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Case Report

Supracoronary approach for keratoma removal in horses: Two cases

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Summary

Two competitive horses were presented for examination of chronic lameness; one associated with a hoof-wall deformity, the other with a firm mass over the dorsal pastern region. Radiographs revealed moderately radiopaque masses associated with both deformities. The lesions were characterised ultrasonographically as noninvasive, well-circumscribed heterogeneous masses. Computed tomographic examination of the second case revealed a well-defined, partially mineralised, bi-lobed mass with associated bony resorption of the underlying middle phalanx. Both patients were anaesthetised and the keratomas surgically removed via approaches 1–2 cm proximal to the coronary bands. Both horses were stall-sound 2 days after surgery and returned successfully to an equal level of competition by 8 months. A supracoronary approach is a viable alternative to partial or complete hoof wall resection for the removal of nonsolar keratomas from the foot of a horse.

Introduction

Differential diagnoses for soft tissue masses of the distal limb include neoplasia such as fibroma, squamous cell carcinoma (Berry *et al*. 1991) and malignant melanoma (Kunze *et al*. 1986). Given the rarity of neoplasia in this area (Lloyd *et al*. 1988; Chan and Munroe 1997; Valentine *et al*. 2000), hoof wall keratoma must be considered. Keratomas can present as masses with or without hoof wall deformation (Chan and Munroe 1997; O'Grady and Horne 2001), associated focal lysis of the distal phalanx (P3), or concurrent solar infection and lameness (O'Grady and Horne 2001). They typically originate just distal to the coronary band and extend toward the toe (Chan and Munroe 1997). The masses in this report were located proximal to the coronary band, leaving doubt as to their origin and pathophysiology. Surgical excision is the treatment of choice for each of the differentials and is preferred to conservative management in the case of a keratoma (Bosch *et al*. 2004).

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Case 1

An 18-year-old Thoroughbred gelding used for novice level eventing was referred to the George D. Widener Hospital at the University of Pennsylvania, New Bolton Center in June 2006 with a 2 year history of a mass on the dorsolateral aspect of the right hind foot. The mass had been noticed in early 2004 coincident with moderate right hindlimb lameness. Treatment with rest, nonsteroidal anti-inflammatory drugs and later, a 14 day course of trimethoprim/sulphamethoxazole (25 mg/kg bwt *per os* q. 12 h), had been unsuccessful. After having received antimicrobial treatment the lameness had dissipated but the mass had remained unchanged. Successive episodes of lameness were increasingly severe.

A 3 × 4 cm mass at the dorsolateral aspect of the right hind coronary band caused deformation of the hoof from the coronet to the tip of the toe. The horse was 3/5 lame in the right hindlimb at a trot in a straight line (Ross and Dyson 2002). Lameness was abated by 80% with perineural analgesia of the plantar digital nerves at the base of the proximal sesamoid bones. Low plantar digital local anaesthesia at the level of the heel bulbs did not change the lameness. Radiographs revealed a well-circumscribed, heterogeneous, radiopaque mass in the soft tissues dorsal to the distal interphalangeal (DIP) joint and lateral to the extensor process of P3 (**Fig 1**). No deformation, invasion or lysis of P3 was noted, nor was there any evidence of a sclerotic rim of bone adjacent to the mass.

The mass was ultrasonographically characterised as lamellar with heterogeneous echotexture and measured 2.8 cm wide, 3.6 cm long and 1 cm deep. The collateral cartilages, long digital extensor tendon and the collateral ligaments of the DIP joint were ultrasonographically normal. Differential diagnoses included hoof wall

