Diagnostic Workup of Upper-Limb Stress Fractures and Proximal Sesamoid Bone Stress Remodeling

Susan M. Stover, DVM, PhD, Diplomate ACVS

Stress fractures and stress remodeling occur in specific locations within the scapula, humerus, and proximal sesamoid bones of racehorses. Knowledge of locations and of racehorse characteristics that place racehorses at high risk for scapular and humeral stress fractures and remodeling injuries enhances veterinarians' ability to detect scapular and humeral stress lesions. Detection of stress remodeling in proximal sesamoid bones is more challenging because of the small size and location of the lesions. Author's address: JD Wheat Veterinary Orthopedic Research Laboratory, Department of VM:APC, School of Veterinary Medicine, University of California at Davis, One Shields Avenue, Davis, CA 95616; e-mail: smstover@ucdavis.edu. © 2013 AAEP.

1. Introduction
As elite athletes, racehorses are susceptible to overuse injuries from repetitive motions incurred during intense training and competition. Thoroughbred and Quarter Horse racehorses competing over flat racecourses have respective sets of occupational skeletal injuries related to sites of concentrated high stresses incurred during task-specific training and racing activities. These injuries are typically referred to as stress fractures or fatigue fractures when the affected site is associated with the cortex of a long bone and as stress remodeling when the affected site is in a trabecular or subchondral location. The etiopathogenesis and biological repair mechanisms of these repetitive, overuse injuries are similar, irrespective of location. Fundamentally, injured bone tissue is capable of healing and regeneration. However, continued training and racing of horses with stress fractures or sites of active stress remodeling can promote severe or catastrophic fractures or irreversible osteoarthrosis when the underlying bone tissue is weakened and can no longer support the overlying articular cartilage. Thus, it is important to identify stress fractures and sites of stress remodeling in racehorses so that affected racehorses can be appropriately rehabilitated to allow healing and return to athletic performance.

The purpose of this presentation is to enhance awareness of preferential sites and features of stress fractures in the shoulder and arm of the forelimb and of stress remodeling in the proximal sesamoid bones so that lesions in these regions are more likely to be detected and appropriately managed to achieve resolution in racehorses.

2. Unsoundness Associated With a Stress Fracture or Stress Remodeling
Lameness varies markedly in severity between affected horses, from unapparent or only a change in demeanor to non-weight-bearing. Lameness
is most commonly noted immediately after a training or racing event\textsuperscript{3,4} and may appear to resolve within days of onset. Stress fractures and stress remodeling commonly affect bilateral limbs because the repetitive stresses associated with training and racing are incurred by both forelimbs or hind limbs.\textsuperscript{5,6} Consequently, affected horses may not demonstrate a distinct unilateral limb lameness. Instead, bilateral limb lameness may manifest as an unwillingness to perform at an expected level, a change in attitude, or unusual resistive behavior (for example, unwillingness to enter the starting gate). Poorly performing horses should be given a thorough physical examination to rule out non-musculoskeletal causes and elucidate skeletal injuries. Knowledge of the sites predisposed to stress fracture enhances ability to detect these injuries in racehorses.

3. Scapular Stress Fractures

Complete scapular fractures occur through the site of a pre-existing stress fracture.\textsuperscript{2,5} Complete scapular fractures are the cause of 2% and 4% to 8% of fatal musculoskeletal injuries in Thoroughbred and Quarter Horse racehorses, respectively.\textsuperscript{2,5,7–9} Approximately 1 in 2500 Thoroughbred starters and 1 in 1000 Quarter Horse starters incurs a complete scapular fracture during a race.\textsuperscript{10} Although the overall prevalence and incidence of scapular stress fractures in racehorses are unknown, scapular lesions comprised 2% of positive findings for forelimb scintigraphic examinations in Thoroughbred racehorses.\textsuperscript{10}

Approximately 1 in 1000 Quarter Horse starters incurred a complete scapular fracture during a race.\textsuperscript{10} Although the overall prevalence and incidence of scapular stress fractures in racehorses are unknown, scapular lesions comprised 2% of positive findings for forelimb scintigraphic examinations in Thoroughbred racehorses.\textsuperscript{10}

