Pedal bone fractures

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Pedal bone (third phalanx) fractures are traumatic injuries that should be considered in the diagnosis of confirmed foot pain or clinical signs suggesting the foot as the cause of lameness. Causes include collisions with firm objects or kicking walls and both front and hind feet can be affected.

They are typically classified as 6 types:

I: Palmar process fractures.
II: Oblique/parasagittal fracture.
III: Sagittal fractures.
IV: Extensor process fractures.
V: Comminuted fractures.
VI: Solar margin fractures.

Pedal bone fractures are acute in onset and severe, with the exclusion of type VI and some type IV fractures. Clinical signs may include heat in the foot, increased digital pulses, or sensitivity to hoof testers. Cases with an articular component involving the coffin joint will usually have a coffin joint effusion. Some will respond to a palmar digital nerve block but all pedal bone fractures should respond significantly to an abaxial sesamoid nerve block. Articular fractures respond at least partially to a DIPJ block and if there is any ambiguity over articular involvement, contract medium combined with the local anaesthetic followed by radiography is useful.

Type VI fractures can occur in both foals and adults and usually causes a low grade and relatively short term lameness. In adult horses they are almost always in the front feet between the quarters and the toe.

Pedal bone fractures are diagnosed radiographically but radiography directed at the foot is prompted by clinical signs, a lameness that blocks to the foot or suggestive findings on other imaging modalities (i.e. scintigraphy). A normal foot series (lateral, standing DP, dorsoproximal-palmarodistal oblique and palmaroproximal-palmarodistal oblique) may not show the fracture in which case additional views such as a dorsal 45 proximal 45 lateral-palmarodistal medial oblique should be taken. The latter can be obtained with the foot bearing weight, standing on a cassette tunnel, or in a block. Proper foot preparation and packing lessens the incidence of potentially confusing artefacts that may be mistaken for fracture lines. Radiographic diagnosis can be challenging as the x-rays must pass parallel to the fracture line for the line to be visible. If a fracture is suspected but not confirmed radiographically, then repeat radiographs after 10 days are recommended at which time osteolysis may aid identification of the fracture line. Separate centres of ossification of the palmar processes should not be confused with type I fractures. Radiographs can underestimate the severity of the fracture, such as comminution and MRI or CT will give a more accurate picture of the fracture configuration. Scintigraphy can be useful to assess the significance of a chronic fracture. Many fractures remain radiographically evident long after healing is complete.

Treatment often is dictated by the presence of an articular component (types II and III) where return of joint congruity in the coffin joint is a main principle of treatment. Lag screw fixation of type III fractures will give the best return of articular congruity but this must be offset against the risk of infection due to an inability to completely sterilise the hoof wall prior to placing the screws. Many type II fractures, although articular, will respond to nonsurgical treatment.

Nonsurgical treatment options, often used in combination, include remedial farriery (Rim shoes or shoes with multiple clips) or foot casts and all in conjunction with box rest/exercise restriction. Both shoeing and casting can also be used as adjunct treatments post surgical repair. Neurectomies can be considered for cases with chronic lameness. A bar shoe should be considered when a horse with a healed pedal bone fracture is returning to work.

The prognosis for articular fractures (types II and III) is worse than for nonarticular fractures (types I and VI) because of the likelihood of osteoarthritis within the coffin joint. Nonarticular fractures carry a good prognosis if a long enough period of convalescence is undertaken. Comminuted fractures (type V) carry a poor, but not hopeless, prognosis.

Four cases will be presented and their different methods of management discussed.

NOTES
Magnetic resonance (MR) imaging has become more available in equine veterinary practice in recent years and has provided us with a better understanding of the pathogenesis of palmar heel pain in lame horses. Injury to the deep digital flexor tendon (DDFT) within the foot is now recognised as a common cause of lameness in sport horses (Dyson et al. 2005; Mair and Kinns 2005; Dyson and Murray 2007). It has been suggested that DDFT lesions in the digit are primarily degenerative and may be a sequel to vascular compromise (Blunden et al. 2009). Different types of DDFT injury can be evident on MR scans with core lesions, sagittal splits and dorsal fibrillation being the most common (Blunden et al. 2009). Injuries to the DDFT frequently occur in association with other pathological changes to the podotrochlear apparatus (Dyson et al. 2007).

