A STUDENT HANDBOOK ON SLOW VIRUS INFECTIONS AND PRION DISEASES IN DOMESTIC ANIMALS

By

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PREFACE

The book has been constructed to highlight on the various important issues related to slow virus diseases and prion infections in dometic animals. The book provides an overview on the relevant area of focus. The author also duly acknowledges the various researches as carried out by the investigators worldwide on the related issues as discussed in this text.

Dr. Subha Ganguly

Satarupa Roy

DEDICATION

This book is dedicated to the Students of Veterinary and Animal Sciences and has been composed exclusively for providing firsthand knowledge on the related issues for the development of science, education and technology. I also want to express my indebtnesss towards my Parents and family members for their constant encouragement in preparing this Book.

Dr. Subha Ganguly

Satarupa Roy

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Chapter 1: Slow Virus infections

Introduction

Different members of retroviruses infect human beings, cattle, cats, primates, rodents and avian species. The family has 6 genera out of which only two genera i.e. *Lentivirus* and *Spumavirus* have been recognized officially. All the viruses of veterinary importance are grouped under type C group of retroviruses. Lentiviruses differ from retroviruses in structure and genome. The envelope glycoprotein epitopes are group specific (encoded by gene *gag*).

Genus Lentivirus

Properties of Visna and Maedi viruses

Maedi and Visna diseases appear to caused by the same or very closely related lentiviruses (lentus meaning slow). The virus is non-oncogenic and exogenic. The agents isolated from Maedi (causing dyspnoea) and Visna (causing Wasting disease) are similar morphologically and biologically. Maedi virus produces pneumotropic and visna neurotropic lesions in sheep and goats respectively. The virus is not sensitive to interferons. Both agents are antigenically similar. The mutation rate of these viruses is very high. There is considerable antigenic drift I n both the viruses.The nucleocapsid of these viruses is cylindrical type. Visna and maedi viruses share nucleic acid sequences. These viruses replicate in sheep cell cultures causing syncitial type of CPE followed by cellular degeneration.

The poor immune response in Lentivirus infgections is due to death of T-helper cells, death of antigen presenting cells, reduced lymphokine production, production of suppressor factors and due to genetic variation afeecting viral epitopes.

Pathogenesis of maedi in sheep:

Maedi and maedi-like viruses have been reported from several countries in Europe and USA in sheep. In some countries it is known as ovine progressive pneumonia. The disease occurs in 3-4 years old sheep. Early symptoms of the disease are loss of condition and increased respiration. Later on there is dyspnoea, head may be extended with each respiration, nostrils are dialated and there is slight nasal discharge and cough. Experimentally, incubation period is about 2 years in sheep. The course of the disease is 4-6 months and the animal may survive for about a year. Generally, there is secondary bacterial pneumonia. Microscopically, the lungs become enlarged and diffusely thickened. In advanced cases, the lung alveoli are blocked. There may be arthritis and lymphoid tumors in mammary glands.

Pathogenesis and clinical symptoms of visna in sheep and goats:

Visna virus causes meningoencephalitis, a chronic progressive neurological disease in sheep and goats affecting CNS. In nature, the virus affects sheep below two years of age. The incubation period after intracerebral inoculation in sheep and goats is several years and clinical symptoms occur

in 2-6 years. The disease starts with weakness of hips and hind limbs with difficulty in walking. There is progressive weight loss, trembling of facial muscles, paralysis and death.

The course of the disease is few months to several years. The virus persists in infected animals even though there is cellular and humoral immune response. Isolation of the virus is possible during initial stage from leucocytes, brain and CSF. There is continuous mutation of the virus. The viral antigen may be absent due to stoppage of replication of virus beyond p[roviral stage. The virus can be recovered from milk of infected ewes. Ther is no indication of vertical transmission. Transmission of the virus is through contact, by inhalation and by ingestion of contaminated faeces and other materials.

Clinical symptoms of caprine arthritis an encephalomyelitis:

Arthritis occurs in goats over one year old and encephalomyelitis in kids of 2-4 months age. In older goats, encephalomyelitis progresses slowly in months or years. Hock and shoulder joints are swollen and become painful. The disease becomes more severe in winter months. Thickening of shoulder joints make movement difficult. There is synovitis of joints with synobvial cell hyperplasia and infiltration of lymphocytes, plasma cells and macrophages. There may be mild interstitial pneumonia. Kids are infected by feeding on infected colostrums an milk.

