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Clinical and histopathological features of *Burkholderia cepacia* complex dermatitis in dogs: a series of four cases

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Background – The *Burkholderia cepacia* complex (Bcc) is an emerging cause of opportunistic infections. Deep pyoderma associated with Bcc infection has been reported previously in dogs receiving ciclosporin.

Objective – To report the clinical and histopathological features of four additional cases of Bcc dermatitis in dogs, one of which progressed to septicaemia.

Animals – Four dogs with a skin culture yielding growth of Bcc and skin biopsies for histopathological investigation.

Methods and materials – Retrospective review of medical records and skin biopsies and PCR for *Burkholderia* on DNA extracted from paraffin-embedded skin and liver to confirm Bcc sepsis.

Results – Three different breeds and one mixed breed dog were represented. Two dogs were receiving ciclosporin and one was receiving oclacitinib. One dog had no evidence of immunosuppression. One dog was bathed two days prior to onset of skin lesions. Three dogs presented with dorsally orientated ulcers, crusts and draining tracts; one dog had infection localized to a surgical site. The main histological feature from skin biopsies was severe neutrophilic folliculitis and furunculosis with marked neutrophilic to pyogranulomatous dermatitis. Intracellular Gram-negative and Warthin–Starry positive rods were present in three of four cases. Three dogs were successfully treated with systemic fluoroquinolones or trimethoprim sulfamethoxazole. The Bcc isolate in one dog was resistant to all tested systemic antimicrobials. This dog developed septicaemia and was euthanized.

Conclusions and clinical importance – Bcc skin infections can occur in immunocompetent and immunocompromised dogs. Bcc isolates may be extensively antimicrobial resistant, presenting a challenge for clinical management. Cutaneous infection may progress to life-threatening sepsis.

Introduction

The *Burkholderia cepacia* complex (Bcc) includes at least 18 species of aerobic, Gram-negative rods that are ubiquitous in the environment but can cause opportunistic infections.[1,2] *Burkholderia multivorans* and *B. cenocepacia* are most often associated with Bcc infections in humans, particularly cystic fibrosis patients.[1,3] Contaminated medical devices and medications have been associated with nosocomial outbreaks.[4–6] Bcc infections present a clinical challenge due to frequent antimicrobial resistance, which may be both intrinsic and acquired.[3] Immunocompromised humans also may develop fulminating and often fatal pneumonia, and sepsis termed "cepacia syndrome."[7]

A published case series described six dogs receiving ciclosporin with Bcc-associated deep pyoderma.[8] Affected dogs were successfully treated with antimicrobials. The objective of this case series is to describe clinical and histopathological features of four additional cases of Bcc dermatitis in dogs. In one of these cases, cutaneous infection with an extensively drug-resistant Bcc strain progressed to septicaemia.

Case Reports

Four dogs with both a skin culture yielding growth of *Burkholderia cepacia* and skin biopsies for histopathological investigation were identified. Medical records were searched for information regarding patient signalment, clinical features, treatment and outcome.

Signalment and history

Table 1 summarizes patient signalment and history. Dogs 1, 2 and 3 had a history of allergic dermatitis. Dog 1 and 2 were receiving oral ciclosporin (in combination with ketoconazole for Dog 1) at the time of diagnosis; ciclosporin was started for Dog 2 after the onset of dorsal ulcers and

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crusts potentially associated with Bcc dermatitis. Dog 3 was receiving oclacitinib at the time of diagnosis. This dog had undergone surgery for excisional biopsy of a subcutaneous mass on the ventral neck at another veterinary hospital two days prior to presentation. The dog presented to the authors' institution for evaluation of incisional dehiscence and was concurrently diagnosed with atypical hypoadrenocorticism (glucocorticoid insufficiency) based on: mentation change, hypothermia and hypotension; results of an adrenocorticotropic hormone stimulation test; and normal serum electrolytes. Dog 2 was bathed at home two days prior to the onset of skin lesions. Dog 4 was not receiving immunomodulatory medications and was reported to be systemically healthy.