Whilst these DDFT injuries have been well documented and well correlated with histopathological changes (Dyson et al. 2003; Blunden et al. 2006, 2009; Murray et al. 2006) there is still little information in the literature about the prognosis for return to athletic soundness in horses with DDFT injury in the foot. It is generally considered that these injuries have a guarded prognosis for return to athletic soundness (Dyson 2003). Dyson et al. (2005) reported a retrospective study of 199 horses that had undergone MRI examination. DDFT tendinitis was diagnosed in 59% of horses and considered to be the primary injury in 65 horses (33%). Only 13 of 47 (28%) horses with primary lesions of the DDFT that were followed-up 6–36 months later had returned to full athletic function without recurrent lameness. Smith et al. (2007) reported the results of a retrospective study in which lesions in the DDFT in 22 navicular bursae of 20 horses were identified and debrided endoscopically. Follow-up information (≥6 months) was available for 15 animals of which 9 had returned to preoperative levels of performance.

A retrospective study of lame horses with DDFT injury in the foot diagnosed using low-field MRI (0.27 Tesla) at The Liphook Equine Hospital between December 2003 and December 2007 is currently being undertaken. The aim of this study is to determine the prognosis for athletic soundness for horses with lameness caused by DDFT injury within the foot and to test the hypothesis that the prognosis for horses with tendon injury at or above the level of the navicular bone is better than that of horses with DDFT injury at the level of the tendon’s insertion.

This larger study follows on from a pilot study in which the case records of all horses scanned at The Liphook Equine Hospital between December 2003 and May 2005 were reviewed. Horses in which lameness was localised to the foot with diagnostic analgesia and in which lesions in the DDFT were considered to be the primary cause of lameness were included in the study. Only lesions that could be classified as core lesions, parasagittal or sagittal splits or dorsal border lesions were included. Horses with a diffuse increase in signal in the DDFT were not included. Follow-up information was obtained by telephone survey at least 8 months after diagnosis. The outcome was classified as good if the horse had resumed full athletic function and returned to its previous level of work, moderate if the horse was sound but in lighter work and poor if the horse had suffered from persistent or recurrent lameness or had been subjected to euthanasia due to lameness.

In this period 160 horses underwent MRI examination for the investigation of foot lameness at The Liphook Equine Hospital. DDFT lesions were identified in 66 horses (41%). Thirty-six horses had concurrent pathology and were not included in this study. In 30 horses DDFT lesions were considered to be the sole cause of lameness. Of these 30 horses, 19 horses had sagittal or parasagittal splits in the DDFT, 6 horses had dorsal border changes (short splits or fibrillation) and 5 horses had core lesions. Three of 30 (10%) horses had a good outcome, 15/30 (50%) horses had a moderate outcome and 12/30 (40%) horses had a poor outcome. No horses with tendon lesions involving the tendon’s insertion (n = 4; 2 splits and 2 core lesions) had a good outcome. In conclusion, at the current time, the prognosis for return to former levels of athletic function in horses with DDFT injuries in the foot is considered guarded. Further large studies are needed to determine what effect the type and location of injury have on the prognosis and to determine the effect of different management strategies.

References
Keratomas: Diagnosis and treatment

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Definition and aetiology
Keratomas are hyperplastic keratin masses that normally arise from the epidermal keratin-producing cells of the stratum germinativum of the coronary band but can also originate from any point on the inner surface of the hoof wall or the sole. Keratomas are situated between the stratum medium of the hoof wall and the underlying distal phalanx (P3). The aetiology is unknown although they are thought to result from excess horn production by the germinal epithelial cells, initiated either by trauma or a subsolar abscess, but have also been diagnosed when there has been no known hoof wall insult.

Clinical signs and diagnosis
Clinical signs include lameness (varying from mild to severe) localised to the foot. The lameness is a result of either the development of a secondary subsolar abscess or has been attributed to the pressure on the underlying sensitive laminae and P3. Pressure from the keratoma may also cause outward deviation of the overlying hoof wall and thinning or discolouration of the white line at the sole. Radiographs of the P3 typically demonstrate a clearly defined, smoothly margined semicircular radiolucent defect in the solar margin of the distal phalanx. Surrounding marginal sclerosis is sometimes present. However, not all cases present with this radiographical abnormality, either because the keratoma is not large enough to cause sufficient bone resorption or because the keratoma has not extended distally enough. New bone formation on the dorsal aspect of P3 has also been observed. Space occupying lesions such as keratomas have also been observed on MR images and multiple keratomas within one foot are rare but have been reported.

