ANZCVS EQUINE CHAPTER PROCEEDINGS

2018

The Proceedings of the 2018 Equine Chapter Meeting at the ANZCVS Science Week

5 July - 7 July 2017

QT Gold Coast International Hotel
Gold Coast, Queensland, Australia

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ACKNOWLEDGEMENTS

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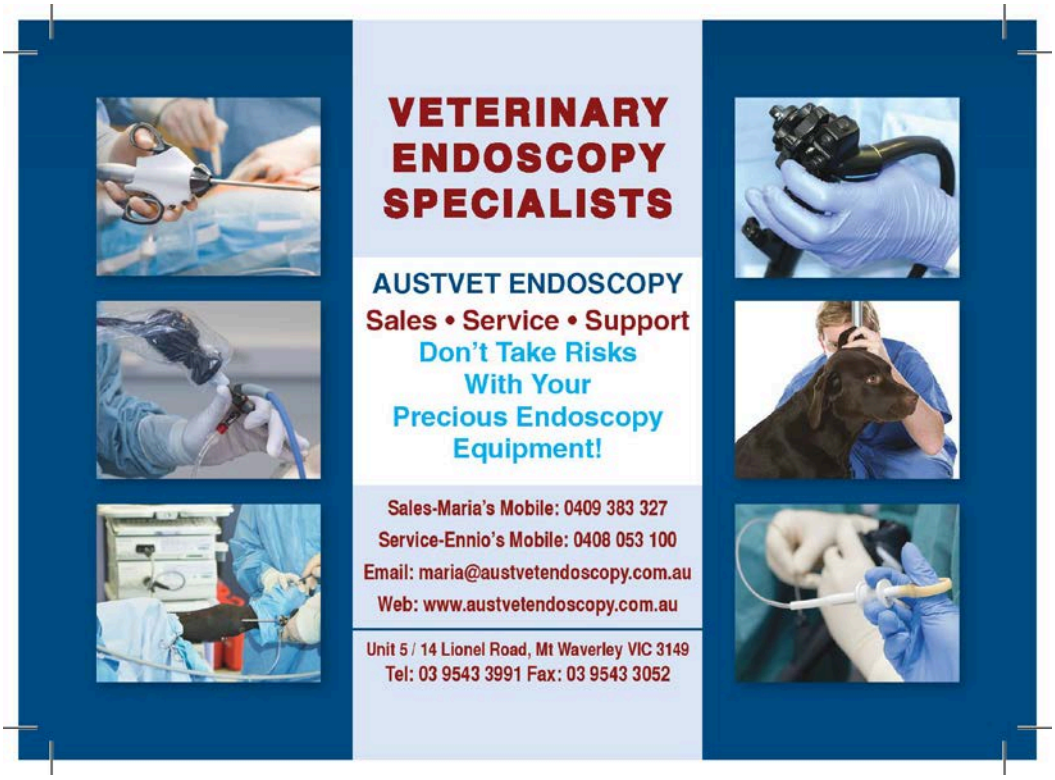

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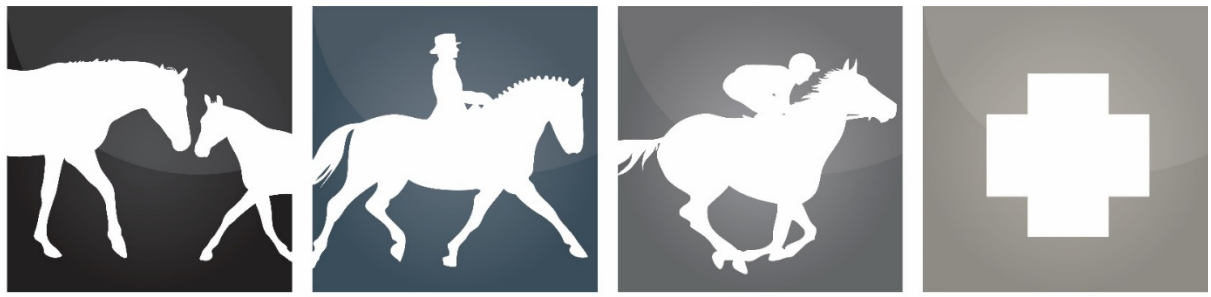

CAMDEN EQUINE CENTRE

A SPECIALIST SERVICE OF THE UNIVERSITY OF SYDNEY

SCIENTIFIC PROGRAM

Thursday 5th July 2019

8.00	**Plenary Forum: Pipeline 1**
9.00	Professor Chris Sanchez – Equine Pain Management University of Florida
9.20	
9.55	
10.00	**MORNING TEA IN TRADE DISPLAY AREAS**
10.30	Dr Catherine Russell – Colic in the Post Partum Mare Scone Equine Hospital
10.50	
11.25	
11.30	Professor Chris Sanchez – Equine Pain Management 'Evidence Based Therapy' University of Florida
11.50	
12.25	
12.30	**LUNCH – GROUND FLOOR AND FIRST FLOOR**
1.30	Dr Matt Stewart – Diagnosis and Management of Septic Arthritis University of Illinois
2.15	
2.15	Dr Andrew Dart – Emergency Treatment of Equine Colic University of Sydney
3.00	
3.00	**AFTERNOON TEA IN TRADE DISPLAY AREAS**
3.30	Professor Chris Sanchez – Ocular Manifestations of Systemic Disease University of Florida
3.50	
4.25	
4.30	Associate Professor Fatima Nasrallah – Pathophysiology, Diagnosis and Treatment of Traumatic Brain Injury in Humans Queensland Brain Institute and Centre for Advanced Imaging
4.50	
5.30	**EQUINE CHAPTER ANNUAL GENERAL MEETING**
6.00	**HAPPY HOUR – STINGRAY LOUNGE, GROUND FLOOR**

Friday 6th July 2018

8.00	**Plenary Forum: Pipeline 1**
9.00	Dr Luke Wells-Smith – Diagnosis and Management of Acute Laminitis Motion - Equine Podiatry Consulting
9.20	
9.55	
10.00	**MORNING TEA IN TRADE DISPLAY AREAS**
10.30	Professor Chris Sanchez – Foal Diagnostics, Therapeutics, and Prognostics University of Florida
10.50	
11.25	
11.30	Dr Keely Wilson – Emergencies during anaesthesia: when not to keep calm and carry on Murdoch University
11.50	
12.25	
12.30	**LUNCH – GROUND FLOOR AND FIRST FLOOR**
	Invited Research Presentations and Research Abstracts
1.30	Invited Research Abstract: Voriconazole-containing Thermogel for subconjunctival injection in horses. Dr Rosemary Cuming, Murdoch University.
2.00	Osteogenic capacities of equine stem cell populations are dependent on intrinsic bone morphogenetic protein (BMP) signaling activity. Dr Matt Stewart.
2.20	Effects of sample handling on plasma adrenocorticotropic hormone (ACTH) stability in horses with normal and elevated ACTH concentrations. Dr Alison Stewart.
2.40	Biological variation of routine haematology and biochemistry measurands in the horse. Dr Megan Wright.
3.00	**AFTERNOON TEA IN TRADE DISPLAY AREAS**
3.15	Invited Research Abstract: Novel anti-inflammatory therapies for the treatment of equine SIRS. Dr Jenni Bauquier, University of Melbourne.
3.45	Differences in cell marker expression by equine bone marrow-derived mesenchymal stem cells associated with blood antigen type and breed. Dr Chris Riley.
4.05	The effect of internal lag screw fixation on compression and loading responses of type III distal phalangeal fractures in horses. Dr Matt Stewart.
4.25	Septic peritonitis in the Post-Partum Mare. Dr Katie Offer.
4.45	Prevalence and risk factors associated with equine asthma in South Australian horses. Dr Alexandra Jaarsma
5.05	Insulin dysregulation in horses with systemic inflammatory response syndrome. Dr Alison Stewart.
5.30	**COLLEGE ANNUAL GENERAL MEETING**

Saturday 7th July 2018

8.00	Professor Chris Sanchez – Neonatal Sepsis/SIRs therapy University of Florida
8.20	
8.55	
9.00	Dr Alison Stewart – Equine Neurology Case studies and discussion University of Queensland
9.20	
9.55	
10.00	Dr Steve Zedler – Emergency Management of Fractures University of Queensland
10.30	
10.55	
11.00	**MORNING TEA IN TRADE DISPLAY AREAS**
11.30	**Plenary Forum: Pipeline 1**
12.30	**LUNCH – GROUND AND FIRST FLOOR**
1.30	Dr Darien Feary – Metabolic Emergencies of the Equine Endurance Athlete University of Sydney
1.50	
2.15	Dr Meg Brownlow - Exertional Heat Illness in Thoroughbred Racehorses University of Sydney
2.30	
3.00	**AFTERNOON TEA IN TRADE DISPLAY AREAS**
3.30	Associate Professor Helen Liley – Human neonatal encephalopathy and sepsis University of Queensland, Mater Children's Hospital
3.50	
4.25	
4.30	Professor Chris Sanchez – Literature Review 'Top 10 Emergency Articles' University of Florida
4.50	
5.30	
7.00	**COLLEGE AWARDS DINNER – BALLROOM**

Pain management in Horses – Is there Anything New?

Professor L. Chris Sanchez

College of Veterinary Medicine, University of Florida, Gainesville, FL, USA

OVERVIEW

Colic and lameness are two of the most clinically and economically important medical problems facing horses and their owners. Pain is a critical component of each disease process and its alleviation is critical to a successful outcome. A limited number of analgesics are available for use in horses and many are associated with significant untoward effects. This talk will focus on methods used for recognition of pain in horses and available options for analgesic drug use.

EQUINE PAIN SCORING SYSTEMS

Useful pain scoring systems should include the following traits: clearly defined assessment criteria, usable by all observers, simple and quick to use, sensitive, identified strengths and weaknesses, and validated. Possible deficiencies include bias, inter- and intra-observer variability. A lack of agreement between observers is one of the flaws of simple scoring systems such as the visual analogue scale (VAS), in which pain is scored on a numerical scale, when used in humans. Continuous video assessment allows for quantification of either time budgets (locations within the stall, ear position, head position, eating, lying down, etc.) or events (vocalizing, stomping feet, shifting weight, etc.). This relatively objective form of analysis has been performed on horses following arthroscopy.(Price, Catriona et al. 2003) A numerical rating system was used to show the beneficial effect of a butorphanol constant rate infusion for analgesia following colic surgery.(Sellon, Roberts et al. 2004) Facial pain scoring systems have recently been proposed for use in horses.(Dalla Costa, Minero et al. 2014, Gleerup, Forkman et al. 2015) Similar scoring systems have been used in humans and rodents and clearly show promise for use in horses as well. Objective measures such as vital signs, plasma cortisol concentration, and force plate analyses alleviate the subjective nature of assessment. But, vital signs and cortisol are affected by a variety of factors in addition to pain, including hydration status, perfusion, sepsis and/or endotoxaemia, fear, and anxiety; thus, they are not specifically useful indicators of pain.

ANALGESIC AGENTS

Non-steroidal anti-inflammatory drugs (NSAIDs)

A complete discussion of NSAID use in the horse is beyond the scope of these proceedings. Flunixin and phenylbutazone are used most commonly. Firocoxib has demonstrated COX-1 sparing effects in the horse; an initial loading dose is important for analgesia. Acetaminophen provides an alternative option and was recently shown to have similar efficacy to flunixin in a foot pain model when used alone and similar efficacy to phenylbutazone when combined with firocoxib.(Foreman, Foreman et al. 2015)

Opioids

All opioids have the potential for adverse effects in horses, including increased locomotion, excitement, and decreased gastrointestinal motility. Butorphanol has been the most widely used

opioid in horses. Intravenous use can be associated with excitement, ataxia, and increased locomotion when used alone. For short procedures, it is best combined with a α-2 agonist. Intramuscular administration results in decreased systemic availability (37%). When used as a constant rate infusion (CRI), behavioural and gastrointestinal adverse effects are reduced (relative to single injection) in normal horses and one report demonstrated decreased weight loss, improved recovery characteristics and earlier discharge from the hospital when administered for 24 hours after colic surgery.(Sellon, Roberts et al. 2004) Morphine has been used with varying success in horses. Some practitioners and researchers have reported fantastic results, while others have concerns. It's a great analgesic, but has a short duration of action and, as with all opioids, delays gastrointestinal transit. Buprenorphine appears to have a good safety and efficacy profile in horses, with the advantage of a longer (8-12 hour) duration of action.(Love, Pelligand et al. 2015) Buprenorphine is a fantastic option, as it has all of the positive attributes needed for a good opioid – the major downside is cost in the US. Transdermal fentanyl patches would theoretically provide a fantastic route of opioid administration in horses. Unfortunately, uptake from the patches is highly variable and extremely high plasma concentrations (associated with agitation in some horses) are needed for MAC reduction(Thomasy, Steffey et al. 2006) or visceral or somatic anti-nociception in healthy horses.(Sanchez, Robertson et al. 2007) Tramadol is an analogue of codeine, but it has less abuse potential and fewer cardiorespiratory side-effects than drugs classified as opioids. Tramadol has a short half-life and very low oral bioavailability (~3%) in horses and does not appear to provide effective analgesia when used alone. In a clinical setting, tramadol alone failed to provide pain relief in horses with naturally occurring laminitis but did appear to help when used in combination with ketamine.(Guedes, Matthews et al. 2012)

Alpha$_2$-adrenoceptor agonists

Alpha$_2$-adrenergic agonists are frequently used for both sedation and short-term analgesia. These drugs are not ideal for prolonged analgesic therapy, as they cause an immediate and profound decrease in gastrointestinal motility, amongst other cardiovascular effects, and have a relatively short duration of action. Importantly, one should note that sedative effects may require lower dosages and/or last longer than analgesic effects with the majority of drugs in this class. Alpha$_2$-adrenergic agonists provide dose-dependent visceral and somatic anti-nociception of varying duration, as well as an opioid-sparing effect. Thus, a combination of an alpha$_2$-adrenergic agonist and opioid provide a variety of commonly used multimodal analgesic protocols.

Sodium channel blockers

Lidocaine is an aminoamide local anaesthetic which prevents propagation of action potentials by binding to voltage-gated sodium channels. Lidocaine, administered as an intravenous infusion, is commonly used in horses for its potential analgesic, prokinetic and anti-inflammatory properties. Clinical signs of toxicity in conscious horses include skeletal muscle tremors, altered visual function, anxiety, ataxia, and collapse. Moderate evidence in clinical and research settings support its use as a visceral and somatic analgesic agent. Drug accumulation can be noted in a clinical setting after prolonged administration and lower infusion rates should be used in horses receiving highly protein bound drugs.

N-methyl-D-aspartate antagonists

Ketamine is a non-competitive N-methyl-D-aspartate receptor antagonist and can modulate central sensitization and exert an anti-hyperalgesic effect at sub-anaesthetic doses. Anti-nociception has not

been demonstrated in healthy horses receiving infusions of ketamine (Fielding, Brumbaugh et al. 2006) but in clinical settings there seems to be some beneficial effects (Wagner, Mama et al. 2011, Guedes, Matthews et al. 2012). In laminitic horses, addition of ketamine resulted in decreased blood pressure, decreased forelimb offloading frequency and increased forelimb load, relative to tramadol alone. (Guedes, Matthews et al. 2012)

Antispasmodic medications

N-butylscopolammonium bromide (NBB) has both anticholinergic and antispasmodic properties and is labelled for the treatment of spasmodic colic. NBB administration also decreases rectal tone, facilitating rectal examination and may be useful in horses with oesophageal obstruction.

Multimodal therapy

In severely painful horses, combination of ketamine with lidocaine and/or butorphanol could potentially provide additional analgesia, relative to infusion of a single drug. In healthy horses, butorphanol-containing combinations resulted in delayed total gastrointestinal time and reduced faecal output,(Elfenbein, Robertson et al. 2014) thus one should pay careful attention to faecal output when using said combinations.

REFERENCES

Dalla Costa, E., M. Minero, D. Lebelt, D. Stucke, E. Canali and M. C. Leach (2014). "Development of the Horse Grimace Scale (HGS) as a pain assessment tool in horses undergoing routine castration." PLoS One 9(3): e92281.

Elfenbein, J. R., S. A. Robertson, R. J. MacKay, B. KuKanich and L. Sanchez (2014). "Systemic and anti-nociceptive effects of prolonged lidocaine, ketamine, and butorphanol infusions alone and in combination in healthy horses." BMC Vet Res 10 Suppl 1: S6.

Fielding, C. L., G. W. Brumbaugh, N. S. Matthews, K. E. Peck and A. J. Roussel (2006). "Pharmacokinetics and clinical effects of a subanesthetic continuous rate infusion of ketamine in awake horses." Am J Vet Res 67(9): 1484-1490.

Foreman, J. H., C. R. Foreman and B. E. Bergstrom (2015). Medical alternatives to conventional cyclooxygenase inhibitors for treatment of acute foot pain in a reversible lameness model in horses. Journal of Veterinary Internal Medicine.

Gleerup, K. B., B. Forkman, C. Lindegaard and P. H. Andersen (2015). "An equine pain face." Vet Anaesth Analg 42(1): 103-114.

Guedes, A. G., N. S. Matthews and D. M. Hood (2012). "Effect of ketamine hydrochloride on the analgesic effects of tramadol hydrochloride in horses with signs of chronic laminitis-associated pain." Am J Vet Res 73(5): 610-619.

Love, E. J., L. Pelligand, P. M. Taylor, J. C. Murrell and J. W. Sear (2015). "Pharmacokinetic-pharmacodynamic modelling of intravenous buprenorphine in conscious horses." Vet Anaesth Analg 42(1): 17-29.

Price, J., S. Catriona, E. M. Welsh and N. K. Waran (2003). "Preliminary evaluation of a behaviour-based system for assessment of post-operative pain in horses following arthroscopic surgery." Veterinary Anaesthesia and Analgesia 30(3): 124-137.

Sanchez, L. C., S. A. Robertson, L. K. Maxwell, K. Zientek and C. Cole (2007). "Effect of fentanyl on visceral and somatic nociception in conscious horses." J Vet Intern.Med 21(5): 1067-1075.

Sellon, D. C., M. C. Roberts, A. T. Blikslager, C. Ulibarri and M. G. Papich (2004). "Effects of continuous rate intravenous infusion of butorphanol on physiologic and outcome variables in horses after celiotomy." J Vet Intern.Med. 18(4): 555-563.

Thomasy, S. M., E. P. Steffey, K. R. Mama, A. Solano and S. D. Stanley (2006). "The effects of i.v. fentanyl administration on the minimum alveolar concentration of isoflurane in horses." Br.J Anaesth. 97(2): 232-237.

Wagner, A. E., K. R. Mama, E. K. Contino, D. J. Ferris and C. E. Kawcak (2011). "Evaluation of sedation and analgesia in standing horses after administration of xylazine, butorphanol, and subanesthetic doses of ketamine." J Am Vet Med Assoc 238(12): 1629-1633.

Notes

Post Partum Colic

Dr Catherine Russell

Clovelly Intensive Care Unit, Scone Equine Hospital, 106 Liverpool Street, Scone, NSW.

Colic in the post-partum period is common and often challenging to the attending veterinarian. Stage 2 labour is explosive and rapid, normally lasting 10-60 minutes. With the powerful abdominal contractions plus the long limbed foal there is much potential for damage to the urogenital and gastrointestinal tract and their associated vasculature as well as the diaphragm and pelvic canal. It is vital to attempt to obtain a diagnosis as prompt medical and surgical treatment will significantly affect the mare's prognosis. Transport of a mare with uncomplicated post-partum haemorrhage is contraindicated and this complicates the decision process and adds pressure to the on-farm veterinarian.

