PESTE DES PETITS RUMINANTS

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Summary: Peste des petits ruminants (PPR) is a viral disease of goats and sheep characterised by necrotising and erosive stomatitis, enteritis and pneumonia. The causative agent is a member of the family Paramyxoviridae and the genus Morbillivirus.

The disease was reported for the first time in 1942 in Côte d'Ivoire and the geographical spread of the disease has grown since then. In general, it occurs in African countries lying between the Sahara and the Equator, in the Middle East and in India. The disease was ignored in these different areas because it was confused with rinderpest or with Pasteurella infections which is an usual complication of PPR.

The disease is on the List A of the International Animal Health Code of the OIE. With the exception of Oman, no reports have indicated the occurrence of PPR in the OIE Middle East Member Countries since the last conference, held in Damascus in 1993.

In terms of the diagnosis, new methods used for the antibody (C-ELISA) and/or antigen (Immunocapture ELISA, PCR) detection of the disease are described. The control of PPR in the region needs to be organised in a network taking into consideration the epidemiology of the disease.

In endemic areas a realistic strategy should be based on whole flock vaccination and sustained for many years.

The preferred vaccine, according to research carried out so far, is the attenuated homologous vaccine developed by Diallo et al, 1989.

1. INTRODUCTION

Peste des petits ruminants is a highly contagious viral disease which affects sheep and goats. It has an increasingly significant economic impact on a number of countries in Africa and Middle East, and is often associated with high morbidity and mortality (1).

The disease carries the reference number A 050 in the OIE disease numbering system. Each case should be reported to the OIE.

The disease was first described in Côte d'Ivoire (2) and had different synonyms such as pneumoenteritis complex, erosive stomatitis and enteritis of goats, goat catarrhal fever, Kata or pseudo-rinderpest.

The casual agent belongs to the morbillivirus genus of the family Paramyxoviridae and possesses antigenic links with the viruses of rinderpest, canine distemper, phocine distemper and human measles (3, 4) (Fig.1).

The half-life of a hydrated virus held at 37°C is about 2 h; at 50°C, infectivity is destroyed within 30 min (6).

PPR virus is also sensitive to lipid solvents and to low pH. A virus present in lymph nodes is protected from pH changes after death, and it can be recovered from the lymph nodes of carcasses held for 8 days at 4°C.

Clinically, PPR resembles rinderpest and is characterised by ocular and nasal discharges, erosive stomatitis and diarrhoea. PPR outbreaks occur one or two weeks after purchase and stocking.

The incubation period is about six days. The onset of the disease is marked by a sudden dullness and pyrexia, with high body temperature (40°-41°C). The different mucous membranes are congested with a serious ocular and nasal
discharge. The animal is reluctant to eat. Two days after the onset of the disease, tiny, greyish necrotic spots develop on the tongue, the inner surface of the lips, the cheek mucosae and the gums.

![Diagram of virus relationships](image)

*Fig. 1: Diagram on the relationship of the different morbilliviruses according to the genetic sequences of the nucleocapsid proteins (5)*

The necrotic lesions coalesce very quickly and form plaques with thick, white dead tissues causing a highly fetid smell in the mouth. The disease also shows signs of bronchopneumonia characterised by a dry cough, which becomes soft, moist and productive very quickly. The ocular and nasal discharges become mucopurulent, and can make the eyelids stick together and partially block the nasal openings and thus render breathing difficult. Diarrhoea starts after the drop of the fever and the animal becomes weak. Death follows prostration within 10 to 15 days after the onset of the disease. In some cases, the animal survives and the above-mentioned acute symptoms will regress and disappear within 3 to 4 weeks. The dry crusts around the eyes and the muzzle may persist for a long time. In the hyperacute form, the disease may appear within 4 to 6 days.

The mortality rate in the acute form is about 20 to 60% and can reach 100% in the hyperacute form. However, in some instances, the symptoms can be very mild or absent, which makes the clinical diagnosis of the disease difficult or impossible. All suspected cases should be confirmed by laboratory tests.

*Pasteurella* pneumonia almost invariably supervenes in acute cases and complicates the clinical and postmortem findings. In addition, a wide range of other secondary bacterial invaders have been isolated.

PPR virus circulates in a belt lying across Africa, immediately south of the Sahara (7). It exists in the Middle East, in India and Bangladesh. It was ignored before in those areas, because it was confused with rinderpest or with *pasteurella* infection which is a usual complication of PPR.

As for rinderpest, there is no vector for PPR and the transmission of the virus occurs by close contact between an infected animal and a susceptible one. In Africa, goats are more affected, while in India, sheep are struck more often than goats (8).

Recent reports from countries in the Middle Eastern region, notably Cyprus, Egypt, Kuwait, Qatar, Sudan and Syria, indicate that no PPR outbreaks have been registered in the area since the last conference in 1993. Only Oman has reported the occurrence of PPR on an annual basis.

