Disorders of the infraspinatus tendon and bursa in three horses

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Case Description—3 horses with penetrating wounds to the shoulder area were examined because of forelimb lameness.

Clinical Findings—All horses had physical examination findings (decreased cranial phase of the stride, swelling in the shoulder region, and signs of pain on manipulation of the shoulder) that were suggestive of problems in the upper portion of the forelimb. Injury to the biceps tendon or bursa was the primary differential diagnosis in each instance, but no abnormalities involving those structures were found. Radiographic and ultrasonographic imaging revealed injuries to the caudal eminence of the greater tubercle of the humerus, the infraspinatus tendon, and the infraspinatus bursa. Examination with ultrasound was more sensitive than radiography at detecting both osseous and soft tissue changes.

Treatment and Outcome—All 3 horses responded favorably to treatment with antimicrobials and nonsteroidal anti-inflammatory drugs. Although initial response to standing lavage was favorable in 1 horse, endoscopic lavage was later required. Standing removal of fracture fragments was performed in 2 horses. Ultrasonographic imaging was helpful in monitoring the response to treatment and changes in the affected structures. All 3 horses eventually became sound after treatment.

Clinical Relevance—Infraspinatus bursitis and tendonitis should be included in the differential diagnoses of horses with shoulder lameness. Diagnosis and monitoring should include ultrasonographic monitoring. The prognosis for return to soundness after appropriate treatment appears to be good. (*J Am Vet Med Assoc* 2006;229:549–556)

A 4-year-old Arabian mare weighing 375 kg (827 lb; horse 1) was referred to the University of California at Davis VMTH with a 1-day history of severe right forelimb lameness. The horse was at pasture and had no obvious signs of traumatic injury that were evident to the owner or referring veterinarian. On initial examination, the horse had signs of anxiety and was trembling and sweating. Physical examination abnormalities included tachycardia (66 beats/min), mild diffuse swelling of the right shoulder region, and grade 4/5[a] lameness of the right forelimb with a decreased cranial phase of the stride. Abnormal findings on a CBC and serum biochemical analysis included leukocytosis (14,920 cells/mL; reference range, 5,000 to 11,600 cells/mL), neutrophilia (11,578 cells/mL; reference range, 2,600 to 6,800 cells/mL), mild hyperfibrinogenemia (500 mg/dL; reference range, 100 to 400 mg/dL), mild hyperglobulinemia (4.8 g/dL; reference range, 1.7 to 4.7 g/dL), and high serum activities of creatine kinase (786 U/L; reference range, 119 to 287 U/L) and aspartate aminotransferase (635 U/L; reference range, 138 to 409 U/L). No radiographic abnormalities were detected in standing mediolateral and humeral skyline projections of the shoulder or in mediolateral projections of the humerus and elbow joint. A small (< 1 cm in length) healing skin wound was noticed over the triceps musculature, caudal and distal to the right shoulder joint. The wound edges were well granulated with no evidence of drainage. Complete ultrasonographic examination of the right shoulder was performed with a 10-MHz linear[b] and 6-MHz curvilinear[c] transducer at a scanning depth of 4 to 5 cm. The infraspinatus bursa was severely distended with echogenic fluid, and the synovial membranes of the structure were thickened (width, 4 mm). Bursal distension was observed cranial to the infraspinatus tendon and dorsal to the caudal eminence of the GT but was most evident caudal to the infraspinatus tendon (**Figure 1**). The skin wound was in close proximity to the bursa, but no communicating tract between the wound and infraspinatus bursa was detected. Mild cortical roughening of the caudal eminence of the GT was also noticed. Ultrasound-guided aspiration of the bursal contents yielded a large volume of purulent material with a protein concentration of 7.0 g/dL (reference range, < 2.0 g/dL) and a nucleated cell count of 2,904,000 cells/mL (reference range, < 20,000 cells/mL), with 96% nondegenerate neutrophils. No bacteria were detected. The fluid was severely purulent and was submitted for aerobic and anaerobic microbial culture and susceptibility testing.

A clinical diagnosis of septic infraspinatus bursitis was made, and ultrasound-guided, through-and-through lavage of the bursa was performed with the horse standing, on the evening of admission. Potassium penicillin G (22,000 U/kg [10,000 U/lb], IV), gentamicin sulfate (6.6 mg/kg [3 mg/lb], IV), and phenylbutazone (4.4 mg/kg [2 mg/lb], IV) were administered prior to surgery. Adequate sedation and analgesia were achieved with doses of detomidine (0.005 mg/kg [0.002 mg/lb], IV) and butorphanol (0.01 mg/kg [0.005 mg/lb], IV) administered intermit-

Abbreviations

VMTH	Veterinary medical teaching hospital
GT	Greater tubercle

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tently during the procedure. After local infiltration of the skin with a 2% lidocaine solution, a 12-gauge, 3-inch stainless steel needle[d] was advanced 3 to 4 cm into the distended bursa caudal to the infraspinatus tendon and slightly distal to the caudal eminence of the GT. The bursa was distended with 40 mL of sterile lactated Ringer's solution. A second 12-gauge, 3-inch stainless steel needle was inserted into the bursa slightly distal and caudal to the first needle for fluid egress. An 18-gauge, 3.5-inch spinal needle was placed in the infraspinatus bursa cranial to the tendon at the level of the caudal eminence of the GT for fluid egress (**Figure 2**). All needles were placed with ultrasound guidance. A fluid pump was used to lavage the bursa with 7 L of lactated Ringer's solution. Excellent egress was obtained through the cranial and caudal needles. Amikacin sulfate (500 mg) was instilled into the bursa after lavage. Portals were left open to promote drainage. The horse tolerated the procedure well. A treatment regimen of potassium penicillin G (22,000 U/kg, IV, q 6 h), gentamicin sulfate (6.6 mg/kg, IV, q 24 h), and phenylbutazone (2.2 mg/kg [1 mg/lb], IV, q 12 h) was continued.