Clinical presentation

Skin lesions were initially dorsally distributed in dogs 1, 2 and 4, consisting of ulcers (often deep or crateriform), draining tracts and crusts (Figure 1). Within three weeks of initial onset, Dog 1 also developed haemorrhagic crusts on the face and in the inguinal region. In Dog 3, the edges of the incision from the excisional biopsy on the ventral neck were erythematous to black with peri-incisional swelling and serosanguinous discharge. Dogs 1, 2 and 3 had systemic signs at the time of hospital presentation—these are summarized in Table 1. Signs of systemic illness resolved for Dog 3 following emergency stabilization, surgical debridement of the incisional site and initiation of corticosteroid therapy for treatment of atypical hypoadrenocorticism.

Cytological findings

Results of impression smears from ulcers were recorded in the medical record for dogs 1, 2 and 4. Inflammatory cells (neutrophils, macrophages) with large numbers of intracellular and extracellular bacterial rods were noted in all cases. Extracellular bacterial cocci also were noted on impression smears from Dog 1.

Histopathological findings

Histopathological features of skin biopsies from dogs 1, 2 and 4 were similar: severe neutrophilic luminal folliculitis and furunculosis with marked neutrophilic and histiocytic to pyogranulomatous dermatitis (Figure 2). Gram and Warthin–Starry stains revealed Gram-negative and Warthin–Starry positive bacterial rods within histiocytes for dogs 1, 2 and 4 (Figure 3a). Intracellular Gram-negative and Warthin–Starry positive bacterial rods also were present within the hair follicle outer root sheath for Dog 2 (Figure 3b). Three weeks following initial skin biopsies, Dog 1 was euthanized due to progressive skin lesions and systemic illness (fever, inappetence and weight loss). Postmortem examination revealed septicaemia evidenced by acute hepatitis and circulating intrahistiocytic Gram-negative, Warthin–Starry positive bacterial rods. Similar intrahistiocytic bacteria were identified in the skin, liver, lung, spleen and lymph nodes.

Histopathology of tissue submitted at the time of excisional biopsy from Dog 3 showed that pyogranulomatous cellulitis was the cause of the mass effect on the ventral neck. This dog subsequently underwent surgical revision and closure of the dehisced excisional biopsy site. Tissue was again submitted for histopathology, which showed neutrophilic and histiocytic cellulitis and myositis with granulation tissue and fibrin. Aerobic, anaerobic, mycobacterial and fungal cultures were performed at the time of surgical revision, with only Bcc isolated on aerobic culture.

Bacterial culture and susceptibility

Antimicrobial susceptibility data for the four Bcc isolates are summarized in Table 2. Meticillin-susceptible *Staphylococcus pseudintermedius* (MSSP) also was isolated from Dog 1.

Polymerase chain reaction

Conventional PCR was performed using HotStarTaq 2× Mastermix (Qiagen; Hilden, Germany) on DNA extracted from paraffin-embedded skin and liver from Dog 1 using the DNeasy Tissue and Blood kit (Qiagen; Hilden, Germany) following the manufacturer's instructions with genus-specific *Burkholderia* primers (gro1-CTGGAAGACATCGCGATC, gro2- GTCGATGATCGTCGTGTT, final concentration: 0.2 mM). The protocol for thermal cycling was 94°C for 15 min followed by 25 cycles of 94°C for 30 s, 50°C for 30 s and 72°C for 60 s using a Veriti thermal cycler (Applied Biosystems; Waltham, MA, USA). DNA extracted from *Burkholderia cepacia* ATCC 25416 was used as a positive control. PCR for the *histone 3.3* gene was performed under the same conditions as a positive extraction control. PCR of the DNA extractions from

Table 1. Signalment and historical information for four dogs with *Burkholderia cepacia* complex dermatitis.

Dog	Breed	Age (years)	Gender	Weight (kg)	Concurrent disease	Medications	Bathing history	Systemic signs
1	Rough collie	6	Male castrate	41	Allergic dermatitis	Ciclosporin (2.4 mg/kg/day) Ketoconazole (2.4 mg/kg/day)	No	Vomiting, lethargy, inappetence
2	Smooth-coated fox terrier	2	Male castrate	15.6	Allergic dermatitis	Ciclosporin (4.6 mg/kg/day)	Yes–two days prior	None
3	Pug/beagle cross	6	Male castrate	12.5	Allergic dermatitis; Atypical hypoadrenocorticism	Oclacitinib (dose unknown)	Surgery two days prior	Dull mentation, recumbency, hypothermia, hypotension
4	Dobermann pinscher	0.75	Male intact	45	None	None	No	None

