

## AN OUTBREAK OF PESTE DES PETITS RUMINANTS IN GOATS IN REAWALPINDI

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### ABSTRACT

An outbreak of peste des petits ruminants (PPR) in goats was recorded in the area of Rawalpindi city. Sick animals showed serous ocular and nasal discharge, fever up to 106°F, erosive lesions in mouth, diarrhoea and pneumonia. Mortality rate was 80%. Appropriate samples were collected for laboratory analysis. Results of competitive ELISA and immunocapture ELISA determined that the animals were suffering from PPR. Vaccination using tissue culture rinderpest virus (TCRV) was successful to curtail the infection in goats in that area.

### INTRODUCTION

Peste des petits ruminants (PPR), is an acute contagious disease caused by Morbillivirus of the family Paramyxoviridae. It occurs in Africa, the Arabian Peninsula, most of the Middle Eastern countries, India, Pakistan and Afghanistan (Shaila *et al.*, 1989; Taylor *et al.*, 1990; Russain, 1996). The disease affects mainly small ruminants, more severe in goats than in sheep and is rapidly fatal in young animals. The morbidity rate can be up to 100% and in severe outbreaks, with 100% mortality. In milder outbreaks, the mortality rate may not exceed 50%. Infected animals present clinical signs (fever, oculonasal discharge, erosive lesions in mouth, diarrhoea and pneumonea) similar to rinderpest (RP) in cattle from which it must be differentiated.

### OUTBREAK

An outbreak occurred in goats in Rawalpindi during 2<sup>nd</sup> week of June, 1997. The disease started in an herd of 24 goats and 20 of them died within a period of 15 days. There were about 100 goats belonging to different farmers in that particular area and the disease spreaded to other goats in the neighborhood. The sick goats did not respond to symptomatic treatment prescribed by a veterinarian. Clinical signs observed in sick animals were refusal to eat but excessive water intake, body temperature up to 106°F that became sub-normal after onset of diarrhoea, thick tenacious nasal discharge,

profuse watery diarrhoea (dark green colour) on second day of illness which becomes mixed with shreds of mucus membrane on third day, erosions and cheesy material in oral cavity, recumbency, followed by death within 4-5 days of illness.

When the disease was reported during 1<sup>st</sup> week of July 1997, about 30 goats had already died either due to this disease or were sold to the butchers during terminal stages of illness. Four goats (age between six months to five years) were found sick at the time of investigation. Body temperature was 105-106 °F and goats had thick, tenacious nasal discharge but little lachrymal secretions. There were erosions on upper and lower gums and upper part of the tongue and the lesions were covered with a white cheesy material. All sick animals had cough.

### LABORATORY ANALYSIS

Blood samples were collected from four sick as well as three recovered goats. Infected material from gums and nasal cavities of four sick goats was collected with sterile cotton swabs. Intestines of two already slaughtered sick animals were also examined. Mesenteric lymph nodes and selective parts of intestine were collected for laboratory analysis.

Serum samples were processed for the detection of antibodies against PPR and RP using competitive ELISA (Anderson *et al.*, 1991). All recovered and two sick animals were positive for the presence of antibodies to PPR. Remaining samples were analyzed for the presence

of antigen against PPR and RP using immunocapture ELISA (Libeau *et al.*, 1991). PPR virus antigen was detected in all except two nasal swabs. Results were negative for the presence of antibodies or antigen to RP.

A sick goat (one year old) showed clinical signs as mentioned above, was slaughtered on 3<sup>rd</sup> day of illness for postmortem examination. At necropsy, emphysema was observed in certain areas of lungs. Mesenteric lymph nodes were enlarged and gelatinous. Pharyngeal lymph nodes, tonsils, spleen and mesenteric lymph nodes were collected and analyzed by immunocapture ELISA. These were found positive for PPR but negative to RP virus antigen.

## DISCUSSION

Laboratory analysis of samples collected from sick and dead animals during this outbreak confirmed the diagnosis as PPR. About 70 goats in the area were vaccinated with tissue culture rinderpest virus vaccine (TCRV, Pirbright UK) on July 15, 1997. There was no sick animal when the area was again visited 3 weeks following vaccination. The source of infection could not be determined during these investigations.

Peste des petits ruminants was first described in Cote d'ivoire (Gargadennec and Lalanne, 1942) but it also occurred in intertropical Africa (Lefevre and Diallo, 1990), the Arabian peninsula (Taylor *et al.*, 1990) and in most of the Middle Eastern countries such as Jordan and Lobanon (Lefevre *et al.*, 1991). PPR in goats in a newly emerging disease in Pakistan. The disease was first recorded as a RP like disease in goats during 1991 (Pervez *et al.*, 1993) and as PPR like disease during 1992 (Athar *et al.*, 1995; Ayaz *et al.*, 1997). Both these reports were based upon clinical, epidemiological and postmortem findings of infected animals. The first laboratory confirmed outbreak of PPR occurred in Lahore (1994) and the samples from infected animals were analyzed at the World Reference Laboratory for Rinderpest, UK (unpublished data). A report on the use of counter immunoelectrophoresis to detect antibodies against PPR in goats has been made (Tahir *et al.*, 1998). A sero-epidemiological investigation in the Northern Areas of Pakistan, using competitive ELISA indicated high incidence (28%) of PPR antibodies in local goat population (Hussain 1996). The authors have also diagnosed two PPR outbreaks in goats in districts Talagang and Mardan during 1996-97. Control of such outbreaks by vaccinating the animals with TRCV also supported that PPR virus is circulating in goat population of Pakistan. Several outbreaks similar to PPR

have also been observed in other parts of the country causing high mortalities in goats. However, true picture about the prevalence of this disease in Pakistan is not clear mainly due to lack of proper diagnostic facilities and poor reporting system. Most field veterinarians confuse PPR with pleuropneumonia which is another limiting factor to control PPR in the country.

PPR virus also causes mild infection in cattle and the presence of antibodies can neutralize RP virus vaccine (Anderson *et al.*, 1993). This is a serious consideration since international agencies are planning to fund a national project for the eradication of RP in Pakistan. Presence of PPR virus in the livestock population may affect the success of this project and the authorities should look into its significance before the commencement of this project.

Use of TCRV in small ruminants can make it difficult to determine their role as a reservoir for the transmission of rinderpest virus to the large animals. A specific vaccine, containing live PPR virus, is now commercially available for the control of this disease. It is important that the veterinary authorities in Pakistan should introduce this vaccine for the control of PPR in goats.

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