The Pharma Innovation Journal 2019; 8(4): 318-321

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277- 7695
ISSN (P): 2349-8242
NAAS Rating: 5.03
TPI 2019; 8(4): 318-321

www.thepharmajournal.com
Received: 14-02-2019
Accepted: 18-03-2019

Peer Rayees Aziz
Teaching Associate, Department of Veterinary Medicine, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Shiv Kumar Sharma
Associate and Head, Department of Veterinary Medicine, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Shantanu Kumar Kuldeep
Teaching Associate, Department of Veterinary Surgery and Radiology, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Hiteshwar Singh Yadav
Teaching Associate, Department of Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Naresh Kuntal
Teaching Associate, Department of Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Correspondence
Peer Rayees Aziz
Teaching Associate, Department of Veterinary Medicine, College of Veterinary and Animal Sciences, Navania, Vallabhnagar, Udaipur, Rajasthan, India

Hemato-biochemical and electrolyte alterations in a flock of goats affected with peste des petits ruminants

Peer Rayees Aziz, Shiv Kumar Sharma, Shantanu Kumar Kuldeep, Hiteshwar Singh Yadav and Naresh Kuntal

Abstract
A flock having 24 goats with the history of dullness, depression, anorexia, diarrhea, shivering, nasal discharge and matting of eyelids came in the Veterinary Clinical Complex of CVAS, Navania, Vallabhnagar, Udaipur for treatment. Clinical examination revealed high fever (104^0F - 106^0F), increased respiratory rate, tachycardia, oculo-nasal discharge, dyspnoea, coughing, sneezing, non-haemorrhagic diarrhea, rough hair coat, halitosis, severe dehydration and emaciation. In one goat there was presence of corneal opacity (Unilateral) which is also an additional sign of PPR. Superficial lymph nodes were also palpable. Because of diarrhea the hind quarters were soiled with feces. Feces were checked for parasitic ova but result was negative. Diagnosis of PPR was done based on clinical signs and symptoms. Blood samples of the affected goats were collected and were examined for hemato-biochemical and electrolytes. All the values of hematological parameters such as Hb, PCV, TEC, TLC and Lymphocytes were decreased except Neutrophils which was increased. The biochemical parameters such as AST, ALT, ALKP, BUN, Creatinine and Bilirubin were increased whereas Albumin, Glucose and TP were decreased. The serum electrolytes were analysed among which Sodium and Chloride were increased whereas Potassium was decreased.

Keywords: PPR, goats, hemato, biochemical, electrolyte imbalances

Introduction

India is a vast country with a population of 137.17 million goats and 65.06 million sheep, of which Rajasthan shares 21.67 million and 9.08 million goats and sheep respectively. Goat rearing is an integral part of farming system in India. Peste des petits ruminants is a disease of small ruminants (sheep and goats). It is caused by a Morbillivirus of family Paramyxoviridae. It is a highly contagious, infectious and an economically important disease which causes high morbidity and mortality rates. Kids over 4 months and under 1 year of age are most susceptible to the disease. The World Organization for Animal Health has recently identified PPR as a serious notifiable and economically important trans-boundary viral disease of sheep and goats to jeopardize with high morbidity and mortality (Diallo *et al*., 2007; Folitse *et al*., 2017; Birindwa *et al*., 2017) [9, 11, 6]. Clinically, the disease is characterized by high fever, oculo-nasal discharges, necrotizing and erosive stomatitis, diarrhoea, and dyspnea, bronchopneumonia followed by either death or recovery from the disease (Balamurugan *et al*., 2012; Sharma *et al*., 2012; Jaisree *et al*., 2018) [2, 26, 13]. Animals that are unprotected by means of devoid of immunization has the morbidity rate can be up to 100% and mortality may be 20% to 90% particularly in goats (Torsson *et al*., 2016) [28].

Transmission of the PPR virus in nature occurs primarily through direct contact with goats from low altitude pasture lands to high infected animals and by inhalation of the infectious pasture lands in summers and from high altitude to low aerosol produced by a combination of sneezing and altitude pasture lands in winters is common (Mahajan *et al*., 2013; Baazizi *et al*., 2017) [18, 1]. Infection rates in goats rise with age, and the disease, which varies in severity, is rapidly fatal in young animals and has a controversial effect on the sex (Nargesi *et al*., 2012) [20]. The small stock management practices, seasons, transportation, infected animals that newly introduce into the flock are the major risk factors for the presence or absence of PPR outbreaks. Alongside this, considerations of breed susceptibility, immune-competence of hosts, and existing parasitic infections also jeopardize the condition (Pope *et al*., 2013) [21]. Excluding clinical signs, and history, complete blood count can also be used for diagnosis that tends to be more useful in countries where diagnostic cost is considered as a burden (Tariq *et al*., 2014) [27].

