"LUMPY JAW" IN EXOTIC HOOF STOCK: A HISTOPATHOLOGIC INTERPRETATION WITH A TREATMENT PROPOSAL

David A. Fagan, D.D.S., James E. Oosterhuis, D.V.M., and Kurt Benirschke, M.D.

Abstract: "Lumpy jaw" of artiodactyls and macropods, although often considered a manifestation of actinomycosis, is actually an osteomyelitis, perhaps commencing with a dental root abscess or trauma to the jaw. Anaerobes may be cultured from the lesions, and vegetable matter may be impacted in them. It is a chronic disease and difficult to treat. Successful treatment may include draining the abscess, cleaning the cavity with saline and antibiotics, flushing with a combination of hydrogen peroxide–sodium hypochlorite and Betadine, and apicoectomy and endodontic filling when active inflammation has subsided.

Key words: "Lumpy jaw," actinomycosis, chronic alveolar osteomyelitis, compound apicoectomy, surgical fistulation, exotic hoof stock.

INTRODUCTION

The term "lumpy jaw" has been used to describe an assortment of clinical ailments that involve the facial bones of hoof stock, particularly chronic actinomycosis lesions common in domestic sheep and cattle, or macropods. Lumpy jaw lesions in exotic ungulates are often assumed to represent actinomycosis that require opening, drainage, flushing, and healing by second intention.

Lumpy jaw does not describe an actual disease, nor does it represent a morphologic diagnosis. It is merely a colloquialism used to identify anatomic facial bone abnormalities. These chronic, expansive, bony lesions are actually the initial clinical manifestation of chronic alveolar osteomyelitis, most often involving dentition. It is usually associated with overlying, diffuse, soft tissue cellulitis, which may be located some distance away from the dental source of infection.

Chronic alveolar osteomyelitis more correctly describes the morphologic equivalent for this common anatomical abnormality (Fig. 1a–c). It describes the disease in exotic ungulates more precisely in etiologic and pathogenetic terms.

These lesions have been difficult to treat. Anaerobic bacteria may persist in dentinal tubules of root canal walls, leading to treatment failure.^{1,8} Lesions must be cleaned with endodontically accepted disinfecting irrigation to eliminate organisms. Although endodontic irrigants may permeate dentinal tubules, their effectiveness may depend on the type of bacteria present.²

CASE REPORTS

Case 1: Transcaspian urial (Ovis vignei arkal)

This mature, approximately 2-yr 6-mo, 34-kg female presented with a severe gore to the left caudal thigh but was otherwise healthy. The wound healed without incident. Four months later, the animal represented with an intermandibular swelling. Radiography confirmed a cystic bony structure at the base of the ramus of the left mandible (Fig. 2). There was no clinical evidence of infection. The animal was released and observed for another month. Because the mandibular swelling did not improve, treatment was started to prepare for apicoectomy of the involved molar tooth with endodontic therapy, debridement, and closure of the wound (Fig. 3). Culture of the abscess identified light gram-positive cocci, heavy gram-negative rods, heavy probable Bacteroides species, light probable microaerophilic streptococcus, light alphastreptococcus, and heavy Fusobacterium species. The urial was anorexic after the oral surgery and unable to cope with hospital confinement. Despite medical management, the animal's condition deteriorated, and it was euthanized.

Radiographs (see Fig. 2) suggested localized inflammatory response that could have been caused by a direct traumatic blow to the lower jaw, so the mandibular swelling may have resulted from traumatic injury to the lower border of the mandible during the confrontation, which also resulted in the reported gore wound.

Case 2: Himalayan tahr (*Hemitragus jemlahicus*)

This approximately 3-yr and 6-mo old, 32.5-kg female was euthanized after 8 mo of veterinary care

From the Colyer Institute, PO. Box 26118, San Diego, California 92196-0118, USA (Fagan); the Zoological Society of San Diego—San Diego Wild Animal Park, Escondido, California 92027, USA (Oosterhuis); and the Department of Pathology and Reproductive Medicine, University of California, San Diego Medical Center, San Diego, California 92103-8321, USA. (Benirschke). Correspondence should be directed to Dr. Fagan.



Figure 1. a. A view of the right side of the shaved face of a male Alpine ibex showing the typical clinical presentation of a chronic, expansive, mandibular osteomyelitis. The slight depression in the center of the lump is the weakest point of this bony expansion, and this is the location where this abscess will eventually rupture and drain, below the lower border of the mandible. b. Radiographic view of the mandibular right first molar of the Alpine ibex seen clinically in a. Note that the radiolucency is clearly of dental origin, which involves and appears to be limited to the periapical region of this single molar tooth. c. Initial view of the unshaved face of the Alpine ibex seen in a. Note that the anatomical distortion of the rear portion of the mandible is essentially visually unnoticeable. Palpation of the lower border of the mandible is the only dependable method of early detection of these lesions.



