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Reflecting on the past and fast forwarding to present day information on PPR

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Abstract

Peste des petits ruminants (PPR) is a highly contagious and economically important viral disease affecting goats, sheep and wild ruminants that is caused by a Morbillivirus that belongs to the family Paramyxoviridae. PPR spreads quickly in susceptible ruminant species, and the highest number of outbreaks occurs in sheep and goats. Morbidity and mortality are usually high, and PPR can create epidemics that can cause up to 100% mortality in susceptible sheep and goat populations. Virus isolation is the gold standard for the diagnosis of PPR. Current control of the disease mainly includes isolation and disinfection of the contaminated environment, and administration of a live-attenuated vaccine, which provides a strong immunity. Mass vaccination of sheep and goats in endemic countries might be a pragmatic approach to control PPR in the first phase of disease eradication. In the future, the development of a marker vaccine with a robust companion test may help in serosurveillance to identify infection in vaccinated animals to control PPR disease.

Key words: PPR, Kata, Goat plague, Vaccination

Introduction

The first report of PPR was made in the Ivory Coast, West Africa, in 1942. Today, PPR is quite common in both Africa and parts of Asia. PPR, kata, or goat plague is caused by peste des petits ruminants virus (PPRV), a morbillivirus belongs to family Paramyxoviridae closely related to the morbilliviruses of rinderpest, measles (in humans), carnivores, and marine mammals. The disease occurs mostly in goats and sheep and has spread extensively across national and continental boundaries in the last few decades (Gibbs *et al*. 1979). The incubation period of the disease is 4–6 days, but can be up to 14 days. Clinical infection varies, and may include fever, oculo-nasal discharges, oral erosions, pneumonia and diarrhoea (Naznin, *et al*. 2014). PPR is considered to be the most significant economic threat to the development of sustainable sheep and goat production across the developing world, particularly in Africa and Asia. Infection rates in enzootic areas are generally high (above 50%) and can be up to 90% of the flock during outbreaks. There is no significant seasonal variation in the prevalence of the disease but because maternal antibodies are lost at about 4 months of age, the number of susceptible animals is likely to increase 3 to 4 months after peak kidding and lambing seasons (Diallo *et al*. 2007).

Modes of transmission

PPRV is transmitted mainly by aerosols when animals live in close contact. Large amounts of the virus are present in exhaled air and in all body excretions and secretions including faeces, saliva, ocular and nasal discharges, and urine that can contaminate fomites (Parida *et al*. 2016). Diarrheic feces are especially infectious. Infection is mainly by inhalation but could also occur through the conjunctiva and oral mucosa.

Risk factors and immune mechanisms

Kids over 4 months and under 1 year of age are most susceptible to the disease, corresponding to waning maternal antibodies from immune dams. In a particular flock, the risk of an outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity. The causative agent, Peste des

petits ruminants virus (PPRV), is considered sensitive to abiotic environmental factors, and it does not survive long outside of a host.

Clinical findings

The disease can be peracute, acute, or subacute. The peracute and acute forms are seen mainly in goats and are similar to rinderpest in cattle except that severe respiratory distress is a common feature of caprine PPR. Signs generally appear 3 to 6 days after being in contact with an infected animal. A high fever (above 40°C) is accompanied by dullness, sneezing, and serous discharge from the eyes and nostrils (Banyard *et al*. 2010). A day or two later, discrete necrotic lesions develop in the mouth and extend over the entire oral mucosa, forming diphtheritic plaques. There is profound halitosis and the animal is unable to eat because of a sore mouth and swollen lips (Hoy *et al*. 2012). Nasal and ocular discharges become mucopurulent and the exudate dries up, matting the eyelids and partially occluding the external nares. Diarrhea develops 3 to 4 days after the onset of fever (Li *et al*. 2017). It is profuse and feces may be mucoid and blood tinged. Dyspnea and coughing occur later, and the respiratory signs are aggravated when there is secondary bacterial pneumonia. Erosions have been described in the vulva and prepuce. Death usually occurs within 1 week of the onset of illness and earlier in peracute cases. Subacute forms are more common in sheep but they also occur in goats. The signs and lesions are less marked and a few animals may die within 2 weeks, but most recover.

