Haemato-Biochemical Changes in Canine Demodicosis

Moneesh Thakur¹*, Hriyadesh Prasad¹, Kalyan Sarma¹, Radhika Thakur², A.K. Samanta³, Albert Debbarma¹, Arindam Bhowmik¹, Prasenjit Debnath¹ and Abhijit Deka⁴

¹Department of Veterinary Medicine, ³Department of Animal Nutrition, ⁴Department of Veterinary Pathology, College of Veterinary sciences and Animal Husbandry, Selesih, Aizawl, Central Agricultural University, Mizoram-796015, India
²Department of Basic Sciences, College of Forestry, Dr. Yashwant Singh Parmar University of Horticulture and Forestry, Nauni, Solan, Himachal Pradesh-173230, India

*Corresponding author

A B S T R A C T

Twenty four dogs brought to Teaching Veterinary Clinical Complex C.V.Sc. & A.H. Selesih suffering from demodectic mange were included in the present study. These were randomly allotted to four groups irrespective of sex, breed and age. Ten apparently healthy dogs were chosen to serve as apparently healthy dogs were chosen to serve as control. Various haematobiochemical parameters like Hb, PCV, TLC, TEC, DLC, MCV, MCH, blood glucose, total protein, albumin, globulin, A:G ratio of affected dogs and control were studied. In all the four affected groups, the mean values of Hb, PCV and TEC were significantly lower (P<0.01) and MCV and MCH were significantly higher (P<0.01). The mean blood glucose levels, total protein and albumin was significantly lower (P<0.01) with a significant increase (P<0.01) in mean plasma globulin in all the dogs suffering from demodicetic mange when compared to corresponding values of healthy control group. Leucocytosis, neutropilia, eosinophilia and lymphopenia were also observed in affected group. A significant decrease (P<0.01) was also observed in albumin: globulin ratio in affected animals in contrast to healthy control group.

Keywords: Mange, Dogs, Sex, Breed, Age

Introduction

Dog is the first carnivore to be domesticated and have been utilized for hunting, patrolling in police services, in wars and as companion. The development of dog is as obscure as evolution of man himself and its presence in social status as a pet has been increasing continuously as companion in most of the countries. The pet has got the inherent psychological instinct for companionship like human urge and the emotional bondage of a woman to its pet is often more closer than a mother to her child. Being the largest organ,
skin protects the animal from external injuries and gives a charming look to the animal. It can also be said that the skin is the mirror of dog’s body to some extent. Of the various clinical problems in dogs, dermatological complaints are recurrent and complicated.

Skin disorders are among the most common health problems, they vary from acute self-limiting problems to chronic or long-lasting problems requiring life-time treatment (Munjal, 2012). Inadequate prevalence data on common disorders have hampered efforts to prioritize health reforms of dogs (Neill et al., 2014).

Canine demodicosis is a common non-contagious inflammatory parasitic dermatosis characterized by excessive proliferation of D. canis within the hair follicles and sebaceous glands (Caswell et al., 1997; Ihrke et al., 2005; Scott et al., 2001; Singh et al., 2001; Verde et al., 2005).

Receptivity of dogs to demodicosis is influenced by numerous intrinsic and extrinsic factors. Intrinsic factors like hereditary predisposition, alterations in skin’s structure and biochemistry, immunological disorders, breed, age and hormonal status (hypothyroidism and hyperadrenocorticism). Extrinsic factors include alimentation, fitness, presence of stress factors and presence of other diseases or pathogens (Mederle et al., 2010; Singh et al., 2014).

Scott et al., 2001 was classified demodicosis into localized and generalized forms based on the extent of the affected body area. In localized form, skin lesions are restricted to one body area with good prognosis and the majority of cases spontaneously resolved without miticidal treatment. Generalized form further classified into juvenile-onset and adult-onset, skin lesions spread over the whole body and have a poor prognosis.

Materials and Methods

Animals attending Teaching Veterinary Clinical Complex College of Veterinary Sciences and Animal Husbandry, Selesih, Aizawl, Mizoram for various skin infections were routinely subjected to skin scrapping examination. Twenty four dogs confirmed for demodectic mange infection were included in present study. In addition, ten apparently healthy dogs were chosen to serve as control. The affected dogs, irrespective of sex, breed and age were allotted randomly to four groups, (Group A, N= 6), (Group B, N= 6), (Group C, N= 6) and healthy dogs (N= 10) were allotted to control group for haemato-biochemical investigations.

From each dog under study, approximately 6ml of blood was collected on day 0 through cephalic and recurrent tarsal vein puncture for haemato-biochemical estimations. For haematological study approximately 1ml of blood was kept separately. For biochemical analysis, a portion of blood was centrifuged at 1500 rpm for 15 to 20 minutes and plasma was harvested. The plasma was kept in deep freezer (-20°C) till further use. Haematobiochemical parameters like Hb, PCV, TLC, TEC, DLC, MCV, MCH, blood glucose, total protein, albumin, globulin, A:G ratio of affected and healthy animals were studied. Haemogram and leukogram were studied as per the procedure described by Jain (1986).

