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Rinderpest

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Definition

Rinderpest is usually described as an acute, febrile, virus disease of ruminants, especially cattle and water buffalo, characterized by inflammation, hemorrhage, necrosis and erosion of the digestive tract accompanied by a wasting, frequently bloody diarrhea and a mortality approaching 90%. This is an excellent definition when fully virulent strains of the virus infect completely susceptible cattle populations. However in countries where rinderpest is enzootic and particularly where vaccination has been carried out for many years, a much different picture evolves. In these countries it is quite rare to see the fulminating form of the disease. Instead rinderpest takes on a smouldering, less acute characteristic with milder lesions and much lower mortality. Naturally attenuated strains of the virus are sometimes responsible for the milder forms of the disease. These milder forms of rinderpest complicate field diagnosis. It should also be kept in mind that all ruminant game animals are probably susceptible to rinderpest as are Asiatic domestic pigs and African wart hogs. European type domestic pigs are also susceptible but the disease frequently is subclinical. The American peccary and the white tailed deer were shown to be susceptible at the Plum Island Animal Disease Center.

History and Current Status

Rinderpest, also known as cattle plague, probably has caused more losses in cattle and water buffalo than any other single disease in the history of livestock production. It has been compared to the vast bubonic plague epidemics that decimated human populations until the 19th century.[19] Starvation and suffering of large human populations followed the enormous cattle losses in affected countries. Deaths of large numbers of cattle and water buffalo not only caused marked decreases in meat and milk but also resulted in a decrease in work oxen which were, and still are in many developing countries, the main source of farm power.

Accounts of cattle plague go back as far as the seige of Troy in 1184 B.C. and the disease has often been associated with the ravages of war and the movements of armies since that time. Charlemagne is said to have introduced cattle plague into France when he returned with his armies in 810 A.D. During that same year the scourge appeared in Britian. From the 9th century to the times of Napoleon, epizootics of rinderpest swept over Europe about every 50 years, each time severely reducing the cattle populations in the countries affected. The latest outbreak of rinderpest in Britain occurred in 1865, when infection was imported from Russia across the Baltic Sea. The last panzootic of rinderpest in Western Europe occurred in 1870, after the Franco-Prussian War.

However in 1913, during the Balkan War, the infection was introduced into several Balkan countries from Turkey and during World War I parts of Russia were infected. After the war, the disease spread to Poland where it persisted for several years.

The first Veterinary College was established in 1762 by Bourgelat at Lyon, France, as a result of the serious losses from the rinderpest epizootic that terminated in 1750.[6]

The only recent outbreak of rinderpest in Western Europe occurred in Belgium in 1920, when infected cattle from India

were introduced to the port of Antwerp. The disease spread to over 200 premises but was rapidly eradicated by the veterinary authorities.

Fortunately the only occurrence of rinderpest in the Americas was a limited outbreak in Brazil in 1921, probably from zebu cattle imported from India.

A single outbreak of rinderpest also occurred in Australia in 1923, on a dairy farm near Perth. As soon as the outbreak was diagnosed, all cattle, sheep, goats, and pigs within a 1-mile radius of the infection were slaughtered and rigid quarantine was imposed in an area about 30 miles around the infected farm. These measures proved effective and the disease was completely eradicated within 2 months. Nevertheless, the outbreaks in Australia and Brazil serve to remind us that as long as rinderpest exists anywhere in the world, we must remain constantly on the alert to prevent its introduction into the United States.

Currently, rinderpest occurs in tropical Asia and Africa, in the Middle East, Pakistan, India, Burma and Southeast Asia. Little detailed information is available concerning the status of the disease in China.[2] A report of a visit to China by two American veterinarians in 1974, stated that rinderpest was eradicated there in 1955.[1] The most recent epizootic in the Middle East occurred in 1969. Turkey became reinfected in 1970, after an interval of 38 years during which rinderpest was not present in that country.

