Emerging Diseases

Chaired by Andy Durham

Sponsored by Equine Veterinary Journal

08.30–08.55

The 2006 outbreak of equine infectious anaemia in Ireland: New insights into an old disease

A. Cullinane*, M. Quinlivan, F. Cook†, M. Nelly, H. Patterson, R. Kenna, S. Gildea, M. Garvey, P. Lyons, M. Flynn, P. Galvin, M. Neylon and K. Jankowska

Virology Unit, Irish Equine Centre, Johnstown, Naas, Co. Kildare, Ireland; and †Gluck Equine Research Centre, Department of Veterinary Science, University of Kentucky, Lexington, Kentucky 40546, USA.

Equine infectious anaemia (EIA) is caused by a lentivirus of the *Retroviridae* family. The high incidence of outbreaks of EIA in Romania indicates that within the EU there is a risk of virus spreading from Eastern Europe. In 2006 an outbreak occurred in Ireland, the source of which appears to have been contaminated plasma that was imported from Italy. More than 30 cases were confirmed by virological testing. Two clusters were identified linked to a veterinary practice and a referral hospital. Equine infectious anaemia is a notifiable disease and more than 1500 horses were subject to movement restrictions by the Department of Agriculture. During the 12 months after the disease was identified our laboratory tested over 57,000 samples for EIA. The comparative sensitivity of the agar gel immunodiffusion (AGID), 4 different ELISAs and the immunoblot was evaluated. The nucleotide sequence of the gag gene of this virulent strain of EIA virus (EIAV) was determined and 2 specific and sensitive real time PCR and RT-PCR assays were developed. Viral RNA and DNA were detected by RT-PCR and PCR respectively in all *post mortem* tissues examined from infected animals. Viral RNA and DNA were also detected in nasal secretions, genital swabs and saliva (RNA only) for the first time. Horses remain EIAV carriers for life thus the detection of seropositive horses and their removal from the population is the basis of effective disease control and eradication. Control measures within the EU are based on an incubation period of up to 90 days and in the United States on a period of 60 days. The serological data from the Irish outbreak suggests that the usual incubation period for this strain of EIAV is approximately 37 days but may be longer than 60 days in a minority of cases. One horse had a possible incubation period of between 120 and 157 days. The data accumulated during this outbreak has important implications for the prevention and control of a notifiable disease.

NOTES

08.55–09.20

Glanders

Ulrich Wernery

Central Veterinary Research Laboratory, PO Box 597, Dubai, United Arab Emirates.

Glanders is a disease of equidae which has been eradicated from most countries in the world. However, it is a re-emerging disease and still exists in parts of Asia, Africa and South America, and odd cases are reported from time to time in other countries. No cases have been recorded in the Middle East for many years. The last cases reported in this region were in Turkey in 1998 (OIE, annual report). The Turkish Veterinary Authorities carried out large scale testing of equidae in 2000/2001 using CFT and mallein tests. Positive animals were destroyed, and no cases have been reported since. In 2004 a glanders outbreak occurred in horses in the UAE in post import facilities. The disease was reported to the OIE. In 2009–10 new outbreaks occurred in Bahrain and Kuwait. The sources of all 3 outbreaks have not yet been determined. Another outbreak is currently observed in India and some lions were killed in Tehran zoo suffering from glanders. Re-occurrence of glanders has also been reported from Brazil and Pakistan. Intensive research which followed the outbreaks of glanders in Dubai and other GCC states resulted in the adoption of serological tests other than the CFT recommended by the OIE. By way of acknowledgement of these contributions, the OIE designated the CVRL as an OIE reference laboratory for glanders.

From a scientific point of view, disease events over the past several years have significantly increased our knowledge and concern over glanders and the risk of more widespread global distribution of the disease.

During the presentation the different glanders lesions will be presented and the methods to diagnose the disease will be discussed.

NOTES

09.20–09.45

Piroplasmosis

Paul Phipps

Wildlife Zoonoses and Vector Borne Disease Research Group, Virology Dept., Animal Health and Veterinary Laboratories Agency, New Haw, Addlestone, Surrey KT15 3NB, UK.

Equine piroplasmosis is a peracute, acute or chronic tickborne disease of equids, including horses, asses, mules and zebras which is caused by infection with 2 distinct intra-erythrocytic protozoan parasites, *Babesia caballi* and *Theileria equi*. Symptoms of acute disease may include fever, icterus, colic, diarrhoea, haemolytic anaemia and oedema of the lower body and limbs as well as respiratory distress arising from pulmonary oedema. Chemotherapeutic treatment is difficult and, due to the toxicity of babesicides, care must be taken in the administration of the correct dosage. Recovery results in a long lasting, low grade persistent infection with absence of clinical signs thus providing a reservoir for infection of tick vectors. Twelve species of hard ticks from 3 genera, *Dermacentor*, *Hyalomma* and *Rhipicephalus* have been identified as vectors of equine piroplasmosis and tick to tick transmission is both transovarial and transtadial in the case of *B. caballi* whereas *T. equi* is transmitted transtadially only. The disease has a wide geographical range according to the distribution of tick vectors and is endemic in Africa, South and Central America, The Middle East, Asia and Southern Europe. Equine piroplasmosis is reportable to The World Organisation for Animal Health (OIE) and since 2009 has been a notifiable disease in the Republic of Ireland. Some countries, where competent tick vectors exist in the absence of disease transmission, require certification of a negative serological test result using either Competition Enzyme Linked Immunosorbent Assay (cELISA), Indirect Fluorescent Antibody Test (IFAT) or Complement Fixation Test (CFT) as a pre-import requirement. AHVLA has been an internationally recognised equine piroplasmosis testing laboratory for over 30 years and has carried out pre-export testing on behalf of clients in the UK, the Republic of Ireland and throughout Europe as well as serological surveys for clients worldwide during this time. As part of this international trade testing, AHVLA has found evidence of maternally derived anitibodies in foals of foreign born dams as well as both iatrogenic and transplacental transmission of equine piroplasmosis in horses resident in the UK. The UK has always reported freedom from autochthonous equine piroplasmosis due to a lack of competent tick vectors. Recent evidence, however, suggests that the geographical range of *Dermacentor reticulatus*, vector of *Babesia caballi*, is expanding both in Northern Europe and within the UK.

