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Case report

Septic Osteitis and Osseous Sequestration of the Distal Phalanx Associated with a Coronary Band Lesion in a Draft Horse: Clinical, Radiographic and Surgical Findings

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Abstract: Inflammatory disorders of the distal phalanx (P3) associated with lesions of the coronary region of the hoof may involve reactive periosteal bone proliferation and focal osseous separation. This report describes a case of focal inflammatory osteal pathology of P3 in a draft horse presenting with severe forelimb lameness and a localized lesion of the coronary region. Radiographic examination revealed a longitudinal radiopaque formation with irregular margins on the dorsal aspect of P3, associated with a corresponding radiolucent area and a tract containing fluid and gas extending toward the coronary region. Surgical intervention was performed under general anesthesia. A progressive distal hoof wall resection was carried out with preservation of the coronary region, followed by evacuation of purulent material and curettage of the affected bone. A detached lamellar bone fragment consistent with focal osseous sequestration was removed, and debridement was continued until macroscopically viable bone was obtained. The excised tissues were submitted for histopathological evaluation. Hematoxylin–eosin-stained sections demonstrated lamellar bone with periosteal thickening and adjacent fibrous connective tissue showing hemorrhagic infiltration and a mixed inflammatory infiltrate composed predominantly of neutrophils and macrophages. No histological evidence of neoplastic proliferation was identified. The findings were consistent with periostitis and osteitis associated with reactive exophytic bone formation and focal osseous sequestration. This case highlights the diagnostic value of radiography and the importance of complete surgical debridement in inflammatory conditions of the distal phalanx associated with lesions of the coronary region.

Keywords: distal phalanx; equine; coronary region; osseous sequestration; reactive exostosis; periostitis; osteitis; hoof surgery; draft horse

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1. Introduction

Osseous sequestration is defined as the separation of a segment of necrotic bone from adjacent viable osseous tissue as a consequence of vascular compromise, septic inflammation, or direct traumatic insult [1]. Interruption of the intraosseous blood supply results in cortical necrosis, followed by progressive demarcation of the devitalized fragment from surrounding healthy bone. In the horse, sequestrum formation is a recognized sequela of septic and traumatic osteal pathology, and surgical removal of the necrotic fragment is frequently required to achieve definitive resolution [2].

Within the equine digit, the anatomical configuration of the hoof capsule—characterized by its rigid, non-distensible keratinized wall and the close apposition of the dermal lamellae to the distal phalanx (P3)—predisposes to propagation of infectious processes to the underlying bone when drainage is inadequate or delayed [3]. Subsolar abscessation and penetrating solar or coronary injuries may extend beyond the epidermal and dermal layers, leading to septic involvement of deeper structures, including the distal phalanx.

Septic conditions affecting the hoof complex may culminate in septic osteitis or osteomyelitis of P3, radiographically manifested by focal osteolysis, cortical irregularity,

and, in chronic cases, periosteal reaction and sequestrum formation [4]. The pathophysiological progression involves bacterial contamination, inflammatory osteolysis, compromise of local perfusion, and eventual necrosis of cortical bone.

Septic osteitis of the distal phalanx is a well-documented clinical entity in both adult horses and foals and is typically associated with severe weight-bearing lameness, radiographic evidence of bone lysis, and, in advanced stages, separation of necrotic osseous fragments [5]. When medical management and drainage are insufficient, surgical debridement and curettage of devitalized bone are considered essential to eliminate the septic focus and restore functional integrity of the digit.

The present report describes a case of focal septic osteal pathology of the distal phalanx associated with osseous sequestration in a mature draft horse, detailing the clinical presentation, radiographic findings, surgical management, and histopathological confirmation, and discussing the pathophysiological mechanisms implicated in sequestrum formation within the equine hoof.

2. Materials and Methods

A 6-year-old intact male draft horse (Greu de Bucovina type) was presented for evaluation of severe lameness of the left hindlimb. A complete general and orthopedic clinical examination was performed, and the degree of lameness was assessed using a standard lameness grading scale. The distal region of the affected limb was inspected and palpated, and the coronary region was clipped to allow direct evaluation of the skin and underlying structures.

Standard radiographic examination of the distal segment of the affected forelimb was performed using appropriate projections to assess the distal phalanx and adjacent structures.

The horse was placed under general anesthesia and positioned in lateral recumbency. A progressive dorsal hoof wall resection was performed in a distal direction while preserving the coronary region. The area corresponding to the radiographically identified lesion was approached through controlled removal of the hoof wall. After accessing the pathological focus, the contents were evacuated and curettage of the affected region of the distal phalanx was performed. Curettage was continued until all macroscopically altered tissue was removed, and intraoperative assessment of osseous vitality was based on the gross appearance and consistency of the bone.

At the end of the procedure, a topical cicatrizing ointment was applied locally, followed by placement of a compressive bandage. Postoperative management included periodic bandage changes.

The retrieved osseous fragment and associated soft tissue samples were submitted for histopathological examination. The specimens were processed using standard laboratory techniques, and representative serial sections were stained with hematoxylin and eosin for microscopic evaluation.

