Maggots on Board

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Class of 2020

Clinicopathological Conference

July 19, 2019

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Introduction

Pedal osteitis is separated into two forms: septic and non-septic. These forms reference whether the third phalanx (P3) is infected.[8] Regardless of the inciting cause, both septic and non-septic processes result in altered circulation to the affected area of P3.[14] Numerous insults to the hoof, underlying soft tissues, and the bone predispose horses to either form of pedal osteitis. The most common inciting cause in adults is direct introduction of infectious agents via puncture wounds.[14] Other predisposing or inciting events include: contusions, chronic subsolar bruising, thrombi, solar abscesses, fractures of P3, flexor deformities, and keratomas.[8,10,14] In foals the septic form dominates as the most common presentation due to hematogenous origin.[13]

To differentiate between forms and other differential diagnoses, gross, radiographical, advanced imaging, and even surgical evaluation may be necessary. [9, 5, 11,12] This is due to the fact that there are many differentials that may cause a severe lameness that localizes to the foot, and that to be classified as septic, P3 must be infected.[8]

Septic pedal osteitis is radiographically characterized by osteolysis and demineralization of P3 due to infection of the bone and can be contributed to by infection of the surrounding tissues.[9] Sequestration of type VI fractures of the solar margin is a common sequela to the septic form due to devascularization and dissecting purulent tracts.[14] Septic pedal osteitis often warrants surgical intervention and intense management post operatively, as often these are chronic cases with high loads of mixed or pure cultures of environmental pathogens.[9,2]

History and Presentation

Rio is a 23-year-old, Peruvian Paso gelding, who was used for light trail rides. He presented to the MSU-CVM equine surgery department for severe lameness of the right thoracic limb. Previous to Rio's presentation, he was being boarded for an extended period of time while

his owners were having his pasture maintained. The owner had no knowledge of his signs, until she found him severely lame (4+/5) and underweight on January 2, 2019. On January 7th Rio presented to his referring veterinarian where an abscess was pared out the sole of his right front foot. The abscess extended from the medial heel bulb to the center of the toe. On January 8th, the abscess produced a draining tract that ruptured through the medial coronary band. Rio's referring veterinarian treated him with: phenylbutazone, flunixin meglumine, misoprostal, and daily ab-axial sesamoid blocks for comfort. He showed no improvement and was referred to the MSU-CVM equine surgery service on January 19th, 2019.

Upon presentation Rio was quiet, alert, and responsive. He weighed 757lb with a body condition score of 3/9. His vital perimeters were as follows: temperature of 99.6 F, heart rate of 60 beats per minute, and respiratory rate of 28 breaths per minute. His heart rate was mildly increased likely due to stress and discomfort. His mucous membranes were pink, slightly tacky, with a capillary refill time of less than 2 seconds. No murmurs or arrhythmias were auscultated and his lung sounds were normal. Borborygmi was decreased in all four quadrants. Digital pulses were normal in both of his pelvic limbs and increased in both thoracic limbs. Rio displayed a severe 4+/5 lameness of the right thoracic limb. The medial aspect of his right front coronary band was swollen and warm. The defect on the sole where the abscess had been pared out, extended from the toe to the medial heel bulb. Application of hoof tester revealed pain at the toe, lateral wall, and medial wall.

Pathophysiology

Both septic and non-septic pedal osteitis have a basis in the disruption of vascular supply to the third phalanx.[14] Inciting causes can be anything that alters the mechanical loading of P3 or

the pressure within the hoof capsule such as: puncture wounds, contusions, chronic subsolar bruising, thrombi, solar abscesses, fractures, flexor deformities, and keratomas.[3,10,14]

The inciting cause in non-septic pedal osteitis induces hyperemia and localized pressure which causes inflammation. The altered blood flow and inflammation leads to swelling. This increases the pressure within the capsule of the hoof which further impedes venous drainage.[14] Persistent pressure and inflammation causes lysis of P3, especially around the vascular canals and solar margin.[14] These sites are most commonly affected due to the anatomy of the vasculature and the fact that the periosteal surface and solar margin experience the most direct pressure.[1] The periosteal and solar margin depend on periosteal branches of the dorsal branch of the palmar digital artery, the terminal arterial arch, and the circumflex artery of the sole, while the deeper portions of the third phalanx receive circulation from endosteal and medullary vessels.[1] Due to the third phalanx's unique vasculature, damage to the bone occurs most frequently in the outer surface and rarely effects deeper layers.[1]