Figure 2. Preoperative diagnostic radiograph of the urial confirming the presence of a periapical radiolucency involving the mandibular right first molar. Note the osteoblastic nodules of reactive bony tissue surrounding the apical area of the tooth root, which occurred before complete tissue necrosis.

for chronic, active, necrotizing cellulitis and mandibular alveolar osteomyelitis involving the complex endodontic anatomy (Figs. 7a–c, 8). This animal had lost two mandibular left molars previously. The source of recurrent infection was not identified. This right facial abscess may have caused other systemic issues such as constant pacing causing significant damage to the feet. The organisms identified in this animal's mandibular abscess included heavy gram-positive rods, heavy *Actinomyces pyogenes*, heavy *Peptococcus* species, and heavy *Fusobacterium* species.

The mandibles of the urial and tahr were removed after death, fixed in formalin, and photographed. Muscle and connective tissue were removed, and the mandibles were decalcified in 11% hydrochloric acid until they were soft enough for



Figure 3. Postoperative radiographic view of the urial's completed apicoectomy, endodontic treatment with synthetic particulate bone graft of the involved molar tooth.



Figure 4. This step cross section of the urial's mandible clearly indicates the invasive nature of the expansive bony infection with a focus of infection contained within and emerging from the large, partially necrotic, complex anatomy of the molar pulp chamber. The arrows at the top left indicate healthy periodontal ligament (PDL); on the top right healthy dentin. The arrows in the middle indicate healthy connective tissue on the left and healthy bone on the right. The triple arrow at the bottom left indicates the expanding necrotic tissue surrounding the apical third of this birooted tooth spreading to the adjacent tooth to the bottom right. The double arrow at the bottom right indicates intact dentin surrounding the partially necrotic pulp tissue. Note that the infection spreads easily along the PDL space; then through the interradicular bone to the neighboring tooth. Recall from the text that Basson and others have shown that all this "Dead tissue, in the form of slough and necrosis . . . delays healing and promotes infection."

sectioning. They were washed and step sectioned for study and photography. Selected portions were embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Some sections were further stained with the Brown and Brenn bacterial stain (IMEB Inc., International Medical Equipment, San Marcos, California 92069, USA) and Grocott methenamine silver fungal stain.⁷

Figures 4–6 indicate a partially necrotic vascular bed surrounding the apical region of an actively growing tooth root of the Transcaspian urial. This sagittal section also demonstrates two more developing apical abscesses in adjacent teeth because this aggressive bacterial infection spreads along the periodontal ligament space to infect the alveolar bone.^{2,8} Similar histopathology was identified in the tahr.

DISCUSSION

The etiology of chronic alveolar osteomyelitis remains complex and incompletely understood. However, it is clear that one or more of the following



Figure 5. This sagittal section of the urial's mandibular dentition demonstrates additional developing abscesses because this destructive bacterial infection spreads the length of the periodontal ligament space to infect adjacent alveolar bone. All of this "Dead tissue, in the form of slough and necrosis . . . delay(s) healing and promote(s) infection."8 The arrow at the top left indicates infected pulp tissue linked with the severely necrotic first molar periapical abscess. The triple arrow at top middle indicates healthy dentin, and the arrows at the top right and lower left indicate healthy alveolar bone. The remaining three arrows along the bottom indicate the molar's root abscess; a developing apical abscess involving premolar 4; and incipient interradicular bone necrosis between these two teeth. Note that premolars 3, 2, and 1 also appear to have varied degrees of apical tissue necrosis, which represents a typical spreading of the infective process "down stream" along the path of arterial flow.

four common clinical ailments can form the focus of infection, which eventually results in the expansive lumpy jaw osteomylelitis process of the alveolar bone: 1) any aggressive periapical abscess, 2) any chronic periodontal abscess, 3) a localized traumatic injury to the mandible or maxilla, and 4) a genetic or developmental defect (example, enamel dysplasia).

