
Kirsten M. Neil, BVSc, MS, DACVIM; Jane E. Axon, BVSc, DACVIM; Paddy G. Todhunter, BVSc, MS, DACVS; Paul L. Adams, BVSc; John P. Caron, DVM, MVS, DACVS; Angus R. Adkins, BVSc

Objective—To determine the clinical characteristics and outcome of foals with septic osteitis of the distal phalanx.

Design—Retrospective case series.

Animals—22 foals.

Procedures—Information obtained from medical records included signalment; clinical, laboratory, and radiographic findings; treatment method; and outcome. Foals included in the study had lameness referable to the foot, radiographic evidence of localized lysis or focal loss of bone density of the distal phalanx, and suppurative discharge or necrosis of the affected bone evident at surgery. Foals with a history or evidence of penetrating wounds or subsolar abscessation were excluded.

Results—Mean age of foals at initial evaluation was 40.8 days (range, 3 to 122 days). Twenty-one (95%) foals had lameness as the primary complaint. Lesions consistent with septic osteitis of the distal phalanx localized to specific areas of the bone on the basis of radiographic and surgical findings were located on the solar margin or toe (14/22 [64%]), extensor process (5/22 [23%]), and palmar or plantar process (3/22 [13%]). Hind limbs (18/26 [69%]) affected limbs were more frequently affected. Two foals had >1 affected limb, 2 had additional sites of osteomyelitis, and 4 had concurrent septic arthritis. Surgical debridement and regional antimicrobial perfusion were performed during general anesthesia. Extensor process lesions were not debrided. Nineteen of 22 (86%) foals survived to be discharged from hospital, and 16 horses reached racing age. Eleven of 16 had race starts, of which 8 had official race starts and 3 had unofficial race starts.

Conclusions and Clinical Relevance—Septic osteitis of the distal phalanx should be considered as a source of lameness in foals with signs referable to the foot and does not necessarily preclude a career in racing. Although infection may occur secondary to bacterial penetration of the hoof or sole, the distal phalanx should also be considered as a potential site for hematogenous septic arthritis or osteomyelitis in foals. (J Am Vet Med Assoc 2007;230:1683–1690)

Pedal osteitis is an inflammatory condition that results in demineralization of the distal phalanx.1 Two classifications of pedal osteitis are recognized: septic and nonseptic.2 Purulent discharge originating from the distal phalanx, with associated radiographic signs of localized osteolysis, distinguishes septic osteitis from inflammation associated with noninfectious causes.3 Inflammation and infection is referred to as osteitis rather than osteomyelitis because the distal phalanx does not contain a medullary cavity.4

Septic pedal osteitis in adult horses has been associated with conditions such as hoof wall avulsion, fractures, abscesses, and laminitis; however, penetrating wounds and subsolar abscesses remain the leading causes.3,5,6 Although often reported in adult horses, this condition has not been well characterized in foals. In foals, the most common cause of osteomyelitis is hematogenous dissemination of bacteria,7,8 even in the absence of signs of systemic disease.9 Acute inflammation occurs after bacterial colonization of bone, with subsequent ischemia, necrosis, and possible sequestrum formation,10 and osseous lesions are often accompanied by septic arthritis.9,11,12 These lesions can develop rapidly, in some instances in as little as 2 to 3 days.9

The objective of the study reported here was to determine the clinical and radiographic findings, treatment, short-term survival rate, and future athletic performance in a group of foals with septic osteitis of the distal phalanx of suspected hematogenous origin.

Criteria for Selection of Cases

The medical records of foals <6 months of age that were treated for septic osteitis of the distal phalanx at

Abbreviation

PMMA Polymethylmethacrylate

From the Scone Veterinary Hospital, 106 Liverpool St, Scone, NSW 2337, Australia (Neil, Axon, Todhunter, Adams, Adkins); and the Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824-1314 (Caron). Dr. Neil’s present address is Goulburn Valley Equine Hospital, 905 Goulburn Valley Hwy, Congupna, VIC 3632, Australia. Dr. Todhunter’s present address is Newcastle Equine Hospital, 905 Goulburn Valley Hwy, Congupna, VIC 3632, Australia. Dr. Neil’s present address is Goulburn Valley Veterinary Medicine, Michigan State University, East Lansing, MI 48824-1314 (Caron). Dr. Neil’s present address is Goulburn Valley Equine Hospital, 905 Goulburn Valley Hwy, Congupna, VIC 3632, Australia. Dr. Todhunter’s present address is Newcastle Equine Hospital, 905 Goulburn Valley Hwy, Congupna, VIC 3632, Australia.