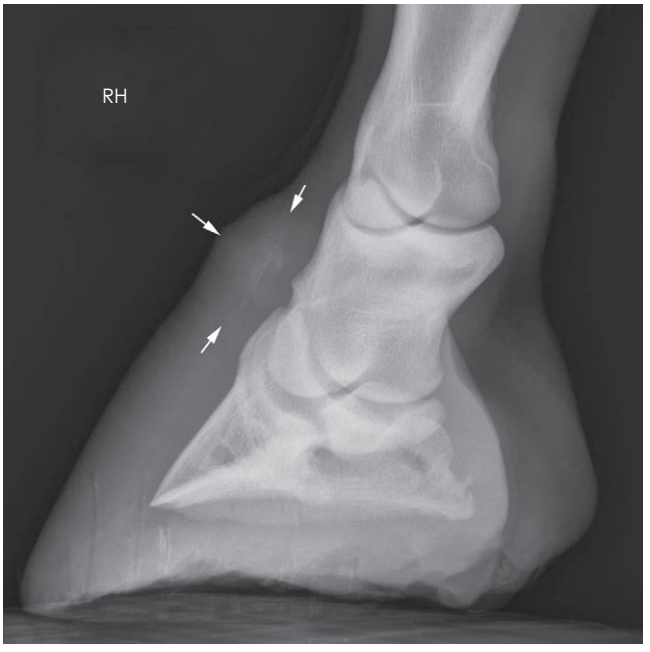

Fig 1:* Case 1: *Dorsomedial plantarolateral radiograph of the right hind foot; mass indicated by arrows.

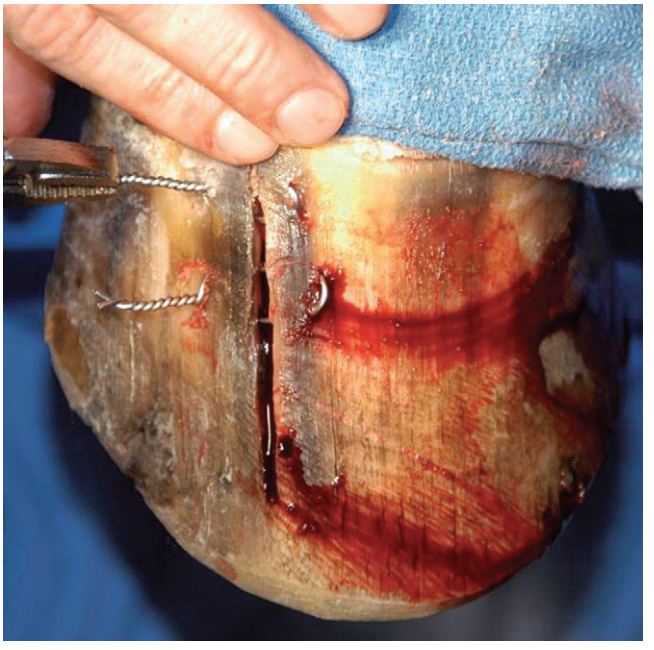

Fig 2:* Case 1: *Placement of stainless steel hoof-wall sutures. The defect is for drainage only.

keratoma, calcified haematoma, fibroma, squamous cell carcinoma and malignant melanoma (Sundberg *et al*. 1977; Berry *et al*. 1991; Seahorn *et al*. 1992; Valentine *et al*. 2000; O'Grady and Horne 2001).

The horse was placed under general anaesthesia in left lateral recumbency and an Esmarch tourniquet was applied to the proximal third of the metatarsus. The right hindlimb was clipped and prepared pursuant to standard aseptic technique, from the proximal metatarsus to the foot. The foot itself was thoroughly cleaned and then scrubbed with chlorhexidine. An iodine povacrylex/isopropyl alcohol solution (DuraPrep)[1] was then applied and allowed to dry. Perineural analgesia was performed at the level of the base of the lateral proximal sesamoid bone. The surgical site and hoof were covered with an iodophor impregnated antimicrobial incise sheet (Ioban)[1]. A proximally convex crescent-shaped skin incision was made over the dorsolateral aspect of the middle phalanx (P2) 2 cm proximal to the coronary band. The body of the mass was isolated with periosteal elevators. A vascular pedicle originating from the sensitive lamina of the hoof wall was double-ligated and transected. The mass was placed in 10% neutral-buffered formalin and submitted for histopathological analysis. The subcutis and skin were closed in 2 layers.