Haemorrhage

Haemorrhage in the post partum mare is reported to occur more commonly in multiparous, older mares though young mares are affected. It may occur peripartum but is more commonly seen immediately after birth with a mean reported time of death at 8.5 hours.[1] It has been reported to affect 2-3% of post partum Thoroughbred mares and cause 40% of post partum deaths.[2]
Haemorrhage is most commonly from the uterine artery (77%) and less commonly from the internal pudendal, internal iliac and caudal mesenteric arteries. A post mortem study reported rupture to be more likely near a curve or a flexure of an artery. Studies have shown haemorrhage is a result of degeneration and weakness of the arterial wall.[1,3] Haemorrhage may be contained within the broad ligament as a haematoma or may rupture the broad ligament resulting in hemoperitoneum.
Mares have variable clinical signs from obtunded mentation, to violent colic. Recumbency, sweating, flank watching, marked tachycardia, normal temperature and pale mucous membranes with prolonged CRT are often reported. Pudendal haemorrhage is associated with marked vulval and perineal swelling. Variable damage to the pelvic canal may be seen, particularly following prolonged dystocia. Tachycardia at admission, during hospitalisation, and haemorrhage localised to the vagina were significantly associated with outcome.[3]
Leukopenia is commonly reported and leukopenia that failed to resolve within 3 days was reported with complications. Invasive diagnostic testing is controversial with concerns of dislodging a clot and causing fatal haemorrhage. Arnold et al reported this did not occur in the 22 mares in their study where rectal examination was performed and it is this author's preference to attempt to confirm the diagnosis.[3] When haemorrhage is not confined to the broad ligament, abdominal ultrasound will show a moderate to large volume fluid with a characteristic rouleux effect (like swirling smoke). Rarely a haematoma is observed, transabdominally, caudal to the kidney or located in the wall of the uterus. Careful uterine palpation may confirm this however a full bladder can be mistaken for a haematoma. Peritoneal fluid should be sampled and analysed as haemoperitoneum may be observed following rupture of a uterine wall and associated vessel. Serial sampling is recommended in mares with pyrexia or failure of the peripheral white cell count to normalise within 2-3 days.
Treatment of uncomplicated haemorrhage relies on keeping the mare calm and quiet. I do not recommend transport unless there are other complications, or it is not possible to treat on farm.

Transport may increase the mare's blood pressure causing the clot to dislodge resulting in fatal haemorrhage.
Prognosis of mares that were admitted to a referral hospital are reported at 88%.[3] Of the survivors, 49% were reported to produce a foal. Resolved haematomas of the broad ligament are intermittently found during routine palpation of the mare.

Uterine disorders

Tears and lacerations
Most uterine tears occur in stage 2 of delivery but may occur at any stage and especially during dystocia. It is reported to be the 3rd most common cause of death in mares (6%) in the post-partum period. In 163 mares admitted to a referral hospital post-partum 5.5% were diagnosed with uterine tear. [4]
Mare's present with clinical signs that are not specific but often include depression, colic, tachycardia and fever. The presence of retained foetal membranes complicates and may delay diagnosis. Uterine tears are notoriously difficult to palpate per vagina especially those of the tips of the uterine horns and have been successfully identified preoperatively in 24% of cases.[5] Uterine body tears are less common (26%) but reported to be palpated in 100% of cases. [5] A high index of suspicion occurs when this author removes a very clean glove after uterine palpation in the febrile post partum mare. The rapidity of development and severity of clinical signs depends upon the uterine environment and degree of peritonitis.
Ultrasound of the abdomen will show increased abdominal fluid and evidence of peritonitis. If the tear has included an artery of the uterine wall significant haemoperitoneum can be seen. Abdominocentesis is recommended. Fluid will be serosanginous to sanginous to frank haemorrhage with marked elevation of protein and white cell count, and presence of bacteria. Plant material may be seen with large tears. Serial peritoneal samples are recommended if the decision for surgery is not obvious and particularly in cases with haemoperitoneum and development of pyrexia or failure of peripheral leukocyte count to normalise.
Prognosis is reported at 63 - 80%. One study showed no difference in medical (73%) or surgical (76%) treatment on outcome. [5] In our hospital exploratory laparotomy is recommended to allow definitive diagnosis, repair of the lesion and large volume peritoneal lavage. Medical treatment in smaller tears often invokes intensive medical management and significant cost. Laminitis can be a significant complication.
Reports of subsequent fertility are good with one study reporting 13/26 in foal in that breeding season and 23/26 producing at least one foal at some time after surgery.

Uterine cramping and passage of foetal membranes
Normal physiological involution of the uterus and passage of foetal membranes can cause significant signs of colic in some mares, particularly maidens. Oxytocin may not be tolerated causing straining, sweating and recumbency. This source of colic is resolved after the passage of the membranes and with a single dose of anti-inflammatories or xylazine.

Gastrointestinal

Impaction post partum is seen particularly in maiden mares and also following dystocia associated with bruising and pain in the pelvic canal. Temporary nerve damage to the rectum from bruising occurs rarely and will cause signs of impaction and discomfort within 24 hours. Mares may also have difficulty urinating or show signs of ataxia and typically moderate to marked perineal bruising. Manual evacuation of the rectum, catheterisation of the bladder if required, anti-inflammatories and time will resolve most of these mares.

Colon torsion is the most common presentation of surgical colic in post partum mares, reported in 16.6% of mares admitted to a referral hospital.[4] Less commonly are mesenteric tears with ischemic necrosis and/or entrapment of bowel and small intestinal torsion. Mares present with violent colic signs that need to be distinguished from broad ligament haematoma. Distinguishing clinical signs include distended abdomen, rectal findings and abdominal ultrasound. Evaluation of the diaphragm is recommended for rare cases of hernia. Lack of response to pain relief and absence of rectal or ultrasound findings supportive of haemorrhage will support exploratory laparotomy.

Caecal or large colon rupture has been reported to be the cause of death in 19% of post partum mares. The rupture may occur before, during or after birth and mare's will classically present with severe signs of colic, sweating, tachycardia, toxic mucous membranes and ileus. Ultrasound is consistent with peritonitis with progressive scans showing increasing volumes of echogenic peritoneal fluid with strands of fibrin, omentum and particulate material. Peritoneal fluid classically has a leucocytosis with degenerate neutrophils, mixed bacteria and plant material. Care should be taken as some early cases will not show peritonitis presumably due to the rupture being either walled off and held between the side of the abdomen and the caecum, or in early cases with small volume of leakage and dilution from the inflammatory reaction. Other intestinal causes of peritonitis in these mares include gradual necrosis and leakage from an intestinal organ following the tearing of mesentery at foaling.

Rectal prolapse

This is uncommonly seen though usually devastating in the post partum mare. Due to the short mesentery and associated blood supply the mare is at risk of ischemic necrosis of the rectum. Due to telescoping of the mucosa it is not easy to estimate how much is prolapsed and they carry a grave prognosis as the location precludes surgical resection and anastamosis. The horses do not show signs of colic for the first 24 hours and then develop signs of impaction. The affected rectum feels leathery and thickened and blood may be seen on the glove after rectal examination.

References

1. Ueno T, Nambo Y, Tajima Y and Umemura T. Pathology of lethal peripartum broad ligament haematoma in 31 Thoroughbred mares. *Equine vet. J.* 2010:42;529-533.
2. Arnold CE, Payne E M, Thompson JA, Slovis NM and Bain FT Periparturient hemorrhage in mares: 73 cases (1998-2005). *J Am Vet Med Ass* 2008:232;1345-1351
3. Williams NM, Bryant UK. Periparturient Arterial Rupture in Mares: A Postmortem Study. *J Equine Vet Sci* 2012;32:281-284.

4. Dolente BA, Sullivan EK, Boston R and Johnston JK. Mares admitted to a referral hospital for postpartum emergencies: 163 cases (1992-2002). *J Vet Em Crit Care* 2005:15;3;193-200.
5. Javsicas LH, Giguere S, Freeman DE, MVB, Rodgerson DH, and Slovis NH, Comparison of Surgical and Medical Treatment of 49 Postpartum Mares with Presumptive or Confirmed Uterine Tears. *Vet Surg* 2010:39:254–260.

Notes

Diagnosis and Management of Septic Arthritis

Dr Matthew Stewart

University of Illinois

Introduction: By definition, "septic arthritis" refers to an inflammatory disease of joints induced by bacterial or, more rarely, fungal infection. Regardless of etiology, septic arthritis usually presents as an acute onset of joint swelling and heat, with severe lameness in the affected limb(s). Septic arthritis in horses occurs as a consequence of three clinical scenarios:

1. Acute, haematogenous septic arthritis in foals (AHSA): This develops following a bacteraemic or septicaemic episode with vascular delivery of bacteria to the subsynovial vascular plexus of joints and, frequently, adjacent intraosseous capillary beds. Systemically septic foals can present with one or multiple affected joints. Alternatively septic arthritis can develop in foals in the absence of any obvious systemic abnormalities or days after resolution of systemic disease.

2. Trauma: The lower limbs of horses have little or no soft tissue coverage. Therefore, involvement of adjacent synovial structures; joints, tendon sheaths, and bursae, should be considered in any lower limb wound.

3. Iatrogenic infection, following joint injections or surgery: This is a very infrequent occurrence (less than 1.0% in most studies) in both contexts [5,6,12,32]. Several recent studies have demonstrated that a low level of intra-articular contamination is 'the rule' following arthrocentesis [34,35], but rarely results in clinical disease. Administration of intra-articular agents can increase the likelihood of joint sepsis [14,32].

Diagnosis: The case history usually provides a clear indication of the initial cause of septic arthritis (concurrent or recent sepsis, limb wound, injection, surgery etc). As noted above, clinical signs reflect acute onset of articular and peri-articular swelling, heat and lameness. In foals, osteomyelitic lesions in the vertebrae can present as acutely paraplegic cases.

Grossly abnormal synovial fluid is the primary initial diagnostic indicator of joint sepsis. Aspirates appear turbid, in stark contrast to normal synovial fluid. In acute cases, synovial fluid volumes are increased. However, in chronic cases, the intra-synovial space can become loculated, complicating fluid aspiration. In trauma cases, confirmation of joint involvement can usually be made by distending the suspected joint space with sterile saline, using a portal distant from the wound bed, at the time of fluid collection. The clinical pathological criteria for a suppurative synovial fluid finding varies somewhat between labs, but the following indices are representative:

Greater than 40,000 nucleated cells/ml

Greater than 90% neutrophils

Greater than 3.0 gm total protein/dl

+/- positive Gram stain findings (bugs visible in app. 25% of cases)

Positive culture, via blood culture bottle (app. 50% success)

Plasma and/or synovial Serum Amyloid A (SAA) concentrations are gaining prominence as a diagnostic tool for early identification of sepsis [15,17,20,25] and for monitoring responses to therapy [15]. Elevations in synovial fibrinogen D-dimer concentrations have also shown value in distinguishing septic from non-septic joint disease [24], but this assay is not yet commercially available.

Ultrasonographic imaging of infected joints is useful to assess the extent of intra-synovial fibrin deposition and identify pockets of loculated fluid for diagnostic aspiration [2]. Radiological assessment of affected joints is indicated to determine whether there is any skeletal/osseous involvement, but sufficient bone resorption needs to have occurred to be detectable; this process requires, at minimum, several days. In equivocal, or intractable cases, scintigraphy and MRI are very useful to identify lesions prior to them becoming radiologically evident [8,11].

Treatment of septic arthritis: Treatment of septic arthritis will obviously occur in the context of managing any associated problems such as systemic sepsis, local sepsis or trauma.

Antibiotic coverage is the most important aspect of treatment for septic arthritis. Identification of the causative organism(s) and sensitivity profiles are extremely useful for case management. However, culture and sensitivity procedures routinely require several days and, all too frequently, no pathogens are grown from samples. Although some 'educated guess-timates' can be made regarding the likely pathogen(s), broad-spectrum coverage is usually opted for; amikacin or gentamicin, combined with first generation cephalosporins (cephalothin), penicillin or ampicillin.

Musculoskeletal infections present several challenges for antimicrobial therapy:

1. bacteria are often resistant to routine antibiotics
2. tissue penetration can be reduced by local vascular compromise, ischemic or necrotic tissue, and residual debris.
3. Establishing effective MIC levels at the site of infection may require administrating prohibitively expensive and potentially toxic doses of antibiotics.

To mitigate these limitations, several variants of 'local antibiotic delivery' have been developed to provide extremely high concentrations of antibiotic to the infected joint. These strategies include repeated intra-articular injections [29,36], distal limb perfusion via intravenous or intraosseous routes [7,9,27,36,37], delivery via intra-articular catheters [13], constant intra-articular infusion of antibiotic [1,19,21], and placing local antibiotic-impregnated beads within peri-articular tissues [10]. The applicability of any given local delivery strategy to clinical cases varies with the target joint, infection severity and response to initial treatment, and budgetary constraints.

Joint lavage decreases gross contamination and reduces the levels of intra-articular inflammatory cells and mediators. In the face of intra-articular inflammation, articular cartilage will lose proteoglycan content within 12-24 hours [31]. Arthroscopic lavage offers several advantages over through-and-through needle lavage, providing simultaneous large bore, high volume lavage, removal of foreign bodies, fibrin and other debris, along with evaluation of the joint surface and surrounding structures [22,38]. In chronic, severe, or persistent cases, an arthrotomy is superior to any type of lavage alone, although ascending contamination and fistula formation are recognized sequele [3,4,29]. While acknowledging reports of outstanding clinical success following joint lavage [38], high volume lavage is not, in itself, anti-bacterial and is most unlikely to remove bacteria colonizing the synovium. In my

experience, without effective antibiotic delivery, the beneficial responses to articular lavage are transient.

Prognosis for equine septic arthritis: Many factors impact the outcome of individual septic arthritis cases. Accepting this, the published literature indicates that survival of foals treated for septic arthritis ranges from 33% to 80% [16,22,23,26,28,33]. In several studies, multi-joint involvement and systemic illness both negatively influenced prognosis [16,23,30,33]. Foals that survive septic arthritis are less likely to race, take longer to begin racing and make fewer starts [23,30]. Success rates in adult horses treated for septic arthritis is higher; 75-85% [18,22,28]. In adult cases, multi-joint involvement and systemic disease is far less likely and some of these cases develop following relatively small inoculate burdens following arthrocentesis, and tend to be very responsive to therapy.

References

1. Adams SB, Lescun TB (2000) How to Treat Septic Joints with Constant Intra-articular Infusion of Gentamicin or Amikacin. 46th AAEP proceedings p 188- 192
2. Beccati K, et al (2015). Ultrasonographic findings in 38 horses with septic arthritis/tenosynovitis. Vet Radiol Ultrasound 56: 68–76
3. Bertone AL, et al (1987) Comparison of various treatments for experimentally induced equine infectious arthritis. Am J Vet Res 48:519-529
4. Bertone AL (1992) Arthrotomy versus arthroscopy and partial synovectomy for treatment of experimentally induced infectious arthritis in horses. Am J Vet Res 53:585-591
5. Borg H, et al (2013) Postoperative septic arthritis after elective equine arthroscopy without antimicrobial prophylaxis. Vet Surg 42: 262–266
6. Brunsting JY, et al (2018) Incidence and risk factors of surgical site infection and septic arthritis after elective arthroscopy in horses. Vet Surg 47:52–59
7. Butt TD et al (2001) Comparison of 2 techniques for regional antibiotic delivery to the equine forelimb: intraosseous perfusion vs. intravenous perfusion. Can Vet J 42: 617-622
8. Easley J, et al (2011) Magnetic resonance imaging findings in horses with septic arthritis. Vet Radiol Ultrasound 52: 402–408
9. Errico JA, et al (2008) Comparison of two indirect techniques for local delivery of a high dose of an antimicrobial in the distal portion of forelimbs of horses. Am J Vet Res 69:334-342
10. Farnsworth KD, et al (2001) The effect of implanting gentamicin-impregnated PMMA beads in the tarsocrural joint of the horse. Vet Surg 30: 126-131
11. Gaschen L, et al. (2011) Magnetic resonance imaging in foals with infectious arthritis. Vet Radiol Ultrasound 52: 627–633
12. Gillespie CC, et al (2016) Methods and variables associated with the risk of septic arthritis following intra-articular injections in horses: A survey of veterinarians. Vet Surg 45:1071-1076
13. Goodrich LR, et al (1999) How to place an orthopedic drain in a joint or tendon sheath to treat intrasynovial sepsis. 45th AAEP proceedings, p 122-123
14. Gustafson SB, et al (1989) Comparison of the effect of polysulfated glycosaminoglycan, corticosteroids, and sodium hyaluronate in the potentiation of a subinfective dose of Staphylococcus aureus in the midcarpal joint of horses. Am J Vet Res 50:2014-2017
15. Haltmayer E, et al (2017) Course of serum amyloid A (SAA) plasma concentrations in horses undergoing surgery for injuries penetrating synovial structures; an observational clinical study. BMC Vet Res13:137
16. Hepworth-Warren KL, et al (2015) Bacterial isolates, antimicrobial susceptibility patterns, and factors associated with infection and outcome in foals with septic arthritis: 83 cases (1998–2013). J Am Vet Med Assoc 246:785–793
17. Jacobsen S, et al (2006) Concentrations of serum amyloid A in serum and synovial fluid from healthy horses and horses with joint disease. Am J Vet Res 67:1738–1742

18. Lapointe JM, et al (1992) Septic arthritis in 15 Standardbred racehorses after intra-articular injection. Equine Vet J 24: 430-434
19. Lescun TB, et al (2000) Constant intra-articular gentamicin infusion of the tarsocrural joint in the horse. Am J Vet Res 61: 407- 412
20. Ludwig EK, et al (2016) Serum and synovial fluid Serum Amyloid A response in equine models of synovitis and septic arthritis. Vet Surg 45: 859–867
21. Meagher DT, et al (2006) Evaluation of a balloon constant rate infusion system for treatment of septic arthritis, septic tenosynovitis, and contaminated synovial wounds: 23 cases (2002–2005). J Am Vet Med Assoc 228:1930-1934
22. Meijer MC, et al (2000) Clinical Experiences of Treating Septic Arthritis in the Equine by Repeated Joint Lavage: a Series of 39 Cases. J Vet Med A 47: 351–365
23. Neil KM, et al (2010) Retrospective study of 108 foals with septic osteomyelitis. Aust Vet J 88:4–12
24. Riber T, et al (2011) Synovial fluid D-dimer concentration in foals with septic joint disease. J Vet Intern Med 25:1113–1117
25. Robinson CS, et al (2017) Are serum amyloid A or D-lactate useful to diagnose synovial contamination or sepsis in horses? Vet Record doi: 10.1136/vr.104386
26. Sanchez LC, et al (2008) Factors associated with survival of neonatal foals with bacteremia and racing performance of surviving Thoroughbreds: 423 cases (1982–2007). J Am Vet Med Assoc 233:1446–1452
27. Scheuch BC et al (2002). Comparison of intraosseous or intravenous infusion for delivery of amikacin sulfate to the tibiotarsal joint of horses. Am J Vet Res 63: 374- 380
28. Schneider RK, et al (1992) A retrospective study of 192 horses affected with septic arthritis/tenosynovitis. Equine Vet J 24: 436-442
29. Schneider RK et al (1992). Open drainage, intra-articular and systemic antibiotics in the treatment of septic arthritis/ tenosynovitis in horses. Equine Vet J 24: 412- 214
30. Smith LJ, et al (2004) What is the likelihood that Thoroughbred foals treated for septic arthritis will race? Equine Vet J 36: 452-456
31. Smith RL, Merchant TC, Schurman DJ (1982) In vitro cartilage degradation by Esherichia coli and Staphylococcus aureus. Arthritis Rheum. 25: 441-446
32. Steel CM, et al (2013) Risk of septic arthritis after intra-articular medication: a study of 16,624 injections in Thoroughbred racehorses. Aust Vet J 91:268–273
33. Vos NJ, Ducharne NG (2008) Analysis of factors influencing prognosis in foals with septic arthritis. Irish Vet J 61: 102-106
34. Wahl K, et al (2012) Contamination of joints with tissue debris and hair after arthrocentesis: the effect of needle insertion angle, spinal needle gauge, and insertion of spinal needles with and without a stylet. Vet Surg 41: 391–398
35. Waxman SJ, et al (2015) Effect of needle brand, needle bevel grind, and silicone lubrication on contamination of joints with tissue and hair debris after arthrocentesis. Vet Surg 44:373–378
36. Werner LA, et al (2003) Bone gentamicin concentration after intra-articular injection or regional intravenous perfusion in the horse. Vet Surg 32:559-565
37. Whitehead KJ, et al (1992) Regional limb perfusion for antibiotic treatment of experimentally induced septic arthritis. Vet Surg 21: 367-373
38. Wright IM, et al (2003) Endoscopic surgery in the treatment of contaminated and infected synovial cavities. Equine Vet. J 35: 613-619

Notes

Emergency Treatment of Equine Colic

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It is estimated 1 horse in 10 (10% of the population) will experience colic each year and many episodes of colic will go unnoticed by horse owners. Approximately 80-90% of cases are simple and will respond with minimal medical treatment. Of the remaining 10-20% requiring more aggressive treatment only 1-7 % will require surgery. While there are over 70 different causes of colic reported, approximately 70% are reported to be spasmodic colic (hypermotility). Traditionally spasmodic colic was common and largely a consequence of emboli showers associated with *Strongylus vulgaris* infection and verminous arteritis. With new worming medications verminous arteritis has become very rare yet spasmodic colic is still reported as being the most common diagnosis in horses with colic. This may be historical because the actual cause of medical responsive colics are often not definitively diagnosed.

