Note: This case report is presented as an example of an accepted report that clearly demonstrates the applicant's clinical skills, and nearly meets every requirement as specified for case reports. No case report is perfect. In this case, reviewers noted that reference 7, a thesis paper, should have been listed as an endnote; also, Table 1 does not list a reference for normal values given. Other minor deficiencies are noted; however, in total deficiencies did not warrant rejection of the case report. For purposes of posting this report electronically, resolution of images is lower than that required for an actual case report.

Treatment of a retrobulbar abscess secondary to dental associated infection in a rabbit

000-000-000

INTRODUCTION

This case report describes a 13-month-old female neutered Netherland Dwarf rabbit that was diagnosed with a dental associated infection of the right maxillary third premolar. The rabbit presented with dysphagia, right ocular disease and facial swelling. The rabbit was treated both surgically and medically, and post-operatively with daily wound care. Full resolution of the infection was achieved.

Dental disease is a common problem of pet rabbits. Some authors have postulated that nearly 100% of pet rabbits will develop some form of acquired dental disease (ADD) in their lifetime[1]. Dental disease may affect rabbits of any age and gender and can be a cause of great morbidity and malaise[2]. Often dental disease may lead to infection of teeth and surrounding structures. The development of infection and abscessation often heralds a guarded to poor prognosis for resolution unless affected teeth are extracted, infected tissue is resected and supportive and medical therapy are employed[1-4].

To comprehend the pathophysiology of rabbit dental disease, a sound knowledge of rabbit dental anatomy and physiology is required. This also permits reasonable recommendations to be offered in terms of diagnosis, treatment and prognosis of dental disease conditions.

Rabbits are true herbivores. Their natural diet entails the consumption of large volumes of abrasive high fiber foods[1,2]. These foods, and the volume consumed, naturally promote teeth wear[1]. As an adaptation, rabbits have evolved ever-growing (elodont) teeth[1]. The teeth are also classified as aradicular[5] (without a root) and hypsodont (long crowned)[3]. The elodont feature leads to a dynamic state intrinsic to rabbit dentition which may clinically serve to complicate dental disease syndromes[2]. This type of dentition also allows for a concurrent increase in teeth size with growth of the animal[2].

The rabbit dental formula[6] is 2(I2/1 C0/0 PM3/2 M3/3) = 28. It is useful clinically to divide rabbit dentition into two groups, the incisor teeth set and cheek teeth set (premolars and molars)[3]. Each cheek teeth arcade can be divided into quadrants (upper and lower on right and left). The rabbit mouth also features a relatively long diastema and anisognathism (the lower jaw is narrower than the upper jaw[6]. The maximum open-mouth gape of a rabbit is only 20-25°, (compared to a rat, which is 40°)[7]. This coupled with the long diastema can make inspection of the oral cavity relatively difficult[2].

Each tooth can be further divided into the clinical crown (region of tooth exposed above the gingival margin) and the reserve crown (region of tooth buried below the gingival margin)[3]. The growing portion at the tip of the reserve crown is the apex, which is open in rabbit teeth.

Incisors have a single pulp cavity[3]. The cheek teeth have a single pulp chamber at the tooth apex which diverges into two towards the clinical crown[2].

Enamel is present only on the labial side of the strongly curved maxillary incisors, while the mildly curved mandibular incisors feature enamel on both the labial and lingual aspects of the teeth[6]. Through normal wearing, this feature produces the characteristic chisel shape to the clinical crown tips (in occlusively normal incisor teeth)[6]. Rabbits also periodically 'grind' their teeth to help shape their incisor tips to their characteristic chisel shape [7]. In normal resting occlusion, the mandibular incisor tips lay just caudal to the main maxillary incisors, in the space between the primary maxillary incisors and the peg teeth[6].

In contrast, the cheek teeth are longitudinally straighter and somewhat 'folded' on the buccal side in cross section[2]. This formation creates an increase in the proportion of enamel on the occlusal surface of the tooth (providing increasing grinding efficiency and aiding in reduction of wear rate)[2].

At rest, the opposing cheek teeth quadrants are normally in contact with each other[7]. This observation contrasts to what many authors previously described, where they stated that at rest, the rabbit's opposing cheek teeth quadrants do not normally contact each other[1,8]. In fact, in the normal resting closed mouth gape, both the opposing incisors and cheek teeth are in contact with each other[7].

The rate of teeth growth varies between the different teeth sets and can be influenced by age, pregnancy and diet [7]. The upper incisors for example grow slower than the lower incisors, at rates of 2.0 and 2.4mm/week respectively[9].

Rabbits primarily use a vertical action to 'cut' foliage with their incisors[1,7]. Once typical leaf material is present in the mouth, it is then masticated largely in a horizontal or lateral plane by the cheek teeth. This is important to consider, as dietary factors have been shown to affect a rabbit's normal chewing process[10]. Food is ground by only one side of the cheek teeth at a time[2]. Natural vegetation such as grass compliments a normal horizontal chewing action of the

cheek teeth, while harder and thicker food items (such as pellets, grains or carrots) tend to encourage more vertical and less horizontal movements in mastication[10]. This vertical action can lead to a reduction in tooth wear and also potentially increase forces on teeth in the vertical plane, producing increased pressure on the growing apex of the tooth[3].

There are many proposed causes of dental disease in rabbits. These can include congenital anomalies and acquired dental disease. Congenital causes can include maxillary brachygnathia, mandibular prognathism and jaw or teeth malformation[1,11]. Acquired causes can include trauma, inappropriate nutrition[1], metabolic bone disease[2,7,16,17] ,trauma and neoplasia[1,12,]. These etiologies may occur individually or in combination.

Normally, the continual processes of growth, dental attrition and dietary abrasion shape rabbit's teeth. Some authors suggest that the effects of improper or insufficient wearing of the teeth have the greatest impact on the formation of dental disease in rabbits [1,3,8,14,13]. This interpretation is supported by the "inappropriate diet" theory that most pet rabbits are fed inappropriate diets that do not simulate the abrasive nature of a natural grass diet. Instead, many pet rabbits are offered concentrate feeds which alter normal masticatory movements[1]. As teeth are insufficiently worn by these inappropriate diets, the resultant teeth elongation further reduces proper masticatory capacity[1]. Ultimately, increased pressure applied to the elongated cheek teeth during mastication results in their increased curvature and gradual movement into supporting bone[1].

The "inappropriate diet" theory in part relies upon the 'non-contact between cheek teeth at rest' premise[1,8]. As the cheek teeth are allowed to elongate, their normal resting position alters. Instead of being separated at rest, the upper and lower quadrants now come in contact at their occlusal surfaces. Gradually this is believed to lead to a widening of the normal 'closed' mouth gape of the rabbit and eventually, resting jaw tone prevents further tooth eruption[8]. A slowing of

apical growth coupled with a lack of eruption and increased pressure is believed to lead to intrusion of the apices into underlying apical jaw bone[8].

Yet another hypothesis, the metabolic bone disease theory, states that metabolic bone disease may play a major role in the development of ADD in pet rabbits[2,7,16,17]. Some studies have found certain metabolic bone diseases such as nutritional secondary hyperparathyroidism (NSHP) to be a possible factor in formation of ADD in pet rabbits[2,7,16,17]. These studies suggest that the early loss of supporting alveolar bone (due to underlying NSHP) at the apex of the teeth may allow their intrusion into surrounding bone[7]. This may especially be seen in the lower cheek teeth where the teeth apices can eventually penetrate the ventral mandibular cortex[7]. The associated weakness of surrounding bone affected by NSHP may lead to distortion of the normal tooth socket[7]. Subsequent alteration in teeth shape (increased curvature) and orientation may then lead to cheek teeth malocclusion[7]. Further loss of alveolar bone may then initiate loosening of teeth and widening of periodontal spaces[7]. This in turn may instigate the development of secondary infections, subsequent osteomyelitis and the development of periapical abscessation[7].

Any cause of dental disease that leads to derangements in tooth or teeth (shape, size, structure, constituents) and associated supporting structures, may then lead to improper mastication and ineffective wearing. This may then result in disease in other teeth[7].

The primary skeletal abnormalities that can contribute to ADD such as maxillary brachygnathia and mandibular prognathism have been suggested to occur more commonly in dwarf or lop-eared breeds[8]. In one study, there appeared to be no significant relationship between breed of rabbit and dental disease[7]. Interestingly however, in the same study, significantly more male rabbits than female rabbits had shown signs of dental disease[7].

Regardless of the precise cause of dental disease, the deviation of overgrown cheek teeth results in several possible predictable sites of apical eruptions of teeth through associated cortical bone. In most instances, all cheek teeth apices erupt through their associated cortical bone laterally except for the first and last mandibular cheek teeth, which more often tend to erupt medially[2].

