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In the horse, proximal sesamoidean osteomyelitis is an uncommon condition that can cause severe lameness. In this case, aggressive surgical and medical therapy was necessary to cause significant improvement in lameness in a 6-year-old American Quarter Horse. This case report represents the progression of osteomyelitis into the first reported proximal sesamoidean sequestrum of the horse and its surgical treatment with resulting favorable outcome.

CASE REPORT

Proximal Sesamoid Sequestrum in a Horse

Suzann A. Carson Dunkerley, DVM
R. Reid Hanson, DVM
Jay Humburg, DVM, MS
Department of Large Animal Surgery and Medicine
College of Veterinary Medicine
Auburn University, Alabama 36849-5522

Introduction

Proximal sesamoidean osteomyelitis is an uncommon condition that can cause severe lameness in the horse.[1] The development of such osteomyelitis into a sesamoidean sequestrum has not been reported to the authors' knowledge. While sequestration of the cortex of long bones often carries a rapid healing time and good prognosis once the lesion is curetted, osteomyelitis of the proximal sesamoid was found to heal slowly and carry a poor prognosis for athletic soundness.[2] This may be in part due to the limited blood supply to the proximal sesamoidean bones.[1,3,4] This case report represents the progression of osteomyelitis into the first reported proximal sesamoidean sequestrum of the horse and its surgical treatment with resulting favorable outcome.

Case Report

A 6-year-old, 450-kg American Quarter Horse mare was referred for evaluation of a 2-cm laceration on the plantar-medial aspect of the right hind fetlock. The mare had incurred the wound when cast in a stall 2 weeks earlier. The horse did not improve with stall rest, phenylbutazone (4.4 mg/kg, PO, q24h) and topical wound management during the 2-week period along with procaine penicillin (22,000 IU/kg, IM, q12h) and gentamicin sulfate (4.4 mg/kg, IV, q12h) for 3 consecutive days. The horse was referred to the Auburn University Large Animal Clinic for further evaluation.

The horse had a grade 5/5 right hind limb lameness at admission. There was a 2-cm diameter plantar-medial laceration with minimal drainage and a moderate amount of granulation tissue. Mild swelling was present over the medial proximal sesamoid and a painful response was elicited by palpation of this area. No distention of the fetlock joint capsule was evident.

Lameness was significantly reduced with the plantar and deep metatarsal nerves blocked at the level of the distal end of the second and fourth metatarsus. A 0.5-cm diameter, 4-cm long, grade 2 core lesion of the deep digital flexor tendon (DDFT) was evident with ultrasound examination 4 cm above the fetlock. In addition, a 0.5-cm diameter hypoechoic area in the medial, superficial sesamoidian ligament 1 cm distal to the sesamoid was noted. Three slender, small

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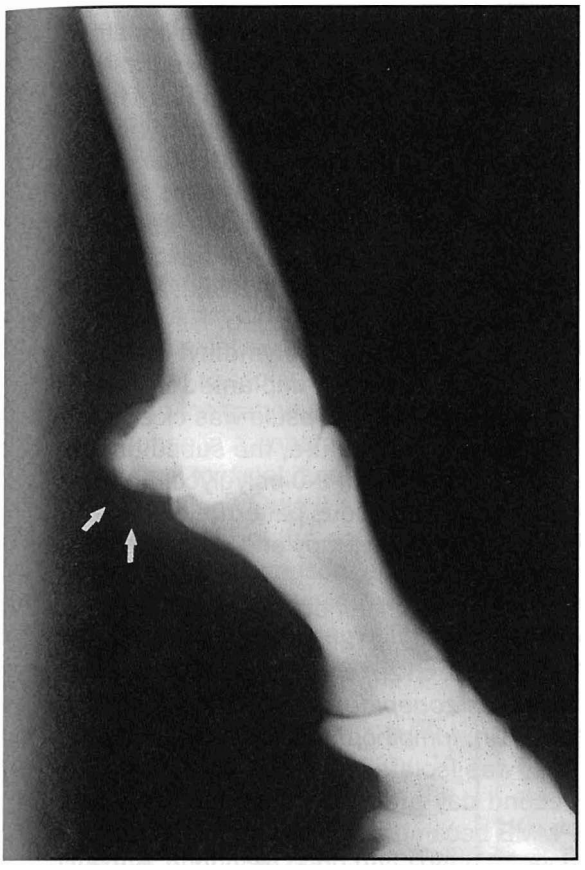

FIG. 1 — Dorsomedial to plantar lateral oblique radiograph 2 weeks after injury to the caudomedial region of the right hind fetlock of a mare. Two basilar chip fractures are noted (arrows).