Presented at the 13th Annual American College of Veterinary Surgeons Symposium, Washington, DC, October 2003.

The authors thank Dr. Lori Bidwell for artistic assistance.

Address correspondence to Dr. Neil.
Scone Veterinary Hospital, New South Wales, Australia, between December 1995 and December 2002 were reviewed. Septic ostitis of the distal phalanx was defined as radiographic evidence of septic ostitis that was confirmed by the presence of suppurative discharge or necrosis of the affected bone at surgery. Foals included in the study had lameness referable to the foot and one or more of the following supportive clinical and laboratory findings: signs of pain on application of finger pressure or hoof testers to the sole, improvement or resolution of lameness with perineural anesthesia, high WBC or absolute neutrophil count, high fibrinogen concentration, or positive results of microbial culture of bone. Foals with a history or evidence of pedal ostitis secondary to penetrating wounds or evidence of subsolar bruising or abscesses were excluded.

Procedures

Medical records were reviewed to obtain signalment, clinical findings, laboratory test results, radiographic findings, treatment method, and outcome. In all cases, radiographic views of the distal phalanx were obtained and included dorsoproximal–palmarodistal oblique views (65ø), lateromedial views, or both. In addition, dorsopalmar (weight-bearing), dorsolateral–palmaromedial oblique (15ø to 60ø), and dorsomedial–palmarolateral oblique (15ø to 60ø) views were obtained in some foals. Radiographic lesions considered supportive of septic ostitis of the distal phalanx included localized areas of osteolysis and focal or diffuse loss of bone density with irregular mottling or irregularity of margins of the lytic area, the presence of 1 or more sequestra, or both. Diagnosis was confirmed at surgery by the presence of necrotic bone and purulent discharge originating from the distal phalanx. Lesions were classified on the basis of the area of the distal phalanx that was affected: solar margin lesions had lysis of the dorsal, solar, or both surfaces of the solar margin that may have been confined to the toe or were extensive and involved the body of the distal phalanx with extension along the solar surface of the solar margin, palmar or plantar process lesions had focal areas of osteolysis confined to the lateral or medial palmar or plantar process and involved the proximal or distal angle of the process, and extensor process lesions had local areas of osteolysis confined to the extensor process. Radiographic findings of bony fragments palmar to the palmar or plantar process or proximal to the extensor process alone and irregularity of the solar margin with widening of vascular channels without evidence of purulent discharge and bone necrosis at surgery were not considered consistent with septic ostitis.

Other information including treatment modalities, culture results and antimicrobial susceptibility data, limb affected, and duration of hospitalization was also obtained from the records. The presence of localized infections, osteomyelitis affecting other bones, and septic arthritis was recorded. Laboratory results recorded included plasma fibrinogen concentration, WBC count, absolute neutrophil count, absolute band neutrophil count, and serum IgG concentration. Not all variables were recorded in medical records of all foals.

Bacteriologic culture results were available for 10 foals. Synovial fluid, blood, or bone underwent bacteriologic culture in enrichment broth or blood culture bottles containing sodium polyanetholesulfonate for subculturing after incubation. Synovial fluid and macerated bone were inoculated directly onto horse blood agar. Samples were also submitted for anaerobic culture. Antimicrobial susceptibility of bacteria was performed by use of the agar disk diffusion method.

Short-term survival was determined as successful discharge of the foal from the hospital. Records obtained from the Australian Stud Book were used to determine whether the foals survived to be parentage verified and whether foals survived to be registered with the Australian Jockey Club and, hence, named. Race records were obtained for Thoroughbred foals that had reached racing age (> 2 years old) at the time of the study. The number of starts, wins, and placings were recorded. For horses racing in Australia, race starts are classified as official and unofficial, the latter defined as race starts with no prize money awarded. Because foals were intended for racing, long-term outcome was classified as successful for horses that raced and a failure for those that did not race. The duration of follow-up ranged from 3 to 10 years.