3. Results

3.1. General Clinical Examination

A 6-year-old intact male draft horse (Greu de Bucovina type), used for forestry traction work in mountainous terrain, was presented on March 14, 2025, for acute-onset lameness of the left hindlimb. According to the anamnesis, the lameness developed suddenly immediately after returning from timber extraction activities. No obvious wound had been identified initially at the hoof level, most likely due to the abundant hair covering the coronary region. Conservative treatment administered for approximately two weeks prior to referral (local cooling and topical anti-inflammatory therapy) resulted in no improvement and progressive worsening of clinical signs.

Orthopedic examination revealed severe lameness of the left hindlimb, graded 4.5–5/5 on the AAEP lameness scale. The horse demonstrated minimal weight-bearing on the affected limb and exhibited a marked pain response during locomotion, particularly when the limb was loaded during the stance phase. At rest, the animal frequently attempted to unload the limb by intermittently lifting the hoof or shifting weight to the contralateral hindlimb.

Following clipping of the hair at the distal portion of the limb to allow better visualization of the integument, a well-demarcated hyperemic area was identified at the dorsolateral aspect of the coronary band (Fig. 1a). The affected region presented mild, diffuse oedema and was markedly painful upon palpation, eliciting a clear withdrawal response. Local tissue temperature appeared mildly increased compared with adjacent areas, suggesting an active inflammatory process. No fluctuation suggestive of a mature abscess

cavity was detected on digital palpation, and careful inspection did not reveal spontaneous drainage, purulent exudate, or a visible fistulous tract. The coronary surface remained relatively dry and intact, with no obvious disruption of the epidermal continuity.

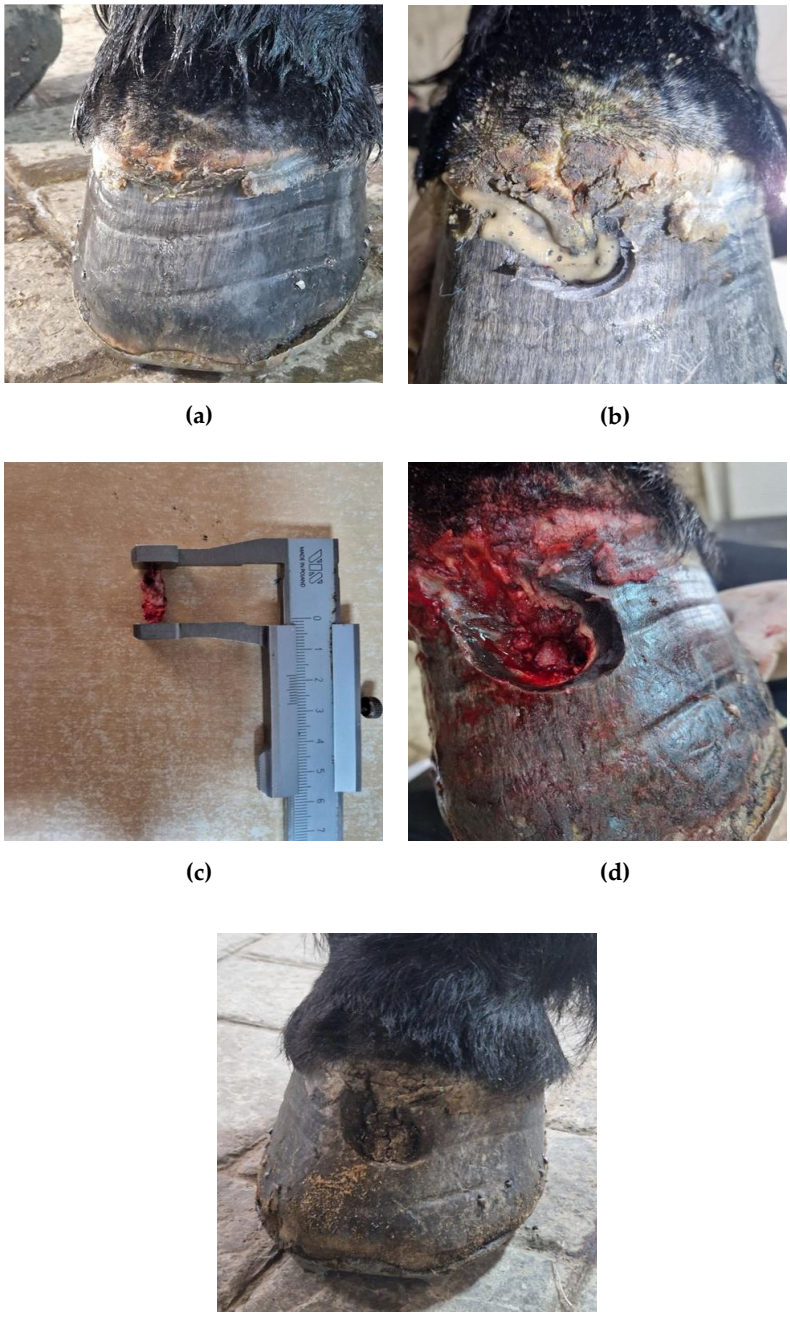


Figure 1. (**a)** Preoperative clinical aspect of the dorsolateral coronary band of the left hindlimb after clipping, showing localized hyperemia and mild oedema; **(b**) Intraoperative view of the subparietal cavity following dorsal hoof wall resection and evacuation of pressurized purulent material; (**c**) Macroscopic appearance of the extracted osseous fragment; (**d)** Dorsal cortical defect of the third phalanx after complete curettage, demonstrating viable bleeding bone; (**e**) Cicatrized lesion located adjacent to the radiographic site at 27 days post-procedure.