In India, severity of the disease is more pronounced in goats than in sheep with a combined susceptible population of about 200 million and thus it is one of the major threats to the small ruminant population of the country. PPR virus has affinity for lymphoid organs contributing to marked immune-suppression as indicated by leucopenia, monocytes depletion and lymphopenia. These observations are predominant particularly during acute phase of disease (Rajak *et al*., 2005) [22]. To describe PPR better and to make a fast and accurate diagnosis so that accurate symptomatic and prophylactic treatment may be given, clinical biochemical and haematological findings need to be examined (Das *et al*. 2015) [8].

Material and Methods

A flock having 24 goats with the history of dullness, depression, anorexia, diarrhea, shivering, nasal discharge and matting of eyelids came in the Veterinary Clinical Complex of CVAS, Navania, Vallabhnagar, Udaipur for treatment. Further clinical examination revealed high fever (104^0F - 106^0F), increased respiratory rate, tachycardia, oculo-nasal discharge, dyspnoea, coughing, sneezing, non-haemorrhagic diarrhea, rough hair coat, halitosis, severe dehydration and emaciation. In one goat there was presence of corneal opacity (unilateral) which is also an additional sign of PPR. Lymph nodes were also palpable. Because of diarrhea the hind quarter was soiled with feces. Feces were check for parasitic ova but result was negative.

Fig 1: Nasal discharge in an affected goat

Fig 2: Corneal opacity in an affected goat

Collection of samples for hematology

Blood was collected aseptically from the jugular vein of each goat by adopting a standard protocol. The haematological parameters like Haemoglobin (Hb), Packed Cell Volume (PCV), Total Erythrocyte Count (TEC), Total Leucocyte Count (TLC) were estimated by Auto- analyser (IDEXX Vet Autoread) and Differential Leucocyte Count (DLC) was carried out by Wright's staining technique. For serum biochemical analysis, blood was collected in a sterilized vacutainer from the affected goats. Serum samples were separated and all the serum samples were subjected to evaluate Total Protein, Albumin, Bilirubin, Glucose, Creatinine, Alkaline Phosphatase, Aspartate Transaminase, Alanine Transaminase, Blood Urea Nitrogen and serum electrolytes (Sodium, potassium and chloride ions) were analyzed by automatic analyzer (IDEXX Vet Test) as per standard diagnostic protocol.

Results and Discussion

The mean values of haematological and biochemical parameters in PPR affected and healthy goats are presented in the table. Present investigation showed significant decrease in values of Hemoglobin, Total Erythrocyte Count and Packed Cell Volume in the PPR affected goats as compared to healthy goats, this could be attributed to the erosion, necrosis and congestion associated with PPR, and also to the different phases of the disease, presence of secondary infections and nutritional status. Similar findings were also reported by Sahinduran *et al*., 2012 [23] Das *et al*., 2015 [8] Bari *et al*., 2018 [4] Begum *et al*., 2018 [5].

The total leucocyte count was significantly decreased in PPR affected goats as compared to healthy goats. Similar findings were also recorded by other several workers in PPR affected goats as compared with apparently healthy goats (Kataria *et al*., 2007, Yarim *et al*., 2006, Sahinduran *et al*., 2012, Sharma *et al*., 2012, Bari *et al*., 2018, Islam *et al*., 2018) [15, 31, 23, 26, 4, 12] which was due to the lymphotropic nature of the virus and also the inhibition of peripheral blood lymphocytes proliferation by PPR virus (Maina *et al*., 2015) [19].

In differential leucocytes count there was decrease in lymphocyte count and corresponding increase of neutrophil count in the PPR affected animals as compared to healthy goats. Similar observations was also made by several workers (Kataria *et al*., 2007, Kul *et al*., 2007, Yarim *et al*., 2006, Sahinduran *et al*., 2012, Sharma *et al*., 2012, Maina *et al*., 2015, Balogun *et al*., 2017, Ugochukwu *et al*., 2018) [15, 16, 31, 23, 26, 19, 3, 29]. This was due to necrosis of the lymphocytes in lymph nodes, spleen and peyer's patches as the virus was lymphotropic like that of rinderpest virus (Scott 1981, Jones 1997) [25, 14].

