History

A 9-year-old Thoroughbred mare was referred for evaluation because of a 1-month history of severe non-weight-bearing left hind limb lameness and pyrexia with rectal temperatures up to 41°C (105.8°F). The metatarsal area of the left hind limb had been swollen for 1 month, and a wound became evident just below the torus carpeus (chestnut) after the first 2 weeks of clinical signs. Findings on radiographs of the tarsus obtained 1 week after the onset of lameness were inconclusive. The referring veterinarian suspected lymphangitis and therefore administered penicillin followed by ceftiofur as well as phenylbutazone and flunixin meglumine without clinical improvement. Two days before referral, the horse was able to bear weight on its left hind limb.

At the time of referral, the horse appeared to walk comfortably but was tachycardic (heart rate, 64 beats/min) and pyrexic (rectal temperature, 38.9°C [102°F]). The left hind limb had firm, warm edema from the area of the metatarsophalangeal joint to the tarsus, with fluctuating areas around a large (6 × 5-cm) wound just below the chestnut. Pus was exteriorized by manual pressure and sent for bacteriologic culture. Serum biochemical analysis and CBC revealed that the horse had moderate leukocytosis (13.11 × 10^9 cells/L; reference range, 6 × 10^9 cells/L to 12 × 10^9 cells/L), hyperproteinemia (94 g/L; reference range, 55 to 75 g/L), and hyperfibrinogenemia (8 g/L; reference range, 2 to 4 g/L). Radiographs of the left tarsus and metatarsal region were obtained (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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Figure 1—Lateromedial (A), dorsoplanter (B), dorsolateral-plantaromedial oblique (C), and dorsomedial-plantarolateral oblique (D) radiographic views of the tarsal and metatarsal regions of a 9-year-old Thoroughbred mare evaluated because of a 1-month history of severe left hind limb lameness and pyrexia.
The fragment of the wound was performed, and the horse was still fully weight bearing at rest. The cannulated screw was removed, the hole was curetted, and the removed bone was sent for histologic examination. The horse was discharged from the hospital. Unfortunately, the horse’s comfort level deteriorated, pyrexia became uncontrollable, and the owner had the horse euthanized at home, making further examination impossible. Histologic evaluation of bone submitted prior to hospital discharge revealed necrotizing osteomyelitis with intralesional bacteria.

Findings in the horse of this report underscore the low diagnostic sensitivity of radiography to detect bony changes early in the disease process of osteomyelitis, as the radiographs made 1 week after the onset of lameness were inconclusive. Because there was a lack of known bone trauma and because of the absence of full-thickness cortical discontinuity, it is hypothesized that osteomyelitis in this horse was hematogenous in origin. The observed wound may have represented a primary source of infection, a secondary draining tract, or because of its location proximal to the sequestrum, a second site of hematogenous spread of infection. The diffuse periosteal new bone formation that surrounded the diaphysis made up the involucrum, which forms around a sequestrum. The involucrum elevates the periosteum, which subsequently deprives the underlying cortex of its external blood supply. The massive diaphyseal sequestrum observed in this horse is likely the result of compromised medullary blood supply.