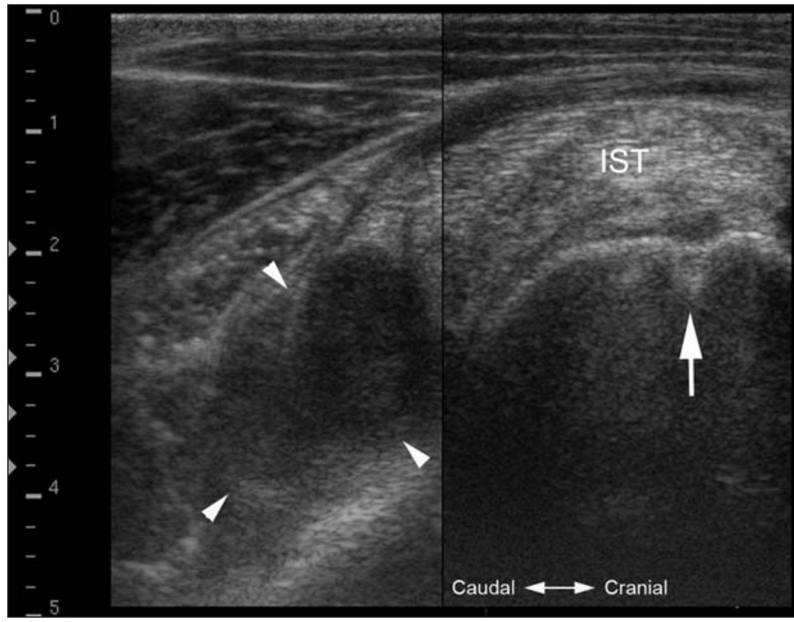

Figure 1—Composite transverse ultrasonographic image of a septic infraspinatus bursa in a horse (horse 1). Notice that the caudal reflection of the infraspinatus bursa is filled with echogenic fluid (arrowheads; dimensions, 2.0 X 3.3 cm). The infraspinatus tendon (IST) had a normal layered appearance at the level at which this image was obtained. The small cortical defect (arrow) of the caudal eminence of the GT grew larger during the next 10 days. Image was obtained with a 10-MHz linear transducer at a scanning depth of 5 cm.

Although the horse's gait improved over the next 24 hours, the lameness worsened slightly over the ensuing few days. Ultrasonographic reevaluation on day 4 revealed persistent distension of the infraspinatus bursa. Echogenic debris floating in the fluid was interpreted as fibrin. Increased cortical roughening of the caudal eminence of the GT was suggestive of osteomyelitis secondary to septic bursitis. Multiple radiographic skyline projections of the GT were obtained, but failed to confirm the ultrasonographic findings. Ultrasound-guided aspiration of the bursa was repeated. The shoulder joint was inadvertently entered on the first attempt, yielding clear yellow synovial fluid with a protein concentration of 4.5 g/dL and a nucleated cell count of 2,300 cells/µL (16% nondegenerate neutrophils). No bacteria were observed. Analysis of the fluid revealed mild inflammation secondary to a regional inflammatory response. A second attempt at bursal centesis yielded opaque, purulent fluid from the infraspinatus bursa. Standing ultrasound-guided bursal lavage was repeated with 6 L of sterile lactated Ringer's solution via the same technique as described previously. Amikacin (500 mg) was again instilled into the bursa after the lavage procedure. Microbial culture and susceptibility testing results from the bursal fluid obtained on day 1 became available and indicated small numbers of *Enterobacter cloacae* with resistance to penicillin but susceptibility to amikacin, ceftiofur, enrofloxacin, gentamicin, and trimethoprim sulfa. Treatment with potassium penicillin was discontinued, and administration of ceftiofur (2.6 mg/kg [1.2 mg/lb], IV, q 12 h) was initiated.

Only mild improvement in the right forelimb lameness was observed over the next 3 days. Ultrasound-guided aspiration of the infraspinatus bursa yielded fluid with high protein concentration (7.4 g/dL). Direct smear of the aspirated fluid revealed 96% nondegenerate-to-moderately degenerate neutrophils, but no bacteria were seen. A nucleated cell count could not be obtained

Figure 2—Photograph depicting instrument placement in a standing horse undergoing lavage of the infraspinatus bursa. The ingress and caudal egress needles have been placed caudal to the infraspinatus tendon, and a cranial egress needle is in place cranial to the infraspinatus tendon. All needles were placed with ultrasound guidance. Similar instrument placement was later used for endoscopy of the infraspinatus bursa. The location of the small skin wound is indicated (arrow). The horse's neck is to the right of the photograph.

because of mucin clot formation in the sample. Intrabursal anesthesia with 3 mL of 2% lidocaine solution and intrasynovial injection of 750 mg of amikacin sulfate elicited noticeable improvement in the right forelimb lameness. A decision was made to perform endoscopic lavage of the bursa with the horse anesthetized on the following day (day 7). After induction of general anesthesia, the horse was positioned in left lateral recumbency and anesthesia was maintained with inhaled isoflurane and oxygen. Ultrasonography was used to identify the bursa, and an 18-gauge, 3.5-inch spinal needle was inserted into the bursa cranial to the infraspinatus tendon and 1 cm distal to the GT. Bursa fluid obtained had a total protein concentration of 6.7 g/dL and a nucleated cell count of 207,000 cells/µL. The bursa was distended with 40 mL of sterile lactated Ringer's solution. An endoscopic portal was created by sharply incising the skin and SC tissues at the needle site. The bursa was penetrated with a sharp obturator inserted in a 12-cm arthroscopic sheath. The obturator was removed, and a 4-mm-diameter, 18-cm, 30° arthroscope was inserted into the bursa. The bursa was distended with a continuous infusion of lactated Ringer's solution. Two 12-gauge, 3-inch stainless steel needles were placed caudally into the bursa for fluid egress. Endoscopic examination of the bursa revealed thickened hyperemic synovium and several areas of bone with an irregular surface at the caudal eminence of the GT. The caudal egress portal was used as an instrument portal. Attempts to debride the bone through this portal were unsuccessful because of restriction of instrument mobility by overlying muscle and the large infraspinatus tendon. Lavage of the bursa was discontinued after 10 L of fluid had been infused because of extravasation of fluid from the bursa into adjacent musculature. The arthroscope was removed, and as much fluid as possible was manually expressed from the bursa. The instrument portals were left open to permit drainage, and an iodine-impregnated drape[e] was placed on the skin to protect the portals during the horse's recovery from general anesthesia.

