KYASANUR FOREST DISEASE (KFD) / Monkey Fever

Kyasanur Forest Disease (KFD) is a serious zoonotic disease, mostly found in southern India. The disease was first reported in 1957 from Shimoga district, Karnataka, which is a primitive sylvan territory in Western Ghats of India. Monkey deaths and human cases have now been reported from three neighbouring states bordering Karnataka viz., Wayanad (2013) and Malappuram districts of Kerala (2014), North Goa district of Goa state (2015) and Sindhudurg district of Maharashtra (2016).

It is also known as "**Monkey Disease / Monkey Fever**" because of its association with monkey deaths.

Etiology:

Caused by KFD virus (KFDV), which belongs to the family of ***Flaviviridae*** and the genus ***Orthoflavivirus*****.** Hard ticks (*Hemaphysalis spinigera*) spread the KFD virus to animals and humans. Monkeys are primarily affected by KFDV.

Affected Specie / Transmission:

Tick-borne: The primary vector for KFDV transmission is the hard-bodied tick ***Haemaphysalis spinigera***

Amplifying hosts: The wild primates most susceptible to fatal infection are the black-faced Hanuman langur (*Semnopithecus entellus*) and the red-faced bonnet macaque (*Macaca radiata*).

Hotspots: When an infected monkey dies, ticks usually drops-off from its body, creating "hotspots" of highly infectious ticks on the forest soil. Humans can get the disease from an infected tick bite or by contact with an infected animal.

Incubation Period: 3 to 8 days after the bite of an infective hard tick.

Clinical Signs:

High fever

Prostration (extreme exhaustion or collapse)

Weakness and lethargy

Watery or bloody diarrhea (hemorrhagic enteritis)

Dehydration and thirst

Bleeding problems (hemorrhagic diathesis)

In terminal stages, monkeys may exhibit photophobia and cyanosis.

Lesions:

Significant systemic lesions includes, Focal hemorrhages in various tissues, extensive hemorrhage within the intestinal wall and necrosis (tissue death) of intestinal epithelial cells, necrosis in the liver and kidneys (parenchymatous organs) and widespread necrosis in lymphoid tissues.

Widespread damage to blood vessel walls is a common feature, which leads to increased vascular permeability, hemorrhages, and disseminated intravascular coagulation (DIC).

In few cases nervous involvement is apparent and lymphohistiocytic meningoencephalitis is observed.

CAPTURE MYOPTAHY (in Deers)

(exertional myopathy, or exertional rhabdomyolysis)

Capture myopathy (CM) is a noninfectious, metabolic disease of wild and domestic animals that can lead to significant morbidity and mortality. The condition is most commonly associated with pursuit, capture, restraint, transport, secondary to other diseases and natural hazards encountered in the environment. It is characterized by **metabolic acidosis**, **muscle necrosis**, and **myoglobinuria**. The condition is nowadays most commonly referred to as CM, exertional myopathy, or exertional rhabdomyolysis

Classically, the initial clinical signs observed in animals suffering from capture myopathy are anxiety, shivering, rapid breathing, bent neck (*torticollis*), dark red urine and hyperthermia. Subsequently to these, muscle stiffness, severe muscle pain, ataxia, paresis, torticollis, prostration, and paralysis are observed. Animals typically become obtund, anorexic, and unresponsive.

Exertional rhabdomyolysis, or CM, in animals is distinguishable from other types of rhabdomyolysis by its pathophysiology, as it affects both **skeletal and cardiac muscles** in response to extreme stress and muscular exertion. Stresses of FEAR and ANXIETY are the triggering mechanisms and involvement of sympathetic nervous and adrenal systems, and exaggerated muscular activity is noticed in most cases.

Muscular Lesions are characterized by acute rhabdomyolysis. Myocytes are markedly swollen, with loss of striations and fragmentation and cleavage of myofibrils. Sarcolemmal nuclei are pyknotic in multiple areas.

CM diagnosis relies strongly on the animal's natural and individual history as well as any clinical signs that have manifested. Increased CK is the most sensitive and specific index of muscle damage in birds and mammals. CK elevation more than 10 times the upper reference limit, myoglobinuria, hyperkalemia, and coagulopathy are important findings.

NOTE:

In domestic Poultry, a similar manifestation is also reportedly termed as "**Exertional myopathy**" resulting from overly strenuous muscular exercise and can be precipitated by preexisting conditions such as **selenium deficiency**. Inadequate energy metabolism and/or mechanical stresses.

Deep pectoral myopathy (also known as **degenerative myopathy** or **green muscle disease**) and **capture myopathy** in birds are the main examples of exertional myopathy in birds. Leg muscle myopathy can occur after transport of poultry.

CHRONIC WASTING DISEASE (CWD in Wild Cervids; Deers etc)

Chronic wasting disease (CWD) is a contagious, transmissible and fatal neurodegenerative **disease of captive and free-ranging cervids, including deer, elk, moose, and reindeer**. It is a member of the transmissible spongiform encephalopathy (TSE) family of diseases, or prion diseases, that includes bovine spongiform encephalopathy; scrapie of sheep and goats; transmissible mink encephalopathy; and kuru, Creutzfeldt-Jakob disease (CJD), and variant CJD of humans.

Chronic wasting disease is **transmitted Horizontally as well as Vertically.**

The main SYMPTOMS of CWD, which occur after a long incubation period up to several years, are **significant weight loss, ataxia, and hypersalivation**. Animals with clinical chronic wasting disease are **>16 months old** and initial signs are subtle changes in behavior and weight loss.Loss of wariness, somnolence, persistent walking, polydipsia and polyuria, and hyperexcitability may be seen among main **Behavioral Changes**. **Aspiration pneumonia may be the only presenting clinical sign and is often the cause of death**.

LESIONS from chronic wasting disease are **seen in the GRAY MATTER of the CNS**. Lesions are **bilaterally symmetrical** and anatomically constant among animals. Spongiform appearance is obvious; vacuolization occurs in neuronal perikarya and neuronal processes. Along with neuronal degeneration, astrocytic hyperplasia and hypertrophy may appear.

DIAGNOSIS based upon clinical signs in not reliable. Diagnosis is based on detection of PrP^{Sc} by ELISA or Western blot, with confirmation by Immunohistochemistry. H&E staining of brains of affected animals reveals **amyloid plaques** that appear as pale, fibrillar, eosinophilic areas of neuropil and are sometimes surrounded by vacuoles (florid plaques).