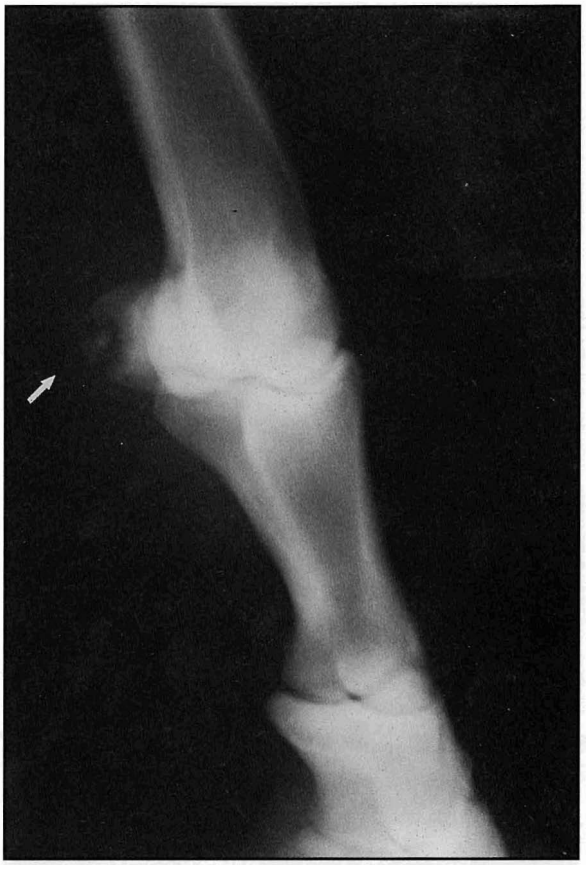

FIG. 2 — Dorsomedial to plantar lateral oblique radiograph 6 weeks after injury to the caudomedial aspect of the right hind fetlock of a mare. Sequestration of the medial proximal sesamoid bone is noted (arrow).

fragments of bone 1 cm from the plantar-medial basilar aspect of the medial, proximal sesamoid were evident on radiographs (Fig. 1). Synovial fluid collected via a dorsal approach to the fetlock joint contained 1,000 WBC/µl. No fluid could be collected from the flexor tendon sheath. A diagnosis of basilar sesamoidian fracture, DDFT core lesion, and sesamoidian desmitis were made.

Treatment

The horse was treated with gentamicin sulfate (4.4 mg/kg, IV, q12h) and procaine penicillin (22,000 IU/kg, IM, q12h) for 5 consecutive days, phenylbutazone (2.2 mg/kg, PO, q12h) for 7 days in addition to daily bandage changes and stall rest. The antibiotics were prescribed, although the cell count in the joint fluid was low, because of the considerable pain and local temperature rise. The horse was not willing to bear weight on the heel until a wooden block was taped to the heel, which improved the lameness to grade 4/5. Thus, a shoe with a 4-cm heel elevation was applied. The horse was discharged with instructions to continue stall rest, phenylbutazone (2.2 mg/kg, PO, q24h), daily rebandaging, and to bring the horse back for a recheck in 1 month.

The horse was re-admitted to the clinic 21 days later. The lameness had improved over the 3-week period prior to admission but suddenly worsened the day before readmission. The horse had a grade 5/5 right hind lameness and the laceration on the plantar-medial aspect of the right hind fetlock was 1 cm in length and was discharging purulent exudate. Radiographs revealed a large 2-cm sequestrum of the plantar aspect of the medial proximal sesamoid bone (Fig. 2).