The formation of infection and abscessation associated with ADD in rabbits may have several causes including endodontic infection, penetrating foreign bodies, trauma to teeth or jaw and hematogenous spread[1]. In rabbits affected with ADD, it is not always possible to define what has lead to the formation of infection. Abscessation involving the tooth apex and periapical tissue, progress to osteomyelitis of the supporting alveolar bone, soft tissue infection and abscess formation may all subsequently result from infection[2].

In regards to the development of tooth infection, one theory put forward is the creation of periodontal pockets from loss of periodontal ligaments secondary to periodontitis[1]. Periodontitis is normally rare in rabbits, but its incidence may increase, as tooth growth is reduced[1]. Once formed, these periodontal pockets may then become inhabited by bacteria, initiate disease and spread to the apex [1].

In another theory for formation of infection, it has been suggested that in some cases of ADD, accompanying alveolar bone demineralization may lead to widening of the periodontal spaces and subsequent loosening of the teeth[7]. This phenomenon may then allow bacteria and food particles to enter the periodontal space and generate periodontitis. Progression of this may lead to the development of periapical infection[7].

Oftentimes, when dental associated abscess form, they do so with a well-developed capsule[2,3]. Within the abscess may be contained combinations of purulent material, necrotic and infected

tissue. Important features of dental associated abscesses in rabbits are the abscess capsule, necrotic tissue (dental, bony or soft tissue) and osteomyelitis[4].

Dental abscesses in rabbits may harbor both mixed aerobic and anaerobic bacteria. *Fusobacterium nucleatum*, *Actinomyces* spp., *Streptococcus* spp., *Peptostreptococcus* spp., *Prevotella* spp., *Pasteurella* spp., *Staphylococcus* spp., *Pseudomonas aeruginosa*, *Enterococcus* spp., *Bacteroides* spp. and other microbes have all been isolated from rabbit dental abscesses[18-20]. This knowledge should influence the type of antimicrobial therapy used ensuring at least that an antibiotic with an anaerobic spectrum is used[2].

Dental disease in rabbits can be graded based upon features such as tooth elongation, malocclusion, tooth growth and the presence of infection[2,7,11,21]. The prevention (or at least minimization) of dental disease in rabbits is likely to center on sound nutritional advice. Access to sunlight may also play a role if metabolic bone disease is an issue. On the veterinarian's part, early recognition of dental anomalies is an important factor in advising on frequency of dental checks.

The typical history of dental disease in rabbits can vary widely. Many of the signs associated with dental disease in rabbits may go unnoticed for some time. This is in part due to the nature of the rabbit as a prey species masking clinical signs of illness[2]. Even in more severe cases when infection or abscessation are present, the typical signs presented in many other species affected by abscessation of any kind (malaise, pyrexia, pain, etc) are often not presented in rabbits[2,3].

Primary signs of dental disease may include[2,3,7,14] malocclusion, dental asymmetry, tooth disfigurement/dystrophy, dental overgrowth and dental spur formation. Enamel dysplasia (enamel ribbing of the incisors) may indicate underlying metabolic bone disease[3,7].

When incisor teeth malocclude and overgrow, they usually follow a particular pattern of overgrowth. The lower incisors tend to protrude rostrally (unimpeded by lack of wear from the upper incisors) [7]. The main upper incisors tend to curl in a caudal direction[7]. The peg teeth will often grow ventrolaterally[7].

With cheek teeth, the array of maldirectioned growth is a little more variable. However, the production of dental spurs that impinge on either the tongue or cheeks is a little more predictable[2]. The maxillary cheek teeth tend to maldirect towards and produce spurs on their buccal sides[2,3]. Hence buccal abrasions may be seen in this scenario. Conversely, the mandibular cheek teeth tend to maldirect towards and produce spurs on their lingual sides[2,3]. Hence lingual abrasions may be seen. In severe cases, medial spurs of the lower cheek teeth may elongate sufficiently to cause laceration of the lingual artery and lead to a fatal hemorrhage[7].

Diseased cheek teeth also often exhibit ‘step-mouth’ or ‘wave-mouth’ which represents the pattern of unevenness of the occlusal plane of any one of the cheek teeth quadrants. This can occur due to uneven wear or uneven growth of neighboring teeth[2].

The secondary signs[2,3,7,11,14,22] that may alert to the presence of ADD may include, but are not limited to; inappetence, dysphagia, loss of body condition, weight loss, change in dietary preference, halitosis, chin wetness/salivation, epiphora/ocular discharge, exophthalmos, nasal discharge, dyspnea, facial masses/swellings, altered mandibular movements, altered gape, perineal soiling (faecal and/or urinary), uneaten cecotrophs, change in fecal consistency and quantity, unkempt coat/lack of grooming and wet/stained forearms,

At physical examination, the face and jawline should be palpated carefully for any unevenness, swelling, discomfort or asymmetry[3]. Any swelling of the face or jaw should include the possibility of ADD and dental abscessation as high on the list of differential diagnoses.

Swellings associated with abscessation may be firm or soft, but usually appear non-painful to touch[2]. Often a ventral mandibular abscess may be hidden by the dewlap, hair growth or by the normal head carriage of the rabbit[2]. Occasionally, a ruptured abscess may be featured by regional wetness and/or foul odor. The size of the facial abscess may not always give an indication of the severity or prognosis of the underlying dental disease problem[3].

Other important differentials to dental disease can include any source of oral or facial pain resulting from trauma, soft tissue ulceration or neoplasia. Other causes of masses and swellings on the face may include infection/abscessation non-dental in origin and neoplasias of cutaneous[23], boney or soft tissue origin[12,24-27]. With unilateral exophthalmos differentials include[2,20,28,29] retrobulbar abscess, orbital abscess, neoplasia, hemorrhage, salivary mucocele, lacrimal apparatus disease, cellulitis, foreign body penetration, trauma, granuloma and cystic structures (including cysticercosis).

Performing full dental examinations in the conscious rabbit can prove difficult. There is often a need to utilize sedation or general anesthesia to facilitate oral inspection[2,3]. Examination of the incisor teeth may be achieved by gently retracting the lips. Inspection of the cheek teeth is more difficult due to many factors such as small oral opening, long diastema, relatively small gape and difficulty in safely restraining the rabbit for oral examination[2]. The use of otoscopes can aid examination of the first few cheek teeth. More specialized equipment such as the human bivalve nasal speculum[a] can facilitate cheek teeth inspection by allowing retraction of the buccae and lingua away from the dental arcades. In sedated or anesthetized animals, inspection can be enhanced by use rabbit specific instrumentation e.g., mouth gags, cheek dilators, tabletop positioning stands[3]. The use of oral endoscopy in anesthetized patients may offer a more detailed dental assessment[3,20,30,31],

Imaging techniques are required to diagnose underlying lesions. The employment of radiographic imaging may be considered a primary diagnostic tool in diagnosing dental and

skull lesions[2,3,7,32-36]. The minimum radiographic projections required include; lateral, left and right lateral obliques and dorsoventral or ventrodorsal views[2,3,35,35]. In the lateral view, the overall impression of the shape of the skull is attained. Ideally the tympanic bullae should be superimposed in this projection[3]. The overall radiodensity of the skull bone can be assessed for evidence of osteopenia, though this can be difficult to quantify without comparison to normals[7]. Radiodensity can sometimes be examined by viewing the line of incisive bone at the rostral portion of the hard palate or the ramus of the mandible[7]. In the lateral view, the incisor teeth occlusion can often be assessed (with mouth closed)[7].

Some authors consider that a lateral projection may allow for assessment of degree of gingival elongation along the cheek teeth crowns[3]. The lateral view also allows for assessment of the maxillary diastema (hard palate). In this view, the apex of the maxillary incisors may be assessed for their potential incursion into the incisive bone of the hard palate[3,7,35].

The cheek teeth normally occlude in a 'zigzag' array[2]. The occlusal surface of the cheek teeth may reveal unevenness, spurs or malformed clinical crowns indicative of dental disease and overall cheek teeth crown length can be assessed[3,7].

In the lateral oblique skull views, both left and right projections are necessary. These views reveal a hemimandible with associated cheek teeth set and its contralateral maxillary cheek teeth quadrant[3]. Each cheek tooth may be identified and assessed individually[3]. Evidence of deformities, curvatures, misalignments, dystrophic calcification, resorptive lesions and erosions can be examined[3,7]. Similarly, each incisor tooth may be individually identified without the effects of superimposition as in the lateral view[3]. The incisors can be examined for evidence of enamel ribbing, while the reserve and clinical crowns can be inspected for elongation[7]. If present, evidence of dystrophic calcification or closure of the pulp cavity may also be localized in the incisors with this view[7]. It is thought that the development of incisor abnormalities follows

a course of elongation, closure of the pulp cavity and then dystrophic calcification of the reserve crown and surrounding bone[7].

