Unusual gait abnormalities affecting an equine musculoskeletal system

Author : Jonathan D C Anderson, Matthew J Chesworth

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Gait abnormalities in the horse usually present as lameness and a systematic approach to the lame horse will facilitate the most accurate and complete diagnosis.

Several more unusual gait abnormalities of the horse have specific characteristics that make them pathognomonic for the underlying pathological process involved. Because of the intimate association between the musculoskeletal and nervous systems, several neuromuscular conditions present with characteristic gait abnormalities, which, if recognised, will prevent misdiagnosis or confusion as to the best management approach.

Neuromuscular disorders

Figure 1. Typical reaction of a horse with shivers when asked to back up or lift the hindlimb. The limb is jerked up and shivers for several seconds prior to it being replaced.

Neuromuscular disorders include shivers, stringhalt, sweeney, tetanus and dropped elbow. All have characteristic clinical features.

Shivers

Shivers is a chronic neuromuscular disorder of horses that has been reported for centuries. Draft breeds are predominantly affected, although it is reported across a variety of horse breeds. It has not been reported in ponies. All ages are affected with a slow progression of disease. Shivers results in a reflex hypertonia of the hindlimb flexor muscles and tail.

Periodic, involuntary spasms of the muscles in the pelvic region, pelvic limbs and tail are often associated with tension or trembling of the hindlimb musculature. When the hindlimb is stimulated and elevated off the ground, the leg hyperflexes and abducts in a spastic state for several seconds before being replaced on to the ground (**Figure 1**). Backing up or turning in a circle often will stimulate the same response and the symptoms can be exacerbated by stress or excitement.

The clinical spectrum of shivers in horses is variable in the degree or manifestation of signs. The diagnosis of a characteristic case of shivers seldom presents a problem; however, the signs of shivers may be intermittent, occasional or latent and difficult to confirm. Shivers may be extremely difficult to detect in the early stages, and careful observation may be required before a diagnosis can be made. Owners may report the horse snatches the limb up when having feet picked out or shod.

The aetiology is unknown and a suggested correlation between shivers and horses with polysaccharide storage myopathy (PSSM) is likely a coincidence as apposed to true correlation, as PSSM tends to affect draft breeds, as does shivers[1]. In the past, breeding away from horses affected with shivers reduced the incidence of shivers in the horse population – suggesting a genetic link, although no genetic link has been established[2].

Shivers is usually diagnosed by the clinical presentation, although early onset or mild cases may be difficult to distinguish from other conditions such as stringhalt. PSSM horses show generalised weakness and abnormal polysaccharide accumulation in the muscle fibres and, if there is breed disposition for PSSM, a muscle biopsy will help clarify concurrent presence of this disease.

Shivers has no cure, with the disease often progressing in severity to the point severely affected horses may not be able to move back at all without the limb abducting so high it causes the horse to lose balance. With progression of the disease, a gradual and progressive atrophy of the muscles of the thigh occurs and this may progress to generalised muscle atrophy. There then may be some response to a high fat, low carbohydrate diet with increasing exercise[2].

Stringhalt

Figure 2. The typical appearance of a horse with classical stringhalt. Note the dorsal aspect of the metatarsophalangeal joint will slap the underside of the abdomen.

Stringhalt is a progressive, ill-defined neuromuscular disorder of the hindlimb that results in mild to severe and often violent hyperflexion of the tarsus. It has been described as a distal axonopathy in which damage to the large, more vulnerable nerve fibres from the muscle spindles in the hindlimb are predominantly affected[3]. In particular, the muscle spindles of the lateral digital extensor tendon appear particularly relevant to the observed clinical signs. The condition can be unilateral or bilateral, can occur in any breed and age of horse and, at its worst, the affected limb may result in the dorsal aspect of the foot hitting the ventral abdomen (**Figure 2**).

Backing the horse up, or turning in a tight circle, may exacerbate the condition. It is differentiated from shivers as the foot contacts the ground immediately as apposed to being held in a spastic state for a few moments.