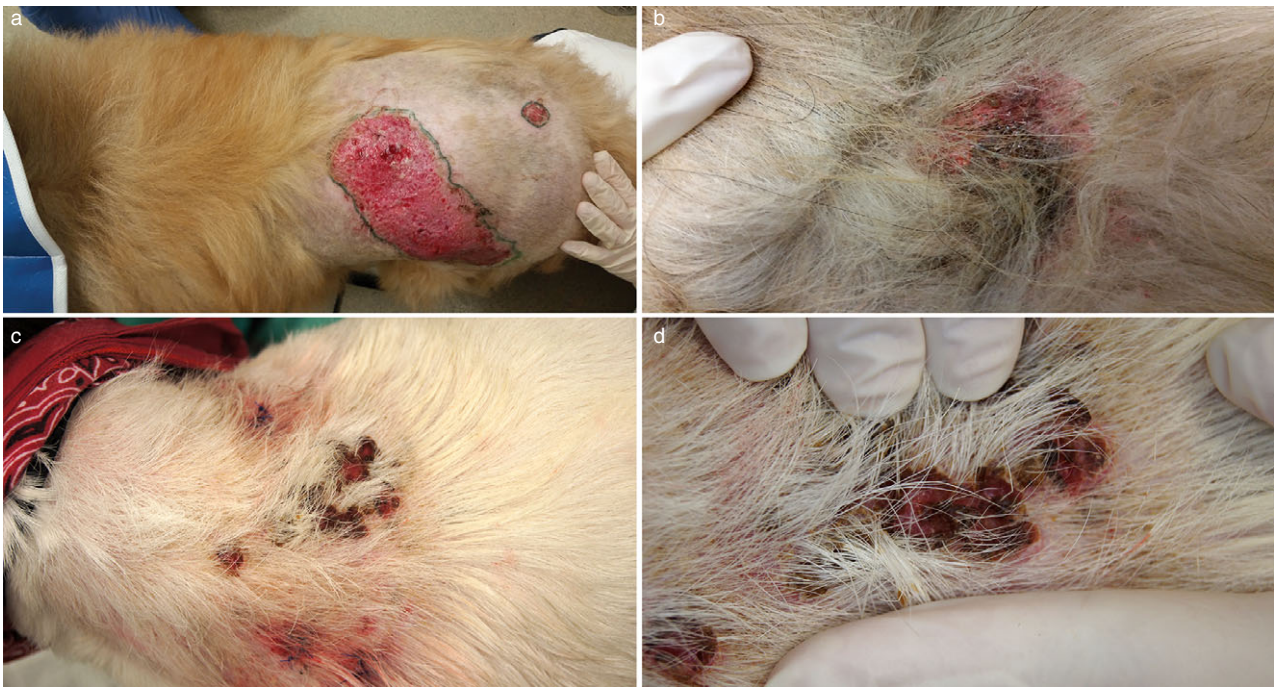

Figure 1. Clinical features of *Burkholderia cepacia* complex cutaneous infection in the dog.
(a) Large area of coalescing ulcers with draining tracts and haemorrhagic exudate over the dorsum and lateral flank and smaller ulcer over the caudal dorsum of Dog 1. The edges of the lesions have been inked, crusts have been removed and the haircoat has been clipped. (b) Focal haemorrhagic crust overlying an ulcer on the dorsum of Dog 1 (photograph taken prior to clipping of haircoat). (c) Multifocal haemorrhagic crusts and ulcers over the dorsum of Dog 2. (d) Close-up of haemorrhagic crusts overlying crateriform ulcers on the dorsal trunk of Dog 2.