Gross inspection of the dorsal hoof wall did not reveal visible cracks, deformities, or structural defects. The hoof capsule maintained a normal conformation and there were no evident defects or discoloration that could immediately suggest an external penetration point or chronic wall pathology. Application of hoof

testers elicited a pronounced pain response localized to the dorsal aspect of the hoof capsule. The most intense reaction occurred in the region topographically corresponding to the coronary lesion observed during the clinical inspection (Fig. 1a). Compression in this area provoked a strong withdrawal reflex and behavioral signs of discomfort, indicating increased sensitivity of the underlying structures. In contrast, application of the hoof testers to the lateral quarters, heel region, and frog elicited either a mild or absent response. This localized pain pattern suggested the presence of a focal pathological process involving the dorsal hoof wall and/or the adjacent subsolar or subcoronary structures.

The clinical appearance of the cicatrized lesion observed at 27 days post-procedure indicates a favorable healing process. The formation of a stable scar adjacent to the radiographic site suggests that the local tissue response progressed normally, with resolution of the initial inflammatory signs and gradual restoration of tissue integrity. Such evolution is consistent with the expected healing pattern following surgical or therapeutic intervention in the affected region. The absence of visible complications, such as excessive swelling, discharge, or tissue necrosis, further supports the effectiveness of the applied treatment and the satisfactory recovery of the area.

3.2 Radiographic Examination

Radiographic examination of the left hindlimb was performed in a latero-medial projection, with careful positioning to optimize visualization of the third phalanx and its relationship to the dorsal hoof wall. The images revealed a distinct longitudinal radiopaque structure located on the dorsal surface of the third phalanx, extending proximodistally along the anterior cortical margin. This structure exhibited irregular, poorly defined margins and heterogeneous opacity, features suggestive of reactive or devitalized osseous tissue rather than organized periosteal proliferation.

Beneath this radiopaque formation, a focal radiolucent area was identified within the dorsal aspect of the third phalanx. The radiolucency was relatively well delineated but irregular in contour, consistent with localized osteolysis. The dorsal cortical bone appeared thinned and focally interrupted, with loss of the normal continuous radiopaque cortical line. The adjacent trabecular bone demonstrated reduced radiodensity and partial disruption of its normal architecture, indicating inflammatory bone rarefaction and remodeling.

In addition, a radiolucent tract extending from the dorsal cortical lesion toward the coronary band was identified. This tract contained discrete radiolucent foci consistent with intralesional gas within the soft tissues and subparietal space, supporting the presence of an active septic process. The orientation of this channel indicated communication between the osseous lesion and the coronary region, compatible with a fistulous tract or drainage pathway through the subparietal tissues. No complete fracture lines, articular surface involvement, or displacement of the distal phalanx were observed, and the distal interphalangeal joint space appeared preserved.

Overall, the radiographic examination revealed changes consistent with a focal inflammatory osteitic process affecting the dorsal surface of the third phalanx (P3). The most prominent findings included focal cortical disruption of the dorsal margin of the distal phalanx accompanied by a well-defined area of localized osteolysis. The lytic region appeared irregular and slightly radiolucent relative to the surrounding cortical bone, suggesting active bone resorption associated with inflammation or infection.

Additionally, a radiolucent tract-like area was visible extending from the dorsal margin of the distal phalanx toward the inner surface of the hoof wall. This radiographic feature was compatible with a subparietal septic cavity located between the dorsal hoof wall and the underlying structures. The presence of this radiolucent pathway suggested a possible drainage route or communication channel between the infected bone surface and the surrounding soft tissues. The cavity appeared to communicate proximally with the region of the coronary band, corresponding topographically with the hyperemic and painful area observed during the clinical examination.

Taken together, these findings were highly suggestive of a localized septic process involving the dorsal aspect of the distal phalanx, characterized by focal osteitis, cortical bone destruction, and secondary reactive bone formation. The radiographic appearance also indicated communication between the osseous lesion and the subcoronary soft tissues, supporting the hypothesis of a septic focus originating from or extending toward the coronary band (Fig. 2a and b). These combined clinical and radiographic findings highlight the importance of early diagnostic imaging in identifying septic involvement of the distal phalanx and guiding appropriate therapeutic management.

3.3 Intraoperative Findings and Surgical Management

Surgical intervention was performed under general anesthesia, with the horse positioned in lateral recumbency. The affected limb was carefully prepared and aseptically draped. Particular attention was given to precise identification and preservation of the coronary band in order to prevent secondary disturbances in horn growth. A progressive distal resection of the dorsal hoof wall was carried out in a controlled, stepwise manner, allowing continuous assessment of the underlying corium and limiting tissue removal to the affected area.

