Slov Vet Res 2007; 44 (4): 123-32
UDC 636.3.09:616.98-07:616-036.22:551.585.53

Case Report

LARGE SCALE OUTBREAKS OF PESTE DES PETITS RUMINANTS IN SHEEP AND GOATS IN THAR DESERT OF INDIA

Anil Kumar Kataria[1]*, Nalini Kataria[2], Ajey Kumar Gahlot[3]

[1]Apex Centre for Animal Disease Investigation, Monitoring and Surveillance, [2]Department of Veterinary Physiology, Radioisotope Laboratory, [3]Department of Clinical Veterinary Medicine, College of Veterinary and Animal Science, Rajasthan Agriculture University, Bikaner-334001, Rajasthan, India

*Corresponding author, E-mail: akkataria1@rediffmail.com

Summary: Severe peste des petits ruminants (PPR) outbreaks were recorded in Thar desert of Rajasthan state (India) affecting large population of sheep and goats over a huge geographical area, taking a toll in tens of thousands of animals. These outbreaks were investigated for epidemiological, clinical, pathological, haematological and biochemical features. The outbreaks started in migratory sheep which returned back from adjacent PPR endemic areas and later spread to goats with estimated case fatality rates of 70% and 80%, respectively. Younger animals were more affected than the adult ones. The stress of migration coupled with low environmental temperatures bolstered by humidity and nutritional deficiency was the reason of precipitation of the disease. Typical clinical and pathological findings were recorded and significant ($P \leq 0.05$) changes were observed in most of the haemato-biochemical parameters. The free-range grazing system had been responsible for the involvements of larger number of flocks in distant areas with prevalence for longer duration. The disease waned with increase in the environmental temperatures.

The present situation warrants strict sero-surveillance and monitoring of PPR together with uninterrupted vaccination of migratory flocks at district or state borders for its effective control.

Keywords: sheep diseases – diagnosis – virology; goat diseases – diagnosis – virology; disease outbreaks; peste-des-petits-ruminants – diagnosis – virology; pathology, clinical; blood chemical analysis: antibodies, viral – blood; enzyme-linked immunosorbent assay – methods; desert climate; sheep; goats

Introduction

Peste des petits ruminants (PPR), an *office international des epizooties* list A disease of sheep and goats caused by morbillivirus is characterized by high morbidity and high mortality rates resulting into heavy economical losses. The disease has been regularly reported from various parts of the world but mainly from Africa, Arabia, the Middle and Near East and the Indian subcontinent (1, 2, 3, 4).

Rajasthan state situated in the north-west part of India is the biggest state in regards to its geographical area and shares a very long international border with Pakistan. The inland borders are shared with other states: Punjab in the north, Haryana and Uttar Pradesh in the north-east, Madhya Pradesh in the south-east and Gujarat in the south-west. The arid region of western part of Rajasthan constitutes famous Thar desert, where ambient temperatures vary widely from subzero in winter to as high as 50 °C in summer in some places. In this area, the livestock remains the mainstay of desert economy over agricultural produce. The sheep are reared mainly for wool and meat, and goats for milk and meat. The migration of small ruminants, especially of sheep, to other adjoining states, is a regular feature during famine and during times of scarcity of feed and fodder in Thar desert. The migration of various sized flocks (usually of few hundreds to several thousands) normally starts in March which is the time of onset of summer in this region. The flocks return back by October, the time of onset of winter, but early return of animals is possible if rain is experienced in the area. The arrival of animals is often accompanied and/or followed by various dis-

Received: 1 October 2007
Accepted for publication: 19 November 2007

ease outbreaks in the unprotected flocks.

Though PPR is considered endemic throughout India (5) this region had never experienced the outbreaks in the past similar to that in the present report involving a wider geographical area with very high morbidity and mortality.

This paper puts on record some field investigations in regards to epidemiology, clinical signs, pathological findings, and laboratory investigations encompassing haemato-biochemical changes and histopathology in sheep and goats affected with PPR during natural outbreaks in the Thar desert.

