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Epidemiology, Haemato-biochemical and Pathological Changes Related to Field Outbreaks of PPR in Small Ruminants in Odisha

P.K. Rath, S.K. Panda, B.P. Mishra, R. Mishra, D.K. Karna

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ABSTRACT

Background: Odisha experiencing sporadic outbreaks of Peste des petits ruminants (PPR) throughout the year. There is a scarcity of available literature on PPR in Odisha till today. This is the first ever detail investigative approach in the state undertaken with an objective to corelate the epidemiological risk factors, haemato-biochemical and pathological changes in natural field outbreaks occurring in eight different districts.

Methods: Fourteen field outbreaks of PPR were evaluated clinically as well as epidemiologically and confirmed through polymerase chain reaction (PCR). Blood, serum, faecal and tissue samples were collected to observe haemato-biochemical and pathomorphological changes to asses disease severity.

Result: Present study concluded an overall mortality rate of 46.81%. Chi-square analysis revealed significant highest prevalence among 7-12 months (46.13%) age, Ganjam breed (45.51%) and females (80.49%). Frequent migration among the border areas along with poor management and helminthic infection was major precipitating factor. There was polycythemia along with neutrophilia and lymphopenia. Significant increase in alanine transaminase (ALT), aspartate aminotransferase (AST), K^{+} and Ca^{+2} along with creatinine, urea and blood urea nitrogen (BUN) BUN was observed in affected flocks. Antero-ventral consolidation of lungs, syncytia and presence of both eosinophilic intranuclear and intracytoplasmic inclusion bodies were major pathological changes.

Key words: Goats, PPR, Pathology, Sheep, Small ruminants.

INTRODUCTION

Peste des petits ruminant (PPR) is an economically important epizootic disease of cloven-hoofed small ruminants caused by genus *Morbillivirus* of *Paramyxoviridae family*. PPR is enzootic in India with consistent outbreaks round the year causing heavy mortality and morbidity (Mahajan *et al.* 2017). The existence of PPR has been recognized in India since 1989 as an epizootic in southern states (Shaila *et al.* 1996).

First occurrence of PPR outbreak in Odisha was reported in a Govt. goat farm stationed at Ghatgaon, Keonjhar (Nayak *et al.* 1997). The acute clinical nature of the disease causes extensive damage in affected small ruminant population with serious economic loss (Chauhan *et al.* 2009). Odisha houses nearly 6.39 million goats and 1.3 million sheep as per 2019 livestock census. Various epidemiological determinants like age, breed, sex, season, inter-species contact, migration, exchange of animals, grazing pattern are important individual risk factors for augmenting the PPR infection among the susceptible flocks (Shuaib *et al.* 2014). Clinical severity of PPR often magnified due to pre-existing parasitic infections (Selvaraju, 2014) owing to poor management as often practiced by rural landless and marginal farmers.

Present study considered as a detailed descriptive investigations on PPR in Odisha, designed to assess clinical, haemato-biochemical profiles, molecular confirmation along with patho-anatomical and histological changes of PPR infected sheep and goats so as to make a fast and accurate diagnosis in evaluating the disease severity by the field veterinarians by refreshing their knowledge.

Department of Veterinary Pathology, College of Veterinary Science and Animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar-751 003, Odisha, India.

Corresponding Author: P.K. Rath, Department of Veterinary Pathology, College of Veterinary Science and Animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar-751 003, Odisha, India. Email: drpkrath78@gmail.com

