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Hemato-biochemical changes and spatial distribution of lesions on body surface in demodicosis affected dogs.

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Abstract
This study was carried out on 24 dogs. All the dogs were randomly divided into 3 equal groups (n=6) viz. A, B, and C based on treatment regimen irrespective of sex, breed and age. Six apparently healthy dogs were taken as control (Group D). Hemato-biochemical investigation were performed to measure TLC, RBS, TSP, Albumin, TSH, T_3 and T_4. The mean values of TLC in groups A, B and C were 13.19±1.14, 13.21±0.87 and 13.43±1.28 respectively,quite high in comparison to healthy group 7.89±0.42.RBS in group A, B and C were 69.71±3.84, 85.80±5.88 and 70.89±4.49 respectively, while 87.27±2.75 was in control. Post treatment value in group A, B and C were 79.03±3.63, 79.61±4.43 and 80.62±2.42 respectively. The total serum protein (g/dl) and albumin (g/dl) of healthy group were 6.12±0.10 and 1.60±0.10 respectively, and in treated group were2.49±0.04,2.65±0.99and2.78±0.09respectively.The mean value T_3 (ng/dl), T_4 (µg/dl) and TSH (ng/ml) were not differ.The spatial distribution of lesions were head, trunk and limb 36.5%, 40.5% and 23% respectively.

Keywords: Demodex, thyroid, albumin, *Serum protein*, lymphadenopathy

Introduction
Demodicosis is an inflammatory skin disease in dogs caused by tiny, cigar-shaped eight-legged mites (Demodex mites), also referred to as demodectic mange. Demodicosis is generally less severe than sarcoptic mange/scabies. *Demodex canis* is the most common species of genus Demodex, besides this two other species *Demodex injai* and *Demodexcornei* also reported in dogs in different countries (Sivajothi *et al.*, 2015) [1].
Demodex mites have shown a long parasitic parallelism with their mammalian hosts without any inflammatory response (Akilov *et al.*, 2004 and Forton *et al.*, 2012) [2, 3]. The host immune system seems to detect and endure the presence of these mites and inhibitory effect on mite proliferation (Ferrer *et al.*, 2014) [4]. While, the exact immunological mechanism that controls mite proliferation in canine are still unknown. Many immunosuppressed dogs never develop demodicosis, and in many cases the above courses may not be found. However, in certain circumstances, such as underdeveloped or impaired immune system, malnutrition or intense stress, the mites reproduce rapidly and increase their population several times than normal (Scott *et al*, 2010) [5] and symptoms appears in sensitive dogs that range from mild irritation and hair loss as small patches in skin to severe. It can lead to skin lesions, alopecia, pruritus, inflammation and immunosuppression. Generally, transmission of demodex mites occurs from mother to puppies during nursing i.e. first day of life, if the puppies isolated just after caesarean section do not have any demodex mite (Greve *et al*., 1966) [6]. Generalized demodicosis developed from localized condition or occurs in older animals related with immunosuppressive disease (Shipstone, 2000) [7], it may be a severe and potentially life-threatening condition. Mild erythema, comedones and scaling lesion observed in mildly affected dogs. Partial or complete alopecia may develop. Multiple coalescing of alopecia and follicular papules shows moderate severity. Follicular castes may be present. Follicular pustules are seen in severe cases. Furunculosis scales, crusts, exudation and focal ulceration are observed in advanced disease. Bilateral ceruminous otitis externa, lymphadenopathy, lethargy and fever may develop in generalized demodicosis.

Materials and methods
In the present investigation skin scrapings and blood samples from dogs attending out door of TVCC of Bihar Veterinary College, Patna, constitute the materials for study.

Clinical diagnosis and scoring of clinical symptoms 0 to 5 based on of demodectic mange was based on physical examination like intense pruritus, popular, eruption, crusting, excoriations, erythema and alopecia. All the scales were added to express as demodicosis induced skin lesion (DISL). The blood were subjected for TLC estimation and serum for random blood sugar, total serum protein, albumin, T3, T4 and TSH.