It has been estimated that prior to 1949, there were some two million deaths from rinderpest each year throughout the world.[19] Since then there has been a dramatic decrease in these losses mainly due to the use of the excellent vaccines now available. In India alone, 200 million cattle were immunized between 1955 and 1967. However, in order to eradicate rinderpest, herd immunity levels must be maintained. This involves annual vaccination of the calf crop and in India there are 25 milion calves produced each year. To maintain the immunity level in this situation is a formidable task.

Etiology

The cause of cattle plague was the subject of great controversy during the early history of the disease. It was frequently confused with cholera, typhus, dysentery and smallpox in man, and foot-and-mouth disease in cattle. That rinderpest was an infectious disease was established only in 1754, when susceptible animals were infected by placing bits of material, previously dipped in morbid discharges, into an incision made in the dewlap.[6] However, it was not until 1899 that rinderpest was proven to be a viral disease when susceptible cattle were infected with bacteria-free filtrates.

Rinderpest virus has been classified as a myxovirus from 120 to 300 millimicrons in size.[15] The essential component of the virus core is ribonucleic acid. Most rinderpest virus particles are spherical in shape. It is a relatively fragile virus and in fluid supernate is not infectious after exposure to 60°C for 5 minutes. However, the virus remains viable for months in the deepfreeze,[19] and freeze dried virus will remain viable for years in the deepfreeze. Strong alkalies, acids and the common disinfectants will destroy the virus. Sunlight is lethal and reconstituted live virus vaccines must be protected from direct sunlight. This can be done by placing the reconstituted vaccines in brown bottles which filter out the ultraviolet rays from the sun. Fortunately there is only one immunological type of rinderpest virus in contrast to foot-and-mouth disease with its 7 types and many subtypes. This means that suitable vaccines will protect cattle against all known strains of rinderpest virus. Although there is only one immunological type, field strains of the virus may vary widely in virulence, ease of transmission and host affinity.

Rinderpest virus is related antigenically to the viruses of human measles and canine distemper, summarized as follows:

1. Distemper and measles antisera neutralize rinderpest virus in vitro.[18],[12]
2. Rinderpest antiserum neutralizes both distemper and measles virus in vitro.[7]
3. Monkeys inoculated with rinderpest virus develop antibodies to rinderpest, canine distemper and occasionally to measles.[3]
4. Dogs inoculated with measles or rinderpest virus develop low levels of antibodies to measles and to rinderpest but not to canine distemper. Nevertheless these same dogs resist challenge to canine distemper virus.[3],[17]
5. Cattle inoculated with canine distemper virus develop distemper antibodies but not rinderpest antibodies and when challenged with rinderpest virus usually are susceptible. A few cattle given massive doses of distemper virus were later immune to rinderpest.[3]
6. Cattle inoculated with measles virus did not develop antibodies to measles, rinderpest or canine distemper, and were later susceptible when challenged with rinderpest virus.[3],[12] (As indicated above, the antigenic relationship of rinderpest, measles and distemper is not complete and findings of *in vitro* serological tests were not always confirmed by *in vivo* biological tests.)

The discovery of the relationship of these three viruses in an outstanding example of scientific observation both in the field and in the laboratory. For example, the relationship of rinderpest and distemper was first suspected when it was observed that dogs in the vicinity of the Veterinary Laboratory at Kabete, Kenya, did not contract distemper. These dogs had been fed meat from the post-mortem room where rinderpest-infected cattle were necropsied. Apparently this exposure to rinderpest infected meat was sufficient to render the dogs resistant to distemper.

Clinical Features

Textbook descriptions of rinderpest usually emphasize the peracute, highly fatal aspects of the disease. This form of the disease usually occurs when highly virulent virus is first introduced into a completely susceptible cattle population.

Only recently has emphasis been placed on the less dramatic forms of the disease. The incidence of these forms is related to the innate resistance of the infected stock and the virulence of the virus involved.