Two general forms of the disease are recognised. Australian stringhalt is associated with ingestion of dandelion (*Taraxacum officinale*), flatweed (*Hypochaeris radicata*) and cheeseweed (*Malva parviflora*). Older and taller horses appear to be predisposed. Hindlimb muscle atrophy and laryngeal paralysis may also be features of the condition. Horses recover if removed from affected pastures, but this may take days to many months to occur. Phenytoin has been reported to decrease the severity of clinical signs[4].

The second, more sporadic, form of stringhalt has an unknown aetiology, but may be associated with injury to the stifle or tarsus and, particularly, trauma to the dorsum of the metatarsus, upward fixation of the patella and painful foot conditions.

Treatment of both forms of the disease, which can recover spontaneously, may be unnecessary as, in many cases, little impact occurs on the horse's performance. If progressive, deterioration of the condition is seen or, in more severe cases, dramatic improvement over a few days has been reported in myotenotomy of the lateral digital extensor tendon[5].

While lateral extensor tenotomy can be performed standing and has fewer incisional complications, better success has been documented when a portion of the muscle is removed in addition to the tenotomy and this necessitates general anaesthesia[5].

A four-week postoperative recovery period means horses are quickly back into work barring any complications of surgery. In some cases, the condition becomes so progressively severe ridden work becomes impossible.

Sweeney/shoulder slip

Sweeney, or shoulder slip, is a characteristic gait abnormality in which loss of collateral support of the shoulder on the medial (subscapularis) and lateral sides occurs. This results in the shoulder region bulging abaxially when weight is placed on the limb. The foot is usually placed medially and the heel rotates laterally as the horse moves. Traditionally thought to be due to damage of the suprascapular nerve, it has been shown it is rather a feature of a brachial plexus injury[6].

Experimental transection of the suprascapular nerve results in profound neurogenic atrophy of the supraspinatus and infraspinatus muscles, but not an abnormal gait, while seven horses euthanised due to instability of the shoulder region had fibrosis of nerves within the brachial plexus and ventral nerve roots and no abnormality affecting the suprascapular nerve as it coursed around the cranial neck of the scapula.

Typically, horses present with a history of blunt trauma and pain-related lameness initially that resolves after one to two weeks. Muscle atrophy occurs within 7 to 10 days and concurrent patchy sweating of the caudal neck, depending on the nerves affected.

With nerve regeneration occurring 1mm each day, progressive improvement in the lameness acts as a good prognostic indicator over the ensuing days following injury[6].

Despite persistent muscle atrophy of the supraspinatus and infraspinatus muscles, normal gait can be restored. However, if after six months of no improvement in the gait occurs then prognosis for return to function is guarded. Dissecting a notch out of the scapular neck, which has been described to remove fibrotic material around the nerve, is not indicated, as there is no evidence the condition is related to compression or fibrosis of the suprascapular nerve.

Tetanus

Tetanus is an infrequent cause of a progressively stiff gait that is frequently fatal if not recognised and treated early. Tetanus, is a disease caused by the toxin tetanospasmin, which is produced by the anaerobic bacteria Clostridium tetani. This organism is almost ubiquitous in the environment and commonly found in soil, where spores are able to survive for several years. Exposure usually occurs through infection of a deep contaminated wound or can occur through lacerations, surgical

sites and the umbilicus of neonates.

Once produced by the bacteria, the toxin gains access to motor neurons and causes blockade of the inhibitory synapses by preventing neurotransmitter release. This, in turn, leads to hypertonia of the striated muscles and can also lead to sympathetic and parasympathetic signs.

Horses present in some cases with localised hypertonia and muscle spasms around a wound. This often progresses to generalised spasticity with clonic muscle spasms. A "sawhorse" stance with an elevated tail head is characteristic of the disease and extensor muscle spasm makes movement difficult. Severe cases will become recumbent with dysphagia and dyspnoea – all three of which are negative prognostic indicators for survival. Further signs include a sardonic grin caused by increased muscle tone of the muscles supplying the lips. Dorsomedial retraction of the ears and wrinkling of the forehead may also be seen. Prolapse of the third eyelid and enophthalmos is also occasionally seen due to hypertonicity of the extraocular muscles.