The horse's lameness improved steadily after the endoscopic procedure. Repeat ultrasound performed 4 days after surgery (day 11) revealed less effusion and synovial thickening in the bursa; however, the cortical defect in the caudal eminence of the GT had enlarged and was 11 mm wide, 4.7 mm deep, and 13 mm long **(Figure 3)**. The infraspinatus tendon had an enlarged cross-sectional area, and a new hypoechoic area was observed adjacent to the cortical defect in the GT. The horse was maintained on a regimen of IV administered antimicrobials for 10 days after the procedure, at which point orally administered enrofloxacin[f] (5 mg/kg [2.3 mg/lb] q 12 h) was instituted. The dose of phenylbutazone was reduced (2.2 mg/kg, PO, q 24 h) 5 days after the procedure because of the substantial improvement in lameness. Recheck ultrasound imaging performed 3 days prior to discharge revealed only mild effusion and synovitis of the infraspinatus bursa. The cortical defect in the GT was slightly wider in dimension, and the degree of infraspinatus tendonitis was unchanged. The horse was discharged to the owner's care 24 days after admission with instructions for stall confinement and continuation of enrofloxacin administration for 8 days and phenylbutazone administration for 5 days. At the time of discharge, the horse was walking well with minimal signs of lameness and a normal cranial phase

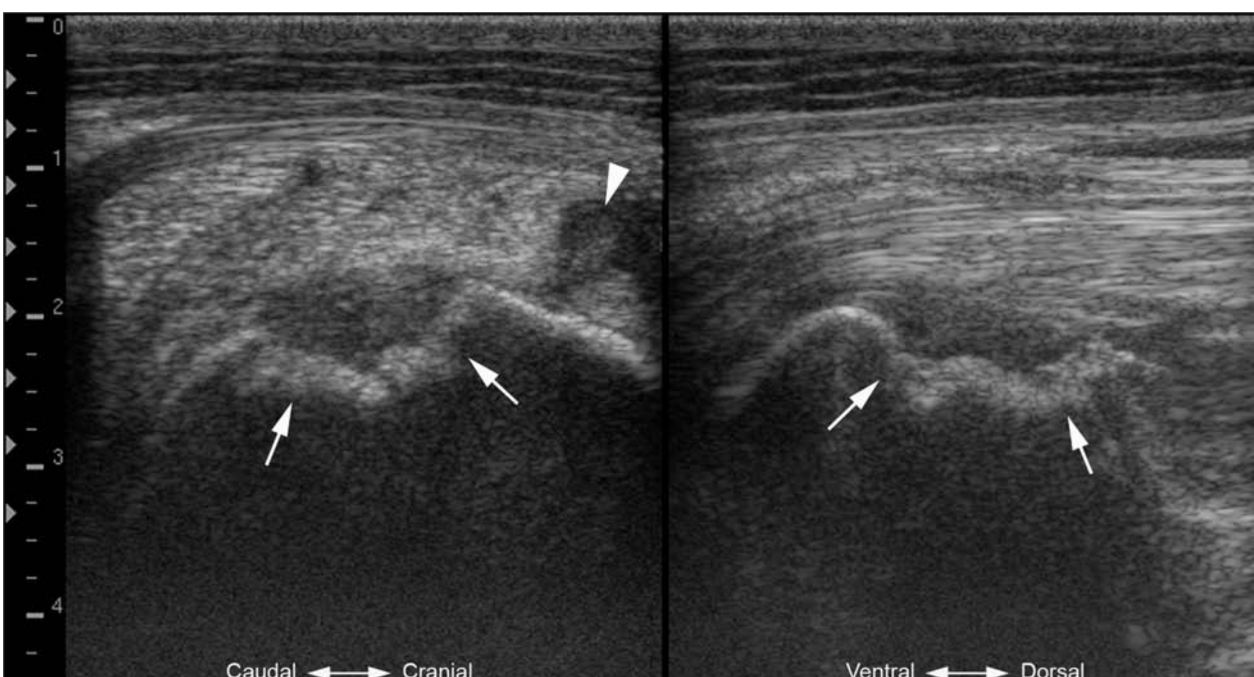

Figure 3—Transverse (left) and longitudinal (right) ultrasonographic images of a destructive lesion (arrows) involving the caudal eminence of the GT that developed secondary to septic infraspinatus bursitis in the same horse as in Figure 1. Notice the increase in size of the bony defect, compared with its configuration in Figure 1. The thickened synovium of the infraspinatus bursa (arrowhead) is evident cranial to the infraspinatus tendon. Image was obtained with a 10-MHz linear transducer at a scanning depth of 4.5 cm.

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of the stride. Recheck lameness examination and ultrasonographic evaluation were recommended in 14 days, but the horse was not returned to the VMTH or to the referring veterinarian for follow-up examination. Multiple attempts to contact the owner were unsuccessful; however, the horse was seen by the referring veterinarian for routine veterinary care 1.5 years after discharge, at which time no forelimb lameness was observed.

