In today's presentation we will cover information regarding the organism that causes Rinderpest and its epidemiology. We will also talk about the economic impact the disease has had in the past and could have in the future. Additionally, we will talk about how it is transmitted, the species it affects, clinical and necropsy signs seen, and diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease as well as actions to take if Rinderpest is suspected.

Rinderpest virus (RPV) is a single-stranded RNA virus in the family Paramyxoviridae, genus Morbillivirus. It is very similar to peste des petits ruminants virus, canine distemper virus, human measles virus, and marine mammal morbilliviruses. There is only one serotype of rinderpest virus, but field strains vary widely in virulence, ease of transmission, and host affinity. Photo (Electron photomicrograph of the riderpest virus) from The Big Picture Book of Viruses: Paramyxoviridae accessed at www.virology.net/Big_Virology/EM/rpv1.JPG.
The word “rinderpest” is German for pestilence or plague of cattle. Accounts of rinderpest virus or cattle plague date back to the siege of Troy in 1184 BC. Since that time RPV has been associated with war and movement of armies. RPV was responsible for the establishment of the first veterinary school in 1762 in Lyon, France. Even today war is a factor in its spread as in the war with Iraq in the early 1990’s RPV entered Turkey when refugees from Iraq brought infected cattle with them. 1885 marked the “Great African Pandemic” which killed 80-90% of the wild ruminants, 2.5 million cattle died in South Africa alone. In the 1960’s RPV was eradicated from most of Europe, China, Russia and the Far East. 1992 saw the establishment of the Global Rinderpest Eradication Program (GREP) by the FAO.

Outbreaks of rinderpest virus can have devastating economic effects. Rinderpest is highly contagious and can destroy entire populations of cattle and buffalo. Outbreaks can lead to famine in areas where cattle are depended upon for meat, milk and draft power. An epidemic in sub-Saharan Africa in the 1980s wiped out most of the cattle. A 1982-1984 outbreak in Africa caused an estimated $500 million as a result of livestock losses and control measures. It is estimated that $100 million is spent annually world-wide for vaccination. (Photo: www.fao.org)

Rinderpest is a serious disease mainly of cattle and domestic buffalo, including water buffalo. Most wild and domestic cloven-footed animals can become infected including zebu, sheep and goats, pigs (Asian pigs appear to be more susceptible than African or European pigs) and wild ungulates including African buffalo, elands, kudus, wildebeests, antelopes, bushpigs, warthogs, giraffes, hippopotamuses. In Africa 13 species of game animal are naturally infected with rinderpest virus and 6 more species can be infected experimentally. Buffalo and wildebeest are the greatest RP spreaders, but without reinfection from cattle rinderpest would probably die out in wild game. (Photos: www.fao.org)

This series of maps from the Global Rinderpest Eradication Program show how well the program has been working at eradicating Rinderpest since the 1980’s. The map in the upper left shows Rinderpest endemic areas in the 1980’s, the map in the upper right is for the 1990’s and the lower center map shows the remaining foci of Rinderpest in the 2000’s. GREP is designed to respond to and address all rinderpest outbreaks in order to reach the goal of complete eradication by the year 2010. Their goals and challenges are to eliminate the last foci of virus persistence, remove doubt about rinderpest persistence, persuade uncommitted countries to endorse GREP, strengthen rinderpest surveillance and emergency preparedness and to ensure cessation of unnecessary mass vaccination.
In most cases the prognosis for Rinderpest is poor. This is especially true where it does not occur and the populations of animals are immunologically naïve. Under these conditions mortality can reach 100%. Animals that recover are immune for life. In endemic areas, newborn animals are protected from 6-11 months of age by maternal antibodies, so the most susceptible are immature or young adult animals. (Photo of calf: P.Roeder at fao.org; historical photo of RPV)

Rinderpest virus is mainly transmitted by direct or close contact with infected animals. Virus is shed in nasal and ocular secretions and in feces, urine, saliva and blood. To a lesser degree contaminated food or water can transmit RPV as well as fomites. (Photos: fao.org)

RPV can be transmitted by aerosol only for very short distances. The most infectious period is 1 to 2 days before the onset of clinical signs and then up to 8 or 9 days after onset of clinical signs. Transmission via arthropod vectors is not known to occur. No chronic carrier state exists and rinderpest virus does not persist in wild populations without the presence of susceptible cattle. Photo: USAID
Clinical Signs