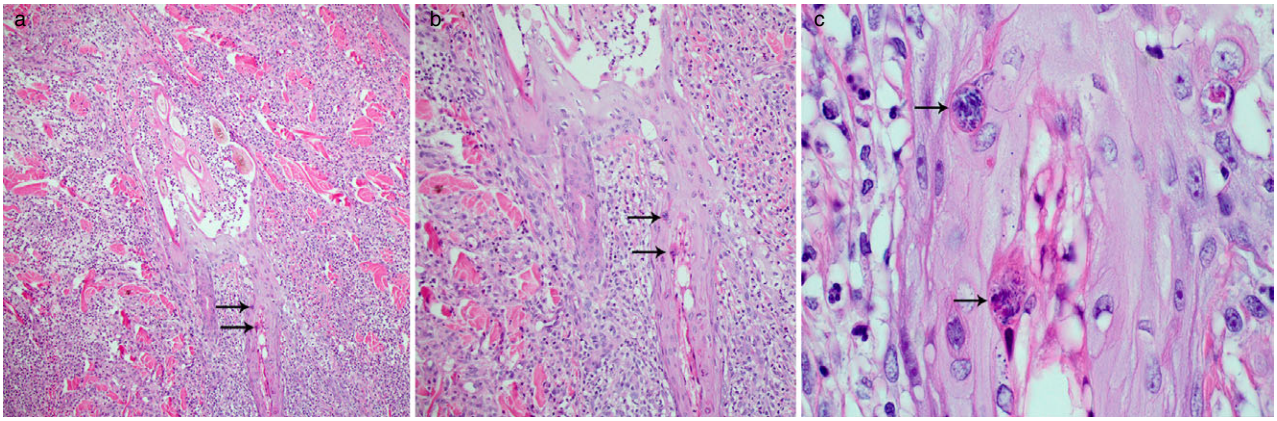

Figure 2. Histological features of *Burkholderia cepacia* complex cutaneous infection in the dog.
(a) Photomicrograph. Severe neutrophilic luminal folliculitis and furunculosis with marked neutrophilic to pyogranulomatous dermatitis. Neutrophils fill the follicular lumen and neutrophils and fewer epithelioid macrophages surround a ruptured hair follicle. Colonies of intracellular bacterial rods distend keratinocytes within the follicular outer root sheath (arrows). Haematoxylin and eosin, ×20 (a) ×40 (b) ×100 (c).

both the skin and liver of Dog 1 were positive for *Burkholderia*, confirming sepsis due to disseminated Bcc infection (Figure 4).

Treatment and treatment outcome

Treatment and outcome for the four dogs is summarized in Table 3. Immunomodulatory medications (ciclosporin with or without ketoconazole and oclacitinib) were stopped following diagnosis of Bcc infection in dogs 1,2 and 3. Dog 3 underwent surgical debridement and flap closure of the previous surgical site, followed by oral trimethoprim sulfamethoxazole post-operatively. The surgical site was fully healed in three weeks. Dogs 2 and 4 were treated with oral fluoroquinolones (despite the lack of *in vitro* susceptibility for Dog 2); Dog 2 also was bathed daily with a shampoo containing 3% chlorhexidine. Both of these dogs had complete resolution of skin lesions within 12 weeks. Dog 1 was treated with oral cephalexin (to address MSSP co-isolated on skin culture), topical medical grade honey and 1% silver sulfadiazine applied under tie-over bandages; euthanasia was pursued due to progression of skin lesions and systemic illness.

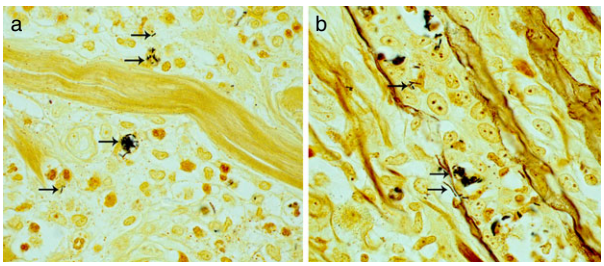

Figure 3. Histological features of *Burkholderia cepacia* complex cutaneous infection in the dog.
(a) Photomicrograph. Numerous positive-staining rods within histiocytes (arrows). Warthin–Starry, ×100. (b) Numerous positive-staining rods free within the hair follicle wall and within follicular outer root sheath keratinocytes (arrows). Warthin–Starry, ×100.

Table 2. Antimicrobial susceptibility of four *Burkholderia cepacia* complex isolates

Antimicrobial	Dog 1	Dog 2	Dog 3	Dog 4
Ceftazidime	R	R	I	S
Cefpodoxime	R	R	R	S
Ticarcillin/clavulanate	R	R	R	R
Enrofloxacin	R	R	R	S
Marbofloxacin	R	R	R	S
Trimethoprim/sulfamethoxazole	R	S	S	S

I intermediate, R resistant, S susceptible.