A 13-year-old Appaloosa mare (horse 2) was referred to the VMTH 4 hours after falling on a fence and incurring a large laceration in the right shoulder region. The referring veterinarian administered gentamicin sulfate and phenylbutazone and sutured the wound prior to transport to protect the exposed underlying bone. On initial examination at the VMTH, the horse was tachycardic (84 beats/min) and tachypneic (24 breaths/min) and had grade 4/5 right forelimb lameness. The sutured laceration measured 60 cm in length and extended dorsally from the right pectoral region to the point of the shoulder, then curved caudally and extended an additional 20 cm. No clinically important hematologic or electrolyte abnormalities were detected.

Subcutaneous emphysema and irregularity of the contour of the GT were noticed on standing mediolateral and lateral oblique (caudolateral to craniomedial) radiographic views of the right shoulder. An SC line block with 2% lidocaine solution was performed in the skin dorsal to the laceration, and the sutures were removed prior to exploration of the wound. The skin was easily reflected away from the underlying muscle bellies, revealing severe trauma to the brachiocephalicus and biceps brachii musculature distal to the humeral tubercles. The wound was thoroughly lavaged, and a 3 × 4 × 2-cm bony fragment was removed from the region of the GT through the open wound. The wound and muscle edges were debrided, a Penrose drain was placed at the distal extent of the wound, and the wound was closed in layers in routine fashion. Arthrocentesis of the shoulder joint was not performed because of the gross contamination of overlying tissues. Treatment with potassium penicillin G, gentamicin sulfate, and phenylbutazone was instituted. The skin flap became necrotic and was removed on day 7, exposing a 10 × 16-cm area of underlying muscle. The open portion of the wound was flushed daily and protected by stent bandaging and sterile laparotomy sponges. The horse continued to walk well until day 9, when the lameness in the affected limb worsened. Ultrasonographic examination of the right shoulder on day 11 revealed a sharp and irregular contour of the caudal eminence of the GT, confirming the origin of the fracture fragment that was removed at admission. The overlying infraspinatus tendon was enlarged and had a large hypoechoic area and irregular fiber pattern adjacent to the fracture bed in the GT. Subcutaneous emphysema from the nearby wound prevented sonographic evaluation of the infraspinatus tendon insertion and the portion of the infraspinatus bursa cranial to the tendon. The caudal portion of the bursa did not appear distended. The lesser and intermediate tubercles and the cranial eminence of the GT were smooth, and no additional fracture fragments were observed. There was no evidence that the wound communicated with the shoulder joint or bicipital bursa. The horse continued to receive gentamicin sulfate administered IV and procaine penicillin G administered IM until day 15, when antimicrobial treatment was changed to orally administered chloramphenicol (20 mg/kg [9.1 mg/lb], q 8 h). Daily treatments included wound cleansing and stent bandage changes. The horse was discharged after 20 days of hospitalization with instructions for stall rest and only minimal walking until the wound was completely healed. Orally administered chloramphenicol and phenylbutazone were continued for 10 and 7 days after discharge, respectively. At the time of discharge, the horse was lame at the walk and had a markedly decreased cranial phase of the stride. A guarded prognosis for return to athletic soundness was given. Follow-up information obtained from the referring veterinarian revealed that the horse gradually improved and returned to its previous use as a trail horse without lameness for approximately 3 years. The skin wound healed well with minimal scarring. The horse was euthanatized for unrelated reasons 4.5 years after discharge.

A 4-year-old Tennessee Walking Horse gelding (horse 3) was referred to the VMTH 4 days after becoming entangled in a tree and incurring a small puncture wound in the left shoulder region. The referring veterinarian administered tetanus toxoid, initiated treatment with procaine penicillin G and phenylbutazone, and began saline (0.9% NaCl) solution wound flushes. Prominent swelling of the left shoulder region with severe lameness and mild serous drainage from the wound were observed initially, but these signs gradually improved until the day of examination at the VMTH, when the lameness acutely worsened. At admission, the horse had a grade 4/5 lameness in the left forelimb and had difficulty extending the limb cranially. There was a small volume of serous drainage from the 2-cm linear wound slightly distal to the point of the shoulder (cranial eminence of the GT). A CBC revealed anemia (PCV, 22%; reference range, 30% to 46%) and fibrinogen in reference range (400 mg/dL; reference range, 100 to 400 mg/dL). Wound exploration with a blunt stainless steel probe revealed a focal area of extension of the wound 2 to 3 cm deeper into the tissues. No synovial fluid was obtained during arthrocentesis of the shoulder joint or bicipital bursa. Amikacin sulfate (250 mg) was instilled into the shoulder joint after the arthrocentesis procedure. No osseous abnormalities were detected on standing mediolateral radiographic projections of the left shoulder. Contrast radiography of the wound revealed contrast material extending distally into SC tissues only. Ultrasonographic evaluation of the left shoulder revealed a large step defect with fragmentation of the caudal eminence of the GT (**Figure 4**). The overlying infraspinatus tendon had a large hypoechoic area with fiber tearing. A second bony fragment was detected cranial to the infraspinatus tendon. A hypoechoic tract extended from the wound to the infraspinatus bursa. The bursal synovium was thickened and edematous, and there was a moderate volume of effusion. Ultrasound-guided aspiration of the bursal contents yielded an insufficient volume of clear yellow fluid for

