Surgical management of bilateral semitendinosus fibrotic myopathy and cranial cruciate ligament disease in a German Shepherd Dog

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CASE REPORT Surgical management is traditionally not recommended for semitendinosus fibrotic myopathy because of the high recurrence rate. A 2-year-old German Shepherd Dog was initially presented for an abnormal bilateral pelvic limb gait. On physical examination, palpably thickened fibrotic bands along the thigh musculature and restricted stifle range of motion were identified bilaterally. A clinical diagnosis of bilateral semitendinosus fibrotic myopathy was based on the distinctive gait anomaly. Conservative management with physiotherapy and non-steroidal anti-inflammatory medications was recommended. The patient was re-presented 3 and 5 years later for left and right cranial cruciate disease, respectively, with no improvement in the gait abnormality. Medial crural fasciotomy and modified triple tibial osteotomy were performed at each time point. The former involving releasing the extensive crural fascial insertion on the tibia. Long-term follow-up 29 and 15 months postoperatively for the respective limbs showed continued resolution of the abnormal gait with only mild restriction in stifle extension.

CONCLUSION Effects of surgical intervention may not be as short-lived as previously described, with medial crural fasciotomy considered as a potential treatment option. Further investigation of the effect of cranial cruciate ligament disease and its implications on the pathogenesis of semitendinosus fibrotic myopathy may also be warranted.

KEYWORDS contracture; cranial cruciate ligament; crural fascia; dogs; fasciotomy; fibrotic myopathy; semitendinosus m.

ABBREVIATIONS CrCL, cranial cruciate ligament; GSD, German Shepherd Dog; LPL, left pelvic limb; ROM, range of motion; RPL, right pelvic limb; TPA, tibial plateau angle; TPLO, tibial plateau levelling osteotomy; TTO, triple tibial osteotomy

Fibrotic myopathy is an abnormal pathological process in which the myotendinous unit becomes irreversibly replaced by fibrous connective tissue, resulting in contracture and permanent dysfunction of the muscle.1,2 Multiple hypotheses regarding the pathogenesis of fibrotic myopathy have been postulated, including direct musculoskeletal trauma, intramuscular injections, development of compartment syndrome, recurring strain, ischaemia, infection, immune-mediated disease or neoplasia.1-3 However, the exact causation remains unclear and may differ according to anatomical location.1 Quadriceps4 and infraspinatus5,5 contracture following musculoskeletal trauma has been frequently reported in the dog. Less commonly, fibrotic replacement of the semitendinosus, 6-8 gracilis, 9-10 supraspinatus, 1 teres minor, 1 iliopsoas, 11-12 sartorius1 and tensor fascia lata1 have been described. The resulting debilitation is reportedly worse when affected muscles span multiple joints.12-14

Semitendinosus fibrotic myopathy is overrepresented in active young male German Shepherd Dogs (GSDs).5-8,13-17 This condition can occur alone or concurrently with gracilis muscle involvement.5,8,10,17 In 61% of cases there is bilateral involvement.12,16 A genetic component is suspected, but a 4-generation pedigree analysis of six dogs with fibrotic myopathy revealed no common ancestry.5 Chronic repetitive strain is speculated to be the most likely cause. Steiss hypothesised that the increased stifle flexion angles associated with the conformation of GSDs place greater stress on muscles during physical activity compared with other breeds.3 Chronic stretching and tearing of the highly vascularised muscles result in local haemorrhage and swelling, which compounds a localised compartment syndrome.5,11,13 Because of the limited regeneration of myofibrils, severely damaged muscles are consequently replaced by dense collagenous connective tissue.11,17,18 The clinical diagnosis of semitendinosus fibrotic myopathy is often based on observation of a distinctive gait abnormality where there is reduced stride length with internal stifle rotation, external hock rotation and rapid internal rotation of the foot during the mid-to-late swing phase.6-8,15-17 Reduced passive and active stifle extension with a characteristic fibrotic thickening in the region of the semitendinosus muscle can be appreciated on physical examination.5-8,13-17

Many surgical treatments for this condition have been attempted; these include myotomy, myectomy and use of autogenous fat graft incorporation or hyaluronate injection into the resection site,13,15 often resulting in a short-lived resolution of the clinical signs with the gait anomaly recurring within weeks to months in all previous reports.5-8 A Z-plasty lengthening procedure was described in an affected cat, which similarly resulted in recurrence within 2 weeks.19 Interestingly, semitendinosus tenotomy in horses affected by fibrotic myopathy has been shown to be effective, with all cases returning to athletic function.20,21 Because of the poor reported response, however, surgical management of this condition is generally not recommended for canine patients.13

Interestingly, the involvement of the crural fascia has never been described as part of the pathogenesis or treatment of fibrotic myopathy, despite its close association with the gracilis and
Bilateral semitendinosus muscles. The crural fascia is a distal continuation of the femoral and stifle fascia and comprises a superficial and deep leaf. The crural fascia could be beneficial in the management of patients afflicted with semitendinosus fibrotic myopathy. This case report details the clinical features and outcomes following treatment of bilateral semitendinosus fibrotic myopathy with concurrent cranial cruciate ligament (CrCL) disease in a GSD.