Results

Signalment—Twenty-two Thoroughbred foals met the inclusion criteria for the study. Mean age of foals at admission was 40.8 days (range, 3 to 122 days). Fourteen foals were male, 7 were female, and sex was not recorded for 1 foal.

Initial complaint—Twenty-one (95.5%) foals had lameness as the primary complaint. Seventeen (80.9%) of those foals had grade 4/5 or 5/5 lameness. Four foals had grade 3/5 lameness, 3 of which had been treated with systemic antimicrobials and nonsteroidal anti-inflammatory drugs prior to referral. Duration of lameness prior to admission was < 1 to 2 days for 18 (85.7%) foals. Three foals had been treated for a prolonged period prior to evaluation (10 to 23 days). The primary complaint for 1 foal was septicemia and failure of passive transfer (IgG concentration, < 800 mg/dL). This foal developed radiographic evidence of septic ostitis and associated clinical signs (grade 3/5 lameness) within 2 days of hospitalization. Fourteen of 22 (63.6%) foals were febrile (rectal temperature, > 38.8°C [101.8°F]) at the time of evaluation.

Localization of lameness was confirmed by evidence of a painful reaction to application of finger pressure or hoof testers to the sole in 16 foals (16/17 [94.1%]). The reaction for 1 foal was not consistent. For this foal, perineural anesthesia of the palmar digital nerve at the level of the proximal sesamoid bones resulted in resolution of lameness. Five foals had lameness localized on the basis of effusion of the distal interphalangeal joint. Perineural anesthesia of the palmar digital nerve at the level of the proximal sesamoid bones resulted in resolution of lameness and associated clinical signs (grade 3/5 lameness) within 2 days of hospitalization. Fourteen of 22 (63.6%) foals had lameness as the primary complaint. Seventeen (80.9%) of those foals had grade 4/5 or 5/5 lameness. Four foals had grade 3/5 lameness, 3 of which had been treated with systemic antimicrobials and nonsteroidal anti-inflammatory drugs prior to referral. Duration of lameness prior to admission was < 1 to 2 days for 18 (85.7%) foals. Three foals had been treated for a prolonged period prior to evaluation (10 to 23 days). The primary complaint for 1 foal was septicemia and failure of passive transfer (IgG concentration, < 800 mg/dL). This foal developed radiographic evidence of septic ostitis and associated clinical signs (grade 3/5 lameness) within 2 days of hospitalization. Fourteen of 22 (63.6%) foals were febrile (rectal temperature, > 38.8°C [101.8°F]) at the time of evaluation.

Localization of lameness was confirmed by evidence of a painful reaction to application of finger pressure or hoof testers to the sole in 16 foals (16/17 [94.1%]). The reaction for 1 foal was not consistent. For this foal, perineural anesthesia of the palmar digital nerve at the level of the proximal sesamoid bones resulted in resolution of lameness. Five foals had lameness localized on the basis of effusion of the distal interphalangeal joint. Hind limbs (18/26 [69.2%] affected limbs) were more frequently affected than forelimbs (8/26 [30.8%]). One foal had 2 affected limbs, and another had 4 affected limbs.

Radiographic lesions—Fourteen of 22 (63.6%) foals had lesions involving the solar margin: 1 foal had osteoly-
sis and focal radiolucency of the solar surface of the solar margin palmar to the dorsal solar margin (toe), and 13 foals had lesions involving the dorsal solar margin (toe; Figures 1 and 2). Of these 13 foals with involvement of the toe, 7 had lesions confined to this area and 6 had extensive lysis and loss of bone density that extended into the body of the distal phalanx and along the dorsal and, in particular, solar surface of the solar margin. Five of 22 (22.7%) foals had lysis and focal loss of bone density of the extensor process. Three (13.6%) foals had focal lysis of the palmar or plantar process, 2 of which involved the distal angle of the solar surface of the process and 1 of which involved the proximal angle of the process (Figure 3). Both foals with > 1 affected limb had lesions involving the toe or toe and solar margin. A sequestrum was identified radiographically in 4 (18.2%) foals with lesions of the solar margin. No foals in the study had fractures of the distal phalanx or radiographic changes consistent with laminitis on the initial radiographic views.

