RINDERPEST

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Abstract

Rinderpest, also known as cattle, plague, was a highly contagious and fatal viral disease of cloven-hoofed animals, particularly cattle, causing severe economic and agricultural losses worldwide. The disease was caused by a morbillivirus of the Paramyxoviridae and was characterized by lymphoid depletion, immunosuppression, and necrotizing lesions of the gastrointestinal tract. Transmission occurred primarily through direct contact, aerosols, or contaminated materials. Following infection, the virus replicated in lymphoid tissues and epithelial cells, leading to leukopenia, mucosal erosions, and severe diarrhoea. Clinical manifestations included fever, depression, oculonasal discharge, oral lesions, and the characteristic "four Ds": depression, dehydration, discharge, and diarrhoea. Diagnosis was based on clinical signs and laboratory techniques such as ELISA and RT-PCR. Effective control strategies, including vaccination, surveillance, and strict movement control, ultimately led to its global eradication in 2011, declared by the Food and Agriculture Organization and World Organisation for Animal Health. This achievement marked a milestone in veterinary medicine and demonstrated the success of coordinated international efforts. Despite eradication, rinderpest remains important as a model for controlling transboundary animal diseases and strengthening global animal health systems.

Keywords: Rinderpest, Pathogenesis, Eradication, Morbillivirus

Introduction

Rinderpest or Cattle plague is an acute or subacute infectious viral disease that causes lymphocyte loss and necrotic lesions in the mouth and gastrointestinal system. Although all species of Artiodactyla (even-toed ungulates) are likely susceptible, the disease primarily affects cattle. The disease has been the foremost cause of death in cattle in most African and Asian countries, including India. It is the most devastating infectious disease of cattle with mortalities higher than 70%. There were significant rinderpest outbreaks in Europe throughout the 18th century, with an estimated 200 million cattle lost. The most recent rinderpest case was found in Kenya in 2001. In 2011, FAO and OIE formally stated that rinderpest had been eradicated worldwide. Rinderpest is the second infectious disease to be eradicated, after smallpox, which the World Health Organization announced eradicated in 1980. Rinderpest has not been reported since June 1995 in India. The scientific underpinning of the worldwide rinderpest eradication is briefly described in this review from a historical perspective.

Etiology

Rinderpest is caused by *Morbillivirus* of family *Paramyxoviridae*. Measles in humans and distemper in carnivores are caused by the same virus that causes rinderpest and peste des petits ruminants, a related disease of tiny ruminants. Animals that are infected release the virus through sweat, urine, faeces and nasal discharges. Aerosol (infected droplets) inhalation or contaminated feed are the two ways that infection is spread.

Pathogenesis

The virus multiplies in the tonsils and local lymph nodes after being inhaled in contaminated droplets that pierce the upper respiratory tract's epithelium. From these locations, the virus spreads throughout the body in mononuclear cells that enter the bloodstream. It is closely linked to leukocytes and only a tiny percentage is free in plasma. The virus replicates in monocytes, lymphocytes, and epithelial cells and has a strong predilection for lymphoid tissues and the mucosa of the mouth. Significant leukopenia is caused by a stunning destruction of lymphocytes in tissues. Both necrosis and apoptosis kill lymphocytes. The disease's hallmarks of localized, necrotic stomatitis and enteritis are directly caused by viral infection and replication in the alimentary tract's epithelial cells. However, as soon as clinical symptoms and lesions appear, the virus quickly declines and is eliminated from the body because to the powerful antibody response it causes soon after infection. Severe dehydration is typically the cause of death, although in less severe cases, activated latent parasite or bacterial infections may be the cause. These infections are made worse by the animal's immunosuppressed state due to the virus's destruction of lymphoid organs.

Clinical Findings

Clinical indications of fever, anorexia, depression, and oculonasal discharge appeared after an incubation period of 3 to 15 days in typical rinderpest cases. These were followed by necrotic lesions on the tongue, buccal mucosa, and gums. Both the soft and hard palates were frequently impacted. The snout looked dry and cracked, and the oculonasal discharge turned mucopurulent. The last clinical symptom, diarrhoea, may be bloody and watery. If one or more animals exhibit clinical symptoms typical of stomatitis-enteritis syndrome, rinderpest should be suspected. The common clinical manifestations are frequently described by the well-known four Ds: depression, dehydration, discharge, and diarrhoea. The animal species affects the clinical symptoms.