Treatment is geared around prevention of further toxin release and supportive therapy to allow time for regeneration of new synapses, returning normal function of the neurons once more. Antibiotics targeting the C tetani bacteria are used, with high-dose penicillin used most commonly. Tetanus antitoxin is also used at high dosages to neutralise unbound toxin before it is able to worsen the condition. This can be injected around the wound site.

Dropped elbow

Inability to bear weight on a forelimb, with resulting dropping of the affected limb's elbow, is a gait abnormality that can be associated with a fractured olecranon or a radial nerve paresis. The presentation is the same for both conditions and the horse can present with marked distress due to the inability to fix the carpus in extension and place weight on the affected limb. Differentiating the two in the field may be difficult, with presence of wounds in the upper antebrachium, history of fighting with other horses, kick marks on other parts of the horse's body or swelling and crepitus (rare) in the region of the elbow all indicating a fracture of the olecranon.

The distress that often accompanies these injuries can obviate a thorough assessment and requires stabilisation of the limb by fixing of the carpus with a caudally located splint. This will often result in the horse becoming more settled and allow transportation to a referral centre or further radiographic assessment of the limb on the yard. The radial nerve innervates the extensor muscles of the forelimb, thus all extensor function is lost when this is damaged. Horses typically stand with the elbow dropped and the affected limb forward with the antebrachium at an angle of 45° to the ground. Because the triceps tendon of insertion is the olecranon, fracture of the olecranon results in loss of the muscle's extensor function, thus resulting in the dropped elbow and a similar appearance as radial nerve paresis. Compression of the brachial plexus between the scapula and the ribs can also result in radial nerve signs[6].

Diagnosis of the condition is by typical appearance and distinguishing a fracture from radial nerve paresis requires radiographs of the elbow. Both craniocaudal and lateral views are required to delineate a fracture, if present. Fractures can be managed conservatively or surgically depending on their orientation and involvement of the articular surface of the elbow joint. Surgical stabilisation is the preferred treatment option in most cases as conservative treatment can result in non-unions or even repeat fracture six months after apparent repair. Treatment for radial nerve paresis is limited to rest and techniques to try to encourage the return of nerve function. Electrical stimulation of the muscles may help maintain muscle mass. Horses that show improvement in clinical signs steadily over the course of the first few days following the incident have a good prognosis for full return to normal function. If no improvement in clinical signs occurs over the course of several days to two weeks, then, in the authors' experience, return to normal function is guarded and any recovery takes several months, if it occurs at all.

Gait abnormalities associated with musculotendinous unit

Figure 3. Peroneus tertius tendon rupture. Note characteristic tarsus and stifle moving independent of one another. Normally, flexion of the stifle should result in flexion of the tarsus. Also note the characteristic dimpling of the gastrocnemius tendon proximal to the tuber calcis.

Disorders of the musculotendinous unit with characteristic gait dysfunction include exertional rhabdomyolysis, PSSM, fibrotic myopathy, peroneus tertius rupture, upward fixation of the patella, as well as lacerations of the superficial and deep digital flexor tendons and suspensory ligament.

Rhabdomyolysis

A stilted, stiff gait affecting one or more limbs can result from a variety of conditions. The most common cause of an acute onset sudden and painful stiff gait is rhabdomyolysis. This term results in a destruction of striated muscle cells and encompasses a wide range of diseases differing in

their causes. Ultimately, however, they lead to similar clinical signs and abnormalities in gait. Broadly speaking, equine rhabdomyolysis can be divided into exertional and non-exertional causes. Sporadic exertional rhabdomyolysis (tying up) is the most common of these myopathies and is seen during exercise – often after one or two days of rest. It is thought this rest period may lead to a build-up of glycogen within the cells, leading to subsequent damage during exercise. Recurrent exertional rhabdomyolysis (RER) is often seen in young animals – in particular, fillies – and a genetic component has been demonstrated in thoroughbreds[7].

Like RER, PSSM falls under the category of chronic exertional rhabdomyolysis. This disease – seen predominately in warmbloods, Irish sports horses, draft horses and quarter horses – has two forms based on the detection of an autosomal dominant point mutation in the glycogen synthase 1 gene that causes unregulated synthesis of glycogen – those that have the gene are classed as type one PSSM and those that don't are classed as type two PSSM. Prevalence of PSSM in Percherons and Belgians is 35% and, in quarter horses, 12%[8].