Other orthopedic conditions—Two (9.1%) foals had additional sites of osteomyelitis. Limbs affected with osteomyelitis differed from those affected with septic pedal osteitis. One foal had proximal sesamoid bone osteomyelitis and nonseptic effusion of 3 metacarpophalangeal or metatarsophalangeal joints. Another foal had bilateral tuber calcaneal osteomyelitis; results of cytologic analysis of calcaneal bursa fluid were within reference limits.

Four (18.2%) foals had concurrent septic arthritis as judged on the basis of high WBC, neutrophil, or total protein concentrations in synovial fluid samples (WBC count, 100 to 217 × 10⁹ cells/L, with > 90% neutrophils [reference range, < 0.5 × 10⁹ cells/L, with < 90% neutrophils]; total protein concentration, 46 to 64 g/L [reference range, < 25 g/L]). Distal interphalangeal (3/4) and metatarsophalangeal (1/4) joints were affected. Foals with septic arthritis of the distal interphalangeal joint had concurrent distal phalangeal extensor process lesions.

Other orthopedic conditions included proximal sesamoid bone fracture, secondary flexor tendon laxity and associated fetlock laxity of the contralateral limb, and flexural deformity of the affected limbs, the latter condition affecting both foals with multiple distal phalanx lesions.

Laboratory results—Abnormal CBC or fibrinogen concentration results were obtained in all 10 foals for which results were available. Abnormal WBC results were obtained in 4 of 10 foals; 3 foals had neutrophilic leukocytosis, ranging from 18.0 to 22.7 × 10⁹ cells/L for WBC count (reference range, 5.2 to 12 × 10⁹ cells/L) and 14.0 ± 15.2 to 10⁹ cells/L for neutrophil count (reference range, 3.2 to 10.6 × 10⁹ cells/L); 1 foal had leukopenia (4.1 × 10⁹ cells/L) because of neutropenia (2.2 ± 10⁹ cells/L). A left shift was evident in 2 foals with WBC count within reference range, ranging from 0.44 to 0.59 × 10⁹ band neutrophils/L (reference range, 0 ± 0.40 × 10⁹ cells/L). Abnormal plasma fibrinogen concentration was identified in 7 of 10 foals, ranging from 5.0 to 9.0 g/L (reference range, 2.0 ± 4.0 g/L).
Bacteriologic culture results—Culture results were available for 10 foals, with positive results obtained in 9 foals. Results of bacteriologic culture of bone were available for 6 foals, of which 5 had positive results. Isolates included Streptococcus spp (n = 3 foals), Escherichia coli (2), Staphylococcus aureus (2), Proteus spp (2), Actinomyces spp (1), and Pseudomonas spp (1). Mixed bacterial isolates were obtained in 4 of 5 samples with positive results. The sample from which a single gram-positive organism was isolated cultured later yielded a mixed population of bacteria. Synovial fluid culture results were available for 4 foals, of which all had positive results with single etiologic agents isolated: Salmonella spp (n = 1 foal), E coli (1), Staphylococcus spp (1), and Actinobacillus spp (1). One foal had the same organism isolated from synovial fluid and blood (Salmonella spp). Overall, the most common organisms isolated were E coli (n = 3 foals), Staphylococcus spp (3), and Streptococcus spp (3).

Surgical treatment—Surgery was performed on all foals by use of general anesthesia. Palmar or plantar perineural analgesia was performed at the level of the proximal sesamoid bones by administration of mepivacaine hydrochloride. The hoof was prepared aseptically for surgery by use of povidone-iodine solution. A tourniquet was placed in the mid–metacarpal–metatar-
concentrations where appropriate, normalization of rectal temperature, or lack of evidence of further lysis on radiographs.