Lesions of Rinderpest

Rinderpest caused gross pathological lesions in the upper respiratory and gastrointestinal tracts, either as erosion and necrosis or as congestion and bleeding, the latter of which resulted in the rectum classic zebra striping. Animals with rinderpest may have edematous, swollen lymph nodes with white necrotic foci in Peyer's patches. Histological abnormalities comprised intracytoplasmic and intranuclear inclusions, as well as lymphoid and epithelial necrosis with viral-induced syncytia.

Diagnosis of Rinderpest

- Before eradication: clinical and pathological findings after initial laboratory confirmation
- After eradication: laboratory confirmation by a WOAH/FAO rinderpest reference laboratory

Diagnosis of Rinderpest Before Eradication

Clinical and pathological symptoms were adequate for diagnosis in endemic areas and upon initial laboratory confirmation of an outbreak prior to the eradication of rinderpest. Laboratory testing was necessary to distinguish rinderpest from other illnesses that can cause stomatitis-enteritis syndrome in places where it was rare or non-existent. These illnesses included bovine viral diarrhoea, East Coast fever, foot-and-mouth disease, infectious bovine rhinotracheitis, papular stomatitis, vesicular stomatitis, and malignant catarrhal fever.

Agar gel immunodiffusion (AGID) assay, antigen capture ELISA, differential immunocapture ELISA (ic-ELISA), RT-PCR, and virus isolation were among the test techniques available for determining the causal agent of rinderpest.

Diagnosis of Rinderpest After Eradication

A susceptible animal infected with the rinderpest virus, with or without clinical symptoms, is considered a case of rinderpest in the post-eradication era. Positive control virus and viral antigen are not accessible for rinderpest diagnosis outside of a WOAH/FAO rinderpest reference laboratory due to global prohibitions on the distribution and use of materials containing rinderpest virus. For non-WOAH/FAO rinderpest reference laboratories, RT-PCR or real-time RT-PCR assay using the established primer sets is advised for preliminary testing of a suspected case of rinderpest. Instead of using a rinderpest positive control, the test can be conducted in parallel using a peste des petits ruminants' virus and published primer sets for the virus. As an alternative, an internal control reaction can be conducted in parallel using bovine actin primers. A WOAH Reference Laboratory for Rinderpest or an FAO Reference Center for Diagnostics of Rinderpest must confirm a rinderpest viral infection.

Prevention and Control of Rinderpest

- Immunity from a laparinized vaccine lasts for one to seven years.
- Goat tissue vaccine: 13 years or a lifetime of immunity
- In countries where rinderpest is exotic, verified outbreaks are managed through appropriate quarantine and animal movement controls, as well as the killing and destruction of all affected and in-contact animals.
- The importation of live infected animals has been the cause of every rinderpest outbreak in virgin areas.
- Vigilant supervision over the introduction of live animals from potentially contaminated areas is therefore crucial to prevention in such places.
- To destroy the viral envelope, contaminated surfaces should be physically cleaned of all animal waste and dirty bedding before being treated with disinfection solutions that comprise solvents with a pH of either high (>10) or low (<3).
- Calves between the ages of six and twelve months received the RP vaccination in endemic areas. The highest levels of herd immunity will result by vaccinating every cow annually.

Conclusion

Rinderpest, devastating viral disease of cattle characterized by rapid spread, severe tissue damage, and high mortality. Its worldwide eradication in 2011 by the Food and Agriculture Organization and World Organisation for Animal Health remains a landmark achievement, highlighting the effectiveness of vaccination, surveillance, and strong international cooperation in controlling infectious diseases.

References

Libeau, G., Diallo, A., Colas, F. and Guerre, L. (1994) 'Rapid differential diagnosis of Rinderpest and Peste des petits ruminants using an immunocapture ELISA', *Veterinary Record*, 134, pp. 300–304.

Radostits, O.M. et al. (2007) *Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs and goats*. 10th edn. London: Saunders Elsevier.

Reid, H.W., 1981. Rinderpest. In *Diseases of Cattle in the Tropics: Economic and Zoonotic Relevance* (pp. 133-152). Dordrecht: Springer Netherlands.

Yamanouchi, K., 2012. Scientific background to the global eradication of rinderpest. *Veterinary immunology and immunopathology*, *148*(1-2), pp.12-15.

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