Treatment
Complete surgical resolution can result in resolution of the lameness. Resection can either be performed via complete hoof wall resection from the coronary band to the sole or by partial hoof wall resection directly over the mass. Surgical excision can be performed under general anaesthesia or under standing sedation and local analgesia. The affected foot is rasped, scrubbed and wrapped in a povidine iodine dressing for 24 h before surgery. Preoperative antibiotics and nonsteroidal anti-inflammatory agents are also recommended. An abaxial sesamoid nerve block is performed and an Esmarch’s bandage is applied immediately prior to surgery.

Complete hoof wall resection (CR)
Two parallel cuts are made on either side of the mass from the solar margin to the level of the coronary band. The cuts are connected distally along the white line and proximally, just distal to the coronary band. All cuts are made to the level of the sensitive laminae. The cut section of hoof wall is levered proximally, exposing the underlying keratoma and allowing surgical excision. A thorough inspection of the underlying tissue is made to confirm complete removal.

Partial hoof wall resection (PR)
This method requires a more accurate identification of the location of the keratoma prior to surgical excision. This is performed using preoperative radiographical markers and the use of a metal probe inserted into the drainage tract should it be present. A ‘window’ is created in the hoof wall, either using a cast saw or a trephine centred over the mass. This window can be enlarged intraoperatively or a second ‘window’ can be made in the sole of the foot to ensure complete removal of the keratoma.

Histopathology
Histopathology is vital for the affirmative diagnosis of a keratoma.

Post operative care
A foot bandage is placed following surgery and changed every 3–4 days or more frequently if required. The hoof wall defect is packed with swabs which can be soaked in gentamicin or metronidazole to decrease the risk of post operative infection. A normal shoe with side clips or a heart bar shoe with a bolted on hospital plate can be applied once the defect is dry and showing some signs of keratinisation. A metal ‘bridging’ plate can be applied across the hoof defect in cases where there is or there is expected to be excessive hoof wall instability. The hoof wall defect should be inspected at least every 3–4 days and once it has keratinised and hardened, the defect can be filled with polymethylmethacrylate. A normal shoe can then be fitted and exercise introduced.

Post operative complications
Fifty percent of horses in a recent study suffered from post operative complications with the complication rate being statistically higher in horses that had undergone CR (71%) as opposed to PR (25%). Complications include recurrence of the keratoma (11%), excessive granulation tissue formation (31%), the development of a hoof wall crack originating from the surgical site (8%), the continued hoof wall deformation (4%), and infection forming underneath the hoof wall filler (4%).

Outcome
Horses undergoing CR were statistically more likely to develop post operative complications compared to those undergoing PR (P<0.01). Ninety-six percent of horses were sound and returned to their previous exercise level 6–36 months post operatively (a median of 10 months following CR and 7 months following PR). The time to post operative soundness was not different between the 2 techniques although the time to post operative attainment of the previous exercise level was significantly less following PR than following CR (P<0.01).

Conclusion
The surgical resection of a keratoma results in favourable to excellent end result with horses returning to soundness and their previous exercise level. PR is the preferred technique as the complication rate is lower and the time to work following surgery is less compared to CR. However the technique is more difficult and care must be made to ensure adequate resection of the entire keratoma.
Further reading

NOTES
Foot penetrations

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Introduction
Penetrating injuries to the foot are common in equine practice and careful assessment and an appreciation of the underlying structures involved is essential to allow the correct management decisions to be made.

Clinical presentation
Many penetrating injuries of the foot involve nails or screws, which can pass through the outer stratum externum to involve underlying structures but other objects, such as stones, glass or wood may be involved. The owner may be present when the incident occurs or find the object on examination of the foot, and the question always arises of whether to remove the object or leave in situ. If the object is removed it is essential that it is kept for further examination and that the location, depth and orientation of the penetration are recalled by the owner as accurately as possible. If the object is kept in place it may help radiographic assessment but there is always the risk of further trauma to the foot by deeper penetration.

Usually an acute lameness develops following a foot penetration, prompting examination by the veterinary surgeon. Occasionally there may be no history of a penetration or the consequences of the injury may take several days to manifest. A good clinical history is mandatory, including tetanus status of the horse and, if in doubt, tetanus antitoxin should be administered.

