

Navicular syndrome in horses – treatment and prognosis rethink

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ABSTRACT

This article covers advances in diagnostic imaging that have led to a better understanding of the pathology responsible for foot lameness, questions the terminology associated with foot pain and discusses the rationale and efficacy of existing and previous treatments for palmar foot pain.

“It has been my experience that wherever you have a persistent obscure lameness in the fore end, the most likely cause is navicular disease” – TV Vet Horse Book (1975).

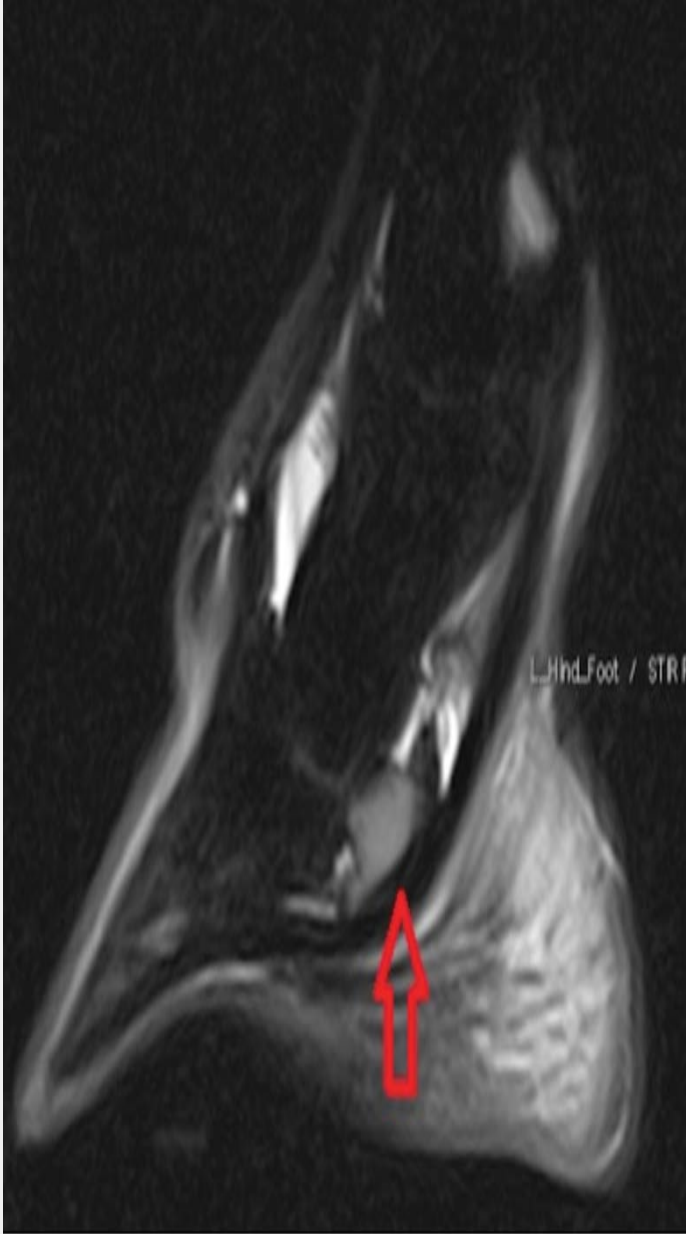
“The tentative diagnosis can be made with confidence on the symptoms, but positive confirmation is possible only by x-ray” – TV Vet Horse Book (1975).

Firstly, let us address the elephant in the room. Navicular disease has, historically, been a blanket term encompassing every lameness that might (or might not) have improved to a palmar digital nerve block and might have had some spurious irregularity of the navicular bone on radiographs. We now know this to be nonsense.

Of course, back in the good old days a perfectly good excuse existed – no way of narrowing the diagnosis further, so treatment was empirical with remedial farriery as the mainstay, supplemented by any number of, often, pointless treatments ranging from surgery through polypharmacy, to eventual neurectomy.