NOTES

09.45–10.10

Hendra and related viruses

James Wood
University of Cambridge, Department of Veterinary Medicine, Madingley Road, Cambridge CB3 0ES, UK.

Emerging diseases: Hendra

Hendra virus first emerged in Brisbane, Australia in 1994, in an outbreak of severe respiratory disease in a racehorse training yard that was first thought to be associated with poisoning rather than any contagious disease. However, the disease spread around the training yard and was quickly associated with a viral infection thought initially to be caused by a novel morbillivirus; during the outbreak, the trainer, who nursed some dying horses intensively, died of the same infection and the vet and stable hand were also infected, the latter developing a flu like illness, before recovering.

A massive investigation of the source of the infection followed on from this outbreak and during these investigations it became clear that 2 other horses had died on one farm in Queensland earlier that year; the farmer, who had assisted in a *post mortem* examination, had also developed an illness, which turned into a relapsing encephalomyelitis from which he died the following year.

After nearly every other species living in Queensland had been ruled out, it turned out that the 3 species of flying fox common in Queensland all had significant antibody prevalences to what had by then been (briefly) termed equine morbillivirus. More detailed phylogenetic investigations of this virus revealed that, while related, it was from a novel paramyxovirus family and the virus was termed Hendra virus, after the suburb in Brisbane where the training yard had been. The virus was classified as Biosecurity Level 4 pathogen, the highest classification, shared with infections like Ebola and Marburg viruses.

There was a considerable international interest in the outbreak and for a few years, all horses leaving Australia had to be seronegative and free from signs of disease for 2 weeks before flying. These restrictions have now been lifted. The disease also resulted in the first formal UK based risk assessment of what you should do if you suspect Hendra (or another BSL4 pathogen) in a large animal. Whole new, minimally invasive *post mortem* protocols had to be devised.

After 1994, 5 years passed with no further equine cases, until 1999 when one other equine case occurred, again in Queensland. Also in 1999, but in Malaysia and Singapore, there was a massive outbreak of a similar virus in pigs, pig farmers and slaughterhouse workers. The respiratory infection in pigs, rapidly transmitted and associated with coughing, caused 265 cases of viral encephalitis, involving 105 fatalities in humans. The causative virus, closely related to Hendra, was termed Nipah virus and it shared with Hendra large fruit bats species as its reservoir hosts.

Since 1999, nearly 50 horses and 4 people have been diagnosed and have died from Hendra virus in Australia, nearly all in Queensland. It seems that, if anything, the frequency of spillover events has increased over the period. The presence of infected host populations, such as in City Botanic Gardens in Melbourne and Sydney has been associated with a great number of misgivings.

Considerable efforts have been made to understand the dynamics of the infection in the reservoir species and why the infection has only recently emerged and why the intensity of spread has apparently increased. There is great interest in the way that flying foxes and humans are apparently living more and more closely together and reportedly on a year round basis. Previously it was thought that flying fox 'camps' were only occupied on a seasonal or sporadic basis. It remains unclear why nearly all the spillover events have been in Queensland, when there are as many (infected) bats and horses in New South Wales.

Preventing disease in equine veterinarians has been a major programme of Equine Veterinarians in Australia and the Department of Primary Industries in Queensland (http://www.dpi.qld.gov.au/4790_2900.htm) and all vets now carry around personal protective equipment to wear when they suspect the disease. The last vet to die from Hendra simply failed to put it on. Progress involving collaborations between Australian and American scientists is now being made towards developing a vaccine against Hendra.

Direct bat to human spillover occurs almost annually in Bangladesh and recent work has demonstrated the presence of a related virus in a common fruit bat in Africa. This talk will explore recent advances in the context of disease emergence, surveillance and prevention.

NOTES

Orthopaedic Outcomes

Chaired by Nigel Woodford

13.30–13.55

Surgical management and outcome of stifle disorders

Michael W. Ross

University of Pennsylvania, New Bolton Center, 382 West Street Road, Kennett Square, Pennsylvania 19348, USA.

Effective surgical management of horses with stifle pain requires accurate diagnosis, but there are no pathognomonic clinical signs. Shortened cranial phase of the stride ('bunny hopping gait' in young horses with bilateral stifle pain), variable degrees of lameness, effusion of one or more joint compartments, positive response to upper limb flexion and other manipulative tests are common clinical signs but diagnostic intra-articular analgesia is essential for diagnosis in mature horses, regardless of the presence of other clinical signs or imaging abnormalities. Stifle pain is a common default diagnosis and diagnostic analgesia, blocking one or more joint compartments, is mandatory. The lateral femorotibial (LFTJ) is independent of the medial femorotibial (MFTJ) and femoropatellar (FPJ) joints. Radiographic and ultrasonographic images are essential in mature horses to identify joint space, osteophytes, subchondral defects, fragments, meniscal and meniscal ligament injuries. Scintigraphy has low sensitivity and computed tomography and magnetic resonance imaging are emerging (not currently available to me). Radiology alone is often diagnostic in young horses with lameness and effusion and manifestations of osteochondrosis (OC) such as osteochondritis dissecans (OCD) of the femoral trochlear ridges or osseous cyst-like lesions of the femur or tibia are often seen. Oblique radiographic images should be obtained to evaluate the medial femoral condyle and to pinpoint fragments in the caudal pouches of the FTJs.

The FPJ and the cranial compartments of the MFTJ and LFTJ can be evaluated separately, if desired; the horse is most commonly positioned in dorsal recumbency. I routinely use fluid irrigation (gas insufflation can be used) and a single portal approach for routine evaluation of the stifle joint, with an initial arthroscopic portal between the middle and medial patellar ligament. Depending on the site of the lesion or if comprehensive examination is required, the septae between the FPJ and each the MFTJ and LFTJ are removed easily using a synovial resector (can be done manually) creating a single joint compartment. This approach is more versatile and consistent than using individual compartment approaches. I struggle in evaluating the caudal compartments of the FTJs, approaches to which have been recently refined (Watts and Nixon 2006). A cranial intercondylar approach to the caudal pouch of the MFTJ (Muurlink *et al.* 2009) was described and can be performed without a switching stick using the single cranial approach and septectomy. Loose fragments in the FPJ most often accumulate in the extensive suprapatellar pouch and removal sometimes requires a long lavage cannula, long Ferris-Smith rongeurs or a separate more proximally positioned instrument portal. Intraoperative radiographs are used if size and number of fragments retrieved differs from preoperative assessment. A synovial resector facilitates removal of embedded fragments at the insertions of the cranial meniscotibial ligaments and various intra-articular blades can be useful to remove meniscal tears. I use microfracture sparingly since aggressive debridement and puncture through calcified cartilage into the subchondral bone can cause a subchondral cyst to occur when one did not exist preoperatively and the technique was not associated with success (Cohen *et al.* 2009). Impressive results were reported using mosaic arthroplasty (Bodó *et al.* 2004) and autologous fibrin laded with growth factors and chondrocytes (Nixon 2001).