With the oblique projections, the interproximal spaces between the cheek teeth can be assessed[3]. These spaces tend to widen with the presence of dental disease in rabbits[7]. The apices of each tooth can be closely examined for evidence of surrounding osteolysis, dystrophic calcification, elongation and penetration into surrounding bone[3,7]. In cheek teeth it is elongation of the reserve crown that appears to be the first change to take place in all rabbits with ADD[7]. Second to this is the loss of the longitudinal radiodense enamel fold of the cheek teeth[7].

A minimum oblique angle of no greater than 30˚ is suggested for lateral oblique views[3]. This especially allows for the vital assessment of the ventral border of each hemimandible[3]. In dental diseased rabbits, the lower cheek teeth apical extremities often show penetration into the ventral mandibular cortex[7]. The loss of the line of the lamina dura at the extremity of the dental socket occurs before this and is thus indicative of early dental disease[7]. As dental disease progresses, the loss of alveolar bone results in a mergence of these sockets and in later stages can lead to their amalgamation[7].

In the dorsonventral/ventrodorsal skull projection, some cases may enable the detection of cheek teeth spurs despite superimposition of maxillary and mandibular cheek teeth sets[2]. The first maxillary premolars can be viewed without superimposition[2]. Their associated surrounding lamina dura should be clearly visible bilaterally as a radiodense line in normal skulls[2]. Similarly, the incisor reserve crowns can be viewed, as can part of the nasal cavity which may be compared bilaterally[2]. The outlines of the mandible can be partially inspected. The zygomatic arches can be inspected for radiodensity and thus may give an indication of presence of osteopenia[7].

Contrast dacryocystorhinograms may be conducted to assess the integrity and patency of the nasolacrimal duct[2]. Advanced imaging via computed tomography is an emerging field in rabbit dental disease diagnostics and may offer more accurate diagnosis and subsequent treatment planning[3,34,36,37]. In cases where orbital disease and exophthalmos are present, ultrasonography (periorbital, ocular) may be employed[19,20,28].

Routine blood biochemistry, hematology and urinalysis do not often contribute to the diagnosis of dental disease in rabbits. However they may aid in the diagnosis of concurrent disease. In one study, most rabbits with dental disease (with or without infection) were found to be mildly anemic[17].

Fine needle aspiration or biopsy of a facial mass that has any degree of suspect underlying dental infection is generally not recommended. Confirmation of dental infection should be made radiographically and not rely just on the aspiration of pus. Similarly, attaining pus samples for microbial culture and sensitivity by these means may not yield effective results due to the likelihood of sampling sterile pus[3]. Furthermore, the break in integrity of the abscess capsule may result in loss of containment of infection and compromise future surgical resection. Samples for culture and sensitivity are likely to be best attained at time of surgery when a piece of the abscess capsule can be collected and submitted for analysis[3,4].

Treatment options for dental associated infections and abscesses can be broadly separated into treatment of the infection and associated dental disease and the management and supportive care of the patient.

The treatment of dental abscesses may be very difficult and lengthy processes[2-4]. Many factors need to be considered for any patient entering dental abscess treatment, especially overall quality of life. Similarly, attentive client compliance and commitment to post operative care are essential to enhance good recovery[3]. Surgical procedures may be extensive, lengthy,

complicated and may require intensive nursing of the patient immediately post operatively[3,4]. In some cases, several successive surgeries may be necessary[3]. The presence of dental abscessation is generally associated with the presence of quite advanced ADD[2]. The possibility for further dental problems to exist concurrently, recur at the infected area, or arise in new areas needs to be considered[2].

Medical therapy alone is largely considered ineffective or palliative[2-4]. There have been no controlled studies reported on the use of antimicrobials in rabbit dental infections. Penicillin G derivatives have been widely used in the treatment of rabbit dental infection due to their activity against typical bacteria expected in dental abscessation. Pharmacokinetic studies of penicillin G in rabbits have been carried out[38].

There are many surgical interventions and treatments described for rabbit dental abscessation. By nature, rabbit pus is of a caseous consistency, making simple open drainage less useful in achieving successful outcomes[2,3]. The placement of wound drains does not appear to produce suitable drainage in rabbit dental abscesses[2]. Many techniques describe the surgical resection of the abscess capsule in conjunction with instilment of the surgical site with various compounds such as clindamycin powder, clindamycin capsules, antibiotic impregnated polymethylmethacrylate beads (AIPMMA beads)[39,40], calcium hydroxide[2,41] , antibiotic preparations such as doxycycline-containing polymer gel [1,19] , sugar and honey solutions[2,42] and bioactive ceramics [40]. All of these treatment modalities have reported varied success rates.

The surgical approach with reported good success rate at healing and resolving dental associated infections relies on the principle of total resection of the abscess and any associated necrotic and infected structures (teeth, bone, soft tissue), suturing the wound open and then allowing the wound to heal by assisted secondary intention[3,4]. The purpose of this approach is to minimize the recurrence of infection by attempting to eliminate infected tissue and avoiding closure of infected wounds[3,4]. This approach is compared similarly to that of the management

of a grade 3 open fracture and osteomyelitis[4]. Follow up wound care (flushing, debriding, suture removal) are generally required for 2 to 4 weeks following surgery[4]. There is little mention of the requirement of use of Elizabethan collars in rabbits post-operatively from these procedures. This is fortunate as rabbits generally require close supervision when Elizabethan collars are fitted. Their presence is often not well tolerated and can prevent the rabbit's consumption of cecotrophs[43].

In terms of surgical difficulty and prognosis for successful resolution, the tooth or teeth associated with the infection and the extent of the infection are important factors[3,4]. Infection associated with the caudal cheek teeth (mandibular cheek teeth 4 and 5, and maxillary cheek teeth 3 to 6) are considered more difficult to access anatomically[3,4]. Anatomical traits that affect access to the last two lower cheek teeth are the presence of the masseter muscle and relative thinness of the mandible in this area[4,44,45]. Anatomically, the last 4 maxillary cheek teeth apices and reserve crowns are located in the ventral bony orbit surrounded by the alveolar bulla of the maxilla[4,44,45]. The alveolar bulla can act as a bony cavity to any periapical infection of the teeth it contains[4]. If infection erupts through the thin bone of the alveolar bulla, retrobulbar infection of soft tissue results[4]. Surgical access to this area is impeded by the eye, periorbital tissues and zygomatic arch. Enucleation, although not ideal, may be required to access this area[1,4,17]. Alternatively, partial resection of the zygomatic arch has been reported to access this area[4], as has a lateral canthotomy procedure[46]. An intraoral approach utilizing endoscopic visualization of the cavity subsequent to extraction of the diseased maxillary cheek tooth has also been described[20].

The overall prognosis for treatment retrobulbar infection in one review was considered guarded to grave[32]. Suggested treatments include enucleation to allow access to the retrobulbar space. Endoscopic assisted methods, both intraoral[21] and extraoral[19] have been utilized in attempt to improve abscess access and visualization and negate the need for enucleation.

CLINICAL REPORT

A 13-month-old female entire Netherland dwarf rabbit presented with a one week history of a reddened right eye and reduced appetite. She had been seen previously at age 6-months for routine neutering. At physical examination, the rabbit was fairly bright, active and responsive. She weighed 1.45 kg and her body condition score was 3/5. Her vital signs were within normal limits. Examination of her right eye revealed mild epiphora, conjunctivitis and subtle exophthalmos and no discernable strabismus. No facial asymmetry could be readily appreciated visually apart from the exophthalmos. Upon facial palpation, a firm, regular, fixed swelling of approximately 10mm diameter was detected ventral to the medial canthus of the right eye. Ophthalmic examination via indirect ophthalmoscopy revealed no anomalies. Intraocular pressures were within normal limits (16mmHg in the right eye and 18mmHg in the left eye). Fluoroscein staining of the right eye yielded no evidence of corneal lesions and her ipsilateral nasolacrimal duct was deemed patent due to presence of dye at the ipsilateral nostril within 1 minute. Incisor occlusion was normal. A brief inspection of her oral cavity with a human bivalve nasal speculum revealed no cheek teeth anomalies, soft tissue abrasions, halitosis or increased salival levels. The remainder of her physical examination was unremarkable. Her usual diet and husbandry was considered satisfactory.

The differential diagnoses for the exophthalmos were retrobulbar abscessation, cyst (including cysticercosis), hemorrhage, granuloma, cellulitis, neoplasia, salivary mucocele, lacrimal apparatus disease, foreign body penetration and trauma.

The rabbit was sedated with fentanyl[b] 0.02mg/kg and midazolam[c] 0.5mg/kg via intramuscular (IM) administration. A more thorough oral inspection with the bivalve nasal speculum revealed no anomalies. Skull radiographs were taken (lateral, left and right lateral oblique and dorsoventral projections). The major findings were dental associated infection of the right maxillary third cheek tooth with widening of the interproximal space and curved deviation of the

neighboring second and fourth cheek teeth seen in the right dorsal-left ventral oblique projection (Fig 1). In the dorsoventral projection, the lateral wall of the right maxilla and zygomatic arch revealed some destruction and reactive bony proliferation (Fig 2). Ocular ultrasound examination was performed and revealed a hypoechoic focus ventromedial to the globe approximately 6.7mm in diameter (Fig 3).