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MATERIALS AND METHODS

There were fourteen outbreaks suggestive of PPR duly investigated in eight districts of Odisha (Chart 1) comprising 361 small ruminants (Goat-286, Sheep-75) during 2014-16. Field diagnosis were mostly done through typical clinical signs suggestive of PPR. Oral and nasal swabs transported in cold conditions to laboratory of the Department of Veterinary Pathology, College of Veterinary Science and Animal Husbandry, Odisha University of Agriculture and Technology (OUAT), Bhubaneswar, Odisa and stored in deep freezer (-80°C). RNA extraction was done through Trizol method (Invitrogen, USA). Quality and quantity assessment of extracted RNA was done by Thermo scientific Nanodrop spectrophotometer from which cDNA was synthesized by iScript cDNA synthesis kit (Bio Rad). Primers used in the

study listed in Table 1. Virus identification was done through PCR with initial denaturation step for 5 min at 95°C, then 30 cycles of denaturation at 95°C for 30 secs, annealing step at 54°C for 40 secs, extension done for about 45 sec at 68°C and final extension step at 68°C for 5 min. PCR products were analyzed through agarose gel.

Blood was collected aseptically from 63 goats (Control-9, Affected-54) and 15 sheep (Control-5, Affected-10) for routine hematology and serum biochemical study.

Fecal samples were collected and examined under light microscopy. Necropsy of dead sheep and goats was conducted at the outbreak site. Representative tissue samples from all organs fixed in 10% neutral formalin and processed for routine hematoxylin-eosin staining in the Department of Veterinary Pathology of the College.

The data recorded for various haemato-biochemical parameters were analyzed by using SAS software (Local, W32_7PRO) through student's *t-test* to observe any differences between infected and control groups. A $p \leq 0.05$ value was accepted as statistically significant.

RESULTS AND DISCUSSION

Epidemiology

Natural field outbreaks suggestive of PPR in several districts of Odisha were duly investigated over a period of 2014-16. Flocks of sheep and goats pertaining to various ages and breeds showing distinct clinical signs indicative of PPR were assessed clinically. Present study concluded a higher morbidity, mortality and case fatality rate in affected goats as compared to sheep (Chart 2) which is in agreement with

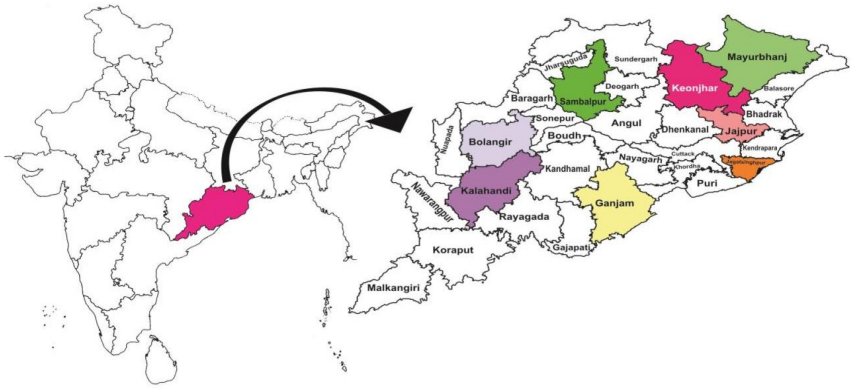

Chart 1: Map showing details of sample collection area.

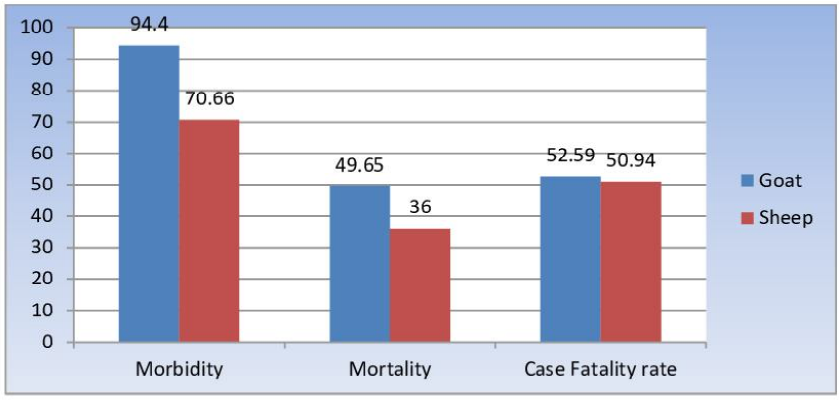

Chart 2: Morbidity, mortality and case fatality rate in sheep and goats infected with PPR.