Diagnosis of rhabdomyolysis is initially based on clinical presentation. Horses may present with a stiff and stilted gait and reluctance to move, which, in some cases, may progress to recumbency. Gait abnormalities in these cases vary depending on severity of muscle damage and it is important to note horses may be asymptomatic. Presence of firm, painful muscles, muscle fasciculations and sweating is highly consistent with exertional rhabdomyolysis and would be confirmed with marked elevation of creatine kinase (CK) and aspartate transaminase (AST) from one to four hours post-exercise. Hindquarters, thoracolumbar and forelimb musculature can be affected, with pain often being seen within 20 minutes of exercise – particularly if the horse has been rested for several days and is on a high carbohydrate diet.

Diagnosis is made in conjunction with persistent elevations of CK and AST and urine analysis, and a muscle biopsy of the semimembranosus or gluteal muscle is useful in refractory cases and in which PSSM or other underlying causes are suspected. Biochemistry results can give important clues to the time of injury in less clinically obvious cases, with muscle enzymes CK (peaking at around four to six hours post-injury) and AST (peaking at 24 hours post-injury) routinely measured in these cases. Lactate dehydrogenase may also be elevated.

Importantly, muscle enzyme elevations may not be a feature of the disease in draft horses, with more mild signs of weakness, exercise intolerance and gait abnormalities being a reason to submit a muscle biopsy for definitive diagnosis. Genetic testing from blood or hair roots allows possible diagnosis of type one PSSM and is available at the University of Minnesota Veterinary Diagnostic Laboratory[8].

Acute management of exertional rhabdomyolysis consists of anti-inflammatories, muscle relaxants and rest for 48 hours, with 5 to 10 minutes of walking over the following week and a gradual build-up of exercise with prolonged relaxed warm-up periods. Fluid therapy, in cases where renal involvement or urinary abnormalities occur, is essential to prevent further renal compromise.

Management strategies for RER or PSSM orientate around rest in a field environment combined with a low carbohydrate/high fat diet. Dietary changes need implementing gradually prior to commencing a change in exercise regimes and both intensity and duration of exercise require a slow and gradual build-up.

Fibrotic myopathy

Fibrotic myopathy is a condition affecting the hindlimb musculature in which there is fibrosis of the muscles caudal to the stifle that prevents their normal function. The most common affected muscle groups are the semimembranosus, semitendinosus and biceps femoris muscles, with the gracilis muscle also being more rarely reported.

Significant scarring of the muscle groups results in an exaggerated protraction of the anterior phase of the stride that causes the foot to "slap" the ground in a sharp caudal direction at the end of the anterior phase of the stride. The fibrosis of the muscle is usually a result of tearing of the particular muscle group that then heals with fibrous scar tissue and represents a true mechanical gait abnormality. Trauma of the muscle groups is seen in horses in which sliding stops or excessive force are placed on the hindlimb that results in tearing of the muscles. Abscessation in the region as a result of intramuscular injections or, for any other reason, may also underlie the condition.

Diagnosis is typically made by the characteristic gait pattern of the horse and, in certain types of quarter horses (specifically those used as cutting horses), it may be bilateral. Confirmation can be made by palpation of fibrous bands of tissue down the caudal aspect of the limb caudal to the stifle, and ultrasound of the muscle groups allows identification of specific muscles involved.

Treatment involves releasing of the constriction caused by the fibrotic tissue. This has traditionally been performed by en-bloc resection of the fibrotic tissue (partial myotenotomy); however, this carries the risk of recurrence as the wound site heals again, incisional dehiscence is common, as is infection of the wound, which also results in recurrence. Transection of the fibrous tissue with a laser (laser fiberotomy), combined with immediate postoperative exercise, has been reported to be successful in resolving the problem and with an improved cosmetic outcome[9].