Specific lesions

OCD lesions in the FPJ are debrided and loose fragments removed; excessive debridement and abrasion arthroplasty are avoided. Prognosis is poor with extensive LTR lesions, coexistent patellar chondromalacia and with intermittent patellar subluxation. Patellar fracture fragments, most common in horses negotiating fences (medial and proximal aspects) are removed; occasionally additional instrument portals are needed. Distal patellar fragments occur after medial patellar desmotomy and are easily removed using standard FPJ portals. Occasionally osteoarthritis (OA) of the FPJ is seen in middle-aged and older horses and prognosis is poor. For a long-term solution I still prefer to debride osseous cyst-like lesions of the medial femoral condyle. Reported success in horses 0–3 years of age (64%) (Smith *et al.* 2005) was similar to that reported for injecting corticosteroids directly into the cyst lining (67%) (Wallis *et al.* 2008). In racehorses choice of technique is often directed by owner/trainer influence - if the horse must train and race soon after the procedure I use arthroscopically assisted injection of corticosteroids (methylprednisolone acetate). Caution is used in operating on older horses with subchondral lucent defects since they usually have substantial OA and prognosis is poor (only 35% of horses >3 years of age returned to soundness [Nixon 2001]). Osteoarthritis of the MFTJ (less commonly the LFTJ) is a common problem causing lameness in older nonracehorses and racehorses. Arthroscopic evaluation in racehorses with severe OA is diagnostic but palliative and seldom curative; if racehorses have severe lameness, do not respond to intra-articular corticosteroids and have narrowing of the MFTJ space I discourage arthroscopic examination. Nonracehorses most commonly develop OA, with varying degrees of articular cartilage damage (early - soft, thin cartilage on the medial femoral condyle; later - full thickness cartilage loss and subchondral bone erosion, osteophyte formation). Coexistent meniscal fraying and tearing is seen in horses with OA, but I see very few horses with meniscal tears as the primary surgical lesion. I marvel at the extensive experience of Walmsley *et al.* (2003). Meniscal injuries most commonly occur in horses with other deterioration of the MFTJ or LFTJ and seem to be associated with rather than the cause of OA or lameness. There are rare exceptions, however. Prognosis in nonracehorses is adversely affected by age, severity of lameness, the presence of a large meniscal tear and the severity of radiological changes (Cohen *et al.* 2009). Few horses have solely a soft tissue lesion diagnosed at arthroscopy; lameness is usually caused by ongoing OA. Rarely, cranial meniscal ligament tears can cause substantial lameness.

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NOTES

13.55–14.20

Medical management and outcome of stifle disorders

Lisa A. Fortier

VMC C3-181, Cornell University, Ithaca, New York, 14853, USA.

Determining if medical or surgical treatments of equine stifle joint disorders are required depends entirely on the diagnosis which in turn is dependent on clinical history, lameness examination results including intra-articular analgesia responses and diagnostic imaging results. The sheer size of the equine stifle presents unique challenges for diagnostic imaging of all methods including ultrasonography, conventional radiography, CT and MRI.

When performing intra-articular (IA) analgesia for diagnostic purposes, it should be remembered that there is a minimal 74% functional communication of the medial femoral tibial joint with the femoropatellar joint in normal horses. In the few horses with OA that have been investigated, the femoropatellar joint communicated with both the medial and lateral femorotibial joints which was different from that of the contralateral 'normal' joint. A more recent study tested the hypothesis that the local analgesic agent mepivacaine diffuses between adjacent equine synovial structures in the hindlimb and with greater frequency than latex, gelatine dye or contrast media. Diffusion from the femoropatellar to medial and lateral femorotibial joints and between the medial and lateral femorotibial joints in both directions were 20/20 (100%). Diffusion from the lateral femorotibial to the femoropatellar joint was 18/20 (90%) and from the medial femorotibial to femoropatellar joints 17/20 (85%). The results suggested greater diffusion of mepivacaine between adjacent synovial structures than indicated in previous studies. Therefore, commonly performed intrasynovial local analgesic techniques in the hindlimb of the horse are not as specific as first thought. The take home point here is that when performing intra-articular anaesthesia of equine stifle joints, each synovial compartment needs to be injected separately to ensure that anaesthesia of the appropriate synovial compartment is obtained.

Our understanding of equine stifle disorders and equine stifle anatomy is continually increasing. This is particularly applicable with respect to soft tissue injuries of the stifle joint. There are very elegant studies describing in detail the radiographic appearance of soft tissue attachments of the stifle joint. Since radiography represents the majority of early diagnostic imaging, understanding the normal anatomic map will aid in the assistance of diagnosing soft tissue injuries of the stifle joint. Similarly, detailed ultrasonographic anatomy of the stifle joint has been reported and is probably the most practically sensitive method of understanding equine stifle soft tissue anatomy given the very limited capacity of performing MRI on equine stifles. Computed tomography has also been investigated as a means to understand disorders of the equine stifle. Regardless of the imaging modality employed, a very astute anatomic knowledge of the equine stifle is required to interpret the results.

Using a combination of clinical history, physical examination findings and imaging modalities, soft tissue injuries to the gastrocnemius, menisci, collateral ligaments, cartilage and cruciate ligaments can be diagnosed, even in heavily muscular breeds. Typically, soft tissue injuries will be treated successfully with conservative therapy such as rest, but biological therapies such as IRAP/PRP/stem cells should be considered to reduce pain and hasten return to function. There are no large clinical reports detailing outcome of medical therapy for disorders of the equine stifle, so the indications for choice of therapy are typically extrapolated from other anatomic sites, such as the carpus.