Scapular stress fractures occur along the typical site and configuration of complete scapular fractures in racehorses (Fig. 1). Complete fractures occur transversely or obliquely at the distal end of the spine of the scapula, dividing the scapula into a large proximal component and a smaller distal component.\textsuperscript{5,7,12} Although comminution, distal propagation into the glenoid, and proximal propagation of incomplete fracture lines are common, these are secondary to the transverse/oblique fracture component. Stress fractures occur predominantly at the distal end of the spine of the scapula in racehorses that died because of a complete scapular fracture.\textsuperscript{5,7} but stress fractures have also been reported to occur in the infraspinous fossa and supraspinous fossa, sites along the course of the transverse/oblique component of the complete fracture.\textsuperscript{4,5,13}

Horses are at highest risk for complete scapular fracture early in their career (2 years of age) or as 5-year-old or older horses, although racehorses of all ages can be affected.\textsuperscript{4,10,13} They occur more commonly in males than females. The right forelimb is affected over twice as frequently as the left forelimb, and bilateral complete fractures affect approximately 9% of horses that died because of a complete scapular fracture.\textsuperscript{10}

Complete scapular fractures occur during racing or during training. Approximately a quarter of affected Quarter Horses and a third of Thoroughbreds had never raced. Only half of unraced horses had completed an official timed work. Horses that incurred complete scapular fracture had fewer high-speed official timed works and races, lower accumulated high-speed distance, and fewer days in active training than age matched control horses.\textsuperscript{14} Consequently, racehorses that are in early high-speed training but behind that of their training cohort, Quarter Horses that had a prolonged lay-up, and Thoroughbreds in high-speed training for a longer duration than that of their training cohort should be examined for signs of scapular stress remodeling.\textsuperscript{14}

Clinical Presentation and Detection

Horses affected with a scapular stress fracture are usually presented because of acute onset of lameness after a race or high-speed work.\textsuperscript{4,13} Lameness is generally rated 2 to 3 of 5 (American Association of Equine Practitioners lameness scale). Importantly, the lameness may appear to resolve quickly. As expected, the lameness does not improve with diagnostic and regional nerve blocks administered at the level of the carpus and distally, although diagnostic blocks should be done with caution because of the risk of catastrophic fracture.

Physical examination is useful in detecting some affected horses. Some horses have responded positively to forelimb abduction and to palpation of the scapular spine (Fig. 2).\textsuperscript{4,13} Ultrasound examination can be useful for detecting irregular periosteal modeling/remodeling, a stair-step in the spine or
cortical surface, or hematoma, but these findings may not be present in horses with acute sub-periosseal bone trauma (Fig. 3). Radiography has not been useful for detection of scapular stress fractures, largely because of limitations related to extensive overlying soft tissues and bony structures of the thorax and neck. Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with scapular stress fracture and is useful in acute and chronic stages of disease (Fig. 4).

Rehabilitation
Conservative therapy of horses with scapular stress fracture has allowed return of racehorses to successful race performance, even after recurrence of a scapular stress fracture. Two months (60 days) of stall confinement, followed by 30 to 60 days of turnout in a small paddock, has been recommended before re-introduction to race training. Response to treatment and length of rehabilitation is guided by follow-up scintigraphic examination. Rehabilitated horses have raced from 6 to 17 months (median, 9 months) after diagnosis of a scapular stress fracture.

