Rinderpest (Cattle Plague)

Rinderpest was the first animal disease to be globally eradicated. Because it was such a scourge and re-emergence remains a possibility, it is vital to maintain current information. Rinderpest was a viral disease of cattle and other ruminants (domestic and wild) characterized by fever, erosive stomatitis, diarrhea, and high morbidity and mortality. In the post-eradication era, testing for rinderpest, preferably using molecular methods, should be considered when an etiologic agent cannot be determined for an infectious disease with characteristic signs of rinderpest.

Historically, rinderpest virus was a scourge that wrought economic havoc throughout Africa, Asia, and Europe. The need to combat rinderpest provided the impetus for the establishment of the first modern veterinary school in Lyon (France) in 1762. After several decades of success in eradicating rinderpest from Europe, the disease recurred unexpectedly in Belgium in 1920, and renewed efforts to eradicate it resulted in the creation of the World Organization for Animal Health (OIE) in 1924. After the creation of the Food and Agriculture Organization (FAO) of the United Nations in 1946, the OIE and FAO signed a cooperation agreement in 1952. Thereafter, the two organizations (FAO and OIE) were major participants in several worldwide campaigns to combat rinderpest, which culminated in global eradication of the disease in 2011. In fact, the last reported rinderpest outbreak occurred in Kenya in 2001, but a 10-year active surveillance period was necessary before global eradication could be declared. Rinderpest is only the second viral disease, after smallpox, to have been successfully eradicated worldwide.

The successful eradication of rinderpest shows that smallpox eradication in 1980 was not an unrepeatable feat and should provide a certain degree of confidence to the international community that concerted, science-based efforts can result in future successes.

Rinderpest virus is biologically similar to the virus of peste des petits ruminants (PPR), which has been targeted by the OIE and FAO as the next animal disease for global eradication.

Rinderpest was a disease of cloven-hoofed animals characterized by fever, necrotic stomatitis, gastroenteritis, lymphoid necrosis, and high mortality. In epidemic form, it was the most lethal plague known in cattle. All wild and domesticated species of the order Artiodactyla were variably susceptible to rinderpest, although dissemination of the virus largely depended on continual transmission among domesticated cattle, buffalo, and yaks. The virus also infected goats and sheep, leading to underdiagnosis of the clinically similar peste des petits ruminants in regions where the two diseases coexisted.

Etiology, Epidemiology, and Transmission of Rinderpest

Rinderpest virus is a *Morbillivirus*, closely related to the viruses causing peste des petits ruminants, canine distemper, and measles. Strains of varying virulence for cattle occurred and could be differentiated genetically. However, a single serotype of the virus existed, and a vaccine prepared from any strain could protect against all strains.

Rinderpest virus is shed in nasal and ocular secretions and can be transmitted during the incubation period (1–2 days before onset of fever). Transmission required direct or close indirect contact between susceptible animals and sick animals shedding the virus. The role of fomites in transmission was negligible, because the virus is fragile, being inactivated within 12 hours of exposure to atmospheric heat and light. There was no carrier state, and recovered animals acquired lifelong immunity. In endemic areas, young cattle became infected after maternal immunity disappeared and before vaccinal immunity began, with possible auxiliary cycles in wild ungulates.

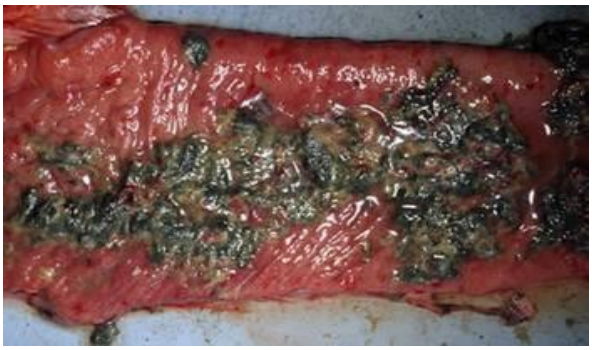
Clinical Findings of Rinderpest

After an incubation period of 3–15 days, fever, anorexia, depression, and oculonasal discharges developed, followed by necrotic lesions on the gums, buccal mucosa, and tongue. The hard and soft palates were often affected. The oculonasal discharge became mucopurulent, and the muzzle appeared dry and cracked.

Diarrhea, the final clinical sign, could be watery and bloody. Convalescence was prolonged and could be complicated by concurrent infections due to immunosuppression. Morbidity was often 100% and mortality was up to 90% in epidemic areas, but in endemic areas morbidity was low and clinical signs were often mild.

Lesions

Rinderpest, necrosis and fibronecrotic exudate



Gross pathologic lesions occurred throughout the GI and upper respiratory tracts, either as areas of necrosis and erosion, or congestion and hemorrhage, the latter creating classic “zebra-striping” in the rectum.

Lymph nodes could be enlarged and edematous, with white necrotic foci in the Peyer’s patches. Histologic lesions included lymphoid and epithelial necrosis with viral-induced syncytia, and intracytoplasmic and intranuclear inclusions were often seen.

Diagnosis of Rinderpest

- It is recommended that post-eradication laboratory diagnosis of rinderpest focus on molecular techniques (such as RT-PCR), which are not only accurate but also allow for phylogenetic analysis to pinpoint the source of any re-emerging virus strain.

Clinical and pathologic findings were sufficient for diagnosis of rinderpest in endemic areas and after initial laboratory confirmation of an outbreak. In areas where rinderpest was uncommon or absent, laboratory tests had to be used to differentiate it from bovine viral diarrhea in particular, as well as East Coast fever,

foot-and-mouth disease, infectious bovine rhinotracheitis, and malignant catarrhal fever. Virus isolation and detection of specific viral antigens in affected tissues using an immunodiffusion test was the standard, but simpler, more rapid and more discriminating tests, such as antigen-capture ELISA and reverse transcription PCR (RT-PCR), were favored toward the end of the eradication campaign. The RT-PCR technique allowed phylogenetic characterization of the virus and helped trace the origin of strains in new outbreaks. A simple lateral flow pen-side test for field use also proved useful in the final stages of the eradication campaign. In the 10-year period between occurrence of the last outbreak and the official declaration of eradication, active rinderpest surveillance in recent endemic areas included the testing of all susceptible cloven-hoofed animals presenting with erosive stomatitis.

Control of Rinderpest

Active immunity to rinderpest was lifelong, whereas maternal immunity lasted 6–11 months. Control in endemic areas was by immunization of all cattle and domestic buffalo > 1 year old with an attenuated cell culture vaccine. In these areas, outbreaks were controlled by quarantine and “ring vaccination” and sometimes by slaughtering. In epidemics, the disease was best eliminated by imposing quarantine and by slaughtering affected and exposed animals. Control of animal movements was paramount to control rinderpest; many outbreaks were due to the introduction of infected cattle to hitherto uninfected herds. The lessons learned from this huge success will be instrumental in the fight against peste des petits ruminants.