CASE REPORT

A 2-year-old male neutered GSD was initially referred for assessment of abnormal pelvic limb gait. The dog was very active and would commonly chase balls for extended periods. He had developed an unusual gait over the previous 3 months and the owners became concerned when he had difficulty jumping into the back of the car. There was no known history of trauma.

On physical examination, the dog had a crouched stance and prominent gait anomaly. During the swing phase of locomotion, both pelvic limbs circumducted with internal rotation of the stifle, external rotation of the hock and a distinctive ‘medial flick’ of the pes tracking towards the midline (Video 1). A shortened cranial stride length was also noted bilaterally. These were more pronounced in the right pelvic limb (RPL) than the left. On physical examination, a taut fibrotic band was palpable within the caudomedial aspect of the thigh in the region of the semitendinosus muscle bilaterally. Stifle extension, hip extension and abduction were restricted. No discomfort was elicited on direct palpation of the thickened fibrotic bands. No abnormalities were identified on neurological examination or in the remainder of the clinical examination.

Survey radiographs of the stifles, hips and lumbar region were performed. Increased soft tissue radio-opacity associated with the fibrotic bands was evident (Figure 1). A shortened cranial stride length was also noted bilaterally. These were more pronounced in the right pelvic limb (RPL) than the left. On physical examination, a taut fibrotic band was palpable within the caudomedial aspect of the thigh in the region of the semitendinosus muscle bilaterally. Stifle extension, hip extension and abduction were restricted. No discomfort was elicited on direct palpation of the thickened fibrotic bands. No abnormalities were identified on neurological examination or in the remainder of the clinical examination.

Survey radiographs of the stifles, hips and lumbar region were performed. Increased soft tissue radio-opacity associated with the fibrotic bands was evident (Figure 1). Ventral bridging spondylosis of the L2–L3 region and moderate joint incongruency in the right coxofemoral joint with mild osteophytosis of the acetabular margin were also present.

FIGURE 1. Mediolateral radiograph showing increased soft tissue opacity of the semitendinosus fibrous band (arrows). Based on the signalment, clinical findings and characteristic gait, a clinical diagnosis of bilateral semitendinosus fibrotic myopathy with concurrent mild right coxofemoral osteoarthritis associated with hip dysplasia was made. The dog was managed conservatively with a 2-week course of robenacoxib (1 mg/kg once daily; Onsior®, Elanco) and physiotherapy was recommended.

The patient re-presented 3 years later for progressive left pelvic limb (LPL) lameness. The dog had continued to run regularly and tolerated well the abnormal mechanical lameness over the preceding 3 years. However, he was noted to have difficulty rising and had been increasingly lame on the LPL over a 4-week period. Lameness worsened after exercise. Gait examination revealed a similar short-stepping crouched gait as previously described, but a grade 3/5 LPL lameness was also apparent. On examination, moderate muscle atrophy was appreciated in the thigh musculature bilaterally. The fibrotic bands were now approximately 2 cm in diameter and extended from the lateral ischiatic tuberosity to the craniomedial border of the proximal tibia. Mild discomfort was also noted on direct palpation of the fibrotic bands. Palpation of the left stifle revealed presence of a medial buttress and significant joint effusion.

Baseline complete blood count and biochemistry revealed no abnormalities. The dog was anaesthetised for radiography and a repeat orthopaedic examination. Marked osteophytosis along the femoral trochlear ridge and patella was present in the left stifle, with obliteration of the infrapatellar fat pad and caudal displacement of the fascial planes indicative of severe joint effusion. Mild reduction in the fat pad and mild osteophytosis were also evident in the right stifle. The tibial plateau angle (TPA) was measured as 27° for both stifles. Although fibrotic restriction of the stifle’s range of motion (ROM) made assessment difficult, a positive cranial drawer sign and tibial compression test were noted on manipulation of the left stifle and a mild drawer movement in the right stifle. Bilateral CrCL disease was diagnosed, with the right side likely to be an early partial tear.

