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Successful Management of Mixed Infection with Generalized Demodicosis, Malasseziosis and Pyoderma in A Pug

J. Shashank[1], G. Tejaswi[2] and K. Satish kumar[3*]

[1]Ph.D, Department of Veterinary Medicine, College of Veterinary Science, Rajendranagar, PVNR TVU, Hyderabad, Telangana, India

[2]PG , Department of Veterinary Medicine, College of Veterinary Science, Rajendranagar, PVNR TVU, Hyderabad, Telangana, India

[3]Professor and University Head, Department of Veterinary Medicine, College of Veterinary Science, Rajendranagar, PVNR TVU, Hyderabad, Telangana, India.

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Abstract

An 8 months old pug breed was presented to the Veterinary Clinical complex (VCC), College of Veterinary Science, Rajendranagar, Hyderabad with a poor general health condition, alopecia, crusted lesions, pruritus, skin scales, moist skin folds, offensive odour and pus exudates. Based on clinical and laboratory findings, the dog was diagnosed with mixed infection of demodicosis malasseziosis and pyoderma. It was treated with oral ivermectin, ketoconazole, amoxycillin+clavulanic acid and atarax tablets, and amitraz, curabless, imidectin spot-on, petben and ketochlor shampoos topically along with supportive therapy. After 60 days of treatment, the dog was found to be negative for mites on microscopic examination of two successive deep skin scrapings, negative for yeasts by cytology with field staining technique and also no microbial growth was found on culture media by which the dog was confirmed to be completely recovered from demodicosis malasseziosis and pyoderma.

Keywords: Demodicosis, Malasseziosis, Pyoderma, Ketoconazole, Ivermectin, Amoxycillin+Clavulanic acid.

Introduction

Nowadays concurrent occurrences of mixed infection by mites, yeast and bacteria are becoming common among dogs (Mueller, 2004) [1]. This may be due to unhygienic management and post-demodicosis complications. Canine demodicosis is an inflammatory parasitic skin disease caused by a proliferation of host-specific follicular mite of the genus *Demodex*. This disease allows the mite to proliferate in the hair follicles and sebaceous glands leading to alopecia, erythema, scaling, hair casting, pustules and secondary infections. Infection is acquired either from an infected animal or object, following immunosuppressive conditions or treatments, and may be related to a genetic immune deficiency (Ferrer *et al.*, 2014, Koch, 2017) [2-3]. The parasite is not considered contagious except during

a few days after birth when puppies acquire mites through direct skin contact with their mother. Canine demodicosis can be divided into two types: localized and generalized according to the extent of lesions. The localized form appears as small patches of alopecia, mild erythema in young dogs and it generally regresses spontaneously without treatment. Whereas, the generalized form is more severe and can even be fatal and may develop from the localized condition or occur in older animals, especially those undergoing severe stress or underlying diseases which require prolonged therapy (Kumari *et al.*, 2018) [4]. Canine demodicosis can be challenging to treat due to several factors such as disease recurrence after treatment, progression to generalized form, immunosuppression and treatment duration. Lymphadenopathy is commonly associated with the disease (Morita *et al.*, 2018, Plant *et al.*, 2011) [5- 6]. The diagnosis is typically based on clinical signs and is confirmed by the presence of mites in deep skin scrapings. Although *Demodex* mites are part of the normal microfauna, it is uncommon to find the mites, even by performing several deep skin scrapings. If a mite is found, this should raise suspicion and additional skin scrapings should be performed. Finding more than one mite is strongly suggestive of clinical demodicosis (Mueller, 2012) [7] and a high number of *Demodex spp.* mites within follicles and sebaceous glands cause canine demodicosis (Shipstone, 2000) [8]. *Malassezia spp.* is a commensal lipophilic yeast and a cutaneous microflora of most warm-blooded animals. These may suddenly act as opportunistic pathogens causing dermatitis or otitis in dogs (Scott *et al.*, 2001) [9]. A change in the skin environment, such as increased sebum or moisture, alteration of epidermal defense system, genetic predisposition, immunodeficiency disorders, long term exogenous corticosteroid and antibiotic administration predisposes to *Malassezia* infections (Kark, 2008, Reberg bruner and Blakemore, 1999) [10-11]. Dogs suffer from a variety of skin infections; canine pyoderma is one of the most common diseases. Pyoderma literally means pus in the skin and can be caused by infectious, inflammatory, neoplastic etiologies; any condition that results in the accumulation of neutrophilic exudates can be termed pyoderma. Most commonly, pyoderma refers to bacterial infections of the skin and it is classified according to the depth of infection as surface, superficial and deep pyoderma. Surface pyodermas are those infections that are restricted to the surface of the skin and not extended into the follicle; it does not extend deeper than the stratum corneum or into the hair follicle. Superficial pyodermas include infections that involve the hair follicle but do not extend into the dermis. Deep pyodermas are infections that extend into the dermis and underlying panniculitis. Pyoderma is caused most commonly by *Staphylococci* as a primary and concurrent infection of demodicosis and yeast dermatitis (Morris, 2010) [12].