Now, however, with the advent of MRI and contrast CT allowing assessment of soft tissues within the hoof capsule, better understanding of diagnostic anaesthetic techniques and retrospective assessment of abnormal navicular radiographs, we,

as a profession, are amply able to dismiss the myth of rampant “navicular disease” that keeps so many of our clients awake at night.





Short tau inversion recovery fast spin-echo sagittal image of a foot showing (top) intense increased signal in the navicular bone (red arrow) consistent with primary navicular bone “inflammation” compared to normal (above).

A study of 702 horses with MRI diagnosis of foot pain concluded response to diagnostic anaesthesia, clinical examination and radiological grading did not generally help differentiate between lameness associated with the podotrochlear apparatus (navicular bone and supporting ligaments) and other causes of foot pain (Parkes et al, 2015).

The present day equivalent of this is proximal suspensory desmitis, a very fashionable diagnosis for pain originating in the palmar/plantar cannon region. I – like many – make this as a diagnosis (often by exclusion) based on the results of nerve blocks, x-rays, ultrasound and scintigraphy, occasionally backed up with CT or MRI examination.

Frequently during the investigation, we find other diagnoses arising from the same area and this must be recognised and recorded so we don't go down the same “navicular pathway”. With the advances in 3D and physiological imaging techniques, more accurate diagnosis will be readily achievable, allowing us to target treatment and offer more accurate prognoses in the same way in

which foot pain is now handled.

Horses that may previously have been diagnosed with navicular disease have undergone MRI examination of their feet with a plethora of specific related and unrelated problems becoming evident. Normal horses have been examined and incidentals/normal variations have been established, allowing confidence in MRI diagnosis. Postmortem examination has also shown excellent correlation with MRI findings, giving further confidence to diagnosis.

The more recent classification is to group primary navicular pathology (navicular bone marrow oedema) together with distal border fragmentation, navicular suspensory desmitis, deep digital flexor tendonitis, impar ligament desmitis, adhesions between the navicular bone and deep digital flexor tendon (DDFT), and navicular bursitis, using the umbrella term “navicular syndrome”.

While many horses will have a combination of these structures injured simultaneously, numerous, as-yet unknown, aetiopathogeneses are involved and individual conditions are likely to respond very differently to different treatments. Is it time we moved away from the term “navicular” with its bogeyman connotations and instead, adopted palmar/plantar foot pain (PFP) or, more accurately, podotrochlear apparatus syndrome (PTAS) to describe this condition?

We could then focus on specific treatments for the individual conditions and, when faced with the combined PFP/PTAS, use a combination of appropriate treatments.

Previous treatments for navicular disease

Farriery

Corrective shoeing has always been essential in treatment of foot conditions and likely accounts for the majority of improvement in lameness seen following combination treatments. More specific farriery is discussed further on.

Surgical

Originally published in 1986, navicular suspensory desmotomy appeared to show promise. The first large-scale study of 118 horses had a success rate of 76% sound at 6 months, dropping to 43% by 36 months (Wright, 1993). Numerous conclusions were drawn from the data available at that time, which can now largely be dismissed – MRI and CT were unavailable at this time and we must assume very few of these animals had primary navicular pathology. So, what was surgery achieving?

Many would argue it is little more than a complicated neurectomy; however, there may be some biomechanical alteration to the forces placed on the navicular bone, DDFT and supporting structures. This may be a useful technique for primary navicular disease; however, as yet, no large-

scale studies have looked at its use in definitively diagnosed cases.

Inferior check ligament (ICL) desmotomy has been suggested as a treatment to allow the pastern and hoof to assume a more normal angle. However, no obvious link exists between the shape of the external hoof and a diagnosis of navicular disease.

Horses in which the navicular bone sits more distally (that is, in closer contact with the distal phalanx than the middle phalanx) seem more prone to developing navicular pathology, and ICL desmotomy is unlikely to significantly lift the position of the bone, suggesting this technique is unlikely to be successful.

