Management of wounds and septic synovial cavities in horses

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Introduction

Traumatic injuries require a careful investigation to determine whether underlying structures such as blood vessels, nerves, muscles, tendons, tendonsheaths, joints or bone are affected. In many cases careful visual inspection and digital exploration of the wound helps the experienced practitioner to determine the extent of the damage. In complicated cases further diagnostic modalities such as radiography, ultrasound or aspiration and analysis of synovial fluid may be required.

However, even traumatic skin wounds without underlying tissue damage are challenging and often labor intensive for both horse owners and equine practitioners. A retrospective study of horses with traumatic wounds recently determined that primary closure was successful in only 24% of horses in which it was attempted. To compound the problem, repair of wounds by second intention is subject to numerous complications that compromise outcome in the horse, including chronic inflammation, poor contraction, development of exuberant granulation tissue (proud flesh), and slow epithelialization.

Debridement is an important step in the initial treatment of a wound, particularly when necrosis, exposed cortical bone, or frayed tendons are present; it can be achieved sharp surgical debridement or hydrosurgically (Versajet). Debridement of non-viable tissue reduces the duration of the inflammatory phase. After thorough debridement, it is advisable to dress the wound briefly in an effort to accelerate biological processes. Bandaging can be combined with topical therapy to enhance the inflammatory response.

Contamination or infection of a synovial cavity (synovial sepsis) is an important complication in traumatic wounds. In most cases, the inoculation of a synovial cavity with micro-organisms may arise following penetration by a traumatic wound.

Clinical signs

Severe lameness, typically in a horse with a wound in the region of a synovial structure. The region of the synovial structure will be swollen as the result of distension of the synovial structure and oedema, hot and painful on palpation (i.e. there will be a local cellulitis). In horses in which the synovial cavity drains through the wound, the severity of lameness and cellulitis may be dramatically reduced. The absence of systemic clinical signs, such as pyrexia, or increases in peripheral blood white blood cell count do not rule out synovial sepsis.

Investigation

Synovial fluid analysis, distension of the synovial cavity to identify leakage and diagnostic imaging are the principal investigative techniques. Of these, the first two are the more important – they are also relatively easily performed.

Synovial fluid analysis

A sample is obtained using aseptic technique from site remote to any wound.

	Normal	**Infected**
Gross characteristics	Clear straw yellow, viscous	Turbid; may be haemorrhagic; reduced viscosity
Cytology (EDTA tube)	$0.2x10^9$/L <10% neutrophils	>$10x10^9$/L >90% neutrophils (may be degenerate & toxic); may find bacteria
Total protein content (EDTA or plain tube)	18-20g/L	>40g/L
Bacteriology (plain tube or broth)	Sterile	50-70% +ve; organism related to pathogenesis

The micro-organisms most commonly isolated from synovial cavities infected following penetration by a traumatic wound are coliforms, Staph spp, Strep spp and anaerobes; following iatrogenic infection the most frequent isolate is Staph aureus. There are a range of possible isolates, including coliforms, Staph spp, Strep spp and Actinobacillus spp from foals with infectious polyarthritis and osteomyelitis syndrome.

Distension of synovial cavity to identify leakage

The synovial cavity is pressurised by distending it with Hartmann's solution injected through the same needle used to obtain a sample. Can be performed using radiographic contrast agent but radiography is seldom required to identify leakage.

Diagnostic imaging

Radiography and ultrasonography are used to identify foreign bodies or concurrent bony or soft tissue injuries. In foals with infectious polyarthritis and osteomyelitis syndrome radiography is used to identify osteomyelitis lesions.

Treatment

Septic synovitis is an emergency and should be treated without delay. The most important elements of treatment are lavage of the synovial cavity with a large volume of Hartmann's solution (ideally >10L) and the administration of antimicrobials. Bandaging, rest and medication of the synovial cavity once infection has resolved are other elements of treatment that require consideration. Wound management techniques and tetanus prophylaxis may also be appropriate.

Lavage may be performed through wide bore needles, cannulae or at endoscopy (arthroscopy, tenoscopy or bursoscopy). A major advantage of using endoscopy is that it allows the synovial cavity to be directly visualised and therefore assessment of any concurrent injuries (e.g. chip fractures), lavage to be directed to specific areas and debridement of infected or devitalised tissues.

Broad spectrum antimicrobials (e.g. penicillin and gentamicin) should be administered systemically pending the results of bacteriology. Local administration (in addition to systemic) is a useful technique for achieving high concentrations of antimicrobial within the infected synovial cavity and associated tissues. The main techniques are intra-synovial injection, intra-synovial depot, intra-venous regional perfusion and intra-osseous perfusion; gentamicin or amikacin (both aminoglycosides) are the agents commonly used for these techniques. Antimicrobials should be administered for long period to avoid recrudescence of infection; a guideline is 14 days following the resolution of infection, the first 7 days parenterally and the last 7 days orally.

The response to treatment should monitored by severity of lameness (NB the masking effect of any NSAIDs administered), sequential synovial fluid analysis and diagnostic imaging.

Prognosis

Reported rates of survival indicate that it is possible to eliminate infection in >80% cases. There is greater variation in the range of rates for return to previous level of performance, with a number of studies reporting no more than 50% due to irreparable damage (e.g. OA, tendon adhesions). Negative prognostic factors are likely to include length of time before treatment, presence of osteomyelitis and pannus formation.