Further diagnostics via blood biochemistry and hematology analyses were out-sourced after blood collection from the lateral saphenous vein. The blood results (Table 1) indicated a mild anemia - hematocrit 0.32L/L (normal 0.35-0.48L/L). Red cell morphology was normochromic with mild anisocytosis. The remainder of the biochemistry and hematology values were within normal limits apart from a mild increase in creatinine kinase (CK), 422IU/L (normal 140-372IU/L) which may have been attributed to handling and restraint for diagnostic tests.

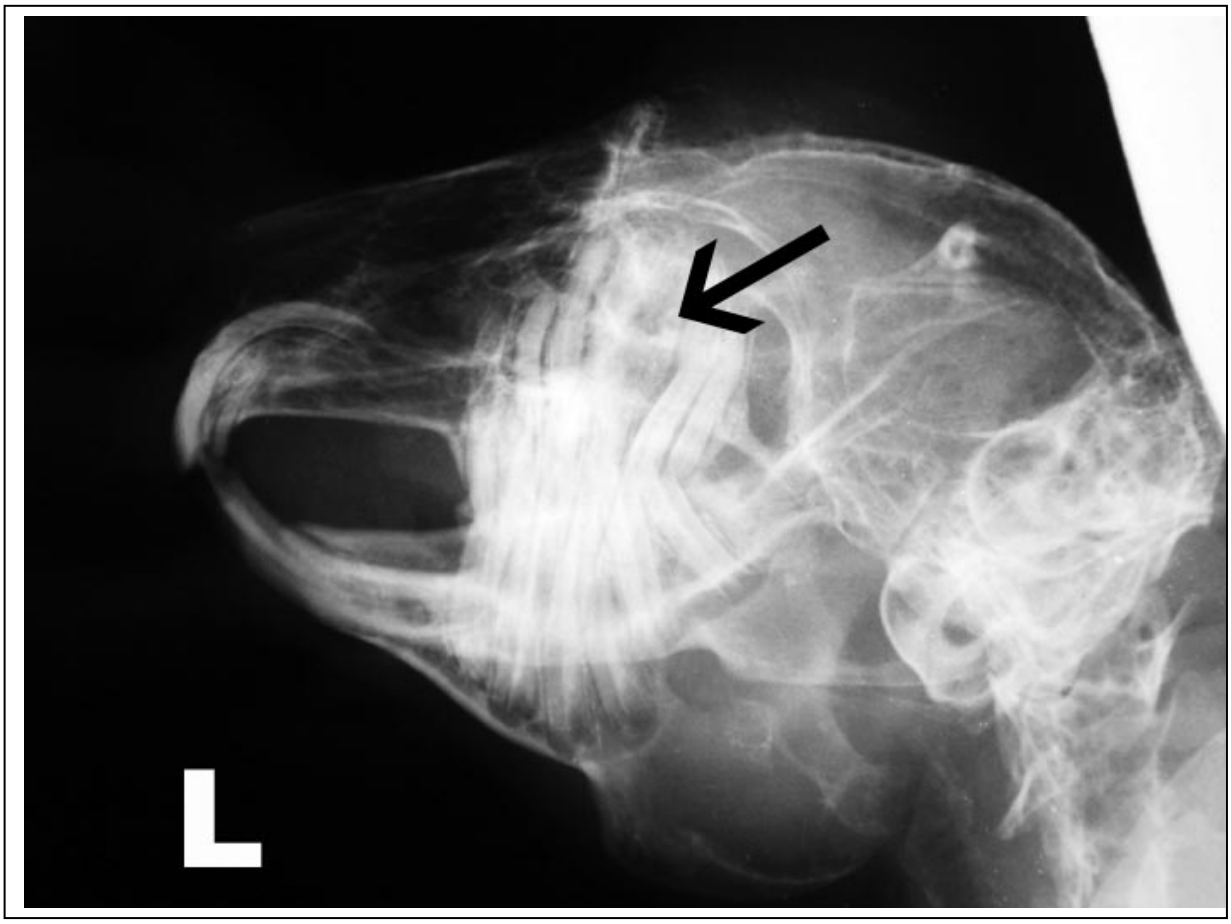

Figure 1. Right dorsal-left ventral oblique skull radiograph. Infection of the right maxillary third cheek tooth is evident with bowing of the second and fourth maxillary cheek teeth and widening of the interproximal space between these teeth (black arrow).

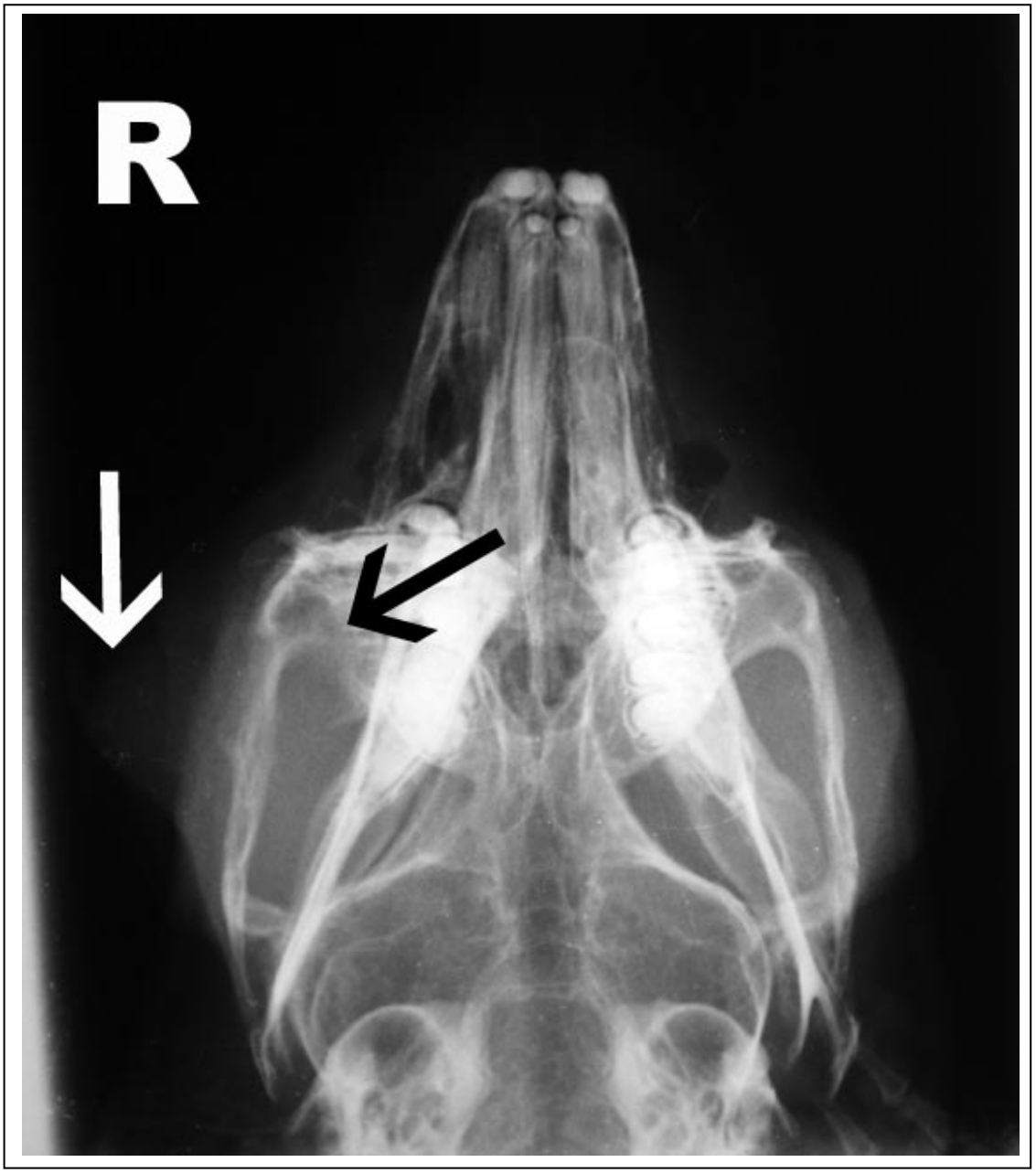

Figure 2. Dorsoventral skull radiograph. Moderate destruction and boney proliferation of the lateral wall of the right maxilla and zygomatic arch is evident (black arrow). Exophthalmos of the right eye is also evident (white arrow).