Finally, there are some stifle disorders that are not amenable to medical therapy including OCD lesions of the distal femoral trochlear ridges in horses showing accompanying clinical signs such as lameness or synovial effusion. Also, for horses with subchondral bone cysts in the distal femoral condyle or proximal tibia that do not communicate with the articular cavity, medical management is not a viable option and surgical intervention should be pursued.

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NOTES

14.20–14.45

Management and outcome of ulnar fractures

Dean W. Richardson

New Bolton Center, University of Pennsylvania, Kennett Square, Pennsylvania 19348, USA.

Introduction

The equine ulna provides an almost pure opportunity for application of the tension band principle because the bone is vestigial distally and strongly loaded in a fairly uniform vector by the triceps insertion on the olecranon process. Wire is extremely strong in tension so even minimal fixation along the tension surface of the caudal ulna can be adequate fixation for many fractures. The advantages of the technique include minimal instrumentation, lower implant costs, possibly smaller incisions and the complete elimination of possible iatrogenic damage to the ulnar articular surface. In young horses, the tension band wiring technique also minimises the risk of interference with growth of the proximal radius.

Plate fixation is unquestionably the more versatile and stronger technique. The principle of the tension band still provides such an enormous advantage that smaller (narrow) plates and smaller screws are still mechanically adequate in even the largest horses.

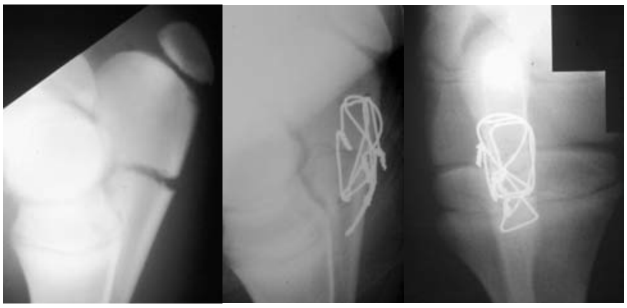

Fig 1: Wire alone technique.

Fig 2: Pins and wire technique.

Case selection for tension band wiring

Although there is no absolute weight limit for this technique, there are distinct limitations for its use dictated by the configuration of the fracture and there are many occasions when plating is both easier and more reliable. The optimal indication for a wiring technique is in the young horse (<6 months) where plating techniques might compromise the proximal radial growth plate. Plating an ulnar fracture using screws affixed to the radius should be avoided if at all possible in any foal less than 6 months old. Tension band wiring is the technique of choice for Salter-Harris I fractures of the proximal olecranon and is highly recommended for repair of simple, minimally displaced fractures at or distal to the level of the humeroradial articulation in young horses. The technique is NOT well suited for comminuted or intrinsically unstable fractures and is usually not used in adult horses with fractures proximal to the joint level if there is enough bone to insert 3 screws into the proximal fragment.

Plating techniques are preferred for larger horses, comminuted fractures and most fractures proximal to the humeroradial articulation.

Surgical approach

A standard approach to the ulna is used between the ulnar head of the deep digital flexor and the *ulnaris lateralis* muscles. With simple wiring of the mid ulna, a very small incision is required. If a pin and wire technique is necessary, the incision is carried proximal to the point of the olecranon. Both pins and wires can be placed in the proximal olecranon without extensive dissection of the triceps insertion. If a plate is bent over the top of the olecranon, do NOT remove the triceps insertion. Get the plate through a narrow slit and work hard to place screws through the soft tissues. (A locking plate is best because it does not have to compress to the bone in order to provide stability.)

Technique

Wire alone (for fractures at or below the humeroradial joint level) (**Fig 1**)

The fracture is held in reduction digitally or with forceps. If there is any obliquity to the fracture plane, a 3.5 or 4.5 mm lag screw is inserted. A 2.5 mm (2.7 or 3.2) bit is used to drill holes transversely through the ulna at least 2–3 cm proximal to and distal to the fracture. Figure-8 loops of wire are passed through the holes and tightened with a wire tightener or vice grips. At least 2 loops of wire are used even in small foals. Usually 3 or 4 wires are used in yearling or adult horses. The wire should be at least 1.25 mm diameter and in larger horses, 1.5 mm wire is used.

In simple oblique fractures extending distal to the joint, one or 2 screws can be used in combination with a simple tension band wiring technique. This can be done through a very small incision.

Pins and wire (for fractures proximal to the joint) (**Fig 2**)

A pin held in a Jacobs chuck is inserted in a proximal to distal direction through the proximal fragment being careful to both centre the pin and allow enough room for a second pin to be placed. The pin size is dependent on bodyweight. In small foals, 3 mm pins are adequate but in an adult horse up to a $^3/_{16}$ inch pin might be used. The 2 pins do not have to be the same size. The fragment is reduced through manipulation (elbow extension with varying amounts of rotation) and the fragment skewered onto the distal bone. It is highly desirable not to completely penetrate the distal cortex, only to engage it securely. Complete penetration can lead to the pin migrating distally in the limb over a period of weeks to months. The second pin is then inserted in a similar manner. One or 2 transverse 2.5–3.2 mm holes are drilled through the ulna approximately 3 cm distal to the fracture. At least 3 wires (sized as above) are passed through the holes and around the proximal protruding pins. A 14 gauge 1.5 or 2 inch

Fig 3: Plating technique.

needle is helpful for wire passage. The wires are tightened alternately to help avoid any tendency to shift the fracture. The pins are cut (*Don't twist the handles of the cutters!*) and tapped down with a mallet and nail set, leaving 6–8 mm above the bone surface. The pin and wire technique can also be used in conjunction with one or 2 lag screws if the fracture is oblique BUT care must be taken to leave enough room for the pins.