Material and methods

A series of PPR outbreaks were observed in the northwest Rajasthan affecting both sheep and goat populations between November 2005 and April 2006. Sheep were mostly Magra, Marwari and Rambouillet breeds and their crosses with local breeds in the organized farms and goats belonged to Marwari breed. The epidemiological observations were recorded in several outbreaks in 21 villages and one organized sheep breeding farm. The clinical signs were observed and post-mortem examination of 58 carcasses was carried out at various places.

The blood samples were drawn from 30 sheep (15 adults and 15 lambs) and 30 goats (15 adults and 15 kids) affected with PPR and 20 healthy sheep (10 adults and 10 lambs) and 20 healthy goats (10 adults and 10 kids) for haemato-biochemical analyses. The most of the adult animals were females but young ones included both male and female. Various haemato-biochemical parameters were determined by standard techniques (6, 7, 8) and serum cortisol by radioimmunometric method using ^{125}I radioimmunoassay kit (RADIM, Spain). The statistical significance for a given parameter was determined between healthy and PPR affected animals. Comparison between healthy and infected herds was made by student's t-test with significant difference considered at $p < 0.05$ (9).

Results

Field Investigation

Epidemiology

The disease first appeared in November 2005 in migratory sheep flocks in Sardarshahar area of Churu district adjoining to Haryana state and later spread to other adjoining districts. The widespread outbreaks were encountered in different flocks untll April 2006. The highest morbidity and mortality rates were recorded during the periods when the lowest night temperatures dropped below zero in January 2006 with high humidity. Because of involvement of very large animal population in huge geographical area in the desert, the exact figures could not be gathered but the available data revealed death of few thousand animals with a case fatality of 70% in sheep and 80% in goats. The young animals of both species had higher case fatality rates than their respective adults. The disease was also recorded at an organized sheep farm where mortality was observed mostly in young animals below 3 months of age which were not vaccinated against PPR, however, vaccinated adult animals also died albeit with less severe clinical and pathological findings.

Clinical signs

The course of the disease was acute and subacute, few of the animals died even in 36 hours of onset of the disease. The affected animals initially were severely depressed with a sudden rise in body temperature reaching almost 42 °C in some cases, and the fever persisted for 7-8 days. From the onset of fever, most animals had a serous nasal discharge which progressively turned into mucopurulent discharge, leading to severe respiratory distress. Areas of erosions were most commonly seen on the visible nasal mucous membranes and muco-cutaneous junctions with inflammation around the mouth (Fig.1). In many of the animals, lesions similar to orf developed at mucocutaneous junction of mouth. The erosive and necrotic stomatitis started as areas of hyperemia at gums, cheeks, dental pad and / or anterior dorsal part of tongue with frothy salivation. The areas later developed into irregular non-haemorrhagic lesions (Fig.2) and in some of the cases circular raised but flat non-bleeding lesions were present on the tongue (Fig.3). There was a great amount of necrotic debris on the older lesions (Fig.4). The individuals with severe oral lesions had visible swelling around mouth. A non-haemorrhagic diarrhoea was observed in all affected animals, developing 2-3 days after onset of the disease. Conjunctivitis was recorded with lachrymal discharge which became mucoid resulting in sticky eyelids. Abortion in pregnant animals was a consistent feature and vulvar mucous membranes had erosive lesions very similar to that in intestinal mucosa.. A subnormal temperature preceded death in animals with severe diarrhoea for few days.

Figure 1: Erosions at muco-cutaneous junction with inflammation around the mouth (PPR, kid)

Figure 2: Irregular non-haemorrhagic oral lesions (PPR, sheep)

Figure 3: Raised and flat circular lesions on tongue (PPR, goat)

Figure 4: Necrotic debris on oral lesions (PPR, sheep)

Pathological findings

Grossly, the carcasses were dehydrated with apparent swelling around the mouth. The lesions in mouth were consistently present in all animals except those who died within short period of 1-2 days. The lesions of respiratory tract included necrotic areas on the mucosa of nostrils and turbinates, severely congested tracheal mucous membrane and white froth in trachea. Lungs were congested and consolidated especially involving antero-ventral parts. Hydrothorax was recorded in few cases.