The presenting lameness is usually moderate to severe but may be less in chronic injuries or with the presence of a draining tract. Increased digital pulse amplitude is usually present. Generalised swelling of the lower limb/digit may develop and there may be effusions of the DIP joint and/or DFTS on palpation. The coronary band and heels should be examined for areas of discomfort or softening indicative of infection tracking proximally. Careful assessment of the solar surface is required. Removal of dirt and purging of the outer horn will assist examination. Hooftesters should be applied over the foot in a careful and consistent manner, testing the ability of the horse to bear weight to protect the horn. In severe cases a hospital plate may be required. Antibiotics are not usually required unless a concurrent cellulitis is present. Most cases resolve satisfactorily but persistent or recurrent drainage should be investigated further (e.g. sequestrum/keratoma). Nail pricks or binds require removal of the nail or shoe and rest and may or may not develop into an abscess.

Diagnostic procedures

Radiography
Radiographic examination is very useful to identify the depth and direction of a penetrating tract, particularly if the object is still present. In the absence of the object, a sterile probe can be introduced into a tract. Ideally a full radiographic examination should be performed but lateralomedial and dorsoproximal-palmaro(plantaro)distal oblique views are the minimum requirements. Radiography can assist in ruling out other causes of acute foot lameness such as fractures as well as detailing sequelea of the penetration such as infective osteitis/osteomyelitis and show gas opacities. Contrast media may be used to outline synovial structures and show communication with tracts but introducing the contrast agent via the tract itself can be unrewarding. The absence of communication of the tract with a synovial structure does not rule out their involvement.

Ultrasound
Ultrasound can be used to assess fluid quantity and echogenicity in synovial structures such as the DIP or DFTS. It is also useful to examine the coronary band/heel bulbs for infections or where excessive soft-tissue swelling may preclude accurate palpation or synovial sample acquisition.

Magnetic resonance imaging
The advent of MRI has revolutionised assessment of conditions of the foot and the use of it in foot penetrations is highly applicable. MRI is extremely good at identifying involvement of soft tissue and bony structures where radiography may fail to do so. The tract is usually easily visualised from its point of penetration to whatever structures are involved. This can be used to direct further diagnostic techniques and case management.

Synoviocentesis
Synoviocentesis of the navicular bursa, DIP joint and/or DFTS may be required. If sepsis of the DIP joint or DFTS is present following a puncture wound to the middle part of the frog then it is highly likely that the navicular bursa is also involved due to anatomical arrangements (i.e. fenestration of the impar or ‘T’ ligament, respectively).

Treatment decisions

Superficial penetrations
Treatment of a subsolar abscess following a superficial penetration requires adequate drainage and removal of affected horn. A sufficiently large drainage hole should be established and flushed. The use of a hot poultice may assist in softening the horn and osmotic solutions (hot tubbing in salty water) may help to draw out fluid. Once drainage has ceased then dry poultices are used to protect the horn. In severe cases a hospital plate may be required. Antibiotics are not usually required unless a concurrent cellulitis is present. Most cases resolve satisfactorily but persistent or recurrent drainage should be investigated further (e.g. sequestrum/keratoma). Nail pricks or binds require removal of the nail or shoe and rest and may or may not develop into an abscess.

Deeper penetrations
Deep solar penetrations may involve the bone, for example P3 and thereby setting up an infective process leading to infective osteitis and/or sequestra formation. Radiography at the time of penetration may be unremarkable but repeat examination is required if clinical signs persist. Removal of the affected portion(s) of bone may be required under standing sedation or general anaesthesia. Once the affected tissue has been removed and drainage established, the condition usually resolves. Samples of tissue (bone/horn) for bacteriology can direct effective antibiotic usage.

Deep penetrations in the middle third of the foot may involve of the DDFT, navicular bursa and bone, impar ligament, DIP joint and possibly the DFTS, depending on depth and orientation. Careful assessment of these cases is required and suitable diagnostic imaging and synoviocentesis techniques are needed. Treatment of a synovial sepsis usually involves arthroscopic techniques to debride and lavage the cavities and remove any foreign material if present. This can be coupled with local delivery techniques of antibiotics both at the time of surgery and in the post operative period. Success rates vary but are around 60% but will reduce with chronicity and multiple structure involvement.
Hoof wall and coronary band injuries

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Traumatic lacerations to the coronary band and hoof wall are relatively uncommon injuries in the horse. The wounds are typically encountered when the horse kicks out or the hoof is trapped between 2 immovable objects. The deeper the wound the more soft tissue trauma and the increased likelihood of involvement of the distal phalanx or contamination of regional synovial structures.