4. Humeral Stress Fractures
Humeral injuries comprised 6% of lesions for Thoroughbred racehorses examined scintigraphically for a musculoskeletal problem and 10% of positive findings for forelimb scintigraphic examinations in Thoroughbred racehorses, with 15% to 29% of horses affected bilaterally. More recently, 20% of racehorses that had a bone scan that included the humerus had a humeral lesion. Three-year-old racehorses are most commonly affected with humeral stress fracture; however, only 32% to 51% of horses had some race experience before diagnosis. Collectively, several studies support a left limb predilection for stress fractures in live racehorses (although this predilection can vary with racetrack surface material) and catastrophic fractures in deceased racehorses; but both forelimbs can be affected and a large proportion of affected racehorses have bilateral lesions.
Complete humeral fractures were the cause of 3% to 9% and 0% to 13% of fatal musculoskeletal injuries in Thoroughbred and Quarter Horse racehorses, respectively.\(^2\),\(^7\)–\(^9\),\(^18\) Complete humeral fractures are known to be associated with a pre-existing stress fracture (Fig. 5).\(^6\),\(^7\) Humeral stress fractures occur at several typical sites on the humerus in racehorses.\(^3\),\(^6\),\(^15\),\(^16\),\(^19\) Because stress fractures at caudoproximal (37% of stress fractures), medial diaphyseal (5%), and craniodistal (31%) sites occur along the typical long oblique catastrophic complete humeral fracture that occurs in racehorses (Fig. 6),\(^6\),\(^7\),\(^16\),\(^20\) the consequences of continuing to train and race on stress fractures at these sites may be more severe than stress remodeling at other sites. Most complete fractures (69–85%) from convenience samples of completely fractured humeri from racehorses extended through a caudoproximal region of stress remodeling.\(^6\),\(^16\)

Complete humeral fractures occur more commonly during training (89–91%) and often at a slow gallop but also occur during racing.\(^6\),\(^17\) However, 49% to 63% of affected horses had not raced before diagnosis of a humeral stress fracture.\(^5\),\(^12\) Risk for complete humeral fracture is highest after an increase in exercise after a 2-month or longer layup.\(^17\)

Clinical Presentation and Detection

Horses affected with a humeral stress fracture are usually presented because of acute onset of lameness after a race or high-speed work but may have a chronic insidious lameness duration of weeks to months.\(^3\),\(^15\) Lameness can range from 1 to 5 of 5 (American Association of Equine Practitioners lameness scale), with most horses having grade 2 to 4 lameness and some horses initially non–weight-bearing.\(^3\),\(^15\) Importantly, the lameness may appear to improve quickly, even within 24 hours.\(^15\) As expected, the lameness does not improve with diagnostic and regional nerve blocks administered at the level of the carpus and distally,\(^15\) although diagnostic blocks should be done with caution because of the risk of catastrophic fracture. Lameness was exacerbated in about half of affected horses after manipulation of the shoulder and elbow joints during flexion, adduction, and abduction of the upper portion of the limb.\(^3\),\(^15\) Horses with severe lameness typically had a shortened cranial phase of the stride at the walk and trot.\(^15\)

Radiographs have been useful for detection of humeral stress fractures but cannot be relied on for diagnosis of acute stress fractures when bone changes are insufficient to alter the radiographic appearance of affected humeri.\(^3\),\(^15\) Radiographic evidence of a stress fracture was observed in 56% to 92% of affected horses, 69% to 80% of caudoproximal stress fractures, and 71% to 100% of craniodistal stress fractures (Fig. 7).\(^3\),\(^15\) In some cases, radiographic detection may have been enhanced because radiography was preceded by scintigraphic diagnosis. It is notable that radiographic findings can be negative when lameness is first noticed but positive 10 days to 12 months later.\(^3\)

The author is unaware of any reports that demonstrated the diagnosis of humeral stress fractures through the use of ultrasound examination. However, ultrasound detection of periosteal new bone may be achievable with knowledge of high-risk horses and sites of predisposition.
Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with humeral stress fracture and is useful in acute and chronic stages of disease (Fig. 8). In two separate studies, none of the horses diagnosed with a humeral stress fracture developed a catastrophic complete humeral fracture in the affected humerus. In contrast, none of the 32 racehorses that developed catastrophic fracture had a known record of a scintigraphic examination.

Rehabilitation
Conservative therapy of horses with humeral stress fracture has allowed resolution of lesions and adaptation of the humerus to the stresses of racing by expansion of the affected cortex (Fig. 9). Racehorses return to racing after stress fracture diagnosis was 7.5 months (range, 5–22 months) for the 77% of horses that raced after fracture. Median number of races after fracture was 8.5 races (range, 1–33 races). No significant difference was found for mean earnings per start between before and after fracture. However, humeral stress fractures can recur at the same or distant sites in the same or contralateral limbs.