Table 1: List of the primers used for the DNA amplification and detection of PPRV gene.

Name	Primer sequence (5'-3')	Reference
NP1	1232 - GTCTCGGAAATCGCCTCGCAGGCT-1255	Couacy-Hymann *et al.* 2002
NP2	1583 - CCTCCTCCTGGTCCTCCAGAATCT - 1560	
NP3	(-106) - ACCAAACAAAGTTGGGTAA - (-87)	Generated during the study
NP4	(+79) - GCTTGGACCTGGGTCCTAAG - (+60)	
Real-time primers		
NP6	502 - GCCTCCATACTAGCACAAGTCTGG - 525	Generated during the study
NP7	624 - TTCCCCAATCACTCTCCTTTG - 604	

Abdalla *et al.* (2012). Higher mortality and morbidity in goats might be due to increased number of new susceptible younger population replaced every year due to their higher production efficiency and adults are replaced regularly through slaughter for meat preference (Gitao *et al.* 2016). Relatively less sample size, increased innate resistance and genetic makeup may also play some roles in the occurrence of low mortality and morbidity rate in sheep (Singh *et al.* 2004). Chi-square analysis pertaining to epidemiological risk factors like age, sex and breed showed a significant variation with highest prevalence among growers (n= 149), Ganjam breed (n = 147) and females (n = 260). Higher occurrence of PPR among the young growers was associated with frequent exposure to contaminated pasture as well as decreased maternal derived protective response as suggested by Aziz *et al.* (2019). Females outnumbered the males in present study as most of poor farmers prefer to keep productive females (Islam *et al.* 2012). Ganjam breed of sheep and goat gracefully retained relatively for longer years by certain farming communities in several pockets of Odisha which makes them more susceptible for getting infected over the time. (Saha *et al.* 2005). There was history of inter-state migration of animals as well as purchasing of small ruminants from local markets near the border areas in all outbreaks which may be regarded as a possible contributing factor for field outbreaks (Kumar *et al.* 2001). Most of the flocks, comprising both sheep and goats, co-housed in natural environment under the open sky outside the village near foothills except rainy season. Faecal sample examinations under light microscopy recorded highest prevalence of strongyle spp. (42.66%) followed by Amphistomes (26.60%), Trichuris (16.97%), coccidia (8.71%) and Strongyloides spp. (5.04%) in 218 (67.49%) affected animals irrespective of their age, breed and sex. There was no vaccination as well as deworming done within one year as per owner's statement. Poor management with lack of proper awareness among the local shepherds most often result in nutritional imbalances and increased helminthic infections among small ruminants thus altering disease susceptibility (Muthuchelvan *et al.* 2017).

Clinical signs

Present study reported various clinical signs such as depression (96.90%), oculo-nasal discharges (Fig 1A) (86.06%), fever (85.44%), diarrhea (79.87%), respiratory difficulty (53.86%), matting of eyelids (47.36%), crusts on nose and eyes (43.03%), stomatitis (38.08%) and nodules

Table 2: Mean ±SE of haematological parameters in goats affected with PPR.

