Review Article

Flexural deformities of the distal interphalangeal joint (clubfeet)

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Summary

A true clubfoot results from a flexural deformity of the distal interphalangeal joint that is characterised by a shortening of the deep digital flexor tendon musculotendinous unit. Flexural deformities are a problem not only in foals but are also responsible for the clubfoot conformation seen in mature horses. Treatment is most successful when the cause is investigated and therapy initiated as early as possible, and when the biomechanical properties of the foot are thoroughly understood. Flexural deformities in foals and mature horses are addressed through appropriate farriery, often combined with surgery.

Introduction

Despite the recent advances in breeding, nutrition and farm management, flexural deformities are still a reasonably common occurrence. A flexure deformity can be defined as a shortening of the musculotendinous unit of the deep digital flexor tendon (DDFT) that results in hyperflexion of a given anatomic region of the limb (Adams 2000; Greet 2000; Hunt 2000, 2011; Greet and Curtis 2003). Flexural deformities have been traditionally referred to as ‘contracted tendons’; however, the primary defect appears to be a shortening of the musculotendinous unit rather than a shortening of just the tendon portion; making ‘flexural deformity’ the preferred descriptive term (Kidd and Barr 2002). Shortening of the musculotendinous unit produces a structure of insufficient length to allow normal alignment of the distal phalanx (P3) relative to the middle phalanx and results in variable clinical signs ranging from an upright hoof angle to a clubfoot. The focus of this paper is to define and recommend therapy for flexural deformities involving the DDFT and the distal interphalangeal joint (DIPJ). The emphasis is on the forelimb unless otherwise stated in the text. The paper is divided into flexural deformities of foals and flexural deformities of mature horses. Severe flexural deformities of foals and mature horses are commonly referred to as clubfeet.

Anatomy review

In the antebrachium, the muscle bellies of the DDFT lie directly on the palmar aspect of the radius and are covered by the muscle bellies of the superficial digital flexor tendon (SDFT) and the flexors of the carpus. The deep digital flexor muscle consists of 3 muscle bellies (the humeral head, the inconsistent radial head and the ulna head), which form a common tendon proximal to the carpus. This tendon, along with the SDFT, passes through the carpal canal and continues down the palmar aspect of the third metacarpal bone. Below the fetlock, at the level of the middle phalanx, the DDFT perforates the tendon of the SDFT, continues distally and inserts on the flexor surface of the distal phalanx (P3). A strong tendinous band known as the accessory ligament of the DDFT (AL-DDFT) originates from the deep palmar carpal ligament and fuses with the DDFT at the middle of the metacarpus (Fig 1). The design and function of the anatomical structures is such that any prolonged shortening of the musculotendinous unit affects the position of the DIPJ. The palmar surface of the distal phalanx is pulled palmarly by this shortened musculotendinous unit, placing the DIPJ in a flexed position. The alignment of the bone within the hoof capsule remains constant while the hoof capsule is pulled with the distal phalanx. The flexed position of the DIPJ combined with the altered load on the foot leads to a rapid distortion of the hoof capsule and thus the clubfoot conformation. It can also be noted from the anatomy that transecting the AL-DDFT, when necessary, lengthens the musculotendinous unit either functionally or by allowing relaxation of the proximal muscle belly associated with the DDFT.
Classification of flexural deformities (clubfeet)

Flexural deformities have been classified as type 1 where the hoof-ground angle is $<90^\circ$ and type 2 where the hoof-ground angle is $>90^\circ$ (Adams 2000). A recent method of classifying flexural deformities using a grading system (Grade 1–4) has been proposed (Redden 2003). Regardless of the method, it would appear beneficial to classify the severity of the flexural deformity to devise an appropriate treatment plan and monitor the response to a given therapy. A grading system would also enhance record keeping as well as improve communication between the veterinarian, farrier and owner with regard to treatment strategies.

A Grade 1 clubfoot has a hoof angle 3–5° greater than the contralateral foot and a characteristic fullness present at the coronet. The hoof-pastern axis generally remains aligned. A Grade 2 clubfoot has a hoof angle 5–8° greater than the contralateral foot, the angle of the hoof-pastern axis is steep and slightly broken forward, growth rings are wider at the heel than at the toe, and the heel may not touch the ground when excess hoof wall is trimmed from the heel. A Grade 3 clubfoot has a broken-forward hoof-pastern axis, often a concavity in the dorsal aspect of the hoof wall, and the growth rings at the heels are twice as wide as those at the toe. A Grade 4 clubfoot has a hoof angle of $>80^\circ$, a marked concavity in the dorsal aspect of the hoof wall, a severe broken-forward hoof-pastern axis, and the coronary band from the toe to the heel has lost all slope and is horizontal with the ground (Fig 2). For simplicity, the author uses a grading system based on the severity or degree of flexion noted in the DIPJ on the lateral radiographic projection to classify flexural deformities.

Flexural deformities in young horses

Flexural deformities in foals can be divided into congenital or acquired deformities. As such, congenital deformities are noted at birth, and acquired deformities generally occur during the first 6 months of life as the foal grows and develops.