The oral cavity was full of white necrotic debris where oral lesions were severe. The lesions in gastrointestinal tract consisted of few erosions in the mucosa from where blood oozed out, large intestines were congested, especially at the caeco-colic junction with streaks of blood on mucosal crests (Zebra-stripes) (Fig.5) though the zebra-stripes were not seen in all the carcasses.

The lymph nodes, especially from mesenteries were severely oedematous, congested and enlarged (Fig.6). In some cases haemorrhages on the internal walls of gall bladder were recorded (Fig.7). Spleen was slightly enlarged in few cases. In some animals liver was studded with necrotic foci. Vaginitis was observed in many animals and vulvar mucus membranes were inflamed and had erosive lesions.

Though the types of lesions were more or less similar in all the animals, there was a variation in the severity and involvement of the organs. The vaccinated animals had less severe lesions on tongue.

Laboratory Investigations

Haemato-biochemical parameters

The mean values of haemato-biochemical parameters in healthy animals and PPR affected sheep and

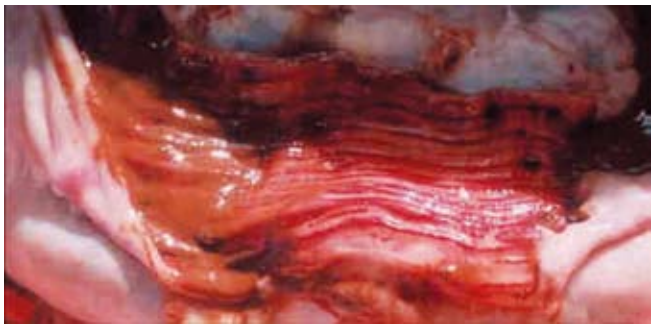

Figure 5: Zebra-stripes in large intestine (PPR, sheep)

Figure 6: Oedematous, congested mesenteric lymph node (PPR, goat)

Figure 7: Haemorrhages on the internal wall of gall bladder (PPR, sheep)

goats are presented in table 1 through 4. All parameters included in the investigation significantly differed between healthy and infected animals in both species in both young and adult animals ($p \leq 0.05$): total erythrocyte count (TEC), total leucocyte count (TLC), haemoglobin (Hb), packed cell volume (PCV), differential leucocyte count (DLC), relative viscosity, specific gravity and erythrocytic sedimentation rate (ESR).

In PPR affected animals of both species and age groups significant ($p \leq 0.05$) changes were recorded in serum biochemical parameters for sodium, potassium, glucose, total serum proteins, albumin, globulin, creatinine, urea and cortisol.

Histopathological changes

Histopathologically in lungs syncytial cells were present in alveolar lamina and inclusion bodies in cytoplasm and nucleus of alveolar macrophages and epithelial cells of bronchioli and bronchi. There was severe congestion of mucosa and submucosa and degeneration and necrosis of intestinal epithelium.

Serology

The sera collected from representative animals in different areas who survived the disease were subjected to c-ELISA which confirmed the presence of specific antibodies.

Discussion

Field Investigations

Epidemiology

The disease first appeared in the sheep flocks which returned back after migration to other states during periods of famine in this desert area. The arrival of flocks was coincided with sudden lowered ambient temperatures. It is expected that the sheep on migration to other states came in contact with native small ruminant population where endemicity of the disease is much higher. The stress of migration and environmental temperature might have precipitated the onset of disease (10). The animals during feed scarcity often become nutritionally deficient resulting into increased susceptibility to infection (5). The spread of disease to other adjoining districts was due to unrestricted movements of the animals as they are reared on free range system of feeding and management covering long distances during the day (10, 11). The serial occurrence of dis-