Plating (**Fig 3**)
Ulnar fractures provide the easiest and most successful long bone plating opportunities in horses. In most cases, a single narrow plate will suffice if it is properly positioned along the caudal surface. There are some guidelines to make it easier:

1. Always attempt to place a lag screw across any oblique fracture before applying the plate. This will provide interfragmentary compression (additional stability) and make it much easier to contour and place the plate.
2. Be meticulous with plate positioning. Common errors include placing the plate too laterally. This makes it very difficult to fully engage the proximal fragment of the olecranon process with long screws. A very common mistake is to have the drill/screw exit the concave medial side of the olecranon. ALWAYS check the position of both ends of the plate before placing the second screw. It is a major error if the distal part of the plate is not properly aligned with the slender ulna.
3. Do not excessively dissect the soft tissues from the proximal ulna. Especially with locking plates, it is best to leave the soft tissues as fully intact as possible. Even with smaller proximal fragments, do not try to wrap the top of the plate too far over the top of the olecranon.
4. Pay close attention to the joint. Don't drill into the articular surface and definitely don't place a screw through it.
5. Try to engage screws in the most cranial and proximal portion of the olecranon whenever possible, especially in fractures with small proximal fragments.
6. In horses less than a year old, try not to engage the radius with a screw. If you must do so to achieve stability, remove that screw as soon as possible after early healing is adequate.
7. Do not allow wounds over the fracture to deter an attempt at repair. Stability is a great means of minimising the risks of contamination.
8. Double plating with the second plate on the lateral surface is an excellent technique for comminuted fractures.
9. Locking plates have major advantages.

Closure and recovery
The closure with either technique is straightforward and reliable. Drains are neither necessary nor desirable unless there is an unusual amount of soft tissue damage. Recovery from general anaesthesia is a particularly high risk with this fracture so special care is always given. Foals should be physically restrained and lifted to their feet. Any special recovery system such as a pool or sling should be used if available. If not, a deep mat with head and tail rope assistance is advisable.

Results

We reported the results of wire fixation (+/- pins and screws) in 22 horses with ulnar fractures (Martin *et al.* 1995). Ages of these horses ranged from 2 weeks to 12 years with a median of 4 months. Fractures healed in 18/22 horses (82%). Long-term follow-up was obtained on 17 horses. Of these, 13 (76%) became athletically sound.

The reported results for plate fixation of ulnar fractures are quite favourable both in terms of fracture healing and athletic soundness. Simple fractures as well as those more distal in the ulna have the best prognosis. Displaced proximal physeal (SH-I) fractures are among the most difficult to repair and severely comminuted fractures can be challenging, especially in large horses.

Nonsurgical treatment of ulnar fractures should only be recommended in minimally displaced fractures distal to the humero-radial articulation. Nonunions/pseudarthoses are common in displaced and unstable ulnar fractures.

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NOTES

14.45–15.10

Tearing of the calcaneal insertions of the superficial digital flexor tendon: Treatment and outcome

Ian M. Wright

Newmarket Equine Hospital, Cambridge Road, Newmarket, Suffolk CB8 0FG, UK.

The hindlimb superficial digital flexor arises from the supracondylar fossa of the femur as a thick round tendon which is incompletely covered with muscle as far as the proximal one-third of the crus. It is intimately attached to the cranial surface of the gastrocnemius and in the distal one-third of the crus it winds around the medial surface of the gastrocnemius to lie caudally. From this point the tendon mediolaterally widens and over the apex of the calcaneus forms a curved fibrocartilagenous cap with a cranial concavity. The calcaneal insertions of the superficial digital flexor emerge from the medial and lateral margins of the cap. The calcaneal bursa is found beneath the SDF tendon from the distal quarter of the crus to the middle of the tarsus. This communicates consistently with a small bursa found between the tendon of insertion of gastrocnemius and the calcaneus. The calcaneal insertions of the SDFT are subsynovial with regard to the calcaneal bursa and form part of its abaxial walls. Distal to the fibrocartilagenous cap the SDF tendon narrows as it passes over the long plantar ligament towards its ultimate insertion via the plantar fibrocartilage on the distal extremity of the proximal phalanx and proximal extremity of the middle phalanx. Its action is to flex the digit and extend the hock which are considered largely to be mechanical effects resulting from the action of other muscles on the stifle joint. The calcaneal insertions are thus critical to its role in providing the caudal arm of the reciprocal apparatus.

This paper documents clinical, ultrasonographic and endoscopic observations in 19 horses with disruption of the calcaneal insertions of the SDF tendon including 7 horses with unstable subluxation of the tendon. These are a cohort of 46 horses referred to Newmarket Equine Hospital between 2005 and 2009 for investigation of lameness accompanied by distension of the calcaneal bursa(e). The animals were used for a variety of purposes; 9 for general riding activities, 4 for flat racing, 3 were eventers, 2 were used for National Hunt racing and one was a hunter. In horses with unstable subluxation of the SDF tendon, clinical signs were all of acute onset with severe lameness and marked anxiety. Six SDF tendons subluxated laterally and one medially. All were manually reducible but displaced readily when animals walked. Horses with unstable SDF tendons had moderate or marked distension of the calcaneal bursa and in 4 there was an additional acquired subcutaneous bursa. Horses with stable SDF tendons exhibited lameness which varied between 2 and 5 out of 10 accompanied by varying degrees of distension of the calcaneal bursa.

In horses with stable SDF tendons, ultrasonography demonstrated disruption and/or heterogeneous tissue adjacent to the calcaneal insertions of the SDF tendon in 9 horses. Extensive disruption of the SDF tendon fibrocartilage was recognised ultrasonographically on the contralateral side to displacement in all horses with unstable subluxation of the SDF tendon. At endoscopy of horses with stable SDF tendons, 6 had tears of the lateral and 6 of the medial calcaneal insertions with torn fibrils extruded into the bursa. Tears varied in proximodistal length and axial/abaxial thickness but all were partial and the fibrocartilagenous cap of the SDF tendon was intact in all of these cases. In these horses the disrupted tendon fibrils and any associated granulomata were removed and the defects debrided to minimise exposure of the synovial environment to disrupted collagenous tissue.

All horses with unstable subluxation of the SDF tendon had extensive proximodistally orientated defects in the fibrocartilage on the contralateral side to the displacement, i.e. 6 medially and one laterally. Defects involved almost the entire proximodistal length of the fibrocartilage but in each case there was proximally and distally a bridge of intact tissue. When the tendon was manually repositioned onto the calcaneus, defects in the fibrocartilage were reduced and when the tendon subluxated the defects opened such that the calcaneus protruded into the intervening space creating a ‘button like’ arrangement that restricted further abaxial displacement of the tendon. At endoscopy the remaining proximal and distal attachments of the fibrocartilage to the SDF tendon were resected together with the associated calcaneal insertions before the free mass of fibrocartilage was removed. In most cases, once this tissue was removed, the tendon and fibrocartilage remaining could readily be displaced by digital manipulation and remained in a stable subluxated position. When this was not possible, a further section of fibrocartilage was resected.