### 2. Diagnosis

#### 2.1. Laboratory diagnosis

As the clinical signs of PPR may vary considerably and be confused with other diseases, particularly with rinderpest and *pasteurella* infections in small ruminants, any clinical diagnosis or suspicion of the disease should be considered as provisional, until a confirmation is made by the laboratory.

Laboratory diagnostic methods have been introduced in some regional laboratories in the Middle East by the Animal Health Division of the International Atomic Energy Agency (IAEA).

The Research Laboratory at the Faculty of Veterinary Medicine at Al Baath University in Hama, Syria, is one of those laboratories providing the region with the new technique which ensures the diagnosis of PPR.
2.1.1. Antibody detection methods

The demonstration of a rising antibody titre or the presence of antibodies associated with the primary immune response are indicators of recent infection. Therefore, serological techniques designed to demonstrate these phenomena may be used to confirm a diagnosis of PPR.

2.1.1.1. The neutralisation test

The neutralisation test is used for antibody detection. The highest neutralisation titre is obtained against the homologous virus. Vero cells are commonly used for this test (9).

2.1.1.2. Competitive ELISA

New reagents have been produced and allow the development of a rapid, sensitive test. The convenient test is the competitive ELISA (C-ELISA) which is based on the use of a monoclonal antibody competing with the test sera in binding to an epitope. Two types of C-ELISA have been developed, one in which the antigen is the purified virus or the sonicated virus infected cells (10). For the second, the antigen (Nucleoprotein N) is a recombinant nucleocapsid protein expressed in baculovirus vector (11).

The test depends on inhibition of binding of a mouse monoclonal antibody to the specific epitope in the presence of positive serum. Inhibition is detected as a reduction in the optical density reading obtained with the conjugate and substrate/chromogen mixture. A threshold value of 50% inhibition is adopted for routine screening.

2.1.2. Antigen detection methods

2.1.2.1. Agar-gel immunodiffusion

The simplest test is the AGID-test to detect PPR antigen. The extract of pathological samples (ocular and nasal swabs, lymph nodes, spleen or lung) is allowed to diffuse, along with known PPR positive antigen, in agar gel against a rabbit PPR hyperimmune serum.

2.1.2.2. The immunocapture ELISA

In this test, two non-overlapping monoclonal antibodies (mAb), directed against the nucleoprotein N, are used. One mAb, the detection antibody, is allowed to react with the test material. Then the formed immunocomplex is captured by the second antibody (capture mAb), previously absorbed onto the ELISA plate, to detect the virus in supernant from infected cells and pathologic samples collected from sick animals. This assay can be performed in one hour on pre-coated plates. There is no cross-reaction between rinderpest and PPR viruses in the test.

2.1.2.3. The Polymerase Chain Reaction

Another possibility for detecting PPR virus is offered by the application of nucleic acid technology. Based on the specific amplification of a virus nucleic acid fragment, this test is very sensitive and the limit of detection has been estimated to be $2 \times 10^3$ PPR virus genome molecules. Combined with the sequencing of amplified products, PCR is now a powerful tool for molecular epidemiology studies of PPR (8).

3. SITUATION IN THE MIDDLE EAST

For the purposes of this paper the Middle East region comprises the states of Afghanistan, Bahrain, Cyprus, Egypt, Iran, Iraq, Jordan, Kuwait, Lebanon, Libya, Oman, Qatar, Saudi Arabia, Somalia, Sudan, Syria, Turkey and the United Arab Emirates, all of which are members of the OIE Regional Commission for the Middle East.

3.1. Peste des petits ruminants in the past
For many years, PPR was considered an African disease localised mainly in West and Central Africa, but some recent evidence seems to indicate that its geographical distribution may be greater than first suspected.

According to the FAO/OIE/WHO Animal Health Yearbooks (1989-1993), the disease has been reported in Benin, Burkina, Burkina Faso, Cameroon, Côte d'Ivoire, Ethiopia, Gambia, Ghana, Guinea, Guinea Bissau, Jordan, Mauritania, Lebanon, Mali, Niger, Nigeria, Oman, Saudi Arabia, Senegal, Sudan, Togo, United Arab Emirates and Yemen Arab Republic.

3.2. Present situation

The present status of PPR in the Middle East is as follows:

Cyprus: PPR is compulsorily notifiable, but this disease has never been recorded in Cyprus.

Egypt: In January 1987 a limited PPR focus appeared in a localised area. In 1989 three foci were recorded as PPR. Since 1989 no cases of PPR have been recorded and Egypt has been free of the disease since that time.

Kuwait: There have not been any reported cases of PPR in the state of Kuwait, either in indigenous flocks or in those imported for slaughter.