Table 1: Haematological values (Mean ± SEM) in healthy and PPR affected sheep

S. No.	Parameter	Sheep			
		Adults		Lambs	
		Healthy (n = 10)	PPR affected (n = 15)	Healthy (n = 10)	PPR affected (n = 15)
1.	TEC (x10^{12}/ l)	7.92±0.23	8.99±0.31*	8.91±0.41	10.0±0.39*
2.	TLC (x10^{9}/ l)	9.98±0.31	6.0±0.37*	11.11±0.22	7.11±0.41*
3.	Hb (g/dl)	10.41±0.61	13.11±0.31*	13.34±0.51	15.12±0.31*
4.	PCV (%)	29.33±0.61	34.12±0.39*	34.12±0.58	37.22±0.34*
5.	DLC (%)				
	Neutrophil	35.3±0.30	61.8±0.30*	36.3±0.22	60.9±0.25*
	Lymphocyte	55.2±0.21	35.2±0.21*	54.8±0.31	35.8±0.20*
	Monocyte	5.3±0.05	2.3±0.01*	5.4±0.01	1.9±0.01*
	Eosinophil	3.1±0.03	1.1±0.01*	3.0±0.01	1.1±0.01*
	Basophil	0.6±0.02	0.2±0.001*	0.5±0.001	0.15±0.001*
6.	Relative Viscosity	4.3±0.03	4.9±0.04*	4.6±0.05	5.0±0.03*
7.	Specific gravity	1.050±0.003	1.059±0.001*	1.055±0.002	1.060±0.001*
8.	ESR (mm/4Hr.)	1.93±0.04	0.20±0.04*	1.48±0.02	0.30±0.03*

1. Figures in parenthesis indicate number of animals.
2. In adults and lambs mean comparison has been made between respective healthy and PPR affected animals for each parameter. All the differences were significant ($p \leq 0.05$) as indicated by asterix.
3. PPR = *Peste des petits ruminants*
4. TEC = Total erythrocyte count
5. TLC = Total leucocyte count
6. Hb = Haemoglobin
7. PCV= Packed cell volume
8. DLC= Differential leucocyte count
9. ESR = Erythrocytic sedimentation rate

Table 2: Biochemical parameters (Mean ± SEM) in healthy and PPR affected sheep

S. No.	Parameter	Sheep			
		Adult		Lambs	
		Healthy (n = 10)	PPR affected (n = 15)	Healthy (n = 10)	PPR affected (n = 15)
1.	Sodium (mmol/L)	130.11±1.89	140.12±2.00*	133.2±1.34	144.2±3.0*
2.	Potassium(mmol/L)	5.3±0.1	8.0±0.10*	5.5±0.10	9.12±0.09*
3.	Calcium(mmol/L)	2.40±0.025	2.25±0.022^{NS}	2.45±0.022	2.32±0.017^{NS}
4.	Phosphorus(g/L)	0.043±0.0034	0.040±0.0007^{NS}	0.045±0.0007	0.043±0.0003^{NS}
5.	Glucose (mmol/L)	2.48±0.072	1.65±0.055*	2.56±0.055	1.54±0.049*
6.	TSP (g/L)	71.2±1.0	61.0±3.1*	78.0±1.1	63.0± 1.1*
7.	Albumin(g/L)	39.4±1.2	23.1±0.9*	41.2±0.9	23.8±0.9*
8.	Globulin(g/L)	36.0±0.9	36.9±0.8*	36.8±0.9	40.1±0.7*
9.	Creatinine(µmol/l)	97.24±3.53	185.64±2.65*	79.56±2.65	185.64±1.76*
10.	Urea(mmol/L)	3.83±0.018	4.99±0.021*	3.33±0.021	4.69±0.019*
11.	Cortisol(mmol/L)	0.165±0.011	0.259±0.019*	0.173±0.008	0.312±0.019*

1. Figures in parenthesis indicate number of animals.
2. In adults and lambs mean comparison has been made between respective healthy and PPR affected animals for each parameter. All the parameters except calcium and phosphorus showed significant differences ($p \leq 0.05$) as indicated by asterix.
3. NS= Non significant difference ($p > 0.05$)
4. PPR = *Peste des petits ruminants*
5. TSP = Total serum proteins
6. NS = Non-significant

