Review of Patterns of Humeral Fracture in Thoroughbred Racehorses

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Humeral fractures in Thoroughbred racehorses have a typical configuration and are preceded by stress fracture. Young racehorses that have a low frequency of races and timed works or that recently returned from an injury are at high risk for humeral fracture. Diagnosis of humeral stress fracture requires nuclear scintigraphy, because early callus is not evident radiographically when risk for complete bone fracture is highest. Authors’ address: J.D. Wheat Veterinary Orthopedic Research Laboratory, Department of Anatomy, Physiology, and Cell Biology, School of Veterinary Medicine, University of California at Davis, Davis, California 95616; e-mail: sksammons@ucdavis.edu. © 2009 AAEP.

1. Introduction
Musculoskeletal injury is the most common cause of death in racehorses. In a study of California racehorses that died between 1990 and 1992, 83% of horses died because of musculoskeletal injuries. Concern about horse welfare and wastage, jockey injuries, and public perception of Thoroughbred racing has influenced researchers to study the potential causes of catastrophic musculoskeletal failure in Thoroughbred racehorses. Increased knowledge may improve understanding of the pathogenesis of injuries and enhance opportunities for injury prevention. The humerus is the third most commonly fractured bone in Thoroughbred racehorses that die during training sessions. Previous studies have attributed racehorse catastrophic fracture occurrence to pre-existing pathology, particularly in the humerus where 77% of complete fractures had evidence of pre-existing stress fractures at necropsy. The stress and complete fracture of the humerus are the subject of this review.

2. Horse Characteristics
Thoroughbred racehorses that die because of complete humeral fracture are predominantly 2- and 3-yr-old horses, and 22% of horses with complete humeral fracture in the current study had no documented races or timed documented works. Only 12% of horses in the study suffered complete fracture of the humerus during a race or timed documented work. Affected horses have lower race and work frequencies. Humeral fractures tend to occur shortly after return from lay-up.

3. Clinical Features
Horses with humeral stress fractures generally produce a grade 2–3 of 5 lameness in the affected limb, and many have a history of more severe lameness (grade 4) up to 24 h post-exercise. Flexion, adduction, or abduction of the shoulder or elbow joints may exacerbate the lameness. Although some humeral stress fractures are discovered because of acute onset of lameness during routine training, some go unnoticed and unexpectedly result in complete fracture.
There is no apparent limb predilection. Although bilateral humeral stress fractures are uncommonly observed in live racehorses, stress fracture callus is usually present bilaterally in cadaveric humeri from horses that had a unilateral complete humeral fracture.

Diagnosis of stress fractures in the proximal aspect of the limb is difficult. Palpation and local anesthesia are generally unrewarding, and radiography is limited in that region of the limb because of superimposition of large muscle mass and thoracic structures (Fig. 1). Scintigraphy is most sensitive for detecting increased bone metabolic activity associated with stress fracture (Fig. 2).

4. Fracture Characteristics

The humerus fractures in a characteristic pattern with a typical underlying pathology. The complete fracture characteristically courses from the caudoproximal cortex in a soft spiral configuration to the caudodistal cortex (Fig. 3). A free bone fragment may be present at the proximal aspect of the distal bone fragment.

For completely fractured humeri, the most common location of periosteal callus is the caudoproximal site, but callus was also observed in medial and craniodistomedial sites (Fig. 4). For live Thoroughbred racehorses with humeral stress fracture callus, 37% were caudoproximal and 17% were craniodistal. Interestingly, callus was not observed in the medial site in live racehorses but was observed in two additional sites: caudodistal and cranioproximal in 42% and 4%, respectively.

5. Fracture Development

Bone Changes

A progression of stages of callus development is apparent in necropsy specimens of completely fractured humeri (Fig. 5). Early callus is <1 mm thick and pink with a rough surface texture. The callus becomes thicker and red in color, and it has a longitudinal pattern to the rough surface texture. In some cases, callus is exuberant and has an irregular contour. With consolidation and remodeling during disease resolution, the callus becomes smooth surfaced and dense, and this results in a blunted contour to the caudal aspect of the humeral neck.
Corresponding changes occur in the bone tissues observed on transverse section using microcomputer tomography (Fig. 6). With disease progression, endosteal trabeculae thicken, new bone forms on the periosteal surface, and the parent cortex becomes more porous. Most completely fractured humeri have a thin, rough-surfaced periosteal callus, some endosteal sclerosis, and cortical porosities. We speculate that humeri with this type of callus are most susceptible to complete fracture, because endosteal and periosteal callus are insufficient to compensate for cortical porosity.

Pathogenesis

In racehorses, complete fractures have been associated with pre-existing incomplete fractures. A bone is susceptible to complete fracture if an incomplete fracture experiences continued loading before sufficient healing. Incomplete fractures, also called fatigue fractures or stress fractures, are not associated with a single event or trauma but instead are associated with repetitive loading activities. Fatigue fractures generally occur within the first year of training in Thoroughbred racehorses (2 yr olds). During a normal physical exercise program, bones sustain damage that are repaired by the bone remodeling process. When exercise intensity is high, the resorptive phase of the remodeling process may exceed the replacement phase and result in transient osteoporosis and a weaker bone. In fact, damaged bone is reabsorbed by osteoclasts within 48–72 h after onset of damage and continues for 2–3 weeks. Unfortunately, the reparative phase is much slower, requiring a 3-mo bone formation phase, and this results in a temporarily weakened bone. Therefore, fatigue fractures may result when the rate of damage accumulation exceeds the rate of replacement when the same level of stress is applied during both periods.

Return from inactivity can also promote stress fracture and related complete fracture. In contrast to fatigue fractures caused by high rates of resorption and low rates of replacement, stress fractures can also occur as a result of insufficient activity. Bones that experience periods of inactivity are prone to reductions in bone mass, which is termed disuse osteoporosis. Therefore, racehorses on lay-up or...
reduced activity for a variety of reasons, including lameness or poor performance, may develop some degree of disuse osteoporosis. When a horse returns to full work after this period of prolonged lack of exercise, the bones may be weakened from a degree of disuse-related osteoporosis and less able to withstand a sudden significant increase in activity. Consequently, these horses may be more susceptible to microdamage accumulation, stress fracture, and complete fracture. In fact, previous evidence suggests that many horses sustain catastrophic humeral fractures soon after return from extended lay-up.

6. Predisposing Factors
Thoroughbred racehorses seem predisposed to complete humeral fracture soon after return to training after an injury. In addition to the presence of underlying pathology, racehorses suffering humeral fracture have fewer career days and therefore, fewer races and works, which may correlate with their younger age compared with horses that may fracture other bones. Horses that suffered complete humeral fracture also tend to have fewer career lay-ups but fracture soon after resuming activity. This history is consistent with the concept that lay-up induces some degree of disuse osteoporosis in humeri. With return to work, these humeri likely have increased susceptibility to microdamage accumulation, stress fracture, and complete fracture.

7. Summary
Young Thoroughbred racehorses are susceptible to humeral stress fracture. Humeral stress fractures predispose the horse to complete humeral fracture. Humeral stress fractures occur at characteristic locations. Stress fractures at only caudoproximal, craniodistomedial, and medial sites have been associated with complete humeral fractures. It is speculated that stress fractures at craniodistal and caudodistal sites are less likely to promote complete fracture; however, this could be related to a higher likelihood of radiographic detection in early stages of callus formation. Thoroughbred racehorses are most likely to incur a complete humeral fracture soon after return from lay-up. Further work to correlate histopathologic changes with exercise may...
allow design of training regimens for the prevention of humeral fractures.

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References