The sequestrum was removed and the involucrum curetted under general anesthesia with the aid of intraoperative radiographs. Aerobic culture of the sequested bone identified *Escherichia coli* and *Pantoea agglomerans* which were sensitive to gentamicin. *Streptococcus zooepidemicus* was

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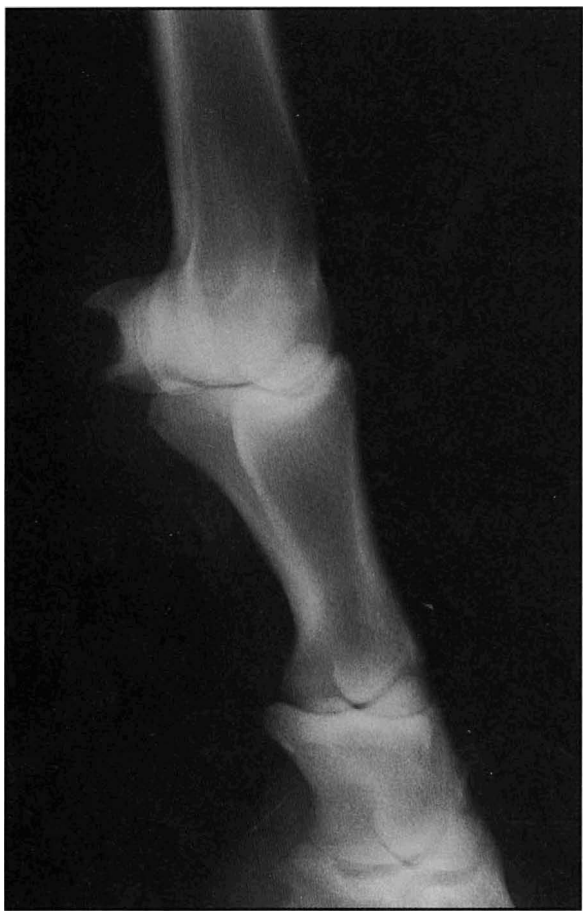

FIG. 3 — Dorsomedial to plantar lateral oblique radiograph of the right hind fetlock of a mare. Post-surgical removal of the sequestrum in the medial, proximal sesamoid bone.

also cultured which was sensitive to penicillin and trimethoprim/sulfa. The mare was treated with gentamicin sulfate (6 mg/kg, IV, q24h), procaine penicillin (22,000 IU/kg, IM, q12h) and phenylbutazone (4.4 mg/kg, PO, q12h) for 3 days. The procaine penicillin was continued an additional 2 days and the phenylbutazone dose was decreased (2.2 mg/kg, PO, q12h) over the following 6 days. The lameness reduced to grade 2/5 during the following 17 days.

On the 18th postoperative day, large amounts of non-viscous fluid began to drain from the original wound site. By the 21st postoperative day the mare had become increasingly lame to grade 5/5. Although the limb had no change in the swelling from the first day of presentation, radiographs showed a mild sesamoid sclerosis with no additional sequestration or fragmentation (Fig. 3). The mare was placed back on phenylbutazone (4.4 mg/kg, PO, q12h) and the bandage continued to be changed daily.

Because of the lack of improvement at 26 days after the initial surgery, the wound was re-explored under general anesthesia The involucrum site was again curetted. Synovial fluid obtained from an intraoperative centesis from the dorsal aspect of the fetlock joint was yellow, clear, and slightly viscous, with a specific gravity of 1.026, a total protein concentration of 3.8 g/dl, 30,000 RBC/µl, and 800 WBC/µl. No toxic neutrophils or bacteria were seen. Three gentamicin sulfate impregnated polymethyl-methacrylate beads (PMMB) were implanted in the involucrum site. The joint capsule was closed with 2-0 polyglycolic acid suture, the subcutaneous tissue was closed with 2-0 polyglycolic acid suture in a simple continuous pattern, and the skin was closed with 0 polypropylene in simple interrupted pattern.

Treatment with trimethroprim-sulfamethoxazole (15 mg/kg, PO, q12h) and phenylbutazone (2.2 mg/kg, PO, q12h) was given for 30 days. *Streptococcus zooepidemicus*, that was sensitive to penicillin, trimethoprim/sulfa and gentamicin sulfate, was isolated from the involucrum. On the second day after the second surgery, the first PMMB became dislodged from the bed and by 7 days all beads had been dislodged. Because the lameness continued to improve to grade 2/5, the heel elevation was lowered to 2 cm. The mare remained at the clinic at the owner's wishes until discharged 38 days after the second surgery.

Six months after discharge, the owner reported that the mare was slightly lame but was comfortable when at pasture with other mares. The mare was being used as a broodmare and was 45 days pregnant. When the raised shoe was removed at 5 months, there was no worsening in the grade of lameness.