The dog was admitted for a left tibial plateau levelling procedure and medial crural fasciotomy to mitigate the effects of the fibrotic myopathy. The patient was positioned in dorsal recumbency and a left craniomedial arthroscopy was performed.21 A completely ruptured CrCL was identified and debrided. A partial meniscectomy was performed following identification of a bucket-handle tear in the caudal horn of the medial meniscus. The joint capsule was lavaged and closed with 2-0 polydioxanone (PDS II, Ethicon) using a simple continuous suture pattern. Exposure for the medial crural fasciotomy was undertaken via medial approach to the tibia with an extension of the previous skin incision down to the level of the distal tibial metaphysis.23 Subcutaneous tissue was incised and the medial crural fascia was identified (Figure 2). The crural fascia was grossly thickened with two major fibrotic bands appreciable within: the first inserted on the cranial margin of the tibia and extended down the proximal tibial diaphysis while the second smaller band inserted down the caudomedial tibial diaphysis distally (Figure 3). The crural fasciotomy incision was made from the cranial edge of the caudal sartorius and continued distally along the medial tibia. The thickened fascial insertions on the tibial crest and along the distal shaft of the tibia were released, which provided an immediate and dramatic increase in stifle extension and pelvic limb abduction (Figure 4). A modified triple tibial osteotomy (TTO) procedure described by Smith22 was performed for treatment of the CrCL disease. This entails the use of three linear osteotomies: the first being a complete osteotomy of the tibial crest with the second and third tibial osteotomies completing a closing wedge resection of the proximal tibia. A 3.5-mm locking TPLO plate was used to stabilise the tibial osteotomy. The tibial crest osteotomy was secured with two 1.6-mm Kirschner wires and tension-band...
fixation using 16G orthopaedic wire. No attempt was made to close the fascial layer. The subcutaneous layer was closed with a simple continuous pattern using 2-0 polydioxanone and the skin layer was apposed with an intradermal pattern using 3-0 poliglecaprone 25 (Monocryl, Ethicon). The postoperative TPA was measured as 6°. A modified Robert-Jones bandage was placed postoperatively and the dog recovered from anaesthesia without complications. He was managed in hospital with a continuous-rate infusion of morphine (0.05–0.2 mg/kg/h; DBL Morphine Sulfate Injection BP, Hospira) and was discharged with a 10-day course of firocoxib (5 mg/kg once daily; Previcox, Merial) and tramadol hydrochloride (3.5 mg/kg twice daily; Tramadol Sandoz SR, Sandoz). A structured physiotherapy program was recommended but was not complied with because of the dog’s temperament. Six weeks’ of restricted activity was advised and passive ROM exercises recommended three times daily.

Examination at the 2-week postoperative recheck showed a grade 2/5 LPL lameness, but the gait abnormality had resolved with normal ROM present within the left stifle. A moderate amount of subcutaneous swelling was noted over the surgical site, but no associated discharge or pyrexia was observed. Serosanguineous fluid was obtained on fine needle aspiration and this was submitted for cytology as well as culture and sensitivity testing. The dog was started empirically on a 5-day course of amoxicillin-clavulanate (22 mg/kg twice daily; Apo-Amoxycillin and Clavulanate, Apotex). Cytology results were consistent with a postoperative seroma. The antibiotics were discontinued when the culture results returned negative.

A week later, the dog was presented for acute non-weight-bearing LPL lameness after escaping from the backyard. Repeat radiography revealed caudal displacement of the proximal tibial fragment associated with breakage and loosening of the two proximal plate screws. Surgery was revised with a 3.5-mm broad locking TPLO plate. No evidence of recurring bridging fibrosis across the released crural fascia was noted intraoperatively.

On examination at 2 weeks and 2 months following revision surgery, the dog showed good LPL function with normal stifle ROM. He walked comfortably and tracked normally in a straight line compared with the short-stepping abnormal RPL gait. Repeat radiography showed evidence of bone healing across the osteotomy. Recurrence of the restrictive fibrotic bands was not evident on palpation.

The patient was seen 14 months after the first surgery for progressive RPL lameness. The owner reported an occasional
non-weight-bearing lameness that worsened after exercise. Orthopaedic examination showed marked muscular atrophy of the thigh muscles bilaterally. Severe restriction in stifle extension and abduction associated with the fibrotic bands was evident in the RPL. Comparatively, there was only a mild reduction in left stifle extension. Marked medial buttress was also evident in both stifles. There was a marked pain response on cranial drawer test and palpable effusion in the right stifle. Repeat radiography showed evidence of significant effusion and osteophytosis around the patella and trochlear ridges of the right stifle.