In horses referred to veterinary hospitals for further investigation, impactions involving the large intestine are most common condition identified. The ascending colon is the most common region of the gastrointestinal tract involved in all forms of colic, followed by small intestine, caecum and small or descending colon. The highest fatalities are associated with strangulating lesions of the intestine, with torsions of the colon being the most common condition, followed by strangulation of the small intestine. Over the last 20 – 25 years the mortality rates for hospitalised patients and patients undergoing surgery have fallen steadily, and over the last 10-15 years, long term survival rates have improved, with many horses returning to their original use. This has improvement in survival rate been associated with by earlier recognition of clinical signs, earlier veterinary intervention and triage, earlier referral accompanied by improvements in surgical and anaesthetic techniques and after care.

The decision on whether a lesion is likely to respond to medical treatment or require surgery is made based on the patient's history, pain and response to analgesics, cardiovascular status, rectal and/or ultrasound examination of the abdomen, the presence of nasogastric reflux and laboratory tests including abdominocentesis. A decision on how to proceed may be made on any one or variable combinations of factor and may not require the completion of all aspects of the clinical examination and ancillary tests. For example a horse that has unrelenting pain that is not responsive to analgesics is likely to be a surgical candidate. In the field, studies would suggest that a clinical examination and cardiovascular status response to analgesics (87%) and a rectal examination (75%) are the most common tests performed while other tests including nasogastric intubation (44%) are performed much less frequently. This is not surprising given many horses with mild signs of colic will respond to the initial treatment. Horses presenting to referral hospitals are likely to be assessed more thoroughly.

During surgery an efficient and systematic approach to resolution of the condition is essential to minimise surgical and anaesthetic time. Surgical techniques are well described, however with experience, surgeons will become more skilled and develop techniques that are associated with less

complications. Where there is strangulated small intestine it is important to remember markedly distended, amotile intestine proximal to the lesion may also be predisposed to adhesion formation so resection of this intestine with the strangulated intestine may reduce adhesion formation, enhance post-operative progressive motility and reduce handling of the intestine. It is important to empty the small intestinal contents into the caecum with minimal handling of the small intestine to reduce adhesion formation and ileus. The importance of keeping the intestine moist and returning the small intestine to the abdomen while other procedures are performed should not be underestimated. Reinspecting the small intestine for progressive motility before closure should be used to help decide appropriate postoperative management.

For small and large intestinal lesions, a pelvic flexure enterotomy to empty the contents of the ascending colon is warranted particularly where there is considerable feed material in the colon. This reduces the volume of food in the intestine while normal progressive motility throughout the intestine resumes. A period of decreased intestinal motility is experienced after most gastrointestinal surgeries.

Assessing intestinal viability following strangulation or torsion of the colon can be challenging. Return of a pink colour and bleeding at the enterotomy site suggests intact blood supply but does not guarantee viability. Return of spontaneous motility or motility after flicking the intestine suggests intact nerve supply and intestinal viability because nerve tissues are the most susceptible to low oxygen tension. It is often assumed when assessing large colon torsions that the severe oedema of the wall and mesentery offers a poorer prognosis than where the colon and mesentery. Where there is oedema of the colon the venous supply has been occluded before the arterial supply allowing the arterial supply to continue while a lack of oedema suggests the arterial and venous supply have been occluded simultaneously and the colon has been without arterial oxygen. Where there is reduced arterial supply, the tissue closest to the root of the mesentery will be more protected than the distal tissues and anoxia and improve the chances of a colon resection being successful.

The long held concept of holding all horses off feed following colic surgery has been questioned in recent times. Feed encourages the return to normal gastrointestinal motility and promotes a return to a balanced bacterial microflora. In routine surgery for simple large intestinal displacements and early return to a normal hay diet spread over the day is appropriate. In small intestinal resection and anastomosis where there is active intestinal motility at the time the abdomen, small handfuls of roughage given intermittently following surgery while assessing motility may promote progressive motility.

Pain management is best managed pre-emptively and ultimately has been shown to reduce the overall dose of medication required over the course of treatment. Where post-operative pain is a concern full dose of flunixen meglumine are warranted for the first 24 hours to assess the recovery then a reduction in frequency rather than dose is appropriate. With appropriate fluid therapy serious renal complications are not common in horses presenting with acute colic. Lignocaine infusion offers an alternative to non-steroidal anti-inflammatory drugs where renal function may be a concern in horses that have been inappetant and dehydrated for days prior to referral.

Notes

Ocular manifestations of Systemic Disease

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Take Home Message

Many systemic inflammatory diseases in horses have ocular signs, and many ophthalmic diseases (or their treatment) can have or result in systemic signs. Thus, it is important to look at the whole horse when considering treatment plans or prognoses. Though the proceedings are organized by specific manifestations, the talk will be entirely case-based.

Ocular Manifestations of Systemic Disease

Neonatal sepsis/SIRS

The septic foal may seed bacteria to various organs, including the eye. The first sign of septic uveitis is usually a green hue to the iris and anterior chamber as fibrin seeps out of the uveal vasculature. Additional signs of uveitis follow and typically include miosis, globe hypotony (low IOP), aqueous flare, conjunctival and episcleral injection. Occasionally hypopyon and hyphema may develop. Ocular signs may be unilateral or bilateral. Systemic antimicrobial therapy and generalized support are critical to survival. The uveitis must be addressed with symptomatic anti-inflammatory therapy if intraocular scarring is to be avoided. As long as no corneal ulcer is present, topical medical treatment should consist of corticosteroids and atropine. If tolerable, systemic flunixin will benefit the eyes. Occasionally, fibrin will completely fill the anterior chamber and be slow to resorb. If it does not improve rapidly, intracameral tissue plasminogen activator (TPA) may be very helpful to hasten its dissolution.

Rhodococcus equi

Ocular manifestations of *R. equi* include uveitis and occasionally hyphema. Signs of uveitis typically include epihora, blepharospasm, photophobia, corneal oedema, miosis, aqueous flare and significant accumulations of fibrin in the anterior chamber. Severe cases can present with panuveitis, endophthalmitis and vitreal abscess formation. If the uveitis is not controlled adequately, intraocular scarring and cataract formation may result. As such, screening thoracic radiographs and/or ultrasonography are recommended in all foals presenting for surgery associated with congenital cataracts. Concurrent *R. equi* infection greatly impacts the success of cataract removal and the well-being of the patient. Medical treatment for *Rhodococcus*-induced uveitis is symptomatic, with topical anti-inflammatory drugs, atropine and judicious intracameral TPA if needed to resolve fibrin.

Salmonellosis

Salmonellosis is a common infectious enteric disease in horses. Septic horses may have signs ranging from conjunctival and scleral haemorrhages to a severe fibrinous iridocyclitis. *Salmonella* spp. have been recovered from the anterior chambers of affected horses. Fibrin clots typically clear with topical and systemic treatment. The most common signs of ocular involvement are the classic signs of anterior uveitis and hypopyon. Most animals with ocular signs are bacteraemic, thus ocular involvement is more common in foals/weanlings. Treatment of salmonellosis-related uveitis should aim to decrease

inflammation, prevent synechia formation, and relieve ocular pain. Topical corticosteroids and atropine are used most commonly.

Lyme

Borrelia burgdorferi is a spirochete that may cause panuveitis in horses. Ocular signs are generally nonspecific and panuveitis (anterior and posterior uveitis) may be mild to severe. Hyphema has been reported. Treatment is symptomatic with topical corticosteroids and atropine and systemic flunixin meglumine. Spirochetes have been found inside the eyes of severe cases of panuveitis. When neurologic signs are present in a systemically ill animal, facial nerve paralysis is prominent which can predispose the patient to exposure keratitis.

Leptospirosis

Both acute iridocyclitis and equine recurrent uveitis (ERU) may be initiated by Leptospiral organisms. The exact mechanism by which Leptospiral organisms induce and sustain recurrent uveitis is not well understood, however, it is believed that some degree of molecular mimicry may trick the immune system into mounting and auto-immune attack against the eye. Bacterial antigens and antibodies have been found in the equine eye. Experimental infections with leptospiral organisms has been associated with the development of clinical uveitis even up to one year after initial infection. However, not every horse infected with lepto will develop uveitis or recurrent uveitis. Peri-papillary depigmented chorioretinal lesions may occur with acute or subacute infections of the retina and choroid. A persistent vitritis similar to ERU may be caused by Leptospiral infection and is responsive to vitrectomy therapy.

Temporohyoid osteoarthropathy

Temporohyoid osteoarthropathy (THO) is a neurologic condition in adult horses characterized by acute onset of CN VII and CN VIII deficits resulting in facial paralysis and/or vestibular signs. This condition occurs secondary to bony proliferation of the temporohyoid joint and stylohyoid bone that leads to fusion of the TH joint. Fusion of this joint can predispose the animal to fracture of the base of the skull, the shaft of the stylohyoid bone or the petrous temporal bone. This, in turn, leads to damage of the inner and middle ear and the previously noted cranial nerves. Prognosis is guarded and the facial paralysis or paresis may be permanent. Exposure keratitis and corneal ulceration are common and slow to heal without a normal eyelid function.

Systemic Manifestations of Ocular Disease

Large Colon and Cecal Impactions

Large colon impactions are a frequent cause of large intestinal non-strangulating obstruction in the horse. Impaction can occur from a variety of factors, most commonly decreased water intake, decreased overall activity (such as stall confinement due to illness or injury), or a variety of other factors. Cecal impactions occur less commonly, but reported risk factors include surgery for non-gastrointestinal reasons. Horses treated for ophthalmic disease often have multiple risk factors for large colon and or cecal dysfunction, including stall confinement, dietary alterations, and potentially decreased gastrointestinal motility associated with sedation and/or anaesthesia, topical administration of atropine, and/or ocular pain. Careful monitoring includes measurement of daily water intake and faecal output and careful monitoring for signs of colic. Medical therapy is typically

successful and any decrease in water intake or faecal output should warrant careful scrutiny and/or intervention with oral electrolytes, decrease in hay provided and, potentially enteral fluids. Such intervention should be accompanied by a decrease in frequency of atropine administration. Tropicamide may be employed topically; however, it is not nearly as effective at addressing intraocular inflammation or producing mydriasis.

Right Dorsal Colitis

Ulceration and/or inflammation of the right dorsal colon is the most common ulcerative disorder associated with protein-losing enteropathy in the horse. This condition is most often associated with systemic non-steroidal drug administration; importantly, this can occur as an idiosyncratic reaction, thus does not necessarily indicate NSAID abuse. Clinical signs include dependent oedema, colic, weight loss and diarrhoea. Clinicopathologic changes often precede clinical signs and include hypoproteinemia secondary to hypoalbuminemia and, infrequently, anaemia. Since ophthalmology patients frequently require long-term, high-dose systemic NSAID administration to control intraocular inflammation, they are at particular risk for the development of RDC. It is recommended that any animal on an NSAID for extended periods of time have blood protein concentrations checked regularly.

References

1) Leiva M, Peña T, Armengou L, Cesarini C, Monreal L. Uveal inflammation in septic newborn foals. J Vet Intern Med. 2010 Mar-Apr;24(2):391-7.
2) Reuss SM, Chaffin MK, Cohen ND. Extrapulmonary disorders associated with Rhodococcus equi infection in foals: 150 cases (1987-2007). J Am Vet Med Assoc. 2009 Oct 1;235(7):855-63.
3) Gerhards H, Wollanke B. [Antibody titers against Borrelia in horses in serum and in eyes and occurrence of equine recurrent uveitis]. Berl Munch Tierarztl Wochenschr. 1996 Aug;109(8):273-8.
4) Walker AM, Sellon DC, Cornelisse CJ, Hines MT, Ragle CA, Cohen N, Schott HC. Temporohyoid osteoarthropathy in 33 horses (1993-2000). J Vet Intern Med. 2002 Nov-Dec;16(6):697-703.
5) Davis JL. Ocular manifestations of systemic disease. In: Gilger BC (ed) Equine Ophthalmology 2nd ed. Philadelphia: Elsevier Saunders 2011:443-469.
6) Cullen CL, Webb AA. Ocular manifestations of systemic disease. Part 3: The horse. In Gelatt KN (ed): Veterinary Opthalmology. Ames, IO: Wiley Blackwell. 2013: 2037-2070.

Notes

Pathophysiology, Diagnosis and Treatment of Traumatic Brain Injury

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Traumatic brain injury (TBI) is a significant cause of morbidity and mortality worldwide[1]. Mortality due to severe TBI can reach 40% with high rates of disability among the survivors[2]. Due to the large numbers of patients involved, it has been estimated that even small improvements in TBI management could have significant impact. Cognitive, behavioural, and emotional impairments are common and particularly disabling post-TBI and can persist into the chronic stage. Widespread injury to the white matter tracts, or diffuse axonal injury is a key feature of TBI and mainly disrupts neuronal networks and impairs information processing which contributes to the cognitive impairments observed post-TBI[3].
Classifying head injuries has been problematic. While certain circumstances suggest major injury, patients with complete absence of clinical findings have been found to have intracerebral haemorrhage on imaging[4]. To date, computed tomography (CT) has been the imaging modality of choice in the first 24 hours after injury due to its short scan time and wide availability. It is superior in evaluating bones and detecting acute subarachnoid haemorrhage or fractures but has its limitations when it comes to diagnosis of soft tissue damage.

Magnetic Resonance Imaging (MRI), on the other hand, has emerged as a very promising method with superior sensitivity to CT. Forty eight to 72 hours after injury, MRI is generally considered to be superior to CT as the ability of MRI to detect hematomas improves over time as the composition of the blood changes. The development of new acquisition sequences such as Fluid attenuated inversion recovery MRI (FLAIR), Magnetisation Transfer Imaging (MTI), Magnetic Resonance Spectroscopy (MRS), and Diffusion Tensor Imaging (DTI), have improved the sensitivity of MRI.

Imaging has several potential roles in the chronic management of head trauma: identifying post-operative sequelae, evaluating the underlying functional abnormalities associated with late complications of head trauma, predicting long-term prognosis, guiding rehabilitation, and monitoring recovery and intervention. There is significant need for objective measures to predict the clinical course of TBI patients. Although some anatomic imaging findings on MRI have been found to be predictive of overall survival, such as cortical thinning and volumetric brain changes, they are not adequately predictive of functional outcome[5].

A number of more advanced MRI methods have emerged that will have significant potential in predicting patient outcome: functional connectivity resting state MRI (rsfMRI) has become a very popular method for the detection of how the different brain regions interconnect and interact by mapping the brain circuitry at rest, and has been applied in both humans and animals. It has major implications for monitoring functional outcome following injury and how neurons reorganise their connections to recover function. Dynamic contrast enhanced MRI (DCE-MRI) is also a very sensitive method to detect blood brain barrier leakiness in the early stages following an injury which may be a very important marker of long-term patient outcome and may have significant implications for acute patient management.

MRI is at the fore of technological advancements and its application in the field of traumatic brain injury will lead to major changes in diagnosis, prognosis, and management and may lead to preventative or pre-emptive treatments in the acute setting. Therefore, MRI may become a more useful tool for the early evaluation of acute brain injury.

In the future, the already significant role of imaging in guiding therapy may grow. Technological improvements continue to reduce scanning time and improve resolution. New methods are being developed to quantify damage on images and perhaps improve predictive power. A growing number of minimally invasive, image-guided techniques are replacing open surgical techniques. MRI in particular will have significant advantages in the non-invasive and safe longitudinal monitoring of patients over time and during interventions.

Most importantly, MRI has huge translational benefit where all sequences can be applied to both animals and humans in a similar manner making all advancements in the human sector applicable to veterinary medicine and vice versa.

References

1. Ghajar J: Traumatic brain injury. The Lancet 356:923-929, 2000
2. Murray GD1 TG, Braakman R, Cohadon F, Dearden M, Iannotti F, Karimi A, Lapierre F, Maas A, Ohman J, Persson L, Servadei F, Stocchetti N, Trojanowski T, Unterberg A.: The European Brain Injury Consortium survey of head injuries. Acta Neurochir 141:223-36, 1999
3. Mesulam M: From sensation to cognition. Brain 121:1013-52, 1998
4. Quayle K, Jaffe D, Kuppermann N, et al: Diagnostic testing for acute head injury in children: when are head computed tomography and skull radiographs indicated? Pediatrics 99:e11, 1997
5. Azian AA1 NA, Shuaib L, Mahayidin M, Ariff AR, Naing NN, Abdullah J.: Computed tomography of the brain in predicting outcome of traumatic intracranial haemorrhage in Malaysian patients. Acta, Neurochir 143:711-20, 2001

Notes

Diagnosis and Management of Acute Laminitis

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Laminitis Defined

Laminitis is defined as inflammation of the lamellar attachment which results in failure of the suspensory apparatus of the distal phalanx within the hoof capsule. There are three stages of laminitis defined as;

- Development phase: clinical signs of laminitis not evident, however the inflammatory process has been initiated
- Acute phase: begins when clinical signs of laminitis become evident
- Chronic phase: begins 72 hours after initial clinical signs of laminitis are apparent

The chronic phase can be divided into chronic unstable and chronic stable based on the clinical and radiographic findings.

Identifying Patients at High Risk of Developing Laminitis

Risk factors associated with the development of laminitis can be categories as;

- Sepsis associated

Horses presenting with a septic focus are at high risk of developing laminitis either while recovering from the underlying septic focus or in a period up to 12 weeks post exposure. Disease processes that appear to be associated with developing laminitis include colitis, retained foetal membranes, cellulitis and pneumonia. While we may not know the exact trigger factor in each individual case, a history of a septic focus in an acute case of laminitis is of serious concern.