Parameters	Goats	Mean±SE	P-value
Hb (gm/dl)	Control (n=9)	8.31±0.16	P<0.0001
	Affected (n=54)	10.29±0.11	
TEC (M/µl)	Control (n=9)	10.64±0.39	P<0.0001
	Affected (n=54)	12.08±0.09	
PCV (%)	Control (n=9)	24.15±0.37	P<0.0001
	Affected (n=54)	35.12±5.4	
TLC (10^3/µl)	Control (n=9)	10.80±0.49	P<0.0001
	Affected (n=54)	20.28±0.20	
MCV (fl)	Control (n=9)	15.95±0.37	P>0.1789
	Affected (n=54)	15.28±0.19	
MCH (pg)	Control (n=9)	6.75±0.10	P<0.0001
	Affected (n=54)	7.70±0.05	
MCHC (g/dl)	Control (n=9)	39.86±0.11	P<0.0001
	Affected (n=54)	51.04±0.09	
DLC			
N %	Control (n=9)	36.44±1.17	P<0.0001
	Affected (n=54)	54.01±0.36	
L %	Control (n=9)	61.00±1.17	P<0.0001
	Affected (n=54)	42.18±0.26	
E %	Control (n=9)	2.38±0.16	P>0.5219
	Affected (n=54)	2.50±0.10	
M %	Control (n=9)	0.22±0.14	P<0.0024
	Affected (n=54)	0.90±0.08	
B %	Control (n=9)	0.00±0.00	P<0.0001
	Affected (n=54)	0.37±0.06	
N/L	Control (n=9)	0.60±0.03	P<0.0001
	Affected (n=54)	1.28±0.01	

*p≤0.05 means significant.

Fig 1: (A) Muco-purulent nasal discharges, (B) Antero-ventral consolidation of lungs in PPR.

on skin (11.76%) with different degree of severity in affected small ruminants. Clinical signs as reported were consistent with Nath *et al.* (2014) and Manimaran *et al.* (2017). There was salivation with fibrinous exudates and erosive lesions on tongue, hard palate, gums, lips as well as yellowish necrotic crusts in oral commissures. Affected animals were showing marked depression with arched back due to increased pain in abdomen due to excessive intestinal peristaltic movement (Bari *et al.* 2018). Most of the acutely affected animals (n=168, 46.53%) died within one week. Early pyrexia subsides with onset of diarrhea after some days of infection characterized with sub normal temperature before death of affected animals owing to severe dehydration (Zakian *et al.* 2016). Multi system involvement of PPRV with varying degree of tropism to epithelial and lymphoid cells is responsible for producing symptoms consistent to pneumo-enteritis and severe immunosuppression (Singh *et al.* 2004).

Table 3: Mean ±SE of haematological parameters in sheep affected with PPR.

Parameters	Sheep	Mean±SE	P-value
Hb (gm/dl)	Control (n=5)	8.40±0.16	P<0.0001
	Affected (n=10)	10.50±0.06	
TEC (M/µl)	Control (n=5)	8.32±0.14	P<0.0001
	Affected (n=10)	12.48±0.24	
PCV (%)	Control (n=5)	21.20±0.38	P<0.0001
	Affected (n=10)	26.44±0.37	
TLC (10^3/µl)	Control (n=5)	23.32±1.76	P<0.0001
	Affected (n=10)	60.66±0.43	
MCV (fl)	Control (n=5)	16.84±0.21	P>0.1789
	Affected (n=10)	22.12±0.20	
MCH (pg)	Control (n=5)	10.84±0.09	P<0.0001
	Affected (n=10)	8.40±0.06	
MCHC (g/dl)	Control (n=5)	45.16±0.09	P<0.0001
	Affected (n=10)	38.18±0.25	
DLC			
N %	Control (n=5)	45.60±0.24	P<0.0001
	Affected (n=10)	55.50±0.42	
L %	Control (n=5)	54.20±0.37	P<0.0001
	Affected (n=10)	43.50±0.22	
E %	Control (n=5)	0.20±0.20	P<0.0001
	Affected (n=10)	0.80±0.29	
M %	Control (n=5)	0.00±0.00	P<0.0001
	Affected (n=10)	0.20±0.13	
B %	Control (n=5)	0.00±0.00	P<0.0001
	Affected (n=10)	0.00±0.00	
N/L	Control (n=5)	0.60±0.03	P<0.0001
	Affected (n=10)	1.28±0.01	

*p≤0.05 means significant.