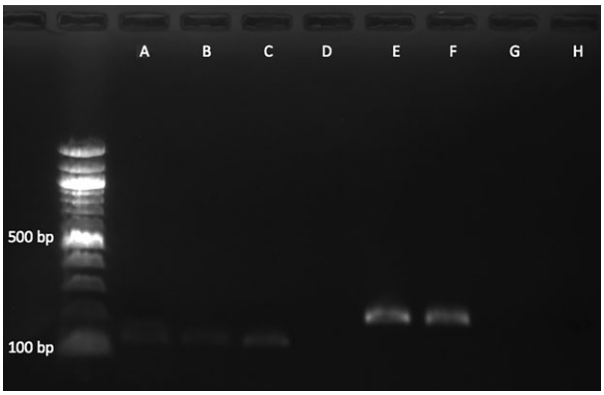

Figure 4. PCR results for the gro1/gro2 PCR (139 bp product) (a) skin (dog 1), (b) liver (dog 1), (c) ATCC 25416, (d) no template control. PCR results for the Histone 3.3 gene (214 bp) for (e) skin (dog 1), (f) liver (dog 1), (g) ATCC 25416 and (h) no template control.

Discussion

This case series confirms the importance of Bcc as an emerging pathogen. In the six cases previously reported, all of the dogs were receiving ciclosporin and four were West Highland white terriers.[8] Although immunocompromised dogs appear to be at greatest risk of infection, Dog 4 in this case series had no documented cause of immunosuppression. *Burkholderia* was not found as a core component of the cutaneous microbiome in one longitudinal study of 40 dogs;[9] opportunistic infection likely follows exposure through an environmental source. Dog 2 was bathed prior to the onset of skin lesions. An otherwise healthy 1-year-old Cardigan Welsh corgi developed dorsal furunculosis associated with Bcc infection following bathing in a previous report.[10] These reports suggest that contaminated shampoos or grooming products could serve as potential exposure sources. Dog 3 developed an incisional infection; although the source of exposure is unknown for this dog, Bcc contamination of chlorhexidine scrub was identified as the putative exposure source in a report of five cats with Bcc cellulitis.[11]

Dogs 1, 2 and 4 initially presented with dorsal truncal skin lesions, particularly deep to crateriform ulcers, that were similar to those reported in another case series.[8] Histologically, cases of Bcc dermatitis have furunculosis but more robust histiocytic to pyogranulomatous interstitial dermal inflammation than seen in cases of furunculosis associated with *Pseudomonas aeruginosa* or *Staphylococcus* spp.[8,10] Bcc is unique as compared with other types of bacteria associated with dorsal furunculosis, such as *P. aeruginosa*, in its ability to survive intracellularly within macrophages and other phagocytic cells.[10,12] In cases of *P. aeruginosa* associated furunculosis, bacterial rods may be noted histologically within inflammatory infiltrates or follicular ostia, but are not typically present intracellularly within histiocytes or the follicular root sheath as in Bcc-associated furunculosis.[8,10] It is important to note that the bacterial rods can be seen on light microscopic examination with careful scrutiny but are easier to visualize with Warthin–Starry or Giemsa stains.

Antimicrobial resistance of Bcc isolates is a concern in human and veterinary medicine. *Burkholderia* spp. have several mechanisms of antimicrobial resistance, including drug efflux pumps, restrictive porins and the ability to alter drug targets. Bcc isolates are inherently resistant to polymyxins and clinical isolates from humans are frequently resistant to chloramphenicol, aminoglycosides, fluoroquinolones, tetracycline, rifampicin and amoxicillin/clavulanic acid.[3] In this case series, Dog 1 developed cutaneous infection with an extensively drug-resistant Bcc strain, precluding systemic antimicrobial therapy. This dog's infection progressed to fulminant septicaemia, which was confirmed via *Burkholderia* PCR performed on

Table 3. Treatment and treatment outcome for four dogs with *Burkholderia cepacia* complex dermatitis

Dog	Topical treatment	Systemic treatment	Treatment duration (weeks)	Treatment outcome
1	Medical grade honey, 1% silver sulfadiazine cream	Cephalexin (24.3 mg/kg twice daily)	Three	Euthanasia
2	3% chlorhexidine shampoo	Marbofloxacin (6.4 mg/kg once daily)	Four (systemic), six (topical)	Complete resolution
3	None	Trimethoprim sulfamethoxazole (19.2 mg/kg twice daily)	One following surgical debridement and closure	Complete resolution
4	3% chlorhexidine shampoo	Enrofloxacin (7.7 mg/kg once daily)	12	Complete resolution

DNA extracted from paraffin-embedded sections of skin and liver. Future studies should investigate molecular characterization and virulence factor expression of *Burkholderia* spp. isolated from veterinary infections.