- Endocrine associated

Horses with either a diagnosed or subclinical endocrine disease such as equine metabolic syndrome or pars pituitary intermedia dysfunction are at high risk of developing laminitis, particularly if they are exposed to other risk factors. Many of these horses have either sub-clinical or clinical chronic laminitis and may develope an acute-on-chronic episode.

- Excess mechanical forces or contralateral limb over load

'Road Founder' is less commonly diagnosed in the 21st century due to a low percentage of horses working on hard surfaces. Occasionally when horses escape and gallop down a bitumen or gravel road they can develop serious contusions to the underlying sensitive corium surrounding the distal phalanx and excess force on the lamellar attachment.

Excess weight bearing on the contralateral limb due to a severe unilateral lameness can initiate an episode of support limb laminitis. The mechanisms under which support limb laminitis occurs is still under investigation. Potential mechanisms include hypoperfusion or excessive strain on the lamellar attachment.

The final aspect to consider is the overall hoof health prior to presentation. Racehorses in training typically have poor hoof health. Frequently they present with minimal vertical depth of hoof, evidence of sub-clinical laminitis and poor wall quality. This weakens the support structures of the foot and reduces the foots ability to resist further destabilisation from a laminitic episode.

Individual cases of laminitis may not be as straight forward as the above classification of risk factors. Horses may be subjected to one or more of the above categories, resulting in a severe episode of laminitis. An example of this would be a broodmare that is obese, showing signs of equine metabolic syndrome that has retained her foetal membranes post foaling. The 'perfect storm' scenario has been described, where multiple risk factors culminate into a severe episode of laminitis. Awareness of this overlap helps to identify at very high risk cases and start to implement a prevention plan.

Clinical Signs of Acute Laminitis

Physical examination of the laminitic horse is very important when developing a diagnosis, however there is no pathognomonic clinical sign for laminitis. The most consistent signs of acute laminitis include; increased intensity of the digital pulse to one or more of the limbs, increased frequency of weight shifting between limbs and hesitant foot placement when walking in a tight circle on a hard surface.

Lameness can range from very mild and difficult to observe, to reluctant to walk, 'saw horse' stance or recumbent and unable to stand. Lameness is not a reliable indicator of the severity of the laminitic episode. Severe lamellar pathology can be present in horses that appear relatively comfortable while horses with mild lamellar pathology may appear very lame and recover rapidly to conservative management.

Extreme care must be exercised when evaluating the laminitic horse when maintained in soft orthotics such as Softrides. Boots with a soft gel base mask lameness even in the most severe cases of laminitis. These boots provide minimal support for the distal phalanx and can encourage excessive weight bearing on an already fragile lamellar attachment.

Radiographic and Venographic Findings in Acute Laminitis

The radiographic findings in the acute laminitic horse can be unrewarding. Many times, the radiographic appearance of the foot is considered 'normal'. In a horse with no previous history of either clinical or sub-clinical laminitis, radiographic measurements will rarely show the anticipated typical signs of laminitis such as phalangeal or capsular rotation.

The key points in radiographing the acute laminitis case include;

- Determine the overall hoof health of the foot
- Determine the underlying conformation of each foot
- Determine if the horse has had previous clinical or sub-clinical laminitic episodes
- To develop baseline measurements to compare in follow up radiographs

It is paramount that when repeating radiographs of the laminitis case that a consistent protocol is followed. This requires the horse to be standing on a level surface, both feet standing on blocks of equal height, use of a radio-opaque marker along the dorsal wall along and marking the exact position of the coronary band-hoof wall interface, an object to account for magnification and a x-ray beam centred on the solar rim of the pedal bone.

Subtle changes in vascular filling patterns with a venogram can be observed in the acute laminitis case. Again, a consistent protocol when performing venograms is paramount. By obtaining baseline vascular filling patterns in the acute laminitis case, monitoring how this changes over a period of time can give valuable information.

Management of Acute Laminitis

The treatment of laminitis over the past 100 years has been controversial to say the least and has been centered around the use of various orthotics to support of the foot. The use of the heartbar shoe and dorsal hoof wall resection were promoted heavily in the 1980's whereas the application of wedged heel cuffs for the prevention and treatment of acute laminitis have been in favour through the 1990's to present. The use of heel elevation has created controversy in the scientific community on the biomechanical effects on the dorsal hoof wall. That being said, the observation of how heel elevation effects hoof growth is likely more important in the laminitic horse than the current in vitro studies. Regardless of what orthotic is used to treat a case of laminitis, it is important to monitor the response with regular clinical, radiographic and venographic assessment.

As with any disease process the holy grail is prevention. The investigation into cryotherapy for the prevention of sepsis associated laminitis has been one of the most important break throughs in laminitis research. The clinical use of cryotherapy in colitis cases has led to a reduction in laminitis development in one particular hospital. The understanding that insulin and diet play a major role in endocrine associated laminitis allows veterinarians to further educate owners on nutrition and to implement management strategies that may reduce the incidence of such cases.

Although there is now a large body of scientific evidence surrounding laminitis, the treatment of this debilitating disease remains challenging. Controlled clinical studies are difficult to perform and present ethical dilemma's. Added to this the heterogeneity of clinical laminitis cases makes critical evaluation difficult. However, systematic evaluation of the effects of pharmaceuticals, orthotics and therapeutic shoes on clinical cases of laminitis would be of great benefit to the equine clinician and farrier.

Notes

Foal Diagnostic, Therapeutics and Prognostics

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How does published research help your day to day practice? This talk will focus on recent research that has clinical application for diagnosis, treatment, and/or prognosis for common foal disorders.

Pneumonia in weanling age foals

- Treatment with gallium maltolate was shown to be non-inferior to macrolide therapy for treatment of foals with subclinical pneumonia.(Cohen, Slovis et al. 2015) In this central Kentucky report, 54 foals with ultrasonographic evidence of pulmonary abscesses (>1cm diameter) were treated with either gallium or clarithromycin/rifampin. Non-inferiority was determined when results from one non-compliant farm were excluded.
- Gamithromycin therapy was shown to be non-inferior to azithromycin/rifampin in foals with moderate bronchopneumonia,(Hildebrand, Venner et al. 2015) though tulathromycin was not.(Rutenberg, Venner et al. 2017) In the gamithromycin trial, foals with total abscess scores of 8-20 cm were included, whereas foals with abscess scores 10-15 cm were used in the tulathromycin study. Both trials were controlled, randomized, double-blinded clinical trials and included placebo groups who were not treated.

Macrolide-associated hyperthermia

- Oral treatment with erythromycin causes an acute, significant decrease in terbutaline-induced sweating in healthy foals.(Stieler, Sanchez et al. 2016) This effect began within two days of initiation of therapy and persisted for at least 10 days after cessation of therapy. This provides strong evidence for anhidrosis as the cause of hyperthermia observed in some macrolide-treated foals.
- In a follow-up study, clarithromycin and azithromycin therapy also suppressed sweating, though not to the same extent as erythromycin.(Stieler Stewart, Sanchez et al. 2017) Rifampin therapy did not alter erythromycin-induced alterations in sweating, but therapy with rifampin alone had no effect.

Factors associated with survival

- In a retrospective study of 1065 foals (≤14 days of age) admitted between 1982 and 2008, overall survival was 72.8%.(Giguere, Weber et al. 2017) Factors associated with non-survival in this population included sepsis, temperature, acid base status, neutropenia, and infectious orthopaedic disorders, and sepsis score. Foals admitted in the 2000s were approximately 3.4 times more likely to survive than foals admitted in the 1980s.
- In a retrospective study of 94 hospitalized foals with neonatal encephalopathy, overall survival was 79.8%.(Lyle-Dugas, Giguere et al. 2017) Foals with concurrent disease, high total calcium and low ALP at admission, and that were recumbent or required treatment with vasopressors/inotropes during hospitalisation were significantly less likely to survive. No other therapy was associated with survival, and vasopressor/inotrope administration was likely a proxy for persistent hypotension.
- In a retrospective evaluation of surgical therapy for umbilical infection, 50/65 foals survived to discharge and 43 remained alive one year after surgery.(Oreff, Tatz et al. 2017) Tachycardia,

increased creatinine concentration, young age at presentation, septic arthritis, multiple pathologies, increased time between arrival and surgery, and postoperative complications were associated with decreased long-term survival in univariable analyses. Multivariable analyses were not performed.

- In an observational study of 65 foals (32 septic, 33 non-septic), 63% had a respiratory alkalosis and 58.5% had a strong ion difference (SID_m) acidosis; both alterations were evident in 32%.(Viu, Armengou et al. 2017) Higher L-lactate and venous pCO_2 concentrations were evident in non-surviving foals.

References

- Cohen, N. D., N. M. Slovis, S. Giguere, S. Baker, M. K. Chaffin and L. R. Bernstein (2015). "Gallium maltolate as an alternative to macrolides for treatment of presumed Rhodococcus equi pneumonia in foals." J Vet Intern Med **29**(3): 932-939.
- Giguere, S., E. J. Weber and L. C. Sanchez (2017). "Factors associated with outcome and gradual improvement in survival over time in 1065 equine neonates admitted to an intensive care unit." Equine Vet J **49**(1): 45-50.
- Hildebrand, F., M. Venner and S. Giguere (2015). "Efficacy of gamithromycin for the treatment of foals with mild to moderate bronchopneumonia." J Vet Intern Med **29**(1): 333-338.
- Lyle-Dugas, J., S. Giguere, M. F. Mallicote, R. J. Mackay and L. C. Sanchez (2017). "Factors associated with outcome in 94 hospitalised foals diagnosed with neonatal encephalopathy." Equine Vet J **49**(2): 207-210.
- Oreff, G. L., A. J. Tatz, R. Dahan, G. Segev, D. Berlin and G. Kelmer (2017). "Surgical management and long-term outcome of umbilical infection in 65 foals (2010-2015)." Vet Surg **46**(7): 962-970.
- Rutenberg, D., M. Venner and S. Giguere (2017). "Efficacy of Tulathromycin for the Treatment of Foals with Mild to Moderate Bronchopneumonia." J Vet Intern Med **31**(3): 901-906.
- Stieler, A. L., L. C. Sanchez, M. F. Mallicote, B. B. Martabano, J. A. Burrow and R. J. MacKay (2016). "Macrolide-induced hyperthermia in foals: Role of impaired sweat responses." Equine Vet J **48**(5): 590-594.
- Stieler Stewart, A. L., L. C. Sanchez, M. F. Mallicote, A. L. Muniz, M. S. Westerterp, J. A. Burrow and K. R. Mac (2017). "Effects of clarithromycin, azithromycin and rifampicin on terbutaline-induced sweating in foals." Equine Vet J **49**(5): 624-628.
- Viu, J., L. Armengou, J. Rios, C. Cesarini and E. Jose-Cunilleras (2017). "Acid base imbalances in ill neonatal foals and their association with survival." Equine Vet J **49**(1): 51-57.

Notes

Emergencies During Anaesthesia: when not to keep calm and carry on.

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The anaesthesia related morbidity and mortality in horses is unmatched to the majority of domestic species that undergo general anaesthesia. Even with the advent of newer anaesthetic agents, better monitoring equipment and adequately trained personnel, the incidence of emergencies and complications has not really changed. However, the timing of these events has now shifted from the majority occurring in the intraoperative period to these now occurring in the postoperative period or recovery phase. Regardless, emergencies can occur in any part of the perioperative period and can lead to devastating consequences. This lecture will discuss the main and also the rare complications and emergencies that can occur in horses undergoing general anaesthesia both in a hospital environment and a field setting.

Preoperative period

Drug administration in the perioperative period can be associated with many complications including: inadvertent administration, allergic reactions and perivascular administration of drugs. Complications associated with particular drug administration are also well described.

Cardiovascular instability and electrolyte abnormalities seen especially in the critically ill patient should have adequate resuscitation prior to induction of general anaesthesia.

Induction and maintenance

The induction phase can result in dramatic cardiovascular events or injury to both horse and personnel. Intubation can be difficult especially when there is underlying pathology present.

Adequate and more advanced monitoring of both the cardiovascular and respiratory system of horses during the maintenance phase of inhalational anaesthesia has rendered this time safer than in the past. However, it is also the longest phase of anaesthesia and complications and emergencies associated with the cardiovascular, respiratory and musculoskeletal systemic as well as problems associated with the equipment used to maintain anaesthesia can all result in emergency situations. In comparison, although the main agents used to maintain anaesthesia in the field and considered fairly safe, when emergencies occur, the ability of the practitioner to respond and delivery appropriate therapy can be limited.

Recovery period

The recovery phase in equine anaesthesia is associated with dramatic emergencies associated with the respiratory, cardiovascular, neurological and musculoskeletal system. Due to the size and nature of the horse, it can be difficult to control situations that can lead to disasters.

Notes

Neonatal Sepsis/SIRs therapy

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DIAGNOSIS AND CAUSATIVE ORGANISMS

Blood culture is the gold standard for diagnosis of bacteremia, but isn't very practical in the field. Identification of a causative organism allows for directed antimicrobial therapy as well as determination of patterns in infection, but is rarely practical in the field. If performed, samples should be collected from a large vein (jugular, cephalic, saphenous) after surgical clip and aseptic preparation into a sterile syringe without anticoagulant and immediately placed into blood culture media. Sterile sample collection from a venous catheter at the time of placement is also acceptable. For those foals receiving antimicrobial therapy prior to sample collection, use of a medium with resins may improve microbial recovery. For any medium, care should be taken to infuse the recommended volume of blood to promote optimum recovery.

Two main factors limit the practical usefulness of blood cultures. First, positive results are usually not available for at least 48 hours following submission. Second, a positive blood culture, while extremely specific, is not very sensitive. Thus, the modified "sepsis score" is often used to identify at risk foals, but accuracy is sub-optimal. Thus, while a "positive" score is supportive of sepsis in a suspected animal, a "negative" score alone should not be used to withhold antibiotic therapy from an at risk foal. A recent study showed that use of a cut point of 7, rather than 11, may improve sensitivity/specificity of the scoring system.(Weber, Sanchez et al. 2014)

Whereas gram-positive organisms predominated in the 1940's-1950, *E. coli* has remained the predominant organism isolated from septic foals regardless of clinic location or methodology since that time. Era and geographic location appear to play a major role in prevalence of other causative organisms. The prevalence of gram positive sepsis has increased in Pennsylvania and California(Marsh and Palmer 2001, Theelen, Wilson et al. 2014); *Actinobacillus* spp accounted for approximately 30% of all isolates in Ohio(Stewart, Hinchcliff et al. 2002), while gram positive organisms were isolated from 40.3% of blood cultures in Australia.(Russell, Axon et al. 2008) A Florida study documented a decrease in gram-negative enteric organisms in the 2000s relative to the 1980s.(Sanchez, Giguere et al. 2008) Systemic fungal infections can also occur. Clinical signs include persistent fever and thrush (white plaques on the tongue). The most commonly implicated organism is *Candida albicans*. Risk factors include prolonged hospitalization and immunodeficiency.

TREATMENT

Antimicrobial therapy

Antibiotics provide the basis of therapy for septic foals. Initially a broad-spectrum bactericidal approach must be used due to the possibility of gram positive and negative organisms. Antimicrobial therapy should begin immediately in any foal in which sepsis is suspected and should not be delayed for referral. In human ICUs, institution of antimicrobial therapy should occur within an hour of admission. Therapy can be altered if necessary when sensitivity data become available. A minimum therapeutic course of two weeks is recommended for bacteremic foals without localizing clinical signs. If localizing signs are present, a minimum course of 4 weeks is recommended.

Antimicrobial sensitivity varies by geographic region and organism. Based on available data, a recommended initial therapeutic approach involves combining amikacin or a third-generation cephalosporin with penicillin, ampicillin, or ceftiofur. The use of amikacin should be tempered in light

of the foal's cardiovascular and renal status. If a foal is severely hypovolemic and azotemic, a safer initial choice would likely involve a cephalosporin, such as ceftiofur, alone. Ceftiofur alone is also a practical initial field approach. With aminioglycoside use, monitoring should include creatinine measurement (approx. every 5 days) for potential renal adverse effects.

Cefpodoxime can provide a reasonable alternative for oral administration. Trimethoprim/sulfa combinations are not recommended as an initial therapy for septic foals, as a bacteriocidal alternative is preferable. Fluoroquinolones, such as enrofloxacin, have an excellent spectrum of activity against gram-negative and some gram-positive organisms but have been associated with arthropathy in foals. Thus, use of this agent should be reserved for those cases with documented resistance to other antimicrobial agents and informed owner consent. One should watch closely for the development of white plaques on the tongue (thrush), which can be the first sign of systemic candidiasis. If this occurs, fluconazole has broad-spectrum activity and is effective for many *Candida* spp.

Cardiovascular support

Cardiovascular support is critical in foals with hypovolvemia, acid-base disorders, septic shock or hypotension. When a foal presents in septic shock, fluid resuscitation is critical. Initial choices commonly include a combination of crystalloid and colloid preparations. In lieu of advanced monitoring, physical parameters, such as development of edema, urine output, vital signs, and temperature of the distal limbs, should be examined carefully during therapy. If blood pressure does not normalize with restoration of plasma volume, vasopressors or inotropes (dobutamine, norepinephrine) should be considered. One must take care not to provide excess crystalloid therapy, especially considering sodium load, as foals can develop oedema quickly.

Septic foals can also develop severe transient hypothalamic-pituitary-adrenal (HPA) axis dysfunction, which is associated with decreased survival(Hart, Slovis et al. 2009). Thus, there is some evidence to support use of low-dose hydrocortisone therapy in septic foals with HPA axis dysfunction.(Hart, Barton et al. 2011)

Anti-acid therapy

Uncommonly, sick foals can develop gastric ulcers in the non-glandular or glandular region of the stomach. The use of prophylactic anti-acid therapy is controversial and highly dependent upon clinician preference. Severely ill, predominantly recumbent patients frequently have predominantly alkaline pH patterns than their normal cohorts. Thus, glandular ulcer disease in sick neonates is likely not a strictly an acid-related problem and factors such as alterations in mucosal perfusion may contribute. In addition, gastric alkalinization may contribute to bacterial translocation and has been associated with increased incidence of diarrhea(Furr, Cohen et al. 2012). In situations where acid suppression is indicated (such as need for long-term NSAID use or documented ulceration), primary options for acid suppression in the neonatal foal include omeprazole and ranitidine. Sucralfate remains a possible alternative for ulcer prophylaxis, especially in foals receiving non-steroidal anti-inflammatory drugs, without altering intragastric pH; its effectiveness is unknown in foals.

Immunoglobulin therapy

Foals with documented failure of transfer of passive immunity should be treated with either plasma or colostrum, depending upon the timing. Foals with a serum IgG concentration less than 400 mg/ dL generally require intervention with colostrum or a commercial oral IgG product if less than 12-18 hours of age, or with intravenous plasma thereafter. Foals with a serum IgG between 400 and 800 mg/ dL should receive intervention if other factors are present that may predispose to disease, such as prematurity, dysmaturity, over-crowding, other diseased foals on the farm, or early signs of sepsis.

Colostral quality should be ensured prior to administration. Good quality colostrum should have an IgG concentration greater than 3000 mg/dl, which is typically reflected by a colostral specific gravity greater than 1.060 (colostrometer), a reading of >23% on a sugar refractometer or >16% on an alcohol refractometer. Commercially available frozen equine plasma offers a convenient source of plasma free of alloantibodies and infectious agents. Generally, administration of 1 litre of plasma will result in a 200-300 mg/dL increase in serum IgG in a 50-kg foal.