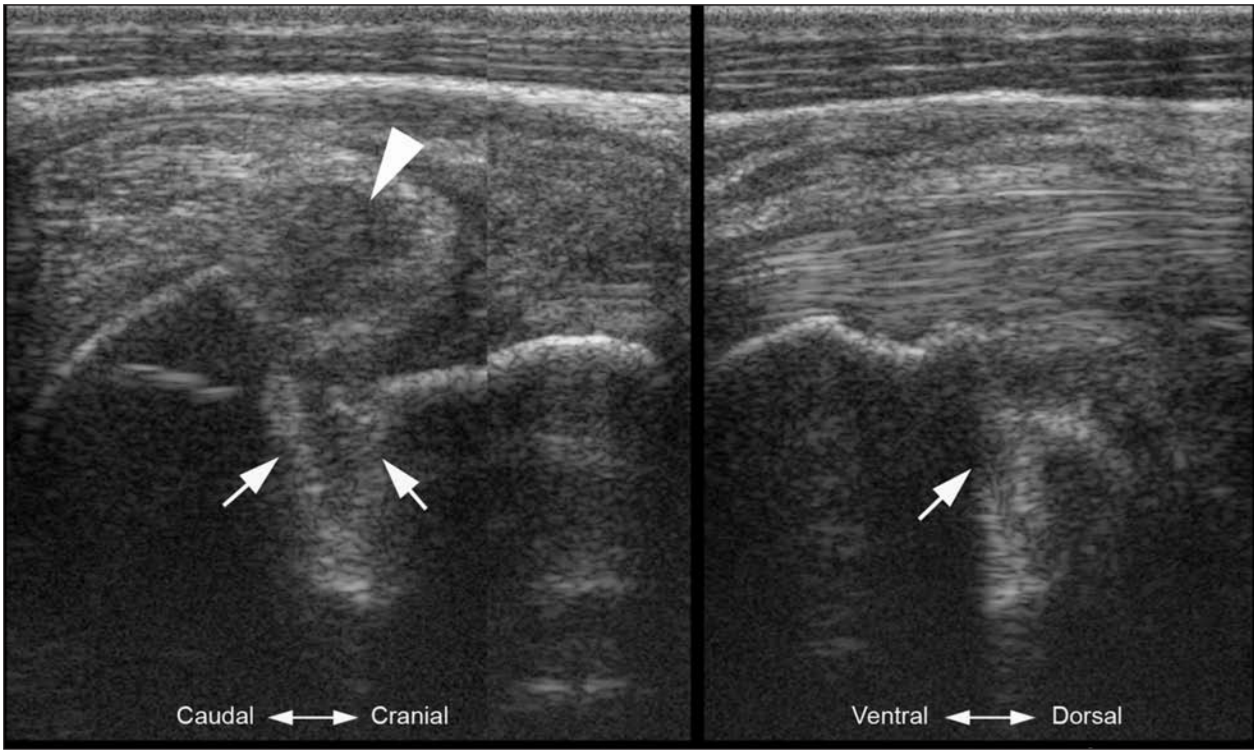

Figure 4—Transverse (left) and longitudinal (right) ultrasonographic images of a fracture (arrows) of the caudal eminence of the GT of the humerus in horse 3. Notice the large hypoechoic area (arrowhead) in the infraspinatus tendon adjacent to the fracture. Image was obtained with a 10-MHz linear transducer at a scanning depth of 5 cm.

cytologic evaluation or culture, but a direct smear of the fluid was interpreted as indicating mild purulent inflammation with 96% nondegenerate neutrophils. No micoorganisms were detected in the fluid. The horse was maintained on a regimen of procaine penicillin G, gentamicin sulfate, and phenylbutazone.

The fracture fragments from the caudal eminence of the GT were removed during a standing procedure while the horse was sedated with small doses of detomidine (0.005 mg/kg, IV) and butorphanol (0.01 mg/kg, IV), repeated as needed for maintenance of sedation and analgesia. After the skin was anesthetized, a 19-gauge, 1.5-inch hypodermic needle was placed with ultrasound guidance to locate the largest fracture fragment while avoiding the skin wound and tract to the infraspinatus bursa. The needle was used as a guide for incision of tissues to expose the fracture fragment. Three fracture fragments were removed from the GT with Rongeur forceps. The largest fragment was 3 cm in diameter. The shoulder joint capsule was entered during removal of that fragment and was subsequently lavaged with 1 L of sterile lactated Ringer's solution. Polymethylmethacrylate beads impregnated with 500 mg of amikacin were placed in the incision. A stent bandage was used to protect the wound and incision site. The horse's gait improved immediately after the procedure, and continued improvement was observed over the next 72 hours, although the shortened cranial phase of the stride persisted. The antimicrobial regimen was changed to chloramphenicol (25 mg/kg [11.4 mg/lb], PO, q 8 h) administration on day 4 because the horse would not tolerate IM injections. Other daily treatments included basic wound care and stent bandage changes.

On day 6, the lameness worsened, and the stent bandage was soaked with clear fluid. Arthrocentesis of the shoulder joint yielded fluid with a cloudy yellow appearance, protein concentration of 5.1 g/dL, and nucleated cell count of 24,600 cells/µL, 98% of which were nondegenerate neutrophils. No organisms were observed on a direct smear of the fluid, and culture yielded no growth of bacterial organisms. Distension of the shoulder joint with 80 mL of saline solution resulted in leakage of fluid from the incision site, confirming that there was direct communication with the shoulder joint. The joint was lavaged with 2 L of lactated Ringer's solution, and 500 mg of amikacin sulfate was instilled into the joint. New polymethylmethacrylate beads impregnated with 500 mg of amikacin were placed in the incision, and a stent bandage was placed. The horse's lameness improved during the next 3 days. Repeat arthrocentesis of the shoulder joint on the day of discharge from the hospital (day 9) revealed an improved total protein concentration (3.8 g/dL) and nucleated cell count (1,000 cells/µL; 69% nondegenerate neutrophils). There was no leakage of fluid from the incision site after distension of the shoulder joint with 60 mL of saline solution. Amikacin sulfate (500 mg) was instilled into the joint. The horse was discharged and the owner given instructions to continue stall confinement with brief periods of hand walking. The horse was continued on a regimen of orally administered chloramphenicol for an additional 14 days and phenylbutazone for 7 days, after which the dose of phenylbutazone was decreased and administered for an additional 10 days. The owner reported that the horse gradually improved and returned to its previous use as a trail horse with no lameness. Communication with the referring veterinarian 2 years after discharge from the hospital confirmed the absence of residual lameness.