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Résumé

Contexte – Le complexe *Burkholderia cepacia* (Bcc) est une cause émergente d'infections opportunistes. Les pyodermites profondes associées à Bcc ont été décrites précédemment chez des chiens recevant de la ciclosporine.

Objectifs – Décrire les données cliniques et histopathologiques de quatre cas supplémentaires de dermatite à Bcc chez des chiens, dont un a évolué en septicémie.

Sujets – Quatre chiens avec une culture cutanée permettant la croissance de Bcc et des biopsies cutanées pour examen histopathologique.

Matériel et méthode – Une revue rétrospective des données médicales et des biopsies cutanées et de PCR pour Burkholderia sur des extraits d'ADN de peau et de foie montés dans la paraffine pour confirmer le sepsis à Bcc.

Résultats – Trois différentes races et un chien croisé ont été décrits. Deux chiens recevaient de la ciclosporine et un recevait de l'oclacitinib. Un chien n'avait pas de signe d'immunosuppression. Un chien a été lavé deux jours avant l'apparition des lésions. Trois chiens présentaient des ulcères, croûtes et fistules sur le dos ; un chien avait une infection localisée à un site chirurgical. La principale donnée histopathologique des biopsies cutanées était une folliculite et furonculose neutrophilique sévère avec une dermatite neutrophilique à pyogranulomateuse marquée. Des bacilles Gram négatives et Warthin-Starry positives intracellulaires étaient présentes dans trois des quatre cas. Trois chiens étaient traités avec succès avec des fluoroquinolones systémiques ou de la triméthoprine sulfaméthoxazole. La souche de Bcc chez un chien était résistante à tous les antimicrobiens testés. Ce chien a développé une septicémie et a été euthanasié.

Conclusion et importance clinique – Les infections cutanées à Bcc peuvent se développer sur des chiens immunocompétents et immunodéprimés. Les souches de Bcc peuvent montrer des résistances antimicrobiennes importantes, ce qui représente un défi pour la gestion clinique. L'infection cutanée peut évoluer en septicémie mortelle.

Resumen

Introducción – el complejo *Burkholderia cepacia* (Bcc) es una causa emergente de infecciones oportunistas. La pioderma profunda asociada con la infección por Bcc ha sido reportado previamente en perros que reciben ciclosporina.

Objetivo – reportar las características clínicas e histopatológicas de cuatro casos adicionales de dermatitis por Bcc en perros, uno de los cuales progresó a septicemia.

Animales – cuatro perros con un cultivo de piel produciendo crecimiento de Bcc y biopsias de piel para investigación histopatológica.

Métodos y materiales – revisión retrospectiva de registros médicos y biopsias de piel y PCR para Burkholderia en DNA extraído de piel e hígado en bloques de parafina para confirmar la sepsis por Bcc.

Resultados – los perros eran de tres razas diferentes y uno mestizo. Dos perros estaban recibiendo ciclosporina y uno estaba recibiendo oclacitinib. Un perro no tenía evidencia de inmunosupresión. Un perro fue bañado dos días antes del inicio de las lesiones de la piel. Tres perros presentaron úlceras, costras y conductos drenantes en distribución dorsal; un perro tuvo infección localizada en una zona de intervención quirúrgica. La principal característica histológica de las biopsias cutáneas fue una foliculitis neutrofílica severa y forunculosis con marcada dermatitis neutrófilica a piogranulomatosa. Tres de los cuatro casos presentaron bacilos intracelulares Gram-negativos y positivos con Warthin-Starry. Tres perros fueron tratados con éxito con fluoroquinolonas sistémicas o trimetoprim sulfametoxazol. El aislado Bcc de un perro era resistente a todos los antimicrobianos sistémicos probados. Este perro desarrolló septicemia y fue sacrificado.
Conclusiones e importancia clínica – las infecciones cutáneas por Bcc pueden ocurrir en perros inmunocompetentes e inmunocomprometidos. Los aislados de Bcc pueden ser ampliamente resistentes a antimicrobianos, presentando una gran dificultad para el manejo clínico. La infección cutánea puede progresar a una sepsis potencialmente mortal.