Haemato-biochemical examinations

Statistical analysis of various hematological parameters conducted through student's *t-test* showed significant variations as depicted in Table 2 and 3. Severe diarrhoea resulting severe dehydration and haemoconcentration with polycythaemia consistent with increased Hb, TEC and PCV (Islam, 2015). There was leucocytosis mostly neutrophilia and relative lymphopenia attributed to secondary infection (Das *et al.* 2015) as well as stress due to increased cortisol level (Kataria *et al.* 2007). Alterations in serum biochemical parameters in both sheep and goat as analyzed through student *t-test* illustrated in Table 4 and 5 which is in accordance with Malik *et al.* 2018. Acute to chronic inflammation in response to viral replication and damage to microvasculature results in derangement of serum biochemical parameters (Aziz *et al.* 2019). Damage to renal tissue characterized by glomerular atrophy, degeneration and necrosis results in leakage of protein molecules leading to decreased total protein in affected animals (Begum *et al.* 2018). Significant increase in creatinine was related to extensive muscle damage with elevation of cortisol level (Kataria *et al.* 2007) while higher concentrations of urea attributed to increased breakdown of proteins and haemoconcentration due to severe diarrhea. Functional as well as structural damage to liver by the PPR virus was attributed to an elevated AST and ALT levels in affected small ruminants in the present study (Aziz *et al.* 2019). Hypoglycemic condition was observed in affected small ruminants might be due to functional damage of liver thereby impairing glycogenolysis (Aziz *et al.* 2019) as well as anoxia and in-appetence (Kataria *et al.* 2007). Mean ±SE of sodium

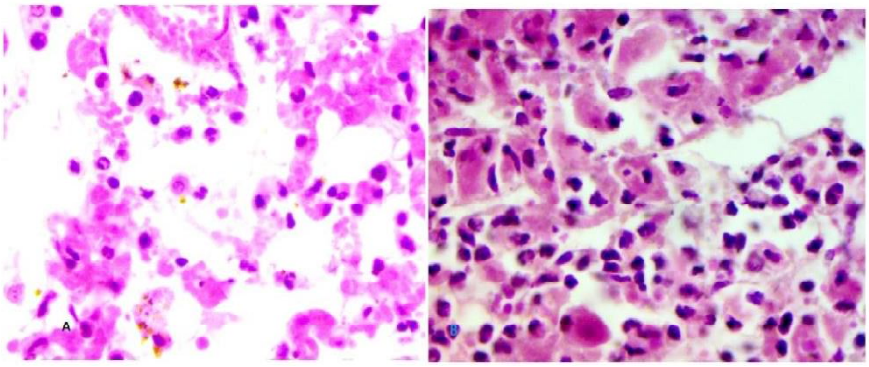

Fig 2: (A) Intranuclear eosinophilic inclusion body in alveolar macrophage of lungs (H&E-100X), (B) intracytoplasmic eosinophilic inclusion body in hepatocytes (H&E-100X).

and potassium concentration was found significantly higher in affected sheep and goat as a reflection of haemoconcentration (Islam *et al.* 2018) and nephropathy (Islam *et al.* 2018).

Gross and histopathology

Necropsy was conducted only in 43 (25.44%) animals comprising 32 goats and 11 numbers of sheep due to field limitations. Morbid changes as found in the present study were in consonance with Zakian *et al.* (2016). Similar necropsy lesions were reported earlier in concurrent infections of PPR and Contagious caprine pleuropneumonia (CCPP) in a goat flock byShanmugavadivu *et al.* (2021) confined mostly to digestive and respiratory system. Most of the carcasses were severely dehydrated and emaciated. Necropsy revealed soiling of hind quarters along with erosive and ulcerative lesions on oral cavity, plugging of the nose by dried exudates, matting of eyelids with encrustations. Yellowish white false membrane, ulcerations on tongue and hard palate were observed in most of the carcasses. There were frothy exudates in trachea along with antero-ventral consolidation (Fig 1B) and hepatization of lungs showing red to grey areas with firm consistency suggestive of fibrinopurulent bronchopneumonia. Hyperemia and haemorrhages in the gut mucosa with congestion consistently seen near the ileo-cecal valve, caeco-colic junction and rectum. Erosive and ulcerative vulva-vaginitis was evident in two cases during necropsy.