Blood biochemical viz. blood glucose, total protein, albumin, globulin were estimated using specific diagnostic kits.

Results and Discussion

The mean values of Hb (g/dl), MPV (fl), TLC, TEC, DLC (%), PCV(%), MCV (fl), MCH (pg), blood glucose (mg/dl), total protein (g/dl), albumin (g/dl), globulin (g/dl), A:G ratio are illustrated in Table 1.
The mean values of Hb, PCV and TEC were significantly lower (P<0.01) and MCV and MCH were significantly higher (P<0.01) in dogs suffering from demodectic mange as compared to control group indicating macrocytic anaemia in affected groups. This anaemia might be due to stress arising from the disease. Similar findings were reported by Gupta and Prasad (2001) and Soodan et al., (2005). The demodectic dogs in general had significantly higher (P<0.01) TLC, neutrophils and eosinophilic count than healthy control. Leukocytosis along with neutrophilia and eosinophilia concurred with the findings of Sharma et al., (2005). The generalized inflammation and response of leucocytes to prolonged antigenic stimulus in the form of chronic demodex mite infection may be responsible for leukocytosis; eosinophilia may be a reflection of hypersensitivity to persistent demodex mites in tissues. Dhume et al., (2002) Lymphopenia reported in affected animals during present study stimulated the findings of Nair and Nauriyal (2007). Lymphopenia might be due to the reason that cell mediated immunity plays an important role in fighting against demodectic mites.

### Table 1 Haematobiochemical changes ±in demodectic dogs and control group (Mean±SE)

| Parameters          | Control (n=10) | Group A (n=6) | Group B (n=6) | Group C (n=6) |
|---------------------|---------------|--------------|--------------|--------------|
| Hb (g/dl)           | 12.22 ±0.04   | 10.24 ±0.11**| 10.47 ±0.09**| 10.16 ±0.14**|
| PCV (%)             | 38.4± 0.60    | 31.78 ±0.43**| 32.66 ±0.33**| 31.33 ±0.40**|
| TLC (x 10³/µl)      | 9.25 ±0.06    | 11.35 ±0.11**| 11.74 ±0.13**| 11.68 ±0.07**|
| TEC (x10⁶/µl)       | 6.04 ±0.06    | 4.14 ±0.21**  | 4.0 ±0.21**   | 4.33 ±0.19**  |
| Neutrophils (%)     | 69.1 ±0.62    | 80.44 ±0.50** | 79.77 ±0.70**| 80.22 ±0.52**|
| Lymphocytes (%)     | 27.10 ±0.32   | 10.56 ±0.33** | 11 ±0.57**   | 11.33 ±0.37**|
| Monocytes (%)       | 0.80 ±0.20    | 1.04 ±0.24    | 1.22 ±0.27   | 0.77 ±0.28   |
| Eosinophils (%)     | 2.6 ±0.26     | 5.33 ±0.62**  | 8.0 ±0.28**  | 7.8 ±0.51**  |
| Basophils (%)       | 0.0 ±0.0      | 0.0 ±0.0      | 0.0 ±0.0     | 0.0 ±0.0     |
| MCV (fl)            | 62 ±0.47      | 75± 0.53**    | 81 ±0.87**   | 71 ±0.59**   |
| MCH (pg)            | 21± 0.69      | 23± 0.63**    | 25 ±0.82**   | 23± 0.53**   |
| Blood Glucose (mg/dl)| 94.5 ±0.65    | 82.1± 1.4**   | 84.66 ±1.0** | 77.33 ±0.47**|
| Total Protein (g/dl)| 6.36 ±0.03    | 6.36± 0.03**  | 5.32 ±0.05** | 5.26 ±0.04** |
| Albumin (g/dl)      | 3.03 ±0.04    | 1.62± 0.05**  | 1.55 ±0.05** | 1.55 ±0.07** |
| Globulin (g/dl)     | 3.3± 0.04     | 3.72± 0.02**  | 3.72± 0.04** | 3.73± 0.05** |
| A:G Ratio           | 0.91 ±0.01    | 0.41 ±0.01**  | 0.4 ±0.01**  | 0.41± 0.01** |

*Significant at 5% (P<0.05)
**Significant at 1% (P<0.01)
n = No. of animals in each group

The mean blood glucose levels in affected animals was significantly lower (P<0.01) than that of healthy control, indicating hypoglycaemia in them which might be due to increased need for glucose during inflammatory reactions as suggested by Sharma (2006) and Gupta (2008). A significant decrease (P<0.01) in mean total protein levels in demodectic dogs as compared to the healthy group indicated hypoproteinaemia which was in agreement with the observations of Biswas et al., (2012) and Solanki et al., (2007). The mean value of plasma albumin revealed a significant decrease (P<0.01) as against a significant increase (P<0.01) in mean plasma globulin in
all affected groups indicating hypoalbuminemia and hyperglobinemia respectively. A significant decrease (P<0.01) was also observed in albumin: globulin ratio in contrast to healthy control group due to decrease in plasma albumin and relative increase in plasma globulin concentration. This finding was in concurrence with the observations of Biswas et al., (2002) and Jyotsna and Gupta (2005). Decreased levels of plasma albumin in the present study may be result of excessive breakdown of proteins due to trauma to skin and proliferation of mites. Elevated plasma globulin level may be attributed to chronic skin disease.