Table 1: Hemato-biochemical and electrolyte levels in the PPR affected goats.

S. No.	Parameters	Normal Range	Range in case of PPR	Test (PPR Cases) Value (Mean±SE)
1.	Hb (g/dl)*	8-12 (10)	3.9-6.6	5.47±0.1439
2.	PCV (%)	22-38 (30)	15-21.03	17.88±0.3572
3.	TEC ($\times 10^6$/µL)	8-18 (13)	2.5-4.4	3.33±0.1144
4.	TLC ($\times 10^3$/µL	4-13 (8.5)	2-3-13	2.51±0.1178
5.	Lymphocytes ($\times 10^3$/µL)	3-9 (6)	1.98-4	2.55±0.1397
6.	Neutrophils ($\times 10^3$/µL)	1-5 (3)	4-9	6.44±0.2248
7.	TP (g/dl)	6.4-7.0 (6.7)	3-6	4.18±0.1582
8.	Albumin (g/dl)	2.7-3.8 (3.25)	1-2	1.72±0.0895
9.	Bilirubin (mg/dl)	0-0.9 (0.45)	1-2.99	1.74±0.1222
10.	Glucose (mg/dl)	50-75 (62.5)	26-44	33.96±1.0555

11.	AST(µ/L)**	160-500 (330)	564-700	597.96±7.3550
12.	ALT (µ/L)	15.3-52.3 (33.8)	22-28	25.17±0.3544
13.	ALKP (µ/L)	27-387 (207)	395-505	423.87±6.5162
14.	BUN (mg/dl)	12-28 (20)	24-56	30.87±2.1010
15.	Creatinine (mg/dl)	0.9-1.8 (1.35)	2-4	3.19±0.1359
16.	Cl (mEq/L)	105-120 (112.5)	119-136.09	130.42±1.0620
17.	Na (mEq/L)	142-155 (148.5)	165-190	177.25±1.5985
18.	K (mEq/L)	3.9-6.3 (5.1)	2-3.09	2.50±1.1045

Value with Superscript *significantly decreased values at P<0.05 and value with superscript ** significantly increased at P< 0.05 level of significance on the basis of student's T- test.

In biochemical study, Total Protein and Albumin level were significantly lower in PPR affected goats as compared to healthy goats which is due to nephritic damage to the glomeruli that causes increase in permeability of capillary walls of the glomerulus leading to passing of high level of protein from blood to urine. Similar findings were also recorded by others workers (Kataria *et al*., 2007, Das *et al*., 2015, Bari *et al*., 2018, Islam *et al*., 2018, Begum *et al*., 2018) [15, 8, 4, 12, 5].

Bilirubin was significantly higher in PPR affected goats as compared to healthy goats. Similar findings were also recorded by Yarim *et al*., 2006 [31] and Das *et al*., 2015 [8].

The high values for Blood Urea Nitrogen and Creatinine in the serum are indicative of a functional damage to the kidney. Under normal conditions, these substances are not supposed to be present in the blood at high levels, because the function of the kidneys is to excrete them through the urine. But when there is any functional defect or damage to the kidney, such as in PPR infection (possibly due to disruption of the Na^+ - K^+ pump), these substances are found at high levels in the serum; this is in consonance with the findings of Kataria *et al*., 2007 [15], Sahinduran *et al*., 2012 [23], Sharma *et al*., 2012 [26], Balogun *et al*., 2017 [3]. An increased level of the creatinine highlight an increased tropism of the virus towards kidneys.

There was a sharp drop in the blood glucose level (hypoglycaemia), this condition occurs whenever there is a difficulty with the conversion of glycogen to glucose (glycogenolysis). The liver is normally a principal store house for glycogen, but due to the functional impairment as a result of PPR infection, hence the short fall (FAO 1999, Kataria *et al*., 2007, Das *et al*., 2015, Saliki 2015, Balogun *et al*., 2017, Begum *et al*., 2018) [10, 15, 8, 24, 3, 5].

Liver damage, as a result of PPR infection is further confirmed by an excessively high level of AST, ALT and ALKP when compared with the normal physiological values (Daramola *et al*., 2005, Waziri *et al*., 2010, Sahinduran *et al*., 2012, Balogun *et al*., 2017, Begum *et al*., 2018) [7, 30, 23, 3, 5] , these are serum enzymes ordinarily found in the liver, but when there is damage to the liver they are found at high levels in the serum.