In septic pedal osteitis all previous statements about the pathogenesis of non-septic pedal osteitis still play a role, however P3 becomes infected. The most common way micro-organisms are introduced is through a direct puncture wound that terminates in the bone, this contributes to the septic form commonly presenting with a draining tract that originates in the bone.[14] Bony lysis is more severe in the infected area due to high inflammation, infection, and pressure which often leads to focal complete lysis, pathologic type VI fractures of the solar margin, and subsequent sequestra due to devitalization and dissecting purulent debris.[14] These sequestra become niduses to perpetuate the disease process because they lack vascular supply.[15]

There are some specific considerations when laminitis is the predisposing factor. Laminitis causes a disruption of the function of the laminae which suspend the third phalanx

within the hoof capsule. This disruption may cause a rotational drop, or a collapse which may either be symmetrical or asymmetrical.[10] The drop of P3 causes an increase in pressure on the subsolar structures and an increase in the weightbearing responsibility of the remaining functional lamina.[10] The increase in pressure on subsolar structures predisposes to bruising and disruption of vascular supply.[10] Rupture of laminae results in hemorrhage and seroma formation under the wall and sole; collapse of P3 and increased pressure results in shearing and disruption of vascular supply.[10] The pooled blood and compromised vascular supply makes the laminitic hoof the perfect environment for microorganisms to grow once introduced.[14] Laminitis also plays a role in treatment and management of cases post operatively due to support limb laminitis.[7]

Diagnostic Approach and Considerations

The diagnostic approach to septic pedal osteitis is based mainly upon radiographic imaging and gross findings that exclude the non-septic form and other differential diagnoses. The initial physical exam is very important and should include a thorough evaluation of the affected foot. The vital perimeters will provide information regarding the patient's comfort or even the possibility of sepsis. The evaluation of the foot may reveal a draining tract, heat, swelling, or a puncture wound.[1,14] This disease will present as a lameness and although Rio was severely lame that is not always the case. Physical exam may not reveal the cause. In a study of 18 horses, at presentation horses ranged from lameness scores of 0.5 to 4.5 with a mean of 3.27.[1]

In horses with less obvious lameness a full evaluation may be needed to localize the lameness to the foot. The majority of horses presenting with this disease will be lame at the walk or trot.[1,6,] Once the affected limb is identified the methodical use of hoof testers is needed to produce a repeatable response in the suspect area of the foot. If a strong repeatable response is not seen the use of regional anesthetic blocks may be used to determine which structures may be

the source of pain.[6] Horses presenting with septic pedal osteitis should block to an abaxial sesamoid block.[8] Once the painful area is identified it should be evaluated again to ensure no puncture wounds, draining tracts, or other signs of inflammation were over looked. When evaluating the hoof grossly fully evaluate the coronary band and the solar surface as these are the most common places for draining tracts to terminate.[2]

Radiology is best pursued before pairing out any abscesses or draining tracts, because air artifacts will be present afterwards, though radiographs taken after pairing can still be of great diagnostic importance. [9] If the horse presents with a foreign penetrating object radiographs should be taken before the removal of the object to determine depth of penetration and the structures it could be penetrating.[9,15] Radiology is also sometimes used in conjunction with paring to determine depth of draining tracts.[1,9] The most useful radiographic views are lateromedial, dorsopalmar, and most importantly a dorso 60 degree proximal palmar distal oblique.[1,15,13] You want to be able to thoroughly evaluate the solar margin and vascular canals as these areas are those most affected.[14] Radiographs are useful in determining if there is pedal osteitis and whether it is septic or nonseptic, as pedal osteitis is defined radiographically by bony lysis of P3 around the solar margin and vascular canals; type VI fractures, and sequestration may also be apparent.[14,13,9] Radiographically the non-septic form will have much milder bony changes, predominately widened vascular canals and permeative lysis of the solar margin.[14] The septic form will have focal complete lysis in the affected area. Sequestration and fractures are much more commonly in the septic form.[13,9] Sequestra are radiographically defined by: distinct margins, sclerosis, and involucrum; however, sequestra may be very small and not apparent on radiographs.[1] In one study, distinctly marinated bone was identified on radiographs and

sequestrum was suspected, however when there was an absence of sclerosis and involucra, sequestration was confirmed with histopathology.[1]

