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Rinderpest In Cattle

A Graduation Project Submitted to the Department Council of the Internal and Preventive Medicine-College of Veterinary Medicine/ University of Al-Qadisiyah in a partial fulfillment of the requirements for the Degree of Bachelor of Science in Veterinary Medicine and Surgery.

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فَتَعَٰلَى ٱللَّهُ ٱلْمَلِكُ ٱلْحَقُّ ۗ وَلَا تَعْجَلْ بِٱلْقُرْءَانِ مِن قَبْلِ أَن يُقْضَىٰٓ
إِلَيْكَ وَحْيُهُۥ ۖ وَقُل رَّبِّ زِدْنِى عِلْمًا ﴿١١٤﴾

صَدَقَ اللهُ العَظِيمُ

من سورة طه

Certificate of Supervisor

I certify that the project entitled (----------) was prepared by ---------- under my supervision at the College of Veterinary Medicine / University of Al-Qadisiyah.

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Dedication

To… He who illuminated the universe with his light… He alone is glorified and transfigured. We prostrate

Thankful for the blessing.

To … the most honorable of God's creation … the Messenger of God, Muhammad, may God's prayers and peace be upon him.

To… Iraqi blood… which perfumes the beloved soil of Iraq

(our righteous martyrs)

To … those who taught us the path of life … and accompanied us in our path

To…whom we have an asset…to what is dearest to us…(our dear fathers)

To… who gave herself a torch that burns… and embraced us with warmth and tenderness

She lit candles for us on the path…(Our dear mothers).

To … those who have a bond with us (our brothers).

To … those who have enhanced the joy in our face, and your words illuminate … and spread

With joy in our papers, you were the best torch that lit our path…

(Our best teachers).

To … those who were with us in the solution and travel … (our female colleagues and colleagues).

To… everyone who shared our joys and sorrows… our fatigue and our comfort.

To all of them we dedicate this humble effort

Abstract

The principal features of rinderpest virus infection of cattle are described. Following a brief historical introduction, the current classification and physical characteristics of the virion are detailed. The characteristics of epizootic and enzootic occurrences of rinderpest in cattle and the role of other species in the transmission of the disease are elucidated. Clinical signs and pathogenesis are considered with reference to the course of viral replication and excretion. The role of active and passive humoral immunity in protection against the disease and the development of live and killed immunogens capable of inducing protective immunity are discussed. Methods of diagnosing rinderpest and the diseases likely to create differential diagnostic problems are considered. It is concluded that the application of control procedures, which includes systematic vaccination with tissue culture propagated virus, could ensure the global eradication of rinderpest.

Chapter one (Introduction)

Rinderpest (cattle plague) is caused by a morbillivirus of the family Paramyxo- Viridae, which cause diseases affecting mammals, including man. Rinderpest Virus affects mainly ungulates, both wild and domestic, as does peste des Petits ruminants virus to which, with measles virus, it is most closely related. Other related viruses are largely defined by the genera which they were first Associated with but this is proving to be a simplification and host specificity Is still ill-defined: canine distemper virus, which also affects a number of Other carnivore families with epidemics reported in African lions and pinnipeds [Roelke-Parker ME et al. 1996,Packer C. 2008]; phocid distemper virus [Baumgartner W et al. 2003]; cetacean morbillivirus [Kennedy S.2005]; measles in humans [Baumgartner W et al. 2003] and a newly discovered felid morbillivirus [Oshitani H. 2010]. Rinderpest in cattle and buf-Faloes is marked by fever with ocular and nasal discharges and is capable of Causing high morbidity and mortality rates from oral and gastrointestinal Tract ulceration, diarrhoea, dysentery, dehydration, protein loss and immuno-Suppression resulting from lymphocyte depletion. Pathogenesis in wildlife Species can be highly variable, for example, a significant proportion of lesser Kudu (Tragelaphus imberbis imberbis) develop ocular lesions, including corneal Opacity and/or panophthalmitis causing total blindness, often leading to Mortality without evident gastrointestinal lesions or diarrhoea. Rinderpest and measles most probably had their origins in an environment Where cattle and humans were living in close

proximity; probably the cattle Herds of Central or South Asia some 10 000 years ago at the time of domesti-Cation of the wild aurochs. However, in contradiction to this understanding, Molecular clock analysis indicates that the divergence of rinderpest and the Related measles virus might not have occurred until as recently as the eleventh Or twelfth centuries [Oshitani H. 2010].