• Incubation period
  - 3-15 days, usually 4-5 days

• Four forms of disease
  - Classic, Peracute, Subacute, Atypical

The incubation period as well as clinical disease varies with the strain of virus, dosage, and route of exposure. Following natural exposure, the incubation period ranges from 3 to 15 days but is usually 4 to 5 days. Clinically, RPV can occur in four different forms: the classical form, the peracute form, the subacute form, and the atypical form. (Photo: Newsletter of the Tropical Medicine Association)

Clinical Signs

• Classic form
  - Fever, depression, anorexia
  - Constipation followed by hemorrhagic diarrhea
  - Serous to mucopurulent nasal/ocular discharge
  - Necrosis and erosion of the oral mucosa
  - Enlarged lymph nodes
  - Death in 6-12 days

The classical form of Rinderpest virus is most common and consists of fever, constipation followed by watery hemorrhagic diarrhea; serous to mucopululent nasal and/or ocular discharge, necrotic oral erosions, enlarged lymph nodes, dehydration and death in 6-12 days. Photo of mouth: http://www.vetmed.ucdavis.edu/vetext/INF-DA/INF-DA_Rinderpest.html

Clinical Signs

• Peracute
  - Young animals, high fever with congested mucous membranes, death in 2-3 days

• Subacute
  - Mild clinical signs with low mortality

• Atypical
  - Irregular fever, mild or no diarrhea
  - Immunosuppression leading to secondary infections

Peracute cases usually occur in young animals that show a high fever, congested mucous membranes resulting in death in 2-3 days. The subacute form of Rinderpest virus shows mild clinical signs combined with low mortality rates. The atypical form is characterised by irregular pyrexia and mild or no diarrhea. Immunosuppression due to the virus’s lymphotropic tendency can lead to secondary infections as well as emergence of latent infection.

Post Mortem Lesions

• Esophagus
  - Brown and necrotic foci

• Omasum
  - Rare erosions and hemorrhage

• Small intestine, abomasum, cecum and colon
  - Necrosis, edema and congestion
  - “Tiger striping”

Brown necrotic or eroded areas are found in the esophagus. Rare erosions and hemorrhage are found in the omasum. The abomasum shows signs of congestion and edema. The small intestine, cecum and colon generally have signs of obvious necrosis and edema and erosions. Colonic ridges may be congested, this is referred to as “tiger striping”. Tiger striping can occur in other diarrheas and probably results from tenesmus. Top photo of intestine: http://www.vetmed.ucdavis.edu/vetext/INF-DA/INF-DA_Rinderpest.html lower photo: USDA)

Post Mortem Lesions

• Lymph nodes
  - Swollen and edematous

• Gall Bladder
  - Hemorrhagic mucosa

• Lungs
  - Emphysema, congestion and areas of pneumonia

The lymph nodes are generally swollen and edematous. The gall bladder may show petechial to ecchymotic hemorrhages. Lungs may show emphysema, congestion and signs of pneumonia. (Photo: Hemorrhagic mucosa of gall bladder from the Gray Book)
**Differential Diagnosis**
- Infectious bovine rhinotracheitis
- Bovine viral diarrhea
- Malignant catarrhal fever
- Foot and mouth disease
- Bluetongue
- Salmonellosis
- Paratuberculosis
- Peste des petits ruminants

Other diseases somewhat similar to rinderpest are infectious bovine rhinotracheitis, bovine viral diarrhea, malignant catarrhal fever, foot and mouth disease, bluetongue, salmonellosis, perhaps Johne’s Disease and in goats and sheep, peste des petits ruminants. (Photo: www.fao.org)

**Sampling**
- Before collecting or sending any samples, the proper authorities should be contacted
- Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

**Diagnosis**
- Clinical
  - Rapidly spreading acute febrile illness in all ages of animals
  - Accompanying clinical signs consistent with RPV
- Laboratory Tests
  - Isolation and confirmation of virus

Clinical diagnosis can be made if a disease with the appropriate clinical signs is recognized in all age groups of a herd. An entire herd may become infected in less than a month, which is in contrast to most BVD outbreaks in the US where only individual animals are sick at one time. Laboratory diagnosis of RPV in non-endemic regions requires isolation and confirmation of the virus.