Table 3: Haematological values (Mean ± SEM) in healthy and PPR affected goats

S. No.	Parameter	Goats			
		Adults		Kids	
		Healthy (n = 10)	PPR affected (n = 15)	Healthy (n = 10)	PPR affected (n = 15)
1.	TEC ($x10^{12}$/ l)	8.22±0.41	0.11±0.51*	9.00±0.38	11.31±0.39*
2.	TLC ($x10^{9}$/ l)	9.31±0.81	5.93±0.38*	10.99±0.31	6.43±0.22*
3.	Hb (g/dl)	10.12±0.31	13.12±0.41*	11.88±0.21	13.88±0.34*
4.	PCV (%)	28.22±1.0	33.12±0.91*	32.34±1.01	37.12±0.93*
5.	DLC				
	Neutrophil	36.1±0.12	62.4±0.21*	37.0±0.22	66.3±0.31*
	Lymphocyte	55.3±0.16	34.2±0.20*	54.0±0.15	32.8±0.41*
	Monocyte	5.1±0.03	2.1±0.02*	5.0±0.01	2.10±0.01*
	Eosinophil	3.0±0.04	1.0±0.01*	3.0±0.01	1.12±0.01*
	Basophil	0.5±0.001	0.1±0.001*	0.4±0.001	0.15±0.001*
6.	Relative Viscosity	4.0±0.04	4.8±0.03*	4.25±0.06	4.9±0.02*
7.	Specific gravity	1.050±0.001	1.055±0.001*	1.054±0.001	1.059±0.001*
8.	ESR (mm/4Hr.)	1.4±0.03	0.27±0.03*	1.53±0.04	0.30±0.04*

1. Figures in parenthesis indicate number of animals.
2. In adults and lambs mean comparison has been made between respective healthy and PPR affected animals for each parameter. All the differences were significant ($p \leq 0.05$) as indicated by asterix.
3. PPR = *Peste des petits ruminants*
4. TEC = Total erythrocyte count
5. TLC = Total leucocyte count
6. Hb = Haemoglobin
7. PCV= Packed cell volume
8. DLC= Differential leucocyte count
9. ESR = Erythrocytic sedimentation rate

Table 4: Biochemical parameters (Mean ± SEM) in healthy and PPR affected goats

S. No.	Parameter	Goats			
		Adults		Kids	
		Healthy (n = 10)	PPR affected (n = 15)	Healthy (n = 10)	PPR affected (n = 15)
1.	Sodium (mmol/L)	131.12±1.31	141.1±0.71*	133.0±1.7	145.2±0.61*
2.	Potassium(mmol/ L)	5.6±0.09	7.9±0.05*	5.9±0.09	8.1±0.13*
3.	Calcium(mmol/ L)	2.46±0.032	2.42±0.005^{NS}	2.475±0.05	2.42±0.05^{NS}
4.	Phosphorus(g/L)	0.0583±0.0004	0.056±0.0009 ^{NS}	0.057±0.0009	0.055±0.0009^{NS}
5.	Glucose (mmol/L)	2.45±0.044	2.101±0.038*	2.59±0.038	2.15±0.028*
6.	TSP (g/L)	70.7±0.9	60.0±1.0*	77.1±1.0	61.0± 0.2*
7.	Albumin(g/L)	36.2±0.1	21.0±0.3*	36.3±0.3	22.0± 0.3*
8.	Globulin(g/L)	34.1±0.1	39.0±0.6*	34.3±0.2	38.0±0.8*
9.	Creatinine(µmol/l)	97.24±7.70	167.96±3.53*	114.92±2.65	238.68±7.95*
10.	Urea(mmol/L)	2.49±0.049	3.83±0.019*	2.05±0.066	3.33±0.0166*
11.	Cortisol(mmol/L)	0.168±0.009	0.284±0.022*	0.165±0.008	0.259±0.008*