I saw my first case of rinderpest in the Northern Frontier District of Kenya in 1952. This was toward the end of a relatively mild outbreak in an area where many of the cattle had been vaccinated. We had difficulty in finding a sick animal. This was a 3-year old zebu showing a temperature of 103°F, slight diarrhea and not much else. After a great deal of haggling, we purchased the animal from the owner, probably paying 3 times its value. I was very skeptical that the animal really had rinderpest but we later isolated virus from specimens we took to the laboratory. This convinced me that rinderpest need not be the peracute infection I had been led to believe by reading the literature on the disease.

The incubation period following natural exposure to rinderpest ranges from 3 to 15 days which further emphasizes the variations that may occur. Typically the first sign of rinderpest is a rise in temperature to 104°F or above. However, the first sign can be missed except in dairy cows where milk yield falls almost simultaneously with the initial temperature rise. Next there is a general restlessness accompanied by loss of appetite, cessation of rumination and rapid breathing.

Clear nasal and lacrimal discharges appear 24-48 hours after the initial temperature rise. Visible mucous membranes become congested and the animal is often constipated.

After 2 or 3 days, the temperature falls with the onset of severe diarrhea which may be the first sign observed by the owner of grazing cattle. Leukopenia begins at this time, involving mainly lymphocytes followed by a marked shift to a predominance of immature neutrophils.[10,19] Nasal and lacrimal discharges become purulent. The muzzle is dry and lesions begin in the mouth and nose as grey, necrotic, pinhead size areas that later coalesce and erode with raw red areas underneath.

Very rarely at about this time the animal goes berserk, becomes aggressive and charges anything in its path. I saw one such case in Kenya which we had to shoot for the safety of the personnel at the laboratory. Most of the time the animal becomes very depressed at the height of the febrile reaction.

Severe straining accompanies the diarrhea which is foetid and often blood stained. The rectal mucosa becomes congested and eroded. Dehydration in acute cases is severe and spectacular. Death usually occurs in 6-12 days after the onset of illness and the animal is prostrate and emaciated. Surviving animals go through a long period of convalescence.

Peracute cases may die 2 to 3 days after the onset of the fever and may show no other signs or lesions.

In milder forms of rinderpest, many signs such as fever, inappetence, nasal and lacrimal discharges, depression, and diarrhea are often transient or absent or so mild as to be missed and recovery is the rule.[19] The skin form of rinderpest was reported to be common in the 18th and 19th centuries and the lesions were compared to those of human smallpox. Today the occurrence of such skin lesions in cattle is rare although they are reported in Thailand buffalo.

Post-Mortem Findings

As with clinical features, the post-mortem lesions vary considerably with the innate resistance of cattle and the virulence of the virus. Rinderpest virus has an affinity for lymphoid and epithelial tissues and the lesions reflect this.

Lymph nodes are generally swollen, edematous and congested. The Peyer's patches are swollen, hemorrhagic and necrotic. In some instances the entire patch may slough out leaving a raw crater. The lymphatic tissue at the cecocolic junction is similarly and invariably involved. Microscopically there is massive destruction of lymphoid follicles and disappearance of mature lymphocytes which are partially replaced by plasma cells, macrophages occasional neutrophils, and an eosinophilic, acellular matrix.[10]

The gastrontestinal tract shows the effect of epithelial involvement. Early lesions occur in the mouth as grey, necrotic, pinhead size areas which coalesce and erode leaving raw, red floors. The first appearance of these lesions involves the inner surface of the lower lip, the adjacent gum, cheeks and the anterior ventral surface of the tongue. These lesions are nonvesicular. Oral lesions may extend to the hard palate, pharynx and upper portions of the esophagus. The initial microscopic lesions in the oral epithelium include syncytical cell formation and necrosis of cells just above the basal layer. The rumen and reticulum rarely show lesions but the omasum may be eroded and hemorrhagic.