Oman: The disease was reported in Oman in 1983, and since then outbreaks of PPR have been very common in the country. The number of outbreaks and cases since 1987 are summarised as follows:

<table>
<thead>
<tr>
<th>Year</th>
<th>No. outbreaks</th>
<th>No. cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>126</td>
<td>29037</td>
<td>1425</td>
</tr>
<tr>
<td>1988</td>
<td>131</td>
<td>8654</td>
<td>452</td>
</tr>
<tr>
<td>1989</td>
<td>80</td>
<td>2096</td>
<td>130</td>
</tr>
<tr>
<td>1990</td>
<td>72</td>
<td>2994</td>
<td>120</td>
</tr>
<tr>
<td>1991</td>
<td>118</td>
<td>5103</td>
<td>508</td>
</tr>
<tr>
<td>1992</td>
<td>149</td>
<td>4425</td>
<td>253</td>
</tr>
<tr>
<td>1993</td>
<td>166</td>
<td>3112</td>
<td>230</td>
</tr>
<tr>
<td>1994</td>
<td>147</td>
<td>2541</td>
<td>297</td>
</tr>
</tbody>
</table>

Qatar: no cases of PPR have been reported in the country.

Sudan: PPR was observed for the first time in eastern Sudan in February 1971 at the Ethiopian border not far from the Dinder National Park. The disease spread in January 1972 to the west and to the north, reaching Khartoum and Gazira state in the centre of the country. Another outbreak was recorded in 1974. Sudan remained free from the disease until 1989 when it was reported again in the central area of the country between Rufaa and Wad Rawa. The disease spread to the Khartoum state and to the Northern Dafur in Western Sudan in 1990. Since 1991 there have been no reports of the disease in the country (12).

Syria: no cases of PPR were reported in the country. The last registered PPR case was in 1987.

No report has been received from the following countries: Afghanistan, Bahrain, Iran, Iraq, Jordan, Lebanon, Libya, Saudi Arabia, Somalia, Turkey and the United Arab Emirates.

3.2.2. The different strategies existing for the control of PPR in Member Countries are as follows:

Cyprus: to prevent the introduction of PPR into Cyprus the current veterinary control programme ensures a complete safeguard against the introduction of the disease. If PPR occurred in Cyprus, the stamping out policy would be practised. The following requirements are in force to ensure the safety of the importation of animals and animal products:

For the import of small ruminants: a) the imported animals should originate from a PPR-free territory; b) the disease must not have occurred during the last 12 months; c) no PPR vaccination must have been practised within 12 months preceding the export of the animals.
For the import of fresh meat: The exporting country must have been free from PPR for at least 12 months and the fresh meat obtained from animals slaughtered at least 12 months after the last occurrence of PPR.

**Egypt**: No vaccination against PPR must have been practised because sheep and goats are used as indicators for verification of the presence/absence of circulating RP virus.

**Oman**: The disease is controlled by yearly vaccination. In recent years, the disease has only been reported among nonvaccinated and especially young weaned kids. The number of vaccinated animals is as follows:

<table>
<thead>
<tr>
<th>Year</th>
<th>No. vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>75717</td>
</tr>
<tr>
<td>1988</td>
<td>504941</td>
</tr>
<tr>
<td>1989</td>
<td>445845</td>
</tr>
<tr>
<td>1990</td>
<td>474356</td>
</tr>
<tr>
<td>1991</td>
<td>25213</td>
</tr>
<tr>
<td>1992</td>
<td>353742</td>
</tr>
<tr>
<td>1993</td>
<td>380250</td>
</tr>
<tr>
<td>1994</td>
<td>433820</td>
</tr>
</tbody>
</table>

Strict veterinary quarantine procedures are in place but should be tightened further.

**Sudan**: Further studies in the epidemiology of PPR in the Sudan are necessary to develop an effective control policy.

**Syria**: For the prevention and control of PPR in the country the following procedures are in force: a) the imported ruminants should originate from a PPR-free territory, and the disease must not have occurred in the area in question within the last 36 months before importation; b) the imported ruminants must be clinically examined at the border before entering the country and a 15-21 day quarantine is necessary to ensure that the imported animals are free from PPR; c) no vaccination procedures are used against PPR in Syria.

### 4. Prevention and control

The increasing role of PPR worldwide and in the Middle East region requires a network like WARECC (West Asia Rinderpest Eradication Campaign Coordination).

The procedures for the protection of **PPR free territories** include prohibiting the importation of live animals (especially small and wild ruminants) from endemic infected areas and areas at risk. The occurrence of an epidemic in these areas should be treated by using a stamping out strategy for all infected areas animals and animals in contact with them.

The procedure for the eradication of PPR in the **endemic territories** consists of vaccinating all animals in the infected area followed by seromonitoring and surveillance to ensure that the vaccination programme has been done effectively.

Taking advantage of the close relationship between rinderpest virus and PPR virus, rinderpest vaccine has been used to protect small ruminants against PPR in all regions where the disease is endemic (13). Now an effective homologous attenuated vaccine is available (14). Its use is being recommended in all countries where rinderpest does not exist. However, for countries where PPR does not exist but where a risk of contamination is present sanitary measures and serosurveillance at the border with contaminated neighbours should be maintained.
REFERENCES