1. Figures in parenthesis indicate number of animals.
2. In adults and lambs mean comparison has been made between respective healthy and PPR affected animals for each parameter. All the parameters except calcium and phosphorus showed significant difference ($p \leq 0.05$) as indicated by asterix.
3. NS= Non significant difference ($p > 0.05$)
4. PPR = *Peste des petits ruminants*
5. TSP = Total serum proteins
6. NS = Non-significant

ease outbreaks had also been recorded for a long duration in different flocks in various adjoining districts in west Bengal (10). The highest morbidity and mortality rates were recorded during the periods when the lowest night temperatures dipped below zero in January 2006 with high humidity. In the present outbreaks humidity might have played the role in disease occurrence as it may be one of the factors allowing thriving and multiplication of PPR virus in nature (10). The environmental stress particularly hot and humid is also held responsible for precipitation of the disease (12).

Because of involvement of very large animal population in huge geographical area in desert the all figures could not be gathered regarding morbidity and mortality nevertheless the available data suggested a case fatality of 70% in sheep and 80% in goats. The PPR antibody prevalence is low (10- 30%) in goat population in the northern parts of the country suggesting higher risk of infection in goats (5). Moreover, the recovery rate of goats infected with PPR virus is less in comparison to that in sheep (13). In the Thar desert sheep and goats are reared in unorganized sector by small and marginal farmers and these animals are used as cash crops without any inputs. Neither these animals are supplemented with feed additives nor usually vaccinated against the diseases in which cost is incurred. This area never experienced PPR before hence all of the animals in these outbreaks were also not protected against it. The non-vaccination of these animals explains the very high morbidity and mortality rates. In the present serial outbreaks both species of small ruminants were affected whereas in some earlier reported outbreaks only goats died and sheep were not affected at all (10, 14). In contrast to the present observation a significantly higher mortality rate was recorded in sheep than in goats in Cameroon (15).

The disease was also recorded at an organized sheep farm where mortality was observed mostly in young animals below 3 months of age which were not vaccinated against PPR. It has been suggested that new born animals become susceptible to PPR virus infection at three to four months of age (16). However, few vaccinated adult animals also died albeit with less severe clinical and pathological findings.

Clinical signs

Though the course of the disease was acute and subacute with appearance of clinical signs and development of typical lesions, in cases where animals died within 1-2 days of onset of disease no lesions were recorded. Most of the affected animals had very high body temperatures for long periods. The circular raised, but flat lesions on dorsum of tongue were seen only in few cases. The development of lesions on muco-cutaneous junction of mouth very similar to that in orf or contagious ecthyma needs to be differentiated. The presence of mucopurulent discharge and froth in trachea led to respiratory occlusion and development of a typical obstructive sound during breathing. The amount of necrotic debris in oral cavity depended on the extent and age of the lesions. Unlike in other outbreaks typical overt oral lesions were recorded in the present outbreaks in sheep as well as in goats (17).

A continuous profuge diarrhoea caused severe dehydration in the affected animals. The kids were more severely affected due to dehydration and showed prostration for longer duration as compared to lambs. These animals had more subnormal temperatures before death.

Pathological findings

The mouth lesions were present in all of the animals except those who died within 1-2 days of appearance of disease. Even the vaccinated animals at the organized sheep breeding farm had lesions on tongue. Lungs in all the animals showed congestion and consolidation but hydrothorax was recorded in few cases only. No animal had any kind of lesion in abomasums (17) and heavy haemorrhages in large intestines (18) were not observed in any of the animals.

The haemorrhages recorded on the luminal wall of gall bladder in the present outbreak in some cases has not been reported to the best of our knowledge but presence of thick granular bile had been reported (14). The necrotic foci in liver of some animals could have developed due to some secondary bacterial infection of haematogenous route.

Though the types of lesions were more or less similar in all the animals but there was variation in the severity and involvement of the organs.

Treatment

The affected animals were given antibiotics to control secondary bacterial infections along with anti-inflammatory drugs. Many animals which received proper treatment in early stages of the disease were saved. It was observed that higher fatality rates in PPR affected animals was more due to secondary bacterial infections than the disease itself

which could have been due to immune suppression associated with morbillivirus infections (19).