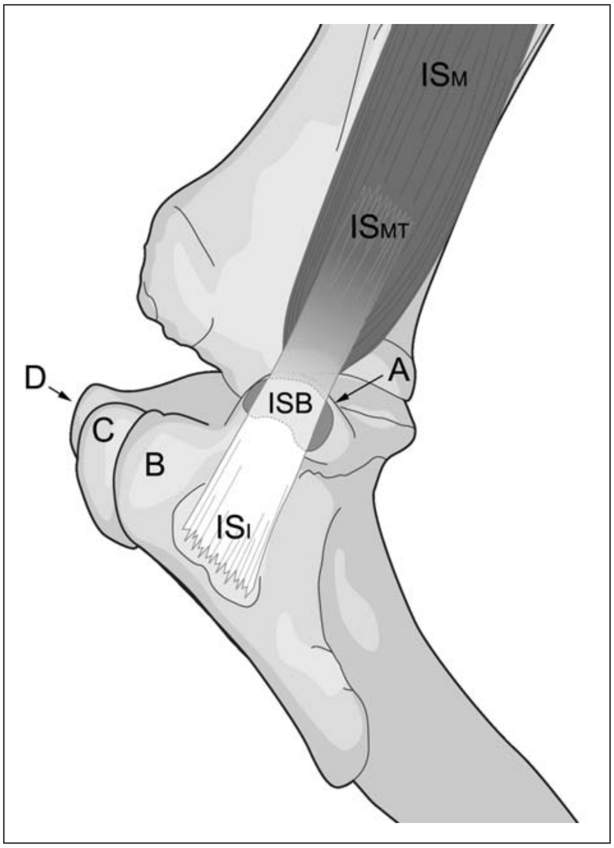

Figure 5—Diagram of the lateral view of the region of the infraspinatus tendon and bursa, including the humeral tubercles in the left forelimb of a horse. Cranial is to the left. A = Caudal eminence of GT. B = Cranial eminence of the GT. C = Intermediate tubercle. D = Lesser tubercle. IS_M = Infraspinatus muscle. IS_{MT} = Musculotendinous junction of the infraspinatus muscle. IS_I = Infraspinatus tendon insertion. ISB = Infraspinatus bursa.

Discussion

The anatomy and normal ultrasonographic appearance of the infraspinatus tendon and bursa have been described.[1,2] The infraspinatus tendon provides lateral support to the shoulder as it extends from the infraspinatus muscle and courses over the shoulder joint and caudal eminence of the GT to its primary insertion on the dorsolateral aspect of the humerus **(Figure 5)**.[1,2] The infraspinatus bursa is located between the infraspinatus tendon and caudal eminence of the GT. The bursa is not clearly visible in most horses and, in our experience, is rarely distended except when septic, in which instance ultrasonographic imaging typically reveals distension in the caudal portion of the bursa to be more prominent than in the cranial portion.

To the authors' knowledge, disorders of the infraspinatus bursa or tendon have not been described as a cause of lameness in the horse, although inflammation of the infraspinatus bursa was briefly described in 1 textbook.[3] In contrast, abnormalities involving the biceps tendon and bicipital bursa have frequently been reported.[4–13] The occurrence and treatment of septic bicipital bursitis have also been well documented,[5,6,8–11,13,14] and it is known that in many instances, the condition develops secondary to an external wound.[5,6,10,13] All horses in the present study had skin wounds that were indicative of a penetrating traumatic injury. Injury to the biceps tendon, with or without communication with the bicipital bursa, was the primary differential diagnosis in each of these 3 horses; however, no abnormalities involving those structures were found. Physical examination findings, including decreased cranial phase of the stride, heat and swelling in the shoulder region, and resentment of manipulation of the shoulder, suggested a problem with the upper portion of the limb, but no localizing physical examination findings specific to the infraspinatus tendon or bursa were detected. A combination of radiographic and ultrasonographic imaging was necessary to determine the source of lameness. Ultrasound was the definitive diagnostic imaging modality for diagnosis of the infraspinatus disorders, but it was also the only imaging modality that enabled identification of the osseous abnormalities in 2 of the 3 horses.

Radiography is one of the most common diagnostic tools used to evaluate the equine shoulder but often provides limited information.[15–17] In the present report, radiographs failed to reveal the destructive lesion of the caudal eminence of the GT in horse 1 and the fractures of the caudal eminence in horse 3. Even with the high-output radiographic systems used in many hospitals, evaluation is primarily limited to mediolateral and caudolateral to craniomedial (lateral oblique) projections because of the large size of adult horses. The caudal eminence is difficult to project on these views because of its relative caudal and lateral location and superimposition of other osseous structures.[15] Skyline radiographic projections of the humeral tubercles have been described,[16] but those views could not be obtained in 2 of the 3 horses in the present study because of signs of severe pain that precluded the shoulder flexion required to obtain the views. Skyline views did not reveal the lytic lesion in horse 1 and may not have detected the abnormalities involving the caudal eminence in the other 2 horses. Skyline views best reveal abnormalities of the cranial eminence of the GT, the intermediate tubercle, and the lesser tubercle. Although fractures of the humeral tubercles have frequently been reported,[18–22] reports of fractures of the caudal eminence of the GT are rare.[20] Because the bony contour of the caudal eminence is readily visible with ultrasound, it is possible that fractures of the caudal eminence will be recognized more frequently with increased use of ultrasound to evaluate the shoulder.