The low levels of potassium ions in the infected goats resulted from the dehydration arising from the symptomatic diarrhoea due to PPR infection, this will lead to metabolic acidosis as a result of the drop in pH level in the gastrointestinal tract (reason for increase in serum chloride level). This pH change will in turn stimulate a compensatory mechanism leading to respiratory alkalosis, and hence the rapid respiration for the expiration of carbon dioxide. This condition will result in an increase in blood sodium that will bind to bicarbonates to serve as buffer during PPR infection to aid in the reduction of the pH (Lewis 2016). This drop in potassium will automatically affect the Na^+-K^+ pump mechanism which will evidently disrupt the functionality of most of the systems (especially nervous, cardiovascular and urinary systems) (Das *et al*., 2015) [8].

Conclusion

In the present study, PPRV infection alter haemato-biochemical and electrolyte parameters in goats which is manifested as anaemia, low hematocrit value, hypoalbuminemia, hypoglycemia, lymphocytopenia, hypo proteinemia, azotemic, hypokalemia, neutrophilia, hyper bilirubinaemia, hyper chloremia, hypernatremia. Besides this high level of AST, ALT and ALKP were also evaluated. These valuable data obtained in the present study may be helpful in the diagnosis and treatment (symptomatic) of the disease. In addition to these clinical, and haemato-biochemical parameters, veterinary practitioners should kept in mind for the possible occurence of new outbreaks and for its diagnosis.

References

1. Baazizi R, Mahapatra M, Clarke BD, Ait-Oudhia K, Khelef D, Parida S. Peste des petits ruminants (PPR): A neglected tropical disease in Maghreb region of North Africa and its threat to Europe. PloS One, 2017, 12(4).
2. Balamurugan V, Saravanan P, Sen A, Rajak KK, Venkatesan G, Krishnamoorthy P *et al*. Prevalence of peste des petits ruminants among sheep and goats in India. Journal of Veterinary Science. 2012, 13(3).
3. Balogun FA, Fasanmi OG, Oladipo TA, Popoola MA, Olona JF, Adeoye YD. Field evaluation and confirmation of acute peste des petits ruminant outbreak in a flock of West African dwarf goats in Ibadan, Nigeria. International journal of veterinary science and medicine. 2017; 5(2):175-180.
4. Bari MS, Rana EA, Ahaduzzaman M, Al Masud, A, Das T, Hasan T. Hemato-biochemical parameters of Pesti-des Petits Ruminants (PPR) affected goats in Chittagong, Bangladesh. Journal of Advanced Veterinary and Animal Research. 2018; 5(2):211-217.
5. Begum S, Nooruzzaman M, Parvin M, Mohanto N, Parvin R, Islam MR *et al*. Peste des petits ruminants virus infection of Black Bengal goats showed altered haematological and serum biochemical profiles. Onderstepoort Journal of Veterinary Research. 2018; 85(1):1-10.
6. Birindwa BA, George GC, Ntagereka BP, Christopher O, Lilly BC. Mixed infection of peste-des-petits ruminants and Capripox in goats in South Kivu, Democratic Republic of Congo. Journal of Advanced Veterinary and Animal Research. 2017; 4(4):348-355.
7. Daramola JO, Adeloye AA, Fatoba TA, Soladoye AO. Haematological and biochemical parameters of West African Dwarf goats. Livestock Research for Rural Development. 2005; 17(8):3.
8. Das S, Nath R, Balamurugan V, Choudhury R, Devi M. Haematobiochemical analysis of goats naturally infected