Medicinal

Phenylbutazone remains the most common treatment, with the drawbacks of a potential to develop right dorsal colitis, the competition withhold time and the fact deterioration of existing pathology is likely to occur without the protective mechanism of pain and inflammation.

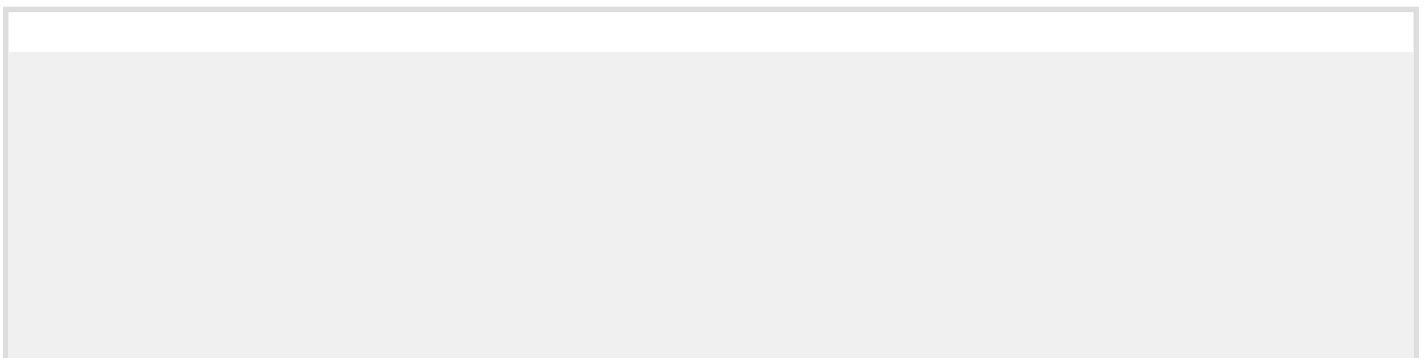
Warfarin has been used at a dose rate of 0.018mg/kg daily, increasing by increments of 20% until the stage one prothrombin time is increased by two seconds to four seconds. Warfarin administration has been associated with fatal haemorrhage in 1% of treated horses.

It is aimed at reducing thrombosis and decreasing blood viscosity, although the only evidence of any vascular compromise is vascular changes within the bone (dilated venules entrapped in fibrous marrow) are only seen in horses with navicular disease. A “successful response” to treatment was seen in up to 80% of patients.

Isoxsuprine hydrochloride (0.6mg/kg to 1.2mg/kg twice a day by mouth) may increase distal limb circulation and has been credited with long-term efficacy in 40% to 87% of horses.

As with warfarin, the assumption was an ischaemic pathogenesis, now largely disproven, hence both these drugs have all but disappeared from clinical use.

But what were we treating?







Chronic foot lameness: lateromedial radiographs pre (top) and five weeks post (above) palmar digital neurectomy surgery, showing traumatic luxation of the distal interphalangeal joints necessitating euthanasia. Luxation occurred in both forefeet.

Now we are able to make a specific diagnosis, treatment can focus on addressing the specific underlying pathology, making many previous treatments redundant.

For example, distal border fragmentation of the navicular bone has been well recognised radiographically; however, only recently has been shown to be unlikely associated with existing lameness. Treatments targeted at this are, therefore, unlikely to be necessary or effective.

The following is a list of the existing and potentially future treatments available to treat both primary navicular bone pathology and the combined “navicular syndrome”/PPF.

Existing treatments

Remedial farriery

The use of a rolled toe, raised heel bar shoe is initially advised. The rolled toe eases breakover, raising the heel by 2° to 4°, unloads the DDFT, navicular suspensory and impar ligaments, reducing the matrix force of the DDFT, pushing the navicular bone into the palmar aspect of the DIP joint, and an egg or straight bar shoe provides caudal support.