If a subsolar process such as an abscess or seroma is noted, that portion of the sole should be paired out and all draining tracts should be explored. This is best facilitated with regional anesthetics such as lidocaine or bupivacaine in an abaxial sesamoid block.[1,2,9] Radiology can be used in conjunction with paring and grossly exploring draining tracts by using malleable radiopaque probes or contrast agents.[1,9] This allows some visualization as to where the tract is originating whether it be in the bone or soft tissue; however, it is important to remember that this may not be helpful as radiographs are 2D images of 3D structures. Using probes and contrast may help you rule out differentials such as: septic arthritis, septic tenosynovitis, and septic navicular bursitis.[9]

Advanced imaging modalities, such as contrast radiographs, computed tomography, magnetic resonance imaging, and venography, have also been utilized to determine whether the third phalanx is infected, extent of the bony lysis, and whether local synovial structures are involved.[5,9,11,12]

Rio's history at presentation was very suggestive of a chronic infectious process of the foot. A solar abscess was located and pared prior to his presentation by his farrier, with reports of a draining tract at the medial coronary band. His lameness was evident at the walk and he showed no improvement despite treatment of the abscess and medical pain management. Hoof testers revealed pain that localized to the medial toe and quarter of the right forefoot. Radiographic evaluation was then pursued. A lateral, dorsopalmar, navicular skyline, and dorso 45 and 60 degree proximal palmarodistal oblique were taken. The images were suggestive of predisposing laminitis and showed an irregular medial solar margin, severe focal bony lysis of

approximately 25% of P3, and severe soft tissue swelling centered at the coronary band. These images were highly suggestive of septic pedal osteitis and a commuted tomography scan was scheduled for the next morning with the intent of determining the extent of bony damage, which would immediately be followed by surgical resection of the medial hoof wall, debriding of the diseased soft tissue, and removal of diseased and sequestered bone.

Treatment and Management

Rio was placed under general anesthesia for the CT scan and surgery. He was placed in right lateral recumbency and an abaxial sesamoid block was performed on the right limb. A partial hoof wall resection was performed over the medial quarter using half-round nippers, and an abscess tract which was separating the sensitive and insensitive laminae was debrided with rongeurs and a curette. This tract originated in the bone, and the dorsal tissues were infected down to the bone. The affected bone was soft, friable, and appeared to be covered in granulation tissue. This infected bone was debrided until firm healthy bone remained. A hoof knife was used to debride the sole which revealed remnants of the solar abscess. Cultures of the infected bone were collected for culture and sensitivity. Another described method of debriding infected soft tissue and bone in the case of septic pedal osteitis is through trephination.[9] In cases of very focal less extensive cases, a trephine can be used to remove two to three portions of hoof wall or sole in which to debride through, through this method there is compromised visualization, and it is important to place one trephination in an area to allow ventral drainage.[9]

Post-operatively Rio was hospitalized for management of his right forefoot for approximately 2 months. The mainstays of his post-operative management included bandaging, continued debridement, antibiotic therapy, pain management, control of support limb laminitis, and controlled weight gain.

There are three options described for protection of the defect post operatively, a foot cast, a light water-proof bandage, and a hospital plate.[1,2,9] A light water-proof bandage was selected as it allows for easier access to the defect, and the ability to change the bandage frequently with less effort. This bandage method was modified several times thorough the course of Rio's treatment, as it allowed us to modify our topical treatments as needed. The bandage always consisted of gauzes to pack the defect, kerlix, vet wrap, and duct tape to protect it from moisture and elasticon around the top to keep stall shavings out. When bandaging the foot, special care was always taken to avoid pressure on the coronary band. Use of multiple topical antibiotics are described in the literature including: metronidazole, ampicillin, and benzylpenicillin.[1,13] Throughout Rio's time at the hospital this bandaging method was adapted with using multiple antimicrobial methods. Metronidazole tablets made into a paste were used first for two days after the initial bandage change when the tissue filling the defect began to look discolored and have a foul odor, after two days, the method was switched to using a diluted betadine solution on gauze to pack the wound, later clavamox powder was added onto the diluted betadine gauze, AMD antimicrobial sponges were later utilized for a few days, and once the defect had completely keratinized (36 days post-surgery) full strength betadine gauzes were utilized. Other antimicrobial treatments were given systemically and regionally.