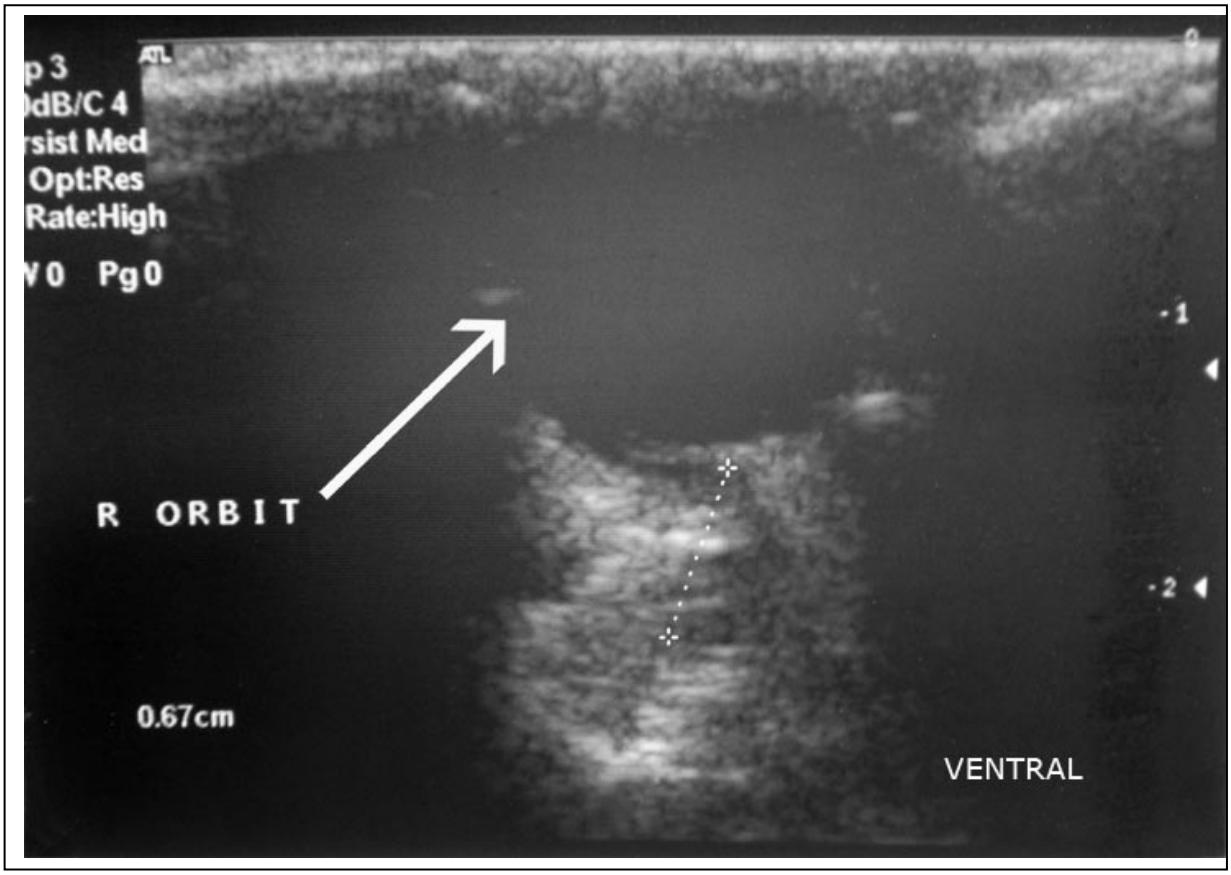

Figure 3. Ultrasonographic image of right eye. The retrobulbar hypoechoic focus 6.7mm in diameter (dotted lines) is evident ventral to the globe.

Table 1. Rabbit blood biochemistry and hematology results

TEST	RESULT	REFERENCE RANGE	
RBC	5.8	4.8 - 7.3 x10 ^12 /L	
HAEMOGLOBIN	133	103 - 155 g/L	
HAEMATOCRIT	0.32	0.35 - 0.48 L/L	LOW
MCV	67	65 - 76 fL	
MCH	23	20 - 25 pg	
MCHC	330	295 - 339 g/L	
PLATELET COUNT	127	103 - 583 x10 ^9 /L	
WCC	6.1	3.4 - 16.2 x10 ^9 /L	
HETEROPHIL %	22	%	
HETEROPHIL	1.3	0.7 - 10.2 x10 ^9 /L	
LYMPHOCYTE %	65	%	
LYMPHOCYTE	4	1.5 - 8.8 x10 ^9 /L	
MONOCYTE %	11	%	
MONOCYTE	0.7	0 - 1.4 x10 ^9 /L	
EOSINOPHIL %	0	%	
EOSINOPHIL	< 0.1	0 - 0.5 x10 ^9 /L	
BASOPHIL %	2	%	
BASOPHIL	0.1	0 - 0.8 x10 ^9 /L	
SODIUM	143	132 - 150 mmol/L	
POTASSIUM	4.6	4.3 - 6.3 mmol/L	
CHLORIDE	104	85 - 105 mmol/L	
BICARBONATE	19	mmol/L	
NA:K RATIO	31.1		
ANION GAP	20.6	mmol/L	
GLUCOSE, SERUM	8.6	6.0 - 8.8 mmol/L	
UREA	6.1	3.3 - 8.1 mmol/L	
CREATININE	0.11	0.07 - 0.23 mmol/L	
CALCIUM	3.8	2.2 - 4.6 mmol/L	
PHOSPHATE	1.1	1.0 - 1.6 mmol/L	
CA:P RATIO	3.8		
PROTEIN, TOTAL	61	49 - 71 g/L	
ALBUMIN	36	27 - 36 g/L	
GLOBULIN	25	24 - 33 g/L	
A:G RATIO	2.2		
BILIRUBIN, TOTAL	0	0 - 25 umol/L	
ALP	49	IU/L	
AST	33	33 - 99 IU/L	
ALT	64	55 - 260 IU/L	
CK	422	140 - 372 IU/L	HIGH
CHOLESTEROL	1.6	0.1 - 1.9 mmol/L	
GAMMA GT	8	IU/L	

Following the imaging and blood results, a diagnosis of retrobulbar abscessation secondary to dental associated infection of the right maxillary third cheek tooth was made. Although neoplasia could not be ruled out, it seemed less likely given the patient's age.

Treatment options at this stage involved planning access to the infected tooth and associated infected tissue and abscess for resection. In an attempt to circumvent enucleation, a lateral approach dorsal to the zygomatic arch was to be carried out. A combination of intraoral and extraoral tooth extraction was planned.

The rabbit was sent home with oral meloxicam[d] analgesia 0.3mg/kg q12h and a hypromellose type ophthalmic lubricant[e] to reduce the risk of exposure keratitis to be applied every 8h to the exophthalmosed right eye pending surgery which was scheduled for four days time. Antibiosis was not instigated at this stage as samples for cytological and microbial culture and sensitivity were scheduled for collection at time of surgery.

On re-examination of the patient four days later, the rabbit was bright and alert. She had maintained a moderate appetite. The facial swelling was now more pronounced (about 20mm diameter) and could be appreciated without facial palpation. Her exophthalmos had remained stable.

The rabbit was premedicated with fentanyl[b] 0.025mg/kg, ketamine[f] 5mg/kg and glycopyrrolate[g] 0.02mg/kg all via IM administration. The cephalic vein was cannulated with a 24 gauge IV catheter and the rabbit was commenced on perioperative crystalloid fluid therapy at a rate of 10mL/kg/h. The rabbit was pre-oxygenated with 100% oxygen for 5 minutes via face mask, 5 minutes after premedication. A 2.5mm uncuffed endotracheal tube was passed blindly into the trachea and she was maintained on isoflurane[h] and oxygen. Ocular lubricant was placed into both eyes. Intraoperative regional analgesia was provided by application of bupivicaine[i] at less than 1mg/kg over the surgery site. The anesthesia and recovery was smooth. Post-operatively

the rabbit was offered analgesia via buprenorphine[j] 0.03mg/kg and meloxicam[l] 0.5mg/kg both by subcutaneous (SC) injection.

The rabbit was placed in left lateral recumbency on a heating pad for the surgery. A lateral approach was made over the facial swelling (Fig 4). The abscess capsule was encountered and dissected free from surrounding tissue up to the level of the maxilla. The dorsal rim of the zygomatic arch was encountered and was found to be relatively soft and was partially debrided with rongeurs thus improving access. A piece of abscess capsule was collected and a swab taken for cytological and microbial analysis (after this point procaine benzylpenicillin[k] 60,000IU/kg was administered IM). Further entry into the abscess capsule was made (Fig 5). Pus and debris were gently extracted utilizing a bone curette. A connection to the alveolar bulla was identified with a 3mm perforation present in its lateral wall. Further excavation of the bony wall with an 18 gauge hypodermic needle and flushing with sterile saline revealed the lytic remains of the reserve crown of the right maxillary third cheek tooth which were removed with further curettage and flushing.

