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Peste des Petits Ruminants (PPR)

ESGPIP

ETHIOPIA SHEEP AND GOAT PRODUCTIVITY IMPROVEMENT PROGRAM

Further information:

Ethiopia Sheep and Goat Productivity Improvement Program (ESGPIP)

Tel. +251 011 416 6962/3
Fax: +251 011 416 6965
E-mail: *pvamrf_ethiopia@ethionet.et*

FOREWORD

This technical bulletin titled "*Peste des Petits Ruminants (PPR)*" is the twentieth in a series of technical bulletins produced by the Ethiopia Sheep and Goat Productivity Improvement Program (ESGPIP) as an extension support tool to improve the productivity of sheep and goats in Ethiopia.

PPR is one of the most important diseases that seriously hinder sheep and goat production in Ethiopia. Knowledge of the disease in terms of the symptoms observed and prevention methods is important in combating the disease and consequently improve sheep and goat productivity. This technical bulletin is the first in a series on important diseases affecting sheep and goat productivity.

Kebele Development Agents (KDA's) should use this technical bulletin as an extension aid to train producers on recognition of the disease and above all to help them take preemptive measures to prevent the occurrence of the disease.

At this juncture, I would like to thank all those involved in the preparation and review of this technical Bulletin.

Desta Hamito (Prof.)
Chief of Party
ESGPIP

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TABLE OF CONTENTS

Peste des Petits Ruminants (PPR)

Prepared by: Sileshi Zewdie **Edited by: Alemu Yami, R.C. Merkel and L. Dawson**

1. Introduction

Peste des Petits Ruminants is common in Ethiopia. PPR is a highly acute contagious and infectious viral disease of goats and sheep that is clinically similar to rinderpest and is characterized by fever, erosive stomatitis, diarrhea, conjunctivitis, gastroenteritis, and pneumonia. The name is French for "disastrous disease of small ruminants". Goats are usually more severely affected than sheep.

The presence of PPR can have a serious impact on livestock production and trade. Economic losses are due to loss of production, death, abortion and cost of controlling the disease. The presence of the disease can limit local trade and export.

2. Organism

Peste des petits ruminants (PRR) is a paramyxovirus of the genus Morbillivirus. It is antigenically very similar to the Rinderpest virus. Other members of the genus include measles virus and canine distemper virus.

3. History

PPR is one of the constraints of small ruminant production in Ethiopia. PPR entered Ethiopia in 1989 in the southern Omo River valley from where it moved east to Borana then northwards along the Rift Valley to Awash. The disease then spread northwards into the central Afar Region and eastwards into the Ogaden. In 1997, a survey conducted at Debre Zeit abattoir demonstrated high sero prevalence rates of 85.7 percent in animals from the pastoral areas, 43.2 percent from sedentary farms and 32.9 percent from mixed farms. Strains of PPR virus that cause only sub-clinical disease have been identified in several areas of the country.

4. Animals affected

Whereas clinical disease is seen in both sheep and goats, goats are more susceptible to PPR than sheep. Cattle are susceptible to infection but they do not exhibit clinical signs. Cattle do not, however, play a role in the spread of PPR because they are apparently unable to transmit the disease to other animals.

5. Morbidity/Mortality

The morbidity and mortality rates from PPR can be up to 100% in severe outbreaks. In milder outbreaks, morbidity is still high but the mortality rate may be closer to 50%. Severity depends upon the susceptibility of the population with young animals (4-8 months) usually having more severe cases. Poor nutritional status, stress of movement and concurrent parasitic and bacterial infections enhance the severity of clinical signs. The incidence of PPR in an endemic area is similar to that of rinderpest in that a low rate of infection exists continuously. When the susceptible population builds up, periodic epidemics occur with almost 100% mortality

6. Transmission

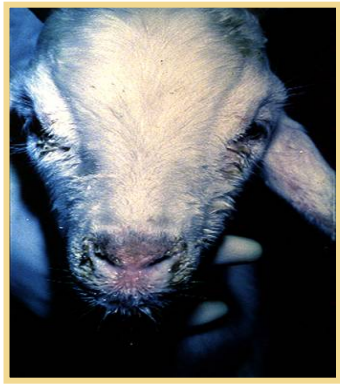
Transmission of PPR requires close contact. The virus is present in eye, nose, and mouth discharges as well as feces. Most infections occur through inhalation of aerosols from sneezing and coughing animals. Animals may be infectious during the incubation period. There is no known carrier state.

7. Clinical Signs

Most cases of PPR are acute, with a sudden fever that may last for 5-8 days before the animal either dies or begins to recover. The characteristic signs begin with a clear discharge from the nose that becomes grey and sticky. The discharge from the nose may remain mild or may progress to severe inflammation of the mucous membrane of the nose characterized by the presence of exudates that crust over, blocking the nostrils causing respiratory distress. The nasal mucous membranes may develop small areas of erosion. The conjunctiva may be congested with matted eyelids. The mucous membranes in the mouth may also be eroded.