Laboratory Investigations

Haemato-biochemical parameters

The mean values of haemato-biochemical parameters in healthy animals corroborated the earlier findings in sheep and goats of arid tract (20, 21, 22). The severe dehydration in the affected animals was evidenced by increased viscosity and specific gravity which led to polycythaemia (23). Severe leucopoenia could have been due to the inhibition of peripheral blood lymphocytes proliferation by PPR virus (19). A marked lymphocytopoenia, monocytopoenia, neutrophilia and eosinopoenia in present investigation could have been due to the combined effect of virus infection and stress as evidenced by elevated cortisol levels (24).

The increased mean values of sodium and potassium reflected haemoconcentration. The total serum protein values decreased but globulin concentration increased indicating immune response towards infection. The higher globulin concentration was achieved at the expense of compensatory fall in albumin levels.

Serum cortisol was recorded higher in PPR affected stock indicating stress. Cortisol causes increase in blood glucose levels due to glycogenolytic property but in present investigation, decreased glucose levels could have been due to animals not being fed since the onset of infection. The higher levels of cortisol causing muscle wasting resulted in increased serum creatinine levels and an increased urea concentration reflected protein breakdown and haemoconcentration simultaneously.

Acknowledgements

The authors are thankful to Joint Director, CADRAD, Indian Veterinary Research Institute, Bareilly (UP) for serological confirmation of the disease.

References

1. Obi TU, Durojaiye OA, Kasali OB, Akpavie S, Opasina, DB. Peste des petits ruminants (PPR) in goats in Nigeria: clinical, microbiological and pathological features. Zentralbl Vet Med B 1983; 30: 751-61.

2. Al-Naeem A, Elzein EMA, Al-Afaleq AI Epizootiological aspects of peste des petits ruminants and rinderpest in sheep and goats in Saudi Arabia. Rev Sci Tech Off Int Epizoot 2000; 19: 855-8.

3. Taylor WP, Al-Busaidy S, Barrett T. The epidemiology of peste des petits ruminants in the Sultanate of Oman. Vet Microbiol 1990; 22: 341-52.

4. Shaila MS, Purushothaman V, Bhavsari D, Venugopal K, Venketesan RA. PPR of sheep in India. Vet Rec 1989; 125: 602.

5. Singh RP, Saravanan P, Sreenivasa BP, Singh RK, Bandyopadhyay SK. Prevalence and distribution of peste des petits ruminants virus infection in small ruminants in India. Rev Sci Tech Off Int Epizoot 2004; 23: 807-19.

6. Oser BL. Hawk's physiological chemistry. 14th ed. New Delhi: Tate Graw Hill publishing, 1976.

7. Ranade VG, Joshi PN, Pradhan S. A text book of practical physiology. 3rd ed. Pune: Pune Vidyarthi Griha Prakashan, 1982: 299-342.

8. Schalm OW, Jain NC, Carroll EJ. Veterinary haematology. 3rd ed. Philadelphia: Lea and Febiger, 1975: 15- 81.

9. Snedecor GW, Cochran WG. Statstical methods. 6th ed. New Delhi: Oxford and IBH Publishing, 1967: 45-83.

10. Mondal AK, Chottopadhyay AP, Sarkar SD, Saha GR, Bhowmik K. Report on epizootiological and clinicopathological observations on peste des petits ruminants (PPR) of goats in west Bengal. Indian J Anim Health 1995; 34:145-8.

11. Kumar GS, Rathore BS Mehrotra ML. Epidemiological observations on Peste des petitis ruminants in north India. Indian J Anim Sci 1999; 69: 365-8.

12. Wosu LO. Seasonality of occurrence of peste des petits ruminats disease in Nigeria: a preliminary report. Mysore J Agric Sci 1995; 29: 158-62.

13. Shankar H, Gupta VK, Singh N. Occurrence of peste des petits ruminants like disease in small ruminants in Uttar Pradesh. Indian J Anim Sci 1998; 68: 38-40.

14. Shaila MS, Peter AB, Varalakshmi P, Apte M, Rajendran MP, Anbumani SP. Peste des petits ruminants in Tamilnadu goats. Indian Vet J 1996; 73: 587-8.