Discussion and Conclusion

In the horse, proximal sesamoidean osteomyelitis is an uncommon condition that can cause severe lameness.[1] The development of this osteomyelitis into a sesamoidean sequestrum has not been reported to the authors' knowledge. Sequestration of the cortex of long bones carries a rapid healing time and good prognosis once the lesion is curetted. In comparison, osteomyelitis of the proximal sesamoid was found to heal slowly and carry a poor prognosis for athletic soundness.[2] This may be in part due to underly-

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ing causes including limited vascular supply and an extension of desmitis or osteomyelitis into the synovial space of the fetlock joint and the synovial sheath of the deep flexor tendon.[1,3,4]

The blood supply to the proximal sesamoid bones are supplied from multiple branches of the medial and lateral palmar/plantar digital arteries.[1,5,6] After entering the sesamoid bones on the non-articular, abaxial surface, the major vessels occupy vascular canals that course through the trabecular architecture of the bones in a general lateral to medial, proximal to distal, and posterior to anterior direction.[5] Little if any blood supply originates from the soft tissue attachments of the suspensory apparatus.[5] Compromise of this vascular supply could increase the susceptibility of the region to sepsis from hematogenous spread of bacteria.[7]

In this case, aggressive surgical and medical therapy was necessary to cause significant improvement in the lameness. Impregnated PMMBs may have aided in antibiotic penetration of the diseased tissue despite having remained in the defect only 4 days. Regional limb perfusion with antibiotics may have been an alternative method of treatment.[8] Subsequent fracture of the proximal sesamoid was a concern and despite improvement of clinical signs and ultrasonography which showed smaller DDFT and sesamoidian ligament lesions, a poor prognosis for an athletic career was given and use as a broodmare was recommended. ■

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Morris Animal Foundation

Diseases of the Nervous System

Diseases of the nervous system in horses are frequently devastating. These conditions are especially perilous because they can present suddenly or as isolated incidents in the early stages, and thus can result in severe injuries to the affected horses and their riders. Furthermore, some equine neurodegenerative diseases do not respond well to treatment and carry a very poor prognosis.

Among the most commonly diagnosed equine neurodegenerative diseases are equine protozoal myelitis, equine degenerative myeloencephalopathy, cervical stenosis or "wobbler" syndrome, and conditions associated with infections by equine herpes virus 1. Another neurodegenerative disease has been added to this list recently. This condition, called equine motor neuron disease (EMND), was originally described in horses in the Northeastern US in the early 1990s, but is now known to occur worldwide.

EMND occurs sporadically, and the available epidemiological data do not support a heritable component. Quarter Horses appear to have a somewhat increased risk over other horse breeds. The clinical presentation of EMND is non-specific. Horses show progressive weakness, muscle wasting, fasciculations and muscle tremors, weight loss, hyperesthesia, and prolonged recumbency. Serum biochemical changes are usually limited to mild increases in the activities of enzymes that are released after muscle damage or deterioration (creatinine kinase and aspartate aminotransferase). Electromyography studies show positive sharp waves and fibrillations suggestive of muscle denervation.

Characteristic histopathological changes of EMND can be appreciated in the motor neurons of the spinal cord and the brain stem, and they include chromatolysis, swelling, prominent neurofilaments, and the accumulation of eosinophilic cytoplasmic inclusions. The appearance of these changes, along with characteristic denervation atrophy in muscle biopsies, are pathognomonic for EMND.

Nobody knows the cause of EMND. Recent studies suggest that a deficiency of dietary anti-oxidants and/or excess of potential metallic neurotoxins may be associated with the disease. This, along with the characteristic pathological changes and anecdotal responses to treatment with Vitamin E, suggests that EMND is caused by oxidative damage to neurons. The resemblance of EMND to amyotrophic lateral sclerosis (ALS or Lou Gehrig's disease) in humans, and the fact that a familial type of ALS is caused by a mutation in the SOD1 gene encoding a copper and zinc superoxide dismutase, also support the contention that EMND is an oxidative disease.

To clarify the possible nutritional and oxidative etiology of EMND, Dr. Tom Divers and his colleagues at Cornell University College of Veterinary Medicine have embarked on a project entitled "High Copper/Iron Diets and the Risk of Motor Neuron Disease in Vitamin-E Deficient Horses." This Morris Animal Foundation-sponsored study is being cosponsored by Ruth Anne and Paul Leibman and Trustee Deborah Carter in memory of her dressage horse Ramon.

The results from this study will improve our understanding of EMND, and will lead to effective strategies for prevention of this dreadful disease.

Rob Hilsenroth, DVM