Gentamycin (6.6mg/kg IV q24) and Procaine Penicillin G (2200 IU/kg IM q12) were used as initial systemic antibiotic therapies until the culture and sensitivity returned. Gentamycin was also used once a day in regional limb perfusion through the right cephalic vein with the use of a tourniquet 2 days post-operatively. The culture revealed a heavy mixed infection of *Proteus mirabilis*, *Streptococcus uberis*, *Streptococcus dysgalactia ssp equisimilis*, and an unidentified

anaerobic gram positive cocci. Systemic antimicrobials were then switched to Chloramphenicol (50mg/kg PO q6) which he received for 26 days.

Despite aggressive antimicrobial treatment the tissue filling the defect began to become discolored and foul smelling. It was decided to further debride the tissue with a curette, standing using abaxial sesamoid blocks, and order medical grade maggots. Medical grade maggots are not a very common therapy used in animal medicine but their use is increasing in popularity due to an increase in antimicrobial resistance.[3] Medical maggots most commonly *Phaenicia sericata* are raised and sterilized in a laboratory setting.[3] They debride necrotic tissue via three main mechanisms: secretion of proteolytic and digestive enzymes, ingesting and digesting microbes, and dissolving biofilms.[3] Medical grade maggots are stimulate healthy tissue growth. A special bandage, called a "maggot cage," utilizing women's pantyhose was fashioned to allow airflow to the maggots but keep them contained to the area of the defect and two days later they were removed, and healthy granulation tissue was left.

Rio was very painful for the first two to three of days post-operatively. Ketamine at 2.5 mL IV twice daily for two days was used to combat potential wind up pain, and Acepromazine at 10 mg/kg IM q8 for two days was used for its tranquilization effects. After the first two days Rio was maintained on Flunixin Meglumine at 2.2 mg/kg IV q12 until his catheter was pulled and then he was given the same dose orally. He received this dose for thirty days until he was backed off of it slowly. After 10 days of this course of NSAIDs, Ranitidine at 7mg/kg PO q8 was added as a preventative measure against gastrointestinal adverse reactions.

Throughout Rio's stay he was monitored for support limb laminitis, by having his digital pulses checked every 4 hours, and he received ice bracelets in limbs with bounding pulses. However, near the end of his stay he became lame on the left front foot due to a hematoma. It

was pared out and an epson salt soaker boot was utilitized and he recovered quickly. He also received corrective trimming and shoing throughout his stay and continues to be rechecked and reshod here regularly.

Case Outcome

Rio's right front foot is completely keratinized and working on growing the hoof wall back, this process takes months usually between 6 to 12, as new hoof wall growth moves down from the coronary band at a rate of 10mm per month.[2,9] He was still lame at his presentation on June 21st, 2019 (his most recent presentation) with a lameness score of 3/5 on the right. This lameness was mainly attributed to his right shoe becoming loose. The shoe was pulled and replaced, his lameness on then reduced significantly. His owner reported that his comfort level has improved tremendously on that foot since discharge. That day he was re-evaluated radiographically and was found to have a medial to lateral imbalance of the right hoof due to the resection defect on the medial side. This imbalance was corrected via corrective trimming and shoeing. There was some detached hoof wall on the right hoof, on the medial aspect and heel which was removed. The shoe was nailed in the lateral aspect of the hoof wall and glued on the medial, then the wall defect was filled with acrylic. He was also re-evaluated on the left front there was no evidence of pathology in the subsolar region, and the previously noted negative palmar angle was subjectively improved but still present. He was correctively trimmed on the left front and an area of white line disease was paired with a hoof knife, he was then reshod. He presented that day (June 21st) for a left hindlimb lameness which scored 4/5. A solar abscess in the medial sole was found paired out with a hoof knife and treated with Epson salts and a sugar betadine bandage, he was reshod with extended heels on his rear feet and discharged on June 29th. He is continuing to gain is body condition back and his owner is happy with his progress.

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