

Breaking the biofilm: A persistent case of deep pyoderma in a Bullykutta dog

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Abstract

An eleven-month-old male Bullykutta dog was presented with a complaint of persistent skin infection in spite of treatment. The history revealed recurrence over four months, with the latest episode persisting for more than one month. Clinical examination showed hard, adherent crusts with erythema, alopecia, scaling and draining tracts of blood oozing lesions localized at the dorsum. Skin scrapings tested negative for mites, and no fleas or flea dirt's were observed. Direct impression cytology revealed cocci, clusters of bacilli, and neutrophilic infiltration. Trichogram analysis identified trichorrhexis, with all hairs in the telogen phase. Culture confirmed *Staphylococcus* and *Pseudomonas* spp. ABST results showed sensitivity to ceftriaxone and resistance to other drugs. Based on history of draining tracts of blood oozing lesions and laboratory investigations the present was diagnosed as deep pyoderma with a mixed bacterial infection. Treatment began with cephalexin but was later switched to Sulphamethoxazole and trimethoprim due to recurrence. A 10-day regimen of Sulphamethoxazole and trimethoprim combined with omega fatty acid supplements and adjunctive topical therapy resulted in complete resolution and effectively preventing further recurrence of infection.

Keywords: Deep pyoderma, Bullykutta

Introduction

Canine pyoderma is the most frequently occurring bacterial skin infection in dogs. It is primarily associated with staphylococcal species, which naturally inhabit the skin of healthy dogs as common colonizers (Pinchbeck *et al.*, 2006). Causes for recurrent pyoderma infection include ectoparasitic infestations, allergic skin diseases such as atopic dermatitis and food allergic dermatitis where pyoderma is a secondary causative, endocrine conditions, keratinization disorders, genodermatoses (follicular dysplasia, color dilution alopecia, sebaceous adenitis), immunodeficiency (congenital, acquired), bacterial hypersensitivity, resistant strains of *Staphylococcus* sp., non-staphylococcal pyoderma such as *Pseudomonas* leading to prolonged or recurring infections, (Peter, 2005 ; Loeffler and Lloyd, 2018).

Case History and Observations

An eleven-month-old male Bullykutta dog was presented to the Dermatology Unit of Madras

Veterinary College Hospital with major complaint of crusting lesions with erythematous patches and scales, folliculitis, and draining tracts of blood oozing lesions localized all over the dorsum region of the body for the past one month but there is recurrence of infection since four months. Upon clinical examination the lesion is with hard crusts and erythematous patches with loss of hair over the lesion. Deep skin scraping was negative for mites. No traces of flea and flea dirt which ruled out parasitic infestation. Trichogram revealed trichorrhexis, follicular casts and hairs in telogen phase. Direct impression cytology revealed cocci, sheets of bacilli and neutrophilic infiltration. Culture and ABST revealed causatives as *Staphylococcus* and *Pseudomonas* species and sensitive for cephalosporins in ABST. Based on diagnosis it is confirmed as deep pyoderma involving dermis layer.

Treatment and Discussion

Treatment is initiated with cephalexin as there is recurrence observed antibiotic was changed to Trimethoprim and sulpha methoxazole, along with omega fatty acids supplements with biotin and niacinamide and topical adjunctives such as 2% chlorhexidine spray and

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ketoconazole- chlorhexidine shampoo and mupirocin ointment. The patient showed significant improvement with no recurrence observed during follow-up visits. Regular follow-ups were scheduled to monitor recovery and ensure early detection if recurrence occurs. This

case highlights the importance of prompt diagnosis and treatment in managing recurrent deep pyoderma were there are draining tracts indicating damage to dermis layer which is due to biofilm formation and cutaneous barrier dysfunction



Fig.1 Initial presentation: Bullykutta with adherent crusts and scaling along with hyperpigmentation and deep folliculitis