An indirect approach has been described and adopted by the authors in which the tendon of origin of the affected muscle groups is identified ultrasonographically and this is transected in a standing procedure, usually with a successful outcome[10]. Reported success rates for treatment vary and partial resolution, at the very least, often occurs – probably as a result of inadequate transection of all affected tissues.

Peroneus tertius rupture

The peroneus tertius (PT) forms part of the hindlimb reciprocal apparatus in the horse and is responsible for coordinating flexion and extension of the hock and stifle. As the tarsus is extended,

the normal PT is stretched. Complete or partial rupture of the PT is usually traumatic (blunt trauma or laceration) in origin and has a characteristic gait in which the stifle flexes without flexion of the hock when the limb moves forward. When passively flexing the limb, the hock can be moved independently from the stifle and results in a characteristic dimpling effect of the gastrocnemius tendon on the plantar tarsus (**Figure 3**). If traumatic in origin, acute lameness is usually associated with the injury and the characteristic gait is diagnostic.

Ultrasound can be used to determine the site of the rupture that occurs most commonly in the mid-tibial region, but also occurs at its origin on the lateral aspect of the femur or its insertion on the dorsal aspect of the distal tarsal region.

While traditionally it has been thought the site of rupture is an important prognostic indicator, more recently, the site of rupture, degree of lameness, ultrasonographic size of the lesion, the age of the horse, the rehabilitation type and type of injury had no influence on the outcome11. However, the higher the level of performance the horse was in at the time of injury, or if an additional structure was injured at the time of injury, then there was an 11 times and 14.6 times respectively less likelihood of these horses returning to its intended use[11].

Treatment of PT rupture orientates around rest and an ultrasonographically guided return to exercise. Premature return to exercise can result in recurrence, even when a functional recovery has been achieved.

Hypertrophy of the adjacent structures may help to support the healing PT – giving a false impression of full recovery. Closed, traumatic rupture of the PT requires three months of total rest, with light work resuming after and normal exercise resuming six months after injury. However, again, ultrasound should be used to assess the state of healing of the PT and, in some cases, horses may not be able to resume exercise for eight months and take 10 months to return to previous exercise levels. Nevertheless, 70% of horses can return to their previous levels of exercise after between six and 10 months, although caution should be exercised if the horse was a performance horse at the time of injury, or if additional structures were involved in the original injury[11].
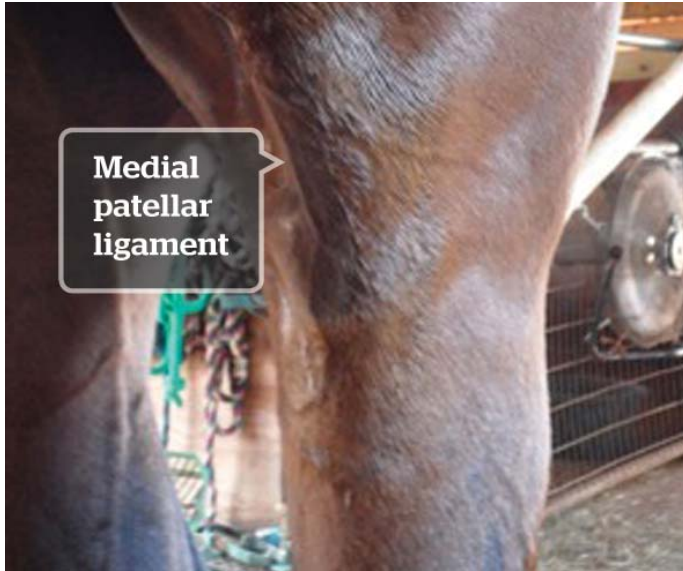
Upward fixation of the patella

Figure 4. Location of the medial patellar ligament on the medial aspect of the stifle. The medial patellar ligament is best located by palpating the tight finger thickness middle patellar ligament at its insertion on to the tibia and moving medially to feel the medial patellar ligament as it continues proximal from its insertion with the middle patellar ligament on the tibia.

Intermittent upward fixation of the patella is a relatively common condition in horses in which the medial patellar ligament fails to disengage from its position over the fibrocartilage on the medial trochlear ridge of the femur, thereby preventing the stifle from flexing (**Figure 4**).