These etiologic factors often occur in combination in our personal experience. For example, a localized periodontal abscess can progress into a periapical abscess. An enamel or dentin dysplasia such as the dens in dente, often results in an irreversible pulpitis with a periapical abscess. Any irreversible pulpitis or periapical abscess may eventually develop into a chronic alveolar osteomyelitis originating at the apex of the infected tooth. A traumatic blow to the lower border of the mandible often results in a localized injury with inflammatory consequences, which can then become infected and develop into an osteomyelitis. It is important to note



Figure 6. This medial-lateral step section view of the mandible establishes the relationship of the expansive bony lesion to the apex of a typical, trirooted, mandibular first molar marked with a triple-headed arrow at the top right. The double arrow at the top left indicates the adjacent birooted, mandibular, right premolar 4. The arrow at the bottom right identifies the periapical cystic cavity associated with this molar tooth. The middle arrow along the bottom identifies a pathologic fracture of the lower border of the mandible (the straight edge is a processing artifact). The arrow at the lower left identifies the apical portion of the anterior root of premolar 4.

that all these etiologic factors occur with regularity, involve the animal's dentition, are difficult to detect without a detailed intraoral examination including manual palpation of the jaw and dental radiography. All can eventually result in a chronic, expansive, alveolar osteomyelitis of dental origin as shown in Figures 9, 10.

There may be additional etiologic factors or combinations thereof that are as yet unidentified and which contribute to the formation of chronic alveolar osteomyelitis. For example, carious or developmentally deformed teeth enable intraoral organism and foreign bodies from food material to be impacted into the dental pulp chamber during mastication.

There may be additional etiologic factors or combinations thereof that are as yet unidentified and which contribute to the formation of the lumpy jaw lesion. For example, carious or developmentally deformed teeth enable intraoral organism and foreign bodies from food material to be impacted into the dental pulp chamber during mastication. An understanding of the pathogenesis of these lesions is important because successful clinical management and treatment depend on the interruption of this progressive sequence.

Alveolar osteomyelitis typically develops in the following sequence: inflammation of the alveolar bone occurs secondary to one of the factors listed above. If the lesion was not infected initially, sooner or later infection of the site will most likely develop. In the clinical experience of the authors, these lesions are generally of mixed microbial type, and they develop within the body of the facial bones. The animal's immune system responds and attempts to wall off and contain the infection with fibroplasias. The process eventually results in the formation of a cystic cavity. Bacterial growth (particularly anaerobic), continues within the cystic cavity. Intermittently, but regularly, the pressure within the cystic cavity exceeds the surrounding local capillary bed pressure. When this occurs, the lesion expands with more active inflammation and fibroplasia. Eventually, this partially contained expansion erodes into the nutrient canals of the adjacent cortical layer of bone and diffuse cellulitis results. It is only at this time that outwardly demonstrable lumpy jaw becomes evident.

Historically, the treatment for lumpy jaw type lesions has been limited to repeated episodes of opening the lesion, flushing and draining the contained debris, while anticipating the eventual granulation and closure of the wound. However, in time, this therapy usually requires repetition and eventually needs additional treatments. Once the lumpy jaw lesion is recognized to be a chronic alveolar osteomyelitis of dental origin, with one or more tooth root abscesses as the source of the recurrent infection, the clinician is able to treat and cure the lumpy jaw. Detailed analysis and examination of the histologic sections from these cases strongly suggests that the management of chronic alveolar osteomyelitis requires early diagnosis by palpation and oral radiography, aggressive and regular systemic antibiotic therapy, aggressive and regular, and occasionally, prolonged localized antimicrobial debridement of the infected site.

The authors have treated a large number of these lesions (see Table 1) using a combination of clinically proven treatment modalities, which have collectively been termed "the beta infusion technique" which consists of the injection of disinfecting solutions after surgical fistulation, with later compound apicoectomy.

This developing, two-phase treatment protocol requires minimal intervention and handling, so is especially useful defined by the limitations commonly associated within the treatment of the highly excitable and easily injured exotic herbivores. The first phase of treatment (the beta infusion phase) involves the surgical fistulation of the lateral wall of the "lump" mass with placement of a fixed, solid catheter at a location that will eventually provide adequate access to the apical roots of the involved

Figure 7. a. This decalcified cross section of the coronal third of a typical herbivore birooted premolar demonstrates the multichambered anatomy (in this case six individual chambers) of herbivore dentition. These convoluted and interconnected chambers are one of the major reasons for the difficulty in effectively treating "lumpy jaw" tooth-related, facial infections. The top double arrow indicates healthy dentin. The double arrow on the lower left indicates two of the three pulp chambers with healthy pulp tissue remaining. The middle arrow on the bottom indicates necrotic pulp tissue in the middle chamber only with healthy pulp tissue in the other two chambers indicated by the double arrow on the bottom right. This

dentition. This requires familiarity with standard apicoectomy surgical access, as well as the ability to efficiently obtain multiple intraoral dental radiographs during the surgical procedure. The clinical objective is to convert the chronic, invasive osteomyelitis into a contained, externally draining fistulous tract. This tract connects the apical abscess of the associated tooth (teeth) directly to the exterior of the animal's face. If the patient is an exhibit animal, and if the issue of facial cosmetics may eventually become a management concern, it is essential to correct the anatomical distortion caused by the swelling of the cortical bony plate at this time.