The wound healing characteristics of hoof wall avulsions are slightly unique when compared to wound healing of other tissues or body locations. Outcome and long-term soundness is often difficult to predict at initial presentation. Wound healing is, by comparison, slow. There is no contribution of contraction to the speed of wound healing and the prolonged treatment often required can sometimes result in significant costs.

Like any healing tissue, wound healing consists of 3 phases: inflammation, repair and maturation. However, wounds involving the hoof capsule show limited swelling and do not contract. Epithelialisation is different and comes from progressive downward growth from the coronary band rather than local proliferation. Wounds of the frog or sole behave in a similar way to other epithelial wounds where re-epithelialisation comes from the margins of the wound via migration.

Wounds involving the coronary band may involve epithelialisation from 2 sources, therefore the final result depends on what type of epithelium filled the defect. For this reason horny spurs can occasionally form proximal to the coronary band, where the germinal epithelial cells of the coronary band have migrated proximally. Similarly, conservative management or failure of repair of the coronary band will result in hoof defects growing distally.

The proximity of the hoof to the ground means that the wounds quickly become contaminated, so first aid should be directed at haemostasis, decontamination, and preserving the structure of the hoof. The ultimate aim is to prevent further contamination. If contamination is light the wound should be covered with a sterile water based gel before clipping and scrubbing of the skin. As with any wound, disinfection, copious scrubbing of the skin, and dilute antiseptics are advised. Detergents, concentrated antiseptics (especially iodine) and other caustic agents will damage deeper tissues and delay wound healing.

It is not unusual for wounds of the coronary band to contain foreign material, particularly wood. Careful digital exploration and ultrasound examination is useful in locating such material but small splinters below the coronary band are difficult to locate. Wounds of the coronary band and heel bulb should be meticulously closed whether by primary or secondary closure. Method of closure and choice of suture size/pattern should be applicable to the tension relief required. Debridement should be kept to a minimum (without leaving nonviable tissue) due to the relative absence of elasticity of neighbouring tissues. In heavily contaminated wounds and wounds with extensive tissue loss, repair is best delayed until a granulation bed is obtained. Undermining of the adjacent coronary band with thinning of the underlying hoof wall is one possible way to reduce tension in coronary band repairs.

Topical and systemic antibiotics therapy is indicated in most cases. High bacterial loads is one of the major inhibitors of wound healing. Distal limb regional and intraosseous perfusions are useful adjuncts when treating or preventing wound sepsis of the these injuries.

Avulsion injuries and resultant shearing of the epidermal laminae from the dermal laminae usually results in some of the stratum germinativum remaining adherent to the lamellar corium. These residual cells are responsible for the rapid epithelialisation and keratinisation. Hoof avulsion injuries rarely involve the deeper structures and usually result in detachment of a portion of the hoof wall, normally in the heel or quarter.

Lameness is usually marked and proportional to the amount of hoof wall involved.

Treatment is usually straightforward. After appropriate restraint, regional anaesthesia, cleaning and disinfection, the detached hoof wall is simply excised. It is advised that if there is any horn remaining attached at the coronary band and underlying corium, it should be left in place to provide stability. No horn should be left with square edges. The margins should always be thinned or tapered distally. The area is covered using wet to dry antibacterial dressings until sufficiently healed. Once keratinisation is evident, hoof hardeners can be applied to enhance the drying process. Hoof acrylic could later be applied to the hoof defect and a bar-shoe with quarter clips fitted. A suggested guideline is to wait until 0.5 cm of growth has occurred from the coronary band before considering using acrylcs.

Wounds involving the heel bulb and coronary band should invariably be treated by primary closure. Treatment with bandaging alone will usually result in poor functional results, horny spurs, permanent hoof cracks and hoof wall defects. The initial costs of a surgical repair are offset by the ongoing costs incurred with prolonged bandaging; costs that are often underestimated.

Any wound deep enough to increase movement and cause instability of a hoof wall should be immobilised in a cast (standing application if appropriate). Wounds of the heel bulb and quarters at good examples. A cast should either finish just below the fetlock or just above the coronary band. A cast should never finish at the mid-pastern as this will result in rubs at the dorsoproximal or caudoproximal border of the cast. Two or 3 weeks cast immobilisation is required. Once the cast is removed the foot should be shod with a heart bar shoe and quarter clips with the heel under the coronary band defect floated to reduce stress at coronary band repair.

Hoof injuries are best protected with a wet to dry dressing (petroleum gauze and antibiotic ointment is a good primary dressing) and the final layer covered with duck tape.

Further reading

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