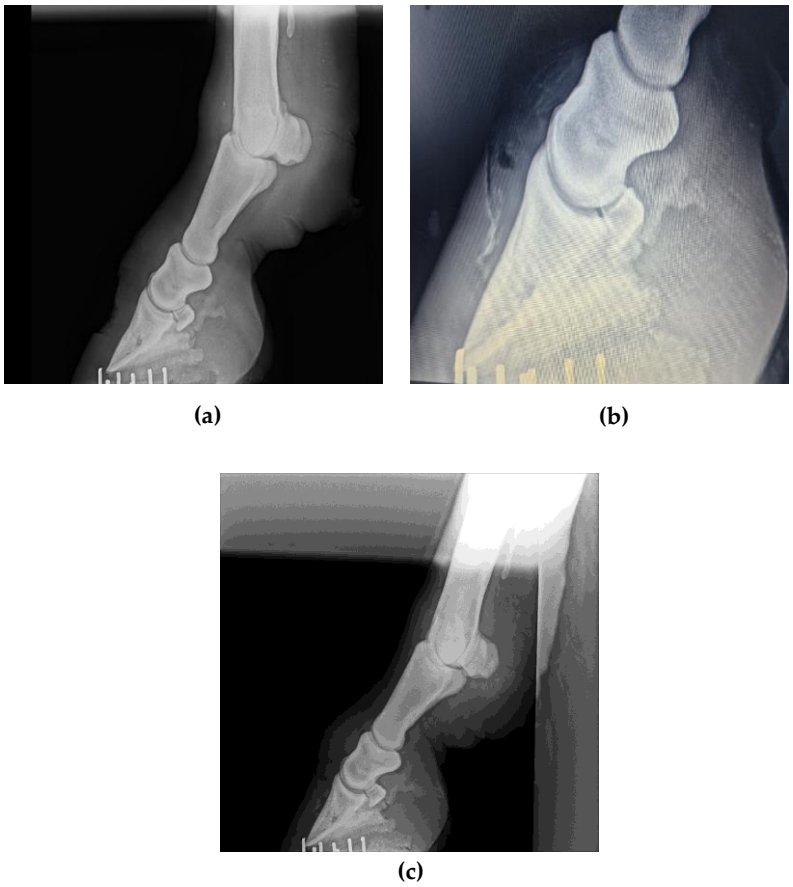


Figure 2. Preoperative latero-medial radiograph of the left hindlimb showing (**a**) dorsal radiopaque lesion of the third phalanx with associated osteolysis; (**b**) Gas-containing tract toward the coronary band; (**c**) Latero-medial radiograph obtained 27 days postoperatively showing restoration of the dorsal cortical margin of the third phalanx and complete disappearance of the previously observed radiolucent lesion.

The purulent material had accumulated within a confined subparietal compartment located between the dorsal hoof wall and the underlying corial tissues. This space had been effectively sealed by the rigid hoof capsule, preventing spontaneous drainage and allowing progressive accumulation of exudate. As a result, substantial internal pressure had developed within the lesion prior to surgical decompression.

The pressurized evacuation of purulent exudate, together with the presence of intralesional gas, strongly confirmed the existence of an active septic process within an anatomically inextensible compartment. Such conditions are characteristic of deep subparietal abscessation, where the rigid structure of the hoof capsule limits expansion and promotes the buildup of pressure, thereby exacerbating pain and facilitating the extension of infection toward adjacent soft tissues and osseous structures. The sudden decompression achieved through dorsal wall resection therefore represented both a diagnostic confirmation of the septic focus and a critical therapeutic step in relieving intracompartimental pressure and enabling thorough drainage and debridement of the infected cavity.

Following complete evacuation of the purulent content, a well-defined subparietal cavity became clearly visible within the dorsal aspect of the hoof capsule (Fig. 1b). The cavity was located between the inner surface of the dorsal hoof wall and the underlying corial tissues, extending proximally toward the coronary band. Its margins were relatively distinct, indicating the presence of a localized pathological compartment formed as the septic process progressed. Careful inspection revealed that this cavity communicated directly with the dorsal cortical surface of the third phalanx (P3), confirming the radiographic suspicion of osseous involvement.

The internal surface of the cavity was lined by markedly inflamed soft tissue characterized by hyperemia and friable inflammatory granulation tissue. In several areas, partially organized necrotic debris adhered to the surrounding tissues and the exposed bone surface. The dorsal cortex of the distal phalanx was directly visible through the surgical window and presented evident pathological alterations, including focal cortical irregularity, superficial bone erosion, and localized loss of cortical continuity. These macroscopic findings were consistent with an inflammatory osteitic lesion affecting the dorsal margin of P3.

Thorough surgical debridement was subsequently performed. Curettage of the affected osseous surface was carried out using sharp bone curettes in order to remove all infected and devitalized material. Necrotic bone fragments, inflammatory granulation tissue, fibrinous deposits, and other devitalized debris were meticulously excised until a clean and stable bone surface was obtained. The curettage was continued carefully until only firm, viable bone remained, characterized by a more uniform appearance and resistance to the curette.

During this process, a free osseous fragment measuring approximately 1.83 cm in length was identified within the cavity and subsequently extracted (Fig. 1c). The fragment was irregularly shaped, with rough and heterogeneous surfaces and poorly defined margins. It was not firmly attached to the surrounding bone structures and could be removed without resistance, suggesting that it had previously detached from the dorsal cortical surface of the third phalanx. Macroscopic examination indicated that the fragment was composed of devitalized or reactive bone. Based on its appearance and location, it was considered compatible either with a sequestrum formed secondary to focal septic osteitis or with a reactive exophytic bone fragment that had subsequently become detached during the progression of the inflammatory process.