A 1° decrease in palmar angle causes a four-fold increase in peak force in the navicular bone. Trimming is essential in maintaining or correcting the palmar angle; however, trimming alone is rarely capable of immediately redressing the abnormal forces on the foot.

Different shoes are available for many of the common diagnoses now made on MRI examination. The shoe type should not be prescriptive, but this formula can be used as a starting point and the shoe tailored to the individual's needs.

It is always best to involve the farrier in the original decision-making; in most cases, he or she is far more familiar with your patient's hoof than you and infinitely more capable of shoeing the horse.

Medical

Bisphosphonates

Bisphosphonates have become the treatment of choice for primary bone inflammation within the hoof capsule. Initially thought to inhibit osteoclastic activity (and, hence, bone resorption), their effect is now realised to be far more complex. They are also considered to be good analgesics for bone-related pain.

Concerns have been raised over their potential to increase the risk of stress fracture, particularly in young Thoroughbred racehorses in training; however, this population is relatively distinct from horses suffering "navicular syndrome". Published data is available for tiludronic acid as a treatment for navicular disease; however, this study was carried out before the advent of MRI (Frevel et al, 2014; Denoix et al, 2003).

A dose rate of 1mg/kg is effective in reducing lameness and returning horses to their previous level of activity two months to six months post-treatment. The shorter duration of pre-existing lameness, the better the prognosis. An updated study would be beneficial looking specifically at the response to treatment for horses diagnosed with navicular disease using MRI.

Clodronic acid is also licensed for treatment of navicular disease in the horse at 1.2mg/kg IM. Efficacy was assessed by comparing lameness grades before and after treatment in 146 horses. Treated horses had a significantly higher success rate (reduction in lameness) than horses in the control group and efficacy was sustained over 180 days in 66%.

There has been a trend to delivering these drugs via intravenous regional perfusion (IVRP) to the affected area to reduce costs. The clinical results are unpublished, but effects seem similar to a full

systemic dose.

Concerns have been raised over the potential toxicity of bisphosphonates to articular cartilage, with one study showing tiludronic acid administered at 50mg by IVRP in the distal limb raised synovial fluid concentrations above a previously determined safety level in a small number of horses (Hunter et al, 2015). However, no differences in synovial fluid parameters were found between treated and control limbs, suggesting this is probably a safe technique.

Botulinum toxins

Botulinum toxin A has been shown to inhibit the release of neurotransmitters when injected intra-articularly in dogs. Inhibition of substance P, calcitonin and gene-related peptide interfere with the pain pathway reducing lameness. Botulinum toxin B has been researched as a treatment for horses with degenerative changes in the podotrochlear apparatus of horses.



Palmar-proximal to palmar-distal oblique radiographic view of the navicular bone of the horse that suffered distal interphalangeal joints luxation. While there is clearly medullary sclerosis and areas of lysis within the navicular bone, there is also marked irregularity of the flexor cortex associated with fibrocartilage erosion. There was also marked calcification of the navicular suspensory apparatus. This is a severe case of “navicular syndrome” or palmar/plantar foot pain/podotrochlear apparatus syndrome.

Its safety was first confirmed in a small number of horses prior to evaluation of the potential to reduce lameness in affected animals. 3U/kg to 4.4U/kg of botulinum toxin type B was injected aseptically into the navicular bursa and lameness was assessed at 7 days and 14 days.

In two horses, transient increase in lameness occurred at day two, which resolved spontaneously.

All horses showed improved lameness grade, but none became sound. Further research is

required to titrate the dose and examine the longer-term efficacy of this promising treatment.

Intrabursal medication

Intrabursal medication with corticosteroid (100mg methylprednisolone acetate) and hyaluronic acid (20mg) has shown good results, depending on the pathology identified using MRI.

Looking at “navicular syndrome” as a whole (including navicular bone injury, collateral sesamoid desmitis, deep digital flexor tendonitis, navicular bursitis, adhesions and so on) a favourable response was seen following intrabursal medication, with 75% of 101 horses returning to previous use for mean nine months and 35% sound when re-examined.