Concurrently, animals will most likely have profuse, non-hemorrhagic diarrhea resulting in severe dehydration, which may progress to emaciation, difficult breathing and die within 5-10 days. Bronchopneumonia with coughing is common late in the disease. Abortion may be seen in pregnant animals. The prognosis of acute PPR is usually poor. The severity of the disease and outcome in the individual is correlated with the extent of the mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor when extensive necrosis and secondary bacterial infections result in a fetid odor from the animal's mouth. Respiratory involvement is also a poor prognostic sign.

Depression, hemorrhage, diarrhea.



Discharge from the eyes, nose, mouth and erosion in the mouth

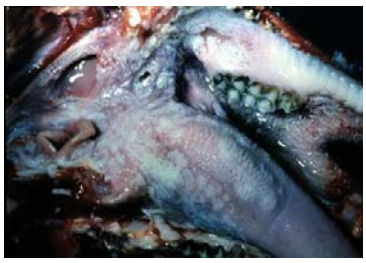
Close up view of mouth lesions

Figure 1. Clinical signs

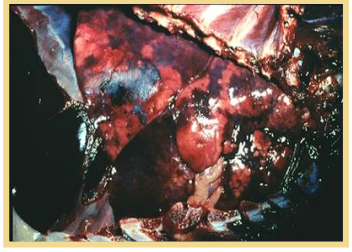
8. Post mortem lesions

In dead animals, the eyes and nose will have a dirty white/grey discharge and the animal's rear will often be covered with bad smelling, watery feces. Post mortem lesions are similar to rinderpest, with inflammatory and necrotic lesions in the oral cavity and throughout the gastro intestinal tract. In severe cases the hard palate, pharynx and upper esophagus also have lesions. The carcass is generally emaciated.

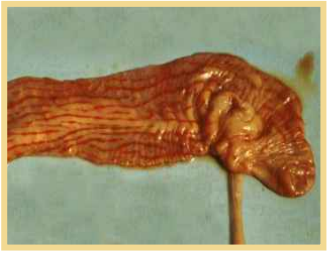
The most severe lesions are seen in the large intestine, with congestion and "zebra stripes" of congestion on the mucosal folds of the posterior colon. Erosive lesions may also occur in the vulva and vaginal mucous membranes. There is pus in the lungs. Congestion and enlargement of the spleen may be seen. The lymph nodes are generally congested and enlarged.



Necrotic lesions in the oral cavity



Pneumonia



zebra stripes on intestine

Figure 2. Post-mortem lesions

9. Diagnosis

Differential diagnosis include: rinderpest, contagious caprine pleuropneumonia, bluetongue, Pasteurellosis, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis, Nairobi sheep disease and mineral poisoning. The case history, geographic location and the combination of clinical signs can help differentiate some of these diseases.

To confirm PPR outbreak some laboratory tests have to be carried out at the National Animal Health Diagnostic & Investigation Center or at the nearby Animal Health Regional Laboratory

Development agents should inform the nearby animal health personnel about the outbreak so that appropriate samples should be taken. For PPR diagnosis swabs of the mucous membrane of eye, nose, mouth and rectal discharges should be collected. Whole blood must be collected in heparinized tubes. Samples may also be taken of the spleen, large intestine and lungs. These samples should be transported under refrigeration.

10.Treatment

There is no treatment for PPR but it helps to give antibiotics to stop secondary bacterial infections.

11.Prevention and Control

Barns, tools and other items that have been in contact with the sick animals must be cleansed and disinfected with common disinfectants (phenol, sodium hydroxide 2%, virkon) as well as alcohol, ether, and detergents. The virus can survive for long periods of time in chilled or frozen tissues.

New animals should be quarantined for three weeks before allowing them to mix with the flock. In a case of PPR outbreak, animals with signs of PPR should be isolated immediately and sheep and goats around the outbreak area should be vaccinated as soon as possible. Vaccine for PPR is effective. Vaccinate before start of the rainy season. In endemic areas sheep and goats should be vaccinated annually. Vaccine for PPR is produced by the National Veterinary Institute. Carcasses of dead animals and contaminated items should be buried or burned.

The role of Kebele Development Agents in prevention & control of Peste des Petits Ruminants (PPR)

- **Advise farmers/pastoralists to keep newly purchased sheep and goats separate from other animals for about three weeks.**
- **Advise farmers/pastoralists to isolate animals with signs of PPR immediately and to move their healthy animals to other clean area.**
- **Immediately report disease outbreaks to the nearest Office of Agriculture and Rural Development.**
- **Arrange with the Office of Agriculture and Rural Development to vaccinate all sheep and goats that have been in contact with sick animals. Observe the vaccinated animals very closely every day. If any animal shows signs of PPR put it with the sick ones that have been isolated.**
- **Arrange with the community and the Office of Agriculture & Rural Development for annual vaccination of sheep and goats against PPR.**
- **Provide regular public awareness education on the prevention and control of PPR.**

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