The removal of this fragment was considered a critical step in the surgical management of the lesion, as retained sequestra or devitalized bone can act as persistent foci of infection and significantly impair the resolution of septic processes within the hoof. Necrotic or poorly vascularized bone fragments provide an ideal substrate for bacterial colonization, protecting microorganisms from both host immune responses and antimicrobial therapy. Consequently, failure to remove such fragments may lead to chronic infection, delayed healing, or recurrence of clinical signs.

Complete elimination of the fragment, combined with thorough curettage of the surrounding tissues, allowed effective decontamination of the cavity and facilitated the establishment of a viable environment for subsequent healing. Careful curettage ensured the removal of infected and devitalized tissue, while preserving as much healthy bone and surrounding structures as possible. This approach promotes the formation of healthy granulation tissue and supports progressive tissue regeneration within the affected area.

Furthermore, the removal of the sequestrum reduces mechanical irritation and prevents continued inflammatory stimulation of the adjacent tissues. By eliminating the primary source of infection and restoring adequate drainage and aeration of the cavity, the procedure enhances the effectiveness of postoperative treatments and significantly improves the prognosis. In cases involving the distal phalanx and hoof structures, such surgical debridement is particularly important, given the limited vascular supply and the enclosed anatomical environment that can favor the persistence of septic material.

Curettage was continued until all visibly altered bone tissue had been eliminated and a uniformly bleeding cortical surface was obtained. The presence of punctate hemorrhage from the bone surface was considered an important indicator of adequate debridement and tissue viability, suggesting that the remaining bone retained an intact vascular supply. Achieving this endpoint is essential in the surgical management of septic osseous lesions, as it indicates that devitalized or infected bone has been successfully removed.

At this stage, contact between the curette and the remaining bone produced a clear, resonant sound, consistent with healthy and structurally stable osseous tissue (Fig. 1d). This auditory feedback is frequently used intraoperatively as an additional indicator that the curettage has reached viable cortical bone, distinguishing it from the dull sound typically associated with necrotic or weakened bone.

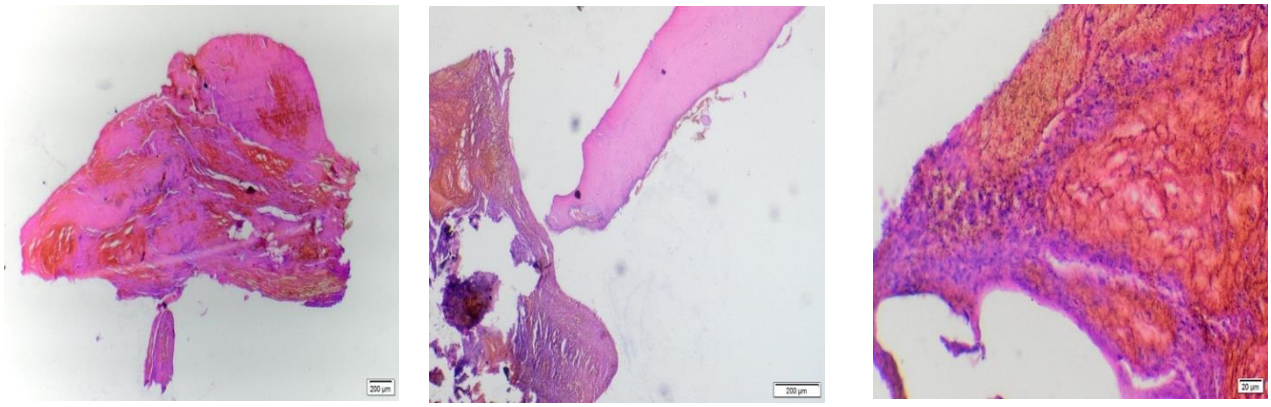
Careful inspection of the surgical cavity confirmed the absence of residual purulent material, sinus tracts, or necrotic bone fragments. The margins of the defect appeared regular and clean, with no evidence

of further cortical instability or ongoing bone destruction. Ensuring complete removal of infected and devitalized tissues at this stage is crucial to minimize the risk of persistent infection and to create favorable conditions for subsequent healing, granulation tissue formation, and gradual restoration of the affected structures.

Following extensive lavage of the surgical cavity with sterile isotonic solution, a topical antimicrobial and cicatrizing ointment was applied to the exposed corial and osseous surfaces. A sterile compressive bandage was placed to protect the surgical site and to support controlled healing. Postoperatively, systemic anti-inflammatory therapy was administered to control pain and inflammation, and broad-spectrum antimicrobial therapy was instituted to address the septic component of the lesion. The bandage was scheduled for regular changes, with local cleansing and reapplication of topical medication. Strict environmental hygiene and limited activity were recommended during the initial healing phase to prevent contamination and mechanical stress on the regenerating hoof wall.

3.4 Histopathological examination

Histopathological examination of the extracted osseous fragment was performed on serial sections stained with Hematoxylin–Eosin. At low magnification (4× objective), the specimen consisted predominantly of mature lamellar bone with largely preserved cortical architecture, although focal surface irregularities were evident (Fig. 3a). The periosteum was markedly thickened and expanded by oedema, with partial loss of the normal distinction between the outer fibrous and inner osteogenic layers. In certain areas, the external cortical surface appeared uneven, consistent with reactive bone remodeling in an inflammatory context.



