Research Paper

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Kyasanur Forest Disease - an Emerging Threat in Kerala

Medical Science

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ABSTRACT

Kysanur Forest Disease (KFD), The monkey born zoonotic disease is becoming an emerging threat in the land of kerala as there was few reports on the incidence of this disease both in monkeys and human being in years 2013 and in the ongoing year 2014. This article is a briefing about the epidemiology, clinical manifestations, necropsy findings, diagnostic methods, treatments and preventive measures of this disease.

INTRODUCTION

KFD is also referred to as monkey fever. It is an infectious bleeding disease in monkey and human, caused by highly pathogenic virus called KFD virus (KFDV). KFDV is zoonotic in origin belongs to family Flaviviridae and it is transmitted primarily by infective tick, Haemphysalisspinigera. KFDV is an enveloped spherical virion particle and the genome is made of single stranded positive sense RNA (Banerjee, 1988). KFDV is ranked as a high risk category pathogen requiring Biosafety level -4 handling.

EPIDEMIOLOGY

KFDV is enzootic to India and maintained in ticks, mammals, and birds. It causes febrile illness in humans and was first recognized in in 1957 in Kyasanur Forest of Shimoga District, Karnataka State, India (Upadhyaya et al., 1957). The virus has been isolate dromnatuarally infected Semnopithecus entellus (langur), Macaca radiate (bonnet monkey), Rattusblanfordi, Rattusratus(rat)Suncusmurinus(shrew) and a bat Rhinolophusrouxi. Neutralizing antibodies have been found in cattle, buffaloes, goats, wild boars, porcupines, squirrels, rats, mice and a number of bird species. The first epidemic season of KFD in human was observed in Jan-May 1954 when four villages were affected in Karnataka.

In Kerala the reported cases are as follows, two incidences of human infections are there one case from Noolpuzha of Wayanad district in May 2013 and three case from Nilambur of Malappuram disrtict in June 2014. KFDV infection in monkeys are reported from Mannar, Alappuzha District in April 2014.

TRANSMISSION

Large species of animals are thought to be reservoir hosts for the disease. Rodents, shrews, monkeys and birds upon tick bite become reservoir for this virus. The vector for disease transmission is Haemaphysalis spinigera, a forest tick (Varma , 2001). Humans get infection from the bite of nymphs of the tick. Human is dead end host in the natural cycle of the virus. Deforestation, subsequent cattle grazing in those areas and the low susceptibility of cattle for KFDV lead to conclude that cattle is the large mammal reservoir for vector maintenance and propagation.

CLINICAL SYMPTOMS

In monkeys: KFDV infection causes severe febrile illness ,haemorrhagic enteritis (Parvi, 1989). When infected monkeys die, ticks drop from the body, thereby generating hotspots of infectious ticks that further spred the virus. In enzootic areas, KFDV circulates through small mammals (rat, shrews, ground birds) and ticks.

In humans cases were found among persons who visited forests to collect firewood, grass, other forest products and cattle graz-

ers. Clinically, Human disease us characterized by an incubation period of 3-8 days, followed by sudden chills, high fever, frontal headache, heightened sensitivity to light and continuous fever for 12 days or longer often associated with diarrhea, vomiting, cough, severe pain in neck, low back and extremities, accompanied by severe prostration. Papulo-vesicular eruption of the soft palate (blisters on upper, inner mouth) is an important diagnostic sign in some patients. Bleeding signs such as in the gum, nose (epistasis), cough (hemoptysis), gastrointestinal bleeding resulting in dark feces (melena) and fresh blood in the feces are common. The convalescent phase constituting the recovery after KFD's onset is generally prolonged, may be up to 4 weeks. Relapse of the symptoms, often observed after 1 to 2 weeks of the first febrile period, lasts for 2 to 12 days and a case fatality rate is more than 30%. During infection by KFDV , virus titre remains high for 10 days after onset of symptoms, as reported by Bhat et al. (1991). However , Upadhyaya et al. (1975) found that viremia in patients lasted for 12-13 days of illness and unlike most other Flavi viruses, remains high during the first 3-6 days. Leucopenia and accompanying thrombocytopenia are constant haematological features in KFD. Intra-alveolar haemorrhage, resulting into secondary infection and massive gastrointestinal haemorrhages are terminal complications that could lead to death. (Devendra et.al.,2013)

POSTMORTEM FINDINGS

In monkeys gross findings are blood clots in the anus, hemorrhage in lungs, moderate swelling and pallor of the renal cortex , brain, and adrenals. Non purulent encephalitis with focal microgliosdis, perivascular cuffing are the common lesions in brain. Anal hemorrhage, pallor of adrenal cortex, focal liver necrosis with cytoplasmic inclusion bodies, necrosis in small and large intestines are common (Adhikari et.al 1993). Histologically liver shows focal hepatocellular degeneration, fatty changes, necrosis, degenerative changes in central and midzonal cells, including vacuoles and pigments with the presence of eosinophil cytoplasmic inclusions. In the kidneys there were marked degenerative changes in the tubules. Pulmonary hemorrhage, depletion of malphigian follicles, sinus histocytosis, erythrophagocytosis, mild myocarditis,and encephalitis are the prominent lesions. Phagocytosis of nuclear material and red blood cells are present in the peripheral blood. An increase in the nuclear debris is also seen in the lymph glands of some infectde monkeys. In humans gross findings are pallor of the liver, kidneys, adrenals and brain. Degenerative changes in liver, kidney with mild myocarditis and encwphalitis. PPattnaik, 2006).

DIAGONOSIS

Diagnosis is primarily syndromic and serological. Being a very stable virus in the blood, the diagnosis is made by virus isolation from blood or by serologic testing using ELISA, (Mouryaet .al, 2012). Other serological tests include HA, CFT, virus neutralization test and mass tag PCR.

TREATMENT

There is no specific treatment for KFD, but supportive therapy , includes analgesics and antipyrectics, intravenous fluids for those with hypotension, blood transfusion or fresh –frozen plasma and platelets for thos4 with hemorrhagic symptoms, antibiotics for bronchopneumonia, and corticosteroids and anticonvulsants for neurological symptoms (shellaberger,1991). A formalin inactivated KFDV vaccine produced in chick embryo fibroblasts has been licensed and currently in use in the endemic areas in Karnataka state and shows effective prevention (Gudadappa et.al.,2013).

PREVENTION AND CONTROL

A timely supportive therapy , such as careful precautions for patients with bleeding disorder and maintenance of hydration is important and reduces KFD mortality in humans. Prophylaxis by vaccination, as well as preventive measures like protective clothing, tick control, and mosquito control atre advised. An attenuated live vaccine is now available.

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