Chapter two (Structure of the virus)

While the majorty of RPV particles are pleomorphic (Fenner et ah,1993b), the common shape is an enveloped spheroid, 100-300 nm in Diameter. However, few particles are enveloped filamentous virions. The Envelopes are bristled with minute projections, which are the surface Glycoproteins (H and F proteins) representing the virus-specific external Antigens (Ags) (McCulough et al., 1991; Forsyth and Barrett, 1995). The Virus has four other structural proteins that are situated internally and are Associated with the nucleocapsid (N), the phosphprotein (P), the matrix (M) And the virus polymerase or the large (L) protein. Additional viral functional Proteins are the Cand V respectively, are part of the genome internal proteins (Rima, 1983).

The causative agent

The causative agent of RP was one of the first micro-organisms to be Recognized as a filterable virus. The agent is a member of the genus Morbillivirus of the family paramyxoviridae. The genus includes other Major viruses namely; canine distemper (CD) and human measeles (HM). These have been chronicled with RPV for centuries as virulent plagues for Their host species (McCullough et al., 1991; Fenner et al., 1993a; Ismail et al,1994). The fourth terrestrial morbillivirus – peste des petits

ruminants (PPR) Virus – was only recognized in the 1940s and has been regarded as a distinct Member of the genus (Gibbs et ah, 1979). Other members of the Morbillivirus genus infect marine mammals (dolphin morbillivirus, DMV, Phocid distemper vims, PDV, porpoise morbillivirus, PMV). More recently, Morbillivirus Abs were detected in the sera of Atlantic pinniped and many Cetacean species (Duignan et al., 1994). These viruses are antigenically Related (Barrett et al., 1993c).

Serological and molecular biological studies showed closer Relationship of RPV to human measles virus (HMV) than to peste des petits Ruminants virus (PPRV) (Haas and Barrett, 1996) while PDV is more closely Related to CDV (Barrett et al., 1993c; Osterhaus et al., 1995). Albeit these intergenus variations among morbilliviruses, RP shares With them the same physio-chemical and some antigenic properties and Produce similar cytopathic effects (CPE) in cell cultures.

DIAGNOSIS

Classical rinderpest resulting from contact exposure has an incubation period of 3–15 days, dependent on The virus strain and the degree of exposure.

For the purposes of the OIE Terrestrial Animal Health Code, the incubation period for rinderpest is 21 days.

Clinical diagnosis

Clinical signs of rinderpest have not been seen since 2001. A milder form of the disease, with the potential To regain classical characteristics, used to occur in association with endemic situations in East Africa.

Classical acute or epizootic form

- Clinical disease is characterized by an acute febrile attack within which prodromal and erosive Phases can be distinguished
- Prodromal period (time between onset of fever and first appearance of oral lesions) lasts on Average 3 days
    i. affected animals develop a pyrexia of between 40°C and 41.5°C together with partial Anorexia, depression, reduction of rumination, constipation, lowered milk production, Increase of respiratory and cardiac rate, congestion of visible mucosa, slight serous to Mucopurulent ocular and nasal discharges, and drying of the muzzle
- Erosive phase with development of necrotic mouth lesions

i. at height of fever: flecks of necrotic epithelium appear on the lower lip and gum and in rapid Succession may appear on the upper gum and dental pad, on the underside of the tongue, On the cheeks and cheek papillae and on the hard palate; erosions or blunting of the cheek Papillae
ii. necrotic foci can fuse to form larger patches; material works loose giving rise to shallow, Nonhaemorrhagic mucosal erosions