PROGNOSIS/OUTCOMES

Most retrospective reports cite short-term survival for hospitalized septic neonates near 70%. Various factors have been associated with short-term survival, but have not necessarily been consistent between studies. One report evaluating long-term survival and performance, bacteremic Thoroughbred foals were as likely to start a race as their maternal siblings.(Sanchez, Giguere et al. 2008)

REFERENCES

Furr, M., N. D. Cohen, J. E. Axon, L. C. Sanchez, L. Pantaleon, E. Haggett, R. Campbell and B. Tennent-Brown (2012). "Treatment with histamine-type 2 receptor antagonists and omeprazole increase the risk of diarrhoea in neonatal foals treated in intensive care units." Equine Veterinary Journal **44**: 80-86.

Hart, K. A., M. H. Barton, M. L. Vandenplas and D. J. Hurley (2011). "Effects of low-dose hydrocortisone therapy on immune function in neonatal horses." Pediatr.Res. **70**(1): 72-77.

Hart, K. A., N. M. Slovis and M. H. Barton (2009). "Hypothalamic-Pituitary-Adrenal Axis Dysfunction in Hospitalized Neonatal Foals." Journal of Veterinary Internal Medicine **23**(4): 901-912.

Marsh, P. S. and J. E. Palmer (2001). "Bacterial isolates from blood and their susceptibility patterns in critically ill foals: 543 cases (1991-1998)." J.Am.Vet.Med Assoc. **218**(10): 1608-1610.

Russell, C., J. Axon, A. Blishen and A. Begg (2008). "Blood culture isolates and antimicrobial sensitivities from 427 critically ill neonatal foals." Aust.Vet J **86**(7): 266-271.

Sanchez, L. C., S. Giguere and G. D. Lester (2008). "Factors associated with survival of neonatal foals with bacteremia and racing performance of surviving Thoroughbreds: 423 cases (1982-2007)." J.Am.Vet Med.Assoc. **233**(9): 1446-1452.

Stewart, A. J., K. W. Hinchcliff, W. J. A. Saville, E. Jose-Cunilleras, J. Hardy, C. W. Kohn, S. M. Reed and J. J. Kowalski (2002). "Actinobacillus sp bacteremia in foals: Clinical signs and prognosis." Journal of Veterinary Internal Medicine **16**(4): 464-471.

Theelen, M. J., W. D. Wilson, J. M. Edman, K. G. Magdesian and P. H. Kass (2014). "Temporal trends in prevalence of bacteria isolated from foals with sepsis: 1979-2010." Equine Vet J **46**(2): 169-173.

Weber, E. J., L. C. Sanchez and S. Giguere (2014). "Re-evaluation of the sepsis score in equine neonates." Equine Vet J.

Notes

Equine Neurology Case Studies and discussion

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INTRODUCTION

Neurological localization should be performed prior to diagnostic procedures and treatment. Many cases can be diagnosed by identification of pathognomonic or typical clinical signs, and with experience diagnosis can often be obtained from the neurological examination. Video clips of various neurological horses will be presented with the opportunity for audience participation on lesion localization and differential diagnoses.

CEREBRAL LESIONS

Unilateral cerebral lesion may result in: blindness of contralateral (opposite) visual field, but usually normal PLR and pupil size. Contralateral hemi-paraesthesia and conscious proprioceptive deficits of thoracic and pelvic limbs are seen at rest with cerebral lesions (compared to unconscious proprioceptive deficits that are seen during movement due to a cerebella lesion). There may be compulsive circling and neck bend towards the lesion.

Depending on size of lesion there will be increasing depression, and then decreased level and content of consciousness. Stupor or coma require a bilateral lesion. There may be ipsilateral papilledema. Terminally there may be a decerebrate posture (opisthotonus, extended thoracic and pelvic limbs), coma and convulsions. Brainstem lesions can result in cranial nerve signs.

Cerebral dysfunction may be due to viral encephalitis, verminous myeloencephalitis, brain tumour, cholesteosteatoma, pituitary hyperplasia with or without signs of pars intermedia dysfunction; brain abscess (frequently Strangles), fungal granuloma, etc. CT and MRI are useful diagnostic modalities to allow the localization and prognostication of cerebral masses. Brain surgery is possible with intensive post-operative management.

Cranial nerves

1. On	Olfactory
2. Old	Optic
3. Olympuses'	Oculomotor
4. Towering	Trochlear
5. Tops	Trigeminal
6. A	Abducens
7. Fat	Facial
8. Voluptuous	Vestibulocochlear
9. German	Glossopharyngeal
10. Vaulted	Vagus
11. And	Accessory
12. Hopped	Hypoglossal

Cranial nerve exam

- Menace (2,7)
- Pupil size (2,3, symp)
- PLR (2,3)
- Dolls eye (8,3,4,6)
- Pathologic nystagmus
- Facial symmetry
  - Temporal/masseter (5)
  - Expressive muscles (7)
- Palpebral (5,7)
- Retractor oculi (5,6)
- Gag reflex (9,10)
- Tongue tone (12)

Radial nerve paralysis possibly with humeral or olecranon fracture or even entire brachial plexus avulsion (which looks identical but has a much poorer prognosis). Unable to weight bear (inability to extend elbow, carpus and fetlock). During motion, elbow drops, toe drags, but the pectoral muscles may be able to advance the leg forward half a stride.

SPINAL CORD DISEASES

The five major causes of spinal cord disease in the horse are: cervical vertebral stenosis, equine

protozoal myeloencephalitis (Americas), equine degenerative myeloencephalopathy, EVH-1 myeloencephalopathy, and trauma.

Cervical Vertebral Stenosis commonly referred to as "Wobbler syndrome"
Signalment: can be seen in any horse at any age but is typically seen in young, fast growing horses, most commonly males and Thoroughbreds. Commonly seen when training begins. Clinical Signs: abnormal gait, most obvious on the hind limbs, but because it is a cervical lesion, forelimbs will also be affected. Forelimb gait abnormalities may be subtle; gait abnormalities are usually symmetrical and result in signs of ataxia, weakness, dysmetria and spasticity. The slap test is usually abnormal on both sides.
Gait abnormalities seen: when turned in small circles, at sudden stops, when walked down hill or led over obstacles. Diagnosis: localization of a cord lesion to the cervical region, radiographic detection of vertebral canal narrowing usually possible, otherwise myelogram. Some large bore CT scans can image the entire cervical spine, of which some can be performed standing.

Equine Degenerative Myeloencephalopathy (EDM) Clinically looks similar to CVS; EDM is a diffuse degenerative disease of the equine spinal cord and caudal brain stem. It primarily affects young horses, from birth to 2 yrs. Symmetric tetraparesis and ataxia.

Equine Motor Neuron Disease (EMND) A neurodegenerative disorder of the lower motor neurons resulting in type 1 muscle fiber atrophy. Clinical signs: involuntary fine muscle fasciculations, tendency to lie down frequently, marked weight loss (muscle atrophy) despite a ravenous appetite. Continually shift weight on hind limbs (seem unable to lock their stifles). Short strided gait but no ataxia. Lipofuscin deposits are often observed on the retina. Ophthalmoscopic examination reveals 80% have a mosaic, reticulated “chicken wire” pattern.

EVH-1 Myeloencephalitis due to Equine Herpes Virus I & rarely IV Rapid onset of ataxia and paresis, that often progresses to recumbency; may be concomitant signs of an upper respiratory infection, often occurs 2-8 days after a febrile period. Neurologic deficits of the pelvic limbs often worse than the thoracic limbs, often bladder paralysis with possible urine overflow incontinence, dog sitting, poor tail and anal tone and sometimes fecal retention. Occasionally cerebral signs, which has a very poor prognosis. Signs may stabilize quickly (within 24 hours), but complete recovery may take up to 18 months; some horses gradually deteriorate and require euthanasia.

Spinal Cord Trauma Peracute onset; non-progressive with clinical signs depends upon the level of injury (T2 - S2 injury involves hind limbs only); and from ataxia to acute recumbency. Diagnosis from history, radiology, CT, CSF analysis - see engulfment of RBC by macrophages and yellow tinged CSF (xanthochromia) if over 48 hours old.

Viral encephalitis: Kunjin, Murray valley, Ross River, Hendra
Affected horses often asymptomatic, but can be ataxic, weak, have paresis of the hind limbs, and are occasionally tetraplegic and recumbent. Fever occasionally documented. May have cerebral signs such as depression, stupor, blindness, hyperesthesia or hyperexcitability.

BOTULISM An afebrile progressive muscular paralysis can appear in 3-7 days after ingestion of pre-formed toxin. Initially restlessness, dysphagia, decreased tongue and tail tone, incoordination, ataxia, shaking as they rise or become recumbent due to muscle weakness, inability to rise; sternal recumbency with head at the flanks then lateral recumbency with periods of paddling. Eventually respiratory muscle paralysis and death.

TETANUS (LOCKJAW) Incubation period 2-4 weeks after a deep wound with anaerobic conditions. Initially stiff gait, then sawhorse stance, prolapse of the 3rd eyelid, flared nostrils, lips retracted, erect ears. Signs are exaggerated with stimuli. In lateral recumbency, a stimulus can cause painful whole body muscle contractions. Respiratory paralysis in terminal stages.

References: Large Animal Internal Medicine, 5th Ed, Smith, B. Mosby 2015; Equine Internal Medicine, 4th Ed, Reed SM, Bayly W, Sellon D. Elsevier 2017; Large Animal Neurology, 2nd Ed, Mayhew J. Wiley 2008; Equine Neurology, 2nd Ed, Furr M, Reed S. 2015, Wiley.

Notes

Emergency Management of Fractures

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Appropriate first aid and timely referral is the first step in ensuring the best possible outcome and minimizing the expense of treating horses that sustain a fracture. Conversely, mismanagement and delayed referral can make what was a straightforward repair a costly and sometimes futile effort due to exacerbation of the damage to the bone and soft tissue, infection and development of supporting limb laminitis. Over the last decade, considerable advancements have occurred in the field of equine orthopaedics such as development of locking compression plates, minimally invasive methods for fracture repair, and improved methods for local antimicrobial delivery, along with better anaesthesia and recovery protocols. While some fractures such as open unstable radius fractures in adults still carry a poor prognosis, care should be taken to avoid being overly pessimistic about what is repairable.

Timely application of an appropriate splint is an essential first step for most complete fractures located between the pastern and elbow or proximal tibia. If there is overt instability the limb should be splinted immediately before moving the horse or performing any treatment or diagnostics such as radiographs. Splinting the limb protects the bone ends and soft tissue from further damage. It will also improve the horses comfort and calm an animal that is distressed by the inability to place the limb. The horse will typically relax once they can place the limb on the ground and care should be taken to avoid giving too much sedation. Acepromazine is contraindicated in some horses as it may result in collapse of an animal with high levels of catecholamines and or hypovolemia associated with, sweating or haemorrhage. Alternatively, if the handler and person applying the splint are skilled and work quickly the animal can be restrained with a twich. Length and position(s) of the splint(s) depends on the fracture location. A convenient reference on the topic is available online. http://www.vetmed.ucdavis.edu/ceh/local_resources/pdfs/pubs-HR29-3-Chart-sec.pdf

Splints are applied over a padded bandage with several overlapping layers of strong inelastic tape to prevent movement. Enough padding should be applied to protect the soft tissue without compromising the stability of the splint. Generally 2-4 layers of combine dressing or cotton wool are adequate for lower limb injuries and 6-12 layers when splinting the upper limb. The padding should be moulded to the limb to create a uniform tube thus allowing even contact of the splint. The splint should be strong enough to be effective but as light as possible to avoid it having a pendulum effect on the fractures. Appropriate splints range from commercially available purpose built splints such as the Kimzey Leg Saver, lightweight pine board and PVC pipe.

If the fracture is open or the bone is protruding the skin should be clipped and cleaned with a surgical antiseptic to prevent contamination and a sterile primary dressing is applied over the bone end(s). Exposed bone that is grossly contaminated should be cleaned with saline. The bone and soft tissue should not be exposed to soaps or surgical disinfectants as these substances are cytotoxic. While there are no definitive studies on horses, saline lavage is current standard practice in humans for this reason. Antimicrobial treatment is indicated if the fracture is open or if the skin is severely compromised. Again there is a lack of definitive studies on treatment protocols in horses. In humans with open fractures contaminated with soil, administration of antimicrobials including a penicillin and aminoglycoside within 3-6 hours of injury, saline lavage, thorough debridement and closure of the wound within 24 hours is currently recommended and associated with a decrease in infection rates. The animal should be adequately hydrated before administering potentially nephrotoxic drugs.

Laminitis associated with unilateral weight bearing can occur within 48 hours and can be catastrophic. The contralateral foot should be evaluated carefully before proceeding with definitive

treatment of the fracture. The coronary band should be palpated for a cleft that would indicate sinking. Radiographs should be evaluated critically, noting that in the acute phases the coffin bone will displace distally rather than rotated within the hoof capsule and position can change with weight bearing. Obtaining a good lateral radiograph with the horse weight bearing on a block can prove to be difficult in these cases.

Unilateral weight bearing results in laminitis due to ischemia. Experimentally, walking has been shown to improve perfusion within the foot. Encouraging the animal to move periodically is believed to be beneficial in clinical cases. However, the necessary frequency of doing so is unknown and must be weighed up against the potential for further injury in the fractured limb. Based on some un-published in vitro loading studies altering the position of the foot with a heel wedge does not appear to be beneficial. Currently we are investigating more definitive fixation methods to protect the lamellae in the supporting limb.

Compared to fractures in the limbs, fractures of the skull and mandible present a number of potential problems including respiratory obstruction, ocular damage, contamination from the nasal passage and mouth, neurological damage, and dehydration due to inability or reluctance to drink. Fractures affecting the orbit should be carefully palpated with a gloved hand inserted under the eyelids to assess the configuration and identify any foreign material or shards of bone. If the bone is impinging on the globe it may be reduced by applying pressure from the inside of the orbit. Note that considerable force can be required to completely reduce the fracture. The cornea should be assessed for ulceration and treated appropriately. Exposure keratitis may occur if there is proptosis paralysis of the eyelids. This can be managed with a temporary tarsorrhaphy or topical treatment with ointment of artificial tears. Fractures associated with the nasal passages, paranasal sinuses, and basisphenoid can cause respiratory obstruction due to haemorrhage and swelling. Note that this can occur hours to days after the injury. Airflow should be assessed and monitored. If necessary a tracheostomy may be performed. Alternatively for fractures of the nasal passages and paranasal sinuses nasopharyngeal tube(s) can be inserted and sutured or taped in place. These have the advantage of avoiding a surgical procedure and acting as stents in the airway. Fractures of the mandible and maxilla are often open and communicating with the oral cavity and thus prone to heavy contamination from oral secretions and feed. While little can be done about normal oral secretions, feed should be withheld to minimize movement at the fracture from chewing and impacting the fracture line with feed material. Medical treatment can include administration of intravenous fluids to correct dehydration if indicated and broad spectrum antimicrobials. NSAIDs can be given in appropriated doses for pain relief. .

For transporting equine fracture patients, trucks and large goose neck trailers are more stable and preferable to typical horse floats. The horse should be stably confined to the smallest area possible and given some freedom of movement of its head for balance. Foals and under some specific circumstances adult horses may be transported recumbent with an attendant. Road safety regulations negate this option in many areas. Sedation and even anaesthesia are necessary in some specific circumstances. The horse should never given enough room to get up and down unless it is incapable of standing. Young foals may be carried short distances by two people who each grasp a forearm of the foal and lock arms under the abdomen. The foal will typically remain calm if kept close to the mare.

References

C. E. MEDINA-TORRES, C. UNDERWOOD, C. C. POLLITT, et al. The effect of weightbearing and limb load cycling on equine lamellar perfusion and energy metabolism measured using tissue microdialysis. Equine Veterinary Journal 48 (2016) 114–119

Margaret C. Mudge, VMDa,Lawrence R. Bramlage, DVM, MS. Field Fracture Management. Vet Clin Equine 23 (2007) 117–133

Marissa Srour, BS; Kenji Inaba, MD; Obi Okoye, et al. Prospective Evaluation of Treatment of Open Fractures Effect of Time to Irrigation and Debridement JAMA Surg. 2015;150(4):332-336. doi:10.1001/jamasurg.2014.2022

Suzanne Stewart, Dean Richardson, Ray Boston, and Thomas P. Schaer Risk Factors Associated With Survival to Hospital Dischargeof 54 Horses With Fractures of the Radius Veterinary Surgery 44 (2015) 1036–1041

Andrew van Eps, BVSc, PhDa, Simon N. Collins, PhDa, Christopher C. Pollitt, BVSc, PhD Supporting Limb Laminitis Vet Clin Equine 26 (2010) 287–302

Notes

Metabolic Emergencies of the Equine Endurance Athlete

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The veterinarians' role is integral to endurance riding competition worldwide. Endurance is the only equestrian sport where horses must pass a veterinary examination before, and after each phase of 20-40km during competition, and also upon completion of the ride. The challenges of endurance exercise take place under variable weather conditions and terrain, over distances from 80 to 160km in one day, or up to 400km over multiple days. The welfare of endurance horses relies on the knowledge and experience of riders, and is protected by veterinary examination of horses during competition. As endurance riding has grown as a sport worldwide, speeds of competition at international events have increased, and so has the need for the education of veterinarians about the treatment of endurance related illness and injury. The philosophy of veterinary examination is to detect metabolically compromised horses showing several signs of fatigue at an early stage and withdraw them from competition, but horses that show inconsistent lameness are generally allowed to continue (Robert, 2014).

Horses can be eliminated from competition by the veterinary team based on metabolic or musculoskeletal-related parameters. Elimination rates from endurance competition worldwide range from 10% to 60% depending on the course terrain, speed, and level of competition (Robert, 2014). Lameness is consistently the most common reason for elimination and accounts for an average of 30% of all horses entered in a ride. Elimination for metabolic reasons is less common (8-10% of horses). Despite these stringent controls, the veterinary team at high level endurance competitions anticipates the need to provide emergency treatment to at least 10% of the field for metabolic conditions.

It is widely understood that metabolic problems in endurance horses generally occur secondary to fluid and electrolyte deficits and their associated acid-base abnormalities, metabolic heat accumulation, and substrate depletion (Foreman, 1998). Metabolic problems in endurance horses primarily affect the cardiovascular, gastrointestinal, and musculoskeletal systems, with secondary renal and occasionally neurologic effects. A simplified list of metabolic problems includes poor recovery, colic, exertional myopathy, synchronous diaphragmatic flutter, heat exhaustion, exhausted horse syndrome, and metabolic encephalopathy. In reality, horses can show a spectrum of these clinical sings depending on the degree of metabolic compromise and the organ system most affected.

If detected early, horses may initially show delayed heart rate recovery (HR >64 bpm after 30 minutes recovery period), mild alterations in mental alertness and mild fluid and electrolyte deficits. Once eliminated from further competition, these mildly affected horses will usually recover with cessation of exercise and voluntary food and water intake. These horses should be monitored closely, as they can require treatment at a later time. Horses with more marked fluid and electrolyte deficits tend to show more specific and obvious clinical signs such as abdominal pain, muscle stiffness and/or myoglobinuria, and cardiac arrhythmia (atrial fibrillation most common) and/or generalised exhaustion. Horses showing these signs are metabolic emergencies and require immediate evaluation and treatment. Severely affected horses can rapidly progress to irreversible cardiovascular shock, coma, and death despite aggressive emergency therapy. Thankfully, the incidence of death due metabolic problems in endurance horses in much less common today than it was before stringent veterinary control and education became integral to endurance competition worldwide. Horses can also pass all veterinary inspections and complete the ride with no clinical evidence of metabolic compromise, but go on to develop clinical problems (most commonly colic) within 1 or 2 hours of completing the ride.

