potential disadvantages in managing animals with muscle contracture, particularly quadriceps contracture. Glucocorticoids have many potential side effects, including delayed wound and bone healing, osteoporosis, altered growth, muscle weakness and atrophy, and increased infection risk.[31,32] This would make treatment undesirable in juvenile animals and those recently subjected to major trauma or surgery. Clinical signs of iatrogenic hyperadrenocorticism may develop,[32] although cats appear relatively resistant to these side effects. Further investigation of the role that steroid treatment may play in management of muscle contractures, particularly in the cat, are warranted. Ideally, this should include muscle biopsy of clinical cases to determine whether there is a significant inflammatory component to muscle injury in this species. Steroid treatment could be considered as an adjunct to conventional multimodal surgical, and physical therapy in patients where clinical improvement is not progressive with conventional approaches.

Conclusions

Bilateral quadriceps contracture can be successfully managed in cats. Conventional treatment with early intervention, surgical mobilisation, physical therapy, analgesia and maintaining joint flexion remains the most appropriate initial therapy based on current evidence. Prednisolone therapy appeared to be of benefit in this case, which had been refractory to conventional treatments. Muscle biopsies should be considered in cats with muscle contractures at this or any site to determine the nature of pathological changes and whether there are inflammatory lesions.

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