Most reports on ultrasonography of the equine shoulder have focused on imaging the biceps tendon and bicipital bursa.[5,7,8,23–25] The processes of diagnosis and monitoring in the horses of the present report emphasize the importance of performing a complete ultrasonographic examination of the shoulder region.[2,26] At the University of California at Davis VMTH, ultrasonographic examination of the shoulder region includes evaluation of the biceps tendon, bicipital bursa, the lesser and intermediate tubercles and the cranial and caudal eminences of the GT, supraspinatus tendons of insertion, infraspinatus tendon and bursa, scapula (including the scapular spine and the infraspinous and supraspinous fossae), and shoulder joint surfaces.[26] All structures can be reliably imaged with a high-frequency (7- to 14-MHz) linear transducer designed for musculoskeletal imaging at a scanning depth of 4 to 8 cm. A midrange-frequency (4- to 8-MHz) microconvex transducer is useful for imaging some structures and for ultrasound-guided interventional procedures. The examination is performed in the same manner regardless of results of intra-articular anesthesia, results of intrabursal anesthesia, or clinician suspicion of injury to a specific structure. Lesions of shoulder joint structures are difficult to localize on the basis of physical examination, and routine imaging of all structures allows the ultrasonographer to become familiar with normal appearance and increases the likelihood of recognizing abnormalities in affected horses. Evaluation of the contralateral limb for comparison can also be helpful in differentiating normal from abnormal findings.

Ultrasound-guided procedures, both diagnostic and therapeutic, were important in the diagnosis and treatment of 2 of the 3 horses in this report. In our experience, ultrasound-guided procedures are best performed by a single person to maximize the hand-eye coordination necessary for successful needle placement. In horse 1, the shoulder joint was inadvertently entered during attempts to aspirate fluid from the infraspinatus bursa despite the fact that the ultrasonographer was guiding the needle that was being advanced by a second clinician. In such a scenario, the needle can easily be advanced outside the ultrasound beam and into deeper structures without the ultrasonographer's knowledge. Biopsy guides were not used in the horses of this report; in our experience, biopsy guides can assist with needle placement but do not guarantee needle visibility on the ultrasound monitor and may restrict redirection of the needle upon movement of the horse.

Treatment of any infected synovial structure is aimed at eliminating the causative agent and removing the inflammatory mediators.[27–29] Long-term antimicrobial treatment was used in all 3 of the horses in the present report because of the direct communication of wounds with bone or synovial structures. Bone and synovial infections that develop in association with a penetrating traumatic wound are most often polymicrobial.[30] All horses were initially treated IV with broad-spectrum antimicrobials and were switched to oral formulations several days prior to discharge so that clinical response could be assessed while the horse remained in the hospital. Continued antimicrobial treatment after discharge was considered to be necessary in all horses, and oral formulations were chosen for ease of administration. Orally administered enrofloxacin was chosen in horse 1 on the basis of microbial susceptibility results. Chloramphenicol was chosen in horses 2 and 3 because of its broad spectrum of activity, its relative affordability, and the susceptibility patterns of common musculoskeletal wound contaminants.[31] Neither antimicrobial is commonly associated with the development of colitis at the authors' institution, compared with less expensive oral formulations such as trimethoprim sulfa. Trimethoprim sulfa was not considered for use in any of the horses because of its narrow spectrum against bone and synovial pathogens.[31] Chloramphenicol use is limited to horses with serious and potentially life-threatening infections in the authors' hospital when an oral formulation is required. Owners and staff are thoroughly counseled in the safe handling of this drug to avoid potential human health concerns.[32]

Antimicrobial treatment should be used in conjunction with through-and-through lavage to improve the clinical outcome of synovial infections.[27,28] The standing lavage procedures used in horse 1 were easy to perform with ultrasound guidance and yielded excellent flow of fluid from the egress portals placed caudal and cranial to the infraspinatus tendon. Although initial results were promising, the improvement in lameness and cytologic aspects of the infraspinatus bursa fluid were less than optimal, and endoscopy of the infraspinatus bursa was ultimately required for successful treatment. The advantages of endoscopy in the treatment of infected synovial structures include visualization of the synovial structure and high-volume targeted lavage, which facilitate removal of fibrin and other inflammatory debris.[29] Endoscopic imaging also enabled confirmation of the destructive lesion of the caudal eminence of the GT, but did not allow for additional debridement of that lesion because of the close proximity of the overlying infraspinatus tendon. Exposure and debridement of the abnormal bone could have been attempted with open tenotomy of the infraspinatus tendon but would have resulted in open bursotomy and potential lateral instability of the joint. The invasiveness of that procedure, challenges of postoperative management of open bursotomy wounds, and exposure to environmental contaminants were considered to outweigh the potential benefits. Endoscopy of the bursa was not performed in horse 2 because of severe overlying tissue damage and infection or in horse 3 because of the favorable clinical response observed after the standing lavage procedures.

In summary, infraspinatus bursitis and infraspinatus tendonitis should be included in the differential diagnoses of shoulder lameness in horses. The absence of radiographic findings does not rule out a fracture, particularly involving the caudal eminence of the GT. Ultrasonographic examination of the shoulder is essential for diagnosis and treatment of infraspinatus disorders and is also useful for identifying abnormalities of the caudal eminence. The infraspinatus tendonitis was considered to have developed secondary to fractures of the caudal eminence in 2 of the 3 horses and to the destructive lesion of the caudal eminence that developed during the course of treatment in the third horse. Infraspinatus bursitis seems to be more commonly associated with penetrating injuries, as is reported for bicipital bursitis. High-volume endoscopic lavage of the infraspinatus bursa should be considered in horses with septic or contaminated bursitis or in those that do not respond adequately to standing lavage procedures. Endoscopy may also reveal or confirm bony lesions that are not visible radiographically. Results suggest that injuries to the infraspinatus tendon and caudal eminence of the GT have good prognosis with adequate treatment.

a. Lameness grading scale 1–5, American Association of Equine Practitioners, Lexington, Ky.
b. Vingmed System 5, GE Medical Systems, Milwaukee, Wis.
c. Ausonics Impact, Universal Ultrasound, Bedford Hills, NY.
d. Jorgensen Laboratories, Loveland, Colo.
e. Ioban, 3M Health Care, Saint Paul, Minn.
f. Baytril, Bayer Corp/HAVER Animal Health, Kansas City, Mo.