Materials and Methods

The present investigation was carried out in the diagnostic laboratory, Veterinary Clinical Complex; the dog was brought with a history of skin lesions associated with pruritus. Detailed clinical examination revealed poor general health condition, thickening of the skin, moist skin folds, offensive odor, skin scales, alopecia, crusted lesions, erythema and pus exudates (Fig. 1 and 2). Laboratory examination was done with a deep skin scraping, cytology with a field staining and swab culture along with hematological analysis. Before collection of skin scraping, the scalpel blade was dipped in liquid paraffin and collection of scrapings was continued until there was a slight ooze of blood from dermal capillaries. The material was digested with 10 ml of 10% KOH and kept under flame for five minutes. It was centrifuged at 10000 RPM for 5 minutes and the supernatant was discarded. A drop of sediment was taken on a microscopic slide, placed the cover slip and examined under 10X of the microscope, and for cytology, field staining was done and examined under 100X objective of the microscope for the detection of microorganisms (Rosenkrantz, 2009)[13]. Another sample was also collected using a sterile swab and processed for culture and antibiogram.

Results

Microscopic examination of skin scrapings and cytology revealed *Demodex canis* mites and *Malassezia* yeasts, cocci organisms, respectively. *Staphylococcus* organisms were isolated by culture examination (Fig. 3 to 5). Based on the clinical history and laboratory findings, the present case was diagnosed as a mixed infection with *Demodicosis*, *Malasseziosis* and Pyoderma. Conducted antibiotic sensitivity tests revealed that *Staphylococcus spp.* was more sensitive to Amoxicillin+Clavulanic acid (Fig. 6). Complete blood picture count revealed increased white blood cell counts (Table.1). Based on clinical and laboratory findings, treatment was initiated with oral medications with TOXO-MOX (Amoxicillin+Clavulanic acid) – 250 mg, ½ tablet, twice daily for 14 days, Tab. Ivermectin-6 mg, ½ tablet on alternate days for 2 weeks. Tab. Atarax (Anti-histamine) -10mg, ½ tablet daily for 14 days, Tab. Ketoconazole-200 mg, 1/4 tablet, once daily for 14 days. Medicated bath with Petben and Ketochlor shampoos, twice weekly followed by 1 dose of Imidectin spot-on, topical application of 0.05% Amitraz solution weekly once for 2 weeks and Curabless cream once daily for 14 days. Supportive therapy with Hepamust (Pet Mankind) syrup, @4 ml BID for 1 month, Immuncare (Vetrina) syrup, @4 ml BID for 1 month and Absolute Salmon oil (Drools) syrup, @4 ml BID for two months. After one month of treatment, the dog showed clinical improvement *i.e.*, the general skin lesions were improved, pruritus and pyoderma were controlled. Microscopic examination revealed a decreased number of mites in skin

scrapings, and yeasts on field staining, but complete recovery and regrowth of hair were noticed after two months of therapy (Fig. 7 to 9).