5. Proximal Sesamoid Bone Stress Remodeling
Proximal sesamoid bone fracture (with or without associated metacarpal bone fracture) is the most frequent catastrophic (fatal) musculoskeletal injury in racehorses, accounting for 45% to 50% of Thoroughbred injuries and 37% to 40% of Quarter Horse injuries. Although small displaced abaxial, apical, and basilar fragments are common in racehorses, these findings do not lead to catastrophic fractures of the proximal sesamoid bones. Complete transverse or oblique fractures of the proximal sesamoid bones disrupt transmission of force from the suspensory ligament to the distal sesamoidean...
ligaments and are the most common cause of fetlock injuries that are catastrophic because they disrupt the integrity of the suspensory apparatus.\(^\text{21}\)

The most common configuration for catastrophic fetlock injury is biaxial proximal sesamoid bone fracture.\(^\text{2,9,21}\) Complete, transverse, articular fracture that separates the body from the base of the medial proximal sesamoid bone is accompanied by complete, oblique or transverse, articular fracture of the lateral proximal sesamoid bone (Fig. 10).\(^\text{21}\)

Fractures often have some comminution and disruption of adjacent soft tissues. Although other proximal sesamoid bone fracture configurations also disrupt the suspensory apparatus, the focus of this report will be on medial proximal sesamoid bone fracture because of lesions that probably predispose to fracture and initiation of suspensory apparatus failure.\(^\text{22,23}\)

Two lesions probably predispose to medial proximal sesamoid bone fracture.\(^\text{22,23}\) Both lesions are foci of bone resorption that occur in response to the local accumulation of microdamage acquired from the repetitive load cycles associated with training and racing. The first lesion that was recognized occurs on the palmar margin of the proximal sesamoid bone (Fig. 11).\(^\text{22}\) The second lesion is more common and occurs consistently within a subchondral, abaxial location within the medial proximal sesamoid bone (Fig. 12).\(^\text{23}\) Both lesions are large enough to create a stress riser sufficiently large to initiate bone fracture but small enough, and, in difficult locations, to detect with the use of radiography.

**Clinical Presentation and Detection**

The clinical signs that distinguish horses with impending catastrophic fracture of the proximal sesamoid bones from horses that can race successfully without injury is challenging. However, several pieces of evidence demonstrate that some features of horse signalment and history and some clinical signs are associated with increased risk for catastrophic fracture.

Racehorses that have catastrophic failure of the fetlock suspensory apparatus, including horses with proximal sesamoid bone fractures, are typically older horses (\(\geq 5\) years old), more likely to be male than female, have had a long racing career (\(\geq 20\)

---

**Fig. 10.** Dorsopalmar radiograph of proximal sesamoid bone specimens illustrates a common fracture configuration. The medial proximal sesamoid bone incurs a relatively simple transverse fracture that splits a larger proximal fragment from the base. The lateral proximal sesamoid bone fracture varies more but commonly has an oblique fracture that separates the apex from the body.

**Fig. 11.** Dorsal (articular) surface of the proximal sesamoid bones from the fractured leg of a racehorse with fractures of the medial and lateral proximal sesamoid bones. The fracture surfaces of the medial proximal sesamoid bone have been opened to visualize the stress-remodeling lesion (ellipses) on the palmar aspect of the bone.
races), and have experienced prolonged high exercise intensity.\textsuperscript{24,25} Furthermore, horses that had a catastrophic fetlock injury were 3 times more likely to have had a palpable abnormality of the suspensory ligament; they were 2 times more likely to have had an abnormality of the fetlock joint on pre-race inspection; and the pre-race veterinary inspector was 8 to 14 times more likely to have considered the horse to be at higher risk.\textsuperscript{9,24} The usefulness of these findings is tempered by the low specificity of the findings; that is, a much larger number of horses had a similar abnormality but did not receive a subsequent injury in the associated race.\textsuperscript{26} However, when the long-term performance of horses is considered, 45\% of horses with signs of mild suspensory apparatus injury had a training remission caused by a severe musculoskeletal injury within 3 months compared with 14\% of horses without the same signs.\textsuperscript{27} Thus, horses with mild injuries to the suspensory apparatus are much more likely to drop out of the racing pool.