The surgical fistulation of the lesion phase requires minimal "hands on" time and only inter-

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situation is representative of a classic "single point infection" involving the dental pulp "from the crown down," as opposed to a periapical infection, which initially infects the dental pulp "from the apex up." Crown down infections originate through abraded, developmental, or genetic "holes" in the chewing surface of the tooth-similar to a "dens in dente," or abraded occlusal surface. Compare this view of the dental pulp with the connected pulp chamber configurations seen in b and c. H&E, $\times 8$. b. Note the isthmus connecting the adjacent dental pulp chambers in the middle third of a typical herbivore premolar tooth (now only one convoluted chamber). Compare this crosssectional anatomy to that seen in a and c, and observe the ease with which any dental pulp chamber infection can spread. Proceeding clockwise from the top left, the arrows indicate healthy pulp; the isthmus now connecting the pulp chambers of this birooted tooth; healthy periodontal ligament (PDL); healthy dentin; a processing artifact, which separated the PDL from the dentin; and finally healthy connective tissue at the bottom left. H&E, $\times 8$. c. Note that the tooth root anatomy at the apical third of a typical herbivore premolar tooth is now two disconnected chambers. Compare with that seen in a and b. Note how easily it would be for an infection to invade up into the tooth along the vascular path of one root and then spread into the adjacent pulp canals further up into the tooth through the vascular anastomosis, which occur there. The double arrow at the top indicates healthy pulp tissue in the now bifurcated roots. The arrows on the bottom indicate healthy dentin on the right and healthy bone between the two root apices to the left. The necrotic pulp within this tooth is entirely contained to the coronal third of the tooth by the vascular health of the remaining vital pulpal tissues within the tooth. In time, this will either stimulate the formation of a "secondary dentin bridge" to wall off and isolate these infected tissues, or the infection will continue to spread and result in the complete infective necrosis of all the tooth's pulpal tissue, thereby creating an infective periapical abscess leading to the inevitable formation of another "lumpy jaw" lesion. H&E, ×8.





Figure 8. A higher power view of the necrotic dental pulp tissue similar to the region seen in Figure 7a. Note the interplay between healthy and diseased tissues. Recall what O'Brien said: "Dead tissue, in the form of slough and necrosis, can, if present in a wound, delay healing and promote infection. Debridement describes any method by which such materials are removed and, as a consequence, the potential to achieve wound healing enhanced."⁹ H&E, $\times 40$.

mittent follow-up procedures, generally once or two times a week. This continues for 6-8 wk and allows flushing of the internal bony site with proven disinfecting agents to establish a sealed tubular, draining fistulous tract connecting the tooth root abscess to the exterior surface of the face. This flushingdebridement effect is accomplished in three steps: first, with sterile water (H_20) to verify the patency of the drain path; second, simultaneously from two separate source syringes, one with hydrogen peroxide (H₂O₂), and the other with 5.25% sodium hypochlorite until no further debris is discharged, and this creates an effective, vigorously foaming, gaseous flushing medium of $H_2 + O_2 + Cl_2$ gas bubbles with some HCl + NaCl + H_2O ; third, rinse the depths of the wound with a 2% Betadine solution (Povidone-Iodine U.S.P., 10% with 1% available iodine) which functions as a broad-spectrum antiseptic and antimicrobial.4-6,9,10 All this assists in the elimination of local infective material, while the animal's immune system completes the healing process in about 6-8 wk. This procedure enables the animal to remain with its group or with minimal separation. It also requires minimal time in hospital.

In those cases with pronounced facial soft tissue cellulitis, direct injection of the 2% Betadine solution into and around the infection site soft tissue will rapidly facilitate its prompt resolution. This remains an effective treatment used a number of

Figure 9. A photomicrograph of the dynamic osteoclastic–osteoblastic activity involving the immunologic response of the animal to the bacterial contamination of the tissues in the apical region of the tooth. The two short arrows at the top left show degenerating dentin debris within the pulp chamber. The long arrow at the top right identifies healthy dentin near the apex of the infected tooth. The arrow at the bottom right indicates a mass of regenerating new bone around the apex of the tooth originating on top of a mass of old bone at the lower left. H&E, \times 40.

years ago to manage acutely infected bite wounds in cases where prolonged daily antibiotic therapy was not possible. Analysis of mouse tissues thusly treated demonstrated that although the Betadine has a temporary detrimental impact on healthy tissue, it does not irreversibly damage them. The invasive microorganism die immediately, and the host's ability to regenerate was significantly improved. Until another treatment alternative is developed and be-

Figure 10. A higher power view of the Brown and Brenn–stained colonies of gram-positive organisms invading the dental pulp chamber of the tahr. These organisms are typical of cocci and can only be eliminated with the techniques associated with traditional endodontic therapy, "but their effectiveness is dependent on the type of bacteria found within the tubules.²" H&E, \times 40.