(a) **(b)** **(c)**

Figure 3. Representative histological section showing (**a**) Mature lamellar bone with marked periosteal thickening; (**b**) Periosteal oedema, vascular congestion, and mixed inflammatory infiltrate; (**c**) Dense neutrophilic and macrophagic inflammatory infiltration without evidence of neoplastic changes

At intermediate magnification (10× objective), the periosteum appeared markedly thickened due to pronounced interstitial oedema and cellular infiltration. The periosteal connective tissue was expanded by clear spaces separating collagen bundles, consistent with fluid accumulation within the extracellular matrix. Within this edematous stroma, a mixed inflammatory infiltrate was evident, composed predominantly of neutrophils and macrophages, with occasional scattered lymphocytes (Fig. 3b). The neutrophils were often arranged in small aggregates or diffusely distributed throughout the periosteal tissue, indicating an active acute inflammatory component, while the presence of macrophages suggested ongoing phagocytosis and tissue cleanup associated with a subacute inflammatory stage.

The vascular component of the periosteum and adjacent connective tissue was markedly altered. Numerous small and medium-sized blood vessels were dilated and congested with erythrocytes, reflecting vascular hyperemia and increased local blood flow typical of inflammatory reactions. Endothelial cells lining these vessels appeared mildly swollen, and in some areas the perivascular space was expanded by inflammatory cells and oedema fluid.

Multifocal areas of recent hemorrhage were also observed within the periosteal tissue. These consisted of small accumulations of extravasated erythrocytes dispersed within the connective stroma, likely resulting

from increased vascular permeability and minor vascular damage associated with the inflammatory process. The hemorrhagic foci were generally limited in extent and did not show evidence of significant organization.

In addition to the previously described inflammatory alterations, a mild fibroblastic proliferation was also evident within the periosteal connective tissue layer. Numerous spindle-shaped fibroblasts were identified interspersed among the collagen fibers of the periosteum. These cells appeared elongated with tapered cytoplasmic processes and oval nuclei, and they were arranged in small aggregates as well as short, interlacing bundles, indicating an increase in fibroblastic activity within the connective tissue matrix.

The surrounding extracellular matrix exhibited moderately organized collagen fibers, between which these fibroblasts were distributed, suggesting the initiation of reactive connective tissue remodeling. Such fibroblastic proliferation is commonly associated with the early stages of tissue repair, where fibroblasts actively participate in the synthesis of new extracellular matrix components, particularly collagen, as part of the reparative response.

This proliferative fibroblastic response likely represents an early reparative mechanism triggered by the adjacent inflammatory and destructive processes affecting the periosteal surface and underlying bone tissue. The presence of activated fibroblasts in the periosteal connective tissue indicates that the tissue is attempting to restore structural integrity and stabilize the affected area, counteracting the damage induced by the ongoing inflammatory reaction.

Furthermore, the coexistence of inflammatory changes and fibroblastic proliferation reflects the dynamic interplay between tissue injury and the activation of local reparative mechanisms, a process that is commonly observed in periosteal reactions associated with osteitic or osteomyelitic conditions. In the presence of inflammatory stimuli, the periosteum responds through a cascade of cellular and molecular events that aim to contain tissue damage and initiate structural repair.

The periosteum is a highly vascularized and metabolically active connective tissue layer, composed of an outer fibrous layer and an inner cambium layer rich in mesenchymal progenitor cells. Due to this cellular composition, it plays a crucial role in bone homeostasis, repair, and remodeling. When exposed to inflammatory mediators released during infection or bone injury, the periosteum becomes activated, leading to proliferation of fibroblasts and recruitment of progenitor cells capable of differentiating into osteogenic lineages.

In such conditions, fibroblasts within the periosteal connective tissue increase their synthetic activity, producing extracellular matrix components, particularly collagen fibers, which contribute to the formation of a provisional reparative framework. This early fibroplastic response not only stabilizes the affected tissue but also creates a microenvironment that supports subsequent phases of tissue repair, including osteoblastic activity and potential new bone formation.

Additionally, inflammatory mediators and local vascular changes may stimulate angiogenesis and enhanced cellular turnover within the periosteum, further supporting the regenerative process. These mechanisms represent a coordinated attempt by the periosteal tissue to counteract the destructive effects of inflammation and restore structural integrity to the affected bone surface.

Therefore, the simultaneous presence of inflammatory infiltrates and fibroblastic proliferation in the periosteal connective tissue is indicative of an active and ongoing reparative response, reflecting the intrinsic capacity of the periosteum to participate in both inflammatory defense and early stages of bone tissue regeneration.

At higher magnification (20× objective), the inflammatory infiltrate was more clearly characterized by dense aggregates of neutrophils distributed within the periosteal connective tissue and adjacent inflammatory stroma (Fig.3c). Many of these neutrophils exhibited morphological features consistent with degenerative change, including nuclear swelling, karyolysis, and fragmentation of nuclear material. In several areas, neutrophils appeared partially disintegrated, forming small accumulations of cellular debris intermixed with fibrin and inflammatory exudate. These findings were indicative of an intense acute inflammatory response associated with tissue destruction.