The emergency treatment of endurance horses occurs in the field at the ride base, or on the course, with limited facilities or supplies, sometimes in the dark, and often with no ability to perform blood sample analysis. At championship and elite level competition, an on-site 'hospital' is set up and a team of treatment veterinarians are designated to manage multiple horses requiring emergency treatment. The use of on-site point of care monitors for blood analysis can be extremely valuable in some cases, although unfortunately, changes in laboratory parameters can be mild and do not necessarily reflect the severity of metabolic compromise, electrolyte deficits, or differentiate between successful and eliminated horses.

The general principles of emergency treatment of metabolic problems of endurance horses are logically centred on fluid and electrolyte therapy, and active cooling if necessary. Horses can develop large fluid deficits through sweating (10-15 L/hr), and because horses lose relatively more chloride, potassium, and lesser amounts of calcium in sweat, they tend to (but not always) develop a hypochloremic metabolic alkalosis. Although sodium concentrations in sweat are similar to plasma (isotonic), hypotonic rehydration through voluntary water intake can result in a significantly descresed plasma sodium concentration. Hyperventilation as a form of heat loss causing respiratory alkalosis can compound the metabolic alkalosis. As fluid and electrolyte losses continue, horses can develop complex acid-base abnormalities with additional metabolic acidosis due to inadequate tissue perfusion. Dehydration also compounds the problem of overheating in exhausted horses, as extracellular fluid deficits prevent adequate heat loss via sweating. The widespread use of oral electrolyte supplementation of endurance horses during competition is certainly indicated and can attenuate electrolyte deficits and stimulate earlier voluntary water intake (Sampieri, et al, 2006). However, commercial electrolyte pastes have relatively low electrolyte concentrations and may not prevent these metabolic problems.

Mildly affected horses that have some audible intestinal borborygmi present may be managed with single or multiple (every 30-60 mins) administration of isotonic enteral fluids containing NaCl and KCl in 2:1 ratio via nasogastric tube (e.g. 30g NaCl + 15g KCl in 5L H_2O). This may be adequate to prevent deterioration of metabolic status and to restore the horse's appetite and thirst response. The addition of glucose at 14g/L or 2 tbsp/L increases the intestinal absorption of water and electrolytes and also provides a source of energy. Checking for reflux is essential, as is frequent reassessment of metabolic status. The development of ileus can occur at any stage, and it is not uncommon for endurance horses to start refluxing following replacement fluid therapy, either enteral or parenteral. Any horse that is more compromised than this will benefit from intravenous (IV) fluid and electrolyte replacement.

What type and how much fluid should I give?

The traditional approach to IV fluid therapy in endurance horses is to administer large volumes (>30L) of an isotonic replacement fluid. Therefore, the use of any commercial balanced polyionic replacement fluid such as Lactated Ringer's solution or Hartmann's is generally the first line of therapy. Another appropriate choice is 0.9% sodium chloride (normal saline) due to its relatively high chloride concentration and acidifying effects. This is a logical approach based on the most likely fluid, electrolyte, and acid-base derangements associated with endurance exercise, and in the absence of laboratory parameters on which to base more specific therapy. Treatment of metabolic conditions in endurance horses is one of the few indications for the acidifying effects of 0.9% sodium chloride (unless the horse is markedly hypovolaemic and has a metabolic acidosis). However, 0.9% sodium chloride is not available in 5L bags and may not be the most practical choice in the emergency field setting, nor the most appropriate choice in horses that are not hypochloremic.

When blood parameters cannot be measured, routine supplementation of IV fluids with a source of calcium, potassium and magnesium in dehydrated endurance horses is advisable. The most common and practical approach is to add ½ to 1 x 500ml bag of a commercial cattle milk fever preparation such as Calcigol Plus (Ca, Mg and glucose) to a 5L bag of isotonic replacement fluids. Excessive use of calcium in endurance horses may not be warranted, since it plays a role in cell death and apoptosis,

particularly with reperfusion injury (Fielding, 2015). Supplementation with 5% dextrose is indicated in exhausted and neurologic horses.
The body water deficit is often greater than 30 L and the severely dehydrated horse may require 50-100 L over a period of 24 hours. Approximately 1 hour is required to administer a 10-L bolus through a 14-gauge intravenous catheter and standard administration set; multiple catheters or those with large bores (10 gauge) can be used for more rapid administration. Fluid therapy should only be ceased when the thirst reflex and appetite has returned, and when clinical signs have resolved.

How do I treat colic in an endurance horse?

Colic is a common problem identified in eliminated as well as successful endurance horses. Managing endurance horses with colic is not the same as the common colic in the field and can be challenging because many show significant levels of pain. It is very important for treatment veterinarians to recognise that endurance horses with colic have associated metabolic compromise, they rarely have a surgical lesion, and typically respond to aggressive medical therapy (Fielding, 2015; Fielding and Dechant, 2012).

All horses exhibiting signs of colic should have a nasogastric tube passed immediately to check for reflux resulting from ileus. The first line of treatment is fluid therapy along with pain management. Fluid therapy is almost always required because abdominal pain is associated with inadequate gastrointestinal perfusion due to significant fluid and electrolyte deficits and shunting of blood away from splanchnic circulation to exercising muscles for long periods of time. The enteral route for fluid therapy can be attempted if intestinal borborygmi is present, but this is often not ideal because of reduced GIT function associated with ileus. Ideally, intravenous fluids supplemented with calcium and potassium should be administered as outlined previously. Again, it is not uncommon for horses to start refluxing following IV rehydration, so frequent checking for reflux is essential.
Pain management with alpha-2 agonists (xylazine, detomidine) and butorphanol may need to be repeated frequently. Flunixin meglumine (0.5 to 1 mg/kg) can be administered intravenously once the horse is sufficiently rehydrated. Differentiating between ileus (common) and surgical lesions (uncommon) can be challenging and referral to a surgical facility is warranted if the horse is not responding adequately to rehydration and pain management. The most common surgical lesion in endurance horses is small intestinal volvulus (Alexander, 2012). Therefore careful evaluation and prompt referral is essential if a surgical lesion is suspected. Enteritis has also been implicated as a cause of colic in endurance horses during competition (Fielding and Dechant, 2012). If a functional ileus is confirmed, then a continuous rate infusion of lignocaine for 24-48 hours may support GIT motility, reduce inflammation, and assist with providing visceral analgesia.

How do I treat exertional myopathy in an endurance horse?

There are two types of exertional myoapthy that occur depending on when it manifests during the ride. **Type A** (acute myopathy) affects horses during the early stages of the ride (after 5 to 10 km) with signs of muscle stiffness, pain, and reluctance to continue exercising (Harbison and Holt, 1998). Treatment of these depends on the severity of clinical signs. Pain relief with or without fluid therapy (enteral or parenteral) is needed. Cases with myoglobinuria will generally require IV fluid therapy to protect the kidney from myoglobin-induced renal injury. Mild cases often resolve with minimal treatment.

Type B exertional myopathy occur during later stages of the ride (after 60k or more), with a slower onset of signs. Type B exertional myopathies are more common in the endurance horse than other equine athletes and are associated with the multisystemic effects of exhaustion. Intravenous fluids should be administered until urine output is achieved and the urine is grossly clear. Supplementation of calcium in humans with rhabdomyolysis is controversial because of the negative effects of excessive

extracellular calcium on already compromised muscle cells. Low doses of flunixin meglumine (0.5 mg/kg IV) are indicated for inflammation once hypovolemia is resolved and diuresis is achieved. Adequate fluid resuscitation is indicated before release of affected horses because exertional rhabdomyolysis has been associated with severe renal failure in both humans and horses. Horses with myopathies should be monitored closely and reevaluated within 48 hours for azotemia and progress of increased muscle enzymes.

How do I treat SDF in an endurance horse?

Synchronous diaphragmatic flutter (SDF), commonly referred to as "thumps", is typically associated with hypocalcemia, and concurrent metabolic alkalosis, hypochloremia, and hypokalemia are often also present and have been implicated in its development. In many clinical situations there are no measurable electrolyte or acid-base abnormalities (Nagy, 2012). SDF is generally transient and is not a life-threatening condition per se, but it does indicate a metabolic imbalance that if allowed to progress, may become serious (Robert). Many affected horses are otherwise stable hemodynamically and meet all the criteria for adequacy of perfusion. Despite these findings, some horses with SDF may require treatment with intravenous fluids supplemented with calcium for resolution of clinical signs. Calcium gluconate can be added to 5-L bags of crystalloid (0.5 mL of 23% calcium gluconate per kilogram per 5-L bag), and fluids with dilute calcium can be administered as a bolus. Adverse effects of rapid calcium administration include bradycardia and warrant slow administration. Many mildly affected horses recover spontaneously without specific treatment as long as they are rested and are drinking and eating (particularly lucerne hay) (Fielding, 2015).
SDF is more common in horses competing in hot, humid climates, even at the beginning and throughout the ride, and resolve with rest alone. It seems logical that hyperventilation significantly contributes to alkalosis in these situations.

SDF is a recurrent problem for some horses during competition that appear otherwise metabolically normal. Dietary manipulation of the calcium/phosphorus ratio is recommended for these horses, although the possibility of previous phrenic nerve injury and other mechanical factors have been hypothesized, but unproven, causes of chronic, recurrent SDF.

How do I treat an exhausted endurance horse?

If fatigued, dehydrated horses continue to exercise at the same intensity the physiological state of fatigue becomes pathological and fluid, electrolyte and energy deficits require veterinary intervention. The clinical signs of exhausted horse syndrome are varied and multisystemic. The exhausted horse is often adipsic, anorexic, with signs of depressed, generalised weakness, glazed eyes, and symptoms of severe hypovolaemia or shock, ileus, colic, and persistently elevated rectal temperature. Rectal temperature may be misleading, anal tone in the exhausted horse often is reduced and the rectum dilated with air.

Aggressive isotonic fluid therapy with volumes up to 60- 100 L in 24 hrs may be necessary to replace the overall fluid and electrolyte deficits in exhausted horses. Electrolyte and dextrose supplementation is indicated. Evidence of systemic inflammation warrants the administration of flunixin meglumine (0.5-1 m/kg IV) once rehydration and diuresis have been established. Oxygen therapy if available may make the difference in collapsed horses and should be administered at high flow rates (10-15L/min) via nasal insufflation. Providing shade and supplementary cooling will usually be necessary depending on the climate/time of day and clinical status of the patient.

Sequelae of exhausted horse syndrome if not treated rapidly and aggressively early in the disease process include colic, colitis, renal failure, laminitis, coagulative hepathopathies, terminal central

nervous system signs, convulsions and death. Cryotherapy applied to the front and hind feet is indicated for the prevention of laminitis in exhausted horses.
Recovered exhausted horses should not travel for 24-48 hrs and need to be rested for several weeks. Prevention of this condition by diligent veterinary examination during vet checks and rider attention is so very important.

How do I treat a neurologic endurance horse?

Central nervous system disorders can accompany any serious metabolic problem in endurance horses. Neurologic signs are not common, but if present indicate life-threatening, and likely multifactorial metabolic compromise. The exact cause and pathogenesis of metabolic encephalopathy in endurance horses has not been determined, but one or a combination of several disturbances is likely. Possible contributing causes include heat exhaustion, hyponatraemia or hypernatraemia, hypocalcaemia, hypoglycaemia, and hyperammonaemia (Phillipart, 2008).

Management of metabolic encephalopathy must address all possible contributing factors rapidly and aggressively, while maintaining the safety of the public and the horse. Horses are often disoriented, ataxic, agitated, or alternatively may be depressed, head pressing, blind, have seizures or propulsive walking. Sedation may be required to facilitate treatment, particularly if the horse is hyperthermic. Consideration of the effect of sedation on the cardiovascular system and head position contributing to cerebral oedema warrants careful dosing of sedative drugs. Obtaining a venous blood sample for immediate or later analysis should be attempted.

Rapid and aggressive cooling (if indicated) with iced water, and volume expansion with IV fluid therapy should be initiated with multiple, large bore catheters. The immediate goals of therapy are to increase circulating blood volume, cardiac output, cerebral perfusion, and tissue oxygenation, while simultaneously addressing likely deficits in electrolytes and energy substrate. The author's preference is to administer 4ml/kg (2L) of hypertonic (7.2%) NaCl initially, and along with isotonic crystalloid fluids supplemented with Ca, K, and Mg, and 5-10% dextrose. Historically, the use of hypertonic (7.2%) NaCl in dehydrated endurance horses has not been recommended because of the theoretical concern that it will be ineffective and potentially detrimental because of significant intracellular fluid deficits. A more recent clinical study by Fielding and Magdesian (2011) comparing the effects of hypertonic (7.2%) NaCl and isotonic (0.9%) NaCl in metabolically compromised endurance horses demonstrated more rapid restoration of vascular volume, shorter time to urination, and more dilute urine in horses that received hypertonic (7.2%) saline. The additional beneficial osmotic and inflammatory-modulating effects of hypertonic (7.2%) NaCl are particularly indicated in neurologic horse to rapidly improve cerebral perfusion, reduce cerebral oedema, and reduce neuronal cytotoxic oedema.

Other treatments that may be required include diazepam for seizure control, corticosteroids to reduce capillary permeability and provide cell membrane stabilisation, and if possible intranasal oxygen therapy. Non-steroidal anti-inflammatory drugs such as flunixin, meloxicam, or ketoprofen are indicated once the horse is rehydrated and urinating. Blood sample analysis is important, as is checking for reflux and monitoring for sequalae such as diarrhoea, laminitis, renal failure, and coagulopathy. Prognosis for endurance horses affected with metabolic encephalopathy can be guarded.

In general, the prognosis for horses requiring emergency veterinary treatment for metabolic conditions during endurance competition is good. Most horses that are treated respond clinically within 2 to 4 hours and are fully recovered by the following morning. Rest and careful feeding should be continued for several days, and the horse is given a mandatory rest period of at least 60 days before it can compete again. Prevention of serious metabolic compromise is usually possible by insuring the horse is truly fit and ready to compete, both mentally and physically. Providing adequate acclimatization and recovery time post-transport is important. Finally, adequate rider and veterinary education will assist the recognition of early signs of fatigue and when to stop exercising. This is the

responsibility of riders and veterinarians, and considerably more importance and resources should be directed toward education.

References and recommended reading

Foreman JH. The exhausted horse syndrome. *Vet Clin North Am Equine Pract*. 1998;14:205-219.

Whiting JM. The exhausted horse. In: Robinson NE, Sprayberry KA, eds. Current therapy in equine medicine, 6th ed. St Louis: Saunders; 2009.p. 926-929.

Harbison B and Holt J. Management and Intensive Care of the Equine Endurance Athlete. Post Graduate Foundation in Veterinary Science: University of Sydney. 1998.

Schott HC. Challenges of Endurance Exercise: Hydration and Electrolyte Depletion. In.Proceedings of the 17th Kentucky Equine Research Nutrition conference. 2010.p. 94-111.

Sampieri F., Schott HC., Hinchcliff KW., Geor RJ., Jose-Cunilleras E. Effects of oral electrolyte supplementation on endurance horses competing in 80 km rides. *Equine vet J. Suppl*. 2006;38(S36):19-26.

Fielding CL and Madesian KG. Critical Care and Fluid therapy.Cpt 44. In Smith BP.ed. Large Animal Internal Medicine, 5th ed. Missouri: Elseiver;2015.p. 1369-1390.

Fielding CL and Magdesian KG. A comparison of hypertonic (7.2%) and isotonic (0.9%) saline for fluid resuscitation in horses: a randomized, double-blinded, clinical trial. *J Vet Internal Med*. 2011;25:1138-1143.

Robert, C. Veterinary aspects of training and racing endurance horses. In: Hinchcliff, KW, Kaneps, AJ, Geor, RJ, eds. Equine sports medicine and surgery.2nd ed.; Edinburgh: Saunders/Elsevier; 2014. p. 1083-1106.

Philippart MA, Robert C. Neurologic probems of metabolic origin in endurance horses. *Prat* Vet Equine 2008;40:59-68.

Nagy A., Dyson s., Murray JK. A veterinary review of endurance riding as an international competitive sport. *The Vet J*. 2012:194:288-293.

Nagy A, Murray JK, and Dyson SJ. Decriptive epidemiology and risk factors for elimination from Federation Equestre Internationale endurance rides due to lameness and metabolic reasons (2008-2011). *Equine Vet J.* 2014;46:38-44.

Alexander GR and Haines GR. Surgical colic in racing endurance horses. *Equine vet Educ*.2012;24:193-199.

Fielding CL and Dechant JE. Colic in competing endurance horses presenting to referral centres: 36 cases. *Equine Vet J.* 2012; 44:472–475.

Notes

Exertional Heat Illness in Thoroughbred Racehorses – a race between time and temperature

Dr Meg Brownlow

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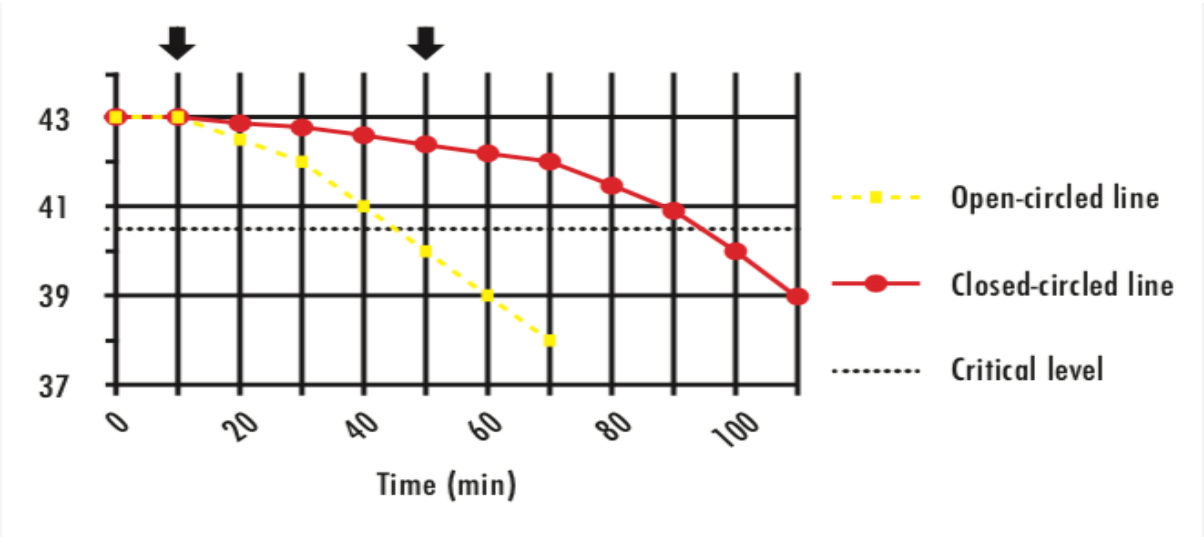
A UNIQUE MEDICAL CHALLENGE BECAUSE OF THE TIME-SENSITIVE NATURE OF TREATMENT. BEST PRACTICE: EARLY DETECTION – RAPID RESPONSE

Exertional heat illness (EHI) is a condition that occurs typically when athletes perform strenuous exercise in warm conditions, but because it is primarily related to metabolic heat production the risk of EHI is often underestimated in cooler conditions. EHI occurs in all athletic species, the human, the greyhound dog and a wide range of sporting horses, but the thoroughbred racehorse is at the high end of the spectrum for EHI because of the intensity of exercise and concomitant higher degree of metabolic heat production. In all species the result is significant elevation of core body temperature (>41°C), which causes a complex pathophysiology with significant effects on the central nervous system, initiation of the systemic inflammatory response syndrome (acute sepsis), and coagulopathies. The end stage (heat stroke) is characterized by widespread organ dysfunction and death.