Fig.2 Erythematous crusting all over dorsum



Fig.3 Lesions localized only over dorsum

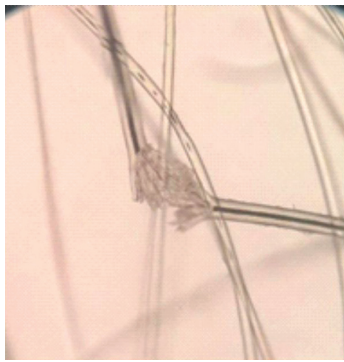


Fig. 4 Trichorrhexis nodosa

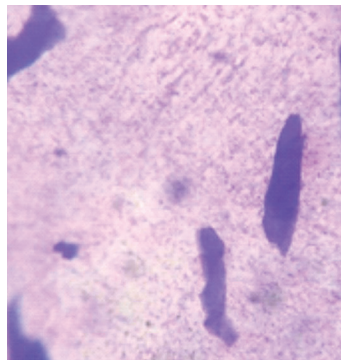


Fig. 5 Cytology impression with rods

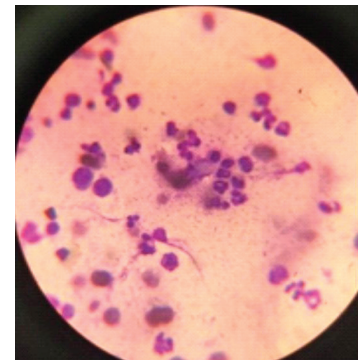


Fig. 6 Cytology impression with neutrophil infiltration and cocci



Fig. 7.1 On cetrimide agar the colonies appeared as yellowish mucoid colonies which were suspected for *Pseudomonas* sp

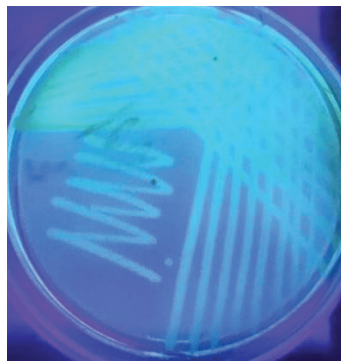


Fig.7.2 Under UV light colonies exhibit fluorescence which were presumptive of *Pseudomonas* sp

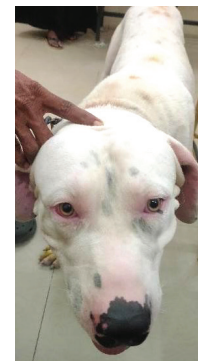


Fig. 8 After treatment

Canine pyoderma, a prevalent skin infection, manifests through primary lesions like papules and pustules, with secondary lesions including crusting, epidermal collarettes, alopecia, scaling, erythema, pruritus, lichenification, and hyperpigmentation (Chaudhary *et al.*, 2019). The predominant bacterial agents responsible for pyoderma include *Staphylococcus intermedius* and *Staphylococcus aureus* (Scott *et al.*, 1998), though deep pyoderma often involves opportunistic pathogens such as *Pseudomonas*, *Proteus*, *Escherichia coli*, *Actinomyces*, *Actinobacillus*, *Fusobacterium*, and *Mycobacterium* spp. (Paradis *et al.*, 2001). Deep pyoderma, the most severe variant, extends into the dermis and subcutaneous tissue, manifesting as draining sinuses, nodules, hemorrhagic crusts, and painful swelling, with a risk of systemic spread and bacteremia (Loeffler, 2018). Rather *et al.* (2021) reported that the bacterial persistence in pyoderma was largely attributed to extracellular polymeric substances (EPS), which created a protective barrier

aiding survival and bacterial slime enhances adhesion to host cells, further complicating treatment efforts. Cerasela (2013) and Hill & Moriello (1994) reported that *Staphylococci* produced protein A, a virulence factor that triggered the complement cascade, leading to neutrophil recruitment and heightened inflammation and additionally, protein A contributed to both immediate and delayed hypersensitivity reactions, exacerbating tissue damage and immune dysregulation. Andrade *et al.* (2022) reported that the biofilm production was a key factor in chronic infections, with 51% of *S. pseudointermedius*, 94.6% of *S. aureus*, and 88.9% of *S. coagulans* isolates demonstrating biofilm-forming capability. Most species of *Pseudomonas* are known to readily form biofilms, which play a significant role in causing biofilm-associated infections that often become recurrent and lead to chronic skin infection (Vetrivel *et al.*, 2021). First-line options for Methicillin-susceptible *Staphylococcus pseudointermedius* (MSSP) included clindamycin, cefalexin, and amoxicillin-clavulanate

(Loeffler *et al.*, 2025). Mupirocin, an antibiotic derived from *Pseudomonas fluorescens*, selectively inhibits iso-leucyl transfer RNA synthetase, disrupting bacterial protein synthesis. This case illustrates the intricate interplay between biofilm formation, cutaneous barrier dysfunction, and antimicrobial resistance in recurrent deep pyoderma. Successful resolution through a multifaceted approach incorporating targeted antimicrobial therapy, adjunctive topical treatments, and omega fatty acid supplementation highlights the necessity of comprehensive management strategies for persistent infections.

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