The rabbit was placed into sternal recumbency and had its mouth held open with cheek dilators and mouth gag. The right maxillary third cheek tooth was identified and the soft tissue attachments were loosened using a Crossley cheek teeth luxator[m]. The remaining part of the tooth was extracted intraorally utilizing cheek teeth extraction forceps[n] and was found to have pus and debris in its shortened reserve crown.

The rabbit was placed back into left lateral recumbency. The cavity was flushed further with saline. To aid in exploration of the cavity, a 1.9mm rigid endoscope was inserted into the space. A communication with the oral cavity was confirmed (oro-orbital fistula) and also enabled identification of further areas for debridement. Following further curettage and saline flushing, the cavity was flushed with 0.05% chlorhexidine[o] solution.

Figure 4. Facial mass in patient representing abscessation and extension of infection from infected upper third cheek tooth.

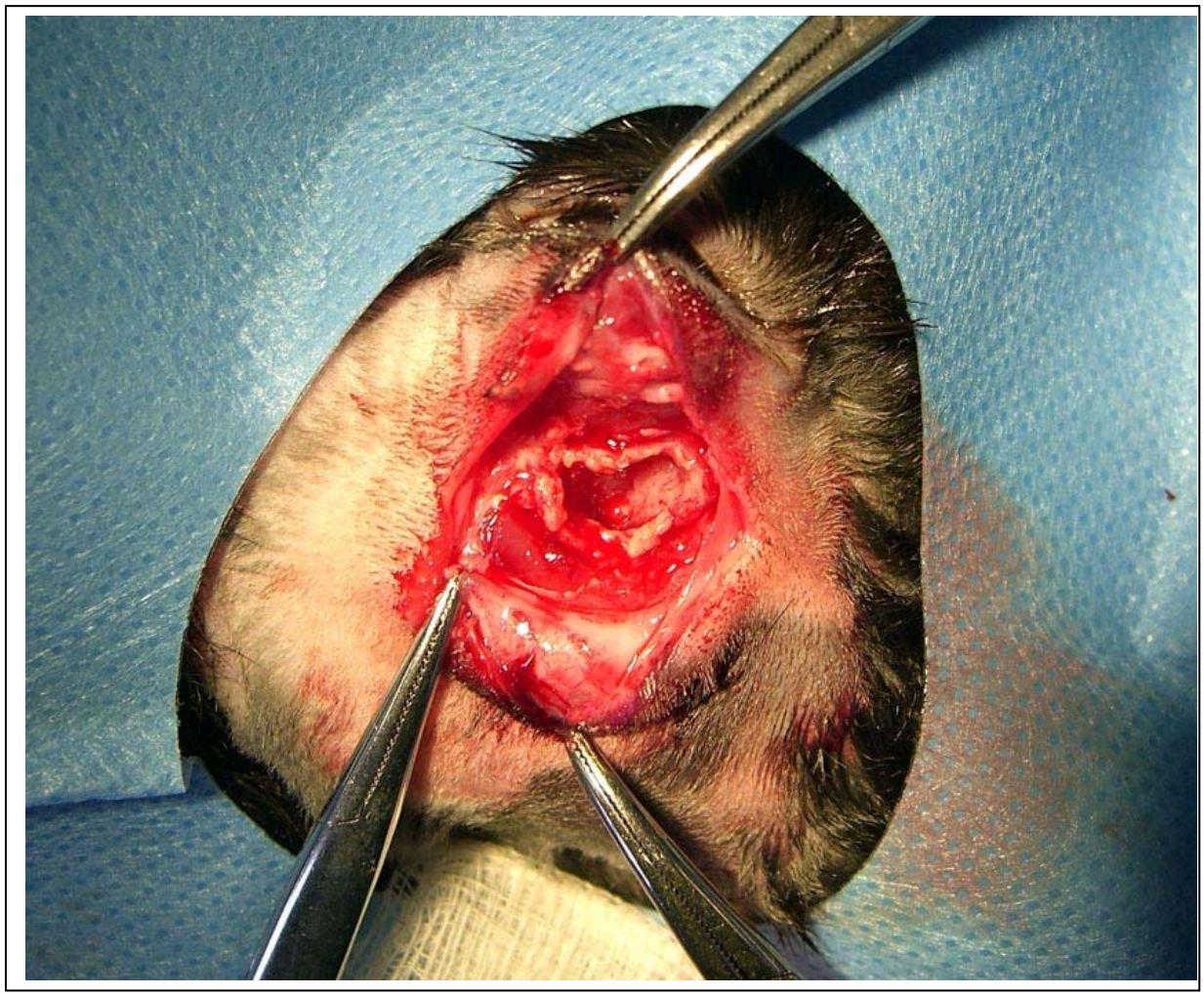

Figure 5.
Intraoperative photograph depicting opened facial abscess. Part of the abscess capsule is visible centrally in the wound

Due to the presence of an oro-orbital fistula through the alveolar bulla a decision was made to place an indwelling tube to facilitate post-operative flushing and thus minimize food debris collecting in the site. Thus, to aid in the flushing of the wound and post-operative management, the terminal portion of a Luer lock extension tube[p] was shortened and inserted to just enter the cavity of the alveolar bulla and then tunneled caudally under the skin to exit caudoventral to the eye (Fig 6). The tube was sutured into the wound utilizing synthetic absorbable 4/0 suture. The end of the tube containing a Luer lock fitting was long enough to be secured onto the dorsal cranium (via synthetic nonabsorbable 4/0 suture) and rest between the ears. The affected area was to be left open to heal by second intention. The surgical site was marsupialized with synthetic nonabsorbable 4/0 suture in continuous pattern. Manuka honey[q] was instilled into the surgical site and the area was covered with an adhesive transparent semi-occlusive polyurethane film[r].

The cytological analysis of the swab was performed in-house (Gram and modified Wright-Geimsa stains) and revealed masses of degenerative cells and many bacteria of mixed morphotypes including gram-negative rods, gram-positive cocci and gram positive branching rods thus confirming the diagnosis of abscessation.

The rabbit was provided analgesia via buprenorphine 0.03mg/kg q8h SC for the next 24h and offered oral meloxicam 0.5mg/kg q12h for the next seven days. The wound was flushed externally with 0.05% chlorhexidine[o] twice daily and with sterile saline via the indwelling tube twice daily. The rabbit was discharged from hospital 24h after surgery with the same wound care instructions and was to be rechecked on a daily basis in the short term.

The rabbit was maintained on parenteral procaine benzylpenicillin 60,000IU q48h by SC pending the aerobic/anaerobic culture and sensitivity tests which were out-sourced. Six days after submission, the results of the bacterial culture revealed a heavy mixed growth of

Figure 6. Intraoperative photograph depicting indwelling flush tube inserted into the retrobulbar abscess site to assist with post-operative flushing.

facultative and obligate anaerobes. The only colony isolated in aerobic culture was a Pasteurellaceae type organism which was susceptible to penicillin.

The rabbit maintained a good appetite in the days post-operatively. The rabbit was actively seen to move her mouth and swallow when sterile saline was instilled into the tube. By day three, saline was seen to overflow externally over the surgery site when saline was instilled into the tube. The rabbit appeared to tolerate the indwelling tube satisfactorily for the first 4 days but unfortunately removed the tube from the wound on day five post surgery. The tube was removed in total at this point. A decision was made to exclude the use of an Elizabethan collar to avoid this dilemma due to her initial tolerance of the tube and this was weighed against her potential intolerance of the E-collar.

External twice daily flushing with 0.05% chlorhexidine was maintained up until day 18 when the formation of a hard scab in the area reduced this requirement (Fig 7). No further debridement or curettage of the wound was carried out during this time as the wound appeared clean. Her subtle exophthalmos had remained stable by day 18. The antibiotic regimen was maintained with procaine benzylpenicillin 60,000IU q48h by SC injection for four weeks and then reduced to twice weekly for the next 8 weeks. Weekly rechecks were carried out over this time. By eight weeks post surgery, there was no appreciable exophthalmus. Most of the hair had regrown at the surgery site. Antibiotics were ceased three months after surgery. Dental examinations revealed normal occlusion during this time.

The rabbit has remained clinically well for the 17 months since initial diagnosis and no further complications had been reported. Recheck examinations have been carried out every three months since the surgery.