The dog was admitted for a modified TTO and concurrent medial crural fasciotomy. The approach to the RPL was as previously described. There was no evidence of meniscal injury and a partial meniscectomy was not performed. The fibrotic bands on the medial aspect of the limb were more thickened and taut compared with those seen when the LPL surgery was undertaken. A marked improvement in the stifle ROM was similarly achieved following release of the crural fascial attachments. The modified TTO was performed and stabilised with a 3.5-mm broad locking TPLO plate and tension-band wire. Closure of the incision and postoperative care were as described for the LPL. On postoperative radiographs (Figure 5), the TPA was measured as 5°.

At the 2-week recheck, the dog had a grade 3/5 RPL lameness, but showed no evidence of the anomalous gait and had good ROM in the right stifle. Moderate subcutaneous swelling was similarly noted over the surgical site, with no evidence of associated discharge or pyrexia. Results of needle aspiration for cytology as well as culture and sensitivity testing were negative for infection. Hence, the swelling was managed as a seroma.

On the 8-week postoperative radiographs, bone healing was evident. However, a grade 1/5 RPL lameness still persisted, with osteoarthritis suspected to be the main contributor to the dog’s discomfort. He was managed with firocoxib and gabapentin (3 mg/kg twice daily; Neurontin, Pfizer) for 4 weeks and was tapered off following resolution of lameness.

A follow-up examination was performed 29 and 15 months post-surgery in the LPL and RPL, respectively. Palpable thickening within the semitendinosus muscle could be appreciated bilaterally, but there was only mild restriction of both stifles in extension. Movement was unencumbered, with no recurrence of the mechanical lameness noted bilaterally. The pelvic limbs displayed good cranial stride length and tracked within the sagittal plane without circumduction. Occasional lameness following prolonged vigorous exercise was noted, but there was an overall improvement in gait and function following surgery.

**DISCUSSION**

We describe the use of bilateral medial crural fasciotomy and modified TTO for treatment of clinically diagnosed semitendinosus fibrotic myopathy and concurrent CrCL disease in a GSD. Most of the studies in the literature depict a poor response to surgical intervention of gracilis or semitendinosus contracture. In the biggest case series of 18 dogs managed surgically with myotomy or tenomyectomy the dogs showed recurrence of the abnormal gait within a median period of 2 months postoperatively.1 Interestingly, Henderson et al.2 recently detailed their management protocol for chronic fibrotic myopathy where aggressive resection of all fibrotic bands was undertaken followed by a 1-month intensive in-hospital rehabilitation and 6-month outpatient physiotherapy program thereafter. Most patients returned to an acceptable function albeit at a lower level but information regarding long-term recurrence was unavailable. To our knowledge, our study marks the first case in the literature where there has been good functional outcome following surgical management with no recurrence in the long-term.

The classical gait and presence of fibrotic bands are consistent with semitendinosus or gracilis fibrotic myopathy. Although similar, differentiating the two depends on a solid appreciation of muscle anatomy. The gracilis muscle originates from the pelvic symphysis whereas the semitendinosus muscle arises from the lateral angle of the ischiatic tuberosity and passes deep to the gracilis muscle.22 Both muscles attach separately onto the cranial border of the tibia, but unite caudally to send a distal reinforcing band to the calcaneal tuberosity.22 Functionally, both act as flexors of the stifle and extensors of the hip and tarsus. The use of ultrasound or advanced imaging such as computed tomography or magnetic resonance imaging can be useful to delineate the region of pathology.10,14 These were not used in this case because the origin of the fibrotic bands could be clearly identified anatomically to that of the semitendinosus muscle at the ischiatic tuberosity. The distinct extension of this thickened band proximally along the medial thigh towards the ischiatic tuberosity also differentiated it from the periarticular medial buttress.

The superficial and deep leaves of the crural fascia unite to form three distinct fibrous strands medially: one inserts on the cranial border of the tibia, another extends along the caudal border of the cranial tibial muscle and the last contributes to the common calcaneal tendon.22 The first two correlate anatomically...
to the two distinctive fibrotic bands seen intraoperatively, which suggests extensive fibrotic involvement of the crural fascia in the disease process that has not been previously described. Moreover, this technique may not be limited to semitendinosus muscle contractures and could possibly extend to use in dogs affected by sartorius or gracilis fibrotic myopathy because of their contribution to the medial crural fascia.