Result and discussion

The most common site of lesions were head, trunk and limb36.5%,40.5% and 23% respectively. The head regions periorbital, perioral, chin, calvaria; trunk region includes abdomen dorsum, neck, anal region and limbs comprises both fore and hind. It is evident from the data the most frequently involved region was trunk followed by head and least one is limb area Sakina and Mandial, (2011) reported that the head region is most affected area with lesions, 77.14% on face, 71.42% in peri-orbital region and 57.14% on calvaria in contrast to our findings. Pereira, *et. al*. (2012) reported 96.7% lesions on the head, 83.3% in limbs and 63.3% on the trunk in contrast to our findings. The hematological studies revealed a significantly (P<0.05) increased TLC in dogs with demodicosis. The mean values of TLC (10^3/mm) were 7.89±0.42, 13.28± 0.60and 9.73± 0.58 in apparently healthy, demodectic dogs before and after treatment, respectively. Elevated TLC, in all Demodex spp. infected groups might be due to prolonged antigenic stimulus. Similar justification also made by Patel *et al.,* (2005) [9], Dadhich and Khanna, (2008) [10], Singh *et al.,* (2011) [11], Sakina *et al.,* (2012) [8], Reddy *et al.,* (2014) [12] and Beigh *et al.,* (2013) [13]. After treatment, marked reduction in TLC were found in all treated groups and mean TLC values for these groups vary non-significantly (p>0.05) comparing with normal healthy groups showing positive response in different treatment groups. The decrease in TLC following therapy may be due to resolution of infection by using miticidal therapy. Leukocytosis with neutrophilia and lymphopenia, which were highly significant (p<0.05) changes observed in the present study were in accordance with the findings of Pradhan *et al.,* (2012) [14] and Arora *et al.,* (2013) [15]. The mean value of random blood sugar infected group A, B and C were 69.71±3.84, 85.80±5.88 and 70.89±4.49respectively, while the mean value of apparently healthy group was 87.27±2.75. Post treatment value of random blood sugar significantly changed in group A, B and D to 79.03±3.63, 79.61±4.43 and 80.62±2.42 respectively. The mean value of RBS of clinically infected groups significantly lower (p<0.05) in comparison to apparently healthy control group A, group C except group B, showed hypoglycaemia which might be due to increase consumption glucose during inflammatory reactions as suggested by Sakina *et al.,* (2012) [8]. The mean value of treated group showed nonsignificant (p>0.05) changes in comparison with healthy control. The lowering of RBS was seen attributed to stress induced reduction of blood glucose in infected dogs. After treatment dogs were appeared almost physically nearer to the healthy control group. Treated group, B and C treated with methanolic extract of moringa and N-acetyl-l-cysteine as antioxidants showed better recovery towards healthy group D in comparison to treated group A. Variation in mean value of total serum protein (g/dl) and albumin (g/dl) values of healthy group were 6.12±0.10 and 1.60±0.10 respectively, changes in mean TSP value of clinically infected group A, B and C were 8.15±0.10, 8.01±0.39and 7.80±0.14 and after therapy were 7.47±0.22, 6.61±0.49and 5.82±0.08, respectively. The mean value of albumin of infected group A, B and C noted as 2.080.05, 2.17±0.16 and 2.01±0.06 respectably and treated group were 2.49±0.04, 2.65±0.99and 2.78±0.09respectively. There was a significant (p<0.05) increase in total proteins and significant (p<0.05) decrease in albumin in demodectic dogs before therapy compared with healthy dogs and after therapy.In present study, dogs with demodicosis had elevated total serum protein and reduced albumin levels compared to healthy control group dogs. These findings agreed with Sakina *et al*., (2012) [8], Chakraborty and Pradhan, (2015) [16] and Haleem *et al*., (2015) [17] who reported increased total serum proteins but decreased albumin in dogs with demodicosis. Increased total serum protein might be due to excessive break down of protein due to trauma of skin by proliferation of mites, hypoalbuminemia could be attributed to loss of albumin through injured skin in chronic skin disease disorder as reported by Jain, (1986) [18] and Sakina *et al*., (2012) [8]. The mean value T_3 (ng/dl), T_4 (µg/dl) and TSH (ng/ml) are depicted in table 4.9. The mean value T_3 (ng/dl), T_4 (µg/dl) and TSH (ng/ml) were 1.59 ± 0.05, 2.66 ±0.09 and 2.36 ± 0.08 in apparently healthy; 1.15 ±0.03, 2.35± 0.04 and 3.68 ± 0.11 in demodectic infected dogs before therapy and 1.63 ± 0.06, 2.62 ± 0.04 and 2.10 ± 0.06 after treatment, respectively. There was statistically significant (p<0.05) decrease in T3 and T4 values and increased TSH values in infected group compared to healthy and treated groups. Finding of the present study were similar to Mederle *et al.,* (2010) [19] and Haleem *et al.,* (2015) [17], who considered presence of an association between hypothyroidism and demodicosis, presumably because of suppression of the immune system allowing proliferation of mites. Thus, the increased numbers of mites and/or microbes due to immunosuppression could cause skin disease like demodicosis. After therapy, the values reached to normal limits which could be due to improved immunity levels in dogs because of therapeutic management.

Conclusion

Hence, from present study it can be concluded that the above described biochemical parameters may be considered as prognostic marker in the treatment of clinical demodicosis in dogs. Further research is required to measure the role of different inflammatory markers in the clinical case of demodicosis in dogs.

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