Removal of the mass left a large amount of dead space under the bulging hoof wall defect. To allow distal drainage, a 4 cm long, 3 mm wide full-thickness vertical groove was burred through the apex of hoof wall deformity using a high-speed rotary tool (Dremel High Speed Cutter)[2]. The cavity was packed with a collagen sponge (Ultrafoam Collagen Sponge)[3] soaked in amikacin (1.5 g; Amiglyde-V)[4]. The disruption of hoof wall integrity and resultant instability was countered by placement of 1.25 mm steel-wire suture across the groove in a shoe-lace pattern (**Fig 2**) and application of a 2-layer reinforced dorsal hoof wall covering. The sutures were not tightened enough to close the gap or occlude drainage, but were secure enough to decrease shearing forces on the hoof wall. A urethane-soaked (Equi-Thane Adhere)[5] linear carbon fibre fabric patch[6] was bonded to the hoof wall with its fibres running perpendicular to the defect. A piece of stiff plastic tubing was placed in the distal-most aspect of the gap, to be pulled before the adhesive hardened, in order to maintain a distal opening for drainage. Kevlar/carbon-fibre meshwork (Cobra Sox)[6] saturated with Equithane was placed over the entire repair to provide support in torsion and compression and protection from dorsal impact. The foot was left bare of any type of shoe, supportive device or cast, and was placed in a bandage with a duct tape covering. The limb was then placed in a modified Robert-Jones-style bandage extending from the ground (including the foot) to the tarsus.

The gelding recovered from general anaesthesia without incident. Stall confinement was initiated, phenylbutazone (3 mg/kg bwt *per os* q. 12 h) was administered and daily bandage changes were performed. Although moderately painful in the immediate post operative period, by 48 h the gelding was ambulatory with a greatly improved comfort level as judged by decreased heart rate, increased voluntary motion and weight borne on the affected limb. Bandage changes revealed minimal bleeding and adequate drainage. The gelding was discharged 5 days after surgery with instructions for strict stall rest, a tapering course of oral phenylbutazone and bandage changes every 3 days. One

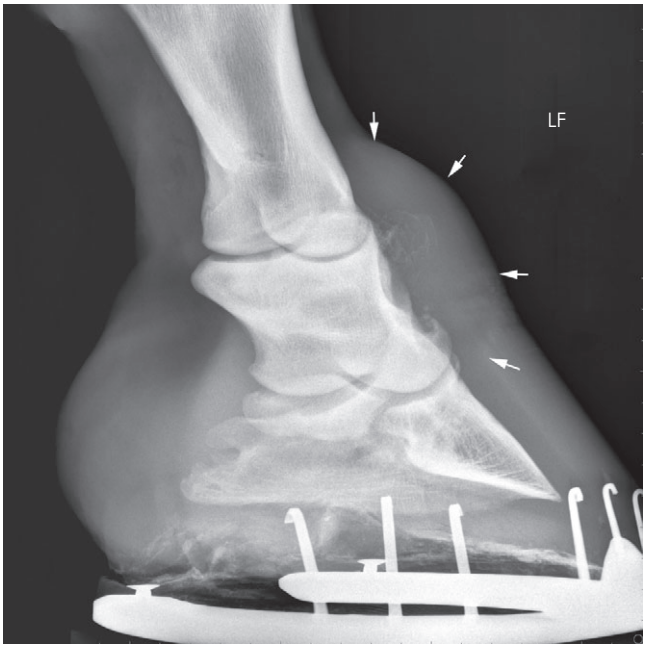

Fig 3: Case 2: Dorsomedial palmarolateral radiograph of the left front pastern; mass indicated by arrows.

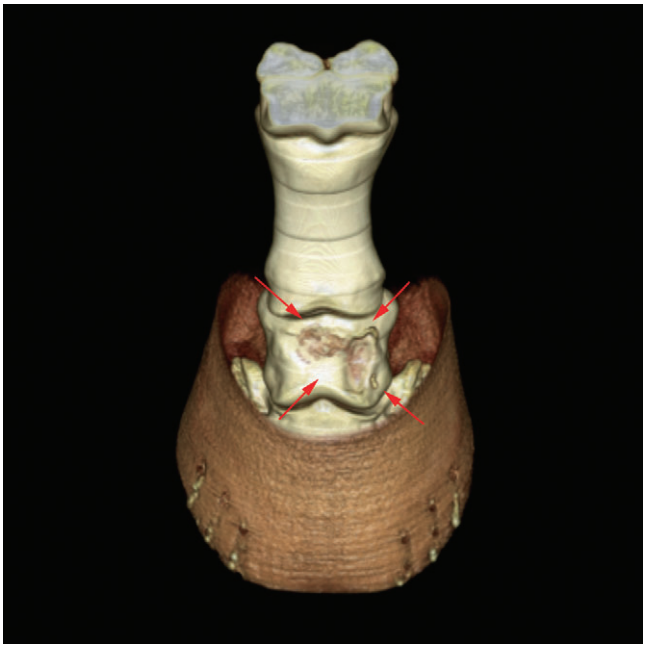

Fig 4: Case 2: Computed tomographic reconstruction; loss of bone from the dorsal cortex of the middle phalanx is noted.

month later the hoof patch was reset and a glue-on shoe (Sigafoos Series I)[7] was applied. The gelding was sound at this time. Over the next year, the coronary band returned to its normal conformation and the hoof wall defect was pushed down and out of the hoof by healthy new growth. The gelding was gradually returned to work and was competing successfully at the same level 8 months after surgery. There has been no keratoma recurrence.