Interspersed among the neutrophilic aggregates were numerous activated macrophages. These cells displayed abundant eosinophilic cytoplasm and enlarged, often vesicular nuclei, consistent with a reactive and phagocytically active phenotype. In some macrophages, cytoplasmic vacuoles containing cellular debris or erythrocyte remnants were observed, reflecting ongoing phagocytosis of necrotic tissue and inflammatory detritus. The coexistence of neutrophils and macrophages suggested a transition between acute suppurative inflammation and early stages of tissue clearance and repair.

Despite the abundance of inflammatory cells, no well-organized abscess structure was identified at this magnification. Specifically, there was no clear central cavity surrounded by a defined pyogenic membrane or a peripheral fibrous capsule. Instead, the inflammatory process appeared relatively diffuse and infiltrative within the affected periosteal and connective tissues.

Importantly, careful examination of the cellular morphology revealed no evidence of nuclear atypia, abnormal mitotic figures, or pleomorphism that might indicate a neoplastic process. The nuclei of fibroblasts, inflammatory cells, and vascular endothelial cells maintained normal architecture and chromatin distribution. Furthermore, there were no atypical cellular proliferations or disorganized tissue patterns suggestive of sarcomatous or other neoplastic transformation. These observations supported the interpretation that the lesion represented a purely inflammatory and reactive process rather than a proliferative neoplasm.

Overall, the histopathological features were consistent with inflammatory periostitis and osteitis associated with reactive bone formation. The examined sections demonstrated clear evidence of an active inflammatory process involving both the periosteum and the superficial cortical layers of the bone. The periosteum was markedly expanded by oedema and infiltrated by mixed inflammatory cells, predominantly neutrophils and macrophages, accompanied by vascular congestion and multifocal hemorrhages. These findings indicated an ongoing inflammatory reaction affecting the periosteal tissues.

In addition to the inflammatory component, the cortical surface of the bone exhibited areas of irregularity and focal resorption compatible with osteitic changes. The presence of necrotic debris, inflammatory exudate, and degenerating neutrophils further supported the diagnosis of an active infectious or septic process affecting the osseous structures. Macrophage infiltration and phagocytic activity suggested ongoing removal of necrotic material and tissue breakdown products.

Evidence of early reparative activity was also present. Mild fibroblastic proliferation within the periosteal connective tissue, together with the presence of reactive bone formation along the cortical surface, indicated that the lesion had initiated a localized reparative response. This reactive osteogenesis likely represented a biological attempt to stabilize and remodel the affected bone in response to inflammation and tissue damage.

Importantly, no histological features suggestive of neoplastic proliferation were observed. Cellular morphology remained consistent with inflammatory and reparative processes, without nuclear atypia, abnormal mitotic figures, or disorganized cellular growth patterns.

Taken together, these microscopic findings supported the diagnosis of focal inflammatory periostitis and osteitis accompanied by reactive bone formation, most consistent with a secondary response to a localized septic process affecting the dorsal aspect of the distal phalanx.

3.5 Clinical and Radiographic Outcome

The horse was re-evaluated 27 days postoperatively. The owner returned primarily for a complimentary tax-free follow-up radiographic examination, performed at the clinician's insistence in order to objectively assess lesion evolution, coinciding with a separate dental procedure scheduled for another horse.

At clinical examination, the horse showed complete resolution of lameness in the affected forelimb. No pain was elicited on palpation of the coronary band or dorsal hoof wall. The surgical defect of the hoof capsule exhibited progressive keratinization and ongoing horn regeneration, with satisfactory cicatrization and absence of purulent discharge. No signs of recurrent infection or local inflammation were observed.

Follow-up latero-medial radiography demonstrated complete resolution of the previously described radiolucent area at the dorsal aspect of the third phalanx (Fig. 2c). The dorsal cortical margin appeared continuous and well-defined, with restoration of normal radiodensity of the adjacent trabecular bone. No residual osteolysis, gas accumulation, or fistulous tract toward the coronary band was identified.

These findings indicated complete clinical recovery and radiographic resolution of the septic osteitic process within 27 days following surgical debridement. The clinical image illustrates a focal defect located on the dorsal aspect of the hoof wall in close proximity to the coronary band. The lesion appears as a vertically oriented fissure associated with localized disruption of the hoof horn and partial loss of the external keratinized layer. The surrounding horn tissue shows irregular margins and structural disorganization, suggesting previous mechanical disruption and subsequent degradation of the hoof wall architecture.

This type of defect is consistent with a penetrating or traumatic lesion of the coronary region, which may serve as an entry point for bacterial contamination into the deeper structures of the hoof capsule. In working horses, particularly those operating in forestry environments, small penetrating injuries produced

by wooden splinters or sharp environmental debris may remain initially unnoticed due to the dense hair covering the coronary band and the rigid structure of the hoof capsule.

Once microbial contamination occurs, the confined anatomical environment of the hoof capsule can facilitate the accumulation of inflammatory exudate within the subparietal space, promoting the development of a localized septic process. Progressive pressure generated by purulent material within this inextensible compartment may lead to separation of the hoof wall layers and formation of a visible fissure or defect, similar to the one observed in this case.

Furthermore, the proximity of the lesion to the coronary band is of particular clinical significance. The coronary region represents the primary site of horn production, and traumatic or septic damage at this level may compromise the integrity of the newly forming hoof wall. Such lesions may therefore persist or enlarge over time as the hoof grows, creating a pathway through which infection can propagate toward deeper tissues.