Table 4: Mean± SE of serum biochemical parameters in goats affected with PPR.

Parameters	Goats	Mean±SE	P-value
Total protein (g/dl)	Control (n=9)	7.36±0.15	P<0.0001
	Affected (n=54)	4.72±0.12	
Albumin (g/dl)	Control (n=9)	4.56±0.10	P<0.0001
	Affected (n=54)	2.83±0.07	
Globulin (g/dl)	Control (n=9)	2.80±0.08	P<0.0001
	Affected (n=54)	1.89±0.05	
A: G	Control (n=9)	1.63±0.04	P<0.0001
	Affected (n=54)	1.52±0.02	
Creatinine (mg/dl)	Control (n=9)	1.06±0.05	P< 0.0001
	Affected (n=54)	3.16±0.07	
Urea (mg/dl)	Control (n=9)	30.12±0.17	P<0.0001
	Affected (n=54)	39.87±0.77	
BUN (mg/dl)	Control (n=9)	13.50±0.14	P<0.0001
	Affected (n=54)	15.62±0.07	
Glucose (mg/ml)	Control (n=9)	53.52±0.32	P<0.0001
	Affected (n=54)	42.65±0.18	
ALT (IU/L)	Control (n=9)	30.44±0.12	P<0.0001
	Affected (n=54)	47.87±0.12	
AST (IU/L)	Control (n=9)	181.00±0.81	P<0.0001
	Affected (n=54)	236.4±0.52	
K (mmol/L)	Control (n=9)	5.29±0.07	P<0.0001
	Affected (n=54)	5.88±0.03	
Na (mmol/L)	Control (n=9)	134.3±0.26	P<0.0001
	Affected (n=54)	142.5±0.17	
	Affected (n=54)		

*p≤0.05 means significant.

Microscopically, tongue lesions consisted of necrosis of squamous epithelial cells, sloughing of lingual papillae with ulcerations and infiltrations of mononuclear cells with syncytia. Histopathological examination of lungs revealed presence of sero-fibrinous exudates in alveoli and alveolar necrosis with infiltration of mononuclear cells and congestion. Other characteristic microscopic findings were inter-alveolar haemorrhage, congestion of inter alveolar septal area, intranuclear eosinophilic inclusions (Fig 2A) in alveolar macrophage of lungs and presence of syncytia in affected pulmonary parenchyma. Microscopic lesions as evident in different organs were consistent with gross changes in different organs found during necropsy of affected animals. Presence of intracytoplasmic inclusions (Fig 2B) in hepatocytes, hepatic necrosis and infiltration of mononuclear cells and neutrophils with syncytia formations were observed through liver microscopy. Villous atrophy, congestion, desquamation of muscle layer of intestine was major microscopic changes in intestine. There was infiltration