The symptoms of encephalomyelitis in young kids are progressive weakness, trembling and in the later stage paralysis. Affected goats show progressive wasting. There is no fever and the animal maintains good appetite and eye sight.

Antibodies are detected by indirect FA, ID and ELISA tests.

Pathogenesis of equine infectious anaemia:

The disease is also known as swamp fever because it mostly occurs in low lying humid and swampy areas where insects are found in large numbers and transmits the disease by biting the horses. The arthropod vectors are tabanid and stable flies and mosquitoes. The incubation period of the disease is 2-21 days.

The symptoms include recurrent fever, anaemia, jaundice, anorexia, progressive weakness, loss of weight and oedema. In acute and subacute cases, there are haemorrhages and enlargement of spleen, lymph nodes and kidneys. There are peticheal haemorrhages on mucous membranes. There is decrease in PCV, blood platelets and RBC count. Tissue macrophages and other cells carry the virus. Cell associate viraemia persists during the entire course of the disease which continues lifelong. There is successive antigenic variation in the virus. The virus has number of antigenically distinct strains.

The disease can spread vertically by transplacental infection and by horizontal mode by consumption of infected materials. Isolation of the virus can be made by citrated blood, serum or leucocytes or in primary equine leucocyte cultures.

The disease can be diagnosed by history, clinical symptoms, blood examination and by pathological lesions. Antibodies in the infected animals can be detected by gel diffusion test also known as Coggin's test. Another reliable test is by inoculation of serum or whole blood from infected horse to uninfected susceptible horse.

Chapter 2: Prions

Prions

Prions are transmissible agents causing degenerative changes in central nervous system. They are composed of glycoproteins but do not contain nucleic acids. Prions are capable of multiplication but do not evoke any immune response. They cause slow virus diseases having very long incubation period. The diseases are characterized by degenerative neurological changes. Prions are also known as atypical or unconventional viruses. They are sensitive to proteases. They are proteinaceous infectious particles and cause diseases like scrapie in sheep.

Prions cause diseases which are slow and having prolonged incubation periods:

(a) Scrapie disease in sheep
(b) Bovine spongiform encephalopathy.
(c) Transmissible mink encephalopathy.
(d) Wasting disease of mule deer.
(e) Mad cow disease
(f) Kuru disease in humans.
(g) Creutdzfeldt-Jacob disease in humans.

Properties of agents causing scrapie disease in sheep:

Scrapie is a slow progressive disease of CNS of sheep and occasionally of goats. It is caused by filterable agent. It is resistant to boiling, UV irradiation, formalin treatment and exposures to strong acids and alkalies, but is susceptible to phenol and ether. It is not affected by nucleases. The agent is intimately associated with host cell components. There are two distinct scrapie disease agent. The agent has no nucleic acid but has protein which may be drawn from host.

Pathogenesis of scrapie disease in sheep:

The incubation period of the disease is 2-3 years naturally and 6-9 months intracerebrally after experimental inoculation. The disease infects sheep and goats of 3 years old. Infection in sheep by contact is uncommon but spreads by vertical transmission. The infected animals show nervous signs, staggering gait and then unable to move and die after some months. There is irritation on the skin which leads to rubbing against a post. There is spongiform degeneration nd vacuolation of cytoplasm. The agent in mice is maximum in spleen after intracerebral inoculation. Diagnosis of the disease is made with infected brain materials. Diagnosis cannot be made on the basis of serological and virological procedures.

Bovine spongiform encephalopathy (BSE):

This disease was first recognized in Great Britain in 1986. Majority of the cases were reported in dairy cows. The symptoms and lesions of the disease are similar to that of scrapie. Diagnosis of the disease is difficult. Confirmation is made by examination of sections of infected brain tissues. For control measures, the infected animals are recommended for slaughter. The agent is very resistant to sterilization by different means. BSE epidemics have raised public concern about human health hazards.

Transmissible mink encephalopathy:

The disease affects the minks by feeding the contaminated and infected sheep meat. The symptoms and lesion of the disease resemble that of scrapie.

Mad cow disease:

The disease was reported in England among the cattle population in 1999-2000. It appears to be similar to BSE. Slaughter of infected cows and prohibition of beef export is the control measure to be adopted during its outbreak.

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