15. Ndamukong KJ, Sewell MM, Asanji MF. Disease and mortality in small ruminants in the North West Province of Cameroon. Trop Anim Health Prod 1988; 21: 191-6.

16. Srinivas RP, Gopal T. Peste des petits ruminants (PPR): a new menace to sheep and goats. Livestock Advisor 1996; 21: 22-6.

17. Kumar A, Singh SV, Rana R, Vaid RK, Misri J, Vihan VS. PPR outbreak in goats: epidemiological and therapeutic studies. Indian J Anim Sci 2001; 71: 815-8.

18. Singh VP, Chum VK, Mondhe KS. Peste des petits ruminants: an outbreak in sheep in Rajasthan. Indian Vet J 1996; 73: 466-7.

19. Heaney J, Barrett T, Cosby SL. Inhibition of in vitro leukocyte proliferation by morbilliviruses. J Virol 2002; 76: 3579-84.

20. Kataria AK, Kataria N, Gahlot AK. Observations on some blood analytes in Marwari sheep. Vet Pract 2005; 6: 39-40.

21. Kataria AK, Kataria N, Ghosal AK. Haematological changes associated with season in Marwari goats. Indian Vet J 1992; 69: 552-4.

22. Kataria AK, Kataria N, Bhatia JS, Ghosal AK. Blood metabolic profile of Marwari goats in relation to seasons. Indian Vet J 1993; 70: 761-2.

23. Bhikane AU, Kulkarni DD, Narladkar BW, Ali MS. Clinico-pathological and therapeutic investigations on peste des petits ruminants in goats. Indian Vet J 1997; 74: 377-9.

24. Kataria N, Kataria AK. Use of blood analytes in assessment of stress due to drought in camel. J Camel Pract Res 2004; 11: 129-33.

OBSEŽNI IZBRUHI KUGE MALIH PREŽVEKOVALCEV PRI OVCAH IN KOZAH V PUŠČAVI THAR V INDIJI

A. K. Kataria, N. Kataria, A. K. Gahlot

Povzetek: V puščavi Thar v Radžastanu (Indija) smo naleteli na resne izbruhe kuge malih prežvekovalcev (PPR). Bolezen je prizadela populacijo ovac in koz, ki živi na obsežnem geografskem področju, in je pokončala več deset tisoč živali. Izbruhe bolezni smo analizirali iz epidemiološkega, kliničnega, patološkega, hematološkega in biokemičnega vidika. Izbruhi so se najprej pojavili v nomadskih čredah ovac, ki so se vračale s sosednjih področij z endemično prisotno kugo malih prežvekovalcev, kasneje pa se je razširila še na koze. Ocenjena smrtnost pri ovcah je 70-odstotna in pri kozah 80-odstotna. Mlajše živali so bile bolj prizadete kot odrasle. Naglo širjenje bolezni je pogojeval stres zaradi migracije v kombinaciji z nizkimi temperaturami in visoko vlago v okolju ter pomanjkanjem hrane. Ugotovili smo tipične klinične in patološke najdbe, pri večini hematoloških in biokemičnih parametrov pa smo ugotovili statistično značilne spremembe ($P \leq 0.05$). Zaradi sistema proste paše je bilo prizadetih veliko čred tudi na oddaljenih predelih, bolezen pa se je tam bistveno dlje zadrževala. Število obolenj se je zmanjševalo v odvisnosti z naraščanjem temperature v okolju.
Trenutna situacija v zvezni državi Radžastan zahteva strog nadzor bolezni PPR in serološko pregledovanje v kombinaciji z neprekinjenim cepljenjem nomadskih čred na ogroženih področjih in državnih mejah.

Ključne besede: ovce, bolezni – diagnostika – virologija; koze, bolezni – diagnostika – virologija; epidemije; peste-des-petits-ruminants – diagnostika – virologija; klinična patologija; kri, kemične analize; protitelesa, virusna – kri; encimsko vezan imunosorbentni test – metode; puščave; ovce; koze