Case 2

A 12-year-old Hanoverian gelding was referred to the George D. Widener Hospital at the University of Pennsylvania, New Bolton Center with a history of persistent left forelimb lameness. Treatment with corticosteroid injections of the corresponding distal and proximal interphalangeal joints and multiple i.m. injections of polysulphated glycosaminoglycans had failed. The gelding had recently developed a bulge on the dorsal aspect of the left front pastern. Magnetic resonance imaging of the foot and pastern region had been performed at another referral centre 3 weeks prior to presentation. The radiologist's report had described a mass located just dorsal to P2 and P3 and had diagnosed villonodular synovitis.

At presentation the gelding displayed *grade 2/5* left front limb lameness and had a firm, nonpainful mass on the dorsal aspect of the left front pastern. The owner wanted the mass removed and elected not to have a detailed lameness examination performed. Radiographs showed soft tissue thickening over the dorsal proximal aspect of P2 (**Fig 3**). Organised mineralisation was visible within the thickened area. There was well-marginated lysis in the dorsolateral portion of the diaphysis of P2 and proximal irregular periosteal reaction. Primary differential diagnoses for the mass included soft tissue neoplasia, resolving haematoma, granuloma and keratoma.

The gelding was placed under general anaesthesia in right lateral recumbency. Computed tomography was performed using an 8 slice mobile CT scanner (EQUS I)[8] at 120 kVp and 1.25 mm slice thickness (**Fig 4**). A well-defined, partially mineralised bi-lobed mass was seen overlying the dorsolateral aspect of the mid-diaphysis of P2. The mass was deep to the common digital extensor tendon, noninvasive and measured 4.5 cm wide by 2.3 cm deep by 4.5 cm long.

Presurgical preparation of the limb was performed as described in *Case 1*. An inverted 'T' shaped skin incision was made directly over the mass. The vertical incision extended from mid-pastern to 1 cm proximal to the coronary band and the horizontal incision was 7 cm long. The common digital extensor tendon was transected in an inverted 'V' just distal to the insertion of the extensor branches of the suspensory ligament and retracted distally. The mass was excised as described in *Case 1* and submitted for histopathological analysis. The extensor tendon was reapposed using No.1 polyglactin 910 (Vicryl)[9] in a cruciate pattern. The skin was closed in 2 layers and the distal limb was placed in a fibreglass cast.

The gelding recovered from general anesthesia without incident and was sound at the walk immediately after surgery. Stall confinement was initiated and both phenylbutazone (3 mg/kg bwt *per os* q. 12 h for 5 days) and procaine penicillin (22,000 iu/kg bwt i.m. q. 12 h for 3 treatments) were administered. The gelding was discharged 5 days later with instructions for stall rest and a

tapering course of oral phenylbutazone. Four weeks later the cast was removed and the animal was sound at a walk. Gradual reintroduction to work was commenced after 2 months of rest. The gelding was sound competing successfully as an upper-level dressage horse one year after surgery. There has been no keratoma recurrence.

The masses were grossly characterised as disorganised with inwardly-folded lamellar structure. They ranged from tan-yellow on the periphery to dark brown in the centre. Histopathologically, the masses were composed of large amounts of lamellar keratin devoid of viable cellularity and were diagnosed keratomas.