Zusammenfassung
Hintergrund – Der *Burkholderia cepacia* Komplex (Bcc) ist eine neu entstehende Ursache opportunistischer Infektionen. Eine tiefe Pyodermie im Zusammenhang mit Bcc Infektionen wurden bereits bei Hunden, denen Ciclosporin verabreicht wurde, beschrieben.
Ziel – Ein Bericht über die klinischen und histopathologischen Merkmale von vier weiteren Fällen einer Bcc Dermatitis bei Hunden, von denen sich eine zur Septikämie entwickelte.
Tiere – Vier Hunde mit einer Hautkultur, die ein Wachstum von Bcc ergab und Hautbiopsien zur histopathologischen Untersuchung.
Methoden und Materialen – Eine retrospektive Review der Krankenakten und Hautbiopsien und PCR auf Burkholderia aus DNA, die aus Haut, die in Paraffin eingebettet war, extrahiert wurde sowie aus Leber, um eine Bcc Sepsis zu bestätigen.
Ergebnis – Drei unterschiedliche Rassen und ein Mischlingshund waren repräsentiert. Zwei Hunde erhielten Ciclosporin und einer Oclacitinib. Ein Hund zeigte keinerlei Evidenz einer Immunsupprimierung. Ein Hund wurde zwei Tage bevor sich die Hautveränderungen entwickelten, gebadet. Drei Hunde wurden mit dorsal auftretenden Ulzera, Krusten und Fisteln vorgestellt; bei einem Hund bestand eine Infektion an einer Operationswunde. Die hauptsächlichen histologischen Merkmale der Hautbiopsien bestanden aus einer markanten neutrophilen Follikulitis und Furunkulose mit einer markanten neutrophilen bis pyogranulomatösen Dermatitis. Es traten intrazelluläre Gram-negative und Warthin-Starry positive Stäbchen in drei der vier Fälle auf. Drei Hunde wurden erfolgreich mit systemischen Fluoroquinolonen oder mit Trimethoprim Sulfamethoxazol behandelt. Das Bcc Isolat eines Hundes war resistent auf alle getesteten systemischen Antibiotika. Der Hund entwickelte eine Septikämie und wurde eingeschläfert.
Schlussfolgerungen und klinische Bedeutung – Bcc Infektionen können bei immunkompetenten und immunkompromittierten Hunden auftreten. Bcc Isolate können weitegehend auf Antibiotika resistent sein, wodurch sie eine Herausforderung für das klinische Management darstellen. Eine kutane Infektion kann sich dadurch zu einer lebensbedrohlichen Sepsis entwickeln.

要約
背景 – *Burkholderia cepacia* complex(Bcc)は、新たな日和見感染症の原因菌である。 Bcc感染に関連した深在性膿皮症が、シクロスポリン投与犬において報告されている。
目的 – 本研究の目的は、犬におけるBcc皮膚炎の追加4症例(1症例が敗血症に進行した)における臨床および組織病理学的特徴を報告することである。
被験動物 – 組織病理学的調査のため、Bcc培養をもたらす皮膚培養および皮膚生検を実施した犬4頭。
方法および材料 – 医療記録及び皮膚生検に対する後ろ向き研究を実施し、Bcc敗血症を確認するために、パラフィン包埋された皮膚および肝臓から抽出したDNA上の*Burkholderia*に対するPCRを実施した。
結果 – 異なる3犬種および雑種犬1頭が示された。 2頭はシクロスポリンを、1頭はオクラシチニブを投与されていた。 1頭の犬では免疫抑制の証拠が認められなかった。 1頭の犬は皮膚病変が発症する2日前に入浴していた。 3頭の犬は背部に潰瘍、痂皮および排膿を認めた。 1頭の犬は手術部位に限局した感染を有していた。皮膚生検による主な組織学的特徴は、重度の好中球性毛包炎および細菌性肉芽腫性皮膚炎に対して顕著な好中性を示す癒腫症であった。 4例のうち3例は、細胞内グラム陰性およびワルチン=スターリー染色陽性の桿菌を認めた。 3頭の犬はフルオロキノロンまたはスルファメトキサゾール・トリメトプリムによる全身療法で順調に治療された。 1頭の犬のBcc分離株は、試験した全身性抗菌剤に耐性であった。この犬は敗血症を発症し、安楽死した。
結論と臨床的重要性 – Bcc皮膚感染は、免疫適応及び免疫不全の犬において起こり得る。 Bcc分離株は広範囲に抗菌剤耐性を有し、臨床管理の課題となっている。皮膚感染症は、生命を脅かす敗血症に進行する可能性がある。