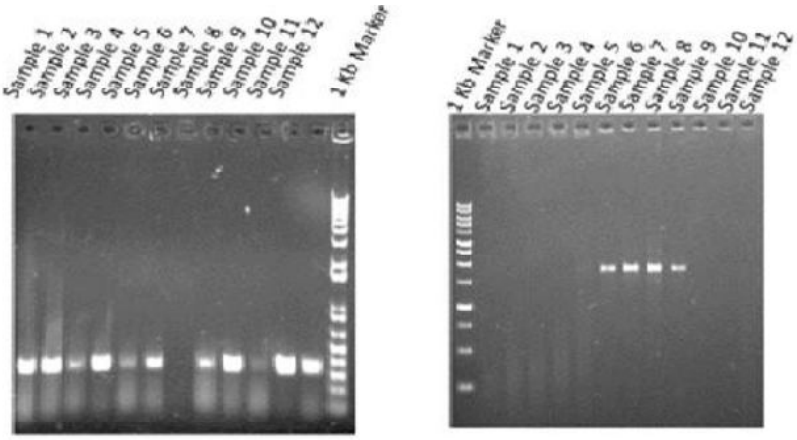

Fig 3: Agarose gel electrophoresis of some positive samples amplified using primers specific to partial N gene sequences (352 bp) left and for full length N gene (1764 bp) right.

Table 5: Mean± SE of serum biochemical parameters in sheep affected with PPR.

Parameters	Goats	Mean±SE	P-Value
Total protein (g/dl)	Control (n=5)	7.14±0.02	P<0.0001
	Affected (n=10)	5.66±0.04	
Albumin (g/dl)	Control (n=5)	3.94±0.05	P<0.0001
	Affected (n=10)	3.52±0.04	
Globulin (g/dl)	Control (n=5)	3.20±0.04	P<0.0001
	Affected (n=10)	2.14±0.04	
A:G	Control (n=5)	1.23±0.03	P<0.0001
	Affected (n=10)	1.65±0.04	
Creatinine (mg/dl)	Control (n=5)	1.06±0.06	P<0.0001
	Affected (n=10)	2.39±0.02	
Urea (mg/dl)	Control (n=5)	31.01±0.42	P<0.0001
	Affected (n=10)	43.65±0.07	
BUN (mg/dl)	Control (n=5)	14.08±0.12	P<0.0001
	Affected (n=10)	18.69±0.05	
Glucose (mg/ml)	Control (n=5)	52.64±0.11	P<0.0001
	Affected (n=10)	37.28±0.23	
ALT (IU/L)	Control (n=5)	29.08±0.22	P<0.0001
	Affected (n=10)	52.00±0.17	
AST (IU/L)	Control (n=5)	171.8±0.37	P<0.0001
	Affected (n=10)	242.6±0.16	
K^+ (mmol/L)	Control (n=5)	5.64±0.07	P<0.0001
	Affected (n=10)	6.36±0.04	
Na^+ (mmol/L)	Control (n=5)	132.0±0.06	P<0.0001
	Affected (n=10)	140.8±0.35	

*p≤0.05 means significant.

of inflammatory cells in the sub-mucosa of villi as well as muscle layer of intestine along with depletion of splenic pulp and congestion. Microscopic changes observed were in agreement with Jagtap *et al.* (2012) and Manimaran *et al.* (2017).

Molecular confirmation by PCR

Clinical samples (n=70, oral swabs-42 and nasal swabs-28) were analyzed for PCR based detection of viral nucleic acid by using primers (Table 1) targeting partial (352bp) as well as full (1764bp) nucleocapsid (N) gene for amplification. All the samples were screened PCR positive (Fig 3) for PPRV. Molecular based confirmation for PPR virus is the most reliable and extensively used technique for diagnosis due to its high sensitivity and specificity (Kumar *et al.* 2014; Pandey *et al.* 2020).

CONCLUSION

In conclusion, clinical, haemato-biochemical alterations along with gross and histopathological lesions may be used in field condition to arrive at a fast-reliable diagnosis in PPR with confirmation by molecular based technique like PCR so as to assess the disease severity and further planning the treatment regimen. Pre-existing parasitic infestation associated with poor management might play as an important contributing factor in precipitating the disease. Though there are sporadic outbreaks occurring round the year, but this is a detail investigative study in the region. Awareness among the farmers and all the stakeholders needs to be addressed by refreshing the knowledge on PPR epidemiology, pathology and pathogenesis to limit the disease spread so as to achieve the control over the disease and ensure livelihood security.

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