Discussion

Keratoma is a rare condition of the equine hoof (Sundberg *et al*. 1977; Lloyd *et al*. 1988; Reeves *et al*. 1989; Berry *et al*. 1991). In a review by Sundberg *et al*. (1977), keratomas accounted for 1.3% of 236 neoplasms diagnosed from 1322 biopsies or necropsies over a 5 year period. Keratomas typically originate from the keratinocyte-producing *stratum basale* of the coronary band (Reeves *et al*. 1989). They are also reported to arise from both the inner cornified layer of the hoof wall and rarely from the sole itself (O'Grady and Horne 2001). They grow between the hoof wall's inner stratum and P3 (Lloyd *et al*. 1988; O'Grady and Horne 2001). They are usually solitary masses comprised of characteristic concentric sheets of squamous epithelial cells, abundant keratin and few viable cells (Lloyd *et al*. 1988). They can be oblong or spherical (O'Grady and Horne 2001), appear tan/yellow to brown on cross-section (Sundberg *et al*. 1977; Lloyd *et al*. 1988; Reeves *et al*. 1989), and are often associated with local infection (Lloyd *et al*. 1988; Boys Smith *et al*. 2006). Although their aetiology remains obscure (O'Grady and Horne 2001), they are thought to arise from trauma to the hoof wall (Lloyd *et al*. 1988) or chronic irritation of the inner laminae (Reeves *et al*. 1989). Despite being located predominantly proximal to the foot, both of the masses described had vascular or filamentous connections to the sensitive lamellar tissues of the foot and thus do not necessarily challenge proposed aetiologies.

Keratomas commonly cause lameness when present (Lloyd *et al*. 1988; Valentine *et al*. 2000). Lameness manifests only when they become large enough to impinge on the laminar dermis of P3 or are associated with acute subsolar infection (Seahorn *et al*. 1992). Although a keratoma is not typically visible radiographically unless mineralised, its presence can be suspected with a radiolucent defect in P3 (Lloyd *et al*. 1988). The defect occurs due to pressure necrosis as the mass grows in a finite space. This radiolucency is distinguished from osteomyelitis by its distinct margination, lack of sclerotic rim and history of gradual onset of lameness. Computed tomographic imaging can show underlying bony reaction but is not necessary for diagnosis.

The treatment of choice for keratomas is surgical excision via complete or partial hoof wall resection (Boys Smith *et al*. 2006). Bosch *et al*. (2004) reported a return to equal or higher level of performance in 83% of patients managed surgically, as opposed to 42% of those managed conservatively. A study by Boys Smith *et al*. (2006) compared complete and partial hoof wall resection techniques for keratoma removal, and found fewer surgical complications and a more rapid return to athletic activity with the latter technique. Post operative complications of significance include loss of hoof wall stability, excessive formation of granulation tissue and recurrence (Bosch *et al*. 2004; Boys Smith *et al*. 2006). Partial resection of the hoof wall is preferred as it limits post operative morbidity, iatrogenic instability and thus post operative lameness.

The cases reported here are unusual due to the locations of the keratomas and their atypical radiographic characteristics. Keratomas commonly manifest as lameness of the affected foot, and presurgical diagnosis is often predicated on radiographic presence of the classic radiolucent halo of bony resorption keratomas can cause. These conditions are not always pre-existing and were not noted to be a feature of the disease in the cases reported here. The masses described in this report had mineralised density on radiographs and no associated halo of bony resorption (*Case 1*). Although keratoma-induced bony resorption was present in *Case 2*, it was located on P2, which, to the authors' knowledge, has not been reported previously. Both masses were histopathologically identified as keratomas, proving that this lesion can be located above the hoof wall. In addition, keratoma must be a differential diagnosis for a lameness-causing mass of the foot or pastern region, even without the presence of radiographic abnormalities of P3.

The successful resolution of these cases documents the possibility of an extramural approach to keratoma excision. It offers the distinct advantage of maximising post operative hoof wall integrity, minimising the convalescent period and decreasing the time to return to work. The post operative morbidity, especially protracted lameness, classically associated with hoof wall resection is avoided. Immediate cosmesis is also improved. Although chosen in these cases because of how far proximal the masses were located, it is possible that this approach could be used for any hoof wall keratoma.

Acknowledgements

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Manufacturers' addresses

[1]3M, St. Paul, Minnesota, USA.
[2]Dremel, Racine, Wisconsin, USA.
[3]Davol Inc., Warwick, Rhode Island, USA.
[4]Fort Dodge Animal Health, Overland Park, Kansas, USA.

[5]Vettec, Oxnard, California, USA.
[6]Atkins & Pearce, Covington, Kentucky, USA.
[7]SoundHorse Technologies, Unionville, Pennsylvania, USA.
[8]Universal Medical Systems Inc., Solon, Ohio, USA.
[9]Ethicon, Somerville, New Jersey, USA.

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