References

1. Dyce KM, Sack WO, Wensing CJG. The forelimb of the horse. In: Dyce KM, Sack WO, Wensing CJG, eds. *Textbook of veterinary anatomy*. 2nd ed. Philadelphia: WB Saunders Co, 1996;575–578.
2. Tnibar MA, Auer JA, Bakkali S. Ultrasonography of the equine shoulder: technique and normal appearance. *Vet Radiol Ultrasound* 1999;40:44–57.
3. Stashak TS. Lameness. In: Stashak TS, ed. *Adams' lameness in horses.* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2002;910.
4. Bohn A, Papageorges M, Grant BD. Ultrasonographic evaluation and surgical treatment of humeral osteitis and bicipital tenosynovitis in a horse. *J Am Vet Med Assoc* 1992;201:305–306.
5. Pugh CR, Johnson PJ, Crawley G, et al. Ultrasonography of the equine bicipital tendon region: a case history report and review of anatomy. *Vet Radiol Ultrasound* 1994;35:183–188.
6. Vatistas NJ, Pascoe JR, Wright IM, et al. Infection of the intertubercular bursa in horses: four cases (1978–1991). *J Am Vet Med Assoc* 1996;208:1434–1437.
7. Gillis CL, Vatistas NJ. Biceps brachii tendinitis and bicipital (intertubercular) bursitis. In: Robinson NE, ed. *Current therapy in equine medicine 4.* Philadelphia: WB Saunders Co, 1997;12–14.
8. Reef VB. Musculoskeletal ultrasonography. In: Reef VB, ed. *Equine diagnostic ultrasound.* Philadelphia: WB Saunders Co, 1998; 39–186.
9. Reimer JM. Synovial structures. In: Reimer JM, ed. *Atlas of equine ultrasonography.* St Louis: CV Mosby Co, 1998;88–90.
10. Gough MR, McDiarmid AM. Septic intertuberal (bicipital) bursitis in a horse. *Equine Vet Educ* 1998;10:66–69.
11. Tudor RA, Bowman KF, Redding RW, et al. Endoscopic treatment of suspected infectious intertubercular bursitis in a horse. *J Am Vet Med Assoc* 1998;213:1584–1585.
12. Hawe C, McDiarmid AM. Tendonitis of the biceps brachii and intertubercular (bicipital) bursitis in a Thoroughbred racehorse. *Equine Vet Educ* 1999;11:60–62.
13. Fugaro MN, Adams SB. Biceps brachii tenotomy or tenectomy for the treatment of bicipital bursitis, tendonitis and humeral osteitis in 3 horses. *J Am Vet Med Assoc* 2002;220:1508–1511.
14. Adams MN, Turner TA. Endoscopy of the intertubercular bursa in horses. *J Am Vet Med Assoc* 1999;214:221–225.
15. Dyson S. Interpreting radiographs 7: radiology of the equine shoulder and elbow. *Equine Vet J* 1986;18:352–361.
16. Adams SB, Blevins WE. Shoulder lameness in horses—part I. *Compend Contin Educ Pract Vet* 1989;11:64–70.
17. Butler JA, Colles CM, Dyson SJ, et al. The shoulder, humerus and elbow. In: Butler JA, Colles CM, Dyson SJ, et al, eds. *Clinical radiology of the horse*. 2nd ed. Oxford, England: Blackwell Science Ltd, 2000;205–228.
18. Allen D, White NA. Chip fracture of the greater tubercle of a horse. *Compend Contin Educ Pract Vet* 1984;6:S39–S41.
19. Dyson S. Sixteen fractures of the shoulder region in the horse. *Equine Vet J* 1985;17:104–110.
20. Yovich JV, Aanes WA. Fracture of the greater tubercle of the humerus in a filly. *J Am Vet Med Assoc* 1985;187:74–75.
21. Adams R, Turner TA. Internal fixation of a greater tubercle fracture in an adolescent horse: a case report. *Equine Vet Sci* 1987; 7:174–176.
22. Adams SB, Blevins WE. Shoulder lameness in horses—part II. *Compend Contin Educ Pract Vet* 1989;11:190–196.
23. Crabill MR, Chaffin MK, Schmitz DG. Ultrasonographic morphology of the bicipital tendon and bursa in clinically normal Quarter Horses. *Am J Vet Res* 1995;56:5–10.
24. Cauvin ERJ. Soft tissue injuries in the shoulder region: a systematic approach to differential diagnosis. *Equine Vet Educ* 1998; 10:70–74.
25. McDiarmid AM. The equine bicipital apparatus—review of anatomy, function, diagnostic investigative techniques and clinical conditions. *Equine Vet Educ* 1999;11:63–68.
26. Whitcomb MB. How to perform a complete ultrasound exam of the equine shoulder, in *Proceedings.* 49th Annu Conv Am Assoc Equine Pract 2003;49:42–49.
27. McIlwraith CW. Treatment of infectious arthritis. *Vet Clin North Am Large Anim Pract* 1983;5:363–379.
28. Spurlock GH. The management of open joint injuries. *Vet Clin North Am Large Anim Pract* 1989;5:563–573.
29. Wright IM, Smith MRW, Humphrey DJ, et al. Endoscopic surgery in the treatment of contaminated and infected synovial cavities. *Equine Vet J* 2003;35:613–619.
30. Schneider RK, Bramlage LR, Moore RM, et al. A retrospective study of 192 horses affected with septic arthritis/tenosynovitis. *Equine Vet J* 1992;24:436–442.
31. Moore RM, Schneider RK, Kowalski J, et al. Antimicrobial susceptibility of bacterial isolates from 233 horses with musculoskeletal infection during 1979–1989. *Equine Vet J* 1992;24: 450–456.
32. Page SW. Chloramphenicol 1. Hazards of use and the current regulatory environment. *Aust Vet J* 1991;68:1–2.