The palmar and subchondral locations of the foci of stress remodeling create challenges in lesion detection. Synovial effusion and synovial fluid abnormalities are unlikely to result directly from stress-remodeling lesions in the proximal sesamoid bones because neither lesion is intra-articular or intra-thecal. However, synovial effusion of the fetlock joint may be present because other degenerative lesions (eg, proximodorsal proximal phalangeal osteochondral fragments, score lines, palmar osteochondral disease) usually accompany proximal sesamoid bone lesions. Similarly, intra-synovial anesthesia may not result in improvements in lame-

ness unless lameness is caused by other attendant intra-synovial degenerative changes. However, regional anesthesia that desensitizes the fetlock region is likely to improve any observed lameness or result in enhancing lameness in the contralateral limb because stress remodeling is often bilateral.

Radiographs have not been useful for detection of lesions that might predispose to catastrophic fracture of the proximal sesamoid bone.\textsuperscript{21} Paradoxically, among all proximal sesamoid bones from horses that had a catastrophic proximal sesamoid bone fracture, the likelihood of bone fracture was reduced by half when the bone had radiographic evidence of osteophytes and to less than a quarter when the bone had prominent vascular channels.\textsuperscript{21} The two sites of stress remodeling are in difficult locations to visualize radiographically because of overlying bone tissue and contours (Fig. 13). Whereas the abaxial site appears to be associated with abaxial vascular channels, the subchondral site appears to be associated with basilar vascular channels.

Radiographs are useful for detecting other lesions of the fetlock that usually (but not always) accompany proximal sesamoid bone disease. Horses that sustained catastrophic fracture of the proximal sesamoid bones often have degenerative lesions of the metacarpal condyle (palmar osteochondral disease), osteochondral degeneration or fragmentation of the dorsoproximal margin of the proximal phalanx, and other articular cartilage degenerative changes. However, it is not known how often these changes are present without proximal sesamoid bone disease.
Ultrasound examination might be useful for detecting abnormal bone contours on the palmar aspect of the proximal sesamoid bone. Unfortunately, this region of the bone is normally irregular in contour; therefore, distinction of contours caused by lesions from normal contours could be difficult. The author is unaware of any reports that demonstrated the diagnosis of proximal sesamoid bone stress remodeling through the use of ultrasound examination.

Magnetic resonance imaging has potential for detection of stress-remodeling lesions. Technology and image acquisition and analysis software enhancements for standing magnetic resonance imaging machines may make detection of stress-remodeling lesions in the proximal sesamoid bone feasible in horses without the risks associated with general anesthesia.

Clinical computed tomography currently requires general anesthesia, and, in our experience, the small foci of stress remodeling in the proximal sesamoid bones makes lesion detection challenging. Enhancements in computed tomography technology could increase chances of lesion detection.

Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with stress remodeling (Fig. 14). The challenge is differentiating changes associated with adaptive remodeling of the proximal sesamoid bone in response to the increased loads associated with training and racing from pathologic or maladaptive remodeling. Furthermore, it can be difficult to differentiate increased activity in the palmar aspect of the metacarpal condyle from increased activity in the proximal sesamoid bone, particularly because palmar osteochondral disease often accompanies subchondral proximal sesamoid bone lesions. Because flexion of the fetlock moves the proximal sesamoid bones proximal to the metacarpal condyle, a lateral image of the flexed fetlock may allow differentiation of proximal sesamoid activity from metacarpal condylar activity.

Rehabilitation
Bone lesions are capable of healing. Affected horses need time without intensive training and racing to allow resolution of bone lesions. Clinically, the difficulty is detecting lesions so that healing progression can be monitored to guide rehabilitation of affected horses.

Acknowledgment
This work was supported by the California Horse Racing Board Racing Safety Program.

References
3. Mackey VS, Trout DR, Meagher DM, et al. Stress fractures of the humerus, radius, and tibia in horses: clinical features...