- Gastrointestinal signs appear when the fever drops or about 1–2 days after the onset of mouth Lesions

i. diarrhoea is usually copious and watery at first; later may contain mucus, blood and shreds of Epithelium; accompanied, in severe cases, by tenesmus

- Diarrhoea or dysentery leads to dehydration, abdominal pain, abdominal respiration, and weakness

- In terminal stages of the illness, animals may become recumbent for 24–48 hours prior to death; Death usually occurs 6–12 days following onset of fever
- Deaths will occur but mortality rate will be variable; may be expected to rise as the virus gains Progressive access to large numbers of susceptible animals O depending on the strain of RPV, initial mortality rates may be as low as 10–20% or in the Order of 90% in highly susceptible animals. Animals in areas with endemic peste des petits Ruminants (PPR) may show resistance to RPV and lower mortality
- Some animals die while showing severe necrotic lesions, high fever and diarrhoea, others after a Sharp fall in body temperature, often to subnormal values
- In rare cases, clinical signs regress by day 10 and recovery occurs by day 20–25

Peracute form

- No prodromal signs except high fever (>40–42°C), sometimes congested mucous membranes, And death within 2–3 days .
- This form occurs in highly susceptible young and newborn animals

Mild subacute or endemic form

- Clinical signs limited to one or more of the classic signs
- Usually no associated diarrhoea
- May show a slight, serous, ocular or nasal secretion
- Fever: variable, short-lived (3–4 days) and not very high (38-40°C)

- No actual depression; animals may continue to graze, water and trek
- Low or no mortality, except in highly susceptible species (buffalo, giraffe, eland, and lesser kudu)

i. in these wild species: fever, nasal discharge, typical erosive stomatitis, gastroenteritis, and Death

Atypical form

- Irregular pyrexia and mild or no diarrhoea

i. fever may remit slightly in the middle of the erosive period, and
ii. 2–3 days later, return rapidly to normal accompanied by a quick resolution of the mouth Lesions, a halt to the diarrhoea and an uncomplicated convalescence

- The lymphotropic nature of RPV leads to immunosuppression and favours recrudescence of Latent infections and/or increased susceptibility to other infectious agents

Transmission

The spread of RPV is most often due to introduction of live infected Animals into RP-free areas (Losos, 1986). Exposure of healthy, susceptible Animals to infected droplets, either in the breath of a sick animal or in its Virus-rich secretions or excretions, leads to infection, thus close contact Between infected and susceptible animals is essential for successful Transmission.

Although the precise site of entry of RPV field strains to the animal

Body in natural infection is not defined, the bulk of evidence indicates that Infection occurs principally by inhalation (Provost, 1958; Scott,

1964;Plowright, 1965). Ingestion of food contaminated by the discharge of animals Showing clinical or subclinical infection or are incubating the disease may Also be an important mode of infection especially in pigs (Scott et al, 1959).However, it is acceptable that successful transmission by oral routes in Species other than wild and domestic pigs is erratic (Losos, 1986). Cattle Drenched experimentally with high virulent RPV-infected material failed to Induce infection. Scott et al, 1959 suggested that this might be attributed to The intact skin or to the stratified squamous nature of the epithelium of the Gut.

Among different animal species, cattle can transmit infection to sheep And goats (Zewart and Macadam, 1967). Infected goats, however, can Transmit infection to susceptible cattle (Macadam, 1968) while sheep can't (Plowright, 1952). Nonetheless, contact transmission among experimentally Infected sheep occurs but rarely. Camels could acquire infection from sick cattle although it was not Possible to transmit the virus from infected camels to either susceptible Camels or cattle (Taylor, 1968). In spite of rare exceptions, it is doubtful that recovered animals act as Carriers for more than a few days (Joshi etal, 1984). It appears, however, That ability of domestic pigs to acquire infection relatively easily by ingesting Uncooked contaminated meat is an important factor in the transmission of the Vims (Losos, 1986).