Congenital flexure deformities

Congenital flexural deformities are present at birth, may involve a combination of joints (e.g. carpus, metacarpophalangeal and DIP joints) and are characterised by abnormal flexion of these joints and their inability to extend. Proposed aetiologies of congenital flexural deformities include malpositioning of the fetus.
in utero, nutritional mismanagement of the mare during gestation, teratogens in various forages ingested by the mare and maternal exposure to influenza virus, or the deformities could be genetic in origin (Kidd and Barr 2002; Hunt 2011). The affected foal tends to walk on the toe of the hoof capsule, is unable to place the heel on the ground and assumes a so-called ‘balerina’ stance. Treatment of foals with a congenital flexural deformity varies with the severity of the deformity. A mild to moderate flexural deformity in which the foal can readily stand, nurse and ambulate is generally self-limiting and resolves without treatment. Brief intervals of exercise once or twice daily in a small paddock on firm footing for the first few days of life may be all that is necessary for the deformity to resolve. If the condition is severe or has not improved by the third day post foaling, i.e. administration of oxytetracycline (2–3 g) repeated every other day if necessary is frequently beneficial (Madison et al. 1994). A variety of bandaging techniques and splints are used, along with physical therapy, to ‘stretch’ the involved area to hasten recovery. Foals with severe congenital flexural deformities usually do not have just one isolated structure or joint that is responsible for the deformity, therefore, in the author’s opinion, the use of a toe extension is not indicated.

**Acquired flexural deformities**

Acquired flexural deformities generally develop when the foal is aged 2–6 months and generally involves the DIPJ initially. The aetiology of this deformity is unknown, but speculated causes include genetic predisposition, improper nutrition (i.e. overfeeding, excessive carbohydrate [energy] intake, unbalanced minerals in the diet) and excessive exercise. A recent study looked at grazing patterns in a small number of foals and showed that foals with long legs and short necks had a tendency to graze with the same limb protracted (van Heel et al 2006). Fifty percent of the foals developed uneven feet with a higher heel on the protracted limb, leading researchers to feel there may be a possible correlation between conformational traits and an acquired flexural deformity. It is the current author’s opinion that a large contributing factor to this syndrome is contraction of the muscular portion of the musculotendinous unit caused by a response to pain, the source of which could be physical dysplasia or trauma from foals exercising on hard ground. Discomfort may follow aggressive hoof trimming where excessive sole is removed, rendering the immature structures within the hoof capsule void of protection and susceptible to trauma and bruising. Any discomfort or pain in the foot or lower portion of the limb coupled with reduced weightbearing on the affected limb appears to initiate the flexor withdrawal reflex, which seems to cause the flexor muscles proximal to the tendon to contract, leading to an altered position of the DIPJ. This shortening of the musculotendinous unit shifts weightbearing to the dorsal half of the foot causing a decrease in sole depth and bruising of the sole, reduced growth of the dorsal aspect of the hoof wall, and excessive hoof wall growth at the heel to compensate for the shortening of the musculotendinous unit. As the flexural deformity may be secondary to pain in these cases, it is essential that the source of pain should be carefully evaluated by physical examination and by localisation using regional analgesia and diagnostic imaging techniques.

A genetic component must also be considered for acquired flexure deformities, as some mares consistently produce foals that develop a flexural deformity in the same limb as the dam or grand dam in which a similar deformity is present. The genetic component of the flexural deformity may be the ultimate determinant of the severity of the deformity. A genetic component should also be considered in the aetiology of acquired flexural deformities, although there is no scientific evidence for this at present. Some mares appear to consistently produce foals that develop flexural deformities, sometimes in the same leg as was affected in their dam or grand dam.

**Mild acquired flexural deformities**

**Clinical signs**

The initial clinical sign of a flexural deformity may be abnormal wear of the hoof at the toe, which is often discovered by the farrier during routine hoof care. Closer investigation may reveal that the dorsal hoof wall angle is increased and that after the heels of the hoof capsule have been trimmed to a normal length, the heels may no longer contact the ground. A prominent coronary band may or may not be present at this stage. Most foals affected to this degree have a mildly broken-forward hoof-pastern axis. Increased palpable digital pulse, heat in the affected foot, and signs of pain when a hoof tester is applied to the dorsal aspect of the toe are not uncommon clinical findings. These findings are generally the result of trauma or excessive weightbearing on the toe.

**Treatment**

Conservative treatment, such as correcting the nutritional status of the foal (i.e. weaning the foal to avoid possible excessive nutrition from the mare), restricting exercise to reduce trauma, judiciously administering a nonsteroidal anti-inflammatory drug (NSAID) to relieve pain, administering oxytetracycline to facilitate muscle relaxation, and carefully trimming the hoof is, in the author’s opinion, a good starting point. NSAIDs should be administered short-term and should be used judiciously in foals due to the potential side effects, such as gastroduodenal irritation and nephrotoxicity. For analgesia, the author will administer firocoxib (0.1 mg/kg bwt q.24 h) or flunixin meglumine (1.1 mg/kg bwt q.24 h) combined with a gastric protectant. Hoof trimming begins with lowering the heels from the middle of the foot palmarly until the hoof wall at the heels and the frog are on the same plane. The bars...