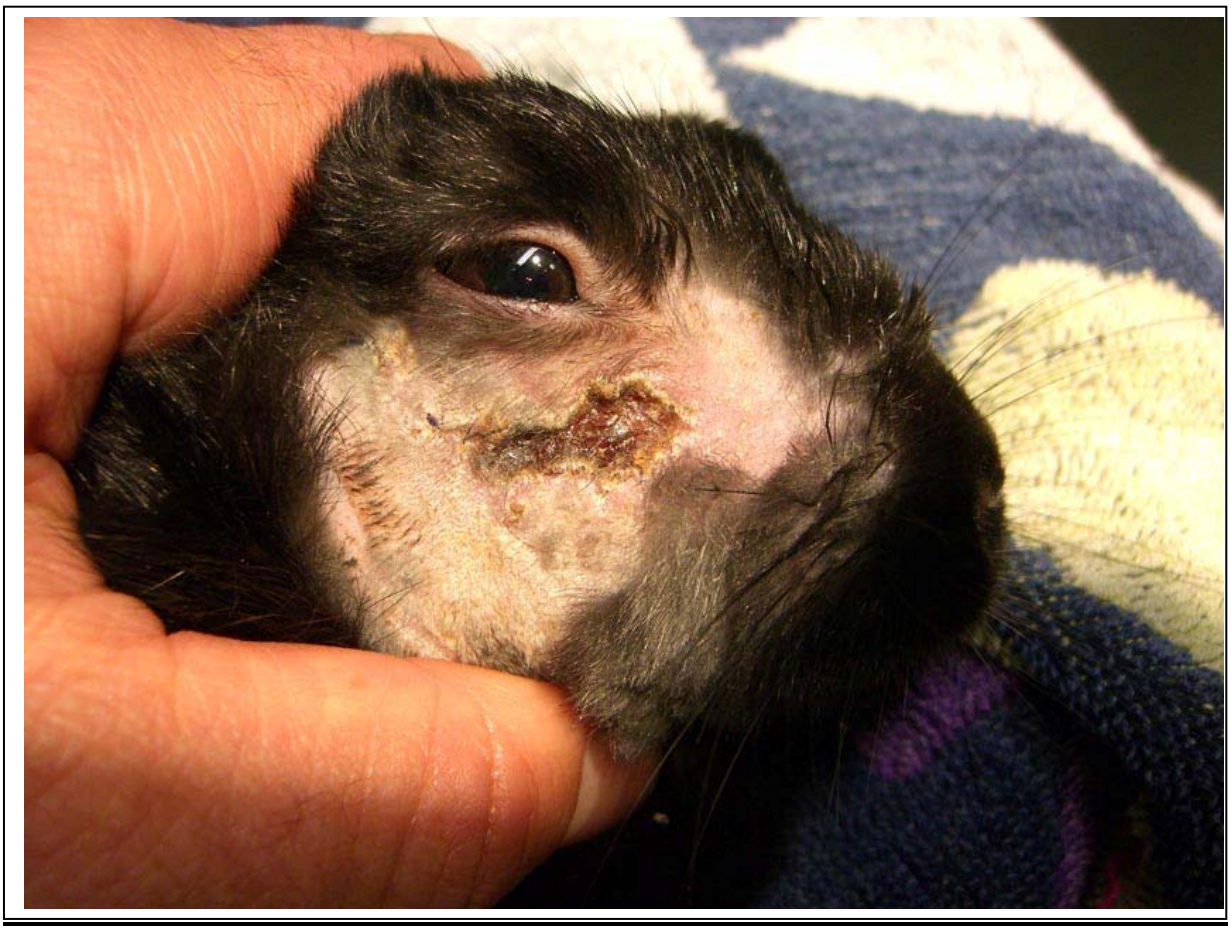

Figure 7. Surgery site 18 days post surgery

DISCUSSION

This case of a 13-month-old Netherland dwarf rabbit represents a case of dental associated infection and retrobulbar abscess that was difficult to treat due to limited access of the infected area.

The initial suspicion of retrobulbar abscessation was based on the physical exam findings. Skull radiographs supported the diagnosis of retrobulbar abscessation secondary to dental associated infection, while ocular ultrasonography added further support to this diagnosis.

A definitive diagnosis would require sampling of the mass effect. This was not carried out prior to surgery as this may have compromised the surgery process by potentially breaking the integrity of the abscess capsule which may have resulted in loss of containment of infection. Pus in these cases is often also sterile. Sample collection was scheduled during surgery so that the abscess capsule (which often yields better results) could be attained. For this same reason, antibiosis was not commenced until after samples were collected at surgery.

Advanced imaging via Computed Tomography was not performed but may have assisted in conformation of the retrobulbar abscess and also help define the extent of the abscess and bone deformation in greater detail. This would have aided the planning of surgical access.

The initial anemia was considered mild in nature and may have been a result of systemic inflammation. It was not followed up with further hematological analysis due to the positive progress of the patient, although this may have been useful to monitor in the weeks following initial diagnosis.

The decision to hold off surgery for four days was based on the bright status of the patient and hospital scheduling. In preference, the surgery would have been conducted as soon as

possible. Pending surgery, a decision was made to commence analgesia via oral meloxicam and hypromellose ophthalmic lubricant to minimize the risk of exposure keratitis. Antibiosis was held off pending sample collection at surgery.

Growth of the abscess was evident in the period from initial presentation to time for presentation for surgery, though this did not alter the surgical plan. The surgical access to maxillary cheek teeth is considered difficult without enucleation. Fortunately in this case the abscessed area was well defined and access could be simplified with partial removal of the infected zygomatic arch. Access and debridement of the source of the infection was also fortunate in that it appeared that the tooth infection had resulted in rupture of the lateral wall of the alveolar bulla, as is often predicted with this tooth. The resulting abscess had developed lateral to this making surgical resection less complicated than expected. Removal of the remaining portion of the tooth intraorally was also likely to have been less complicated due to partial loss of its reserve crown in the infected alveolar socket.

The use of rigid endoscopy was beneficial in exploring the abscess cavity and identifying remaining tooth fragments and areas to debride. The resulting oro-orbital fistula through the alveolar bulla posed potential problems for the collection of food debris and subsequent contamination of the orbit and delay in healing. The insertion of an indwelling tube to facilitate post-operative flushing of the fistula may have assisted in preventing contamination of the socket. It may have been more beneficial had the tube remained in place for longer than 5 days, however, the presence of overflow after day 3 may have suggested that the fistula was closing or becoming clogged with debris. Placement of an Elizabethan collar may have helped to prevent this, but may have also brought other complications from collar placement.

Due to the presence of the fistula, AIPPMA beads were not utilized in this case. The fistula could have potentially been closed utilizing bioactive ceramic compounds or antibiotic-containing polymer gels. Due to the introduction of the indwelling flush tube, none of these

treatment options were utilized. If the flushing had proved unsuccessful, or if the infection had returned, then repeat surgical debriding of the area coupled with closure of the fistula using these compounds would have been explored. The application of the Manuka honey and polyurethane film dressing immediately post operative was carried out only once as a post-procedural dressing. Continued external flushing of the wound with 0.05% chlorhexidine was maintained twice daily until a hard scab had formed.

Antibiosis was carried out for an extended period due to the location of the infection. Ideally, follow up radiographs of the skull could have been performed in an attempt to monitor the affected area. Repeat radiographs were not carried out due to the positive progress of the patient. Further evaluation via repeat ocular ultrasound was not considered due to lack of recurrence of retrobulbar swelling and resolution of exophthalmos.

The overall prognosis for treatment of dental associated retrobulbar infection is guarded to grave. This case represents a successful resolution of such infection sparing the need for enucleation.

SUMMARY

A 13-month-old Netherland dwarf rabbit was diagnosed with retrobulbar abscessation secondary to dental associated infection. The rabbit presented with a reduced appetite, conjunctivitis, exophthalmos and a palpable facial swelling. The diagnosis was based on skull radiographs and ocular ultrasonography and confirmed at the time of surgery with cytological and microbiological assessment. Acutely the case was managed with meloxicam and ophthalmic hypromellose. At surgery, the abscess and capsule were resected and the infected right maxillary third cheek tooth was extracted both extraorally and intraorally. External rigid endoscopy of the surgical site enabled thorough exploration of the oro-orbital fistula created during surgery. An indwelling tube was inserted into the fistula to facilitate flushing post-

surgically. Procaine benzylpenicillin antibiosis was instituted and was supported by culture and sensitivity results. Post-operative wound care was carried out and apparent resolution of the infection attained. 17 months after initial diagnosis there appears to be no further complications.