Much can be learned from a review of EHI in humans, where it is one of the most common causes of sudden death in athletes and remains a significant cause of mortality in the defence forces. Historically, investigators affiliated with the Israeli Defence Force have been leaders in the field of EHI study. Although there are differences in type of exercise and a greater variety of cofounders with human EHI, the approach to treatment and prevention strategies is essentially the same. Human patients die because they are misdiagnosed, not treated early enough or the cooling modality is not appropriate to achieve rapid reversal of hyperthermia. Evidence is overwhelming that immediate and aggressive cooling after diagnosis of EHI ensures survival with limited sequelae. The situation in racehorses is similar, with the condition under-reported and often misdiagnosed. The spectrum of clinical signs acts along a continuum from Level 1 to Level 4; horses can enter at any level depending on the interplay between individual animal factors and climatic conditions operating on the day.

WHY IS EHI REGARDED AS AN EMERGENCY?



This figure illustrates the concept of the **critical thermal maximum** (from Armstrong, 2003). The vertical axis shows the temperature in degrees centigrade and the horizontal axis shows elapsed time up to 100 minutes. The dotted line represents the critical thermal maximum, defined as the temperature above which cell damage may occur (close to 41°C), resulting in multi-organ damage with subsequent increased morbidity and mortality.

The open-circled yellow line and the attendant arrow at 43°C represents an early cooling intervention within 10 minutes of clinical signs being recognized. Note that this early intervention reduces the temperature below the critical thermal maximum at approximately 30 minutes post initiation of the intervention. Comparatively, a late intervention (closed red circles) allows body temperature to remain above the critical thermal maximum for an extended period (80 minutes). Studies have shown that when cooling is delayed there is a significant increase in organ damage, morbidity and mortality after 30 minutes.

The expert consensus statement for EHI in humans is EARLY RECOGNITION – RAPID RESPONSE (Belval et al., 2017). The individual must be cooled as rapidly as possible, ideally within 30 minutes. Horses are no different. To aid in early recognition in thoroughbred racehorses the author has investigated the use of the infrared thermometer to gauge skin temperature. The 'hot' horse (Tsk > 39°C) is considered to be Level 1 and prodromal for escalation to higher levels of EHI. At this stage there are vague signs of CNS dysfunction, which can be confusing, and misdiagnosis is common. There may be irritability, restlessness, head-nodding, and characteristically horses are very difficult to restrain. They have elevated heart and respiratory rates and their perceived level of exertion immediately after racing is always high. Skin vasodilation is most extensive and on humid days horses may sweat profusely, with liquid sweat running off the horse and onto the ground. On hot days with low levels of humidity they may be dry-coated but this is not anhidrosis; sweat simply evaporates very efficiently under these conditions. There are also clinical signs of central blood volume contraction due to re-distribution of blood flow to the skin.

The cooling modality must be directly available: it dictates the cooling rate which also dictates outcome. Racing NSW has developed a mobile spray unit based on a 180-litre plastic water tank with a pump capable of providing 18 litres of ice-cold water per minute, feeding into a large diameter hose. The unit is powered by a 17.5-volt battery, which enables continuous use for approximately 5 to 6 hours. These units have been a 'game-changer' in the treatment of EHI and have become the cornerstone of management at the racetrack. The author has also had much success with the alpha-2 agonist detomidine (Zoetis, US). This is used when horses are displaying definite signs of CNS dysfunction and it appears to completely reverse CNS signs within minutes. Horses are then able to be cooled with minimal risk to horse and handler. The author has also developed a cooling collar, which over the past summer has proved to be most valuable as an adjunct to whole body cooling.

References

Armstrong, L.E. (2003). *Exertional Heat Illness* Champaign, IL, Human Kinetics.

Belval, L.N.., Casa, D.J. et al.,(2018). Consensus statement – Prehospital care of exertional heat stroke. *Prehospital Emergency Care*, May-June 22(3): 392-397.

Notes

Human neonatal Encephalopathy and Sepsis

Associate Professor Helen Liley

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Among live born infants in 2016, an estimated 1.7 million deaths worldwide were attributed to neonatal disorders (GBD 2016) Neonatal encephalopathy accounted for about 30% and sepsis plus other infections for 14%. In Australia, there are about 1500 neonatal deaths per year (2.6 per 1000 live births vs 12.3 per 1000 live births worldwide) with only about 3.1% attributed to infection and 5.2% to hypoxia-ischaemia, (AIHW 2010-2015) demonstrating preventability where standards of antenatal, intrapartum and neonatal care are high.

In years gone by, neonates were thought to be first exposed to both commensal bacteria and potential pathogens when fetal membranes ruptured or labour commenced, (excepting certain pathogens, the so called "TORCH" infections; toxoplasmosis, rubella, CMV, hepatitides and "other", that were known to cause placental and fetal infection). Placental transfer of maternal antibodies and the neonate's unprimed innate immunity were thought to be the only mechanisms to help the newborn to sort commensals from pathogens at the time of birth and to survive. However, studies (especially those using contemporary microbial nucleotide analytic methods which find organisms not detected by usual culture techniques) now demonstrate fetal exposure to microbes from early in pregnancy. Each phase of establishment of the fetal and neonatal microbiome is now considered to contribute to later resilience to pathogens. These processes can be affected by maternal acquisition of pathogens, antibiotic exposure, prematurity, mode of birth and exposure to maternal milk.

In Australia, the most common pathogens causing severe or life-threatening human neonatal sepsis are *Streptococcus agalactiae* (Group B Streptococcus, or GBS) and various gram negative organisms, however a larger range of organisms not detected by routine culture may account for some of the larger number of infants who are investigated for signs of sepsis and elevated inflammatory markers, while an unknown proportion have a sterile inflammatory syndrome. Strategies for prevention (either based on screening for maternal carriage or on recognition of risk factors, combined with intrapartum antibiotic prophylaxis) have reduced the rate of early onset GBS infection by 5-10 fold to about 0.2-0.3 per 10,000.

After the first week, the pattern of pathogenic organisms implicated in neonatal sepsis and the risk factors vary depending on gestation and whether the baby is at home exposed to community organisms or remains hospitalised, at risk of nosocomial infection.

Neonatal Hypoxic Ischaemic Encephalopathy is sometimes due to sepsis (about 1/170 cases of moderate or severe HIE in a recent global study), but causes include other acute, "sentinel" intrapartum events. In about half of cases in high resource countries, the antecedent causes are obscure. Outcomes for moderate or severe HIE have improved with induced hypothermia and neonatal intensive care. Other potential adjunct therapies are in various stages of investigation.

References

1: GBD 2016 Causes of Death Collaborators. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017 Sep 16;390(10100):1151-1210.

2. Australian Institute of Health and Welfare Perinatal Data Visualisations https://www.aihw.gov.au/reports/mothers-babies/perinatal-data-visualisations/contents/data-visualisations Accessed 23 June 2018

Notes

Top 10 Equine Papers of 2018 with a Focus on Emergency Medicine

Associate Professor L. Chris Sanchez

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This talk will summarize information from several relevant papers from the late 2017-early 2018 literature, with an emphasis on those requiring emergency care. Titles are hyperlinked to each paper online, many of which are available as open access. If you would like the full reprint of any paper which is not available to you online, please email me at sanchezl@ufl.edu .

- Cerebrospinal fluid Lyme multiplex assay results are not diagnostic in horses with neuroborreliosis.(Johnson, Johnstone et al. 2018) The title says it all
- Efficacy of the early administration of valacyclovir hydrochloride for the treatment of neuropathogenic equine herpesvirus type-1 infection in horses. (Maxwell, Bentz et al. 2017)
    - Valacyclovir administration decreased viral shedding and viremia as well as severity (but not risk) of ataxia.
    - Study evaluated prophylactic (prior to challenge) and treatment at onset of fever.
    - Antiviral treatment starting at onset of fever (must monitor closely) could be beneficial in an outbreak situation.
- A missense mutation in MYH1 is associated with susceptibility to immune-mediated myositis in Quarter Horses. (Finno, Gianino et al. 2018)
    - Mutation in *MYH1* highly associated with susceptibility to IMM phenotype in Quarter Horses.
    - Novel report of both mutation in *MYH1* and link between myosin mutation and autoimmune disease.
- Evaluation of digital cryotherapy using a commercially available sleeve style ice boot in healthy horses and horses receiving i.v. endotoxin. (Burke, Tomlinson et al. 2018)
    - Contrary to prior reports, use of this commercially available boot decreased mean skin and lamellar temperature in control- and endotoxin-treated horses to temperatures previously reported to reduce likelihood of laminitis.
    - Endotoxin-treated horses had colder skin and lamellae than controls.
    - This boot does not include the foot.
- Clinical outcomes after colopexy through left ventral paramedian incision in 156 thoroughbred broodmares with large colon disorders (1999-2015). (Broyles, Hopper et al. 2018)
    - Ninety-three percent of 156 TB broodmares treated with a colopexy via left ventral paramedian incision survived to hospital discharge. One-year survival and production of a live foal after surgery were 78% and 66%, respectively.
- Factors affecting the prognosis for uterine torsion: the effect of treatment based on measurements of serum progesterone and estradiol concentrations after surgery. (Satoh, Higuchi et al. 2017)
    - Only included seven mares, progesterone/estradiol concentrations in two.
    - All corrected via ventral midline celiotomy; survival 57% mare and foal
- Mare and foal survival and subsequent fertility of mares treated for uterine torsion.(Spoormakers, Graat et al. 2016)
    - Included 189 mares
    - Mean gestation at diagnosis – 283d; 77% prior to d320
    - Survival: mare 90.5%; foal 82.3%
    - Prior to day 320, standing flank laparotomy had higher mare and foal survival
- Caecal intussusception in the horse: Ultrasonographic findings and survival to hospital discharge of 60 cases (2009–2013). (Paulussen, Broux et al. 2018)

    - Caecocolic intussception more common than caecocaecal.
    - Ultrasonographic differentiation possible in 92%
    - In operated horses, survival to discharge was 80% for caecocaecal and 61% for caecocolic intussception.
- Two reports of eosinophilic encephalitis in horses
    - Histologic characterization in Florida horses: S. neurona and H. gingivalis most common. (Zoll, Prakoso et al. 2018)
    - Neurological changes associated with H. gingivalis (Sousa, Neto et al. 2018)
- Oesophageal disorders in horses: Retrospective study of 39 cases. (Bezdekova and Janalik 2018)
    - Young horses and simple obstruction most common
    - Oesophagitis and aspiration pneumonia most common complications
- Prognostic Value and Development of a Scoring System in Horses With Systemic Inflammatory Response Syndrome (Roy, Kwong et al. 2017)
    - SIRS associated with higher mortality in acute GI disease
- Comparison of jugular and transverse facial venous sinus blood analytes in healthy and critically ill adult horses. (Lascola, Vander Werf et al. 2017)
    - Sites can be used interchangeably for blood gas analysis in healthy horses
    - Small differences noted in haematocrit, ionized calcium, and glucose

References

- Bezdekova, B. and P. Janalik (2018). "Oesophageal disorders in horses: Retrospective study of 39 cases." Equine Veterinary Education **30**(2): 94-99.
- Broyles, A. H., S. A. Hopper, J. B. Woodie and A. J. Ruggles (2018). "Clinical outcomes after colopexy through left ventral paramedian incision in 156 thoroughbred broodmares with large colon disorders (1999-2015)." Vet Surg **47**(4): 490-498.
- Burke, M. J., J. E. Tomlinson, A. T. Blikslager, A. L. Johnson and B. L. Dallap-Schaer (2018). "Evaluation of digital cryotherapy using a commercially available sleeve style ice boot in healthy horses and horses receiving i.v. endotoxin." Equine Vet J.
- Finno, C. J., G. Gianino, S. Perumbakkam, Z. J. Williams, M. H. Bordbari, K. L. Gardner, E. Burns, S. C. Peng, S. A. Durward-Akhurst and S. J. Valberg (2018). "A missense mutation in MYH1 is associated with susceptibility to immune-mediated myositis in Quarter Horses." Skeletal Muscle **8**.
- Johnson, A. L., L. K. Johnstone and D. Stefanovski (2018). "Cerebrospinal fluid Lyme multiplex assay results are not diagnostic in horses with neuroborreliosis." Journal of Veterinary Internal Medicine **32**(2): 832-838.
- Lascola, K. M., K. Vander Werf, S. Freese, A. Morgera, D. J. Schaeffer and P. Wilkins (2017). "Comparison of jugular and transverse facial venous sinus blood analytes in healthy and critically ill adult horses." Journal of Veterinary Emergency and Critical Care **27**(2): 198-205.
- Maxwell, L. K., B. G. Bentz, L. L. Gilliam, J. W. Ritchey, N. Pusterla, R. Eberle, T. C. Holbrook, D. McFarlane, G. B. Rezabek, J. Meinkoth, C. Whitfield, C. L. Goad and G. P. Allen (2017). "Efficacy of the early administration of valacyclovir hydrochloride for the treatment of neuropathogenic equine herpesvirus type-1 infection in horses." American Journal of Veterinary Research **78**(10): 1126-1139.
- Paulussen, E., B. Broux, T. van Bergen, L. Lefere, D. De Clercq and G. van Loon (2018). "Caecal intussusception in the horse: Ultrasonographic findings and survival to hospital discharge of 60 cases (2009-2013)." Equine Veterinary Education **30**(5): 241-246.
- Roy, M. F., G. P. S. Kwong, J. Lambert, S. Massie and S. Lockhart (2017). "Prognostic Value and Development of a Scoring System in Horses With Systemic Inflammatory Response Syndrome." Journal of Veterinary Internal Medicine **31**(2): 582-592.

- Satoh, M., T. Higuchi, S. Inoue, T. Gotoh, H. Murase and Y. Nambo (2017). "Factors affecting the prognosis for uterine torsion: the effect of treatment based on measurements of serum progesterone and estradiol concentrations after surgery." J Equine Sci **28**(4): 163-167.
- Sousa, S. H., R. Neto, A. S. Leonardo, A. C. L. Camara and M. B. Castro (2018). "Neurological Changes in Horses Naturally Infected With Halicephalobus gingivalis." Journal of Equine Veterinary Science **60**: 1-5.
- Spoormakers, T. J., E. A. Graat, F. ter Braake, T. A. Stout and H. J. Bergman (2016). "Mare and foal survival and subsequent fertility of mares treated for uterine torsion." Equine Vet J **48**(2): 172-175.
- Zoll, W. M., D. Prakoso, M. Dark, J. Liu, H. Stockdale-Walden and M. T. Long (2018). "Histologic characterization of eosinophilic encephalitis in horses in Florida." J Vet Diagn Invest **30**(3): 442-446.

Notes

RESEARCH SESSION

Sponsored by The University of Queensland and The University of Sydney

Invited Research Abstract: Voriconazole-containing Thermogel for subconjunctivial injection in horses.

R. Cuming, E. Abarca, A. Stewart, A. Wooldridge, S. Duran, W. Ravis, J. Ramapuram

Auburn University, Auburn, Alabama.

Introduction

Keratomycosis is a significant ocular disease of horses and humans worldwide. More economic and effective treatment modalities are needed to improve the prognosis associated with keratomycosis. This study aimed to determine *in vitro* release profiles, transcorneal permeation and ocular injection characteristics of a voriconazole-containing thermogel for subconjunctival space (SCS) injection in horses.

Materials and methods

i) *In vitro* voriconazole release. Liquid (4°C) poly (DL-lactide-co-glycolide-b-ethylene glycol-b-DL-lactide-co-glycolide) (PLGA-PEG-PLGA) (+0mg, 1mg and 5mg voriconazole) was placed in glass vials at 34.5°C to induce gelation. Physiological buffered saline (PBS) was added and samples were collected for 28 days. Voriconazole concentrations in the collected PBS samples were measured via high performance liquid chromatography (HPLC). **ii)** *In vitro* permeation. Permeation of voriconazole (1.5% solution, 0.3% and 1.5% thermogel) through equine corneas and sclerae was determined using a Franz Cell diffusion chamber. PBS was collected for 24 hours and voriconazole concentrations measured via HPLC. **iii)** Characterisation of thermogel subconjunctival space (SCS) injection. Ten normothermic *ex vivo* equine eyes were injected with liquid thermogel (4°C) and the resultant gel deposit described. **iv)** *In vivo* ocular toxicity. SCS thermogel injections were performed in a horse 1 week and 2 hours pre-euthanasia. Toxicity was evaluated via ocular inflammatory scores and histopathology.

Results

i) Voriconazole was released from the PLGA-PEG-PLGA thermogel for >21 days in all groups. Release followed first order kinetics. **ii)** Voriconazole diffused through the cornea and sclera in all groups. Permeation was greater through the sclerae than corneas. **v)** Voriconazole-thermogel was easily able to be injected into the dorsal SCS where it formed a discrete gel deposit which was identifiable by both visual and ultrasonographic assessment. i**v)** Voriconazole-thermogel was easily injected *in vivo* and did not induce adverse reactions in a single pilot horse.

Relevance to equine clinical practice

Voriconazole-containing thermogels have potential application in the treatment of keratomycosis. They are inexpensive and can be easily injected into the SCS by equine clinicians in both hospital and ambulatory settings. Further research is needed, and is currently underway, to evaluate their performance *in vivo*.

Notes

Osteogenic capacities of equine stem cell populations are dependent on intrinsic bone morphogenetic protein (BMP) signalling activity.

Dr Matthew Stewart

INTRODUCTION: In recent decades, stem cell therapies have become increasing prominent in the treatment of musculoskeletal conditions, in both people and veterinary species. In equine practice, stem cell therapies primarily utilize progenitor populations isolated from bone marrow (BM) and adipose tissue (ADI). Although adipose-derived stem cells offer a very large and easily accessible resource, the biological activities and responses of ADI cells are less robust than those of BM cells in many experimental contexts [1,2].

In previous work, we have compared the osteogenic capacities of equine BM and ADI cell populations, derived from the same donors. BM cells were substantially more capable of osteogenic differentiation than ADI cells in standard osteogenic medium. Further, we established that osteogenic medium stimulates up-regulation of several osteo-chondrogenic BMP ligands in BM cells and that inhibition of endogenous BMP signaling in osteogenic BM cultures is sufficient to block differentiation. In light of these findings, we addressed the hypothesis that the relatively poor osteogenic capacity of ADI cells is a result of inadequate endogenous BMP ligand expression and/or induction. To test this hypothesis, we addressed the following questions:

1. Is basal osteo-chondral BMP expression equivalent in BM and ADI cells?
2. Is BMP ligand induction under osteogenic conditions equivalent in BM and ADI cells?
3. Does exogenous BMP-2 ligand supplementation stimulate ADI cell osteogenesis?

MATERIALS & METHODS: Four donor horses were used in this project, under institutional IACUC approval. Bone marrow aspirates were collected from the 5^{th} or 6^{th} sternebrae. Adipose tissue was collected from the fat depot adjacent the tail butt. Donor horses were sedated prior to collections and the collection sites were desensitized with local anesthetic. Both the bone marrow aspirates and adipose cells isolated by collagenase digestion were seeded at low density in DMEM/10% FBS, supplemented with antibiotics and expanded through two passages. P3 cells were transferred to control (as above) or osteogenic culture medium (DMEM/10% FBS supplemented with dexamethasone, beta glycerophosphate and ascorbic acid), for up to 10 days.

Expression of BMP ligands and of osteogenic genes was assessed by quantitative PCR. Induction of alkaline phosphatase (ALP) activity was measured by an enzymatic assay and generation of mineralized matrix was monitored by Alizarin Red staining. Significance of pair-wise comparisons was determined using t tests, while multi-variable analyses were carried out by ANOVA.