with peste des petits ruminants. International Journal for Research in Emerging Science and Technology. 2015; 2(9):19-24.
9. Diallo A, Minet C, Le Goff C, Berhe G, Albina E, Libeau G. The threat of peste des petits ruminants: progress in vaccine development for disease control. Vaccine. 2007; 25(30):5591-5597.
10. FAO (Food and Agriculture Organisation). Recognizing Peste des Petits Ruminants Field Manual, 1999.
11. Folitse RD, Amemor E, Rejoice EN, Emikpe BO, Tasiame W. Pattern of peste des petits ruminants (PPR) distribution in Ghana (2005-2013). Bulgarian Journal of Veterinary Medicine. 2017; 20(1):51-57.
12. Islam M, Pathak DC, Das S, Rahman T, Sarma S, Borgohaign, R. Hematological and biochemical alterations in goats due to paste des petits ruminant's viral infection. 2018; 6(3):710-713.
13. Jaisree S, Aravindhbabu RP, Roy P, Jayathangaraj MG. Fatal peste des petits ruminants disease in Chowsingha. Transboundary and Emerging Diseases. 2018, 65(1).
14. Jones TC, Hunt RD, King NW. Veterinary Pathology. Edn 3, Baltimore, Lipincott, Williams and Wilkins, 1997, 442-450.
15. Kataria AK, Kataria N, Gahlot AK. Large scale outbreaks of Peste des petits ruminants in sheep and goats in Thar desert of India. Slovenian Veterinary Research. 2007; 44(4):123-132.
16. Kul O, Kabakci N, Atmaca HT, Ozkul O. Natural Peste des petits ruminants virus infection: Novel pathogenic findings resembling other morbilivirus infections. Veterinary Pathology. 2007; 44:470-486.
17. Lewis JL. Metabolic Acidosis. Merck Veterinary Manual. Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Kenilworth, NJ; USA, 2016.
18. Mahajan S, Agrawal R, Kumar M, Mohan A, Pande N. Incidence of Peste des petits ruminants in nomadic sheep and goat of Jammu region. Veterinary World. 2013; 6(7):384-387.
19. Maina SM, Gitao CG, Gathumbi PK. Hematological, Serological and Virological findings in Sheep and Goats Experimentally Infected with lineage iii peste des petits ruminants virus isolates in Kenya. Journal of Experimental Biology and Agricultural Sciences. 2015; 3(1):81-88.
20. Nargesi I, Kolveiri MP, Maghsoudi O. Survey on Peste des Petits Ruminants (PPR) in small ruminants. Annals of Biological Research. 2012; 3:4842-4844.
21. Pope RA, Parida S, Bailey D, Brownlie J, Barrett T, Banyard AC. Early events following experimental infection with peste-des-petits ruminants virus suggest immune cell targeting. PloS One. 2013; 8(2):e55830.
22. Rajak KK, Sreenivasa BP, Hosamani M, Singh RP, Singh SK, Singh RK *et al*. Experimental studies on immunosuppressive effects of peste des petits ruminants (PPR) virus in goats. Comparative Immunology, Microbiology & Infectious Diseases. 2005; 28:287-296.
23. Sahinduran S, Albay MK, Sezer K, Ozmen O, Mamak N, Haligur M *et al*. Coagulation profile, hematological and biochemical changes in kids naturally infected with Peste des petits ruminants. Tropical Animal Health and Production. 2012; 44:453-457.
24. Saliki JT. An overview of Peste des Petit Ruminants. Merck Veterinary Manual. Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Kenilworth, NJ, USA, 2015.
25. Scott GR. Rinderpest and peste des petits ruminants. In: Viral Diseases of Food Animals. Gibbs, EPJ. Edn. Vol. II, Disease Monographs. Academic Press, New York, 1981, 401-425.
26. Sharma CS, Mehta HK, Prakash MM, Shukla PC. Studies on clinico-haemato-biochemical changes in peste des petits ruminants in goats. Veterinary Practitioner. 2012; 13(2):322-325.
27. Tariq A, Shahzad A, Kausar R, Fiaz UA, Jan SS. An easy Approach towards Diagnosis of Peste Des Petit Ruminant (PPR) through Clinical Examination. Research Journal for Veterinary Practitioners. 2014; 2(1):1-3.
28. Torsson E, Kgotlele T, Berg M, Mtui-Malamsha N, Swai ES, Wensman JJ *et al*. History and current status of peste des petits ruminants virus in Tanzania. Infection Ecology & Epidemiology. 2016; 6(1):32701.
29. Ugochukwu ICI, Ugochukwu EI, Chukwu CC. Haematological parameters and serum biochemical assay of West African Dwarf goats infected with peste des petits ruminants virus in Nsukka, Enugu State. Comparative Clinical Pathology. 2018; 27(1):13-19.
30. Waziri MA, Ribadu AY, Sivachelvam N. Changes in the serum proteins, hematological and some serum biochemical profiles in the gestation period in the Sahel goats. Veterinarski Arhiv. 2010; 80:215-24.
31. Yarim GF, Nisbet C, Yazici Z, Gumusova SO. Elevated serum total sialic acid concentrations in sheep with Peste des petits ruminants. Medycyna Weterynaryjna. 2006; 62(12):1375-1377.