Pathogenesis

Rinderpest virus field isolates affect both lymphoid and epithelial Cells and they have a very high affinity to those of the upper respiratory and Alimentary tract (Thiery, 1965a; Bansal and Joshi, 1979; Blood and Radostits, 1989). In cattle infection occurs readily via the respiratory tract (Plowright, 1964; Liess and Plowright, 1964). The virus passes through

the Epithelium of the upper and less frequently, the lower portions of the tract to Establish foci of proliferation in the draining lymph nodes from which it Disseminates via the blood to other lymphoid tissues. In the blood, the virus Is intimately associated with the leucocytes and only small portions being Free in the plasma rendering it filterable (Losos, 1986).

The course of infection was found to have four main phases namely; Incubation, prodromal, mucosal and convalescence (Plowright, 1968). A Fifth phase following the mucosal and proceeding the convalescent period Was described by Scott (1981).

The incubation period in natural infection varies between 3 and 9 days Depending on the virus strain, the dose and the route of infection while Experimentally it may be as short as 1 to 4 days (Losos, 1986). Virus strains That induce long incubation periods, unlike those with short incubation Periods, cause mild infections and low spreading outbreaks. Viraemia, which Is a characteristic of the incubation phase, begins on a slow rising manner on The second and third days of infection (Liess and Plowright, 1964) and Generalization occurs by the end of the incubation period when the virus Becomes established throughout the gastrointestinal tract. Taylor et al., (1965)identified low-grade viraemia in one experimental animal indicated By the recovery of the virus from the spleen on the 7n Day of infection. Using tissue culture techniques, it was found that RPV proliferated in The pharyngeal and submaxillary lymph nodes and in the palatal tonsils by The 3rd Day of infection and relatively higher virus titres were demonstrated in The mucosae of the upper respiratory tract (Taylor et al., 1965). Some Experimental animals, nevertheless, showed highest virus titres in their Bronchial or costocervical lymph nodes indicating involvement of the lower

Respiratory tract as well. On the other hand, no infectivity could be 'demonstrated in the mucosae of bronchi or lung parenchyma associated with These lymph nodes reinforcing the presumptive fact that the virus passes Through the mucosae without proliferating or producing local lesions (Bedson and Duckworth, 1962). Contrary to this, Plowright (1965) did Observe proliferation of the virus in the lung and the bone marrow as well. The prodromal phase is the period between the onset of fever and the Appearance of mucosal lesions in the mouth; it usually lasts 5 to 7 days. During this phase high virus titres were detected in the lymphoid tissues of The gastrointestinal tract. The virus appeared in the trubinate mucosa on the 5" day of infection and high titres were detected in the lungs towards the end Of this period. Fever, the characteristic feature of this phase, occurred after The virus had been disseminated through the body and was associated with The release of virus and tissue breakdown products into the circulation. Body Temperature of infected animals may reach 40.5 to 41.5°C in some epizootics. Series of variable symptoms may be encountered during the course of The disease depending on the virulence of the virus strain and the Susceptibility of the host. Multiple mixed infection aggravates the clinical Illness of RP (Blood and Radostits, 1989). The disease may take different Forms, a peracute form in which death ensues one to two days after Appearance of clinical signs, an acute form in which symptoms last four to Seven days or a mild form in which the animal may live up to three weeks or Longer (Losos, 1986). Hypovirulent strains which cause only 10% Symptomatic infection in cattle were known.

In general the symptoms of the prodromal phase are drop in milk Yield in lactating cows, staring coat, depression or restlessness. Sometimes there is a clear serous or seromucoid conjunctival or /and nasal discharge.