Fig. 1 and 2: Crusted lesions, erythemas, moist skin folds with thickening and pus exudates

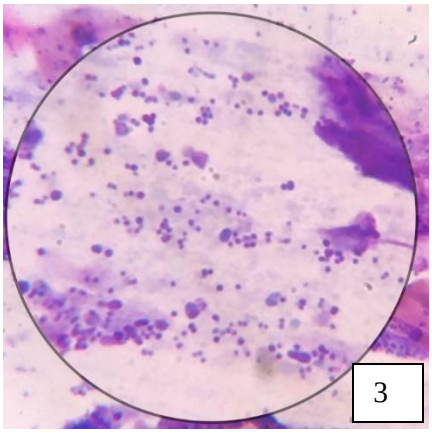

Fig. 3: *A. Malassezia* yeast B. Cocci bacteria under microscope (100X)

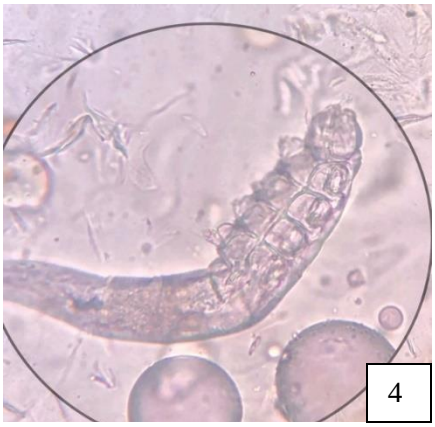

Fig. 4 Demodex canis under 10X

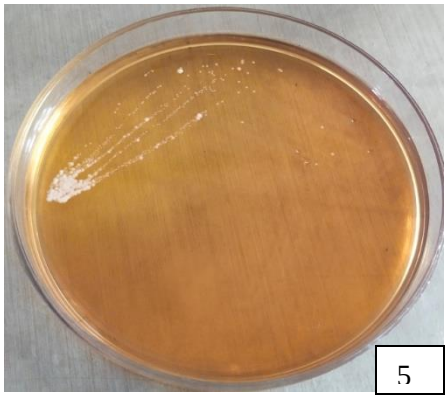

Fig. 5: Growth of *Staphylococcus* organism on MSA

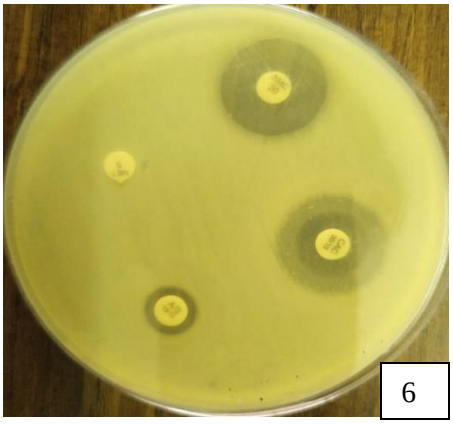

Fig. 6: *Staphylococcus* showing sensitive to

Fig. 7, 8 and 9: Clinical improvement of dog after treatment

Table 1: Hematological parameters

Parameter	**Before treatment**	**After treatment**	**Normal ranges**
Hemoglobin (g/dl)	13.2	14.6	12-18
PCV (%)	40	45	37-55
RBC (10^6/µl)	6.52	7.14	5.5-8.5
Platelets (10^5/µl)	2.36	2.48	2-5
WBC (10^3/µl)	21.8	14.30	6-17
Neutrophils (%)	92	70	55-80
Lymphocytes (%)	30	32	12-30
Monocytes (%)	2	3	3-10
Eosinophils (%)	3	1	2-10
Basophils (%)	0	0	0-1