RESULTS: Under basal conditions, expression of BMP-2 was consistently 4-5 times higher in BM cells than in ADI cells, while BMP-4 expression was 6-7 times higher in BM cells. Expression of BMPs-6 and -7 was negligible in both cell types. After transfer into osteogenic medium, BM cells generated a mineralized matrix, associated with significant increases in ALP expression and activity and up-regulation of Runx2 and Osterix, while these changes were negligible in ADI cultures (Fig 1). BMP-2 expression increased fourfold in BM cells by day 5 and by over 100-fold by day 10. Induction of BMP-4 expression followed a similar profile. In stark contrast, there was little or no change in expression of either BMP by ADI cells (fourfold or less); comparable to basal expression in BM cells.

Supplementing osteogenic ADI cultures with 100 ng BMP-2/ml significantly increased Runx2, Osterix and ALP expression (Fig 2). The effects of BMP-2 supplementation on matrix mineralization were less prominent. Of note, exogenous BMP-2 administration variably stimulated endogenous BMP-2

expression by 3-10 fold, indicative of a positive feedback regulatory loop. Exogenous BMP-2 had no effect on BMPs-4, 6 or 7.

DISCUSSION/CONCLUSION: This study was conducted to determine whether the relatively poor osteogenic capacity of equine adipose-derived stem cells is a result of low endogenous BMP ligand expression and/or induction. In support of this, BMP-2 and -4 ligand expression was significantly lower in ADI cells than in BM cells under basal conditions, BMP ligand induction was substantially more robust in BM than in ADI cells under osteogenic conditions, and, finally, exogenous BMP-2 administration significantly increased the expression several osteogenic indices in ADI cells. Collectively, these findings suggest that the differential osteogenic capacities of BM- and ADI-derived stem cells derive from differences in endogenous BMP activities. Although ADI cells have several clinical advantages with regard to ease of collection and isolation, exogenous BMP stimulation will likely be necessary if ADI cells are used to support bone repair. The results also suggest that regulation of BMP signaling represents one mechanism by which stem cell lineage predispositions are determined.

REFERENCES: 1. Im G, et al. Do adipose tissue-derived mesenchymal stem cells have the same osteogenic and chondrogenic potential as bone marrow-derived cells? Osteoarthritis Cartilage 2005;13:845–53. 2. Toupadakis CA, et al. Comparison of the osteogenic potential of equine mesenchymal stem cells from bone marrow, adipose tissue, umbilical cord blood, and umbilical cord tissue. Am J Vet Res 2010;71:1237–45.

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Notes

Effects of sample handling on plasma adrenocorticotrophic hormone (ACTH) stability in horses with normal and elevated ACTH concentrations.

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Introduction: Basal ACTH concentration is recommended to diagnose pituitary pars intermedia dysfunction (PPID); however, previous studies have questioned accuracy when sample handling is less than ideal. Objective: to describe the effects on ACTH concentration and subsequent diagnosis of PPID after exposure of samples to various temperatures, separation techniques and storage conditions.

Materials and Methods: Animals: 16 mature horses, 4 with ACTH concentrations considered negative for PPID (<35 pg/mL, group N), 6 with equivocal ACTH concentrations (35- 100 pg/mL, group E) and 6 with ACTH concentrations positive for PPID (>100 pg/mL, group P).

Blood was collected in EDTA tubes and stored for 2, 4, 8, 12, 24 or 48 hours at -20°C, 4°C, 20°C, 30°C; and for 10, 20, 30, 60 and 120 minutes at 40°C or 70°C, as whole blood or after plasma separation by centrifugation or gravity. The effects of freeze-thaw cycles, storage in a silica-containing tube and hemolysis were also investigated. ACTH concentrations were analysed using a chemiluminescent assay and effects of different factors determined by linear mixed effects models.

Results: In group N, processing had a clinically limited effect; however, in groups E and P, temperature, time, separation methods, freeze-thaw cycles and hemolysis induced significant ($P<0.05$) changes in ACTH concentrations resulting in false negative and false positive results.

Relevance to Equine Practice: Further studies are required, but it appears that ACTH should be measured from non-haemolysed samples kept at 4°C, centrifuged within 2 hours and analyzed within 8 hours of collection to prevent misdiagnosis of PPID.

Notes

Biological variation of routine haematology and biochemistry measurands in the horse.

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Introduction
Clinical pathology results are typically interpreted by referring to population-based reference intervals, however the use of individualised (subject-based) reference intervals is more appropriate for measurands with a high degree of variation between individuals. Intrinsic biological variation represents physiological fluctuations over time, and contributes to the total variation of a laboratory result. Knowledge of biological variation can improve clinical decision making by allowing earlier detection of changes from disease processes and providing evidence of clinically significant change in serial results.

As no data are currently available on biological variation of equine haematology or serum biochemistry measurands, the first objective of this study was to determine such variation by repeated sampling of a group of healthy horses representing the mixture of horses seen in the general equine practice environment. Secondary objectives were to calculate indices of individuality to determine whether population-based or individualised reference intervals are suitable, and to calculate reference change values for each measurand, as a guide for clinicians in identifying significant changes in serial results.

Materials and Methods
In a prospective cohort study, thirty-nine privately owned horses were sampled by jugular venipuncture for analysis of haematology and biochemistry measurands at weekly intervals for 6 weeks. Haematology measurands were analysed on the day of collection. Serum was frozen and biochemistry analyses were performed on thawed samples in batches. Duplicate results were obtained and the coefficient of variation was calculated for analytical variation, within-subject variation and between-subject variation were determined. The index of individuality and reference change values were derived for each measurand.

Results
Haematology (red blood cell count, mean corpuscular haemoglobin and mean cell volume) and biochemistry measurands (total protein, globulins, albumin, gamma-glutamyl transferase, aspartate aminotransferase) demonstrated high individuality, indicating that individualised reference intervals are more appropriate for evaluation of these measurands. Two haematology (mean corpuscular haemoglobin concentration and platelets) and three biochemistry measurands (chloride, glucose and sodium) had low individuality, indicating that the use of traditional population-based reference intervals is appropriate for these measurands. Remaining measurands had intermediate individuality, suggesting interpretation of the reference change value should occur with consideration of the population-based reference interval.

Relevance to Clinical Equine Practice
The use of individualised reference intervals is justified for many measurands in horses due to the high individual variation observed for these measurands. This research provides strong evidence that the application of reference change values to serial results can aid in the assessment of individual patient health and response to therapy in horses. Accordingly, these findings emphasise the importance of

the principles of biological variation and the use of serial results for improved clinical decision making and patient management in equine practice.

Ethical animal research

The study was approved by the Animal Care and Ethics Committee, Charles Sturt University (CSU), Wagga Wagga, NSW, Australia (Approval number: A16076). Horse owners provided informed consent for the collection of blood for analysis for research purposes.

Notes

Invited Research Abstract: Novel anti-inflammatory therapies for the treatment of equine SIRS.

Dr Jenni Bauquier

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The systemic inflammatory response occurs commonly in horses with gastrointestinal disease. Current treatments can be inadequate in controlling the inflammatory response, therefore there is a need for novel anti-inflammatory treatments.
Initially, whole blood assays were used to screen different drugs. The novel anti-inflammatory agents (rolipram, azithromycin, ethyl pyruvate, and metformin) were added to aliquots of whole blood (from 4 healthy horses) diluted 1:1 with cell culture medium over a range of concentrations, after which LPS was added (*E. Coli* O55:B5; 1 µg/ml). Samples were then incubated at 37°C (98.6°F) for 21 hours. The anti-inflammatory effect was measured by determining tumour necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) concentrations in supernatant. Inhibition of TNF-α production occurred for all drugs, with rolipram being most efficacious, having an IC_{50} of 1.2 µM (95% CI 0.4-3.5 µM). However, its use may be limited in horses due to deleterious side effects, therefore further agents were investigated. The role of bacterial toxins other than LPS was also included in further investigations.

A further novel drug, BIRB-796, works intracellularly, inhibiting the enzyme p38 MAPK in inflammatory cells and reducing transcription of inflammatory cytokines. Using the same model as above, increasing concentrations were added to equine whole blood diluted 1:1 with cell culture medium, and stimulated with either lipopolysaccharide (LPS), lipoteichoic acid (LTA) or peptidoglycan (PGN). Supernatant concentrations of TNF-α and IL-1β were measured. BIRB-796 was then administered to 6 healthy horses at 0.5 mg/kg IV for safety analysis. Following this, the same formulation or placebo was administered IV at the same dose to 6 horses in a cross-over design, followed by 30 ng/kg LPS IV over 30 minutes. Clinical parameters were measured and blood samples taken every 30 minutes for 6 hours for leukocyte count and measurement of TNF-α.

BIRB-796 showed good potency *in vitro* with IC_{50} values for TNF-α inhibition (geometric mean (95% CI)) of 0.07 (-0.18-0.25) µM for LPS, 0.03 (-0.21-0.27) µM for LTA and 0.46 (-0.69-1.60) µM for PGN. Maximum inhibition values (mean ± SEM) were 66.7 (±9.2)%, 71.4 (±8.5)% and 73.1 (±12.2)% for LPS, LTA and PGN respectively.

No abnormalities were found during or up to 24 hours after IV administration of BIRB-796 to healthy horses. When pre-treated with BIRB-796, horses given IV LPS had significant improvements in heart rate, rectal temperature, blood pressure, leukocyte count, and TNF-α concentration.

BIRB-796 significantly reduced the systemic effects of LPS. This compound might be a useful treatment in equine clinical cases of SIRS. Further evaluation in a clinical setting is needed to confirm its value in the management of equine SIRS.

Notes

Differences in cell marker expression by equine bone marrow-derived mesenchymal stem cells associated with blood antigen type and breed.

Christopher B. Riley[1], J. Lacy Kamm[1], Natalie Parlane[2], Erica Gee[1], Keren E. Dittmer[1], C. Wayne McIlwraith[3]

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Introduction

There is significant variability in the clinical outcomes following therapeutic use of mesenchymal stem cells (MSCs) for equine musculoskeletal disease. Among the possible causes are differences in the immunogenic response to MSCs. The characterization of MSCs from different donor sources is important in the identification of less immunogenic allogeneic donor MSCs, and may facilitate standardisation of clinical treatments. The aim of this study was to define marker expression differences between two breeds, and horses with different erythrocyte antigen profiles.

Materials and Methods

MSCs derived from the bone marrow of 18 Thoroughbred (TBs) and 18 Standardbreds (STBs), including 8 blood erythrocyte Aa, Ca, and Qa antigen negative (EAN) horses, were evaluated. From each horse MSCs from passages 2, 4, 6, and 8 were labelled and evaluated by flow cytometry. Cell surface expression of CD11a/18, CD44, CD90 and major histocompatibility (MHC) class II antigens were assessed. Trilineage assays for differentiation into adipogenic, chondrogenic and osteogenic cells lines were used to verify characterization of the MSCs.

Results

Differences in MSC marker expression between breeds and blood antigen types over time were significant. Standardbreds had lower expression of MHC class II antigens than did TBs at passages 2, 4 and 6, and may be the less immunogenic MSC donor as compared to TBs. CD90 antigen expression was significantly higher in EAN STBs compared to blood erythrocyte antigen positive STBs for all passages. All MSC samples showed high expression of CD44 and low expression of CD11a/18.

Relevance to Clinical Equine Practice

Blood erythrocyte antigen negative STBs are preferred donors of MSCs for allogenic treatment.

This work was approved by the Massey University Animal Ethics Committee (MUAEC protocol 15/13), and funded by the New Zealand Equine Trust and the C. Wayne and Nancy Goodman McIlwraith Equine Orthopaedic Scholarship Fund.

Notes

The effect of internal lag screw fixation on compression and loading responses of type III distal phalangeal fractures in horses.

Dr Matthew Stewart

Introduction: Type III distal phalangeal fractures in horses can be treated conservatively or surgically. Intuitively, fracture reduction should improve repair and mitigate secondary arthritis; however, retrospective outcomes do not support this. This study was carried out to determine the effects of lag screw stabilization and loading on these fractures.

Materials & methods: Type III fractures were created in 12 paired digits. Fractures were reduced with 4.5 or 5.5mm cortical screws in lag fashion, or left as non-reduced controls. Fracture reduction was assessed by comparing pre- and post-reduction fracture gap measurements. Effects of incremental compressive loading and terminal unloading were measured from serial fluoroscopic images.

Results: 5.5mm screws significantly reduced fracture gaps. 4.5mm screws did not. Under load, articular fracture gaps in all groups were compressed, and were closed at 540 Kg loads. Distally, loading increased fracture gaps in 'Control' and '4.5mm' groups. Reduction with 5.5mm screws significantly constrained distal expansion. After loading was removed, articular fracture gaps in Controls were wider than the residual gaps in reduced phalanges, although not significantly.

Discussion/conclusion: Only 5.5 mm screws reliably reduced the fracture gaps, and were also more effective than 4.5 mm screws in stabilizing distal fracture gaps under load. Fluoroscopic images clearly showed that loading the articular surface compressed the fracture gap, with complementary expansion of the fracture gap distally. These findings explain the success of conservative management of these fractures, although lag screw fixation does prevent cyclical fracture compression-expansion.

Acknowledgment: There are no proprietary interests or conflicts to report.

Notes

Septic Peritonitis in the Post Partum Mare.

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Introduction

Septic peritonitis in the post-partum period is a potentially life-threatening condition that requires early diagnosis and aggressive treatment. The inciting cause most frequently involves compromise or rupture of either the urogenital and gastrointestinal tracts, but the presentation of these cases can be frustratingly similar. The aims of this study were; 1) to report the most frequent causes of septic peritonitis and associated outcome in the post-partum mare and 2) to evaluate the influence of various clinical and clinicopathological parameters on primary diagnosis.

Materials and Methods

Records were reviewed for cases of peritonitis in mares presented to Scone Equine Hospital from 2005-2017. Cases were included if they presented 7 or less days post-partum, had a final diagnosis confirmed by exploratory laparotomy or post mortem examination, and either a peritoneal white cell count (WCC) $\geq 10x10^9$ cells/l or degenerate neutrophils or plant material present in a peritoneal fluid sample. Clinical findings and clinicopathological data was analysed using a commercially available statistics program (JMP11, Carey, NC). When evaluating the influence of parameters on primary diagnosis, diagnoses were split into two categories; gastrointestinal (GIT) and uterine disease. Univariable analysis was completed using Wilcoxon/Kruskal-Wallis Tests or Fisher's exact test as appropriate. Statistical significance was considered at $P<0.05$.

Results

101 cases of peritonitis in the postpartum mare were admitted during this period, and 67 horses met the inclusion criteria. Of these, 37 (55%) suffered a uterine rupture, 14 (21%) a GIT rupture, 6 (9%) a mesenteric rent with strangulating small intestine, 3 (4%) a mesocolic tear and avascular necrosis of the small colon, 2 (3%) a uroperitoneum and 5 (7%) 'other', which included rectal prolapse, vaginal tears, and necrosis of the GIT and urogenital tracts. Uterine cases presented significantly longer post foaling (median 2 days IQR 1-3 days) than GIT cases (median 1 day, IQR 1-1 day) (P=0.0026). PCV was significantly higher in the GIT group (median 50.9%, IQR 47-64.9%) compared to the uterine group (median 45.5%, IQR 37-54.1%) (P= 0.0059). Peritoneal lactate (mmol/l) was significantly higher in the uterine (median 9.8, IQR 7.5-16.7) than GIT group (median 3.8, IQR 2.9-4.7) (P=0.0016) and the GIT group had a significantly lower peritoneal RBC ($x10^{12}$/l) than the uterine (GIT median 0.22, IQR 0.14-1.31 vs uterine median 0.71, IQR 0.29-6.7) (P=0.0243). Survival to hospital discharge was more likely in uterine (68%) than GIT group (4%) (P<0.001, OR 49.8). Heart rate on admission, peripheral WCC or band neutrophil count, blood lactate and the presence of toxic changes in neutrophils on haematology or peritoneal WCC was not associated with final diagnosis.

Relevance to clinical equine practice

Uterine tears were the most common cause of septic peritonitis in the post-partum mare in this study. The GIT group were more likely to present earlier and have a higher PCV and peritoneal RBC count and lower peritoneal lactate on admission, but no single clinical or clinicopathological

parameter could be used to definitively differentiate the various causes of peritonitis. Exploratory laparotomy therefore remains a useful tool to establish the definitive diagnosis.

Notes

Prevalence and risk factors associated with Equine Asthma in South Australian horses.

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Introduction: Equine asthma is a common disease of domesticated horses, especially in the Northern Hemisphere. However, there are few studies of Australian horses and the prevalence of equine asthma in these horses is unknown.

Materials and methods: An online survey ("Survey Gizmo") was disseminated to South Australian horse owners via an email and social media campaign and was available for 6 weeks. An email reminder was sent after 3 weeks. Respiratory disease suggestive of equine asthma (during the previous year) was defined according to the "horse owner assessed respiratory signs index" (HOARSI). Risk factors were evaluated using univariable and multivariable logistic regression models.

Results: 439 responses were received, of which 361 were usable. These included 134 mares, 220 geldings and 7 stallions of varying breeds and disciplines with mean (±sd) age of 11.9 (±6.7) years. 236/361 (65%) horses were identified as having equine asthma, according to the HOARSI score, despite only 32% owners reporting that they thought their horse was affected by respiratory disease and only 8.6% horses receiving a veterinary diagnosis. Cases were further classified as mild (136; 57.6%), moderate (87; 36.9%) or severe (13; 5.5%). Clinical signs were more prevalent in summer (70%) than in other seasons. The majority (63%) of horses were housed outdoors all year round. The final multivariable logistic regression model revealed the following risk factors for equine asthma: age, use and time at pasture, whilst feeding of wet hay reduced the risk.

Relevance: Equine asthma is a common, under-diagnosed issue affecting South Australian horses.

This study has been approved by the Human Research Ethics Committee of the University of Adelaide in accordance with the NHMCR National Statement on Ethical Conduct in Human Research.

Notes

Insulin dysregulation in horses with systemic inflammatory response syndrome.

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Introduction: Systemic inflammation is a cause of insulin dysregulation in many species but the insulin and glucose dynamics in adult horses diagnosed with systemic inflammatory response syndrome (SIRS) are poorly documented. We hypothesised that in horses with SIRS, insulin and glucose dynamics would be altered and associated with survival.

Materials and Methods: Prospective study enrolling adult horses diagnosed with SIRS admitted to a referral hospital in which serum insulin and glucose concentrations were measured. Horses were grouped by outcome (survival, hyperinsulinemia and hyperglycemia) and compared with $p < 0.05$ considered significant.

Results: Fifty-eight horses were included in the study and 36 (62%) survived. At admission, 21 horses (36%) were hyperinsulinemic and 44 horses (88%) were hyperglycemic, with survivors having significantly higher serum insulin and a significantly lower serum glucose concentration. Horses diagnosed with hyperinsulinemia at any time during hospitalization were 4 times more likely to survive whereas horses that were hyperglycemic at any time during hospitalization were 5 times less likely to survive. Serum glucose concentration and presence of hyperglycemia both were associated with severity of disease. Insulin/glucose ratio, reflecting insulin secretion, was significantly higher in survivors whereas glucose/insulin ratio, reflecting peripheral tissue insulin resistance, was significantly lower in non-survivors. Only in survivors was there a significant correlation between serum insulin and glucose concentrations.

Relevance to Clinical Equine Practice: Hyperinsulinemia and hyperglycemia are common features of SIRS in horses but those presenting with relative hypoinsulinemia and corresponding hyperglycemia suggestive of endocrine pancreatic dysfunction have a worse prognosis.

Notes