Nine of 12 horses with stable SDF tendons returned to work at or greater than the previous level of performance. Six of 7 horses with unstable SDF tendons returned to work of which 4 were considered to have returned to their previous level of performance. Two of these subsequently sustained similar injuries to their contralateral SDF tendons 23 and 31 months post surgery. Both were managed in a similar manner and again returned to work.

In the literature to date, lesions of the calcaneal insertions for the SDF tendon have been restricted to injuries resulting in displacement and/or instability of the tendon. The results of this study suggest that if these insertions are disrupted in part that stability is maintained but that lameness accompanied by distension of the calcaneal bursa result. Removal of the disrupted tissue was associated with clinical improvement and a return to work in a majority of horses.

Although a well recognised and documented condition, records of only 8 horses with lateral (Scott *et al.* 1982; Meagher and Aldrete 1989; Stashak 2002) and one with medial (Reiners *et al.* 2000) displacement of the SDF tendon could be found in the literature. There is one further case of lateral instability associated with fracture of the calcaneus (Scott 1983). Instability has been considered to result from disruption of the retinacular insertions of the SDF tendon to the calcaneus (Dik and Leitch 1995; Reiners *et al.* 2000; McIlwraith 2002; Stashak 2002; Latimer 2004; Auer 2006). However, there has been no morphological description of the lesions involved and the results of the current study suggest that the above may be a simplification of the pathogenesis. In all cases of instability evaluated in this series there was also substantial longitudinal disruption of the fibrocartilage and based on this observation, it was hypothesised that the surgery would lead to stable subluxation of the SDF tendon. In the author’s experience, the most critical feature of displacement of the SDF tendon from the calcaneus has been the stability of the subluxation. Those horses which present with the tendon permanently (usually laterally) subluxated can, with conservative management, return to useful work. By contrast those with unstable subluxation, in which the tendon spontaneously reduces and displaces remain permanently compromised and frequently with continued anxiety.

Although there are few reports of reduction and surgical stabilisation in the literature (Scott *et al.* 1982; Meagher and Aldrete 1989; Reiners *et al.* 2000; Stashak 2002), representing a total of 9 documented cases, this has generally been regarded as the treatment of choice for unstable displacement. Results have often been disappointing with the prognosis ascribed to the ease of reconstruction of the torn retinaculum (Dyson 2003).

In the current study, 2 discrete injuries have been identified. The first involves intrathecal (with the respect of the calcaneal bursa) tearing of the insertion of the calcaneal insertions of the SDF tendon. The second occurs in conjunction with disruption of the fibrocartilagenous cap of the SDF tendon and results in unstable subluxation of the same. Evaluation and understanding of the tissue damage involved has permitted more rational intervention than previously available and this is supported by the results obtained. Surgery aims to remove disruptive and inhibitory tissue thus creating a 'stable' subluxation.

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NOTES

Orthopaedic Outcomes

Chaired by Nigel Woodford

16.00–16.25

Management and outcome of septic synovitis in the foal

Richard Payne

Rossdales Equine Hospital, Cotton End Road, Exning, Newmarket, Suffolk CB8 7NN, UK.

Introduction

Septic synovitis is a serious and potentially life-threatening condition. Successful management depends upon early accurate diagnosis and immediate treatment. Any delay in these steps may significantly worsen the prognosis for survival.

In the neonatal foal such infection is most often the result of haematogenous spread of bacteria. Bacteria may enter the synovial cavity direct, or they may infect bone in the physeal region, causing destructive osteomyelitis, with subsequent erosion to the articular surface of the joint.

Bacterial entry via the umbilical stump is the most likely source for haematogenous spread, but the possibility of entry via the gastrointestinal tract and respiratory system should not be ignored. For these reasons a thorough and detailed physical examination should be carried out.

The most common Gram-negative pathogens are *Escherichia coli*, *Actinobacillus* spp, and *Salmonella* spp. The more common Gram-positive organisms are *Staphylococcus*, *Streptococcus* and *Rhodococcus* spp. Although rare, mycoplasma and chlamydial infection has also been reported.

Risk factors

Foals which are ‘immunologically naïve’, are at greater risk of development of septic synovitis. This may be as a consequence of being premature or dysmature, or more commonly a result of failure to absorb sufficient colostral antibody.

There also seems to be a ‘window of risk’ at 6–10 weeks of age, at a stage when passive immunity is waning and before sufficient endogenous antibody has developed.

Simple management factors, such as lack of attention to disinfection of the umbilical stump may also increase the risk of synovial infection.

Pathophysiology

Bacterial colonisation of synovial membrane results in a marked inflammatory reaction, with influx of serum protein and white blood cells (predominantly neutrophils). Fibrin deposited within the synovial cavity further impairs synovial membrane function and harbours bacteria.

Diagnosis

A diagnosis is reached by putting together a ‘jigsaw’ of information. This includes the history, physical findings and results of synoviocentesis and imaging.

Foals are frequently affected in the first 30 days of life. Clinical signs include acute lameness, synovitis, local heat, swelling and pain, together with pyrexia. In some cases physical findings are subtle. The umbilicus warrants careful examination and an ultrasound scan can be helpful to assess the stump, arteries and vein. This should be done *before* joint lavage, so that if umbilical removal is deemed necessary, it can be carried out under the same anaesthetic.

Synoviocentesis

This should be carried out after standard aseptic preparation. Adequate restraint is essential and mild sedation (e.g. butorphanol with detomidine), with the foal recumbent, may be helpful.

Synovial fluid should be analysed for total and differential white cell count and total protein estimation. Cytology should also be carried out.

A small ‘aliquot’ of synovial fluid should ideally be placed in a bacterial culture medium bottle. Successful culture can be frustrating, but it may help dictate appropriate antibiotic therapy.

A simple Gram-stain of synovial fluid may be helpful at least to demonstrate the presence of bacteria, and to determine whether there is Gram-positive or Gram-negative infection. The role of PCR techniques in detection bacteria in synovial fluid is likely to become increasingly important.