NOTATIONS

a. Human bivalve nasal speculum. Model 26030, Welch Allyn USA

b. Fentanyl injection. DBL Mayna Pharma, Acacia Ridge QLD

c. Midazolam (Hypnovel), Roche Dee Why NSW

d. Meloxicam (Metacam oral), Boehringer Ingelheim, North Ryde NSW

e. Hypromellose (Tears Naturale), Alcon, Frenchs Forest NSW

f. Ketamine injection, Parnell Labs, Alexandria NSW

g. Glycopyrrolate (Glycosate Vet), Nature Vet, Glenorie NSW

h. Isoflurane, VCA, Kings Park, NSW

i. Bupivicaine injection, Pfizer, West Ryde NSW

j. Buprenorphine, (Temgesic), Reckitt Benckiser, West Ryde NSW

k. Procaine benzylpenicillin (Propen), Troy Laboratories, Smithfield NSW

l. Meloxicam (Metacam injection), Boehringer Ingelheim, North Ryde NSW

m. Crossley cheek teeth luxator, iM3, Lane Cove NSW

n. Cheek teeth extraction forceps, iM3, Lane Cove NSW

o. Chlorhexidine (Chore C), Juror, Rutherford NSW

p. Luer lock extension tube with injection site. Codman US Corporation, Santa Anna CA

q. Manuka Honey, Nature's Goodness, Magellan NSW

r. Inclusive transparent dressings, Johnson & Johnson, Langhorne, PA

REFERENCES

1. Crossley DA. Oral biology and disorders of lagomorphs. *Vet Clint Exit Anim.* 2003; 6:629-659
2. Harcourt-Brown F. Dental disease. In *Textbook of rabbit medicine*. Oxford, UK: Butterworth-Heinemann. 2002;165-206
3. Capello V, Gracis M, Lennox AM (eds). *Rabbit and Rodent Dentistry Handbook*. Lake Worth FL. Zoological Education Network Inc. 2005
4. Capello V. Clinical technique: Treatment of periapical infection in pet rabbits and rodents. *J Exot Pet Med*. 2008; 17(2):124-131
5. Hobson P. Dentistry. In*, Manual of rabbit medicine and surgery*. BSAVA Publications, Gloucester. 2006; pp184-196
6. Crossley DA. Clinical aspects of Lagomorph dental anatomy: the rabbit (*Oryctolagus cuniculus).* J Vet Dent. 1995;12(4):131-135
7. Harcourt-Brown FM. Metabolic bone disease as a possible cause of acquired dental disease in pet rabbits. 2006. Thesis for Fellowship diploma from Royal College Veterinary Surgeons, London
8. Crossley DA. Small mammal dentistry Pt 1. In Quesenberry KE, Carpenter JW eds*. Ferrets. Rabbits and Rodents: Clinical medicine and surgery 2nd Ed*. Saunders (Elsevier) USA. 2004;370-379
9. Shadle AR. The attrition and extrusive growth of the four major incisor teeth of domestic rabbits. *J mammol*. 1936;17:15-21
10. Weijs WA, Dantuma R. Functional anatomy of the masticatory apparatus in the rabbit *(Oryctolagus cuniculus). Neth J Zool.* 1981;31:99-147
11. Verstraete FM, Osofsky A. Dentistry in Pet Rabbits. *Compend Contin Educ Pract Vet*. 2005;9:671-684
12. Whitten KA, Popielarczyk MM, Belote DA, McLeod GC, Mense MG. Ossifying fibroma in a miniature rex rabbit (*Oryctolagus cuniculus*). *Vet Pathol*. 2006; 43(1):62-64

13. Reiter AM. Pathophysiology of dental disease in the rabbit, guinea pig and chinchilla. *J Exot Pet Med*. 2008; 17(2):70-77

14. Capello V. Diagnosis and treatment of dental disease in pet rabbits and rodents: A review. *Ex Mamm Med and Surg* 2004;2(2): 12-19

15. Harcourt-Brown FM. The progressive syndrome of acquired dental disease in rabbits. *J Exot Pet Med*. 2007; 16(3):146-157

16. Harcourt-Brown FM. Calcium deficiency, diet and dental disease in pet rabbits. *Vet Rec* 1996;139:567-571

17. Harcourt-Brown FM, Baker SJ. Parathyroid hormone, haematological and biochemical parameters in relation to dental disease and husbandry in pet rabbits. *J Small Anim Prac*. 2001;42:130-136

18. Tyrell KL, Citron DM, Jenkins JR, Goldstein EJ. Periodontal bacteria in rabbit mandibular and maxillary abscesses. *J Clin Microbiol* 2002;40(3):1044-1047

19. Ward ML. Diagnosis and management of a retrobulbar abscess of periapical origin in a domestic rabbit. *Vet Clin Exot Anim* 2006;9:657-665

20. Martínez-Jiménez D, Hernández-Divers SJ, Dietrich UM, Williams CO, Blasier MW, Wilson H, Frank PM. Endosurgical treatment of a retrobulbar abscess in a rabbit. *J Am Vet Med Assoc.* 2007;230(6):868-872

21. Harcourt-Brown FM. Diagnosis, treatment and prognosis of dental disease in pet rabbits. *In Pract* 1997;19:407-421

22. Lennox AM. Diagnosis and treatment of dental disease in pet rabbits. *J Exot Pet Med*. 2008; 17(2):107-113

23. von Bomhard W, Goldschmidt MH, Shofer FS, Perl L, Rosenthal KL, Mauldin EA. Cutaneous neoplasms in pet rabbits: a retrospective study. *Vet Pathol*. 2007;44(5):579-588

24. Gillett CS, Gunther R. Mandibular mucoepidermoid carcinoma in a rabbit. *Lab Anim Science* 1990;40(4): 422-423

25. Walter JH,Gobel T. Ameloblastic fibroma of odontogenic origin in a young rabbit. [German]. *Kleintierpraxis* 1992;37(9): 633-638

26. Walberg J. Osteogenic sarcoma with metastasis in a rabbit (*Orycytolagus cuniculus*). *Lab Anim Sci*. 1981;31(4):407–408

27. Renfrew H, Rest JR, Holden AR. Extraskeletal fibroblastic osteosarcoma in a rabbit (*Oryctolagus cuniculus*). *J Small Anim Pract*. 200;42(9):456-458

28. O'Reilly A, McCowan C, Hardman C, Stanley R. *Taenia serialis* causing exophthalmos in a pet rabbit. *Vet Ophthal* 2002;5(3):227-230

29. Wagner F, Fehr M. Common ophthalmic problems in pet rabbits. *J Exot Pet Med*. 2007; 16(3):158-167

30. Hernandez-Divers SJ. Clinical technique: Dental endoscopy of rabbits and rodents. 2008. *J Exot Pet Med*. 2008; 17(2):87-92

31. Taylor M. Endoscopy as an aid to the examination and treatment of oropharyngeal disease of small herbivorous mammals. *Semin Avian Exot Pet Med* 1999;8:139-141

32. Harcourt-Brown FM. A review of clinical conditions in pet rabbits associated with their teeth. Vet Rec 1995;137:341-346

33. Crossley DA. Rodent and rabbit radiology. In DeForge DH & Colmery BH eds. *An atlas of veterinary dental radiology*. Ames Iowa State University Press. 2000:247-260

34. Silverman S, Tell LA. Domestic rabbit. In *Radiology of Rodents, Rabbits and Ferrets. An atlas of normal anatomy and positioning.* Elsevier Saunders USA. 2005:159-230

35. Gracis M. Clinical technique: Normal dental radiography of rabbits, guinea pigs and chinchillas. *J Exot Pet Med*. 2008; 17(2):78-86

36. Capello V, Lennox AM, Widmer W. Rabbit. In *Clinical Radiology of Exotic Companion Mammals***.** Blackwell Wiley USA. 2008:54-167

37. Capello V, Cauduro A. Clinical technique: Application of Computed Tomography for diagnosis of dental disease in the rabbit, guinea pig and chinchilla. *J Exot Pet Med*. 2008; 17(2):93-101

38. Welch WD, Lu YS, Bawdon RE. Pharmacokinetics of penicillin-G in serum and nasal washings of *Pasteurella multocida* free and infected rabbits. *Lab Anim Sci* 1987;37:65-68

39. Divers SJ. Mandibular abscess treatment using antibiotic impregnated beads. *Exotic DVM* 2000;2(5):15-29

40. Aikin S. Small mammal dentistry Pt 2. In Quesenberry KE, Carpenter JW eds*. Ferrets. Rabbits and Rodents: Clinical medicine and surgery 2nd Ed*. Saunders (Elsevier) USA 2004;379-382

41. Remeeus Pg, Verbeek M. The use of calcium hydroxide in the treatment of abscesses in the cheek of the rabbit resulting from a dental periapical disorder. *J Vet Dent*. 1995;12(1):19-22

42. Harcourt-Brown F. Honey to treat rabbit abscesses. *Exotic DVM* 2002;3(6):13-14

43. Donnelly T, Brown C. Restraint collars in exotic pets and birds. *The Veterinarian* 2006; pp60-62

44. Barone R, Pavaux C, Blin PC, Cuq P. *Atlas d'anotomie du lapin*. 1973. Masson et Cie

45. Popesko P, Rjtova V, Horak J. *A colour atlas of anatomy of small laboratory animals Vol 1: Rabbit, Guinea Pig*. Wolfe Publishing, London

46. Visigalli G, Cappelletti A, Nuvoli S. A surgical approach to retrobulbar abscessation in a pet dwarf rabbit. *Exotic DVM* 2008;10(1):11-14