Shorter duration of pre-existing lameness and primary DDFT injuries/bursitis resulted in a better prognosis. Some of these horses underwent pressurisation of the navicular bursa as an adjunct treatment to breakdown adhesions between the collateral sesamoidean ligaments, DDFT and navicular bone.

Interestingly, horses that immediately returned to work had a significantly poorer prognosis (71% returned to work for mean 5.5 months, only 17% sound at follow-up) compared to horses that underwent a 6-month rest and rehabilitation programme prior to return to work (84% returned to intended use for mean 19 months, 71% sound at follow-up).

Horses with multiple pathologies had the poorest prognosis, whereas all horses with navicular fibrocartilage erosion only returned to previous use.

Surgical

Navicular busoscopy

When extensive fibrillation or tearing of the dorsal aspect of the DDFT is evident on MRI, bursoscopy to assess the damage and debride loose tendon fibrils is effective in reducing inflammation responsible for bursitis. Using a transthecal approach, the theory also exists that the pain from distension of the bursa is reduced by decompressing the bursa, allowing fluid into the digital flexor tendon sheath.

Whether this communication remains long-term is debatable. If we are to return to the navicular suspensory desmotomy, should we be performing this bursoscopically like the majority of other desmotomies nowadays?

Navicular decompression

Decompression of the navicular bone by drilling multiple (three) holes in a proximal to distal direction under arthroscopic guidance has been proposed as a treatment for primary navicular

bone pain, targeting increased intraosseous pressure (IOP) caused by medullary sclerosis or osteonecrosis.

The study (Jenner and Kirker-Head, 2011) was based on experience from human medicine where IOP is associated with increased signal on short tau inversion recovery MRI images consistent with a “bone marrow oedema pattern” and decompression resulted in significant reduction in maximum IOP. Reports are limited to experimental work in healthy horses, so no conclusions can be drawn regarding its efficacy.

Mild short-lived lameness was associated with the procedure, but no long-term lameness or adverse effects. It would stand to reason this technique would only be appropriate for primary navicular bone pathology seen on MRI scan, and not necessarily have as much beneficial effect on horses with a combination of conditions (navicular syndrome).

In human patients treated for osteonecrosis with decompression drilling, a 73% success rate with mean 11-year follow-up was reported, indicating the persistent long-term effect of the surgery. Although difficult to draw any parallel conclusions from this human work, this technique, if proved to be successful in clinical cases of navicular oedema/inflammation, might provide the single best long-term treatment for the condition.

Neurectomy

When all else fails, palmar digital neurectomy remains a simple and effective way of returning a horse to soundness. It is not a technique to be taken lightly and extensive counselling of the owner prior to surgery is essential.

Surgical complications (for example, neuroma formation), altered gait (tripping), careful management (to avoid solar penetration or invasive infection) and catastrophic breakdown injuries (DDFT rupture and distal interphalangeal joint [DIP] luxation) need to be discussed. With accurate diagnosis, despite treatment failure, the feasibility of neurectomy can be more carefully assessed, leading to a reduction in catastrophic breakdown injuries or potentially performing unilateral neurectomy where applicable, reducing all other risks.

Palmar foot pain is still an area attracting a huge amount of research. We by no means have the final answers to any of the big questions regarding aetiopathogenesis, diagnosis, treatment or prognosis, but we are getting closer. This article highlights the need for a large-scale multicentre study looking at treatment response to specific therapies for specific conditions and, overall, for the blanket PFP/navicular syndrome/PTAS so we can finally offer a more accurate prognosis and a targeted treatment.

In the interim, rest, corrective shoeing, bisphosphonates and intrabursal medication with

corticosteroid and hyaluronan all delivered in combination should give a fairly good long-term prognosis, with bursoscopic surgery reserved for refractory cases or those where dorsal margin DDFT damage seems prevalent.

- **Some drugs mentioned are used under the cascade**

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