摘要

背景 — 洋葱伯克氏菌复合体(*Burkholderia cepacia* complex ,Bcc)是一种新出现的条件致病性感染原。先前有报道称,对于服用环孢素的犬,洋葱伯克氏菌复合体感染可导致其发生深层脓皮病。

目的 — 报告另外四例犬Bcc皮炎的临床和组织病理学特征,其中一例出现败血症。

动物 — 四只犬皮肤培养长出 Bcc,皮肤活检用于组织病理调查。

材料与方法 — 回顾性研究病历记录,皮肤和肝脏活检后进行石蜡包埋,并用PCR方法检测伯克氏DNA提取物,以确认 Bcc 败血症。

结果 — 选取了不同品种的三只犬和一只混血犬。两只犬服用环孢素;一只服用奥拉替尼;一只没有发现存在免疫抑制;一只犬在洗澡后两天出现了皮肤病变。三只犬身体背侧出现溃疡、结痂以及瘘管;一只犬的感染发生在于外科手术部位。皮肤活检的主要组织学特征是严重的中性粒细胞毛囊炎和疖病,伴有明显的中性到脓性肉芽肿性皮炎。细胞内革兰氏阴性和Warthin–Starry银染色阳性的杆菌,存在于四个病例中的三个。三只犬通过全身性氟喹诺酮类药物或甲氧苄氨嘧啶-磺胺甲恶唑成功治愈。一只犬的Bcc分离菌株对所有检测的全身性抗菌药具有耐药性,这只犬患上败血症后被安乐死。

结论和临床价值 — Bcc 感染可以发生于免疫不全和免疫低下的犬。 Bcc 菌株可能对抗菌药广泛耐药,这对于临床管理提出了挑战。皮肤感染可能会导致危及生命的脓毒症。

Resumo

Contexto – O complexo *Burkholderia cepacia* (CBC) é uma causa emergente de infecções oportunistas. Piodermites profundas associadas a infecções por CBC já foram reportadas em cães recebendo ciclosporina.

Objetivo – Relatar as características clínicas e histopatológicas de quatro casos de dermatites por CBC em cães, tendo um deles progredido para septicemia.

Animais – Quatro cães apresentando culturas de lesões cutâneas com crescimento de CBC e biópsia cutânea para exame histopatológico.

Métodos e materiais – Estudo retrospectivo dos históricos clínicos, biópsias cutâneas e PCR para Burkholderia de DNA extraído de pele fixada em parafina e fígado para confirmar a sepse por CBC.

Resultados – Três cães de diferentes raças e um cão sem raça definida foram incluídos. Dois cães estavam recebendo ciclosporina e um cão estava recebendo oclacitinib. Um cão não apresentava nenhum sinal de imunossupressão. Um dos cães foi banhado dois dias antes do surgimento das lesões cutâneas. Três cães apresentavam lesões ulceradas, crostas e tratos drenantes na região dorsal; um cão apresentava infecção localizada em incisão cirúrgica. A principal característica histopatológica das biópsias cutâneas foi foliculite neutrofílica e furunculose com dermatite neutrofílica a piogranulomatosa marcante. Bastões Gram-negativos intracelulares e Warthin-Starry positivos estavam presentes em três dos quatro casos. Três cães foram tratados com sucesso com fluorquinolonas ou sulfa com trimetoprim. O CBC isolado em um dos cães foi resistente a todos os antimicrobianos sistêmicos testados. Este cão desenvolveu septicemia e foi eutanasiado.

Conclusões e importância clínica – As infecções cutâneas por CBC podem ocorrer tanto em cães imunocompetentes quanto em imunossuprimidos. Os isolados de CBC podem apresentar resistência extensiva a antimicrobianos, representando um desafio no manejo clínico do caso. Infecções cutâneas podem progredir para sepse com risco de morte.