In the long-term follow-up, fibrotic bands within the semitendinosus muscles were still palpable with mild stifle restriction noted in both pelvic limbs. The palpable thickening could possibly be explained by the fibrotic remnants that were left in situ following crural fasciotomy. Moreover, the accessory tendons extending to the common calcaneal tuberosity were not released and could provide a source of continuous strain and ongoing foci for bridging fibrosis.\(^{5,6,13}\) The development of osteoarthritis and associated periarthritis fibrosis from chronic CrCL disease would also likely be a contributing factor in the mildly reduced ROM in the stifles. Compared with other previous surgical techniques described,\(^{6-9,16,19}\) medial crural fasciotomy is a simple technique that is quick to perform. The minimal amount of soft tissue on the medial aspect of the tibia allows for easy identification of the medial crural fascia and its attachments. In this case, a medial approach to the tibia was required to perform the modified TTO, which facilitated the concurrent medial crural fasciotomy. Seroma formation in the postoperative period occurred in both instances following surgery. Medial crural fasciotomy involves the release of the crural fascial attachment to the tibia and extends beyond the mid-shaft of the tibia. The fascial layer was not re-apposed in order to reduce the recurrence of fibrosis. However, the lack of apposition results in potential dead space over the tibia medially, which can result in the production of interstitial fluid from inflammatory mediators that accumulates and forms a seroma, as seen in this case.\(^{21}\) Techniques such as postoperative bandaging or cold compression are advocated to reduce the incidence of associated seroma formation.\(^{26}\)

The effect of the concurrent tibial plateau levelling procedure remains a confounding factor. Although the marked improvement in stifles ROM postoperatively is largely attributed to the fascial release, it is also important to consider some contribution to this area from the TTO, because of the resulting cranial rotation of the distal limb relative to the tibial plateau. The CrCL acts as the primary passive constraint in preventing cranial tibial displacement during weight-bearing.\(^{27}\) Similarly, the semitendinosus muscle has been found to be an agonist of this function by operating as an active stabiliser to oppose cranial tibial thrust.\(^{22,26}\) The CrCL have been identified as containing a high density of mechanoreceptors and proprioceptors.\(^{28}\) Multiple electromyographic studies using various animal models have identified a neurophysiological reflex arc between the CrCL and the caudal thigh muscles whereby increased ligamentous loads similarly trigger increased contractions and recruitment of these muscles as a protective mechanism.\(^{20,29}\) An ex vivo study evaluating the biomechanics of the semitendinosus muscles showed a two-fold increase in tensile forces within the muscles when the CrCL was transected compared with when it was intact.\(^{29}\) Moreover in CrCL-deficient stifles, it was found that the mean cranial tibial displacement increased from 2.1 to 7.2 mm following the release of the semitendinosus muscle.\(^{29}\) These findings suggest that a significantly increased strain is likely to be placed on the semitendinosus muscle in the event of CrCL disease. Hence, the elimination of the cranial tibial thrust with the modified TTO may serve to diminish repetitive stress on the semitendinosus muscle and could play a possible role in explaining the lack of recurrence in this case. Conversely, it could be postulated that any compromise to the CrCL would increase semitendinosus muscle strain and may act as a trigger early in the disease process of fibrotic myopathy.

A number of limitations need to be highlighted in this report. Firstly, this case study was based on two procedures performed on a single patient with no controls. A greater number of cases with a control group would be required to assess the repeatability and validity of the results seen. Secondly, histopathology of the diseased tissue was not performed. However, the signalment of an active GSD with the classical gait and palpable fibrotic bands were considered to be sufficient for a clinical diagnosis of semitendinosus fibrotic myopathy.\(^{5,6,8,13}\) Thirdly, assessment of function was based on subjective clinical evaluation and objective measurements of function such as goniometry, kinematic gait or force-plate analysis were not used. These would have been ideal for critical evaluation of kinetic parameters and to quantify the degree of improvement in the ROM.

Although a positive long-term outcome was achieved in this case, it is unclear which of the two, the release of the crural fascia or correction of the CrCL disease, played a more significant role. Replication of the crural fasciotomy in the absence of the tibial osteotomy procedure should be investigated in future cases of semitendinosus muscle contracture to assess the true benefit of this technique.

CONCLUSION

In conclusion, both the crural fascia and CrCL disease may be implicated as part of the fibrotic myopathy disease process and more work needs to be done to identify the underlying aetiology as knowledge of the disease remains limited and hypothetical. The lack of long-term recurrence in the case presented here suggests that surgical intervention may not be as ineffective as previously reported, but further investigations into the techniques described here are warranted.

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REFERENCES