Discussion

Shipstone (2000) [8] proposed the critical factors for the appearance of demodectic mange as the genetic predisposition of breed and immunosuppressive conditions. Localized demodicosis is a common mild and benign self-limiting disease, however, the generalized form initiates with the progression of multifocal, erythematous, partially alopecic, crusted macules that eventuate in plaques and can be life threatening if left untreated. Malassezia pachydermatis belongs to the resident flora in various mammals and birds. However, it may also cause diseases, especially otitis and dermatitis, as an opportunistic pathogen (Guillot and Bond, 1999, Machado *et al.*, 2003) [14-15]. Two mechanisms that have been suggested to trigger overgrowth of the yeast are alterations in host defense mechanisms and changes in the cutaneous microenvironment. A disrupted epidermal barrier renders the skin more prone to bacterial and yeast infections. Diseases that can cause a decrease in cutaneous barrier function and are commonly associated with malassezia dermatitis are hypersensitivity diseases (especially atopic dermatitis), parasitic infestation and keratinization disorders (Chen and Hill, 2005) [16]. Mite infestation causes itching and scratching, which predisposes the skin to bacterial invasion and further leads to the development of pyoderma. Leukocytosis might have resulted from toxins released due to tissue damage, necrosis and secondary bacterial infections. Bacteria or yeast irritates and stimulates the mast cells for the release of more histamines which acts as a chemotactic, leading eosinophils from the bone marrow to the circulation resulting in eosinophilia (Dimri *et al.*, 2000) [17]. In the present case, for treating *Demodicosis, Malasseziosis* and Pyoderma, we used oral ivermectin, ketoconazole and Amoxicillin+Clavulanic tablets, respectively. Ivermectin acts by interfering with the nerve and muscle functions of parasites. The drug binds to glutamate -gated chloride channels of nerve and muscle cells, this binding pushes the channels open, which increases the flow of chloride ions and hyper-polarizes the cell membranes, paralyzing and killing the parasites (Martin *et al.*, 2021)[18]. Ketoconazole is the most commonly used drug as acts by binding to cytochrome P450, which inhibits the synthesis of ergosterol, an important component of the fungal cell membrane. This results in alterations of cellular permeability and activity of various membrane enzymes (Gabal, 1986) [19]. Ketoconazole also has anti-inflammatory properties and acts on keratinisation process (Levine, 1982) [20]. Amoxicillin+Clavulanic acid has an increased spectrum of activity against gram-negative bacteria due to the presence of the "suicide" drug, clavulanic acid. Clavulanic acid irreversibly binds to f-lactamases, allowing the amoxicillin fraction to interact with the bacterial pathogen. This combination usually has excellent bactericidal activity against B-lactamase-producing *Staphylococci, E. coli, Klebsiella spp., Pseudomonas spp., Enterobacter spp.,* Penicillins have greater stability to lactamases, they have greater activity against *Staphylococci* and gram-negative

bacterial (Petricia and Dowling 1996) [21]. Amitraz acts by inhibiting monoamine oxidase, prostaglandin synthesis and by stimulating the alpha 2 adrenergic receptors of the arthropod nervous system. In the present study, the combination of ivermectin and Amitraz has given good efficacy for the management of generalized demodicosis (Mueller, 2004) [1] (Kumari *et al.,* 2018) [4]. Used Curabless by its antibacterial and antifungal activity as given good results in the present study. Hepamust is a highly palatable herbal liver tonic, used might be due to its powerful antioxidant, which prevents entry of toxins inside the hepatocytes and their subsequent damage. Absolute Salmon oil contains omega-3 fatty acids and it supports in the maintenance of healthy skin and coat. The rationale for immune-modulator therapy includes the stimulation of enhanced immune surveillance and altered response to bacterial allergens that leads to diminished recurrence (Ihrke, 2005) [22].

Conclusion

The dog was completely recovered within 60 days of treatment. It can be concluded from the present study that combined infection of *Malasseziosis, Demodicosis* and Pyoderma can be effectively controlled by oral medications with Ketoconazole, Ivermectin and TOXO-MOX (Amoxicillin+Clavulanic acid) followed by topical application of Amitraz, curabless and Imidectin spot on with Petben and ketochlor shampoos. In conclusion, it is extremely important to critically evaluate dermatological disease during each examination with the proper baseline diagnostic testing. It is also essential to understand the risks, benefits and possible side effects of all therapies administered when creating a long-term treatment.

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