Laboratory diagnosis

Laboratory confirmation of PPR can be accomplished by virus isolation, PCR, ELISA, detection of viral antigen by fluorescent antibody testing, virus neutralization or AGID; detection of rising antibody titer by ELISA or virus neutralization; and histopathologic evaluation, including immunohistopathology (Jaisree *et al.* 2017). Virus isolation is most successful in the first days of infection (often before the onset of diarrhea). Blood should be taken in heparin. The lymph nodes and spleen are reliable sources of virus, and tears and ocular discharges are also reliable sources. Lymph node biopsy after day 3 of infection is the most reliable means of diagnosis in a living goat. Tissues should be shipped frozen on ice to the laboratory for virus isolation or detection of antigen (Anderson and McKay, 1994). Detection of a rising antibody titer can help diagnose the disease retrospectively. In PPR, lesions of the oral cavity and intestines characterized by erosive and ulcerative stomatitis and enteritis, and the presence of pathognomonic syncytial cells and inclusion bodies, are identical with those of rinderpest in cattle (Scott, 1990; Barker *et al.* 1993).

Treatment

Treatment of goats showing clinical signs of PPR is of little value and is generally symptomatic (Abubakar, *et al.* 2016). Supportive treatment includes fluid therapy for dehydration and antibiotics to prevent secondary bacterial infections. Lesions around the eyes, nostrils, and mouth should be cleaned and good nursing provided.

Prevention and control

An effective vero-cell line based live attenuated indigenous freeze-dried vaccine can be used to prevent PPR.
PPR can be controlled by keeping following points in view:

- Focused vaccinations in high-risk populations of sheep and goats
- Mass vaccination campaigns to achieve 70% to 89% herd immunity
- An understanding of the cultural and socioeconomic circumstances of owners
- A keen watch on the endemic nature of PPR in neighbouring countries
- Coordinated efforts from all stakeholders
- Proper funding and execution of control programs

Vaccines against PPR

The first homologous PPR vaccine was developed using the live-attenuated Nigerian strain PPRV Nig 75/1 (Diallo *et al.* 1989). Similarly, three other homologous PPR vaccines using Indian isolates of PPRV have been developed and evaluated recently by the Indian Veterinary Research Institute (IVRI; Mukteswar, India) and Tamil Nadu University of Veterinary and Animal Science (TANUVAS; Chennai, India).

Salient features of peste des petits ruminants vaccine (Sungri/96)

Vero cell-based live-attenuated peste des petits ruminants vaccine targeting goats and sheep belonging to Lineage 4 (Asian lineage) in phylogeny. Freeze-dried vaccine is presented in 50/100 doses per vial in diluents 1M $MgSO_4$ /0.85% normal saline solution having shelf life more than 1

year at 4ºC. Stability of the diluted vaccine is at least 8 h. Dose is 1.0 ml of reconstituted vaccine administered via subcutaneous route in neck region. Duration of immunity lasts for more than 6 years.

Conclusion

Peste des petits ruminants (PPR) is a highly contagious and infectious viral disease of domestic and wild small ruminants characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis and pneumonia. Goats are usually more severely affected than sheep. The virus is primarily transmitted through aerosol and direct contact between infected and susceptible animals. The magnitude of the disease depends on several factors such as the virulence of the PPR virus strain, species of animal, age, gender, breed, host immune status and previous population exposure to PPRV. In some cases, particularly in the mild form of the disease, affected animals develop coughing and diarrhoea, and spontaneous recovery may occur within 10–15 days of infection. Sheep and goats in endemic regions may develop lifelong immunity following natural infection, but naive animals may allow continuous circulation of the virus to establish an endemic situation. Screening tests for PPR, and effective preventive and eradication measures, should be routinely carried out in sheep and goat flocks in regions with a high disease prevalence, to control the outbreak and improve animal productivity.

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