Other antimicrobials used included trimethoprim sulfadimidine (30 mg/kg [13.6 mg/lb], PO, q 12 h), rifampin (5 mg/kg, PO, q 12 h), azithromycin dihydrate (10 mg/kg, PO, q 24 h), enrofloxacin (2.5 mg/kg [1.14 mg/lb], PO, q 12 h), and ampicillin sodium (20 mg/kg [9.1 mg/lb], IV, q 8 h). Foals with lesions of the toe, solar margin, or palmar process of the distal phalanx were treated with topically administered metronidazole. One foal was treated with only topically administered metronidazole and did not receive systemic antimicrobial administration.

Twelve of 19 (63.2%) foals were treated with further systemically administered antimicrobials after discharge from the hospital for an additional 7 to 21 days. Mean duration of treatment with systemically administered antimicrobials (including after discharge) was 19.0 days (range, 4 to 42 days).

Additional treatments—Other supportive treatments were administered on an individual basis. All foals were administered flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IV, once) prior to or during surgery; however, continued use of nonsteroidal anti-inflammatory drugs was not routine. Four foals received phenytoin (2.2 mg/kg [1 mg/lb], PO, q 12 h) for 1 to 5 days after surgery. Postoperative care consisted of bandage changes and wound flushing with saline solution or 2% povidone-iodine. This was performed daily initially, then every 2 to 3 days after granulation tissue was evident. Foals for which fenestration of the sole was performed had the fenestrated sole removed 2 to 4 weeks after surgery when new sole had grown. Additional treatments included stall confinement; IV administration of fluids, electrolytes, or plasma; and oral administration of cimetidine hydrochloride (18 mg/kg [8.2 mg/lb], q 8 h) or ranitidine hydrochloride (6.6 mg/kg, q 8 h). Radiography was repeated after surgery in 12 foals on the basis of assessment of lack of response to treatment, including lack of improvement or resolution of lameness, continued suppurative discharge from the site of debridement, high fibrinogen or WBC concentrations, or continued pyrexia.

Outcome—Overall, 86.4% (19/22) of foals survived to be discharged from hospital. Of the 3 foals that did not survive to be discharged, all were euthanatized because of progression of osteitis and lack of response to treatment. Two of those foals had minimal laminar attachment of the distal phalanx to the hoof wall with subsequent instability of the distal phalanx within the hoof. The other foal developed severe digital flexor tendon laxity of the contralateral unaffected limb. Two of the 3 foals that did not survive to be discharged had been treated for prolonged periods prior to referral and subsequent surgical debridement.

At the time of discharge, no lameness was evident in 12 foals. Seven foals were mildly lame (grade 1/5) at the time of discharge, and all were less lame than at admission. Survival to discharge from hospital was favorable for all lesion types (Figure 4).

In the 17 foals in which surgical debridement of bone lesions was performed, follow-up radiographs obtained 1 to 4 months after discharge from the hospital revealed smooth margins at the bony defect. Although large defects were often still evident, there was no associated lameness. All 5 foals for which repeat surgical debridement was required survived to be discharged from the hospital.

After discharge, 3 foals died or were euthanatized before they were 1 year of age and were not registered. Only 1 of these foals was euthanatized for reasons related to septic pedal osteitis. That foal had quadrilateral pedal bone involvement and was euthanatized 1 month after discharge because of hoof contracture. One foal was sold as a weanling and later died from unrelated causes. One foal was euthanatized 8 months after discharge because of extensive osteochondritis dissecans and degenerative joint disease of both metacarpophalangeal joints, which was not believed to be directly related to the septic pedal osteitis.

Of the long-term survivors, 11 of 16 horses had raced at the time of the study. Eight of these horses had official race starts, with a range of 2 to 37 starts (mean, 13.4 starts). All horses had won at least 1 race (mean, 2.9 wins; range, 1 to 9 wins). An additional 3 horses had unofficial race starts. Prognosis for racing was poorer for foals with solar margin lesions than those with extensor process and palmar-plantar process lesions (Figure 4). Of the 5 foals that required repeat surgical debridement, 4 survived to race age, of which 2 foals subsequently raced. For foals with concurrent septic arthritis, 3 of 4 survived to race age, of which 2 foals subsequently raced.