Imaging

This is an essential. Most commonly this involves radiographic examination. Where bone or physeal infection is involved, there may be a ‘lag period’ of several days (after the onset of clinical signs) before radiographic evidence can be demonstrated. Repeated radiographic examination is indicated particularly if there is a poor response to initial treatment.

Computed tomography scan (if available, and where economics allow) is an excellent way to look for early signs of bone infection. Nuclear scintigraphy, using radio-labelled leucocytes is also a helpful method for detecting physeal infection.

Treatment

Early aggressive treatment is essential to prevent permanent joint/synovial cavity dysfunction. The potential cost, risks, complications and prognosis should be discussed carefully with the owner in advance.

Systemic antibiotics

These are essential. Ideally these should be ‘broad spectrum’ and bactericidal - unless the results of culture and sensitivity allow therapy to be more specifically tailored. Gentamicin should be avoided if renal compromise is suspected. Intravenous antibiotics (via a catheter) may be preferable in neonates with small muscle mass.

Intrasynovial injection

Intrasynovial injection of antibiotics is also an effective means of increasing the local concentration of antibiotic in a joint. Gentamicin or amikacin are commonly used and may be injected at the conclusion of joint lavage.

The use of gentamicin impregnated polymethylmethacrylate beads or bioabsorbable gentamicin impregnated collagen sponge can also be effective. In selected cases the use of intraosseous antibiotics or regionally perfused intravenous antibiotics may also be worth considering.

Synovial cavity lavage

Synovial cavity lavage is essential to remove inflammatory cellular debris, degradative enzymes and bacteria. Polyionic buffered saline (e.g. Hartmann's solution) should be used. Lavage is accomplished by one of 2 methods.

i) Through and through lavage

Ingress and egress needles (or catheters/cannulae) are placed into the affected synovial cavity and it is flushed through. In small foals (<100 kg) this may be carried out under sedation.

Needle/catheter flushing is best used early in the course of infection (<48 h), before significant pannus and fibrin accumulates within the joint. If there is no improvement, or a lapse in infection, arthroscopic lavage should be considered.

In rare selected 'problem' cases indwelling polyurethane catheters may be left in a joint to facilitate repeated or continuous lavage. This carries a risk of retrograde infection entering the joint and requires very careful supervision and aseptic nursing.

ii) Arthroscopic lavage

This allows inspection of the joint as well as lavage and debridement. Fibrin and pannus may be removed, articular cartilage can be assessed and, if there is a seeding adjacent subchondral bone infection, debridement under arthroscopic control may be carried out.

Arthrotomy

In cases of chronic, nonresponsive septic arthritis, creation of open drainage may be considered (Schneider 1992b). High standards of post operative care will be required.

Post operative management

Careful and continual re-appraisal of the foal's condition should be made in the first few days post operatively. If there is any doubt about clinical progress, repeat synoviocentesis and radiography should be performed. The possibility of multiple joint involvements should always be kept in mind.

Short-term judicious use of NSAID's may be helpful in improving comfort and encouraging return to function. High doses should be avoided as they could mask clinical signs of pain, swelling and lameness. Where they are used, concurrent use of anti-ulcer medication (Omeprazole) is indicated.

Prognosis

Schneider *et al.* (1992b) reported a 45% survival rate for foals with septic synovitis. More recently 84% survival to discharge has been reported (Smith *et al.* 2004) in a group of 69 Thoroughbred foals, with 48% of these surviving foals achieving their intended purpose of racing. Early diagnosis and aggressive treatment offers the best chance of a favourable outcome.

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NOTES

16.25–16.50

Sacroiliac conditions - management and outcome

Leo Jeffcott

Faculty of Veterinary Science, University Veterinary Teaching Hospital, 410 Werombi Road, Camden, New South Wales 2570, Australia.

Sacroiliac problems are an important cause of poor performance in both equestrian and racing horses. Pain originating from the sacroiliac has long been recognised as the underlying basis for this, but specific diagnosis has traditionally been difficult to achieve. This has been attributed to the often low grade or chronic clinical signs and the inaccessible location of the joint. Consequently, diagnosis of sacroiliac dysfunction has largely been based on a process of elimination of other conditions in the thoracolumbar spine and hindlimbs. This is despite the many innovations in diagnostic imaging technology.

Functional anatomy

It is crucial to have a good appreciation of the structure and anatomy of the sacroiliac area to understand the likely pathogenesis of sacroiliac dysfunction. The joint is unlike any other diarthroidial joint in the body as it is designed for gliding movements, not weightbearing. It transfers the forces from the horse's quarters and hindlimbs to the thoracolumbar spine assisted by the interconnection of the ligaments, particularly the dorsal sacroiliac ligament and the sacrotuberous ligament. These ligaments are anchored through the thoracolumbar and gluteal fascia and the hamstring ligament complex.

Conditions involved in sacroiliac dysfunction

There are a wide range of conditions that are referred that are associated with sacroiliac problems:

- Jumper's Bump or Hunter's Bump
- Wing of ilium fracture
- Other sites of pelvic fracture
- Wing of sacrum fracture
- Distal hindlimb lameness (hock, stifle)
- Thoracolumbar injury or pathology
- Dorsal sacroiliac ligament desmitis
- Sacrolumbar disc injury
- Acute sacroiliac injury ± subluxation
- Chronic sacroiliac dysfunction

Chronic sacroiliac dysfunction

This commonly encountered problem has been well described in the literature and there appears to be 2 clinical manifestations. The first occurs in performance horses, still in work, where the main clinical findings are pain and poor performance, with the signs usually responsive to periarticular analgesia (Dyson and Murray 2003). Although pathological studies have not yet been done, it is assumed that the pain originates from the ligamentous structures surrounding the joint. The other condition is more debilitating and exhibits more marked gait changes with asymmetry of the hindquarters and associated pathological joint changes. However, these degenerative changes have been reported in racehorses that died or were subjected to euthanasia at the racetrack (Haussler *er al.* 1999), making interpretation in clinical cases of chronic sacroiliac dysfunction difficult. This presentation will concentrate on the latter problem as this is where the author's experience lies.