**Diagnosis**
- Samples to Collect
  - Live animals
    - Viremia drops when fever falls and diarrhea begins
    - Blood sample
    - Swabs of lacrimal fluid
    - Necrotic tissue of oral cavity
    - Aspirations of superficial lymph nodes
  - Dead animals
    - Spleen, lymph node, tonsil

Samples that should be collected in live animals must be taken while the animal is in the febrile phase of illness because this is when viremia is at its peak level. Blood should be collected and stored in heparin or EDTA for serum analysis. Swabs of lacrimal fluid may be collected, as well as necrotic tissue of the oral cavity and aspirations of superficial lymph nodes. In dead animals, collect tissues from the spleen, lymph nodes and tonsils.

**Treatment**
- No known treatment
- Diagnosis usually means slaughter of effected animals
- Supportive care with antibiotics in rare cases of valuable animals
- Preventative measures are key

There is no known treatment for Rinderpest virus infection, this, combined with the high morbidity rates, accounts for the devastating nature of the disease. A diagnosis of RPV usually means slaughter of the affected animals and significant economic loss. In rare cases, supportive care and antibiotic therapy can help in the treatment of especially valuable animals. Because of the lack of effective treatment, preventative measures are of key importance.
Public Health Significance

- Rinderpest virus does not cause disease in humans

Prevention and Control

Recommended Actions

- Notification of Authorities
  - Federal:  
    Area Veterinarian in Charge (AVIC)  
    www.aphis.usda.gov/vs/area_offices.htm  
    State veterinarian  
    www.aphis.usda.gov/vs/sregs/official.htm  
- Quarantine

Disinfection

- Chemical  
  - Glycerol and lipid solvents
- Natural  
  - pH 2 and 12  
  - For at least 10 minutes  
  - Optimal survival for the virus is at pH 6.5-7

Vaccination

- Most commonly used vaccines  
  - Cell-culture-adapted  
  - Colostral immunity interferes with vaccination  
    - Vaccinate calves annually for 3 years  
    - Heat stability of vaccine an issue

Rinderpest virus is not known to cause disease in humans.

If RPV is suspected authorities should be contacted immediately. The State Veterinarian and Federal Area Veterinarian in Charge for each specific area can be found at the above web site. If an outbreak occurs, the area should be quarantined.

Rinderpest virus is rapidly inactivated at pH 2 and 12 (10 minutes); optimal for survival is a pH of 6.5-7. The virus is inactivated by glycerol and lipid solvents. Iodophore and chlorine dioxide disinfectants are particularly effective against the virus.

The most commonly used vaccine is the cell-culture-adapted vaccines. This is a safe vaccine for many species and produces life-long immunity in cattle (animals challenge-inoculated 7 years after vaccination were protected). In endemic areas where cattle have been vaccinated, colostral immunity will interfere with the vaccination of calves up to 11 to 12 months of age. Because the duration of colostral immunity is variable, the recommendation is to vaccinate calves annually for 3 years. One of the biggest problems with the cell-culture-adapted vaccine has been stability. It must be kept cold until used and many sites where vaccination must occur are very remote, making refrigeration difficult. Researchers at Plum Island in the early 1990's greatly increased the stability of the vaccine by modifying the stabilizers and lyophilization process. This change in production is now being used in some production facilities in Africa.
In endemic areas vaccination is often performed annually at the national herd level. When spot epidemics arise slaughter is coupled with ring vaccination. Wildlife, sheep, and goats should be monitored serologically. Serological monitoring of sheep and goats could be complicated by using Rinderpest vaccine to protect goats against peste des petits ruminants. High-risk countries (those trading with, or geographically close to, infected countries) can protect themselves by having all susceptible animals vaccinated before they enter the country or vaccinating the national herd, or both. Rinderpest free areas must place import restrictions on susceptible animals and uncooked meat products from infected areas.

Additional Resources

Internet Resources

- World Organization for Animal Health (OIE) website
  - www.oie.int
- USAHA Foreign Animal Diseases – “The Gray Book”
  - www.vet.uga.edu/vpp/gray_book
- Food and Agriculture Organization of the United Nations
  - www.fao.org

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