Figure 4—Illustration of the location of lesions (A = extensor process, B = solar margin [toe], and C = palmar or plantar process) in 22 foals with septic pedal osteitis. Among foals with lesion A, 4 of 5 were discharged from hospital and 3 of 4 had race starts (2 official, 1 unofficial). Among foals with lesion B, 12 of 14 were discharged from hospital and 6 of 11 had race starts (5 official, 1 unofficial). Among foals with lesion C, 3 of 3 were discharged from hospital and 2 had race starts (1 official, 1 unofficial).
Discussion

Unlike adult septic pedal osteitis in which a primary cause is often identified,3,5,6 there was no evidence of subSolar abscesses, subSolar bruising, or sinus tracts consistent with foreign body penetration in the foals in this series. Although septic pedal osteitis may have developed in some of these foals as a result of undetected puncture wounds, none were identified in any foal at surgery, suggesting that many of these foals acquired the infection hematogenously. Further evidence to support this contention was found in the observation of concurrent osteomyelitis, septic arthritis, and multiple distal phalangeal lesions in a number of affected foals. Septicemia and failure of passive transfer, common predisposing factors for septic arthritis and osteomyelitis in foals,14-16 were also evident, with bacteremia detected in 1 foal.

In retrospective studies8,17 of septic arthritis-osteomyelitis in foals, the femur, tibia, and third metacarpal-metatarsal bones are most frequently affected. Involvement of the distal interphalangeal joint appears rare, with only single foals affected in 3 retrospective studies11,12,18 of 66, 69, and 93 foals with septic arthritis. Distal phalangeal lesions have been alluded to previously in association with septic arthritis of the distal interphalangeal joint in 2 foals; however, it is unknown whether the lesions were hematogenous in origin.9 Accurate classification of lesions of the distal phalanx into presently recognized categories of septic arthritis-osteomyelitis3,5,6 requires further investigation. Lesions described in this study may require a new lesion categorization.

Predisposing factors in adults for the development of septic and nonseptic pedal osteitis have been described1,3,5,6 with both conformational and biomechanical factors implicated. Laminitis has been documented as a primary condition predisposing to distal phalangeal infection in adults.1 Although laminar changes were evident in 2 foals in this study, these changes were not evident on the initial radiographic examination and appeared to be a consequence of the primary bone infection, with destabilization following laminar damage and loss of lamellar attachments of the distal phalanx to the hoof capsule. A similar phenomenon has been reported to be associated with septic pedal osteitis in a stallion.19 Of interest, osteolysis of the distal portion of the distal phalanx has been reported secondary to flexural deformity in foals,20 a condition that affected both of the foals with multiple distal phalangeal lesions in the present study. However, foals in the aforementioned study20 differed in that there were no signs of bone infection and resolution of radiographic changes occurred following treatment of flexural deformity.

The pathogenesis of septic osteomyelitis in foals has been postulated to relate to the vascular arrangement of metaphyseal and physeal regions of bone, with sluggish blood flow and low oxygen tensions favoring lodgment of bacteria.8,10,21 Osteitis affecting other bones is common in the distal portions of the limbs of horses secondary to trauma or extension of local infection.22 The vascular network of the distal phalanx is extensive,3,23 with arterial branches from the palmar digital artery, terminal arterial arch, and circumflex artery supplying the parietal surface and the solar border. Large vascular channels radiate from the solar canal; however, similar branches do not penetrate the palmar processes.24 Whether the anatomic arrangement of the vasculature of the distal phalanx predisposes this bone to lodging or sedimentation of bacteria after hematogenous dissemination requires further investigation. Conversely, it has been suggested that the distal phalangeal vasculature may provide an effective defense mechanism against sepsis because despite the long duration and extensiveness of septic pedal osteitis in adults, lesions often remain superficial.3 This is in contrast to the nature of infections in foals in the present study and, perhaps, relates to the differing etiology of the infection in this age group.