Because of the reciprocal apparatus when the stifle cannot flex, the hock cannot either, so the horse's leg becomes stuck in an extended position until the medial patellar ligament unhooks. The horse will be unable to move the leg forward without dragging the toe and it may result in distress for the horse when asked to move. Especially common in young horses prior to, or at the initiation of, training, a higher incidence occurs in Shetland ponies, miniature horses and standardbreds. The inciting cause is commonly the resting of a fit horse due to injury, with resultant loss of quadricep muscle tone and laxity of the medial patellar ligament. Usually, bilateral with one side more severely affected, the condition can be subtly seen as the buckling of the hindlimb when the horse decelerates or is going downhill, or irritation under the saddle with resultant adverse behaviour.

Diagnosis of persistent fixation is from the typical history and obvious gait or description of the gait when seen by the owner or trainer. Intermittent fixation is more difficult to diagnose. Physical manipulation of the patella region may reproduce the classical signs and backing up and being asked to move off sharply or turning in a tight circle may result in signs. The patella may appear to move abnormally as it catches on the medial trochlear ridge.

It is important underlying conditions, such as developmental orthopaedic disease of the femoropatellar and femorotibial joints and medial patellar ligament desmitis, are ruled out as

underlying causes of pain and resultant fixation.

Treatment strategies progress through a series of increasingly invasive stages. Increasing muscle mass and fitness is the first essential component of resolution and may be all that is required in some cases. Horses intermittently catching the patella may respond to an internal sclerosing agent being injected in small increments in multiple locations within the medial patellar ligament. Iodine in a sesame seed or almond oil base is most commonly used and, in conjunction with exercise, will enable successful resolution. For those horses not responding after two weeks to injections, or for more persistent cases, medial patellar ligament splitting can be successful.

A number 15 scalpel blade is used to make multiple stab incisions into the medial patellar ligament (**Figure 5**). The principle is increasing the bulk of tissue within the ligament will prevent the laxity, decrease its elasticity and prevent it from becoming permanently engaged over the medial trochlear ridge.
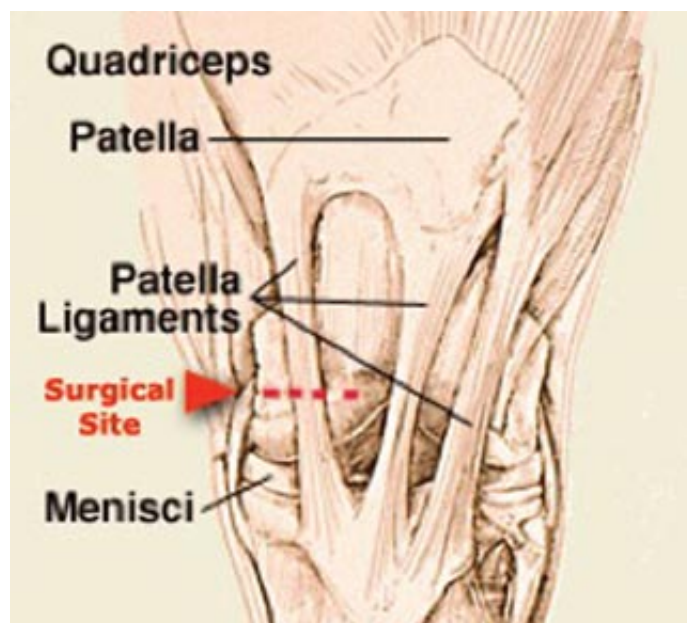

Figure 5. Anatomy of the ligaments of the dorsal aspect of the stifle region showing the location for transection of the medial patellar ligament on the medial aspect of the femoropatellar joint.

For permanently fixated cases, or those not responding to either of the two aforementioned treatments, medial patellar ligament desmotomy is required. The ligament is transected at its insertion on to the tibia via a stab incision. Complications of this procedure include fragmentation of the apex of the patella some years later that can lead to more persistent lameness.

This is the only procedure that provides immediate correction of the problem; however, given the lack of complications associated with the other two procedures, this is left as a last resort by the authors, unless the horse has permanent upward fixation, in which case, it is the only option to

resolve the condition. Many horses mature out of the condition and the prognosis is usually good – especially if fitness can be maintained.