The abomasum is frequently severely congested and edematous. The pyloric region is one of the most consistently involved areas of the digestive tract with erosions leaving a raw bleeding surface.

Lesions in the small intestines are generally mild except for the Peyer's patches.

The large intestine is often severely and spectacularly affected particularly in the caecum, the cecocolic junction, and the rectum. Hemorrhage and erosions occur along the crests of the longitudinal folds of the mucosa of the large intestines where they form parallel streaks often called zebra or tiger striping.

Diagnosis

Accurate and rapid diagnosis is essential to control and eradication and is the most important consideration for rinderpest and other exotic diseases as far as the United States is concerned.

The first line of defense, next to preventing entry, is in the field. Cooperation of cattlemen, practicing veterinarians and State and Federal veterinary officials would be absolutely essential in order to make an early presumptive

diagnosis of rinderpest which could then be rapidly confirmed at the Plum Island Animal Disease Center. Unfortunately, the clinical and gross pathological features of rinderpest alone cannot be used to make a positive diagnosis. Other diseases, particularly the virus diarrhea-mucosal disease complex present features indistinguishable from rinderpest. Thus when rinderpest is suspected in the field, laboratory tests must be carried out immediately. Delay could be disastrous.

The above is not meant to imply that every case of mucosal disease should be submitted to Plum Island as this would overburden facilities there. This decision should be left to the State and Federal authorities who would consider all factors involved with particular attention to recent animal movements.

Fortunately there are excellent laboratory procedures for rinderpest diagnosis.[19] Isolation and identification of the virus in tissue cultures of calf kidney cells appears to be the most reliable method. The following points should be kept in mind:

1. The optimal period for collecting samples for isolating rinderpest virus is during the febrile stage. Animals that have been sick for several days and dead animals are poor subjects for sampling.
2. Suitable early cases should be bled and then slaughtered for collection of lymph nodes, spleen and tonsil samples.
3. Blood buffy coat (white blood cells) is the preferred source of cirus for isolation in tissue culture. Defibrinated blood should be forwarded to the laboratory on ice but care should be taken not to freeze it. Spleen suspensions can also be used to isolate virus in tissue culture. Lymph nodes may be toxic to tissue culture but should be frozen and sent to the laboratory for possible isolation in susceptible cattle. Blood from these cattle can then be used for tissue culture isolation.
4. Cytopathic changes are evident in 3-12 days if rinderpest virus is present.
5. The specificity of the cytopathic effect is demonstrated by the absence of cytopathic changes in cultures containing immune serum.
6. Other methods of laboratory diagnosis are agar gel diffusion, complement-fixation, and hemagglutination inhibition of measles virus in monkey erythrocytes.

In addition to virus diarrhea-mucosal disease complex there is a long list of other diseases that might be confused clinically with rinderpest including malignant catarrh, bovine petechial fever, East Coast fever, trypanosomiasis, Johnes disease, foot-and-mouth disease, hemorrhatic septicemia and coccidiosis.

Epizootiology

Epizootiogically, rinderpest is a relatively straightforward disease. Transmission is usually between sick and healthy animals through inhalation of virus-laden aerosols and the movement of infected animals is primarily responsible for the spread of the disease. Nevertheless, some highly invasive strains of the virus appear to spread as readily by indirect contact as does foot-and-mouth disease.

Fortunately, from the standpoint of control, persistent or chronic carriers of rinderpest have not been identified. Experimentally virus could not be isolated from cattle beyond 16 days after initial exposure. French and British workers in Africa have demonstrated that some vaccinated cattle with low antibody levels may become temporarily reinfected in the nasal mucosa for periods up to 14 days. These temporary reinfections are clinically inapparent and are only important if the cattle happen to come in contact with susceptible cattle. The possibility of transient reinfections in vaccinated cattle has been acknowledged in the import restrictions of some European countries.