The presence of a focal defect in this region supports the hypothesis of a communication pathway between the external environment and the deeper subparietal structures, which may ultimately allow extension of the septic process to the dorsal cortical surface of the distal phalanx. This mechanism is consistent with the radiographic and intraoperative findings described in the present case, where a tract connecting the coronary region with the dorsal surface of P3 was identified.

Overall, the clinical appearance of the hoof lesion provides important evidence supporting the presumed pathogenesis of the condition, namely a traumatic coronary penetration followed by subparietal infection and secondary septic osteitis of the distal phalanx. Such lesions highlight the importance of careful inspection of the coronary band and dorsal hoof wall in horses presenting with severe lameness of unclear origin.

4. Discussion

Inflammatory conditions of the distal phalanx associated with lesions of the coronary region represent a diagnostic and therapeutic challenge due to the confined anatomical environment of the hoof capsule and the potential for rapid extension of infection into osseous structures. Penetrating injuries of the hoof, even when not immediately apparent, are well recognized as a source of deep-seated infection and subsequent osteal involvement [6]. In the present case, although a penetrating tract was not externally visible at the time of examination, radiographic evidence of a communication pathway containing gas between the dorsal cortex of P3 and the coronary region strongly supported the presence of a septic process originating from the coronary area.

Solar and subparietal penetrations have been associated with progression toward deeper structures, particularly when early drainage is inadequate or delayed [7]. The rigid, non-distensible nature of the hoof capsule favors accumulation of purulent material under pressure, leading to vascular compromise of adjacent tissues. In this case, the intraoperative finding of pressurized purulent exudate mixed with gas confirmed the existence of a septic focus within a confined subparietal compartment, consistent with mechanisms previously described in penetrating hoof injuries.

Septic pedal osteitis has been documented as a complication of penetrating wounds and subsolar abscessation, frequently requiring surgical intervention when conservative management fails [8]. In reported case series, aggressive debridement of necrotic bone and restoration of drainage were considered essential for resolution and return to function. The surgical approach adopted in the present case, consisting of progressive dorsal hoof wall resection and thorough curettage of the affected cortical bone, is in accordance with these recommendations.

Although septic osteitis of the distal phalanx has been more extensively described in foals, similar pathological mechanisms apply to mature horses when bacterial contamination results in cortical necrosis and osteolysis [9]. The radiographic findings observed in this case—focal osteolysis, cortical disruption, and a distinct radiopaque formation—are consistent with inflammatory bone remodeling and possible sequestrum formation secondary to localized vascular compromise.

The extracted osseous fragment was macroscopically detached and corresponded radiographically to the longitudinal radiopaque structure identified preoperatively. Sequestrum formation is recognized as a consequence of localized bone necrosis, followed by demarcation and separation of the devitalized fragment

from surrounding viable bone [10]. The histopathological findings of lamellar bone with periosteal thickening and mixed inflammatory infiltration support the interpretation of an inflammatory osteal process associated with reactive bone proliferation rather than neoplastic transformation.

Furthermore, marginal cortical devitalization has been reported in association with traumatic or septic processes, leading to focal separation of bone fragments [11]. Although no fracture line was identified radiographically in the present case, the possibility of microtrauma or focal cortical disruption secondary to a penetrating wooden foreign body cannot be excluded, particularly given the horse's use in forestry traction work [12]. The absence of retained foreign material at the time of surgery does not preclude a prior transient penetrating insult that initiated the inflammatory cascade [12].

The complete radiographic resolution of the osteolytic area and restoration of a continuous dorsal cortical margin within 27 days indicate that early and thorough surgical debridement can result in favorable outcomes, even in cases involving focal osseous sequestration. These findings underscore the importance of prompt radiographic assessment in cases of unexplained severe lameness associated with coronary region abnormalities and highlight the necessity of complete removal of necrotic and devitalized bone to prevent chronic infection or persistent osteitis.

Overall, this case reinforces the pathophysiological link between localized septic processes within the hoof capsule and focal cortical necrosis of the distal phalanx, culminating in osseous sequestration. Accurate radiographic diagnosis combined with meticulous surgical debridement appears to be critical for achieving rapid functional recovery in similar cases.

5. Conclusions

This case illustrates that inflammatory pathology of the distal phalanx associated with lesions of the coronary region may culminate in focal cortical necrosis and osseous sequestration, even in the absence of an externally visible penetrating wound. The confined anatomical environment of the hoof capsule facilitates accumulation of septic material under pressure, predisposing to vascular compromise and localized osteitis.

Radiographic examination proved essential for identifying cortical disruption, focal osteolysis, and the presence of a detached osseous fragment in communication with the coronary region. Early recognition of these changes allowed timely surgical intervention.

Complete surgical debridement, including removal of the detached lamellar bone fragment and curettage to macroscopically viable bleeding bone, resulted in rapid clinical recovery and radiographic resolution of the lesion within 27 days. Histopathological evaluation confirmed the inflammatory nature of the lesion and excluded neoplastic proliferation.

Prompt diagnostic imaging combined with meticulous surgical management appears critical for achieving favorable functional outcomes in cases of septic osteal involvement of the distal phalanx associated with coronary region lesions.

Supplementary Materials: No supplementary material is provided, as all information and data are presented in the manuscript.

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