Principles of treatment of septic pedal osteitis in foals follow those of treatment of osteomyelitis affecting other bones. Surgical debridement of lesions is routinely performed in adults with septic pedal osteitis.3,5,6 Ischemia is an important aspect of osteomyelitis because organisms thrive in a vascular bone such that antimicrobial treatment alone may be unsuccessful. Typical recommendations are for surgical debridement of affected bone, provided mechanical stability of the affected joint is not jeopardized; however, some authors do not advocate debriding lesions if the affected bone is close to the joint.3,9,10 In the present study, all lesions were surgically debrided except for extensor process lesions. Foals with extensor process lesions did not have debridement of those lesions because of individual surgeon’s preferences, in part because of concerns with creating instability of the distal phalanx within the hoof capsule secondary to debridement of extensive lesions. Furthermore, affected foals in this series had good clinical response to treatment without debridement of these lesions. These foals underwent distal interphalangeal joint lavage because of the likelihood of concurrent septic arthritis. Regional IV perfusion and antimicrobial-impregnated PMMA beads were used in an attempt to provide high local concentrations of antimicrobials, as has been reported in other studies.25-27 The use of topically administered metronidazole in the present study was in accordance with recommendations for treatment of septic pedal osteitis in adults and associated with the potential difficulty in isolating anaerobic organisms.5 Fenestration of the sole was performed to retain the structural support of the hoof capsule and underlying sole while allowing for adequate drainage of the infected material. This was thought to have decreased the occurrence of hoof contracture, with this complication reported in only 1 foal in this study.

In adults, foot infections, including those associated with septic pedal osteitis, are usually caused by mixed bacterial populations after introduction of environmental microbes.3,5,28 In the present study, mixed bacterial populations were evident from bacteriologic culture of bone in 4 of 5 cases for which results were available. This appeared to be more consistent with infection secondary to bacterial penetration of the hoof or sole; however, none of the foals had evidence of penetrating wounds or changes in the solar corium evident at surgery. Furthermore, 1 of the foals from which mixed organisms were isolated had septic pedal osteitis affecting all 4 limbs, a finding that is...
less consistent with penetrating wounds. These culture results may be attributed to the requirement for a solar approach at surgery because areas of the sole directly over the affected bone are opened prior to samples being taken for culture and are therefore exposed to potential environmental contamination. Substantial bacterial populations are known to persist despite presurgical disinfection of the hoof.20 Conversely, the involvement of multiple organisms may also reflect the pathogenesis of the disease process in these foals. Mixed infections of hematogenous origin may have occurred in some of these foals, as in previous reports12,30,31 of mixed-organism septicemia and septic arthritis in foals. The most common bacterial isolates from foals with septic arthritis-osteomyelitis are E coli, Actinobacillus spp., and Streptococcus spp.8,9 In 1 study11 of 66 foals, Enterobacter spp were the most common isolates, with E coli cultured from 27% of joints. In another study,11 Salmonella spp were the most common bacteria isolated. Similar organisms were isolated from the foals in the study reported here. Although results were not available for all foals, the high proportion of positive culture results in this study highlights the importance of obtaining samples from not only affected bone but also the distal interphalangeal joint when extensor process lesions are suspected.

In this study, 11 of 16 of the surviving foals that reached racing age raced, and of these, 8 had official race starts and 3 had unofficial race starts. These results are comparable to the Australian average for which approximately 60% of the total population of Thoroughbred foals born in Australia start in a race.8 Although the number of official starters was less than the Australian average, the success of these starters further suggests that prognosis for racing is not necessarily unfavorable. Furthermore, at the time of the study, 3 of the nonstarters were < 4 years old, so the actual proportion of foals with septic pedal osteitis that subsequently race may be underestimated.

Two of the 3 foals that did not survive to be discharged from hospital in this study had been treated for prolonged periods prior to referral. The rapid progression of septic pedal osteitis and sequelae, such as hoof contracture and laminar detachment, highlights the importance of early diagnosis and implementation of appropriate treatment. Angular limb and flexural deformities of the affected and contralateral limbs may also occur.

The absence of evidence of penetrating wounds or subsole abscission in these foals suggested infection of hematogenous origin. As such, septic pedal osteitis should be considered as a potential component of septic arthritis-osteomyelitis in foals with lameness referable to the foot. Aggressive early treatment of septic pedal bone osteitis is essential to avoid life-threatening complications. Septic pedal osteitis in foals does not necessarily preclude a career in racing.

References

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