Table 1. List of some of the species treated by the authors, while developing the beta infusion technique with compound apicoectomy into a reasonably effective clinical protocol. A few additional photographs of the developing protocol can be found at the following Web site: www.colyerinstitute.org. Additional general background information concerning these species can be found on the Ultimate Ungulate Web Site.¹²

Aepyceros melampus-Impala Ammotragus lervia-Aoudad, Barbary sheep Antidorcas marsupialis—Springbok Antilocapra americana—Pronghorn Camelus dromedaries-Dromedary, Arabian camel Capra caucasica-West Caucasian tur Capra ibex-Alpine ibex Connochaetes gnou-White-tailed gnu Damaliscus pygargus-Bontebok Elaphodus cephalophus-Western tufted deer Gazella dama-Addra gazelle Gazella dorcas-Dorcas gazelle Gazella leptoceros-Slender-horned gazelle Gazella rufifrons-Red-fronted gazelle Gazella soemmerringi-Soemmerring's gazelle Gazella thomsonii-Thomson's gazelle Hemitragus jemlahicus-Himalayan tahr Mazama americana-Mexican Red brocket Muntiacus muntjac-Indian muntjac Neotragus moschatus-Zulu Suni Ovis aries-Mouflon Ovis canadensis-Bighorn sheep Oryx dammah-Scimitar-horned oryx Pudu mephistophiles-Northern pudu Tragelaphus eurycerus-Bongo Tragelaphus spekei-Sitatunga

comes readily available, this Betadine infusion technique provides a practical, effective, and inexpensive method to treat the diffuse cellulitis associated with lumpy jaw infections while eliminating the continued expansion of the alveolar osteomyelitis.

The second phase of the protocol is scheduled when the alveolar osteomyelitis has been eliminated or is contained with the dental pulp infection confined to the draining fibrous tract extending from the apex of the tooth to the exterior, or both. This phase is referred to as a compound apicoectomy, which restores the integrity of both the dentition and the alveolar bony defect, with a combination of traditional dental root apex amputation or apicoectomy copiously irrigated with chlorhexidine.¹¹ Effective endodontic therapy with access to the dental pulp chamber through the apex of the surgically prepared tooth (see Fig. 7a–c) is essential, and retro-filling of the apical aspect of the tooth root is a critical second step. It is best done with generally accepted dental restorative materials such as a glass ioniomer cement or a dental composite resin but not dental amalgam. Finally, if circumstances indicate,³ the bony defect in the jaw can be restored with one or more of the commonly accepted bony grafting materials such as Bioglass (Consil, Nutramax Laboratories Inc., Baltimore, Maryland 21236, USA). This synthetic bone grafting particulate material can be moistened with a broad-spectrum antibiotic solutions such as enrofloxacin, amikacin. Sometimes medical grade calcium sulfate (CapSet, LifeCore Biomedical, Chaska, Minnesota 55318-3051, USA) can be a very effective source of localized calcium. This bone grafting material is usually replaced with normal bone matrix within 4 mo. In some instances, the surgical access wound is allowed to granulate in, and heals naturally by second intention.

CONCLUSIONS

Chronic alveolar osteomyelitis in exotic hoof stock is, by definition, a virulent, aggressive, disfiguring, difficult-to-resolve, infective process in a fractious, frequently fragile, and often impossibleto-hospitalize endangered hoofed animal. The cure treatment is often worse than the illness because the application of many of the reasonable and clinically preferred treatment modalities frequently may results in debilitating diarrhea, repeated facial trauma, irresolvable foot abscesses, broken fractured limbs, evulsion of the lip, stress-induced abortion, antibiotic-resistant septicemia, clinical frustration, extraordinary hospitalization costs, and occasionally iatrogenic death of the patient. These bony infections are often terminal if they are not treated and resolved.

A developing clinical protocol based on proven techniques is presented, which provides the clinician with a method to treat, manage, and eliminate one of the most difficult dental infections of exotic hoofed stock. Although it is not easy to apply or guaranteed to always produce instant success, it will achieve the desired results when applied early with vigor, determination, and imagination.

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