Flexor tendon and suspensory ligament rupture

Catastrophic injury to the digital flexor tendons and suspensory ligament can present in horses of any age and discipline; however, when associated with exercise, it is most commonly seen in jumping or racing horses where maximal extension of the distal limb occurs. Alternatively, they can present as a deep wound or laceration to the palmar/plantar surface of the limb. Palmar digital neurectomy may also lead to the rupture of structures such as the deep digital flexor tendon (DDFT) due to lack of sensory feedback as a lesion progresses. Spontaneous rupture of the superficial digital flexor tendon (SDFT) in aged horses is also recognised[12].

Several telltale signs linked to the gait of these horses are available to the clinician on presentation in most situations. These help distinguish whether structures have truly ruptured, or if some function remains. They also help to distinguish SDFT (**Figure 6**), DDFT (**Figure 7**) and suspensory ligament (**Figure 8**) rupture from each other.

Figure 6. Superficial digital flexor tendon breakdown injury. Note the marked swelling of the palmar aspect of the distal third of the metacarpal region usually associated with acute onset of pain and lameness and diffuse swelling of the limb that may extend to the proximal carpal region including the carpal sheath. Once stabilised, comfort can be achieved within 24 to 48 hours of injury. Image: Barny Fraser, Rainbow Equine Hospital.

Figure 7. Rupture of the deep digital flexor tendon characterised by elevation of the toe. This occurs usually in conjunction with rupture of the superficial digital flexor tendon and is a career-ending injury. Image: Barny Fraser, Rainbow Equine Hospital.

Figure 8. Rupture of the suspensory apparatus resulting in dropping of the metacarpophalangeal joint and near horizontal angulation of the pastern region. This is a career-ending injury, with return to athletic soundness extremely unlikely. Image: Barny Fraser, Rainbow Equine Hospital.

Cases typically present as an acute onset of non-weight bearing lameness; however, comfort levels can improve relatively quickly if incomplete rupture occurs. Concurrent heat and diffuse and painful swelling at the site of injury often occurs.

Complete rupture of the SDFT results in a hyperextension of the fetlock joint, which moves closer to the ground relative to the contralateral joint when the limb is loaded at walk; however, this is not always evident when the horse is standing. Rupture of the DDFT leads to a characteristic elevation

of the toe from the ground when the limb is weight bearing. The level of fetlock often remains normal. If both SDFT and DDFT rupture are apparent, as seen in some severe lacerations, dropping of the fetlock – in combination with elevation of the toe from the ground – occurs.

Laceration or rupture of the suspensory ligament causes a hyperextension of the fetlock similar to that of the SDFT rupture. Palpation of the structures and ultrasound will rapidly distinguish between the two if needed. If suspensory ligament rupture is in combination with SDFT and DDFT laceration, the fetlock contacts the ground when weight bearing, with marked elevation of the toe from the horizontal. This is often very distressing for the horse.

While rupture of any of the three structures is usually a career-ending injury, a fibrotic union can form between the ruptured tendon ends and a return to soundness can be achieved with surgical and conservative management of SDFT ruptures.

Initial treatment involves early anti-inflammatory therapy, including NSAIDs, and cold water therapy, combined with strict rest. A Robert Jones bandage or bandage cast can be useful until further assessment of structures can be assessed. If the SDFT is confirmed to be completely lacerated, and the lacerated ends are intact, surgical debridement and suturing enables the best chance of successful healing in the shortest time frame.

Otherwise, box rest alone, in conjunction with either casting or Robert Jones bandaging, has been effective in restoring a functional tendon, although return to soundness is unlikely. Spontaneous ruptures in aged horses can heal, with conservative management consisting of three to four months of box rest alone[12].

For DDFT and suspensory ligament lacerations, a palmar/plantar splint is important to support the metacarpophalangeal or metatarsophalangeal joint if the DDFT or the suspensory ligament is involved, and palmar/plantar shoe extensions are useful to correct the hyperextension of the coffin joint. Rupture of the DDFT and suspensory ligaments carry a hopeless prognosis for return to comfort.

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