Experimentally, European-type domestic swine can be infected through ingestion of infected meat.[20] Clinically these infections are usually mild or inapparent. These swine can then transmit rinderpest to in-contact cattle. Thus it is possible, but not probable, that domestic swine could act as they do in foot-and-mouth disease as the introduction of rinderpest to a country previously free from the disease. Some breeds of Asiatic pigs are highly susceptible and are a major factor in the epizootiology of the disease where they exist in rinderpest areas.

Rinderpest in sheep and goats has on a few occasions caused severe losses but is not generally considered important in the transmission to cattle. However, a few years back in Sudan, a strain of rinderpest virus from sheep was highly invasive for cattle. This outbreak was controlled by vaccinating the sheep, goats and cattle in the areas with tissue culture attenuated vaccine. In West Africa, a disease called by the French "peste des petit ruminants" which affects sheep and goats is apparently caused by a strain of rinderpest virus which is virulent for sheep and goats but is naturally attenuated for cattle. Although sheep and goats must be considered in the epizootiology of rinderpest the significance of these animals in the perpetuation of the disease is probably not a major factor.

In enzootic areas rinderpest is usually confined to weaned immature animals because adult cattle are immune from vacciantion or survival from outbreaks. Calves up to 10 months old which are nursing immune mothers may be protected by colostral antibodies. Rinderpest in the immature animals, which are in the process of losing their colostral immunity, is often subclinical or mild and the mortality is low. Nevertheless, these animals serve to perpetuate the infection. They further complicate the picture because even low levels of colostral antibody may block the response to attenuated live virus vaccines. It is often stated that an 80 percent immunity level in a population is sufficient to prevent an epidemic or an enzootic. In this regard rinderpest is similar to human measles. Even if 100 percent of adults are immune, the disease will persist if a high percentage of immature or juveniles are susceptible.

There is a marked difference in the innate resistance of different cattle populations to rinderpest. The reasons for

this have not been fully explored. Native breeds in enzootic areas are generally more resistant than imported breeds. This is probably because of their long association with the disease and "survival of the fittest." Nevertheless, a few native breeds like the Ankole of E. Africa and the N'dama of W. Africa remain highly susceptible. Japanese black cattle are also highly susceptible.

The wide variation of susceptibility is of practical importance when selecting an attenuated live virus vaccine. Goat attenuated (caprinized) vaccine will kill a high percentage of Ankole and N'dama cattle. Even the more attenuated lapinized vaccine may kill up to 40 percent of Japanese black and Korean cattle.

Control and Eradication

I will deal with control and eradication of rinderpest in the event of its introduction into countries like the U.S. where the disease has not previously existed.

One cannot overemphasize the importance of prompt diagnosis. We cannot deal satisfactorily with a new disease problem until we know the cause and the extent of the disease.

If there is any doubt in the field about a suspect disease being rinderpest the disease should be dealt with as rinderpest until laboratory tests are carried out. At this stage strict quarantine should be sufficient. Control of cattle movements is highly effective in stopping the spread of rinderpest.

If the laboratory tests are positive for rinderpest a "stamping out" program should be initiated immediately. All sick and in-contact animals should be slaughtered and disposed of as in foot-and-mouth disease. Some authorities might argue that "stamping out" methods are overly drastic for rinderpest but my contention is that in a limited outbreak in the U.S., this would be the quickest, safest and least expensive method. **There is little doubt that all ruminant game animals in the U.S. are susceptible to rinderpest.** The American peccary is also susceptible. The introduction of rinderpest into our highly susceptible game population would undoubtedly cause heavy losses in these animals and would certainly greatly complicate control and eradication in cattle. In the case of widespread, multiple foci outbreaks of rinderpest in the U.S. with wide game animal involvement, it would probably be necessary to resort to vaccination. In the case of rinderpest this is a highly effective alternative. Tissue culture attenuated rinderpest vaccine is one of the safest and most effective immunizing agents ever devised.

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