Pathological changes

At *post mortem* examination in horses with severe clinical signs of chronic sacroiliac dysfunction signs of cartilage degeneration accompanied by articular extensions are seen mostly on the caudal margin of the joint. The increased area of the joint could be up to 30% of the total joint surface area. These changes were presumed to have arisen from prolonged mild instability of the sacroiliac joint with a greater than normal range of movement.

Functional instability

The presence of these degenerative changes may indicate a response to altered mechanical load or *functional instability* and an increased neutral zone. They may also result in altered nerve stimulation at the medial aspect of the joint and altered contraction of the specialised middle gluteal in the horse.

Management and outcome

The treatment of functional instability at the sacroiliac remains largely empirical due to the lack of a complete understanding of its primary pathogenesis. It should always be based on clinical presentation, but includes use of rest, anti-inflammatory medication and exercise (Haussler 2003). Reduction of pain associated with sacroiliac dysfunction is important, but it should be noted that complete rest may be contraindicated due to the possible adverse effects of reduced pelvic and hindlimb muscle function and worsening functional instability of the sacroiliac joint.

Nonsteroidal anti-inflammatory medication can result in temporary improvement in performance (Jeffcott *et al.* 1985), however, it is ideal to rehabilitate the horse effectively, so improvement is consistent and lasting. A specific rehabilitation programme based on biomechanical findings may achieve this. Exercise to build up the muscles of the back and hindquarters is more appropriately recommended. The use of exercise to manage sacroiliac dysfunction has generally been nonspecific and there is a clear need for more specific muscle re-training to enhance functional stability of the region, and ultimately improve performance, in individual cases. The use of manual mobilisation and/or manipulation techniques which can be applied to the equine pelvis and sacroiliac joint may improve joint kinematics.

It is interesting to note that the clinical picture once recognised is not usually progressive. Treatment appears to be difficult and is usually aimed at overcoming the sacroiliac damage by progressively building up the muscles of the quarters and back. Improvement in muscular tone and fitness tends to counteract the clinical signs of poor hindlimb impulsion. In mild cases this type of management has been successful although once fit, the horse must be kept fit all the time and not allowed to rest or it would lose muscle tone and return to the original state.

Conclusion

Although sacroiliac dysfunction is still something of an anathema to equine veterinarians, many of the difficulties relating to diagnosis and management have greatly improved in recent years. Much progress has been made in research and there is a better appreciation of the functional anatomy of the sacroiliac joint and the type of pathological lesions that can develop. This is complemented by the improvements in clinical evaluation, local analgesia and the use of modern diagnostic imaging modalities. The current thrust of research in biomechanics, kinesiology and various forms of physiotherapy bode well for the future.

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NOTES

16.50–17.15

Management and outcomes of upward fixation of the patella

Oliver Crowe

B&W Equine Group, Byams Farm, Willesley, Nr Tetbury, Gloucestershire GL8 8QU, UK.

Upward fixation of the patella occurs when the horse fails to release the patella from its 'fixed' position, with the medial patella fibrocartilage hooked over the medial trochlea of the femur. The reciprocal apparatus is then locked preventing flexion of the limb. It has been reported that the condition is more common in horses with a straight hindlimb conformation as upward fixation requires stifle extension of approximately 145°, an angle more readily achieved when ordinary extension approaches that. Others report that the condition is common in Warmbloods, Thoroughbreds, Standardbreds and ponies and may have an hereditary predisposition.

Upward fixation occurs most commonly in young horses, those that are out of condition or have other causes of hindlimb weakness that prevents the quadriceps from effectively 'unlocking' the patella. It is also seen more commonly in animals that are stabled or that have suffered trauma to the stifle region. Lameness may develop if the condition becomes persistent.

Treatment involves identifying and correcting underlying causes of the condition and introducing an exercise programme to strengthen the muscles of the thigh. Horses should be carefully assessed for primary lameness (leading to disuse atrophy), neurological dysfunction, muscle disease or other causes of ill-thrift before commencing a training programme. If possible, horses should be turned out rather than box rested and nonsteroidal anti-inflammatory drugs administered if necessary to allow training. Applying hind shoes with heel wedges reduces the resting extension of the limb and decreases the chance of achieving 145° of extension.

Training regimes should be tailored according to individual circumstances but ridden trot up hills has been recommended. As young horses are most often affected, training may involve lungeing or long reining in horses that have not been backed. Increases in workload should be gradual to avoid exhaustion and any deterioration in lameness or other signs of disease carefully monitored.

Surgical intervention should be reserved for horses that fail to respond to conservative management and increased maturity. The traditional treatment by medial patella desmotomy has been shown to have a relatively high risk of complications. Tnibar proposed medial patellar ligament splitting to avoid those complications. The original description reported creating multiple longitudinal incisions in the proximal third of the medial patella ligament under general anaesthesia.

I find the procedure easier in the standing sedated horse with local analgesic injected subcutaneously. Identification of the ligament is easier and it is more easily penetrated by a number 63 tendon splitting blade when under tension. The procedure should be performed under strict asepsis as it is possible to penetrate the femoropatellar joint. I usually penetrate the ligament between 10 and 20 times, mostly in a craniocaudal direction from the cranial margin. Care should be taken as you move proximally not to direct the incisions axially toward the medial femur. Antimicrobials and phenylbutazone are administered perioperatively and all skin incisions left to heal by second intention. Horses are treated as outpatients and discharged with instructions to box rest overnight to allow a stable clot to form in each of the incisions. They should then be turned out and entered into a graduated exercise programme. Horses often suffer a transient (1–2 weeks) mild lameness and work should not be increased significantly until that has resolved.

Treatment results in the medial patella ligament increasing in thickness to approximately 2–3 times its normal size. So far there have been no incidents of patella fragmentation recognised following this procedure.

If medial patella ligament splitting fails to resolve the condition (at least 2 months of training should be undertaken before considered a treatment failure) then a medial patella desmotomy could be undertaken. The risk of patella fragmentation with subsequent femoropatellar osteoarthritis should be discussed with the owner prior to surgery. The procedure is carried out with the horse standing and local analgesic injected around the medial patella ligament. The horse should be box rested for at least 12 weeks post operatively with lead walking introduced during the second half of that period. The femoropatellar joint is destabilised by the procedure and even with careful post operative management, patella fragmentation has been seen in a number of cases. As such, this should be considered a treatment of last resort for refractory cases.

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