Inflammation of the conjunctivae, buccal and nasal mucosae follow and there May be hyperaemia of the vaginal mucosae and swelling of the vulva. Lacrimation and salivation become profuse and blepharospasm, which is an Uncommon feature in RP, may be seen in some cases. Patchy superficial Necrosis and erosion of the epithelium inside the nares as well, can be seen in Some animals. Muzzle becomes dry and animals are commonly constipated. The mucosal phase, which extends between eight to 12 days, is Identified by appearance of the mouth lesions when the temperature drops And diarrhoea starts. Discrete, slightly raised grayish necrotic lesions (1 to 5 Mm in diameter) develop leading to stomatitis. The involvement of the Dorsum of the tongue is also common (Liess and Plowright, 1964). Similar Lesions are seen on the nasal, vulval and vaginal mucosae. Necrosis may be Widespread inside the nares and on the turbinates, often allied with secondaiy Bacterial infection. The respiratory involvement are represented by laboured, Painful delayed expiration accompanied by grunting. Diarrhoea usually Appears on the 4^{th} To 7U Day of reaction with mucus and/or streaked with Blood and of foetid odour. Tenesmus is common. Death ensues due to Dehydration.

In enzootic areas where resistance to infection is high, subacute and Skin forms are common (Blood and Radostits, 1989). In the subacute form Temperature reaction is mild (Liess and Plowright, 1964). The animal shows Low degree of anorexia and malaise and the inflammation is catarrhal without Dysentery (Maurer et al, 1955, 1956). The skin form on the other hand, is Rare and is characterized by erythematous thickening of the skin (Mares,1956; Thiery, 1956a). Noticeably, the quantity of the virus in tissues usually Drops rapidly during viraemia. Signs and lesions similar to those of cattle Develop in sheep and goats infected with RPV skin form. In the convalescent phase, which extends between 13 to 16

days, Resolution of the mouth lesions is of dramatic speed. The necrotic deposits of The epithelia begin to clear by the 3rd To 5th Days following their appearance (Plowright, 1964) and complete healing occurs within 48 hours. However, With exceptions, some buccal papillae often remain reddened and eroded up To the end of the second week following the first clinical reaction. Contrary to the findings of Plowright (1964) and Liess and Plowright (1964) in which Complete clearance of the virus in cattle was observed between days 9 and 10 Following first reaction, Scott (1955) could recover the old laboratory Kabete ' 0 ' strain from the spleen and lymph nodes of infected cattle 16 days Postinfection.

Differential diagnosis

For presumptive diagnosis RP infection should be differentiated from PPR, bovine viral diarrhoea (BVD) and mucosal disease (MD), malignant Catarrhal fever (MCF), blue tongue (BT) and foot and mouth disease (FMD). To this end AGID, CIEF, VN, immunocapture ELISA, c-ELISA and RT-PCR are used for differential diagnosis (Diallo et al., 1995).

Vaccination

Vaccination against RP confers solid lifelong immunity (Plowright,1957) and all age groups of animals respond to vaccination. However, calves Born of immune dams should not be vaccinated under three months of age (Brown, 1958). On the other hand, the impact of some concurrent infections May, however, cause vaccination failure (Twinamasiko and Kakaire, 1994).Several vaccines have been used while others are under trials. These include:

1. The caprinized vaccine: it was a goat – adapted RPV vaccine (Provost et Al., 1958) used in Zebu cattle and in areas where a degree of natural Immunity is not anticipated (Blood and Radostits, 1989). The vaccine is Sufficiently virulent to produce undesirably severe reactions, particularly In calves, buffalo and British breed of cattle.
2. The lapinized vaccine: it was the Nakamura III rabbit-adapted RPV strain (Nakamura et al., 1955). The vaccine had been attenuated and used for Zebu-type breeds, in which a solid immunity for two years was induced. It was claimed that the vaccine strain could be maintained and Transported in rabbits where there is no refrigeration (Blood and Radostits, 1989).
3. A lapinized-avianized vaccine: has been adapted and used in pigs (Losos,1986).
4. The avianized vaccine: though cheap, stable and liable for varyingDegrees of attenuation, the cultivation process is tedious (Provost et ah,1961). The immunity produced by this vaccine lasts for at least 16 Months.
5. The measles vaccine: this vaccine protects calves against RP at an age When ordinary RP vaccines are ineffective (Blood and Radostits, 1989). The vaccine is also effective in adult cattle.
6. Cell-culture vaccine (CCV): this was a virulent Kabete '0 ' strain that had Been attenuated to produce a vaccine (Plowright and Ferris, 1959). The Vaccine is cheap, easy to be produced, can be freeze-dried and safer than All other RP vaccines in all situations (Blood and Radostits, 1989). The Vaccine is administered sc, however, intranasal (in) trails were conducted (Provost et al., 1972). Different combinations of CCV with other Vaccines are

avialable (Kathuria et al, 1976; Jeggo et al., 1987), but their Usage in Africa has not been successful and therefore not recommended.

7. The thermostable vaccines: these include:
   a. Mariner's ultra-dry vero-cell derivative of Plowright strain (Mariner's-RBOK-BK-VERO): the vaccine is characterized by thermostability And vials of the vaccine were found to be potent after exposure to 30°C for 30 days (Mariner et al., 1990).
   b. PARC thermovax RP vaccine: it is heat-stable clones of Plowright vaccine virus selected at Internationale d'elevage et de medecine veterinaire (IELMVT) and has been incorporated by PARC (Scott and Provost, 1992). Both vaccines are now under trial in some tropical countries.
8. Inactivated vaccines: many inactivated RP vaccines were produced (Blood and Radostits, 1989), however, the duration of immunity they Elicited is short.
9. Recombinant (subunit) vaccines: these are regarded as the future Vaccines. The RP-vaccina (Yilma, et al., 1988) and capripoxviruses (Romero et ah, 1993; Ngichabe et ah, 1997) recombinant vaccines are Under field trials. Nonetheless, other recombinant vaccines namely; a Chimaeric RPV/PPRV vaccine (Bulletin of Institute for Animal Health, UK, 1997c) and a reporter RP-vaccinia recombinant vaccine (Barrett et Al., unpublished data) are on their way to be tested.

Chapter Three (Conclusions)

In the light of these findings, the Joint Committee concluded the following:

i. Rinderpest as a freely circulating viral disease has been eliminated from the World; and

ii. The presence of virulent or attenuated rinderpest virus in laboratories consti-Tutes a potential threat to global biosecurity.

Recommendations

1) A resolution should be taken forward by FAO and OIE, for adoption by their Governing bodies, declaring global rinderpest eradication and implementing Subsequent necessary measures.
2) Guidelines on rinderpest virus sequestration as agreed by the Joint Committee In consultation with the OIE Biological Standards Commission should be imple- Mented by national veterinary authorities, OIE and FAO.
3) FAO and OIE should, as a matter of urgency, continue to work in close collabo-Ration on the following:
    a. Develop a strategic plan to guide the post-eradication activities at interna-Tional level;
    b. Complete an analysis of the risks of re-emergence of rinderpest virus, and its Consequences;
    c. Prepare an international contingency plan based on the risk analysis;
    d. Set up a joint FAO/OIE Advisory Body on rinderpest, defining its terms of ref-Erence and membership; this Advisory Body may set up subcommittees, for Example to monitor rinderpest research activities.

4) National veterinary authorities should update national contingency plans in Line with the guidelines for rinderpest virus sequestration and the international Contingency plan.
5) FAO and OIE should establish an appropriately funded mechanism for over-Sight and approval of facilities holding rinderpest virus containing material, in Conjunction with national regulatory authorities and, where appropriate, with Other international organizations.
6) FAO and OIE should maintain archives of existing documents (including disease Status dossiers); digitization of files should be considered where possible, as well As identification of documentation that should be made publicly accessible.

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