Acknowledgment

The help rendered by Miss Radhika Thakur (Ph.D. Scholar, Statistics), College of Forestry, Department of Basic Sciences, Dr. Yashwant Singh Parmar University of Horticulture and Forestry, Nauni, Solan (Himachal Pradesh) for statistical analysis in the present study is duly acknowledged.

References

Baker, H. and Frank, O. 1968. Clinical Vitaminology: Methods and Interpretation. Interscience Publishers, London. p 295.

Biswa, L., Mukhopadhyay, S.K., Bhattacharya, M.K., and Roy, S. 2002. Interacademia, 6: 734-736.

Caswell, J.L., J.A. Yager, W.M. Parker and P.F. Moore. 1997. A prospective study of the immunophenotype and temporal changes in the histologic lesions of canine demodicosis. Vet Pathol., 34: 279-287.

Dhume, G.V., Shardode, D.B., Dakshinkar, N.P., Shrikhande, G.B. 2001. Haematobiochemical investigation in canine demodicosis. The blue cross book vol 19, 16-17.

Gupta, A. 2008. Investigation on dietary and environmental component vis- A- vis skin health in dogs. M.V.Sc. thesis. CSK himachal Pradesh Krishi Vishva Vidyalaya, Palampur, India.

Gupta, N. and Prasad B. 2001. Clinicodiagnostics and therapeutic management of acariasis in dogs. Indian Vet. Med., 21: 73-75.

Ihrke, P.J. 2005. Small Animal Dermatology, Canine and Feline demodicosis. Proceeding of the NAVC North American Veterinary Conference, Orlando, Florida, pp: 279-280.

Jain, M.C. 1986. Hemolytic anemia associated with some infectious agents. Schalm’s Veterinary Hematology, 4th Ed., Lea and Febiger, Philadelphia, pp. 599-601.

Jyotsana, S. and Gupta, S.K. 2005. Serum protein profile in demodectic mange. Indian Vet. Med. J., 28: 35-37.

Merderle, N., Gh. Darabus, I. Oprescu, S. Morariu, M. ILIE, D. Indre and O. Mederle. 2010. Review Article, Diagnosis of canine demodicosis. Sci. Parasitol., 11: 20-23.

Munjal, R.S. 2012. Common dermatological diseases by bacteria and fungi in pet dogs. Ind. J. Fund. Appli. Life Sci., 2: 207-209.

Nair, S.S. and Nauriyal, D.S. 2007. Diagnostic significance of hematological changes associated with various canine dermatoses. Intaspolivet, 8(1): 68-72.

Neill, D.G., D.B. Church, P.D. McGreevy, P.C. Thomson and D.C. Brodbelt. 2014. Prevalence of Disorders Recorded in Dogs Attending Primary-Care Veterinary Practices in England. PLOS ONE, 9: 1-16.

Scott, D.W., W.M. Miller and C.E. Griffin, 2001. Parasitic skin diseases. In: Muller, K. (ed) Small Animal Dermatology, 6th
Sharma, S.A., Ahmed, N.M., Thankichalam, M., and Sundararaj, A. 2005. Haematobiochemical changes in canine demodicosis. Indian Vet. J., 82: 396-401.

Sharma, S.K. 2006. Etiology haematobiochemical and therapeutics of skin disease in canine. M.V.Sc. thesis. Sher-E-Kashmir University of Agric. Sciences and Technology, Jammu (J&k), India.

Singh, S.K. and U. Dimri. 2014. The immune-pathological conversions of canine demodicosis. Vet. Parasitol., 203: 1-5.

Singh, S.K., U. Dimri, M.C. Sharma, D. Swarup, B. Sharma, H.O. Pandey and P. Kumari. 2011. The role of apoptosis in immunosuppression of dogs with demodicosis. Vet. Immunol. Immunopathol., 144: 487-492.

Solanki, J.B., Hasnani, J.J., Patel, D.M., Patel, P.V. and Raval, S.K. 2007. Canine demodicosis in Anand J. Vet. Parasitol, 2191): 79-80.

Soodan J.S., Yadav, A., and Khajuria J.K. 2005. Comparative efficacy of some caricides against demodicosis in dogs. Intas Polivet, 6(2): 335-337.

How to cite this article:
Moneesh Thakur, Hriyadesh Prasad, Kalyan Sarma, Radhika Thakur, A.K. Samanta, Albert Debbarma, Arindam Bhowmik, Prasenjit Debnath and Abhijit Deka. 2018. Haematobiochemical Changes in Canine Demodicosis. Int.J.Curr.Microbiol.App.Sci. 7(11): 2958-2962. doi: https://doi.org/10.20546/ijcmas.2018.711.338